Appendix A: Change notice – Regulation 22

Interest holder	Tamboran B2 Pty Ltd	**EMP Title**	Beetaloo Sub-basin Multi-well Drilling, Stimulation and Well Testing Program EMP	**Unique EMP ID**	ORI10-3	**Mod #**	2	**Date**	25 January 2022
Brief Description	1. Inclusion of the chemical risk assessment by EHS Support on behalf of Condor Energy (Appendix E.1).								
Geospatial files included?	Attached.								

Does the proposed change result in a new, or increased, or potential or actual environmental impact or risk?	**If an INCREASE in the existing potential or actual environmental risk, is it provided for in the EMP?**	**Does the proposed change require additional mitigation measures to be included?**	**Has additional stakeholder engagement been conducted?**	**Does it require additional environmental performance standards and measurement criteria?**	**Does it affect compliances with Sacred Site Authority Certificates?**	**Does it affect current rehabilitation, weed fire, wastewater, erosion and sediment control, spill or emergency response plans?**	**Will the environmental outcome continue to be achieved and will the impacts and risks be managed to ALARP and acceptable?**
No. Well construction, operation, maintenance and management is covered by the Well Operations Management Plan (WOMP) and Part B of the Code. There are no new or increased environmental impacts or risks through the addition of the new chemical suite.	N/A No increased impact or risk with sufficient design details included in the EMP and WOMP.	No. Existing mitigation measures are in place covering chemical risk assessment, including spill and wastewater management.	N/A Additional stakeholder engagement is not required, as the use of chemicals has previously been communicated with stakeholders.	No. Environmental performance standards within the existing approved EMP are sufficient .	No. Amendment to AAPA Certificates C2022/02 and C2020/003 is not required.	No. Tamboran operates all sites under a suite of management plans which contain common elements for all operating sites. Existing plans have been updated and remain valid and appropriate to cover any potential risk covered under the proposed modification, in alignment with the existing approved EMP.	Yes. There will be no material change to this activity, as expected volumes of chemicals, waste and wastewater management remain within the ranges outlined in the approved EMP. Mandatory groundwater monitoring required by the Code as outlined in *Table 31 Monitoring program summary*, will be met.
Additional contextual information	Provides Tamboran greater flexibility around service provider for hydraulic fracturing and stimulation activities.						

Interest holder	Tamboran B2 Pty Ltd	EMP Title	Beetaloo Sub-basin Multi-well Drilling, Stimulation and Well Testing Program EMP	Unique EMP ID	ORI10-3	Mod #	2	Date	25 January 2023

Current EMP text

Executive Summary

Table 4 Chemicals that may be added to the sand proppant during stimulation

Material name	Typical volume	Maximum volume	Unit	Storage area
Acetic Acid - 60% PH control	3,000	6,000	L	Stimulation chemical storage area
BE-9 Biocide	17,000	34,000	L	Stimulation chemical storage area
Caustic Soda Liquid pH control/ buffer	15,000	30,000	L	Stimulation chemical storage area
DCA-11001 Breaker Activator	5,000	10,000	L	Stimulation chemical storage area
DCA-13002 Breaker	300	600	kg	Stimulation chemical storage area
DCA-13003 Breaker	10,000	20,000	L	Stimulation chemical storage area
DCA-16001 Clay Stabiliser	42,000	84,000	L	Stimulation chemical storage area
DCA-17001 Corrosion Inhibitor	1,000	2,000	L	Stimulation chemical storage area
DCA-19001 Crosslinker	600	1,200	kg	Stimulation chemical storage area
DCA-19002 Crosslinker	10,000	20,000	L	Stimulation chemical storage area
DCA-23001 Friction Reducer	5,000	10,000	kg	Stimulation chemical storage area
DCA-23003 Friction Reducer	18,000	36,000	L	Stimulation chemical storage area
DCA-25005 Gelling Agent	35,000	70,000	kg	Stimulation chemical storage area
DCA-30001 Scale Inhibitor	15,000	30,000	L	Stimulation chemical storage area
DCA-32002 Surfactant	15,000	30,000	L	Stimulation chemical storage area
DCA-32014 Surfactant	200	400	L	Stimulation chemical storage area
FE-2 Buffer	200	400	kg	Stimulation chemical storage area
Hydrochloric Acid - 32%	50,000	150,000	L	Stimulation chemical storage area
100 Mesh Sand- Proppant	91,000	182,000	kg	Stimulation chemical storage area
40/70 Sand- Proppant	1,650,000	3,300,000	kg	Stimulation chemical storage area
30/50 Sand- Proppant	610,000	1,220,000	kg	Stimulation chemical storage area

Amended EMP text

Executive Summary

Table 4 Chemicals that may be added to the sand proppant during stimulation

Material name	Typical volume	Maximum volume	Unit	Storage area
Acetic Acid - 60% PH control	3,000	6,000	L	Stimulation chemical storage area
BE-9 Biocide	17,000	34,000	L	Stimulation chemical storage area
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DCA-11001 Breaker Activator	5,000	10,000	L	Stimulation chemical storage area
DCA-13002 Breaker	300	600	kg	Stimulation chemical storage area
DCA-13003 Breaker	10,000	20,000	L	Stimulation chemical storage area
DCA-16001 Clay Stabiliser	42,000	84,000	L	Stimulation chemical storage area
DCA-17001 Corrosion Inhibitor	1,000	2,000	L	Stimulation chemical storage area
DCA-19001 Crosslinker	600	1,200	kg	Stimulation chemical storage area
DCA-19002 Crosslinker	10,000	20,000	L	Stimulation chemical storage area
DCA-23001 Friction Reducer	5,000	10,000	kg	Stimulation chemical storage area
DCA-23003 Friction Reducer	18,000	36,000	L	Stimulation chemical storage area
DCA-25005 Gelling Agent	35,000	70,000	kg	Stimulation chemical storage area
DCA-30001 Scale Inhibitor	15,000	30,000	L	Stimulation chemical storage area
DCA-32002 Surfactant	15,000	30,000	L	Stimulation chemical storage area
DCA-32014 Surfactant	200	400	L	Stimulation chemical storage area
FE-2 Buffer	200	400	kg	Stimulation chemical storage area
Hydrochloric Acid - 32%	50,000	150,000	L	Stimulation chemical storage area
100 Mesh Sand- Proppant	91,000	182,000	kg	Stimulation chemical storage area
40/70 Sand- Proppant	1,650,000	3,300,000	kg	Stimulation chemical storage area
30/50 Sand- Proppant	610,000	1,220,000	kg	Stimulation chemical storage area
Alcohols, C11-14-iso-, C13-rich,ethoxylated- Surfactant	5285	10570	L	Stimulation chemical storage area
Sodium (C14-16) olefin sulfonate - Surfactant	4658	9316	L	Stimulation chemical storage area
Diisobutyl glutarate - plasticiser	627	1254	L	Stimulation chemical storage area
Diisobutyl succinate - plasticiser	209	418	L	Stimulation chemical storage area
Diisobutyl adipate- plasticiser	179	358	L	Stimulation chemical storage area
sodium thiosulphate- stabilising agent	4763	9527	L	Stimulation chemical storage area
sodium sulphate stabilising agent	913	1827	L	Stimulation chemical storage area

Interest holder	Tamboran B2 Pty Ltd	EMP Title	Beetaloo Sub-basin Multi-well Drilling, Stimulation and Well Testing Program EMP	Unique EMP ID	ORI10-3	Mod #	2	Date	25 January 2023

Current EMP text	Amended EMP text				
	sodium sulphite stabilising agent	794	1588	L	Stimulation chemical storage area
	Ethylene Glycol- Crosslinker	5112	10225	L	Stimulation chemical storage area
	Choline Chloride- Claystabiliser	10301	20603	L	Stimulation chemical storage area
	Glutaraldehyde- Biocide	14930	29859	L	Stimulation chemical storage area
	Ammonium Sulphate- Breaker	4479	8958	L	Stimulation chemical storage area
	Polyacrylamide- Friction reducer	4479	8958	L	Stimulation chemical storage area
	Sodium polyacrylate- gelling agent	746	1493	L	Stimulation chemical storage area
	Sodium bisulfite- stabiliser	149	299	L	Stimulation chemical storage area
	Alkyl Alcohol- surfactant	149	299	L	Stimulation chemical storage area
	2-Propenoic acid, homopolymer, ammonium salt- biocide	149	299	L	Stimulation chemical storage area
	Potassium persulfate-braker	149	299	L	Stimulation chemical storage area
	2-Ethoxy-naphthalene-surfactant	149	299	L	Stimulation chemical storage area
	Sodium Gluconate- stabiliser	8576	17152	L	Stimulation chemical storage area
	Boric -Crosslinker	4288	8576	L	Stimulation chemical storage area
	Potassium Hydroxide- pH control	10745	21491	L	Stimulation chemical storage area
	Mannanase- Cross linker	2	4	L	Stimulation chemical storage area
	Ammonium Persulphate- breaker	7451	14902	L	Stimulation chemical storage area
	Talc- buffer	384	769	L	Stimulation chemical storage area
	Sodium Bromate- breaker	50441	100881	L	Stimulation chemical storage area
	Hepta sodium phosphonate- Emulsifier	3176	6351	L	Stimulation chemical storage area
	DISTILLATES, HYDROTREATED LIGHT- friction reducer	54231	108462	L	Stimulation chemical storage area
	Guar Gum- Viscocity regulator	15141	30282	L	Stimulation chemical storage area
	Polyoxyethylene nonylphenol ether- surfactant	4466	8933	L	Stimulation chemical storage area
	Quaternary ammonium compounds, bis(hydrogenated tallow alkyl)dimethyl, salts with bentonite- biocide	4466	8933	L	Stimulation chemical storage area
	1,6-Hexanediol- cross linker	447	893	L	Stimulation chemical storage area

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Current EMP text	Amended EMP text

Quartz or Organophilic phyllosilicate- proppant	1084	2167	L	Stimulation chemical storage area
HydroChloric Acid- pH control	44715	89430	L	Stimulation chemical storage area
N-Benzyl-Alkylpyridinium Chloride- pH control	28	57	L	Stimulation chemical storage area
Formic Acid- corrosion inhibitor	38	76	L	Stimulation chemical storage area
Sodium erythorbate- scaler prohibitor	334	668	L	Stimulation chemical storage area
Citric Acid- pH control	15878	31756	L	Stimulation chemical storage area
Acetic Acid- pH control	15878	31756	L	Stimulation chemical storage area
Isopropanol- clay management	83	167	L	Stimulation chemical storage area
Ethoxylated C12-C16 Alcohol - surfactant	57	114	L	Stimulation chemical storage area
Ethoxylated Decanol - surfactant	19	38	L	Stimulation chemical storage area
Cinnamaldehyde- biocide	57	114	L	Stimulation chemical storage area
Ethoxylated Tallow Alkyl Amine - surfactant	9	19	L	Stimulation chemical storage area
Methanol- corrosion inhibitor	2	4	L	Stimulation chemical storage area
Polyacrylamide - friction reducer	49093	98186	L	Stimulation chemical storage area
Polyethylene glycol trimethylnonyl ether - clay manager	87	173	L	Stimulation chemical storage area
Water in Additive- stabiliser	66804	133607	L	Stimulation chemical storage area
Potassium Sorbate Food Grade- corrosion inhibitor	14	29	L	Stimulation chemical storage area
Mannanase (Mannan endo-1,4-beta-mannosidase)- cross linker	2	4	L	Stimulation chemical storage area
Nonoxynol-9- surfactant	9	19	L	Stimulation chemical storage area
2-Ethylhexanol PO/EO polymer- stabiliser	9	19	L	Stimulation chemical storage area
Corn Oil- friction reducer	662	1325	L	Stimulation chemical storage area

3.11.2.1 Results of risk assessment

The results of the chemical hazard and exposure analysis are provided in Appendix E.

A Tier 1 assessment was undertaken on all chemicals except for light petroleum distillate (CAS# 64742-47-8). Certain chemicals (14 from Slick Water, 17 from Hybrid and 15 from High Velocity Friction Reduced) require standard flowback water and wastewater disposal controls to ensure the risk of management is low. These controls are consistent with the requirements outlined in the Code of Practice and summarised in section 6.5 of this plan. It must be noted that none of these chemicals were identified to be persistent and bioaccumulative.

3.11.2.1 Results of risk assessment

The results of the chemical hazard and exposure analysis are provided in Appendix E, Appendix E.1.

A Tier 1 assessment was undertaken on all chemicals except for light petroleum distillate (CAS# 64742-47-8). Certain chemicals (14 from Slick Water, 17 from Hybrid and 15 from High Velocity Friction Reduced) require standard flowback water and wastewater disposal controls to ensure the risk of management is low (Appendix E). These controls are consistent with the requirements outlined in the Code of Practice and summarised in section 6.5 of this plan. It must be noted that none of these chemicals were identified to be persistent and bioaccumulative.

Interest holder	Tamboran B2 Pty Ltd	EMP Title	Beetaloo Sub-basin Multi-well Drilling, Stimulation and Well Testing Program EMP	Unique EMP ID	ORI10-3	Mod #	2	Date	25 January 2023

Current EMP text	Amended EMP text
An assessment of the potential valid environmental and human health exposure pathways is summarised in Table 18. The exposure pathways assessment considered the: • properties of the chemicals. • site setting and physical separation distances between receptors (environmental and human) and the activity as outlined in section 3.1. • lack of protected flora and fauna and high conservation value areas in the vicinity of the activity as outlined in section 4.2. • description of the activity and summary of controls as provided in section 3 and section 6.5. The exposure pathway assessment identified only one partially complete exposure pathway; the on-site release of particulates and vapour during chemical mixing and flowback evaporation. The limited number of valid pathways is consistent with the limited size and duration of the proposed activities. A Tier 2 assessment was conducted on hydrotreated light petroleum distillate, which was classified as a bioaccumulative and toxic substance. As per NICNAS 2017 and DOE 2017 guidance, the Margin of Exposure (MOE) approach was used to assess the health risk to workers. For each occupational activity scenario (i.e. transport and storage, mixing/blending of hydraulic fracturing chemicals, evaporation of flowback and cleaning and maintenance), an MOE was derived by comparing the point of departure (e.g. No Observed Adverse Effects Level [NOAEL]) for long-term health effects from the critical toxicological study to the estimated total human internal dose from all routes of exposure. Based on the calculated MOEs, the chemical is of low concern for workers (refer to individual toxicity profile for further detail). A summary of the Tier 2 risk assessment is provided in Appendix E.	An assessment of the potential valid environmental and human health exposure pathways is summarised in Table 18. The exposure pathways assessment considered the: • properties of the chemicals. • site setting and physical separation distances between receptors (environmental and human) and the activity as outlined in section 3.1. • lack of protected flora and fauna and high conservation value areas in the vicinity of the activity as outlined in section 4.2. • description of the activity and summary of controls as provided in section 3 and section 6.5. The exposure pathway assessment identified only one partially complete exposure pathway; the on-site release of particulates and vapour during chemical mixing and flowback evaporation. The limited number of valid pathways is consistent with the limited size and duration of the proposed activities. A Tier 2 assessment was conducted on hydrotreated light petroleum distillate which was classified as a bioaccumulative and toxic substance (see Appendix E) and glutaraldehyde (CAS number 111-30-8), which is identified in the Tier 1 assessment as having a potential avian wildlife exposure (see Appendix E.1). As per NICNAS 2017 and DOE 2017 guidance, the Margin of Exposure (MOE) approach was used to assess the health risk to workers. For each occupational activity scenario (i.e. transport and storage, mixing/blending of hydraulic fracturing chemicals, evaporation of flowback and cleaning and maintenance), an MOE was derived by comparing the point of departure (e.g. No Observed Adverse Effects Level [NOAEL]) for long-term health effects from the critical toxicological study to the estimated total human internal dose from all routes of exposure. Based on the calculated MOEs, the chemical is of low concern for workers (refer to individual toxicity profile for further detail). A summary of the Tier 2 risk assessment is provided in Appendix E and Appendix E.1.
3.11.1 Chemical types and quantities **Table 16 Anticipated chemical volumes use in the drilling and stimulation process**	**3.11.1 Chemical types and quantities** **Table 16 Anticipated chemical volumes use in the drilling and stimulation process**

Material name	Typical volume	Maximum volume	Unit	Storage area
Acetic Acid - 60% pH control	3,000	6,000L	L	Stimulation chemical storage area
BE-9 Biocide	17,000	34,000	L	Stimulation chemical storage area
Caustic Soda Liquid pH control	15,000	30,000	L	Stimulation chemical storage area
DCA-11001 Breaker Activator	5,000	10,000	L	Stimulation chemical storage area
DCA-13002 Breaker	300	600	kg	Stimulation chemical storage area
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DCA-16001 Clay Stabiliser	42,000	84,000	L	Stimulation chemical storage area
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DCA-25005 Gelling Agent	35,000	70,000	kg	Stimulation chemical storage area

Material name	Typical volume	Maximum volume	Unit	Storage area
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Current EMP text				
DCA-30001 Scale Inhibitor	15,000	30,000	L	Stimulation chemical storage area
DCA-32002 Surfactant	15,000	30,000	L	Stimulation chemical storage area
DCA-32014 Surfactant	200	400	L	Stimulation chemical storage area
FE-2 pH Buffer	200	400	kg	Stimulation chemical storage area
Hydrochloric Acid - 32%	50,000	150,000	L	Stimulation chemical storage area
100 Mesh Sand	91,000	182,000	kg	Stimulation chemical storage area
40/70 Sand	1,650,000	3,300,000	kg	Stimulation chemical storage area
30/50 Sand	610,000	1,220,000	kg	Stimulation chemical storage area
Sodium Chloride- weighting agent	15,000	30,000	kg	Completion chemical storage area
ALDACIDE G Biocide	500	1,000	L	Completion chemical storage area
OXYGON Oxygen scavenger	100	200	kg	Completion chemical storage area
BARACOR 100 corrosion inhibitor	2,000	4,000	L	Completion chemical storage area
CON-DET wetting agent	50	100	kg	Drilling chemical storage area
SAPP- sodium Acid Phosphate cement treatment	50	100	kg	Drilling chemical storage area
Bentonite- lubricant	3,000	6,000	kg	Drilling chemical storage area
Caustic Soda-pH control	1,400	2,800	kg	Drilling chemical storage area
EZ MUD DP or EZ MUD Liquid- drilling mud	2000	4,000	kg	Drilling chemical storage area
ALDACIDE G Biocide	336	672	kg	Drilling chemical storage area
STOPPIT Loss of circulation material	1,000	2,000	kg	Drilling chemical storage area
Soda Ash- drill mud conditioner	350	700	kg	Drilling chemical storage area
BARACOR 100 Corrosion inhibitor	250	500	kg	Drilling chemical storage area
Sodium Chloride (Flossy Salt)- weighting agent and formation inhibitor	96,000	192,000	kg	Drilling chemical storage area
Barite- weighting agent	500	1,000	kg	Drilling chemical storage area
BARACARB loss of circulation material	500	1,000	kg	Drilling chemical storage area
Citric Acid- pH control	500	1,000	kg	Drilling chemical storage area
BARADEFOAM HP Drilling fluid/foam	500	1,000	kg	Drilling chemical storage area
Sodium Bicarbonate- pH buffer	500	1,000	kg	Drilling chemical storage area

Amended EMP text				
DCA-25005 Gelling Agent	35,000	70,000	kg	Stimulation chemical storage area
DCA-30001 Scale Inhibitor	15,000	30,000	L	Stimulation chemical storage area
DCA-32002 Surfactant	15,000	30,000	L	Stimulation chemical storage area
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40/70 Sand- Proppant	1,650,000	3,300,000	kg	Stimulation chemical storage area
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Alcohols, C11-14-iso-, C13-rich,ethoxylated- Surfactant	5285	10570	L	Stimulation chemical storage area
Sodium (C14-16) olefin sulfonate - Surfactant	4658	9316	L	Stimulation chemical storage area
Diisobutyl glutarate - plasticiser	627	1254	L	Stimulation chemical storage area
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Ethylene Glycol- Crosslinker	5112	10225	L	Stimulation chemical storage area
Choline Chloride- Claystabiliser	10301	20603	L	Stimulation chemical storage area
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Alkyl Alcohol- surfactant	149	299	L	Stimulation chemical storage area
2-Propenoic acid, homopolymer, ammonium salt- biocide	149	299	L	Stimulation chemical storage area
Potassium persulfate- braker	149	299	L	Stimulation chemical storage area

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Current EMP text				
PERFORMATROL- polymer fluid system	500	1,000	kg	Drilling chemical storage area
SOURSCAV- mud additive treat H2S contamination	500	1,000	kg	Drilling chemical storage area
DRIL-N-SLIDE- Casing lubricant	500	1,000	kg	Drilling chemical storage area
STEELSEAL- corrosion inhibitor	500	1,000	kg	Drilling chemical storage area
BARAZAN D or BARAZAN D PLUS- viscosity increaser	4,150	8,300	kg	Drilling chemical storage area
PAC L Loss of circulation material	2,300	4,600	kg	Drilling chemical storage area
Potassium Chloride- weighting agent and formation inhibitor	22,500	45,000	kg	Drilling chemical storage area
GEM CP/GP Shale stabiliser	500	1,000	kg	Drilling chemical storage area
QUIK-FREE – drilling additive	500	1,000	kg	Drilling chemical storage area
BAROFIBRE, BAROFIBRE Superfine and BAROFIBRE COARSE Loss of circulation material	500	1,000	kg	Drilling chemical storage area
BaraBlend-657 Loss of circulation material	500	1,000	kg	Drilling chemical storage area
N-DRIL HT PLUS filtration control additive	500	1,000	kg	Drilling chemical storage area
DEXTRID LTE filtration control additive	4,600	13,800	kg	Drilling chemical storage area
BARABUF pH buffer	500	1,000	kg	Drilling chemical storage area
BORE-HIB shale stabiliser	500	1,000	kg	Drilling chemical storage area
BDF 933 or BaraLube W-933 drilling lubricant	864	1,728	kg	Drilling chemical storage area
BAROLIFT sweeping agent	500	1,000	kg	Drilling chemical storage area
OXYGON Oxygen scavenger	500	1,000	kg	Drilling chemical storage area
ENVIRO-THIN filtration control additive	500	1,000	kg	Drilling chemical storage area
Lime pH buffer	500	1,000	kg	Drilling chemical storage area
BDF 677 Clay stabiliser	4,770	9,540	kg	Drilling chemical storage area
BDF 988 Clay stabiliser	3,390	6,780	kg	Drilling chemical storage area
SARALINE 185V- Synthetic based mud	299,800	599,600	kg	Drilling chemical storage area
NOVATEC P emulsifier for SBM	13,110	26,220	kg	Drilling chemical storage area

Amended EMP text				
2-Ethoxy-naphthalene-surfactant	149	299	L	Stimulation chemical storage area
Sodium Gluconate-stabiliser	8576	17152	L	Stimulation chemical storage area
Boric -Crosslinker	4288	8576	L	Stimulation chemical storage area
Potassium Hydroxide- pH control	10745	21491	L	Stimulation chemical storage area
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DISTILLATES, HYDROTREATED LIGHT-friction reducer	54231	108462	L	Stimulation chemical storage area
Guar Gum- Viscocity regulator	15141	30282	L	Stimulation chemical storage area
Polyoxyethylene nonylphenol ether-surfactant	4466	8933	L	Stimulation chemical storage area
Quaternary ammonium compounds, bis(hydrogenated tallow alkyl)dimethyl, salts with bentonite- biocide	4466	8933	L	Stimulation chemical storage area
1,6-Hexanediol- cross linker	447	893	L	Stimulation chemical storage area
Quartz or Organophilic phyllosilicate- proppant	1084	2167	L	Stimulation chemical storage area
HydroChloric Acid- pH control	44715	89430	L	Stimulation chemical storage area
N-Benzyl-Alkylpyridinium Chloride- pH control	28	57	L	Stimulation chemical storage area
Formic Acid- corrosion inhibitor	38	76	L	Stimulation chemical storage area
Sodium erythorbate-scaler prohibitor	334	668	L	Stimulation chemical storage area
Citric Acid- pH control	15878	31756	L	Stimulation chemical storage area
Acetic Acid- pH control	15878	31756	L	Stimulation chemical storage area
Isopropanol- clay management	83	167	L	Stimulation chemical storage area
Ethoxylated C12-C16 Alcohol - surfactant	57	114	L	Stimulation chemical storage area

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Current EMP text

NOVATEC S emulsifier SBM	5700	11,400	kg	Drilling chemical storage area
Calcium Chloride weighting agent SBM	37,000	74,000	kg	Drilling chemical storage area
VG SUPREME clay viscosifier SBM	11,350	22,700	kg	Drilling chemical storage area
M-I BAR weighting agent SBM	193,500	169,500	kg	Drilling chemical storage area
NOVATEC F emulsifier SBM	3,610	7,220	kg	Drilling chemical storage area
NOVATEC transferred emulsifier SBM	1770	1770	kg	Drilling chemical storage area
Waste drilling fluids	2,500	2,500	m^3	Drilling mud sump
Completion fluids	1.4	1.4	ML	Drilling mud sump/on-site tank
Condensate	160	320	KL	Condensate storage area
Diesel	250	500	KL	Diesel storage tanks
Hydraulic oil	1,000	3,000	L	Workshop
Engine oil	1,000	3,000	L	Workshop
Degreasers	100	300	L	Workshop
Flowback	<10	13.8	ML	Flowback tanks

Amended EMP text

Ethoxylated Decanol - surfactant	19	38	L	Stimulation chemical storage area
Cinnamaldehyde- biocide	57	114	L	Stimulation chemical storage area
Ethoxylated Tallow Alkyl Amine - surfactant	9	19	L	Stimulation chemical storage area
Methanol- corrosion inhibitor	2	4	L	Stimulation chemical storage area
Polyacrylamide - friction reducer	49093	98186	L	Stimulation chemical storage area
Polyethylene glycol trimethylnonyl ether - clay manager	87	173	L	Stimulation chemical storage area
Water in Additive-stabiliser	66804	133607	L	Stimulation chemical storage area
Potassium Sorbate Food Grade- corrosion inhibitor	14	29	L	Stimulation chemical storage area
Mannanase (Mannan endo-1,4-beta-mannosidase)- cross linker	2	4	L	Stimulation chemical storage area
Nonoxynol-9- surfactant	9	19	kg	Stimulation chemical storage area
2-Ethylhexanol PO/EO polymer- stabiliser	9	19	kg	Stimulation chemical storage area
Corn Oil- friction reducer	662	1325	kg	Stimulation chemical storage area
Sodium Chloride-weighting agent	15,000	30,000	kg	Completion chemical storage area
ALDACIDE G Biocide	500	1,000	L	Completion chemical storage area
OXYGON Oxygen scavenger	100	200	kg	Completion chemical storage area
BARACOR 100 corrosion inhibitor	2,000	4,000	L	Completion chemical storage area
CON-DET wetting agent	50	100	kg	Drilling chemical storage area
SAPP- sodium Acid Phosphate cement treatment	50	100	kg	Drilling chemical storage area
Bentonite- lubricant	3,000	6,000	kg	Drilling chemical storage area
Caustic Soda-pH control	1,400	2,800	kg	Drilling chemical storage area
EZ MUD DP or EZ MUD Liquid- drilling mud	2000	4,000	kg	Drilling chemical storage area
ALDACIDE G Biocide	336	672	kg	Drilling chemical storage area
STOPPIT Loss of circulation material	1,000	2,000	kg	Drilling chemical storage area
Soda Ash- drill mud conditioner	350	700	kg	Drilling chemical storage area

Interest holder	Tamboran B2 Pty Ltd	EMP Title	Beetaloo Sub-basin Multi-well Drilling, Stimulation and Well Testing Program EMP	Unique EMP ID	ORI10-3	Mod #	2	Date	25 January 2023

Current EMP text	Amended EMP text				
	BARACOR 100 Corrosion inhibitor	250	500	kg	Drilling chemical storage area
	Sodium Chloride (Flossy Salt)- weighting agent and formation inhibitor	96,000	192,000	kg	Drilling chemical storage area
	Barite- weighting agent	500	1,000	kg	Drilling chemical storage area
	BARACARB loss of circulation material	500	1,000	kg	Drilling chemical storage area
	Citric Acid- pH control	500	1,000	kg	Drilling chemical storage area
	BARADEFOAM HP Drilling fluid/foam	500	1,000	kg	Drilling chemical storage area
	Sodium Bicarbonate- pH buffer	500	1,000	kg	Drilling chemical storage area
	PERFORMATROL- polymer fluid system	500	1,000	kg	Drilling chemical storage area
	SOURSCAV- mud additive treat H2S contamination	500	1,000	kg	Drilling chemical storage area
	DRIL-N-SLIDE- Casing lubricant	500	1,000	kg	Drilling chemical storage area
	STEELSEAL- corrosion inhibitor	500	1,000	kg	Drilling chemical storage area
	BARAZAN D or BARAZAN D PLUS- viscosity increaser	4,150	8,300	kg	Drilling chemical storage area
	PAC L Loss of circulation material	2,300	4,600	kg	Drilling chemical storage area
	Potassium Chloride- weighting agent and formation inhibitor	22,500	45,000	kg	Drilling chemical storage area
	GEM CP/GP Shale stabiliser	500	1,000	kg	Drilling chemical storage area
	QUIK-FREE – drilling additive	500	1,000	kg	Drilling chemical storage area
	BAROFIBRE, BAROFIBRE Superfine and BAROFIBRE COARSE Loss of circulation material	500	1,000	kg	Drilling chemical storage area
	BaraBlend-657 Loss of circulation material	500	1,000	kg	Drilling chemical storage area
	N-DRIL HT PLUS filtration control additive	500	1,000	kg	Drilling chemical storage area
	DEXTRID LTE filtration control additive	4,600	13,800	kg	Drilling chemical storage area
	BARABUF pH buffer	500	1,000	kg	Drilling chemical storage area
	BORE-HIB shale stabiliser	500	1,000	kg	Drilling chemical storage area

Interest holder	Tamboran B2 Pty Ltd	EMP Title	Beetaloo Sub-basin Multi-well Drilling, Stimulation and Well Testing Program EMP	Unique EMP ID	ORI10-3	Mod #	2	Date	25 January 2023

Current EMP text

Amended EMP text

BDF 933 or BaraLube W-933 drilling lubricant	864	1,728	kg	Drilling chemical storage area
BAROLIFT sweeping agent	500	1,000	kg	Drilling chemical storage area
OXYGON Oxygen scavenger	500	1,000	kg	Drilling chemical storage area
ENVIRO-THIN filtration control additive	500	1,000	kg	Drilling chemical storage area
Lime pH buffer	500	1,000	kg	Drilling chemical storage area
BDF 677 Clay stabiliser	4,770	9,540	kg	Drilling chemical storage area
BDF 988 Clay stabiliser	3,390	6,780	kg	Drilling chemical storage area
SARALINE 185V- Synthetic based mud	299,800	599,600	kg	Drilling chemical storage area
NOVATEC P emulsifier for SBM	13,110	26,220	kg	Drilling chemical storage area
NOVATEC S emulsifier SBM	5700	11,400	kg	Drilling chemical storage area
Calcium Chloride weighting agent SBM	37,000	74,000	kg	Drilling chemical storage area
VG SUPREME clay viscosifier SBM	11,350	22,700	kg	Drilling chemical storage area
M-I BAR weighting agent SBM	193,500	169,500	kg	Drilling chemical storage area
NOVATEC F emulsifier SBM	3,610	7,220	kg	Drilling chemical storage area
NOVATEC transferred emulsifier SBM	1770	1770	kg	Drilling chemical storage area
Waste drilling fluids	2,500	2,500	m^3	Drilling mud sump
Completion fluids	1.4	1.4	ML	Drilling mud sump/on-site tank
Condensate	160	320	KL	Condensate storage area
Diesel	250	500	KL	Diesel storage tanks
Hydraulic oil	1,000	3,000	L	Workshop
Engine oil	1,000	3,000	L	Workshop
Degreasers	100	300	L	Workshop
Flowback	<10	13.8	ML	Flowback tanks

Appendix F Spill Management Plan

Spill Management Plan Appendix A Chemical volumes per well and storage areas (based on maximum 3 wells per pad)

Appendix F Spill Management Plan

Spill Management Plan Appendix A Chemical volumes per well and storage areas (based on maximum 3 wells per pad)

Material name	Typical volume	Maximum volume	Unit	Storage area

Interest holder	Tamboran B2 Pty Ltd	EMP Title	Beetaloo Sub-basin Multi-well Drilling, Stimulation and Well Testing Program EMP	Unique EMP ID	ORI10-3	Mod #	2	Date	25 January 2023

Current EMP text

Material name	Typical volume	Maximum volume	Unit	Storage area
Acetic Acid - 60% pH control	3,000	6,000L	L	Stimulation chemical storage area
BE-9 Biocide	17,000	34,000	L	Stimulation chemical storage area
Caustic Soda Liquid pH control	15,000	30,000	L	Stimulation chemical storage area
DCA-11001 Breaker Activator	5,000	10,000	L	Stimulation chemical storage area
DCA-13002 Breaker	300	600	kg	Stimulation chemical storage area
DCA-13003 Breaker	10,000	20,000	L	Stimulation chemical storage area
DCA-16001 Clay Stabiliser	42,000	84,000	L	Stimulation chemical storage area
DCA-17001 Corrosion Inhibitor	1,000	2,000	L	Stimulation chemical storage area
DCA-19001 Crosslinker	600	1,200	kg	Stimulation chemical storage area
DCA-19002 Crosslinker	10,000	20,000	L	Stimulation chemical storage area
DCA-23001 Friction Reducer	5,000	10,000	kg	Stimulation chemical storage area
DCA-23003 Friction Reducer	18,000	36,000	L	Stimulation chemical storage area
DCA-25005 Gelling Agent	35,000	70,000	kg	Stimulation chemical storage area
DCA-30001 Scale Inhibitor	15,000	30,000	L	Stimulation chemical storage area
DCA-32002 Surfactant	15,000	30,000	L	Stimulation chemical storage area
DCA-32014 Surfactant	200	400	L	Stimulation chemical storage area
FE-2 pH Buffer	200	400	kg	Stimulation chemical storage area
Hydrochloric Acid - 32%	50,000	150,000	L	Stimulation chemical storage area
100 Mesh Sand	91,000	182,000	kg	Stimulation chemical storage area
40/70 Sand	1,650,000	3,300,000	kg	Stimulation chemical storage area
30/50 Sand	610,000	1,220,000	kg	Stimulation chemical storage area
Sodium Chloride- weighting agent	15,000	30,000	kg	Completion chemical storage area
ALDACIDE G Biocide	500	1,000	L	Completion chemical storage area
OXYGON Oxygen scavenger	100	200	kg	Completion chemical storage area
BARACOR 100 corrosion inhibitor	2,000	4,000	L	Completion chemical storage area
CON-DET wetting agent	50	100	kg	Drilling chemical storage area
SAPP- sodium Acid Phosphate cement treatment	50	100	kg	Drilling chemical storage area
Bentonite- lubricant	3,000	6,000	kg	Drilling chemical storage area
Caustic Soda-pH control	1,400	2,800	kg	Drilling chemical storage area

Amended EMP text

Acetic Acid - 60% PH control	3,000	6,000	L	Stimulation chemical storage area
BE-9 Biocide	17,000	34,000	L	Stimulation chemical storage area
Caustic Soda Liquid pH control/ buffer	15,000	30,000	L	Stimulation chemical storage area
DCA-11001 Breaker Activator	5,000	10,000	L	Stimulation chemical storage area
DCA-13002 Breaker	300	600	kg	Stimulation chemical storage area
DCA-13003 Breaker	10,000	20,000	L	Stimulation chemical storage area
DCA-16001 Clay Stabiliser	42,000	84,000	L	Stimulation chemical storage area
DCA-17001 Corrosion Inhibitor	1,000	2,000	L	Stimulation chemical storage area
DCA-19001 Crosslinker	600	1,200	kg	Stimulation chemical storage area
DCA-19002 Crosslinker	10,000	20,000	L	Stimulation chemical storage area
DCA-23001 Friction Reducer	5,000	10,000	kg	Stimulation chemical storage area
DCA-23003 Friction Reducer	18,000	36,000	L	Stimulation chemical storage area
DCA-25005 Gelling Agent	35,000	70,000	kg	Stimulation chemical storage area
DCA-30001 Scale Inhibitor	15,000	30,000	L	Stimulation chemical storage area
DCA-32002 Surfactant	15,000	30,000	L	Stimulation chemical storage area
DCA-32014 Surfactant	200	400	L	Stimulation chemical storage area
FE-2 Buffer	200	400	kg	Stimulation chemical storage area
Hydrochloric Acid - 32%	50,000	150,000	L	Stimulation chemical storage area
100 Mesh Sand- Proppant	91,000	182,000	kg	Stimulation chemical storage area
40/70 Sand- Proppant	1,650,000	3,300,000	kg	Stimulation chemical storage area
30/50 Sand- Proppant	610,000	1,220,000	kg	Stimulation chemical storage area
Alcohols, C11-14-iso-, C13-rich,ethoxylated-Surfactant	5285	10570	L	Stimulation chemical storage area
Sodium (C14-16) olefin sulfonate - Surfactant	4658	9316	L	Stimulation chemical storage area
Diisobutyl glutarate - plasticiser	627	1254	L	Stimulation chemical storage area
Diisobutyl succinate - plasticiser	209	418	L	Stimulation chemical storage area
Diisobutyl adipate- plasticiser	179	358	L	Stimulation chemical storage area
sodium thiosulphate- stabilising agent	4763	9527	L	Stimulation chemical storage area
sodium sulphate stabilising agent	913	1827	L	Stimulation chemical storage area
sodium sulphite stabilising agent	794	1588	L	Stimulation chemical storage area

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Current EMP text				
EZ MUD DP or EZ MUD Liquid- drilling mud	2000	4,000	kg	Drilling chemical storage area
ALDACIDE G Biocide	336	672	kg	Drilling chemical storage area
STOPPIT Loss of circulation material	1,000	2,000	kg	Drilling chemical storage area
Soda Ash- drill mud conditioner	350	700	kg	Drilling chemical storage area
BARACOR 100 Corrosion inhibitor	250	500	kg	Drilling chemical storage area
Sodium Chloride (Flossy Salt)- weighting agent and formation inhibitor	96,000	192,000	kg	Drilling chemical storage area
Barite- weighting agent	500	1,000	kg	Drilling chemical storage area
BARACARB loss of circulation material	500	1,000	kg	Drilling chemical storage area
Citric Acid- pH control	500	1,000	kg	Drilling chemical storage area
BARADEFOAM HP Drilling fluid/foam	500	1,000	kg	Drilling chemical storage area
Sodium Bicarbonate- pH buffer	500	1,000	kg	Drilling chemical storage area
PERFORMATROL- polymer fluid system	500	1,000	kg	Drilling chemical storage area
SOURSCAV- mud additive treat H2S contamination	500	1,000	kg	Drilling chemical storage area
DRIL-N-SLIDE- Casing lubricant	500	1,000	kg	Drilling chemical storage area
STEELSEAL- corrosion inhibitor	500	1,000	kg	Drilling chemical storage area
BARAZAN D or BARAZAN D PLUS- viscosity increaser	4,150	8,300	kg	Drilling chemical storage area
PAC L Loss of circulation material	2,300	4,600	kg	Drilling chemical storage area
Potassium Chloride- weighting agent and formation inhibitor	22,500	45,000	kg	Drilling chemical storage area
GEM CP/GP Shale stabiliser	500	1,000	kg	Drilling chemical storage area
QUIK-FREE – drilling additive	500	1,000	kg	Drilling chemical storage area
BAROFIBRE, BAROFIBRE Superfine and BAROFIBRE COARSE Loss of circulation material	500	1,000	kg	Drilling chemical storage area
BaraBlend-657 Loss of circulation material	500	1,000	kg	Drilling chemical storage area

Amended EMP text				
Ethylene Glycol- Crosslinker	5112	10225	L	Stimulation chemical storage area
Choline Chloride- Claystabiliser	10301	20603	L	Stimulation chemical storage area
Glutaraldehyde- Biocide	14930	29859	L	Stimulation chemical storage area
Ammonium Sulphate- Breaker	4479	8958	L	Stimulation chemical storage area
Polyacrylamide- Friction reducer	4479	8958	L	Stimulation chemical storage area
Sodium polyacrylate- gelling agent	746	1493	L	Stimulation chemical storage area
Sodium bisulfite- stabiliser	149	299	L	Stimulation chemical storage area
Alkyl Alcohol- surfactant	149	299	L	Stimulation chemical storage area
2-Propenoic acid, homopolymer, ammonium salt- biocide	149	299	L	Stimulation chemical storage area
Potassium persulfate- braker	149	299	L	Stimulation chemical storage area
2-Ethoxy-naphthalene- surfactant	149	299	L	Stimulation chemical storage area
Sodium Gluconate- stabiliser	8576	17152	L	Stimulation chemical storage area
Boric -Crosslinker	4288	8576	L	Stimulation chemical storage area
Potassium Hydroxide- pH control	10745	21491	L	Stimulation chemical storage area
Mannanase- Cross linker	2	4	L	Stimulation chemical storage area
Ammonium Persulphate- breaker	7451	14902	L	Stimulation chemical storage area
Talc- buffer	384	769	L	Stimulation chemical storage area
Sodium Bromate- breaker	50441	100881	L	Stimulation chemical storage area
Hepta sodium phosphonate- Emulsifier	3176	6351	L	Stimulation chemical storage area
DISTILLATES, HYDROTREATED LIGHT- friction reducer	54231	108462	L	Stimulation chemical storage area
Guar Gum- Viscocity regulator	15141	30282	L	Stimulation chemical storage area
Polyoxyethylene nonylphenol ether- surfactant	4466	8933	L	Stimulation chemical storage area
Quaternary ammonium compounds, bis(hydrogenated tallow alkyl)dimethyl, salts with bentonite- biocide	4466	8933	L	Stimulation chemical storage area

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Current EMP text				
N-DRIL HT PLUS filtration control additive	500	1,000	kg	Drilling chemical storage area
DEXTRID LTE filtration control additive	4,600	13,800	kg	Drilling chemical storage area
BARABUF pH buffer	500	1,000	kg	Drilling chemical storage area
BORE-HIB shale stabiliser	500	1,000	kg	Drilling chemical storage area
BDF 933 or BaraLube W-933 drilling lubricant	864	1,728	kg	Drilling chemical storage area
BAROLIFT sweeping agent	500	1,000	kg	Drilling chemical storage area
OXYGON Oxygen scavenger	500	1,000	kg	Drilling chemical storage area
ENVIRO-THIN filtration control additive	500	1,000	kg	Drilling chemical storage area
Lime pH buffer	500	1,000	kg	Drilling chemical storage area
BDF 677 Clay stabiliser	4,770	9,540	kg	Drilling chemical storage area
BDF 988 Clay stabiliser	3,390	6,780	kg	Drilling chemical storage area
SARALINE 185V- Synthetic based mud	299,800	599,600	kg	Drilling chemical storage area
NOVATEC P emulsifier for SBM	13,110	26,220	kg	Drilling chemical storage area
NOVATEC S emulsifier SBM	5700	11,400	kg	Drilling chemical storage area
Calcium Chloride weighting agent SBM	37,000	74,000	kg	Drilling chemical storage area
VG SUPREME clay viscosifier SBM	11,350	22,700	kg	Drilling chemical storage area
M-I BAR weighting agent SBM	193,500	169,500	kg	Drilling chemical storage area
NOVATEC F emulsifier SBM	3,610	7,220	kg	Drilling chemical storage area
NOVATEC transferred emulsifier SBM	1770	1770	kg	Drilling chemical storage area
Waste drilling fluids	2,500	2,500	m^3	Drilling mud sump
Completion fluids	1.4	1.4	ML	Drilling mud sump/on-site tank
Condensate	160	320	KL	Condensate storage area
Diesel	250	500	KL	Diesel storage tanks
Hydraulic oil	1,000	3,000	L	Workshop
Engine oil	1,000	3,000	L	Workshop
Degreasers	100	300	L	Workshop
Flowback	<10	13.8	ML	Flowback tanks

Amended EMP text				
1,6-Hexanediol- cross linker	447	893	L	Stimulation chemical storage area
Quartz or Organophilic phyllosilicate- proppant	1084	2167	L	Stimulation chemical storage area
HydroChloric Acid- pH control	44715	89430	L	Stimulation chemical storage area
N-Benzyl-Alkylpyridinium Chloride- pH control	28	57	L	Stimulation chemical storage area
Formic Acid- corrosion inhibitor	38	76	L	Stimulation chemical storage area
Sodium erythorbate- scaler prohibitor	334	668	L	Stimulation chemical storage area
Citric Acid- pH control	15878	31756	L	Stimulation chemical storage area
Acetic Acid- pH control	15878	31756	L	Stimulation chemical storage area
Isopropanol- clay management	83	167	L	Stimulation chemical storage area
Ethoxylated C12-C16 Alcohol - surfactant	57	114	L	Stimulation chemical storage area
Ethoxylated Decanol - surfactant	19	38	L	Stimulation chemical storage area
Cinnamaldehyde- biocide	57	114	L	Stimulation chemical storage area
Ethoxylated Tallow Alkyl Amine - surfactant	9	19	L	Stimulation chemical storage area
Methanol- corrosion inhibitor	2	4	L	Stimulation chemical storage area
Polyacrylamide - friction reducer	49093	98186	L	Stimulation chemical storage area
Polyethylene glycol trimethylnonyl ether - clay manager	87	173	L	Stimulation chemical storage area
Water in Additive- stabiliser	66804	133607	L	Stimulation chemical storage area
Potassium Sorbate Food Grade- corrosion inhibitor	14	29	L	Stimulation chemical storage area
Mannanase (Mannan endo-1,4-beta-mannosidase)- cross linker	2	4	L	Stimulation chemical storage area
Nonoxynol-9- surfactant	9	19	L	Stimulation chemical storage area
2-Ethylhexanol PO/EO polymer- stabiliser	9	19	L	Stimulation chemical storage area
Corn Oil- friction reducer	662	1325	L	Stimulation chemical storage area
Sodium Chloride- weighting agent	15,000	30,000	kg	Completion chemical storage area
ALDACIDE G Biocide	500	1,000	L	Completion chemical storage area
OXYGON Oxygen scavenger	100	200	kg	Completion chemical storage area

Interest holder	Tamboran B2 Pty Ltd	EMP Title	Beetaloo Sub-basin Multi-well Drilling, Stimulation and Well Testing Program EMP	Unique EMP ID	ORI10-3	Mod #	2	Date	25 January 2023

Current EMP text	Amended EMP text				
	BARACOR 100 corrosion inhibitor	2,000	4,000	L	Completion chemical storage area
	CON-DET wetting agent	50	100	kg	Drilling chemical storage area
	SAPP- sodium Acid Phosphate cement treatment	50	100	kg	Drilling chemical storage area
	Bentonite- lubricant	3,000	6,000	kg	Drilling chemical storage area
	Caustic Soda-pH control	1,400	2,800	kg	Drilling chemical storage area
	EZ MUD DP or EZ MUD Liquid- drilling mud	2000	4,000	kg	Drilling chemical storage area
	ALDACIDE G Biocide	336	672	kg	Drilling chemical storage area
	STOPPIT Loss of circulation material	1,000	2,000	kg	Drilling chemical storage area
	Soda Ash- drill mud conditioner	350	700	kg	Drilling chemical storage area
	BARACOR 100 Corrosion inhibitor	250	500	kg	Drilling chemical storage area
	Sodium Chloride (Flossy Salt)- weighting agent and formation inhibitor	96,000	192,000	kg	Drilling chemical storage area
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	BARACARB loss of circulation material	500	1,000	kg	Drilling chemical storage area
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	Sodium Bicarbonate- pH buffer	500	1,000	kg	Drilling chemical storage area
	PERFORMATROL- polymer fluid system	500	1,000	kg	Drilling chemical storage area
	SOURSCAV- mud additive treat H2S contamination	500	1,000	kg	Drilling chemical storage area
	DRIL-N-SLIDE- Casing lubricant	500	1,000	kg	Drilling chemical storage area
	STEELSEAL- corrosion inhibitor	500	1,000	kg	Drilling chemical storage area
	BARAZAN D or BARAZAN D PLUS- viscosity increaser	4,150	8,300	kg	Drilling chemical storage area
	PAC L Loss of circulation material	2,300	4,600	kg	Drilling chemical storage area
	Potassium Chloride- weighting agent and formation inhibitor	22,500	45,000	kg	Drilling chemical storage area
	GEM CP/GP Shale stabiliser	500	1,000	kg	Drilling chemical storage area

Interest holder	Tamboran B2 Pty Ltd	EMP Title	Beetaloo Sub-basin Multi-well Drilling, Stimulation and Well Testing Program EMP	Unique EMP ID	ORI10-3	Mod #	2	Date	25 January 2023

Current EMP text	Amended EMP text				
	QUIK-FREE – drilling additive	500	1,000	kg	Drilling chemical storage area
	BAROFIBRE, BAROFIBRE Superfine and BAROFIBRE COARSE Loss of circulation material	500	1,000	kg	Drilling chemical storage area
	BaraBlend-657 Loss of circulation material	500	1,000	kg	Drilling chemical storage area
	N-DRIL HT PLUS filtration control additive	500	1,000	kg	Drilling chemical storage area
	DEXTRID LTE filtration control additive	4,600	13,800	kg	Drilling chemical storage area
	BARABUF pH buffer	500	1,000	kg	Drilling chemical storage area
	BORE-HIB shale stabiliser	500	1,000	kg	Drilling chemical storage area
	BDF 933 or BaraLube W-933 drilling lubricant	864	1,728	kg	Drilling chemical storage area
	BAROLIFT sweeping agent	500	1,000	kg	Drilling chemical storage area
	OXYGON Oxygen scavenger	500	1,000	kg	Drilling chemical storage area
	ENVIRO-THIN filtration control additive	500	1,000	kg	Drilling chemical storage area
	Lime pH buffer	500	1,000	kg	Drilling chemical storage area
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	BDF 988 Clay stabiliser	3,390	6,780	kg	Drilling chemical storage area
	SARALINE 185V- Synthetic based mud	299,800	599,600	kg	Drilling chemical storage area
	NOVATEC P emulsifier for SBM	13,110	26,220	kg	Drilling chemical storage area
	NOVATEC S emulsifier SBM	5700	11,400	kg	Drilling chemical storage area
	Calcium Chloride weighting agent SBM	37,000	74,000	kg	Drilling chemical storage area
	VG SUPREME clay viscosifier SBM	11,350	22,700	kg	Drilling chemical storage area
	M-I BAR weighting agent SBM	193,500	169,500	kg	Drilling chemical storage area
	NOVATEC F emulsifier SBM	3,610	7,220	kg	Drilling chemical storage area
	NOVATEC transferred emulsifier SBM	1770	1770	kg	Drilling chemical storage area
	Waste drilling fluids	2,500	2,500	m^3	Drilling mud sump
	Completion fluids	1.4	1.4	ML	Drilling mud sump/on-site tank
	Condensate	160	320	KL	Condensate storage area
	Diesel	250	500	KL	Diesel storage tanks
	Hydraulic oil	1,000	3,000	L	Workshop

Interest holder	Tamboran B2 Pty Ltd	EMP Title	Beetaloo Sub-basin Multi-well Drilling, Stimulation and Well Testing Program EMP	Unique EMP ID	ORI10-3	Mod #	2	Date	25 January 2023

Current EMP text	Amended EMP text
	<table><tr><td>Engine oil</td><td>1,000</td><td>3,000</td><td>L</td><td>Workshop</td></tr><tr><td>Degreasers</td><td>100</td><td>300</td><td>L</td><td>Workshop</td></tr><tr><td>Flowback</td><td><10</td><td>13.8</td><td>ML</td><td>Flowback tanks</td></tr></table>
Appendix	
Appendix E Chemical Risk Assessment AECOM (2019). *Beetaloo 2019 Campaign - Hydraulic Fracturing Chemical Risk Assessment*, dated 16-Dec-2019.	**Appendix E Chemical Risk Assessment** AECOM (2019). *Beetaloo 2019 Campaign - Hydraulic Fracturing Chemical Risk Assessment*, dated 16-Dec-2019. **Appendix E.1 Chemical Risk Assessment – Condor** EHS Support (2023) *Hydraulic Stimulation Chemical Risk Assessment Tamboran Resources Northern Territory Tenements*, dated 12 January 2023.

Appendix E.1 Chemical Risk Assessment – Condor

Hydraulic Stimulation Chemical Risk Assessment

Tamboran Resources Northern Territory Tenements

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Acronyms

AICIS	Australian Industrial Chemicals Introduction Scheme, 2022
CAS	Chemical Abstracts Service
COPC	constituent of potential concern
CRA	Chemical Risk Assessment
DoEE	Department of the Environment and Energy
EMP	Environment Management Plan
EP	Exploration Permit
LC50/EC50	lethal concentration 50 / effect concentration 50
NEPC	National Environment Protection Council
NEPM	National Environment Protection (Assessment of Site Contamination) Measure
NICNAS	National Industrial Chemicals Notification and Assessment Scheme
NOAEL	no observed adverse effect level
NOEC	no observed effect concentration
NT	Northern Territory
PBT	persistent (P), bioaccumulative (B) and toxic (T)
SDS	safety data sheet

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Units of Measure

Area	
ha	hectare
m^2	square metres
Density	
kg/m^3	kilograms per cubic metre
Electrical Conductance	
µS/cm	microsiemen per centimetre
dS/m	decisiemen per metre
mS/cm	millisiemen per centimetre
mV	millivolt
Length	
µm	micrometres
cm	centimetres
km	kilometres
m	metres
mm	millimetres
Mass	
µg	micrograms
g	grams
kg	kilograms
mg	milligrams
t	metric tonnes
Concentration by Mass	
µg/kg	microgram per kilogram
mg/kg	milligram per kilogram

Pressure	
kPa	Kilopascals
Pa	Pascals
Temperature	
°C	degrees Celsius
°F	degrees Fahrenheit
K	Kelvin
Velocity	
m/s	metres per second
Volume	
µL	microlitres
cL	centilitres
cm^3	cubic centimetre
GL	gigalitre
L	litres
m^3	cubic metre
mL	millilitres
ML	megalitre
Concentration by Volume	
µg/L	microgram per litre
mg/L	milligram per litre
ppmv	parts per million by volume
ppbv	parts per billion by volume

1 Introduction

Condor Energy ("Condor") has been retained by Tamboran B2 Pty Ltd ("Tamboran") to provide hydraulic stimulation services for pilot wells located within Tamboran's tenements within the Beetaloo Sub-basin of the Northern Territory (NT). Tamboran recently acquired NT exploration permits (EPs) 98, EP117 and EP76 (**Figure 1-1**) within the Beetaloo Sub-basin from Origin Energy B2 Pty Ltd ("Origin"). There are no homesteads within 10 km of the proposed worksites.

Prior to the transfer of assets to Tamboran, Origin prepared Environment Management Plans (EMP) for EP98, EP117 and EP76 to progress exploration activities across their respective tenements. The EMPs cover various exploration activities, which include undertaking seismic surveys, drilling targeted exploration wells and subsequent hydraulic fracturing of these wells (Origin, 2021a; Origin, 2021b). Tamboran is also developing an updated EMP for EP98, EP117 and EP76; however, at this time the Origin EMP was used as the basis for this evaluation. For the purposes of this assessment, it is assumed that the environmental controls relevant to hydraulic stimulation under the updated Tamboran EMP will be effectively the same as that within the current EMP.

Under the Code of Practice: Petroleum Activities in the Northern Territory 2021 ("the Code"), an EMP is required for oil and gas activities. Hydraulic stimulation (or fracturing) activities were reviewed in the *Independent Scientific Inquiry into Hydraulic Fracturing of Onshore Unconventional Reservoirs in the Northern Territory* report issued on 27 March 2018 (NT, 2018). The Inquiry concluded that the risks associated with unconventional onshore shale gas extraction in the NT could be appropriately managed provided all the recommendations of its report were adopted and implemented. The NT Government accepted all 135 recommendations and announced the lifting of a previous moratorium on exploration on 17 April 2018. Of the 135 recommendations, 35 were required to be implemented prior to the commencement of exploration, with the remaining recommendations required to be implemented prior to the commencement of production. The development of an EMP is a key component of meeting these requirements. The EMP documents the relevant natural environment, proposed activities and methods to manage the environmental impacts and risks associated with proposed activities, including how to address regulatory obligations and relevant report recommendations that have underpinned the Code of Practice: Onshore Petroleum Activities in Northern Territory 2021.

Condor is undertaking planning for the hydraulic stimulation program and has retained EHS Support Pty Ltd ("EHS Support") to prepare a Chemical Risk Assessment (CRA) to reflect the proposed stimulation fluids for potential use in EP98, EP117 or EP76. Tamboran is currently planning to undertake hydraulic stimulation of existing wells at the at the Amungee NW-2H site, and also plan to undertake further exploration activities, including drilling wells and undertaking hydraulic stimulation within other areas of EP98, EP117 and EP76 in the future. This CRA documents the relevant EMP requirements utilising the chemicals present in the proposed hydraulic stimulation formulations for future stimulation activities. This formulation is presented in **Appendix A**. Chemicals listed in this table with a volume of 0 were not assessed in this CRA. Additional updates to this CRA may be required in the future, with the evaluation of any additional proposed chemicals/revised chemical concentrations, and this will be undertaken where applicable.

This CRA evaluates potential hazards associated with chemicals and the potential for human and environmental receptor exposure, and where potentially hazardous chemicals have complete exposure pathways, quantification of the potential risks. This CRA is supported by a broader

evaluation of environmental conditions and risks and recommended avoidance, mitigation and management strategies.

This CRA for the hydraulic stimulation activities developed as part of the EMP meets the requirements of the NT Code of Practice as well as being in general accordance with the following:

- Petroleum Operations, Department of Environment, Parks and Water Security (DEPWS), Environment Management Plan Content Guideline (NT, 2021);
- Department of the Environment and Energy, Exposure Draft - Chemical Risk Assessment Guidance Manual: for chemicals associated with coal seam gas extraction (DoEE, 2017);
- Australian Industrial Chemicals Introduction Scheme, 2022 (AICIS) (which has progressively replaced NICNAS below, since 31 August 2020);
- National Industrial Chemicals Notification and Assessment Scheme (NICNAS), National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia (NICNAS, 2017a);
- enHealth Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth, 2012); and
- National Environment Protection (Assessment of Site Contamination) Measure 1999 (ASC NEPM); Schedule B4, Site-specific health risk assessment methodology (NEPC, 2013).

Reference has also been made to the relevant information available within the Petroleum Onshore Information Northern Territory (POINT) online mapping and data catalogue.

The chemicals assessed in this CRA have been compiled from several formulations that have been used (or are planned for use) in the Beetaloo Sub-Basin and potentially in other tenements and basins. The lists of chemicals assessed are presented in **Appendix A** and were provided by Condor. **Appendix A** also includes maximum concentrations that potentially would be used in a hydraulic stimulation. It should be noted that the compiled lists of chemicals have been assessed as “one formulation” (noting that they contain a number of separately used components that are applied at various stages during the stimulation process) with maximum concentrations provided by Condor. This is a conservative assessment for the hydraulic stimulation program because the actual concentration of individual chemicals will potentially be less, and there may be fewer chemicals represented in a selected formulation.

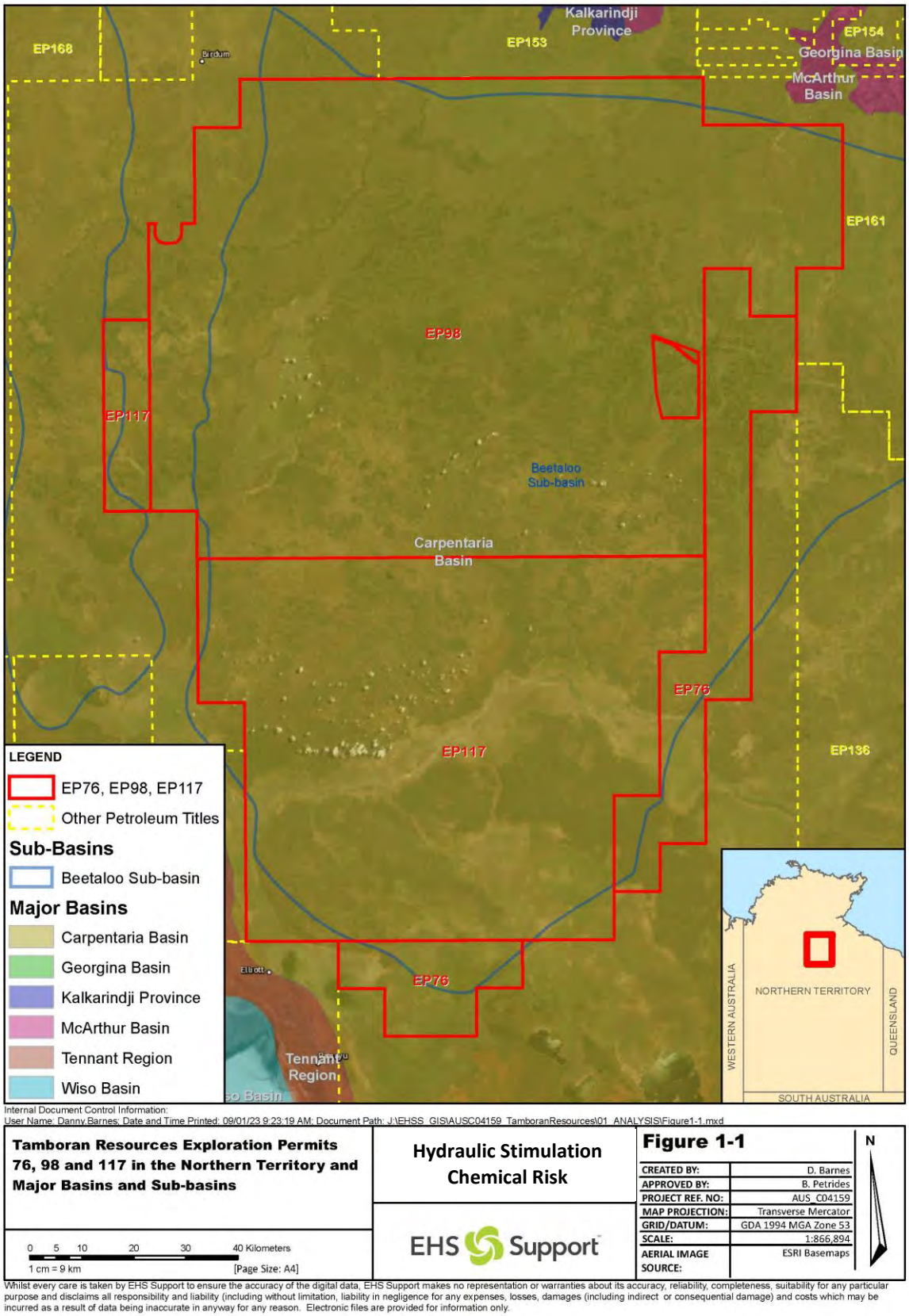

Figure 1-1 Location of Tamboran Tenements

2 Tier Assessment

A tiered assessment was conducted on the compiled hydraulic fracturing fluid systems using screening of the potential human health and ecological hazards that should be considered for potential exposure to the hydraulic fracturing fluids during transportation, hydraulic fracturing activities (including storage) and subsequent treatment and disposal of flowback. The tiered assessment includes the following steps:

- Tier 1 – Identify chemicals of low human health and ecological concern that do not require additional evaluation in the tier assessment process.
- Tier 2 – Chemicals that are not identified as a low human health and ecological concern and therefore require an additional risk assessment to characterise potential risks. This is done using a quantitative evaluation of the risks based on the potential complete exposure pathways and Tier 1 assessment.

2.1 Conceptual Exposure Model

The EMP developed for EP98, EP117 and EP76 will provide an overview of the proposed hydraulic stimulation program, which is similar to that which will be utilised by Condor's other NT tenements. The stimulation process involves pumping slurry, primarily consisting of water and sand (proppant) plus a minor volume of a specific blend of chemicals down the well to a specific geological target at sufficient pressure to create fractures in the target geological formation. Proppant keeps the fractures open after the fluid pressure is released, thereby improving the wells productive potential. Chemicals used in hydraulic stimulation fluid systems are designed to optimise stimulation outcomes and are commonly found in food and other household domestic products.

Casing bullhead fracture stimulations are typically implemented in shale development with a pump down plug and perforation technique for fluid diversion. This is achieved by pumping down a bridge plug and perforating guns on wireline to the desired depth in the horizontal wellbore. The plug is set, and the zone is perforated. The tools are then removed from the well, and the fracture stimulation treatment is pumped in. This process is repeated until all target zones in the well are complete.

The life cycle of chemicals used during the hydraulic fracturing of wells includes the following general categories:

- Transportation of chemicals – from the supplier warehouse to the well lease and between well leases.
- Hydraulic fracturing activities – storage of chemicals, usage (e.g., blending, injecting) and subsequent recovery of fluids (including storage in produced water and flowback fluid treatment tanks) at the well lease and associated vendor chemical additives.
- Disposal and management – recovered vendor chemical additives in wastes and hydraulic fracturing flowback.

Throughout the life cycle of chemical additive products, without adequate management controls in place, there is the potential for human and environmental receptors to be exposed to the chemical additives. Based on an evaluation of the life cycle of products and chemicals, environmental conditions in the areas of development, anticipated populations and location selection, the following potentially complete exposure pathways were identified:

- Transportation of chemicals:
  - Human and environmental receptor exposure to chemicals as a result of accidental release during transport from supplier warehouse to well lease or between well leases (i.e., truck rollover).
  - Human and environmental receptors exposed to surface water bodies that received runoff from an accidental release during transportation.
- Hydraulic fracturing activities:
  - Human and environmental receptor exposure to chemicals as a result of accidental release during the storage and preparation of products on the well lease for hydraulic fracturing activities.
  - Human and environmental receptor exposure to residual chemicals (vendor chemicals) in recovered materials as a result of an accidental release from storages (treatment tanks) on the well lease.
  - Human and environmental receptors exposed to surface water bodies that received runoff from an accidental release during hydraulic fracturing activities.
- Treatment, Storage and disposal:
  - Human and environmental receptor exposure to chemicals as a result of accidental release during transport of surplus chemicals and wastes (i.e., flowback) from the well lease to a disposal/management facility.
  - Human and environmental receptor exposure to chemicals as a result of accidental release of stored wastes and/or flowback.
  - Human and environmental receptors exposed to surface water bodies that received runoff from an accidental release of stored wastes and/or flowback.

To assess the unmitigated risks from the improbable scenario where some fluids were to overflow the bunded area, a range of release scenarios are considered comprising:

- Smaller release volumes of 1,000 L and 100,000 L, which would reflect small scale releases; and
- An improbable release out of the bunded area (1,000,000 L).

Appendix B provides an assessment of the potential for effects on groundwater associated with a release of hydraulic fracturing fluid, waste or flowback to the land surface scenarios. The results of this assessment showed that the travel times for surface releases to reach groundwater are very long, thereby providing ample opportunity for containment and remedial action. Therefore, the potential for impacts to groundwater is considered low.

Both mitigated and unmitigated risks from an overland flow scenario from a release have been assessed as part of the assessment. Typical pads for shale development in the Beetaloo range from 5.5 -10 ha with typically 1 m high berm walls surrounding any inground treatment tanks and/or double-lined aboveground tanks to contain and manage the risk from potential releases. In the absence of this structure, a major release could have the potential to migrate a distance off the well pad. However, with these measures, any releases would be limited to the potential for incidental/minor spillage outside the fluid storage and containment area. In the context of a potential release scenario of 1,000 L outside of the containment and storage area, the maximum affected area of spreading will be less than 0.4 ha and limited to the proximity of the release area.

Therefore, given the planned management control of the construction of a bunded area surrounding treatment tanks, the potential for a complete exposure pathway to surface water bodies associated with runoff from an accidental release is considered unlikely and not assessed further.

The risks associated with the transport of chemicals and wastes is considered to be managed to a level as low as reasonably practicable. This is because the potential for a release is controlled through the implementation of a traffic/transport management plan, including use of designated trucking routes, vehicle signage, vehicle management systems (to manage speed and driving behaviour/habits). In the unlikely event of a vehicular accident, incident and spill response procedures will be implemented. In this context, this scenario is not assessed further.

The management of chemicals and wastes will be conducted at the well lease using drums, intermediate bulk containers and engineered tanks designed to contain the fluids. No permanent storage of chemicals, flowback or wastes will be conducted in ponds or sumps, and therefore the potential for releases is considered limited. Wastewater will be managed through the use of engineered treatment tanks that will contain liquids and may have the potential for exposures to avian receptors; however, this exposure route is unlikely given that tanks would not harbour fish or other vertebrates that would be attractive to avian species, or that would give rise to incidental ingestion of water during feeding. In the unlikely event of a release to the ground, the potential for exposures (other than workers) is limited. The well pad sites are fenced to limit access to the public and prevent entry by livestock and large native fauna. If materials are spilled to the ground, then investigation, remediation and rehabilitation activities will be immediately implemented to address soil impacts. In this context, exposure during and post-activity are unlikely.

Lastly, chemical exposures to workers are controlled through engineering, management controls and personal protective equipment, which are focused on elimination and mitigation of the potential for dermal contact and potential for incidental ingestion (therefore, the exposures are considered unlikely). In addition, Safe Work Australia and Condor Occupational Safety Guidance are used to minimise human health exposure.

2.2 Tier 1 Assessment

The Tier 1 assessment includes an evaluation of the human health and environmental hazards of the chemicals in the two hydraulic fracturing fluid systems. The objective of the Tier 1 assessment is to identify chemicals of low human health and ecological concern that do not require additional chemical risk assessment in the Tier 2 assessment. A persistent, bioaccumulative and toxic (PBT) assessment was conducted because of specific concerns for substances that can be shown to persist for long periods in the environment, bioaccumulate in food chains and that can give rise to toxic effects after a longer time and over a greater spatial scale than chemicals without these properties.

Furthermore, a regulatory review was conducted to determine if the chemicals were identified as potential chemicals of concern in AICIS or NICNAS. Additional information is provided in the risk assessment dossiers (**Appendix C**) and safety data sheets (SDSs) (**Appendix D**) for the compiled hydraulic fracturing fluid systems. This information can be used for emergency responders, health and safety managers and environmental hazard clean-up teams.

As per the DEPWS Environment Management Plan Content Guideline (DEPWS, 2021), the Tier 1 assessment included the following:

- Name of chemical;
- Chemical purpose;
- Chemical Abstract Service (CAS) number;
- Total mass in kg;
- Approximate downhole concentration for that chemical expressed in mg/L;

- Appropriate ecotoxicity (aquatic and oral values) data including for acute lethal concentration 50 / effect concentration 50 (LC50/EC50) and chronic no observed effect concentration (NOEC) data where available; and
- Information on the biodegradation and bioaccumulation potential of organic chemicals.

The results of the Tier 1 assessment for the hydraulic fracturing fluid system formulations noting which chemical additives were assessed, the information used for the assessment and the chemicals categorised as Tier 1 or Tier 2, is presented in **Table 1** (attached). **Table 1** also includes discussion on Tier 1 assessment findings and whether a chemical was retained for further evaluation in the Tier 2 assessment. Observed recovery of drilling, well development and hydraulic fracturing fluids chemicals in flowback from other regional operators of oil and gas petroleum tenements is approximately 20 percent or less of the injected fluid chemical concentration. The concentration declines have been attributed to dilution by pore water within the shales, sorption, complexation and decay (bio-decay, hydrolysis). For the purposes of the Tier 1 and Tier 2 assessments, the higher injected fluid concentrations have been considered.

The following general approach was used to screen the constituents of potential concern (COPCs):

- A chemical was identified by AICIS (2022) or NICNAS (NICNAS, 2017a; NICNAS, 2017b) as a chemical of low concern, the PBT assessment did not identify a PBT substance and no human health hazard was identified; therefore, a Tier 2 assessment was deemed not to be warranted.
- If the chemical was not categorised by NICNAS as a chemical of low concern (either because it needed further evaluation or was not included in the 2017 NICNAS assessment) but was not a PBT substance and no human health hazard was identified, then a Tier 2 assessment was deemed not to be warranted.
- If the chemical satisfied the toxicity criteria for the PBT assessment because of aquatic toxicity values or a human health hazard was identified, the potential for complete exposure pathways was then assessed to determine the potential for risk (an incomplete pathway precludes an exposure occurring and an associated potential risk). In this context, site setting and management protocols associated with the action were evaluated, and if the pathway was incomplete, a Tier 2 assessment was not deemed to be warranted. Key controls limiting the potential for exposure included:
    - Implementation of the management controls within the EMP, which ensures the well site is located away from surface water (the current location is greater than 10 km away from the mapped watercourse, precluding a surface release from impacting surface water).
    - Maintenance of access control restrictions during hydraulic fracturing activities that will prevent access by the public, livestock and large native fauna.
    - Australia SafeWork Place and Condor Occupational Safety Guidance will be used to minimise human health exposure.

The outcome of the Tier 1 assessment identified the chemicals of low human health and environmental concern. Based on this outcome, no further management or mitigation is considered necessary for the majority of the chemicals. The following section presents the chemical(s) that could potentially pose significant hazards or risks evaluated in the Tier 2 Assessment.

2.3 Tier 2 Assessment

Of the chemicals evaluated for the hydraulic fracturing system formulation, glutaraldehyde (CAS number 111-30-8) was carried through to Tier 2 assessment. Chemicals identified in the Tier 1 assessment with a high ecotoxicity hazard assessment and therefore having a potential avian wildlife

exposure to fluids stored in treatment tanks were carried through to a Tier 2 assessment. Glutaraldehyde (CAS number 111-30-8) satisfied this criterion and had the requisite toxicity data for a Tier 2 assessment. No chemicals were identified in the Tier 1 assessment for a human health Tier 2 assessment.

2.3.1 Avian Wildlife

Potential exposure to selected chemical additives and/or flowback in treatment tanks by avian wildlife was assessed for representative avian species. **Appendix E** presents the outcomes of the Tier 2 assessment for this chemical (glutaraldehyde [CAS number 111-30-8]).

The potential exposure pathway for avian wildlife was assessed based on the potential ingestion of waters containing the selected chemicals (including flowback) from treatment tanks that were used for storage during the hydraulic fracturing activities of approximately three weeks. If a chemical was included in multiple fluid systems (e.g., glutaraldehyde), the maximum injected concentration (present in any of the fluid systems) was used in the Tier 2 assessment. Potential dietary intake of water containing these chemicals was compared to toxicity reference values developed specifically for avian wildlife to estimate a hazard quotient; a potential hazard quotient threshold level less than 1 indicates there are no unacceptable exposures to the avian species.

The hazard quotient for all the assessed avian species was orders of magnitude less than the threshold hazard quotient level of 1 (**Appendix E**). Therefore, there were no unacceptable exposures to the avian species. In addition, as a further conservative consideration, even if the potential exposure period is expanded to one year, the hazard quotient for the assessed avian species still will be orders of magnitude less than the threshold hazard quotient level of 1.

3 Summary and Risk Management

The goal of the CRA was to demonstrate that potential risks associated with hydraulic stimulation chemicals proposed for use by Condor across Tamboran's EP98, EP117 and EP76 tenements have been eliminated or reduced as much as is reasonably practicable to potentially exposed human and ecological receptors.

The life cycle of the hydraulic stimulation fluid system chemicals was assessed specifically for hydraulic stimulation operations and included:

- Activities associated with hydraulic stimulation chemical mixing and use at the well pad; and
- Management of flowback water (i.e., stored on-site) during or after the completion of hydraulic stimulation activities at the well pad.

The hydraulic stimulation chemicals within the life cycle (i.e., mixing, usage and storage) may result in potential exposure to human receptors and the environment through accidental releases. These potential releases, whilst unexpected, are considered to have a very low probability of occurrence and are constrained by the EMP requirements to managing risk, existing legislative requirements and the ongoing mitigating of potential impacts.

Condor and Tamboran have developed and implemented a range of systems and plans to control the transportation and storage of chemicals during field development and operational activities. This includes personnel induction and training, effective traffic management and routing to minimise the potential for accidents and spill management planning and response equipment. These systems and processes are considered effective in lowering the probability of occurrence of consequence associated with transportation incidents.

The human health and ecological hazard mitigation information provided in the chemical risk assessment dossiers and SDSs primarily focuses on safe handling, transportation and worker protection.

Based on the outcomes of this assessment, no further management controls are considered necessary.

4 Limitations

EHS Support has prepared this report in accordance with the usual care and thoroughness of the consulting profession for the use of Condor and only those third parties who have been authorised in writing by EHS to rely on the report. It is based on generally accepted practices and standards at the time it was prepared. No other warranty, expressed or implied, is made as to the professional advice included in this report. It is prepared in accordance with the scope of work and for the purpose outlined in the Proposal email dated 2 August 2022 and subsequent emails.

The methodology adopted and sources of information used by EHS Support are outlined in this report. EHS Support has made no independent verification of this information beyond the agreed scope of works, and EHS Support assumes no responsibility for any inaccuracies or omissions. No indications were found during our investigations that the information contained in this report as provided to EHS Support was false.

This report was prepared through January 2023 and is based on the information reviewed at the time of preparation. EHS Support disclaims responsibility for any changes that may have occurred after this time.

This report should be read in full. No responsibility is accepted for the use of any part of this report in any other context or for any other purpose, or by third parties. This report does not purport to give legal advice. Legal advice can only be given by qualified legal practitioners.

5 References

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Tables

Table 1
Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Ethoxylated branched C13 alcohol	78330-21-9	926	4894	166.43	**Aquatic Toxicity** Freshwater fish: 2 species, 720 to 1,500 µg/L. Freshwater crustaceans: 2 species, 590 to 860 µg/L. Freshwater rotifers: 1 species, *Brachionus calyciflorus* , 1,300 µg/L Freshwater algae, diatoms and blue-green algae: 6 species, 200 to 8,700 µg/L. Freshwater mesocosms: 4 NOEC data for multiple species tests were 80, 80, 320, and 330 µg/L, although replication was insufficient to meet OECD (1992) requirements. Normalised data were 380, 380, 320, and 1,520 µg/L. **Chronic Toxicity** -No studies available Freshwater fish: 2 species, 720 to 1,500 micrograms per litre (µg/L). Freshwater crustaceans: 2 species, 590 to 860 µg/L. Freshwater rotifers: 1 species, Bra*chionus calyciflorus, 1* ,300 µg/L Freshwater algae, diatoms and blue-green algae: 6 species, 200 to 8,700 µg/L. Freshwater mesocosms: 4 NOEC data for multiple species tests were 80, 80, 320, and 330 µg/L, although replication was insufficient to meet OECD (1992) requirements. Normalised data were 380, 380, 320, and 1,520 µg/L. **Terrestrial Toxicity** -No studies are available **$PNEC_{water}$** . 0.14 mg/L **$PNEC_{sediment}$** . 11.95 mg/kg sediment wet weight **$PNEC_{soil}$** . 10.54 mg/kg soil dry weight	Qualitative Assessment: Human Health Hazard -Low concern Ecological Hazard - moderate toxicity PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Property: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Property: BCF for AEs have a reported range of <5-387.5 PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Ethoxylated branched C13 alcohol is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Sodium thiosulphate	7772-98-7	1690	8050	150.00	**Aquatic Toxicity** **Acute Aquatic** -96-hr LC_{50} *Lepomis macrochirus* - 510 mg/L -96-hr LC_{50} *Salmo gairdneri* - 770 mg/L -72-hr EC_{50} *Pseudokirchneriella subcapitata* - >100 mg/L -48-hr EC_{50} *Daphnia magna* - 230 mg/L **Chronic Studies** - No data are available. **Terrestrial Toxicity** - No studies are available **$PNEC_{water}$** - 1.0 mg/L **$PNEC_{soil}$** - No data available	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Dissociates completely in aqueous media PBT Assessment: Does not meet the criteria for biodegradation.	Environmental Fate Properties: Dissociates to ions that are ubiquitous in environment PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Sodium thiosulphate not a PBT substance. Qualitative assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical dissociates completely in aqueous media to ions that are ubiquitous in the environment. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Sodium sulphate	7757-82-6	2700	2466	28.76	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Pimephales promelas* 7,960 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} - *Daphnia magna* - 4,736 mg/L **Chronic Aquatic - Invertebrate** -7-day - $LOEC_{50}$ - *Ceriodapnia dubia* 1,109 mg/L **Terrestrial Toxicity** No data available **$PNEC_{water}$** - 11 mg/L **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Dissociates completely in aqueous media PBT Assessment: Biodegradation is not applicable to these inorganic ions.	Environmental Fate Properties: Will dissociate to sodium and sulfate ions which are not expected to bioaccumulate. PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that sodium sulphate is not a PBT substance. Qualitative assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical dissociates completely in aqueous media to ions that are ubiquitous in the environment. It does not bioaccumulate. Human health hazards and ecological hazards are of low concern. Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

Table 1
Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Sodium Sulphite	7757-83-7	2630	2088	25.00	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} Golden Orfe - 316 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - 89* (59) mg/L -72-hr EC_{50} *Desmodesmus subspicatus* - 43.8* (29)mg/L * test substance sodium disulphite; adjusted concentration for sodium sulphite in parentheses **Chronic Toxicity** -34-day NOEC Zebrafish >316 mg/L. -21-day NOEC *Daphnia magna* >10* (6.6) mg/L -EC_{10} *Desmodesmus subspicatus* 33.3* (22) mg/L * test substance sodium disulphite; adjusted concentration for sodium sulphite in parentheses **Terrestrial Toxicity** No adequate studies were located. **$PNEC_{water}$** - 0.7 mg/L (NOEC for invertebrates) **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - low concern. PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Dissociates completely in aqueous media PBT Assessment: Does not meet the criteria for biodegradation.	Environmental Fate Properties: Dissociates to ions that are ubiquitous in environment PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Sodium Sulphite is not a PBT substance. Qualitative assessment indicated low concern to human health and to aquatic life. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical dissociates completely in aqueous media to ions that are ubiquitous in the environment. It does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Ethylene glycol	107-21-1	1110	5509	156.29	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Pimephales promelas* - >72,860 mg/L -96-hr LC_{50} *Oncorhynchus mykiss* - 22,810 mg/L and 24,591 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - >100 mg/L, 46,300 mg/L (20°C), 51,000 mg/L (24°C) -48-hr EC50 *Ceriodaphnia dubia-affinis* - 25,800 mg/L (20°C), 10,000 mg/L (24°C) **Acute Aquatic - Algae and other aquatic plants** -96-hr IC_{50} *Selenastrum capricornutum* - 10,940 mg/L -96-hr NOEC *Selenastrum capricornutum* - 10,000 mg/L **Chronic Aquatic - Fish** -7-day NOEC *Pimephales promelas* - 15,380 mg/L **Chronic Aquatic - Invertebrate** -7-day NOEC *Ceriodaphnia dubia* - 8,590 mg/L **Chronic Aquatic - Algae** -72-hr NOEC *Pseudokirchneriella subcapitata* - >100 mg/L **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - 10 mg/L **$PNEC_{soil}$** - 0.13 mg/kg soil dry weight (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - Repeated exposures may cause kidney toxicity Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Property: Readily biodegradable. PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: -Calculated log Kow is -1.36 -BCF in golden ide (*Leuciscus idus melanotus*) after 3 days exposure was 10 PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that ethylene glycol is not a PBT substance. Qualitative Assessment indicated potential hazard to human health (e.g., kidney toxicity). The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Australia WorkSafe and Condor Occupational Health & Safety procedures will be used to minimise human health exposure. Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

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Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Glutaraldehyde	111-30-8	1130	16871	470.14	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} Bluegill Sunfish - 13 mg/L -96-hr LC_{50} *Oncorhynchus mykiss* - 10 mg/L **Acute Aquatic - Invertebrate** -48-hr LC_{50} *Daphnia magna* - 14.87 mg/L and 14 mg/L **Acute Aquatic - Algae and other aquatic plants** -72-hr EC_{50} *Scenedesmus subspicatus* - 0.375 mg/L (biomass), 0.6 (growth rate), 0.025 (NOEC) -72-hr EC_{50} *Scenedesmus subspicatus* - 0.92 mg/L (biomass), 0.61 (growth rate), 0.33 (NOEC) -72-hr EC_{50} *Scenedesmus subspicatus* - 0.61 mg/L (growth rate) **Chronic Aquatic - Fish** -97-day LOEC *Oncorhynchus mykiss* - 5 mg/L -97-day NOEC *Oncorhynchus mykiss* - 1.6 mg/L **Chronic Aquatic - Invertebrate** -21-day NOEC *Daphnia magna* - 5 mg/L **Terrestrial Toxicity** **Earthworms** -14-day LC50 - >500 mg/kg soil dry weight **Soil microoganisms** -28-day EC50 - 360 mg/kg soil dry weight - > 593 mg/kg soil dry weight -28-day EC10 - 1.5 mg/kg soil dry weight - 11.5 mg/kg soil dry weight **Avian** -single dose (oral gavage) LC50 Mallard duck - 206 mg/kg -5-day dietary NOEC - Mallard duck - >2,500 ppm **Terrestrial Plants:** -19-day EC_{50} - *Avena sativa* (oats) - >1,000 mg/kg soil dry weight; NOEC - ≥1000 (emergence rate, dry matter, shoot length) -19-day EC_{50} - *Brassica napus* (rapeseed) - >1,000 mg/kg soil dry weight; NOEC - ≥1000 (emergence rate), 500 (dry matter), 250 (shoot length) -19-day EC_{50} - *Vicia sativa* (vetch) - >1,000 mg/kg soil dry weight (emergence rate and shoot length); 901 mg/kg soil dry weight based on dry weight; NOEC - ≥1000 (emergence rate), 125 (dry matter), 125 (shoot length) **$PNEC_{water}$** - 0.0025 mg/L (Chronic algae) **$PNEC_{soil}$** -1.5 mg/kg soil dry weight (Chronic soil organisms)	Qualitative Assessment: Human Health Hazard - Corrosive; skin/respiratory sensitizer Ecological Hazard - Very toxic to aquatic life with long lasting effects. Moderatly toxic to birds on acute basis. PBT Assessment: Substance exhibits higher toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable. PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Property: The log Kow at different pH values range from -0.36-0.80. PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 2	NICNAS Assessment (2018) Human Health - potentially harmful to public health in event of transport spill. - potentially harmful to workers health in event of industrial incident Environment -Potentially harmful to the environment in the event of transport spill PBT Assessment: The overall conclusion is that glutaraldehyde is not a PBT substance. Qualitative Assessment indicated potential hazard to human health (e.g., skin irritant). The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical and does meet the screening criteria for toxicity. This chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted for aquatic receptors. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Australia SafeWork Place and Condor Occupational Safety Guidance will be used to minimise human health exposure. Therefore, a Tier 2 assessment was not warranted for human receptors. Chemicals with a high ecotoxicity hazard assessment have a potential avian wildlife exposure to chemicals stored in treatment tanks. Therefore a Tier 2 assessment was conducted for avian receptors.	A quantitative risk characterisation was used to assess the risk to avian receptors from potential exposure to gluteraldehyde (**Appendix E**). There were no unacceptable potential risks to avian receptors as a result of ingestion of waters stored in treatment tanks.
Diammonium peroxidisulphate	7727-54-0	1260	9388	234.63	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Oncorhynchus mykiss* - 76.3 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - 120 mg/L **Acute Aquatic - Algae** -72-hr EC_{50} *Phaedactylum tricornutum* - 320 mg/L **Chronic Aquatic - Invertebrate** -21-day NOEC *Daphnia magna* 20.8 mg/L **Terrestrial Toxicity** No terrestrial toxicity studies identified. **$PNEC_{water}$** - 0.4 mg/L **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - Moderate concern (irigating to eyes, skin, and respiratory tract.) Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Dissociates completely in aqueous media PBT Assessment: Not applicable	Environmental Fate Properties: Inorganic salt that dissolves to respective cations and anions. PBT Assessment: Does not meet the screening criteria for bioaccumulation. Diammonium peroxidisulphate is not a PBT substance	Tier 1 (NICNAS/ Qualitative/PBT/Exposure Assessment)	NICNAS has assessed diammonium peroxidisulphate in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment. PBT Assessment: The overall conclusion is that sodium diammonium peroxodisulphate is not a PBT substance. Qualitative assessment indicated moderate potential human hazard Moderate concern (iritating to eyes, skin, and respiratory tract). The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical dissociates completely in aqueous media to its respective cations and anions. It does not bioaccumulate Therefore, a Tier 2 assessment was conducted for potential exposures to humans. Management: Australia SafeWork Place and Condor Occupational Safety Guidance will be used to minimise human health exposure. Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

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Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Hydrotreated light petroleum distillate	64742-47-8	850	46096	1707.75	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LL_{50} *Oncorhynchus mykiss* - 2-5 mg/L -96-hr NOEL *Oncorhynchus mykiss* - 2.0 mg/L **Acute Aquatic - Invertebrate** -48-hr EL_{50} *Daphnia magna* - 1.4 mg/L -48-hr NOEL *Daphnia magna* - 0.3 mg/L **Acute Aquatic - Algae** -72-hr EC_{50} *Raphidocelis subcapitata* - <1-3 (average value of 2) mg/L -72-hr EC_{50} *Selenastrum capricornutum* - 3.7 mg/L **Chronic Aquatic - Invertebrate** -21-day NOEL *Daphnia magna* 0.48 mg/L -21-day EL_{50} (reproduction)- 0.89 mg/L **Terrestrial Toxicity** No terrestrial toxicity studies identified. **$PNEC_{water}$** - 0.005 mg/L **$PNEC_{soil}$** - 0.32 mg/kg dry weight	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - Low toxicity PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Inherently biodegradable PBT Assessment: Does not meet the screening criteria for persistence	Environmental Fate Property: EPISUITE estimated BCF = 3.162 L/kg wet-weight PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that hydrotreated light petroleum distillate is not a PBT substance. Qualitative assessment indicated low concern for human and ecological hazards. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. This chemical is inherently biodegradable and does not meet the PBT assessment criteria for toxicity or bioaccumulation. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Australia SafeWork Place and Condor Occupational Safety Guidance will be used to minimise human health exposure. Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Guar Gum	9000-30-0	NA	NA	476.80	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Oncorhynchus mykiss* - 218 mg/L **Acute Aquatic - Invertebrate** -48-hr LC_{50} *Daphnia magna* - 42 mg/L -96-hr LC_{50} *Daphnia magna* - <6.2 mg/L **Chronic Aquatic** -No chronic studies available **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - 0.006 mg/L (Acute *Daphnia*) **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - Low concern to fish, moderate acute toxicity to invertebrates PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable. PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: Not expected to bioaccumulate. PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (NICNAS/ PBT/ Exposure Assessment)	NICNAS Assessment (2018) Human Health - unlikely to cause harm to public - unlikely to cause harm to workers Environment -Potentially harmful to the environment in the event of transport spill NICNAS: Identified as chemical of low concern for human health in National assessment of chemicals associated with coal seam gas extraction in Australia, Tech Report Number 11 (NICNAS, 2017) PBT Assessment - The overall conclusion is that guar gum is not a PBT substance. Qualitative assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable, is not expected to bioaccumulate, and does not meet the PBT criteria for toxicty. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

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Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Quartz or Organophillic phyllosilicate	14808-60-7	NA	NA	34.13	**Aquatic and Terrestrial Toxicity** -No studies are available. -Expected to be low concern for toxicity to aquatic organisms. **$PNEC_{water}$** - not derived **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Hazard: Inhalation: silicosis and lung cancer in humans. Oral/dermal: low concern. Ecological Hazard: Low concern PBT Assessment: Substance exhibits higher toxicity than that established by regulatory guidance.	Environmental Fate Properties: Not relevant PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: inorganic complex not expected to bioaccumulate PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative Assessment/ PBT)	PBT Assessment: The overall conclusion is that Crystalline silica, quartz is not a PBT substance. Qualitative Assessment indicated hazardous to human health by the inhalation pathway; not hazardous by the oral/dermal route. Management: Australia WorkSafe and Condor Occupational Health & Safety procedures will be used to minimise human health exposure. Therefore a Tier 2 Assessment is not warranted.	NA
Hydrochloric acid	7647-01-0	1190	53211	1408.10	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Lepomis macrochirus* - pH 3.25-3.5 (20 mg/L) **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - pH 4.92 (0.45 mg/L) **Acute Aquatic - Algae and other aquatic plants** -72-hr EC_{50} *Chlorella vulgaris* - pH 4.7 (growth rate) (0.73 m/L) 72-hr EC_{10} *Chlorella vulgaris* - pH 4.7 (0.364 mg/L) **Chronic Aquatic** -No chronic studies available **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - not derived **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - Corrosive; respiratory irritant Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Dissociates completely PBT Assessment: Not applicable.	Environmental Fate Property: Not expected to bioaccumulate. PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (NICNAS/ Qualitative Assessment/ PBT)	NICNAS Assessment (2018) Human Health - unlikely to cause harm to public - potentially harmful to workers health in event of industrial incident Environment -Potentially harmful to the environment in the event of transport spill PBT Assessment - The overall conclusion is that hydrochloric acid is not a PBT substance. Qualitative Assessment indicated potential hazard to human health (e.g., corrosive). Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Australia SafeWork Place and Condor Occupational Safety Guidance will be used to minimise human health exposure. Therefore, a Tier 2 assessment was not warranted.	NA
Citric acid	77-92-9	1670	26516	500.0	**Aquatic Toxicity** **Acute Aquatic - Fish** -48-hr LC_{50} *Leuciscus idus melanotus* (golden orfe) - 440 mg/L and 760 mg/L -96-hr LC_{50} *Lepomis macrochirus* (fathead minnow)- >100 mg/L **Acute Aquatic - Invertebrate** -24-hr EC_{50} *Daphnia magna* - 85 mg/L (un-neutralised test solution) 1,535 mg/L in neutralised solution **Acute Aquatic - Algae and other aquatic plants** -8-day EC_0 *Scenedesmus quadricauda* - 640 mg/L **Chronic Aquatic** -No chronic studies available **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - 0.44 mg/L (Acute Daphnia) **$PNEC_{soil}$** - 0.002 mg/kg soil dry weight (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard -Eye irritant Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: log K_{ow} is -1.61 to -1.80 PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (NICNAS/ PBT/ Exposure Assessment)	NICNAS: Identified as chemical of low concern for human health in National assessment of chemicals associated with coal seam gas extraction in Australia, Tech Report Number 11 (NICNAS, 2017) Qualitative Assessment indicated potential hazard to human health (e.g., eye irritant). PBT Assessment: The overall conclusion is that citric acid is not a PBT substance. The estimated injected concentration did exceed the PNECs for this chemical. This chemical is readily biodegradable, does not bioaccumulate, and does not meet the PBT assessment criteria for toxicity. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Australia WorkSafe and Condor Occupational Health & Safety procedures will be used to minimise human health exposure.	NA

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Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Acetic acid	64-19-7	1040	16513	500.0	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Oncorhynchus mykiss* - (test substance potassium acetate) >300.82 mg/L (as acetate ion) -96-hr LC_{50} *Danio rerio* - (test substance potassium acetate) >300.82 mg/L (as acetate ion) -96-hr LC_{50} *Oncorhynchus mykiss* - (test substance acetic acid) 64.8 mg/L (measured) -96-hr LC_{50} *Oncorhynchus mykiss* - (test substance acetic acid) 31.3 mg/L - 67.6 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - (test substance potassium acetate) >300.82 mg/L (as acetate ion) -48-hr EC_{50} *Daphnia magna* - (test substance acetic acid) 79.5 mg/L (measured) -48-hr EC_{50} *Daphnia magna* - (test substance acetic acid) 18.9 mg/L (measured) **Acute Aquatic - Algae and other aquatic plants** -72-hr EC_{50} *Desmodesmus subspicatus* - 486.5 mg/L **Chronic Aquatic - Fish** -21-day *Oncorhynchus mykiss* study - measured NOEC 57.2 mg/L (60% acetic acid) and 34.3 mg/L (100% acetic acid) **Chronic Aquatic - Invertebrate** -21-day *Daphnia magna* reproduction study measured NOEC 80 mg/L (60% acetic acid) and 31.4 mg/L (100% acetic acid) -21-day *Daphnia magna* reproduction study measured NOEC 22.7 mg/L (100% acetic acid) **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - 3.0 mg/L (E(L)C50 test fish or *Dapnhia magna*) **$PNEC_{soil}$** - 0.04 mg/kg soil dry weight (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - Corrosive, respiratory irritant Ecological Hazard - moderate actute toxicity to aquatic organisms. PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Hazard Assessment: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Property: Low K_{ow} is -0.17 PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (NICNAS/ PBT/ Exposure Assessment)	NICNAS Assessment (2018) Human Health - potentially harmful to public health in event of transport spill. - potentially harmful to workers health in event of industrial incident Environment -unlikely to cause harm to environment PBT Assessment: The overall conclusion is that acetic acid is not a PBT substance. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable, does not bioaccumulate, and does not meet the PBT criteria for toxicity to aquatic organisms. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Australia WorkSafe and Condor Occupational Health & Safety procedures will be used to minimise human health exposure. Therefore, a Tier 2 assessment was not warranted.	NA
Isopropanol	67-63-0	800	67	2.6	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Pimpephales promelas* - 9,640 mg/L **Acute Aquatic - Invertebrate** -24-hr EC_{50} *Daphnia magna* >10,000 mg/L **Chronic Aquatic - Invertebrate** -16 day NOEC *Daphnia magna* 141 mg/L -21 day NOEC *Daphnia magna* 30 mg/L -7-day NOEC *Scenedesmus quadricauda* is 1,800 mg/L **Terrestrial Toxicity** -EC_{50} lettuce seed germination test - 2,100 mg/L **$PNEC_{water}$** - 0.3 mg/L **$PNEC_{soil}$** - 0.014 mg/kg soil dry weight (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard -Low concern Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: Bioaccumulation of isopropanol is not expected to occur based on its log K_{ow} value of 0.05 and its calculated BCF value of 1. PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative Assessment/PBT)	PBT Assessment: The overall conclusion is that isopropanol is not a PBT substance. Qualitative assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and does not bioaccumulate. Therefore, a Tier 2 assessment was not warranted. Management: mplementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Alcohols, C12-16, ethoxylated	68551-12-2	930	53	1.79	**Aquatic Toxicity** Toxicity values (NOECs) utilised in development of ANZECC water quality guideline for alcohol ethoxylates: -Freshwater fish: 2 species, 720 to 1,500 µg/L -Freshwater crustaceans: 2 species, 590 to 860 µg/L. -Freshwater rotifers: 1 species, *Brachionus calyciflorus* , 1,300 µg/L -Freshwater algae, diatoms and blue-green algae: 6 species, 200 to 8,700 µg/L. - Freshwater mesocosms: 4 NOEC data for multiple species tests were 80, 80, 320, and 330µg/L, although replication was insufficient to meet OECD (1992) requirements. Normalised data were 380, 380, 320, and 1,520 µg/L **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - 0.140 mg/L (ANZECC Water Quality Guideline for alcohol ethoxyates) **$PNEC_{soil}$** - 0.0 to 10.7 mg/kg dry weight soil (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - Harmful to aquatic life with long lasting effects PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Property: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Property: Log K_{ow} range from <5 to 387.5 PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/ PBT/ Exposure Assessment)	PBT Assessment: The overall conclusion is that Alcohols, C12-16, ethoxylated is not a PBT substance. Qualitative Assessment indicated low concern to human health; however harmful effects to aquatic life. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

Table 1
Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Cinnamaldehyde	104-55-2	1041	59	1.79	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Brachydanio rerio* - >3.9 mg/L to < 5.5 mg/L -96-hr LC_{50} *Poecilia reticulata* - >3.5 mg/L to < 6.5 mg/L - 96 hr LC_{50} - *Lepomis machrochirus* - > 20 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - 3.21 mg/L to 11.5 mg/L **Acute Aquatic - Algae and other aquatic plants** -72-hr EC_{50} *Desmodesmus subspicatus* - 31.6 mg/L -72-hr EC_{50} *Chlorella vulgaris* - 16.09 mg/L **Chronic Toxicity** -21-day EC_{50} - *Daphnia magna* - 0.402 mg/L -28-day NOEC - estimated for fish - 15.159 mg/L **Terrestrial Toxicity** -5-day LOEL - *Colinus virginianus* - 1% w/w **$PNEC_{water}$** - 0.152 mg/L (chronic fish) **$PNEC_{soil}$** - 0.075 mg/kg dry weight soil (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard -Skin/eye irritant; skin sensitizer Ecological Hazard - Toxic to aquatic life PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable. PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Property: log K_{ow} is 2.107 PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative Assessment/ PBT/ Exposure Assessment)	PBT Assessment: The overall conclusion is that cinnamaldehyde is not a PBT substance. Qualitative Assessment indicated potential hazard to human health (e.g., skin irritant). The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. This chemical is readily biodegradable, does not bioaccumulate, and does not meet the PBT assessment criteria for toxicity. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Australia WorkSafe and Condor Occupational Health & Safety procedures will be used to minimise human health exposure.	NA
Methanol	67-56-1	790	1	0.06	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} Bluegill - 15,400 mg/L -96-hr LC_{50} *Salmo gairdneri* - 20,100 mg/L -96-hr LC_{50} *Pimphales promelas* - 28,100 mg/L **Acute Aquatic - Invertebrate** -96-hr EC_{50} *Daphnia magna* - 18,620 mg/L -48-hr EC_{50} *Daphnia magna* - >10,000 mg/L **Acute Aquatic - Algae and other aquatic plants** -96-hr EC_{50} *Selenastrum capricornutum* -~22,000 mg/L -10-14 d EC_{50} *Chlorella pyrenoidosa* - 28,400 mg/L **Chronic Aquatic** -No chronic studies available **Terrestrial Toxicity** 35-d EC_{50} Earthworm *Eisenia fetida* - 17,199 mg/kg soil dry weight 63-d EC_{50} Earthworm *Eisenia fetida* - 26,646 mg/kg soil dry weight 28-d EC_{25} *Folsomia candida* - 2,842 mg/kg soil dry weight (test results) 28-d NOEC (reproduction) *Folsomia candida* - 1,000 mg/kg soil dry weight (derived graphically) 14-d EC_{50} *Hordeum vulgare* - 15,492 mg/kg soil dry weight 14-d NOEC (seedline emergence) *Hordeum vulgare* - 12,000 mg/kg soil dry weight (derived graphically) 14-d EC_{25} *Hordeum vulgare* - 2,538 mg/kg soil dry weight (test results) 14-d NOEC (shoot dry mass) *Hordeum vulgare* - 1,555 mg/kg soil dry weight (derived graphically) 14-d EC_{25} *Hordeum vulgare* - 2,823 mg/kg soil dw (test results) 14-d NOEC (root dry mass) *Hordeum vulgare* - 2,592 mg/kg soil dw (derived graphically) 14-d EC_{25} *Hordeum vulgare* - 4,885 mg/kg soil dw (test results) 14-d NOEC (shoot length) *Hordeum vulgare* - 2,592 mg/kg soil dw (derived graphically) 14-d EC_{25} *Hordeum vulgare* - 5,752 mg/kg soil dw (test results) 14-d NOEC (rott length length) *Hordeum vulgare* - 4,320 mg/kg soil dw (derived graphically) **$PNEC_{water}$** - 10 mg/L (Acute *Daphnia)* **$PNEC_{soil}$** - 100 mg/kg soil dry weight (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - Low concern if used at <3% Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: -Calculated log K_{ow} - 1.36 -BCF in *Cyprinus carpio* 1.0, BCF *Leuciscus idus* <10 PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (NICNAS/ Qualitative Assessment/ PBT)	NICNAS Assessment (2018) Human Health - potentially harmful to public in event of transport spill or pond leak - potentially harmful to workers when mixing and/or cleaning or in event of industrial accident Environment -unlikely to cause harm to environment PBT Assessment: The overall conclusion is that methanol is not a PBT substance. Qualitative assessment indicated low concern to human health The estimated injected concentration did not exceed the ecotoxicity values or PNECs for this chemical. This chemical is readily biodegradable, does not bioaccumulate, and does not meet the PBT assessment criteria for toxicity. Therefore, a Tier 2 assessment was not warranted. Management: Australia SafeWork Place and Condor Occupational Safety Guidance will be used to minimise human health exposure. Therefore, a Tier 2 assessment was not warranted.	NA

Table 1
Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Sodium (C14-16) olefin sulfonate	68439-57-6	1054	4910	146.68	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Danio rerio* (Zebra Fish) - 4.2 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Ceriodaphnia dubia* - 4.53 mg/L **Acute Aquatic - Algae** -72-hr EC_{50} *Skeletonema costatum* - 5.2 mg/L **Chronic Aquatic - Invertebrate** -21-day NOEC *Daphnia magna* -6.3 mg/L -21-day NOEC *Daphnia magna* -2.42 mg/L **$PNEC_{water}$** - 0.08 mg/L (Acute Fish) **$PNEC_{soil}$** - 0.002 mg/kg dry weight soil (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: -Measured log K_{ow} -1.3 PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Sodium (C14-16) olefin sulfonate is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Diisobutyl glutarate	71195-64-7	966	606	19.75	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Oryzias latipes* - 3.7 mg/L **Acute Aquatic - Invertebrate** -24-hr LC_{50} *Daphnia magna* - 17 mg/L **Acute Aquatic - Algae** -72-hr EC_{50} *Selenastrum sp* . - 2.8 mg/L **Chronic Aquatic - Invertebrate** -21-day NOEC *Daphnia magna* 5.6 mg/L **Chronic- Algae** -72-hr NOEC *Selenastrum capricornutum* - 2mg/L **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - 0.04 mg/L (Acute Fish) **$PNEC_{soil}$** - 0.13 mg/kg dry weight soil (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: -Measured log K_{ow} -4.3 PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Diisobutyl glutarate is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Diisobutyl succinate	925-06-4	978	204	6.58	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Oryzias latipes* - 3.7 mg/L **Acute Aquatic - Invertebrate** -24-hr LC_{50} *Daphnia magna* - 17 mg/L **Acute Aquatic - Algae** -72-hr EC_{50} *Selenastrum sp* . - 2.8 mg/L **Chronic Aquatic - Invertebrate** -21-day NOEC *Daphnia magna* 5.6 mg/L **Chronic- Algae** -72hr NOEC *Selenastrum capricornutum* - 2 mg/L **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - 0.04 mg/L (Acute Fish) **$PNEC_{soil}$** - 0.13 mg/kg dry weight soil (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: -Measured log K_{ow} -4.3 PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Diisobutyl succinate is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

Table 1
Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Diisobutyl adipate	141-04-8	951	170	5.64	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Oryzias latipes* - 3.7 mg/L **Acute Aquatic - Invertebrate** -24-hr LC_{50} *Daphnia magna* - 17 mg/L **Acute Aquatic - Algae** -72-hr EC_{50} *Selenastrum sp* . - 2.8 mg/L **Chronic Aquatic - Invertebrate** -21-day NOEC *Daphnia magna* 5.6 mg/L **Chronic- Algae** -72hr NOEC *Selenastrum capricornutum* - 2 mg/L **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - 0.04 mg/L (Acute Fish) **$PNEC_{soil}$** - 0.13 mg/kg dry weight soil (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: -Measured log K_{ow} -4.3 PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Diisobutyl adipate is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and does not bioaccumulate. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Sodium Gluconate	527-07-1	1790	15351	270.07	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Oryzias latipes* - >100 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - >1000 mg/L **Acute Aquatic - Algae** -72-hr E_rC_{50} *Selenastrum capricornutum* - > 1000 mg/L **Chronic Aquatic - Algae** -72-hr NOEC *Selenastrum capricornutum* - 560 mg/L **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - 5.6 mg/L (Acute Fish) **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: -Measured log K_{ow} -5.99 PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Sodium Gluconate is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is of lo ecoloical concern, readily biodegradable, and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Boric Acid	10043-35-3	1489	6385	135.03	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} Fathead minnow - 79.7 mg/L **Acute Aquatic - Invertebrate** -96-hr LC_{50} *Legumia recta* (Black sandshell mussel) - 147 mg/L -96-hr LC_{50} *Hyalella azteca* -64 mg/L **Acute Aquatic - Algae** -72-hr EC_{50} *Pseudokirchneriella subcapitata* - 52.4 mg/L **Chronic Aquatic - Fish** -72-hr LC_{10} *P. promelas* - 3.5-47 mg B/L -72-hr LC_{10} freshwater fish- 21.6 mg B/L -34-day NOEC (Biomass) *Danio rerio* - 1.8 mg B/L -32-day NOEC (Mortality) *Pimephales promelas* - 11 mg B/L **Chronic Aquatic- Invertebrate** -14-day NOEC (Reproduction) *Daphnia magna* - 2.4 mg B/L **Chronic Aquatic - Algae** -72-hr NOEC *Pseudokirchneriella subcapitata* - 17.5 mg B/L -4-day NOEC (Growth) *Pseudokirchneriella subcapitata* -2.8 mg B/L **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - 940 µg/L (ANZG water quality guideline) **$PNEC_{soil}$** - 5.7 mg/kg soil dry weight	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Dissociates completely PBT Assessment: Not applicable for this inorganic compound	Environmental Fate Properties: -BCF <0.1-10.5 L/kg (reported for borates in fish and oysters). PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Boric Acid is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical dissociates completely and it does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

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Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Potassium Hydroxide	1310-58-3	2044	21963	338.37	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Gambusia affinis* (mosquito fish) - 80 mg/L -96-hr LC_{50} *Gambusia affinis* (mosquito fish)- 125 mg/L -24-hr LC_{50} *Carassius auratus* (goldfish)-160 mg/L -48-hr LC_{50} *Leuciscus idus melanotus* - 189 mg/L **Acute Aquatic - Invertebrate** -48-hr LC_{50} *Ceriodaphnia cf. dubia* - 40 mg/L **Chronic Toxicity** No studies available **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - not derived **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - Limited toxicity Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable-dissociates completely PBT Assessment: Criteria is not applicable for the inorganic ions.	Environmental Fate Properties: Essential to all living organisms. PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative Assessment/PBT)	PBT Assessment: The overall conclusion is that Potassium Hydroxide is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical dissociates completely and does not bioaccumulate. Management: No additional management required, Tier 1 screening satisfied.	NA
Mannanase	37288-54-3	1420	3	0.07	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hour LC50 *Oncorhynchus mykiss* (rainbow trout) - >105.8 mg/L **Acute Aquatic - Invertebrate** -48-hour EC50 *Daphnia Magna* - >105.8 mg/L **Acute Aquatic-Algae** -72-hour EC50 *Raphidocelis subcapitata* (green algae) - >105.8 mg/L **Chronic Toxicity** -72 hr NOEC - *Raphidocelis subcapitata* - 26.5 mg/L **Terrestrial Toxicity** No studies available **$PNEC_{water}$** - 0.139 mg/L **$PNEC_{soil}$** - 0.002 mg/kg soil dry weight (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - low concern Ecological Hazard - low acute and chronic toxicity PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence	Environmental Fate Properties: Log K_{ow} = -1.3 PBT Assessment: Does not meet the criteria for bioaccumulation	Tier 1 (Qualitative Assessment/PBT)	PBT Assessment: The overall conclusion is that Mannanase is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did not exceed the ecotoxicity values or PNECs for this chemical. In addition, this chemical is readily biodegradable and does not bioaccumulate. Management: No additional management required, Tier 1 screening satisfied.	NA
Sodium Bromate	7789-38-0	3339	168421	1588.39	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hour LC_{50} *Morone saxatillis* (striped bass) - 30.8 mg/L -48-hour LC_{50} *Morone saxatillis* (striped bass) - 605.0 mg/L -24-hour LC_{50} *Leiostomus xanthurus* -698.0 mg/L **Chronic Toxicity** -10-day LC_{50} *Morone saxatillis* (striped bass) - 92.6 mg/L -10-day LC_{50} *Leiostomus xanthurus* - 278.6 mg/L **Terrestrial Toxicity** No studies available **$PNEC_{water}$** - 0.308 mg/L **$PNEC_{soil}$** - NA	Qualitative Assessment: Human Health Hazard - moderate concern Ecological Hazard - low acute and chronic toxicity concern to aquatic organisms PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Dissociates completely PBT Assessment: Does not meet screening criteria for persistence	Environmental Fate Properties: Low K_{ow} value PBT Assessment: Does not meet the criteria for bioaccumulation	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Sodium bromate is not a PBT substance. Qualitative Assessment indicated moderate concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical dissociates completely and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

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Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Hepta sodium phosphonate	22042-96-2	1400	4446	100.00	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Gambusia affinis* (mosquito fish) - 180-252 (mean:216) mg active/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Chironomus tentans* - 7,589 mg active/L **Chronic Toxicity** -60-day NOEC *Oncorhynchus mykiss* - 25.6 mg active acid/L **Terrestrial Toxicity** -14-day LC_{50} Mallard duck (*Anas platyrhynchos*)- >454 mg/kg -14-day LC_{50} Bobwhite quail (*Colunus virginianus*)- >454 mg/kg **$PNEC_{water}$** - 0.31 mg DTPMP sodium salt/L **$PNEC_{soil}$** - 40 mg DTPMP sodium salt/kg soil dry	Qualitative Assessment: Human Health Hazard - Low toxicity Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Not readily biodegradable PBT Assessment: Does meet the screening criteria for persistence.	Environmental Fate Properties: BCF values in fish studies are <10 and <94 for concentrations 18.8 and 2.03 mg/L respectively PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative Assessment/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that hepta sodium phosphonate is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical and is not readily biodegradable. However, this chemical is of low ecological concern and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. No additional management required, Tier 1 screening satisfied.	NA
Polyoxyethylene nonylphenol ether	9016-45-9	1050	4690	140.65	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Pimephales promelas* - 0.218 mg/L -96-hr LC_{50} *Lepomis macrochirus* - 1.3 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Ceriodaphnia dubia* - 0.328 mg/L -48-hr LC_{50} *Daphnia* - 1.8 mg/L **Acute Aquatic- Algae** -48-hr EC_{50} *Pseudokirchneriella subcapitata* - 20-50 mg/L **Chronic Toxicity-Fish** -21-day NOEC *Oncorhynchus mykiss* - 0.048 mg/L -7-day NOEC *Ceriodaphnia dubia* - 0.285 mg/L **Chronic Toxicity- Invertebrates** -6-day NOEC *Daphnia Magna* - 1.0 mg/L **Chronic Toxicity-Algae** -96-hr NOEC *Pseudokirchneriella subcapitata* - 8 mg/L -120-hr (5 day) EC_{50} *Scenedesmus Opoliensis* - 37.4 mg/L **Terrestrial Toxicity** No data available. **$PNEC_{water}$**- 0.00096 mg/L **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - Low to moderate toxicity Ecological Hazard - moderate concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: BCF values in fish studies are <1.4 L/Kg PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative Assessment/PBT Exposure Assessment)	PBT Assessment: The overall conclusion is that Polyoxyethylene nonylphenol ether is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

Table 1
Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Quaternary ammonium compounds, bis(hydrogenated tallow alkyl)dimethyl, salts with bentonite	68953-58-2	NA	NA	140.65	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} Freshwater rainbow trout- > ca. 500 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - > 100 mg/L -96-hr EC_{50} *Daphnia magna* - 300 mg/L -48-hr EC_{50} *Daphnia magna* - <500 mg/L -72-hr EC_{50} *Skeletonema costatum* - 23.8 mg/L -72-hr EC_{50} *Skeletonema costatum* - 82.3 mg/L -72-hr EC_{50} *Skeletonema coastatum* - >1,000 mg/L **Acute Aquatic- Algae** -72-hr EC_{50} *Scenedesmus subspicatus* - >100 mg/L **Chronic Toxicity** -21-day NOEC *Daphnia magna* - 3.2 mg/L -72-hour NOEC *Scenedesmus subspicatus* - 100 mg/L **Terrestrial Toxicity** -14-day NOEC earthworms- 1000 mg/kg -14-day LC_{50} earthworms- >1000 mg/kg -EC_{50} *Tritium gestivum* - >100 mg/kg -EC_{50} *Raphanus sativus* ->100 mg/kg -NOEC *Tritium gestivum* - 100 mg/kg -NOEC *Raphanus sativus* - 100 mg/kg -LC_{50} *Lepidum sativum* - 9 mg/kg -LOEC *Lepidum sativum* - 1 mg/kg **$PNEC_{water}$** - 0.064 mg/L **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - Low toxicity Ecological Hazard - low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Not readily biodegradation PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: Insoluble in water and is not bioavailable PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Quaternary ammonium compounds, bis(hydrogenated tallow alkyl)dimethyl, salts with bentonite is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical and is not readily biodegradable. However, this chemical of low ecological conern, is insoluble in water and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
1,6-Hexanediol	629-11-8	960	429	14.06	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Leuciscus idus* - 4,460-10,000 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - >500 mg/L **Acute Aquatic-Algae** -72-hr EC_{50} *Desmodesmus subspicatus* - 5,940 mg/L **Chronic Toxicity** -72h EC_{10} *Desmodesmus subspicatus* - 1,180 mg/L -96h NOEC *Leuciscus idus* - 2,200 mg/L **Terrestrial Toxicity** -EC_{50} *Psudomonas putida* - >10,000 mg/L **$PNEC_{water}$** - 50 mg/L **$PNEC_{soil}$** - 0.67 mg/L	Qualitative Assessment: Human Health Hazard - Low toxicity Ecological Hazard - low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: Log K_{ow} is low. PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (NICNAS/ Qualitative Assessment/PBT/Exposure Assessment)	NICNAS has assessed sodium benzoate in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment (NICNAS, 2019). PBT Assessment: The overall conclusion is that 1,6 Hexanediol is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did not exceed the ecotoxicity values or PNECs for this chemical. In addition, this chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Sodium erythorbate	6381-77-7	1702	568	10.51	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Oncorhynchus mykiss* - >100 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - >100 mg/L **Acute Aquatic-Algae** -72-hr EC_{50} *Raphidocelis subcapitata* - >160 mg/L **Chronic Toxicity** - 72-hr NOEC - *Raphidocelis subcapitata* - 20 mg/L **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - 0.2 mg/L **$PNEC_{soil}$** - 0.027 mg/kg soil dry weight (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - low concern Ecological Hazard - low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Inherently Biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: -log K_{ow} - -3.29 and estunated BCF (0.8933). PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative Assessment/PBT)	PBT Assessment: The overall conclusion is that Sodium erythorbate is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is inherently biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

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Evaluation of Compiled List of Chemicals
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Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Ethoxylated Decanol	26183-52-8	880	17	0.6	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Cyprinus carpio and Danio rerio* - 1.2 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - 0.39 mg/L to 0.91 mg/L **Acute Aquatic-Algae** -72-hr EC_{50} *Desmodesmus subspicatus* - 0.18 mg/L to 1.8 mg/L (growth rate) **Chronic Toxicity** - 10-day NOEC *Lepomis macrochirus* - 0.16 mg/L -30-day NOEC *Lepomis macrochirus* - > 0.33 mg/L -21-day NOEC - *Daphnia magna* - 0.77 mg/L -72-hr NOEC *Desmodesmus subspicatus* - 0.4 mg/L **Terrestrial Toxicity** -NOEL *Eisenia fetida* - >1,000 mg/kg soil dry weight (acute toxicity) **$PNEC_{water}$** - 0.016 mg/L **$PNEC_{soil}$** - 1 mg/kg soil dry weight (acute toxicity study)	Qualitative Assessment: Human Health Hazard - Low acute toxicity Ecological Hazard - moderately toxic to aquatic organisms PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily Biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: Measured log K_{ow} 3.51 PBT Assessment: Does not meet the screening criteria for bioaccumulation	Tier 1 (Qualitative Assessment/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Ethoxylated Decanol is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Ethoxylated Tallow Alkyl Amine	61791-26-2	958	9	0.3	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LL_{50} *Danio rerio* - >100 mg/L **Acute Aquatic - Invertebrate** -48-hr LL_{50} *Daphnia magna* - 12.41 mg/L **Acute Aquatic-Algae** -72-hr LL_{50} *Pseudokirchnerella subcapitata* - 39.7 mg/L **Chronic Toxicity** Algae - 72-hr EC_{10} - 7.08 mg/L **Terrestrial Toxicity** No studies available **$PNEC_{water}$** - 0.071 mg/L **$PNEC_{soil}$** - 3.14 mg/kg soil dry weight (Equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - low toxicity Ecological Hazard - low acute aquatic toxicity PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties Structure indicates no potential for bioaccumulation PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative Assessment/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Ethoxylated Tallow Alkyl Amine is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Polyoxyethylene glycol trimethylnonyl ether	127087-87-0	1050	91	2.73	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Pimephales promelas* - 0.218 mg/L -96-hr LC_{50} *Lepomis macrochirus* - 1.3 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Ceriodaphnia dubia* - 0.328 mg/L -48-hr LC_{50} *Daphnia* - 1.8 mg/L **Acute Aquatic- Algae** -48-hr EC_{50} *Pseudokirchneriella subcapitata* - 20-50 mg/L **Chronic Toxicity-Fish** -21-day NOEC *Oncorhynchus mykiss* - 0.048 mg/L -7-day NOEC *Ceriodaphnia dubia* - 0.285 mg/L **Chronic Toxicity- Invertebrates** -6-day NOEC *Daphnia Magna* - 1.0 mg/L **Chronic Toxicity-Algae** -96-hr NOEC *Pseudokirchneriella subcapitata* - 8 mg/L -120-hr (5 day) EC_{50} *Scenedesmus Opoliensis* - 37.4 mg/L **Terrestrial Toxicity** No data available. **$PNEC_{water}$**- 0.00096 mg/L **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - Low to moderate toxicity Ecological Hazard - moderate concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: BCF values in fish studies are <1.4 L/Kg PBT Assessment: Does not meet the criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Polyoxyethylene nonylphenol ether is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

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Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Potassium Sorbate Food Grade	24634-61-5	1360	20	0.45	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Danio rerio* - >500 mg/L (mortality) to > 1250 mg/L (mortality) -96-hr LC_{50} *Oncorhynchus mykiss* - >1000 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - 750 mg/L to 982 mg/L (mobility) **Acute Aquatic-Algae** -72-hr EC_{50} *Desmodesmus subspicatus* - 480 mg/L (growth rate) **Chronic Toxicity** -72-hr NOEC *Desmodesmus subspicatus* - 8.46 mg/L -21-d NOEC *Daphnia magna* - 50 mg/L **Terrestrial Toxicity** -14-day LC_{50} *Eisenia fetida* - 864 mg/kg soil dry weight -14-day NOEC *Eisenia fetida* - 582 mg/kg soil dry weight -31-day NOEC *Brassica rapa* - >100 mg/kg soil dry weight -39-day NOEC *Avena sativa* - >100 mg/kg soil dry weight **$PNEC_{water}$** - 0.169 mg/L **$PNEC_{soil}$** - 1 mg/kg soil dry weight	Qualitative Assessment: Human Health Hazard - low acute toxicity Ecological Hazard - low toxicity to aquatic organisms PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence	Environmental Fate Properties: Measured BCF in fish is 0.007 at ph 6.5 PBT Assessment: Does not meet the criteria for bioaccumulation	Tier 1 (Qualitative Assessment/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Potassium Sorbate Food Grade is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did not exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and not expected to bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted for aquatic receptors. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Sodium Benzoate	532-32-1	1500	0	0.01	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hour LC_{50} *Pimephales promelas* - 484 mg/L (mortality) -96-hour LC_{50} *Pimephales promelas* - >100 (mortality) **Acute Aquatic - Invertebrate** -96-hour LC_{50} *Daphnia magna* - >100 mg/L (mortality) **Acute Aquatic-Algae** -72-hour EC_{50} *Raphidocelis subcapitata* - >30.5 mg/L (growth rate) **Chronic Toxicity** -72-hour EC_{10} *Raphidocelis subcapitata* - 6.5 mg/L (growth rate) -144-hour NOEC - *Danio rerio* - 10 mg/L **Terrestrial Toxicity** No studies available **$PNEC_{water}$** - 0.65 mg/L **$PNEC_{soil}$** - 0.06 mg/kg	Qualitative Assessment: Human Health Hazard - low concern Ecological Hazard - low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: log K_{ow} - 1.88 PBT Assessment: Does not meet the criteria for bioaccumulation	Tier 1 (NICNAS/ Qualitative Assessment/ PBT)	NICNAS has assessed sodium benzoate in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment (NICNAS, 2019). PBT Assessment: The overall conclusion is that methanol is not a PBT substance. Qualitative assessment indicated low concern to human health The estimated injected concentration did not exceed the ecotoxicity values or PNECs for this chemical. This chemical is readily biodegradable, does not bioaccumulate, and does not meet the PBT assessment criteria for toxicity. Therefore, a Tier 2 assessment was not warranted. Management: No additional management required, Tier 1 screening satisfied.	NA
Formic Acid	64-18-6	1220	0.0000015	1.19	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Brachydanio rerio* (Zebrafish) - 130 mg/L -96-hr LC_{50} *Oncorhynchus mykiss* (Rainbow trout) - 3,500 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - 365 mg/L to 540 mg/L **Acute Aquatic-Algae** -72-hr EC_{50} *Pseudokirchneriella subcapitata* - 1,240 mg/L **Chronic Toxicity** -21-d NOEC *Daphnia* - 100 mg/L **Terrestrial Toxicity** No data available **$PNEC_{water}$** -10 mg/L **$PNEC_{soil}$** - 4.13 mg/kg soil dry weight (equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - low acute toxicity Ecological Hazard - low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence	Environmental Fate Properties: Not excpected to bioaccumulate. log K_{ow}= -2.1 PBT Assessment: Does not meet the criteria for bioaccumulation	Tier 1 (NICNAS/ Qualitative Assessment/ PBT)	NICNAS has assessed formic acid in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment (NICNAS, 2019). PBT Assessment: The overall conclusion is that Formic Acid is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did not exceed the ecotoxicity values or PNECs for this chemical. This chemical is readily biodegradable and does not bioaccumulate. Management: No additional management required, Tier 1 screening satisfied.	NA

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Alkylpyridine Quat	68909-18-2	1104	31	0.89	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC50 *Cyprinodon variegatus* - 14.1 mg/L **Acute Aquatic - Invertebrate** -48-hr EC50 *Daphnia magna* - 3.1 mg/L **Acute Aquatic-Algae** - 72-hr EC50 *Pseudokirchneriella subcapitata* - 0.47 mg/L **Chronic Toxicity** No studies available. **Terrestrial Toxicity** No studies available. **$PNEC_{water}$** - 0.00047 mg/L **$PNEC_{soil}$** - 0.0063 mg/kg soil dry weight (Equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard - Skin corrosion causing skin burns and eye damage. Ecological Hazard - Exhibits significant acute and chronic aquatic toxicity PBT Assessment: Substance exhibits higher toxicity than that established by regulatory guidance.	Environmental Fate Properties: Inherently biodegradable. PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: Not excpected to bioaccumulate. log K_{ow}= -2.1 PBT Assessment: Does not meet the criteria for bioaccumulation	Tier 1 (Qualitative Assessment/ PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that alkylpyridine quat is not a PBT substance. Qualitative Assessment indicated potential hazard to human health (e.g., skin irritant). The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical and does meet the screening criteria for toxicity. This chemical is inherently biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted for aquatic receptors. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Australia SafeWork Place and Condor Occupational Safety Guidance will be used to minimise human health exposure. Therefore, a Tier 2 assessment was not warranted.	NA
2-Ethylhexanol PO/EO polymer	64366-70-7	NA	NA	0.3	**Aquatic Toxicity** -LC_{50}/EC_{50} > 100 mg/L for test most sensitive test species **Chronic Toxicity** No studies available. **Terrestrial Toxicity** No studies available. **$PNEC_{water}$** - 0.1 mg/L **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - low acute toxicity Ecological Hazard - low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence	Environmental Fate Properties: Not excpected to bioaccumulate. PBT Assessment: Does not meet the criteria for bioaccumulation	Tier 1 (ACIS/Qualitative Assessment/ PBT)	AICIS Assessment (2022): Chemical unlikely to require further regulation to manage risks to environment. PBT Assessment: The overall conclusion is that 2-Ethylhexanol PO/EO polymer is not a PBT substance. Qualitative assessment indicated low concern for human and ecological health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is readily biodegradable and not expected to bioaccumulate. Therefore, given this information and the AICIS Assessment findings, a Tier 2 assessment was not warranted Management: No additional management required Tier 1 screening satisfied.	NA
Ammonium sulphate	7783-20-2	1770	7928	141.04	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hour LC_{50} *Onchorhyncus mykiss , Salmo gairdneri* - 53 mg/L -96-hour -LC_{50}*Prosopium williamsoni* - 57.2 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Daphnia magna* - 169 mg/L -48-hr EC_{50} *Ceriodaphnia acanthina* - 121.7 mg/L **Chronic Toxicity** -30-day EC_{10} *Lepomis macrochirus* 5.29 mg/L -10-week EC_{10} *Hyallella azteca* - 3.12 mg/L -18-day EC_{50} - *Chlorella vulgaris* - 2,700 mg/L -5-day EC_{50} - *Chlorella vulgaris* - 1,605 mg/L **Terrestrial Toxicity** No data were available **$PNEC_{water}$** - 0.312 mg/L **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - low concern Ecological Hazard - low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Dissociates completely in aqueous media PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Property: Log Kow is -5.1 PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that ammonium sulphate is not a PBT substance. Qualitative assessment indicated low concern to human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical dissociates completely and does not bioaccumulate. Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

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Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Sodium polyacrylate	9003-04-7	NA	NA	23.51	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} for *Brachydanio rerio, Salmo giardneri, Leucisucus idus, and Lepomis macrhochirus* are dependent on molecular weight and range from >200 to > 10000 mg/L **Acute Aquatic - Invertebrate** -46-hr EC_{50} for *Daphnia magna* are dependent on molecular weight and range from >200 mg/L to >276 mg/L **Acute Aquatic-Algae** -72-hr EC_{50} (molecular weight of 8,000) *Selenastrum capricornutum* - 40 mg/L -96-hr EC50 (molecular weight of 78,000) *Selenastrum capricornutum* - 44 mg/L **Chronic Toxicity** Fish -32-day NOEC (molecular weight of 4,500) *Pimephales promelaas* - 56 mg/L -28-day NOEC (molecular weight of 4,500) *Brachydanio rerio* - >450 mg/L -14-day NOEC (molecular weight of 78,000) *Brachydanio rerio* - >400 mg/L Invertebrate -21-day NOEC for *Daphnia magna* dependent on molecular weight and range from > 12 to > 450 mg/L Algae -96-hr NOEC for *Scenedesmus subspicatus* are dependent on molecular weight and range from 32.8 to 180 mg/L **Terrestrial Toxicity** -14-day EC0 to *Eisenia foetida foetida* - 1,000 mg/L -14-day EC0 - *Eisenia foetida andrei* - 1,000 mg/L -21-day NOEC - *Brassica rapa* - 1,000 mg/L **PNECwater** - 1.2 mg/L **PNECsoil -** 25 mg/kg	Qualitative Assessment: Human Health Hazard - low concern Ecological Hazard - low toxicity concern for aquatic organisms, terrestrial invertebrates, and plants. PBT Assessment: Does not meet the screening criteria for toxicity.	Environmental Fate Properties: Not readily biodegradable. PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Property: Not expected to bioaccumulate due to their high molecular weights PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Sodium polyacrylate is not a PBT substance. Qualitative assessment indicated low concern for human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical and is not readily biodegradable. However, this chemical does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Sodium bisulfite	7631-90-5	1348	201	4.7	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Leuciscus idus* - 316 mg/L -96-hr LC_{50} *Salmo gairdneri* - 147-215 mg/L -96-hour LC_{50} *Brachydanio rerio* - 464-1,000 mg/L **Acute Aquatic - Invertebrate** -48-hour EC_{50} *Daphnia magna* - 88.8 mg/L **Acute Aquatic-Algae** -96-hour EC_{50} *S. subspicatus* - 43.9 mg/L -72-hour EC_{10} *S. subspicatus* - 33.3 mg/L **Chronic Toxicity** -Chronic toxicity studies on sodium sulfite -34-day NOEC *Danio rerio* - >316 mg/L -21-day NOEC *Daphnia magna* - >10 mg/L No chronic studies are available on sodium bisulfite **Terrestrial Toxicity** No studies available **$PNEC_{water}$** - 0.8 mg/L **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - low concern Ecological Hazard - low to moderate toxicity concern to aquatic organisms PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: An inorganic compound that dissociates completely to ionic species and sulfur dioxide gas. PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Property: Not expected to bioaccumulate because its dissociates species are inorganic ions and a gas PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/PBT)	PBT Assessment: The overall conclusion is that Sodium bisulfite is not a PBT substance. Qualitative assessment indicated low concern for human receptors and low to moderate concern for aquatic receptors. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

Table 1
Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Alkyl Alcohol	56-81-5	1261	188	4.7	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} *Oncorhynchus mykiss* - 54,000 mg/L -96-hr LC_{50} *Pimephales promelas* - 885 mg/L -96 hrLC_{50} - *Carassius auratus* - >5000 mg/L **Acute Aquatic - Invertebrate** - 24-hr EC_{50} *Daphnia magna* - >10,000 -48 hr - LC_{50} *Daphnia magna* - 1995 mg/L **Chronic Toxicity** -NOEC - fish - > 100 mg/L - NOEC - *Daphnia magna* - 897 mg/L **Terrestrial Toxicity** -No studies available **$PNEC_{water}$** - 18 mg/L **$PNEC_{soil}$** - 0.24 mg/kg soil dry weight (Equilibrium partitioning method)	Qualitative Assessment: Human Health Hazard-low concern Ecological Hazard-low toxicity concern to aquatic organisms PBT Assessment: Does not meet the screening criteria for toxicity.	Environmental Fate Properties: Readily biodegradable. PBT Assessment: Does not meet the screening criteria for persistence	Environmental Fate Property: Measured log K_{ow} is -1.75 PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/PBT)	PBT Assessment: The overall conclusion is that Alkyl Alcohol is not a PBT substance. Qualitative assessment indicated low concern for human and ecological health. The estimated injected concentration did not exceed the ecotoxicity values or PNECs for this chemical. Therefore, a Tier 2 assessment was not warranted Management: No additional management required Tier 1 screening satisfied.	NA
2-Propenoic acid, homopolymer, ammonium salt	9003-03-6	NA	NA	4.7	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-hr LC_{50} for *Brachydanio rerio, Salmo giardneri, Leucisucus idus, and Lepomis macrhochirus* are dependent on molecular weight and range from >200 to > 10000 mg/L **Acute Aquatic - Invertebrate** -46-hr EC_{50} for *Daphnia magna* are dependent on molecular weight and range from >200 mg/L to >276 mg/L **Acute Aquatic-Algae** -72-hr EC_{50} (molecular weight of 8,000) *Selenastrum capricornutum* - 40 mg/L -96-hr EC50 (molecular weight of 78,000) *Selenastrum capricornutum* - 44 mg/L **Chronic Toxicity** Fish -32-day NOEC (molecular weight of 4,500) *Pimephales promelaas* - 56 mg/L -28-day NOEC (molecular weight of 4,500) *Brachydanio rerio* - >450 mg/L -14-day NOEC (molecular weight of 78,000) *Brachydanio rerio* - >400 mg/L Invertebrate -21-day NOEC for *Daphnia magna* dependent on molecular weight and range from > 12 to > 450 mg/L Algae -96-hr NOEC for *Scenedesmus subspicatus* are dependent on molecular weight and range from 32.8 to 180 mg/L **Terrestrial Toxicity** -14-day EC0 to *Eisenia foetida foetida* - 1,000 mg/L -14-day EC0 - *Eisenia foetida andrei* - 1,000 mg/L -21-day NOEC - *Brassica rapa* - 1,000 mg/L **PNECwater** - 1.2 mg/L **PNECsoil** - 25 mg/kg	Qualitative Assessment: Human Health Hazard - low concern Ecological Hazard - low toxicity concern for aquatic organisms, terrestrial invertebrates, and plants. PBT Assessment: Does not meet the screening criteria for toxicity.	Environmental Fate Properties: Not readily biodegradable. PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Property: Not expected to bioaccumulate due to their high molecular weights PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that 2-Propenoic acid, homopolymer, ammonium salt is not a PBT substance. Qualitative assessment indicated low concern for human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. It is not readily biodegradable; however, it is not expected to bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA

Table 1
Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Potassium persulfate	7727-21-1	1390	208	4.7	**Aquatic Toxicity** **Acute Aquatic - Fish** -96-h LC_{50} *Oncorhynchus mykiss* (Rainbow trout) - 76.3 mg/L (mortality) -96-h LC_{50} *Oncorhynchus mykiss* (Rainbow trout) - 163 mg/L (mortality) -96-hr LC_{50} *Oncorhynchus mykiss* - 76.3 mg/L **Acute Aquatic - Invertebrate** -48-h EC_{50} *Daphnia magna* - 120 mg/L **Acute Aquatic-Algae** -72-h EC_{50} *Raphidocelis subcapitata* - 83.7 mg/L -72-h EC_{50} *Raphidocelis subcapitata* - 116 mg/L **Chronic Toxicity** -21-d NOEC *Daphnia magna* - 20.8 mg/L -120-h NOEC *Raphidocelis subcapitata* - 154 mg/L (biomass) -120-h NOEC *Raphidocelis subcapitata* - 23.5 mg/L (biomass) -120-h NOEC *Raphidocelis subcapitata* - 6.92 mg/L (biomass) **Terrestrial Toxicity** Persulphates are not expected to be distributed to the terrestrial compartment and consequently not to cause toxicity to terrestrial organisms and plants. **$PNEC_{water}$** - 0.416 mg/L **$PNEC_{soil}$** - no derived	Qualitative Assessment: Human Health Hazard- low concern Ecological Hazard- low toxicity concern to aquatic receptors PBT Assessment: Does not meet the screening criteria for toxicity.	Environmental Fate Properties: Expected to biodegrade PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Property: Not expected to bioaccumulate PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that Potassium persulfate is not a PBT substance. Qualitative assessment indicated low concern for human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical. However, this chemical is expected to biodegrade and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
2-Ethoxy-naphthalene	93-18-5	1241.3	185	4.7	**Aquatic Toxicity** **Acute Aquatic - Invertebrate** -72-h EC_{50} *Daphnia magna* - 3.9 mg/L (mobility) **Chronic Toxicity** -No studies available **Terrestrial Toxicity** -No studies available **$PNEC_{water}$** - 0.039 mg/L **$PNEC_{soil}$** - 1.61 mg/kg soil dry weight (Equilibrium paritioning method)	Qualitative Assessment: Human Health Hazard- low acute oral and dermal toxicity. Ecological Hazard- aquatic toxicity is unlikely to occur due to insoluble nature. PBT Assessment: Does not meeting criteria for toxicity.	Environmental Fate Properties: Readily biodegradable. PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Property: Not expected to bioaccumulate. PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/PBT/Exposure Assessment)	PBT Assessment: The overall conclusion is that 2-Ethoxy-naphthalene is not a PBT substance. Qualitative assessment indicated low concern for human health. The estimated injected concentration did exceed the ecotoxicity values or PNECs for this chemical.] However, this chemical is readily biodegradable and does not bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	NA
Nonoxynol-9	26571-11-9	1050	10	0.3	**Aquatic Toxicity** **Acute Aquatic - Fish** - 95-hr LC_{50} *Pimephales promelas* (Fathead minnow) - .128 mg/L - 96-hr LC_{50} *Lepomis macrochirus* (Bluegill) - 1.3 mg/L **Acute Aquatic - Invertebrate** -48-hr EC_{50} *Ceriodaphnia dubia* (Water flea) - .328 mg/L -48-hr LC_{50} *Daphnia magna* - 1.8 mg/L **Acute Aquatic-Algae** -48-hr EC_{50} *Pseudokirchneriella subcapitata* - 20-50 mg/L **Chronic Toxicity** -21-day NOEC *Oncorhynchus mykiss* (Rainbow trout) - .048 mg/L -7-day NOEC *Ceriodaphnia dubia* - .285 mg/L -6-day NOEC *Daphnia Magna* - 1.0 mg/L -96-hr NOEC *Pseudokirchneriella subcapitata* - 8 mg/L -120-hr (5-d) EC_{50} *Pseudokirchneriella subcapitata* -37.4 mg/L **Terrestrial Toxicity** -No data were available. **$PNEC_{water}$** - 10 mg/L **$PNEC_{soil}$** - unavailable	Qualitative Assessment: Human Health Hazard-low to moderate oral acute toxicity and low dermal toxicity Ecological Hazard- moderate toxicity concern to aquatic receptors PBT Assessment: Does not meet the criteria for toxicity	Environmental Fate Properties: Readily biodegradable PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Property: It is not expected to bioaccumulate. Low potential to adsorb to soil or sediment. PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (Qualitative/PBT)	PBT Assessment: The overall conclusion is that Nonoxynol-9 is not a PBT substance. Qualitative assessment indicated low to moderate concern for human health. The estimated injected concentration did not exceed the PNECs for this chemical. Therefore, a Tier 2 assessment was not warranted. Management: No additional management required, Tier 1 screening satisfied.	NA

Table 1
Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Chemical Name	CAS Number	Density (kg/m^3)	Chemical Mass in Fluid (kg)	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Biodegradation	Bioaccummulative	Screening	Discussion	Outcome of Tier 2 Assessment
Talc	14807-96-6	2700	1038	12.1	**Aquatic Toxicity** Acute Aquatic -96-h LC_{50} Unnamed fish species - 89,581 mg/L (QSAR) -48-h LC_{50} Daphnid species - 36,812 mg/L (QSAR) -96 h LC50 Freshwater algae - 7,203 mg/L Chronic Aquatic - Fish No data available **Terrestrial Toxicity** No data available. **$PNEC_{water}$** - 72 mg/L **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - Low concern Ecological Hazard - Low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Biodegradability is not relevant because inorganic substance. PBT Assessment: Does not meet the screening criteria for persistence.	Environmental Fate Properties: Bioaccumulation not expected to occur based on its log K_{ow} value of -9.4. PBT Assessment: Does not meet the screening criteria for bioaccumulation.	Tier 1 (NICNAS/Qualitative Assessment/PBT)	NICNAS has assessed talc in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment (NICNAS, 2019). PBT Assessment: The overall conclusion is that talc is not a PBT substance. Qualitative assessment indicated low concern to human health The estimated injected concentration did not exceed the ecotoxicity values or PNECs for this chemical. Therefore, a Tier 2 assessment was not warranted. Management: No additional management required, Tier 1 screening satisfied.	NA
Polyacrylamide	250852-02-3	NA	NA	141.04	**Aquatic Toxicity** No data available **Chronic Toxicity** No data available **Terrestrial Toxicity** No data available **$PNEC_{water}$** -not derived **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - low acute toxicity Ecological Hazard - low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Not readily biodegradable PBT Assessment: Does meet the screening criteria for persistence	Environmental Fate Properties: Not excpected to bioaccumulate because of expected very high molecurlar weight and poor water solubility PBT Assessment: Does not meet the criteria for bioaccumulation	Tier 1 (NICNAS/ Qualitative Assessment/ PBT)	NICNAS: Identified polyacrylamide (25085-02-3) as a polymer of low concern for human health in in IMAP Tier 1 assessment PBT Assessment: The overall conclusion is that polyacrylamide (25085-02-3) is not a PBT substance. Qualitative Assessment indicated low concern to human health. This chemical is not readily biodegradable; however, it is not expected to bioaccumulate. Aquatic toxicity studies were not available; however, this chemical is expected to have low concern for aquatic toxicity becuase of its very high molecular weight and poor water solubility. Therefore, a Tier 2 Assessment is not warranted. Management: No additional management required, Tier 1 screening satisfied.	
Polyacrylamide	9005-05-8	NA	NA	1545.96	**Aquatic Toxicity** **Acute Aquatic - Fish** -LC_{50} for Fathead minnow, rainbow trout, and blue gill sunfish are dependent on ioinic charge and range from >100 to 840 mg/L **Acute Aquatic - Invertebrate** -EC_{50} for *Daphnia magna* (ionic charge -39) - 470 mg/L **Chronic Toxicity** No Studies Available **Terrestrial Toxicity** No data available **$PNEC_{water}$** -0.1 mg/L **$PNEC_{soil}$** - not derived	Qualitative Assessment: Human Health Hazard - low acute toxicity Ecological Hazard - low concern PBT Assessment: Substance exhibits lower toxicity than that established by regulatory guidance.	Environmental Fate Properties: Not expected to biodegrade due to high molecular weight PBT Assessment: Does meet the screening criteria for persistence	Environmental Fate Properties: Not excpected to bioaccumulate because of expected very high molecurlar weight and water solubility PBT Assessment: Does not meet the criteria for bioaccumulation	Tier 1 (NICNAS/ Qualitative Assessment/ PBT)	NICNAS: Identified polyacrylamide (9003-05-8) as a polymer that poses no unreasonable risk to the environment. PBT Assessment: The overall conclusion is that polyacrylamide (25085-02-3) is not a PBT substance. Qualitative Assessment indicated low concern to human health. The estimated injected concentration did exceed the PNEC or aquatic toxicity value. This chemical is not readily biodegradable; however, it is not expected to bioaccumulate. Additionally, the potential exposure to aquatic receptors is considered incomplete (refer to text). Therefore, a Tier 2 assessment was not warranted. Management: Implementation of Constraints Protocol in EMP will be required to prevent accidental discharge/release. Therefore, a Tier 2 assessment was not warranted.	

Table 1
Evaluation of Compiled List of Chemicals
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Table Notes:
°C = degrees Celsius
µg/L = microgram per litre
AICIS = Australian Industrial Chemicals Introduction Scheme
ANZECC = Australian and New Zealand Environment Conservation Council
Ca:Mg = calcium:magnesium
CaCO3 = calcium carbonate
CAS = Chemical Abstract Service
CFT = Chemical Fracture Tracer
dw = dry weight
EC_0 = The concentration of a substance that is estimated to be lethal to 0% of the test organisms
EC_{50} = effects concentration of half the maximal response
ECHA = European Chemicals Agency
EG = ethylene glycol
EMP = Environmental Management Plan
GFT = Gas Fracture Tracer
HCO3- = bicarbonate
IMAP = Inventory Multi-tiered Assessment and Prioritisation
kg/L = kilogram per litre
Kow = n-octanol-water partition coefficient
L = litre
LC_{50} = lethal concentration of 50 percent of population
LOEC = lowest observed effects concentration
mg/kg = milligram per kilogram
mg/L = milligrams per litre
Na+ = Sodium ion
NA = not applicable
NICNAS = National Industrial Chemicals Notification and Assessment Scheme
NOEC = no observed effect concentration
NOELR = no observed effect loading rate
PBT = persistence, bioaccumulative, toxic
PEG - polyethylene glycol
PNEC = predicted no effect concentration
TGK = toxicity threshold (growth inhibition)
WAF = Water Accommodated Fraction Analysis
Additional NICNAS cher
Silica dioxide
Sodium Chloride
Tributyl tetradecyl phosphonium chloride
UVCB = unknown or variable composition, complex reaction products or biological materials

AICIS. 2022. Chemicals unlikely to require further regulation to manage risk to environment; Evaluation statement. 30 May.
Australian and New Zealand Environment and Conservation Council and Agriculture and Resource Management Council of Australia and New Zealand (ANZECC & ARMCANZ). (2000). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Canberra, ACT: Author.
NICNAS 2017, Chemicals of low concern for human health based on an initial assessment of hazards, Project report prepared by the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) as part of the National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia, Commonwealth of Australia, Canberra.
NICNAS 2018, Human health Tier II assessment, Project report prepared by the National Industrial Chemicals Notification and Assessment Scheme (NICNAS) as part of the National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia, Commonwealth of Australia, Canberra.
Organisation for Economic Co-operation and Development (OECD). (1992). Test No. 301: Ready Biodegradability. (Biodégradabilité Facile.) Paris: OECD Publishing.
Soucek, D.J. (2007). Comparison of hardness and chloride regulated acute effects of sodium sulfate on two freshwater crustaceans. Environ. Toxicol. Chem. 26: 773-779.
Australian Industrial Chemicals Introduction Scheme. 2021. Chemical Information Database. Available online at:

Appendix A Compiled List of Chemicals

CONFIDENTIAL INFORMATION - ONLY TO BE USED FOR REGULATOR NOTIFICATION (QLD FORMAT)

08-January-2023

Pre Frac NOC for Tamboran (Hybrid system with Borate Crosslinked and High Viscosity Friction Reducer fluid systems with 15% HCL Acid Spearhead, 100 Mesh & 40/70 Sand)

Total injected fluid volume (kilolitres):		**31755.702**		
Comprising of:				
Base fluid type (e.g. water)		**Litres**		**% of total volume**
Makeup Water		29749793.160		93.683%
Proppant type (e.g. sand)	**Proppant size**	**Kilograms**	**Litres**	**% of total volume**
Sand	20/40 Sand	0.000	0.000	0.00000%
Sand	100 Mesh	347222.222	130998.845	0.41252%
Sand	40/70	3905895.692	1473603.332	4.64044%

Any wet chemical constitutes:	**Litres**	**CAS Number**	**% of total volume**
Alcohols, C11-14-iso-, C13-rich,ethoxylated	5285.099	78330-21-9	0.016643%
Sodium (C14-16) olefin sulfonate	4658.053	68439-57-6	0.014668%
Diisobutyl glutarate	627.046	71195-64-7	0.001975%
Diisobutyl succinate	209.015	925-06-4	0.000658%
Diisobutyl adipate 141-04-8	179.156	141-04-8	0.000564%
sodium thiosulphate	4763.355	7772-98-7	0.015000%
sodium sulphate	913.330	7757-82-6	0.002876%
sodium sulphite	793.893	7757-83-7	0.002500%
Ethylene Glycol	4963.172	107-21-1	0.015629%
Glutaraldehyde	14929.657	111-30-8	0.047014%
Ammonium Sulphate	4478.897	7783-20-2	0.014104%
Polyacrylamide	4478.897	25085-02-3	0.014104%
Sodium polyacrylate	746.483	9003-04-7	0.002351%
Sodium bisulfite	149.297	7631-90-5	0.000470%
Alkyl Alcohol	149.297	56-81-5	0.000470%
2-Propenoic acid, homopolymer, ammonium salt	149.297	9003-03-6	0.000470%
Potassium persulfate	149.297	7727-21-1	0.000470%
2-Ethoxy-naphthalene	149.297	93-18-5	0.000470%
Sodium Gluconate	8576.225	527-07-1	0.027007%
Boric Acid	4288.112	10043-35-3	0.013503%
Potassium Hydroxide	10745.265	1310-58-3	0.033837%
Ammonium Persulphate	7450.906	7727-54-0	0.023463%
Talc	384.295	14807-96-6	0.001210%
Sodium Bromate	50440.588	7789-38-0	0.158839%
Hepta sodium phosphonate	3175.570	22042-96-2	0.010000%
DISTILLATES, HYDROTREATED LIGHT	54230.768	64742-47-8	0.170775%
Guar Gum	15141.114	9000-30-0	0.047680%
Polyoxyethylene nonylphenol ether	4466.405	9016-45-9	0.014065%
Quaternary ammonium compounds, bis(hydrogenated tallow alkyl)dimethyl, salts with bentonite	4466.405	68953-58-2	0.014065%
1,6-Hexanediol	446.641	629-11-8	0.001406%
Quartz or Organophilic phyllosilicate	1083.668	14808-60-7	0.003413%
HydroChloric Acid	44715.156	7647-01-0	0.140810%
N-Benzyl-Alkylpyridinium Chloride	28.391	68909-18-2	0.000089%
Formic Acid	37.854	64-18-6	0.000119%
Sodium erythorbate	333.810	6381-77-7	0.001051%
Citric Acid	15877.851	77-92-9	0.050000%
Acetic Acid	15877.851	64-19-7	0.050000%
EGMBE	0.000	111-76-2	0.000000%
Isopropanol	83.279	67-63-0	0.000262%
Ethoxylated C12-C16 Alcohol	56.781	68551-12-2	0.000179%
Ethoxylated Decanol	18.927	26183-52-8	0.000060%
Cinnamaldehyde	56.781	104-55-2	0.000179%
Ethoxylated Tallow Alkyl Amine	9.464	61791-26-2	0.000030%
Methanol	1.893	67-56-1	0.000006%
Potassium Chloride	0.000	7447-40-7	0.000000%
Polyacrylamide	49092.948	9003-05-08	0.154596%
Polyethylene glycol trimethylnonyl ether	86.584	127087-87-0	0.000273%
Water in Additive	62324.751	7732-18-5	0.196263%
Potassium Sorbate Food Grade	14.385	24634-61-5	0.000045%
Sodium Benzoate	0.288	532-32-1	0.000001%
Mannanase (Mannan endo-1,4-beta-mannosidase)	2.158	37288-54-3	0.000007%
Nonoxynol-9	9.464	26571-11-9	0.000030%
2-Ethylhexanol PO/EO polymer	9.464	64366-70-7	0.000030%

*Note: display all values to 3 significant figures.

Total	**100.0000%**

Appendix B Assessment of Potential Release to Surface

Potential Risk to Groundwater from Hypothetical Water Releases

Exploration Permits EP76, EP98 & EP117

Prepared for:

Prepared by:

EHS Support

January 2023

Document Control

PROJECT DETAILS

Project No.:	AUS_C04159
Report Revision No.:	B
Date of Issue:	9 January 2022
Project Manager:	Joe Hayes
Project Director:	Nigel Goulding

REPORT DETAILS

Title:	Potential Risk to Groundwater from Hypothetical Water Releases
Main Author(s):	Chris Smitt/Ben Petrides
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Client:	Condor Energy
Client Contact:	Andrew McKenzie

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9 January 2023	1	Condor Energy	Andrew McKenzie, Scott Ross	e

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Acronyms

CLA	Cambrian Limestone Aquifer
E&A	exploration and appraisal
EP	exploration permit
Ma	million years ago
mbgl	metres below ground level
NT	Northern Territory
POINT	Petroleum Onshore Information Northern Territory

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Units of Measure

Area	
ha	hectare
m^2	square metres
Density	
kg/m^3	kilograms per cubic metre
Electrical Conductance	
µS/cm	micro Siemen per centimetre
mV	millivolt
Length	
µm	micrometres
cm	centimetres
km	kilometres
m	metres
Mass	
µg	micrograms
kg	kilograms
mg	milligrams
t	metric tonnes
Concentration by Mass	
µg/kg	microgram per kilogram
mg/kg	milligram per kilogram
Pressure	
kPa	kilopascals
Pa	Pascals
Temperature	
°C	degrees Celsius
°F	degrees Fahrenheit
K	kelvin
Velocity	
m/d	metres per day
m/s	metres per second
L/s	Litres per second
Volume	
µL	microlitres
cm^3	cubic centimetre
GL	gigalitre
L	litres
m^3	cubic metre
mL	millilitres
ML	megalitre
Concentration by Volume	
µg/L	microgram per litre
mg/L	milligram per litre
ppmv	parts per million by volume
ppbv	parts per billion by volume

1 Introduction

This report provides an assessment of the potential for impacts on groundwater associated with Tamboran B2 Pty Ltd shale gas activities within exploration permits (EP) EP76, EP98 and EP117 in the Northern Territory (NT). In particular this assessment focusses on the area of the four proposed exploration and appraisal (E&A) wells; two at Amungee NW located in the centre of EP98 and two at the Valkerri 76 S2 site located in the central region of EP76. Both of these locations are located within the Amungee Mungee pastoral station. This assessment also focusses on the exploration well Beetaloo W-1, drilled in September 2016 in the centre of EP117.

For the purpose of this assessment, the primary mode of potential impact was identified as an accidental release to the land surface and the resulting radial land flow and sub-surface infiltration. The technical assessment and modelling is provided in the following sections.

1.1 Objective

The objective of this assessment is to define the potential extent of the area impacted by a release or "spill" of fluids and the likelihood of migration to groundwater. Specifically, the following objectives were addressed:

1. Using three spill scenarios (1,000 L, 100,000 L and 1 ML), determine the maximum pooled area in which a spill would inundate.
2. Over the size of the pooled area, determine infiltration rates to gain an understanding of vertical groundwater movement and associated travel time.
3. Evaluate the potential impacts on groundwater and other receptors of interest.

1.2 Scope of Work

To meet the objectives described above, the following work tasks were undertaken:

1. Establish applicable soil/aquifer characteristics within the areas of interest based on a literature review, available stratigraphic information from the Petroleum Onshore Information Northern Territory (POINT)1 web-based data catalogue and other literature (as appropriate).
2. Assess the water pooling area on a flat surface using the formulae proposed by Grimaz et al. (2007).
3. Assess the infiltration capacity of surface soils and ponding time using the analytical Green-Ampt infiltration equation (Green and Ampt, 1911).
4. Assess the infiltration velocity and depth once surface soils become saturated using Darcy's Law (Darcy, 1856).
5. Qualitatively evaluate the potential impacts on groundwater and other receptors of interest.

[1] NT. 2022. Petroleum Onshore Information, NT. Available online at: https://point.nt.gov.au/weave/point.html?deviceType=Desktop . Accessed December 2022.

1.3 Area of Interest

This assessment of the potential for impacts on groundwater associated with shale gas activities in the Northern Territory is applicable to EP76, EP98 and EP117 only. The EPs are shown on **Figure 1-1**, along with the major Basins and Sub-basins.

1.3.1 Receptors of Interest

The sites were chosen based on the geological, environmental, cultural, and social suitability of the site. The approximate buffer distances to the nearest environmental and community receptors are provided in **Table 1-1**.

Table 1-1 Buffer distances to sensitive receptors

Receptor	EP76 – Valkerri 76 S2	EP98 – Amungee NW	EP117 – Beetaloo W-1
Closest pastoral bore	4 km	11.4 km	7.5 km
Nearest homestead	27 km	50 km	12 km
Nearest community	65 km (Daly Waters)	100 km (Jingaloo)	16 km (Jingaloo)
Bullwaddy Conservation Reserve	40 km	30 km	-
Lake Woods	161 km	125 km	-
Frew Ponds	-	-	45 km
Lake Woods	-	-	60 km
Nearest mapped watercourse (Newcastle Creek)	20 km	13 km	8.5 km
Aboriginal protected areas	8 km	7 km	-

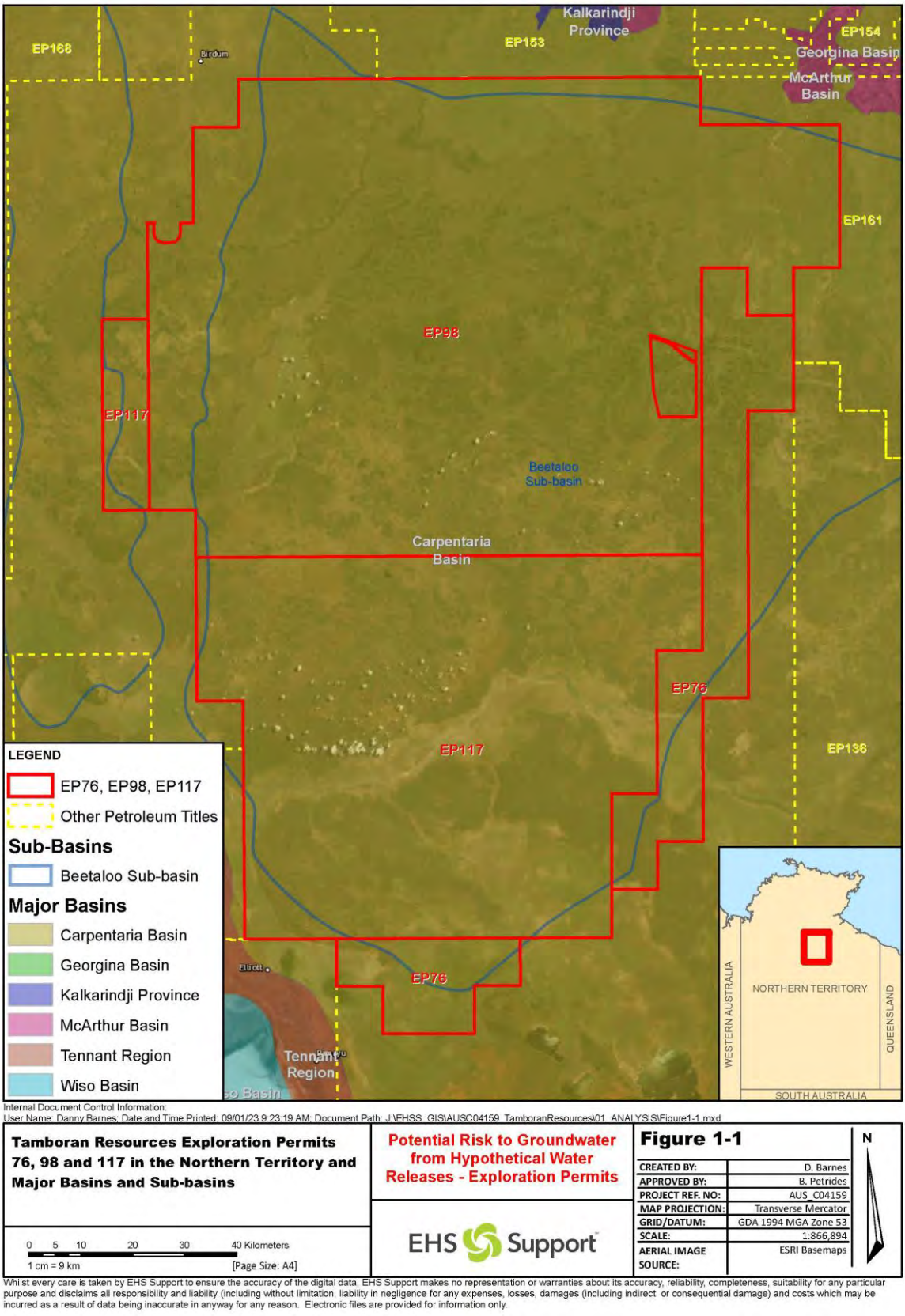


Figure 1-1 Tamboran B2 Pty Ltd Exploration Permits 76, 98 and 117 in the Northern Territory and Major Basins and Sub-basins

2 Overview of Hydrogeology/Geology

2.1 Geology

The Beetaloo Sub-basin comprises a thick sequence of mudstone and sandstone formations (Roper Group) that were deposited approximately 1,500-1,300 million years ago (Ma). The Roper Group is estimated to reach in excess of 5,000 m in thickness in the centre of the Sub-basin and estimated to be thinner outside the formally defined Beetaloo Sub-basin. The Roper Group is overlain unconformably by the yet to be formally defined Neoproterozoic Group. Unconformably overlying the Neoproterozoic group is the Georgina Basin (Cambrian) sedimentary package, which includes widespread extrusive flood basalts and a thick limestone sequence that forms the Cambrian Limestone Aquifer (CLA), a significant water supply aquifer. The Georgina Basin is capped unconformably by a thin section of Cretaceous mudstone and sandstone (Albian aged approx. 100–113 Ma) and recent alluvial and laterite deposits.

The proposed E&A wells will be completed in the Velkerri formation. Organic richness within the Velkerri formation is generally confined to three to four main shale intervals, the A, A-B, B and C shales. The existing Amungee NW-1H and Velkerri 76 S2-1 wells have been completed in the Velkerri B shale.

The Velkerri Formation Amungee Member is overlain with thick series of low permeability units (mudstone, siltstones, tight sandstone and Volcanic units), which include the Velkerri Formation Wyworrie Member, Kyalla Formation, Hayfield Formation, and Antrim Plateau Volcanics. These formations provide thick and multilayered effective geological barriers, with the Gum Ridge Formation separated from the target formations by >1,500 m.

2.2 Basins and Sub-basins

Table 2-1 presents Tamboran B2 Pty Ltd tenements and the associated geological basins (sub-basins where relevant). **Table 2-2** provides a summary of the basins and the inter-relationships. **Figure 2-1** presents EP76 and relevant basins, **Figure 2-2** presents EP98 and relevant basins and **Figure 2-3** presents EP117 and relevant basins.

Table 2-1 Basins and Sub-basins Relevant to the Areas of Interest

Exploration Permit	Owner	Basin(s)	Sub-Basin
EP76	Tamboran B2 Pty Ltd (77.5%) and Falcon Oil & Gas Australia (22.5%)	Carpentaria	Beetaloo
EP98	Tamboran B2 Pty Ltd (77.5%) and Falcon Oil & Gas Australia (22.5%)	Carpentaria	Beetaloo
EP117	Tamboran B2 Pty Ltd (77.5%) and Falcon Oil & Gas Australia (22.5%)	Carpentaria	Beetaloo

Table 2-2 Basin Summary and Relationships

Basin	Age (Ma)	Thickness (km)	Lithology	Relationship
Carpentaria	65 – 205	5	Sedimentary: sandstone, mudstone, limestone	Unconformably overlies the sedimentary rocks of Palaeoproterozoic Murphy Inlier, Paleo-Mesoproterozoic McArthur and South Nicholson basins, Neoproterozoic to Palaeozoic Georgina Basin and Palaeozoic Daly Basin.
Wiso	360 – 540	<0.3 to 3	Sedimentary: dolostone, limestone, shale, sandstone, siltstone.	Faulted against Palaeo-Neoproterozoic metamorphic rocks of the Aileron Province to the south. Unconformably overlies Palaeoproterozoic rocks of the Tanami Region to the west, Tennant Region to the east, and the Palaeo-Mesoproterozoic Birrindudu Basin to the northwest. Cretaceous rocks of the Carpentaria Basin cover its northern margin.
Georgina	355 – 850	3.7	Sedimentary: dolostone, limestone, shale, sandstone, siltstone.	Unconformably overlies Palaeoproterozoic Murphy, Warramunga and Davenport provinces, Palaeo-Mesoproterozoic McArthur and South Nicholson basins and Lawn Hill Platform, and in fault contact with Palaeo-Neoproterozoic Aileron Province. Interpreted to be contiguous with Neoproterozoic to Palaeozoic Wiso and Daly basins that developed as distinct depocentres isolated by basement highs formed from the Cambrian Kalkarindji Province. Unconformably overlain by Mesozoic Carpentaria and Eromanga basins.
Daly	470 – 520	1	Sedimentary: limestone, dolostone, sandstone, siltstone, conglomerate	Unconformably overlies the Palaeoproterozoic Pine Creek Orogen and Palaeo-Mesoproterozoic Birrindudu Basin to the north and east and Neoproterozoic Victoria Basin to the west. Overlain by Mesozoic Carpentaria Basin on its southern margin
Victoria	700 – 850	0.950	Sedimentary: dolostone, sandstone, limestone, shale.	Unconformably overlies Palaeoproterozoic Pine Creek Orogen and Palaeo-Mesoproterozoic Birrindudu Basin. Unconformably overlain by Neoproterozoic Wolfe Basin, Neoproterozoic to Palaeozoic Wiso Basin, Palaeozoic Daly Basin and Cambrian Kalkarindji Province.

Basin	Age (Ma)	Thickness (km)	Lithology	Relationship
Beetaloo Sub-basin	1,320 – 1,650	10	Sedimentary and minor volcanic: dolostone, sandstone, shale, felsic and mafic volcanic rocks.	The Beetaloo Sub-basin is a structural component of the Proterozoic greater McArthur Basin. It consists of two discrete subsurface volumes of sedimentary rock, typically bounded by faults, containing the thickest preserved formations that host significant hydrocarbon resources. Significant thicknesses of Mesoproterozoic sediment accumulated in the Beetaloo Sub-basin relative to adjacent areas (Munson, 2016). The sub-basin lies entirely under the cover of younger basin sediments of the Neoproterozoic Centralian A Superbasin, the Paleozoic Centralian B Superbasin (including the Georgina, Wiso and Daly basins) and the Mesozoic Carpentaria Basin.
McArthur	1,430 – 1,800	12	Sedimentary and minor volcanic: dolostone, sandstone, shale, felsic and mafic volcanic rocks.	Unconformably overlies Palaeoproterozoic Pine Creek Orogen, Murphy Province and Arnhem Province to the northwest, southeast and northeast respectively. Unconformably overlain by the Palaeozoic Arafura, Georgina and Mesozoic Carpentaria basins. Interpreted to be contiguous under cover with the Palaeo-Mesoproterozoic Birrindudu Basin and Tomkinson Province.
Birrindudu	1,550 – 1,780	10	Sedimentary: sublithic arenite, quartz arenite, siltstone, shale, conglomerate, stromatolitic chert, limestone, glauconitic sandstone.	Unconformably overlies Palaeoproterozoic Pine Creek Orogen to the north. Unconformably overlain by Palaeozoic Wiso and Daly basins to the east; by Cambrian Ord Basin to southwest; by Neoproterozoic Wolfe Creek Basin to west and Neoproterozoic Victoria Basin to the north; and in places, by Cambrian Kalkarindji Province and patchy sedimentary rocks of basin-margin Mesozoic sandstone. Towards the south is underlain by Palaeoproterozoic metasediments and granites of Tanami Region. In northwest, in faulted contact with Palaeozoic–Mesozoic Bonaparte Basin and Palaeoproterozoic rocks of Halls Creek Orogen.

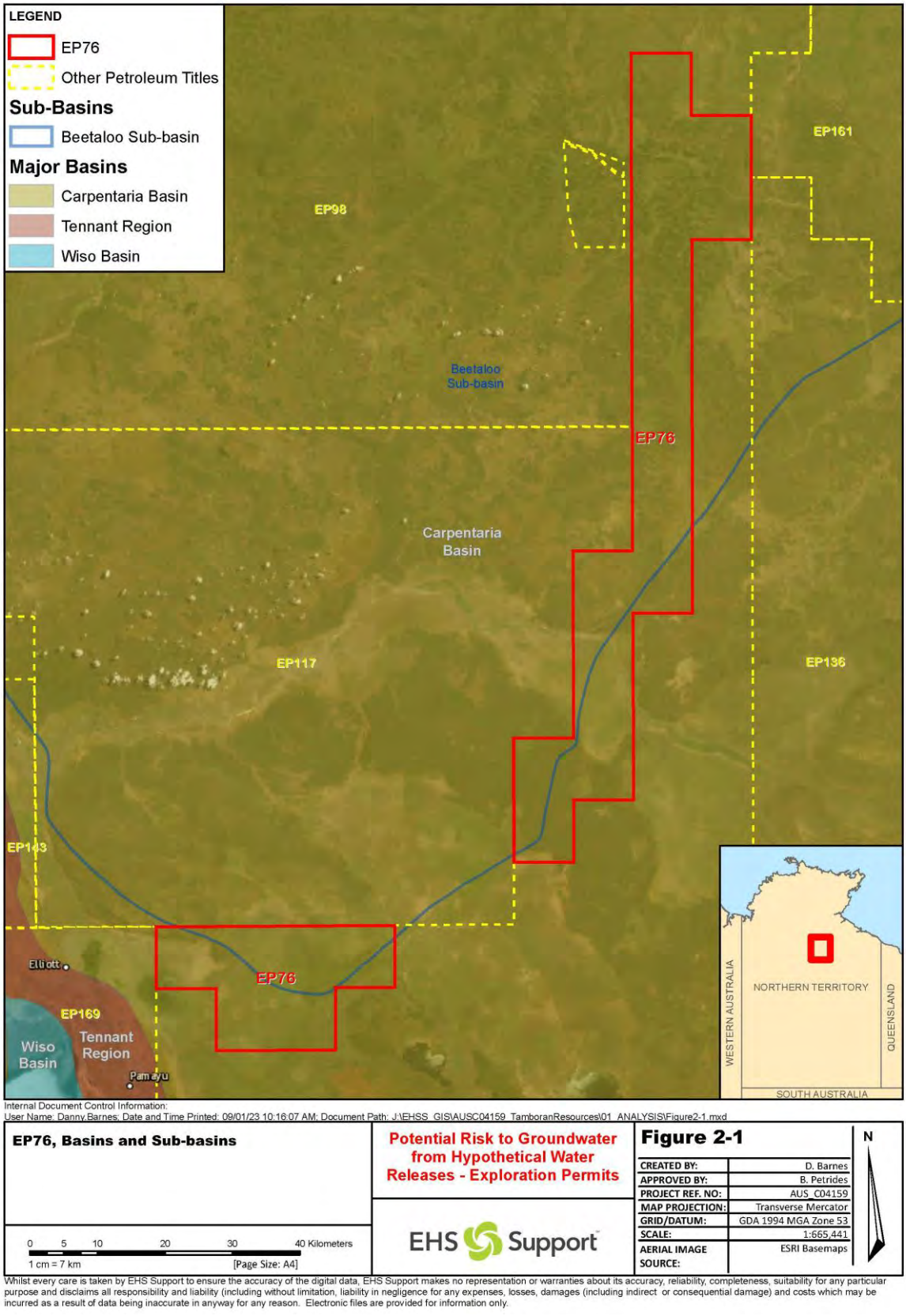


Figure 2-1 EP76, Basins and Sub-Basins

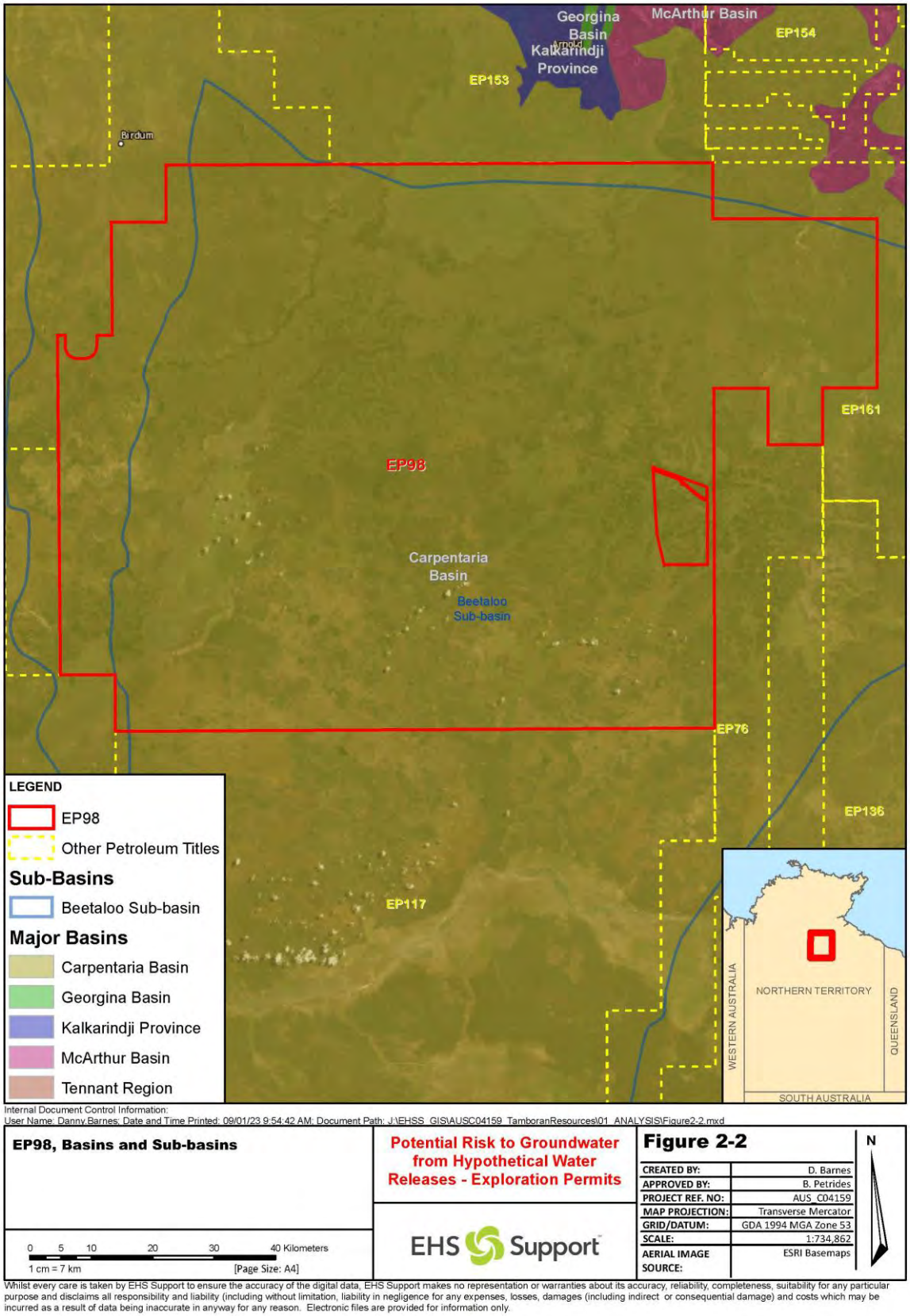


Figure 2-2 EP98, Basins and Sub-Basins

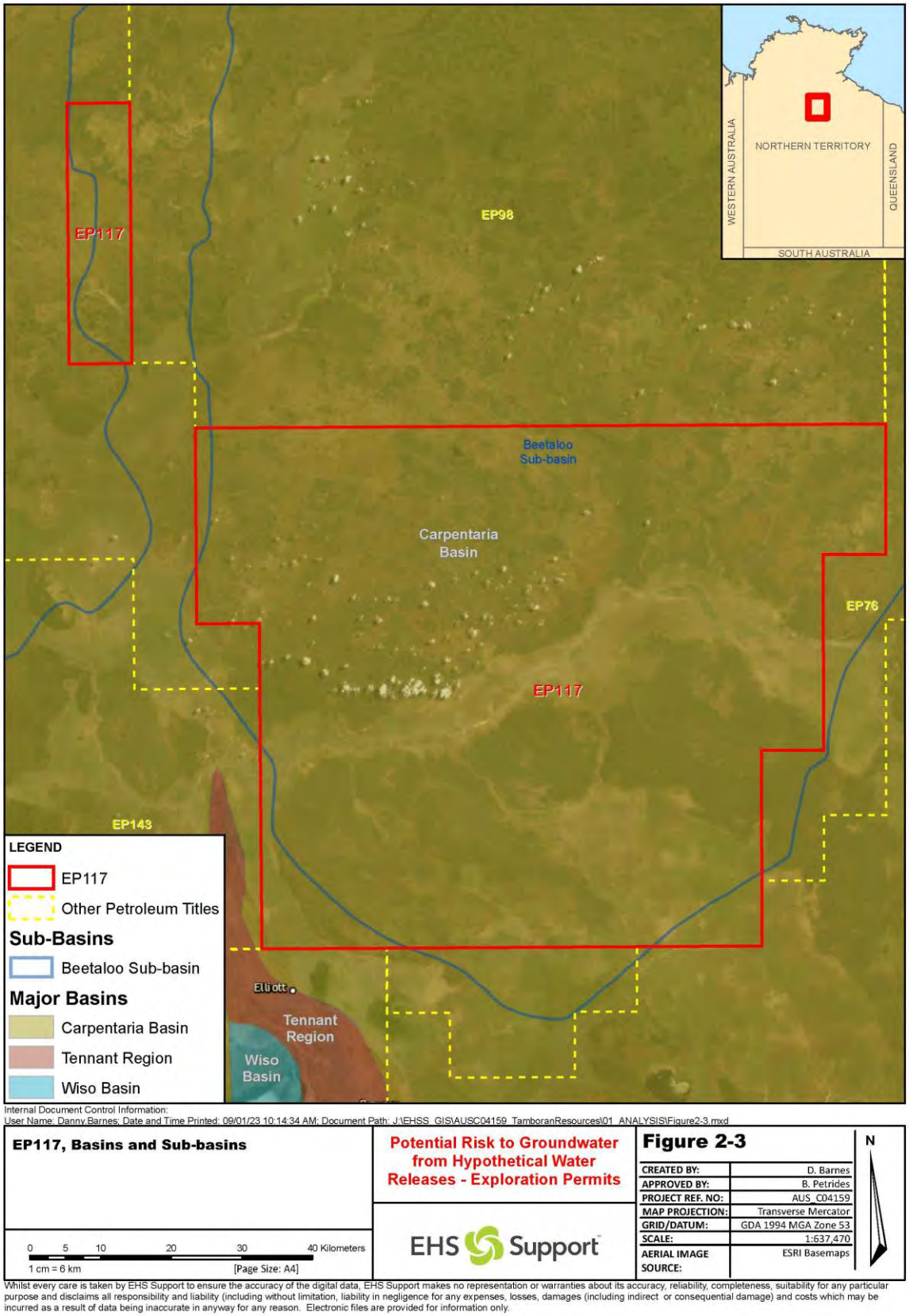


Figure 2-3 EP117, Basins and Sub-Basins

2.3 Stratigraphic Overview in Each Exploration Permit

The shallow (<100 m) hydrostratigraphic sequence within each EP was evaluated by reviewing petroleum drillholes, where present, groundwater extraction licence well construction logs, and other stock and domestic supply well construction logs. These shallow sequences are most susceptible to impacts associated by a release or "spill" of fluids. The breakdown of available information is presented in **Table 2-3**.

Table 2-3 Available Stratigraphic Information from Existing Drillholes and Wells

Exploration Permit	# of Petroleum Drillholes	# of Groundwater Extraction Licenced Wells	# of Other Registered Use Wells
EP76	1	1	23
EP98	9	3	149
EP117	3	1	83

Figure 2-4 shows the petroleum drillholes, groundwater extraction licenced wells and stock and domestic supply wells in each of the exploration permits.

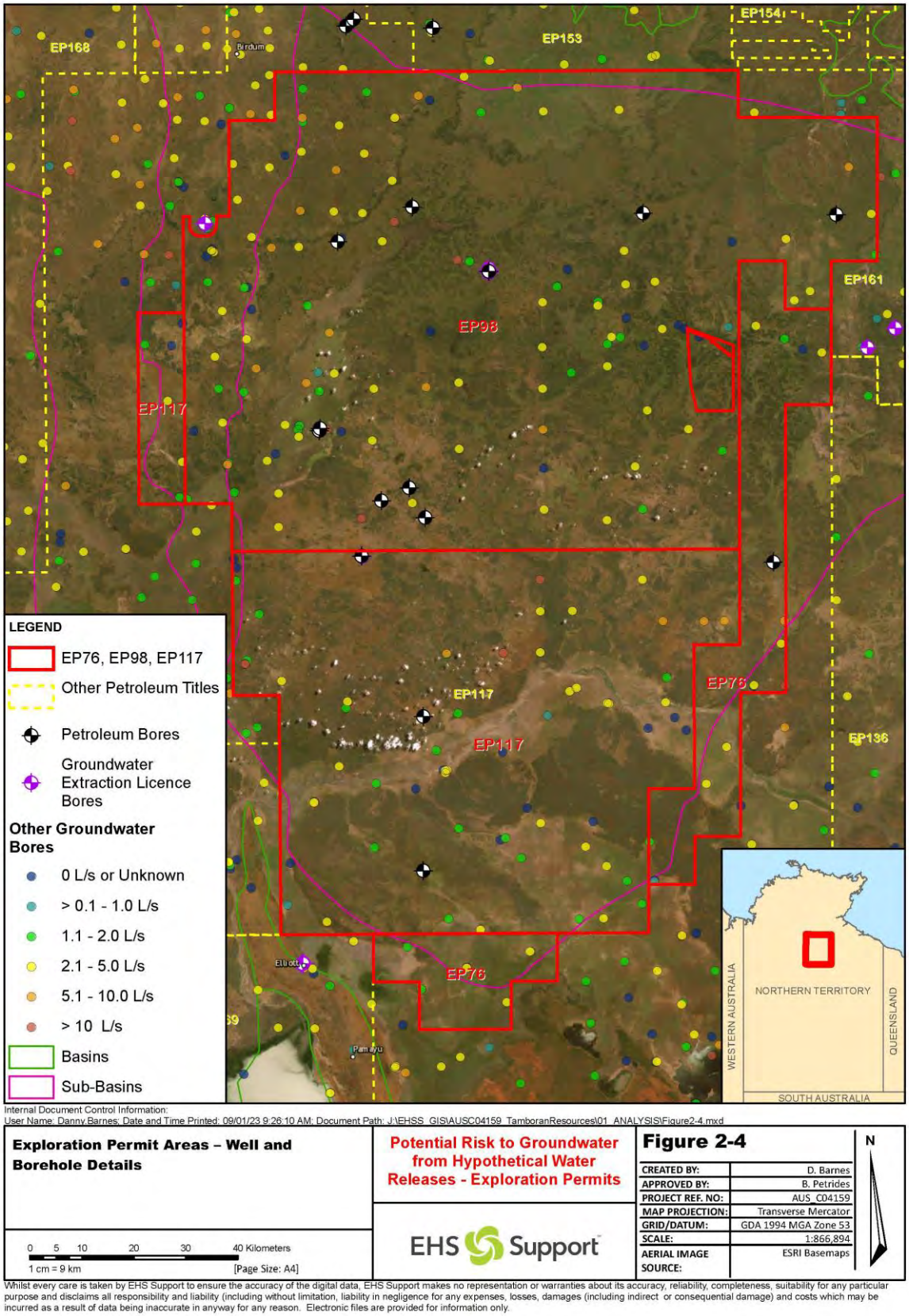


Figure 2-4 Exploration Permit Areas – Well and Borehole Details

2.3.1 Exploration Permit 76

EP76 covers an area of approximately 1,880 km^2. One petroleum well has been drilled in this EP; Velkerri 76 S2-1 and based on the basic well completion report (**Figure 2-5**) the generalised lithology is described in **Table 2-4**.

In this EP, the Anthony Lagoon Formation, comprising sandstone and dolomitic/siltstone/limestone and the Gum Ridge Formation comprising fossiliferous siltstone and chert and limestone form the major aquifer in the region. Groundwater yields in these fractured and karstic rocks have been recorded between 5.0 and 15.0 L/sec.

Table 2-4 EP76 – Generalised Stratigraphy

Depth From (mbgl)	Depth to (mbgl)	Lithology	Hydrogeological Unit
0	60	Undifferentiated sediments (Clay)	-
60	350	Limestone	Anthony Lagoon Formation/Gum Ridge Formation

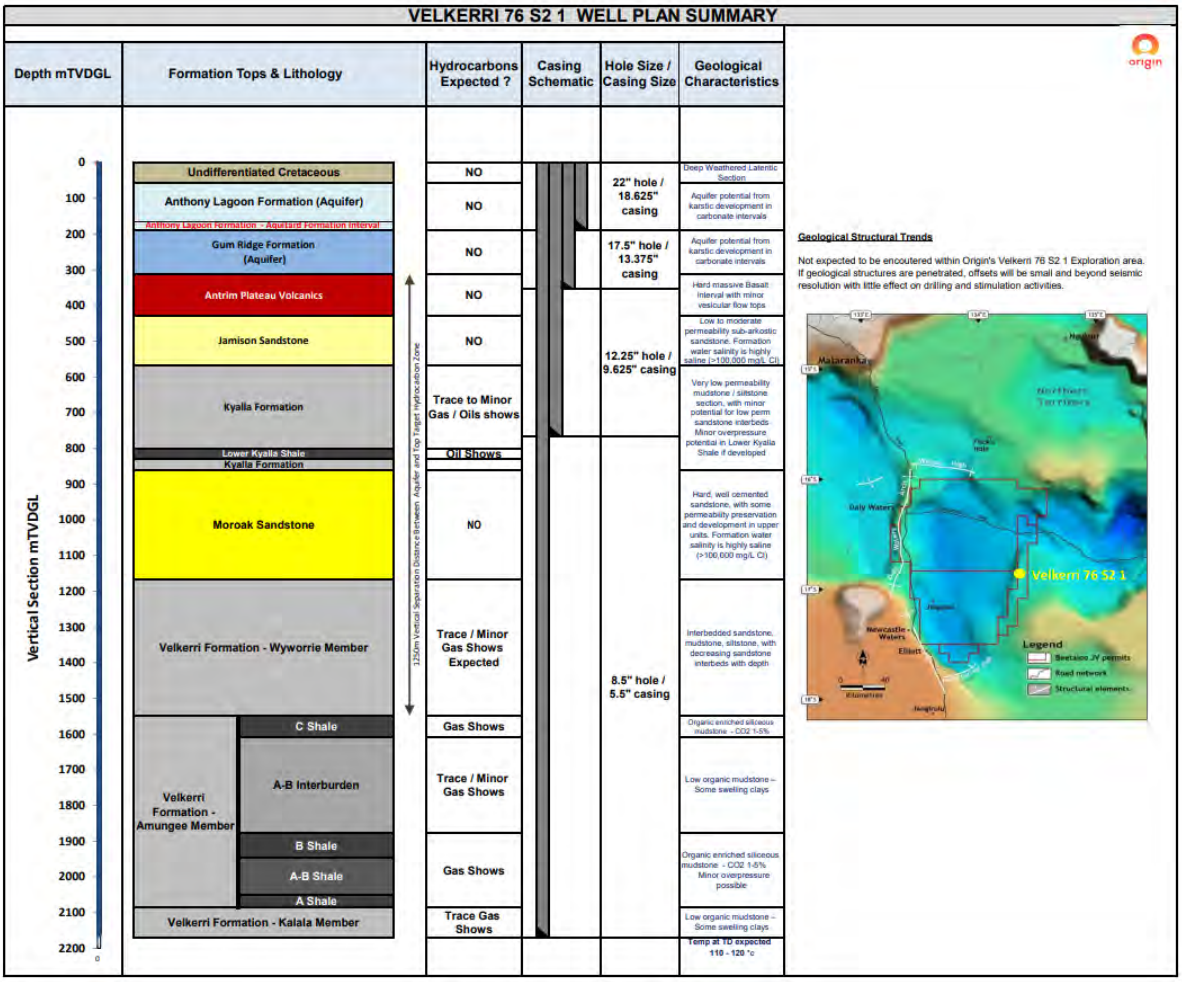


Figure 2-5 Well Plan Summary – Velkerri 76 S2-1

2.3.2 Exploration Permit 98

EP98 covers an area of approximately 10,124 km^2. Fifteen petroleum wells have been drilled in this EP: Chanin 1, Kalala South 1, Amungee (Amungee NW1, Amungee NW 1H, Amungee NW 1H Re-entry, Amungee NW-2H), Ronald 1, Burdo 1, Balmain 1, Mason 1, Shortland 1, Jamison 1, and Shenandoah (Shenandoah 1, Shenandoah 1A, Shenandoah 1A Re-entry). Based on the basic well completion reports, the generalised lithology is described in **Table 2-5** and shown on **Figure 2-6**.

Table 2-5 EP98 – Generalised Stratigraphy

Depth From (mbgl)	Depth to (mbgl)	Lithology	Hydrogeological Unit
0	80	Undifferentiated sediments (Clay)	-
80	220	Limestone	Gum Ridge Formation*

*Historically this unit has been mapped as the Tindall Limestone

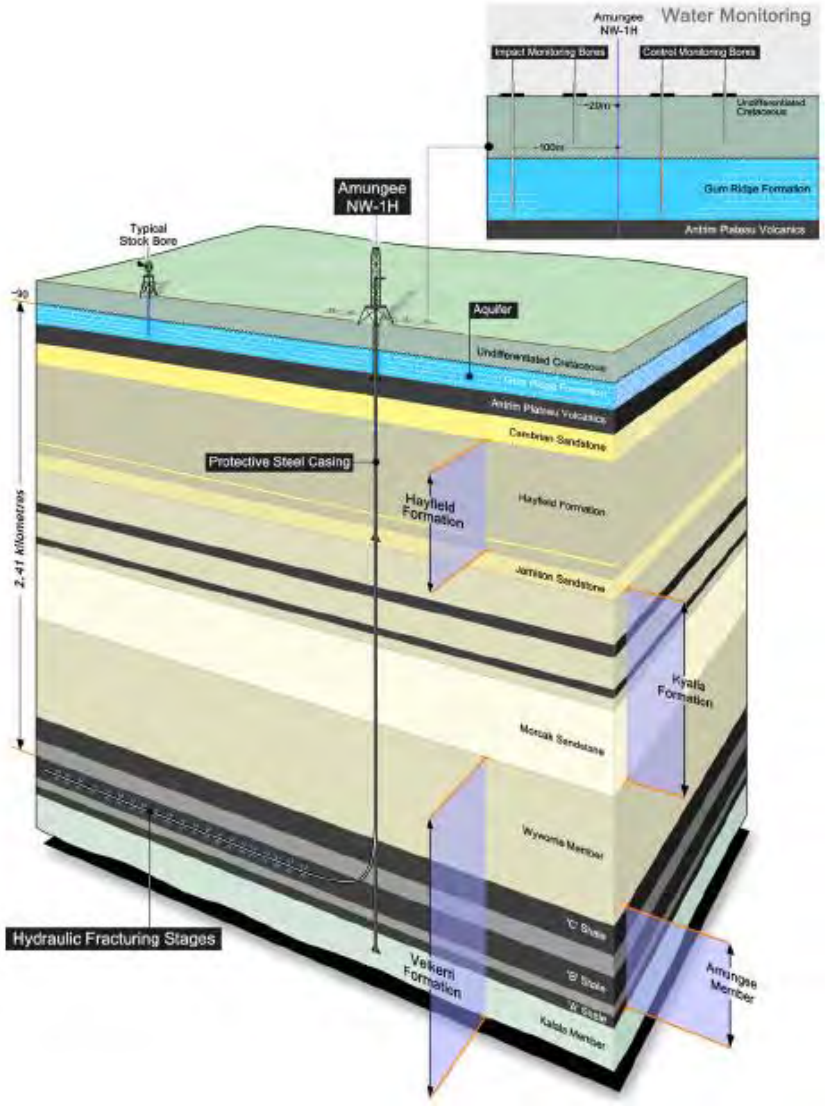


Figure 2-6 Schematic of the Existing Amungee NW-1H Well

2.3.3 Exploration Permit 117

EP117 covers an area of approximately 6,375 km^2. One petroleum well has been drilled in this EP; Beetaloo W-1 and based on the basic well completion report (**Figure 2-7**) the generalised lithology is described in **Table 2-6.**

Table 2-6 EP117 – Generalised Stratigraphy

Depth From (mbgl)	Depth to (mbgl)	Lithology	Hydrogeological Unit
0	116	Undifferentiated sediments (Clay)	-
116	436	Limestone	Anthony Lagoon Formation/Gum Ridge Formation

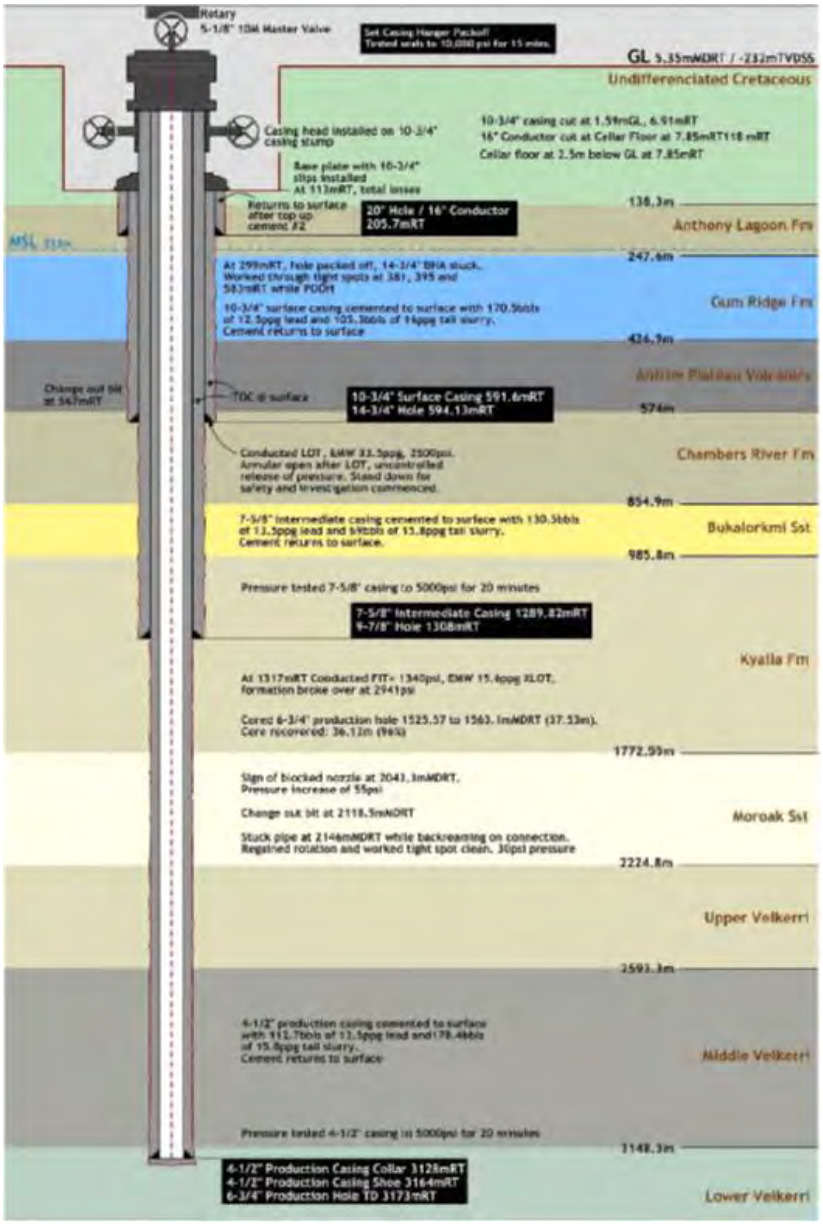


Figure 2-7 As Drilled Schematic of Beetaloo W-1

2.4 Hydrogeology

Within the Beetaloo exploration area, groundwater use is primarily from the CLA with minor, localised use from formations where shallower groundwater is intersected, where the CLA is too deep, or where the CLA is absent from erosion. This includes:

- Overlying Cretaceous sediments where it is saturated in the central-south of the Beetaloo Sub-basin;
- Antrim Plateau Volcanics in the north-west; and
- Bukalara Sandstone in the north-east.

The CLA, comprising the Gum Ridge Formation and the Anthony Lagoon Formation, is an extensive regional aquifer system that forms the principal water resource in the Beetaloo Sub-Basin.

In the vicinity of the Amungee NW site, the Anthony Lagoon Formation is interpreted as being eroded by the Base Cretaceous unconformity. At Amungee NW the Gum Ridge Formation is the upper water bearing aquifer unit with a standing water depth of approximately 106 m below ground level (mbgl).

At Velkerri 76 S2 and Beetaloo W-1, the Anthony Lagoon Formation forms the upper water bearing aquifer with the groundwater level at approximately 89 mbgl and 73 mbgl respectively.

The limestone in the Gum Ridge Formation is commonly fractured and cavernous with recorded bore yields up to 100 L/s from this aquifer. At both Amungee NW and Velkerri 76 S2, yields in excess of 20 L/sec were achieved with minimal (<1 m) aquifer losses.

Approximately 80% of groundwater bores drilled in the basin screen the CLA, and the aquifer supplies water for the pastoral industry and local communities, including Elliot, Daly Waters, Larrimah, and Newcastle Waters. The CLA contains a significant but largely undeveloped groundwater resource with the sustainable yield from the Georgina Basin estimated at 100,000 ML/year (NALWTF, 2009). Existing groundwater use in the Beetaloo Sub-Basin is estimated at 6,000 ML/year, primarily used for agricultural production (Foulton and Knapton, 2015).

The Antrim Plateau Volcanics conformably underlies the CLA in the north and central part of the Beetaloo Sub-Basin. Much of the Sub-Basin consists of sequences of massive basalt flows with negligible primary porosity. The north-west portion of the Sub-Basin forms a marginal aquifer where the formation is shallow and fractured; however, reported use is primarily from a sandstone sequence at the contact with the Gum Ridge Formation. There is no reported use within the three petroleum EPs held by Origin.

The Bukalara Sandstone forms a fractured and weathered aquifer where it outcrops beyond the north-east margin of the Beetaloo Sub-Basin. The formation consists of quartz sandstone with shale interbeds and probable enhanced permeability in these areas due to jointing within the sandstone. No use is reported from the formation away from the north-east margin of the Beetaloo Sub-Basin where it is at considerable depth. This unit, if present, will be protected through intermediate casing and cement.

The regional groundwater flow direction in the CLA is north-west toward Mataranka, where the aquifer discharges into the Roper River and supports significant groundwater dependent ecosystems (aquatic, riparian and floodplain), including the Roper River at Elsey National Park and Red Lily/57 Mile Waterhole. These discharge features occur around 100 km north-west of the Beetaloo Sub-Basin. Dry season flow in the Roper River has been gauged at 95,000-126,000 ML/yr and provides an

estimate of the magnitude groundwater discharge from the CLA. Large decadal changes in the discharge to the Roper River suggest that most recharge input occurs close to the discharge zone (i.e., beyond the Beetaloo Sub-Basin region). Groundwater recharge mechanisms to the CLA are poorly characterised but are likely to be dominated by infiltration through sinkholes and preferential recharge through soil cavities.

3 Analytical Assessment (Methodology)

Liquid releases on a permeable soil surface undergo three main processes that control the extent of the release and the subsequent environmental impacts. These processes are:

- Overland flow (runoff);
- Evaporation; and
- Infiltration.

In this assessment, overland flow (also referred to as runoff) is assessed along with infiltration.

3.1 Lateral Spreading of Fluid/Runoff

Runoff of water as a fluid dynamical process has concurrently been an important research topic with surface water hydrology and is typically described with the use of the Saint Venant equations (Woolhiser and Liggett, 1967). However, only recently has runoff been coupled with surface infiltration at a spatial scale that can be applicable to point source flows, such as release from a pipeline. Esteves et al. (2000) provides a list of theoretical models that include the basic elements of a liquid release on land.

The approach adopted for this assessment is a progression of the Green and Ampt (1911) model (**Section 3.2.1**). In essence, the Green and Ampt model approximates the curved soil moisture profiles allowing the calculation of the soils' infiltration capacity. The remaining water balance component is therefore runoff. This is visually presented in **Figure 3-1**.

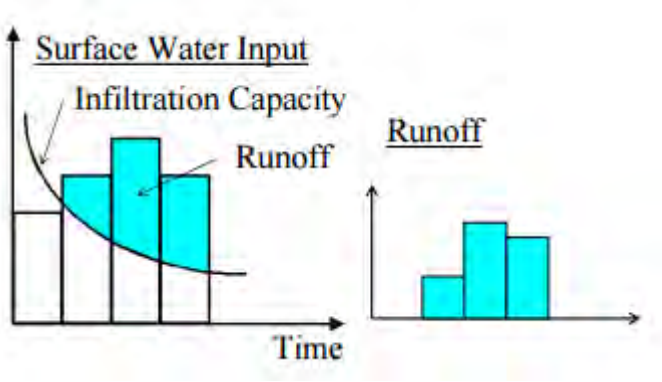


Figure 3-1 Conceptualisation of the Green and Ampt Model and the Remaining Runoff

Due to the regional approach and the complexity of this assessment, slight modifications to mathematical theory behind this and similar models were undertaken to predict the regional scale flow characteristics from a point source.

Whilst the Green and Ampt (1911) equation was used to assess the initial infiltration depths, modifications to the algorithm developed by Grimaz et al. (2007) and the Manning Kinematic Equation were adopted to model the remaining water assumed to be runoff. These analytical steps are provided in **Section 3.1.1**.

3.1.1 Water Pooling on Flat Surfaces

For instantaneous releases on flat surfaces (and assuming this water bypasses any bunded walls), the formulae (Equation 1) proposed by Grimaz et al. (2007) was used to estimate the area of the

pool of liquid on flat ground. This method is used for oil spills but can allow for water by varying the liquid properties (primarily viscosity and permeability).

$$A_{pool} \cong 2.3782 \frac{Q^{4/5}}{(k_i k_r)^{1/5}} \quad (1)$$

Where:

A_{pool} = the area of the pool of liquid on the surface [m^2]
Q = the total amount of liquid released [m^3]
k_i = the intrinsic permeability of soil [m^2]
k_r, = the relative permeability of the liquid [-].

The values of k_r, which vary with different grades of water saturation of soil, are shown in **Table 3-1**. For the conservative nature of this assessment, a k_r value of 0.3 will be assumed.

Table 3-2 provides the intrinsic permeability values used for sand and clay soil profiles. Sand and clay were chosen as these represent the extremes of potential infiltration and therefore bound the conditions observed in soils within the Area of Interest.

Table 3-1 Relative Permeability k_r, for Different Scenarios of Accidental Release

Soil Situation	k_r
Dry: long time without rainfall in warm regions and in hot seasons	1
Slightly wet: long time without rainfall in other regions or seasons	0.9
Very wet: from 2 hours to 2 days after strong rainfall	0.3
Completely saturated: during strong rainfall with ponds on surface	0

Table 3-2 Values of Intrinsic Permeability and Kinematic Viscosity for Sand and Clay

Soil situation	k_i
k_i = intrinsic permeability of soil (m^2)	
Sand	1.00E-08
Clay	1.00E-13

3.2 Infiltration into Unsaturated Zone

The spilt fluid will not only tend to spread out over the surface of the soil and evaporate but will also penetrate into the ground (unless it is impermeable). Infiltration to the unsaturated zone, and in particular infiltration capacity and time for ponding to occur, can be determined using the Green and Ampt (1911) infiltration equation.

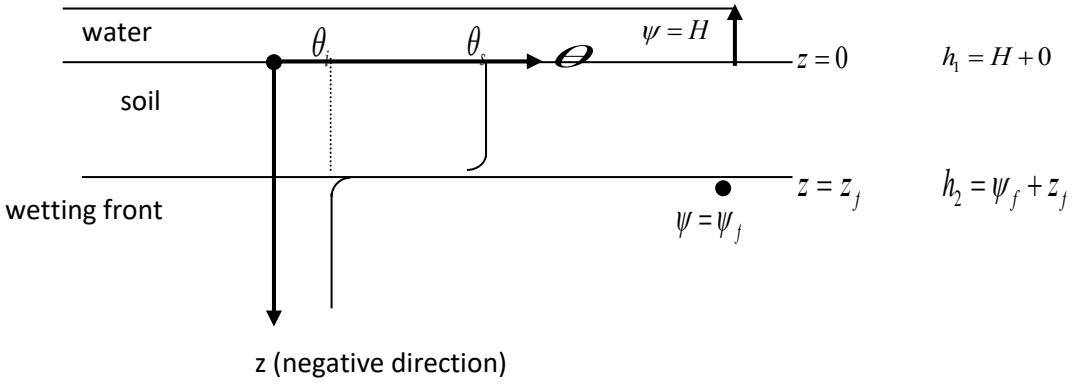
The infiltration rate actually experienced in a given soil depends on the amount and distribution of soil moisture and on the availability of water at the surface with a maximum rate at which the soil in a given condition can absorb water. This upper limit is called the infiltration capacity, f_c, and is a limitation on the rate at which water can move into the ground. If surface water input is less than infiltration capacity, the infiltration rate will be equal to the surface water input rate (w). If irrigation (analogous to a release) intensity exceeds the ability of the soil to absorb moisture, infiltration occurs at the infiltration capacity rate until the soil is saturated and ponding and associated runoff occurs. Infiltration capacity declines over time until a steady state is reached.

Several processes combine to reduce the infiltration capacity. The filling of fine pores with water reduces capillary forces drawing water into pores reducing the storage potential of the soil. Clay swells as it becomes wetter, and the size of pores is reduced. Coarse-textured soils such as sands have large pores which water can easily drain, while the fine pores in clays retard drainage. If the soil particles are held together in aggregates by organic matter or a small amount of clay, the soil will have a loose, friable structure that will allow rapid infiltration and drainage.

The calculation of infiltration at a point combines the physical conservation of mass (water) principle expressed through the continuity equation with quantification of unsaturated flow through soils, expressed by Darcy's equation. The downward hydraulic gradient inducing infiltration is from a combination of the effect of gravity, quantified by the elevation head, and capillary surface tension forces, quantified by the pressure head (negative due to suction) being lower at depth due to lower moisture content. If the water input rate is greater than the saturated hydraulic conductivity (i.e., w is greater than K_{sat}), at some point in time the water content at the surface will reach saturation. At this time, the infiltration capacity drops below the surface water input rate and runoff is generated. This time is referred to as the ponding time. After ponding occurs, water continues to infiltrate, and a zone of saturation begins to propagate downward into the soil as the wetting front. After ponding, the infiltration rate is less than the water input rate and the excess water accumulates at the surface and becomes infiltration excess runoff. As time progresses and the depth of the zone of saturation increases, the contribution of the suction head to the gradient inducing infiltration is reduced, so infiltration capacity is reduced. Once the soil profile is completely saturated no further water can infiltrate.

3.2.1 Green and Ampt Infiltration Model

The Green and Ampt (1911) model (Equation 2) is an approximation of the infiltration process described above and was utilised to assess infiltration capacity and time for ponding for various soils.



$$q = -K_s \frac{dh}{dz} = -K_s \frac{h_2 - h_1}{z_2 - z_1} = -K_s \frac{(\psi_f + z_f) - (H + 0)}{z_f - 0} = -K_s \frac{\psi_f + z_f - H}{z_f} \qquad (2)$$

Where

H = the depth of ponding, cm
Ks = saturated hydraulic conductivity (cm/s)
q = flux at the surface (cm/h) and is negative
f = suction at wetting front (negative pressure head)

θ_i = initial moisture content (dimensionless)

θ_s = saturated moisture content (dimensionless)

The following assumptions are implicit in the Green and Ampt equation:

- As water infiltrates, the wetting front advances at the same rate with depth, which produces a well-defined wetting front.
- The volumetric water content remains constant above and below the wetting front as it advances.
- The soil-water suction immediately below the wetting front remains constant with both time and location as the wetting front advances.

3.2.2 Darcy Infiltration Model

Once the soil has become permanently saturated (i.e., established) from a constant head driving behind the wetting front or when the Green and Ampt *flux (q) becomes constant*, Darcy's Law can be applied to determine the rate at which water can infiltrate vertically. This is shown in Equation 3.

$$qD = \frac{-K_{h,v}\frac{\Delta h}{\Delta l}}{n} \quad (3)$$

Where

qD = specific discharge of groundwater or Darcy Flux (m/day)
Kh,v = average hydraulic conductivity (vertical [Kv] or horizontal [Kh]) of the saturated sediment (m/day)
$\Delta h / \Delta l$ = hydraulic gradient driving the fluid (-)
η = effective porosity (-)

4 Analytical Assessment (Results)

This section presents the results of the assessment outlined in **Section 1.2** and the methodology (described in **Section 3.1** and **Section 3.2**) for determining:

- Lateral spreading/overland flow (**Section 4.1**);
- Infiltration into unsaturated zone (**Section 0**); and
- Infiltration rates under saturated flow conditions (**Section 4.3**).

4.1 Overland Flow

4.1.1 Overland Flow on Flat Surfaces

To assess the unmitigated risks from the improbable scenario where some fluids were to overflow the bunded area, a range of release scenarios are considered comprising:

1. Smaller release volumes of 1,000 L and 100,000 L, which would reflect small scale releases, and
2. An improbable release out of the bunded area (1,000,000 L).

Section 2 presents a summary of the recorded shallow lithology in each EP based on petroleum drillholes, licenced groundwater extraction wells, and stock and domestic supply wells. For modelling purposes, the shallow stratigraphy in each EP has been simplified. It is noted that this simplification allows for a more conservative evaluation of infiltration, as most surficial sediments in the Areas of Interest are composed of either natural clays or clays derived from weathering of the host rock.

Table 4-1 presents the simplified stratigraphy in each EP adopted for modelling, and model input parameters are provided in **Table 4-2**. It is noted that the shallow stratigraphy across the Areas of Interest are considered to be laterally equivalent and/or comprise similar hydraulic properties; these can be grouped into two main categories:

1. Low permeability formations including the Anthony Lagoon Beds.
2. Higher permeability formations including the Gum Ridge Limestone and Tindall Limestone.

For the purposes of assessing surface water pooling, soil properties reflective of a clay and more permeable sandier soils have been applied to Equation 1. These parameters are presented in **Table 3-1** and **Table 3-2.**

Table 4-1 Simplified Shallow Stratigraphy

Exploration Permit	Lithology	Hydrogeological Unit
EP76 & EP117	Clay overlying Limestone	Anthony Lagoon Beds/Gum Ridge Formation
EP98	Clay overlying Limestone	Gum Ridge Formation/ Tindall Limestone

Table 4-2 Modelling Input Parameters

Parameter	Anthony Lagoon Beds	Gum Ridge Limestone / Tindall Limestone /	Literature Source
Exploration Permit	EP76	EP76, EP98	
Porosity	0.482*	0.4**	* Dingman, 1994 **Knapton, 2006
Hydraulic Conductivity (K_{sat}) (m/d)	$8.6x10^{-4}$	0.864	Freeze, R. A., & Cherry, J. A. (1979).
Air-Entry Tension (cm)	40.5	12.1	Dingman, 1994
Saturated Tension (cm)	30.78	9.2	Dingman, 1994
Intrinsic permeability (m^2)	$1x10^{-13}$	$1x10^{-16}$	Dingman, 1994

Sources:
Dingman, S.L. 1994. Physical Hydrology Edition 5, Macmillan Publishing Company, 1994 ISBN 002329745X, 9780023297458 575 pages
Freeze, R.A. and Cherry, J.A. 1979. Groundwater. Prentice-Hall, Inc., Englewood Cliffs.
Knapton. 2006. Regional Groundwater Modelling of the Cambrian Limestone Aquifer System of the Wiso Basin, Georgina Basin and Daly Basin. Technical Report No. 29/2006A Department of Natural Resources, Environment & The Arts, Alice Springs.

Without the inclusion of bunding, a catastrophic release (1 ML) could impact an area of up to 94.7 ha if the surface geology remained consistent of a tight clay/sit representative of the Anthony Lagoon Beds. In the event of a smaller scale release of 1,000 L and prior to any bunds being established, these impacts would be highly localised being 0.4 ha (about half the size of a soccer field). It should be again stated, the above is a very conservative assessment.

Table 4-3 Model Results - Pooled Water Area

Stratigraphic Unit	Volume Released (L)	Volume Released (m^3)	Area (m^2)	Radius (m)	Comment
Anthony Lagoon Beds	1,000	1	3769.2	34.6	Releases of 1 to $100m^3$ improbable to over topping bunding walls.
	100,000	100	150054.3	218.5	
	1,000,000	1,000	946778.5	549.0	
Gum Ridge Limestone / Tindall Limestone	1,000	1	946.8	17.4	Releases of 1 to $100m^3$ improbable to over topping bunding walls.
	100,000	100	37691.9	109.5	
	1,000,000	1,000	237820.0	275.1	

4.2 Green and Ampt Infiltration Model

In addition to potential overland flow, infiltration into the sub-surface would occur. In the case of releases that are not contained within the bunded area, the infiltration rate would be slow due to the limited head of fluids within the release area, while in the bunded area, the retention of release fluids would provide a higher head as liquids could be present up to the height of the surrounding walls.

The results of the Green and Ampt Infiltration equation are discussed below and shown in **Figure 4-1**.

Recalling from **Section 2** and **Section 4.1.1** above, there are two distinct hydrogeological units (siltstone/clay and limestone) that extend across the Areas of Interest. The assessment therefore is based upon the time to infiltrate through both these formations.

Assuming the sub-surface is similar to the lower permeable units as defined in **Table 4-1** and **Table 4-2**, the results indicate that the ground would become quickly saturated (the infiltration capacity of the soils are exceeded). As a result, and spill would unlikely move to any significant depth as the majority of the water would run off, however for the water that remains and not inclusive of evaporation, any spill will take approximately between 40 days (Limestone) and >1000 days (Siltstone) to move through the top 1m. After 40 days it would be assumed the water on the surface would have evaporated. This is based on a saturated hydraulic conductivity of a siltstone/clay (K = 0.000001 cm/s [0.00086 m/d]) and for a limestone (K = 0.001 cm/s [0.864 m/d]).

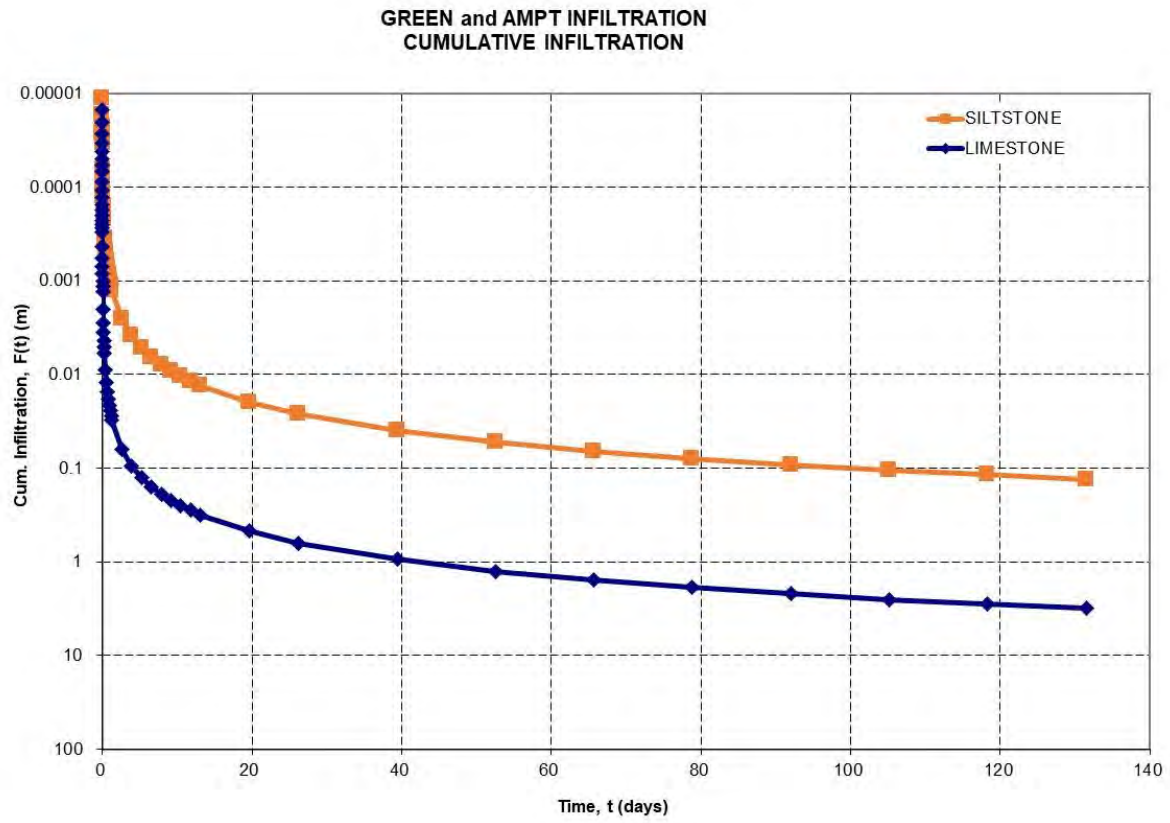


Figure 4-1 Results of the Green and Ampt Analytical Model for Limestone and Siltstone

Note: Siltstone equivalent to the Anthony Lagoon Beds. Permeable sandstone equivalent to the Gum Ridge Limestone / Tindall Limestone.

4.3 Darcy Infiltration Model

The results of the Darcy infiltration modelling are discussed as follows and shown in **Figure 4-2**. Adopting the same assumptions as presented in **Section 3.2.1**, (i.e., the sub-surface is similar to the units described in **Table 4-1** and hydraulic properties defined in **Table 4-2**) and that the water is available in the surface to act as a driving head (i.e., a consistent leak), the results indicate water will take approximately 400 days to move through the first 10 m and then approximately another 2,000 days to move through another 50 m (siltstone/clay). If the subsurface was equivalent to a limestone which is more permeable, water would take 200 days to reach 50 m depth, or the approximate depth of the water table.

It should be noted that this evaluation is highly conservative as it assumes the sub-surface is completely saturated and has a constant driving head. However, in reality the driving head will be removed, either by evaporation or remediation, well before the predicted travel time is reached.

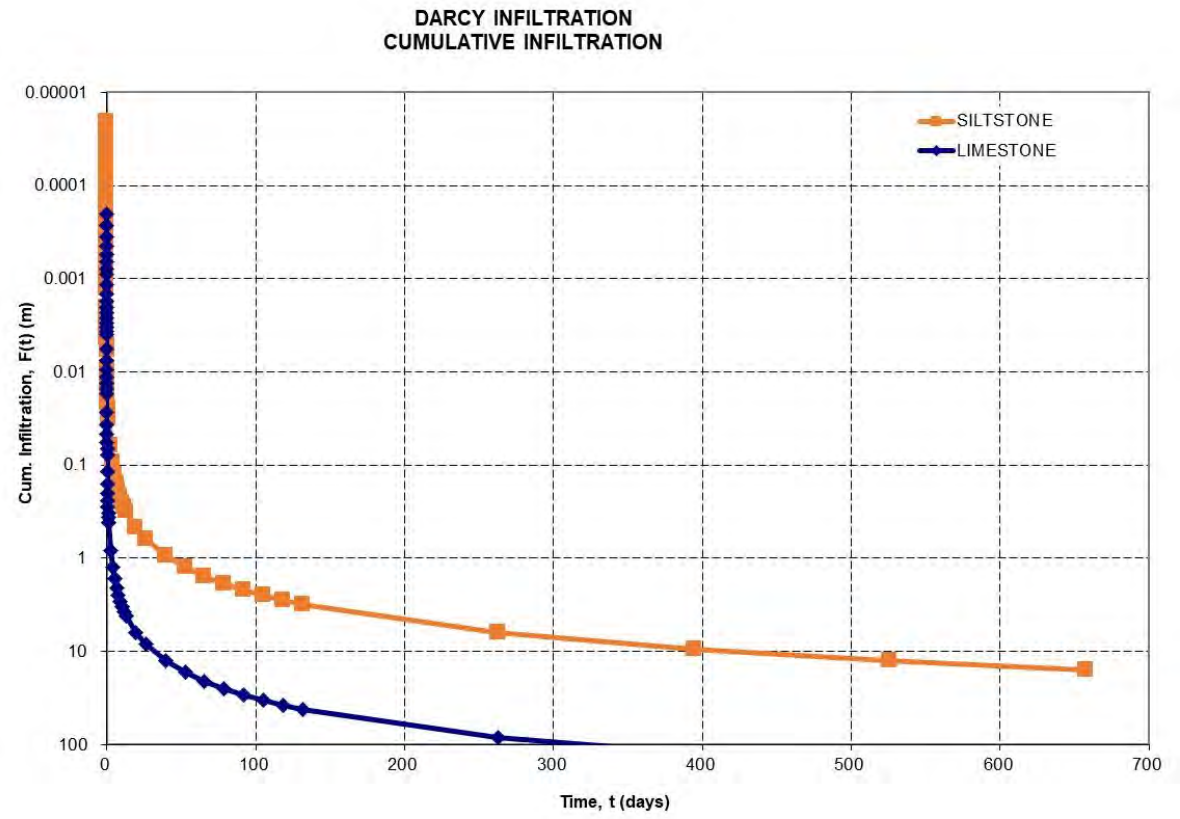


Figure 4-2 Results of the Darcy Analytical Model for Limestone and Siltstone

Note: Siltstone equivalent to the Anthony Lagoon Beds. Permeable sandstone equivalent to the Gum Ridge Limestone / Tindall Limestone.

5 Discussion

The results of this assessment present a very conservative estimate of the potential impacts to surface environmental receptors and groundwater. Its conservatism is inherent in the assumption that some of the scenarios considered that no risk mitigation measures were adopted and that the water releases were catastrophic.

In the context of smaller scale releases outside of the bunded area, this assessment indicates that spills of up to 1,000 L would only migrate a radial distance of 35 m. A catastrophic spill of 1,000,000 L on a relatively impermeable surface could migrate radially 549 m; however, this level of spill is considered highly unlikely with the assessment being highly conservative.

In the context of potential impact to groundwater via infiltration, modelling using both Green and Ampt (1911) and Darcy's equations (1856) (to assess unsaturated and saturated soils) has been conducted based on highly conservative assumptions. It has been determined that water would take 2,000 days to move through 50 m of siltstone/clay and 200 days for a lithology consistent with limestone. However, the modelling does not consider the capacity of the formation to retain water. In this context and based on the finite volume of water in the compound, it is not anticipated that a single release would infiltrate to groundwater.

With reference to potential sensitive receptors listed in **Table 1-1**, for the highly conservative and catastrophic release of 1,000,000 L of fluid, no sensitive receptors would be impacted.

6 Limitations

EHS Support Pty Ltd (EHS Support) has prepared this report in accordance with the usual care and thoroughness of the consulting profession for the use of Condor and only those third parties who have been authorised in writing by EHS Support to rely on the report. It is based on generally accepted practices and standards at the time it was prepared. No other warranty, expressed or implied, is made as to the professional advice included in this report. It is prepared in accordance with the scope of work and for the purpose outlined in the Proposal email dated 2 August 2022.

The methodology adopted and sources of information used by EHS Support are outlined in this report. EHS Support has made no independent verification of this information beyond the agreed scope of works and EHS Support assumes no responsibility for any inaccuracies or omissions. No indications were found during our investigations that information contained in this report as provided to EHS Support was false.

This report was prepared in December 2022 and January 2023 and is based on the information reviewed at the time of preparation. EHS Support disclaims responsibility for any changes that may have occurred after this time.

This report should be read in full. No responsibility is accepted for use of any part of this report in any other context or for any other purpose or by third parties. This report does not purport to give legal advice. Legal advice can only be given by qualified legal practitioners.

This report contains information obtained by inspection, sampling, testing or other means of investigation. This information is directly relevant only to the points in the ground where they were obtained at the time of the assessment. The borehole logs indicate the inferred ground conditions only at the specific locations tested. The precision with which conditions are indicated depends largely on the frequency and method of sampling, and the uniformity of conditions as constrained by the project budget limitations. The behaviour of groundwater and some aspects of contaminants in soil and groundwater are complex. Our conclusions are based upon the analytical data presented in this report and our experience. Future advances in regard to the understanding of chemicals and their behaviour, and changes in regulations affecting their management, could impact on our conclusions and recommendations regarding their potential presence on this site.

Where conditions encountered at the site are subsequently found to differ significantly from those anticipated in this report, EHS Support must be notified of any such findings and be provided with an opportunity to review the recommendations of this report.

Whilst to the best of our knowledge information contained in this report is accurate at the date of issue, sub-surface conditions, including groundwater levels can change in a limited time. Therefore, this document and the information contained herein should only be regarded as valid at the time of the investigation unless otherwise explicitly stated in this report.

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Appendix C Risk Dossiers

1,6 HEXANEDIOL

This dossier on 1,6 hexanediol presents the most critical studies pertinent to the risk assessment of 1,6 hexanediol in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed 1,6-hexanediol in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment.

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Hexane-1,6-diol

CAS RN: 629-11-8

Molecular formula: $C_6H_{14}O_2$

Molecular weight: 118.17 g/mol

Synonyms: alpha,omega-Hexanediol, HDO, Hexamethylene glycol, Hexamethylenediol, Hexane-1,6-diol, Adipol

SMILES: C(CCCO)CCO

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-Chemical Properties of 1,6 hexanediol

Property	Value	Klimisch Score	Reference
Physical state at 20oC and 101.3 kPa	Solid colourless crystalline	2	ECHA
Melting Point	39.5-42.1^{o}C	3	ECHA
Boiling Point	250^{o}C @ 101.3 kPa	3	ECHA
Density	960 kg/m3@ 20^{o}C	2	ECHA
Vapour Pressure	0.1Pa @ 25^{o}C	2	ECHA
Partition Coefficient (log K_{ow})	0@25^{o}C	2	ECHA
Water Solubility	1000 g/L @ 20^{o}C	2	ECHA
Flash Point	136^{o}C @ 101.3 hPa	2	ECHA
Auto flammability	320^{o}C @ 101.3 hPa	2	ECHA
Viscosity	Not applicable	-	ECHA
Henry's Law Constant	Not applicable	-	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

1,6-Hexanediol is expected to degrade in the environment, has a low potential for adsorption, and is unlikely to bioaccumulate. Specific data are discussed below.

B. Biodegradation

Degradation studies were conducted according to OECD guideline 301C using municipal activated sludge without preconditioning. After 28 days a DOC removal of 98% and a biological oxygen demand of 95% (BOD/ThOD) was measured. This result is supported by a literature study, which showed a DOC removal > 90% after 7 days in a test according to OECD 301A also using municipal activated sludge. [Kl Score = 3] (ECHA). Therefore, the substance is expected to biodegrade rapidly.

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

1,6-Hexanediol is not expected to adsorb to suspended solids and sediment based upon the K_{oc} of 1.01 and the log K_{oc} of 0.004 as calculated by use from EPISUITE™ using the MCI method.

If 1,6 hexanediol is released to water, it is not expected to absorb to suspended soils and sediments based on its high water solubility and low K_{oc} value.

D. Bioaccumulation

No bioaccumulation studies were conducted. Due to the low log K_{ow}, of 0, bioaccumulation in organisms is not expected [Kl. score = 2].

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

1,6 hexanediol has low acute toxicity and it is rapidly excreted via urine. It is not irritating to the skin or the eye. 1,6 hexanediol is not a skin sensitizer nor is genotoxic.

B. Metabolism

An oral gavage study in Chinchilla rabbits were administered 2 mmol per kilogram body weight (mmol/kg bw) of 1,6 hexanediol via oral gavage. Roughly 4-9 % of the administered dose was excreted as glucuronide in the urine. Another urinary metabolite was adipic acid, which is the product that results from oxidation of both the hydroxyl groups of the parent compound (ECHA) [KI score =1].

C. Acute Toxicity

The oral LD_{50} in rats is approximately 3,000 milligrams per kilogram (mg/kg) (ECHA) [Kl. score = 2]. The 8-hour LC_{50} in rats is >3.3 mg/L air (ECHA) [Kl. score = 2]. The dermal LD_{50} in rabbits is >2,500 mg/kg (ECHA) [Kl. score = 2].

D. Irritation

Application of 1 millilitre (mL) to the skin of rabbits for 20 hours under occlusive conditions was not irritating. The mean of the 24, 48, and 72-hour scores were 0.00 for both erythema and oedema (ECHA) [Kl. score = 2].

Instillation of 0.1 mL into the eyes of rabbits was not irritating. The mean of the 24, 48, and 72-hour scores were: 0.00 for corneal opacity; 0.00 for iridial lesions; 1.70 for conjunctival redness; and 1.00 for chemosis (ECHA) [Kl. score = 1].

E. Sensitisation

1,6-Hexanediol was not considered a skin sensitizer when tested in a guinea pig maximization test (ECHA) [Kl. score = 1].

F. Repeated Dose Toxicity

Oral

Male and female Wistar rats were dosed with 0, 100, 400, or 1,000 mg/kg 1,6-hexanediol by oral gavage for 28 days. There were no substantial treatment-related effects regarding feed consumption, body weight, body weight gain, clinical chemistry parameters, clinical signs, gross pathology, or histopathology. The no observed adverse effects level (NOAEL) for this study is 1,000 mg/kg-day, the highest dose tested (ECHA) [Kl. score = 1].

Male and female Wistar rats were dosed with 0, 100, 400, or 1,000 mg/kg 1,6-hexanediol by oral gavage for 91-92 days (male and female respectively). There were no treatment effects observed in the female group, so the NOAEL was determined to be 1,000 mg/kg/day. The NOAEL for this study is 400 mg/kg-day based on reduced body weight in male rats(ECHA) [Kl. score = 1].

Inhalation

There are no studies available.

Dermal

There are no studies available

G. Genotoxicity

In Vitro Studies

The *in vitro* genotoxicity studies on 1,6 hexanediol are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on 1,6 hexanediol

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (S. typhimurium strains	-	-	2	ECHA

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (S. typhimurium strains	-	-	2	ECHA
Mammalian cell gene mutation (HGPRT, Chinese hamster V79 cells)	-	-	1	ECHA
Chromosomal aberrations (Chinese hamster V79 cells)	-	-	1	ECHA

*+, positive; -, negative

In Vivo Studies

No studies available

H. Carcinogenicity

No studies available.

I. Reproductive Toxicity/Developmental Toxicity

A reproductive/developmental toxicity screening (OECD TG 421) study has been conducted on 1,6-hexanediol. Male and female Wistar rats were dosed with 0, 100, 400, or 1,000 mg/kg-day by oral gavage for four weeks. There was no indication of reproductive or developmental toxicity. The 1,000 mg/kg males had reduced body weights and body weight gain. The NOAEL for reproductive and developmental toxicity is 1,000 mg/kg-day, the highest dose tested. The NOAEL for parental toxicity is 1,000 mg/kg-day for females and 400 mg/kg-day for males (ECHA) [Kl. score = 1].

Male and female Wistar rats were dosed with 0, 100, 400, or 1,000 mg/kg 1,6-hexanediol by oral gavage for 56 days. There were no treatment-related effects on oestrous cycle length and the number of cycles that were obtained. Sperm motility, the incidence of abnormal sperm in the cauda epididymis, and the sperm head counts in the testis and cauda epididymis were similar between treated and control males. The NOAEL for reproductive and developmental toxicity endpoints is 1,000 mg/kg-day, the highest dose tested (ECHA) [Kl. score = 1].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for 1,6 hexanediol follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

The lowest NOAEL from these studies is 400 mg/kg-day based on the absence of treatment-related effects in a reproductive and developmental toxicity study.

The NOAEL of 400 mg/kg-day from the four-week reproductive and developmental toxicity study will be used to determine the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 10
UF_D (database uncertainty) = 1
Oral RfD =400/(10 x 10 x 1 x 10 x 1) = 400/1000 = 0.4 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (0.4 x 70 x 0.1)/2 = 1.4 mg/L

B. Cancer

There is no evidence that 1,6 hexanediol is a carcinogen.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

1,6 hexanediol does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

1,6 hexanediol has low acute and chronic aquatic toxicity to algae, fish, and invertebrates.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on 1,6 hexanediol .

Table 3: Acute Aquatic Toxicity Studies on 1,6 hexanediol

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Leuciscus idus	96- hour LC_{50}	4,460-10,000	2	ECHA
Daphnia magna	48-hour EC_{50}	>500	2	ECHA
Desmodesmus subspicatus	72-hour EC_{50}	5,940	2	ECHA

Chronic Studies

The 72h EC_{10} for Desmodesmus subspicatus, also known as Scenedesmus subspicatus, is 1,180 mg/L(ECHA) [KI Score =2].

The 96h no observed effect concentration (NOEC) for Leuciscus idus is 2,200 mg/L based on mortality (ECHA) [KI Score =2].

C. Terrestrial Toxicity

The EC_{50} for Pseudomonas putida is >10,000 mg/L based on growth inhibition.

D. Calculation of PNEC

The PNEC calculations for 1,6 hexanediol follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E(L)C_{50} values are available for fish (4,460 mg/L) , *Daphnia* (>500 mg/L), and algae (5,490 mg/L).NOEC/72h EC_{10} values from long-term studies are available for algae (1,180 mg/L) and fish (2,200 mg/L). On the basis that the data consists of short-term results from three trophic levels and long-term results from two trophic levels, an assessment factor of 10 has been applied to the lowest reported E(L)C_{50} value of 500 mg/L for Daphnia. The E(L)C_{50} value is used because the value for Daphnia is lower than the NOEC values for all other trophic levels, including fish and algae. The $PNEC_{aquatic}$ is 50 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 32.03 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (0.82/1280) \times 1000 \times 50$
$= 32.031 mg/kg$

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1000 \times BD_{solid}]$

= 0.8 + [(0.2 x 0.0404/1000 x 2400]
= 0.82 m^3/m^3

Where:

Kp_{sed} = solid-water partition coefficient (L/kg)
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]
$Kp_{sed} = K_{oc} \times f_{oc}$
=1.01 x 0.04
= 0.0404 L/kg

Where:

K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for1,6 hexanediol calculated from EPISUITE™ using the MCI is 1.01 L/kg.
F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.67 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
= (0.02/1500) x 1000 x 50
= 0.67 mg/kg

Where:

Kp_{soil} = soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]
$Kp_{soil} = K_{oc} \times f_{oc}$
= 1.01 x 0.02
= 0.02 m^3/m^3

Where:

K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for 1,6 hexandiol calculated from EPISUITE™ using the MCI is 1.01 L/kg.
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

1,6 hexanediol is readily biodegradable and thus does not meet the screening criteria for persistence.

1,6 hexanediol has a low K_{ow}. Thus, 1,6 hexanediol does not meet the criteria for bioaccumulation.

The NOECs from the chronic aquatic toxicity studies on 1,6 hexanediol are > 0.1 mg/L. The acute $E(L)C_{50}$ values from the acute aquatic toxicity studies on 1,6 hexanediol are > 1 mg/L. Thus, 1,6 hexanediol does not meet the criteria for toxicity.

The overall conclusion is that 1,6 hexanediol is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Not Classified

B. Labelling

None

A. Pictogram

None

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

B. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

C. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

D. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established a value for this substance.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

E. Transport Information

1,6 Hexanediol is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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2-ETHOXY-NAPHTHALENE

This dossier on 2-ethoxy-naphthalene presents the most critical studies pertinent to the risk assessment of 2-ethoxy-naphthalene in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): 2-ethoxynaphthalene

CAS RN: 93-18-5

Molecular formula: $C_{12}H_{12}O$

Molecular weight: 172.2 g/mol

Synonyms: 2-ethoxy-naphthalene; 2-ethoxynaphthalene; Naphthalene, 2-ethoxy-; Bromelia; Ethyl β-naphtholate; Ethyl β-naphthyl ether

SMILES: O(C=1C=CC=2C=CC=CC2C1) CC

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-chemical Properties of 2-ethoxy-naphthalene

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	White powder	1	ECHA
Melting Point	35-37.1°C @ 96.93 kPa	1	ECHA
Boiling Point	300°C @96.88 kPa	1	ECHA
Density	1241.3 kg/m^3@ 20°C	1	ECHA
Vapour Pressure	0.518 Pa @ 25°C	2	ECHA
Partition Coefficient (log K_{ow})	3.75 @ 25°C	1	ECHA
Water Solubility	0.00001 g/L @ 30°C	1	ECHA
Flash Point	140.6 °C	1	ECHA
Auto flammability	Not applicable because the substance is a solid	-	ECHA
Viscosity	Not applicable because the substance is a solid	-	ECHA
Henry's Law Constant	Not applicable because the substance does not have an ionic structure	-	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

2-ethoxy-naphthalene is readily biodegradable in the environment. The substance will strongly adsorb to soil or suspended sediments and is insoluble in water. However, 2-ethoxy-naphthalene is not expected to bioaccumulate.

B. Biodegradation

An OECD Guideline 301 D (Ready Biodegradability: Closed Bottle) test was conducted to determine the biodegradability of 2-ethoxynaphthalene. The results of this study demonstrated that 2-ethoxy-naphthalene undergoes 33.45% biodegradation after 42 days of incubation at 20 ± 1 °C (ECHA)[KI. score =1].

These results indicate the 2-ethoxy-naphthalene is inherently biodegradable. If a chemical is found to be readily or inherently biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

An OECD Guideline 121 (Estimation of the Adsorption Coefficient K_{oc} on soil and on sewage sludge using high performance liquid chromatography HPLC) study was performed to determine the log K_{oc} for 2-ethoxy-naphthalene. The log K_{oc} value of 2-ethoxy-naphthalene was determined to be 3.490 ± 0.003 (K_{oc} = 3090) at 25°C. This log K_{oc} value indicates that 2-ethoxy-naphthalene has a strong sorption to soil and sediment and therefore has negligible to slow migration potential to ground water (ECHA)[KI. score =1).

The half-life period of 2-ethoxy-naphthalene in soil is estimated to be 30 days (720 hrs). Based on this half-life value of 2-ethoxy-naphthalene, it is concluded that the chemical is not persistent in the soil environment and the exposure risk to soil dwelling animals is moderate to low (ECHA).

D. Bioaccumulation

The bioconcentration factor (BCF) of 2-ethoxy-naphthalene was estimated using the EPISuite program (BCFBAF (v3.01) model) developed by the US EPA. The bioconcentration factor (BCF) of 2-ethoxy-naphthalene was estimated to be 136.6 L/kg whole body wet weight at 25°C. This result indicates that 2-ethoxy-naphthalene is not expected to bioaccumulate (ECHA) [KI. score =2].

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

2-ethoxy-naphthalene has low acute oral and dermal toxicity. This substance is not irritating to the skin, but it is slightly irritating to the eye. 2-ethoxy-naphthalene is not a skin sensitiser. 2-ethoxy-naphthalene is not mutagenic or genotoxic. There are no studies available to evaluate the carcinogenic potential of this substance. 2-ethoxy-naphthalene is not a reproductive or developmental toxicant.

B. Acute Toxicity

Oral

An OECD Guideline 423 (Acute Oral Toxicity-Acute Toxic Class Method) study was conducted female Sprague-Dawley rats exposed to 300 or 2000 mg/kg of 2-ethoxy-naphthalene via oral gavage. Gross pathological examination did not reveal any abnormalities in animals from 300 mg/kg and 2000 mg/kg dose groups. Therefore, the acute oral LD_{50} value of 2-ethoxy-naphthalene was considered to be >2000 mg/kg body weight (ECHA) [KI. score =1].

Inhalation

There are no studies available.

Dermal

An OECD Guideline 402 (Acute dermal toxicity) study was conducted using male and female Sprague-Dawley rats exposed to 2000 mg of 2-ethoxy-naphthalene via semi occlusive dressing for 24 hours. It was concluded that the acute dermal median lethal dose (LD_{50}) of 2-ethoxy-naphthalene was considered to be >2000 mg/kg body weight (ECHA) [KI. score =1].

An acute dermal toxicity study was conducted using rabbits exposed to 5,000 mg/kg bw/day of 2-ethoxy-naphthalene. The acute dermal LD_{50} value was considered to be >5,000 mg/kg bw (ECHA)[KI. score =2].

C. Irritation

Skin

An OECD Guideline 402 (Acute dermal toxicity) study was conducted using male and female Sprague-Dawley rats exposed to 2000 mg/kg of 2-ethoxy-naphthalene via occlusive dressing for 24 hours. Administration of the test item did not result in any signs of toxicity and mortality during the study period of 14 days. Animals exhibited normal body weight gain through the study period of 14 days. Gross pathological examination did not reveal any abnormalities attributable to the treatment. The overall irritation score of the substance was determined to be 0 and no erythema and oedema (skin irritation) were observed at the end of 14 days after patch removal. Hence, it was concluded that 2-ethoxy-naphthalene (CAS No. 93-18-5) was not-irritating to the skin of rats under the experimental conditions tested (ECHA) [KI. score =1].

Eye

An *in vivo* eye irritation study was conducted using New Zealand white rabbits exposed to a single exposure of 0.1 grams or undiluted 2-ethoxy-naphthalene. The individual mean score for animal nos. 1, 2 and 3 at 24, 48, 72 hours for corneal opacity, iris, conjunctiva and chemosis were found 1.00, 0.00, 2.00, 1.00; 1.00, 0.00, 2.00, 1.33, and 1.00, 0.00, 2.00, 1.33, respectively. The effects observed in all the animals were fully reversible within an observation period of 21 days. 2-Ethoxy-naphthalene was estimated to be slightly irritating to eyes. (ECHA) [KI. score =2].

D. Sensitisation

An Open Epicutaneous Test (OET) was performed on guinea pigs to assess the skin sensitisation potential of 3,10,30, or 100 % of 2-ethoxy-naphthalene. It was observed that none of the guinea pigs

induced contact sensitisation at challenge concentration of 2%. Thus, 2-ethoxy-naphthalene was considered to be not sensitising on skin of guinea pigs when tested via an Open Epicutaneous Test (OET) (ECHA) [KI. score =2].

E. Repeated Dose Toxicity

Oral

A sub chronic repeat dose oral toxicity study was performed using male and female FDRL rats exposed to 5.1 mg/kg (males) or 5.7 mg/kg (females) 2-ethoxy-naphthalene via their feed diluted in cotton seed oil for 90 days. Administration of 2-ethoxy-naphthalene for 90 days at a level in excess of at least 100 times the maximum estimated daily dietary intake in man evoked no adverse effect on growth, food consumption, haematology, blood chemistry, liver and kidney weights or on gross and microscopic appearance of major organs at autopsy. Hence, the No Observed Adverse Effect Level (NOAEL) for 2-ethoxy-naphthalene is considered to be 5.1 mg/kg bw/day in males and 5.7 mg/kg bw/day in females (ECHA) [KI. score =2].

A sub chronic repeat dose oral toxicity study was performed using rats exposed to 5 mg/kg bw/day of 2-ethoxy-naphthalene via oral gavage. There were no significant alterations were noted at the tested dose level. The NOAEL for 2-ethoxy-naphthalene was reported to be is 5.0 mg/kg bw/day (ECHA) [KI. score =2].

A sub chronic repeat dose oral toxicity study was conducted using rats exposed to 1,000 mg/kg bw/day (2%) of 2-ethoxy-naphthalene via their feed for 60 days. During the 2 months study period, the treated rats developed cataracts and 2-ethoxy-naphthalene was considered to be cataractogenic. Based on these observations, the NOAEL for 2-ethoxy-naphthalene was reported to be < 1000 mg/Kg/day (ECHA)[KI. score =2].

Inhalation

There are no studies available.

Dermal

There are no studies available.

F. Genotoxicity

In vitro Studies

The *in vitro* genotoxicity studies on 2-ethoxy-naphthalene are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on 2-ethoxy-naphthalene

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
OECD Guideline 471 Bacterial Reverse Mutation Assay (Salmonella typhimurium strains TA 100, TA 102, TA 98, TA 1535, and TA 1537	-	-	1	ECHA
Salmonella typhimurium TA 98, TA 100, TA 1535, TA 1537	-	-	2	ECHA

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Salmonella typhimurium TA 98, TA 100, T1535, TA 1537 and E. coli WP2 uvr A pKM 101	-	-	2	ECHA
Bacterial reverse mutation assay (S. typhimurium TA 98 TA 100 TA 1535, TA 1537, TA 15838	-	-	2	ECHA
In vitro cytogeneticity chromosome aberration study in mammalian cells (human peripheral blood lymphocytes and Chinese hamster fibroblast cell line, CHL)	-	-	2	ECHA
OECD Guideline 473 *In vitro* mammalian chromosome aberration test (human peripheral blood lymphocytes) **	-	-	1	ECHA
OECD Guideline 473 *In vitro* mammalian chromosome aberration test (Chinese hamster fibroblast cell line, CHL) ***	-	-	2	ECHA

*+, positive; -, negative
**Methyl 2-naphthyl ether (CAS RN 93-04-9)
***4-methoxybenzaldehyde (CAS RN 123-11-5)

In vivo Studies

A drosophila sex linked recessive lethal mutation (SLRL) assay was conducted to determine the mutagenic potential of 25 mM of 2-ethoxy-naphthalene in male drosophila melanogaster exposed to 2-ethoxy-naphthalene via their oral feed. Sex linked recessive lethal mutation were noted in the chromosomes. 2-ethoxy-naphthalene gave negative gene mutation results in the Drosophila SLRL test performed using male Drosophila melanogaster species (ECHA) [KI. score =2].

An *in vivo* micronucleus assay was performed to determine the mutagenic nature of 2-ethoxy-naphthalene in male and female NMRI mice exposed to 0,344, 603, 861 mg/kg of 2-ethoxy-naphthalene via intraperitoneal route of exposure for 24 hours. The micronucleus assay was performed using bone marrow smears of male and female NMRI mice. 2-ethoxy-naphthalene failed to produce genetic effects in this micronucleus assay (ECHA)[KI. score =2].

G. Carcinogenicity

Oral

There are no studies available.

Inhalation

There are no studies available.

Dermal

There are no studies available.

H. Reproductive Toxicity

A sub chronic oral feeding study was conducted using male and female FDRL rats exposed to 0, 5.1, 5.7 mg/kg bw/day of 2-ethoxy-naphthalene daily for 90 days. There were no adverse effects on body weight and food consumption or food efficiency throughout the administration period. Similarly, no effect on haematological parameters and organ weight of treated male and female rats were observed as compared to control. In addition, there were no gross pathological or histopathological changes observed in the treated male and female rats in liver, kidneys, stomach, small and large intestines, spleen, pancreas, heart, lungs, bone marrow, muscle, brain, spinal cord, bladder, adrenals, thyroid, pituitary, gonads, salivary glands, and lymph nodes as compared to control. Therefore, the NOAEL was considered to be 5.1 mg/kg bw/day for males and 5.7 mg/kg bw/day for females (ECHA) [KI. score =2].

A 28 day repeat oral toxicity study was conducted using male and female Sprague-Dawley rats exposed 0, 125, 250, and 500 mg/kg bw/day of 2-methoxynaphthalene via oral gavage. The results showed that methyl 2-naphthyl ether significantly increased the level of testosterone in the 500 mg/kg body weight/day group as well as it significantly increased the level of estrogen in the 250 mg/kg body weight/day group. The relative and absolute organ weight of ovaries decreased when treated with 125, 250 or 500 mg/kg body weight/day. In similarity, the relative and absolute organ weight of uterus decreased in the 125 or 500 mg/kg body weight/day groups. No significant changes in were detected in hematology, clinical biochemistry, mortality organ weight, and no effects were observed in water consumption, ophthalmoscopic examination or locomotor activity. In male rats, the relative organ weights of the testes and epididymides increased when rats were treated with 500 mg/kg body weight/day. Histopathology performed on reproductive organs after treatment with 500 mg/kg body weight/day did not reveal any toxic lesions as compared to control. Hence, NOAEL was considered to be 250 mg/kg bw/day when Sprague Dawley rats were exposed daily to test material by oral route for 28 days. (ECHA)[KI. score =1].

I. Developmental Toxicity

<u>Oral</u>

An OECD Guideline 414 (Prenatal developmental toxicity) study was conducted using New Zealand White rabbits exposed to 2-ethoxy-naphthalene via oral gavage for 15-30 days. The test material dissolved in 0.5% Carboxymethyl cellulose in dose concentration 0, 3, 10 and 50 mg/kg/day and administered by daily gavage through gestation day 6 to 28 to mated females (25/dose group).The preliminary range-finding study (0, 10, 60 and 300 mg/kg/day) was performed, Based on preliminary range-finding study findings, 0, 3, 10 and 50 mg/kg/day were selected for the main study. There were no maternal death or necropsy findings at any dose levels. There was a significant reduction in the body weight gain during the treatment period in the high dose group (50 mg/kg). The food consumption was comparable to the vehicle control group. The reduction in body weight during the treatment period was considered treatment related. One rabbit aborted in the high dose group, there were 2 non pregnant rabbits in control, 4 in low dose group, 3 in mid dose group and 4 in the high dose group. There was one complete resorption in mid dose group. At the end, at least 20 litters were observed in each of the dose groups. The maternal data parameters comprising of implantations, early and late resorptions, pre- and post-implantation loss in all the treatment groups were comparable to the vehicle control group. The mean number of corpora lutea, implantation and live foetus were significantly lower in high dose group (50 mg/kg bw/day) when compared with the control group. Observed decrease in corpora lutea at 50 mg/kg bw/day is considered as biological variation because the treatment was initiated after the implantation (gestation day 6). Therefore, the decrease observed in the absolute uterine weight, implantation and live foetus reported at this dose level are also considering as biological variation as these observations are directly correlated

with the decrease in the number of the corpora lutea. Hence, the NOAEL for developmental toxicity was considered to be 50 mg/kg/day (ECHA)[KI. score =2].

An OECD Guideline 414 (Prenatal developmental toxicity) study was conducted using Crl:CD BR VAF/Plus rats exposed to , 150, 300, 600, or 1000 mg/kg/day of 2-ethoxy-naphthalene via oral gavage for 15-30 days. Mortality was observed in the 1000 mg/kg/day dose group. Clinical signs such as tremors, uncoordinated movements, recumbent posture, languidness, cold body and decreased body weight gain were observed. Foetal body weights were decreased at 600 and 1000 mg/kg/day. Skeletal variations were seen at the 300 mg/kg/day and at higher doses. The skeletal variations manifested as increased unossified sternebrae, seventh cervical ribs, and misaligned sternebrae. Hence, the NOELs for maternal and developmental toxicity were considered to be 150 mg/kg/day (ECHA) [KI. score =2].

Inhalation

There are no studies available.

Dermal

There are no studies available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for 2-ethoxy-naphthalene follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

A 90-day sub chronic repeat dose oral toxicity study was performed using male and female FDRL rats exposed to 5.1 mg/kg (males) or 5.7 mg/kg (females) 2-ethoxy-naphthalene via their feed diluted in cotton seed oil for 90 days. . No adverse effect on growth, food consumption, haematology, blood chemistry, liver and kidney weights or on gross and microscopic appearance of major organs at autopsy was observed. The NOAEL for 2-ethoxy-naphthalene was reported to be is 5.1 mg/kg bw/day for males and 5.7 mg/kg bw/day for females (ECHA) [KI. score =2]. These NOAELs were supported by a 28-day sub chronic repeat dose oral toxicity study which reported no significant alterations at the dose level of 5.0 mg/kg bw/day in rats. The NOAEL of 5.1 mg/kg bw/day will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 10
UF_D (database uncertainty) = 1
Oral RfD = 5.1/(1 x 10 x 1 x 1 x 1) = 5.1/1000 = 0.005 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (0.005 x 70 x 0.1)/2 = 0.017 mg/L

B. Cancer

There are no studies available to evaluate the carcinogenic potential of 2-ethoxy-naphthalene.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

2-ethoxy-naphthalene does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

2-ethoxy-naphthalene is of low aquatic and terrestrial toxicity concern.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on 2-ethoxy-napthalene. Limited studies have been conducted since the substance is highly insoluble in water and aquatic toxicity is unlikely to occur (ECHA).

Table 3: Acute Aquatic Toxicity Studies on 2-ethoxy-napthalene

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Daphnia magna	72-h EC_{50}	3.9 (mobility)	1	ECHA

Chronic Studies

There are no studies available.

C. Terrestrial Toxicity

The test material 2-ethoxy-naphthalene is considered to have negligible direct or indirect exposure to soil. The half-life period of 2-ethoxy-naphthalene in soil is estimated to be 30 days (720 hrs). Based on this half-life value, it is concluded that the chemical is not persistent in the soil environment and the exposure risk to soil dwelling animals is moderate to low (ECHA).

D. Calculation of PNEC

The PNEC calculations for 2-ethoxy-napthalene follow the methodology discussed in DEWHA (2009).

PNEC Water

Because of the insolubility of the substance, experimental results are available for one trophic level (invertebrates). An acute EC_{50} value is available for Daphnia magna (3.9 mg/L). On the basis that the data consists of one short-term study for one trophic level and that the substance is not persistent in the environment, an assessment factor of 100 has been applied to the lowest reported EC_{50} value of 3.9 mg/L for invertebrates. The EC_{50} value is used because the value for invertebrates is the only value available for this substance. The $PNEC_{aquatic}$ is 0.039 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 1.832 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (60.1/1280) \times 1000 \times 0.039$
$= 1.832$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1000 \times BD_{solid}]$
$= 0.8 + [(0.2 \times 60.1/1000 \times 2400]$
$= 60.1\ m^3/m^3$

Where:
Kp_{sed} = solid-water partition coefficient (L/kg)
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]
$Kp_{sed} = K_{oc} \times f_{oc}$
$= 3090 \times 0.04$
$= 123.6$ L/kg

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for 2-ethoxy-naphthalene was determined from an OECD Guideline 121 study. The K_{oc} value was reported to be 3090 L/kg.
F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 1.61 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
= (61.8/1500) x 1000 x 0.039
= 1.61 mg/kg

Where:
Kp_{soil} = soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]
$Kp_{soil} = K_{oc} \times f_{oc}$
= 3090 x 0.02
= 61.8 m^3/m^3

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for 2-ethoxy-naphthalene was determined from an OECD Guideline 121 study. The K_{oc} value was reported to be 3090 L/kg.
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

2-ethoxy-napthalene is readily biodegradable and thus does not meet the screening criteria for persistence.

The estimated BCF for 2-ethoxy-naphthalene is 136.6 L/kg. Thus, 2-ethoxy-napthalene does not meet the criteria for bioaccumulation.

Because of the insoluble nature of the substance and the low potential for aquatic toxicity, there are no data from chronic aquatic toxicity studies for 2-ethoxy-naphthalene. The acute EC_{50} values from a single acute aquatic toxicity study on 2-ethoxy-naphthalene is > 1 mg/L. Thus, 2-ethoxy-naphthalene does not meet the criteria for toxicity.

The overall conclusion is that 2-ethoxy-naphthalene is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H411: Aquatic Chronic 2

H315: Skin irritation 2

H319: Eye irritation 2/2A

B. Labelling

Warning

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

There are no workplace exposure standards for 2-ethoxy-naphthalene in Australia.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

2-ethoxy-naphthalene is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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ACETIC ACID

This dossier on acetic acid presents the most critical studies pertinent to the risk assessment of acetic acid in its use in coal seam or shale gas extraction activities. It does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed acetic acid in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment.

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Acetic acid

CAS RN: 64-19-7

Molecular formula: $C_2H_4O_2$

Molecular weight: 60.1 g/mol

Synonyms: Acetic acid, ethanoic acid, ethylic acid, methane carboxylic acid, vinegar acid

SMILES: CC(=O)O

II. PHYSICO-CHEMICAL PROPERTIES

Key physical and chemical properties for the substance are shown in Table 1.

Table 1: Overview of the Physico-Chemical Properties of Acetic Acid

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Colourless liquid with a pungent odour.	2	ECHA
Melting Point	16.64°C @ 101.3 kPa	2	ECHA
Boiling Point	117.9°C @ 101.3 kPa	2	ECHA
Density	1040 kg/m^3 @ 25°C	2	ECHA
Vapour Pressure	2079 Pa @ 25°C	2	ECHA
Partition Coefficient (log K_{ow})	-0.17 @ 20°C	2	ECHA
Water Solubility	602.9 g/L @ 25°C	2	ECHA
Viscosity	1.056 mPa s @ 25°C	2	ECHA
Dissociation constant (pKa)	4.756 @ 25°C	2	ECHA

Acetic acid readily dissociates in aqueous media to the acetate ($H_3C2O_2^-$) and hydrogen (H^+) ions.

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

The acetate ion of acetic acid is readily biodegradable, is not expected to bioaccumulate, and has a low potential to adsorb to soil.

B. Partitioning

The pKa of acetic acid is 4.76, indicating that this substance will exist partially in anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts (PubChem).

Volatilization of acetic acid from water and moist soil surfaces is not expected to be an important fate process given a Henry's Law constant of 0.21 pascal cubic metre per mole (Pa-m^3/mol) (ECHA). Acetic acid is expected to volatilise from dry soil surfaces based upon its vapour pressure.

Hydrolysis is not expected to be an important environmental fate process since this substance lacks functional groups that hydrolyse under environmental conditions(PubChem).

C. Biodegradation

Acetic acid was readily biodegradable in a non-acclimated freshwater study. Degradation was 96% after 20 days (Price et al., 1974; ECHA) [Kl. score = 2]. Acetic acid is also readily biodegradable under anaerobic conditions (Kameya et al., 1995) [Kl. score = 2].

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017a).

D. Environmental Distribution

No experimental data are available for acetic acid. Using KOCWIN in EPISuite™ (USEPA, 2017), the estimated K_{oc} values from log K_{ow} and the molecular connectivity index (MCI) are 1.153 and 1.0 L/kg, respectively. Based on these values, acetic acid has a low potential for adsorption to soil and sediment and is expected to have very high mobility in soil.

Acetic acid is highly soluble in water and dissociates completely in aqueous solution to acetate and its hydrogen ion. However, the chemistry of the receiving water compartment, such as its pH and the presence of metal ions, may affect the speciation and partitioning of this substance and its buffering capacity (DoEE, 2017b).

E. Bioaccumulation

There are no bioaccumulation studies on acetic acid. Bioaccumulation of acetic acid is not expected to occur because acetic acid dissociates completely in aqueous solution to acetate and its hydrogen ion.

Both ions are ubiquitous in the environment. Acetate is naturally found in eukaryotic and prokaryotic cells and is involved in their biochemical pathways.

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Acetic acid is a corrosive liquid. Depending on the concentration, aqueous solutions of acetic acid are either corrosive, irritating, or non-irritating to the skin, eyes, and gastrointestinal tract. Vapours from aqueous solutions of acetic acid can cause respiratory irritation. There are no adequate repeated dose toxicity studies on acetic acid. Acetic acid is not genotoxic. Positive findings have been reported in some in vitro genotoxicity studies and are considered to be the result of the pH change in the test system. There are no carcinogenicity studies by the oral or inhalation route. It is not carcinogenic by the dermal route. Animal studies have shown no developmental toxicity from ingestion of acetic acid.

B. Acute Toxicity

Oral

The oral LD_{50} of the sodium salt of acetic acid in rats is 3,310 milligrams per kilogram (mg/kg) (Woodard et al., 1941; ECHA) [Kl. score =2]. The oral LD_{50} of the acetic acid in unfasted rats is 3,530 mg/kg (ECHA) [Kl. score =4]. The oral LD_{50} of the sodium salt of acetic acid in mice is 4,960 mg/kg (Smyth et al., 1951; ECHA) [Kl. score =2].

Inhalation

The 4-hour inhalation LC_{50} in rats for acetic acid vapor is 11.4 milligrams per litre (mg/L). There were clinical signs that were indicative of corrosion (ECHA) [Kl. score = 2].

C. Irritation

Application of a 3.3% or a 10% aqueous solution of acetic acid to the skin of rabbits for 4 hours was slightly irritating. The Primary Dermal Irritation Index scores were 0.5 and 1.1, respectively (Nixon et al., 1990; ECHA) [Kl. score = 2]. Application of a 10% solution of acetic acid to the skin of rabbits for 4 hours under semi-occlusive conditions was slightly irritating (ECHA) [Kl. score = 2].

Instillation of 0.1 mL of a 10% solution of acetic acid to the eyes of rabbits was considered irritating. The mean of the 24-, 48-, and 72-hours scores were: 2.67 for erythema; 1.67 for chemosis; 1.72 for corneal opacity; and a mean of 87% corneal swelling (Jacobs and Martens, 1989; ECHA) [Kl. score = 2]

D. Sensitisation

No studies are available.

E. Repeated Dose Toxicity

Oral

No adequate studies for human health risk assessment are available.

Inhalation

No studies are available.

Dermal

No adequate studies are available.

F. Genotoxicity

In Vitro Studies

The *In Vitro* genotoxicity studies on acetic acid are presented below in Table 2.

Table 2: *In Vitro* Genotoxicity Studies on Acetic Acid

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (*S. typhimurium* strains)	NC	-	2	Ishidate et al. (1984); ECHA
Bacterial reverse mutation (*S. typhimurium* strains)	-	-	2	Zeiger et al. (1992); ECHA
Chromosomal aberrations (CHO cells)	-**	-**	2	Morita et al. (1990); ECHA
Mammalian cell gene mutation (mouse lymphoma L5178Y cells)	-***	-***	2	Seifried et al. (2006); ECHA

*+, positive; -, negative; NC, not conducted.
**A dose-dependent increase in chromosomal aberrations was observed with 10 mM acetic acid (-S9) and 8 mM acetic acid (+S9). These concentrations were close to the cytotoxic limit at which the cells could no longer be evaluated. These effects were abolished by neutralizing the test medium or increasing the buffer capacity. These results suggest that the positive findings are due to the acidic pH of the incubation medium rather than a consequence of an intrinsic clastogenic potential of acetic acid.
***Acetic anhydride (hydrolyses to acetic acid in aqueous media).

In Vivo Studies

No studies are available on acetic acid.

A bone marrow micronucleus study has been conducted on acetic anhydride, which hydrolyses to acetic acid. Male and female SD rats were exposed by inhalation to 0, 1, 5, or 20 parts per million (ppm) acetic anhydride, 6 hours/day, 5 days/week for 13 weeks. The incidence of micronucleated immature erythrocytes was not increased at any exposure concentration (ECHA). [Kl. score = 1]

G. Carcinogenicity

No oral or inhalation studies are available.

No deaths nor skin tumours were seen when acetic acid was applied dermally once a week to CD-1 mice for 32 weeks (Slaga et al., 1975; ECHA) [Kl. score = 4].

H. Reproductive Toxicity

No studies are available.

I. Developmental Toxicity

Pregnant female Wistar rats were dosed with 0 or various concentrations up to 1,600 mg/kg apple cider vinegar (5% acetic acid) by oral gavage on gestational days 6 to 15. There were no maternal or developmental toxicity effects noted at any dose level. The no observed adverse effect level (NOAEL) for maternal and developmental toxicity is 1,600 mg/kg-day (ECHA). [Kl. score = 2]

Pregnant female CD-1 mice were dosed with 0, 16, 74.3, 345, or 1,600 mg/kg apple cider vinegar (5% acetic acid) by oral gavage on gestational days 6 to 15. There were no treatment-related effects on maternal or foetal survival, or on soft or skeletal tissues. There was no effect on the foetal development in the presence of slight maternal toxicity (reduced body weight gain) at 345 mg/kg. At 1,600 mg/kg, there was an increase in the number of litters containing a dead foetus and some reductions in ossification. The NOAELs for maternal and developmental toxicity are 74.3 and 345 mg/kg-day, respectively (ECHA). [Kl. score = 2]

Pregnant female Dutch-belted rabbits were dosed with 0, 16, 74.3, 345, or 1,600 mg/kg apple cider vinegar (5% acetic acid) by oral gavage on gestational days 6 to 18. There were no treatment-related effects on maternal or foetal survival, or on soft or skeletal tissues. There was a reduction in the pregnancy rate in the high-dose group; and a dose-dependent decrease in maternal body weights at >74.3 mg/kg. Some deaths or abortions occurred in all treated groups and some litter losses were reported at >345 mg/kg. Maternal effects were much more noticeable than the effects on foetal development. These findings have been considered a consequence of the bactericidal properties of orally administered acetic acid within the gastrointestinal tract of female rabbits, and not a direct effect on embryonic implantation and development of acetic acid (EU, 2008). It is likely that this accounts for the apparent increased sensitivity of this species to oral administration of acetic acid. The NOAEL for developmental toxicity is 1,600 mg/kg-day; a NOAEL for maternal toxicity was not identified (ECHA). [Kl. score = 2]

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for acetic acid follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

There are no repeated dose toxicity studies that were considered adequate for human health risk assessment.

The Joint FAO/WHO Expert Committee on Food Additives (JECFA) has maintained a group acceptable daily intake (ADI) of "not limited" for acetic acid and its potassium and sodium salts (JECFA).

While concentration of acetic acid will affect pH, and extreme pH values (<4 and >11) may adversely affect health, there are insufficient data to set a health guideline value (ADWG, 2011)

B. Cancer

There are no carcinogenicity studies by the oral or inhalation route. A dermal carcinogenicity study in mice showed no carcinogenic activity when acetic acid was applied to the skin for 32 weeks. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Acetic acid is a flammable liquid.

Acetic acid does not exhibit the following physico-chemical properties:

- Explosivity
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Acetic acid is of moderate acute toxicity concern to aquatic organisms, in part because of the effect of pH changes from the dissociated hydrogen ion. The acetate ion is of low acute toxicity concern to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

Table 3 presents the results of acute aquatic toxicity studies on acetic acid and potassium acetate.

Table 3: Acute Aquatic Toxicity Studies on Acetic Acid and Potassium Acetate

Test Substance	Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Potassium acetate	*Oncorhynchus mykiss*	96-hour LC_{50}	>300.82*	2	ECHA
Potassium acetate	*Danio rerio*	96-hour LC_{50}	>300.82*	2	ECHA

Test Substance	Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Acetic acid	*Oncorhynchus mykiss*	96-hour LC_{50}	64.8 (measured)	4	ECHA
Acetic acid	*Oncorhynchus mykiss*	96-hour LC_{50}	31.3 – 67.6	4	ECHA
Potassium acetate	*Daphnia magna*	48-hour EC_{50}	>300.82*	2	ECHA
Acetic acid	*Daphnia magna*	48-hour EC_{50}	79.5 (measured)	4	ECHA
Acetic acid	*Daphnia magna*	48-hour EC_{50}	18.9 (measured)	4	ECHA
Acetic acid	*Desmodesmus subspicatus*	72-hour EC_{50}	486.5	4	ECHA

*As the acetate ion.

Chronic Studies

In a 21-day fish (*Oncorhynchus mykiss*) chronic study, the measured no observed effect concentration (NOEC) values for 60% and 100% acetic acid were 57.2 and 34.3 mg/L, respectively (ECHA). [Kl. score = 4]

In a 21-day *Daphnia* reproduction study, the measured NOEC for 60% and 100% acetic acid were 80 and 31.4 mg/L, respectively (ECHA). [Kl. score = 4]

In a 21-day *Daphnia* reproduction study, the measured NOEC for 100% acetic acid was 22.7 mg/L (ECHA). [Kl. score = 4]

C. Terrestrial Toxicity

No data are available.

D. Calculation of PNEC

Despite the low Klimisch scores for aquatic toxicity testing (K=4), the PNEC calculations for acetic acid follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. For the acute toxicity studies, data are available on both acetic acid and potassium acetate; both substances dissociate completely in aqueous media to the acetate anion and the corresponding cations (H+ and K+). The toxicity of these substances is expected to be driven by the acetate ion, with the cations having a minor role. The toxicity data on potassium acetate are preferred because of the absence of a potential pH change from the dissociated H+ ion of acetic acid. For the chronic toxicity studies, only acetic acid has been tested for two trophic levels: fish and invertebrates. These studies will not be used to derive the PNEC value; however, an assessment factor of 100 will be applied to the lowest acute $E(L)C_{50}$ values for the acetate ion.

From the potassium acetate studies, acute $E(L)C_{50}$ values (adjusted for acetic acid) are available for fish (300.82 mg/L) and Daphnia (300.82 mg/L). By applying an assessment factor of 100 to the $E(L)C_{50}$ value of 300.82 mg/L from either fish or Daphnia, the $PNEC_{water}$ for acetic acid is 3.0 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 1.9 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1{,}000 \times PNEC_{water}$

$= (0.82/1{,}280) \times 1{,}000 \times 3.0$

$= 1.9$ mg/kg

Where:

$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (cubic metre per cubic metre [m^3/m^3])

BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]

$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1{,}000 \times BD_{solid}]$

$= 0.8 + [(0.2 \times 0.04/1{,}000 \times 2{,}400]$

$= 0.82$ kg/m^3

Where:

Kp_{sed} = solid-water partition coefficient (L/kg).

BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]

$Kp_{sed} = K_{oc} \times f_{oc}$

$= 1.0 \times 0.04$

$= 0.04$ L/kg

Where:

K_{oc} = organic carbon normalized distribution coefficient (L/kg). The K_{oc} for acetic acid calculated from EPISUITE™ using the MCI is 1.0 L/kg .

F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There is no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.04 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$

$= (0.02/1500) \times 1000 \times 3.0$

$= 0.04$ mg/kg

Where:

Kp_{soil} = soil-water partition coefficient (m^3/m^3)

BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]

$Kp_{soil} = K_{oc} \times f_{oc}$

$= 1.0 \times 0.02$

= 0.02 m^3/m^3

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for acetic acid calculated from EPISUITE™ using the MCI is 1.0 L/kg .
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Acetic acid is readily biodegradable; thus, it does not meet the screening criteria for persistence.

Bioaccumulation of acetic acid is not expected to occur because acetic acid dissociates completely in aqueous media to acetate and its hydrogen ion. Both ions are ubiquitous in the environment. Acetate is naturally found in eukaryotic and prokaryotic cells and is involved in their biochemical pathways. The log K_{ow} for acetic acid is -0.17. Thus, acetic acid does not meet the screening criteria for bioaccumulation.

The NOECs from the chronic aquatic toxicity studies on acetic acid are >0.1 mg/L. The EC_{50} values for potassium acetate are > 1 mg/L. Thus, acetic acid does not meet the criteria for toxicity.

The overall conclusion is that acetic acid is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Flammable Liquid Category 3

Skin Corrosion Category 1A

EU:

≥90%: Skin Corrosion 1A

≥25% to <90%: Skin Corrosion 1B

≥10% to <25%: Skin irritant Category 2; Eye irritant Category 2

In addition to the hazard statements corresponding the GHS classifications (if Skin Corrosion 1A or 1B is included), the following non-GHS hazard statement is to be added to the SDS: AUH071: Corrosive to the Respiratory Tract.

B. Labelling

Danger

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. Get medical attention immediately, preferably a physician for an ophthalmologic examination.

Skin Contact

For minor skin contact, avoid spreading material on unaffected skin. Remove and isolate contaminated clothing. Wash the contaminated area of body with soap and fresh water. Get medical attention immediately.

Inhalation

Move person to fresh air. Administer oxygen if breathing is difficult. Do not use mouth-to-mouth method if victim inhaled the substance; give artificial respiration with the air of a pocket mask equipped with a one-way valve or other proper respiratory medical device. Give artificial respiration if victim is not breathing. Get medical attention immediately.

Ingestion

Rinse mouth and lips with plenty of water if person is conscious. Do not induce vomiting. Do not use mouth-to-mouth method if victim had ingested the substance. Obtain medical attention immediately if ingested.

Notes to Physician

Treat as a corrosive due to pH of the material. All treatments should be based on observed signs and symptoms of distress in the patient.

B. Fire Fighting Information

Extinguishing Media

Use water spray or fog, foam, dry chemical or carbon dioxide. Do not use straight streams of water.

Specific Exposure Hazards

Flammable liquid and vapor. Vapours are flammable and heavier than air. Vapours may travel across the ground and reach remote ignition sources causing a flashback fire danger. Emits toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon dioxide, carbon monoxide.

Special Protective Equipment for Firefighters

Structural firefighter's protective clothing provides limited protection in fire situations only; it is not effective in spill situations where direct contact with the substance is possible. Wear chemical protective clothing that is specifically recommended by the manufacturer. It may provide little or no thermal protection. Wear positive pressure self-contained breathing apparatus (SCBA). Move containers from fire area if you can do it without risk.

C. Accidental Release Measures

Personal Precautions

Isolate area. Keep unnecessary and unprotected personnel from entering the area. Use personal protective clothing. Ensure adequate ventilation. Wear respiratory protection if ventilation is inadequate. Do not breath mist, vapours, or spray. Avoid contact with skin, eye, and clothing. Eliminate all sources of ignition.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilled

Eliminate all sources of ignition. All equipment used when handling the material must be grounded. A vapor suppressing foam may be used to reduce vapours. Use clean non-sparking tools to collect absorbed material. Pick up with suitable absorbent material and transfer to a container for chemical waste. For large amounts, dike spillage and pump off product into container for chemical waste. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Prevent exposure to ignition sources (i.e., use non-sparking tools and explosion-proof equipment). Avoid contact with eyes, skin, and clothing. Avoid breathing vapor. Wash thoroughly after handling. Keep

container closed. Use with adequate ventilation. Use proper bonding and/or ground procedures. However, bonding and grounds may not eliminate the hazard from static accumulation. Peroxides may form upon prolonged storage. Exposure to light, heat or air significantly increases peroxide formation. If evaporated to a residue, the mixture of peroxides residue and material vapor may explode when exposed to heat or shock.

Storage

Keep container tightly closed. Store in a cool, well-ventilated area away from heat and light. Storage containers should be grounded and bonded. Fixed storage containers, transfer containers and associated equipment should be grounded and bonded to prevent accumulation of static charge.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace exposure standard for acetic acid in Australia is 10 ppm (25 mg/m^3) as a 8-hr time-weighted average (TWA) and 15 ppm (37 mg/m^3) as a 15-min short-term exposure limit (STEL).

Engineering Controls

Good general ventilation should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation, or other engineering controls to maintain airborne levels below recommended exposure limits. If exposure limits have not been established, maintain airborne levels to an acceptable level.

Personal Protection Equipment

Respiratory Protection:

If workers are exposed to concentrations above the exposure limit, they must use appropriate, certified respirators. If there are no applicable exposure limit requirements or guidelines, use an approved respirator. Selection of air-purifying or positive pressure supplied-air will depend on the specific operation and the potential airborne concentration of the product. For emergency conditions, use an approved positive-pressure self-contained breathing apparatus.

Hand Protection:

Use gloves chemically resistant to this material. Consult the safety data sheet (SDS) for appropriate glove barrier materials.

Skin Protection:

Use protective clothing chemically resistant to this material. Selection of specific items such as face shield, boots, apron, or full body suit will depend on the task.

Eye protection:

Use chemical goggles.

Other Precautions:

Wash hands, forearms, and face thoroughly after handling chemical products, before eating, smoking, and using the lavatory and at the end of the working period. Appropriate techniques should be used to remove potentially contaminated clothing. Wash contaminated clothing before reusing. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

For glacial acetic acid or >80% acetic acid solutions:
UN 2789 (ACETIC ACID, GLACIAL or ACETIC ACID SOLUTION)
Class: 8
Packing Group: II

For ≥50% to 80% acetic acid solutions:
UN 2790 (ACETIC ACID SOLUTION)
Class: 8
Packing Group: II

For >10% to <50% acetic acid solutions:
UN 2790 (ACETIC ACID SOLUTION)
Class: 8
Packing Group: III

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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ALCOHOLS, C12-16, ETHOXYLATED

This dossier on alcohols, C12-16, ethoxylated presents the most critical studies pertinent to the risk assessment of alcohols, C12-16, ethoxylated in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The information presented in this dossier was obtained primarily from the Human & Environmental Risk Assessment on Ingredients of European Household Cleaning Products: Alcohol Ethoxylates (HERA, 2009), and from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch *et al.*, 1997).

For the purpose of this dossier, alcohols, C12-15, ethoxylated (CAS RN 68131-39-5) has been reviewed as a surrogate chemical for ethoxylated C12-C16 alcohol (CAS No. 68551-12-2), where appropriate.

I. SUBSTANCE IDENTIFICATION

Chemical Name: Alcohols, C12-16, ethoxylated

CAS RN: 68551-12-2

Molecular formula: $H–(CH_2)_{12–16}–(OCH_2CH_2)_n–OH$ (where n is the average number of EO units)

Molecular weight: Not available (UVCB substance)

Synonyms: Alcohols, C12-16, ethoxylated, Ethoxylated C12-16 alcohols; polyethylene glycol, dodecyl, tetradecyl, hexadecyl ether

SMILES: Not available (UVCB substance)

Alcohol ethoxylates (AE) are a class of non-ionic surfactant polymers that have the basic structure $C_{x-y}AE_n$. The subscript (x-y) following the 'C' indicates the range of carbon chain units. The hydrocarbon chain can be either linear or branched. AEs also contain an ethylene oxide (E) chain attached to the alcohol. The degree of ethylene oxide (EO) polymerization is indicated by the subscript (n) which indicates the average number of ethylene oxide units. Ethoxylated C12-C16 alcohol (CAS No. 68551-12-2) has an average number of 1 to 6 moles of ethylene oxide units.

II. PHYSICO-CHEMICAL PROPERTIES

No information is available on alcohols, C12-16, ethoxylated. Therefore, data were read across from a similar substance, alcohols, C12-15, ethoxylated (CAS RN 68131-39-5), as shown below.

Table 1: Overview of the Physico-chemical Properties of Alcohols, C12-15, Ethoxylated (1 to 2.5 moles ethoxylated)

Property	Value	Klimisch score	Reference
Physical state at 20°C and 101.3 kPa	Clear liquid with a rancid odor*	2	ECHA
Melting Point	7.22°C (pressure not provided)	2	ECHA

Property	Value	Klimisch score	Reference
Boiling Point	271.11-516.11°C (pressure not provided)	2	ECHA
Density	ca. 930 kg/m^3 @ 20°C	2	ECHA
Vapor Pressure	<1 Pa@ 25°C	2	ECHA
Partition coefficient (log K_{ow})	5.06** @ 25°C	2	ECHA
Water Solubility	0.021 g/L @ 25°C	2	ECHA
Flash Point	165.56°C	2	ECHA
Auto flammability	235°C	2	ECHA
Viscosity	28.1 mPa s (dynamic) @ 20°C	2	ECHA

*Based on alcohols, C12-15, ethoxylated (1 to 2.5 EO) [CAS No. 68131-39-5]
**Weight-averaged log K_{oc} of whole substance based on normalized composition

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Alcohols, C12-16, ethoxylated is readily biodegradable. It has a low potential for bioaccumulation and a moderate potential for absorption to soil and sediment.

B. Biodegradation

There are no studies available for alcohol, C12-16, ethoxylated.

AE homologues with linear hydrocarbon chain lengths from C8 to C15 and mean values ranging from 3-20 EO units are readily biodegradable (HERA, 2009). If a chemical is found to be readily biodegradable, it is categorized as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

Alcohols, C12-C14, ethoxylated (7-8) degraded to 100% in 28 days in a die away screening test (HERA, 2009) [Kl. Score = 2].

Alcohols, C12-15, ethoxylated is readily biodegradable. In an OECD 301B test, degradation of 10 mg/L of alcohols, C12-15. ethoxylated was 72% after 28 days but it failed the 10-day window (ECHA) [Kl. score = 1].

In an OECD 301B test, degradation of 20 mg/L of alcohols, C12-15. ethoxylated was 61% after 28 days but it failed the 10-day window (ECHA) [Kl. score = 1].

A 240 mg/L concentration of alcohol, C12-15, ethoxylated (7 EO) degraded 80- 88% in 28 days when tested using a shake-flask CO_2-evolution test method (ECHA) [Kl. score = 2].

C. Environmental Distribution

There are no experimental data are available for alcohols, C12-16, ethoxylated. Using KOCWIN in EPISuite™ (EPA, 2018), the estimated K_{oc} values for surrogates of alcohols, C12-16, ethoxylated are:

K_{oc} for C12-C16 linear alcohol, ethoxylated (2 EO): 3,920 L/kg (molecular connectivity index, MCI) and 13,530 L/kg (K_{ow}).

The adsorption potential of the alcohols, C12-15 was determined using QSAR-calculations (EPI Suite v4.11) using the KOCWIN v2.00 model based on the Molecular Connectivity Index (MCI) and the log Kow method. Smiles-codes of the unethoxylated alcohols as well as smiles-codes of the alcohol ethoxylates with an ethoxylation of 1 EO, 2 EO and 3 EO of the homologues with chain lengths of C12 and C15 were chosen as representatives of the mixture. The representative structures fall within the applicability domain of both models and thus the calculations are considered valid. The results are given as a range which represents the variation of carbon chain length and the degree of ethoxylation according to substance specifications. The calculated log Koc values range from 2.301 to 3.352 (MCI method) and 2.382 to 3.926 (log Kow method) (ECHA) [KI. score =2].

Based on these K_{oc} values, if released to soil, the alcohols, C12-C16 ethoxylated is expected to adsorb strongly to soil and it is expected to have a low potential for mobility.

D. Bioaccumulation

The potential for bioaccumulation of AEs is considered low due to the biotransformation and excretion of the substance. The various studies present considerable evidence that AEs are rapidly eliminated and metabolised (ECHA).

The BCF values for alcohol ethoxylates in fathead minnows have been reported to range from <5 to 387.5 L/kg (Toll et al., 2000; as cited in ECHA) [KI.score=2]. The uptake rates varied from 330 to 1660 (L x kg/d) and elimination rates varied from 3.3 to 59 per day (Toll et al., 2000; as cited in ECHA) [KI. score=2]. The high concentration in fish is thought to be prevented by an efficient biotransformation of the alcohol ethoxylates, leading to a high elimination rate.

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

The acute toxicity of alcohols, C12-16, ethoxylated is low by the oral and dermal routes. Skin irritation studies in rabbits on alcohols, C12-16, ethoxylated have shown mixed results, but human patch studies on these alcohol ethoxylates do not support a skin irritant classification. Alcohols, C12-16, ethoxylated is expected to be irritating to the eyes of rabbits. Alcohols, C12-16, ethoxylated is not a skin sensitizer. Repeated dose toxicity studies on alcohol ethoxylates similar to alcohols, C12-16, ethoxylated in rats do not indicate any target organ effects. These alcohol ethoxylates are not genotoxic, carcinogenic, and they have a low potential for reproductive and developmental toxicity.

B. Acute Toxicity

There are no acute toxicity studies are available on alcohols, C12-16, ethoxylated.

<u>Oral</u>

The oral LD_{50} in rats for $C_{12\text{-}15}AE_3$ is >5,000 mg/kg (ECHA) [Kl. score = 2]. The oral LD_{50} in rats for $C_{12\text{-}15}AE_7$ is 1,700 mg/kg (HERA, 2009) [Kl. score = 2]. The oral LD_{50} value in rats for $C_{12\text{-}13}AE_{6.5}$ is 2,100 mg/kg (HERA, 2009) [Kl. score = 2]. The oral LD_{50} value in rats for $C_{12\text{-}15}AE_{11}$ is >2,000 mg/kg in males and between 1,000 and 2,000 mg/kg in females (HERA, 2009) [Kl. score = 2]. The oral LD_{50} values in rats for $C_{14\text{-}15}AE_{13}$ in two separate studies are 1,100 and 1,000 mg/kg (HERA, 2009) [Kl. score = 2]. The

relative number of EO units, but not the carbon chain length, appears to influence acute oral toxicity (HERA, 2009).

The acute oral LD_{50} for alcohols, C12-C15, ethoxylated in male and female Wistar rats is >5000-<10,000 mg/kg bw (ECHA) [KI. score = 2].

Inhalation

The 4-hour LC_{50} for alcohols, C12-C15, ethoxylated in male and female Sprague-Dawley rats is > 1,600 mg/m^3 (>1.6 mg/L) (ECHA) [KI. score =2].

Dermal

Acute dermal LD_{50} values of >2,000 mg/kg were determined for $C_{12\text{-}14}AE_3$ and $C_{12\text{-}14}AE_6$ in two separate studies (HERA, 2009) [Kl. score = 2]. The acute dermal LD_{50} of $C_{12\text{-}15}AE_7$ is >2,000 mg/kg (HERA, 2009) [Kl. score = 2].

The acute dermal LD_{50} for alcohols, C12-C15, ethoxylated in male and female Wistar rats >2000 mg/kg bw (ECHA) [KI. score =2].

C. Irritation

Skin

Application of 0.5 mL isotridecanol, ethoxylated (3 EO) to the skin of rabbits for 4 hours under occlusive conditions was considered irritating (ECHA) [Kl. score = 2].

Application of 0.5 mL isotridecanol, ethoxylated (3 EO) to the skin of rabbits for 4 hours under semi-occlusive conditions was not considered irritating (ECHA) [Kl. score = 2].

In a 24-hour human patch test, there was some short-lived redness in some individuals from the application of $C_{12\text{-}14}AE_3$, but there was no scaling or edema in any subjects (HERA, 2009) [Kl. score = 2].

In a standard 4-hour human patch test, the irritation potential of $C_{12\text{-}15}AE_5$ and $C_{12\text{-}15}AE_5$ were compared to 20% sodium dodecyl sulfate (which is classified a skin irritant under GHS). The results showed that neither alcohol ethoxylate should be classified as a skin irritant (Basketter et al., 2004) [Kl. score = 2]. Nonetheless, the substance is classified by ECHA as an irritant (see Section IX).

Eye

Most alcohol ethoxylates tested as the undiluted neat test material are moderately to severely irritating to the eyes of rabbits, with an eye irritation index (EII) ranging from >25 to 50 (HERA, 2009). The alcohol ethoxylates $C_{12\text{-}14}AE_3$, $C_{12\text{-}14}AE_6$, $C_{13}AE_6$, and $C_{12\text{-}14}AE_{10}$ were found to be moderately to severely irritating to the eyes of rabbits (HERA, 2009). In another study, $C_{12\text{-}15}AE_{11}$ was considered moderately to severely irritating to the eyes of rabbits (HERA, 2009).

Some alcohol ethoxylates were reported to be practically or minimally irritating to the eyes of rabbits with EII scores of 0.5 to 15. These alcohol ethoxylates include: $C_{12\text{-}15}AE_3$, $C_{14\text{-}15}AE_7$, $C_{12\text{-}14}AE_{15}$, $C_{14\text{-}15}AE_{18}$, and $C_{13}AE_{20}$ (HERA, 2009).

D. Sensitisation

There are no sensitisation studies are available on alcohols, C12-16, ethoxylated.

In a guinea pig maximization test, $C_{12\text{-}13}AE_{<2.5}$ (CAS No. 66455-14-9) was not considered a skin sensitizer (ECHA) [Kl. score = 2].

In guinea pig maximization tests, $C_{12\text{-}15}AE_3$, $C_{12\text{-}15}AE_7$, and $C_{14\text{-}15}AE_7$ were not considered skin sensitizers (HERA, 2009) [Kl. scores = 2].

E. Repeated Dose Toxicity

Oral

There are no repeated dose toxicity studies are available on alcohols, C12-16, ethoxylated. Data for similar ethoxylates are presented below.

Rats were given in their diet 0%, 0.0313%, 0.0625%, 0.125, 0.25, 0.5 or 1.0% $C_{12\text{-}15}AE_7$ for 90 days. The animals in the ≥0.25% groups showed significantly reduced body weight gain, which was associated with marked decreases in food and water consumption. Relative liver weights were significantly increased in the ≥0.5% male rats and ≥0.25% females. Histopathologic examination showed hepatocytic enlargement in the ≥0.125% groups, suggesting increased liver metabolism based on increased alkaline phosphatase activity at the higher dose levels. The NOAEL was established at 0.0625% in the diet or 102 mg/kg-day (HERA, 2009) [Kl. score = 2].

Rats were fed $C_{12\text{-}14}AE_7$ in the diet at concentrations of 0%, 0.0313%, 0.0625%, 0.125%, 0.25%, 0.5% and 1.0% for 90 days. The animals in the ≥0.25% groups showed significantly reduced body weight gain, which was associated with marked decreases in food and water consumption. Relative liver weights were significantly increased in the ≥0.5% male rats and ≥0.25% females. Histopathologic examination showed hepatocytic enlargement in the ≥0.125% groups, suggesting increased liver metabolism based on increased alkaline phosphatase activity at the higher dose levels. The NOAEL was established at 0.0625% in the diet or 110 mg/kg-day (HERA, 2009) [Kl. score = 2].

Male and female Wistar rats given in their diet 0, 300, 1,000, 3,000, and 10,000 ppm $C_{14\text{-}15}AE_7$ for 90 days. There were no deaths during the study. Mean body weights and feed were lower in 10,000 ppm males and the 3,000 ppm females. Feed consumption was lower in the 10,000 ppm animals and the 3,000 ppm females. Relative liver weights were increased in the ≥3,000 ppm animals, and relative spleen weights were increased in the 10,000 ppm males. Clinical chemistry changes were noted in the 10,000-ppm group and consisted of significantly higher urea, chloride and potassium levels in males, significantly higher urea, chloride and cholesterol in females. Increased total leucocytes and lymphocytes were seen in the 10,000 ppm animals and in the 3,000 ppm males. The 10,000 ppm females showed lower numbers of neutrophils; mean cell volume and mean cell hemoglobin were identified in one or both sexes fed in the ≥3,000 ppm dose groups. In the 1,000 ppm females, there were minor, but statistically significant changes in the liver and kidney weights and plasma urea concentration; these effects were considered to be of no toxicological significance. Histopathologic examination showed no treatment-related effects at any dose level. The NOAEL for this study is 1,000 ppm in the diet, which corresponded to 50 mg/kg-day (HERA, 2009) [Kl. score = 2].

Rats were given in their diet 0, 0.1, 0.5, or 1% $C_{14\text{-}15}AE_7$ for 90 days. Body weights, food intake, organ weights, and hematology and clinical chemistry parameters were similar across groups. The NOAEL

for this study is 1% in the diet, which corresponded to 700 and 785 mg/kg-day for males and females, respectively (HERA, 2009) [Kl. score = 2].

Rats were given in their diet 0, 0.1, 0.5 or 1% $C_{12\text{-}13}AE_{6.5}$ or $C_{14\text{-}15}AE_7$ for two years. Body weight gain was reduced in the 1% males and ≥0.5% females, which was likely due to the reduced food consumption in these animals. At study termination, organ to body weight ratios were increased in the ≥0.5% females (liver, kidney, and brain), 1% females (heart), and 1% males (liver). A dose-related focal myocarditis was observed in males. While focal myocarditis is commonly observed in non-treated aging rats, the incidence in the treated animals were higher than in the controls. The NOAEL was established at 0.1% or 50 mg/kg-day (HERA, 2009) [Kl. score = 2].

Male and female CR rats were given in their diet $C_{14\text{-}15}AE_7$ at 0.1, 0.5 and 1% for two years. A treatment-related body weight depression was observed in females at the two highest treatment levels and in males at the 1% dose level, probably due to the poor palatability of the diet. Relative liver, kidney, heart, and thyroid/parathyroid gland weights were increased in the 1% dietary group at study termination. Histopathological examination showed a dose-related increase in the incidence of focal myocarditis at the 12-month time point, but not at the end of the study at two years. The NOAEL for this study was considered to be 0.5% in the diet, which corresponded to 162 and 190 mg/kg-day for males and females, respectively (HERA, 2009) [Kl. score = 2].

An OECD guideline 422 (Combined repeated dose toxicity study with the reproduction/developmental toxicity screening test) was conducted in male and female Wistar rats exposed to a daily (7 days a week) dose of 100, 300, and 1,000 mg/kg bw/day of alcohols, C12-C15, ethoxylated by oral gavage for 29 (males) -64 days (females). Slightly increased plasma albumin concentrations were observed in males at the 300 and 1000 mg/kg bw/day dose levels, increased plasma urea concentrations were observed in males at the 1000 mg/kg bw/day dose level, decreased plasma cholesterol concentrations in males at the 300 and 1000 mg/kg bw/day levels and increased bile acid concentrations in females at the 1000 mg/kg bw/day dose level were considered as non-adverse since these changes were not associated with any adverse pathological alterations. Non-adverse test item-related morphologic alterations were present in males and females at the 1000 mg/kg bw/day dose level in the liver (macroscopically enlarged liver, centrilobular hypertrophy, increased weights starting at 100 mg/kg bw/day in males and 300 mg/kg bw/day in females), forestomach (squamous cell hyperplasia) and jejunum (vacuolation in the lamina propria), in males starting at 100 mg/kg bw/day in the thyroid gland (follicular cell hypertrophy and increased weights at 1000 mg/kg bw/day) and in females at 1000 mg/kg/day in the adrenal gland (macroscopically enlarged adrenal gland, diffuse cortical hypertrophy, and increased weights at 1000 mg/kg bw/day). There were no toxicologically significant changes were noted in any of the remaining parameters investigated in this study, i.e., mortality, clinical appearance, functional observations (motor activity, grip strength, hearing ability, pupillary reflex and static righting reflex), body weight, food consumption, hematology and clotting parameters, male T4 thyroid hormone. A systemic NOAEL of ≥1000 mg/kg bw/day and a reproductive toxicity NOAEL of ≥1000 mg/kg bw/day was established for this study (ECHA) [KI. score = 1].

Inhalation

There are no studies are available.

Dermal

There are no adequate studies are available.

F. Genotoxicity

In vitro Studies

The genotoxicity studies conducted on alcohol ethoxylates are reviewed in HERA (2009). The results of few of the *in vitro* studies on similar alcohol ethoxylates to alcohols, C12-16, ethoxylated are presented below in Table 2.

Table 2: *In Vitro* Genotoxicity Studies on Selected Alcohol Ethoxylates

Test Substance	Test System	Results*		Klimisch Score	References
		-S9	+S9		
$C_{14\text{-}15}AE_7$	Bacterial reverse mutation (*S. typhimurium* strains)	-	-	2	HERA, 2009
$C_{14\text{-}15}AE_7$	Bacterial reverse mutation (*S. typhimurium* strains)	-	-	2	HERA, 2009
$C_{14}AE_{12}$	Chromosomal aberrations (CHO cells)	-	-	2	HERA, 2009

*+, positive; -, negative

In Vivo Studies

In two separate studies, CD-1 mice were given an intraperitoneal dose of 0, 50, or 100 mg/kg $C_{12\text{-}15}AE_3$ or $C_{12\text{-}14}AE_9$. There were no increases in the frequency of micronuclei in the bone marrow cells (Talmage, 1994) [Kl. score = 2].

Male and female Tunstall rats were given a single oral gavage dose of 0, 250, 500, or 1,000 mg/kg $C_{14\text{-}15}AE_7$. There were no increases in chromosomal aberrations in the bone marrow cells (HERA, 2009 [Kl. score = 2].

G. Carcinogenicity

There are no studies are available on alcohols, C12-16, ethoxylated. Therefore, data from similar substances are presented below.

Male and female Sprague-Dawley rats were given in their diet $C_{12\text{-}13}AE_{6.5}$ in the diet at doses up to 1% (500 mg/kg-day). Reduced food consumption was noted at the higher dose levels (*i.e.*, 0.5 and 1% for females and 1% for males), resulting in a lower body weight gain compared to the control group. No treatment-related histopathology was found and no increase in tumour incidence was observed (HERA, 2009) [Kl. score = 2].

Male and female Charles River rats were given in their diet 0, 0.1, 0.5 or 1% $C_{14\text{-}15}AE_7$ for two years. There were no treatment-related changes in general behaviour and appearance. The survival rate of the test animals was comparable if not better than the controls. Body weights of the 0.5% females and the 1% males and females had significantly lower weight gains than the control. There were no treatment-related effects on organ weights and tumour incidence (HERA, 2009) [Kl. score = 2].

Male and female Sprague-Dawley rats were given in their diet $C_{14\text{-}15}AE_7$ at 0.1, 0.5 and 1% for two years. A treatment-related body weight depression was observed in females at the two highest treatment levels and in males at the 1% dose level, probably due to the poor palatability of the diet. There was no evidence for any carcinogenic activity (HERA, 2009) [Kl. score = 2].

H. Reproductive Toxicity

There are studies are available on alcohols, C12-16, ethoxylated.

CD rats were given in their diet 0, 0.05, 0.1 or 0.5% (approximately 0, 25, 50 or 250 mg/kg-day) $C_{12}AE_6$ in a two-generation reproductive toxicity study. There were no treatment related effects in the parents or pups on general behaviour, appearance, or survival. At 0.5%, there was reduced weight gain in both the parental animals and the pups compared to the controls. Fertility was unaffected by treatment. The NOAEL for reproductive toxicity is 0.5% in the diet, which corresponds to 250 mg/kg-day (HERA, 2009) [Kl. score = 2].

In a two-generation developmental and teratogenicity study, CD rats were given in their diet 0, 0.05, 0.1 or 0.5% $C_{14\text{-}15}AE_7$ (approximately 0, 25, 50 or 250 mg/kg-day). Three of the treated groups were given the test substance continuously throughout the study; in the other three groups the females received the test substance on GD 6-15 and the males were untreated. None of the deaths of parental rats during the study was considered to be compound related. There were no treatment-related changes in behaviour or appearance in the parental rats or pups. Slightly lower body weight gain was noted in the 0.5% continuously treated females. Food consumption was similar for control and treated rats. Fertility, gestation, and viability indices were similar across groups. The average 21-day body weights for the 0.5% continuous treated pups were significantly lower than that of the control. Relative liver weights of the 0.5% continuously treated F_1 parental animals were increased at the 91-day sacrifice; relative liver weights of the 0.5% continuously treated males were also increased at the 60-day and caesarean section sacrifices. There were no treatment-related histopathological lesions in any of the tissues from the F_0 and F_1 generations. The NOAEL for reproductive toxicity is 0.5% in the diet or 250 mg/kg-day (HERA, 2009) [Kl. score = 2].

A sub-acute reproductive and developmental toxicity screening study was completed using male and female Wistar rats exposed to 100, 300, and 1,000 mg/kg bw/day of alcohols, C12-15, ethoxylated via oral gavage for 29 (males)-64 (females) days. All the females had regular cycles of 4 to 5 days. Extended di-oestrous occurred during the mating period in three females of the control group and two females of the mid-dose group (300 mg/kg bw/day) with a regular cycle during premating. One female at 300 mg/kg bw/day had an inconclusive cycle determination during the premating phase. Given their absence of a dose-related incidence, this finding did not indicate a relation with treatment. Length and regularity of the oestrous cycle were considered not to have been affected by treatment with the test item up to 1000 mg/kg bw/day. Mating index was not affected by treatment. The mating indices were 90, 100, 100 and 100% for the control, 100, 300 and 1000 mg/kg bw/day groups, respectively. One female of the control group did not mate. All of the paired females showed evidence of mating within 4 days, except one female at 300 mg/kg bw/day for which mating took 13 days. Hence, precoital time was not affected by treatment with the test item. Number of implantation sites was considered not to be affected by treatment. The mean number of implantation sites were 11.0, 8.9, 12.9 and 12.1 for the control, 100, 300 and 1000 mg/kg bw/day. The relatively low mean number of implantation sites at 100 mg/kg bw/day was attributed to the low number of implantation sites in three females (4, 1 and 2 implantation sites, respectively). In the absence of a dose-related incidence, the relatively low mean number of implantation sites at 100 mg/kg bw/day was considered not to be related to treatment with the test item. One female at 100 mg/kg bw/day and one female at 1000 mg/kg bw/day were not pregnant. In the absence of a dose-related incidence of non-pregnancy, this was considered not to be related to treatment with the test item. The fertility indices were 100, 90, 100 and 90% for the control, 100, 300 and 1000 mg/kg bw/day groups, respectively. It was considered not to be affected by treatment of the animals. Gestation index and duration of gestation were not affected by treatment with the test item up to 1000 mg/kg bw/day. The gestation indices were 100% for all groups. All pregnant females had 21-22

days gestation, except for one female at 100 mg/kg bw/day which only had 19 days of gestation (her litter consisted of 1 pup only). Given the incidental occurrence and lack of a dose-related trend, no toxicological relevance was attributed to this early delivery. No signs of difficult or prolonged parturition and no deficiencies in maternal care were noted among the pregnant females. A NOAEL for systemic toxicity was reported to be ≥1000 mg/kg bw/day ECHA) [KI. score =1].

A two-generation reproductive toxicity study was completed using male and female Fischer 344 rats exposed to 10, 100, and 250 mg/kg bw/day alcohols, C12-15, ethoxylated via dermal exposure. No mortalities were observed in the parental generation, and the five deaths in the F1 adult males and females in the control and treatment groups were not considered to be compound related. In the highest dose group, body weights of both males and females in both treated generations were sporadically decreased compared to controls. There was no effect on maternal body weight during gestational and lactational periods in both generations. At necropsy organ weight differences in liver, lung, kidney, and heart were observed in the F1 generation. However, there were no pathological findings that were associated with these affected organs. There were no compound-related effects on mating and fertility indices and mean gestational length in both generations. No effects on testicular weights, sperm counts and LDH-X activities in F0 and F1 male adults were observed. Macroscopic and microscopic examination of the reproductive organs did not reveal significant differences in the treated groups compared to the controls. A NOAEL for systemic toxicity was reported to be ≥ 250 mg/kg bw/day based on changes in body and organ weights that were not associated with histopathological findings. A reproductive toxicity NOAEL was reported to be ≥ 250 mg/kg bw/day (ECHA)[KI. score = 2].

I. Developmental Toxicity

There are no studies are available on alcohols, C12-16, ethoxylated.

In a two-generation reproductive toxicity study, Charles River rats were given in their diet 0, 0.05, 0.1 or 0.5% (about 0, 25, 50 or 250 mg/kg-day) $C_{12}AE_6$. General behaviour, appearance and survival were unaffected by treatment. At the 0.5% dose level, adults and pups gained less weight than the control rats. In the 0.5% dose group, there was a statistical increase in embryo lethality and soft tissue anomalies and at the 0.1% there was a statistical decrease in mean foetal liver weight. Neither of these effects was considered to be treatment-related by the authors as they showed no dose response characteristics. The NOAEL for maternal toxicity is 50 mg/kg-day. The NOAEL for developmental and teratogenicity is 0.1% in the diet or 50 mg/kg-day (HERA, 2009) [Kl. score = 2].

Pregnant rabbits were given by oral gavage 0, 50, 100 or 200 mg/kg $C_{12}AE$ from gestational days 2 to 16. Nine control rabbits and 31 treated rabbits died during the study. Surviving rabbits at the 200 mg/kg dose group generally showed slight losses of body weight. At 100 and 200 mg/kg, ataxia and a slight decrease in body weight was observed in the pregnant animals. In seven treated and two control rabbits, early deliveries were recorded. There were no treatment-related effects on corpora lutea, implantations, number of live foetuses and spontaneous abortions. The NOAEL for maternal toxicity is 50 mg/kg-day; the NOAEL for developmental toxicity is 200 mg/kg-day (HERA, 2009) [Kl. score = 2].

A developmental toxicity study was conducted using Fischer 344 rats exposed to 10,100,250 mg/kg bw/day alcohols, C12-15 ethoxylated via dermal exposure three days a week from gestation day 0 until weaning. In the highest dose, body weights of both males and females in both treated generations were sporadically and not always statistically significant decreased compared to controls. At necropsy organ weight differences in liver, lung, kidney, and heart were observed in the F1 generation, but no pathological findings were associated with the affected organs. There were no

treated related effects reported for the foetuses. The NOAEL for developmental toxicity was reported to be ≥ 250 mg/kg bw/day and the NOAEL for maternal toxicity was reported to be ≥ 250 mg/kg bw/day. The NOAEL for fetotoxicity was reported to be ≥250 mg/kg bw/day (ECHA) [KI.score =2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for alcohols, C12-16, ethoxylated follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

Two-year dietary studies in rats have been conducted on alcohol ethoxylates $C_{12\text{-}13}AE_{6.5}$ and $C_{14\text{-}15}AE_{7}$ (HERA, 2009). The lowest NOAEL from these studies is 50 mg/kg-day based on increased organ weights. The NOAEL of 50 mg/kg-day will be used to derive an oral reference dose and drinking water guidance value for alcohols, C12-16, ethoxylated.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:
UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1

Oral RfD = 50/(10 x 10 x 1 x 1 x 1) = 50/100 = 0.5 mg/kg-day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,
Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

where:
Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)

Drinking water guidance value = (0.5 x 70 x 0.1)/2 = 1.8 mg/L

B. Cancer

Several alcohol ethoxylates similar to alcohols, C12-16, ethoxylated were not carcinogenic to rats in a two-year dietary study. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Alcohols, C12-16, ethoxylated does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidizing potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

There are no aquatic toxicity studies for ethoxylated C12-C16 alcohol. The aquatic toxicity of other AEs has been extensively evaluated in numerous studies on fish, daphnids and algae as well as microorganisms. A review of the acute studies indicates that invertebrates are somewhat more sensitive to AEs than fish and algae. AEs have moderate chronic toxicity to aquatic life.

B. Aquatic Toxicity

There are no acute aquatic toxicity studies for ethoxylated C12-C16 alcohol. The aquatic toxicity of other AEs has been extensively evaluated in numerous studies on fish, daphnids and algae as well as microorganisms. Table 3 lists the results of acute aquatic toxicity studies on read across substance alcohols, C12-C15, ethoxylated (1 to 2.5 EO) [CAS No. 68131-39-5], alcohols, C12-C14, ethoxylated (2 EO) [CAS No. 68439-50-9] and alcohols, C12-C15, branched and linear, ethoxylated [CAS No. 106232-83-1].

Table 3: Acute Aquatic Toxicity Studies on Ethoxylated C12-C16 Alcohol[a,b,c]

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Oncorhynchus mykiss (Rainbow Trout)	96-hr LC_{50}	1.3 – 1.7[a]	2	ECHA
Danio Rio	96-hr LC_{50}	1.2[b]	2	ECHA
Danio Rio	96-hr LC_{50}	2[b]	2	ECHA
Zebrafish	96-hr LC_{50}	>2[c]	2	ECHA
Daphnia magna	48-hr EC_{50}	0.14[a]	2	ECHA
Daphnia magna	48-hr EC_{50}	0.23[a]	2	ECHA
Daphnia magna	48-hr EC_{50}	0.53[b]	2	ECHA
Daphnia magna	48-hr EC_{50}	2.84[b,d]	2	ECHA
Daphnia magna	48-hr EC_{50}	1.2[e]	1	ECHA
Daphnia magna	48-hr EC_{50}	>2[b]	2	ECHA
Daphnia magna	48-hr EC_{50}	>2[c]	2	ECHA
Daphnia magna	48-hr EC_{50}	0.23	2	ECHA
Pseudokirchneriella subcapitata	72-hr EC_{50}	0.75[a]	2	ECHA

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Pseudokirchneriella subcapitata	72-hr EC_{50}	>2[c]	2	ECHA
Pseudokirchneriella subcapitata	72-hr EC_{50}	0.41[b]	2	ECHA
Desmodesmus subspicatus (green algae)	72-hr EC_{50}	0.778[b]	2	ECHA
Desmodesmus subspicatus (green algae)	72-hr EC_{50}	0.87[e]	1	ECHA
Desmodesmus subspicatus (green algae)	72-hr EC_{50}	1.3[e]	1	ECHA

a: Read across to alcohols, C12-C15, ethoxylated (1 to 2.5 EO) CAS No. 68131-39-5
b: Read across to alcohols, C12-C14, ethoxylated (EO 2) CAS No. 68439-50-9
c: Read across to alcohols, C12-C15, branched and linear, ethoxylated (CAS No. 106232-83-1)
d: alcohols, C12-C14, ethoxylated (EO 1) CAS No. 68439-50-9 as WAF (water accommodated fraction)
e: alcohols, C12-C14, ethoxylated (EO 4 or EO 6) CAS No. 68439-50-9

A review of the acute studies indicates that invertebrates are somewhat more sensitive to AEs than fish and algae. As concluded in HERA (2009), the Danish EPA (2001) found that the acute toxicity of AE to invertebrates varies, with EC50 values from 0.1 mg/l to more than 100 mg/l for linear AE and from 0.5 mg/l to 50 mg/l for branched AE. The toxicity is species specific and may vary between 0.29 mg/l and 270 mg/l for the same linear AE (Lewis and Suprenant 1983, quoted in Danish EPA 2001). The most commonly used invertebrates for testing are Daphnia magna and Daphnia pulex, and they are also among the most sensitive invertebrates to AE. The Danish EPA (2001) found that some AE are very toxic to invertebrates, i.e., linear AE of C12-15 EO1-8 and branched AE with a low degree of branching, i.e. < 10-25%. They concluded that branching of the alkyl chain reduces the toxicity of AE to invertebrates, as also observed for algae (Danish EPA, 2001). However, the data used to reach this conclusion is from specially synthesized AE which have been shown to have a significantly higher toxicity than the AE made from a technical alcohol which are used commercially (Kaluza and Taeger, 1996).

Chronic studies

In developing a water quality guideline for AEs (ANZG, 2018), the toxicity data was normalized for a specific alkyl chain length or a specific number of EO groups. The NOECs listed below were normalized to an alkyl chain length of C13.3 and EO of 8.2. There were chronic data for 13 species that belonged to 7 taxonomic groups (fish, crustacea, blue alga, diatoms, green alga, protozoa, and worms).

Freshwater fish: 2 species, 720 to 1,500 µg/L.

Freshwater crustaceans: 2 species, 590 to 860 µg/L.

Freshwater rotifers: 1 species, Brachionus calyciflorus, 1,300 µg/L.

Freshwater algae, diatoms and blue-green algae: 6 species, 200 to 8,700 µg/L.

Freshwater mesocosms: 4 NOEC data for multiple species tests were 80, 80, 320 and 330 µg/L, although replication was insufficient to meet OECD (1992) requirements. Normalized data were 380, 380, 320 and 1,520 µg/L.

C. Terrestrial Toxicity

There are no studies are available. The substance is readily biodegradable. Therefore, soil is not expected to be a compartment of concern. Thus, the risk to terrestrial macroorganisms is regarded to be negligible (ECHA).

D. Calculation of PNEC

The PNEC calculations for ethoxylated C12-C16 alcohol follow the methodology discussed in DEWHA (2009).

PNEC water

The ANZECC water quality guideline (2018) for freshwater is: "A high reliability trigger value of 140 mg/L was derived for AE (normalized data) using the statistical distribution method with 95% protection."

For the purposes of calculating the PNEC values for sediment and soil, the $PNEC_{water}$ will be 0.14 mg/L.

PNEC sediment

There are no toxicity data for sediment-dwelling organisms. Nonetheless, a $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 0.0875 mg/kg sediment wet weight.

The calculations are as follows:
$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= 0.800/1280 \times 1000 \times 0.140$
$= 0.0875$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 kg/m^3[default]
$PNEC_{water}$ = 0.002 mg/L

$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1000 \times BD_{solid}]$
$= 0.8 + [(0.2 \times 156.8)/1000 \times 2400]$
$= 0.800\ m^3/m^3$

And:
Kp_{sed} = solid-water partition coefficient (L/kg)
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 kg/m^3[default]
$Kp_{sed} = K_{oc} \times f_{oc}$
$= 3920 \times 0.04$
$= 156.8$ L/kg

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for alcohols, C12-16, ethoxylated based on the molecular connectivity index (MCI) is 3,920 L/kg (USEPA, 2018).
f_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 7.32 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
$= (78.4/1500) \times 1000 \times 0.14$
$= 7.32 mg/kg$

Where:
Kp_{soil} = soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 kg/m^3 [default]

$Kp_{soil} = K_{oc} \times f_{oc}$
$= 3920 \times 0.02$
$= 78.4\ m^3/m^3$

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for alcohols, C12-16, ethoxylated based on the molecular connectivity index (MCI) is 3,920 L/kg (USEPA, 2018).
f_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU Reach Criteria methodology (DEWHA, 2009; ECHA, 2017).

Alcohols, C12-16, ethoxylated is readily biodegradable and thus does not meet the screening criteria for persistence.

The bioconcentration factors (BCF) in fish for ethoxylated alcohols (which includes alcohols, C12-16, ethoxylated) have been reported to range from <5 to 387.5. Thus, alcohols, C12-16, ethoxylated does not meet the screening criteria for bioaccumulation.

The chronic NOEC values for alcohols ethoxylates are >0.1 mg/L. Thus, alcohols, C12-16, ethoxylated do not meet the criteria for toxicity.

The overall conclusion is that alcohols, C12-16, ethoxylated is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

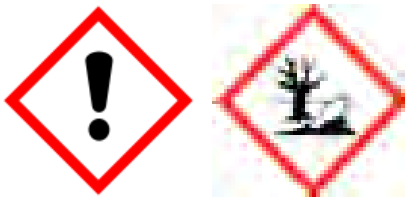
A. Classification

H400: Very toxic to aquatic life
H412: Harmful to aquatic life with long lasting effects

B. Labelling

Warning

C. Pictogram



X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Rinse immediately with plenty of running water. If easy to do, remove contact lenses. Get medical attention.

Skin Contact

Wash with soap and water. Get medical attention if symptoms occur.

Inhalation

Treat symptomatically. Move to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink plenty of water. Never give anything by mouth to an unconscious person. Seek medical attention.

B. Fire Fighting Information

Extinguishing Media

Water spray, dry chemical, foam. Do not use water jet.

Specific Exposure Hazards

May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon oxides.

Special Protective Equipment for Firefighters

Self-contained breathing apparatus and full protective clothing must be worn in case of fire.

C. Accidental Release Measures

Personal Precautions

Wear appropriate personal protective equipment. Do not breath mist or aerosol.

Environmental Precautions

Prevent from entering sewers, waterways, or low area

Steps to be Taken if Material is Released or Spilled

Absorb spill with inert absorbent material, then place in a container for chemical waste.

D. Storage And Handling

General Handling

Protect against moisture. Shut containers immediately after taking product because product takes up the humidity of air. No special precautions are necessary beyond normal good hygiene practices.

Other Handling Precautions

Wash hands thoroughly after handling. Avoid breathing mists or aerosols.

Storage

Keep container closed.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for alcohols, C12-16, ethoxylated.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection:
Wear respiratory protection if ventilation is inadequate.

Hand Protection:
Chemical resistant protective gloves.

Skin Protection:
Body protection must be chosen depending on activity and possible exposure.

Eye protection:
Chemical safety goggles.

Other Precautions:
Handle in accordance with good industrial hygiene and safety practice. Eyewash fountains and safety showers must be easily accessible.

F. Transport Information

UN: UN 1993

Class:3

Packaging Group: II

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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ALCOHOLS, C11-14-ISO-, C13-RICH, ETHOXYLATED

This dossier on alcohols, C11-14-iso-, C13-rich, ethoxylated presents the most critical studies pertinent to the risk assessment of alcohols, C11-14-iso-, C13-rich, ethoxylated in their use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The information presented in this dossier was obtained primarily from the Human & Environmental Risk Assessment on Ingredients of European Household Cleaning Products: Alcohol Ethoxylates (HERA, 2009). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Alcohols, C11-14-iso-, C13-rich, ethoxylated

CAS RN: 78330-21-9

Molecular formula: Not available (UVCB substance)

Molecular weight: 233.46 g/mol

Synonyms: Ethoxylated branched C11-14, C13-rich alcohols; alpha-Alkyl-omega-hydroxypoly(oxypropylene) and/or poly(oxyethylene) polymers where the alkyl chain contains a minimum of six carbons, minimum number average molecular weight (in amu) 1,100

SMILES: C.C.[*]C.CCCCCCCCCCOCC

II. PHYSICO-CHEMICAL PROPERTIES

Alcohol ethoxylates (AEs) are a class of non-ionic surfactants that have the basic structure $C_{x-y}AE_n$. The subscript (x-y) following the 'C' indicates the range of carbon chain units. The hydrocarbon chain can be either linear or branched. AEs also contain an ethylene oxide (E) chain attached to the alcohol. The degree of ethylene oxide polymerization is indicated by the subscript (n) which indicates the average number of ethylene oxide units. Alcohols, C11-14-iso-, C13-rich, ethoxylated has an average number of 7 moles of ethylene oxide units.

No information is available on alcohols, C11-14-iso-, C13-rich, ethoxylated. Thus, data were read across from a similar substance, alcohols, C12-15, ethoxylated, as shown below.

Table 1: Overview of the Physico-Chemical Properties of Alcohols, C11-14-iso-, C13-rich, ethoxylated[1]

Property	Value	Klimisch score	Reference
Physical state at 20°C and 101.3 kPa	Clear liquid with a rancid odour*	2	ECHA
Melting Point	7.22°C @ 101.3 kPa	2	ECHA
Boiling Point	287°C @ 101.3 kPa	1	ECHA
Density	926 kg/m^3 @ 15.56°C	1	ECHA
Vapour Pressure	Negligible	-	ECHA

Property	Value	Klimisch score	Reference
Partition coefficient (log K_{ow})	5.06* @25 ^{O}C	2	ECHA
Water Solubility	0.007 – 0.063 g/L @ 25 ^{o}C	1	ECHA
Flash Point	165.56 ^{o}C	2	ECHA
Auto Flammability	235 ^{o}C	2	ECHA
Viscosity	28.1 mPa s (dynamic) @ 20^{o}C	1	ECHA

1 – Based on alcohols, C12-C15, ethoxylated (1 to 2.5 EO) [CAS RN 68131-39-5]

*Based on alcohols, C12-14, ethoxylated (1 to 2.5 EO) [CAS RN 68439-50-9]

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Alcohols, C11-14-iso-, C13-rich, ethoxylated is readily biodegradable. They are slightly soluble and have high adsorption potential to soil and sediment. However, they have a low potential to bioaccumulate.

B. Biodegradation

No studies are available on alcohols, C11-14-iso-, C13-rich, ethoxylated.

AEs with a typical alkyl chain (e.g., C12 to C15) will normally reach more than 60% ultimate degradation in standardized tests for ready biodegradability (HERA, 2009). For example, alcohols, C12-15, ethoxylated is readily biodegradable. In an OECD 301B test, degradation was 72% in 28 days, but failed the 10-day window (ECHA) [Kl. score = 1].

An alcohol, C12-15, ethoxylated (7 EO) degraded 80 to 88% in 28 days when tested using a shake-flask CO_2-evolution test method (ECHA) [Kl. score = 2].

If a chemical is found to be inherently biodegradable or readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

No experimental data are available for alcohols, C11-14-iso-, C13-rich, ethoxylated.

Using KOCWIN in EPISUITE™ (USEPA, 2019), the estimated K_{oc} values for alcohols, C1.1-14-iso-, C13-rich, ethoxylated were 5649 L/kg (MCI) and 20,085 L/kg (K_{ow}). However, as described in ECHA, one should keep in mind that surfactancy (the fact that surfactants tend to stay in the boundary layer between the phases) and dissociation is not considered in the EPISUITE™ estimations. Therefore, calculated K_{oc} values should be used with caution.

If released to soil, these K_{oc} values indicate a high potential for both adsorption and low potential for mobility. If released to water, based on these K_{oc} values and slight solubility, this substance is expected to strongly adsorb to suspended solids or sediment.

D. Bioaccumulation

There are no bioaccumulation studies on this substance. The BCF values for AEs in fathead minnows have been reported to range from <5 to 387.5 (Toll et al., 2000). The uptake rates varied from 330 to 1,660 (L x kg/d) and elimination rates varied from 3.3 to 59 per day (Toll et al., 2000). The high concentrations in fish are thought to be prevented by an efficient biotransformation of the alcohol ethoxylates, leading to a high elimination rate. Thus, it can be stated that bioaccumulation of AEs is regarded to be negligible as the surfactants will be rapidly metabolised (ECHA).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Overall, AEs are not expected to be systemically toxic. The available datasets for AEs ranging from C6–C18 and EO3–EO12 are considered representative of the AE category and were used to assess alcohols, C11-14-iso-, C13-rich, ethoxylated.

The acute toxicity of similar AEs is low by the oral and dermal routes. The skin irritation rabbit studies show that the degree of irritation depends on the testing conditions and length of the exposure period. Human patch studies on AEs do not support a skin irritant classification and alcohol ethoxylates in this group are not considered skin sensitisers. Alcohols, C11-14-iso-, C13-rich, ethoxylated is expected to be irritating to the eyes of rabbits. Repeated dose toxicity studies on AEs similar to alcohols, C11-14-iso-, C13-rich, ethoxylated in rats do not indicate any target organ effects. These AEs are not genotoxic, carcinogenic, and have a low potential for reproductive and developmental toxicity.

B. Metabolism

In rats, AEs are readily absorbed in the gastrointestinal tract (i.e., oral absorption has been estimated to be >75%) and rapidly excreted via the urine and faeces after oral application. The alkyl chain length appears to have an impact on the metabolism. AEs with longer alkyl chains are excreted at a higher proportion into expired air and less in urine. Also, ethoxy chain length impacts the proportions excreted via the urine, the faeces, and the expired air with more being excreted via the faeces and expired in the air with longer ethoxy chain length (HERA, 2009).

The same trends were observed when AEs were administered dermally, with the only difference being that adsorption was slower and less of the total administered compound was absorbed (HERA, 2009).

C. Acute Toxicity

No acute toxicity studies are available on alcohols, C11-14-iso-, C13-rich, ethoxylated.

The oral LD_{50} in rats for $C_{12\text{-}15}AE_3$ is >5,000 milligrams per kilogram (mg/kg) (ECHA) [Kl. score = 2]. The oral LD_{50} in rats for $C_{12\text{-}15}AE_7$ is 1,700 mg/kg (HERA, 2009) [Kl. score = 2]. The oral LD_{50} value in rats for $C_{12\text{-}13}AE_{6.5}$ is 2,100 mg/kg (HERA, 2009) [Kl. score = 2]. The oral LD_{50} value in rats for $C_{12\text{-}15}AE_{11}$ is >2,000 mg/kg in males and between 1,000 and 2,000 mg/kg in females (HERA, 2009) [Kl. score = 2]. The oral LD_{50} values in rats for $C_{14\text{-}15}AE_{13}$ in two separate studies are 1,100 and 1,000 mg/kg (HERA, 2009) [Kl. score = 2]. The relative number of ethoxylate (EO) units, but not the carbon chain length, appears to influence acute oral toxicity (HERA, 2009).

An acute dermal LD_{50} values of >2,000 mg/kg were determined for $C_{12\text{-}14}AE_3$ and $C_{12\text{-}14}AE_6$ in two separate studies (HERA, 2009) [Kl. score = 2]. The acute dermal LD_{50} of $C_{12\text{-}15}AE_7$ is >2,000 mg/kg (HERA, 2009) [Kl. score = 2].

D. Irritation

Skin

Application of 0.5 millilitres (mL) $C_{12\text{-}13}AE_{<2.5}$ (CAS RN 66455-14-9) to the skin of rabbits for 24 hours under occlusive conditions was considered irritating (ECHA) [Kl. score = 2].

Application of 0.5 mL alcohols $C_{12\text{-}13}$, branched and linear, <2.5 EO to the skin of rabbits for 4 hours under occlusive conditions was not considered irritating (ECHA) [Kl. score = 2].

In a 24-hour human patch test, there was some short-lived redness in some individuals from the application of $C_{12\text{-}14}AE_3$, but there was no scaling or oedema in any subjects (HERA, 2009) [Kl. score = 2].

In a standard 4-hour human patch test, the irritation potential of $C_{12\text{-}15}AE_5$ and $C_{12\text{-}15}AE_5$ were compared to 20% sodium dodecyl sulfate, which is classified a skin irritant under GHS. The results showed that neither AE should be classified as a skin irritant (Basketter et al., 2004) [Kl. score = 2].

Eye

Most AEs tested as the undiluted neat test material are moderately to severely irritating to the eyes of rabbits, with an eye irritation index (EII) ranging from >25 to 50 (HERA, 2009). The AEs $C_{12\text{-}14}AE_3$, $C_{12\text{-}14}AE_6$, $C_{13}AE_6$, and $C_{12\text{-}14}AE_{10}$ were found to be moderately to severely irritating to the eyes of rabbits (HERA, 2009). In another study, $C_{12\text{-}15}AE_{11}$ was considered moderately to severely irritating to the eyes of rabbits (HERA, 2009).

Some AEs were reported to be practically or minimally irritating to the eyes of rabbits with EII scores of 0.5 to 15. These AEs include: $C_{12\text{-}15}AE_3$, $C_{14\text{-}15}AE_7$, $C_{12\text{-}14}AE_{15}$, $C_{14\text{-}15}AE_{18}$, and $C_{13}AE_{20}$ (HERA, 2009).

E. Sensitisation

No sensitisation studies are available on alcohols, C11-14-iso-, C13-rich, ethoxylated.

In a guinea pig maximisation test, $C_{12\text{-}13}AE_{<2.5}$ (CAS RN 66455-14-9) was not considered a skin sensitiser (ECHA) [Kl. score = 2].

F. Repeated Dose Toxicity

Oral

No repeated dose toxicity studies are available on alcohols, C11-14-iso-, C13-rich, ethoxylated.

Rats were given 0%, 0.0313%, 0.0625%, 0.125%, 0.25%, 0.5% or 1.0% $C_{12\text{-}15}AE_7$ in their diet for 90 days. The animals in the $\geq$0.25% groups showed significantly reduced body weight gain, which was associated with marked decreases in food and water consumption. Relative liver weights were significantly increased in the $\geq$0.5% male rats and $\geq$0.25% females. Histopathologic examination showed hepatocytic enlargement in the $\geq$0.125% groups, suggesting increased liver metabolism on the basis of increased alkaline phosphatase activity at the higher dose levels. The no observed

adverse effect level (NOAEL) was established at 0.0625% in the diet or 102 mg/kg/day (HERA, 2009) [Kl. score = 2].

Rats were fed $C_{12-14}AE_7$ in the diet at concentrations of 0%, 0.0313%, 0.0625%, 0.125%, 0.25%, 0.5% and 1.0% for 90 days. The animals in the ≥0.25% groups showed significantly reduced body weight gain, which was associated with marked decreases in food and water consumption. Relative liver weights were significantly increased in the ≥0.5% male rats and ≥0.25% females. Histopathologic examination showed hepatocytic enlargement in the ≥0.125% groups, suggesting increased liver metabolism on the basis of increased alkaline phosphatase activity at the higher dose levels. The NOAEL was established at 0.0625% in the diet or 110 mg/kg/day (HERA, 2009) [Kl. score = 2].

Rats were given 0%, 0.1%, 0.5% or 1% $C_{12-13}AE_{6.5}$ in their diet for two years. Body weight gain was reduced in the 1% males and ≥0.5% females, which was likely due to the reduced food consumption in these animals. At study termination, organ to body weight ratios were increased in the ≥0.5% females (liver, kidney and brain), 1% females (heart), and 1% males (liver). A dose-related focal myocarditis was observed in males. While focal myocarditis is commonly observed in non-treated aging rats, the incidence in the treated animals were higher than in the controls. The NOAEL was established at 0.1% or 50 mg/kg/day (HERA, 2009) [Kl. score = 2].

Inhalation

No studies are available.

Dermal

No adequate studies are available.

G. Genotoxicity

In Vitro Studies

The genotoxicity studies conducted on alcohol ethoxylates are reviewed in HERA (2009). The results of few of the *in vitro* studies on similar alcohol ethoxylates to alcohols, C11-14-iso-, C13-rich, ethoxylated are presented in Table 2.

Table 2: *In Vitro* Genotoxicity Studies on Selected Alcohol Ethoxylates

Test Substance	Test System	Results*		Klimisch Score	Reference
		-S9	+S9		
$C_{14-15}AE_7$	Bacterial reverse mutation (*S. typhimurium* strains)	-	-	2	HERA, 2009
$C_{14-15}AE_7$	Bacterial reverse mutation (*S. typhimurium* strains)	-	-	2	HERA, 2009
$C_{14}AE_{12}$	Chromosomal aberrations (CHO cells)	-	-	2	HERA, 2009

*+, positive; -, negative

In Vivo Studies

In two separate studies, CD-1 mice were given an intraperitoneal dose of 0, 50, or 100 mg/kg $C_{12-15}AE_3$ or $C_{12-14}AE_9$. There were no increases in the frequency of micronuclei in the bone marrow cells (Talmage, 1994) [Kl. score = 2].

Male and female Tunstall rats were given a single oral gavage dose of 0, 250, 500, or 1,000 mg/kg $C_{14-15}AE_7$. There were no increases in chromosomal aberrations in the bone marrow cells (HERA, 2009) [Kl. score = 2].

H. Carcinogenicity

No studies are available on alcohols, C11-14-iso-, C13-rich, ethoxylated. Based on the available data, chemicals in this group are not considered carcinogenic.

Male and female Sprague-Dawley rats were given $C_{12-13}AE_{6.5}$ in their diet at doses up to 1% (500 mg/kg/day). Reduced food consumption was noted at the higher dose levels (i.e., 0.5% and 1% for females and 1% for males), resulting in a lower body weight gain compared to the control group. No treatment-related histopathology was found and no increase in tumour incidence was observed (HERA, 2009) [Kl. score = 2].

Male and female Charles River rats were given 0, 0.1, 0.5 or 1% $C_{14-15}AE_7$ in their diet for two years. There were no treatment-related changes in general behaviour and appearance. The survival rate of the test animals was comparable if not better than the controls. Body weights of the 0.5% females and the 1% males and females had significantly lower weight gains than the control. There were no treatment-related effects on organ weights and tumour incidence (HERA, 2009) [Kl. score = 2]

Male and female Sprague-Dawley rats were given $C_{14-15}AE_7$ in their diet at 0.1%, 0.5% and 1% for two years. A treatment-related body weight depression was observed in females at the two highest treatment levels and in males at the 1% dose level, probably due to the poor palatability of the diet. There was no evidence for any carcinogenic activity (HERA, 2009) [Kl. score = 2].

I. Reproductive Toxicity

No studies are available on alcohols, C11-14-iso-, C13-rich, ethoxylated. Based on the data available, the chemicals of this group are not considered to cause reproductive toxicity.

CD rats were given 0%, 0.05%, 0.1% or 0.5% (approximately 0, 25, 50, or 250 mg/kg/day) $C_{12}AE_6$ in their diet in a two-generation reproductive toxicity study. There were no treatment related effects in the parents or pups on general behaviour, appearance or survival. At 0.5%, there was reduced weight gain in both the parental animals and the pups compared to the controls. Fertility was unaffected by treatment. The NOAEL for reproductive toxicity is 0.5% in the diet, which corresponds to 250 mg/kg/day (HERA, 2009) [Kl. score = 2].

In a two-generation developmental and teratogenicity study, CD rats were given 0%, 0.05%, 0.1% or 0.5% $C_{14-15}AE_7$ in their diet (approximately 0, 25, 50 or 250 mg/kg/day). Three of the treated groups were given the test substance continuously throughout the study; in the other three groups the females received the test substance on GD 6-15 and the males were untreated. None of the deaths of parental rats during the study was considered to be compound-related. There were no treatment-related changes in behaviour or appearance in the parental rats or pups. Slightly lower body weight gain was noted in the 0.5% continuously treated females. Food consumption was similar for control

and treated rats. Fertility, gestation and viability indices were similar across groups. The average 21-day body weights for the 0.5% continuous treated pups were significantly lower than that of the control. Relative liver weights of the 0.5% continuously treated F_1 parental animals were increased at the 91-day sacrifice; relative liver weights of the 0.5% continuously treated males were also increased at the 60-day and caesarean section sacrifices. There were no treatment-related histopathological lesions in any of the tissues from the F_0 and F_1 generations. The NOAEL for reproductive toxicity is 0.5% in the diet or 250 mg/kg/day (HERA, 2009) [Kl. score = 2].

J. Developmental Toxicity

No studies are available on alcohols, C11-14-iso-, C13-rich, ethoxylated. Based on the data available, the chemicals of this group are not considered to cause developmental toxicity.

In a two-generation reproductive toxicity study, Charles River rats were given 0, 0.05, 0.1 or 0.5% (about 0, 25, 50 or 250 mg/kg/day) $C_{12}AE_6$ in their diet. General behaviour, appearance and survival were unaffected by treatment. At the 0.5% dose level, adults and pups gained less weight than the control rats. In the 0.5% dose group, there was a statistical increase in embryo lethality and soft tissue anomalies, and at the 0.1% there was a statistical decrease in mean foetal liver weight. Neither of these effects was considered to be treatment-related by the authors as they showed no dose response characteristics. The NOAEL for maternal toxicity is 50 mg/kg/day. The NOAEL for developmental and teratogenicity is 0.1% in the diet or 50 mg/kg/day (HERA, 2009) [Kl. score = 2].

Pregnant rabbits were given 0, 50, 100 or 200 mg/kg $C_{12}AE_6$ by oral gavage from gestational days 2 to 16. Nine control rabbits and 31 treated rabbits died during the study. Surviving rabbits at the 200 mg/kg dose group generally showed slight losses of body weight. At 100 and 200 mg/kg, ataxia and a slight decrease in body weight was observed in the pregnant animals. In seven treated and two control rabbits, early deliveries were recorded. There were no treatment-related effects on corpora lutea, implantations, number of live foetuses and spontaneous abortions. The NOAEL for maternal toxicity is 50 mg/kg/day; the NOAEL for developmental toxicity is 200 mg/kg/day (HERA, 2009) [Kl. score = 2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for alcohols, C11-14-iso-, C13-rich, ethoxylated follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

<u>Oral</u>

A two-year dietary study in rats has been conducted on $C_{12\text{-}13}AE_{6.5}$ (HERA, 2009). The NOAEL from this study is 50 mg/kg/day based on increased organ weights. The NOAEL of 50 mg/kg/day will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10

UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1
Oral RfD = 50/(10 x 10 x 1 x 1 x 1) = 50/100 = 0.5 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:
Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (0.5 x 70 x 0.1)/2 = 1.8 mg/L

B. Cancer

The AEs $C_{12-13}AE_{6.5}$ and $C_{14-15}AE_7$ were not carcinogenic to rats in a two-year dietary study. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Alcohols, C11-14-iso-, C13-rich, ethoxylated does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Alcohols, C11-14-iso-, C13-rich, ethoxylated has moderate chronic toxicity concern to aquatic life.

B. Aquatic Toxicity

Acute Studies

There are no acute aquatic toxicity studies for ethoxylated C12-C16 alcohol. The aquatic toxicity of other AEs has been extensively evaluated in numerous studies on fish, daphnids and algae as well as microorganisms. Table 3 lists the results of acute aquatic toxicity studies on read across substance alcohols, C12-C15, ethoxylated (1 to 2.5 EO) [CAS RN 68131-39-5], alcohols, C12-C14, ethoxylated (2 EO) [CAS RN 68439-50-9] and alcohols, C12-C15, branched and linear, ethoxylated [CAS RN 106232-83-1].

Table 3: Acute Aquatic Toxicity Studies on Ethoxylated C12-C16 Alcohol[a,b,c]

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Oncorhynchus mykiss (Rainbow Trout)	96-hr LC_{50}	1.3 – 1.7[a]	2	ECHA
Danio Rio	96-hr LC_{50}	1.2[b]	2	ECHA
Danio Rio	96-hr LC_{50}	2[b]	2	ECHA
Zebrafish	96-hr LC_{50}	>2[c]	2	ECHA
Daphnia magna	48-hr EC_{50}	0.14[a]	2	ECHA
Daphnia magna	48-hr EC_{50}	0.23[a]	2	ECHA
Daphnia magna	48-hr EC_{50}	0.53[b]	2	ECHA
Daphnia magna	48-hr EC_{50}	2.84[b,d]	2	ECHA
Daphnia magna	48-hr EC_{50}	1.2[e]	1	ECHA
Daphnia magna	48-hr EC_{50}	>2[b]	2	ECHA
Daphnia magna	48-hr EC_{50}	>2[c]	2	ECHA
Daphnia magna	48-hr EC_{50}	0.23	2	ECHA
Pseudokirchneriella subcapitata	72-hr EC_{50}	0.75[a]	2	ECHA
Pseudokirchneriella subcapitata	72-hr EC_{50}	>2[c]	2	ECHA
Pseudokirchneriella subcapitata	72-hr EC_{50}	0.41[b]	2	ECHA
Desmodesmus subspicatus (green algae)	72-hr EC_{50}	0.778[b]	2	ECHA
Desmodesmus subspicatus (green algae)	72-hr EC_{50}	0.87[e]	1	ECHA
Desmodesmus subspicatus (green algae)	72-hr EC_{50}	1.3[e]	1	ECHA

a: Read across to alcohols, C12-C15, ethoxylated (1 to 2.5 EO) CAS No. 68131-39-5
b: Read across to alcohols, C12-C14, ethoxylated (EO 2) CAS No. 68439-50-9
c: Read across to alcohols, C12-C15, branched and linear, ethoxylated (CAS No. 106232-83-1)
d: alcohols, C12-C14, ethoxylated (EO 1) CAS No. 68439-50-9 as WAF (water accommodated fraction)
e: alcohols, C12-C14, ethoxylated (EO 4 or EO 6) CAS No. 68439-50-9

A review of the acute studies indicates that invertebrates are somewhat more sensitive to AEs than fish and algae. As concluded in HERA (2009), the Danish EPA (2001) found that the acute toxicity of AE to invertebrates varies, with EC50 values from 0.1 mg/L to more than 100 mg/L for linear AE and from 0.5 mg/L to 50 mg/L for branched AE. The toxicity is species specific and may vary between 0.29 mg/L and 270 mg/L for the same linear AE (Lewis and Suprenant 1983, quoted in Danish EPA 2001). The most commonly used invertebrates for testing are Daphnia magna and Daphnia pulex, and they are also among the most sensitive invertebrates to AE. The Danish EPA (2001) found that some AEs are very toxic to invertebrates (i.e., linear AE of C12-15 EO1-8 and branched AE with a low degree of branching, < 10-25%). They concluded that branching of the alkyl chain reduces the toxicity of AE to invertebrates, as also observed for algae (Danish EPA, 2001). However, the data used to reach this conclusion is from specially synthesised AEs, which have been shown to have a significantly higher toxicity than the AE made from a technical alcohol which are used commercially (Kaluza and Taeger, 1996).

Chronic Studies

In developing a water quality guideline for AEs (ANZG, 2018), the toxicity data was normalised for a specific alkyl chain length or a specific number of EO groups. The no observed effect concentrations (NOECs) listed below were normalised to an alkyl chain length of C13.3 and EO of 8.2.

Freshwater fish: 2 species, 720 to 1,500 micrograms per litre (μg/L).

Freshwater crustaceans: 2 species, 590 to 860 μg/L.

Freshwater rotifers: 1 species, *Brachionus calyciflorus*, 1,300 μg/L

Freshwater algae, diatoms and blue-green algae: 6 species, 200 to 8,700 μg/L.

Freshwater mesocosms: 4 NOEC data for multiple species tests were 80, 80, 320, and 330 μg/L, although replication was insufficient to meet OECD (1992) requirements. Normalised data were 380, 380, 320, and 1,520 μg/L.

C. Terrestrial Toxicity

No studies are available.

D. Calculation of PNEC

The PNEC calculations for alcohols, C11-14-iso-, C13-rich, ethoxylated follow the methodology discussed by DEWHA (2009).

PNEC Water

The ANZG water quality guideline (2018) for freshwater is: "*A high reliability trigger value of 140 μg/L was derived for AE (normalised data) using the statistical distribution method with 95% protection.*"

For the purposes of calculating the PNEC values for sediment and soil, the $PNEC_{water}$ will be 0.14 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 11.95 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (109/1280) \times 1000 \times 0.14$
$= 11.95$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (cubic metre per cubic metre [m^3/m^3])
BD_{sed} = bulk density of sediment (kilograms per cubic metre [kg/m^3]) = 1,280 [default]

$K_{sed\text{-}water}$ = 0.8 + [(0.2 x Kp_{sed})/1,000 x BD_{solid}]
= 0.8 + [(0.2 x 226/1,000 x 2,400]
= 109 m^3/m^3

Where:
Kp_{sed} = solid-water partition coefficient (litres per kilogram [L/kg])
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]

Kp_{sed} = K_{oc} x f_{oc}
= 5,649 x 0.04
= 226 L/kg

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for alcohols, C11-14-iso-, C13-rich, ethoxylated calculated from EPISUITE™ using the MCI is 5,649 L/kg. The MCI method is preferred to the K_{ow} method due to the surfactant properties of the substance.
F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 10.54 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil}$ = (Kp_{soil}/BD_{soil}) x 1,000 x $PNEC_{water}$
= (113/1,500) x 1,000 x 0.14
= 10.54 mg/kg

Where:
Kp_{soil} = soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]

Kp_{soil} = K_{oc} x f_{oc}
= 5,649 x 0.02
= 113 m^3/m^3

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for alcohols, C11-14-iso-, C13-rich, ethoxylated calculated from EPISUITE™ using the MCI is 5,649 L/kg.
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Alcohols, C11-14-iso-, C13-rich, ethoxylated is readily biodegradable and thus does not meet the screening criteria for persistence.

The measured BCF in fish for AEs, which includes alcohols, C11-14-iso-, C13-rich, ethoxylated, have been reported to range from <5 to 387.5. Thus, alcohols, C11-14-iso-, C13-rich, ethoxylated does not meet the screening criteria for bioaccumulation.

The chronic NOEC values for AEs are >0.1 mg/L. Thus, alcohols, C11-14-iso-, C13-rich, ethoxylated does not meet the criteria for toxicity.

The overall conclusion is that alcohols, C11-14-iso-, C13-rich, ethoxylated is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Eye Irritant Category 2

Aquatic Chronic Toxicity Category 3

B. Labelling

Danger! According to the classification provided by companies to ECHA in Classification, Labelling and Packaging (CLP) notifications, this substance is very toxic to aquatic life, causes serious eye damage, is harmful if swallowed, is harmful to aquatic life with long lasting effects and causes skin irritation.

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Self-contained breathing apparatus and full protective clothing must be worn in case of fire.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Protect against moisture. Shut containers immediately after taking product because product takes up the humidity of air. No special precautions are necessary beyond normal good hygiene practices.

Wash hands thoroughly after handling. Avoid breathing mists or aerosols.

Storage

Keep container tightly closed.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for alcohols, C11-14-iso-, C13-rich, ethoxylated.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required if ventilation is adequate.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Alcohols, C11-14-iso-, C13-rich, ethoxylated is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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ALKYLPYRIDINE

This dossier on alkylpyridine presents the most critical studies pertinent to the risk assessment of alkylpyridine in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): 1-benzyl-1-methyl-2H-pyridin-1-ium; chloride

CAS RN: 68909-18-2

Molecular formula: $C_{12}H_7ClNR1R2R3R4R5$, where R1-5 are alkyl groups

Molecular weight: 221.72 g/mol

Synonyms: Alkylpyridine; Et Me derivs., chlorides, Pyridinium, methyl-1-(phenylmethyl)-, chloride, N-Benzylpicolinonium chloride, Pyridinium, methyl-1-(phenylmethyl)-, chloride (1:1)

SMILES: C[N+]1(CC=CC=C1) CC2=CC=CC=C2.[Cl-]

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-chemical Properties of Alkylpyridine

Property	Value	Klimisch Score	Reference
Physical state at 20oC and 101.3 kPa	Liquid	1	ECHA
Melting Point	-57.27°C @ 101.3 kPa	1	ECHA
Boiling Point	116.34°C @ 101.3 kPa	1	ECHA
Density	1,104 kg/m^3	2	ECHA
Vapour Pressure	200 Pa @ 20°C	2	ECHA
Partition Coefficient (log K_{ow})	3 @ 25°C	2	ECHA
Water Solubility	100 g/L @ 30°C	2	ECHA
Flash Point	55°C	1	ECHA
Auto flammability	There is no evidence of self-ignition at temperatures up to 400°C@ 101.49 kPa	1	ECHA
Viscosity	47.9 mm^2/s (static) @ 38°C	1	ECHA
Henry's Law Constant	This endpoint is not technically feasible due to the UVCB nature of the substance	-	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

N-benzyl-alkylpyridium chloride is inherently biodegradable. Components show variable sorption to soils and sediments. It is not expected to bioaccumulate based on the experimental log K_{ow}.

B. Biodegradation

The ready biodegradation of N-benzyl-alkylpyridium chloride in seawater was determined according to OCED guideline 306 (Biodegradability in Seawater). The rate of degradation was estimated at 13% in seawater assay. The substance was considered likely to be inherently biodegradable (ECHA) [Kl Score=3].

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

A screening test conducted in accordance with OECD 121 indicated that due to its multi component nature, N-benzyl-alkylpyridium chloride displayed a range of Log K_{oc} values from <1.25 to 5.40. The substance is considered to be a UVCB substance comprising multiple components, of similar chemical functionality, in varying proportions. A quantitative assessment of these components would therefore present considerable technical difficulty as there is not considered to be an analytical method that is sufficiently sensitive, and so a more detailed assessment in accordance with OECD 106 for example would not be technically possible. For the purposes of this dossier, a log K_{oc} is estimated to be a midpoint of the range stated above (i.e., approximately 3).

D. Bioaccumulation

No bioconcentration studies have been conducted on N-benzyl alkylpyridium chloride. N-benzyl alkylpyridium chloride is not expected to bioaccumulate based on the experimental log K_{ow} of 3 (ECHA) [Kl. score = 1].

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Very little information exists regarding the specific hazards associated with N-benzyl alkylpyridium chloride. Thus, the information provided in this section is taken from data collected for quaternary ammonium compounds in general.

Significant absorption of quaternary ammonium compounds is unlikely due to their highly ionic nature. As the substance is corrosive (i.e., pH=1.2), very little toxicity data are available except for acute toxicity data showing a LD_{50} in rats that is approximately 50 mg/kg-day.

B. Metabolism

No toxicokinetic data are available for these substances, however the data on related quaternary ammonium compounds are summarised below.

Absorption

Significant absorption of quaternary ammonium compounds is unlikely due to their highly ionic nature. WHO (1998) reports the oral absorption of quaternary ammonium compounds in general to be poor. A published Canadian review of the toxicity of the quaternary ammonium compound didecyldimethylammonium chloride (DDAC) notes experiments in rats in which up to 99% of orally administered radioactivity was recovered in the faeces and less than 2.5% in the urine (ECHA 2020).

The dermal absorption of quaternary ammonium compounds is likely to be low based on the chemical structure, ionic nature, molecular weight, and lack of lipophilicity of the substance. Absorption of this group of substances through skin is also indicated to be very low based on an absence of reports of systemic effects following dermal exposure (WHO, 1998). However, it is noted that the substance is corrosive, therefore it is possible that systemic absorption may occur following significant accidental dermal exposures resulting in skin burns, where the normal barrier integrity of the skin is compromised. Buist et al. (2007) reported very low dermal penetration (0.5%) for the quaternary ammonium compound DDAC in human skin in vitro over a 48-hour period.

No data are available for absorption following inhalation exposure; however, it is considered unlikely that absorption by his route of exposure would be significant. Although not relevant to the human risk assessment, the WHO document notes that the systemic absorption of quaternary ammonium compounds following parenteral administration is 'possible'.

Distribution

No data on distribution are available. However, given the water solubility of the substance, it is likely to be widely distributed via the circulation if absorbed.

Metabolism

No data are available for the substance; however significant metabolism is not predicted given the likely poor systemic absorption. A published Canadian review of the toxicity of the quaternary ammonium compound DDAC reports some oxidative metabolism of the decyl sidechain, but no molecular cleavage by N-dealkylation (Henderson, 1992).

Excretion

Data indicate that quaternary ammonium compounds are largely excreted in the faeces (WHO, 1998; Henderson, 1992). The poor absorption and chemical nature of the substance (specifically the lack of lipophilicity) indicate that substance quaternary ammonium compounds have no or little potential for bioaccumulation.

C. Acute Toxicity

The oral LD_{50} in rats is 50.1 milligrams per kilogram (mg/kg, HPVIS) [Kl. score = 2]. There are no acute inhalation or dermal toxicity studies on N-benzyl-alkylpyridium chloride.

D. Irritation

There are no studies available. However, N-benzyl-alkylpyridium chloride is considered corrosive based on its pH of 1.2 (ECHA).

E. Sensitisation

There are no studies available.

F. Repeated Dose Toxicity

Oral

There are no studies available.

Inhalation

There are no studies available.

Dermal

There are no studies available.

G. Genotoxicity

In vitro Studies

The *in vitro* genotoxicity studies on alkylpyridine are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on alkylpyridine

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (S. typhimurium strains)	-	-	1	ECHA
Mammalian cell gene mutation (mouse lymphoma L5178Y cells)	-	-	1	ECHA
Chromosomal aberrations (human lymphocytes)	-	-	1	ECHA

*+, positive; -, negative

In Vivo Studies

There are no studies available.

H. Carcinogenicity

Oral

There are no studies available.

Inhalation

There are no studies available.

Dermal

There are no studies available.

I. Reproductive Toxicity

There are no studies available.

J. Developmental Toxicity

Oral

There are no studies available.

Inhalation

There are no studies available.

Dermal

There are no studies available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

No data are available on N-benzyl-alkylpyridium chloride to derive oral toxicological reference and drinking water guidance values.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Alkylpyridine does not exhibit the following physico-chemical properties:

- Flammability
- Oxidising potential

The substance is classified as flammable (Flam. Liquid 3).

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

N-benzyl-alkylpyridium chloride exhibits significant acute and chronic aquatic toxicity. Sediment dwelling organisms are far less sensitive to the substance perhaps based on combined effects of biodegradation and binding to the sediment matrix.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on alkylpyridine.

Table 3: Acute Aquatic Toxicity Studies on Alkylpyridine

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Cyprinodon variegatus	96-hr LC_{50}	14.1	1	ECHA
Daphnia magna	48-hr EC_{50}	3.1	1	ECHA
Pseudokirchneriella subcapitata	72-hr EC_{50}	0.47	1	ECHA

Chronic Studies

There are no studies available.

C. Terrestrial Toxicity

There are no studies available.

D. Calculation of PNEC

The PNEC calculations for alkylpyridine follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three two trophic levels. Acute EC_{50} values are available for Cyprinodon variegatus (14.1 mg/L), Daphnia (3.1 milligrams per litre [mg/L]), and algae (0.47 mg/L). On the basis that the data consists of short-term results from three trophic levels, an assessment factor of 1,000 has been applied to the lowest reported E(L)C50 value of 0.47 mg/L for algae. The $PNEC_{water}$ is 0.00047 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 0.0073 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (20/1280) \times 1000 \times 0.00047$
$= 0.0073$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1000 \times BD_{solid}]$
$= 0.8 + [(0.2 \times 40/1000 \times 2400]$
$= 20\ m^3/m^3$

Where:
Kp_{sed} = solid-water partition coefficient (L/kg)
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]
$Kp_{sed} = K_{oc} \times f_{oc}$

= 1000 x 0.04
= 40 L/kg

Where:

K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for alkylpyridine is estimated to be 1000 L/kg.

F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.0063 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
= (20/1500) x 1000 x 0.00047
= 0.0063 mg/kg

Where:

Kp_{soil} = soil-water partition coefficient (m^3/m^3)

BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]

$Kp_{soil} = K_{oc} \times f_{oc}$
= 1000 x 0.02
= 20 m^3/m^3

Where:

K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for alkylpyridine was estimated to be 1000 L/kg.

F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (ADWG, 2011; DEWHA, 2009; ECHA, 2017).

N-benzyl alkylpyridium chloride is estimated to be ultimately biodegradable and thus does not meet the screening criteria for persistence.

No bioconcentration studies are available for N-benzyl alkylpyridium chloride. However. the measured log K_{ow} for N-benzyl alkylpyridium chloride is 3; thus, N-benzyl alkylpyridium chloride does not meet the screening criteria for bioaccumulation.

The acute EC_{50} values for alkylpyridine in algae is <1 mg/L. Thus, alkylpyridium meets the screening criteria for toxicity.

The overall conclusion is that alkylpyridium is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H226-Flammable Liquid 3

H314-Skin Corrosion 1B: Causes severe skin burns and eye damage

H318-Eye damage 1

H400-Aquatic Acute 1: Very toxic to aquatic life

H410- Aquatic Chronic 1

B. Labelling

Warning

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes. Consult physician.

Skin Contact

Wash thoroughly with soap and water. Consult physician.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, alcohol resistant foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The are no workplace exposure standards for alkylpyridine in Australia.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Alkylpyridine is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

ADWG. (2011). National Water Quality Management Strategy. Australian Drinking Water Guidelines, Section 6, Australian Government, National Health and Medical Research Council, Natural Resource Management Ministerial Council. Updated January 2022. Available: https://www.nhmrc.gov.au/about-us/publications/australian-drinking-water-guidelines

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AMMONIUM SULFATE

This dossier on ammonium sulfate (CAS RN 7783-20-2) presents the most critical studies pertinent to the risk assessment of the substance in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed ammonium sulfate in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment.

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): diazanium sulfate

CAS RN: 7783-20-2

Molecular formula: $H_8N_2O_4S$

Molecular weight: 132.14 g/mol

Synonyms: ammonium sulfate, diammonium sulfate, sulfuric acid diammonium salt, mascagnite

SMILES: [NH4+].[NH4+].[O-]S(=O)(=O)[O-]

II. PHYSICAL AND CHEMICAL PROPERTIES

Table 1: Physico-chemical Properties of Ammonium Sulfate

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	solid	2	ECHA
Melting Point	> 280°C (pressure not provided)	2	ECHA
Boiling Point	Not applicable as substance is solid	1	ECHA
Density	1770 kg/m^3 @ 25°C	2	ECHA
Vapour Pressure	0 Pa @ 25°C	2	ECHA
Partition Coefficient (log K_{ow})	-5.1 @ 25°C	2	ECHA
Water Solubility	767 g/L @ 25°C	2	ECHA
Flash Point	Not applicable as substance is solid	1	ECHA
Auto flammability	Not applicable as substance is solid	1	ECHA
Viscosity	Not applicable as substance is solid	1	ECHA

Property	Value	Klimisch Score	Reference
Dissociation constant (pKa)	9.25 @ 25°C	2	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Ammonium sulfate dissociates in aqueous media to the ammonium ion (NH_4^+) and sulfate anion (SO_4^{2-}). Ammonium sulfate is an inorganic ionic substance that is not expected to adsorb or bioaccumulate. Ammonium sulfate is hydrophilic, and it has high mobility in the soil.

B. Biodegradation

Given the fact the ammonium sulfate is an inorganic substance, biodegradation testing is not applicable.

C. Environmental Distribution

Ammonium sulfate is water soluble so it is mainly expected to partition to aqueous phase. Based on its log K_{ow}, it is not expected to adsorb substantially to the soil phase.

D. Bioaccumulation

No experimental data were available for bioaccumulation or bioconcentration of ammonium sulfate. Based on the high water solubility and the ionic nature, ammonium sulfate is not expected to adsorb or bioaccumulate to a significant extent. In addition, due to the log K_{ow} of –5.1 bioaccumulation is not expected (ECHA).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Ammonium sulfate exhibits low acute toxicity by the oral, inhalation and dermal routes. It is not irritating to the skin and eyes; and it is not a skin sensitiser. In repeated dose toxicity studies, dose-related changes were not observed in rats given ammonium sulfate in feed for 52-weeks. Ammonium sulfate is not genotoxic and is not carcinogenic. No reproductive or developmental effects were observed in read-across studies.

B. Acute Toxicity

<u>Oral</u>

In an OECD Guideline (401) study, Gassner rats were exposed to ammonium sulfate via oral gavage. The LD_{50} was determined to be 4,250 mg/kg bw/day in male and female rats (ECHA) [KI score = 2].

In an OECD Guideline (423 Acute Oral Toxicity) study Wistar rats were exposed to ammonium sulfate via oral gavage. The LD_{50} in rats was determined to be > 2000 mg/kg bw/day (ECHA) [KI score = 2].

Inhalation

In an OECD Guideline 433 (Acute Inhalation Toxicity: Fixed Concentration Procedure) study Sprague-Dawley rats were exposed to ammonium sulfate via nose only aerosol inhalation. The resulting LC_0 was determined to be 3.5 mg/m^3 after 4 hours of exposure (ECHA) [KI score = 2].

Dermal

In an OECD Guideline 434 (Acute Dermal Toxicity) study Wistar rats were exposed to ammonium sulfate via open coverage. The LD50 for this study was determined to be > 2000 mg/kg bw/day (ECHA) [KI score = 2].

C. Irritation

Skin

Vienna White rabbits were exposed to ammonium sulfate for up to 20 hours and they were observed for 8 days. There were no signs of clinical toxicity, so ammonium sulfate is not considered irritating to the skin (ECHA) [KI score = 2].

Eye

Ammonium sulfate was placed on the eyes of Vienna White rabbits without rinsing for 8 days. All of the observed effects were considered reversible, so this substance is not considered an eye irritant (ECHA) [KI score 2].

D. Sensitisation

A guinea pig maximisation test was used to determine if ammonium sulfate is a skin sensitiser. The animals did not show any signs of toxicity throughout the study period. [KI. score = 1]. Ammonium sulfate is not sensitising to the skin of guinea pigs (ECHA) [KI score = 1].

E. Repeated Dose Toxicity

Oral

In an OECD 453 (Combined Chronic Toxicity/Carcinogenicity) study Fischer 344 rats were continuously exposed to ammonium sulfate via their feed for 52weeks.

In the chronic study, groups of 10 rats/sex were fed a diet containing the test substance (purity not given) at concentrations of 0, 0.1, 0.6, or 3% for 1 year. These concentrations corresponded to average daily intakes of 0, 42, 256, and 1527 mg/kg bw/day for males and 0, 48, 284, and 1490 mg/kg bw/day for females, respectively.

No mortality was found in any groups throughout the treatment period. No test substance-related change in the body weights was found. Absolute and relative kidney weights were increased at the high dose level for both sexes. Absolute spleen weights were decreased and relative liver weights were increased in high dose males. No dose-related changes were found in the other organs.

The NOAEL for females was determined to be 284 mg/kg bw/day and the NOAEL for males was determined to be 256 mg/kg bw/day (ECHA) [KI score = 1].

Inhalation

Rats were exposed via whole body inhalation of ammonium sulfate for 8 hours a day over a 14-day treatment period. The NOEC was determined to be 300 mg/m^3 (ECHA) [KI score = 2].

Dermal

No data were available**.**

F. Genotoxicity

In vitro Studies

The results of the *in vitro* genotoxicity studies on ammonium sulfate based are presented in Table 2.

Table 2: *In Vitro* Genotoxicity Studies on Ammonium Sulfate

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
OECD Guideline 471 (Bacterial Reverse Mutation Assay) S. typhimurium TA 1535, TA 1537, TA 98 and TA 100	-	-	2	ECHA
OECD Guidline 476 (In vitro Mammalian Cell Gene Mutation Test) Chinese hamster lung fibroblasts (V79)	-	-	1	ECHA
OECD Guidline 473 (In vitro Mammalian Chromosome Aberration Test) human lymphocytes	-	-	2	ECHA

*+, positive; -, negative.

In vivo Studies

An *in vivo* mammalian somatic cell study also known as the cytogenicity/erythrocyte micronucleus cell test was conducted using ddY mice exposed to ammonium sulfate. The results showed that ammonium sulfate is not genotoxic to mice as there were no adverse effects observed (ECHA) [KI score = 2].

G. Carcinogenicity

Oral

A chronic oral toxicity and carcinogenicity study was conducted in rats, similar to the requirements of OECD TG 453. For investigation of the carcinogenic potential, groups of 50 rats/sex were fed a diet containing the test substance (purity not given) at concentrations of 0, 1.5, or 3% for 2 years. These concentrations corresponded to average daily intakes of 0,

564.1, and 1288.2 mg/kg bw/day for males and 0, 4649.9, and 1371.4 mg/kg bw/day for females respectively.

No macroscopic changes were recorded by gross pathology, except for massive nodular or focal lesions suggesting neoplastic changes. At histopathological examination, non-neoplastic and neoplastic lesions were noted in the control and treatment groups, with no significant inter-group difference in their incidences or severity.

The authors concluded that the no observed adverse effect level of ammonium sulfate was the 0.6% diet, which is equivalent to 256 and 284 mg/kg bw/d in males and females, respectively, and the compound is noncarcinogenic under the conditions of the study. There was no evidence of a long-term carcinogenic activity of the test substance.

Data on purity of the test substance are lacking; however, since no adverse effects were observed, this is not considered to affect the evaluation of the carcinogenic potential of ammonium sulfate in an adverse manner (ECHA) [Kl. Score = 1].

Inhalation

No studies are available.

Dermal

No studies are available.

H. Reproductive Toxicity

Oral

Read across of data for ammonium phosphate (7783-28-0) was conducted to screen for the reproductive and developmental toxicity effects of ammonium sulfate. A one generation reproductive toxicity study was conducted using Sprague Dawley rats exposed via oral gavage. The NOAEL for reproductive toxicity was determined to be 1500 mg/kg bw/day (ECHA) [KI score = 1].

I. Developmental Toxicity

An OECD Guideline 422 (Combined Repeated Dose Toxicity) study was conducted using Sprague Dawley rats exposed via oral gavage to a read across substance, ammonium phosphate (7783-28-0), for two weeks. A NOAEL could not be established for maternal toxicity based on inflammatory/degenerative stomach changes recorded during histopathological examination. The foetal NOAEL was determined to be 1,500 mg/kg bw/day (ECHA) [Kl. Score = 1].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for ammonium sulfate follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

The NOAEL from a rat 52-week oral feeding study was reported to be 256 mg/kg bw/day for males based on the actual dose received. This NOAEL will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1
Oral RfD = 256/(10 x 10 x 1 x 1 x 1) = 256/100 = 2.56 mg/kg/day.

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2021)
Proportion of water consumed = 10% (ADWG, 2021)
Volume of water consumed = 2L (ADWG, 2021)
Drinking water guidance value = (2.56 x 70 x 0.1)/2 = 8.96 mg/L

B. Cancer

Ammonium sulfate is not considered a carcinogen. Thus, a cancer reference value will not be calculated for this substance.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Ammonium does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Ammonium sulfate is of low acute concern to aquatic life. Algae is more tolerant than fish or invertebrates.

B. Aquatic Toxicity

In aqueous solution, ammonium salts are completely dissociated into NH_4^+ and a corresponding anion. This equilibrium depends on temperature, pH and ionic strength of the water in the environment. Un-ionized NH_3 species exists in the aquatic environments and the fraction (NH_3/(NH_3 +NH_4^+)) steeply increases with elevated pH value or temperature. It is well known that toxicity to aquatic organisms has been attributed to un-ionized ammonia (NH_3) species, and NH_4^+ species is considered to be non- or significantly less-toxic (Emerson et al., 1975 in ECHA). However, recent developments in assessing ammonia toxicity clearly show that in contrast to earlier assumptions where un-ionized ammonia was considered to be the toxic component, both the uncharged and charged molecule are toxic. Therefore, a joint toxicity model has been proposed, with ammonia causing most toxicity at high pH values and ammonium ion also contributing to toxicity at lower pH values (U.S. EPA 1999, OECD 2007 in ECHA).

It is generally accepted, that the principal toxic component of ammonium salts such as ammonium chloride or -sulphate is ammonia, rather than the corresponding anion (see also: OECD 2004, SIDS ammonium chloride or OECD 2007 ammonium sulphate). Therefore, toxicity values for ammonium salts (such as: ammonium -sulphates, phosphates, carbonates, chlorides or nitrates), where the major toxic component is ammonia, can be considered as equivalent, therefore read-across to those substances is possible. Consequently, the aquatic toxicity data compiled for ammonium sulfate comprises the total topic of ammonia toxicity. Species mean chronic values (SMCV) as described in ECHA were considered as relevant endpoints.

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on ammonium sulfate.

Table 3: Acute Aquatic Toxicity Studies on Ammonium Sulfate[1]

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Onchorhyncus mykiss, Salmo gairdneri	96-hour LC_{50} mortality	53	1	ECHA
Prosopium williamsoni	96-hour LC_{50}	57.2	1	ECHA
Ceriodaphnia acanthina	48-hour EC_{50} mobility	121.7	1	ECHA
Daphnia magna	48-hour EC_{50} mobility	169	1	ECHA

1 - Acute toxicity results were normalized to pH 8 and ammonium sulfate.

Chronic Studies

Chronic values were normalized to 25°C. As indicated, plants (algae) are more tolerant than fish or invertebrates to ammonia.

Fish: A 30-day study was conducted to determine the toxicity of ammonium sulfate to *Lepomis macrochirus.* The EC_{10} for ammonium sulfate was determined to be 5.29 mg/L (ECHA) [KI score 1].

Invertebrates: A 10-week study was conducted to determine the toxicity ammonium sulfate to *Hyallella azteca*. The EC_{10} for ammonium sulfate was determined to be 3.12 mg/L based on reproduction (ECHA) [KI score = 1].

Algae: An 18-day study was conducted to determine the toxicity of ammonium sulfate to *Chlorella vulgaris*. The EC_{50} value for ammonium sulfate was determined to be 2,700 mg/L (ECHA) [KI score = 2].

A 5-day study was conducted to determine the toxicity of ammonium sulfate to *Chlorella vulgaris*. The EC_{50} value for ammonium sulfate was determined to be 1,605 mg/L based on the growth rate (ECHA) [Kl. Score = 2].

C. Terrestrial Toxicity

No reliable studies available.

D. Calculation of PNEC

The PNEC calculations for ammonium sulfate follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute $E(L)C_{50}$ values are available for fish (53 mg/L) and invertebrates (121.7 mg/L). NOEC values from long-term studies are available for fish (5.29 mg/L), invertebrates (3.12 mg/L) and algae (1,605 mg/L). On the basis that the data consists of short-term results from two trophic levels and long-term results from three trophic levels, an assessment factor of 10 has been applied to the lowest reported EC_{10} value of 3.12 mg/L for invertebrates. Therefore, the $PNEC_{water}$ is 0.312 mg/L.

PNEC Sediment

No reliable experimental toxicity data on sediment organisms are available. Ammonium sulfate dissociates completely in water with its environmental distribution is dominated by its high water solubility. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as ammonium sulfate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{sediment}$. Based on its properties, no adsorption of ammonium sulfate to sediment is to be expected, and the assessment of this compartment will be covered by the aquatic assessment.

PNEC Soil

No reliable experimental toxicity data on terrestrial organisms are available. The environmental distribution of ammonium sulfate is dominated by its water solubility. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as ammonium sulfate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{soil}$. Based on its properties, ammonium sulfate is not expected to significantly adsorb to soil, and the assessment of this compartment will be covered by the aquatic assessment.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2008).

Ammonium sulfate is an inorganic salt that dissociates completely to ammonium and sulfate ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions. For the purposes of this PBT assessment, the persistent criteria are not considered applicable to ammonium sulfate or its dissociated ions.

The estimated log K_{ow} is for ammonium sulfate is equal to -5.1. This value suggests that ammonium sulfate is not expected to bioaccumulate (ECETOC, 2000). Therefore, ammonium sulfate does not meet the screening criterion for bioaccumulation.

The NOEC or EC10 values from chronic aquatic toxicity studies are > 0.1 mg/L. The acute $E(L)C_{50}$ values for fish and invertebrates are > 1 mg/L. Thus, ammonium sulfate does not meet the criteria for toxicity.

The overall conclusion is that ammonium sulfate is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Acute Toxicity: H302: Harmful if swallowed

Irritation: H315: Causes skin irritation

Eye: H318: Cause serious eye damage

STOT: H335: May cause respiratory irritation

B. Signal word

Danger

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. Get medical attention immediately, preferably a physician for an ophthalmologic examination.

Skin Contact

For minor skin contact, avoid spreading material on unaffected skin. Remove and isolate contaminated clothing. Wash the contaminated area of body with soap and fresh water. Get medical attention. Launder contaminated clothing before reuse.

Inhalation

Move person to fresh air. Administer oxygen if breathing is difficult. Do not use mouth-to-mouth method if victim inhaled the substance; give artificial respiration with the air of a pocket mask equipped with a one-way valve or other proper respiratory medical device. Give artificial respiration if victim is not breathing. Get medical attention immediately.

Ingestion

Do not induce vomiting. Get medical attention immediately.

Notes to Physician

All treatments should be based on observed signs and symptoms of distress in the patient.

B. Fire Fighting Information

Extinguishing Media

Use water spray or fog, foam, dry chemical or carbon dioxide.

Specific Exposure Hazards

Emits toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon dioxide, carbon monoxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Isolate area. Keep unnecessary and unprotected personnel from entering the area. Use personal protective clothing. Ensure adequate ventilation. Wear respiratory protection if ventilation is inadequate. Do not breath mist, vapours or spray. Avoid contact with skin, eyes and clothing. Eliminate all sources of ignition.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

Eliminate all sources of ignition. Pick up with suitable absorbent material and transfer to a container for chemical waste. For large amounts: dike spillage and pump off product into container for chemical waste. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep away from heat, sparks and flame. Avoid contact with eyes, skin and clothing. Avoid breathing vapour. Wash thoroughly after handling. Keep container closed. Use with adequate ventilation.

Storage

Keep container tightly closed. Store away from heat and light.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for ammonium sulfate.

Engineering Controls

Good general ventilation should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation or other engineering controls to maintain airborne levels below recommended exposure limits. If exposure limits have not been established, maintain airborne levels to an acceptable level.

Personal Protection Equipment

Respiratory Protection: If workers are exposed to concentrations above the exposure limit, they must use appropriate, certified respirators. If there are no applicable exposure limit requirements or guidelines, use an approved respirator. Selection of air-purifying or positive pressure supplied-air will depend on the specific operation and the potential airborne concentration of the product. For emergency conditions, use an approved positive-pressure self-contained breathing apparatus. The following should be effective types of air-purifying respirators: organic vapour cartridge with a particulate pre-filter.

Hand Protection: Use gloves chemically resistant to this material. Consult the SDS for appropriate glove barrier materials.

Skin Protection: Use protective clothing chemically resistant to the material. Selection of specific items such as face shield, boots, apron or full body suit will depend on the task.

Eye Protection: Use chemical goggles.

Other Precautions: Wash hands, forearms and face thoroughly after handling chemical products; before eating, smoking and using the lavatory; and at the end of the working period. Appropriate techniques should be used to remove potentially contaminated clothing. Wash contaminated clothing before reusing. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

UN number: 20506 (Solid). This UN number is for ammonium hydrogen sulfate.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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BORIC ACID (CAS No. 10043-35-3)
SODIUM TETRABORATE DECAHYDRATE (BORAX) (CAS No. 1303-96-4)

This dossier presents the most critical studies pertinent to the risk assessment of two boron compounds (boric acid and borax) in their use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): boric acid

CAS RN: 10043-35-3

Molecular formula: BH_3O_3

Molecular weight: 61.84 g/mol

Synonyms: orthoboric acid; boracic acid; borofax; boron hydroxide; boron trihydroxide

Chemical Name (IUPAC): disodium bicyclo[3.3.1]tetraboroxane-3,7-bis(olate)

CAS RN: 1303-96-4

Molecular formula: $B_4Na_2O_7$

Molecular weight: 381.4 g/mol

Synonyms: sodium tetraborate decahydrate; borax; monosodium metaborate; sodium borate; sodium borate (NaBO2); sodium diborate; sodium meta borate; sodium metaborate; sodium tetraborate

SMILES: B(O)(O)O

II. Physical AND Chemical Properties

Limited measured data are available for borax. In the environment, borax is expected to dissociate and/or hydrolyse to release boric acid at neutral pH. Therefore, measured data available for boric acid have been presented as analogue data for this substance.

Key physical and chemical properties for boric acid are shown in Table 1.

Table 1: Overview of the Physico-Chemical Properties of Boric Acid

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	White, odorless, crystalline solid	2	ECHA
Melting Point	>100°C (decomposes)	1	ECHA
Boiling Point	Not Applicable	-	ECHA
Density	1489 kg/m^3 @ 20°C	1	ECHA
Vapor Pressure	0 Pa @ 25°C	1	ECHA
Partition Coefficient (log K_{ow})	Not Applicable, substance is inorganic	-	ECHA
Water Solubility	48.8 g/L @ 20°C	1	ECHA
Dissociation Constant (pKa)	8.94 @ 20°C	1	ECHA

Boron is almost exclusively found in the environment in the form of boron-oxygen compounds, which are often referred to as borates. The high strength of the B-O bond relative to those between boron and other elements makes boron oxide compounds stable compared to nearly all non-oxide boron materials. Indeed, the B-O bond is among the strongest found in the chemistry of naturally occurring inorganic substances (ECHA).

In the environment, borates and compounds of boric acid will dissociate and/or hydrolyse to form the same boron species. For example, when borax dissolves in dilute solutions, it dissociates into Na^+ ions and the tetraborate anion ($B_4O_5(OH)_4^{2-}$). Boric acid ($B(OH)_3$) is formed following acid catalysed hydrolysis of the tetraborate anion. Under alkaline conditions, dilute solutions of the tetraborate anion depolymerise rapidly to the mononuclear borate anion ($B(OH)_4^-$) (NICNAS, 2019).

Boric acid is a Lewis acid that acts as a weak monoprotic acid by accepting OH^- and not as a proton donor (pKa 9.14). Therefore, at the near neutral pH of most environmental systems and at low concentrations (<0.025 mol B/L), the neutral mononuclear species ($B(OH)_3$) will dominate and only a small proportion of boron will exist as the borate monoanion, $B(OH)_4^-$. Therefore, boric acid is in equilibrium with borate anions in the environment. Both species are very stable as they do not undergo biotransformation or redox reactions under normal environmental conditions (NICNAS, 2019).

Exposure to borates are often expressed in terms of boron (B) equivalents based on the fraction of boron in the source substance on a molecular weight basis. The B equivalents used are a generic designation rather than a designation of the element boron. The factor for converting boric acid to B-equivalents is 0.1748. The factor for converting borax to B-equivalents is 0.2149.

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Borax will transform into boric acid in the aquatic environment. Boric acid is in equilibrium with borate anions in the environment. Degradation is not applicable to inorganic borates. Boric acid is

highly soluble in water. Some partitioning to soil and sediment does occur, however, this adsorption is pH dependent and has a low potential for bioaccumulation.

B. Partitioning

Borax will transform into boric acid in the aquatic environment. Boric acid is in equilibrium with borate anions in the environment. Both species are very stable as they do not undergo biotransformation or redox reactions under normal environmental conditions. Boric acid is highly water soluble, and it tends to remain in surface waters. Although some partitioning from water to soil and sediment does occur, the adsorption is pH dependent with the greatest adsorption occurring under alkaline conditions (pH 7.5 to 9.0) (NICNAS, 2019).

C. Biodegradation

Degradation is not applicable to inorganic borates. Inorganic borates are not subject to hydrolysis, photodegradation, or biodegradation (ECHA). They are subject to chemical transformation processes (adsorption, complexation, precipitation, fixation) once released into the environment (ECHA).

D. Environmental Distribution

The Kp value for boron compounds was calculated as the median of all measured Kp values from the Geochemical Mapping of Agricultural and Grazing Land Soil project ("GEMAS project): 2.19 L/kg dry weight (ECHA) [Kl. Score = 2]. The chemistry of boron in soils and aquatic systems is simplified by the absence of oxidation- reduction reactions or volatilisation. Redox processes can mobilise Fe oxides and Mn oxides, which may lead to a release of boron in aquatic systems. Generally, sediments are characterised with higher pH values than the soil matrix, which increases the boron sorption capacity (ECHA).

If released to soil, based on this low Kp value, low vapour pressure and high water solubility, boric acid and borax are considered relatively mobile in the environment, under certain conditions (ECHA).

E. Bioaccumulation

The WHO review of boron (1998) noted, "highly water soluble materials are unlikely to bioaccumulate to any significant degree and that borate species are all present essentially as un-dissociated and highly soluble boric acid at neutral pH". Bioconcentration factors (BCFs) of <0.1 to 10.5 L/kg have been reported from laboratory tests of fish and oysters (Hamilton and Wiedmeyer, 1990; Thompson et al., 1976).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Borax exhibits low acute toxicity by oral and dermal routes. Boric acid exhibits low acute toxicity by oral, dermal, and inhalation routes. Neither substance is a skin or eye irritant, nor a skin sensitiser. Borax will predominantly exist as un-dissociated boric acid in aqueous media at physiological pH. The developing foetus and the testes are the two most sensitive targets of boron toxicity in multiple species. The testicular effects include reduced organ weight and organ-to-body weight ratio, atrophy, degeneration of the spermatogenic epithelium, impaired spermatogenesis, reduced fertility, and sterility. The developmental effects from boron exposure include high prenatal mortality; reduced foetal body weight; and malformations and variations. Repeated inhalation exposure to read-across substance boron oxide resulted in slight irritation to the respiratory tract,

but no systemic toxicity. Boric acid was not genotoxic, and boric acid and borax were not carcinogenic to rodents.

A. Toxicokinetics

Boric acid is not metabolised in either animals or humans, owing to the high energy level required (523 kJ/mol) to break the B - O bond. Other inorganic borates convert to boric acid at physiological pH in the aqueous layer overlying the mucosal surfaces prior to absorption. Most of the simple inorganic borates exist predominantly as undissociated boric acid in dilute aqueous solution at physiological and environmental pH, leading to the conclusion that the main species in the plasma of mammals is un-dissociated boric acid. Since other borates dissociate to form boric acid in aqueous solutions, they too can be considered to exist as un-dissociated boric acid under the same conditions. Additional support for this derives from studies in which more than 90% of administered doses of inorganic borates are excreted in the urine as boric acid. Absorption of borates via the oral route is nearly 100%. For the inhalation route also 100 % absorption is assumed as worst-case scenario. Dermal absorption through intact skin is very low with a percent dose absorbed of 0.226 ± 0.125 in humans. Using the % dose absorbed plus standard deviation (SD) for boric acid, a dermal absorption for borates of 0.5% (rounded from 0.45%) can be assumed as a worse case estimate (ECHA).

In the blood boric acid is the main species present and is not further metabolised. Boric acid is distributed rapidly and evenly through the body, with concentrations in bone 2 to 3 times higher than in other tissues. Boric acid is excreted rapidly, with elimination half-lives of 1 hour in the mouse, 3 hours in the rat and <27.8 hours in humans and has low potential for accumulation. Boric acid is mainly excreted in the urine (ECHA).

B. Acute Toxicity

The oral LD_{50} of borax in rats is > 2,500 mg/kg (ECHA) [Kl. score = 1]. The oral LD_{50} of boric acid in rats is 3,450 mg/kg (ECHA) [Kl. score = 1].

There are no acute inhalation studies on borax. In a read-across study for borax, the 4-hour inhalation LC_{50} value for disodium tetraborate pentahydrate in rats is >2.04 mg/L (ECHA) [Kl. score = 1]. The 4-hour inhalation LC_{50} value for boric acid in rats is >2.01 mg/L. The mass median aerodynamic diameter (MMAD) was 2.8 µm (ECHA) [Kl. score = 1]. In another study, the 4-hour inhalation LC_{50} value for boric acid in rats was >2.03 mg/L (ECHA) [Kl. score = 1].

The dermal LD_{50} of borax in rabbits is >2,000 mg/kg (ECHA) [Kl. score = 2]. The dermal LD_{50} of boric acid in rabbits is >2,000 mg/kg (ECHA) [Kl. score = 1].

C. Irritation

Application of 0.5 g. of borax to the skin of rabbits for 4 hours under occlusive conditions was not irritating. The mean erythema and oedema scores were 0.00 (ECHA) [Kl. scores = 2]. Application of 0.5 g. of boric acid to the skin of rabbits for 24 hours under occlusive conditions was not irritating. The mean of the 24- and 72-hour scores were 0.13 for erythema and 0.00 for oedema (ECHA) [Kl. scores = 1].

Disodium tetraborates are eye irritants. Instillation of 0.08 mL of read-across substance disodium tetraborate pentahydrate into the eyes of rabbits was slightly irritating. The mean of 24-, 48-, and

72-hour scores were 0.22 for corneal opacity; 0.22 for iridial lesions; 2.8 for conjunctival redness; and 1.89 for chemosis. The effects were fully reversible (ECHA) [Kl. score = 1].

Boric acid induced mild conjunctivae redness and chemosis and minor effects on the iris. The effects were reversible within 7 days (ECHA). Instillation of 100 mg of boric acid into the eyes of rabbits was slightly irritating. The mean of 24-, 48-, and 72-hour scores were 0.00 for corneal opacity; 0.11 for iridial lesions; 0.94 for conjunctival redness; and 0.56 for chemosis (ECHA) [Kl. score = 1].

D. Sensitisation

There are no skin sensitisation studies on borax. Read-across substance disodium tetraborate pentahydrate was not a skin sensitiser to guinea pigs in a Buehler test (ECHA) [Kl. score = 1].

Boric acid was not a skin sensitiser to guinea pigs in a Buehler test (ECHA) [Kl. score = 1]. Sodium tetraborate pentahydrate was not a skin sensitiser to guinea pigs in a Buehler test (ECHA) [Kl. score = 1]. Sodium tetraborate decahydrate was not a skin sensitiser to guinea pigs in a Buehler test (ECHA) [Kl. score = 1].

E. Repeated Dose Toxicity

<u>Oral</u>

Male and female Sprague-Dawley (SD) rats were given boric acid in their feed at doses of 0, 52.5, 175, 525, 1,750 or 5,250 ppm B equivalents for 90 days. The average intake has been estimated to be approximately 0, 2.6, 8.8, 26, 87.5 or 262.5 mg B/kg-day, respectively (EPA, 2004). By week six, all of the animals in the highest dose died. Clinical signs in the top two dose levels were rapid respiration, inflamed eyes, swollen paws, and desquamated skin on the paws and tails. There was also reduced food consumption and body weight gain. The 1,750 ppm females showed reduced liver, spleen ovary, and adrenal weights; the 1,750 ppm males showed reduced liver, spleen, kidney, testes, and adrenal weights. The adrenals of 4 of the 1,750 ppm males showed minor increases in lipid content and size of the cells in the zona reticularis. Atrophied testis (complete atrophy of the spermatogenic epithelium and decreased in the size of the seminiferous tubules) was seen in all of the 1,750 ppm males. One 525 ppm male had partial testicular atrophy. The no observable adverse effects level (NOAEL) for this study is 175 ppm boron or 8.8 mg B/kg-day (Weir and Fisher, 1972). [Kl. score = 2]

Male and female SD rats were given in their diet borax at doses of 0, 52.5, 175, 525, 1,750 or 5,250 ppm B equivalents for 90 days. The average intake has been estimated to be approximately 0, 2.6, 8.8, 26, 87.5 or 262.5 mg B/kg-day, respectively (EPA, 2004). By week 6, all of the animals in the highest dose died. Clinical signs in the top two dose levels were rapid respiration, inflamed eyes, swollen paws, and desquamated skin on the paws and tails. There was also reduced food consumption and body weight gain. The 1,750 ppm females showed reduced liver, spleen and ovary weights; the 1,750 ppm males showed reduced liver, spleen, kidney, testes, and brain weights. The adrenals of the majority of the 1,750 ppm males and females showed slight to moderate increases in lipid content and size of the cells in the zona reticularis. Atrophied testis (complete atrophy of the spermatogenic epithelium and decreased in the size of the seminiferous tubules) was seen in all of the 1,750 ppm males. Four 525 ppm males had partial testicular atrophy. Spermatogenic arrest was found in one 525 ppm male. The NOAEL for this study is 175 ppm boron or 8.8 mg B/kg-day (Weir and Fisher, 1972). [Kl. score = 2]

Male and female B6CF1$_1$ mice were given in the diet 0, 1,200, 2,500, 5,000, 10,000 or 20,000 ppm boric acid for 13 weeks (control and highest dose group) or 16 weeks (remaining dose groups). These dietary levels correspond to approximately 0, 34, 70, 141, 281 and 563 mg B/kg-day for males, respectively; and 0, 47, 97, 194, 388 and 776 mg B/kg-day for females, respectively (EPA, 2004). There was mortality (8/10 males; 6/10, females) in the 20,000 ppm, as well as hyperkeratosis and acanthosis. One male also died in 10,000 ppm group. Degeneration or atrophy of the seminiferous tubules occurred in the ≥5,000 ppm males. Minimal to mild extramedullary haematopoiesis of the spleen was observed in all dose groups. The LOAEL for this study is 1,200 ppm, corresponding to 34 and 47 mg B/kg-day for males and females, respectively (NTP 1987). [Kl. score = 2]

Male and female SD rats were given in their diet 0, 117, 350 or 1,170 ppm boric acid for two years. The average intake has been estimated to be approximately 0, 5.9, 17.5 or 58.5 mg B/kg-day, respectively (EPA, 2004). The 1,170 ppm rats had decreased food consumption during the first 13 weeks of the study and suppressed growth throughout the study. Signs of toxicity in the 1,170 ppm animals included swelling and desquamation of the paws, scaly tails, inflammation of the eyelids, and bloody discharge from the eyes. All of the 1,170 ppm males had testicular atrophy at the 6-, 12- and 24-month time points. The seminiferous epithelium was atrophied, and the tubular size in the testes was decreased. There were significant decreases in the absolute and relative testes weights. Brain and relative thyroid weights were increased. The NOAEL for this study is 350 ppm B equivalents or 17.5 mg B/kg-day (Weir and Fisher, 1972). [Kl. score = 2]

Male and female B6C3F$_1$ mice were given up to 20,000 ppm boric acid in their feed for 13 weeks (NTP, 1987). Eight out of the ten males and six out of the ten females from the 20,000-ppm group died and one of the ten males from the 10,000-ppm group died before end of study. Symptoms included nervousness, hunched appearance, dehydration, foot lesions and scaly tails. Incidences of extra medullary haematopoiesis of the spleen observed of varying severity in all dose groups for both males and females and hyperkeratosis and/or acanthosis of the stomach observed at the highest dose only in both males and females. At doses > 5,000 ppm (142 mg B/kg bw for the male), degeneration or atrophy of the seminiferous tubules was observed. The NOAEL for this study is 34 mg B/kg-day (NTP, 1987). [Kl. score = 2]

Inhalation

Male and female rats were exposed by inhalation to 0, 77, 175, or 470 mg/m^3 boron oxide. The exposures were 6 hours/day, 5 days/week for 24, 12, and 10 weeks for the 77, 175, and 470 mg/m^3 concentrations groups, respectively. The mass median aerodynamic diameter (MMAD) were 2.5, 1.9, and 2.4 µm for the 77, 175, and 479 mg/m^3 concentrations groups, respectively. There was no evidence of systemic toxicity. Some of the 470 mg/m^3 had reddish exudate from the nose. As these animals were covered with dust, this effect may have been local irritation of the nose and from the animals scratching the nose. The NOAEL for systemic toxicity is 470 mg/m^3, the highest exposure concentration tested. The NOAEL for localised effects (irritation) is 175 mg/m^3 (ECHA). [Kl. score = 2]

Dermal

No studies are available.

F. Genotoxicity

In vitro Studies

There are no *in vitro* genotoxicity studies on borax. Table 2 presents the results of the *in vitro* genotoxicity studies on boric acid.

Table 2: *In vitro* Genotoxicity Studies on Boric Acid

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (*S. typhimurium* strains)	-	-	1	ECHA
Bacterial reverse mutation (*S. typhimurium* strains)	-	-	2	ECHA
Mammalian cell gene mutation (mouse lymphoma L5178Y cells)	-	-	1	ECHA
Chromosomal aberrations (Chinese hamster ovary cells)	-	-	1	ECHA
Chromosomal aberrations (Chinese hamster ovary cells)	-	-	1	ECHA
Chromosomal aberrations (human peripheral lymphocytes)	NS	+	2	ECHA
Unscheduled DNA synthesis (rat liver cells)	NA	-	1	ECHA

*+, positive; -, negative; NA, not applicable; NS, not specified.

In vivo Studies

No studies are available on borax.

Male and female Swiss Webster mice were given two daily doses of 0, 225, 450, 900, 1,800, or 3,500 mg/kg boric acid. The frequency of micronucleated polychromatic erythrocytes were not increased at any dose level (ECHA) [Kl. score = 1].

G. Carcinogenicity

Oral

Male and female SD rats were given disodium tetraborate decahydrate (borax) or boric acid in their diet at doses of 0, 117, 350, or 1,170 ppm as B equivalents (approximately 0, 5.9, 17.5, or 58.5 mg B/kg-day) for two years. There was no mention of tumours in the report. Nevertheless, NTP (1987) concluded that this study provided adequate data on the lack of carcinogenic effects of boric acid in rats (Weir and Fisher, 1972; EPA, 2004).

Male and female $B6C3F_1$ mice were given 0, 2,500, or 5,000 ppm boric acid in their diet for 103 weeks. The dietary levels are equivalent to 0, 446, or 1,150 mg/kg-day boric acid or 0, 78.1, or 201.3 mg B/kg-day. There was no evidence of carcinogenicity (NTP, 1987). [Kl. score = 2]

H. Reproductive Toxicity

A three-generation reproductive toxicity study was conducted in SD rats with boric acid. Male and female rats were fed a diet containing 0, 117, 350 or 1,170 ppm boron (approximately 0, 5.9, 17.5 or 58.5 mg B/kg-day, respectively). In the lower two dose groups, there were no treatment-related effects on reproduction. Litter size, progeny weights, fertility, live birth indices, lactation, appearance were similar to the controls. No gross abnormalities were noted in these two dose groups. The 1,170-ppm dose group were found to be sterile, and there were no litters from mating the treated females with control males. Lack of viable sperm was found in the atrophied testes of all 1,170 ppm males. Decreased ovulation was also seen in the majority of the ovaries of the 1,170 ppm females. The NOAEL for this study is 350 ppm boron or approximately 17.5 mg B/kg-day (Weir and Fisher, 1972). [Kl. score = 2]

A three-generation reproductive toxicity study was conducted in Sprague-Dawley rats with borax. Male and female rats were fed a diet containing 0, 117, 350 or 1,170 ppm boron (approximately 0, 5.9, 17.5 or 58.5 mg B/kg-day, respectively). In the lower two dose groups, there were no treatment-related effects on reproduction. Litter size, progeny weights, fertility, live birth indices, lactation, appearance were similar to the controls. No gross abnormalities were noted in these two dose groups. The 1,170-ppm dose group were found to be sterile, and there were no litters from mating the treated females with control males. Lack of viable sperm was found in the atrophied testes of all 1,170 ppm males. Decreased ovulation was also seen in the majority of the ovaries of the 1,170 ppm females. The NOAEL for this study is 350 ppm boron or approximately 17.5 mg B/kg-day (Weir and Fisher, 1972). [Kl. score = 2]

In a continuous breeding protocol, male and female CD-1 mice were given in their diet 0, 1,000, 4,500 or 9,000 ppm boric acid in their feed. The authors estimated that the average daily intakes were: 0, 26.6, 111, and 220 mg B/kg-day to males; and 0, 31.8, 152, 257 mg B/kg-day to females. Boric acid consumption did not differ among the groups. There were no litters in the 9,000 ppm breeding pairs. At 4,500 ppm, there was a successful first litter, after which there was a progressive decrease in fertility; only one pair produced a fourth and fifth litter. All fertility indices were affected in the 4,500-ppm group. A complete crossover mating trial was conducted using control mice and the 4,500-ppm mice. The results showed that the probable cause of the reduced fertility was a decrement in male fertility. A dose-related decrease in body, testicular and epididymal weights was observed in the 4,500 and 9,000 ppm F_0 males. Sperm count was significantly decreased in these two dose groups, and percent motile sperm was decreased in all dose groups. Testicular histopathology showed seminiferous tubular atrophy in the 9,000 ppm males and partial atrophy of the seminiferous tubules in the 4,500 ppm males. There were no histopathologic changes in the 4,500 ppm females. No statistically significant decreases in mating index, fertility index, or live pups/litter in the 4,500 ppm females, but the number of days to litter in this dose group was increased. Oestrous cyclicity was unaffected. Reproductive organ weights were unaffected, but relative maternal liver and kidney/adrenal weights were reduced. An F_1 fertility trial was performed using offspring from the 1,000-ppm groups. There was no decreases in mating, fertility or reproductive performance. The F_2 adjusted live pup weight was slightly, but significantly, reduced from controls. A clear NOAEL for reproductive toxicity in males was not seen in this study. The 1,000 ppm males had decreased sperm motility in the F_0 generation and decreased sperm concentration in the F_1 generation. Decreased F_2 pup relative body weight was statistically significant from controls. The NOAEL in this study for females is 1,000 ppm boric acid or 32 mg B/kg-day). The LOAEL in this study for males is 1,000 ppm or 27 mg B/kg-day; a NOAEL was not established (Fail et al. 1991). [Kl. score = 2]

I. Developmental Toxicity

No studies are available on borax.

Pregnant female SD rats were given 0, 0.1, 0.2 or 0.4% boric acid in their feed on gestational days (GD) 0 to 20 or 0.8% boric acid on GD 6 to 15. The average amounts of boric acid ingested were estimated to be 0, 78, 163, 330 or 539 mg/kg-day (0, 13.6, 28.5 or 57.7 mg B/kg-day), respectively. Effects on the pregnant rats were altered food and/or water intake at ≥0.2% boric acid, increased liver and kidney weights relative to body weights at ≥0.2%, reduced weight gain at ≥0.4%, and increased corrected weight gain at 0.4% boric acid. There was a reduction in foetal body weights in all treated groups (94, 87, 63, and 47% of control weight, respectively). Increased malformations occurred at ≥0.2%, and prenatal mortality was increased at 0.8%. There was a dose-response for altered skeletal morphology in rats (≥0.1%), and specific findings were significantly elevated above controls at ≥0.2%. Specifically, there was an increased incidence of short rib XIII (a malformation) and a decreased incidence or rudimentary or full rib(s) at lumbar I (an anatomical variation) (Heindel et al. 1992). [Kl. score = 2]

Pregnant female SD rats (dams) were given 0, 0.025, 0.005, 0.075, 0.1 or 0.2% boric acid in their feed on GD 0 to 20. Approximately half of the dams were terminated on GD 20, and the remaining dams delivered their litters. Pup growth and viability were monitored until postnatal day (PND) 21. The average amounts of boron ingested on GD 20 were 0, 3.3, 6.3, 9.6, 13.3, and 25 mg B/kg-day], respectively. The average amounts of boron ingested on PND 21 were : 0, 3.2, 6.5, 9.7, 12.9, and 25.3 mg B/kg-day, respectively. There were no maternal deaths and no treatment-related clinical signs. Maternal body weights were similar across all groups during gestation. However, decreased maternal body weights (GD 19 and 20 at sacrifice) and decreased maternal body weight gain (GD 15-18 and GD 0-20) were statistically significant in trend tests. There was a 10% reduction in gravid uterine weight (statistically significant) in the 0.2% group. Corrected maternal weight (maternal gestational weight minus reduced gravid uterine weight) was unaffected by treatment. Feed intake in the 1,000 ppm dams was minimally affected and only during the first three days of dosing. Water consumption was higher in the treated groups after GD 15. The number of corpora lutea and uterine implantation sites, and the percentage of preimplantation loss were similar across all groups. Increased relative kidney weights were increased in the 0.2% group. There were no differences in the viability of the offspring between treated and controls. On GD 20, foetal body weight was 94% and 88% of controls in the 0.1% and 0.2% groups, respectively; recovery was complete at birth (~GD 22). The incidence of short rib XIII was increased on GD 20 in the ≥0.1% groups, but only in the 0.2% group at PND 21. The incidence of wavy rib was increased on GD 20 in the ≥0.1% group; the reversibility of this effect was confirmed on PND 21. There was a slight decrease in extra lumbar ribs in the 0.2% group on GD 20, and extra lumbar ribs were seen in the 0.2% group on PND 21. The developmental NOAEL was considered to be 0.075% boric acid or 9.6 mg B/kg-day on GD 20; and 0.1% boric acid or 12.9 mg B/kg-day on PND 21 (Price et al. 1996a). [Kl. score = 1]

Pregnant Swiss mice were given in their diet 0, 0.1, 0.2 or 0.4% boric acid on GD 0 to 17. The average amounts of boric acid ingested were estimated to be 248, 452 or 1,003 mg/kg-day (0, 43.4, 79.0 or 175.3 mg/B/kg-day), respectively. Maternal toxicity consisted of mild kidney lesions (≥0.1%), increased water intake and relative kidney weights (0.4%), and decreased water intake during treatment. Foetal body weights were reduced in the ≥0.2% groups, and there were increased incidences of resorptions and malformed foetuses per litter in the 0.4% group. The LOAEL for maternal toxicity is 248 mg/kg-day boric acid or 43.4 mg B/kg-day; a NOAEL was not established. The NOAEL for developmental toxicity is 248 mg/kg-day boric acid or 43.4 mg B/kg-day (Heindel et al., 1992). [Kl. score = 2]

Pregnant female New Zealand rabbits were dosed by oral gavage with 0, 62.5, 125 or 250 mg/kg boric acid (0, 10.9, 21.9 or 43.7 mg B/kg) during GD 6-19. Feed intake was in the 250 mg/kg maternal animals during the exposure period, but it was increased in the ≥125 mg/kg dose groups. In the 250 mg/kg group, maternal body weights during GD 9-30, weight gain during GD 6-19, gravid uterine weight, and number of corpora lutea per dam were significantly reduced. In the ≥125 mg/kg groups, maternal corrected gestational weight gain was increased compared to controls. Maternal liver weights were unaffected by treatment. In the 250 mg/kg group, relative, but not absolute, kidney weights were increased, although no effects in the kidney were noted in the histopathological examination. Prenatal mortality was increased in the 250 mg/kg group (90% resorptions/litter versus 6% for controls); the proportion of pregnant females with no live foetuses was increased (73% versus 0%), and live litter size was reduced (2.3 foetuses versus 8.8). Thus, there were only 14 live foetuses (6 live litters) available for evaluation in the 250 mg/kg group. The percentage malformed foetuses/litter was increased in the 250 mg/kg group, primarily due to cardiovascular defects (72% versus 3% of controls). There was no definitive maternal or developmental toxicity in the 62.5 or 125 mg/kg dose groups. The NOAEL for maternal and developmental toxicity is 125 mg/kg-day boric acid or 21.9 mg B/kg-day (Price et al. 1996b). [Kl. score = 1]

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for boric acid follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

Non-Cancer

An oral reference dose was not derived for boric acid or borax.

The Australian drinking water guideline value for boron (4 mg/L) may be applicable (ADWG, 2011). The health-based ADWG value was based on a tolerable daily intake (TDI) of 0.16 mg/kg bw. This TDI is based on the NOAEL of 9.6 mg/kg bw/day for foetal bodyweight effects in a rat developmental study (Price et al. 1996a) with an uncertainty factor of 60 (10 for interspecies and 6 for human intraspecies).

Cancer

There was no evidence of carcinogenicity in rat and mouse chronic studies conducted on borax and/or boric acid. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Borax and boric acid do not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Borax and boric acid have low acute and chronic toxicity to aquatic organisms.

A. Aquatic Toxicity

In ecotoxicological tests for boron, the exposure concentrations are expressed as boron equivalents (i.e., mg B/L). This is because boric acid and borate salts will have the same boron speciation when dissolved in environmental matrices. Therefore, in the following sections toxicological values are given as mg B/L regardless of the form of boron that was tested

Acute Studies

Borax will transform into boric acid in the aquatic environment. Table 3 lists the results of acute aquatic toxicity studies conducted on boric acid.

Table 3: Acute Aquatic Toxicity Studies on Boric Acid

Test Species	Endpoint	Results (mg B/L)	Klimisch score	Reference
Fathead minnow	96-hr LC_{50}	79.7	2	ECHA
Legumia recta (Black sandshell mussel)	96-hr LC_{50}	147	2	ECHA
Hyalella azteca	96-hr LC_{50}	64	2	ECHA
Pseudokirchneriella subcapitata	72-hr EC_{50}	52.4	1	ECHA

Chronic Studies

Long-term effects (LC_{10}) on freshwater fish ranged from 3.5 to 47 mg B/L. Adequate long-term LC_{10} of 21.6 mg B/L was found for the freshwater fish *P. promelas* in a study according to EPA OPPTS 850.1400 (ECHA) [Kl. Score = 2].

Long-term effects (LC10/no observed effect concentration [NOEC]) on reproduction on freshwater vertebrates ranged from 6.6 to 32 mg B/L based on several well-accepted guideline studies (ECHA) [Kl. Scores =1 or 2].

Boric acid has been evaluated for its toxicity towards the freshwater alga *Pseudokirchneriella subcapitata* (formerly *Selenastrum capricornutum*) in an Alga growth inhibition test according to Organisation for Economic Cooperation and Development (OECD) 201 under GLP requirements. The exposure duration was 72 hours under static conditions. The NOEC growth rate determined from the study was 17.5 mg B/L (ECHA) [Kl. Score = 1].

The ANZG water quality guideline (2021) derived a very high reliability default guideline value (DGVs) for (dissolved) boron in freshwater from 22 chronic (long-term) toxicity data, comprising eight fish, two amphibians, three crustaceans, one bivalve, three macrophytes, one green microalga, three diatoms, and one blue–green alga. The summary of representative data used by ANZG to develop a water quality guideline for boron is presented in Table 4. These values are noted to be consistent with those reported in ECHA. Additional chronic aquatic toxicity data is found in the ANZG Technical Brief (2021).

Table 4: Chronic Aquatic Toxicity Studies on Boron[1]

Test Species	Endpoint	Results (mg B/L)
Danio rerio	34-day NOEC (Biomass)	1.8
Pimephales promelas	32-day NOEC (Mortality)	11
Daphnia magna	14-day NOEC (Reproduction)	2.4
Pseudokirchneriella subcapitata	4-day NOEC (Growth)	2.8

1 - The DGVs are based on toxicity data for boron as either boric acid, H_3BO_3 (CAS 10043-35-3), or borax, $Na_2B_4O_710H_2O$ (CAS 1303-96-4), in freshwater.

In the chronic toxicity dataset, fish sensitivity to boron ranged from the least sensitive species in the dataset (*Melanotaenia splendida*, LC10 102 mg/L) to the third most sensitive species in the dataset (*Danio rerio*, NOEC 1.8 mg/L). Of the crustaceans, *D. magna* was best represented in the literature, with 18 published NOEC values (ranging from 2.4 mg/L to 29 mg/L) for six different endpoints from six different publications. The final NOEC of 2.4 mg/L used in the DGV derivation was lower than that for *C. dubia* (NOEC 5.6 mg/L) and for the amphipod *H. azteca* (NOEC 6.6 mg/L).For *P. subcapitata*, there were three separate studies available with toxicity data for boron. The toxicity values from these studies ranged from a NOEC of 2.8 mg/L to a NEC of 27 mg/L, varying with endpoint, duration and test medium used. Boron was least toxic to *P. subcapitata* when tested in algal growth medium with added $NaHCO_3$, suggesting that carbonate addition may have influenced boron toxicity. Therefore, although NECs are preferred to NOECs or EC10s (Warne et al., 2018), in this instance, a reliable NOEC of 2.8 mg/L was the most sensitive toxicity value for *P. subcapitata* (ANZG, 2021).

B. Sediment Toxicity

Limited sediment toxicity data are available for boric acid and boron containing compounds in general (NICNAS, 2019).

Chronic toxicity values for the effects of boric acid on sediment-dwelling invertebrates have been obtained for a freshwater midge (*Chironomus riparius*, harlequin fly), a freshwater bivalve (*Lampsilis siliquoidea*, fatmucket clam), and the aquatic worm (*Lumbriculus variegatus,* California blackworm). The respective toxicity values for these species are as follows: 28 d NOEC = 37.8 mg B/kg; 21 d LC25 (survival) = 363.1 mg B/kg; and 28 d NOEC = 100.8 mg B/kg (NICNAS, 2019).

Due to the high water solubility of boron and its low partitioning to sediment, sediment toxicity testing for boron is particularly challenging as it is difficult to ensure that exposure is through the solid phase (i.e., sediment) and not from the aqueous boric acid in the overlying water (NICNAS, 2019).

C. Terrestrial Toxicity

Ecotoxicological tests with plants and soil invertebrates have recorded modest chronic toxicity values (NOECs/ECs) in the range of 15.3 to 84.0 and 5.2 to 315 mg total B/kg, respectively (ECHA, 2008). However, to predict the potential toxicity of boron to plants and soil organisms, measuring the total boron concentration may be unsuitable. Instead, potential toxicity is better predicted using boron concentrations in the soil solution (extractable boron) (Mertens, et al., 2011). In Australia, it is generally accepted that boron toxicity will pose a risk to terrestrial plants when soil concentrations exceed 15 mg/kg of extractable boron (NICNAS, 2019).

D. Calculation of PNEC

PNEC Water

The ANZG water quality guideline (2021) derived a very high reliability DGV for (dissolved) boron in freshwater. The DGVs for 99, 95, 90 and 80% species protection are 340 µg/L, 940 µg/L, 1,500 µg/L and 2,500 µg/L, respectively. The 95% species protection level for boron in freshwater (940 µg/L) is recommended for adoption in the assessment of slightly-to-moderately disturbed ecosystems. (ANZG, 2021).

PNEC Sediment

Limited sediment toxicity data are available for boric acid and boron containing compounds in general (NICNAS, 2019). Due to the high water solubility of boron and its low partitioning to sediment, sediment toxicity testing for boron is particularly challenging as it is difficult to ensure that exposure is through the solid phase (i.e., sediment) and not from the aqueous boric acid in the overlying water (NICNAS, 2019). K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as boric acid and borax. Therefore, the equilibrium partitioning method cannot be used to calculate the $PNEC_{sed}$. As a result, the assessment of this compartment will be covered by the aquatic assessment.

PNEC Soil

In the ECHA REACH database (ECHA), a $PNEC_{soil}$ was derived for boron using the species sensitivity distribution method and an assessment factor of 2. The $PNEC_{soil}$ was determined to be 5.7 mg/kg soil dry weight.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU Reach Criteria methodology (DEWHA, 2009; ECHA, 2017).

Borax is an inorganic compound that dissociates completely to boric acid and the borate anion in aqueous media. Biodegradation is not applicable to these inorganic compounds; both boric acid and borate are also ubiquitous and are present in most water, soil and sediment. For the purposes of this PBT assessment, the persistent criteria are not considered applicable.

A BCF of <0.1-10.5 L/kg has been reported for borates in fish and oysters. This data suggests that boric acid does not bioaccumulate in the aquatic environment. Thus, boric acid and borax do not meet the criteria for bioaccumulation.

The chronic toxicity data on boric acid has a NOEC > 0.1 mg/L. Acute $E(L)C_{50}$ values are > 1 mg/L. Thus, borax and boric acid do not meet the criteria for toxicity.

The overall conclusion is that borax and boric acid are not PBT substances.

IX. CLASSIFICATION AND LABELLING

A. Classification

Reproductive toxicity (Category 1B), H360

B. Labelling

Danger

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Ingestion

Rinse mouth with water and then drink plenty of water. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Not combustible. May emit hazardous vapours under fire conditions. Depending on conditions, decomposition products may include the following: borane/boron oxides.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing to prevent skin contact.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Avoid inhalation of dusts. Avoid substance contact. Ensure adequate ventilation.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilled

Soak up with inert absorbent material.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for boric acid.

Boron oxide (CAS No. 1303-86-2) has an exposure standard of 10 mg/m^3 time weighted average (TWA)

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is required when dusts are generated.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Boric acid is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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CINNAMALDEHYDE

This dossier on cinnamaldehyde presents the most critical studies pertinent to the risk assessment of cinnamaldehyde in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch *et al.*, 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): 3-phenylacryladehyde

CAS RN: 104-55-2

Molecular formula: C_9H_8O

Molecular weight: 132.16 g/mol

Synonyms: Cinnamaldehyde; (2E)-3-phenylprop-2-enal; 3-phenylacrylaldehyde; cinnamal; (E)-cinnamaldehyde; 3-phenylpropenal; cinnamic aldehyde; phenylacrolein; cinnamylaldehyde; 3-phenyl-2-propenal; trans-cinnamaldehyde; (E)-3-phenylpropenal; (E)-3-phenyl-2-propenal; 3-phenylacrolein; 3-phenyl-2-propenaldehyde; 3-phenyl-2-propen-1-al; acrolein, 3-phenyl-; 2-propenal, 3-phenyl-; 2-propenal, 3-phenyl-, (2E)-

SMILES: C1=CC=C(C=C1)C=CC=O

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-chemical Properties of Cinnamaldehyde

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Light colorless clear liquid	1	ECHA
Melting point	-18°C @ 96.990 kPa	1	ECHA
Boiling point	>250°C @ 96.990 kPa	1	ECHA
Density	1,041 kg/m^3 @ 20°C and 96.75 kPa	1	ECHA
Vapor pressure	3.853 Pa @ 25°C	2	ECHA
Partition coefficient (log K_{ow})	2.107±0.0017 @ 25°C	1	ECHA
Water solubility	2.865 g/L @ 25°C	1	ECHA
Flash point	105°C @ 96.83 kPa	1	ECHA
Auto flammability	Not auto-flammable	1	ECHA
Viscosity	22.12 mPa s @ 20°C 18 mPa s @ 40°C	1	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Cinnamaldehyde is expected to biodegrade and not expected to bioaccumulate to any significant extent. It has a low potential to adsorb to soil or sediment.

B. Biodegradation

Cinnamaldehyde is readily biodegradable. In an OECD 301B test, degradation of cinnamaldehyde was 89% after 7 days, 94% after 14 days, and 100% after 28 days, indicating ready biodegradation (ECHA) [Kl. score = 2]. In an OECD 301D test, biodegradation was 24.98% after 5 days. The BOD_5 value was 0.635 mg O_2/mg (ECHA) [Kl. score = 1].

If a chemical is found to be inherently biodegradable or readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

No experimental data are available for cinnamaldehyde. Using KOCWIN in EPISUITE™ (EPA, 2018), the estimated K_{oc} value from log K_{ow} of 2.107 is 55.82 L/kg. The estimated K_{oc} value from the molecular connectivity index (MCI) is 36.82 L/kg. Based on this estimated value, cinnamaldehyde is expected to have very high mobility in soil. If released to water, based on the K_{oc} value and its high water solubility, it is also not expected to adsorb to suspended solids and sediment.

D. Bioaccumulation

A bioaccumulation study in fish was conducted to estimate the bioconcentration factor (BCF) value for cinnamaldehyde. The BCF value was calculated using a log K_{ow} of 1.9 and a regression derived equation. The estimated BCF value for cinnamaldehyde was determined to be 8 which indicates that this chemical is non-bio accumulative in aquatic organisms (ECHA) [KI. score =2].

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Cinnamaldehyde is of relatively low acute toxicity by the oral, dermal, and inhalation routes of exposure. It is an irritant to skin and eyes and is considered a sensitizer per the guinea pig maximization test. Oral repeat dose studies suggest that cinnamaldehyde has relatively low toxicity. There are no studies on the inhalation routes of exposure. Dermal repeat studies suggest that cinnamaldehyde has low toxicity. Cinnamaldehyde was not mutagenic in *in vitro* and *in vivo* genotoxicity tests, and it is not carcinogenic. Cinnamaldehyde is a not a reproductive or developmental toxicant.

B. Metabolism

Male Fischer 344 rats were given doses of 5, 50, and 500 mg/kg bw/day of cinnamaldehyde by oral gavage for seven days. Cinnamaldehyde was rapidly absorbed within the body and distributed to the gastrointestinal tract, the kidneys, the liver, and a small amount distributed to fat. Benzoic acid is the major metabolic of cinnamaldehyde. After 24 hours more than 80% of cinnamaldehyde is excreted in the urine and a small amount (<7%) is excreted in the faeces (ECHA) [KI. score =2].

The metabolism of 2 and 250 mg/kg bw/day of cinnamaldehyde was evaluated using male and female CD-1 mice exposed via the intraperitoneal route of exposure for 72 hours. About 94% of the administered dose was recovered in the urine after 72 hours. Less than two percent of the administered dose was remained in the mice after 72 hours. The major urinary metabolites were hippuric acid, 3-hydroxy-3-phenylpropionic acid, benzoic acid, and benzyl glucuronide (ECHA) [KI. score = 2].

C. Acute Toxicity

The 14-day acute oral LD_{50} in male and female Osborne-Mendel rats administered 2220 mg/kg bw/day of cinnamaldehyde via oral gavage was determined to be 2,220 mg/kg bw/day (ECHA) [Kl. Score = 2].

An acute oral toxicity study was conducted using male and female guinea pigs given cinnamaldehyde by oral gavage. The LD_{50} was determined to be 3400 mg/kg bw/day (ECHA) [KI. score =2].

Inhalation

There are no acute inhalation studies are available for cinnamaldehyde. An acute inhalation LC_{50} was predicted for cinnamaldehyde using the QSAR toolbox. The 4-hour LC50 in male and female Wistar rats exposed to cinnamaldehyde was predicted to be 68.889 ppm (ECHA) [KI. score =2].

Dermal

An OECD Guideline (Acute Dermal Toxicity) study was conducted using male and female albino Wistar rats exposed to cinnamaldehyde using occlusive dressing for 14 days. The dermal LD_{50} was determined to be is >2,000 mg/kg bw/day (ECHA) [Kl. Score = 2].

D. Irritation

Skin

Application of 0.1 mL of cinnamaldehyde to the skin of New Zealand white rabbits for 4 hours under semi-occlusive conditions was considered slightly-to-moderate irritating. The primary dermal irritation index (PDII) for cinnamaldehyde after 24, 48, and 72 hours was determined to be 3.25. This data indicates that cinnamaldehyde was moderately severely irritating to the skin of New Zealand white rabbits(ECHA) [Kl. score = 2].

An OECD Guideline 439 (In Vitro Skin Irritation: Reconstructed Human Epidermis Test method) study was conducted using non-transformed keratinocytes in a human skin model. The man tissue viability for cinnamaldehyde, when compared to the control, was determined to be 4.1%. This data indicates that cinnamaldehyde is considered to be irritating to human skin (ECHA) [KI. score =1].

Cinnamaldehyde, at doses of 0.02, 0.1%, and 0.8% in ethanol, was applied to the skin (upper arm) of healthy humans over a six-week period Cinnamaldehyde was determined to be severely irritating to the skin based on results from a human patch test (ECHA)[KI. score =2].

Eye

Instillation of 0.1 mL cinnamaldehyde to the eyes of New Zealand rabbits for 24 hours was considering irritating. The mean of the 24-, 48-, and 72-hours scores were: 1.00 for corneal opacity,

0.00 for iridial lesions, 2.00 for conjunctival redness, and 1.22 for chemosis. All effects were resolved by Day 14 of the observation period (ECHA) [Kl. score = 1].

The ocular irritation potential of cinnamaldehyde was determined using an OECD 492 guideline (Reconstructed Human Cornea-like Epithelium RhCE test method for identifying chemicals not requiring classification and labelling for eye irritation or serious eye damage) study. The mean tissue viability of cinnamaldehyde was determined to be 4.1 %. Cinnamaldehyde was determined to be irritating to the human eye (ECHA) [KI. score =1].

Instillation of 8% of cinnamaldehyde to the human eye was determined to be irritating (ECHA)[KI. score =2].

E. Sensitisation

Cinnamaldehyde was considered a skin sensitizer when tested in a guinea pig maximization test (ECHA) [Kl. score = 2].

F. Repeated Dose Toxicity

Oral

Male and female F344 rats were given in their diet 0, 4,100, 8,200, 16,500, or 33,000 ppm cinnamaldehyde (microcapsulated) for three months in a study conducted by the National Toxicology Program. The average daily intake was 0, 275, 625, 1,300, and 4,000 mg/kg-day for males, and 0, 300, 570, 1,090, and 3,100 mg/kg bw/day-day for females. There was no mortality during the study. Mean body weights were reduced in the ≥16,500 ppm animals as a result of decreased feed consumption from unpalatability of the dosed feed. There was a non-significant increase in serum bile acid concentration at all dose levels suggesting an effect on the liver, but there were no corresponding histopathologic effects. An increase in lesions of the forestomach mucosa was seen in the ≥8,200 ppm animals and included squamous epithelial hyperplasia. There was also chronic active inflammation in the 33,000 ppm males and the ≥16,500 ppm females. The NOAEL was considered to be 4,100 ppm, which corresponds to 275 and 300 mg/kg bw/day in males and females, respectively (Hooth et al., 2004; as cited in ECHA) [Kl. score = 1].

Male and female rats were fed in their diet 0, 1,000, 2,100, or 4,100 ppm cinnamaldehyde for 12 weeks. The average daily intake was 0, 50, 100, or 200 mg/kg bw/day-day. There were no significantly differences between treated and control animals in urine sugar and albumin, blood haemoglobin levels, growth, food intake, or other physiological criteria. The NOAEL for this study is 4,100 ppm for males and females, which corresponds to 200 mg/kg bw/day (ECHA) [Kl. score = 2].

Male and female F344 rats were given in their diet 0, 1,000, 2,100, or 4,100 ppm cinnamaldehyde (microcapsulated) for two years in a study conducted by the National Toxicology Program. The average daily intake was 0, 50, 100, or 200 mg/kg bw/day. The survival of the 4,100 ppm males was greater than the controls. The mean body weights of the 4,100 ppm animals were generally less he controls throughout the study. Feed consumption of the ≥2,100 ppm males and the 4,100 ppm females was less than the controls at the beginning and end of the study. There were no non-neoplastic lesions that were considered to be treatment related. The NOAEL for this study is 4,100 ppm for males and females, which corresponds to 200 mg/kg bw/day (Hooth et al., 2004; as cited in ECHA) [Kl. score = 1].

Male and female B6C3F$_1$ mice were given in their diet 0, 1,000, 2,100, or 4,100 ppm cinnamaldehyde (microcapsulated) for two years in a study conducted by the National Toxicology Program. The average daily intake was 0, 125, 270, or 540 (males) and 570 (females) mg/kg bw/day. Mean body weights of the ≥2,100 ppm animals were generally less than the controls throughout the study. There were no non-neoplastic lesions that were considered to be treatment related. Incidences of minimal olfactory epithelial pigmentation was significantly increased in the 4,100 ppm males and the ≥2,100 ppm females. The NOAEL for this study is 1,000 ppm in males and females, which corresponds to 125 mg/kg bw/day, based on reduced body weights at 270 mg/kg bw/day (Hooth et al., 2004; as cited in ECHA) [Kl. score = 1].

An oral subacute toxicity was conducted using male and female B6C3F1 mice exposed 0,656, 1310,2620, 5250, or 10,500 mg/kg bw/day cinnamaldehyde for 14 days (2 weeks: 5 days/week for a total of 12 doses). There were no significant differences in body weight, liver weight, spleen weight, and kidney weight. There were no statistical differences in organ: body weight ratios between surviving treated mice and the control mice. All of the mice in the two highest dose groups, as well as the all the female mice and three male mice from the 2620 mg/kg bw/day dose group, died withing the first two days of dosing. There were no clinical signs or gross lesions observed in the surviving mice or the dead mice. Mild forestomach hyperplasia was observed in both sexes of mice exposed to cinnamaldehyde. Minimal kidney nephropathy was observed in the mice exposed to dose of more than 1310 mg/kg bw/day. A NOAEL of 656 mg/kg bw/day was established for this study. A LOAEL of 1,310 mg/kg bw/day was established in this study based on body weight, organ weight, and histopathological examinations (ECHA) [KI. score = 2].

Inhalation

There are no studies are available. As shown in Table 1, cinnamaldehyde has a low vapor pressure which suggests that the generation of inhalable vapours is low. Under normal conditions, human exposure to cinnamaldehyde by the inhalation route of exposure is highly unlikely.

Dermal

A dermal sub chronic dermal toxicity study was conducted using female Balb/c mice exposed to 25µl 25 percent (v/v) solution of cinnamaldehyde for 4-5 days. The NOAEL was determined to be 25µl (ECHA) [KI. score =2].

A dermal sub chronic dermal toxicity study was conducted using mice exposed to 750 mg/kg bw/day 3D (intermittent) of cinnamaldehyde. A LOAEL value of 750 mg/kg/3D was established for mice exposed to cinnamaldehyde for three days (ECHA) [KI. score =2].

G. Genotoxicity

In Vitro Studies

The *in vitro* genotoxicity studies on cinnamaldehyde are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on cinnamaldehyde

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
OECD Guideline 471 Bacterial Reverse Mutation Assay (S. typhimurium TA 98, TA100, TA 102, TA 1535, TA1537)	-	-	1	ECHA

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
OECD Guideline 471 (*In Vitro* Mammalian Chromosome Aberration Test)	-	-	1	ECHA
OECD Guideline 476 (*In Vitro* Mammalian Cell Gene Mutation Test using the Hprt and xprt genes)	-	-	1	ECHA
Bacterial reverse mutation assay (Salmonella typhimurium TA97, TA98, TA100, TA1335, and TA1537)	-	-	2	ECHA
In vitro mammalian cell micronucleus test	-	-	2	ECHA

*+, positive; -, negative

In Vivo Studies

Male and female B6C3F$_1$ mice were administered in their feed 0, 4,100, 8,200, 16,500, or 33,000 ppm cinnamaldehyde (microcapsulated) for three months in a study conducted by the National Toxicology Program. The average daily intake was 650, 1,320, 2,550, and 5,475 mg/kg bw/day for males, and 0, 625, 1,380, 2,680, and 5,200 mg/kg bw/day for females. There were no increases in the frequency of micronucleated normochromatic erythrocytes in the peripheral blood in the treated animals compared to the controls (ECHA) [Kl. score = 2].

A mouse bone marrow micronucleus test was used to evaluate the genotoxic potential of cinnamaldehyde in ddY mice. Male mice were given oral doses of 0, 250, 313, and 500 mg/kg of cinnamaldehyde for 24 hours. Cinnamaldehyde did not induce any gene mutations in male ddY mice (ECHA) [Kl. score = 2].

H. Carcinogenicity

Male and female F344 rats were administered in their diet 0, 1,000, 2,100, or 4,100 ppm cinnamaldehyde (microcapsulated) for two years in a study conducted by the National Toxicology Program. The average daily intake was 0, 50, 100, or 200 mg/kg bw/day-day. The tumour incidences were similar between the treated and control animals. A NOAEL of 200 mg/kg bw/day (4100 ppm) was reported for this study (Hooth et al., 2004; as cited in ECHA) [Kl. score = 2].

Male and female B6C3F$_1$ mice were administered in their diet 0, 1,000, 2,100, or 4,100 ppm cinnamaldehyde (microcapsulated) for two years in a study by the National Toxicology Program. The average daily intake was 0, 125, 270, or 540 (males) and 570 (females) mg/kg bw/day-day. The tumour incidences were similar between the treated and control animals. The NOAEL was considered to be 4100 ppm (540 mg/kg bw/day for males and 570 mg/kg bw/day females (Hooth et al., 2004; as cited in ECHA) [Kl. score = 1].

An OECD Guideline 451 (Carcinogenicity study) was conducted in male and female Fischer 344 rats exposed to 0, 235, 470, 940, 1880, 3750 mg/kg bw/day of cinnamaldehyde by oral gavage for 16 days. There were no effects observed at the lowest dose level while all the animals in the two highest dose groups died within the first seven days of dosing. There was minimal to moderate forestomach hyperplasia observed in the males who received a dose of ≥470 mg/kg bw/day. A NOAEL of 235 mg/kg bw/day was reported in this study based on no occurrence of hyperplastic lesions or forestomach hyperplasia. There was clear evidence of distended gastrointestinal tracts in

animals who were given doses of 1880 or 3750 mg/kg bw/day as well as slightly decreased body weights in females of the 940 mg/kg bw/day dose group. The target organ toxicity value was reported to be 470 mg/kg bw/day (ECHA) [KI. score = 2].

I. Reproductive Toxicity

There are no adequate studies are available.

J. Developmental Toxicity

Pregnant female CD-1 mice were dosed by oral gavage with 0 or 1,200 mg/kg bw/day cinnamaldehyde on gestational days 6 to 13. The dams were allowed to deliver, and the pups were weaned up to postnatal day 3. There was no effect on maternal survival or body weight development and all 34 litters were viable. The number of liveborn per litter, the survival and birthweight of pups and their weight gain was not affected by treatment. The LOAEL for maternal and developmental toxicity is 1,200 mg/kg-day (ECHA) [Kl. score = 2].

An OECD Guideline 414 (Prenatal Developmental Toxicity) study was conducted in Wistar rats exposed to 0, 125, 250, 500 mg/kg bw/day of cinnamaldehyde by oral gavage from gestation day five to gestation day 19. The NOAEL for maternal systemic toxicity was reported to be 250 mg/kg bw/day. This effect level was based on mortality, clinical signs of toxicity, statistically/biologically significant decreased in body weight on gestation day 17 and gestation day 20. There were significant decreased in food intake on gestation day 8 and 11 and several gross/histopathology findings. The NOAEL for developmental toxicity was reported to be 250 mg/kg bw/day based on decreased fetal body weights observed in the 500 mg/kg bw/day (ECHA) [KI. score =1].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for cinnamaldehyde follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

<u>Oral</u>

A two-year oral repeat dose study was conducted by the national toxicology program in male and female F344 rats. The lowest NOAEL from this study was reported to be 4,100 ppm which corresponds to a dose level 200 mg/kg bw/day.
The NOAEL of 200 mg/kg bw/day will be used for determining the oral Reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

- UF_A (interspecies variability) = 10
- UF_H (intraspecies variability) = 10
- UF_L (LOAEL to NOAEL) = 1
- UF_{Sub} (subchronic to chronic) = 1
- UF_D (database uncertainty) = 1

Oral RfD = 200/ (10 x 10 x 1 x 1 x 1) = 200/100 = 2 mg/kg bw/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,
Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:
Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)

Drinking water guidance value = (2x 70 x 0.1)/2 = 7 mg/L

B. Cancer

Cinnamaldehyde was not carcinogenic to rats or mice when given in the diet for two years. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Cinnamaldehyde does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidizing potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Cinnamaldehyde has low chronic toxicity potential to aquatic organisms. Since cinnamaldehyde is readily biodegradable in water, it was reported to be non-toxic to aquatic fish, invertebrates, and algae at environmentally relevant concentrations.

B. Aquatic Toxicity

Acute Studies

Table 2 lists the results of acute aquatic toxicity studies on cinnamaldehyde.

Table 2: Acute Aquatic Toxicity Studies on Cinnamaldehyde

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Danio rerio (Brachydanio rerio)	96-hr LC_{50}	4.3 (mortality)	1	ECHA
Danio rerio (Brachydanio rerio)	96-hr LC_{50}	2.35 (mortality)	1	ECHA

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Danio rerio (Brachydanio rerio)	96-hr LC_{50}	>3.9- <5.5 (mortality)	1	ECHA
Poecilia reticulata (Guppy fish)	96-hr LC_{50}	>3.5- <6.5	2	ECHA
Lepomis macrochirus (Bluegill fish)	96-hr LC_{50}	>20	2	ECHA
Daphnia magna	48-hr EC_{50}	3.21	2	ECHA
Daphnia magna	48-hr EC_{50}	3.86	2	ECHA
Daphnia magna	48-hr EC_{50}	11.5	2	ECHA
Desmodesmus subspicatus	72-hr EC_{50}	31.6	2	ECHA
Chlorella vulgaris	72-hr EC_{50}	16.09	2	ECHA

Since the test chemical is readily biodegradable in water, the chemical was considered to be non-toxic to aquatic fish, invertebrates and algae at environmentally relevant concentrations (ECHA).

Chronic Studies

In an OECD Guideline 211 (Daphnia magna reproduction test) study, the 21-day EC_{50} was reported to be 0.402 mg/L based on reproduction (ECHA) [KI. score =2].

Based on a prediction completed using ECOSAR version 1.11, a long-term toxicity value for fish was predicted for cinnamaldehyde. Based on effects observed in a flow through freshwater system in fish, the NOEC value for the substance was estimated to be 15.159 mg/L for fish for 28 days of exposure duration. (ECHA) [KI. score = 2].

C. Terrestrial Toxicity

In a short-term toxicity study to birds (avoidance [repellency] test), the 5-day LOEL value was 1% w/w for *Colinus virginianus* (Northern Bobwhite Quail). (ECHA) [KI. score = 2].

D. Calculation of PNEC

The PNEC calculations for cinnamaldehyde follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute $E(L)C_{50}$ values are available for fish (2.35 mg/L), *Daphnia* (3.21 mg/L), and algae (16.09 mg/L). Results from a chronic study in fish was reported to be 15.159 mg/L. On the basis that the data consists of short-term results from three trophic levels and chronic studies on one trophic levels, an assessment factor of 100 has been applied to the lowest reported NOEC of 15.159 mg/L for fish. The $PNEC_{water}$ is 0.152 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 0.179 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (1.51/1280) \times 1000 \times 0.152$
$= 0.179$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1000 \times BD_{solid}]$
$= 0.8 + [(0.2 \times 1.47/1000 \times 2400]$
$=1.51\ m^3/m^3$

Where:
Kp_{sed} = solid-water partition coefficient (L/kg)
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]
$Kp_{sed} = K_{oc} \times f_{oc}$
$= 36.82 \times 0.04$
$=1.47$ L/kg

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for Cinnamaldehyde based on the molecular connectivity index (MCI) is 36.82 L/kg (EPA, 2019).
F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.075 mg/kg soil dry weight.
The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
$= (0.74/1500) \times 1000 \times 0.152$
$= 0.075$

Where:
Kp_{soil} = soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]
$Kp_{soil} = K_{oc} \times f_{oc}$
$= 36.82 \times 0.02$
$= 0.74$

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for cinnamaldehyde based on the molecular connectivity index (MCI) is 36.82 L/kg (EPA, 2019).
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU Reach Criteria methodology (DEWHA, 2009; ECHA, 2017).

Cinnamaldehyde is readily biodegradable; thus, it does not meet the screening criteria for persistence.

Based on a measured log K_{ow} of 2.107± 0.0017, cinnamaldehyde does not meet the screening criteria for bioaccumulation.

The NOEC from a chronic fish study was>0.1 mg/L. The acute E(L)C_{50} values for cinnamaldehyde are >1 mg/L. Thus, cinnamaldehyde does not meet the criteria for toxicity.

The overall conclusion is that cinnamaldehyde is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H315-Skin Irritant Category 2
H319-Eye Irritant Category 2
H317-Skin Sensitizer Category 1
H312-Aquatic Acute Toxicity Category 2
H335-STOT SE3

B. Labelling

Warning!

According to the classification provided by companies to ECHA in REACH registrations this substance causes serious eye irritation, is harmful to aquatic life with long lasting effects, is harmful in contact with skin, causes skin irritation and may cause an allergic skin reaction.

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

First check the victim for contact lenses and remove if present. Flush victim's eyes with water or normal saline solution for 20 to 30 minutes while simultaneously calling a hospital or poison control centre. Do not put any ointments, oils, or medication in the victim's eyes without specific instructions from a physician. IMMEDIATELY transport the victim after flushing eyes to a hospital even if no symptoms (such as redness or irritation) develop. SKIN: IMMEDIATELY flood affected skin with water while removing and isolating all contaminated clothing. Gently wash all affected skin

areas thoroughly with soap and water. If symptoms such as redness or irritation develop, IMMEDIATELY call a physician and be prepared to transport the victim to a hospital for treatment.

Skin Contact

IMMEDIATELY flood affected skin with water while removing and isolating all contaminated clothing. Gently wash all affected skin areas thoroughly with soap and water. If symptoms such as redness or irritation develop, IMMEDIATELY call a physician and be prepared to transport the victim to a hospital for treatment.

Inhalation

IMMEDIATELY leave the contaminated area; take deep breaths of fresh air. If symptoms (such as wheezing, coughing, shortness of breath, or burning in the mouth, throat, or chest) develop, call a physician and be prepared to transport the victim to a hospital. Provide proper respiratory protection to rescuers entering an unknown atmosphere. Whenever possible, Self-Contained Breathing Apparatus (SCBA) should be used; if not available, use a level of protection greater than or equal to that advised under Protective Clothing.

Ingestion

DO NOT INDUCE VOMITING. If the victim is conscious and not convulsing, give 1 or 2 glasses of water to dilute the chemical and IMMEDIATELY call a hospital or poison control centre. Be prepared to transport the victim to a hospital if advised by a physician. If the victim is convulsing or unconscious, do not give anything by mouth, ensure that the victim's airway is open and lay the victim on his/her side with the head lower than the body. DO NOT INDUCE VOMITING. IMMEDIATELY transport the victim to a hospital. (NTP, 1992)

Notes to Physician

Symptoms of exposure to this compound may include inflammation and erosion of gastrointestinal mucosa. The vapor or mist causes irritation of the eyes, mucous membranes and upper respiratory tract. ACUTE/CHRONIC HAZARDS: This chemical may be harmful by inhalation, ingestion or skin absorption. It may cause irritation of the skin, eyes, upper respiratory tract, and mucous membranes. When heated to decomposition it may emit toxic fumes of carbon monoxide and carbon dioxide.

Medical Conditions Aggravated by Exposure

Irritation properties of the substance may aggravate asthma and/or other respiratory conditions.

Emergency Personnel Protection

Personal protective equipment must be used in accordance with known hazards of the substance.

B. Fire Fighting Information

Extinguishing Media

This chemical is combustible. Fires involving this material can be controlled with a dry chemical, carbon dioxide or Halon extinguisher.

Specific Exposure Hazards

May ignite after a delay period in contact with NaOH.

Special Protective Equipment for Firefighters

Use respiratory protection equipment as deemed necessary by hazards associated with the substance.

C. Accidental Release Measures

Personal Precautions

Wash hands before eating, drinking, chewing gum, using tobacco or using the toilet. Remove clothing immediately if substance gets inside. Then wash thoroughly and put on clean clothing.

Environmental Precautions

Do not release to discharge into open drains or waterways.

Steps to be Taken if Material is Released or Spilled

If you spill this chemical, FIRST REMOVE ALL SOURCES OF IGNITION. Then, use absorbent paper to pick up all liquid spill material. Contaminated clothing and absorbent paper should be sealed in a vapor-tight plastic bag for eventual disposal. Solvent wash all contaminated surfaces with 60-70% ethanol followed by washing with a soap and water solution. Do not re-enter the contaminated area until the Safety Officer (or other responsible person) has verified that the area has been properly cleaned.

Wastewater from contaminant suppression, cleaning of protective clothing/equipment, or contaminated sites should be contained and evaluated for subject chemical or decomposition product concentrations. Concentrations shall be lower than applicable environmental discharge or disposal criteria. Alternatively, pre-treatment and/or discharge to a POTW is acceptable only after review by the governing authority. Due consideration shall be given to remediation worker exposure (inhalation, dermal and ingestion) as well as fate during treatment, transfer and disposal.

Do not contaminate water by cleaning of equipment or disposal of wastes

D. Storage and Handling

General Handling

Do not use, pour, spill or store near heat or open flame.

Other Handling Precautions

Observe label precautions. Immediately change contaminated clothing. Apply preventive skin protection. Wash hands and face after working with substance.

Storage

STORAGE PRECAUTIONS: You should keep this material in a tightly closed container under an inert atmosphere and store it at refrigerated temperatures. (NTP, 1992)

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure limit for cinnamaldehyde.

Engineering Controls

Good general ventilation should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation, or other engineering controls to maintain airborne levels below recommended exposure limits. If exposure limits have not been established, maintain airborne levels to an acceptable level.

Personal Protection Equipment

Respiratory Protection:
Where the neat test chemical is weighed and diluted, wear a NIOSH-approved half face respirator equipped with an organic vapor/acid gas cartridge (specific for organic vapors, HCl, acid gas and SO2) with a dust/mist filter. (NTP, 1992)

Hand Protection:
Chemical resistant gloves.

Skin Protection:
For agricultural use requirements, PPE required for early entry to treated areas that is permitted under applicable Worker Protection Standards and that involves contact with anything that has been treated, such as plants, soil, water, is: Coveralls, waterproof gloves, shoes plus socks.

Eye protection:
Protective eyewear shall be worn at all times.

Other Precautions:
None other specific precautions are stipulated.

F. Transport Information

Cinnamaldehyde is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

UN 1993

Class: 3

Packaging Group: II

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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CITRIC ACID

This dossier on citric acid presents the most critical studies pertinent to the risk assessment of citric acid in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The information presented in this dossier was obtained primarily from the OECD-SIDS documents on citric acid (OECD, 2001a, b) and the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed citric acid in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment[1].

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): 2-hydroxypropane-1,2,3-tricarboxylic acid

CAS RN: 77-92-9

Molecular formula: $C_6H_8O_7$

Molecular weight: 192.122 g/mol

Synonyms: citric acid; 1,2,3-propanetricarboxylic acid, 2-hydroxy-; 2-hydroxy-1,2,3-propanetricarboxylic acid

SMILES: C(C(=O)O)C(CC(=O)O)(C(=O)O)O

Citric acid is a ubiquitous natural substance that is an intermediate in the basic physiological tricarboxylic acid (TCA) cycle in every eukaryote cell.

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-Chemical Properties of Citric Acid

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	White crystalline odorless solid	2	ECHA
Melting Point	153°C @ 101.3 kPa	2	ECHA
Boiling Point	Not available due to substance decomposition	2	ECHA
Density	1670 kg/m^3@ 20°C (relative density)	2	ECHA
Vapor Pressure	2.21 x 10^{-6} Pa @ 25°C	2	ECHA
Partition Coefficient (log K_{ow})	-1.5 to -1.8 (temperature not indicated)	2	ECHA

[1] https://www.industrialchemicals.gov.au/chemical-information/search-assessments?assessmentcasnumber=77-92-9+

Property	Value	Klimisch Score	Reference
Water Solubility	592 g/L @ 20 °C (very soluble)	2	ECHA
Flash Point	345°C @ 101.3 kPa	4	ECHA
Flammability	Not flammable	2	ECHA
Auto flammability	1010°C	4	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Citric acid is readily biodegradable. It is not expected to bioaccumulate. Due to its high-water solubility, citric acid is unlikely to adsorb to soil or sediment.

B. Biodegradation

Citric acid can be considered readily biodegradable based on the results of the ready and inherent aerobic biodegradation studies listed in Table 2.

If a chemical is found to be readily biodegradable, it is categorized as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

Table 2: Biodegradation Studies on Citric Acid (OECD 2001a, b)

Test System	Results*	Notes	Klimisch Score
Modified Sturm	97% (CO_2 evolution); 100% (DOC removal)	Readily biodegradable; exposure period not stated	2
Closed Bottle Test	BOD_{30}/COD Ratio = 90%	Readily biodegradable	2
BOD_5/COD Ratio	BOD_5 = 526 mg; COD = 728 mg; BOD_5/COD Ratio = 0.72	Readily biodegradable; concentration of test substance and activated sludge not stated	2
BOD_1/ThOD Ratio	BOD_1/ThOD Ratio = 13%	-	2
BOD_{20}/ThOD Ratio	BOD_{20}/COD Ratio = 98%	Readily biodegradable; initial test substance concentration 720 mg/L	2
Zahn-Wallen Test	85%, 1 day (DOC removal)	Inherently biodegradable	2
Zahn-Wallen Test	98%, 7 days (DOC removal)	Inherently biodegradable	
Coupled Units Test	93% (COD removal)	Ultimately biodegradable; exposure period not stated.	2

C. Environmental Distribution

No experimental data are available for citric acid. Using KOCWIN program in EPISuite™ (EPA, 2016), the estimated K_{oc} value from the K_{ow} value of -1.08 is 0.3617 L/kg.

Based on this K_{oc} value, citric acid is not expected to adsorb to soil if released and has a high mobility. If citric acid is released to water, it is not expected to adsorb to suspended soils or sediment based on its K_{oc} value and rapid hydrolysis.

D. Bioaccumulation

The log K_{ow} for citric acid is -1.5 to -1.8. Thus, citric acid is not expected to bioaccumulate.

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Citric acid exhibits low toxicity by the oral and dermal routes. It is an eye irritant, but slightly to non-irritating to the skin. No adequate studies were found to evaluate the sensitization potential of citric acid. Minimal toxicity and no carcinogenic effects were observed in rats given oral doses of citric acid for up to two years. Citric acid was not mutagenic to bacteria, but *in vitro* studies using human lymphocytes showed genotoxic effects. *In vivo* genotoxicity studies were negative. There were no reproductive or developmental effects in rats given oral doses of citric acid.

B. Acute Toxicity

Oral

The acute oral LD50 in male and female Füllinsdorf albino (SPF) mice exposed to 0,3,4.2, 6, 8.5, and 13 g/kg bw of citric acid via oral gavage was reported to be 5,400 mg/kg bw/day (ECHA) [KI. score =2].

The acute oral LD50 in male ICR-JCL male rats was reported to be 11,700 mg/kg (ECHA) [Kl. score = 2].

The acute oral LD50 values in SD-JCL male mice are 5,400 and 5,790 mg/kg (ECHA) [Kl. score = 2].

Inhalation

There are no reliable studies available.

Dermal

The acute dermal LD_{50} value in rats is >2,000 mg/kg (ECHA) [Kl. score = 1].

C. Irritation

Skin

Application of 0.5 g citric acid powder to the skin of New Zealand white rabbits for 4 hours under semi-occlusive conditions was slightly irritating. The mean of the 24, 48, and 72-hour scores were: 0.3 for erythema and 0.0 for oedema (ECHA) [Kl. score = 1].

Application of citric acid powder to the intact skin of New Zealand white rabbits for 4 hours under semi occlusive conditions was reported to be non-irritating based on a primary dermal irritation index (PDII) score of 0.33/2 (ECHA) [Kl. score = 1].

Application of a 30% solution of citric acid to the intact skin of New Zealand white rabbits was found reported to slightly irritating to rabbits with intact (abraded skin) and non-irritating to rabbits with non-abraded skin based on a primary dermal irritation index (PDII) scores of 0.8/8 and 0/8 respectively (ECHA) [KI. score = 2].

Application of a 50% aqueous solution of citric acid to New Zealand white rabbits for 4 hours under occlusive conditions was reported to be non-irritating (ECHA) [KI. score = 2].

Eye

Instillation of a 30% aqueous solution of citric acid into the eyes of New Zealand white rabbits produced well defined to moderate conjunctival irritation that did not fully resolve after the 14-day observation period (ECHA) [KI. score =1]. Given the fact that the 30% solution effects would have been allowed to dissipate for 21 days, it likely that the test substance would not be considered irritating to the eyes (ECHA).

Instillation of a 10% solution of citric acid into the eyes of New Zealand white rabbits was associated with weak to moderate conjunctival effects, which resolved after 7 days (ECHA) [Kl. score = 1].

Respiratory

In a study preliminary to the evaluation of antitussive agents, citric acid was chosen as most consistent in the cough response elicited as measured by the mean number of coughs produced with five inhalations in human volunteers (ECHA). 10% citric acid gave the highest number of positive reactors.

In a study to develop a method for the use of citric acid in testing antitussive medicines with human volunteers, a training period was used to determine the concentration of citric acid solution able to produce 3-6 coughs after one inhalation (ECHA). There were three test periods one hour apart. 5 inhalations were administered at 3-minute intervals in each test period. The number of coughs was counted after each inhalation. Each subject was given a placebo tablet after the first test period but was informed that they could receive either a placebo or an anti-tussive tablet.

The total number of coughs after each inspiration over the three test periods was compared among subjects and between test periods and inspirations. Statistical variance and F-values were analyzed.

The concentration of citric acid producing between 3 and 6 coughs after a single inhalation was found to vary from 5% to 25%. Adaptation to the citric acid aerosol occurred during the initial training period, but further adaptation during the test period was low, except between the first and second inhalation.

Some reduction in response between the first and second test periods might be attributable to a placebo reaction. It was concluded that the administration of citric acid to induce coughing using the method described would be useful in evaluating antitussive medicines, providing that a double-blind trial using a placebo was used.

A study was conducted to evaluate the effect of inspiratory flow rate on the cough response in humans to citric acid (ECHA). It was considered by the authors that the cough response to citric acid is produced mainly by irritation of the larynx and trachea. Variations in the inspiratory flow rate might lead to changes in deposition of the drug, and consequently in the cough threshold. The effect of inspiratory flow rate was studied in 11 healthy non-smoking volunteers aged 23 to 29 years (9

male, 2 female). The citric acid was administered by inhalation of a nebulized solution via apparatus which limited and measured the inspiratory flow rate to 50, 100 and 150 l/minute of increasing concentrations of citric acid.

The test was finished when a cough was produced after each inhalation at one concentration (cough threshold) or the maximum concentration was reached. Each concentration was given at three different flow rates. The exposures were repeated on 3 days at least 48 hours apart.

The mean cough threshold was determined to be 21 (±9-54) mg/l at an inspiratory flow rate of 50 l/min and 43 (±13-141) mg/l at 150 l/minute. It was concluded that inspiratory flow rate should be controlled when cough challenges with citric acid are performed.

Inhalation of citric acid was shown to cause cough and bronchoconstriction in the guinea pig. The bronchoconstriction seems to involve cholinergic and capsaicin sensitive neurons (ECHA).

Citric acid was seen to elicit a cough response in the guinea pig (ECHA) in a study in which the time-response relationship observed with citric acid showed a maximum response around 5 to 10 minutes of exposure for isolated coughs and a fade in response as the exposure continued.

D. Sensitisation

In a skin prick test, with very limited provided details, it was reported that citric acid, caused positive results in 3 of 91 patients whereof one of the patients also reacted to benzoic and propionic acids (ECHA) [KI. score =4].

In a skin sensitisation, study with limited details, citric acid was concluded to not be a skin irritant or a sensitizer when tested to human volunteers (ECHA) [KI. score = 4]. At induction, patches of 4 % citric acid in a cuticle cream were applied onto the skin of 56 human volunteers, under a semi-occlusive dressing, three times a week for three weeks. At challenge, 4 % citric acid in a cuticle cream was applied dermally to 56 human volunteers two weeks after the last induction (ECHA) [KI. score =4].

E. Repeated Dose Toxicity

<u>Oral</u>

Male and female rats were administered 2000, 4000, 8000, and 16000 mg/kg bw/day of citric acid via oral gavage daily for five successive days. A NOAEL of 4000 mg/kg bw/day was established for both male and female rats based on overall clinical signs, mortality, and body weight. A LOAEL of 8000 mg/kg bw/day was established for male and female rats based on clinical signs, increased mortality, and body weight gain. A 10-day LD_{50} value of 55560 ± 0.44 mg/kg bw/day was also reported in rats (gender not specified) (ECHA) [KI. score = 2].

Mice were administered 1000, 2000, 4000, and 8000 mg/kg bw/day of citric acid via oral gavage daily for ten successive days. A NOAEL of 1000 mg/kg bw/day was established based on clinical signs, mortality, and body weight. A LOAEL of 2000 mg/kg bw/day was established based on clinical signs, increased mortality, and body weight gain (ECHA) [KI. score = 2].

Male rats were given 0, 1.2, 2.4, or 4.8% citric acid in their feed for 6 weeks. The daily intakes were reported to be 1,150, 2,260, or 4,670 mg/kg-day. The high-dose animals had mild blood and urine

parameter changes and slight degeneration of the thymus gland and spleen. The NOAEL is 2.4% in the diet or 2,260 mg/kg-day (OECD, 2001a, b). [Kl. score = 4]

Rats were given 3% or 5% citric acid in their diet for two years. The estimated daily intakes were 1,200 and 2,000 mg/kg/day, respectively. A slight decrease in growth was reported in the 2% group, but no tissue abnormalities in the major organs. The NOAEL is 1,200 mg/kg/day (OECD, 2001a,b). [Kl. score = 4]

Inhalation

No studies are available.

Dermal

No studies are available.

F. Genotoxicity

In vitro Studies

Table 2 presents the results of the *in vitro* genotoxicity studies on citric acid.

Table 2: *In vitro* Genotoxicity Studies on Citric Acid

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
In vitro mammalian cell micronucleus test (lymphocytes: peripheral human)	-	+	2	ECHA
Bacterial Reverse Mutation Assay (S. typhimurium TA 1535, TA 100, TA 98, TA 1537, TA 92, and TA 94)	-	-	2	ECHA
Comet assay (human lymphocytes)	+	NA	2	ECHA
Chromosome aberration test (human peripheral lymphocytes)	+	NA	2	ECHA

*+, positive; -, negative; NA, not applicable

Citric acid was not mutagenic in bacterial reverse mutation assays with strains of *S. typhimurium* or *E. coli* with and without metabolic activation (OECD, 2001a,b; ECHA). [Kl. score = 2]

Peripheral human lymphocytes were treated with 50 to 3,000 µg/ml citric acid. A statistically significant dose-dependent increase in the micronuclei was observed. In another set of studies by the same laboratory, there was a statistically significant and dose-related increase in the number of cells with aberrations, including sister chromatid unions. The study authors reported that the pH of the medium was unchanged (ECHA). [Kl. score = 2]

In vivo Studies

Citric acid was reported to be non-mutagenic in a rodent dominant lethal assay when male Sprague-Dawley rats were given either a single oral dose of citric acid (1.2, 12.0, or 120 mg/kg) or a single oral

dose on five consecutive days (300, 500, or 3,500 mg/kg) (OECD 2001a,b; as reported in ECHA) [Kl. score = 2].

There were no treatment related increases in cells with chromosomal aberrations in observed in the bone marrow of male Sprague-Dawley rats given either a single oral dose of citric acid (1.2, 12.0, or 120 mg/kg) or a single oral dose on five consecutive days (300, 500, 3000, or3,500 mg/kg) (ECHA) [Kl. score = 2].

G. Carcinogenicity

Oral

There was no evidence of carcinogenicity in rats given 3% or 5% citric acid in feed (1,200 or 2,000 mg/kg/day, respectively) for two years (OECD, 2001a, b). [Kl. score = 4]

In a rat feeding study, animals dosed with 5% citric acid in the diet did not show an excess of tumors in comparison with control animals when tested over a period of 2 years (Horn et al., 1957; as reported in ECHA). However, there was limited evidence that high doses of citrate salts increased the incidence of tumors produced by co-administration of known bladder carcinogens (Inouea et al., 1988; Ono et al., 1992; de Camargo et al., 1991; Fukushima et al.,1986; Behnke et al., 1964; as reported in ECHA). Where citric acid or citrate salts were administered alone during these studies, no dose-related tumors were noted (ECHA).

H. Reproductive Toxicity

In a non-standard repeat dose dietary study (duration and frequency not specified), 5% citric acid in feed did not affect either the number of young born to mice or rats or their subsequent survival up to the point of weaning (ECHA). [Kl. score = 4]

In a reproductive toxicity study, 1.2% w/w citric acid was administered in feed given daily to male and female rats over a period of 90 weeks and it was reported that citric acid did not give rise to any reproductive effects (ECHA).

The no adverse effect level (NOAEL) for reproductive toxicity in rats has been reported as 2500 mg/kg/bw/day (Kim et al, 2013 citing Citric acid SIDS initial assessment report (OECD SIDS, 2001; as cited in ECHA).

I. Developmental Toxicity

Hamsters were administered citric acid via oral gavage daily from gestation day 0 to gestation day 10 resulted in a NOAEL of > 272 mg/kg bw/day based on teratogenicity (ECHA) [KI. Score=2].

Wistar rats were exposed to citric acid by oral gavage from gestation day 6 to gestation day 15. A NOAEL of >295 was established for this study based on teratogenicity (ECHA) [KI. Score =2].

Albino CD-1 mice were exposed to citric acid by oral gavage from gestation day 6 to gestation day 15. A NOAEL of >241 mg/kg bw/day was established for this study based on teratogenicity (ECHA) [KI. score =2].

Pregnant female rats were dosed by oral gavage with 0, 2.95, 13.7, 63.6, or 295 mg/kg citric acid on GD 6-15. No maternal or developmental effects were noted. The NOAEL for maternal and

developmental toxicity is 295 mg/kg-day, the highest dose tested (OECD, 2001a, b; ECHA). [Kl. score = 2]

Pregnant female rats were dosed by oral gavage with 0, 2.41, 11.2, 52, or 241 mg/kg citric acid on GD 6-15. No maternal or developmental effects were noted. The NOAEL for maternal and developmental toxicity is 241 mg/kg-day, the highest dose tested (OECD, 2001a, b; ECHA). [Kl. score = 2]

Pregnant female rabbits were dosed by oral gavage with 0, 4.25, 19.75, 91.70, or 425 mg/kg citric acid on GD 6-18. No maternal or developmental effects were noted. The NOAEL for maternal and developmental toxicity is >425 mg/kg-day, the highest dose tested (OECD, 2001a, b; as cited in ECHA)[KI. score =2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for citric acid follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

In a two-year dietary study, the only effect seen in rats fed either 3 or 5% citric acid (approx. 1,200 or 2,000 mg/kg/day) was a slight decrease in growth in the 5% dose group. In the absence of statistical analysis of the body weight gain data, a conservative approach was taken, and the 5% dose group was considered an LOAEL. The NOAEL of 3% citric acid in the diet (1,200 mg/kg/day) will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

- UF_A (interspecies variability) = 10
- UF_H (intraspecies variability) = 10
- UF_L (LOAEL to NOAEL) = 1
- UF_{Sub} (subchronic to chronic) = 1
- UF_D (database uncertainty) = 1

Oral RfD = 1,200/(10 x 10 x 1 x 1 x 1) = 1,200/100 = 12 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)

Drinking water guidance value = (12 x 70 x 0.1)/2 = 42 mg/L

B. Cancer

Citric acid was not carcinogenic to rats in a chronic dietary study. Thus, no cancer reference value was derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Citric acid does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidizing potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Citric acid is of low toxicity concern to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

The 48-hour LC_{50} values in Leuciscus idus melanotus (golden orfe) from two separate laboratories were 440 mg/L and 760 mg/L (ECHA) [Kl. scores = 2].

The 96-hour LC_{50} in Lepomis macrochirus (fathead minnow) is >100 mg/L (ECHA) [Kl. score = 2).

The 24-hour EC_{50} in Daphnia is 85 mg/L in un-neutralized test solution and 1,535 mg/L in a neutralized solution (OECD, 2001a,b; as cited in ECHA). [Kl. score = 2]

The 8-day toxicity threshold value (EC_0) of 640 mg/L and a NOEC of 425 mg/L was determined for citric acid in Scenedesmus quadricauda (ECHA; OECD, 2001a,b). [Kl. score = 2]

Chronic Studies

Citric acid is essential in the Krebs cycle (or TCA cycle), which in turn is an essential chemical cycle that takes place in all living organisms to generate energy, via the generation of adenosine triphosphate (ATP). This means that citric acid is naturally present inside all living organisms, and it is very unlikely that it will be found in the environment at concentrations high enough to exert hazards to organisms (ECHA). Short-term aquatic toxicity data indicate that citric acid is of low toxicity. Further, the substance is readily biodegradable, has a log K_{ow} <3 and is highly soluble. Therefore, it is very unlikely to persist in the environment long enough to cause long-term effects. As a result, the completion of chronic studies was not required, and no studies are available.

C. Terrestrial Toxicity

No studies are available.

D. Calculation of PNEC

The PNEC calculations for citric acid follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for two trophic levels. Acute E(L)C_{50} values are available for fish (440 mg/L) and *Daphnia* (1,535 mg/L, neutralized). On the basis that the data consist of short-term results from two trophic levels, an assessment factor of 1,000 has been applied to the lowest reported effect concentration of 440 mg/L for fish. The $PNEC_{water}$ is 0.44 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 0.277 mg/kg wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (0.807/1280) \times 1000 \times 0.44$
$= 0.277$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]

$K_{sed\text{-}water} = 0.8 + [0.2 \times Kp_{sed}/1000 \times BD_{soilid}]$
$= 0.8 + [0.2 \times 0.014/1000 \times 2400]$
$= 0.807\ m^3/m^3$

Where:
Kp_{sed} = solid-water partition coefficient (L/kg).

BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]

$Kp_{sed} = K_{oc} \times f_{oc}$
$= 0.3617 \times 0.04$
$= 0.014$ L/kg

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for citric acid is estimated to be 0.3617 L/kg.
F_{oc} = fraction of organic carbon suspended sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.002 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
$= (0.007/1500) \times 1000 \times 0.44$
$= 0.002$ mg/kg

Where:
Kp_{soil} = soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]

$Kp_{soil} = K_{oc} \times f_{oc}$
$= 0.3617 \times 0.02$
$= 0.007\ m^3/m^3$

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for citric acid is estimated to be 0.3617 L/kg.
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU Reach Criteria methodology (DEWHA, 2009, ECHA, 2017).

Citric acid is readily biodegradable; thus, it does not meet the screening criteria for persistence.

The log K_{ow} values for citric acid are -1.5 to -1.8. Thus, citric acid does not meet the screening criteria for bioaccumulation.

There are no chronic aquatic toxicity studies on citric acid. The acute $E(L)C_{50}$ values for citric acid are >1 mg/L in fish and invertebrates. Thus, it does not meet the screening criteria for toxicity.

The overall conclusion is that citric acid is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

The information in this section is for a citric acid solution.

A. Classification

H315: Causes skin irritation
H319: Causes serious eye irritation
H335: May cause respiratory irritation
Eye irritation-category 2A
Skin irritation-category 2
Specific target organ toxicity (single exposure)- category 3

B. Labelling

Warning

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

In the case of contact, immediately flush eyes with plenty of water for at least 15 minutes. If symptoms persist, seek medical advice.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention if respiratory irritation develops or if breathing becomes difficult.

Ingestion

Rinse mouth with water and then drink plenty of water. Never give anything by mouth to an unconscious person. If symptoms develop, seek medical advice.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

No data are available.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilt

Pick up with absorbent material. Dispose of contaminated material as prescribed.

D. Storage and Handling

General Handling

No special measures necessarily provided product is used correctly.

Other Handling Precautions

Avoid eye and skin contact.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for citric acid.

Engineering Controls

None

Personal Protection Equipment

Respiratory Protection:

Respiratory protection is not required.

Hand Protection:

Chemical resistant protective gloves.

Skin Protection:

Body protection must be chosen depending on activity and possible exposure.

Eye protection:

Safety glasses with side-shields.

Other Precautions:

Handle in accordance with good industrial hygiene and safety practice. Eyewash fountains and safety showers must be easily accessible.

F. Transport Information

Citric acid is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods Code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

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ACRYLAMIDE/SODIUM ACRYLATE COPOLYMER (CAS NO. 25085-02-3)
ACRYLAMIDE/AMMONIUM ACRYLATE COPOLYMER (CAS NO. 26100-47-0)
ACRYLAMIDE, SODIUM ACRYLATE POLYMER (CAS NO. 25987-30-8)
2-PROPENOIC ACID, POTASSIUM SALT, POLYMER WITH 2-PROPENAMIDE (CAS NO. 31212-13-2)
ACRYLATE TERPOLYMER (CAS NO. 903573-39-7)[1]
SILICONE BASED EMULSION NEUTRALISED POLYACRYLIC BASED STABILISER (NO CAS NO.)

This group contains a sodium salt of a polymer consisting of acrylic acid, methacrylic acid or one of their simple esters and three similar polymers. They are expected to have similar environmental concerns and have consequently been assessed as a group. Information provided in this dossier is based on acrylamide/sodium acrylate copolymer (CAS No. 25085-02-3).

This dossier on acrylamide/sodium acrylate copolymer and similar polymers presents the most critical studies pertinent to the risk assessment of these polymers in their use in coal seam gas activities. This dossier does not represent an exhaustive or critical review of all available data. Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): 2-Propenoic acid, sodium salt, polymer with 2-propenamide

CAS RN: 25085-02-3

Molecular formula: $(C_3H_5NO.C_3H_4O_2.NA)_x$-

Molecular weight: No information is available. Based on the type and intended use of the copolymer, the molecular weight would likely range from 100,000 to > 3,000,000 daltons (Hamilton et al., 1997).

Synonyms: Acrylamide/sodium acrylate copolymer; 2-propenamide, polymer with 2-propenoic acid, sodium salt; 2-propenoic acid, sodium salt, polymer with 2-propenamide; 2-Propenamide-sodium 2 propenoate copolymer; sodium acrylate acrylamide polymer; sodium acrylate-acrylamide copolymer

SMILES: Not applicable.

II. PHYSICAL AND CHEMICAL PROPERTIES

No information is available.

III. ENVIRONMENTAL FATE PROPERTIES

No studies are available. The acrylamide/sodium acrylate copolymer is not expected to be readily biodegradable. The physico-chemical properties of the copolymer would preclude it from undergoing significant biodegradation (Guiney et al., 1997). Biodegradation is limited due to the very high molecular weight and the low water solubility of the copolymer. The copolymer will likely bind tightly to organic matter found within soils and sediments (Guiney et al., 1997). The copolymer is not expected to bioaccumulate because of its poor water solubility and high molecular weight.

[1] CAS name: 2-Propenoic acid, polymer with sodium 2-hydroxy-3-(2-propen-1-yloxy)-1-propanesulfonate (1:1) and alpha-sulfo-omega-(2-propen-1-yloxy)poly(oxy-1,2-ethanediyl) ammonium salt (1:1), sodium salt

IV. HUMAN HEALTH HAZARD ASSESSMENT

No studies are available.

NICNAS has assessed acrylamide/sodium acrylate copolymer in an IMAP Tier 1 assessment and considers it a "polymer identified as a low concern to human health by application of expert validated rules[2]."

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

No toxicological reference values or drinking water guidance values were developed.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Acrylamide/sodium acrylate copolymer does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

No studies are available. Acrylamide/sodium acrylate copolymer is expected to be a low concern for toxicity to aquatic organisms (Guiney et al., 1997). Due to its poor solubility and high molecular weight, it is not expected to be bioavailable. It does not contain any reactive functional groups (i.e., cationic groups).

A. Calculation of PNEC

No PNEC values were calculated.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2008).

Acrylamide/sodium acrylate copolymer is not readily biodegradable; thus, it meets the screening criteria for persistence.

Acrylamide/sodium acrylate copolymer is expected to have a very high molecular weight and poor water solubility. It is not expected to be bioavailable. Thus, this copolymer does not meet the criteria for bioaccumulation.

There are no aquatic toxicity studies on acrylamide/sodium acrylate copolymer. It is expected to have low concern for aquatic toxicity because of its very high molecular weight and poor water solubility. Thus, the copolymer does not meet the criteria for toxicity.

The overall conclusion is that acrylamide/sodium acrylate copolymer is not a PBT substance.

[2] https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/tier-i-human-health-assessments#cas-A_25085-02-3

IX. CLASSIFICATION AND LABELLING

A. Classification

Not classified.

B. Labelling

No signal word.

C. Pictograms

None.

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 5 minutes. If symptoms persist, seek medical advice.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention if respiratory irritation develops or if breathing becomes difficult.

Ingestion

Rinse mouth with water and then drink plenty of water. Never give anything by mouth to an unconscious person. If symptoms develop, seek medical advice.

B. Fire Fighting Information

Extinguishing Media

Water fog, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Burning produces harmful and toxic fumes. Heat from fire may melt, decompose polymer and generate flammable vapours. Combustion products may include: Nitrogen oxides, carbon monoxide, carbon dioxide and unburned hydrocarbons (smoke). Dust can accumulate static charges which can cause an incendiary electrical discharge. Fine dust dispersed in air in sufficient concentrations, and in the presence of an ignition source, is a potential dust explosion hazard.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Potential combustible dust hazard. Avoid generating dust. Creates dangerous slipping hazard on any hard smooth surface.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

Scoop up and remove.

D. Storage and Handling

General Handling

Avoid dust accumulation in enclosed space. Avoid generating dust; fine dust dispersed in air in sufficient concentrations, and in the presence of an ignition source is a potential dust explosion hazard. Electrostatic charge may build up during handling. Equipment, container and metal containers should be grounded and bonded.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place. Use adequate ventilation to avoid excessive dust accumulation. Store away from excessive heat and away from strong oxidising agents. Take measures to prevent the build-up of electrostatic charge.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure limit for acrylamide/sodium acrylate copolymer.

Engineering Controls

Use in a well-ventilated area. Avoid creating dust. Take precautionary measures against static charge.

Personal Protection Equipment

Respiratory Protection: Not normally needed; however, if significant exposures are possible, then the following respirator is recommended: Dust/mist respirator.

Hand Protection: Normal work gloves.

Skin Protection: Normal work coveralls.

Eye Protection: Wear safety glasses or goggles to protect against exposure.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Eyewash fountains and safety showers must be easily accessible.

F. Transport Information

Acrylamide/sodium acrylate copolymer is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII.REFERENCES

Department of the Environment, Water, Heritage and the Arts (DEWHA). (2009). Environmental risk assessment guidance manual for industrial chemicals, Department of the Environment, Water, Heritage and the Arts, Commonwealth of Australia.

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CRYSTALLINE SILICA, QUARTZ (CAS No. 14808-60-7)
CRYSTALLINE SILICA, CRISTOBALITE (CAS No. 14464-46-1)
CRYSTALLINE SILICA, TRIDYMITE (CAS No. 15468-32-3)
NON-CRYSTALLINE SILICA (IMPURITY) (CAS No. 7631-86-9)
DIATOMACEOUS EARTH (CAS No. 61790-53-2)
DIATOMACEOUS EARTH, CALCINED (CAS No. 91053-39-3)

This dossier on crystalline silica, quartz, cristobalite and tridymite; non-crystalline silica (impurity); diatomaceous earth; and diatomaceous earth, calcined presents the most critical studies pertinent to the risk assessment of these substances in their use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

For the purpose of this dossier, crystalline silica, quartz (CAS No. 14808-60-7) has been reviewed as representative of crystalline silica cristobalite and tridymite, and non-crystalline silica (impurity). Crystalline silica, quartz is also considered representative of diatomaceous earth and diatomaceous earth, calcined, as they both consist mainly of silicon dioxide.

NICNAS has assessed crystalline silica in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment.

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): dioxosilane

CAS RN: 14808-60-7

Molecular formula: SiO_2

Molecular weight: 60.084 g/mol

Synonyms: Cristobalite, Dioxide, Silicon

SMILES: O=[Si]=O

II. PHYSICO-CHEMICAL PROPERTIES

Silica is an off-white granule that occurs naturally in various crystalline and amorphous or other non-crystalline forms. Crystalline silica is characterised by silicon dioxide (SiO_2) molecules oriented in fixed, periodic patterns to form stable crystals. The primary crystalline form of silica is quartz. Other crystalline forms of silica include cristobalite, tripoli and tridymite. Particle size is a key determinate of silica toxicity, since toxicity is restricted to particles that are small enough to be deposited into the target regions of the respiratory tract (OECD, 2011).

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Crystalline silica is characterised by silicon dioxide (SiO_2) molecules oriented in fixed, periodic patterns to form stable crystals. The primary crystalline form of silica is quartz. It is a stable solid under typical environmental conditions. It will not biodegrade, bioaccumulate, nor will it sorb to sediments or soils.

B. Biodegradation

No data are available. Based on the crystalline form of the substance, it is not expected to biodegrade.

C. Environmental Distribution

No experimental data are available for crystalline silica. As a stable inorganic solid, it is not soluble in water, and it will not sorb to soils or sediment.

D. Bioaccumulation

There are no bioaccumulation studies on crystalline silica.

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Human exposure to crystalline silica via inhalation can lead to silicosis, lung cancer and pulmonary tuberculosis (WHO, 2000).

B. Acute Toxicity

No adequate acute oral, dermal or inhalation exposure studies are available for quartz, cristobalite or tridymite.

Most acute toxicity studies for quartz or cristobalite were conducted using intratracheal instillation. Intratracheal instillation is the introduction of the substance directly to the trachea and is used to test respiratory toxicity of a substance.

Single intratracheal instillation of quartz caused inflammatory effects and formation of discrete silicotic nodules in rats, mice and hamsters (IARC, 2012; WHO, 2000). Other effects like oxidative stress, cellular proliferation and increases in water, protein and phospholipid content of rat lungs, apoptosis (programmed cell death) and lung cancer were also noted.

In an acute dose study, rats were dosed once with 0, 0.75, 1.5, 3.0, 6.0 or 12 mg/kg bw/day quartz by intratracheal instillation (Seiler et al., 2001). The lowest observed adverse effect level (LOAEL) of 0.75 mg/kg bw/day was derived from these studies.

Two other similar studies of single intratracheal instillation of quartz reported higher LOAELs in rats (3 and 40 mg/kg bw/day) based on inflammation and fibrosis (Saffiotti et al., 1996).

C. Irritation

No data available.

D. Sensitisation

No data available.

E. Repeated Dose Toxicity

Oral

No data available.

Inhalation

Repeated inhalation exposure of crystalline is known to cause adverse effects (IARC, 2012). Silicosis has been identified as the main non-cancer effect of silica exposure, although available epidemiologic data as well as animal data provide evidence for several other effects associated with silica exposure, such as silicotuberculosis, enlargement of the heart (cor pulmonale), interference with the body's immune system and damage to the kidneys (Health Canada, 2013).

Dermal

No data available.

F. Genotoxicity

No data available.

G. Carcinogenicity

Oral

No data available.

Inhalation

The International Agency for Research on Cancer (IARC) has classified crystalline silica as a Group 1 carcinogen, as there was sufficient evidence for carcinogenicity in experimental animals and sufficient evidence for carcinogenicity of inhaled crystalline silica from occupational sources (IARC, 1997; IARC, 2012).

H. Reproductive Toxicity

No data available.

I. Developmental Toxicity

No data available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicity information on crystalline silica is inadequate and/or unreliable for deriving toxicological reference and drinking water guidance values for this substance.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Crystalline silica does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Although no data are available, crystalline silica is expected to exhibit low acute toxicity to aquatic organisms.

B. Aquatic Toxicity

No aquatic toxicity data were available.

C. Terrestrial Toxicity

No terrestrial toxicity data were available.

D. Calculation of PNEC

No PNEC values were calculated.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2008).

Crystalline silica is an inorganic mineral. Thus, biodegradation is not applicable to this substance. For the purposes of this PBT assessment, the persistent criteria are not considered applicable to crystalline silica.

As an inorganic complex it is not expected to bioaccumulate. Thus, crystalline silica does not meet the screening criteria for bioaccumulation.

Crystalline silica is not expected to cause adverse effects in environmental receptors. Thus, this substance does not meet the screening criteria for toxicity.

Therefore, crystalline silica is not a PBT substance.

IX. CLASSIFICATION AND LABELING

A. Classification

H373 – may cause damage to organs through prolonged or repeated exposure.

B. Labelling

Warning

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. Get medical attention if symptoms persist.

Skin Contact

For minor skin contact, avoid spreading material on unaffected skin. Remove and isolate contaminated clothing. Wash the contaminated area of body with soap and fresh water. Launder contaminated clothing before reuse.

Inhalation

Move person to fresh air. Get medical attention if respiratory irritation develops or breathing becomes difficult.

Ingestion

Rinse mouth. Do not induce vomiting. Get medical attention if symptoms occur.

Notes to Physician

All treatments should be based on observed signs and symptoms of distress in the patient.

B. Fire Fighting Information

Extinguishing Media

Use extinguishing media appropriate for surrounding material.

Specific Exposure Hazards

Reacts with hydrofluoric acid (HF) forming toxic gas (SiF_4).

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Isolate area. Keep unnecessary and unprotected personnel from entering the area. Use personal protective clothing. Ensure adequate ventilation. Wear respiratory protection if ventilation is inadequate. Do not breath mist, vapours or spray. Avoid contact with skin, eyes and clothing. Eliminate all sources of ignition.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

Pick up mechanically – vacuum up. Avoid generating dust. If formation of dust cannot be avoided, use respiratory filter device. Dispose of the material collected according to regulations.

D. Storage And Handling

General Handling

Handle in accordance with good industrial hygiene and safety practice. Avoid contact with eyes, skin and clothing. Avoid dust formation. Do not breathe dust. Wash thoroughly after handling. Use with adequate ventilation.

Storage

Provide adequate exhaust ventilation at places where dust is formed. Keep airborne concentrations below exposure limits. Keep containers tightly closed in a dry, cool, well-ventilated area.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has established an occupational exposure standard for exposure to crystalline silica of an 8-hour time weighed average (TWA) exposure limit of 0.05 mg/m^3.

Engineering Controls

Good general ventilation should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation or other engineering controls to maintain airborne levels below recommended exposure limits. If exposure limits have not been established, maintain airborne levels to an acceptable level.

Personal Protection Equipment

Respiratory Protection: If workers are exposed to concentrations above the exposure limit, they must use appropriate, certified respirators. If there are no applicable exposure limit requirements or guidelines, use an approved respirator. Selection of air-purifying or positive pressure supplied-air will depend on the specific operation and the potential airborne concentration of the product. For emergency conditions, use an approved positive-pressure self-contained breathing apparatus. The following should be effective types of air-purifying respirators: organic vapour cartridge with a particulate pre-filter.

Hand Protection: Use gloves chemically resistant to this material. Consult the SDS for appropriate glove barrier materials.

Skin Protection: Use protective clothing chemically resistant to this material. Selection of specific items such as face shield, boots, apron or full body suit will depend on the task.

Eye Protection: Use chemical goggles.

Other Precautions: Wash hands, forearms and face thoroughly after handling chemical products; as well as before eating, smoking and using the lavatory; and at the end of the working period. Appropriate techniques should be used to remove potentially contaminated clothing. Wash contaminated clothing before reusing. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Crystalline silica is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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ETHOXYLATED DECANOL

This dossier on ethoxylated decanol presents the most critical studies pertinent to the risk assessment of ethoxylated decanol in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Decan-1-ol, ethoxylated

CAS RN: 26183-52-8

Molecular formula: $(C_2H_4O)nC_{10}H_{22}O$ (UVCB)

Molecular weight:202.33 g/mol (monomer)

Synonyms: Ethoxylated decanol; decyl alcohol, ethoxylated; Poly(oxy-1,2-ethanediyl), .alpha.-decyl-.omega.-hydroxydecyl alcohol; ethoxylated alpha-decyl-omega-hydroxypoly(oxy-1,2-ethanediyl); polyethylene glycol decyl ether; decyl alcohol ethoxylated; 2-decoxyethanol l

SMILES: C(CCCOCCO)CCCCCC

II. PHYSICO-CHEMICAL PROPERTIES

Key physical and chemical properties for the substance are shown in Table 1.

Table 1: Overview of the Physico-Chemical Properties of Ethoxylated Decanol

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Clear liquid with characteristic mild odor	1	ECHA
Melting Point	-27°C @ 101 kPa	1	ECHA
Boiling Point	224°C @ 101 kPa	1	ECHA
Density	880 kg/m³ @ 25 °C	2	ECHA
Vapor Pressure	100 Pa @ 20 °C	2	ECHA
Partition Coefficient (log K_{ow})	3.51 @25 °C	2	ECHA
Water Solubility	0.0000759-0.000082 g/L @ 25 °C	2	ECHA
Flash Point	118.7°C @ 101.3 kPa	2	ECHA
Auto flammability	220°C @ 101.3 kPa	2	ECHA
Viscosity	13.911 mm²/s @ 25°C	2	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Ethoxylated decanol is readily biodegradable. It is not expected to bioaccumulate and has a low tendency to adsorb to soil or sediment.

B. Biodegradation

An OECD Guideline 301 B (Ready Biodegradability: CO_2 Evolution Test) was performed. Ethoxylated decanol (6 EO) was tested for ready biodegradability according to OECD 301B. The degradation of the test item was 83% within 28 days (after acidification). The biodegradation of the test item reached the criterion for ready biodegradability (ECHA) [Kl. score = 1].

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

The adsorption potential of ethoxylated decanol was determined using EPIWIN QSAR model (SRC KOCWIN v2.01). Determination of the K_{oc} for the mixture was not possible but the K_{oc} value for the pure homologues in the mixture were calculated. The Log K_{oc} values from the KOCWIN calculation for ethoxylated decanol ranged from 68.45-127.1 L/kg (MCI method) and 75.71-231.5 9l (log K_{ow} method) (ECHA) [KI. score = 2].

The K_{oc} for ethoxylated decanol was determined using a more specific QSAR method where the adsorption of several radio-labelled specific alcohol ethoxylates homologues were investigated in activated sludge and river water solids. The K_{oc} value for ethoxylated decanol was determined to be 1057-1462 L/kg and the log K_{oc} value was determined to be 3-3.2 at 25 °C. These values indicate that ethoxylated decanol has low mobility in soil (ECHA) [KI.score = 2].

D. Bioaccumulation

A bioconcentration factor (BCF) value of 237 L/kg at 24- hours was determined using the fathead minnow (ECHA) [Kl score = 2].

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Ethoxylated decanol has low acute toxicity by the oral route of exposure and limited acute toxicity by the dermal route. It has moderate acute toxicity by the inhalation route of exposure. It is not a skin and eye irritant nor is it a skin sensitiser. Repeated exposure studies in rodents caused limited toxicity. There are data available to evaluate carcinogenic effects of decanol, ethoxylate although the lack of mutagenic effects suggests that decanol, ethoxylate is not expected to be a carcinogen. Ethoxylated decanol is not expected to have an effect on reproduction based on findings in animals exposed to similar compounds. There was no evidence of developmental toxicity observed in animals exposed to ethoxylated decanol by the dermal route of exposure.

B. Acute Toxicity

Oral

An OECD Guideline 401 (Acute Oral Toxicity) study was performed using male and female Sprague-Dawley rats. Decanol, ethoxylate was administered to the rats via oral: gavage at a dose of 5,050 mg/kg bw/day. The LD_{50} was determined to be > 5,050 mg/kg bw based on clinical signs of toxicity which included decreased activity, diarrhea, piloerection, and polyuria (ECHA) [Kl score = 1].

Inhalation

An OECD Guideline 403 (Acute Inhalation Toxicity) study was performed using male and female Sprague-Dawley rats exposed to an aerosol of ethoxylated decanol via the inhalation route of exposure. The mass median aerodynamic diameter was 1.90 ± 1.82. The four-hour LC_{50} was determined to be > 1,600 mg/m³ air or >1.6 mg/L (ECHA) [Kl score = 2].

An OECD Guideline 403 (Acute Inhalation Toxicity) study was performed using male and female Wistar rats exposed to a vapour of ethoxylated decanol via the inhalation route of exposure. The six-hour LC_{50} was determined to be >100 mg/m3 which represents the calculated saturated vapor pressure (ECHA) [Kl. score = 2].

Dermal

An OECD Guideline 402 (Acute Dermal Toxicity) study was performed using male and female Wistar rats exposed to decanol, ethoxylate via occlusive dressing. A24 hour LD_{50} of > 2,000 mg/kg bw/day was determined for decanol, ethoxylate (ECHA) [Kl. score = 2].

C. Irritation

Skin

An OECD Guideline 404 (Acute Dermal Irritation / Corrosion) was performed using New Zealand White rabbits exposed to decanol, ethoxylate via semiocclusive dressing for four hours. Very slight erythema (max score = 1) was present at each observation through 24 hours in three animals. Oedema (max score = 0) was not observed at any observation timepoint throughout the study. The reported skin irritation results for the test animals indicate that decanol, ethoxylate is not a dermal irritant (ECHA) [Kl score = 2].

Eye

An OECD Guideline 405 (Acute Eye Irritation / Corrosion) study was conducted using New Zealand White rabbits exposed to 0.2 mL of decanol, ethoxylate. The 24-, 48-, and 72-hour cornea opacity score (max score = 4), the iris score (max score = 0), the conjunctivae score (max score =0), and the chemosis (max score = 0) score indicated that the decanol ethoxylate was not irritating to the eye (ECHA) [Kl. score = 2].

D. Sensitisation

An OECD Guideline 406 (Skin Sensitisation) was performed using female Dunkin-Hartley guinea pigs exposed to decanol, ethoxylate via intradermal and epicutaneous routes of exposure.

A study was performed to assess the contact sensitisation potential of the test material in the albino guinea pig. Ten test and five control animals were used for the main study. Based on the results of sighting test, the concentration of the test material for the induction and challenge phases were selected as follows:

- Intradermal Induction: 1% w/v in arachis oil
- Topical Induction: undiluted as supplied
- Topical Challenge: 50% and 25% v/v in arachis oil

The decanol, ethoxylate produced a 0% (0/10) sensitisation rate and was classified as non-sensitiser to guinea pig skin (ECHA) [Kl score = 1].

E. Repeated Dose Toxicity

Oral

An OECD Guideline 408 (Repeated Dose 90-Day Oral Toxicity Study in Rodents) was performed using male and female Wistar rats. The oral repeated dose toxicity of the target substance was estimated based on an adequate and reliable sub chronic oral toxicity key study performed with a structural analogue source substance. Daily oral exposure of male and female rats via the diet for 90 consecutive days to the test substance did not result in any toxicologically relevant effects. The NOAEL was determined to be > 500 mg/kg bw/day, corresponding to the highest dose tested. The result of the key study is further supported by additional (supporting) studies of various structural analogue source substances. Therefore, a systemic NOAEL after oral exposure for the target substance of ≥ 500 mg/kg bw/day was established. The differences in molecular structure between the target and the source substances are unlikely to lead to differences in oral repeated dose toxicity (ECHA) [Kl. score = 2].

Inhalation.

There are no inhalation studies available.

Dermal

There are no dermal repeat dose studies available.

F. Genotoxicity

In vitro Studies

The results of the *in vitro* genotoxicity studies on ethoxylated decanol are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Ethoxylated Decanol

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
OECD Guideline 471 (Bacterial reverse mutation assay) *S. typhimurium* TA 1535, TA 1537, TA 98, TA 100, and TA 1538) **	-	-	2	ECHA
OECD Guideline 482 (Genetic Toxicology: DNA damage and repair unscheduled DNA synthesis in mammalian cells *in vitro*)	-	-	2	ECHA

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
OECD Guideline 473 (*In vitro* mammalian chromosome aberration test)	-	-	2	ECHA

*+, positive; -, negative

In vivo Studies

An OECD Guideline 475 (Mammalian Bone Marrow Chromosome Aberration) test was performed using male and female Sprague-Dawley rats. The rats were administered single doses of 450, 900 and 1500 mg/kg bw/day of decanol, ethoxylate via oral gavage. Post euthanasia, femoral bone marrow smears were prepared. There were no chromosomal aberrations observed post-treatment. Therefore, decanol, ethoxylate was determined to be non-mutagenic *in vivo* (ECHA) [Kl. score = 2].

An OECD Guideline 474 (Mammalian Erythrocyte Micronucleus Test) was performed using male and female CD-1 mice exposed to 100 mg/kg bw/day dose of decanol, ethoxylate via a single intraperitoneal injection. Decanol, ethoxylate was determined to be non-mutagenic *in vivo* (ECHA) [KI. score =2].

An OECD Guideline 474 (Mammalian Erythrocyte Micronucleus Test) was performed using male and female Swiss Webster mice exposed to 200, 400, and 640 mg/kg bw/day of decanol, ethoxylate. Decanol, ethoxylate was determined to be non-mutagenic *in vivo* (ECHA) [KI. score =2].

G. Carcinogenicity

There are no studies are available.

H. Reproductive Toxicity

An OECD Guideline 416 (Two-Generation Reproduction Toxicity) study was performed using male and female Fischer 344 rats. Animals were treated dermally with doses of 1, 10, and 25% (w/v) to shaved dorsal region. The reproductive toxicity of the target substance is estimated based on an adequate and reliable two-generation reproductive toxicity study of a structural analogue source substance with subsequent detailed examination of foetuses. Dermal treatment of pregnant rats with the test substance at doses of 10, 100 and 250 mg/kg bw/day resulted in no maternal toxicity and hence a dermal NOAEL for maternal systemic toxicity of ≥250 mg/kg bw/day. The NOAEL for reproductive toxicity, based on observations in the P0, F1 and F2 generations was determined to be ≥ 250 mg/kg/day [Kl. score = 2].

I. Developmental Toxicity

An OECD Guideline 416 (Two-Generation Reproduction Toxicity Study) was performed using male and female Fischer 344 rats. Animals were treated dermally with doses of 1, 10 and 25% (w/v) to shaved dorsal region. The developmental toxicity of the target substance is estimated based on an adequate and reliable two-generation reproductive toxicity study of a structural analogue source substance with subsequent detailed examination of foetuses. Dermal treatment of pregnant rats with the test substance at doses of 10, 100 and 250 mg/kg bw/day resulted in no maternal toxicity and hence a dermal NOAEL for maternal systemic toxicity of ≥250 mg/kg bw/day. Foetal abnormalities observed include malformations of eyes and front as well as hind limbs. All developmental effects were due to spontaneous occurrence and were considered not to be

treatment-related. The dermal developmental NOAEL was thus determined to be ≥250 mg/kg bw/day. No developmental toxicity is therefore expected for the target substance. As explained in the category justification, the differences in molecular structure between the target and the source substances are unlikely to lead to differences in the developmental toxicity and teratogenicity (ECHA) [KI. score =2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for ethoxylated oleic acid follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

Two-year chronic studies have been conducted in rats given dermal doses of ethoxylated decanol. The lowest NOAEL from these studies is ≥250 mg/kg/day, based on reproductive toxicity. The NOAEL of 250 mg/kg/day will be used for determining the oral Reference Dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:
- UF_A (interspecies variability) = 10
- UF_H (intraspecies variability) = 10
- UFr (route to route variability) = 10
- UF_L (LOAEL to NOAEL) = 1
- UF_{Sub} (subchronic to chronic) = 1
- UF_D (database uncertainty) = 1

Oral RfD = 250/ (10 x 10 x 10 x 1 x 1 x 1) = 250/1000 = 0.25 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:
- Human weight = 70 kg (ADWG, 2011)
- Proportion of water consumed = 10% (ADWG, 2011)
- Volume of water consumed = 2L (ADWG, 2011)

Drinking water guidance value = (0.25 x 70 x 0.1)/2 = 0.875 mg/L

B. Cancer

There are no carcinogenic studies available for ethoxylated decanol. Therefore, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Ethoxylated decanol does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Ethoxylated decanol is moderately toxic to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on decanol ethoxylate.

Table 3: Acute Aquatic Toxicity Studies on Ethoxylated Decanol

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Danio rerio (zebrafish)	96-hour LC_{50}	1.2 (mortality)	2	ECHA
Cyprinus carpio	96-hour LC_{50}	1.2 (mortality)	1	ECHA
Daphnia magna	48-hour EC_{50}	0.39-0.53 (mobility)	2	ECHA
Daphnia magna	48-hour EC_{50}	0.91	2	ECHA
Desmodesmus subspicatus	72-hour EC_{50}	0.18 (growth rate)	2	ECHA
Desmodesmus subspicatus	72-hour EC_{50}	1.8 (growth rate)	2	ECHA
Desmodesmus subspicatus	72-hour EC_{50}	1.6 (growth rate)	2	ECHA

Chronic Studies

Table 4 lists the results of chronic aquatic toxicity studies conducted on decanol ethoxylate.

Table 4: Chronic Aquatic Toxicity Studies on Ethoxylated Decanol

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Lepomis macrochirus (bluegill sunfish)	10-day NOEC	0.16 (mortality)	2	ECHA
Lepomis macrochirus (bluegill sunfish)	30-day NOEC	>0.33 (growth rate)	2	ECHA
Daphnia magna	21-day NOEC	0.77 (reproduction)	2	ECHA
Desmodesmus subspicatus	72-hour NOEC	0.4 (growth rate)	2	ECHA

C. Terrestrial Toxicity

In an acute toxicity test, according to OECD 207, there was no effect on earth worm *Eisenia fetida* was observed up to the highest test item concentration of 1,000 mg/kg soil dw after 13-days. Therefore, the $LC_{50}$was determined to be >1,000 mg/kg dw (ECHA) [Kl. score = 2].

D. Calculation of PNEC

The PNEC calculations for ethoxylated oleic acid follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E (L)C_{50} values are available for fish (1.2 mg/L), invertebrates (0.39 mg/L), and algae (0.18 mg/L). Results from chronic studies are available for fish (0.16 mg/L), invertebrates (0.77 mg/L) and algae (0.4 mg/L). On the basis that the data consists of short-term and long-term studies from three trophic levels, an assessment factor of 10 has been applied to the lowest reported NOEC value of 0.16 mg/L for fish. The $PNEC_{water}$ is 0.016 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. However, it can be expected that the substance will be mineralized under environmental conditions within a short time period. Long-term exposure of sediment organisms to ethoxylated decanol and/or degradation products of this substance is therefore unlikely (ECHA). Therefore, a $PNEC_{sed}$ was not calculated.

PNEC Soil

There is only one acute toxicity study using terrestrial receptors (i.e., NOAEL >1000 mg/kg soil). Given the limited data for the soil compartment, an assessment factor of 1000 was applied to derive a $PNEC_{soil}$ of 1 mg/kg dw.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Decanol, ethoxylated is readily biodegradable; thus it does not meet the screening criteria for persistence.

The BCF value for ethoxylated decanol is 237 L/kg. Therefore, ethoxylated decanol does not meet the screening criteria for bioaccumulation.

The NOECs from the chronic aquatic toxicity studies on ethoxylated decanol are > 0.1 mg/L. ethoxylated decanol. Thus, decanol, ethoxylate does not meet the screening criteria for toxicity.

Therefore, decanol, ethoxylate is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Acute toxicity (ingestion)-category 4

Eye damage-category 1

Skin irritation-category 2

H302-Harmful if swallowed

H3180 Causes serious eye damage

H315- Causes skin irritation

B. Labelling

Danger

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. Get medical attention immediately, preferably a physician for an ophthalmologic examination.

Skin Contact

For minor skin contact, avoid spreading material on unaffected skin. Remove and isolate contaminated clothing. Wash the contaminated area of body with soap and fresh water. Get medical attention. Launder contaminated clothing before reuse.

Inhalation

Move person to fresh air. Administer oxygen if breathing is difficult. Do not use mouth-to-mouth method if victim inhaled the substance; give artificial respiration with the air of a pocket mask equipped with a one-way valve or other proper respiratory medical device. Give artificial respiration if victim is not breathing. Get medical attention immediately.

Ingestion

Do not induce vomiting. Get medical attention immediately.

Notes to Physician

All treatments should be based on observed signs and symptoms of distress in the patient.

B. Fire Fighting Information

Extinguishing Media

Use water spray or fog, foam, dry chemical or carbon dioxide.

Specific Exposure Hazards

Emits toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon dioxide, carbon monoxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Isolate area. Keep unnecessary and unprotected personnel from entering the area. Use personal protective clothing. Ensure adequate ventilation. Wear respiratory protection if ventilation is inadequate. Do not breath mist, vapours or spray. Avoid contact with skin, eyes and clothing. Eliminate all sources of ignition.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

Eliminate all sources of ignition. Pick up with suitable absorbent material and transfer to a container for chemical waste. For large amounts: dike spillage and pump off product into container for chemical waste. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep away from heat, sparks and flame. Avoid contact with eyes, skin and clothing. Avoid breathing vapor. Wash thoroughly after handling. Keep container closed. Use with adequate ventilation.

Storage

Keep container tightly closed. Store away from heat and light.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for ethoxylated decanol.

Engineering Controls

Good general ventilation should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation or other engineering controls to maintain airborne levels below recommended exposure limits. If exposure limits have not been established, maintain airborne levels to an acceptable level.

Personal Protection Equipment

Respiratory Protection: If workers are exposed to concentrations above the exposure limit, they must use appropriate, certified respirators. If there are no applicable exposure limit requirements or guidelines, use an approved respirator. Selection of air-purifying or positive pressure supplied-air will depend on the specific operation and the potential airborne concentration of the product. For emergency conditions, use an approved positive-pressure self-contained breathing apparatus. The following should be effective types of air-purifying respirators: organic vapor cartridge with a particulate pre-filter.

Hand Protection: Use gloves chemically resistant to this material. Consult the SDS for appropriate glove barrier materials.

Skin Protection: Use protective clothing chemically resistant to the material. Selection of specific items such as face shield, boots, apron or full body suit will depend on the task.

Eye protection: Use chemical goggles.

Other Precautions: Wash hands, forearms and face thoroughly after handling chemical products, as well as before eating, smoking and using the lavatory and at the end of the working period. Appropriate techniques should be used to remove potentially contaminated clothing. Wash contaminated clothing before reusing. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

The substance is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

UN 1993

Class: 3

Packaging Group: II

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCE

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DIAMMONIUM PEROXODISULPHATE

This dossier on diammonium peroxodisulphate presents the most critical studies pertinent to the risk assessment of diammonium peroxodisulphate in its use in coal seam or shale gas extraction activities. It does not represent an exhaustive or critical review of all available data. The information presented in this dossier was obtained primarily from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Diammonium peroxodisulphate

CAS RN: 7727-54-0

Molecular formula: $H_8N_2O_8S_2$

Molecular weight: 228.21 g/mol

Synonyms: Ammonium persulfate; Diammonium peroxydisulfate; Diammonium peroxydisulphate; Diammonium persulfate; Peroxydisulfuric acid (((HO)S(O)2)2O2), ammonium salt (1:2); Peroxydisulfuric acid (((HO)S(O)2)2O2), diammonium salt; Peroxydisulfuric acid, diammonium salt; ammonium persulphate

SMILES: [NH4+].[NH4+].[O-]S(=O)(=O)OOS(=O)(=O)[O-]

II. PHYSICO-CHEMICAL PROPERTIES

Key physical and chemical properties for the substance are shown in Table 1.

Table 1: Overview of the Physico-Chemical Properties of Diammonium Peroxodisulphate

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	White, odourless, crystalline solid	1	ECHA
Melting Point	ND. Decomposes at ca. 120°C at 100.66 kPa	1	ECHA
Boiling Point	ND. Decomposes at ca. 393 K (= 120°C) at 100.79 kPa	1	ECHA
Density	1260 kg/m³ at 20°C	1	ECHA
Vapour Pressure	0 Pa @ 25°C	2	ECHA
Partition Coefficient (log K_{ow})	Not applicable as substance is inorganic salt	-	ECHA
Water Solubility	850 g/L @ 25°C	2	ECHA
Viscosity	ND. Substance is a solid at room temperature	-	ECHA

Property	Value	Klimisch Score	Reference
Dissociation constant (pKa)	Diammonium persulfate dissociates completely to ammonium cation and persulfate anion when it is dissolved in water.	-	ECHA

ND = not determined

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Diammonium peroxodisulphate dissociates in aqueous media to the ammonium cation and persulfate anion. Biodegradation is not applicable to inorganic compounds. Diammonium peroxodisulphate is not expected to bioaccumulate; it will dissociate to ions that are ubiquitous in the environment. Diammonium peroxodisulphate is not expected to adsorb to soil or sediment because of its dissociation properties and high water solubility.

B. Partitioning

Persulfates dissociate in water to the corresponding cation and persulfate anion. Hydrolysis is temperature and pH dependent. The persulfate anion, independent from the cation, undergoes decomposition in normal water or acid conditions, readily oxidizing water to oxygen, producing acid conditions. All degradation products are ubiquitous to the environment (ECHA).

Diammonium peroxodisulphate was shown to be hydrolytically stable at 10 °C and pH 4, 7 and 9, a minor hydrolysis was observed at 25 °C, whereas a very strong hydrolysis at 60 °C was observed within 4 days. The DT_{50} at pH 4 and 60 °C was determined to be 27.2 h, at pH 7 and 9 and 60 °C the DT_{50} was determined to be 36.5 h. The DT_{50} at environmentally relevant temperature (12 °C) and pH 7 was extrapolated to be 1698.18 h (70.76 d). (ECHA) [Kl. Score = 1].

C. Biodegradation

Biodegradation is not applicable to inorganic compounds.

D. Environmental Distribution

No experimental data are available for diammonium peroxodisulphate. Persulfates are soluble in water and their vapour pressures are negligible. Thus, persulfates released into the environment are distributed into the water compartment in ionic form of the cation and persulfate ion. Persulfates are not expected to sorb to soil due to their dissociation properties, instability (hydrolysis) and high water solubility. They behave as free ions and decompose into sulphate and bisulphate ions. All decomposition products are ubiquitous in the environment (ECHA).

E. Bioaccumulation

There are no bioaccumulation studies on diammonium peroxodisulphate. Substances of the Persulfate Category are inorganic salts sharing the same anionic persulfate moiety. Persulfates are very soluble in water and are not expected to bioaccumulate in soil or aqueous solutions. They will decompose into organic sulphate or bisulphate (ECHA).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Diammonium peroxodisulphate exhibits moderate acute toxicity by the oral route, and low acute toxicity by the inhalation and dermal routes. In humans, diammonium peroxodisulphate has the potential for skin irritation; it is also a skin sensitiser to guinea pigs and humans. Human exposure to persulfates (including diammonium peroxodisulphate) have been linked to a variety of skin and respiratory complaints indicative of sensitisation. Repeated oral exposure to diammonium peroxodisulphate resulted in irritation to the gastrointestinal tract; and respiratory irritation was seen in rats repeatedly exposed by inhalation to diammonium peroxodisulphate. It is not genotoxic or carcinogenic. It is not a reproductive or developmental toxicant.

B. Acute Toxicity

The oral LD_{50} values in rats are 300 and 700 milligrams per kilogram (mg/kg) for males and females, respectively (ECHA) [Kl. score = 1].

The inhalation 4-hour LC_{50} in rats is >2.95 milligrams per litre (mg/L). Particles sizes of <10 micrometre (μm) and <7 μm were 96.6% – 97.4% and 84.6% – 86%, respectively (ECHA) [Kl. score = 1].

The dermal LD_{50} in rats is >2,000 mg/kg (ECHA) [Kl. score = 1].

C. Irritation

Application of 0.5 g. diammonium peroxodisulphate to the skin of rabbits for 4 hours under occlusive conditions was not irritating. The mean of the 24-, 48-, and 72-hours scores were 0.00 for both erythema and oedema (ECHA) [Kl. score = 2].

Studies in humans indicate that persulfates have the potential for skin irritation (NICNAS, 2001). Calnan and Schuster (1963) reported skin irritation in a human patch test with 5% diammonium peroxodisulphate. Jordan (1998) reported that a mixture with 17.5% persulfates (ammonium, potassium, and sodium) induced skin irritation in human subjects from patches applied under occlusive conditions.

Instillation of 0.1 mL diammonium peroxodisulphate into the eyes of rabbits was considered slightly irritating. The mean of the 24-, 48-, and 72-hours scores were: 1.33 for corneal opacity; 0.00 for iridial lesions; 1.00 for conjunctival redness; and 0.33 for chemosis (ECHA) [Kl. score = 1].

D. Sensitisation

Diammonium peroxodisulphate was considered a skin sensitiser in a guinea pig maximization test (ECHA) [Kl. score = 2].

Human exposure to persulfates have been linked to a variety of skin and respiratory complaints indicative of sensitisation. The complaints consist of immediate and delayed contact hypersensitivity, contact urticarial, rhinitis, bronchitis, and asthma (NICNAS, 2001).

E. Repeated Dose Toxicity

Oral

Male and female CR-CD rats were fed 0, 100, 300, or 600 parts per million (ppm) diammonium peroxodisulphate in their diet for 28 days. The estimated daily intakes are 0, 13, 41, and 82 mg/kg-day. There were no treatment-related effects. The no observed adverse effects level (NOAEL) is 82 mg/kg-day, the highest dose tested (ECHA) [Kl. score = 2].

Male and female CR strain rats were fed 0, 300, 1,000 or 3,000 ppm sodium persulfate in their diet for 90-days. On day 48 of the study, the dietary concentration of the group receiving 1,000 ppm was increased to 5,000 ppm for the remainder of the study. Body weights was decreased in the two highest dose groups during the last six weeks of treatment. There were no treatment-related effects on urinalysis, clinical chemistry or hematology parameters. Histopathological findings were limited to the 3,000-ppm group only and consisted of necrosis and atrophy of the gastrointestinal tract epithelial lining. The absence of the gastrointestinal lesions in the group receiving 1,000 ppm for 8 weeks, followed by 5,000 ppm for 5 weeks, indicates that the lesions are related both to concentration in diet (dose) and length of exposure. A clear NOAEL for this study is 300 ppm, which is estimated to be 22 mg/kg-day. Another NOAEL may be the 1,000-ppm dietary group for an 8-week exposure period. (ECHA; OECD, 2005a,b). [Kl. score = 2].

Inhalation

Male and female SD rats were exposed (whole-body) to 0, 5, 10.3, or 25 milligrams per cubic metre (mg/m^3) diammonium peroxodisulphate dust by inhalation, 6 hours/day, 5 days/week for 13 weeks. Additional groups of animals were exposed for 13 weeks, followed by either a 4- or 13-week recovery period. The MMAD was 2.5, 2.7, and 2.5 µm for the 5, 10, and 25 mg/m^3 groups, respectively. No deaths occurred during the study that were considered to be exposure-related. The 25 mg/m^3 animals showed increased respiration rates, as well as a few of the 25 mg/m^3 animals. This clinical sign disappeared during the first few weeks of the recovery period. Body weights of the 25 mg/m^3 animals were significantly lower during most of the exposure period; by the end of the recovery period the body weights were comparable to the controls. Lung weights were increased in the 25 mg/m^3 animals at the end of the 13-week exposure period but were similar to controls after 6 weeks in the recovery period. Histopathologic changes indicative of irritation was seen in the trachea and bronchi/bronchioles in the 25 mg/m^3 animals; these lesions were not seen after 6 weeks in the recovery period. The NOAEL for this study is 10.3 mg/m^3 (ECHA). [Kl. score = 1]

Dermal

No studies are available.

F. Genotoxicity

In vitro Studies

There are no available genotoxicity studies on diammonium peroxodisulphate. The *in vitro* genotoxicity studies on sodium persulfate are presented below in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Sodium Persulfate

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (*S. typhimurium* strains)	-	-	1	ECHA
Unscheduled DNA synthesis (rat hepatocytes)	NA	-	1	ECHA

*+, positive; -, negative; NA, not applicable

In vivo Studies

Sodium persulfate did not induce micronuclei in the bone marrow cells of male and female mice given a single intraperitoneal injection of 0, 85, 169, or 338 mg/kg sodium persulfate (ECHA) [Kl. score = 2].

G. Carcinogenicity

A 51-week dermal study in female SENCAR mice exposed to 0.2 ml of a 200 milligrams per millilitre (mg/mL) solution of diammonium peroxodisulphate showed that diammonium peroxodisulphate is neither a tumour promoter nor a complete carcinogen when applied to the skin (OECD, 2005a,b; ECHA). [Kl. score = 2]

H. Reproductive and Developmental Toxicity

A reproductive and developmental toxicity screening study (OECD 421) has been conducted on diammonium peroxodisulphate. Male and female Crl:CD (SD)GS BR rats were fed 0, 40, 100, or 250 mg/kg diammonium peroxodisulphate in their diet. In the parental animals, there was no treatment-related mortality, clinical signs, body or organ weight changes, or effects seen in gross necropsy. There were no effects on reproductive performance, fertility, foetal anomalies, foetal viability, spermatogenesis, spermatogenic cycle. The NOAEL for reproductive and developmental toxicity and parental toxicity is 250 mg/kg-day, the highest dose tested (ECHA). [Kl. score = 1]

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for diammonium peroxodisulphate follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Toxicological reference values were not derived. Diammonium peroxodisulphate dissociates in water to ammonium and persulfate ions. The persulfate ions will further hydrolyse to sulphate ions.

The Australian drinking water guideline value for sulphate is 500 mg/L based on health. Concentrations of > 500 mg/L can have purgative effects. There is also an Australian drinking water guideline value for sulphate of 250 mg/L based on aesthetics; it is the taste threshold (ADWG, 2011).

B. Cancer

There are no valid carcinogenicity studies on diammonium peroxodisulphate. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Diammonium peroxodisulphate does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Diammonium peroxodisulphate is of low toxicity concern to aquatic and terrestrial organisms.

NICNAS has assessed diammonium peroxodisulphate in an IMAP Tier 1 environmental assessment and it was concluded that it poses no unreasonable risk to the environment[1].

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on diammonium peroxodisulphate.

Table 3: Acute Aquatic Toxicity Studies on Diammonium Peroxodisulphate

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Oncorhynchus mykiss	96-hour LC_{50}	76.3	1	ECHA
Daphnia magna	48-hour EC_{50}	120	1	ECHA
Phaeodactylum tricornutum	72-hour EC_{50}	320	1	ECHA

Chronic Studies

Long-term toxicity testing to fish was considered scientifically unjustified, due to the results obtained in the short-term toxicity to fish studies, the substance physical-chemical properties and hydrolysis behaviour (ECHA).

An OECD Guideline 211 (Daphnia magna Reproduction Test) was performed and yielded a 21-day NOEC of 20.8 mg/L based on reproduction (ECHA) [Kl Score = 1].

An OECD Guideline 201 (Alga, Growth Inhibition Test) study was performed and yielded a NOEC of 32 mg/L (ECHA) [Kl. Score = 1].

[1] https://www.industrialchemicals.gov.au/chemical-information/search-assessments?assessmentcasnumber=7727-54-0

C. Terrestrial Toxicity

No terrestrial toxicity studies are available.

Persulfates are not expected to be distributed into the terrestrial compartment and consequently not to cause toxicity to terrestrial organisms and plants (ECHA).

D. Calculation of PNEC

The PNEC calculations for diammonium peroxodisulphate follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E(L)C_{50} values are available for fish (76.3 mg/L), Daphnia (120 mg/L), and algae (136 mg/L). Results from chronic studies are available for invertebrates (20.8 mg/L) and algae (32 mg/L). On the basis that the data consists of short-term results from three trophic levels and long-term results from two trophic levels, an assessment factor of 50 has been applied to the lowest reported NOEC value of 20.8 mg/L for invertebrates. The $PNEC_{water}$ is 0.4 mg/L.

PNEC Sediment

No experimental toxicity data on sediment organisms are available. Diammonium peroxodisulphate dissociates completely in water with its environmental distribution is dominated by its high-water solubility. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as diammonium peroxodisulphate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{sediment}$. Based on its properties, no adsorption of diammonium peroxodisulphate to sediment is to be expected, and the assessment of this compartment will be covered by the aquatic assessment.

PNEC Soil

No experimental toxicity data on terrestrial organisms are available. The environmental distribution of diammonium peroxodisulphate is dominated by its water solubility. Sorption of diammonium peroxodisulphate should probably be regarded as a reversible situation, i.e., the substance is not tightly nor permanently bound. K_{oc} and K_{ow} parameters do not readily apply to inorganics, such as diammonium peroxodisulphate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{soil}$. Based on its properties, diammonium peroxodisulphate is not expected to significantly adsorb to soil, and the assessment of this compartment will be covered by the aquatic assessment.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Diammonium peroxodisulphate is an inorganic salt that dissociates to respective cations and anions. Biodegradation is not applicable to these inorganic ions. For the purposes of this PBT assessment, the persistent criteria are not considered applicable to this inorganic salt.

Diammonium peroxodisulphate is not expected to bioaccumulate; it will dissociate to ions that are ubiquitous in the environment. Thus, the substance does not meet the screening criteria for bioaccumulation.

Chronic aquatic toxicity data is > 0.1 mg/L and acute aquatic toxicity data is >1 mg/L. Thus, diammonium peroxodisulphate does not meet the screening criteria for toxicity.

The overall conclusion is that diammonium peroxodisulphate is not a PBT substance.

IX. CLASSIFICATION AND LABELING

A. Classification

Oxidising Solid Category 3
Acute Toxicity Category 4 [Oral]
Skin Irritant Category 2
Eye Irritant Category 2
Skin Sensitiser Category 1
Respiratory Sensitisation Category 1
STOT SE Category 3 [Respiratory Irritation]

B. Labelling

Danger

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. Get medical attention.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention if respiratory irritation develops or if breathing becomes difficult.

Ingestion

Rinse mouth with water and then drink plenty of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: sulphur oxides.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Use personal protective clothing. Avoid dust formation. Ensure adequate ventilation. Do not breathe dust. Wear respiratory protection if ventilation is inadequate. Avoid contact with skin, eye, and clothing.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilled

Scoop up and remove.

D. Storage and Handling

General Handling

Good general ventilation should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation, or other engineering controls to maintain airborne levels below recommended exposure limits.

Other Handling Precautions

Avoid eye and skin contact. Avoid creating or inhaling dust.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place. Do not store with alkalis, acids, or reducing agents.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace exposure standard for diammonium peroxodisulphate in Australia is 0.01 mg/m^3 as a time-weighted average (TWA) peak exposure. A peak limitation is defined by Safe Work Australia as a maximum or peak airborne concentration of a substance determined over the shortest analytically practicable period of time which does not exceed 15 minutes.

Engineering Controls

Ensure adequate ventilation. Localized ventilation should be used to control dust levels below permissible exposure limits.

Personal Protection Equipment

Respiratory Protection:

Use respiratory protection when airborne concentrations are expected to be high.

Hand Protection:

Chemical resistant protective gloves.

Skin Protection:

Body protection must be chosen depending on activity and possible exposure.

Eye protection:

Safety glasses with side-shields.

Other Precautions:

Handle in accordance with good industrial hygiene and safety practice. Eyewash fountains and safety showers must be easily accessible. Remove and wash contaminated clothing before re-use. Contaminated work clothing should not be allowed out of the workplace.

F. Transport Information

UN1444 AMMONIUM PERSULPHATE

Class: 5.1

Packing Group: III

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed

XIII. REFERENCES

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DIISOBUTYL ADIPATE (CAS RN 141-04-8)
DIISOBUTYL GLUTARATE (CAS RN 71195-64-7)
DIISOBUTYL SUCCINATE (CAS RN 925-06-4)

This group contains information on diisobutyl adipate (CAS RN 141-04-8), diisobutyl glutarate (CAS RN 71195-64-7) and diisobutyl succinate (CAS RN 925-06-4). They are expected to have similar environmental concerns and have consequently been assessed as a group. Information provided in this dossier is based on diisobutyl adipate (CAS RN 141-04-8).

This dossier presents the most critical studies pertinent to the risk assessment of the diisobutyl compounds and their use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Hexanedioic acid, 1, 6-bis(2-methylpropyl) ester

CAS RN: 141-04-8

Molecular formula: $C_{14}H_{26}O_4$

Molecular weight: 258.18 g/mol

Synonyms: diisobutyl adipate; adipate, diisobutyl; adipic acid, diisobutyl ester; Hexanedioic acid, bis(2-methylpropyl) ester; hexanedioic acid, 1,6-bis(2-methylpropyl) ester; hexanoic acid, dibutyl ester; diisobutyl hexanedioate

SMILES: CC(C)COC(=O)CCCCC(=O)OCC(C)C

Chemical Name (IUPAC): Pentanedioic acid, bis(2-methylpropyl) ester

CAS RN: 71195-64-7

Molecular formula: $C_{13}H_{24}O_4$

Molecular weight: 244.17 g/mol

Synonyms: diisobutyl glutarate; glutaric acid, diisobutyl ester; pentanedioate, bis(2-methylpropyl);

SMILES: CC(C)COC(=O)CCCC(=O)OCC(C)C

Chemical Name (IUPAC): Butanedioic acid, bis(2-methylpropyl) ester

CAS RN: 925-06-4

Molecular formula: $C_{12}H_{22}O_4$

Molecular weight: 230.16 g/mol

Synonyms: diisobutyl succinate; butanedioate, bis(2-methylpropyl); butanedioic acid, 1,4-bis(2-methylpropyl) ester; succinic acid diisobutyl ester; bis(2-methylpropyl) butanedioate

SMILES: CC(C)COC(=O)CCC(=O)OCC(C)C

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-Chemical Properties of Diisobutyl Adipate (CAS RN 141-04-8)

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Liquid	1	ECHA
Melting Point	<-20.0°C (pressure not provided)	-	ECHA
Boiling Point	284.5 °C @98.1 kPa	1	ECHA
Density	951 kg/m^3 @ 20°C	1	ECHA
Vapour Pressure	15.1 Pa @ 20°C	1	ECHA
Partition Coefficient (log K_{ow})	4.3 @ 30°C	2	ECHA
Water Solubility	0.0427 g/L @ 25°C	1	ECHA
Flash Point	157 °C @ 101.3 kPa	1	ECHA

Table 2: Overview of the Physico-Chemical Properties of Diisobutyl Glutarate (CAS RN 71195-64-7)

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Liquid	-	ECHA
Melting Point	-21.8 °C (predicted average) (pressure not provided)	-	CompTox
Boiling Point	271 °C (predicted average) (pressure not provided)	-	CompTox
Density	966 kg/m^3 (predicted average) (temperature not indicated)	-	CompTox
Vapour Pressure	485 Pa (predicted average) (temperature not indicated)	-	CompTox
Partition Coefficient (log K_{ow})	3.34 (temperature not indicated)	-	CompTox
Water Solubility	0.264 g/L (predicted average) (temperature not indicated)	-	CompTox
Flash Point	120 °C (pressure not provided)	-	CompTox

Table 3: Overview of the Physico-Chemical Properties of Diisobutyl Succinate (CAS RN 925-06-4)

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Liquid	-	ECHA
Melting Point	-25.0 °C (predicted average) (pressure not provided)	-	CompTox
Boiling Point	252 °C (predicted average) (pressure not provided)	-	CompTox
Density	978 kg/m^3 (predicted average) (temperature not indicated)	-	CompTox
Vapour Pressure	950 Pa (predicted average) (temperature not indicated)	-	CompTox
Partition Coefficient (log K_{ow})	2.84 (temperature not indicated)	-	CompTox
Water Solubility	0.552 g/L (predicted average) (temperature not indicated)	-	CompTox
Flash Point	110 °C (pressure not provided)	-	CompTox

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Diisobutyl adipate is readily biodegradable. It is not expected to bioaccumulate. It has a moderate potential to adsorb to soil or sediment.

B. Biodegradation

Diisobutyl adipate is readily biodegradable in water. Using the OECD 301C Readily Biodegradability: Modified MITI Test (ECHA), approximately 86-95% of the material was biodegraded by 28 days (ECHA). [Kl. Score = 1].

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

No experimental data are available for diisobutyl adipate. Using KOCWIN in EPISuite™ (USEPA, 2017), the estimated K_{oc} value from log K_{ow} is 1293 L/kg. The estimated K_{oc} value from the molecular connectivity index (MCI) is 246.5 L/kg. Based upon these K_{oc} values, if released to soil, diisobutyl adipate is not expected to significantly adsorb to soil and has a moderate potential for mobility.

D. Bioaccumulation

No experimental data are available for diisobutyl adipate. Using the bioconcentration factor/bioaccumulation factor (BCFBAF) model in EPISuite™ (USEPA, 2017), the estimated BCF for diisobutyl adipate is 268.7 L/kg based on a regression based estimate. Based on this BCF value, this substance has a low potential for bioaccumulation.

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Diisobutyl adipate exhibits low acute toxicity by the oral route. Diisobutyl adipate is non-irritating to the skin and eyes. Diisobutyl adipate are not a skin sensitiser. In repeated dose toxicity study (28-day Oral), the no observed effect level (NOEL) for systemic toxicity was determined to be 1,000 mg/kg bw/day. Diisobutyl adipate is not genotoxic and is not carcinogenic. In a reproductive toxicity study, the no observed adverse effect level (NOAEL) was 1,000 mg/kg/day for reproduction in male and female rats and 300 mg/kg/day for the F1 generation.

B. Acute Toxicity

Oral

An OECD 401 Acute Oral Toxicity test was conducted. The acute oral LD_{50} of diisobutyl adipate was determined to be 12.1 ml/ kg bw (ECHA). [Kl. Score = 2]

Dermal

No experimental data are available for diisobutyl adipate.

Inhalation

No experimental data are available for diisobutyl adipate.

C. Irritation

Skin

Skin irritation testing was conducted under OECD 404: Acute Dermal; Irritation / Corrosion guidelines. Mice were exposed twice a day for 14 days with 100% diisobutyl adipate. At 100%, diisobutyl adipate was non-irritating to the skin (ECHA). [Kl. Score = 2].

Eye

Eye irritation testing was conducted under OECD 405: Acute Eye Irritation guidelines. Rabbits were exposed to 100% diisobutyl adipate. At 100%, diisobutyl adipate was non-irritating to the eye (ECHA). [Kl. Score = 2].

D. Sensitisation

Skin sensitization testing was conducted under OECD 406: Skin Sensitization guidelines. Diisobutyl adipate was applied to humans. Following the first application, a challenge dose was applied at 12 hrs. with a rechallenge at 24 hrs. There were no indication that diisobutyl adipate was a skin sensitizer (ECHA). [Kl. Score = 2].

E. Repeated Dose Toxicity

Oral

An OECD 407: Repeated Dose 28-day Oral Toxicity Study was conducted in rodents. Sprague Dawley rats were administered dose levels of 0, 20, 140, 1,000 milligrams per kilogram body weight per day (mg/kg bw/day) via oral gavage for 28 days. No effects were observed at any dose. Therefore, the NOEL for systemic toxicity was determined to be 1,000 mg/kg bw/day, the highest tested dose (ECHA). [Kl. Score = 2].

Inhalation

No data were available.

Dermal

No data were available.

F. Genotoxicity

In vitro Studies

The results of the *in vitro* genotoxicity studies on diisobutyl adipate are presented in Table 4.

Table 4: *In vitro* Genotoxicity Studies on Diisobutyl Adipate

Test System[1]	Results*		Klimisch Score	Reference
	-S9	+S9		
OECD Guideline 471 (Bacterial Reverse Mutation Assay) S. typhimurium (TA98, TA100, TA135, TA1537, and TA1538)	-	-	1	ECHA
OECD Guideline 472 (Genetic Toxicity: E. coli, Reverse Mutation Assay)	-	-	1	ECHA

*+, positive; -, negative.

In vivo Studies

No data available.

G. Carcinogenicity

No data available.

H. Reproductive Toxicity

Oral

An OECD 421: Reproduction / Developmental Toxicity Screen Test was conducted in rodents. Sprague Dawley rats were administered dose levels of 0, 100, 300, 1,000 mg/kg bw via oral gavage daily for 14 days during the premating exposure period. Treatment continued for 42 days in males and to day 3 of lactation for females.

Copulation, ovulation, fertility, maintenance of pregnancy, and parturition and lactation were not affected by the test compound.

Reproductive parameters (i.e., duration of gestation, number of corpora lutea, implantations and resorptions, litter size, and sex ratio distribution) were comparable among all four groups including controls. In the 1,000 mg/kg group, pup weight on postnatal days 0 and 4 was slightly decreased along with viability on postnatal day 4. Thus, the NOEL was considered to be 1,000 mg/kg/day for reproduction in male and female rats and 300 mg/kg/day for the F1 generation.

Concerning maternal and paternal general toxicity, no mortalities occurred in any group. There were no toxic effects of this chemical on the general condition of male and female animals. Slight suppression of body weight gain was observed in males in 1,000 mg/kg group, while body weight change in females and food consumption in male and female animals in all compound-treated groups were comparable to those in the controls. Macroscopic findings at necropsy and histological findings for the internal genitalia showed no abnormalities. Kidney weights were increased in males and females of the 1,000 mg/kg groups as compared to the control values. Thus the NOEL for general toxicity of this chemical in parent animals was considered to be 300 mg/kg/day (ECHA). [Kl. Score = 2].

Dermal

No data available.

I. Developmental Toxicity

No data available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for diisobutyl adipate follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

A subchronic repeat dose oral toxicity study was conducted in rodents. No effects were observed at any dose. Therefore, the NOEL for systemic toxicity was determined to be 1,000 mg/kg bw/day, the highest tested dose). The NOEL of 1,000 mg/kg bw/day will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 10
UF_D (database uncertainty) = 1
Oral RfD = 1000/(10 x 10 x 1 x 10 x 1) = 1000/1000 = 1 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (1 x 70 x 0.1)/2 = 3.5 mg/L

B. Cancer

Studies on carcinogenicity were not available. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Diisobutyl adipate does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Diisobutyl adipate is of low toxicity concern to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

Table 5 lists the results of acute aquatic toxicity data for diisobutyl adipate.

Table 5: Acute Aquatic Toxicity Studies on Diisobutyl Adipate

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Oryzias latipes	96-hour LC_{50}	3.7	1	ECHA
Daphnia magna	24-hour LC_{50}	17	1	ECHA
Selenastrum sp.	72-hour EC_{50}	2.8	1	ECHA

Chronic Studies

Long-term aquatic toxicity test of diisobutyl adipate was conducted in invertebrates. The chronic toxicity to Daphnia magna (OECD 211) was studied with a 21-d reproduction test in a semistatic system. The test solution was renewed 3 times per week. The 21-day no observed effect concentration (NOEC) was determined to be 5.6 mg/L for reproduction and survival of the adult test animals (ECHA) [Kl. Score = 1].

Diisobutyl adipate has also been evaluated for its toxicity towards the fresh water algae *Selenastrum capricornutum* in an Alga growth inhibition test according to OECD 201 under GLP requirements. The exposure duration was 72 hours under static conditions. The 72-hr NOEC (biomass) determined from the study was 2 mg/L (ECHA) [Kl. Score = 1].Sediment Toxicity

No data available

C. Terrestrial Toxicity

No data available

D. Calculation of PNEC

The PNEC calculations for siloxanes follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E(L)C_{50} values are available for fish (3.7mg/L), invertebrates (17 mg/L) and algae (2.8 mg/L). Results from chronic studies are also available for two trophic levels (invertebrates and algae), with the lowest NOEC value being 2 mg/L for algae. On the basis that the data consists of short-term studies for three trophic levels and long-term results studies for two trophic levels, an assessment factor of 50 has been applied to the lowest reported NOEC of 2 mg/L for algae. The $PNEC_{water}$ is 0.04 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 0.17 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1{,}000 \times PNEC_{water}$

$= (5.53/1{,}280) \times 1{,}000 \times 0.04$

$= 0.17$ mg/kg

Where:

$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (cubic metre per cubic metre [m^3/m^3])

BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]

$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1{,}000 \times BD_{solid}]$

$= 0.8 + [(0.2 \times 9.86/1{,}000 \times 2{,}400]$

$= 5.53\ m^3/m^3$

Where:

Kp_{sed} = solid-water partition coefficient (L/kg).

BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]

$Kp_{sed} = K_{oc} \times f_{oc}$

= 246.5 x 0.04

= 9.86 L/kg

Where:

K_{oc} = organic carbon normalized distribution coefficient (L/kg). The K_{oc} for diisobutyl adipate calculated from EPISUITE™ using the MCI is 246.5 L/kg .

F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There is no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.13 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$

= (4.93/1500) x 1000 x 0.04

= 0.13 mg/kg

Where:

Kp_{soil} = soil-water partition coefficient (m^3/m^3)

BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]

$Kp_{soil} = K_{oc} \times f_{oc}$

= 246.5 x 0.02

= 4.93 m^3/m^3

Where:

K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for diisobutyl adipate calculated from EPISUITE™ using the MCI is 246.5 L/kg .

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Diisobutyl adipate is readily biodegradable and thus does not meet the screening criteria for persistence.

Based on a measured log K_{ow} of 4.3 diisobutyl adipate does not meet the screening criteria for bioaccumulation.

The lowest chronic NOEC for diisobutyl adipate is >0.1 mg/L. The acute $E(L)C_{50}$ values are >1 mg/L. Thus, diisobutyl adipate does not meet the screening criteria for toxicity.

The overall conclusion is that diisobutyl adipate is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

None

B. Signal word

No signal word

C. Pictogram

Not applicable

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product safety data sheet (SDS) for additional information and for confirmation of the information provided herein.

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. Get medical attention immediately, preferably a physician for an ophthalmologic examination.

Skin Contact

For minor skin contact, avoid spreading material on unaffected skin. Remove and isolate contaminated clothing. Wash the contaminated area of the body with soap and fresh water. Get medical attention. Launder contaminated clothing before reuse.

Inhalation

Move person to fresh air. Administer oxygen if breathing is difficult. Do not use mouth-to-mouth method if victim inhaled the substance; give artificial respiration with the air of a pocket mask equipped with a one-way valve or other proper respiratory medical device. Give artificial respiration if victim is not breathing. Get medical attention immediately.

Ingestion

Do not induce vomiting. Get medical attention immediately.

Notes to Physician

All treatments should be based on observed signs and symptoms of distress in the patient.

B. Fire Fighting Information

Extinguishing Media

Use water spray or fog, foam, dry chemical or carbon dioxide.

Specific Exposure Hazards

Emits toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon dioxide, carbon monoxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Isolate area. Keep unnecessary and unprotected personnel from entering the area. Use personal protective clothing. Ensure adequate ventilation. Wear respiratory protection if ventilation is inadequate. Do not breath mist, vapours or spray. Avoid contact with skin, eyes and clothing. Eliminate all sources of ignition.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

Eliminate all sources of ignition. Pick up with suitable absorbent material and transfer to a container for chemical waste. For large amounts: dike spillage and pump off product into container for chemical waste. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep away from heat, sparks and flame. Avoid contact with eyes, skin and clothing. Avoid breathing vapour. Wash thoroughly after handling. Keep container closed. Use with adequate ventilation.

Storage

Keep container tightly closed. Store away from heat and light.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for diisobutyl adipate.

Engineering Controls

Good general ventilation should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation, or other engineering controls to maintain airborne levels below recommended exposure limits. If exposure limits have not been established, maintain airborne levels to an acceptable level.

Personal Protection Equipment

Respiratory Protection: If workers are exposed to concentrations above the exposure limit, they must use appropriate, certified respirators. If there are no applicable exposure limit requirements or guidelines, use an approved respirator. Selection of air-purifying or positive pressure supplied-air will depend on the specific operation and the potential airborne concentration of the product. For emergency conditions, use an approved positive-pressure self-contained breathing apparatus. The following should be an effective type of air-purifying respirator: organic vapour cartridge with a particulate pre-filter.

Hand Protection: Use gloves chemically resistant to this material. Consult the SDS for appropriate glove barrier materials.

Skin Protection: Use protective clothing chemically resistant to the material. Selection of specific items such as face shield, boots, apron or full body suit will depend on the task.

Eye Protection: Use chemical goggles.

Other Precautions: Wash hands, forearms and face thoroughly after handling chemical products; before eating, smoking and using the lavatory; and at the end of the working period. Appropriate techniques should be used to remove potentially contaminated clothing. Wash contaminated clothing before reusing. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

UN number: none

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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DIETHYLENE TRIAMINE PENTA(METHYLENE PHOSPONIC ACID), SODIUM SALT

This dossier on diethylene triamine penta(methylene phosphonic acid), sodium salt (DTPMP sodium salt) presents the most critical studies pertinent to the risk assessment of DTPMP sodium salt in coal seam or shale gas extraction activities. It does not represent an exhaustive or critical review of all available data. The information presented in this dossier was obtained primarily from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA), and from the OECD-SIDS documents on the Phosphonic Acid Compounds Group 3 category, which includes DTPMP and its sodium salts (OECD, 2004a,b). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): [bis[2-[bis(phosphonomethyl)amino]ethyl]amino]methylphosphonic acid; sodium salt

CAS RN: 22042-96-2

Molecular formula: $C_9H_{28}N_3O_{15}P_5.xNa$

Molecular weight: Not applicable. This substance is a UVCB substance.

Synonyms: Diethylene triamine penta(methylene phosphonic acid), sodium salt; [[(phosphonomethyl)imino]bis[(ethylenenitrilo)bis(methylene)]]tetrakisphosphonic acid, sodium salt phosphonic acid, ((bis(2-(bis(phosphonomethyl)amino)ethyl)amino)methyl)-, sodium salt; hepta sodium salt of diethylene triamine penta (methylene phosphonic acid)

SMILES: [Na+].OP(=O)(O)CN(CCN(CP(=O)(O)O)CP(=O)(O)O)CCN(CP(=O)(O)O)CP(=O)(O)[O-]

II. PHYSICO-CHEMICAL PROPERTIES

Key physical and chemical properties for the substance are shown in Table 1.

Table 1: Overview of the Physico-Chemical Properties of DTPMP (CAS-RN 15827-60-8) and DTPMP Sodium Salt

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Brown liquid	2	ECHA
Melting Point	>450°C (DTPMP) (pressure not provided)	1	ECHA
Boiling Point	>480°C (DTPMP) (pressure not provided_	1	ECHA
Density	1300 to 1400 kg/m^3 (DTPMP) @ 20°C	2	ECHA
Vapour Pressure	Negligible	2	ECHA
Partition Coefficient (log K_{ow})	-3.4 (DTPMP) (temperature not provided)	2	ECHA
Water Solubility	>520 g/L @ 25°C (DTPMP)	2	ECHA
Dissociation Constant (pKa)	1.03 – 12.58 (temperature not provided)	2	ECHA

DTPMP can ionise by loss of a hydrogen ion up to six times. Thus, it is a strong complexing agent and is highly hydrophilic. The sodium salts of DTPMP will dissolve readily in water to give a speciation state that is dictated by the pH of the aqueous medium. DTPMP has 10 possible ionisation states. Eight pK_a values were reported by Martell and Sillen (1968): 2.8, 4.45, 5.5, 6.38, 7.17, 8.15, 10.1, and 12.04, which were measured in 0.1 M potassium chloride. In a source giving no experimental details, DTPMP is described as having 10 pK_a values: 1.03, 2.08, 3.11, 4.15, 5.19, 6.23, 7.23, 8.30, 11.18, and 12.58 (Tomson et al., 1994).

At pH 7, DTPMP will be almost fully ionised in water five times, with a majority of the molecules ionised six times, and some seven or eight times.

DTPMP, sodium salt (CAS RN 22042-96-2) is a UVCB substance (unknown variable composition or biological substance) that can potentially have 1-10 sodium salts.

This dossier contains information on DTPMP (CAS RN 15827-60-8), as well as the sodium salts of DTPMP. The read-across of the acid to the sodium salts is justified because sodium is not significant with respect to the properties under consideration in this dossier. In dilute aqueous conditions of defined pH, a salt will be completely dissociated and will behave no differently to the parent acid, at the identical concentration of the particular speciated form present. Thus, some properties (measured or expressed in aqueous media) for a salt can be directly read-across (with suitable mass correction) to the parent acid and vice versa; the effect of the sodium ion, in this case, will not be significant. In biological systems and the environment, polyvalent metal ions will be present, and the phosphonate ions show very strong affinity to them (OECD, 2004b).

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

DTPMP sodium salt is not biodegradable, and it adsorbs strongly to sediment and soil. However, there are degradation modes operative in the environment which could prevent long-term persistence. DTPMP sodium salt has a low potential for bioaccumulation.

B. Partitioning

As discussed earlier, DTPMP acid and its salts behave in aqueous medium in accordance with the pH and composition of the medium. DTPMP acid and its salts will partition primarily to water and suspended sediments. It is highly soluble.

Photodegradation in the presence of common metal ions has been observed. Half-lives less than 1 hour were measured for sodium salt of DTPMP in water at pH 3, pH 5-6 and at pH 10, irradiated by a middle pressure mercury lamp emitting between 190 and 600 nanometres. Half-lives were found to be shorter in the presence of iron ions at environmentally relevant concentrations (Lesueur et al., 2005).

C. Biodegradation

In a Zahn-Wellens/EMPA (OECD 302B) test, there was no biodegradation after 28 days (ECHA) [Kl. score = 2]. There was also no biodegradation after 28 days in an OECD 301E test (ECHA) [Kl. score = 1].

Using [^{14}C]-DTPMP, there was 64% and 62.6% biodegradation in riverbank soil and silt loam soil, respectively, after 148 days (ECHA) [Kl. score = 2].

There are degradation modes operative in the environment that could prevent long-term persistence. For instance, although biodegradation in soil has not been demonstrated for DTPMP and its salts, the role of abiotic removal processes is significant. The key data for soil adsorption are from the study by Michael (undated). There is no evidence for desorption occurring. Effectively irreversible binding is entirely consistent with the known behaviour of complexation and binding within crystal lattices. Largely irreversible binding is interpreted as a removal process; 5% remaining after 40 to 50 days, which is equivalent to a half-life of 10 days (Monsanto internal report, cited by Gledhill and Feijtel, 1992). This abiotic removal rate is used in the chemical safety assessment of DTPMP and its salts. The available weight of evidence shows that removal from solution to a non-bioavailable bound form, and abiotic mechanisms, are important in the environmental exposure and risk assessment (ECHA).

D. Environmental Distribution

DTPMP sodium salt adsorbs strongly to inorganic surfaces, soils, and sediments. The nature of the adsorption is believed to be primarily due to interaction with inorganic substrates and not to organic carbon (OECD, 2004b).

A K_{oc} value of 9,748 was obtained for DTPMP by evaluating $K_{p(sediment\text{-}water)}$ data from a study by Michael (1979).

Based on this K_{oc} value and its solubility value (> 520 g/L), and assuming no biodegradability, if released to water DTPMP sodium salt will partition primarily to water and suspended sediments.

E. Bioaccumulation

DTPMP exhibits a low potential for bioaccumulation. After 28 days, the BCF values in carp were <10 and <94 for concentrations of 18.8 and 2.03 milligrams per litre (mg/L), respectively (ECHA). [Kl. score = 1]

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

The acute toxicity of DTPMP sodium salt is low by the oral and dermal routes. DTPMP sodium salt is slightly irritating to the skin and non-irritating to the eyes. DTPMP is not a skin sensitiser. Rats given repeated oral doses of DTPMP sodium salt in their diet showed alterations in iron and calcium homeostasis as evidenced by certain haematological changes and bone density, respectively. The changes in calcium homeostasis were not sufficient to alter serum calcium levels. While one *in vitro* genotoxicity study showed a positive response, other genotoxicity studies conducted both *in vitro* and *in vivo* showed no mutagenic or genotoxic response. Rat studies given high oral DTPMP sodium salt by oral gavage showed a low potential for reproductive and developmental toxicity.

B. Acute Toxicity

<u>Oral</u>

The oral LD_{50} of the heptasodium salt of DTPMP (CAS RN 68155-78-2) in rats was >10 mL/kg, which was calculated to be equivalent to >5,838 mg active salt/kilogram (kg) (ECHA) [Kl. score = 1]. The oral LD_{50} of a heptasodium salt of DTPMP was <15 mL/kg or <6,881 mg active salt/kg (ECHA) [Kl. score = 1]. The oral LD_{50} of a heptasodium salt of DTPMP was >5,000 mg/kg or >1,650 mg active salt/kg

(ECHA) [Kl. score = 2]. The oral LD_{50} of a heptasodium salt of DTPMP was >9,000 mg/kg or >3,870 mg active salt/kg (ECHA) [Kl. score = 2].

Inhalation

No inhalation studies are available.

Dermal

The dermal LD_{50} of a sodium salt of DTPMP was >10 mL/kg, which was calculated to be >5,838 mg active salt/kg or >4,602 mg parent acid/kg (ECHA) [Kl. score = 1]. The dermal LD_{50} of a sodium salt of DTPMP was >2,000 mg/kg, which was calculated to be >860 mg active salt/kg (ECHA) [Kl. score = 2]. The dermal LD_{50} of a heptasodium salt of DTPMP was >5 mL/kg, which was calculated to be >2,145 mg active salt/kg (ECHA) [Kl. score = 2].

C. Irritation

Application of 0.5 millilitres (mL) DTPMP sodium salt to the skin of rabbits for four hours under semi-occlusive conditions was only slightly irritating. The primary dermal irritation index was 0.75 (ECHA). [Kl. score = 1]

Instillation of 0.1 mL DTPMP sodium salt into the eyes of rabbits was not irritating (ECHA). [Kl. score = 1

D. Sensitisation

DTPMP sodium salt was not a skin sensitizer in a guinea pig maximization test (ECHA). [Kl. score = 2]

E. Repeat Dose Toxicity

Oral

Male and female Wistar rats were given 0, 100, 1,000, or 10,000 parts per million (ppm) DTPMP sodium salt in their diet for 90 days. The calculated daily intakes were: 0, 8.2, 82.3, and 841.9 mg/kg-day for males; and 0, 9.2, 92.3, and 902.6 mg/kg-day for females. There were no deaths during the study. At 10,000 ppm, minor changes were seen in haematological parameters (red blood cell count was significantly increased; mean cell volume and mean cell haemoglobin concentration were significantly decreased). Total serum iron was decreased in the 10,000 ppm females only, while total serum iron binding capacity was increased in the 10,000 ppm males only. A reduction in iron complexes and reduced pigmentation for age was noted in the spleens of the 10,000 ppm animals. The changes in haematological parameters and serum iron and binding capacity were considered by the study authors to be perturbations of iron homeostasis as a result of the iron binding capacity of DTPMP, which is a chelating agent. Bone density was significantly increased in both sexes in the 10,000-ppm dose group, and the incidence of microlithiasis (formation of minute calculi) in the kidney was reduced in all dose groups; these changes were considered indicative of the effect of the test material on calcium homeostasis due to its chelating ability. There was, however, no change in calcium plasma levels. The no observed adverse effect level (NOAEL) for this study is 1,000 ppm based on the changes in hematology and bond density; this corresponds to 82.5 and 92.3 mg/kg-day for males and females, respectively (ECHA, OECD 2002a). [Kl. score = 1]

Inhalation

No studies are available.

Dermal

No studies are available.

F. Genotoxicity

The results of the *in vitro* genotoxicity studies on DTPMP sodium salts are presented in Table 2.

In vitro Studies

Table 2: *In vitro* Genotoxicity Studies on DTPMP Sodium Salts

Test System	Results*		Klimisch Score	References
	-S9	+S9		
Bacterial reverse mutation (S. typhimurium and E. coli strains)	-	-	1	ECHA, OECD (2004a, b)
Mammalian cell gene mutation (mouse lymphoma L5178Y cells)	-	-	2	ECHA, OECD (2004a, b)
Chromosomal aberration (Chinese hamster lung cells)	**	**	1	ECHA

*+, positive; -, negative
**For the 6-hour pulse treatment and the 24-hour continuous treatment, the results were negative with and without metabolic activation. For the 48-hour continuous treatment, the results were positive, but no information is provided on whether this occurred with and/or without metabolic activation.

In vivo Studies

Male and female SD rats were given a single oral dose of an aqueous solution containing 19.7% DTPMP sodium salt (neutralized to pH 7) at doses of 0, 200, 660, and 1,970 mg active acid/kg. At the high dose, 25% of the animals died, and there were mild clinical signs of toxicity and reduced body weights in both sexes. There was no evidence of chromosomal aberrations in the bone marrow cells in either sex at any dose level (ECHA; OECD, 2004a,b). [Kl. score = 2]

G. Carcinogenicity

No studies are available.

H. Reproductive Toxicity

A reproductive toxicity study was conducted on DTPMP sodium salt in rats via the diet. The females were treated over two generations and males over one generation. concentrations were 0, 300, 1,000, and 3,000 ppm; the daily intakes were calculated to be 0, 28, 97, and 294 mg/kg-day for males and 0, 32, 108, and 312 mg/kg-day for females. The 3,000 ppm F0 females delivered fewer live pups with lower body weights (both effects were not statistically significant). Pregnancy rate (not statistically significant) and a reduced pup weight (statistically significant) was seen in the F2a litters from the 3,000 ppm dams. These changes were not seen in the F1 litters or replicated in the F2b

litters. The NOAEL for reproductive toxicity was determined to be 3,000 ppm, which corresponds to 294 and 312 mg/kg-day for males and females, respectively (ECHA; OECD 2004a,b). [Kl. score = 2]

A three-generation reproductive toxicity study was conducted on DTPMP sodium salt in rats via the diet. The concentrations were 0, 300, 1,000, and 3,000 ppm. There was no systemic, reproductive or developmental toxicity at any dose level. The NOAEL for this study is 3,000 ppm, which was calculated to be 275 mg/kg-day for males and 310 mg/kg-day for females (ECHA). [Kl. score = 2]

I. Developmental Toxicity

Pregnant female SD rats were dosed by oral gavage with 0, 500, 1,000, or 2,000 mg/kg DTPMP during GD 6-15. Toxicity was observed in the 2,000 mg/kg dams as evidenced by an approximate 30% decrease in body weight gain and by the appearance of soft stools. There was no developmental toxicity. The NOAELs for maternal and developmental toxicity were 1,000 and 2,000 mg/kg-day, respectively (ECHA; OECD 2004a,b). [Kl. score = 2]

Pregnant female CD-1 mice were dosed by oral gavage with 0, 100, 500, or 1,000 mg/kg DTPMP during GD 6-15. There were no maternal or developmental toxicity. The NOAEL for maternal and developmental toxicity is 1,000 mg/kg-day, the highest dose tested (ECHA). [Kl. score = 2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for DTPMP follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

A 90-day dietary study was conducted on a sodium salt of DTPMP using rats. The rats of both sexes showed changes in hematology and bone density that were indicative of alterations in iron and calcium homeostasis, due to the chelating ability of DTPMP. The NOAEL for this study was 10,000 ppm, which corresponds to 82.5 and 92.3 mg/kg-day for males and females, respectively. The NOAEL of 82.5 mg/kg-day will be used for determining the oral Reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:
UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 3
UF_D (database uncertainty) = 1

Oral RfD = 82.5/(10 x 10 x 1 x 3 x 1) = 82.5/300 = 0.3 mg/kg-day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:
Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)

Drinking water guidance value = (0.3 x 70 x 0.1)/2 = 1.0 mg/L

B. Cancer

There are no carcinogenicity studies on DTPMP and its sodium salts. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

DTPMP sodium salt does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

DTPMP and its sodium salts are of low toxicity concern to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

No acute toxicity studies are available for the sodium salts of DTPMP. Table 3 lists the results of acute aquatic toxicity studies on DTPMP.

Table 3: Acute Aquatic Toxicity Studies on DTPMP

Test Species	Endpoint	Results (mg active acid/L)	Klimisch Score	Reference
Oncorhynchus mykiss	96-hour LC_{50}	180 - 252 (mean: 216)	2	ECHA
Chironomus tentans	48-hour EC_{50}	7,589	2	ECHA

Algal studies have also been conducted on DTPMP and its sodium salts, but the results have not been provided because of the following confounding factors:

1. Algal growth may be stimulated by the presence of supplementary phosphorus released by the photolytic degradation of phosphonic acids.
2. Algal growth may be inhibited by the complexation of micronutrients (trace metals) by phosphonic acids. This inhibition is an algistatic rather than algicidal effect. Under the standard test conditions used for most studies, the trace metals will be fully and strongly bound to the DTPMP, with the strong possibility that their bioavailability will have been reduced considerably.

Chronic Studies

The 60-day NOEC of DTPMP in *Oncorhynchus mykiss* was determined to be 25.6 mg active acid/L (ECHA). [Kl. score = 1]

The value of 25.6 mg equivalent active acid/L can be converted to units of mg DTPMP-xNa salt/L at relevant conditions of pH by considering the ionisation state of DTPMP (CAS No. 15827-60-8) at the 25.6 mg/L concentration. At pH 6 (the expected value of the test medium), DTPMP is ionised six times (pK_a6 = pH 6.23). Also for the calculation, the number of hydrogen atoms substituted by the sodium salt is removed, which is seven. The calculation is as follows:

MW of DTPMP-7Na / MW of DTPMP = 573.2 + ((21.982 – 1.008) x 6) / 573.2 = 1.22

25.6 mg DTPMP/L x 1.22 = 31 mg DTPMP-xNa/L

C. Terrestrial Toxicity

The 14-day dietary LC_{50} values to the Mallard duck (*Anas platyrhynchos*) and Bobwhite quail (*Colunus virginianus*) are >454 mg/kg; there was no mortality at the highest dose tested (OECD, 2004a, b).

D Calculation of PNEC

The PNEC calculations for DTPMP sodium salt follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for two trophic levels for DTPMP, but not for the sodium salts of DTPMP. Acute E(L)C50 values are available for fish (216 mg/L) and invertebrates (7,589 mg/L). Results are available for a fish chronic study (31 mg DTPMP sodium salt/L). On the basis that the data consists of short-term results from two trophic levels and long-term results from onetrophic level, an assessment factor of 100 has been applied to the chronic NOEC of 31 mg/L for fish. The $PNEC_{water}$ is 0.31 mg DTPMP sodium salt/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 46 mg DTPMP sodium salt/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (188/1280) \times 1000 \times 0.31$
$= 46$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [0.2 \times (Kp_{sed}/1000) \times BD_{solid}]$
$= 0.8 + [0.2 \times (390/1000) \times 2400]$
$= 188\ m^3/m^3$

Where:
Kps_{ed} = solid-water partition coefficient (L/kg).
BD_{solid} = bulk density of the solid phase (kg/m3) = 2,400 [default]
$Kp_{sed} = K_{oc} \times f_{oc}$
$= 9,748 \times 0.04$
$= 390$ L/kg

Where:
K_{oc} = organic carbon normalized distribution coefficient (L/kg). The K_{oc} for DTPMP was estimated to be 9,748 L/kg (OECD 2004a,b).

F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 40 mg DTPMP sodium salt/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
$= (195/1500) \times 1000 \times 0.31$
$= 40$ mg/kg

Where:
Kpsoil = soil-water partition coefficient (m^3/m^3)
BDsoil = bulk density of soil (kg/m3) = 1,500 [default]

$Kp_{soil} = K_{oc} \times f_{oc}$
$= 9,748 \times 0.02$
$= 195\ m^3/m^3$

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for DTPMP was estimated to be 9,748 L/kg (OECD 2004a,b).
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

DTPMP and its sodium salts are not readily biodegradable; thus, they meet the screening criteria for persistence.

The BCF values from a fish study are <10 and <94 for concentrations of 18.8 and 2.03 mg/L, respectively. Thus, DTPMP sodium salt does not meet the screening criteria for bioaccumulation.

The NOEC from a chronic fish study on DTPMP is >0.1 mg/L. Thus, DTPMP and its sodium salts do not meet the screening criteria for toxicity.

The overall conclusion is that DTPMP sodium salt is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Metal Corrosive Category 1

B. Labelling

Warning

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Ingestion

Rinse mouth with water and then drink plenty of water. Never give anything by mouth to an unconscious person.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide carbon dioxide nitrogen oxides phosphorus oxides, phosphine.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilled

Soak up with inert absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for DTPMP sodium salt.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

UN 3265 CORROSIVE LIQUID, ACIDIC, ORGANIC N.O.S. (diethylene triamine penta(methylene phosphonic acid) sodium salt)

Class: 8

Packing Group: III

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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ETHYLENE OXIDE/PROPYLENE OXIDE COPOLYMER (CAS RN 9003-11-6)
ETHYLENE OXIDE/PROPYLENE OXIDE COPOLYMER (CAS RN 9082-00-2)
2-ETHYLHEXANOL EO/PO POLYMER (CAS RN. 64366-70-7)

This group contains information on ethylene oxide/propylene oxide copolymers (CAS RN 9003-11-6 and CAS RN 9082-00-2) and 2-ethylhexanol EO/PO polymer (CAS RN 64366-70-7). They are expected to have similar environmental concerns and have consequently been assessed as a group.

This dossier presents the most critical studies pertinent to the risk assessment of EO/PO copolymer in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the Cosmetic Ingredient report (CIR, 2008) , the Dow Company report (Dow, 2014) and ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed poloxalene (CAS RN 9003-11-6) in an IMAP Tier 1 assessment and considers it a polymer of low concern[1]. AICIS has assessed oxirane, methyl-, polymer with oxirane, mono(2-ethylhexyl) ether (CAS RN 64366-70-7) and also considers it a polymer of low concern.[2]

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Oxirane, methyl-, polymer with oxirane

CAS RN: 9003-11-6

Molecular formula: $(C_3H_6O.C_2H_4O)_x$-

Molecular weight: Variable (polymer)

Synonyms: ethylene oxide, propylene oxide block polymer; poloxalene; poloxamer; polyethylene glycol, propoxylated; polyethylene-polypropylene glycol; polyoxyethylene-oxy-propylene; oxirane, 2-methyl-, polymer with oxirane; oxirane, methyl-, polymer with oxirane

SMILES: Not applicable

The generic CAS RN 9003-11-6 refers to polymers that are synthetic block copolymers of ethylene oxide and propylene oxide. There are over 50 various amphiphilic non-ionic block polymers of hydrophobic propylene oxide (PO) and hydrophilic ethylene oxide (EO) (CIR, 2008). These copolymers consist of a central polyoxypropylene molecule, flanked on both sides by two hydrophilic polyoxyethylene chains.

EO/PO copolymers are also known as Poloxamers.

Chemical Name (IUPAC): Oxirane, methyl-, polymer with oxirane, ether with 1,2,3-propanetriol (3:1)

[1] https://www.nicnas.gov.au/chemical-information/imap-assessments/how-chemicals-are-assessed/Low-concern-polymers.
[2] https://www.industrialchemicals.gov.au/sites/default/files/2022-05/EVA00086%20-%20Evaluation%20statement%20-%2030%20May%202022.pdf

CAS RN: 9082-00-2

Molecular formula: $C_3H_8O_3.3(C_3H_6O.C_2H_4O)_x$-

Molecular weight: Variable (polymer)

Synonyms: Ethylene oxide-propylene oxide copolymer ether with glycerol (3:1); ethylene oxide-propylene oxide copolymer glycerol ether; glycerol, ethylene oxide, propylene oxide polymer;

glycerol poly (oxyethylene, oxypropylene) ether; propylene oxide ethylene oxide polymer, ether with glycerol (3:1); glycerol, propylene oxide, ethylene oxide polymer.

SMILES: Not applicable

Chemical Name (IUPAC): Oxirane, methyl-, polymer with oxirane, mono(2-ethylhexyl) ether

CAS RN: 64366-70-7

Molecular formula: C_8-H_{18}-O. (C_3H_6-O.C_2-H_4-O)$_x$-

Molecular weight: 232.35 g/mol (monomer); variable (polymer)

Synonyms: 2-ethylhexanol EO/PO polymer; oxirane, methyl-, polymer with oxirane, monoether with 2-ethylhexanol; oxirane, 2-methyl-, polymer with oxirane, mono(2-ethylhexyl) ether; oxirane, methyl-,polymer with oxirane, mono(2-ethylhexyl) ether2-((1-((2-ethylhexyl)poly-oxy)poly-propan-2-yl)oxy)ethanol; PEG-14 PPG-7 ethylhexyl ether; PEG-3 PPG-7 ethylhexyl ether; PEG-6 PPG-7 ethylhexyl ether; PEG-9 PPG-7 ethylhexyl ether.

SMILES: not applicable

II. PHYSICO-CHEMICAL PROPERTIES

The physico-chemical properties of the EO/PO copolymers are listed in Table 1.

Table 1 Overview of the Physico-chemical Properties of Selected EO/PO Copolymers (CIR, 2008)

Properties	Poloxamer 124	Poloxamer 188	Poloxamer 407
Avg. molecular weight (g/mol)	2090-2360	7680-9510	9840-14600
Description	Colourless liquid	White solid	Solid
Wt. % oxyethylene	46.7 $\pm$ 1.9	81.8 $\pm$ 1.9	73.2 $\pm$ 1.7
Melting point (^{o}C)	16	52	56
Solubility	Soluble in water	Soluble in water	Soluble in water

The Dow Chemical Company's Product Safety Assessment document (Dow, 2014) on their EO/PO copolymer products with CAS RN 9003-11-6 and CAS RN 53637-25-5 states the following: "Polyglycol EP Series Polymers are liquid polyalkylene glycol block copolymers that are colorless to yellow in appearance and odorless or with a mild, ether odor."

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

No studies are available.

The following information is from the Dow Chemical Company's Product Safety Assessment document on their EO/PO copolymer products with CAS RN 9003-11-6 and CAS RN 53637-25-5 (Dow, 2014):

> "Polyglycol EP Series Polymers are non-volatile (do not evaporate) and vary in water solubility. If released to water or soil, they would tend to remain in and be transported with the surface or ground water to which they are emitted and will be adsorbed to soil and sediment particles. Polyglycol EP Series Polymers are unlikely to persist in the environment, as all products are known or expected to be either readily biodegradable (>65% biodegraded in 28 days per OECD 301F test) or inherently biodegradable according to Organisation for Economic and Co-operation and Development (OECD) test guidelines. As such, these products will be efficiently removed during treatment in biological wastewater-treatment facilities.
>
> These products are not expected to accumulate in the food chain (low bioconcentration potential)."

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

The acute toxicity of EO/PO polymers are very low by the oral route. These polymers are not skin irritants or sensitisers. No systemic toxicity was observed in rats given very high oral doses of EO/PO polymers for up to two years. A slight inflammation response was seen in rats that inhaled a very high concentration of an aerosol or dust of these polymers over a two-week period. Repeated dermal applications of an EO/PO polymer to the skin of rabbits produced a slight irritating response, but no systemic toxicity. An EO/PO polymer was not mutagenic when tested in a bacterial reverse mutation assay. No studies are available to evaluate reproductive or developmental toxicity.

B. Acute Toxicity

The oral LD_{50} values in rats for Poloxamer 124, 182, 188, and 235 were 5,000, 5,500, >15,000, and 34,600 mg/kg (Leaf, 1967). No acute dermal or inhalation studies were located.

C. Irritation

The EO/PO copolymers are not skin irritants to laboratory animals or humans (CIR, 2008).

D. Sensitisation

The EO/PO copolymers are not dermal sensitiser (CIR, 2008).

E. Repeated Dose Toxicity

Oral

Rats were fed diets containing 0, 3, or 5% Poloxamer 188 for 6 months. During the study, 2 and 14 animals died in the mid- and high-dose groups, respectively. Deaths were attributed to a combination of infection and inanition. There were no histopathologic effects that were considered to be treatment related (Leaf, 1967).

Rats were fed diets containing Poloxamers 331, 235, or 338 for 90 days. The doses were: 40, 200, or 500 mg/kg Poloxamer 331; 40, 200, or 500 mg/kg Poloxamer 235; 200, 1,000 or 5,000 mg/kg Poloxamer 338. There was no treatment-related mortality. The rats in the 5,000 mg/kg Poloxamer 338 dose group had diarrhoea. No other details were given (Leaf, 1967).

Rats were fed diets containing 0, 3, 5, or 7.5% Poloxamer 188 for two years. There was no treatment-related mortality. At the two higher doses, the rats had continuous moderate diarrhoea, but not other adverse reactions. A small decrease in growth was seen in the 7.5% group (no statistical analysis and not information on the amount of change), but there were no treatment-related histopathological effects at any dose level. The NOAEL for this study is 5% in the diet. Using 0.05 as the fraction of body weight that rats consume per day as food (U.S. EPA), the NOAEL corresponds to 2,500 mg/kg-day (Leaf, 1967).

Male and female rats were fed diets containing 0, 40, 200, or 500 mg/kg Poloxamer 182 for two years. Deaths occurred in all groups of rats due to chronic respiratory infections unrelated to administration of Poloxamer 182. There were no clinical signs of toxicity, and blood and urine chemistry parameters were comparable across all groups. There were no gross pathological changes noted. It is unclear from the summary in CIR (2008) whether a histopathologic examination was conducted. The NOAEL for this study is 500 mg/kg-day (Leaf, 1967).

Inhalation

Male SD rats were exposed by inhalation to 0 or 97 mg/m3 Poloxamer 101 aerosol for 6 hours/day, 5 days/week over a two-week period. A separate group of rats was exposed for two weeks followed by a two-week recovery period. All animals survived until the end of the study. The only adverse effect observed was slight alveolitis in the Poloxamer 101-exposed rats, which subsided by the end of a two-week recovery (Ulrich et al., 1992).

Dermal

New Zealand rabbits were given dermal applications of 0, 100, 300 or 1,000 mg/kg Poloxamer 184 5 days/week for a total of 20 applications. The skin of the treated animals showed slight intradermal inflammatory responses, but no systemic effects (CIR, 2008).

F. Genotoxicity

Poloxomer 407 was not mutagenic to S. typhimurium strains TA97, TA98, TA100, TA1535, and TA1537 in the absence and presence of metabolic activation (CIR, 2008).

G. Carcinogenicity

Oral

There are no studies available.

Inhalation

There are no studies available.

Dermal

There are no studies available.

H. Reproductive Toxicity

There are no studies available.

I. Developmental Toxicity

Oral

There are no studies available.

Inhalation

There are no studies available.

Dermal

There are no studies available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for EO/PO copolymers follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

EO/PO copolymers have been tested in two chronic rat dietary studies. In the first study, the only effect observed was slightly reduced growth in the rats fed 7.5% (3,750 mg/kg-day) EO/PO copolymer; no effects were seen in the 5% (2,500 mg/kg-day) and lower dose groups. No statistical analysis was provided on whether the change in body weight gain was statistically significant, or whether the change is of sufficient magnitude to be considered an adverse effect. For the purposes of this risk assessment, 3,750 and 2,500 mg/kg-day will be considered a LOAEL and NOAEL, respectively. In the second feeding study, there were no effects seen in the rats at oral doses up to 500 mg/kg-day (highest dose tested).

The NOAEL of 2,500 mg/kg-day EO/PO copolymer in the diet will be used to derive an oral reference dose (RfD) and a drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD =2,500 / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1
Oral RfD = 2,500/ (10 x 10 x 1 x 1 x 1) = 2,500/100 = 25 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (25 x 70 x 0.1)/2 = 88 mg/L

B. Cancer

No carcinogenicity studies were located. Therefore, a toxicological reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

EO/PO copolymers do not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

EO/PO copolymers are practically acutely non-toxic to aquatic organisms.

B. Aquatic Toxicity

No studies are available.

The following information is from the Dow Chemical Company's Product Safety Assessment document on their EO/PO copolymer products with CAS RN 9003-11-6 and 53637-25-5 (Dow, 2014):

"[EO/PO copolymers] are practically non-toxic to aquatic organisms (LC_{50}/EC_{50} >100 mg/L for the most sensitive species tested) on an acute basis."

C. Terrestrial Toxicity

There are no studies available.

D. Calculation of PNEC

The PNEC calculations for EO/PO copolymers follow the methodology discussed in DEWHA (2009).

PNEC Water

No experimental studies were found. However, Dow Chemical's Product Safety Assessment document on their EO/PO copolymers indicates that acute toxicity testing has been conducted on these copolymers and the E(L)C50 value for the most sensitive species is >100 mg/L. On the basis of the short-term results, an assessment factor of 1,000 has been applied to the lowest reported effect concentration of 100 mg/L. The $PNEC_{water}$ is 0.1 mg/L.

PNEC Sediment

A $PNEC_{sed}$ value was not calculated for EO/PO copolymers. There are no experimental toxicity data on sediment organisms and a K_{oc} value EO/PO copolymer is unavailable for calculating the $PNEC_{sed}$ using the equilibrium partition method. A K_{oc} value for the EO/PO polymers has not been determined experimentally, and QSAR models are invalid for high molecular weight polymers, such as EO/PO polymers.

PNEC Soil

A $PNEC_{soil}$ value was not calculated for EO/PO copolymers. There are no experimental toxicity data on soil organisms and a K_{oc} value for EO/PO copolymer is unavailable for calculating the $PNEC_{soil}$ using the equilibrium partition method. A K_{oc} value for the EO/PO copolymers has not been determined experimentally, and QSAR models are invalid for high molecular weight polymers, such as EO/PO polymers.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

EO/PO copolymers are readily biodegradable or inherently biodegradable and thus does not meet the screening criteria for persistence.

EO/PO copolymers are expected to have high molecular weights and are not expected to be bioavailable. Thus, the copolymers do not meet the criteria for bioaccumulation.

There are no chronic aquatic toxicity studies on EO/PO copolymers. However, the acute E(L)C50 on these copolymers are >1 mg/L in aquatic organisms based on information from Dow Chemical's

Product Safety Assessment (Dow, 2014). EO/PO copolymers also have a high molecular weight and are not expected to be bioavailable. Thus, they do not meet the screening criteria for toxicity.

The overall conclusion is that EO/PO copolymers are not PBT substances.

IX. CLASSIFICATION AND LABELLING

A. Classification

H412-Aquatic Chronic 3

B. Labelling

Warning

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide and unburned hydrocarbons (smoke).

Dust can accumulate static charges which can cause an incendiary electrical discharge. Fine dust dispersed in air in sufficient concentrations, and in the presence of an ignition source, is a potential dust explosion hazard.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

There are no workplace exposure standards available for EO/PO copolymers in Australia.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

EO/PO copolymers are not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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ETHYLENE GLYCOL

This dossier on ethylene glycol presents the most critical studies pertinent to the risk assessment of ethylene glycol in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed ethylene glycol in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Ethane-1,2-diol

CAS RN: 107-21-1

Molecular formula: $C_2H_6O_2$ ($HOCH_2CH_2OH$)

Molecular weight: 62.07 g/mol

Synonyms: Ethylene glycol; ethane-1,2-diol; 1,2-ethanediol, 2-hydroxyethanol; monoethylene glycol; MEG; glycol alcohol; EG

SMILES: C(CO)O

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-Chemical Properties of Ethylene Glycol

Property	Value	Klimisch Score	Reference
Physical state at 20oC and 101.3 kPa	Colourless and odourless syrupy liquid	2	ECHA
Melting Point	-13^{o}C @ 101.3 kPa	2	ECHA
Boiling Point	197.4^{o}C @ 101.3 kPa	2	ECHA
Density	1110 kg/m3@ 20^{o}C	2	ECHA
Vapour Pressure	12.3 Pa @ 25^{o}C	2	ECHA
Partition Coefficient (log K_{ow})	-1.36 (calculated) @ 25^{o}C	2	ECHA
Water Solubility	1000 g/L @ 20^{o}C	2	ECHA
Flash Point	111^{o}C	2	ECHA
Auto flammability	398^{o}C	2	ECHA
Viscosity	16.1 mPa s @ 25^{o}C	2	ECHA
Henry's Law Constant	0.133 @ 25^{o}C (QSAR)	2	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Ethylene glycol is readily biodegradable, and it is not expected to bioaccumulate. Ethylene glycol has low potential to adsorb to soil and sediment.

B. Biodegradation

Ethylene glycol was readily biodegradable in an OECD 301A test. After 10 days, degradation was 90-100% (ECHA) [Kl. score = 1]. There was 97% degradation after 20 days in a BOD test; and 96% degradation after 28 days in an OECD 301D test (Waggy et al., 1994; OECD, 2004a,b) [Kl. score = 2]. If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

The aerobic degradation of ethylene glycol was measured from grab river water samples at 4, 8 and 20°C. At 20°C, ethylene glycol was completely degraded in three days in all river waters tested; at 8°C, degradation was complete within 14 days. Degradation at 4°C was substantially slower, with degradation of < 20% after 14 days in river samples with limited suspended matter and a starting concentration of 10 mg/L (Evans and David, 1974).

C. Environmental Distribution

No experimental data are available for ethylene glycol. Using KOCWIN in EPISuite™ (USEPA, 2017), the estimated K_{oc} values from the molecular connectivity index (MCI) and from the log K_{ow} are 1 and 0.2239 L/kg, respectively.

Based upon these K_{oc} values, if released to soil, ethylene glycol is expected to have low potential for adsorption and a high potential for mobility. If released to water, based on its K_{oc} and high water solubility values, ethylene glycol is likely to remain in water and not adsorb to sediment. From the water surface, the substance will not evaporate into the atmosphere (ECHA).

D. Bioaccumulation

The calculated log K_{ow} for ethylene glycol is -1.36 (ECHA). The BCF for ethylene glycol in golden ide (*Leuciscus idus melanotus*) after three days of exposure was determined to be 10 (Freitag *et al.*, 1985). Bioaccumulation is not to be expected.

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Following acute ingestion of ethylene glycol, the critical effects in humans in three subsequent stages are central nervous system toxicity, metabolic acidosis and kidney toxicity. The lethal effects of ethylene glycol in human adults occur at oral doses of $\geq$ 1,600 mg/kg. Ethylene glycol is not a skin irritant or a skin sensitiser in laboratory animals. In humans, ethylene glycol may cause skin irritation; there is also a low potential for skin sensitisation. It is not an eye irritant. The kidney is the primary target organ from repeated exposures. The proposed mode-of-action (MOA) for the kidney damage involves the formation of a precipitate or crystals from the ethylene glycol metabolite oxalic acid with calcium in the urine. Ethylene glycol is not genotoxic or carcinogenic to rodents. Ethylene glycol did not affect fertility in animal studies, but it did cause developmental effects. In rodents, the

developmental effects caused by oral doses of ethylene glycol include teratogenic effects (craniofacial and axial-skeletal malformations and variations). In contrast, no developmental toxicity was seen in rabbit studies. The relevant metabolite for the developmental toxicity seen in rodent, but not rabbit, studies appears to be glycolic acid. This metabolite can be reached at higher concentrations in rats than in rabbits. Based on a physiologically-based pharmacokinetic (PBPK) model for ethylene glycol, humans are unlikely to achieve blood levels of glycolic acid necessary for developmental toxicity.

B. Metabolism

Ethylene glycol is almost completely absorbed in laboratory animals by the oral route (OECD, 2004a; Frantz et al., 1996a). A range of 1-51% of ethylene glycol is absorbed by the dermal route based on *in vivo* studies in rodents (Frantz et al., 1996a,b).

The main metabolic pathway for metabolism of ethylene glycol is oxidation via alcohol dehydrogenases and aldehyde dehydrogenases. The main metabolites of ethylene glycol are carbon dioxide, oxalic acid and glycolic acid (OECD, 2004a).

The relevant metabolite for the repeated dose toxicity studies is oxalic acid, which is slowly transported from the liver to the kidneys, where is forms calcium-oxalate crystals (Corley et al., 2005a).

The relevant metabolite for the developmental toxicity seen in rodent, but not rabbit, studies appears to be glycolic acid. This metabolite can be reached at higher concentrations in rats than in rabbits (Carney et al., 1998).

A physiologically-based pharmacokinetic (PBPK) model has been developed for ethylene glycol. When internal dose surrogates were compared in rats and humans over a wide range of exposures, it has been concluded that humans are unlikely to achieve blood levels of glycolic acid necessary for developmental toxicity (Corley et al., 2005b).

C. Acute Toxicity

The oral LD_{50} in rats was reported to be 7,712 mg/kg (ECHA) [Kl. score = 2]. The 6-hour inhalation LC_{50} value for male and female rats was > 2.5 mg/L (Tyl et al., 1995a) [Kl. score = 2]. The dermal LD_{50} for male and female mice is > 3,500 mg/kg (Tyl et al., 1995b) [Kl. score = 2].

Following acute ingestion of ethylene glycol, the critical effects in humans in three subsequent stages are central nervous system toxicity, metabolic acidosis and kidney toxicity (ECHA). The lethal effects of ethylene glycol in human adults occur at oral doses of $\geq$ 1,600 mg/kg (Hess et al., 2004).

D. Irritation

Application of 0.5 mL of ethylene glycol to the skin of rabbits for 23 hours under occlusive conditions was not irritating (Guillot et al., 1982) [Kl. score = 2].

In a Human Repeated Insult Patch Test (HRIPT), ethylene glycol was applied to the skin for 24 hours under occlusive or semi-occlusive conditions for nine times during the induction phase. The induction phase was followed by a rest period of two weeks, followed by a 24-hour challenge on the sixth week of the study. Erythema was seen in a small proportion of the 401 subjects that completed the study. Under the conditions of the study, three subjects had reactions on challenge that were

indicative of possible irritation and/or low-level sensitisation. These three subjects were re-challenged under occlusive or semi-occlusive conditions one or two weeks later. Re-challenge testing was negative for one subject, but the other two subjects were judged to have irritant reactions to ethylene glycol since their reactions were similar or lesser compared to the skin responses observed during the induction period, and the skin reactions were not greater over time after the challenge or re-challenge (ECHA).

Instillation of 0.05 mL of ethylene glycol into the eyes of rabbits was not irritating (ECHA) [Kl. score = 2].

E. Sensitisation

Ethylene glycol was not a skin sensitiser to guinea pigs in a Magnusson and Kligman test (Kurihara et al., 1996) [Kl. score = 2]. In a HRIPT, ethylene glycol was considered to have a low potential for dermal sensitisation in humans (ECHA).

F. Repeated Dose Toxicity

Oral

Male and female Fischer 344 rats were given in their feed 0, 0.32, 0.63, 1.25, 2.5 or 5% ethylene glycol for 13 weeks. Mortality was seen in the 5% males, but not in females. Mean weight gain was significantly decreased in the 2.5 and 5% males; there was no significant differences in female rats. Feed consumption was similar across all groups. A significant increase was seen in the left kidney weight in the 2.5 and 5% dose groups (both sexes); this was not seen in the right kidneys. Mean thymus ratio to terminal body weight was significantly decreased in the 5% males. Serum urea nitrogen levels were significantly increased in the 2.5 and 5% males, and significantly increased in the ≥ 0.32% females. Creatinine levels were decreased in the 0.32% groups and significantly increased in the 2.5 and 5% groups. The 2.5% and 5% male rats had kidneys that were rough, granular and/or pitted appearances. The 5% females showed nephrosis, and the 5% males had clusters of crystals in the brain. The NOAEL for this study is 1.25%, which was estimated to be 600 to 1,000 mg/kg/day (Melnick, 1984) [Kl. score = 2]

Male and female Sprague Dawley rats were given in their drinking water ethylene glycol for 90 days. The concentrations for females were 0, 0.5, 1.0, 2.0 or 4.0% (0, 597, 1,145, 3,087 or 5,744 mg/kg/day). The concentrations for males were 0, 0.25, 0.5, 1.0 or 2.0% (0, 205, 407, 947 or 3,134 mg/kg/day). In the 4% groups, there was mortality and decreased body weights (males only). Significant organ weights were noted only in males. Kidney weights were significantly increased in the 1% and 2% males; heart, liver and lung were significantly decreased in the 2% males. The 4% males also had a significant increase in the brain and gonads relative to body weights. Leukocyte levels were significantly decreased in the 0.5, 2 and 4% females, but not in males. Significant differences were noted in LDH, creatinine, ALT, calcium and glucose in the 1% males; and phosphorus, BUN and creatinine in the 2% males. There were significant increases in phosphorus in the 1% females and glucose in the 0.5 and 4% females. Kidney lesions were seen in the ≥ 2% females and in the ≥ 1% males, with the lesions more prominent in males than in females. The kidney changes consisted of tubular dilation, tubular degeneration, acute inflammation, birefringent crystals in tubules and pelvic epithelium. The NOAEL for this study is 407 mg/kg/day for males. The LOAEL for females is 597 mg/kg/day; a NOAEL was not established (Robinson et al., 1990) [Kl. score = 2]

Male and female B6C3F$_1$ mice were given in their feed 0, 0.32, 0.63, 1.25, 2.5 or 5.0% ethylene glycol for 13 weeks. There was no mortality and no treatment-related effect on mean weight gain and feed consumption. Organ/body weight ratios were similar across all groups. Serum urea nitrogen and creatinine levels were unaffected. Kidney effects were seen in the male, but not female, mice. Kidney lesions were observed in half of the 5% male mice and one mouse in the 2.5% dose level. Lesions were tubular dilation, cytoplasmic vacuolisation and regenerative hyperplasia of tubular cells. There was no evidence of crystal formation in the tubules. These changes were focal, randomly distributed and of minimal to mild severity. Hyaline degenerative of the liver was present in the centrilobular hepatocytes in all of the 2.5% and 5% males. These cells showed cytoplasmic accumulations of non bifringent, eosinophilic (hyaline), globular or crystalline material which resembled erythrocytes in size, shape and tinctorial properties. The NOAEL for this study is 1.25%, which was estimated to be 600 to 1,000 mg/kg/day (Melnick, 1984) [Kl. score = 2].

Male Fischer 344 and Wistar rats were given in their feed 0, 150, 500 or 1,000 mg/kg ethylene glycol for 16 weeks. At 1000 mg/kg, the following effects were seen: mortality in Wistar strain (2/10) with prior clinical observations of emaciation and dermal atonia and macroscopic findings of changes in kidneys (pale, calculi) and small seminal vesicles in these animals; mean body weight losses, lower mean body weights and mean cumulative body weight changes in Wistar strain (weeks 2 – 16); lower mean food consumption in Wistar strain; higher mean water consumption in both F344 and Wistar strains; lower mean specific gravity and higher mean total urine volume in both F344 and Wistar strains; macroscopic findings of pale kidneys, presence of calculi, rough surface and dilated pelvis; higher mean absolute and relative kidney weights in both F344 and Wistar strains; renal macroscopic findings of crystal nephropathy in Wistar and F-344 rats, with more severe nephropathy in Wistar strain than in the F344 strain. At 500 mg/kg, the following effects were seen: lower mean body weights (study weeks 3, 6-8 and 10-12) and mean cumulative body weight changes in the Wistar strain throughout the study with slightly lower mean food consumption throughout the study; higher mean water consumption in the Wistar strain; lower mean urine specific gravity and higher mean total urine volume in the Wistar strain; macroscopic findings in the Wistar strain consisting of predominantly pale kidneys, presence of calculi, rough surface and dilated pelvis; higher mean absolute and relative kidney weight in the Wistar strain; renal macroscopic findings of crystal nephropathy in Wistar and F-344 strains, with more severe nephropathy in the Wistar strain than in the F344 strain. The NOAEL in both the F344 and Wistar rats is 150 mg/kg/day (Cruzan et al., 2004) [Kl. score = 2].

Male Wistar rats were given in their feed 0, 50, 150, 300 or 400 mg/kg ethylene glycol for 12 months. There was mortality in the 300 and 400 mg/kg dose groups (5/20 and 4/20, respectively); the remaining 400 mg/kg animals were euthanised early (Day 203) due to excessive weight loss. The 300 mg/kg animals had increased water consumption and urine volume with decreased specific gravity, most likely due to osmotic diuresis. Calculi (calcium oxalate crystals) were found in the bladder and kidney pelvis in the ≥ 300 mg/kg animals. The ≥ 300 mg/kg rats that died prematurely had transitional cell hyperplasia with inflammation and haemorrhage of the bladder wall. Crystal nephropathy (basophilic foci, tubule or pelvic dilatation, birefringent crystals in the pelvic fornix, or transitional cell hyperplasia) was seen in all of the 400 mg/kg and most of the 300 mg/kg rats. These effects were not seen in the 50 or 150 mg/kg rats. Kidney oxalate levels, the metabolite responsible for the kidney toxicity, was not increased in the 50 and 150 mg/kg animals compared to the controls. The NOAEL for this study is 150 mg/kg/day (Corley et al., 2005) [Kl. score = 1].

Male and female Sprague-Dawley rats were given in their feed 0, 0.1, 0.2, 0.5, 1.0 or 4.0% ethylene glycol for two years. There was significant reduction in growth in the 4% males after week 16, and in the 1% males after week 70. The 4% females did not gain any weight past the first year of the study. Water consumption was double that of the controls in the 4% males that initiated soon after the

start of the study. The 1% males had significant increases in water consumption after 6 months and some increase was observed in the 0.5% males. Females only showed increased water consumption in the 4% group. There was 100% mortality in the 1 and 4% males, while mortality of additional dose levels were below that of the controls. There was 100% mortality in the 4% females, while the 1% females were similar to the controls; the 0.1, 0.2 and 0.5% females were increased compared to the controls. Since the 1 and 4% males and the 4% females all died before the study termination date, there are no data for these groups on terminal organ weight. For males, the terminal organ weights were decreased in all dose levels compared to the controls. For females, the organ weights were similar to the controls. The 1 and 4% males and females had kidneys with stones and crystals. The NOAEL for this study is 0.2% (data was insufficient to calculate the dose) (Blood, 1965) [Kl. score = 2].

Male and female Fischer 344 rats were given in their feed 0, 40, 200 or 1,000 mg/kg ethylene glycol for 24 months. There were numerous adverse effects in the 1,000 mg/kg males and, to a lesser degree, in the 1,000 mg/kg females. The most remarkable effect was the production of urinary calculi in the kidneys, ureters and urinary bladders of the 1,000 mg/kg males, along with the presence of high levels of calcium oxalate in the urine. Increased incidences of tubular cell hyperplasia, tubular dilation, peritubular nephritis and focal granulomatous nephritis occurred in the 1,000 mg/kg males. Other significant findings in these males were markedly lower body weight gain, increased absolute and relative kidney weights, decreased absolute and relative liver weights, various hematopoietic changes and increased water consumption (likely a result of impaired kidney function). Histopathological changes in the 1,000 mg/kg males were mineralisation of the heart, lungs, stomach and vas deferens being the most noteworthy. The various adverse effects in these males resulted in reduced survival; there was increased mortality which became apparent by 8 months, with all males in this group died by month 16. Although calcium oxalate crystals were found in the urine of the 1,000 mg/kg females, no urinary calculi were seen. Absolute and relative kidney weights were increased in these rats. The most significant histopathologic finding in the 1,000 mg/kg females was fatty metamorphosis of the liver. There were transient changes in organ weights, erythroid parameters, water consumption rates and urine specific gravity in the 200 and 40 mg/kg rats; these effects were considered to be statistical artifacts attributable to chance. Focal soft mineralisation was observed in certain organs of the 200 and 40 mg/kg rats, which were considered to be the result of altered calcium metabolism associated with ingestion of ethylene glycol. The NOAEL for this study is considered to be 200 mg/kg/day (DePass et al., 1986a; ECHA) [Kl. score = 2].

Male and female B6C3F_1 mice were given in their feed 0, 6,250 ppm (males only), 12,500 and 25,000 ppm (males and females) or 50,000 ppm (females only) for 103 weeks. These concentrations are approximately equivalent to 0, 1,500, 3,000, 6,000 or 12,000 mg/kg/day. Survival, mean body weights and feed consumption was similar across all groups. There were no treatment-related clinical signs of toxicity. Liver lesions (males only) and arterial hyperplasia (females only) were observed at 12,500 ppm, but no adverse effects were observed at 6,250 ppm. The NOAEL for this study is 6,250 ppm in males, which corresponds to 1,500 mg/kg/day (NTP, 1993) [Kl. score = 2].

Inhalation

No studies are available.

Dermal

No studies in rodents or rabbits are available.

G. Genotoxicity

In vitro Studies

The *in vitro* genotoxicity studies on ethylene glycol are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Ethylene Glycol

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (*S. typhimurium* and *E. coli* strains)	-	-	1	ECHA
Bacterial reverse mutation (*S. typhimurium* and *E. coli* strains)	-	-	2	ECHA
Mammalian cell gene mutation (mouse lymphoma L5178Y cells)	+/-	-	2	McGregor et al. (1991)
Chromosomal aberration (CHO cells)	-	-	2	ECHA

*+, positive; -, negative

In vivo Studies

A dominant lethal study was conducted in F344 rats given 0, 40, 200 or 1,000 mg/kg/day ethylene glycol in feed. There were slight increases in the dominant lethal mutation index in the high-dose and low-dose groups; these appear to be random occurrences and were not considered to be treatment-related. It was concluded that ethylene glycol was not genotoxic in this study (DePass et al., 1986b) [Kl. score = 2].

H. Carcinogenicity

Oral

Male and female Fischer 344 rats were given in their feed 0, 40, 200 or 1,000 mg/kg ethylene glycol for 24 months. There was increased mortality in the 1,000 mg/kg males, starting at 8 months and resulting in all males in this group dead by 16 months. Survival for the 1,000 mg/kg females and the 200 and 40 mg/kg males and females were similar to the controls. The incidence of mononuclear cell leukemia was statistically significantly higher in the 200 mg/kg males compared to the male controls, but not when compared to the pooled controls (males and females). Evaluation of the data by the method of Thomas et al. (2007), however, showed no treatment-related effect. It was concluded that ethylene glycol was not carcinogenic to rats in this study (DePass et al., 1986) [Kl. score = 2].

Male and female B6C3F_1 mice were given in their feed 0, 6,250 ppm (males only), 12,500 and 25,000 ppm (males and females) or 50,000 ppm (females only) ethylene glycol. These concentrations were approximately equivalent to 0, 1,500, 3,000, 6,000 or 12,000 mg/kg/day. Body weights, survival and incidence of tumours were similar between treated and control mice (NTP, 1993) [Kl. score = 2].

Inhalation

No studies are available.

Dermal

No studies are available.

I. Reproductive Toxicity

Ethylene glycol was assessed in a Reproductive Assessment by Continuous Breeding (RACB) protocol (Chapin and Sloane, 1997). The parental mice were administered ethylene glycol via drinking water during pre-mating exposure, cohabitation, pregnancy and lactation. The F_1 generation received prenatal exposure via maternal exposure during gestation, with the exposure continuing during lactation, weaning and mating of F_1 animals and production of an F_2 litter. The doses were 0, 0.25, 0.5 or 1% ethylene glycol, which corresponded to approximately 0, 410, 840 or 1,640 mg/kg/day. No adverse effects were noted in the parental animals at doses up to 1%. There was a small, but statistically significant, effects on the numbers of litters per fertile pair, the number of live pups per litter, and live pup weight in the 1% dose group. Neither the 0.25 nor 0.5% dose groups were significantly affected. The number of live pups per litter was lower in the treated groups, but differences were not statistically significant. Unusual facial features (i.e., shorter snout and wide-set eye) and skeletal defects (shortened frontal, nasal and parietal bones; fused ribs abnormally shaped or missing sternebrae, abnormally shaped vertebrae; and twisting of the spine) were noted on some of the offspring of the treated mice in the 1% group, but not in the controls. The parental NOAEL is 1% (approximately 1,640 mg/kg/day), and the NOAEL for reproductive toxicity is 0.5% (approximately 840 mg/kg/day (Lamb et al., 1985) [Kl. score = 2].

In a three-generation reproductive toxicity study, Fischer 344 rats were given in their diet 0, 40, 200 or 1,000 mg/kg/day ethylene glycol. There were no treatment-related effects on clinical signs of toxicity or survival in the parental animals. There were no significant effects on fertility index, gestation index, gestation survival for all three generations. Mean pup weights for each of the hree generations were similar between treated and control animals. The NOAEL for parental and reproductive toxicity is 1,000 mg/kg/day (DePass et al., 1986b) [Kl. score = 2].

J. Developmental Toxicity

Pregnant Sprague-Dawley rats were dosed by oral gavage with 0, 50, 150, 500, 1,000 or 2,500 mg/kg ethylene glycol during gestational days (GD) 6-15. Maternal toxicity was observed in the 2,500 mg/kg group and consisted of significantly decreased body weights, increased water consumption, decreased uterine weights, increased kidney weights and increased relative liver weights. At 500 mg/kg, there were developmental effects, which included reduced foetal body weights, extra or missing ribs, missing arches and poor ossification in thoracic and lumbar centra. In the 2,500 mg/kg group, in addition to skeletal malformations, there was gastroschisis, hydrocephaly, lateral ventricle dilated (tissue depressed), umbilical hernia and atelectasis. The NOAELs for maternal and developmental toxicity are 1,000 and 500 mg/kg/day, respectively (Neeper-Bradley et al., 1995) [Kl. score = 2].

Pregnant CD rats were dosed by oral gavage with 0, 1,250 2,500 or 5,000 mg/kg ethylene glycol during GD 6-15. In the $\geq$ 2,500 mg/kg groups, the dams had increased relative kidney weights, decreased gravid uterine weight and increased water consumption. Maternal body weight gain was significantly decreased in the 1,250 mg/kg group. Live litter size was significantly decreased in the 5,000 mg/kg group and foetal body weights were decreased in the 1,250 and 5,000 mg/kg groups. Litters with malformed foetuses were observed in the $\geq$ 1,250 mg/kg groups. The LOAELs for maternal and developmental toxicity are 1,250 mg/kg/day; NOAELs were not established (Price et al., 1985) [Kl. score = 2].

Pregnant Fischer 344 rats were given by oral gavage 0, 40, 200 or 1,000 mg/kg ethylene glycol during GD 6-15. No maternal toxicity was observed at any dose level. There were no significant effects on preimplantation loss, foetal length, foetal weight, total implantations or litter size. There was an increased incidence of skeletal alterations in the 1,000 mg/kg group, which consisted of poorly ossified and unossified vertebral centra. No significant increases in the incidence of major malformations were observed. The NOAELs for maternal and developmental toxicity are 1,000 and 400 mg/kg/day (Maronpot et al., 1983) [Kl. score = 2].

Pregnant CD-1 mice were dosed by oral gavage with 0, 50, 150, 500 or 1,500 mg/kg ethylene glycol during gestational days (GD) 6 to 15. There was no maternal toxicity. At 1,500 mg/kg, there were reduced foetal body weights, fused ribs and arches, poor ossification in thoracic and lumbar centra and increased occurrence of an extra 14^{th} rib. At 500 mg/kg, there was slight reductions in foetal body weight and increased incidences of extra ribs. The NOAELs for maternal and developmental toxicity were 1,500 and 150 mg/kg/day, respectively (Neeper-Bradley et al., 1995) [Kl. score = 2].

Pregnant CD-1 mice were dosed by oral gavage with 0, 750, 1,500 or 3,000 mg/kg ethylene glycol during GD 6 to 15. There was a significant decrease in maternal gain, gravid uterine weights and liver weights in the 1,500 mg/kg group. A decreased number of implantation sites per litter was observed in the 1,500 mg/kg group. Significant decrease in liver litter size was observed in the 3,000 mg/kg group and decreased foetal body weights were seen at ≥ 750 mg/kg. Litters with a significant increase in malformed foetuses were observed in the ≥ 750 mg/kg groups. There was a significant dose-related increase in post-implantation loss per litter, though there were no significant pairwise comparisons. The NOAEL for maternal toxicity is 750 mg/kg/day. The LOAEL for developmental toxicity is 750 mg/kg/day; the NOAEL was not established (Price et al., 1985) [Kl. score = 2].

In a short-term reproductive and developmental toxicity screen test, male and female Swiss Crl:CD-1 mice were allowed to mate over a three-day period. The males were dosed by oral gavage from study Day 3 to study Day 20. The Group A females were exposed throughout the 21-day test period; the Group B females were exposed during GD 8-14. The doses were 0, 250, 700 or 2,500 mg/kg ethylene glycol. The Group A females were sacrificed after 19 days of treatment, and the Group B females were allowed to litter and rear to postnatal day (PND) 4. There was no maternal or paternal toxicity. The 2,500 mg/kg females in Group A had significantly fewer liver implants and more dead implants. The 2,500 mg/kg in Group B had significantly lower total litter weights on PND 1 and 4. The NOAELs for parental and developmental toxicity are 2,500 and 700 mg/kg/day (Harris et al., 1992) [Kl. score = 2].

In a Chernoff/Kavlock assay, pregnant CD-1 mice were dosed by oral gavage with 0 or 11,090 mg/kg ethylene glycol during GD 7-14. The females were allowed to litter and rear to PND 3. Ten percent of the maternal animals died. The number of surviving pups per litter (40% survived), birth weight and pup weight gain were reduced. The LOAELs for maternal and developmental toxicity are 11,090 mg/kg; NOAELs were not established (Schuler et al., 1984; Hardin et al., 1987) [Kl. score = 2].

Pregnant female New Zealand White rabbits were dosed by oral gavage with 0, 100, 500, 1,000 or 2,000 mg/kg ethylene glycol on GD 6 to 19. At 2,000 mg/kg, eight of the 17 does (42.1%) died. Maternal body weights and body weight gain were similar across all groups. There was no developmental toxicity. The NOAEL for maternal toxicity is 1,000 mg/kg/day. The NOAEL for developmental toxicity is 2,000 mg/kg/day, the highest dose tested (ECHA) [Kl. score = 2].

Pregnant female CD rats were dosed by oral gavage with 0, 250, 1,250 or 2,250 mg/kg ethylene glycol on GD 6 to 20. At 2,250 mg/kg, maternal body weight, body weight gain, kidney weight and postpartum uterine weight were significantly reduced. At 1,250 mg/kg, the gestational period was

lengthened, and maternal kidney histopathological effects were noted. Developmental toxicity was noted in the 2,250 mg/kg group and included reduced pup weight, reduced viability and increased malformations (primarily hydrocephaly and abnormalities of the axial skeleton). No developmental toxicity was seen in the 1,250 mg/kg group. The NOAEL for maternal and developmental toxicity is 250 mg/kg/day (ECHA) [Kl. score = 2].

Inhalation

Pregnant female CD rats were exposed by inhalation (whole-body) to 0, 150, 1,000 or 2,500 mg/m^3 ethylene glycol aerosol 6 hours/day on gestational days 6 to 15. There was no treatment-related mortality; a dose-related increased in clinical signs (red fur discoloration on the head and neck) was noted, which was considered to be a non-specific indication of stress. Body weights and body weight gain were unaffected by treatment. There was some evidence of treatment-related reductions in ossification of the foetal skeleton at 1,000 and 2,500 mg/m^3 (considered as fetotoxicity). The NOAECs from inhalation exposure cannot be determined due to confounding oral exposure during whole-body exposure. However, there was no maternal or embryotoxicity at 150 mg/m^3 and no teratogenicity at any aerosol concentration tested (Tyl et al., 1995a) [Kl. score = 2].

Pregnant female CD-1 mice were exposed by inhalation (whole-body) to 0, 150, 1,000 or 2,500 mg/m^3 ethylene glycol aerosol 6 hours/day on gestational days 6 to 15. Reduced maternal body weight was observed in the 2,500 mg/m^3 group on GD 12,15 and 18 and in the 1,000 mg/m^3 group on GD 18. Reduced maternal weight gain was also seen during GD 6-12, 6-15 and GD 6-18 for the ≥ 1000 mg/m^3 groups and for GD 5-18 for the 2,500 mg/m^3 group. Terminal body weights were reduced in the ≥ 1,000 mg/m^3 groups. Gravid uterine weight was also reduced in the ≥ 1,000 mg/m^3 groups, so that body weight corrected for gravid uterine weight was unaffected. The number of viable implantations per litter was reduced at 2,500 mg/m^3. The number of non-viable implantations per litter was elevated at ≥ 1,000 mg/m^3 because of a significant increase in late resorptions at 1,000 mg/m^3, and a significant increase in late resorptions and in dead foetuses at 2,500 mg/m^3. The number of early resorptions at 2,500 mg/m^3 was also elevated but not statistically. foetal body weights per litter (male, female and total) were reduced at ≥ 1,000 mg/m^3. There was a significant increase in the incidence of a number of external, visceral and skeletal malformation, as well as skeletal variations, at ≥ 1,000 mg/m^3. There was no observable maternal or developmental toxicity at 150 mg/m^3. However, a NOAEC cannot be determined because of the amount of ethylene glycol that may have been ingested from the presence of ethylene glycol on the fur (Tyl et al., 1995a) [Kl. score = 2].

Pregnant female CD-1 mice were exposed by inhalation (nose-only) to 0, 500, 1,000 or 2,500 mg/m^3. The study also included a group exposed to 2,100 mg/m^3 (not discussed here). Reduced maternal body weight gain were seen in the 2,500 mg/m^3 for GD 9-12, 12-15, 6-15 and 0-18. Absolute kidney weights were increased in the ≥ 1,000 mg/m^3 groups. foetal body weights per litter were significantly reduced for the 2,500 mg/m^3. In the 2,500 mg/m^3, there was a significant increase in one skeletal malformation (fusion of the ribs) and an increased incidence of skeletal variations. No other teratogenic effects were observed. The NOECs for maternal and developmental toxicity are 500 and 1,000 mg/m^3, respectively (Tyl et al., 1995c) [Kl. score = 2].

Dermal

Pregnant CD-1 mice were administered by dermal applications of 0, 400, 1,677 or 3,549 mg/kg ethylene glycol 6 hours/day on GD 6-15. There was minimal, if any, treatment-related maternal toxicity. Copora lutea, total implants, percentage of live foetuses per litter, foetal body weights and incidence of external or visceral malformations were unaffected by treatment. There was, however,

a significant increase in two skeletal variations in the 3,549 mg/kg group. The NOAELs for maternal and developmental toxicity were considered to be 3,549 mg/kg/day (Tyl et al., 1995b) [Kl. score = 2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for ethylene glycol follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2021).

A. Non-Cancer

Oral

The NOAEL from a 24-month rat dietary study was reported to be 200 mg/kg/day based on kidney lesions in male F344 rats at 1,000 mg/kg/day (DePass et al., 1986b). A subsequent 12-month rat dietary study using male Wistar rats reported a NOAEL of 150 mg/kg/day also based on kidney toxicity at 300 mg/kg/day and higher (Corley et al., 2008). The Wistar rat strain was shown to be more sensitive (approximately three-fold) to the kidney toxicity of ethylene glycol than F344 rats (Cruzan et al., 2004). The NOAEL of 150 mg/kg/day will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Snellings et al. (2013) derived an oral reference dose for ethylene glycol using benchmark dose modelling, with toxicokinetic (PBPK modelling) and toxicodynamic data. The human equivalent dose ($[BMDL_{05}]_{HED}$) was calculated to be 150 mg/kg/day.

Oral RfD = $[BMDL_{05}]_{HED}$ / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 1
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1
Oral RfD = 150/(1 x 10 x 1 x 1 x 1) = 150/10 = 15 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2021)
Proportion of water consumed = 10% (ADWG, 2021)
Volume of water consumed = 2L (ADWG, 2021)
Drinking water guidance value = (15 x 70 x 0.1)/2 = 53 mg/L

B. Cancer

Ethylene glycol was not carcinogenic to rats and mice in two-year dietary studies. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Ethylene glycol does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Ethylene glycol is of low toxicity concern to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on ethylene glycol.

Table 3: Acute Aquatic Toxicity Studies on Ethylene Glycol

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Pimephales promelas	96-hour LC_{50}	>72,860	1	Pillard (1995)
Oncorhynchus mykiss	96-hour LC_{50}	22,810 24,591	2	OECD (2004a,b)
Daphnia magna	48-hour EC_{50}	>100	1	ECHA
Daphnia magna	48-hour EC_{50}	46,300	2	Gersich et al. (1986)
Ceriodaphnia dubia-affinis	48-hour EC_{50}	25,800 (20°C) 10,000 (24°C)	2	Cowgill et al. (1985)
Daphnia magna	48-hour EC_{50}	46,300 (20°C) 51,000 (24°C)	2	Cowgill et al. (1985)
Selenastrum capricornutum	96-hour IC_{50} NOEC	10,940 10,000	2	Pillard and DuFresne (1999)

Chronic Studies

Table 4 lists the results of chronic aquatic toxicity studies conducted on ethylene glycol.

Table 4: Chronic Aquatic Toxicity Studies on Ethylene Glycol

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Pimephales promelas	7-day NOEC	15,380	2	Pillard (1995)
Ceriodaphnia dubia	7-day NOEC (reproduction)	8,590	2	Pillard (1995)
Pseudokirchneriella subcapitata	72-hr NOEC	>100 *	2	ECHA

*Read-across to pentaethylene glycol (CAS No. 4792-15-8)

C. Terrestrial Toxicity

No guideline studies have been conducted on ethylene glycol.

D. Calculation of PNEC

The PNEC calculations for ethylene glycol follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E(L)C_{50} values are available for fish (22,810 mg/L), *Daphnia* (>100 mg/L), and algae (10,940 mg/L). NOEC values from long-term studies are available for fish (15,380 mg/L), invertebrates (8,590 mg/L) and algae (10,000 mg/L). On the basis that the data consists of short-term and long-term results from three trophic levels, an assessment factor of 10 has been applied to the lowest reported E(L)C_{50} value of 100 mg/L for Daphnia. The E(L)C_{50} value is used because the value for fish is lower than the NOEC values for all three trophic levels. The $PNEC_{aquatic}$ is 10 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 6.4 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (0.82/1280) \times 1000 \times 10$
$= 6.4$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1000 \times BD_{solid}]$
$= 0.8 + [(0.2 \times 0.04/1000 \times 2400]$
$= 0.82\ m^3/m^3$

Where:

Kp_{sed} = solid-water partition coefficient (L/kg)

BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]

$Kp_{sed} = K_{oc} \times f_{oc}$

= 1 x 0.04

= 0.04 L/kg

Where:

K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for ethylene glycol calculated from EPISUITE™ using the MCI is 1 L/kg.

F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.13 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$

= (0.02/1500) x 1000 x 10

= 0.13 mg/kg

Where:

Kp_{soil} = soil-water partition coefficient (m^3/m^3)

BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]

$Kp_{soil} = K_{oc} \times f_{oc}$

= 1 x 0.02

= 0.02 m^3/m^3

Where:

K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for ethylene glycol calculated from EPISUITE™ using the MCI is 1 L/kg.

F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2008).

Ethylene glycol is readily biodegradable and thus does not meet the screening criteria for persistence.

The measured BCF in fish is 10. Thus, ethylene glycol does not meet the criteria for bioaccumulation.

The NOECs from the chronic aquatic toxicity studies on ethylene glycol are > 0.1 mg/L. The acute $E(L)C_{50}$ values from the acute aquatic toxicity studies on ethylene glycol are > 1 mg/L. Thus, ethylene glycol does not meet the criteria for toxicity.

The overall conclusion is that ethylene glycol is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

STORE Category 2 (target organ: kidney)

B. Labelling

Warning

A. Pictogram

IX. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace exposure standards for ethylene glycol in Australia is as follows: 10 mg/m^3 as an 8-hour TWA for ethylene glycol (particulate); 20 ppm (52 mg/m^3) as an 8-hour TWA for ethylene glycol (vapour). There is also a skin notation indicating that absorption through the skin may be significant source of exposure.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

X. TRANSPORT INFORMATION

Ethylene glycol is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

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FORMIC ACID

This dossier on formic acid presents the most critical studies pertinent to the risk assessment of formic acid in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed formic acid in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment[1] .

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): formic acid

CAS RN: 64-18-6

Molecular formula: CH_2O_2

Molecular weight: 46.025 g/mol

Synonyms: formic acid, methanoic acid, formylic acid, aminic acid

SMILES: OC=O

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-chemical Properties of formic acid

Property	Value	Klimisch Score	Reference
Physical state at 20oC and 101.3 kPa	Clear and colourless organic liquid	1	ECHA
Melting Point	8°C (pressure not provided)	1	ECHA
Boiling Point	100.23°C @ 101.3 kPa	1	ECHA
Density	1220 kg/m^3 @ 20°C	1	ECHA
Vapour Pressure	4,271 Pa @ 20°C	1	ECHA
Partition Coefficient (log K_{ow})	-2.1@ 23°C and pH 7	1	ECHA
Water Solubility	Completely miscible	2	ECHA
Flash Point	49.5°C @ 101.3 kPa	1	ECHA
Auto flammability	528°C @ 100.6-101.0 kPa	1	ECHA
Viscosity	1.8 mPa s @ 20°C	1	ECHA

[1] https://www.industrialchemicals.gov.au/chemical-information/search-assessments?assessmentcasnumber=64-18-6%2C+

Property	Value	Klimisch Score	Reference
Henry's Law Constant	0.017 Pa-m^3/mole @ 20°C	1	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Formic acid is readily biodegradable, is not expected to bioaccumulate, and has a low potential to adsorb to soil or sediment.

B. Partitioning

The pKa of formic acid is 3.7, indicating that this substance will exist partially in anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts (PubChem).

Volatilisation of formic acid from water and moist soil surfaces is expected to be an important fate process given a Henry's Law constant of 0.017 Pa-m^3/mole (ECHA). Formic acid is expected to volatilise from dry soil surfaces based upon its vapour pressure.

Hydrolysis is not expected to be an important environmental fate process since this substance lacks functional groups that hydrolyse under environmental conditions (PubChem).

C. Biodegradation

Formic acid and the formate ion were readily biodegradable in OECD 301 D tests. In the two tests, biodegradation rates of 82% and 92 % related to the biological oxygen demand were estimated. (ECHA) [Kl. score = 1 and Kl. Score =2].

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

D. Environmental Distribution

The log K_{oc} of the non-dissociated species of formic acid was measured to be < 1.25 in a GLP test according to OECD guideline 121 (ECHA) [KI. score =1]. As this value refers to the uncharged molecule, which will only be present under highly acidic conditions, the K_{oc} and log K_{oc} of the dissociated, charged form at realistic environmental pH values was calculated by using the pKa (= 3.70) and the log P_{ow} of the uncharged molecule (= -0.46) for a corrected log K_{oc} according to Franco et al. (2008). For the formate ion which will be present at environmental relevant pH values, slightly higher adsorption rates were estimated (K_{oc} = 31, log K_{oc} = 1.49) (BASF SE, 2009, as cited in ECHA) [Kl. Score = 2].

Based on these values, formic acid has a low potential for adsorption to soil and sediment and is expected to have very high mobility in soil. Likewise, if released to water, formic acid is also not expected to adsorb to suspended solids or sediments.

E. Bioaccumulation

No bioconcentration studies have been conducted on formic acid. Formic acid is not expected to bioaccumulate based on the experimental log K_{ow} of -2.1 (ECHA) [KI. Score = 1].

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Formic acid is metabolized to formate and formate salts in the body. Formic acid rapidly absorbed after ingestion, and it excreted in urine. Formic acid moderate acute toxicity via oral and dermal routes of exposure. Formic acid has high acute toxicity via the inhalation route of exposure. Formic acid is corrosive to the skin and eyes. It is not a skin sensitiser. Formic acid did not elicit systemic toxicity in repeated dose toxicity studies. Formic acid is not a genotoxin, it is not carcinogenic, nor is it a reproductive or developmental toxicant.

B. Metabolism

The toxicokinetic behaviour of formic acid was examined in human volunteers who ingested up to 2 grams of formic acid. Formate and formic acid are both rapidly absorbed, and they reach peak plasma levels withing 10-30 minutes after ingestion. Resorption of the unprotonated acid begins in the stomach. Sodium formate is converted to the unprotonated acid under the pH conditions of the stomach. Formate is eliminated from the plasma with a half-life time of $t_{1/2}$ =45 minutes. Urinary excretion is rapid within the first six hours after ingestion and returns to normal levels at 12 hours after dosing. Urinary excretion is generally low, and it accounts from approximately 2.1-3.3% of the administered dose. The blood pH remains unchanged following single formate or formic acid doses that are equivalent to 3,000 mg formic acid. Urine volume and pH were increased if formate is excreted by the urine (ECHA) [KI. score =2].

Formate is the common metabolite of formic acid and formate salts. Formate is formed from precursors in the intermediary metabolism and is used as an important constituent of the C1 intermediary metabolism which is required for the biosynthesis of amino acids and nucleic acid bases (purines and pyrimidines). Formate may also be formed from ingested methanol via formaldehyde and further oxidation to formate.

Pharmacokinetic models have been established from methanol inhalation studies which allow calculating the time course of all metabolites including formate in good correlation with animal studies. Peak plasma formate levels were reached within 1 hour (rabbits) and 4-5 hours (pigs) after oral administration of potassium diformate. The elimination from blood follows first order kinetics and the blood levels rapidly return to background levels in all species, i.e., formate does not persist or accumulate. However, there are significant species differences in the elimination rates and the elimination half-lives (from plasma): rat (12 minutes) < guinea pig (22 minutes) < rabbit (32 minutes) < humans (45 minutes) < cat (67 minutes) < dog (77 minutes) < pig (87 minutes). This reflects the species differences in the hepatic concentrations of folates and folate-dependent enzymes which affect the formate degradation to CO_2. Only minor quantities are excreted unchanged via urine in all species.

High formate plasma levels may occur in humans under special conditions, i.e., if the formate elimination capacity is exceeded, for example after ingestion of large amounts of formate salts. Photoreceptor toxicity and damage to the eye may occur in humans under such conditions.

Formic acid and formate salts may be absorbed via the oral route. Formic acid may generate vapours that can be taken up by inhalation. Dermal uptake may also occur with formic acid.

Local toxicity due to corrosivity: skin and eye after direct contact; upper inhalation tract after inhalation; mouth, larynx, pharynx, oesophagus, stomach, intestines after oral ingestion.

Dermal absorption of formic acid is known to occur. Systemic toxicity, acidosis, and elevated formate blood levels were described in clinical case reports following incidental poisoning (ECHA) [KI. score =1].

C. Acute Toxicity

Oral

An OECD guideline 401 (Acute Oral Toxicity) study was conducted using male and female Bor: WISW rats who were administered 501, 631, 794, and 1000 mg/kg bw of formic acid. Clinical signs were reported 30 minutes after dosing. Symptoms of unkept fur, hunched posture, stagger, and blood in urine were observed. At times hypothermia, body weight loss and pale limbs were also observed. The acute oral LD_{50} was reported to be 730 mg/kg bw (ECHA) [KI. score =1].

Inhalation

An OECD Guideline 403 (Acute Inhalation Toxicity) study was conducted using male and female Sprague-Dawley rats administered formic acid via whole body vapour inhalation. The clinical signs indicted corrosive properties of formic acid as evidenced by the occurrence of corneal opacity and corrosion of the dorsal nose in some cases. The symptoms persisted until termination 14 days after the rats were exposed to 7.29 mg/L of formic acid. There were no changes in animals that survived. Inflated lungs and dilated hearts were observed in the animals that died. The four-hour LC_{50} was reported to be 7.85 mg/L air (ECHA) [KI. score 1].

Dermal

The acute dermal toxicity was not examined in animals because of the corrosive properties of formic acid. In addition to this, the dermal toxicity of the salts is low, LD_{50} of sodium formate was >2,000 mg/kg (BASF, 2007, as cited in ECHA).

D. Irritation

There is sufficient human data and information from animal testing which indicates that formic acid is corrosive to the skin and causes eye damage. Therefore, skin irritation and corrosion testing was not conducted because there are studies which indicate that formic acid is corrosive (ECHA).

E. Sensitisation

An OECD Guideline 406 (Skin sensitisation) study was conducted using female Hsd Poc: DH guinea pigs exposed to 2 or 5% formic acid via epicutaneous occlusive dressing. Formic acid had no sensitizing effect on the skin of guinea pigs in this study (ECHA) [KI. score =1].

F. Repeated Dose Toxicity

Oral

An OECD Guideline 453 (combined chronic toxicity/carcinogenicity) study was conducted using male and female Crl:HanWist (glx:BRL) BR rats exposed to 0, 50, 400, and 2,000 mg/kg bw/day of potassium formate (1:2) in their feed for 52 weeks. Treatment related findings were noted at 2,000 mg/kg bw/day and included a statistically significant depression of body weight gain and at terminal kill a thickening of the stomach confirmed as basal cell hyperplasia or foveolar epithelium hyperplasia in the majority of the high dose animals. These changes were less pronounced than in a previous 90-day rat study. There was no evidence of systemic target organ toxicity. The NOAEL for local and systemic toxicity was reported to be 400 mg/kg bw/day based on local effects in the stomach and reduced body weight in the high dosed rats. Taking the molecular weights and stoichiometry into account, this corresponds to a NOAEL of 142 mg formic acid/kg bw/day, and 283 mg formate/kg bw/day (ECHA) [KI. score =1].

An OECD Guideline 408 (Repeated dose 90-day toxicity) study was conducted using male and female Crl:CDBR rats exposed to 0, 600, 1200, and 3000 mg/kg bw/day potassium formate in their feed for 13 weeks. There was no overt toxicity observed after 13 weeks of treatment. Minor irritation occurred in the forestomach of both sexes, with effects being seen in the males at all dose levels. A NOAEL was not derived in this study but is considered to be <600 mg/kg bw/day based on irritation of the forestomach and the squamous cell hyperplasia seen at 600 mg/kg bw/day in both sexes. Systemic toxicity was not observed up to 3,000 mg/kg bw/day. A systemic NOAEL or LOAEL could not be derived for this study (ECHA) [KI. score =1].

Inhalation

An OECD Guideline 413 (Subchronic Inhalation Toxicity: 90-day) study was conducted in male and female in Fischer 344 rats exposed to 0,0.015,0.030,0.062, 0.122, or 0.244 mg/L (0, 8, 16, 32, 64, or 128 ppm) formic acid via whole body vapor inhalation (6 hours/day, 5 days/week) for 13 weeks. There were no mortalities nor clinical signs or systemic toxicity in male and female rats exposed to 8, 16, 32, 64, or 128 ppm for 13 weeks (5 days/week, 6 h/day). There were no unusual gross lesions noted during necropsy, organ weights were not affected by treatment. Male and female reproductive parameters (sperm motility, density, and testicular or epididymal weight; length of the oestrous cycle) were not affected. Histopathology revealed increased incidences of squamous metaplasia of the respiratory epithelium and degeneration of the olfactory epithelium in the high-dose male and female rat groups where most of the animals were affected. However, the severity was generally minimal to mild. A systemic LOAEC was not achieved. The authors suggested that the lack of systemic effects in both 2-week and 13-week NTP inhalation studies is possibly related to the rapid metabolizing capacity of formate to CO_2, due to high tetrahydrofolate and 10-formyl tetrahydrofolate dehydrogenase levels in rodents. These levels are much lower in humans who are significantly more sensitive to the formate toxicity. Therefore, caution should be used in considering the results obtained with rodents in determining potential human risks associated with systemic exposure to formic acid. Nevertheless, human experience does not indicate that formic acid represents a significant systemic thread to humans unless at high concentrations following intended or incidental ingestion or large-scale skin contact, where the caustic effect also governs the toxic mode of action. Based on the local histopathological changes in the respiratory tract the NOAEC in this study was determined to be 64 ppm (0.122 mg/l), and the LOAEC was 128 ppm (0.244 mg/l). The systemic NOAEC was 128 ppm (0.244 mg/l), the highest concentration tested (ECHA) [KI. score =1].

An OECD Guideline 413 (Subchronic Inhalation Toxicity: 90-day) study was conducted using male and female B6C3F1 mice exposed to 0, 8,16,32,64, and 128 ppm formic acid via whole body inhalation (5

days/week, 6 hours/day) for 13 weeks. There were no mortalities or treatment-related signs of toxicity in male and female mice exposed to formic acid at up to 128 ppm (0.244 mg/l) for 13 weeks. Systemic toxicity was generally low, but body weight gain was reduced in both sexes at 128 ppm, resulting in terminal body weights that were 16-20% below those of the controls. A small, but statistically significant increase of liver weight was noted in males at 32 and 64 ppm. Findings of histopathology were limited to few cases of minimal degeneration of the olfactory epithelium. Sperm motility and oestrous cycle length were not affected. The NOAEC in this study was determined to be 32 ppm (0.062 mg/l), based on histopathological changes of the respiratory tract (ECHA) [KI. score =1].

Dermal

There are no studies available based formic acid is corrosive.

G. Genotoxicity

In vitro Studies

The *in vitro* genotoxicity studies on formic acid are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Formic Acid

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
OECD Guideline 471 (Bacterial Reverse Mutation Assay) S. typhimurium TA97, TA98, TA100, and TA1535	-	-	1	ECHA
OECD Guideline 479 (Genetic Toxicology: In Vitro Sister Chromatid Exchange Assay in Mammalian Cells) Chinese hamster lung fibroblasts V79	-	-	1	ECHA
OECD Guideline 479 (Genetic Toxicology: In Vitro Sister Chromatid Exchange Assay in Mammalian Cells) mammalian cells: human lymphocytes	-	-	1	ECHA
OECD Guideline 476 (In Vitro Mammalian Cell Gene Mutation Test)	-	-	1	ECHA
OECD Guideline 473 (In Vitro Mammalian Chromosome Aberration Test) Chinese hamster ovary (CHO)	-	-	2	ECHA

*+, positive; -, negative

In vivo Studies

An OECD Guideline 477 (Genetic Toxicology: Sex-linked Recessive lethal Test in Drosophilia melanogaster) study was conducted using male Drosophila melanogaster exposed to 0.1% formic acid via their feed. Following exposure to 0.1% formic acid, the number of mutants was significantly increased compared to the historical controls in this study. However, an increase was also seen with 0.1% formic acid in a subsequent feeding experiment, but without gaining statistical significance. Sodium formate (produced by neutralization of formic acid) at the same molar concentration in the feed was negative in the Drosophila SLRL test. The authors concluded that the mutations observed

with formic acid were related to the acidic pH, rather than to the acid or the formate molecule itself. Therefore, it was concluded that formic acid and sodium formate did not induce mutations in this *in vivo* study (ECHA) [KI. score =1].

H. Carcinogenicity

Oral

An OECD Guideline 453 (Combined Chronic Toxicity/Carcinogenicity) study was conducted using male and female Crl:HanWist(Glx:BRL)BR rats exposed to 0, 50, 400, and 2,000 potassium formate in their feed for 104 weeks. All of the doses of potassium formate were well tolerated including the top dose without effects on clinical condition or survival. Depression of food consumption and body weight with sequel was observed in rats at 2000 mg/kg bw/day. The NOAEL for systemic toxicity was 400 mg potassium diformate/kg bw/day. Adaptive hyperplastic changes in the stomach and the gastro-intestinal tract were seen in rats at 400 and, to a higher extend, at 2000 mg/kg bw/day. The NOEL was 50 mg potassium diformate/kg bw/day for these effects. There was no evidence of a tumorigenic effect in the stomach or any other tissue, i.e. the NOAEL for carcinogenic effects was 2000 mg potassium diformate/kg bw/day. Taking the molecular weights of potassium diformate (130.1) and formic acid (46.03) into consideration, the following dose descriptors are calculated for formic acid from the above figures using a multiplier of 0.354 (46/136 = 0.354): NOEL gastro-intestinal changes:17.7 mg formic acid/kg bw/day; NOAEL systemic toxicity:142 mg formic acid/ kg bw/day; NOAEL carcinogenicity:708 mg formic acid/kg bw/day(ECHA) [KI. score=1].

An OECD Guideline 453 (Combined Chronic Toxicity/Carcinogenicity) study was conducted using male and female Crl:CD-1 (ICR) BR mice exposed to 0, 50, 400, and 2000 mg/kg bw potassium formate (1:2) in their feed for 80 weeks. All of the doses of potassium formate were well tolerated and did not adversely affect clinical conditions or survival, or the pattern or incidence of neoplastic lesions at any dose level. Treatment related changes were limited to high dose males and included minor disturbances of body weight (overall body weight gain reduced by 15%, level of statistical significance not reached) and food consumption (up to 5% increased), and an increased incidence of limiting ridge hyperplasia in the forestomach. The incidence and nature of tumours was not affected by the test substance, i.e., the test substance was not carcinogenic. The NOAEL for local effects and systemic toxicity was 400 mg potassium diformate/kg bw and day in male mice. The systemic NOAEL was 2000 mg potassium diformate/kg bw/day in female mice. The NOAEL for carcinogenicity was 2000 mg potassium diformate/kg bw/day in both sexes. Taking into consideration the molecular weights of potassium diformate (130.1) and formic acid (46.03), the following dose descriptors are calculated for formic acid from the above figures using a multiplier of 0.354 (46/136 = 0.354):NOEL gastro-intestinal changes, systemic toxicity (males):142 mg formic acid/ kg bw/day; NOAEL systemic toxicity, females:708 mg formic acid/ kg bw/day; NOAEL carcinogenicity 708 mg formic acid/ kg bw/day (ECHA)[KI. score =1].

Inhalation

There are no studies available.

Dermal

There are no studies available.

I. Reproductive Toxicity

Oral

A two generation OECD Guideline 416 (Two-generation reproduction toxicity) study was conducted using male and female Wistar rats exposed to 0, 100, 300, and 1000 mg/kg bw/day sodium formate in their feed. There were no clinical signs of toxicity or mortalities in any of the F0 or F1 parental dose groups. Food consumption and body weights were comparable to that of the concurrent controls. Necropsy and pathology revealed no gross findings or organ weight changes that could be treatment related. There were no indications that sodium formate adversely affected fertility or reproductive performance of the F0 and F1 parental animals at dose levels as high as 1000 mg/kg body weight/day. Mating behaviour, conception, gestation, parturition, lactation and weaning as well as sexual organ weights and gross findings of these organs were comparable between the rats of the test substance-treated test groups and the corresponding controls and ranged within the historical control data of the test facility. There were no effects on male and female reproduction organs. Sperm parameters and oestrous cycle were not affected. No test substance induced signs of developmental toxicity were noted in the progeny of the F0 and F1 parents at dose levels as high as 1000 mg/kg body weight/day. The number of delivered pups/litter, the sex ratio, their postnatal survival on days 4 and 21 after parturition, their body weights, and their sexual maturation remained unaffected by the test substance. Clinical and/or gross necropsy examinations of the F1 and F2 pups revealed only findings which were considered to be spontaneous in nature. The type and incidence of findings was within the range of the concurrent and/or the historical controls. Based on the above, the NOAEL values were as follows: NOAEL 1000 mg/kg bw/day for general systemic toxicity for F0 and F1 parental animals; NOAEL 1000 mg/kg bw/day for fertility and reproductive performance for the F0 and F1 parental rats; NOAEL 1000 mg/kg bw/day for developmental toxicity, in the F1 and F2 progeny. For read across purposes, the NOAEL for the formate anion may be calculated, taking into account formula weights. The calculation (1000 mg sodium formate/kg /69 x 45 = 650 mg/kg bw/day) gives a NOAEL of approx. 650 mg formate/kg bw/day (ECHA)[KI.score =1].

Inhalation

An OECD Guideline 413 (Subchronic Inhalation Toxicity:90-day) study was conducted using male and female Fischer 344 rats exposed to 0,0.015, 0.030, 0.122, 0.244 mg/L (0,8, 16, 32, 64, 128 ppm) formic acid via whole body vapor inhalation (5 days/week, 6 hours per day) for 13 weeks. There were no findings that would indicate adverse effects on male and female reproductive organs at any dose in this 13-week inhalation study. A NOAEC of 0.244 mg/L air was established for this study (ECHA)[KI. score =1].

An OECD Guideline 413 (Subchronic Inhalation Toxicity: 90-day) study was conducted using male and female B6C3F1 mice exposed to 0, 0.015, 0.030, 0.062, 0.122, or 0.244 mg/L (0, 8, 16, 32, 64, or 128 ppm formic acid via whole body inhalation (5 days/week, 6h/day) for 13 weeks. In mice, sperm motility values were lower at all concentrations, but no dose-response relationship was seen, and the values were within the range of historical controls. There were no effects in female mice. The NOAEC was therefore 0.244 mg/L, the highest concentration used (ECHA)[KI. score =1].

Dermal

There are no studies available.

J. Developmental Toxicity

Oral

An OECD Guideline 414 (Prenatal Developmental Toxicity Study) was conducted using Himalayan rabbits exposed to 0, 100, 300, and 1000 mg/kg bw/day sodium formate via oral gavage for 22 days. There were no treatment-related effects in mortality, clinical signs, body weight, food consumption, caesarean parameters, and terminal necropsy in the does. The maternal NOAEL is therefore 1000 mg sodium formate/kg bw/day. There were no treatment-related effects in developmental parameters. Foetal weight at birth, sex distribution, placenta weight, pre- and post-implantation loss was not affected. There were no unusual or increased incidences of external, soft tissue or skeletal malformations attributable to the treatment. The developmental NOAEL is therefore 1000 sodium formate mg/kg bw/day or 667 mg/kg bw/day of formic acid. The NOAEL for teratogenicity is also 1000 sodium formate mg/kg bw/day (the highest dose tested) or 667 mg/kg bw/day formic acid. Generally, formate salts are used as test material in studies requiring repeated dosing, due to the corrosivity of formic acid. NOAEL values obtained in such studies may be used to calculate the NOAEL for the formate anion which may be read across to other salts or formic acid, taking into account stoichiometry and formula weights (ECHA) [KI. score =1].

An OECD Guideline 414 (Prenatal Developmental Toxicity) study was conducted using Wistar rats exposed to 0,59,236, 945, g/kg bw/day sodium formate via oral gavage for 17 days. There was no evidence of maternal toxicity, embryo/foetal toxicity, or teratogenicity at dose level in this study. In addition to this, there was no maternal toxicity observed. Gestational parameters were not influenced and there were no effects on the developing foetuses. No malformations or skeletal variations were seen. The NOAEL for maternal and developmental toxicity was 945 mg sodium formate/kg bw/day of sodium formate (the highest dose tested) or 630 mg/kg bw/day formic acid. Generally, formate salts are used as test material in studies requiring repeated dosing, due to the corrosivity of formic acid. NOAEL values obtained in such studies may be used to calculate the NOAEL for the formate anion which may be read across to other salts or formic acid, considering stoichiometry and formula weights (ECHA) [KI. score =1].

Inhalation

There are no studies available.

Dermal

There are no studies available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for formic acid follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

An OECD Guideline 453 (Combined Chronic Toxicity/Carcinogenicity) study was conducted using male and female Crl:HanWist(Glx:BRL)BR rats exposed to 0, 50, 400, and 2,000 potassium formate in

their feed for 104 weeks. The NOAEL for systemic toxicity was 142 mg/kg bw/day. This value will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1
Oral RfD = 142/(10 x 10 x 1 x 1 x 1) = 142/100 = 1.42 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (1.42 x 70 x 0.1)/2 = 4.97 mg/L

B. Cancer

There is no evidence that formic acid is carcinogenic. Therefore, a value for carcinogenicity was not derived in this dossier.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Formic acid does exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Formic acid is of low toxicity to aquatic organisms on an acute and chronic basis.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on formic acid.

Table 3: Acute Aquatic Toxicity Studies on formic acid

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Zebrafish (Brachydanio rerio)	96-hr LC_{50}	130**	1	ECHA
Rainbow trout (Oncorhynchus mykiss)	96-hr LC_{50}	3,500*	2	ECHA
Daphnia magna	48-hr EC_{50}	365**	1	ECHA
Daphnia magna	48-hr EC_{50}	540*	2	ECHA
Pseudokirchneriella subcapitata	72-hr EC_{50}	1,240**	1	ECHA

*Potassium formate

**Ammonium formate

Chronic Studies

In a 21-day *Daphnia* reproduction study, the measured NOEC for formic acid was 100 mg/L (ECHA). [Kl. score = 1]

C. Terrestrial Toxicity

There are no studies available.

D. Calculation of PNEC

The PNEC calculations for formic acid follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E(L)C_{50} values are available for fish (130 mg/L), *Daphnia* (365 mg/L), and algae (1,240 mg/L). Results from long-term studies are available for invertebrates (100 mg/L). On the basis that the data consists of short-term studies for three trophic levels and long-term studies from one trophic level, an assessment factor of 100 has been applied to the available NOEC value of 100 mg/L for invertebrates. The NOEC value is used because the value for invertebrates is lower than the lowest acute E(L)C_{50} values. The $PNEC_{water}$ is 10 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 10.9 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
= (1.40/1280) x 1000 x 10
= 10.9 mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1000 \times BD_{solid}]$
= 0.8 + [(0.2 x 1.24/1000 x 2400]
= 1.40 m^3/m^3

Where:
Kp_{sed} = solid-water partition coefficient (L/kg)
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]
$Kp_{sed} = K_{oc} \times f_{oc}$
= 31 x 0.04
= 1.24 L/kg

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for formic acid was estimated from an OECD guideline 121 study is 31 L/kg(ECHA) [KI. score =1].
F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 4.13 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
= (0.62/1500) x 1000 x 10
= 4.13 mg/kg

Where:
Kp_{soil} = soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]
$Kp_{soil} = K_{oc} \times f_{oc}$
= 31 x 0.02
= 0.62 m^3/m^3

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for formic acid was estimated from an OECD guideline 121 study is 31 L/kg(ECHA) [KI. score =1].
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Formic acid is readily biodegradable and thus does not meet the screening criteria for persistence.

The log K_{ow} formic acid is -2.1. Thus, formic acid does not meet the criteria for bioaccumulation.

The NOECs from the chronic aquatic toxicity studies on formic acid are > 0.1 mg/L. The acute $E(L)C_{50}$ values from the acute aquatic toxicity studies on formic acid are > 1 mg/L. Thus, formic acid does not meet the criteria for toxicity.

The overall conclusion is that formic acid is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H314-Causes severe skin burns and eye damage

Specific target organ toxicity-category 3

Skin corrosion-category 1

Acute toxicity (ingestion)-category 4

Acute toxicity (inhalation)- category 3

STORE Category 2 (target organ: kidney)

B. Labelling

Danger

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace exposure standards for formic acid in Australia is as follows: 9.4 mg/m^3. The short term exposure limit is 19 mg/m^3.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Formic acid is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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GLUTARALDEHYDE

This dossier on glutaraldehyde presents the most critical studies pertinent to the risk assessment of glutaraldehyde in its use in coal seam gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from NICNAS (1994) and the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Glutaraldehyde

CAS RN: 111-30-8

Molecular formula: $C_7H_8O_2$

Molecular weight: 100.12 g/mol

Synonyms: Pentanedial; glutaral; glutaric dialdehyde; 1,3-diformylpropane; 1,5-pentanedial; glutaric aldehyde; glutaric acid dialdehyde; dioxopentane; glutardialdehyde; 1,5-pentanedione; Algicide®C

SMILES: C(CC=O)CC=O

II. PHYSICAL AND CHEMICAL PROPERTIES

Table 1: Overview of the Physico-Chemical Properties of Glutaraldehyde

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa*	Sweetish smelling, clear water liquid	1	ECHA
Melting Point*	-33°C (pressure not provided)	1	ECHA
Boiling Point*	101.5°C @ 98.71 kPa	1	ECHA
Density*	1,130 kg/m^3 @ 20°C	1	ECHA
Vapour Pressure*	21 Pa @ 25°C	1	ECHA
Partition Coefficient (log K_{ow})*	-0.36 @ 23°C and pH 7	1	ECHA
Water Solubility*	Miscible @ 20°C	2	ECHA
Flash Point*	Not measurable	1	ECHA
Auto flammability*	395°C @ ~1,000hPa	1	ECHA
Viscosity*	12.75 mm^2/s (static) at 25°C	1	ECHA
Henry's Law Constant	0.011 Pa m^3/mol at 25°C [QSAR]	2	ECHA

*ca. 50% glutaraldehyde solution (in water)
1 ppm = 4.095 mg/m3
1 mg/m3 = 0.244 ppm

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Glutaraldehyde is considered readily biodegradable. It is also expected to have a low potential for bioaccumulation. The K_{oc} values for glutaraldehyde indicate that it will have low potential for adsorption to suspended solids and sediment in water and moderate adsorption to soil. Glutaraldehyde is not expected to undergo hydrolysis in the environment. Overall, glutaraldehyde shows limited persistence in the environment.

B. Partitioning

In an OECD TG 111 test (hydrolysis as a function of pH), glutaraldehyde was hydrolytically stable at pH 4 and pH 7 but decomposed at pH 9 (ECHA) [Kl. score = 2].

Photolytic degradation of glutaraldehyde occurred in water under sensitised conditions: the half-life was 18 days when equivalent to 36 days of natural sunlight (12 hours/day; sensitised acetone system); and 49 days when equivalent to 34 days of natural sunlight (12 hours/day; sensitised acetonitrile system). There was no photodegradation of glutaraldehyde under darkness or non-sensitised conditions (ECHA) [Kl. score = 2].

C. Biodegradation

Glutaraldehyde was considered readily biodegradable in an OECD 301A (DOC die away test). Degradation was 90-100% in 28 days (ECHA) [Kl. score = 1].

In a simulation test involving aerobic sewage treatment [activated sludge units] (OECD TG 303A), glutaraldehyde degraded 97% after 73 days based on DOC removal (ECHA) [Kl. score = 1].

In an aerobic aquatic metabolism test, [^{14}C]-glutaraldehyde had a half-life of 10.6 hours in the water/sediment system. A minor transformation product was glutaric acid: the maximum yield was 18.9 to 21.5% at 12 hours, which then declined rapidly to 10.1 to 11% by 24 hours; and was not observed at the end of the study period in the aqueous phase (ECHA) [Kl. score = 1].

In an anaerobic aquatic metabolism test, [^{14}C]-glutaraldehyde was rapidly metabolised with the first-order half-life being 7.7 hours. Glutaraldehyde was transformed to 5-hydroxypentanal (ca 37% of applied radioactivity) on day 1; after that, it declined to < 10%; it was not detected at all after 30 days. The second stable transformation product was 1,5-pentanediol (35% of radioactivity on Day 1), which accounted for 70% of the radioactivity at the end of the test. A minor transformation product was a compound formed via Aldol condensation, cyclisation and dehydration. This compound accounted for about 10-20% of total radioactivity from Day 1 onwards (ECHA) [Kl. score = 1].

In an aerobic soil metabolism test, the half-life of the degradation of [^{14}C]-glutaraldehyde was calculated to be 1.7 days, indicating rapid degradation in soil by microbial biotransformation. Degradation products were measured but not identified (ECHA) [Kl. score = 1].

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

D. Environmental Distribution

The organic carbon/water partition coefficients (K_{oc}) values were determined for sediment and four types of soil. The values are as follows: 120 for sediment; 210 for sandy loam; 500 for silty clay loam; 340 for silt loam; and 460 for loamy sand (ECHA; Leung, 2001) [Kl. score = 1].

Based on these K_{oc} values, glutaraldehyde is considered to be moderately mobile in soil. If released to water, based on these K_{oc} values and its water solubility, it has moderate potential for adsorption to suspended solids or sediments.

E. Bioaccumulation

Glutaraldehyde is not expected to bioaccumulate. The measured log K_{ow} at pH 5, 7 and 9 are -0.41, -0.36 and -0.80, respectively (ECHA).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Glutaraldehyde has moderate-to-high acute toxicity by the oral route, low-to-moderate toxicity by the dermal route, and moderate-to-high toxicity by the inhalation route. Acute inhalation exposure may cause respiratory irritation. Glutaraldehyde is corrosive to the skin and eyes; it is also a skin and respiratory sensitiser. Repeated oral exposures via drinking water to rats have resulted in general systemic toxicity, but no target organ effects. In contrast, the upper respiratory tract, particularly the nasal cavity, is the target organ in rodents from repeated inhalation exposure. Glutaraldehyde may exhibit weak genotoxic effects in some *in vitro* tests, whereas the *in vivo* studies consistently show no genotoxic activity. Glutaraldehyde is not a reproductive toxicant; developmental toxicity can occur at maternally toxic doses, but there is no teratogenicity.

B. Toxicokinetics

Dermal Absorption

[1,5-^{14}C]-glutaraldehyde was applied to the skin of male and female F344 rats. Doses were 0.75% and 7.5%: this corresponds to approximately 6.5 and 63 mg/kg for males; and approximately 8.7 and 102 mg/kg for females. The dermal absorption data are presented in Table 2. The results indicate that glutaraldehyde has a low rate of absorption by the dermal route (ECHA).

Table 2: Dermal Absorption Data in Rats on Glutaraldehyde (ECHA)

Sex	Absorption rate constant/hr		% of applied dose	
	Low Dose	High Dose	Low Dose	High Dose
Males	1.5	0.7	0.7	1.3
Females	1.8	0.9	0.3	2.1

An *in vitro* percutaneous absorption study was conducted on glutaraldehyde using excised skin from rats, rabbits, mice, guinea pigs and humans. The skin samples were placed in a flow-through skin penetration chamber, and [^{14}C]-glutaraldehyde was added at doses of 0.75% and 7.5%. The results are presented in Table 3. Glutaraldehyde did not penetrate any of the skin samples to a significant degree, suggesting that only minimal amounts of glutaraldehyde may be available for systemic

uptake and distribution after skin exposure. The results also show that skin absorption was greater for the animal species used in toxicity tests than human skin (ECHA; Frantz et al., 1993).

Table 3: *In vitro* Percutaneous Absorption (mg/cm^2) of Glutaraldehyde (ECHA; Frantz et al., 1993)

Species	Low Dose	High Dose
Animal*	0.006	0.08
Human	0.002	0.02

*Percutaneous absorption in rats, mice, guinea pigs, mice and rabbits were similar to each other and were reported as a single value.

C. Acute Toxicity

The oral LD_{50} values are: 123 to 820 mg/kg in rats; 100 to 352 mg/kg in mice; and 50 mg/kg in guinea pigs (NICNAS, 1994).

The dermal LD_{50} values are: 640 to 2,000 mg/kg in rabbits; > 2,500 mg/kg in rats; and > 4,500 mg/kg in mice (NICNAS, 1994).

The 4-hour inhalation LC_{50} values for glutaraldehyde are listed in Table 4:

Table 4: Acute inhalation LC_{50} values for Glutaraldehyde

Test Material	LC_{50} (males) [mg/L]	LC_{50} (females) [mg/L]	LC_{50} (both sexes) [mg/L]	Reference
50% aq. aerosol	0.52	0.45	-	OECD, 1995
25% aq. aerosol	-	-	0.8	OECD, 1995
50% aq. aerosol	0.35	0.28	-	OECD, 1995
5% soln. vapour	0.096	0.164	-	OECD, 1995

During the exposure period, the animals showed signs of eye and respiratory irritation, as indicated by laboured and audible breathing, and wetness and encrustation around the nose and eyes.

D. Irritation

Glutaraldehyde is corrosive to the skin and eyes of rabbits (NICNAS, 1994; ECHA). Signs of irritation occurred at a concentration of 2% for skin and 0.2% for eyes (NICNAS, 1994). In the acute inhalation studies, rats exposed to aerosols or vapours of glutaraldehyde showed signs of eye and respiratory irritation (OECD, 1995).

E. Sensitisation

Glutaraldehyde is a skin sensitiser to guinea pigs and humans. Information on the individual studies can be found in NICNAS (1994) and in the ECHA REACH database (ECHA).

Asthmatic symptoms, such as wheezing, coughing, chest tightness, breathing difficulties and non-specific hyper-responsiveness have been reported to occur in humans occupationally exposed to glutaraldehyde (NICNAS, 1994). It is unclear whether the asthma is an allergic hypersensitivity response or a result of the aggravation of pre-existing asthma due to the irritating properties of

glutaraldehyde. Nevertheless, glutaraldehyde should be considered a respiratory sensitiser, although one of low potency.

F. Repeated Dose Toxicity

Oral

Male and female Wistar rats were given in their drinking water 0, 100, 500, or 2,000 ppm glutaraldehyde for 90 days. The approximate daily intakes were 0, 3, 15 or 53 mg/kg/day for males, and 0, 4, 19 or 72 mg/kg/day for females. There were no signs of neurotoxicity at any dose level. There was slight impairment of food consumption in the 2,000 ppm animals, as well as slight impairment of body weight and body weight gain. Impaired water consumption was seen in the 100 and 500 ppm females. The NOAEL for males is 500 ppm (15 mg/kg/day). The NOAEL for females is 100 ppm (4 mg/kg/day) since the impaired water consumption in the 100 ppm females was considered a palatability problem and not an adverse effect (ECHA) [Kl. score = 1].

Male and female F344 rats were given in their drinking water 0, 50, 250 or 1,000 ppm glutaraldehyde for 13 weeks. Additional groups of animals were given in their drinking water 0 or 1,000 ppm glutaraldehyde for 13 weeks followed by a 4-week recovery period. The approximate daily intakes were 0, 5, 25 or 100 mg/kg/day for males; and 0, 7, 35 or 120 mg/kg/day for females. Water consumption was reduced in a dose-dependent manner in the $\geq$ 250 ppm males and 1,000 ppm females, which was attributed to an aversion to the taste and/or odour of glutaraldehyde in the water. There was also a reduction in food consumption in the 1,000 ppm animals with a parallel reduction in body weights. It is unclear whether the reduction in food consumption was related to the decreased water consumption. Urine volume was decreased with an increase in specific gravity, along with a slight increase in protein and ketone concentration, in the $\geq$ 250 ppm animals, which was probably related to the decreased water consumption. There were no treatment-related changes in the haematology parameters measured. Blood urea nitrogen was increased in a dose-related manner in the $\geq$ 250 ppm females at the 6-week time point, but not at the 13-week or 17-week time points. Relative kidney weights were increased in a dose-related manner in the $\geq$ 250 ppm males and females and increased absolute kidney weights in the females. Histopathological examination showed no treatment-related effects. The NOAEL is 50 ppm (5 and 7 mg/kg/day for males and females, respectively) based on dose-related increase in kidney weights at $\geq$ 250 ppm (ECHA) [Kl. score = 2].

Male and female Wistar rats were given in their drinking water 0, 100, 500 or 2,000 ppm glutaraldehyde for 12 months. The approximate daily intakes were: 0, 6.4, 30.5, or 116.6 mg/kg/day for males; and 0. 9.6, 46, or 153 mg/kg/day for females. There was no treatment-related mortality. At 2,000 ppm, treatment-related effects included respiratory sounds (both sexes), decrease in body weight (males), decrease in body weight gain (both sexes), decrease in food consumption (both sexes), reduced water consumption (both sexes), lesions within the glandular stomach (both sexes showed erosion/ulceration of the glandular stomach), increased incidence of clear cell foci in the liver (males) and a single case of slight diffuse squamous metaplasia in the epithelium of the larynx (male). At 500 ppm, water consumption was reduced in males which was considered to be a palatability (bad taste) problem and not an adverse effect. No effects were seen in the 100 ppm animals. The NOAEL for this study is 500 ppm, which corresponds to 30.5 and 46 mg/kg/day for males and females, respectively (ECHA) [Kl. score = 1].

Male and female Fischer 344 rats were given in their drinking water 0, 50, 250 or 1000 ppm glutaraldehyde for 104 weeks. The mean glutaraldehyde consumption was 0, 4, 17 and 64 mg/kg/day for males and 0, 6, 25 and 86 mg/kg/day for females. There were no treatment-related mortalities or clinical symptoms of toxicity. In the 250 and 1,000 ppm groups, there was reduction in

body weight and body weight gain; reduction in food and water consumption; increased statistically significant incidence of nucleated erythrocytes and of large monocytes; decreases in alanine aminotransferase (ALT), aspartate aminotransferase (AST), and glutamate dehydrogenase; dose-related decrease in urine volume accompanied by a dose-related increase in osmolality; changes in absolute and relative kidney weight; gastric irritation; increases in bone marrow hyperplasia; and increased incidence of renal tubular pigmentation. The decreased water consumption was considered to be due to the bad taste, smell and/or irritancy of the test substance in the drinking water; thus, it is of no toxicological relevance. As a result of reduced water intake, there are renal physiological adaptation, such as decreased urine, increased osmolality and changes in kidney weight. The haematological and clinical chemistry parameter changes were marginal and were considered to be of no toxicological relevance. The main haematological finding seen at the end of the study, which consisted of the appearance of nucleated erythrocytes and large monocytes in all treated groups (statistically significant for the ≥250 ppm males), was related to the incidence of large granular lymphocytic leukaemia (LGLL) in the spleen. The bone marrow hyperplasia and renal tubular pigmentation are related to the occurrence/incidence of LGLL and were considered by the authors of the study as being secondary to low-grade haemolytic anaemia in animals with LGLL. The NOAEL for this study is 50 ppm which corresponds to 4 and 6 mg/kg/day for males and females, respectively (Van Miller et al., 2002) [Kl. score = 2].

Inhalation

Male and female F344 rats were exposed by inhalation to 0, 0.0625, 0.125, 0.25, 0.5 or 1.0 ppm (0, 0.26, 0.5, 1, 2 or 4.1 mg/m^3) glutaraldehyde for 6.5 hours/day, 5 days/week for 13 weeks. The study focused on the respiratory tract, using histopathology and epithelial cell labelling index as end points. Histopathological lesions in the nasal passages and turbinates were seen at ≥ 0.25 ppm. Treatment-related effects were primarily the respiratory mucosa (nasal cavity and tips of the turbinates) and the olfactory epithelium (dorsal meatus). Hyperplasia, squamous metaplasia, olfactory degeneration, squamous exfoliation (accumulation of keratin, cell debris and bacteria in the lumen of the nasal vestibule) and focal erosions were reported for both sexes, and the severity and incidence of the findings increased with increasing concentration of glutaraldehyde. The NOAEL for this study is 0.125 ppm (Gross et al., 1994) [Kl. score = 1].

Male and female B6C3F$_1$ mice were exposed by inhalation to 0, 0.0625, 0.125, 0.25, 0.5 or 1.0 ppm (0, 0.26, 0.5, 1, 2 or 4.1 mg/m^3) glutaraldehyde for 6.5 hours/day, 5 days/week for 13 weeks. The study focused on the respiratory tract, using histopathology and epithelial cell labelling index as end points. Histopathologic lesions in the nasal passages and turbinates were seen at all exposure concentrations (≥ 0.0625 ppm). Treatment-related lesions were primarily the respiratory mucosa (nasal cavity and tips of the turbinates) and the olfactory epithelium (dorsal meatus). Hyperplasia, squamous metaplasia, olfactory degeneration, squamous exfoliation (accumulation of keratin, cell debris and bacteria in the lumen of the nasal vestibule) and focal erosions were reported for both sexes, and the severity and incidence of the findings increased with increasing test concentration. Furthermore, neutrophilic inflammation was seen at ≥ 0.062 ppm, and squamous metaplasia as well as necrosis were seen in the larynx at 1 ppm. The LOAEL for this study is 0.0625 ppm; a NOAEL was not established (Gross et al*.,* 1994) [Kl. score = 1].

Male and female B6C3F$_1$ mice were exposed by inhalation to 0 or 0.1 ppm (0 or 0.41 mg/m^3) glutaraldehyde for 6 hours/day, 5 days/week for 52 and 78 weeks. Survival was similar between treated and control groups. Hyperplasia of the squamous epithelium lining of the dorsal wall of the nasal passages and the lateral aspect of the atrioturbinate was seen in a greater number of exposed females than in controls. Epidermal erosion and ulceration as well as squamous and inflammatory exfoliation were also seen in the nasal lumens. All of these changes were dependent on the length of

glutaraldehyde exposure. The authors concluded that, since the induced lesions occurred in the more anterior part of the nasal passages, that they were likely the result of an irritation mechanism (Zissu et al., 1998) [Kl. score = 2].

Male and female Fischer 344 rats were exposed by inhalation to 0, 0.25, 0.5, or 0.75 ppm (0, 1, 2, or 3.1 mg/m^3) glutaraldehyde for 6 hours/day, 5 days/week for two years. Survival in the mid- and high-dose females was statistically significantly decreased compared to controls. Mean body weights of all exposed males and the mid- and high-dose females were generally less than those of the controls. Non-neoplastic lesions were limited primarily to the most anterior region of the nasal cavity. Effects included hyperplasia and inflammation of the squamous epithelium; hyperplasia, goblet cell hyperplasia, inflammation and squamous metaplasia of the respiratory epithelium; and hyaline degeneration of the olfactory epithelium. The LOAEL for this study is 0.25 ppm based on hyperplasia and inflammation of the squamous epithelium of the nose in both sexes. A NOAEL was not established (van Birgelen et al., 2000) [Kl. score = 2].

Male and female B6C3F$_1$ mice were exposed by inhalation to 0, 0.0625, 0.125 or 0.25 ppm (0, 0.26, 0.5 or 1 mg/m^3) glutaraldehyde for 6 hours/day, 5 days/week for two years. Survival of the treated animals was similar to controls. Mean body weights of the high-dose females were generally lower than the controls. Non-neoplastic lesions were limited primarily to the anterior region of the nasal cavity; the effects were qualitatively similar to those seen in the rats (see accompanying summary on the two-year rat study by van Birgelen et al. [2000]). Squamous metaplasia of the respiratory epithelium was observed in both sexes of mice while female mice also had inflammation and hyaline degeneration of the respiratory epithelium. The incidence and severity grade (in parentheses) of the hyaline degeneration were: 16/50 (1.4), 35/49 (1.4), 32/50 (1.3) and 30/50 (1.1) for the 0, 0.0625, 0.125 and 0.25 ppm dose groups, respectively. The LOAEL for this study is 0.0625 ppm based on hyaline degeneration of the respiratory epithelium in female mice. A NOAEL was not established (van Birgelen et al., 2000) [Kl. score = 2].

Dermal

Applications of a 50% solution of glutaraldehyde was applied to the skin of male and female SD rats for 13 weeks. The doses were 0, 50, 100 and 150 mg/kg glutaraldehyde. At the application site, there were signs of irritation (scabs, desquamation and very slight or well-defined erythema). There was no treatment-related mortality, clinical signs, body weights, feed consumption and ophthalmoscopic effects. There were no changes in the haematology and clinical chemistry parameters that were considered to be biologically or toxicologically relevant. Organ weights were similar between treated and control animals. Histopathological examination showed treatment-related effects in the skin associated with chronic irritation; no other changes were noted that were considered to be treatment-related. The NOAEL for this study is 150 mg/kg, the highest dose tested (ECHA) [Kl. score = 1].

G. Genotoxicity

In Vitro Studies

Glutaraldehyde may exhibit weak genotoxic effects in some *in vitro* tests. The bacterial reverse mutation assays have been the most consistent. Variable results have been reported for the forward gene mutation tests; and for sister chromatid exchange (SCE), chromosomal aberration and Unscheduled DNA Synthesis (UDS) tests (Vergnes and Ballantyne, 2002).

In vivo Studies

The *in vivo* studies conducted on glutaraldehyde are presented in Table 5. All the studies show that glutaraldehyde is not mutagenic or genotoxic.

Table 5: *In vivo* Genotoxicity Studies on Glutaraldehyde

Test System	Results*	Klimisch Score	Reference
Rat bone marrow (chromosomal aberration)	-	1	ECHA
Rat bone marrow (chromosomal aberration)	-	2	ECHA
Mouse bone marrow (micronucleus)	-	1	ECHA
Rat bone marrow (chromosomal aberration)	-	2	ECHA
Rat germ cell cytogenetic assay (alkaline elution)	-	2	ECHA
Drosophila SLRL Test	-	2	ECHA
Rat liver UDS Assay	-	1	ECHA
Rat germ cell cytogenetic assay (alkaline elution)	-	2	ECHA
Mouse peripheral blood micronucleus study	-	2	Vernes and Ballantyne (2002)
Rat liver UDS Assay	-	2	Mirsalis *et al.* (1989)

* +, positive; -, negative

H. Carcinogenicity

Oral

Male and female Fischer 344 rats were given in their drinking water 0, 50, 250 or 1,000 ppm glutaraldehyde for 104 weeks. The mean glutaraldehyde consumption was 0, 4, 17 and 64 mg/kg/day for males and 0, 6, 25 and 86 mg/kg/day for females. Mortality rates were 25-30% and 19-23% for males and females, respectively, with no dose-related increase. The major cause of death in all dose groups including the controls was LGLL. There was an increased incidence of LGLL in the liver and spleen in all treated females ($\geq$ 50 ppm). The incidence of LGLL was not significantly increased in the treated males compared to the controls. No other treatment-related increased incidence of tumours was seen (Van Miller et al., 2002) [Kl. score = 2].

Male and female Wistar rats were given in their drinking water 0, 100, 500 or 2,000 ppm glutaraldehyde for two years. The mean daily intake of glutaraldehyde was as follows: 0, 6.1, 31.9 and 120.7 mg/kg/day for males; and 0, 10.5, 48.5 and 176.4 mg/kg/day for females. In the high-dose animals, there was mortality (2 males and 9 females) from asphyxia, and mean terminal body weights were significantly decreased compared to the controls. There were no treatment-related neoplastic effects (ECHA) [Kl. score = 1].

Inhalation

Male and female $B6C3F_1$ mice were exposed by inhalation to 0 or 0.1 ppm (0 or 0.4 mg/m^3) glutaraldehyde for 6 hours/day, 5 days/week for 52 and 78 weeks. No exposure-related neoplastic lesions were observed in either males or females (Zissu et al., 1998) [Kl. score = 2].

Male and female Fischer 344 rats were exposed by inhalation to 0, 0.25, 0.5 or 0.75 ppm (0, 1, 2 or 3.1 mg/m^3) glutaraldehyde for 6 hours/day, 5 days/week for two years. Survival in the mid- and high-dose females was statistically significantly decreased compared to controls. Survival of the treated males was similar to controls. No exposure-related neoplastic lesions were observed in either males or females (van Birgelen et al., 2000) [Kl. score = 2].

Male and female B6C3F$_1$ mice were exposed by inhalation to 0, 0.0625, 0.125 or 0.25 ppm (0, 0.26, 0.5 or 1 mg/m^3) glutaraldehyde for 6 hours/day, 5 days/week for two years. Survival of the treated animals was similar to controls. No exposure-related neoplastic lesions were observed in either males or females (van Birgelen et al., 2000) [Kl. score = 2].

I. Reproductive Toxicity

A two-generation reproductive toxicity study was conducted in Wistar rats given 0, 100, 500 and 2,000 ppm glutaraldehyde in their drinking water. The approximately mean daily intake is 0, 12, 58 and 199 mg/kg/day for the parental males and females of the F_0 and F_1 generation during premating. There were no adverse effects on reproductive performance or fertility. Oestrous cycle data, mating behaviour, conception, gestation, parturition, lactation and weaning as well as sperm parameters, sexual organ weights, gross and histopathological findings of these organs were similar between treated and control groups. In the high-dose animals, there was decreased water and/or food consumption; and decreased body weights and/or reduced body weight gains during the premating periods in the F_0 and F_1 parental females during premating, gestation and/or lactation. The high-dose F_1 parental females also had increased the number of erosions/ulcers with microscopic erosion(s) or inflammatory oedema in the mucosa/submucosa of the glandular stomach. There were no adverse effects in the 500 ppm animals except for slight decreases in water consumption due to a palatability (bad taste) problem. Treatment-related signs of developmental toxicity were seen in the progeny of the high-dose F_0 and F_1 parental generation and included impairment in body weight and consequently in organ weights in the respective F_1 and F_2 pups. The NOAEL for reproductive toxicity is 2,000 ppm (199 mg/kg/day), the highest dose tested. The NOAEL for parental systemic toxicity is 500 ppm (58 mg/kg/day). The NOAEL for developmental toxicity is 500 ppm or 58 mg/kg/day (ECHA) [Kl. score = 1].

A two-generation reproductive toxicity study was conducted in Crj: CD(SD) rats given 0, 50, 250 and 1,000 ppm glutaraldehyde in their drinking water. Mean daily intake was not calculated. Parental body weights and body weight gains were significantly reduced at 1,000 ppm at some periods, particularly during pre-mating. Food consumption was significantly reduced at 1,000 ppm for the F_0 and F_1 parental animals during pre-mating and gestation, and F_1 females during lactation. Water consumption was reduced throughout the pre-mating period for the F_0 and F_1 250 and 1,000 ppm parental animals. There was no indication of adverse effects on reproductive performance or fertility at any dose level. For the F_1 1,000 ppm offspring, body weights were reduced from lactation days 21-28. The NOAEL for reproductive toxicity is 1,000 ppm, the highest dose tested. The NOAEL for parental systemic toxicity is 50 ppm. The NOAEL for developmental toxicity is 250 ppm (Neeper-Bradley and Ballantyne, 2000) [Kl. score = 2].

J. Developmental Toxicity

Pregnant Wistar rats were given in their drinking water 0, 50, 250 or 750 ppm (0, 5, 26 or 68 mg/kg) glutaraldehyde from GD 6 to 16. Water consumption was reduced in a dose-related manner in the $\geq$ 250 ppm dams, and was considered not to be a toxic response, but due to the palatability (bad taste) of the drinking test solution. No other maternal effects were seen in the study. There were no significant differences between treated and controls in the sex distribution, placental weights, foetal

weights, malformations or variations. The NOAEL for maternal and developmental toxicity in this study is 68 mg/kg/day, respectively (ECHA) [Kl. score = 1].

Pregnant Wistar rats were dosed by oral gavage with 0, 25, 50 or 100 mg/kg glutaraldehyde on GD 6 to 15. Mortality was significantly increased in the high-dose group (5/26); there were 2/21 deaths in the mid-dose group. Clinical signs (piloerection) occurred in all treated groups in a dose-dependent manner. Maternal body weight gain and feed consumption were significantly reduced in the high-dose dams, but not at the lower doses. The necropsy findings showed evidence of stomach irritation in almost all of the animals that died during the study and in 12/21 of the surviving dams in the high-dose group. The number of implantations per litter, resorptions and dead foetuses per litter, live foetuses per litter and incidence of post-implantation loss per litter was similar across all groups. The mean foetal body weights for male and female foetuses were significantly reduced in the high-dose group; this was attributed to the reduced food consumption of the dams during gestation rather than a direct effect of treatment. There was no evidence of a treatment-related teratogenic effect. The NOAEL for maternal and developmental toxicity is 50 mg/kg/day, respectively (Ema et al., 1992) [Kl. score = 2].

Pregnant Himalayan rabbits were dosed by oral gavage with 0, 5, 15 or 45 mg/kg glutaraldehyde on GD 7 to 19. In the high-dose group, 5/15 died on GD 9-11. Food consumption and body weight gain were also significantly reduced in the high-dose group. Clinical observations in 12/15 high-dose does included soft faces, diarrhoea and blood in the bedding. The mean gravid uterus weight was significantly reduced in the high-dose group. Post-implantation loss was greatly increased (94.3%) in the high-dose group: no viable foetuses in 9/15 of the high-dose does, only early resorptions; only one female gave four alive foetuses on the scheduled date. There were reduced placental and foetal body weights in the only four foetuses. No significant maternal or developmental effects were seen in the mid- and low-dose groups. The NOAEL for maternal and developmental toxicity in this study is 15 mg/kg/day (ECHA) [Kl. Score = 2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for glutaraldehyde follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

The lowest NOAEL values from key toxicity studies on glutaraldehyde are listed in Table 6.

Table 6: Lowest NOAEL Values from Key Toxicity Studies on Glutaraldehyde by the Oral Route

Species/Sex	Study Duration	mg/kg/day	Endpoint	Reference
Rats, female	90/days	4	Decreased body weights, food and water consumption	ECHA
Rats, male	13-wk (drinking water)	5	Increased kidney weights	ECHA

Species/Sex	Study Duration	mg/kg/day	Endpoint	Reference
Rats, male	12-months (drinking water)	30.5	Clinical signs; decreased body weights and food consumption; increased clear cell foci in liver	ECHA
Rats, male	2-yr (drinking water)	4	Reduced body weight, body-weight gain, and food consumption	Van Miller *et al.* (2002)
Rats	2-generation (drinking water)	58	Systemic toxicity	ECHA
Rats	GD 6-16 (drinking water)	68	Developmental toxicity	ECHA
Rats	GD 6-15 (oral gavage)	50	Developmental toxicity	Ema *et al.* (1992)
Rabbits	GD 7-19 (oral gavage)	15	Developmental toxicity	ECHA

The lowest NOAEL from these studies is 4 mg/kg/day based on reduced body weights, body weight gain and feed consumption in male rats from the two-year drinking water study (Van Miller et al., 2002). The NOAEL of 4 mg/kg/day will be used for determining the oral Reference Dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1
Oral RfD = 4/(10 x 10 x 1 x 1 x 1) = 4/100 = <u>0.04 mg/kg/day</u>

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD: Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2 L (ADWG, 2011)
Drinking water guidance value = (0.04 x 70 x 0.1)/2 = <u>0.14 mg/L</u>

B. Cancer

Increased incidence of large granular cell lymphatic leukaemia (LGLL) was observed in all groups of male and female Fischer 344 rats given glutaraldehyde in their drinking water, including the controls (Van Miller *et al.,* 2002). For the males, the incidence of LGLL was not statistically significantly increased. However, for the females, the incidence of LGLL was significantly increased in all treated females (≥ 50 ppm). Inhalation exposure of Fischer 344 rats to glutaraldehyde did not result in an increased incidence of tumours, including LGLL.

LGLL, also known as mononuclear cell leukaemia, is an extremely common spontaneous neoplastic disease of the ageing F344 rat (Stromberg, 1985; Ward *et al.* 1990; Thomas et al., 2007). Consistent features are splenomegaly, anaemia, thrombocytopenia and leukemic infiltration of the spleen, liver, lung, and in an advanced stage, of several other organs. The incidence is variable but has been increasing progressively with time and can exceed 70% in controls in some studies. This compares with background incidence of less than 1% in other strains of commonly used laboratory rats (Haseman et al., 1998; Thomas et al., 2007). The incidence in F344 rats is modulated by a variety of factors not clearly related to carcinogenicity. Corn oil gavage, for example, has been shown consistently to reduce the incidence of MCL in male, but not female, controls (reviewed in Thomas et al., 2007).

The neoplastic mononuclear cells appear to be derived from large granular lymphocytes (LULs) (reviewed in Thomas et al., 2007). The tumour cell is of the NK type in most, if not all, cases. LGL leukaemia, although uncommon, does occur in humans. There are two types: T-LGL leukaemia which has a chronic course characterised by neutropenia, recurrent infections, splenomegaly and accompanying rheumatoid arthritis, and the much rarer NK-LGL leukaemia which has an acute course, more pronounced splenomegaly, and thrombocytopenia. The latter type appears to resemble more closely the disease in the F344 rat than the former. The aetiology of human LGL leukaemia is unknown. There is some evidence that viral infection may play a role but no evidence that a chemically-related increased of LGLL in the F344 rat is indicative of the potential to induce LGL leukaemia in humans.

To extrapolate results from an animal model that has a clear predisposition (high spontaneous rates) to a tumour type to humans, of which this is not the case, seems inappropriate if the mechanism(s) for LGLL formation in that strain is not understood. Although that rat strain may be useful for understanding the disease process in humans, it does not seem reasonable to use the results from that rat strain for risk assessment purposes. There should be confirmation of a putative leukemogenic effect in the F344 rat in another strain before any conclusions are made about the use of this tumour type for human health risk assessment purposes.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Glutaraldehyde does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Glutaraldehyde has a moderate acute toxicity concern to fish and invertebrates, but is highly toxic to algae. It is of low toxicity concern to terrestrial invertebrates and plants. To birds, glutaraldehyde is moderately toxic on an acute basis and slightly toxic on a subacute dietary basis.

B. Aquatic Toxicity

Acute Studies

Table 7 lists the results of acute aquatic toxicity studies conducted on glutaraldehyde.

Table 7: Acute Aquatic Toxicity Studies on Glutaraldehyde

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Bluegill sunfish	96-hr LC_{50}	13	2	ECHA
Oncorhynchus mykiss	96-hr LC_{50}	10	2	ECHA
Daphnia magna	48-hr LC_{50}	14.87	2	ECHA
Daphnia magna	48-hr LC_{50}	14	2	ECHA
Scenedesmus subspicatus	72-hr EC_{50}	0.375 (biomass) 0.6 (growth rate) 0.025 (NOEC)	1	ECHA
Scenedesmus subspicatus	72-hr EC_{50}	0.92 (growth rate) 0.61(biomass) 0.33 (NOEC)	2	ECHA; Leung, 2001
Scenedesmus subspicatus	72-hr EC_{50}	0.61 (growth rate)	2	ECHA

Chronic Studies

The chronic aquatic toxicity studies conducted on glutaraldehyde are listed in Table 8.

Table 8: Chronic Aquatic Toxicity Studies on Glutaraldehyde

Test Species	Endpoint	Results (mg/L)	Kl. score	Reference
Oncorhynchus mykiss	97/day (OECD 210)	LOEC = 5 NOEC = 1.6	1	ECHA
Daphnia magna	21/day	NOEC = 5	1	ECHA

C. Terrestrial Toxicity

Table 9 lists the results of toxicity studies conducted on glutaraldehyde with earthworms, soil microorganisms and birds.

Table 9: Terrestrial Toxicity Studies on Glutaraldehyde

Test Species (method)	Endpoint	Results	Kl. score	Reference
Earthworm *Eisenia fetida* (OECD 207)	14-d LC_{50}	> 500 mg/kg soil dw	1	ECHA
Soil microorganisms* (OECD 216)	28-d EC_{50} 28-d EC_{10}	360 mg/kg soil dw 11.5 mg/kg soil dw	1	ECHA
Soil microorganisms* (OECD 217)	28-d EC_{50} 28-d EC_{10}	> 593 mg/kg soil dw 1.5 mg/kg soil dw	1	ECHA
Mallard ducks	Single-dose (oral gavage) LC_{50}	206 mg/kg	2	ECHA
Mallard ducks	5-d (dietary) NOEC	> 2,500 ppm	1	ECHA

*organic carbon content of soil = 1.34% dry weight

Glutaraldehyde has also been evaluated in a terrestrial plants test: seedling emergence and seedling growth test (OECD TG 208). The test material contained 48.9% glutaraldehyde. The results are as follows:

Avena sativa (oats): 19/day EC_{50} value is > 1,000 mg/kg soil dry weight based on emergence rate, dry weight and shoot length. The NOECs for *Avena sativa* (oats) were $\geq$ 1,000 mg/kg dry weight on all three parameters tested.

Brassica napus (rapeseed): 19/day EC_{50} is > 1,000 mg/kg soil dry weight based on emergence rate and shoot length and 994 mg/kg soil dry weight based on dry weight. The NOECs were $\geq$ 1,000, 500 and 250 mg/kg soil dry weight for emergence rate, dry matter and shoot length, respectively.

Vicia sativa (vetch): 19/day EC_{50} is > 1,000 mg/kg soil dry weight based on emergence rate and shoot length, and 901 mg/kg soil dry weight based on dry weight. The NOECs were $\geq$ 1,000, 125 and 125 mg/kg soil dry weight for emergence rate, dry matter, and shoot length, respectively (ECHA) [Kl. score = 1].

D. Calculation of PNEC

The PNEC calculations for glutaraldehyde follow the methodology discussed in DEWHA (2009).

PNEC water

Experimental results are available for three trophic levels. Acute $E(L)C_{50}$ values are available for fish (10 mg/L), *Daphnia* (14 mg/L) and algae (0.375 mg/L). Results from chronic studies are also available for all three trophic levels, with the lowest NOEC being 0.025 mg/L for algae. On the basis that the data consists of short-term and long-term results from three trophic levels, an assessment factor of 10 has been applied to the lowest reported NOEC of 0.025 mg/L for algae. The $PNEC_{water}$ is 0.0025 mg/L.

PNEC sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 0.006 mg/kg wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
= (3.1/1280) x 1000 x 0.0025
= 0.006 mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [0.2 \times Kp_{sed}/1000 \times BD_{solid}]$
= 0.8 + [(0.2 x 4.8)/1000 x 2400]
= 3.1 m^3/m^3

Where:
Kp_{sed} = solid-water partition coefficient (L/kg).
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]
$Kp_{sed} = K_{0c} \times f_{oc}$
= 120 x 0.04
= 4.8 L/kg

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for glutaraldehyde in sediment is 120.
F_{oc} = fraction of organic carbon suspended sediment = 0.04 [default].

PNEC soil

Experimental results are available for three trophic level. An acute LC_{50} value is available for earthworms (> 500 mg/kg). Results from long-term studies are available for two trophic levels, with the lowest NOEC or EC_{10} being 1.5 mg/kg soil dry weight for soil organisms.

The EC_{10} value is corrected for bioavailability of glutaraldehyde in soil by normalising to the fraction organic carbon matter content (Fom) in the soil using the following equation:

$EC_{10(std)} = EC_{10(exp)} \times Fom_{soil(std)}/Fom_{soil(exp)}$

Where:
$Fom_{soil(std)}$ = 1% (default soil fraction organic matter)
$Fom_{soil(exp)}$ = 1.34% (see Table 9)
$EC_{10(std)}$ = 1.5 mg/kg x 1/1.34 = 1.12 mg/kg

On the basis that the data consists of one short-term result from one trophic level and two long-term results from two additional levels, an assessment factor of 50 has been applied to the lowest reported long-term EC_{10} of 1.12 mg/kg soil dry weight [corrected for organic carbon content] for soil organisms. The $PNEC_{soil}$ is 0.02 mg/kg soil dry weight.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2008).

Glutaraldehyde is readily biodegradable and thus does not meet the screening criteria for persistence.

The log K_{ow} for glutaraldehyde at different pH values ranges from -0.36 to -0.80. Thus, glutaraldehyde does not meet the screening criteria for bioaccumulation.

The lowest NOEC value from chronic aquatic toxicity studies is < 0.1 mg/L. Thus, glutaraldehyde meets the screening criteria for toxicity.

The overall conclusion is that glutaraldehyde is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Acute Toxicity Category 3 [oral]

Acute Toxicity Category 2 [inhalation]

Skin Corrosion Category 1B

Eye Damage Category 1

Respiratory Sensitiser 1A

Skin Sensitiser 1A

STOT Single Exposure Category 3 [respiratory irritation]

Aquatic Acute Category 1

Aquatic Chronic Category 2

The appropriate hazard statements corresponding the GHS classifications are to be added to the SDS, including the non-GHS hazard statement “AUH071: Corrosive to the Respiratory Tract”.

B. Labelling

Danger

C. Pictograms

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

First aid information was obtained from the ECHA REACH database (ECHA).

Eye Contact

Wash immediately and continuously with flowing water for at least 30 minutes. Remove contact lenses after the first 5 minutes and continue washing. Obtain prompt medical consultation, preferably from an ophthalmologist. Eye wash fountain should be located in immediate work area.

Skin Contact

Take off contaminated clothing. Wash skin with soap and plenty of water for 15-20 minutes. Call a poison control centre or doctor for treatment advice. Wash clothing before reuse. Shoes and other leather items which cannot be decontaminated should be disposed of properly. Safety shower should be located in immediate work area.

Inhalation

Move person to fresh air. If a person is not breathing, call an emergency responder or ambulance, then give artificial respiration; if by mouth-to-mouth use rescuer protection (pocket mask, etc.). Call a poison control centre or doctor for treatment advice. If breathing is difficult, oxygen should be administered by qualified personnel.

Ingestion

If the person is fully alert and cooperative, have the person rinse mouth with plenty of water. In cases of ingestion have the person drink 4 to 10 ounces (120-300 mL) of water. Do not induce vomiting. Do not attempt mouth rinse if the person has respiratory distress, altered mental status, or nausea and vomiting. Call a physician and/or transport to an emergency facility immediately. See Note to Physician. Seek medical attention immediately.

Notes to Physician

Maintain adequate ventilation and oxygenation of the patient. May cause asthma-like (reactive airways) symptoms. Bronchodilators, expectorants, antitussives and corticosteroids may be of help. Glutaraldehyde may transiently worsen reversible airways obstruction including asthma or reactive airways disease. Chemical eye burns may require extended irrigation. Obtain prompt consultation, preferably from an ophthalmologist. If the burn is present, treat as any thermal burn, after decontamination. Due to irritant properties, swallowing may result in burns/ulceration of mouth, stomach and lower gastrointestinal tract with subsequent stricture. Aspiration of vomitus may cause lung injury. Suggest endotracheal/oesophagal control if lavage is done. Probable mucosal damage may contraindicate the use of gastric lavage. Inhalation of vapours may result in skin sensitisation. In sensitised individuals, re-exposure to very small amounts of vapour, mist or liquid may cause a severe allergic skin reaction. No specific antidote. Treatment of exposure should be directed at the control of symptoms and the clinical condition of the patient. Have the Safety Data Sheet, and if available, the product container or label with you when calling a poison control centre or doctor, or going for treatment.

Medical Conditions Aggravated by Exposure

Excessive exposure may aggravate pre-existing asthma and other respiratory disorders (e.g., emphysema, bronchitis, reactive airways dysfunction syndrome).

Emergency Personnel Protection

First Aid responders should pay attention to self-protection and use the recommended protective clothing (chemical resistant gloves, splash protection). If the potential for exposure exists, refer to Section 8 of the Safety Data Sheet for specific personal protective equipment.

B. Fire Fighting Information

Firefighting information was obtained from the ECHA REACH database (ECHA).

Extinguishing Media

Use water fog, carbon dioxide, dry chemical or foam to extinguish combustible residues of this product

Specific Exposure Hazards

This material will not burn until the water has evaporated. Residue can burn. Some components of this product may decompose under fire conditions. The smoke may contain unidentified toxic and/or irritating compounds. Combustion products may include, and are not limited to, carbon monoxide and carbon dioxide.

Special Protective Equipment for Firefighters

Wear positive-pressure self-contained breathing apparatus (SCBA) and protective firefighting clothing (includes firefighting helmet, coat, trousers, boots and gloves). Avoid contact with this material during firefighting operations. If contact is likely, change to full chemical resistant firefighting clothing with self-contained breathing apparatus. If this is not available, wear full chemical resistant clothing with self-contained breathing apparatus and fight the fire from a remote location.

C. Accidental Release Measures

Information on accidental release measures was obtained from the ECHA REACH database (ECHA).

Personal Precautions

Use appropriate safety equipment. Evacuate area. Keep upwind of the spill. Ventilate area of leak or spill. Only trained and properly protected personnel must be involved in clean-up operations.

Environmental Precautions

Spills or discharge to natural waterways is likely to kill aquatic organisms. Prevent from entering into soil, ditches, sewers, waterways and/or groundwater.

Steps to be Taken if Material is Released or Spilt

Avoid making contact with spilt material; glutaraldehyde will be absorbed by most shoes. Always wear the correct protective equipment, consisting of splash-proof mono-goggles, or both safety glasses with side shields and a wraparound full-face shield, appropriate gloves and protective clothing. A self-contained breathing apparatus or respirator and absorbents may be necessary, depending on the size of the spill and the adequacy of ventilation.

Small spills: Wear the correct protective equipment and cover the liquid with absorbent material. Collect and seal the material and the dirt that has absorbed the spilt material in polyethylene bags and place in a drum for transit to an approved disposal site. Rinse away the remaining spilt material with water to reduce odour, and discharge the rinsate into a municipal or industrial sewer.

Large spills: In the case of nasal and respiratory irritation, vacate the room immediately. Personnel cleaning up should be trained and equipped with a self-contained breathing apparatus, or an officially approved or certified full-face respirator equipped with an organic vapour cartridge, gloves, and clothing impervious to glutaraldehyde, including rubber boots or shoe protection. Deactivate with sodium bisulphite (2-3 parts [by weight] per part of active substance glutaraldehyde), collect the neutralised liquid and place in a drum for transit to an approved disposal site.

D. Storage and Handling

Information on storage and handling was obtained from the ECHA REACH database (ECHA).

General Handling

Do not get in eyes, on skin, on clothing. Avoid breathing vapour. Do not swallow. Keep container closed. Use with adequate ventilation. Wear goggles, protective clothing and butyl or nitrile gloves. Wash thoroughly with soap and water after handling. Remove contaminated clothing and wash before reuse.

Other Handling Precautions

Do not spray or aerosolise the undiluted form of the product. Full personal protective equipment (including skin covering and full-face SCBA respirator) is required for dilutions or mixtures of the product used in a spray application.

Storage

Do not store in: Aluminium. Carbon steel. Copper. Mild steel. Iron. Shelf life: Use within 12 Months.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace exposure standard for glutaraldehyde in Australia is 0.1 ppm (0.41 mg/m^3) as a peak limitation, with a sensitisation notation. A peak limitation is defined by Safe Work Australia as a maximum or peak airborne concentration of a substance determined over the shortest analytically practicable period of time which does not exceed 15 minutes.

The information below on exposure controls and personal protection was obtained from the Halliburton Safety Data Sheet (SDS) on ALDACIDE® G ANTIMICROBIAL (revision date: 11-Dec-2014).

Engineering Controls

Use in a well-ventilated area. Local exhaust ventilation should be used in areas without good cross ventilation. If vapours are strong enough to be irritating to the nose or eyes, the TLV is probably being exceeded, and special ventilation or respiratory protection may be required.

Personal Protection Equipment

Respiratory Protection: If engineering controls and work practices cannot keep exposure below occupational exposure limits or if exposure is unknown, wear a NIOSH-certified, European Standard EN 149, AS/NZS 1715:2009, or equivalent respirator when using this product. Selection of and instruction on using all personal protective equipment, including respirators, should be performed by an Industrial Hygienist or other qualified professional. Full Facepiece Respirator with Organic vapour cartridge with particulate pre-filter.

Hand Protection: Chemical-resistant protective gloves (EN 374). Suitable materials for longer, direct contact (recommended: protection index 6, corresponding to > 480-minute permeation time as per EN 374): Butyl rubber gloves. (>= 0.7 mm thickness). This information is based on literature references and on information provided by glove manufacturers or is derived by analogy with similar substances. Please note that in practice the working life of chemical-resistant protective gloves may be considerably shorter than the permeation time determined in accordance with EN 374 as a result of the many influencing factors (e.g., temperature). If signs of wear and tear are noticed, then the gloves should be replaced. Manufacturer's directions for use should be observed because of the great diversity of types.

Skin Protection: Butyl coated apron or clothing.

Eye protection: Splash proof chemical mono-goggles or safety glasses with side shield in conjunction with a face shield. Do NOT wear contact lenses.

Other Precautions: Eyewash fountains and safety showers must be easily accessible.

F. Transport Information

For aqueous glutaraldehyde solutions at a concentration that is corrosive (i.e., 30% and higher):

Australia Dangerous Goods

UN3265, Corrosive Liquid, Acidic, Organic, N.O.S. (Contains Glutaraldehyde)

Class 8

Packing Group III

Environmentally Hazardous Substance

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

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GLYCERINE
[GLYCEROL]

This dossier on glycerine presents the most critical studies pertinent to the risk assessment of glycerine in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Glycerol

CAS RN:56-81-5

Molecular formula: $C_3H_8O_3$

Molecular weight: 92.09 g/mol

Synonyms: glycerin; alkyl alcohol; 2-propanol; 1,3-dihydroxy-; propanetriol; 1,2,3-propanetriol

SMILES: OCC(O)CO

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-Chemical Properties of Glycerine

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Clear, water-white, viscous, sweet-tasting hygroscopic liquid	2	ECHA
Melting Point	18.17°C @ 101.3 kPa	2	ECHA
Boiling Point	290°C @ 101.3 kPa	2	ECHA
Density	1,261 kg/m^3@ 20°C	2	ECHA
Vapour Pressure	0.01 Pa @ 20°C	2	ECHA
Partition Coefficient (log K_{ow})	-1.75 @ 25°C (measured)	2	ECHA
Water Solubility	1,000 g/L @ 25°C (completely miscible)	2	ECHA
Flash Point	199 °C	2	ECHA
Auto flammability	370°C	2	ECHA
Viscosity	1,412 mPa s @ 20°C	2	ECHA
Henry's Law Constant	Not Applicable	-	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Glycerine is readily biodegradable. It is not expected to bioaccumulate. Based on the estimated K_{oc} value, glycerine is expected to be highly mobile in sediment and soil.

B. Biodegradation

Glycerine was readily biodegradable in an OECD 301D test. Degradation was 57% after 5 days, 84% after 15 days, and 92% after 30 days (OECD, 2002) [Kl. score = 2].

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

No experimental data are available for glycerine. Using KOCWIN in EPISuite™ (US EPA, 2017), the estimated K_{oc} value from log K_{ow} is 0.1345 L/kg. The estimated K_{oc} value from the molecular connectivity index (MCI) is 1 L/kg.

Based upon these K_{oc} values, if released to soil, glycerine is expected to have low potential for adsorption and a high potential for mobility. If released to water, based on its K_{oc} and high-water solubility, glycerine is likely to remain in water and not adsorb to sediment.

D. Bioaccumulation

No bioconcentration studies have been conducted on glycerine. Glycerine is not expected to bioaccumulate based on the experimental log K_{ow} of -1.75 (ECHA).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Glycerine has virtually no acute toxicity by the oral and dermal routes. It is non-irritating to the skin and eye and is not a skin sensitizer. No systemic toxicity was seen in animals repeatedly exposed by the dermal and inhalation routes, but liver effects were seen in rats given very high doses in the diet. Glycerine is not genotoxic. Lifetime dietary studies showed no carcinogenic effects in rats. No reproductive or developmental effects were seen in animals given high doses of glycerine in the diet.

B. Metabolism

Glycerine is an intermediate in carbohydrate and lipid metabolism in living organisms.

C. Acute Toxicity

The oral LD_{50} values are >5,000 to 58,400 mg/kg in rats, 4,250 to 38,000 mg/kg in mice, 7,750 and 10,000 mg/kg in guinea pigs (OECD, 2002). The oral LD_{50} value of 4,250 mg/kg in mice is not consistent with the range of values found in the available literature and is considered unreliable because of the lack of documentation of the study (OECD, 2002).

All rats died following a 2-hour exposure to saturated vapours of glycerine, while there was no mortality when the exposure was for only one hour (ECHA) [Kl. score = 2].

No deaths were seen in rabbits following dermal application for 8 hours under occlusive conditions. The dermal LD_{50} is >18,700 mg/kg (Hine et al., 1953).

D. Irritation

Application of 0.5 mL glycerine to the skin of rabbits for 24 hours under occlusive conditions was not irritating (Weil and Scala, 1971; ECHA) [Kl. score = 2].

Instillation of 0.1 ml glycerine into the eyes of rabbits was non-irritating (Weil and Scala, 1971; ECHA).

E. Sensitisation

Male guinea pigs were given ten 0.1 mL injections of a 0.1% solution of synthetic or natural glycerine in isotonic saline every other day over 20 days. Following a two-week period, an 0.05 mL injection was given of the 0.1% glycerine solution. There was no sensitising response (Hine et al., 1953).

F. Repeated Dose Toxicity

Oral

Male and female rats were given in their feed 0, 5, or 20% glycerine for 90 days. Glycerine samples from different companies were compared in separate groups of animals. Body weight gain was higher in the treated rats compared to the controls. The 20% males had increased liver weights relative to body weights with histopathologic changes of generalized cloudy swelling and hypertrophy of the parenchymal cells. The 20% females showed increased relative liver weights but had generalized cloudy swelling in the liver. For the liver changes, there were no differences between the three glycerine samples. Relative heart weights were significant reduced in the 20% females from one glycerine sample, and relative kidney weights were increased in the 20% females from another glycerine sample; these changes were not accompanied by histopathological changes. The NOAEL for this study is 5% glycerine in the diet, which corresponds to an estimated daily intake of 4,580 and 6,450 mg/kg-day for males and females, respectively (ECHA) [Kl. score = 2]

Inhalation

Male and female SD rats were exposed by inhalation (nose-only) to 0, 33, 165, or 660 mg/m^3 of aerosolized glycerine 6 hours/day, 5 days/week for 13 weeks. The mass median aerodynamic diameter (MMAD) was <2.0 µm (respirable). The only effect seen was localized irritation of the upper respiratory tract. The NOAEC for systemic toxicity is 660 mg/m3, the highest exposure concentration tested. The NOAEC for localized effects (irritation) is 167 mg/m^3 (Renne, 1992; ECHA) [Kl. score = 2]

Dermal

Rabbits were given dermal applications of 0.5 to 5.4 ml/kg glycerine 8 hours/day for 45 weeks. No effects including irritation were noted. The NOAEL is 5.4 ml/kg, which is calculated to be 5,040 mg/kg-day (ECHA)[Kl. score = 2]

G. Genotoxicity

In vitro Studies

The *in vitro* genotoxicity studies on glycerine are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Glycerine

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (S. typhimurium strains)	-	-	2	Haworth et al., 1983; ECHA
Bacterial reverse mutation (S. typhimurium strains)	-	-	2	Doolittle et al., 1988; ECHA
Mammalian cell gene mutation (CHO cells)	-	-	2	Doolittle et al., 1988; ECHA
Sister chromatid exchange (human lymphocytes)	-	-	2	Doolittle et al., 1988; ECHA
Unscheduled DNA synthesis (rat hepatocytes)	-	-	2	Doolittle et al., 1988; ECHA
Chromosomal aberrations (CHO cells)	-	-	2	Doolittle et al., 1988; ECHA

*+, positive; -, negative

In vivo Studies

There are no studies available.

H. Carcinogenicity

Oral

Male and female Long-Evans rats were given in their feed 0, 5, 10, or 20% glycerine for two years (the 20% group were for 1 year only). The estimated daily intakes are 0, 2,000, 4,000, and 8,000 mg/kg-day for males: and 0, 2,500, 5,000, and 10,000 mg/kg-day for females. Treatment was discontinued after one year for the 20% animals for reasons that were not stated in the report. Data on mortality and clinical observations were not reported. The tumour incidences were similar between treated and control animals (Hine et al., 1953; ECHA) [Kl. score = 2].

Inhalation

There are no studies available.

Dermal

There are no studies available.

I. Reproductive Toxicity

In a two-generation reproductive toxicity study, male and female rats were dosed by oral gavage with 0 or 20% glycerine solution (in water). There were no treatment-related effects on growth, reproductive performance, fertility, and no histopathological changes in the tissues examined. The NOAEL for this study is 20% glycerine in water, which the daily intake was estimated to be 2,000 mg/kg-day (OECD, 2002; ECHA) [Kl. score = 2].

J. Developmental Toxicity

Oral

Pregnant female Wistar rats were dosed by oral gavage with 0, 13.1, 60.8, 282, or 1,310 mg/kg-day glycerine during gestational days 6 to 15. There was no maternal or developmental toxicity. The NOAEL for maternal and developmental toxicity is 1,310 mg/kg-day, the highest dose tested (ECHA). [Kl. score = 2]

Pregnant female CD-1 mice were dosed by oral gavage with 0, 12.8, 59.4, 276, or 1,280 mg/kg-day glycerine during gestational days 6 to 15. There was no maternal or developmental toxicity. The NOAEL for maternal and developmental toxicity is 1,280 mg/kg-day, the highest dose tested (ECHA) [Kl. score = 2]

Pregnant female Dutch rabbits were dosed by oral gavage with 0, 11.8, 54.8, 254.5, or 1,180 mg/kg-day glycerine during gestational days 6 to 18. There was no maternal or developmental toxicity. The NOAEL for maternal and developmental toxicity is 1,280 mg/kg-day, the highest dose tested (ECHA)[Kl. score = 2]

Inhalation

There are no studies available.

Dermal

There are no studies available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for Glycerine follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

Liver effects were seen in male and female rats in a 90-day dietary study, with a NOAEL of 5% glycerine in the diet. This dose corresponds to an estimate daily intake of 4,580 and 6,450 mg/kg-day for males and females, respectively (ECHA). In a two-year dietary study, no effects were seen in male or female rats at a dose of 20% glycerine in the diet. It should be noted, however, that the treatment at the dietary level of 20% was for only one year, while the lower doses (5 and 10%) were for two years. No liver effects were noted at any dose level. The NOAEL for the two-year dietary study is the

20% dietary level which corresponds to estimated daily intakes of 8,000 and 10,000 mg/kg-day, for males and females, respectively (Hines et al., 1953; ECHA).

The NOAEL of 4,580 mg/kg-day from the male rats in the 90-day dietary study will be used for determining the oral Reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 10
UF_D (database uncertainty) = 1
Oral RfD = 4,580/ (10 x 10 x 1 x 10 x 1) = 4,580/1,000 = 4.6 mg/kg-day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (4.6 x 70 x 0.1)/2 =16.1 mg/L

B. Cancer

Glycerine was not carcinogenic to rats in a two-year dietary study. Therefore, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Glycerine does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Glycerine is of low toxicity concern to aquatic and terrestrial organisms. Glycerine as fatty acid glyceride and as metabolite of fatty acid glycerides is part of (almost) all organisms (ECHA).

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on glycerine.

Table 3: Acute Aquatic Toxicity Studies on Glycerine

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Oncorhynchus mykiss	96-h LC_{50}	54,000	2	ECHA
Pimephales promelas	96-h LC_{50}	885	2	ECHA
Carassius auratus	96-h LC_{50}	>5000	2	ECHA
Daphnia magna	24-h EC_{50}	>10,000	1	ECHA
Daphnia magna	48-h LC_{50}	1,955	2	ECHA

Chronic Studies

Glycerine is a naturally occurring substance and part of fish organisms. Chronic studies conducted on fish have determined NOEC values greater than 100 mg/L (ECHA).

Glycerine is used as part of commercial fish feed. And as such shows no hazard towards fish in tested fish feed concentrations up to 7.5% for 12 months. (ECHA)[KI. score =2]

Using USEPA's EPISUITE, the QSAR estimation (ECOSAR v1.11 KOWWIN version 1.67) of chronic fish toxicity resulted in a 30 day chronic value of 9471 mg/L. This result is far above the limit dose of chronic fish testing (100 mg/L). According to this QSAR estimation no chronic hazard for fish can be identified. (ECHA)[KI. score =2].

The chronic toxicity of glycerine to fish was estimated to be 724,000 mg/L based on results from a trend analysis in the OECD QSAR toolbox (version 4.4.1) (ECHA)[KI. score =2].

The chronic toxicity (NOEC) of glycerine to Daphnia magna was estimated to be 897 mg/L based on results from a trend analysis in the OECD QSAR toolbox (version 4.4.1) (ECHA)[KI. score =2].

Using USEPA's EPISUITE, the QSAR estimation (ECOSAR v1.11 KOWWIN version 1.67) of chronic toxicity to Daphnia magna resulted in a 16 day chronic value of 2,230 mg/L (ECHA)[KI. score =2].

C. Terrestrial Toxicity

There are no studies available. Glycerine is considered a primordial biomolecule common to all species (Lehninger, 1970).

D. Calculation of PNEC

The PNEC calculations for glycerine follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for two trophic levels. Acute E(L)C_{50} values are available for fish (885 mg/L) and Daphnia (1,955 mg/L). NOEC values from long term studies are also available for fish (9,471 mg/L) and Daphnia (897 mg/L). On the basis that the data consists of short-term and long-term results from two trophic levels, an assessment factor of 50 has been applied to the lowest reported NOEC value of 897 mg/L for Daphnia. The $PNEC_{water}$ is 18 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 11.5 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (0.82/1280) \times 1000 \times 18$
$= 11.5$ mg/kg

Where:

$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1000 \times BD_{solid}]$
$= 0.8 + [(0.2 \times 0.04/1000 \times 2400]$
$= 0.82\ m^3/m^3$

Where:

Kp_{sed} = solid-water partition coefficient (L/kg)
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]
$Kp_{sed} = K_{oc} \times f_{oc}$
$= 1 \times 0.04$
$= 0.04$ L/kg

Where:

K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for glycerine was calculated from EPISUITE™ using the MCI is 1 L/kg.
F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.24 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
$= (0.02/1500) \times 1000 \times 18$

= 0.24 mg/kg

Where:

Kp_{soil} = soil-water partition coefficient (m^3/m^3)

BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]

$Kp_{soil} = K_{oc} \times f_{oc}$

= 1 x 0.02

= 0.02 m^3/m^3

Where:

K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for glycerine calculated from EPISUITE™ using the MCI is 1 L/kg.

F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Glycerine is readily biodegradable and thus does not meet the screening criteria for persistence.

No bioconcentration studies are available for glycerine. Based on the measured log K_{ow} for glycerine of -1.75,glycerine does not meet the screening criteria for bioaccumulation.

Glycerine as fatty acid glyceride and as metabolite of fatty acid glycerides is part of (almost) all organisms (ECHA). The chronic NOEC values for glycerine in fish and invertebrates are > 0.1 mg/L. The acute E(L)C50 values for glycerine in fish and invertebrates are >1 mg/L. Thus, glycerine does not meet the screening criteria for toxicity.

Therefore, glycerine is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Not Classified

B. Labelling

No signal word

A. Pictogram

None.

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention if respiratory irritation develops or if breathing becomes difficult.

Ingestion

Rinse mouth with water and then drink a plenty of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice. Ensure adequate ventilation. Do not breathe vapours, mists, or gas.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Other Handling Precautions

Avoid inhalation of vapor or mist.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace exposure standards for glycerine (mist) in Australia is as follows 10 mg/m^3 (Time-weighted average, TWA).

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Glycerine is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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GUAR GUM

This dossier on guar gum (CAS RN 9000-30-0) presents the most critical studies pertinent to the risk assessment of the substance in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The information presented in this dossier was obtained from the chemistry database PubChem. Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): disodium;[[[5-(6-aminopurin-9-yl)-3-hydroxyoxolan-2-yl]oxy-methoxyphosphoryl]oxy-oxidophosphoryl] hydrogen phosphate

CAS RN: 9000-30-0

Molecular weight: 535.15 g/mol; 200,000 to 300,000 daltons (Glickman, 1969)

Molecular formula: $C_{10}H_{14}N_5Na_2O_{12}P_3$

Synonyms: GU-052, guar flour, guaran, gum guar, slocose

SMILES:: COP(=O)(OC1C(CC(O1)N2C=NC3=C(N=CN=C32)N)O)OP(=O)([O-])OP(=O)(O)[O-].[Na+].[Na+]

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-Chemical Properties of Guar Gum

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Off-white to yellowish-white powder	-	PubChem
Vapour Pressure	Negligible	-	PubChem
Water Solubility	< 1 g/L @ 20°C (insoluble)	-	PubChem

III. ENVIRONMENTAL FATE PROPERTIES

Guar gum is a carbohydrate polymer consisting of D-mannose and D-galactose sugars from the guar plant or cluster bean. As a high molecular weight polysaccharide polymer, guar gum is expected to have a negligible vapour pressure. If released to air, a negligible vapour pressure indicates guar gum will exist solely in the particulate phase in the atmosphere. Particulate-phase guar gum will be removed from the atmosphere by wet and dry deposition. If released to soil, guar gum is expected to have no mobility since it is a polymer that binds strongly with soil particles. Volatilisation from moist soil surfaces is not expected to be an important fate process based upon a negligible Henry's Law constant. Likewise, guar gum is not expected to volatilise from dry soil surfaces based upon its vapour pressure. If released into water, guar gum is expected to adsorb to suspended solids and sediment (PubChem). Half-life data was not available.

Guar gum is expected to readily undergo microbial biodegradation in the environment (on the basis that it is a polysaccharide and expected to be readily biodegradable), and the potential to bioaccumulate in organisms is considered to be low (DoEE, 2017 and USEPA, 2005).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Guar gum exhibits very low acute toxicity by the oral route. It is non-irritating to the skin and minimally irritating to the eyes. Repeated dose toxicity studies in rats showed minimal toxicity from exposure to guar gum in the diet. Guar gum is not genotoxic or carcinogenic. Oral exposure to guar gum did not affect fertility in rats; nor was there any indication of developmental toxicity in either rats or mice.

NICNAS has assessed Guar Gum in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to human health[1]

B. Acute Toxicity

The oral LD_{50} in rats was reported to be 7,060 mg/kg (Graham *et al.*, 1981) [Kl. Score = 2].

C. Irritation

Guar gum is non-irritating to the skin and minimally irritating to the eyes (McCarty *et al.*, 1990). Nonetheless, ECHA warns that the substance may cause serious eye irritation.

D. Sensitisation

There were reports of workers sensitised to guar gum in a carpet-manufacturing plant. Immediate skin reactivity to guar gum was observed in 8 out of 162 employees, and 11 of 133 participants had serum IgE antibodies to guar gum. These findings are difficult to interpret since carbohydrates, such as guar gum, are generally not associated with allergenicity (Malo, 1990).

E. Repeated Dose Toxicity

Oral

Male and female Osborne-Mendel rats were given diets containing 0, 1, 2, 4, 7.5, or 15% guar gum for 91 days. The average daily intakes are: 0; 580; 1,187; 2,375; 4,561 and 10,301 mg/kg/day for males; and 0; 691; 1,362; 2,762; 5,770 and 13,433 mg/kg/day for females. There were no deaths during the study. Body weights were significantly decreased in the ≥1% females and the ≥7.5% males; biologically significant changes (>10%) were seen in the 7.5% females and the 15% males. Liver weights were decreased in the ≥1% dietary groups. Kidney weights were decreased in the ≥7.5% dietary groups and were borderline significant in the 4% group. The 15% group males had reduced bone marrow cellularity; although the level was within normal limits, several of the rats were at the lower end of the normal range. The NOAEL for this study is 4% in the diet or 2,762 mg/kg/day based on reduced body weights in the female rats (Graham *et al.*, 1981) [Kl. Score = 2].

Male and female F344 rats and $B6C3F_1$ mice were given diets containing 0; 6,300; 12,500; 25,000; 50,000 or 100,000 ppm guar gum for 13 weeks. Mean body weights were decreased in the 100,000 ppm male rats and in the ≥50,000 ppm female mice. A dose-related decrease in feed consumption was observed for male and female rats; male and female mice were comparable or higher than that of controls. There were no compound-related clinical signs or histopathological effects. The NOAELs

[1] https://www.industrialchemicals.gov.au/chemical-information/search-assessments?assessmentcasnumber=9000-30-0%2C+

for this study are 50,000 and 25,000 ppm for rats and mice, respectively. Using the fraction of body weight that rats and mice consume per day as food (0.05 and 0.13, respectively; USEPA), the NOAELs corresponds to 2,500 mg/kg/day for rats and 3,250 mg/kg/day for mice (NTP, 1982) [Kl. Score = 2].

Male and female F344 rats and B6C3F$_1$ mice were given diets containing 0 ppm, 25,000 ppm or 50,000 ppm guar gum for 103 weeks. Mean body weights of the high-dose females were lower than those of the controls after week 20 for mice and week 40 for rats. No compound-related clinical signs or adverse effects on survival were observed. Feed consumption by dosed rats and mice of either sex was lower than that of controls. There were no non-neoplastic histopathological effects in either rats or mice that were treatment-related. The NOAEL for both rats and mice is 25,000 ppm. Using the fraction of body weight that rats and mice consume per day as food (0.05 and 0.13, respectively; USEPA), the NOAELs correspond to 1,250 mg/kg/day for rats and 3,250 mg/kg/day for mice (NTP, 1982) [Kl. Score = 2].

Inhalation

No studies are available.

Dermal

No studies are available.

F. Genotoxicity

In vitro Studies

Guar gum was not mutagenic to *S. typhimurium* strains TA 97, TA 98, TA 100, TA 102, TA 104, TA 1535, TA 1537, and TA1538 in the presence or absence of metabolic activation (Zeiger *et al.*, 1992) [Kl. Score = 2].

In vivo Studies

Guar gum was inactive in a rat bone marrow cytogenetic assay at doses up to 5,000 mg/kg (Johnson *et al.*, 2015) Kl. Score = 4].

In a rat dominant lethal mutation test, rats were dosed by oral gavage with either a single or multiple doses of up to 5,000 mg/kg guar gum. There was no indication of a mutagenic effect by guar gum (Lee *et al.*, 1983) [Kl. Score = 2].

G. Carcinogenicity

Male and female F344 rats were given diets containing 0 ppm, 25,000 ppm or 50,000 ppm guar gum for 103 weeks in an NTP chronic bioassay. There were increased incidences of adenomas of the pituitary in male rats and pheochromocytomas of the adrenal medulla in female rats that were statistically significant, but these differences were considered to be unrelated to guar gum administration. When pituitary adenomas or carcinomas and when pheochromocytomas or malignant pheochromocytomas were combined, the statistical differences disappeared. NTP concluded that, under conditions of this bioassay, guar gum was not carcinogenic for F344 rats (NTP, 1982) [Kl. Score = 2].

Male and female B6C3F$_1$ mice were given diets containing 0 ppm, 25,000 ppm or 50,000 ppm guar gum for 103 weeks in an NTP chronic bioassay. Hepatocellular carcinomas occurred in treated male

mice at incidences that were significantly lower than that in controls. The combined incidence of male mice with either hepatocellular adenomas or carcinomas was also significantly lower in the high-dose group. NTP concluded that, under conditions of this bioassay, guar gum was not carcinogenic for B6C3F_1 mice (NTP, 1982) [Kl. Score = 2].

H. Reproductive Toxicity

Oral

Male and female Osborne-Mendel rats were fed diets containing 0, 1, 3, 4, 7.5, or 15% guar gum for 13 weeks before mating, during mating and throughout gestation. The daily intakes for the female rats during gestation were 0; 700; 1,400; 2,700; 5,200 or 11,800 mg/kg/day. Fertility was unaffected by treatment. There were slightly fewer corpora lutea and implantations in the 15% dietary group, but implantation efficiency was unaffected. The NOAEL for reproductive toxicity is 5,200 mg/kg/day (Collins *et al.*, 1987) [Kl. Score = 2].

I. Developmental Toxicity

Oral

Male and female Osborne-Mendel rats were fed diets containing 0, 1, 3, 4, 7.5, or 15% guar gum for 13 weeks before mating, during mating and throughout gestation. The daily intake for the female rats during gestation were 0; 700; 1,400; 2,700; 5,200 or 11,800 mg/kg/day. There were no deaths during the study. In the 15% group, the number of viable foetuses per litter were slightly reduced but was not statistically significantly different from controls. The authors indicated that the reduction may have been an effect of the decreased number of corpora lutea because the number of resorptions was unaffected in this treatment group. There was no treatment-related effect on foetal development or sex distribution, and there were no teratogenic effects (Collins *et al.*, 1987) [Kl. Score = 2].

Pregnant female rats were dosed by oral gavage with 0, 9, 42, 200 or 900 mg/kg guar gum on GD 6 to 15. There was no maternal or developmental toxicity at any dose level. The NOAEL for maternal and developmental toxicity is 900 mg/kg/day (FDRL, 1973) [Kl. Score = 2].

Pregnant female CD-1 mice were dosed by oral gavage with 0, 8, 37, 170, or 800 mg/kg guar gum on GD 6 to 15. A significant number of deaths (6 out of 29) occurred in the 800 mg/kg dose group. There were indications of maternal toxicity in the surviving high-dose dams. There was no developmental toxicity at any dose level. The NOAELs for maternal and developmental toxicity are 170 and 800 mg/kg/day, respectively (FDRL, 1973) [Kl. Score = 2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for guar gum follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2021).

A. Non-Cancer

Oral

In a two-year NTP chronic bioassay, female rats and mice given 50,000 ppm guar gum in their feed had lower body weights. There were no treatment-related non-neoplastic lesions in either rats or

mice. The NOAEL for this study is 25,000 ppm for rats and mice, which corresponds to 1,250 mg/kg/day for rats and 3,250 mg/kg/day for mice.

The NOAEL of 1,250 mg/kg/day will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1
Oral RfD = 1,250/(10 x 10 x 1 x 1 x 1) = 1,250/100 = <u>13 mg/kg/day</u>

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2021)
Proportion of water consumed = 10% (ADWG, 2021)
Volume of water consumed = 2L (ADWG, 2021)
Drinking water guidance value = (13 x 70 x 0.1)/2 = <u>46 mg/L</u>

B. Cancer

Guar gum was not carcinogenic to rats or mice in two-year dietary studies. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Guar gum does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Guar gum is a polysaccharide polymer. It has low acute toxicity concern for fish but exhibits moderate acute toxicity to invertebrates (*Daphnia*).

B. Aquatic Toxicity

Acute Studies

The 96-hour LC_{50} for *Oncorhynchus mykiss* is 218 mg/L (Biesinger *et al.*, 1976) [Kl. Score = 2].

The 48-hour and 96-hour LC_{50} values for *Daphnia magna* are 42 mg/L and <6.2 mg/L, respectively (Biesinger *et al.*, 1976) [Kl. Score = 2].

Chronic Studies

No studies are available.

C. Terrestrial Toxicity

No studies are available.

D. Calculation of PNEC

The PNEC calculations for guar gum follow the methodology discussed in DEWHA (2009).

PNEC water

Experimental results are available for two trophic levels. The acute LC_{50} values are available for fish (218 mg/L) and *Daphnia* (<6.2 mg/L). No chronic studies are available. On the basis that the data consists of acute studies from two trophic levels, an assessment factor of 1,000 has been applied to the lowest reported LC_{50} value of 6.2 mg/L for *Daphnia*. The $PNEC_{water}$ is 0.006 mg/L.

PNEC sediment

No experimental toxicity data on sediment organisms are available. The K_{ow} and K_{oc} of guar gum cannot be calculated using EPI Suite because the molecular weight of guar gum greatly exceeds the limit of 1,000. Thus, the equilibrium partition method cannot be used to determine a $PNEC_{sediment}$ and the assessment of this compartment will be covered by the aquatic assessment.

PNEC soil

No experimental toxicity data on soil organisms are available. The K_{ow} and K_{oc} of guar gum cannot be calculated using EPI Suite because the molecular weight of guar gum greatly exceeds the limit of 1,000. Thus, the equilibrium partition method cannot be used to determine a $PNEC_{soil}$ and the assessment of this compartment will be covered by the aquatic assessment.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU Reach Criteria methodology (DEWHA, 2009; ECHA, 2008).

Guar gum is a naturally occurring polysaccharide from the guar plant or cluster bean; it expected to be readily biodegradable. Thus it is not expected to meet the screening criteria for persistence.

The potential to bioaccumulate in organisms is considered to be low. Thus guar gum is not expected to meet the criteria for bioaccumulation.

There are no adequate chronic aquatic toxicity studies available on guar gum. The acute LC_{50} values for guar gum are >1 mg/L in fish and invertebrates. Therefore, guar gum does not meet the screening criteria for toxicity.

The overall conclusion is that guar gum is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Acute Aquatic Toxicity Category 2

B. Labelling

Warning!

According to the classification provided by companies to ECHA in CLP notifications, this substance causes serious eye irritation.

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Remove contaminated clothing. Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention if respiratory irritation develops or if breathing becomes difficult.

Ingestion

Rinse mouth with water and then drink plenty of water. Never give anything by mouth to an unconscious person.

Notes to Physician

May cause asthma-like (reactive airways) symptoms.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus for fire fighting.

C. Accidental Release Measures

Personal Precautions

Avoid dust formation.

Environmental Precautions

No special environmental precautions required.

Steps to be Taken if Material is Released or Spilled

Sweep up and dispose in suitable, closed containers.

D. Storage And Handling

General Handling

Avoid creating or inhaling dust.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard specifically for guar gum.

Engineering Controls

Ensure adequate ventilation.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Handle with gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Eyewash fountains and safety showers must be easily accessible.

F. Transport Information

Guar gum is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

ADWG (2021). National Water Quality Management Strategy. Australian Drinking Water Guidelines, Section 6, Australian Government, National Health and Medical Research Council, Natural Resource Management Ministerial Council.

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HYDROCHLORIC ACID

This dossier on hydrochloric acid presents the most critical studies pertinent to the risk assessment of hydrochloric acid in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from OECD-SIDS documents (OECD, 2002a,b) and the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed hydrochloric acid in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment.

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Chlorane

CAS RN: 7647-01-0

Molecular formula: HCl

Molecular weight: 36.46 g/mol

Synonyms: Hydrochloric acid; HCl; chlorane; hydrogen chloride; muriatic acid; chlorohydric acid

SMILES: Cl

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-chemical Properties of Hydrochloric Acid

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Colourless to slightly yellow gas of fuming liquid with pungent, irritating odour.	2	ECHA
Melting Point	-114.22°C	2	ECHA
Boiling Point	-85°C	4	ECHA
Density	1.639 kg/m^3 @ 0°C (gas) 1190 kg/m^3 @ 15°C (liquid)	4	ECHA
Vapour Pressure	4,104 kPa 4,723 kPa @ 25°C	4	ECHA
Partition Coefficient (log K_{ow})	Not applicable	-	-
Water Solubility	Very soluble	4	ECHA
Viscosity	1.7 x 10^{-6} m^2s @ 20°C	1	ECHA

Hydrochloric acid can exist in a gaseous phase at room temperature and pressure. Hydrochloric acid is also very soluble in water and is a strong acid that dissociates completely in water to hydrogen (H^+) and chloride (Cl^-) ions.

III. ENVIRONMENTAL FATE PROPERTIES

Due to its high water solubility, hydrochloric acid will be found predominantly in the aquatic environment where it dissociates completely to hydrogen (H^+) and chloride (Cl^-) ions. Both ions are ubiquitous in the environment (UNEP, 1995).

The addition of hydrochloric acid to an aquatic ecosystem may decrease the pH depending on the buffer capacity of the receiving water. In general, the buffer capacity is regulated by the equilibria between CO_2, HCO_3^- and CO_3^{2-}:

$CO_2 + H_2O \leftrightarrow HCO_3^- + H^+$ ($pKa_1 = 6.35$)

$HCO_3^- \leftrightarrow CO_3^{2-} + H^+$ ($pKa_2 = 10.33$)

A release of hydrochloric acid into the aquatic environment from the use of HCl could potentially increase the chloride concentration and decrease the pH in the aquatic environment. Table 2 shows the amount of hydrochloric acid that would need to be added to bicarbonate solutions to obtain pH values of 6.0 and 4.0. The UNEP (1995) study reported that the 10th percentile, mean and the 90th percentile of bicarbonate concentrations in 77 rivers in North America, South America, Asia, Africa, Europe and Oceania were 20, 106, and 195 mg/L, respectively. The data show that the decrease in pH depends on the buffer capacity (bicarbonate concentration) of the receiving water. The calculated values in Table 2 were confirmed experimentally.

Table 2: Buffer Capacity to Maintain the pH Based on Bicarbonate Concentration from UNEP Monitoring Data (de Groot and van Dijk, 2002; taken from OECD, 2002b)

Initial concentration of HCO_3^-	Final pH	Concentration of HCl required to obtain the final pH value
		Calculated (mg/L)
20 mg/L HCO_3^- (10th percentile 77 rivers)	6.0	8.28
	4.0	11.9
106 mg/L HCO_3^- (mean value of 77 rivers)	6.0	43.9
	4.0	63.2
195 mg/L HCO_3^- (90th percentile 77 rivers)	6.0	80.7
	4.0	116.3

H^+ and Cl^- ions will not adsorb on the particulate matter or surfaces and will not accumulate in living tissues (OECD, 2002a,b).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Hydrochloric acid is a corrosive liquid. Depending on the concentration, aqueous solutions of hydrochloric acid (HCl) are either corrosive, irritating or non-irritating to the skin, eyes and gastrointestinal tract. Vapours from aqueous solutions of HCl can cause respiratory irritation. HCl is not a skin sensitiser. Subchronic inhalation studies show localised irritation to the upper respiratory tract of rats and mice, but no systemic toxicity. No repeated dose toxicity studies have been conducted by the oral route. Positive findings have been reported in some *in vitro* genotoxicity studies, which are considered to be the result of the pH change in the test system. A lifetime inhalation study showed no carcinogenicity in rats exposed to HCl. No adequate reproductive or developmental studies have been conducted on HCl.

B. Acute Toxicity

The oral LD_{50} values in rats were reported to be 238 to 277 mg/kg and 700 mg/kg (OECD, 2002a,b) [Kl. scores = 2 and 4, respectively].

The lethal dose by dermal exposure is > 5,010 mg/kg for rabbits (OECD 2002a,b) [Kl. score = 4].

The LC_{50} values in rats for HCl gas are 40,989 and 4,701 ppm for 5 and 30 minutes, respectively (ECHA) [Kl. score = 2]. The LC_{50} values in rats for HCl aerosol are 31,008 and 5,666 ppm (45.6 and 8.3 mg/L) for 5 and 30 minutes, respectively (ECHA) [Kl. score = 2].

C. Irritation

Application of a 37% aqueous solution of HCl for 1 or 4 hours was corrosive to the skin of rabbits (OECD, 2002a,b) [Kl. score = 2]. Application of 0.5 mL of a 17% solution of aqueous solution of HCl for 4 hours was corrosive to the skin of rabbits (OECD, 2002a,b) [Kl. score = 3]. Moderate skin irritation was observed in rabbits following an application of 0.5 mL of a 3.3% aqueous solution of HCl for five days; no irritation was observed with 0.5 mL of a 1% aqueous solution (OECD, 2002a,b) [Kl. score = 2]. In humans, an aqueous solution of 4% of HCl was slightly irritating, while a 10% solution was sufficiently irritating to be classified as a skin irritant (OECD, 2002a,b).

Instillation of 0.1 mL of a 10% aqueous solution of HCl to the eyes of rabbits resulted in severe eye irritation (ECHA) [Kl. score = 2]. Instillation of 0.1 mL of a 5% solution of HCl produced corneal opacity, iridial lesions, conjunctival redness and chemosis in 3/3 animals at 1 hour and at day one post-instillation. There was no recovery in any animal and the study was terminated on day two (ECHA) [Kl. score = 1].

D. Sensitisation

Hydrochloric acid was not a skin sensitiser in a guinea pig maximisation test (ECHA) [Kl. score = 2].

E. Repeated Dose Toxicity

Oral

No adequate studies were located.

Inhalation

Male and female SD rats and F344 rats were exposed by inhalation to 0, 10, 20, or 50 ppm 6 hours/day, 5 days/week for up to 90 days. Clinical signs were mainly indicative of the irritant/corrosive nature of HCl. Body weights were significantly decreased in the 50 ppm male F344 rats. There were no treatment-related effects on the haematology or clinical chemistry parameters or urinalysis. At study termination, heart, kidney and testes weights were increased in the 100 and/or 50 ppm groups; these changes were considered to be mainly related to the treatment-related effect on body weight. Histopathological examination showed minimal to mild rhinitis in the $\geq$ 20 ppm dose groups of both strains of rats (both sexes). The NOAELs for systemic toxicity and localised irritation (site-of-contact) are 20 and 10 ppm, respectively (ECHA) [Kl. score = 1].

Male and female B6C3F$_1$ mice were exposed by inhalation to 0, 10, 20 or 50 ppm HCl, 6 hours/day, 5 days/week for up to 90 days. Clinical signs were mainly indicative of the irritant/corrosive nature of HCl. Body weights were significantly decreased in the 50 ppm groups. At study termination, absolute liver weights were decreased in the 50 ppm males. Histopathologic examination showed only eosinophilic globules in the nasal epithelium in the 50 ppm animals. The NOAEL for this study is 20 ppm (ECHA) [Kl. score = 1].

Male SD rats were exposed by inhalation to 0 or 10 ppm HCl 6 hours/day, 5 days/week for 128 weeks. Survival and body weights were similar between treated and control groups. There was a higher incidence of hyperplasia of the larynx compared to control, but no serious irritating effects of the nasal epithelium (ECHA) [Kl. score = 2].

Dermal

No studies were located.

F. Genotoxicity

In vitro Studies

Table 3 presents the *in vitro* genotoxicity studies on hydrochloric acid.

Table 3: *In vitro* Genotoxicity Studies on Hydrochloric Acid

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (*S. typhimurium* and *E. coli* strains)	-	-	2	ECHA
Mammalian cell gene mutation (mouse lymphoma L5178Y cells)	-	+	2	ECHA
Chromosomal aberration (CHO cells)	+	+	2	ECHA
Saccharomyces cerevisiae (mitotic recombination	-	-	2	ECHA
E. coli W3110 (pol A+) and P3078 (pol A-) repair assay	-	-	2	ECHA

* +, positive; -, negative

In the mouse lymphoma assay, the mutant frequency increased as the pH was lowered to 6.5 to 6.0 (from increased HCl) in the presence of metabolic activation. A decrease in pH from the addition of HCl to the medium also resulted in clastogenic effects to CHO cells in the absence or presence of metabolic activation. The positive findings in these two studies are considered to be the result of the pH change in the test media.

In vivo Studies

No adequate studies were located.

G. Carcinogenicity

Oral

No studies were located.

Inhalation

Male SD rats were exposed by inhalation to 0 or 10 ppm HCl 6 hours/day, 5 days/week for 128 weeks. Survival and body weights were similar between treated and control groups. There was a higher incidence of hyperplasia of the larynx compared to control, but no serious irritating effects of the nasal epithelium. There was no increased incidence of tumours in the HCl-treated rats compared to controls (ECHA) [Kl. score = 2].

H. Reproductive Toxicity

No studies were located.

I. Developmental Toxicity

No adequate studies were located.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

Repeated dose, reproductive and developmental toxicity studies by the oral route have not been conducted on hydrochloric acid. These toxicity studies would have questionable usefulness because of the corrosive/irritating nature of hydrochloric acid, which would limit the amount of absorbed HCl. Hydrochloric acid dissociates to hydrogen and chloride ions in bodily fluids, and a significant amount of these ions are already ingested in foods. Furthermore, both ions are present in the body and are highly regulated by homeostatic mechanisms. Thus, an oral toxicological reference and drinking water guidance values were not derived from hydrochloric acid.

The Australian drinking water guideline values for pH (6.5 to 8.5) and chloride (250 ppm, aesthetics) may be applicable (ADWG, 2011).

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Hydrochloric acid does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

The hazard of hydrochloric acid for aquatic organisms is caused by the hydrogen ion (H^+). The toxicity values in terms of mg/L are not relevant because of the varying buffering capacity of different test systems and different aquatic ecosystems.

B. Aquatic Toxicity

Acute Studies

The acute aquatic toxicity studies on hydrochloric acid are listed in Table 4.

Table 4: Acute Aquatic Toxicity Studies on Hydrochloric Acid

Test Species	Endpoint	Results	Klimisch Score	Reference
Lepomis macrochirus	96-hour LC_{50}	pH 3.25 – 3.5 (20 mg/L)	2	ECHA; OECD 2002a,b
Daphnia magna	48-hour EC_{50}	pH 4.92 (0.45 mg/L)	1	ECHA
Chlorella vulgaris	72-hour EC_{50} 72-hour EC_{10}	pH 4.7 [growth rate] (0.73 mg/L) PH 4.7 (0.364 mg/L)	1	ECHA

Chronic Studies

No chronic studies are available.

C. Terrestrial Toxicity

No studies are available.

D. Calculation of PNEC

PNEC values were not derived from hydrochloric acid because factors such as the buffer capacity, the natural pH and the fluctuation of the pH are very specific for a certain ecosystem.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2008).

Hydrochloric acid is an inorganic salt that dissociates completely to hydrogen and chloride ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions; both hydrogen and chloride ions are also ubiquitous and are present in water, soil and sediment. For the purposes of this PBT assessment, the persistent criteria are not considered applicable to this inorganic salt.

Hydrogen and chloride ions are essential to all living organisms, and their intracellular and extracellular concentrations are actively regulated. Thus, hydrochloric acid is not expected to bioaccumulate.

No chronic toxicity data exist on hydrochloric acid. The acute EC_{50} values are > 1 mg/L in fish, < 1 mg/L for invertebrates and algae. Thus, hydrochloric acid meets the screening criteria for toxicity.

The overall conclusion is that hydrochloric acid is a PBT substance based on toxicity to invertebrates and algae.

IX. CLASSIFICATION AND LABELLING

A. Classification

For HCl concentrations of >25%:

- Metal Corrosive Category 1
- Skin Corrosive 1B
- STOT SE Category 3 [Respiratory irritant]

In addition to the hazard statements corresponding to the GHS classification for corrosive, the following non-GHS hazard statement is to be added to the SDS: AUH071: Corrosive to the Respiratory Tract.

B. Labelling

Danger

According to the classification provided by companies to ECHA in REACH registrations this substance causes severe skin burns and eye damage, is toxic if inhaled, may damage fertility or the unborn child, causes serious eye damage, may cause damage to organs through prolonged or repeated exposure, may be corrosive to metals and may cause respiratory irritation.

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. Get medical attention immediately, preferably a physician for an ophthalmologic examination.

Skin Contact

For minor skin contact, avoid spreading material on unaffected skin. Remove and isolate contaminated clothing. Wash the contaminated area of the body with soap and fresh water. Get medical attention immediately.

Inhalation

Move person to fresh air. Administer oxygen if breathing is difficult. Do not use mouth-to-mouth method if victim inhaled the substance; give artificial respiration with the aid of a pocket mask equipped with a one-way valve or another proper respiratory medical device. Give artificial respiration if the victim is not breathing. Get medical attention immediately.

Ingestion

Rinse mouth and lips with plenty of water if a person is conscious. Do not induce vomiting. Do not use mouth-to-mouth method if the victim ingested the substance. Obtain medical attention immediately if ingested.

Notes to Physician

Treat as corrosive due to pH of the material. All treatments should be based on observed signs and symptoms of distress in the patient.

B. Fire Fighting Information

Extinguishing Media

Use dry chemical, carbon dioxide, water spray or fog, or foam.

Specific Exposure Hazards

Containers may explode when heated. Emits toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following materials: halogenated compounds, may release dangerous gases (chlorine).

Special Protective Equipment for Firefighters

Structural firefighter's protective clothing provides limited protection in fire situations only; it is not effective in spill situations where direct contact with the substance is possible. Wear chemical protective clothing that is specifically recommended by the manufacturer. It may provide little or no thermal protection. Wear positive pressure self-contained breathing apparatus (SCBA). Move containers from the fire area if you can do it without risk.

C. Accidental Release Measures

Personal Precautions

Ventilate enclosed areas. Do not walk through spilt material. Do not touch damaged containers or spilt material unless wearing appropriate protective clothing. Wear appropriate personal protective equipment, avoid direct contact. Do not breath mist, vapours, or spray. Do not get in eyes, on skin, or on clothing.

Environmental Precautions

Prevent entry into waterways, sewers, basements or confined areas.

Steps to be Taken if Material is Released or Spilt

ELIMINATE all ignition sources (no smoking, flares, sparks or flames in immediate area). As an immediate precautionary measure, isolate spill or leak area for at least 50 meters in all directions. Keep unauthorised personnel away. Stay upwind. Keep out of low areas. Do not get water inside container.

D. Storage and Handling

General Handling

Handle and open container with care. Use only with adequate ventilation. Keep away from heat. Use caution when combining with water. DO NOT add water to corrosive liquid, ALWAYS add corrosive liquid to water while stirring to prevent the release of heat, steam and fumes. Wear appropriate personal protective equipment, avoid direct contact. Do not breath mist, vapours or spray. Do not get in eyes, on skin or on clothing. Do not ingest. Wash thoroughly with soap and water after handling and before eating, drinking or using tobacco.

Storage

Keep contain tightly closed. Store in a cool, dry, well-ventilated place. Keep away from incompatible materials. Keep from direct sunlight. Separate from alkalis. Do not store above 49^{o}C/120^{o}F.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace exposure standard for hydrochloric acid in Australia is 5 ppm (7.5 mg/m^3 as a peak limitation, with a sensitisation notation). A peak limitation is defined by Safe Work Australia as a maximum or peak airborne concentration of a substance determined over the shortest analytically practicable period of time which does not exceed 15 minutes.

Engineering Controls

Good general ventilation should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation or other engineering controls to maintain airborne levels below recommended exposure limits.

Personal Protection Equipment

Respiratory Protection: If workers are exposed to concentrations above the exposure limit, they must use appropriate, certified respirators. Use a properly fitted, air-purifying or air-fed respirator complying with an approved standard if a risk assessment indicates this is necessary. Respirator selection is based on known or anticipated exposure levels, the hazard of the product and the safe working limits of the selected respirator.

Hand Protection: Chemical-resistant, impervious gloves complying with an approved standard should be worn at all times when handling chemical products if a risk assessment indicates this is necessary. Considering the parameters specified by the glove manufacturer, check during use that the gloves are still retaining their protective properties. It should be noted that the time to breakthrough for

any glove material may be different for different glove manufacturers. In the case of mixtures, consisting of several substances, the protection time of the gloves cannot be accurately estimated.

Skin Protection: Personal protective equipment for the body should be selected based on the task being performed and the risks involved and should be approved by a specialist before handling hydrochloric acid.

Eye Protection: Wear chemical splash goggles and face shield.

Other Precautions: Wash hands, forearms and face thoroughly after handling chemical products; before eating, smoking and using the lavatory; and at the end of the working period. Appropriate techniques should be used to remove potentially contaminated clothing. Wash contaminated clothing before reusing. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Australian Dangerous Goods

UN 1789 (HYDROCHLORIC ACID)

Class: 8

Packing Group: II or III

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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HYDROTREATED LIGHT PETROLEUM DISTILLATE

This dossier on hydrotreated light petroleum distillate (CAS RN 64742-47-8) presents the most critical studies pertinent to the risk assessment of this substance in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): 1,4-bis(propan-2-yl)benzene; 7,7-dimethylhexadecane; octadecane

CAS RN: 64742-47-8

Molecular formula: Not available (UVCB substance)

Molecular weight: Not available (UVCB substance)

Synonyms: Distillates, petroleum, hydrotreated light

SMILES: CC(C)C1=CC=C(C=C1)C(C)C.CCCCCCCCCCCCCCCCCC.CCCCCCCCCC(C)(C)CCCCCC

II. PHYSICO-CHEMICAL PROPERTIES

Hydrotreated light petroleum distillate is a UVCB substance (unknown variable composition or biological substance) containing aliphatic (linear, branched, and/or cyclic paraffins) molecules of carbon and hydrogen. Physical and chemical properties were not available for the UVCB hydrocarbon. As a result, information was obtained from a read-across substance (hydrodesulfurised kerosine). Key physical and chemical properties for the substance are shown in Table 1.

Table 1: Overview of the Physico-Chemical Properties of Hydrodesulfurised Kerosine (CAS RN 64742-81-0)

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Liquid	2	ECHA
Melting Point	-49°C (pour point) @ 101.3 kPa.	2	ECHA
Boiling Point[1]	90 to 320°C @ 101.3 kPa	2	ECHA
Density	770 to 850 kg/m^3 @ 15°C	2	ECHA
Vapour Pressure	<1,000 to 37,000 Pa at 37.8°C	2	ECHA
Partition Coefficient (log K_{ow})	1.99 – 18.02 @ 20°C	2	ECHA
Water Solubility	0.000009 – 0.00645 g/L @ 25 °C	-	OECD
Viscosity	1.1 to 2.5 mm^2/s @ 20°C (kinematic)	2	ECHA
Auto flammability	220 - 250°C (for kerosines)	2	ECHA

[1] CAS numbers in this category indicate a boiling point range of 90-320 deg Celsius.

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Representative substances are expected to be readily biodegradable. They are highly insoluble in water and have high adsorption potential. They have a low potential to bioaccumulate.

While sediment and soil are expected to be the main targets for environmental distribution, biodegradation potential is expected to offset sorption. In fact, fugacity modelling suggests that accumulation in sediment is expected to be several orders of magnitude less than 1%, relative to soil, water and air compartments.

B. Partitioning

Based on Henry's Law Constant values > 4.76 x 10^4 Pa-m^3/mol @25 °C, members of this group have the potential to volatilise from water or moist soil surfaces. These chemicals are unlikely to degrade by hydrolysis as they lack a functional group that is hydrolytically reactive. However, in the air, category members have the potential to rapidly degrade through indirect photolytic processes (OECD, 2012).

C. Biodegradation

Kerosine's are readily to inherently biodegradable. In the supporting OECD 301 study, naphtha solvents were readily biodegraded in 28 days but not within the 10-day window. The mean of three samples was 61% theoretical biological oxygen demand on Day 28. In a valid OECD 301F supporting study Kerosine Mid-Blend was not considered readily biodegradable in 28 days, with less than 60% degradation on day 28 (58.6%). However, according to USEPA guidance for biodegradability, it is considered inherently biodegradable because significant degradation occurred). Based on this and the known properties of hydrocarbons in the range C9 to C16, kerosines are often considered not readily biodegradable; but as they can be degraded by microorganisms, they are regarded as being inherently biodegradable.

If a chemical is found to be inherently or readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

D. Environmental Distribution

Standard adsorption/desorption studies are not applicable to petroleum UVCB substances. Mackay Level III modelling indicates that category member constituents partition mostly to the sediment and soil compartments rather than air compartment when an equal emission rate (1000 kg/hr) to the air, water, and soil compartment is assumed. When release occurs only to either the air, or soil compartment, constituents are indicated in the modelling to partition largely to the compartment to which they are released. When released to the water compartment, constituents are indicated by the model to partition to either water or sediment (HPVIS). However, based on the member category low solubility, partitioning to sediment would be expected.

E. Bioaccumulation

No experimental studies are available on the substance. Using BCFBAF in EPISuite™, the estimated BCF of a representative substance is 0.893 L/kg based on the Arnot-Gobas model that includes biotransformation and upper trophic. Thus, bioaccumulation is not expected (ECHA). [Kl. score = 2]

IV. HUMAN HEALTH HAZARD ASSESSMENT

The information presented within this Section was derived in part from read-across substances: hydrodesulfurised kerosine (CAS RN 64742-81-0) and undiluted JP-8 jet fuel (CAS No. 8008-20-6).

A. Summary

The substance has low acute toxicity by the oral and dermal route. It is not irritating to the skin and eyes, but it is a skin sensitiser. Aside from minor changes in body weight, no adverse effects were seen in animals given repeated doses by the oral route. The substance is not genotoxic when tested in both *in vitro* and *in vivo* assays. There is no indication that this substance will cause malformations or have an adverse effect on reproduction and development. This information was derived in part from products of similar structure or composition.

B. Toxicokinetics

The studies of the pharmacokinetics (i.e., absorption, distribution, metabolism and excretion) of kerosine are scarce. There are some *in vitro* and *in vivo* studies available on jet fuels. However, because jet fuel is a complex mixture, these studies use certain constituents of jet fuels as marker compounds to describe the total jet fuel's pharmacokinetics. There are more data available for a number of kerosine constituents, and these can be used as a basis for understanding the pharmacokinetics of kerosine as a whole. There are three ways in which humans are exposed to kerosine: by inhalation; ingestion; and dermal contact. Due to the relatively low volatility of kerosine and jet fuels, dermal exposure can be a more important route of exposure than exposure via inhalation. During many operations involving aircraft fuel tanks there is a significant potential for dermal exposure. Ingestion occurs primarily as a consequence of incidental ingestion.

Groups of five male C3H mice were dosed with a single dermal application of 15 or 60 µL kerosine (30% straight-run hydrotreated and 70% hydrocracked kerosine) spiked with radiolabelled naphthalene or tetradecane, and sacrificed after 96 h exposure (Mobil, 1994). Another group of five male C3H mice were exposed by air to the same compounds and doses in a metabolism cage to determine passive inhalation. The results of the dermal exposure show that 5% of the labelled tetradecane and 15% of the labelled naphthalene were absorbed over 96 h. The inhalation experiments showed that 2.8% of the labelled naphthalene was bioavailable. Comparison of these data with a similar dataset obtained with a 25% concentration of the test compounds diluted in mineral oil, revealed that dilution did not affect the absorption of the test compound.

Four groups of eight male Sprague-Dawley rats were exposed to 1, 4, 8, or 16 mL kerosine through the abdominal skin for 2 h at a skin area of 4, 8, 16 or 64 cm2, respectively (Tsujino et al., 2003). Before, during and after the experiment, blood samples were taken and analysed for trimethylbenzenes and aliphatic hydrocarbons. Trimethylbenzenes were detectable in blood within 5-20 min and showed a dose dependent absorption. High concentrations of aliphatic hydrocarbons were detected in the exposed skin as compared to the blood concentration. The aliphatic hydrocarbon levels were dependent on the amount of kerosine exposed per unit area.

The systemic distribution of kerosine components in the blood and tissues of rats following *in vitro* dermal exposures was investigated, using trimethylbenzenes and aliphatic hydrocarbons (C9-C16) as biomarkers (Tsujinoet al., 2002). The trimethylbenzenes were absorbed through the skin and detected in blood and tissues to a greater extent as compared to the aliphatics. The data indicate that kerosine components are absorbed percutaneously and distributed to the various organs via the blood circulation. Distribution of trimethylbenzenes in blood and tissues following dermal exposure

is (at decreasing concentrations): kidney > blood > liver > adipose > brain > spleen > lung = muscle. Distribution of aliphatics in blood and tissues following dermal exposure is (at decreasing concentrations): blood > adipose > muscle > lung > liver > kidney > spleen > brain.

The inhalation studies demonstrate that the volatile kerosine constituents are well absorbed (31 – 54%) and are distributed mainly in the fat tissue. Aromatics were metabolised at a higher rate than naphthenes, n-alkanes, isoalkanes and 1-alkenes. Dermal application of kerosine or jet fuel generally shows that the aromatics and aliphatics are well absorbed into the skin. Subsequently, the aromatics penetrate the skin at a higher rate than the alkanes. SKINPERM calculations indicate that although skin permeation rates of alkanes, naphthenes and aromatics are more or less comparable, the latency times of alkanes are longer than the latency times of naphthenes and aromatics. After absorption, the kerosine constituents are distributed via the blood circulation to the fat tissue and various organs. Studies with oral exposure to kerosine indicate that gastrointestinal absorption of kerosine is slow and incomplete, resulting in low bioavailability.

C. Acute Toxicity

Kerosines are of low acute toxicity, with an oral LD50 greater than 5,000 mg/kg (rat), a dermal LD50 greater than 2,000 mg/kg (rabbit), and an inhalation LC50 greater than 5.28 mg/L (rat). The most important effects in animals following very high oral doses were slight irritation of the stomach and the gastrointestinal tract. The only adverse effects observed in acute inhalation studies were decreased activity and breathing frequency at very high doses. Dermal application of kerosine did not lead to acute toxic systemic effects. Clinical effects observed were related to dermal irritation rather than to systemic toxicity. The acute toxicity of kerosine is not classified by EU CLP Regulation (EC No. 1272/2008).

Oral

In the key acute oral toxicity study (Klimisch score=1; ARCO, 1992a), groups of fasted (5 per sex), young adult, Sprague Dawley rats were given a single oral dose of undiluted thermocracked kerosine at a dose of 5,000 mg/kg bw and observed for 14 days. There were no treatment related mortalities. All of the study animals exhibited one or more of the following clinical signs: nasal discharge, ocular discharge, abnormal stools, lethargy, stained coat, and alopecia. All animals gained weight during study period. At necropsy, one of the ten animals exhibited visual lesions, the remaining nine showed signs of alopecia in the inguinal and/or perineal regions. The oral LD50 was determined to be greater than 5000 mg/kg in males and females.

In supporting studies conducted on kerosine substances, rats were administered single oral gavage doses of the test substance. The results supported an oral LD50 of > 5,000 mg/kg in males and females.

Inhalation

In the key acute inhalation toxicity study (Klimisch score = 1; API, 1987a), groups of Sprague-Dawley rats, five males and five females, were exposed by inhalation route to straight-run kerosine for 4 hours to their whole body at a single dose of 5.28 mg/L (vapour, analytical). All except one animal had normal growth rates throughout the study. The one exception on day 8 had a body weight less than its starting body weight but by the end of the study normal growth had resumed. All animals exhibited decreased activity during the exposure. Otherwise, there were no treatment-related clinical signs of toxicity. No macroscopic lesions were observed in any animal at post-mortem and no microscopic changes were observed in any lung section examined. The LC50 was greater than 5.28 mg/L.

In supporting studies conducted on kerosine substances, rats were administered single doses of the test substance via inhalation. The LC50s as measured based on mortality and systemic effects do not indicate classification of kerosine as an acute inhalation toxicant. One supporting study on deodorised kerosine showed a lack of systemic effects after repeated exposure to rats (6 hours each day for 4 days) and resulted in an LC50 of > 7.5 mg/L (Carpenter et al., 1976). Another supporting study on deodorised kerosine showed a lack of systemic effects after a single 6-hour exposure to cats and resulted in an LC50 of > 6.4 mg/L (Carpenter et al., 1976).

Dermal

In the key acute dermal toxicity study (Klimisch score=1; ARCO, 1992g), groups of young adult New Zealand White rabbits, five males and five females, were dermally exposed to undiluted thermocracked kerosine for 24 hours to 10% of their body surface area at a dose of 2,000 mg/kg. Animals were then observed for 14 days. There were no mortalities and all animals gained weight during the study. All of the animals exhibited one or more of the following clinical signs during the observation period: dermal irritation (erythema, oedema, eschar, fissuring and/or dried skin) and/or abnormal stools. Apart from skin irritation, there were no other abnormalities noted at necropsy. The dermal LD50 was determined to be greater than 2,000 mg/kg in both males and females.

In supporting studies conducted on kerosine substances, rabbits were administered single dermal doses of the test substance, and results supported a dermal LD50 of > 2,000 mg/kg in males and females.

D. Irritation

Skin

In the key study, young adult rabbits (6 females) were dermally exposed (occlusive coverage) to 0.5 mL of undiluted kerosine/heating oil for 24 hours on both intact and abraded skin sites. Each of the test sites was evaluated for skin responses for 9 days post-exposure and was scored using the Draize scale. The mean erythema score from 24 to 72 hours was 3.46/4 while the mean oedema score from 24 to 72 hours was 2.33/4. While this protocol deviates from current guidelines that state exposure should be semi-occlusive over 4 hours, and to intact skin only, this study is included as key to show the irritating nature of kerosine products.

In another guideline study conducted according to GLP and in accordance with current guidelines, young adult New Zealand White rabbits (3 per sex) were dermally exposed (semi-occlusive coverage) to 0.5 mL of undiluted odourless kerosine, for 4 hours. Animals were observed for seven days after exposure. Irritation was scored based on the Draize method (1959). The mean erythema score from 24 to 72 hours was 0.17/4 while the mean oedema score from 24 to 72 hours was 0/4.

Additional supporting studies are provided on straight run kerosine, odourless kerosine, hydrocracked kerosine, hydrodesulfurised kerosine, Jet Fuel A, Jet Fuel A1, JP-5, and Cherry Point Jet Fuel A. Most of the studies are valid in their methodology, but they differ from the current OECD guidelines in that animals were exposed under occluded conditions for 24 hours instead of semi-occluded conditions for 4 hours. Considering the conditions of the test, results must be interpreted carefully for the purposes of classification and labelling. The mean scores for erythema and oedema have been assessed against the deviations and provided the test would be conducted under standard conditions, the overall weight of evidence indicates that kerosines are irritating to skin. Kerosines are classified as irritating to the skin according to criteria in EU CLP Regulation (EC No. 1272/2008).

Effects on skin irritation/corrosion: irritating

Eyes

Several well-controlled (GLP) animal experiments performed on a variety of kerosines indicate that none of the kerosines and jet fuels tested were more than slightly irritating to the eyes. In addition, a number of short reports on eye irritation studies on JP-5 and JP-8 show no eye irritation whatsoever in rabbits (6 unwashed eyes; 3 washed eyes): all scores 0.0 for up to 7 days (end of the study). None of the hazard assessments of kerosine and jet fuel constituents have resulted in classification for eye irritation.

In the key study selected for primary eye irritation, 0.1mL of undiluted thermocracked kerosine was instilled into the conjunctival sac of the right eye of three female young adult New Zealand White rabbits and observed through 72 hours. Irritation was scored according to the Draize method (1959). There was no evidence of damage to the cornea or iris for all animals over all scoring periods. Mild conjunctivae indicators such as redness, chemosis, and discharge were evident at the one-hour scoring interval, but not at any of the other scoring intervals. Fluorescein staining scores were zero for all study animals over all scoring periods.

The average irritation score was 0.0 for the cornea, iris and conjunctivae.

Based on the evidence, kerosine is not an eye irritant.

E. Sensitisation

In animal assays for skin sensitisation such as the Magnusson-Kligman GPMT and the Buehler assay, kerosines and jet fuels did not trigger a positive response.

In the key dermal sensitisation study (Klimisch score=1; ARCO, 1992q), thermocracked kerosine in mineral oil was tested on male young adult Pig/Hartley guinea pigs using a modified Buehler technique. During the challenge phase, a second exposure of a 1:4 dilution of thermocracked kerosine to induced test animals did not yield higher response grades, severity, or incidence than those associated with the naive challenge control group exposed to thermocracked kerosine. During the challenge phase, exposure of 0.2% DNCB to induction positive control animals elicited significantly higher response grades, severity indices, and incidence over the naive DNCB challenge control group. The vehicle irritation control group was free of dermal irritation during the challenge phase. Therefore, under the conditions of this study, thermocracked kerosine is not considered a delayed contact sensitiser while DNCB induced an appropriate positive response.

Based on test data, there was no evidence of skin sensitisation; therefore, kerosine is not classified for skin sensitisation according to EU CLP Regulation (EC No. 1272/2008)

F. Repeated Dose Toxicity

Oral

In the key oral subchronic study (Klimisch score=1; Mattie et al., 2000), male rats were treated for 70 to 90 days with 0 (1mL of distilled water), 750, 1,500, or 3,000 mg/kg/day of undiluted JP-8 jet fuel, then mated to untreated females (one female at a time). Males were gavaged throughout the cohabitation period and were returned to their individual cage after successful mating. In the second part of the study, female rats were administered the test compound at doses of 0 (1mL of distilled water), 375, 750, or 1,500 mg/kg/day undiluted JP-8 jet fuel for 90-day prior to mating, through

mating, gestation, delivery, and lactation for a total of 21 week. During mating, they were housed with untreated males.

There were no effects on clinical signs or mortality in either sex. Haematology, clinical chemistry, and urinalysis were measured only in females without any effects noted. Body weights in male rats were decreased in a dose-dependent manner and was likely related to nephropathy, which is specific in male rats treated with hydrocarbons, and not relevant for human exposure. In females, body weight was only significantly reduced in the high-dose group. Absolute and relative liver weights were increased in mid- and high-dose females but were not likely biologically significant due to the lack of changes in clinical chemistry or histopathology in the liver. The test compound caused perianal dermatitis (high-dose only) and stomach hyperplasia (mid- and high-dose) in the female rats. There was a dose-related decrease in pup weight that was significant in the 750 mg/kg/day group on postnatal day 4 only and in the 1,500 mg/kg/day group from postnatal day 4 through postnatal day 21 but had recovered by postnatal day 90. There were no treatment-related effects on reproduction or sperm parameters in males. There were no effects on reproduction, gestation, or litter size in females.

The study low observed adverse effect level (LOAEL) for systemic effects is 1,500 mg/kg/day and the no observed adverse effects level (NOAEL) for systemic effects is 750 mg/kg/day, based on reduced body weight in dams and in pups. The LOAEL for adult male rats exposed to JP-8 orally was 750 mg/kg/day due to changes in clinical pathology, body weight, organ weights and the same irritation seen in female rats. The decrease in male rat bodyweight is very likely due to the male rat-specific nephropathy and is therefore not considered for the derivation of the oral NOAEL. The reproduction NOAEL was 3,000 and 1,500 mg/kg/day in males and females, respectively.

Inhalation

In a key subchronic inhalation toxicity study (Klimisch score=1; Mattie et al., 1991), JP-8 jet fuel was administered to 95 male Fisher 344 rats, 75 female Fischer 344 rats, and 100 male and female C57BL/6 mice by dynamic whole body vapour exposure at concentrations of 0, 500 or 1,000 mg/m3(0, 0.5, or 1.0 mg/L) as a vapour for 24 hours per day, 7 days/week for a total of 90 days. The male rats developed hydrocarbon-induced nephropathy at both treatment concentrations. Male rats had decreased body weight and decreased absolute and relative kidney weight at both treatment concentrations. Female rats were unaffected by treatment. In mice, no significant clinical signs of toxicity were noted that differentiated the groups that were treatment-related. The no observed adverse effect concentration (NOAEC) for male rats is difficult to establish, since potential adverse effects may be masked by male rat specific hydrocarbon nephropathy. However, based on the hydrocarbon-induced nephropathy and reduced body weights and increased kidney weights, the lowest observed adverse effects concentration (LOAEC) in male rats is 500 mg/m^3. The LOEC for male mice is also 500 mg/m^3, but it was not treatment related. The NOAEC for female rats and mice is greater than or equal to 1,000 mg/m3. This was the highest dose tested in the study.

In a subacute inhalation toxicity study (Klimisch score = 1; API, 1986), hydrodesulfurised kerosine vapour was administered to 20 Sprague-Dawley rats/sex/concentration by dynamic whole-body exposure at a concentration of 24 mg/m^3 (0.024 mg/L) for 6 hours per day, 5 days/week for 4 weeks. There were no compound related effects in mortality, clinical signs, body weight, haematology, clinical chemistry, organ weights, or gross and histologic pathology. Therefore, the NOAEC is greater than or equal to 24 mg/m^3. This was the highest dose tested in the study.

Dermal

In a key sub-chronic dermal study hydrodesulfurised kerosine was applied at concentrations of 20, 40 or 60% (v/v) at a rate of 1 ml/kg/day to the shorn intrascapular region of groups of 12 individually housed male and female, Sprague-Dawley rats (aged 7-9 weeks). This was equivalent to doses of test material of 165, 330 or 495 mg/kg/day. Dosing was continued for five days a week for 13 weeks. In addition, a group of 12 male and 12 female rats of similar age were administered mineral oil at a dose rate of 1 ml/kg/day; these animals served as vehicle controls. 12 rats/sex/group each in the vehicle controls and high dose group were maintained for a 4-week recovery period. Ingestion of the test material was prevented by using a collar and removal of any residual test or control material from the skin. Animals were observed for clinical signs prior to dosing and 1, 6 and 24 hours after the first dose. Subsequently, observations were made prior to each dose being applied.

Prior to the administration of each dose, the treated skin site was evaluated for dermal irritation using the Draize scoring method. Body weights were recorded prior to the first dose and weekly thereafter. An ophthalmic examination was conducted on each rat prior to application of the first dose and again prior to sacrifice at the end of the study. During the week prior to the first dose, each rat was subjected to a functional observation battery (FOB). The FOB was conducted again 1, 6 and 24 hours after the first dose and at 7 and 14 days. During the study, the FOB, motor activity and startle response testing was conducted on all rats at weeks 4, 8 and 12. At week 14 blood samples were collected from 12 animals/sex/group. Full necropsies were performed at week 14 on 6 rats/sex/group and at week 18 on the recovery rats (vehicle and high dose groups). Each full necropsy included an examination of the external surface of the body and its contents. The remaining six rats of each group were anesthetized with an intraperitoneal injection of Pentothal and transcardially perfused in-situ using 10% neutral-buffered formalin and given a limited necropsy. For these rats, no organs were weighed, and specific tissues were also collected for subsequent microscopic testing.

There was a generally dose-related increase in the incidence and severity of various skin conditions at the treated site. Males seemed to be more sensitive than females as they were affected at all doses, however, the effects indicated very little irritation. Recovery group animals revealed complete recovery in the females and minimal hyperkeratosis in the high dose group males. At necropsy no substance-related observations were made for males in any group. In the females there was a suggestion of a possible treatment-related effect which occurred in 7 rats across all groups and consisted of skin crusts or ulceration at the site of application of test material. Haematological and serum clinical parameters were unaffected by treatment.

All animals survived until scheduled termination. There were no test substance-related effects on survival, clinical observations (apart from skin irritation), neurobehavioral signs or ophthalmological findings. The NOEL for systemic toxicity was >495 mg/kg/day. The LOEL for slight dermal irritation was 165 mg/kg/day, equivalent to ~ 1 mg/cm^2.

G. Genotoxicity

In vitro Gene Mutation in Mammalian Cells

Key in vitro gene mutation studies in mammalian cells were identified. In a study by the American Petroleum Institute (API, 1984b), cultures of mouse lymphoma cells were exposed to hydrodesulfurised kerosine with or without metabolic activation by Aroclor 1254-induced rat liver S9 fraction. Under non-activation conditions the test material induced a good range of toxicities for evaluation (relative growths ranged from 2.8% to 65.3%). None of the assays induced a mutant

frequency that exceeded the minimum criterion (40.8 x 10-6). The test material was not mutagenic under non-activation conditions. In the presence of metabolic activation, a wide range of toxicities was induced (6.1 to 107.9% relative growths). The minimum criterion mutant frequency of 69.0 x 10-6 was not exceeded. The test material was therefore considered non mutagenic under activation conditions. In a study by API (1977) (Klimisch score = 1), mouse lymphoma L5178Y cells were exposed to straight-run kerosine in acetone vehicle at concentrations ranging from 0.04 to 0.065 µL/mL (with metabolic activation) or 0.006 to 0.13 µL/mL (without activation). There was no evidence that straight-run kerosine induced mutant colonies over background levels.

In vitro Cytogenicity in Mammalian Cells

Hydrodesulfurised kerosine was tested in the sister chromatid exchange assay using Chinese hamster ovary cells (API, 1988a). The assay was conducted with Aroclor-induced rat liver S-9 activation system. A small but statistically significant increase in the frequency of sister chromatid exchanges was observed at the high and low concentrations with metabolic activation. These increases appeared to be random and of no biological significance. There were no significant increases observed at any concentration in the absence of metabolic activation. Under the conditions of the study, hydrodesulfurised kerosine is negative in the sister chromatid exchange assay with Chinese hamster ovary cells.

In vivo Cytogenicity

Based on weight of evidence kerosine substances were found to be non-mutagenic through cytogenic investigations.

In six in vivo bone marrow cytogenetic studies in the rat, there were no indications of chromosomal aberrations. Although an in vivo Sister Chromatid Exchange study in the mouse gave positive findings in the male group (but not in the females) the positive findings in the males were associated with signs of toxicity (lethargy and weight loss) at the very high-top dose used in the study (4,000 mg/kg), both on the day of the administration of the kerosine and the day after (when they were sacrificed).

In a rat bone marrow micronucleus assay (API, 1985c, Klimisch score = 1), straight run kerosine (CAS RN 800-20-6) was administered to Sprague Dawley rats. Straight run kerosine was not considered to induce chromosomal aberrations in bone marrow cells of rats. In another bone marrow micronucleus assay (API, 1984b, Klimisch score = 1), hydrodesulfurised kerosine (CAS RN 64742-81-0) was administered to rats. No clinical signs of toxicity were exhibited by the rats, and there was no significant increase in frequency of micronucleated polychromatic erythrocytes in bone marrow as compared to control. In a study by API (1977) (Klimisch score = 1), straight-run kerosine (CAS RN 8008-20-6) was administered to 45 male rats. No significant increase in the frequency of micronucleated polychromatic erythrocytes was observed.

In vivo Gene Mutation

Key in vivo gene mutation studies were identified. In a sperm cell dominant lethal mutation assay (API, 1980b, Klimisch score = 1), Jet Fuel A was administered via inhalation route to male mice at concentrations of 100 or 400 ppm for a 6-hour exposure period, 5 days per week for 8 weeks. Males were mated with females, and the uteri of pregnant females were examined for living and dead implants. Jet Fuel A did not increase the incidence of post-implantation deaths. In another study by API (1973) (Klimisch score = 1), deodorised kerosine was administered subcutaneously to 10 male Swiss-Webster mice in corn oil vehicle or intraperitoneally to 10 Long-Evans rats undiluted at a dose of 1.0 mL/kg. Males were mated with females, and no pattern of decreased pregnancy rate or increased embryo loss was observed in the females.

H. Carcinogenicity

Kerosine is not carcinogenic when animals are exposed via the oral or inhalation route (ECHA).

Male mice were administered dermally 37.5µL of jet fuel A to the shaved backs of 50 mice per dose, twice a week for 2 years or intermittently so that application of the jet fuel was suspended when dermal irritation was noted in 20% of the group and was resumed when irritation resolved in all but 20% of the affected animals. There was a significant increase in tumours at the application site with continuous treatment compared to the control (0% versus 44%), but not with intermittent treatment (0% versus 2%). With continuous treatment, there was a treatment-related increase in dermal tumour incidence compared to controls. However, stopping treatment during dermal irritation nearly eliminated the carcinogenic effect (ECHA) [Kl. Score = 1].

Male and female mice were administered dermally 25 mg of petroleum-derived jet fuel A to the shaved backs of 25 mice, three times a week for 105 weeks. Due to high mortality, jet fuel A application was discontinued during week 62, but surviving animals were observed until study termination. There was a significant increase in tumours at the application site (0%, 26%, and 26% in the controls, JP-4, and jet A groups). The majority of the tumours were squamous cell carcinomas or fibrosarcomas. At the doses tested, there was a treatment-related increase in dermal tumour incidence when compared to controls. The results of the study indicate that there was a treatment-related increase in dermal tumour incidence when compared to controls, therefore it can be concluded that Jet fuel A has a carcinogenic effect on mice at 25 mg dosage (ECHA) [Kl. Score = 1].

Straight-run kerosine (CAS RN 8008-20-6) and hydrodesulfurised kerosine (CAS RN 64742-81-0) were tested in standard 2-year bioassays in mice. The animals, 50 per group, were treated twice weekly with 50 µl straight-run kerosine or with hydrodesulfurised kerosine. It was concluded that both straight-run and hydrodesulfurised kerosine were moderate skin carcinogens (ECHA) [Kl. Score = 2].

In the key carcinogenicity study from NTP, JP-5 navy fuel in acetone was administered to 50 mice dermally at dose levels of 0 (vehicle control), 250, or 500 mg/kg bw/day for up to 103 weeks. There was a significant decrease in survival in females at both treatment doses. Remaining high-dose females were sacrificed at week 90. There was no treatment-related effect on survival in male mice. The LOAEL is 250 mg/kg/day, based on dermatitis and decreased survival in females. No NOAEL can be determined. At the doses tested, there was not a treatment-related increase in tumour incidence when compared to controls (ECHA) [Kl. Score = 1].

The potential influence of skin irritation on tumour development in long-term mouse skin painting studies was investigated as part of the CONCAWE middle distillates programme. The study included straight run hydrotreated kerosine (MD3). The test material was applied to the shorn skin of three groups of 50 male mice for 104 weeks. For the straight run hydrotreated kerosine, skin tumours only developed in the group of animals in which substantial skin irritation occurred during the study. Since no polycyclic aromatic compounds were detected in the straight run kerosine it is concluded that the occurrence of tumours is likely to have been caused by a non-genotoxic mechanism. This conclusion is consistent with reports by others that lighter middle distillates are tumour promotors but not initiators and furthermore that skin irritation plays an important role in skin tumour development. These tumours are probably the consequence of a continuous cycle of cell damage and repair caused by chronic skin irritation. The conclusions gained from this study can be applied to other carcinogenicity studies on kerosines, and they show that tumours are noted in the presence of repeated dermal irritation, and that kerosines lack a genotoxic mechanism of carcinogenicity (ECHA) [Kl. Score = 1].

I. Reproductive Toxicity

There are no specific reproductive toxicity data for the substance but there are data available with ECHA as migrated information which is read-across based on grouping of substances (category approach).

An OECD Guideline 415 One-Generation Reproduction Toxicity study was conducted. This was a reproductive study performed in two parts. In the first part, males were treated for 70 to 90 days with 0 (1mL of distilled water), 750, 1,500, or 3,000 mg/kg/day of undiluted JP-8 jet fuel, then mated to untreated females (one female at a time). In the second part of the study, female rats were administered the test compound at doses of 0 (1mL of distilled water), 375, 750, or 1,500 mg/kg/day undiluted JP-8 jet fuel for 90 -day prior to mating, through mating, gestation, delivery, and lactation for a total of 21 weeks.

There were no changes in clinical signs or mortality in parental animals. Body weights in male rats were decreased in a dose-dependent manner. Terminal body weights were approximately 545 grams, 520 grams, 475 grams, and 315 grams in the control, 750, 1,500, and 3,000 mg/kg/day, respectively. In females, body weight was only significantly reduced in the high-dose group, but the differences were not significant at terminal sacrifice. The body weight in females at 20 weeks (1 week before sacrifice) was approximately 400 grams, 385 grams, 382 grams, and 335 grams in the control, 375, 750, and 1,500 mg/kg/day, respectively. Hematology was not measured in the males and no effects were noted in the females. Clinical chemistry was not measured in the males and no effects were noted in the females. Urinalysis was not measured in the males and no effects were noted in the females. Absolute and relative liver weights were increased in mid- and high-dose females but were not accompanied by any histological findings. The test compound caused perianal dermatitis (high-dose only) and stomach hyperplasia (mid- and high-dose) in the female rats.

There were no treatment-related effects on reproduction or sperm parameters in males. There were no effects on reproduction, gestation, or litter size in females. The lowest NOAEL based on parental body weight was determined to be 750 mg/kg/day.

The F1 generation was not examined for clinical signs though no mention would suggest no significant signs were noted. No mortality was observed. There were no effects on offspring viability. However, there was a dose-related decrease in pup weight that was significant in the 750 mg/kg/day group on postnatal day 4 only and in the 1,500 mg/kg/day group from postnatal day 4 through postnatal day 21. The 1,500 mg/kg/day group recovered by postnatal day 90. The NOAEL based on offspring body weight was determined to be 750 mg/kg/day.

J. Reproductive Toxicity/Developmental Toxicity

In a developmental toxicity study, undiluted JP-8 jet fuel was administered to 30 Sprague-Dawley (Crl:CD) rats/dose by gavage at various volumes to achieve dose levels of 0 (sterile water), 500, 1,000, 1,500, or 2,000 mg/kg bw/day from days 6 through 15 of gestation.

There was a significant decrease in maternal weight gain with doses of 1,000 mg/kg/day or greater. Maternal necropsy weight was significantly different than the control in the 1,500 and 2,000 mg/kg/day groups. There were no apparent clinical signs of toxicity. Reproductive endpoints were not assessed in this study because females were pregnant prior to treatment and did not deliver, so only developmental endpoints can be assessed. Thirteen females (one 1,000 mg/kg/day; three 1,500 mg/kg/day, and nine 2,000 mg/kg/day) were found dead. Although there appears to be a dose-dependent increase in the mortality, necropsy found the cause of death to be related to the

presence of the test compound in the lungs indicating dosing into the lungs instead of the gastrointestinal tract. The maternal LOAEL is 1,000 mg/kg/day, based on reduced body weight gain. The maternal NOAEL is 500 mg/kg/day.

There was a significant decrease in foetal weight in both male and female foetuses dosed with 1,500 and 2,000 mg/kg/day. The test compound did not significantly increase the incidence of malformations or variations compared to the control nor was the sex ratio altered. The developmental LOAEL is 1,500 mg/kg/day, based on reduced foetal weight. The developmental NOAEL is 1,000 mg/kg/day. It can be concluded that the test substance is not toxic to development.

This study received a Klimisch score of 1 and is classified as reliable without restrictions because it was carried out in a method equivalent/similar to OECD TG 414.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for the substance follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

Non-Cancer

The NOAEL for reduced maternal body weight is 500 mg/kg/day, based on reduced body weight in dams and in pups treated under a repeat dose regimen. The NOAEL from this study will be used for determining the oral Reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:
UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 10
UF_D (database uncertainty) = 1

Oral RfD = 500 /(10 x 10 x 1 x 10 x 1) = 500/1,000 = 0.5 mg/kg-day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

where:
Human weight = 70 kg (ADWG, 2011)

Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)

Drinking water guidance value = (0.500 x 70 x 0.1)/2 = 1.8 mg/L

Cancer

There are no carcinogenicity studies on the substance or related hydrocarbons. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

The substance does not exhibit the following physico-chemical properties:

- Explosivity
- Oxidising potential

The substance is classified as a "Flammable Liquid Category 3"

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

The substance is of low acute concern to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

Table 2 lists the results of acute aquatic toxicity studies on hydrotreated light petroleum distillate surrogates.

Table 2: Acute Aquatic Toxicity Studies on Hydrotreated Light Petroleum Distillate Surrogate[2]

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Oncorhynchus mykiss	96-hour LL_{50}	2-5	1	ECHA
Daphnia magna	48-hour EL_{50}	1.4	1	ECHA
Raphidocelis subcapitata	72-hour EC_{50}	<1-3 (average of 2)	1	ECHA
Selenastrum capricornutum	72-hour EC_{50}	3.7	2	ECHA

Chronic Studies

There are no long-term toxicity studies on fish. A single long-term study on invertebrates is discussed below.

In a 21-day semi-static chronic reproductive toxicity test (OECD 211; KS = 1) on *Daphnia magna*, hydrodesulfurised kerosine was evaluated using water accommodated fraction methodology. The

[2] Hydrodesulfurised Kerosine (CAS RN 64742-81-0)

actual loading rates were 0 (control), 0.08, 0.19, 0.48, 1.2 and 3.0 mg/L. Under the conditions of this test, the 21-day chronic reproductive NOEL for kerosine is 0.48 mg/L. The LOEL is 1.2 mg/L. The EL_{50} based on reproduction is 0.89 mg/L (ECHA).

C. Terrestrial Toxicity

There are no terrestrial toxicity studies for this substance.

D. Calculation of PNEC

The PNEC calculations for hydrotreated light petroleum distillate follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available from acute tests on three trophic levels. There is one long term study on a single trophic level organism, *D. magna*.

On the basis that the data consists of short-term studies from three trophic levels and a long-term study from one trophic level, an assessment factor of 100 is applied to the 21-day chronic reproductive NOEL for kerosine of 0.48 mg/L. The $PNEC_{aquatic}$ is 0.005 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 0.36 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (93.4/1280) \times 1000 \times 0.005$
$= 0.36$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3) [calculated]
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]

$K_{sed\text{-}water} = 0.8 + [0.2 \times Kp_{sed}/1000 \times BD_{solid}]$
$= 0.8 + [0.2 \times 193/1000 \times 2400]$
$= 93.4\ m^3/m^3$

And:
Kp_{sed} = solid-water partition coefficient (L/kg).[calculated]
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]

$Kp_{sed} = K_{oc} \times f_{oc}$
$= 4818 \times 0.04$
$= 193$ L/kg

Where:
K_{oc} = organic carbon normalized distribution coefficient (L/kg). The K_{oc} for hydrodesulfurised kerosine calculated from EPISUITE™ using the MCI is 4818 L/kg.
F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no experimental toxicity testing results available for the substance or its noted surrogates. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.32 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
$= (96.4/1500) \times 1000 \times 0.005$
$= 0.32$ mg/kg

Where:
Kp_{soil} = soil-water partition coefficient (m3/m3)
BD_{soil} = bulk density of soil (kg/m3) = 1,500 [default]

$Kp_{soil} = K_{oc} \times f_{oc}$
$= 4818 \times 0.02$
$= 96.4\ m^3/m^3$

And:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for hydrodesulfurised kerosine calculated from EPISUITE™ using the MCI is 4818 L/kg.
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

The substance or similar compounds are readily biodegradable; thus they do not meet the screening criteria for persistence.

Based on the estimated BCF values, derived from EPISuite estimates (BCF = 3.162 L/kg wet-weight) the substance does not meet the screening criteria for bioaccumulation.

The NOEC values from acute and chronic aquatic toxicity studies on the substance indicate it does not meet the screening criteria for toxicity.

Therefore, hydrotreated light petroleum distillates are not PBT substances.

IX. CLASSIFICATION AND LABELLING

A. Classification

Asp. Tox. 1

B. Labelling

Danger

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Rinse immediately with plenty of running water. If easy to do, remove contact lenses. Get medical attention if symptoms persist.

Skin Contact

Wash with soap and water. Get medical attention if symptoms occur.

Inhalation

Treat symptomatically. Move to fresh air. Get medical attention if symptoms persist.

Ingestion

In case of ingestion, always assume that aspiration has occurred. Do not induce vomiting. Rinse mouth with water and then drink plenty of water. Never give anything by mouth to an unconscious person. Seek medical attention.

B. Fire Fighting Information

Extinguishing Media

Foam (Specifically trained personnel only)- Water fog (Specifically trained personnel only)- Dry chemical powder- Carbon dioxide- Other inert gases (subject to regulations)- Sand or earth

Specific Exposure Hazards

None known.

Special Protective Equipment for Firefighters

Self-contained breathing apparatus and full protective clothing must be worn in case of fire.

C. Accidental Release Measures

Personal Precautions

Wear appropriate personal protective equipment.

Environmental Precautions

Do not release to open drains or surface water. Not regarded as dangerous to the environment.

Steps to be Taken if Material is Released or Spilled

Collect free product with suitable means. Transfer collected product and other contaminated materials to suitable containers for recycle, recovery or safe disposal. Absorb spill with inert absorbent material, then place in a container for chemical waste.

D. Storage and Handling

General Handling

Ensure that all relevant regulations regarding explosive atmospheres, and handling and storage facilities of flammable products, are followed.

Other Handling Precautions

Wash hands thoroughly after handling.

Storage

Keep containers tightly closed and properly labelled. Protect from the sunlight5.3. Light hydrocarbon vapours can build up in the headspace of containers. These can cause flammability / explosion hazard.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for the substance.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection:
Respiratory protection is not required.

Hand Protection:
Minimize skin contact.

Skin Protection:
Body protection must be chosen depending on activity and possible exposure.

Eye protection:
Minimize eye contact.

Other Precautions:
Handle in accordance with good industrial hygiene and safety practice. Eyewash fountains and safety showers must be easily accessible.

F. Transport Information

The substance retains UN 1223 transport code is listed as such within the Australian Dangerous Goods (AUS 2018)

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

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ISOPROPANOL

This dossier on isopropanol presents the most critical studies pertinent to the risk assessment of isopropanol in its use in coal seam or shale gas extraction activities. It does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Propan-2-ol

CAS RN: 67-63-0

Molecular formula: C_3H_8O

Molecular weight: 60.1 g/mol

Synonyms: Isopropanol, isopropyl alcohol, 2-propanol, *sec*-propyl alcohol, dimethylcarbinol

SMILES: CC(C)O

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-Chemical Properties of Isopropanol

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Colourless liquid	2	ECHA
Melting Point	-88.5°C; -89.5°C[1]	2	ECHA
Boiling Point	82.5°C; 82.3°C @ 101.3 kPa	2	ECHA
Density	800 kg/m^3 @ 20°C	2	ECHA
Vapour Pressure	4,400 Pa @ 20°C; 6,002 Pa @ 25°C	2	ECHA
Partition Coefficient (log K_{ow})	0.05 @ 25°C	2	ECHA
Water Solubility	Miscible	2	ECHA
Viscosity	2.038 mPa s @ 25°C	2	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Isopropanol is readily biodegradable. It is not expected to bioaccumulate. Isopropanol has a low tendency to bind to soil or sediment.

[1] No information on the atmospheric pressure reported.

B. Partitioning

Isopropanol is miscible in water. Volatilisation from water surfaces or moist soil surfaces is expected to be an important fate process based upon this compound's estimated Henry's Law constant of 0.821 Pa m^3/mole. It is also expected to volatilise from dry soil surfaces based upon its vapour pressure (Pub Chem).

C. Biodegradation

Aerobic biodegradation of isopropanol has been shown to occur rapidly under non-acclimated conditions, based on a result of 49% biodegradation from a 5-day BOD test (Bridie et al., 1979). Additional biodegradation data developed using standardised test methods show that isopropanol is readily biodegradable in both freshwater and saltwater media (72 to 78% biodegradation in 20 days) (Price et al., 1974).

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

D. Environmental Distribution

No experimental data are available for isopropanol. Using KOCWIN in EPI Suite™ (USEPA, 2017), the estimated K_{oc} value from log K_{ow} is 3.478 L/kg. The estimated K_{oc} value from the molecular connectivity index (MCI) is 1.53 L/kg.

E. Bioaccumulation

Bioconcentration of isopropanol in aquatic organisms is not expected to occur based on a measured log K_{ow} of 0.05 (ECHA). Based on this estimated value, the substance is expected to have very high mobility in soil. If released to water, based on this value and its water solubility, it is also not expected to adsorb to suspended solids and sediment.

Volatilisation from water surfaces is expected with half-lives for a model river and model lake of 86 hours and 29 days, respectively (PubChem).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

The acute toxicity of isopropanol is low by the oral, dermal and inhalation routes. At high exposure levels, isopropanol is irritating to the eyes, nose and throat and may cause transient central nervous system depression. It is not a skin sensitiser, but in some individuals, there may be an allergic contact dermatitis due to cross-sensitisation to other alcohols, such as ethanol. Repeated high exposures cause reversible narcotic effects, consistent with other short-chain alcohols. Isopropanol is not genotoxic. Lifetime inhalation studies in rodents showed no carcinogenic effects. The weight-of-evidence indicates that isopropanol is not a reproductive toxicant. In a two-generation reproductive toxicity study, the male mating index was affected by isopropanol exposure; the significance of this effect is, however, unclear. Developmental toxicity can occur at maternally toxic doses; but it is not a teratogen. Isopropanol also does not affect neurobehavioral development.

B. Acute Toxicity

The acute oral LD_{50} of isopropanol has been reported as 4,700 mg/kg, 5,300 mg/kg, 5,500 mg/kg and 5,400 mg/kg in rats; 4,500 mg/kg in mice; and 5,030 mg/kg, 7,800 mg/kg and 7,900 mg/kg in rabbits (ECHA) [KI Score = 2].

The acute dermal LD_{50} in rabbits has been reported to be 12,900 mg/kg (ECHA) [KI Score = 2].

The acute inhalation 8-hour LC_{50} in rats was 19,000 ppm in females and 22,500 ppm in males (ECHA) [KI Score = 2]. Exposure of rats to 16,000 ppm for 8 hours resulted in four deaths out of six animals (ECHA) [KI Score = 2].

In an acute neurotoxicity study, male and female F344 rats were exposed to 0, 500, 1,500, 5,000 or 10,000 ppm isopropanol for 6 hours. A spectrum of behavioural effects indicative of narcosis, defined as a generalised loss of neuromotor and reflex function, was observed in animals of the 10,000 ppm group and to a lesser extent in the 5,000 ppm animals. Recovery from these effects was observed by 24 hours for the 10,000 ppm animals and by 6 hours for the 5,000 ppm animals. A concentration-dependent decrease in motor activity was observed for the 1,500 ppm males and the 5,000 ppm females. The results show that exposure of rats to isopropanol vapour produces transient, concentration-related narcosis and/or central nervous system sedation. The NOAEL for acute neurotoxicity is 500 ppm (ECHA) [KI Score = 2].

C. Irritation

Isopropanol applied to the intact or abraded skin of rabbits and guinea pigs produced negligible irritation. Liquid isopropanol is moderately irritating to the eyes of rabbits. Isopropanol produced little irritation when tested on the skin of six human subjects (ECHA) [KI Score = 1].

D. Sensitisation

There have been reports of isolated cases of dermal irritation and/or skin sensitisation. Except for three case reports, the positive reactions were observed on patch testing patients with contact dermatitis due to ethanol. These patients also had a positive reaction to ethanol.

E. Repeat Dose Toxicity

<u>Oral</u>

In a drinking water study, rats ingested 0.5 to 10% of isopropanol for 27 weeks and showed decreased body weight gain but no gross or microscopic tissue abnormalities (ECHA) [KI score = 3]. Increased formation of hyaline droplets in the proximal tubules was reported in male rats given 1–4% isopropanol in drinking water for 12 weeks (ECHA) [KI Score = 3].

A two-generation reproductive toxicity study has been conducted in rats given isopropanol by oral gavage. Pre-mating exposures were for at least 10 weeks for both generations. The results from this study are presented in the Reproductive Toxicity section (ECHA) [KI Score = 2].

<u>Inhalation</u>

F344 rats and CD-1 mice (both sexes) were exposed to 0, 100, 500, 1,500 or 5,000 ppm isopropanol for 6 hours/day, 5 days/week for 13 weeks. There were no deaths during the study. During and immediately following exposure to 5,000 ppm, ataxia, narcosis, hypoactivity and a lack of startle

reflex were observed in some rats and mice. Narcosis was not observed in rats during exposure following week 2, suggesting some adaptation to isopropanol. During exposures to 1,500 ppm, narcosis, ataxia, and hypoactivity were observed in some mice, whereas only hypoactivity was observed in rats. Immediately following exposures, ataxia and/or hypoactivity were observed in a few rats or mice exposed to 5,000 ppm. Overall, the 1,500 and 5,000 ppm rats and the 5,000 ppm female mice showed increased body weights and/or body weight gain during the study. Liver weights relative to body weight were observed in rats of both sexes and the 5,000 ppm female mice; however, no corresponding microscopic changes were noted in the liver. Histopathological evaluation showed a slight increase in the size and frequency of hyaline droplets in the kidneys of the isopropanol-exposed rats. Excluding the clinical signs of CNS depression, the NOAEL for this study is 5,000 ppm (ECHA) [Kl Score = 1].

In a subchronic neurotoxicity study, male and F344 rats were exposed by inhalation to 0, 100, 500, 1,500 or 5,000 ppm for 13 weeks. Neurobehavioural evaluations included a functional observation battery (FOB), motor activity and neuropathology. Effects of narcosis were observed in the 5,000 ppm groups only. There were no changes in FOB, but increased motor activity was noted in 5,000 female rats at weeks 9 and 13. Neuropathological examination revealed no exposure-related lesions in the nervous system. The NOAEL for acute effects is 500 ppm, and the NOAEL for subchronic neurotoxicity is 1,500 ppm (ECHA) [Kl Score = 1].

An additional subchronic neurotoxicity study was conducted to clarify the increased motor activity findings. Female F344 rats were exposed to 0 or 5,000 ppm of isopropanol vapour for 6 hours/day, 5 days/week. Half of the animals in each group were exposed for 9 consecutive weeks and the other half for 13 consecutive weeks. After 9 weeks of exposure, the motor activity effect was reversible within 2 days after the last exposure. Subtle differences in the shape of the motor activity versus test session time curve were noted in both the 9-week and the 13-week exposed animals, although it was unclear whether these changes were treatment-related. Complete reversibility of these changes did not occur until 1 and 6 weeks after the last exposure in the 9 and 13 week exposure groups, respectively (ECHA) [Kl Score = 2].

Male and female CD-1 mice were exposed by inhalation to 0, 500, 2,500 or 5,000 ppm isopropanol vapour 6 hours/day, 5 days/week for 18 months. An additional group of mice (all exposure levels) were assigned to a recovery group which were exposed to isopropanol for 12 months and then retained until study termination at 18 months. Survival was similar across all groups. Clinical signs were noted in the 5,000 ppm animals and included hypoactivity, lack of a startle reflex, ataxia, prostration and narcosis. Some of the animals in the 2,500 ppm group also showed hypoactivity, lack of a startle reflex and narcosis. Ataxia was the only exposure-related clinical sign that was noted for the 5,000 ppm animals following exposure. There was a concentration-related increase in body weights and body weight gain in both the 2,500 and 5,000 ppm animals (both sexes). There were no exposure-related changes in the haematological parameters at the 12- and 18-month time points. At study termination, there was a concentration-related increase in liver weights in the females, with the 5,000 ppm females being statistically significant. Nonneoplastic lesions were limited to the testes (males) and the kidney. In the testes, enlargement of the seminal vesicles occurred in the absence of associated inflammatory or degenerative changes. The kidney effects included tubular proteinosis and/or tubular dilatation. The incidence of testicular and kidney effects was not increased in the isopropanol-exposed recovery animals. The NOAEL is 500 ppm (ECHA) [Kl Score = 2].

Male and female Fischer 344 rats were exposed to 0, 500, 2,500 or 5,000 ppm isopropanol vapour 6 hours/day, 5 days/week for 24 months. The mortality rates for all male rats were 82, 83, 91 and 100% for the 0, 500, 2,500 and 5,000 ppm groups, respectively. The corresponding values for the female rats were 54, 48, 55 and 69%. The main cause of death for the 5,000 ppm rats (both sexes),

as well as for much of the mortality of the 2,500 ppm male rats, was chronic progressive nephropathy. Clinical signs were seen in the 5,000 ppm animals and included hypoactivity, lack of a startle reflex and narcosis. Some of the 2,500 ppm animals also showed a lack of a startle reflex. Body weight of the 5,000 ppm animals showed an initial decrease; from Weeks 6-72, body weights and body weight gain were increased. A similar pattern was seen in the 2,500 ppm males. Liver weights were increased in the ≥ 2,500 ppm male at 18 months, in the 2,500 ppm males at 24 months and in the 5,000 ppm females at 24 months. Kidney weights were increased in the 5,000 ppm males at 18 months and in the 5,000 ppm females at 24 months. Isopropanol exposure resulted in impaired kidney function, as indicated by various urine chemistry changes in male (2,500 and 5,000 ppm) and female (5,000 ppm) rats. Animals in these groups also exhibited histopathological effects in the kidneys which appeared to be an exacerbated form of chronic progressive nephropathy. The NOAEL is 500 ppm (ECHA) [Kl Score = 1].

Dermal

No studies are available.

F. Genotoxicity

In vitro Studies

The results of the *in vitro* genotoxicity studies on isopropanol are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Isopropanol

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (*S. typhimurium* TA98, TA100, TA1535, TA1537)	-	-	2	ECHA
Bacterial reverse mutation (*S. typhimurium* TA97, TA98, TA100, TA102, TA104, TA1535, TA1537, TA1538)	-	-	2	ECHA
Sister Chromatid Exchange (V79 cells)	-	-	2	ECHA
Mammalian cell gene mutation (CHO/HGPRT)	-	-	1	ECHA
Adenovirus (SA7) cell transformation (Syrian hamster embryo cells)	NA	-	2	ECHA

*+, positive; -, negative; NA, not applicable

In vivo Studies

Male and female ICR mice were given a single intraperitoneal injection of 0, 350, 1,173 or 2,500 mg/kg isopropanol. There were no increases in micronuclei in the bone marrow polychromatic erythrocytes at the 24, 48 or 72-hour post-dosing time points at any dose level (ECHA) [Kl Score = 1].

G. Carcinogenicity

Oral

No studies are available.

Inhalation

The carcinogenic potential of isopropanol was evaluated via inhalation using three strains of mice. Male mice were exposed to 7.5 ppm of isopropanol for 3 to 7 hours/day, 5 days/week for 5 to 8 months. Animals were killed at either 8 or 12 months. There was no significant increase in the number of lung tumours observed (ECHA) [KI Score = 3].

Male and female CD-1 mice were exposed by inhalation to 0, 500, 2,500 or 5,000 ppm isopropanol vapour for 6 hours/day, 5 days/week for 18 months. An additional group of mice (all exposure levels) were assigned to a recovery group which were exposed to isopropanol for 12 months and then retained until study termination at 18 months. There was no increased frequency of neoplastic lesions in any of the isopropanol-exposed animals (ECHA) [KI Score = 1].

Male and female Fischer 344 rats were exposed to 0, 500, 2,500 or 5,000 ppm of isopropanol vapour for 6 hours/day, 5 days/week for 24 months. The mortality rates for all male rats were 82, 83, 91 and 100% for the 0, 500, 2,500 and 5,000 ppm groups, respectively. The corresponding values for the female rats were 54, 48, 55 and 69%, respectively. The main cause of death for the 5,000 ppm rats (both sexes), as well as for much of the mortality of the 2,500 ppm male rats, was chronic progressive nephropathy. The only neoplastic lesion noted was increased interstitial (Leydig) cell adenomas in male rats. The frequency of these tumours, although elevated above the control animals, was within the historical control range of the testing facility and within the range reported for control animals from the National Toxicology Program carcinogenicity studies (ECHA) [KI Score = 1].

H. Reproductive Toxicity

In a two-generation reproductive toxicity study, Sprague–Dawley rats were dosed by oral gavage with 0, 100, 500 or 1,000 mg/kg isopropanol. There were seven parental deaths that were considered treatment-related: two high-dose F_0 females, two F_1 high-dose females, one mid-dose F_0 female, and two low-dose F_1 males. Lactation body weight gain was increased in the 500 and 1,000 mg/kg females in both generations, and liver and kidney weights were increased in the 500 and 1,000 mg/kg groups in both sexes. Centrilobular hepatocyte hypertrophy was noted in some 1,000 mg/kg F_1 males. There were some kidney effects in the 500 and 1,000 mg/kg F_0 males and in all treated F_1 male rats. The kidney effects were characterised by an increased number of hyaline droplets in the convoluted proximal tubular cells, epithelial degeneration and hyperplasia, and proteinaceous casts. Increased mortality occurred in the high-dose F_1 offspring during the early postnatal period; no other clinical signs of toxicity were observed in the offspring from either generation. Offspring body weight, however, in the 1,000 mg/kg group was reduced during the early postnatal period. There was significant mortality in the F_1 weanlings (18/70) before the selection of the F_1 adults. A statistically significant reduction was observed in the F_1 male mating index of the 1,000 mg/kg group (73 versus 97% in the controls). There were no other treatment-related effects on reproduction, including fertility and gestational indices, or histopathology of the reproductive organs. A benchmark dose level of 420 mg/kg/day was calculated (lower bound on dose associated with a 5% response rate) for the decrease in the male mating index (ECHA) [KI Score = 1].

In a one-generation reproductive/embryotoxicity study, male and female Wistar rats were given 0, 0.5, 1.0 or 2.0% isopropanol in their drinking water. The calculated intakes for males were 383, 686 and 1,107 mg/kg/day (pre-mating) and 347, 625 and 1,030 mg/kg/day (18 weeks of treatment). The calculated intakes for females were 456, 835 and 1,206 mg/kg/day (premating); 668, 1,330 and 1,902 mg/kg/day (gestation); and 1,053, 1,948 and 2,768 mg/kg/day (postpartum). An immediate, statistically significant dose-dependent decrease occurred in water intake in the male rats. Intake

was reduced ~5-14% (1% group; premating period) and ~30% (2% group; days 7-11 to end of study). Overall mean feed consumption was significantly lower in treated versus control animals. Male body weights (2% only) were reduced throughout the study. Water consumption was initially reduced in the 1% and 2% females, but the 2% group recovered to only ~70% of the control values (premating); it continued to be reduced during the gestation and lactation period. Mean maternal body weights were reduced (all treated groups) at the start of gestation, with partial recovery during the gestation period except for the 2% group. Overall weight gain during gestation in these groups were similar to the controls. Following parturition from PND 4 onward, the 2% dams had significantly lower body weights. There were no infertile males in any group, and no treatment-related effect on female fertility or on length of gestation. The number of pups/litter on GD 1 was reduced in the 2% group; because it was not replicated in the embryotoxicity portion, an increase in pup mortality during parturition or GD 0, followed by cannibalism of the dead pups by the dam was suggested. No macroscopic abnormalities were seen in females; nor was there any treatment-related histopathological changes seen in the reproductive tissue in the 2% parental animals. Absolute kidney weight and relative kidney, liver and spleen weights were increased in the 2% F_0 males; increased absolute liver and kidney weights and relative liver weights in the 2% F_0 females. In the embryotoxicity portion, there was a statistically significant increase in the total number of pre-implantation losses in the 2% animals. Whole body oedema was seen in 40% of the foetuses in 3/8 litters in the 2% group. No macroscopic abnormalities of the viscera of these foetuses were detected, and the incidence of oedema was not related to gender. In the one-generation portion, postnatal pup survival and in the average pup weight (by PND 7) were decreased in the 2% group. F_1 generation animals of both sexes showed increased relative liver weights at all dose levels, and the 2% males had higher relative kidney weights. A slight but significant decrease in absolute brain weight and increase in relative empty cecum weights in both sexes of the 2% F_1 generation group was observed. No treatment-related gross abnormalities were observed in the F_1 generation animals at necropsy. The NOAEL for reproductive toxicity is 2% in drinking water, the highest dose tested (ECHA) [KI Score = 1]. The effects of isopropanol (2.5% in drinking water) on the reproduction and growth of rats were assessed in a multigenerational study. No reproductive toxicity was observed. The NOAEL for reproductive toxicity is 2.5% isopropanol in drinking water (ECHA) [KI Score = 4].

Isopropanol was administered as a 3% solution in drinking water to Wistar rats. Reduced parental body weight gain, food, and water consumption were observed in the treated animals compared with the controls. Fertility, litter size and pup weights at postnatal days 4 and 21 were reduced in treated animals compared with the controls. In the second generation, the isopropanol concentration was reduced to 2%, and there were essentially no effects (ECHA) [KI Score = 4].

I. Developmental Toxicity

Oral Studies

Isopropanol was given at concentrations of 0, 0.5, 1.25 or 2.5% in the drinking water to female Wistar rats on GD 6 to 16. The calculated intakes of isopropanol during GD 6-16 were 596, 1,242 and 1,605 mg/kg/day. There was an immediate reduction in water intake in the 2.5% dose group, and this was statistically significant throughout the treatment period when compared to controls. A smaller reduction in water intake was also seen in the 1.25% females (statistically significant during GD 6-9), with no change in the 0.5% females. Palatability of the drinking water may have been the problem since water intake significantly increased the first day following the end of the treatment period for all dose groups. Feed consumption patterns paralleled the water consumption during and after treatment in the mid- and high-dose groups. Overall, mean body weights of the 2.5% females were lower than the controls from GD 7 to termination. Effects on weight gain in the 0.5% and 1.25% females were limited to a failure to gain weight during the first (0.5%) and second (1.25%) day of treatment. There were no treatment-related effects in post-implantation loss, mean number of

implantation sites or live foetuses. There was a slight dose-dependent decrease in mean litter weight and a significant decrease in mean foetal weight in the 1.25% and 2.5% groups. A statistically significant increase in variations was observed, indicative of a lower degree of ossification in the treated animals. There was a dose-dependent decrease in the number of foetuses with the 4th sacral arch and a dose-dependent increase in the number of foetuses with less than 2 caudal arches. The sternum also showed reduced ossification because there were increased numbers of foetuses with small, absent or incompletely ossified sternebrae. The NOAEL for maternal and developmental toxicity is 596 mg/kg/day (ECHA) [KI Score = 1].

In a rat developmental study, female Sprague–Dawley rats were dosed by oral gavage with either 0, 400, 800 or 1,200 mg/kg of isopropanol during gestational days 6 to 15. Two dams (8%) died at 1,200 mg/kg and one dam (4%) died at 800 mg/kg. At 1,200 mg/kg, maternal body weights were reduced throughout gestation (GS 0-20; 89.9% of control value), associated with reduced gravid uterine weight. There were no other treatment-related effects on the dams. Foetal body weights per litter were also significantly reduced at the 800 and 1,200 mg/kg dose levels, but there were no teratogenic effects. The NOAEL for maternal and developmental toxicity is 400 mg/kg/day, respectively (ECHA) [KI Score = 1]. In a rabbit developmental study, female New Zealand white rabbits were dosed by oral gavage with either 0, 120, 240 or 480 mg/kg of isopropanol during gestational days 6 to 18. At 480 mg/kg, isopropanol was unexpectedly toxic to pregnant female rabbits, resulting in the deaths of four does (26%). Maternal body weights were significantly reduced during treatment (gestational days 6–18) and were associated with reduced maternal food consumption during this period. Profound clinical signs were noted at 480 mg/kg and included flushed and/or warm ears, cyanosis, lethargy and laboured respiration. No adverse maternal effects were noted at 120 or 240 mg/kg. There were no developmental or teratogenic effects at any dose tested. The NOAELs for maternal and developmental toxicity are 240 and 480 mg/kg/day, respectively (ECHA) [KI Score = 1].

Isopropanol was given by oral gavage to Sprague–Dawley rats from gestational days 6 to 21 in doses of 0, 200, 700 or 1,200 mg/kg. The dams were allowed to deliver, litters were culled on postnatal day (PND) 4, pups were weaned on PND 22, and their dams were killed. Weaned pups were assessed for day of testes descent or vaginal opening, motor activity, auditory startle and active avoidance. The pups were killed on PND 68. Some of the pups were taken from each dose group and were perfused in situ for pathological examination of the central nervous system. There were no biologically significant findings in the behavioural tests, no changes in organ weights and no pathological findings of note. Thus, there was no evidence of developmental neurotoxicity from isopropanol exposure (ECHA) [KI Score = 1].

Inhalation Studies

Pregnant female Sprague Dawley rats were exposed to 0, 3,500, 7,000 or 10,000 ppm isopropanol for 7 hours/day during gestational days 1–19. The animals showed unsteady gait and narcotisation during initial exposures in the mid- and high-dose groups; reduced food consumption and reduced weight gain were also noted in both the mid- and high-dose groups. Foetal body weights per litter were reduced in all dose groups. Exposure to 10,000 ppm also resulted in failure of implantation, fully resorbed litters, increased resorptions per litter and increased incidence of cervical ribs. The NOAEL for maternal toxicity is 3,500 ppm. The LOAEL for developmental toxicity is 3,500 ppm; a NOAEL was not established (ECHA) [KI Score = 2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for isopropanol follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2021).

A. Non-cancer

Oral

The repeated-dose toxicity studies on isopropanol by the oral route are inadequate for the purposes of risk assessment. There is, however, a well-conducted two-generation reproductive toxicity study, in which rats were dosed by oral gavage up to 1,000 mg/kg/day (Bevan et al., 1995). Allen et al. (1998) calculated a benchmark dose level of 420 mg/kg/day (lower bound on dose associated with a 5% response rate for the decrease in the male mating index). The Point of Departure (POD) of 420 mg/kg/day will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 10
UF_D (database uncertainty) = 1
Oral RfD = 420/(10 x 10 x 1 x 10 x 1) = 420/1000 = 0.4 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2021)
Proportion of water consumed = 10% (ADWG, 2021)
Volume of water consumed = 2L (ADWG, 2021)
Drinking water guidance value = (0.4 x 70 x 0.1)/2 = 1.4 mg/L

B. Cancer

Isopropanol was not carcinogenic to rats or mice in chronic inhalation studies. Therefore, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Isopropanol is a flammable liquid.

Isopropanol does not exhibit the following physico-chemical properties:

- Explosivity
- Oxidising potential

VII. ENVIRONMENTAL EFFECTS SUMMARY

A. Summary

Isopropanol is of low toxicity concern to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on isopropanol.

Table 3: Acute Aquatic Toxicity Studies on Isopropanol

Test Species	Endpoint	Results	Klimisch score	Reference
Pimephales promelas	96-hour LC_{50}	9,640 mg/L	2	ECHA
Daphnia magna	24-hour EC_{50}	> 10,000 mg/L	2	ECHA

Chronic Studies

Table 4 lists the results of chronic aquatic toxicity studies on diethanolamine.

Table 4: Chronic Aquatic Toxicity Studies on Isopropanol

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Daphnia magna	16-day NOEC	141 mg/L	4	ECHA
Daphnia magna	21-day NOEC	30 mg/L	4	OECD, 1977a,b
Scenedesmus quadricauda	7-day NOEC	1,800 mg/L	2	ECHA

C. Terrestrial Toxicity

An EC_{50} value of 2,100 mg/L was determined from a lettuce seed germination test (Reynold, 1977) [Kl score = 2].

D. Calculation of PNEC

The PNEC calculations for isopropanol follow the methodology discussed in DEWHA (2009).

PNEC water

Experimental results are available for two trophic levels. Acute $E(L)C_{50}$ values are available for fish (9,640 mg/L) and invertebrates (> 10,000 mg/L). Results from chronic studies are available for

invertebrates (16- and 21-day NOECs for *Daphnia* are 141 and 30 mg/L, respectively). On the basis that the data consists of acute studies from two trophic levels and a chronic study from one trophic level, an assessment factor of 100 has been applied to the lowest reported NOEC of 30 mg/L for invertebrates. The $PNEC_{water}$ is 0.3 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 0.2 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (0.87/1280) \times 1000 \times 0.3$
$= 0.2$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [0.2 \times Kp_{sed})1000 \times BD_{solid}]$
$= 0.8 + [0.2 \times 0.14/1000 \times 2400]$
$= 0.87\ m^3/m^3$

Where:
Kp_{sed} = solid-water partition coefficient (L/kg).
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]
$Kp_{sed} = K_{oc} \times f_{oc}$
$= 3.478 \times 0.04$
$= 0.14$ L/kg

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for isopropanol calculated from EPI Suite™ using Log K_{ow} is 3.478.
f_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.014 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
$= (0.07/1500) \times 1000 \times 0.3$
$= 0.014$ mg/kg

Where:
Kp_{soil} = soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]
$Kp_{soil} = K_{oc} \times f_{oc}$
$= 3.478 \times 0.02$
$= 0.07\ m^3/m^3$

Where:

K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for isopropanol calculated from EPI Suite™ using K_{ow} is 3.478 L/kg.

f_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2008).

Isopropanol is readily biodegradable; thus, it does not meet the screening criteria for persistence.

Based on a measured log K_{ow} of 0.05 and a calculated BCF of 1, isopropanol does not meet the screening criteria for bioaccumulation.

The chronic toxicity data on isopropanol show a NOEC of > 0.1 mg/L. The acute E(L)C_{50} values for isopropanol are > 1 mg/L. Thus, isopropanol does not meet the screening criteria for toxicity.

The overall conclusion is that isopropanol is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Flammable Liquid Category 2

Eye Irritant Category 2

STOT Single Exposure Category 3 [Narcosis]

B. Labelling

Danger

C. Pictogram

X. SAFETY AND HANDLING

A. First Aid

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. Get medical attention immediately, preferably a physician for an ophthalmologic examination.

Skin Contact

For minor skin contact, avoid spreading material on unaffected skin. Remove and isolate contaminated clothing. Wash the contaminated area of body with soap and fresh water. Get medical attention. Launder contaminated clothing before reuse.

Inhalation

Move person to fresh air. If respiratory irritation, dizziness, nausea or unconsciousness occurs, seek immediate medical assistance. Give artificial respiration if victim is not breathing. Do not use mouth-to-mouth method if victim inhaled the substance; give artificial respiration with the air of a pocket mask equipped with a one-way valve or other proper respiratory medical device.

Ingestion

Do not induce vomiting. Get medical attention immediately.

Notes to Physician

If ingested, material may be aspirated into the lungs and cause chemical pneumonitis. Treat appropriately.

B. Fire Fighting Information

Extinguishing Media

Use water spray or fog, foam, dry chemical or carbon dioxide. Do not use straight streams of water.

Specific Exposure Hazards

Highly flammable. Vapours are flammable and heavier than air. Vapours may travel across the ground and reach remote ignition sources causing a flashback fire danger. Emits toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon dioxide, carbon monoxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Isolate area. Keep unnecessary and unprotected personnel from entering the area. Use personal protective clothing. Ensure adequate ventilation. Wear respiratory protection if ventilation is inadequate. Do not breath mist, vapours or spray. Avoid contact with skin, eyes and clothing. Eliminate all sources of ignition.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

Eliminate all sources of ignition. All equipment used when handling the material must be grounded. A vapour suppressing foam may be used to reduce vapours. Use clean non-sparking tools to collect absorbed material. Pick up with suitable absorbent material and transfer to a container for chemical waste. For large amounts: dike spillage and pump off product into container for chemical waste. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Prevent exposure to ignition sources (i.e., use non-sparking tools and explosion-proof equipment). Avoid contact with eyes, skin and clothing. Avoid breathing vapour. Wash thoroughly after handling. Keep container closed. Use with adequate ventilation. Use proper bonding and/or ground procedures. However, bonding and grounds may not eliminate the hazard from static accumulation. Peroxides may form upon prolonged storage. Exposure to light, heat or air significantly increases peroxide formation. If evaporated to a residue, the mixture of peroxides residue and material vapour may explode when exposed to heat or shock.

Storage

Keep container tightly closed. Store in a cool, well-ventilated area away from heat and light. Storage containers should be grounded and bonded. Fixed storage containers, transfer containers and associated equipment should be grounded and bonded to prevent accumulation of static charge. See SDS for suitable materials and coatings.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace exposure standard for isopropanol in Australia is 400 ppm as an 8-hour TWA and 500 ppm as a 15-min STEL.

Engineering Controls

Good general ventilation should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation or other engineering controls to

maintain airborne levels below recommended exposure limits. If exposure limits have not been established, maintain airborne levels to an acceptable level.

Personal Protection Equipment

Respiratory Protection: If workers are exposed to concentrations above the exposure limit, they must use appropriate, certified respirators. If there are no applicable exposure limit requirements or guidelines, use an approved respirator. Selection of air-purifying or positive pressure supplied-air will depend on the specific operation and the potential airborne concentration of the product. For emergency conditions, use an approved positive-pressure self-contained breathing apparatus. The following should be effective types of air-purifying respirators: organic vapour cartridge with a particulate pre-filter.

Hand Protection: Use gloves chemically resistant to this material. Consult the SDS for appropriate glove barrier materials.

Skin Protection: Use protective clothing chemically resistant to the material. Selection of specific items such as face shield, boots, apron or full body suit will depend on the task.

Eye Protection: Use chemical goggles.

Other Precautions: Wash hands, forearms and face thoroughly after handling chemical products; before eating, smoking and using the lavatory; and at the end of the working period. Appropriate techniques should be used to remove potentially contaminated clothing. Wash contaminated clothing before reusing. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

UN 1219 (Isopropanol)

Class 3

Packing Group II

XI. DISPOSAL

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed

XIII. REFERENCES

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MAGNESIUM SILICATE HYDRATE (TALC)

This dossier on magnesium silicate hydrate (talc) presents the most critical studies pertinent to the risk assessment of this substance in its use in coal seam gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed magnesium silicate hydrate (talc) in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): dioxosilane; oxomagnesium; hydrate

CAS RN: 14807-96-6

Molecular formula: $H_2Mg_3O_{12}Si_4$

Molecular weight: 379.27 g/mol

Synonyms: Talcum, oxosilanediol, trimagnesium; dioxido(oxo)silane; hydroxy-oxido-oxosilane, dioxosilane; oxomagnesium; hydrate

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-chemical Properties of Magnesium Silicate Hydrate (Talc)

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	White solid odorless powder	2	ECHA
Melting Point	1,500°C @ 101.3 kPa	2	ECHA
Boiling Point	This substance is a solid that melts above 300°C	-	-
Density	2700 kg/m³ @ 20°C	2	ECHA
Vapour Pressure	0 Pa at 25°C	2	ECHA
Partition Coefficient (log K_{ow})	-9.4 @ 25°C	2	ECHA
Water Solubility	0.0001 g/L @ 25°C; insoluble in water	2	ECHA
Flash Point	ND	-	-
Auto flammability	ND	-	-
Viscosity	Not applicable as substance is a solid.	2	ECHA
Dissociation constant	ND because the substance is insoluble in water	-	ECHA

ND – not determined

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Magnesium silicate hydrate (talc) is an inorganic substance for which biodegradation is irrelevant. Moreover, it will not bioaccumulate and has a low potential to adsorb to soil.

B. Biodegradation

As an inorganic substance, magnesium silicate hydrate (talc) will not biodegrade. Soil and sediment degradation studies are not considered to be applicable as the test material is essentially insoluble in water and consists of materials which occur naturally in these compartments (ECHA).

C. Environmental Distribution

Magnesium silicate hydrate (talc) is insoluble in water. The log K_{OC} of was estimated to be 1.5027 which is equal to a K_{OC} value of 31.82 L/kg using the KOCWIN v2.00 QSAR method (ECHA). Based on this K_{oc} value, if released to soil, magnesium silicate hydrate (talc) is expected to have a low potential for adsorption. If released into water, the substance has a low potential for adsorption to sediment or suspended solids.

D. Bioaccumulation

There is no potential for bioaccumulation. Due to its inherent chemical-physical properties, such as absence of lipophilicity as well as the capability of the organism to excrete absorbed SiO_2 components, bioaccumulation can be disregarded. Magnesium is widespread in living cells and does not bioconcentrate in aquatic organisms (ECHA).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Talc is a mineral composed of hydrated magnesium silicate. Talc is essentially non-toxic by the oral and dermal routes. Talc is non-irritating to the eyes and skin. There was no toxicity or carcinogenic effects in rats. Talc is not genotoxic. No developmental toxicity was reported in pregnant female rats, mice or rabbits given oral doses of talc.

B. Basic Toxicokinetics

Inhalation

To determine the deposition, distribution and clearance of talc, 44 female Syrian golden hamsters received a single 2-hour nose-only exposure to a neutron-activated talc aerosol and sub-groups of 4 animals were then killed at 11 different intervals from 15 minutes to 132 days after exposure.

The talc tested was a commercial baby powder. Nine unexposed control animals were used; four were killed on the day the test animals were exposed and five were killed on the final day of the study. The aerosol exposure system had 7 tiers of exposure ports, and the talc aerosol was passed through a cyclone elutriator to remove particles that were larger than ~10 µm in diameter; the activity median aerodynamic diameter was 6.4-6.9 µm. The mean aerosol concentration was 40 and 75 µg/L at the 15 to 30 and 60 to 90-minute sampling periods, respectively. In the presentation of the results, the γ-ray counts from the controls were expressed as µg talc equivalent, and the γ-ray counts of the exposed animals were not corrected for control values.

Variations among animals killed at the same time were attributed to variations in aerosol concentration at different tiers. The mean pulmonary talc content in the lungs of test animals at various time intervals was 33.08 µg (15 minutes after exposure), 24.08 µg (100 minutes), 42.70 µg (4 hours), 18.75 µg (21 hours), 21.30 µg (2 days), 21.03 µg (after 4 days), 13.85 µg (after 8 days) and 8.95 µg (after 18 days); the mean for the Day 0 control animals was 1.78 µg. The biological half-life of the talc deposited in the lungs was 7 to 10 days. At the time of termination of the final group, i.e., 132 days, there was no statistically significant difference in the talc burden of the lungs of test (3.70 µg) and control (2.30 µg) animals. The amount of talc in the liver, kidneys and lungs was also determined; the only statistically significant differences compared to controls in any of these organs were found in the liver. There was a decrease at 4 hours compared to day 0 controls, an increase at Day 36 compared to both Day 0 and Day 132 controls, and an increase on Day 68 compared to Day 132 controls.

Analysis of the data using the Kruskal-Wallis test showed that there were no significant differences among the mean talc burden values for the liver, kidneys and ovaries, including the control values, and that there was no significant trend, indicating there was no translocation of talc to these tissues.

As noted, no translocation from the respiratory tract to other tissues was found in this study, and the clearance of talc from the lungs was complete within 4 months after exposure.

Oral

In one study, six female Syrian golden hamsters (outbred Ela:ENG strain) were dosed by gavage with 1 mL neutron-activated talc suspended in physiological saline containing 0.6% (w/w) 1% methyl cellulose, and the animals were killed 24 hours after dosing. The talc used was a commercial baby powder.

Four hamsters were dosed similarly with a non-irradiated talc solution. The neutron-activated talc was exposed to an integrated neutron flux of 7 x 1,016 n/cm^2 30 days prior to dosing. The skinned carcass, gastrointestinal (GI) tract, lungs, liver, kidneys and excreta were analysed for isotopes 60 Co and 46 Sc by gamma-ray spectrometry, and the gamma-ray counts were compared with those of four hamsters that were not dosed with talc.

The γ-ray counts of the tissue and excreta of the dose animals were equivalent to a total of 2.94 mg talc. Based on γ-ray counts, 74.5% of the neutron-activated talc was recovered in the faeces and 23.5% was recovered in the GI tract, while 1.91% was recovered in the skinned carcass, 0.09% in the urine, 0.04% in the kidneys and 0.02% in the liver. The amount found in the urine of the hamsters given irradiated talc was statistically significantly increased compared to the controls. No talc was recovered in the lungs (ECHA) [KI score = 2].

In a second oral study, four LACA female mice were given a single oral dose of 40 mg/kg [3H] talc. Two mice were killed at 6 hours and two at 24 hours after dosing. In the mice killed 6 hours after dosing, 95 and 96% of the radioactivity was recovered in the large intestines and faeces, 9 and 7% was recovered in the small intestines and stomach, and 0.7 and 0% in the urine of each mouse. In the two mice killed 24 hours after dosing, 99 and 101% of the radioactivity was recovered in the large intestines and faeces, 4 and 6% was recovered in the small intestines and stomach, and 1.3 and 1.5% in the urine of each mouse. Less than 0.005% of the radioactivity was found in the carcass of any of the mice (ECHA) [KI score = 2].

In a third oral study, three male Wistar albino rats were given a single oral dose and three rats were given six daily oral doses by gavage of 50 mg/kg body wt [3H] talc. After the last dose, urine and

faeces were collected every 24 hours for 4 days and on Day 10; the rats were then killed. Within 24 hours after administration of the single dose, approximately 75% of the radioactivity was recovered in the faeces and only 1% was recovered in the urine. After 96 hours, a total of 95.8% of the dose was excreted in the faeces and 1.7% in the urine, with a total excretion of 97.5% of the dose. No radioactivity was recovered in the liver or kidneys 10 days after a single dose of talc. On Day 10 in the rats given six daily doses of [3H] talc, there was no radioactivity found in the faeces or livers, and there was a trace of radioactivity (< 0.02%) in the kidneys of these rats (ECHA) [KI score = 2].

C. Acute Toxicity

Oral

A single oral dose of 5,000 mg/kg of talc prepared as an 18.3% (w/v) suspension in saline was administered to 10 male rats. All animals survived, and there were no signs of toxicity. In conclusion, the median lethal dose of Talc (Mg3H2(SiO3)4) after a single oral administration to male rats, observed over a period of 14 days is: LD50 > 5,000 mg/kg body weight (ECHA) [KI Score = 2].

Inhalation

Groups of 5 male and female Wistar rats were treated with magnesium hydroxide as aerosol during 4 hours. No mortality or other relevant adverse effects were observed. An inhalatory LC_{50} (4-hour) value for magnesium hydroxide exceeding 2.1 mg/L was determined, being the maximum feasible concentration that could be tested (ECHA) [KI Score = 2].

Dermal

An OECD Guideline 402 (Acute Dermal Toxicity) was performed. Five males and five female Wistar rats were dermally exposed to a single talc dose of 2,000 mg/kg.

Approximately 24 hours before the test, the fur was removed from the dorsal area of the trunk using an electric clipper. Care was taken to avoid abrading the skin, and only animals with healthy intact skin were used. No less than 10% of the body surface was cleared for the application.

The test item was applied at a single dose, uniformly over an area which was approximately 10% of the total body surface. The test item was held in contact with the skin throughout a 24-hour period. At the end of the exposure period the residual test item was not removed.

Under the conditions of this study, single dermal application of the test item magnesium chloride hexahydrate to rats at a dose of 2,000 mg/kg body weight was associated with no mortality. The dermal LD_{50} was determined to be > 2,000 mg magnesium chloride hexahydrate/kg body weight (ECHA) [KI Score = 2].

Dermal

No studies were available.

D. Irritation

Skin

An *in vitro* skin irritation test was carried out with the reconstituted three-dimensional human skin model EPISKIN-SM™ (Skinethic). This skin model consists of normal (non-cancerous), adult human-

derived epidermal keratinocytes (NHEK) which have been cultured to form a multilayered, highly differentiated model of the human epidermis. The NHEK are cultured on chemically modified, collagen-coated cell culture inserts. A highly differentiated and stratified epidermis model is obtained after a 13-day culture period and is comprised of the main basal, supra basal, spinous and granular layers and a functional stratum corneum.

The test item showed no irritant effects. The mean relative tissue viability (% negative control) was ≥ 50% (112.9%) after 15-minute treatment and 42-hour post incubation. The controls confirmed the validity of the study. The mean OD550 of the three negative control tissues was ≥ 0.6. The mean relative tissue viability (% negative control) of the positive control was ≤ 30% (22.6%). The standard deviation of replicate tissues of all dose groups was ≤ 30% (1.4% - 9.4%). It can be concluded that talc is non-irritating to skin (ECHA) [KI Score = 2].

Eye

An OECD Guideline 405 (Acute Eye Irritation / Corrosion) study was performed using magnesium chloride hexahydrate as a surrogate substance for talc. A dose of 0.1 g of the test item was applied at a single dose in the conjunctival sac of one eye of each test animal after pulling the lower lid away from the eyeball. The lids were then gently held together for about 1 second in order to prevent loss of the material. The untreated contralateral eye served as control. Observations of the eye were made at 1, 24, 48 and 72 hours and 4 to 6 days.

Under the conditions of the study, single ocular instillation of the test item magnesium chloride hexahydrate to rabbits at a dose of 0.1 g produced irritant effects, which were fully reversible. Neither mortalities nor significant clinical signs of toxicity were observed. The test item is deemed to be non-irritating to eyes (ECHA) [KI Score = 2].

E. Sensitisation

No experimental data are available on the Talc ($Mg_3H_2(SiO_3)_4$) powder and silicates; however, there is long experience in humans. Data collected from industrial hygiene surveillance over the last 50 years do not indicate any potential for skin sensitisation. Despite the widespread cosmetic use of talc and special studies in volunteers (BIBRA, 1991) there are no indications of any allergenic effect (ECHA) [KI score = 3].

F. Repeated Dose Toxicity

Oral

A study equivalent or similar to OECD Guideline 452 (Chronic Toxicity Studies) was performed using male and female Wistar rats. Wistar rats (16 male and 16 female) were exposed to talc in feed which resulted in an amount taken up of 100 mg/kg/day. After feeding had been carried out for 101 days, the animals were observed until death and subsequently examined histopathologically.

One of the animals treated with talc showed a leiomyosarcoma of the stomach. Sarcomas, which were not associated with the talc treatment, were found in the uterus of two animals. No chronic pathological effect was associated with oral administration of talc over 5 months. No adverse effects were seen on general toxicity endpoints. Under the condition of this study, for a period of 101 days for male and female rats, the NOAEL of talc in a feeding study was 100 mg/kg/day (ECHA) [KI score = 2].

Inhalation

A study equivalent or similar to OECD Guideline 452 (Chronic Toxicity Studies) was performed using male and female Wistar rats. The Wistar rats (12 male and 12 female) were exposed whole body to aerosolised talc at a mean respirable dust concentration of 10.8 mg/m^3 for 7.5 hours per day, 5 days a week for 6 or 12 months.

Ten days after the end of each exposure period, 6 rats per group were killed; 12 rats per group died and 2 rats per group were unaccounted for. The remaining 4 rats per group were killed one year after the end of the exposure period. Minimal fibrosis was observed. Talc exposure led to distinct fibrosis that was comparable with that after exposure to chrysotile in the parallel group. A lung adenoma was detected in 1 of 24 animals treated with talc. In rats exposed by inhalation to 10.8 mg/m^3 Italian talc (grade 00000; ready milled; mean particle size, 25 µm) for 3 months, minimal fibrosis was observed, the degree of which did not change during the observation period after exposure. Animals that were exposed for 1 year had minimal to slight fibrosis, the degree of which had increased to moderate within 1 year after cessation of exposure.

A no observed adverse effect concentration (NOAEC) of 10.8 mg/m^3 was determined (ECHA) [KI Score = 2].

Dermal

No adequate studies for human health risk assessment are available.

G. Genotoxicity

In Vitro Studies

The *in vitro* genotoxicity studies on talc are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Talc

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Mammalian cell gene mutation (rat pleural mesothelial cells (RPMC)).	-*	ND	2	ECHA

*+, positive; -, negative
ND – not determined

Talc did not cause a statistically significant increase in sister chromatid exchanges (SCEs) and was not clastogenic. The test substance is non-mutagenic under the given experimental conditions (ECHA) [KI Score = 2].

In Vivo Studies

A study equivalent or similar to OECD Guideline 478 (Genetic Toxicology: Rodent Dominant Lethal Test) was performed per a rat dominant lethal assay on Sprague Dawley rats. Groups of 10 male rats were dosed by gavage with a single dose or once daily for 5 days with 30, 300, 3,000 or 5,000 mg/kg talc.

There were no dose-response or time trend patterns; talc did not induce dominant lethal mutations in this assay. Therefore, talc was not genotoxic in a rat dominant lethal assay (ECHA) [KI Score = 2].

H. Carcinogenicity

Oral

An OECD Guideline 453 (Combined Chronic Toxicity / Carcinogenicity Studies) was performed. In a feeding study of 16 male and 16 female Wistar rats, talc was added to the diet; this resulted in a dosage rate of 100 mg/kg/day. After feeding had been carried out for 101 days, the animals were observed until death (approximately 614 days) and subsequently examined histopathologically. One of the animals treated with talc showed a leiomyosarcoma of the stomach. Sarcomas, which were not associated with the talc treatment, were found in the uterus of two animals.

However, no differences in tumour incidence were noted between treated animals and 8 male and 8 female control animals fed basal diet throughout (average survival, 641 days).

Inhalation

In a lifetime experiment, three groups of 50 male and 50 female Syrian golden hamsters, 4 weeks of age, were exposed (whole body) by inhalation to an aerosol of talc baby powder that was prepared from Vermont talc by flotation (95% w/w platy talc with trace quantities of magnesite, dolomite, chlorite and rutile) for 3, 30 or 150 minutes per day, 5 days a week for 30 days. The mean aerosol concentration was 37.1 mg/m^3, with a measurable respiratory fraction of 9.8 mg/m^3 and a MMAD of 4.9 µm. A placebo exposed group comprised 25 males and 25 females. Two further groups of hamsters, 7 weeks of age, were exposed to talc aerosol for 30 or 150 minutes per day for 300 days. The mean aerosol concentration was 27.4 mg/m^3, with a measurable respiratory fraction of 8.1 mg/m^3 and a MMAD of 6.0 µm. Another placebo-exposed group comprised 25 males and 25 females. The survivors of the last two talc-exposed groups were killed at the age of 20 months.

No clinical signs of toxicity to talc were observed. The type, incidence and severity of lesions indicated no trend toward a dose-response and no statistically significant differences between exposed and control groups. The incidence of focal alveolar cell hyperplasia (25% in treated groups; 10% in controls) appeared to be affected by treatment, but a two-way weighted analysis showed no significant association. Thus, exposure of hamsters to talc via inhalation did not produce carcinogenic effects (ECHA) [Kl Score = 2].

I. Reproductive Toxicity

An OECD Guideline 416 (Two-Generation Reproduction Toxicity Study) was performed. Groups of 12-15 gravid Dutch-belted female rabbits were dosed orally with 9, 42, 195 or 900 mg/kg bw talc in corn oil on Days 6-18 of gestation. Eight gravid negative controls were given only vehicle and nine gravid positive controls were dosed with 2.5 mg/kg bw of 6-aminonicotinamide on Day 9 of gestation. The dams were killed on Day 29 of gestation. A total of 1/8, 4/15, 2/12, 5/15 and 2/13 dams of the negative control, 9, 42, 195 and 900 mg/kg bw dose groups, respectively, died or aborted before Day 29 of gestation, and the number of live litters for these groups was 6/7, 10/11, 8/10, 10/10 and 7/11, respectively. Details on Results (PO): Administration of up to 900 mg/kg bw talc on Days 6-18 of gestation had no discernible effect on nidation or on maternal survival.

The number of abnormalities did not differ between test and control animals.

Details on Results (F1): Administration of up to 900 mg/kg bw talc on days 6-18 of gestation had no discernible effect on nidation or on foetal survival. The number of abnormalities did not differ between test and control animals.

The NOAEL was considered to be 900 mg/kg bw/day for reproduction toxicity study. A NOAEL of > 900 mg/kg/day was determined for reproduction (ECHA) [KI Score = 2].

J. Developmental Toxicity

A GLP compliant study was performed. Groups of 20-22 gravid albino CD-1 mice and groups of 20-24 gravid Wistar rats were dosed by gavage with 0, 16, 74, 350 or 1,600 mg/kg bw talc as an anhydrous corn oil suspension on days 6-15 of gestation. The mice were killed on Day 17 and the rats on Day 20 of gestation and the number of implantation sites, resorptions sites, and live and dead foetuses, and the live pup body weights were recorded.

Maternal Toxicity: The administration of up to 1,600 mg/kg bw talc in corn oil had no effect on maternal endpoints.

Embryotoxic / Teratogenic Effects: The administration of up to 1,600 mg/kg bw talc in corn oil had no effect on developmental parameters and had no effect on foetal survival.

The NOAEL was considered to be 1,600 mg/kg bw/day for developmental toxicity (ECHA) [KI score = 2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for talc follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2021).

A. Non-Cancer

<u>Oral</u>

The NOAEL of 100 mg/kg/day from a chronic feeding study in rats was used to determine the oral RfD and drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1
Oral RfD = 100/(10 x 10 x 1 x 1 x 1) = 100/100 = 1 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2021)
Proportion of water consumed = 10% (ADWG, 2021)
Volume of water consumed = 2L (ADWG, 2021)
Drinking water guidance value = (1 x 70 x 0.1)/2 = 3.5 mg/L

B. Cancer

The carcinogenicity studies suggest talc is not a carcinogen. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Talc does not exhibit the following physico-chemical properties:

- Flammability
- Explosivity
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Talc is of low toxicity concern to aquatic organisms.

B. Aquatic Toxicity

Table 3 lists the results of the acute aquatic toxicity studies on magnesium silicate hydrate (talc).

Table 3: Acute Aquatic Toxicity Studies on Talc

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Fish (species unnamed)	96-hour LC_{50}	89,581 mg/L (QSAR)	2	ECHA
Daphnid	48-hour LC_{50}	36,812 mg/L (QSAR)	2	ECHA
Algae (species unnamed)	96-hour LC_{50}	7,203 mg/L	1	ECHA

Chronic Studies

No data are available. Short term aquatic toxicity tests reported in the literature on fish (LC_{50} *Brachydanio rerio* (Zebra fish) >100,000 mg/L/24 hr; for talc) show this substance is not toxic to aquatic life. On this basis the need for long term aquatic testing is waived (ECHA).

C. Terrestrial Toxicity

No data are available.

D. Calculation of PNEC

PNEC calculations for talc follow the methodology discussed in DEWHA (2009).

PNEC water

Acute experimental results are available for three trophic levels. Acute E(L)C_{50} values are available for fish (89,581 mg/L), *Daphnia* (36,812 mg/L), and algae (7,203 mg/L). By applying an assessment factor of 100 to the lowest E(L)C_{50} value of 7,203 mg/L from the acute studies, the $PNEC_{water}$ for talc is 72 mg/L.

PNEC sediment

There are no toxicity data for sediment-dwelling organisms. Moreover, the low K_{ow} indicates that talc is not expected to partition to sediments. Therefore, a $PNEC_{sed}$ was not calculated.

PNEC soil

There are no toxicity data for terrestrial or soil organisms. Moreover, talc is biodegradable and due to its low K_{ow}, is not expected to partition to soil. Therefore, a $PNEC_{soil}$ was not calculated.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2008).

Magnesium silicate hydrate (talc) is an inorganic substance and thus, biodegradation is not relevant. For the purposes of this PBT assessment, the persistent criteria are not considered applicable for this substance.

No data are available on bioaccumulation. However, based on the low log K_{ow}, and the inherent chemical-physical properties of magnesium silicate hydrate (talc), bioaccumulation is not expected. Thus, magnesium silicate hydrate (talc) does not meet the screening criteria for bioaccumulation.

Chronic aquatic toxicity data is not available. The E(L)C_{50} values from the acute aquatic toxicity studies on magnesium silicate hydrate (talc) are > 1 mg/L. Thus, magnesium silicate hydrate (talc) does not meet the criteria for toxicity.

Therefore, magnesium silicate hydrate (talc) is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H332- Harmful if inhaled.

B. Labelling

Warning

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. Get medical attention immediately, preferably a physician for an ophthalmologic examination.

Skin Contact

For minor skin contact, avoid spreading material on unaffected skin. Remove and isolate contaminated clothing. Wash the contaminated area of body with soap and fresh water. Get medical attention. Launder contaminated clothing before reuse.

Inhalation

Move person to fresh air. Administer oxygen if breathing is difficult. Do not use mouth-to-mouth method if victim inhaled the substance; give artificial respiration with the air of a pocket mask equipped with a one-way valve or other proper respiratory medical device. Give artificial respiration if victim is not breathing. Get medical attention immediately.

Ingestion

Do not induce vomiting. Rinse out mouth then drink plenty of water. Get medical attention.

Notes to Physician

All treatments should be based on observed signs and symptoms of distress in the patient.

B. Fire Fighting Information

Extinguishing Media

Use water spray or fog, foam, dry chemical or carbon dioxide.

Specific Exposure Hazards

Magnesium oxide, silicon oxides.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Isolate area. Keep unnecessary and unprotected personnel from entering the area. Use personal protective clothing. Ensure adequate ventilation. Wear respiratory protection if ventilation is inadequate. Avoid dust formation. Avoid breathing vapours, mist of gas. Avoid contact with skin, eyes and clothing. Eliminate all sources of ignition.

Environmental Precautions

No specific environmental precautions required.

Steps to be Taken if Material is Released or Spilled

Eliminate all sources of ignition. Pick up with suitable absorbent material and transfer to a container for chemical waste. For large amounts: dike spillage and pump off product into container for chemical waste. Dispose of contaminated material as prescribed.

D. Storage and Handling

General Handling

Keep away from heat, sparks and flame. Avoid contact with eyes, skin and clothing. Avoid breathing vapour. Wash thoroughly after handling. Keep container closed. Use with adequate ventilation.

Storage

Keep container tightly closed. Store away from heat and light. Store in cool place. Keep container tightly closed in a dry and well-ventilated place.

E. Exposure Controls/Personal Protection

Occupational Exposure Standards

Workplace Australia has established an occupational exposure standard for exposure to talc of an 8 hour time weighed average (TWA) exposure limit of 2.5 mg/m^3 (containing no asbestos fibres).

Engineering Controls

Good general ventilation should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation or other engineering controls to maintain airborne levels below recommended exposure limits. If exposure limits have not been established, maintain airborne levels to an acceptable level.

Personal Protection Equipment

Respiratory Protection: If workers are exposed to concentrations above the exposure limit, they must use appropriate, certified respirators. If there are no applicable exposure limit requirements or guidelines, use an approved respirator. Selection of air-purifying or positive pressure supplied-air will depend on the specific operation and the potential airborne concentration of the product. For emergency conditions, use an approved positive-pressure self-contained breathing apparatus. The following should be effective types of air-purifying respirators: organic vapour cartridge with a particulate pre-filter.

Hand Protection: Use gloves chemically resistant to this material. Consult the SDS for appropriate glove barrier materials.

Skin Protection: Use protective clothing chemically resistant to this material. Selection of specific items such as face shield, boots, apron or full body suit will depend on the task.

Eye Protection: Use chemical goggles.

Other Precautions: Wash hands, forearms and face thoroughly after handling chemical products, as well as before eating, smoking and using the lavatory and at the end of the working period. Appropriate techniques should be used to remove potentially contaminated clothing. Wash contaminated clothing before reusing. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Talc is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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MANNANASE

This dossier on mannanase presents the most critical studies pertinent to the risk assessment of mannanase in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA) and a 2005 human and environmental risk assessment (HERA) report for the surrogate chemical α-amylase. Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

For the purpose of this dossier, α-amylase (CAS RN 9000-90-2) and cellulase (CAS RN 9012-54-8) enzymes have been reviewed as surrogates for mannanase, where appropriate.

NICNAS has assessed mannanase in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment[1]

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): mannan endo-1,4-beta-mannosidase IUBMB 3.2.1.78

CAS RN: 37288-54-3

Molecular formula: Not applicable, unknown or variable composition complex reaction products and biological material (UVCB)

Molecular weight: Not applicable, UVCB

Synonyms: Mannanase; beta-mannanase; endo-B-1,4-mannanase

SMILES: Not applicable, UVCB

II. PHYSICO-CHEMICAL PROPERTIES

Enzymes and other proteins are polymers built of different combinations of the 20 common amino acids. The sequence and length of the amino acids in the polymer differ between enzymes, and this determines the 3-dimensional structure, the activity and specificity of the enzyme. The physico-chemical characteristics of enzymes are mainly dependent on the amino acids building the enzyme. Since all enzymes are built up of a combination of the same 20 common amino acids, the physical and chemical characteristics will be very similar across different enzymes.

The majority of the physico-chemical characteristics are not relevant for enzymes e.g., boiling point, therefore, only relevant parameters have been determined and are summarised for mannanase in Table 1.

[1] https://www.industrialchemicals.gov.au/chemical-information/search-assessments?assessmentcasnumber=37288-54-3

Table 1: Overview of the Physico-chemical Properties of mannanase

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Not applicable	-	-
Melting Point	Not applicable	-	-
Boiling Point	Not applicable	-	-
Density	1320 -1420 kg/m^3 (relative density 1.37 ± 0.05) @ 20°C	1	ECHA
Vapour Pressure	0.004 Pa @ 25°C	1	ECHA
Partition Coefficient (log K_{ow})	-1.3 @ 20°C	1	ECHA
Water Solubility	125 g/L @ 25°C	1	ECHA
Flash Point	Not applicable	-	-
Auto flammability	Not applicable	-	-
Viscosity	Not applicable	-	-
Henry's Law Constant	Not applicable	-	-

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Mannanase is readily biodegradable, it has a low octanol-water partition coefficient, and it is highly soluble in water. Mannanase will not absorb to sediment or soil, and it is not expected to bioaccumulate in aquatic or terrestrial organisms (ECHA).

B. Biodegradation

The biodegradability of mannanase was evaluated in a 28-day modified sturm test as per OECD guideline 301 B (readily biodegradability: CO2 evolution test). Mannanase was introduced to the test system at 20 mg Dissolved Organic Carbon (DOC)/L. Mannanase showed the greater part of biodegradation between Day 2 and 7, increasing from 14.0 to 55.1%. Mannanase reached 38.4% within 5 days and 63.0% biodegradation within 9 days. On Day 28, mannanase achieved a total cumulative biodegradation of 73.7 which indicates that mannanase is readily biodegradable under the conditions of this test (ECHA) [KI. score =1].

In two additional studies, two different amylase enzymes were considered readily biodegradable based on the results of OECD 301E tests. In both tests, there was 99% DOC removal of the enzyme after 28 days. Likewise, mannanase is expected to be readily biodegradable (HERA 2005).

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

Because all enzymes are built up of the combination of the same 20 common amino acids, the physical and chemical characteristics are very similar for different enzymes, and hence, read-across from other enzymes should be fully applicable. The log octanol water partition coefficient (K_{ow}) value

of mannanase has not been determined but other enzymes have been analysed and the $LogK_{ow}$ from literature studies was found to be between -3.1 to -2.95. Due to the similar nature of enzymes, this value can also be extrapolated to mannanase (ECHA) [KI. Score = 1]. In addition, the organic carbon partition coefficient (K_{oc}) for similar enzyme glucoamylase was measured <= 1.3 L/kg at 20°C (HERA, 2005).

Based on these values and its high water solubility (125 g/L), mannanase has a low potential to adsorb to sediment or soil (ECHA).

D. Bioaccumulation

Mannanase is not expected bioaccumulate because it is highly soluble in water and a low potential to cross biological membranes (ECHA).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Mannanase has low acute toxicity, it is degraded in the gastrointestinal tract, and it is not readily absorbed by in the respiratory tract. Mannanase is not irritating to the skin, or the eye and it is not expected to be a skin sensitiser based on studies using a surrogate chemical. Mannanase is not genotoxic and there are no carcinogenic studies available to determine if this substance is a carcinogen. Mannanase is not a reproductive toxicant. No developmental toxicity studies were available.

B. Metabolism

Toxicokinetic studies performed on enzymes and UVCBs are very limited, but toxicokinetic information can be derived from the structure of enzymes combined with knowledge available for proteins in general since enzymes are proteins with catalytic activity.

Skin absorption of enzymes is at a toxicologically insignificant level. The enzymes are degraded in the gastrointestinal tract and are absorbed at a very low extent through the respiratory tract; therefore, the total bioavailability of enzymes can be concluded to be extremely low. This is further supported by the physico-chemical data. Enzymes have a low octanol water partition coefficient value, which indicates that they have no bioaccumulation potential. Also, they are expected to be readily biodegraded. Systemic exposure following enzyme exposure due to occupational or consumer exposure levels is not expected to be of toxicological significance (ECHA).

Given the relatively low absorption of enzymes, metabolism and distribution are not expected to be a relevant pathway (ECHA).

C. Acute Toxicity

<u>Oral</u>

As per EU Method B.1 (Acute Toxicity Oral), male and female Sprague-Dawley rats were exposed to 3.32 g/kg or 10 ml/kg bw mannanase by oral gavage. The only clinical sign observed was piloerect coats in all the rats within two minutes after the first dose and throughout the remainder of the first treatment day. No adverse clinical signs were observed on the second day and the overall body weight during the study was considered to be normal. There were no post-mortem abnormalities reported in this study. Therefore, the LD_{50} was reported to be >3.32 g/kg bw (ECHA) [KI. score =1].

No deaths were observed when male and female rats were given an aqueous suspension of 0, 4,000, or 10,000 mg/kg ofα-amylase ("salt free" batch PPY 1316, enzyme derived from B.subtilis by oral gavage. The actual enzyme content of this batch was 239 mg active enzyme protein (aep)/g (HERA, 2005).

In another study, male and female rats were given an aqueous suspension of 0 or 5,000mg/kg α-amylase preparation derived from B.licheniformis, the actual enzyme content of the preparation was 60.13 mg aep/g. There were no deaths (HERA, 2005).

Rats were exposed by inhalation to either 1.6 mg/L of a production α-amylase (from B. subtilis) batch ADTA202-204, a mixture of two batches prepared by the standard production process (45.9% of particles <4.7 µm) or 1.08 mg/L of a "salt-free" α-amylase (from B. subtilis) batch PPY1316, prepared from production batch ADTA202-204 by removal of NaCl (33.3% of particles <4.7 µm), for 4 hours. An air-exposed control group was also included. The actual amount of enzyme protein in the test aerosols was 0.114 mg aep/L (production batch) and 0.258 mg aep/L (salt-free batch). There were no deaths occurred (HERA, 2005).

In another study, rats were exposed to 1.6 mg/L α-amylase (from B. subtilis) preparation (highest concentration attainable) derived from a genetically modified strain of B. subtilis for 4 hours. Total organic solids comprised 83.3% of the test substance (active and inactive enzyme as well as other organic material). There were no deaths (HERA, 2005).

Inhalation

Male and female Sprague-Dawley rats were exposed to 0.45 mg mannanase concentrated dry matter/ L by inhalation of aerosol droplets through the nose for four hours. The particle size distribution was 86% respirable with an aerodynamic diameter of < 7 µm. There were no unscheduled deaths or evidence of a toxic response in this study. Therefore, the LC_{50} was reported to be > 0.45 mg/L air (analytical) (ECHA) [KI. score =1].

Dermal

Acute dermal toxicity studies were not conducted for mannanase. Due to the physicochemical and toxicological properties, the potential of absorption through the skin is expected to be very low (ECHA).

D. Irritation

Skin

α-Amylase (from B. subtilis) has a low potential for irritation to the skin and eyes of rabbits (HERA,2005).

In a human repeat insult patch test (HRIPT), although skin irritation did not appear after a single application, irritation was reported in human volunteers receiving nine topical applications of 1, 2.5, 5 or 10% α-amylase (from B. subtilis) in distilled water. The magnitude of responses increased with increasing concentration such that the use of the 10% concentration was discontinued and was replaced for the rest of the study by a 0.5 % α-amylase. The irritation was thought to be due to residual protease activity present in the amylase preparation (HERA, 2005).

The irritation potential of mannanase to the skin was evaluated in an OECD guideline 404 (Acute Dermal Irritation/Corrosion) study. New Zealand white rabbits were exposed to 0.5 ml of mannanase

via semi occlusive dressing for four hours. The rabbits were observed for dermal and systemic reactions at 1, 24, 48, and 72 hours after patch removal. There were no signs of erythema or oedema during the study period. There were no abnormal clinical signs reported, and the body weight changes were determined to be normal. The primary irritation score was reported to be zero which indicates that Mannanase is not irritating to the skin of rabbits (ECHA) [KI. score = 1].

Eye

The irritation potential of mannanase to the eye was evaluated in an OECD guideline 405 (Acute Eye irritation/Corrosion) study. New Zealand white rabbits were exposed to 0.1 mL of mannanase in one eye. Each treated eye was examined for irritation of the cornea, the iris, and the conjunctiva at 1, 24, 48, and 72 hours after exposure to mannanase. The mean cornea opacity score was reported to zero, the mean chemosis score was reported to be zero, and the mean conjunctiva score was reported to be 0.33. All the reported effects were cleared 48-72 hours after treatment. In this study, mannanase was reported to be non-irritating to the eyes of rabbits (ECHA) [KI. score = 1].

E. Sensitisation

Skin

α-Amylase (from B. subtilis) was not a skin sensitiser to guinea pigs in two different studies (HERA, 2005). In a human repeat insult patch test (HRIPT) reported above. There were no significant reactions indicative of skin sensitisation in the challenge phase (HERA, 2005).

Respiratory

There are no studies available.

F. Repeated Dose Toxicity

Oral

A sub-chronic systemic toxicity study (OECD guideline 408-Repeated dose 90-day oral toxicity study) was performed on Wistar rats. Male and female rats were exposed to 128, 425, and 1,277 mg/kg bw/day of mannanase daily by oral gavage for a total of 13 weeks. Mannanase was well-tolerated at all doses and there were no significant findings of toxicological relevance. The NOAEL for this study was established at ≥1, 277 mg/kg bw/day (highest dose tested) (ECHA) [KI. score =1].

In the 13-week dietary study on a cellulase enzyme (cellulase enzymes cleave the β-1,4-glycosidic bonds in cellulose and for the purpose of risk assessment the structure of amylases would be expected to be relatively similar to that of celluloses given the fact that amylases and cellulases are both enzyme families that hydrolyse polysaccharides, although differing in their substrates), there was reduced body weight gain in rats given 3,000 mg/kg/day. No other adverse effects were observed. The NOAEL for this study is 600 mg/kg/day (HERA, 2005)

Rats and dogs have been given amylase enzymes orally for up to 90 days. These studies were not reported in any detail and the actual amount of enzyme in the formulations tested in these studies is unclear. No findings of toxicological significance were observed in either species exposed to any of the α-amylase formulations tested other than “slight” reductions in food consumption at high dietary doses (>5% of the diet) or irritation of the stomach of rats dosed by oral gavage with >3,000 mg/kg/day (HERA, 2005).

Inhalation

There are no studies available.

Dermal

There are no studies available.

G. Genotoxicity

In vitro Studies

The *in vitro* genotoxicity studies on mannanase are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on mannanase

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation assay (Salmonella typhimurium strains TA1537, TA98, TA1535, TA100; Escherichia coli WP2uvrA)	-	-	1	ECHA
Mammalian chromosome aberration test (human lymphocytes)	-	-	1	ECHA
Mammalian cell gene mutation test (mouse lymphoma L5178Y cells) **	-	-	1	ECHA
Bacterial reverse mutation assay (S. typhimurium strains TA1535, TA1537, TA98, TA100) ***	-	-	N/A	HERA, 2005
Chromosome aberration assay (human lymphocytes and bone marrow) ****	-	-	N/A	HERA, 2005

*+, positive; -, negative
**α-Amylase
***α-Amylase (from B. subtilis and B. licheniformis
****α-Amylase (from B. licheniformis)

In Vivo Studies

There are no studies available.

H. Carcinogenicity

Oral

There are no studies available.

Inhalation

There are no studies available.

Dermal

There are no studies available.

I. Reproductive Toxicity

Two α-amylases, one derived from B. stearothermophilus and one derived from a genetically modified strain of B. subtilis have been evaluated for effects on fertility in one-generation studies in rats. The diets containing 0, 36 or 72 units of α-amylase/g food. No treatment-related effects on fertility or other findings of toxicological significance were observed for either enzyme (HERA,2005).

J. Developmental Toxicity

Oral

There are no studies available.

Inhalation

There are no studies available.

Dermal

There are no studies available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for mannanase follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

Proteins are digested into amino acids by gastric juices, digestive enzymes and pancreatic proteolytic enzymes in the lumen of the gastrointestinal tract. As enzymes are simply a class of proteins, enzymes will undergo the same process as any food source based on proteins. Absorption of enzymes in toxicological significant amounts through the gastrointestinal tract is unlikely (ECHA). Therefore, an oral reference value and DWG value was not derived.

B. Cancer

There are no studies available to determine if mannanase is a carcinogen. A cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Mannanase does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Mannanase has low acute aquatic toxicity to algae, fish, and invertebrates. There are no chronic studies available, but mannanase is to have low chronic toxicity to ecological receptors given its reported physicochemical properties.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on mannanase.

Table 3: Acute Aquatic Toxicity Studies on mannanase

Test Species	Endpoint	Results (mg aep*/L)	Klimisch Score	Reference
Oncorhynchus mykiss (rainbow trout)	96-hour LC_{50}	>105.8 mg/L (55.5 mg aep)	1	ECHA
Daphnia magna	48-hour $_{EC50}$	>105.8 mg/L (55.5 mg aep)	1	ECHA
Raphidocelis subcapitata (green algae)	72-hour EC_{50}	>105.8 mg/L (55.5 mg aep)	1	ECHA
*Scenedesmus subspicatus***	72-hour EC_{50}	112	-	HERA 2005

*Active enzyme protein (aep)/L
**α-Amylase (Termamyl)

Chronic Studies

An OECD Guideline 201 (Alga, growth inhibition) study was conducted using Raphidocelis subcapitata (green algae) that were exposed to mannanase for 72 hours at 25 °C. The 72 hour NOEC was reported to be 26.5 mg/L (equivalent to 13.9 mg/L active enzyme protein) based on growth rate (ECHA)[KI. score =1].

C. Terrestrial Toxicity

There are no studies available. However, mannanase has a very low vapor pressure (0.004 Pa) and a low K_{ow} (<0). Therefore, exposure to agricultural soil via sludge application as well as via aerial deposition is very low (ECHA).

D. Calculation of PNEC

The PNEC calculations for mannanase follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute $E(L)C_{50}$ values are available for fish (55.5 mg aep /L), *Daphnia* (55.5 mg aep /L), and algae (55.5 mg aep /L). Results from chronic studies are available for algae (13.9 mg aep/L). On the basis that the data consists of short-term results from

three trophic levels and long-term results from one trophic level, an assessment factor of 100 has been applied to the NOEC value of 13.9 mg ep/L for algae. The $PNEC_{water}$ is 0.139 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. PNEC values for sediment exposure have not been derived because the enzyme is readily biodegradable, highly water soluble and has a very low potential for adsorption to sediments. Exposure of the sediment to toxicologically significant concentrations of the enzyme is thus not expected (ECHA).

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.002 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
$= (0.026/1500) \times 1000 \times 0.139$
$=0.002$ mg/kg

Where:
Kp_{soil}= soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]
$Kp_{soil} = K_{oc} \times f_{oc}$
$=1.3 \times 0.02$
$=0.026$ m^3/m^3

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The calculated K_{oc} for similar enzymes to mmannanase is <1.3 L/kg (HERA, 2005)
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Mannanase is readily biodegradable and thus does not meet the screening criteria for persistence.

The log K_{ow} for mannanase is -1.3. Thus, mannanase does not meet the criteria for bioaccumulation.

The chronic NOEC value for mannanase is >0.1 mg/L. The acute $E(L)C_{50}$ values from the acute aquatic toxicity studies on mannanase are > 1 mg/L. Thus, mannanase does not meet the criteria for toxicity.

The overall conclusion is that mannanase is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H334: May cause allergy or asthma symptoms or breathing difficulties if inhaled

Respiratory sensitisation-category 1

B. Labelling

Danger

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

A workplace exposure standard is not available in Australia for mannanase

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required if ventilation is adequate.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Mannanase is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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METHANOL

This dossier on methanol presents the most critical studies pertinent to the risk assessment of methanol in its use in coal seam or shale gas extraction activities. It does not represent an exhaustive or critical review of all available data. The information presented in this dossier was obtained primarily from the OECD-SIDS documents on methanol (OECD, 2004a,b), and the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed methanol in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Methanol

CAS RN: 67-56-1

Molecular formula: CH_4O

Molecular weight: 32.04 g/mol

Synonyms: Methyl alcohol, carbinol, wood spirits, wood alcohol, methylol, wood, columbian spirits, colonial spirit, columbian spirit, methyl hydroxide, monohydroxymethane, pyroxylic spirit, wood naphtha.

SMILES: CO

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Physico-Chemical Properties of Methanol

Property	Value	Klimisch score	Reference
Physical state at 20°C and 101.3 kPa	Colourless liquid	2	ECHA
Melting Point	-97.8°C @ 101.3 kPa	2	ECHA
Boiling Point	64.7°C @ 101.3 kPa	2	ECHA
Density	790 kg/m^3@ 20 °C	2	ECHA
Vapour Pressure	16927 Pa @ 25 °C	2	ECHA
Partition Coefficient (log P_{ow})	-0.77	2	ECHA
Water Solubility	>1,000 g/L [miscible]	2	ECHA
Flash Point	9.7°C	2	ECHA
Auto flammability	455°C @ 101.3 kPa	2	ECHA
Viscosity	0.544 – 0.59 mPa s (dynamic)	2	ECHA
Henry's Law Constant	0.461 Pa m^3/mol @ 20 °C	2	ECHA

Methanol is a highly flammable liquid.

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Methanol is readily biodegradable. It has a low adsorptive capacity to soils and is unlikely to bioaccumulate.

B. Biodegradation

Methanol is readily biodegradable. In a closed bottle test using seawater, there was 84% and 95% degradation after 10 and 20 days, respectively (Price et al., 1974; ECHA). [Kl. score = 2]

In a soil test using [^{14}C]-methanol, there was 53.4% degradation under aerobic conditions after 5 days, as measured by CO_2 evolution; and 46.3% degradation under anaerobic conditions after 5 days, as measured by CO_2 evolution (Scheunert et al., 1987; ECHA). [Kl. score = 2]

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

The adsorption of methanol was investigated in three different soil types at 6°C (Lokke, 1984; ECHA). There was slight adsorption with the sandy soils tested (percentage organic matter of 0.09% and 0.1% in the samples) and with the clay soil (percentage organic matter was 0.22%). Methanol solutions of concentrations of 0.1, 1.0, 9 and 90 mg/L were used in one-hour exposure adsorption studies; the K_{oc} values were between 0.13 and 0.61 for all soil types and at all concentrations.

Based upon these K_{oc} values, if released to soil, methanol is expected to have very high mobility. If released into water, due to its high water solubility and low K_{oc}, methanol is not expected to adsorb to suspended solids and sediment in water.

D. Bioaccumulation

The BCF of methanol in *Cyprinus carpio* was determined to be 1.0 (Gluth et al. 1985); in *Leuciscus idus*, the BCF was < 10 (Hansch and Leo, 1985; Freitag et al. 1985). Therefore, the potential for bioaccumulation is low.

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Methanol has low acute oral, dermal and inhalation toxicity in experimental animals but moderate to high acute oral and dermal toxicity in humans. Methanol is metabolised to formate, which is considered to be the ultimate toxicant in acute methanol intoxication in humans. Acute methanol toxicity in humans is characterised by CNS depression, followed by acidosis and ocular injury. Methanol is not irritating to the skin, but it is slightly irritating to the eyes. It is not a skin sensitiser. Repeated exposures by the oral and inhalation routes have not resulted in any systemic toxicity to rodents. In primates, adverse health effects on brain, kidney and heart were observed in chronic inhalation studies. Methanol is not genotoxic or carcinogenic. Conflicting results have been obtained concerning the effect of methanol on reproductive and developmental toxicity in experimental animals. However, it is not considered to have reproductive or developmental toxicity in humans.

B. Toxicokinetics and Metabolism

Several reviews on the metabolism and pharmacokinetics of methanol are available (Kavet and Nauss, 1990; Liesivuori and Savolainen, 1991; Tephly, 1991; IPCS, 1997; OECD, 2004a, b). Methanol is first oxidised to formaldehyde. This initial metabolic step involves different enzymes in rats than in primates and humans, although the rates are similar. A catalase–peroxidase system is primarily responsible for the initial step in rats, whereas alcohol dehydrogenase plays a major role in humans and monkeys. Methanol oxidation can also occur via hepatic microsomal oxidation involving the cytochrome P450 system.

Formaldehyde is converted to formic acid, which is converted to formate and a hydrogen ion. Conversion to formic acid is a two-step process, the second step is irreversible. In the first reaction, formaldehyde combines with reduced glutathione (GSH) to form S-formylglutathione. This is mediated by an NAD-dependent formaldehyde dehydrogenase. In the second reaction, thiolase catalyses the hydrolysis of S-formylglutathione to form formic acid and GSH. A folate-dependent pathway in the liver is responsible for formate metabolism in both rats and primates. Formate first forms a complex with tetrahydrofolate (THF) that is sequentially converted to 10-formyl-THF (by formyl-THF synthetase) and then to CO_2 (by formyl-THF dehydrogenase). THF is derived from folic acid in the diet and is also regenerated in the folate pathway. Although the folate pathway metabolises formate in both rats and monkeys, rats use the pathway more efficiently.

The dermal uptake rate of liquid methanol applied to the forearm of human volunteers was 11.5 mg/cm^2/hr (Dutkiewicz et al., 1980). The dermal flux for methanol in human skin (epidermis) *in vitro* is 8.29 mg/cm^2/hr (Schueplein and Blank, 1971). When 12 human volunteers immersed one hand into a vessel containing neat methanol for up to 16 minutes, the maximum methanol concentration in blood reached 1.9 ± 1.0 hr after exposure. Delivery rates from the skin into blood lagged exposure by 0.5 hours, and methanol continued to enter the blood for 4 hours following exposure. The average derived dermal absorption rate absorption rate was 8.1 $\pm$ 3.7 mg/cm^2/hr. The authors calculated that the maximum concentration of methanol in blood following immersion of one hand in methanol for approximately 20 minutes is comparable to that reached following inhalation exposures to 200 ppm methanol (Batterman and Franzblau, 1997).

C. Acute Toxicity

The acute oral LD_{50} for rats range from 6,200 to 13,000 mg/kg (Kimura et al., 1971; Welch and Slocum, 1943; Deichman and Mergard, 1948; Smyth et al., 1941). The acute dermal LD_{50} for rabbits was reported to be 20 mL/kg (Rowe and McCollister, 1982). The inhalation 4- and 6-hour LC_{50} values in rats are 128.2 and 87.5 mg/L, respectively (BASF, 1980a,b). Sublethal doses, however, produce CNS effects and ocular injury that may result in blindness. This effect has been seen in primates, but not in rodents, and has been attributed to the differences in blood levels of the metabolite, formic acid.

Methanol is metabolised to formate, which is considered to be the ultimate toxicant in acute methanol intoxication in humans. Acute methanol toxicity in humans is characterised by CNS depression, followed by acidosis and ocular injury. Generally, transient CNS effects appear above methanol levels of 200 mg/L and serious ocular symptoms appear above 500 mg/L (OECD, 2004a). This blood concentration can transiently be achieved in an adult person (70 kg) by ingestion of 0.4 mL methanol/kg (approximately 0.32 mg/kg). The minimal acute methanol dose to humans that can result in death is considered to be 300 to 1,000 mg/kg by ingestion, and fatalities have occurred in untreated patients with initial methanol blood levels in the range of 1,500-2,000 mg/L (OECD,

2004a). However, such high blood methanol levels able to cause death are not likely to be achieved through inhalation exposure.

D. Irritation

Methanol is not irritating to the skin of rabbits (BASF, 1975), but it is slightly irritating to the eyes of rabbits (BASF, 1975).

E. Sensitisation

Methanol was not considered a skin sensitiser to guinea pigs (BASF, 1979).

F. Repeated Dose Toxicity

Oral

Male and female Sprague–Dawley rats were dosed by oral gavage with 0, 100, 500 or 2,500 mg/kg of methanol for 90 days. There were no differences in body weight gain and food consumption between treated and control animals. Brain weights were decreased in both sexes in the 2,500 mg/kg dose group. Elevated serum glutamic pyruvate transaminase and alkaline phosphatase were noted in the 2,500 mg/kg dose group, but there were no adverse treatment-related effects in the gross pathology and histopathological evaluation. The NOAEL is 500 mg/kg/day (USEPA, 1986).

Sprague-Dawley rats were given in their drinking water 0, 500, 5,000 or 20,000 ppm methanol for 104 weeks, and then the animals were maintained until natural death. The study was conducted by the Ramazzini Foundation which uses its testing guideline for carcinogenicity studies and not an internationally accepted guideline. Treatment with methanol did not decrease survival. However, there was considerable early mortality; by 18 months, 30% of the male controls had died. In females, there were no differences in survival between controls and treated groups. There was still more early mortality in the females than expected, but it was less pronounced than the males. There was no obvious effect of methanol exposure on water consumption. The 20,000 ppm males and females weighed more than the controls (up to 14% and 7%, respectively) throughout the study. The 5,000 ppm females also weighed more (4%) than the controls at 24 months, but not at earlier time points. There were no body weight differences between the remaining treatment groups and the controls. The calculated methanol doses based on water intake were: 0, 55, 542 and 1,840 mg/kg/day for males; and 0, 67, 630 and 2,250 mg/kg/day for females. Nearly all rats in all dose groups had some pathology in the lung. The finding of lung pathology was consistent regardless of the age at death (not an old age response). The lung pathology included inflammation, dysplasia or tumours. Lung pathology was present in 70-100% of the first 10% of deaths in each group, including controls (70, 80, 80, 100% in males; and 90, 90, 100, 100% in females at 0, 500, 5,000 and 20,000 ppm, respectively). The degree of inflammation in the lungs is difficult to assess because no other lung information was recorded for the rats when a neoplasm in the lung was recorded (Soffritti et al., 2002; Cruzan, 2009; USEPA, 2013a) [Kl. score = 3].

Inhalation

Cynomolgus monkeys or Sprague–Dawley rats were exposed by inhalation to 0, 500, 2,000 or 5,000 ppm (0, 660, 2,620 or 6,552 mg/m^3) methanol for 6 h/day, 5 days/week for 4 weeks. There was no mortality and no clinical signs of toxicity among the monkeys, but there were a few signs of eye and nose irritation in the rats. No differences were seen between treated and control groups in body weight gain and organ weights, with the exception being decreased absolute adrenal weight in the 5,000 ppm female monkeys and increased relative spleen weights in the 2,000 ppm female rats.

These changes were not considered by the authors to be of biological significance. There were no treatment-related effects on the ophthalmoscopy, gross pathology or histopathology. The NOAEL for this study is 5,000 ppm (6,552 mg/m^3) (Andrews et al., 1987) [Kl. score = 4].

Groups of four male rats were exposed by inhalation to 0, 200, 2,000 or 10,000 ppm (0, 262, 2,621 or 13,104 mg/m^3) methanol for 6 hours/day, 5 days/week for 1, 2, 4 or 6 weeks. Additional groups of animals were exposed for 6 weeks followed by a 6-week recovery period. Evaluation of a number of parameters including lung weights, surfactant levels and enzyme activities did not reveal any adverse effects on the lung. No histopathological examinations were performed (White et al. 1983) [Kl score = 2].

Male and female F344 rats were exposed by inhalation to 0, 10, 100 or 1,000 ppm methanol 19.5 hours/day, 7 days/week for 104 weeks. The average methanol doses were: 0, 3.7, 37 and 369 mg/kg/day in males; and 0, 5.9, 60 and 599 mg/kg/day for females. There were no treatment-related clinical signs and no effect on survival or food consumption. Lower body weights were seen in the 1,000 ppm females beginning around Day 259, but after Day 574, there was no difference from controls. Body weights in males were similar across all groups. There were no treatment-related effects on urinalysis, hematology or clinical biochemistry. Nor were there any treatment-related effects on organ weights or gross lesions. Histopathologic examination showed no statistically significant differences between treated and control animals (NEDO, 1985a) [Kl score = 2].

Male and female B6C3F1 mice were exposed by inhalation to 0, 10, 100 or 1,000 ppm methanol 19.5 hours/day, 7 days/week for 78 weeks. The average methanol doses were: 0, 9.8, 95 and 947 mg/kg/day in males; and 0, 8.1, 106 and 1,071 mg/kg/day for females. There were no treatment-related clinical signs and no effect on survival or body weight. Food consumption was decreased slightly between months 7 and 12 in the 1,000 ppm females. Urinalysis, hematology and clinical biochemistry were similar across all groups. No differences were seen in organ weights, gross lesions or histopathology between treated and control mice (NEDO, 1985b) [Kl score = 2].

Dermal

No studies were identified.

G. Genotoxicity

In Vitro Studies

Methanol was not mutagenic to *Salmonella* strains TA97, TA98, TA100, TA1535, TA1537 and TA1538 in *in vitro* bacterial mutation assays with or without metabolic activation (De Flora et al., 1984a,b; Florin et al., 1980; Gocke et al.,1981). Equivocal results were obtained with *Salmonella* strain TA102 in the presence of metabolic activation (De Flora et al., 1984b). Methanol was not mutagenic in a DNA-repair test using various strains of *Escherichia coli* WP2 (De Flora et al., 1984a) and in a forward mutation assay using *Schizosaccharomyces pombe* (Abbondandolo et al., 1980).

Methanol did not induce micronuclei in Chinese hamster lung V79 cells in vitro (Lasne et al., 1984). Methanol was mutagenic in the mouse lymphoma assay in the presence of metabolic activation (McGregor et al., 1985), but it was not mutagenic in a Basc test or in a Drosophila, sex-linked, recessive lethal mutation assay (Gocke et al., 1981). Treatment of primary cultures of Syrian golden hamster embryo cells with methanol did not lead to cell transformation (Heidelberger et al., 1983).

In Vivo Studies

Male C57BL/6J mice were exposed by inhalation to 0, 800 or 4,000 ppm methanol, 6 hours/day for five days. There were no increased frequencies of micronuclei in blood cells; sister chromatid exchanges, chromosomal aberrations, or micronuclei in lung cells; or synaptosomal complex damage in spermatocytes (Campbell et al., 1991).

Normal or folate-deficient mice were given four daily intraperitoneal injections of up to 2,500 mg/kg of methanol. There was no increase in micronucleated erythrocytes in the treated mice compared to the controls (O'Loughlin et al., 1992).

Male and female NMRI mice were given a single intraperitoneal injection of 0, 1,920, 3,200 or 4,480 mg/kg methanol. There was no increase in micronuclei observed in the bone marrow at any dose level (Gocke et al., 1981).

H. Carcinogenicity

The carcinogenicity studies conducted on methanol were reviewed by Cruzan (2009) and by the USEPA (2013a).

Oral

Male and female SD rats were given in their drinking water 0, 500, 5,000 or 20,000 ppm methanol for 104 weeks. This study was conducted by the Ramazzini Foundation, which uses a unique methodology and not the standardised international testing guidelines. There was excessive early mortality, and lung pathology (inflammation, dysplasia, or tumours) was present in 87 to 94% of those dying anytime during the study. An increase in lympho-immunoblastic lymphomas was reported (Soffritti et al., 2002; Cruzan, 2009; USEPA, 2013a) [KI score = 3].

Inhalation

Male and female F344 rats were exposed by inhalation to 0, 10, 100 or 1,000 ppm methanol 19.5 hours/day, 7 days/week for 104 weeks. The average methanol doses were: 0, 3.7, 37 and 369 mg/kg/day in males; and 0, 5.9, 60 and 599 mg/kg/day for females. There was no increase in tumours in the methanol-exposed rats and mice (NEDO, 1985a) [KI score = 2].

Male and female B6C3F1 mice were exposed by inhalation to 0, 10, 100 or 1,000 ppm methanol 19.5 hours/day, 7 days/week for 78 weeks. The average methanol doses were: 0, 9.8, 95 and 947 mg/kg/day in males; and 0, 8.1, 106 and 1,071 mg/kg/day for females. There was no increase in tumours in the methanol-exposed mice (NEDO, 1985b) [KI score = 2].

I. Reproductive and Developmental Toxicity

Based on the data available, methanol is not considered to have reproductive or developmental toxicity in humans (NICNAS, 2013).

The reproductive and developmental toxicity studies were reviewed by the NTP Centre for Evaluation of Risks to Human Reproduction (NTP-CERHR, 2003). Conflicting results have been obtained concerning the effect of methanol on testicular hormones in rats; nevertheless, methanol does not appear to be a male reproductive toxicant. The primate data indicates that methanol is unlikely to be a reproductive hazard in females. Methanol causes developmental effects at very high

exposure levels in both rats (≥ 10,000 ppm) and mice (≥ 2,000 ppm); there is also some evidence that it is a developmental neurotoxicant in rodents, but not in primates.

Blood methanol concentrations associated with serious teratogenic effects and reproductive toxicity are in the range associated with formate accumulation, which is likely to result in metabolic acidosis, and visual and clinical effects in humans (NTP-CERHR, 2003). Other effects (such as subtle, not yet definitive neurological effects observed in primates) may be exhibited at lower inhalation doses and lower methanol blood levels (OECD, 2004).

The limited data available in humans do not show an association of reproductive and developmental toxicity with methanol (NTP-CERHR, 2003). Based on the studies reviewed by the NTP (2003), it concluded that there is evidence to suggest that women with low folate levels may be more susceptible to the adverse developmental effects of methanol, but more information is necessary to clarify this issue (NICNAS, 2013).

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for methanol follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2021).

A. Non-Cancer

Oral

USEPA has derived an oral reference dose (RfD) by using exposure-response data from candidate principal inhalation studies of mice (Rogers et al., 1993) and rats (NEDO, 1987) and route-to-route extrapolation with the aid of the USEPA physiologically based pharmacokinetic (PBPK) model. The decision to use inhalation rather than oral study data is due to limitations in the database of oral studies, including the limited reporting of noncancer findings in the subchronic and chronic oral studies of rats, the determination that developmental effects are the most sensitive effects of methanol exposure. The RfD of 2 mg/kg/day was estimated from the Rogers et al. (1993) study for extra cervical rib incidence in mice (USEPA, 2013a). This RfD will be used for determining the drinking water guidance value.

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD: Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2021)
Proportion of water consumed = 10% (ADWG, 2021)
Volume of water consumed = 2 L (ADWG, 2021)
Drinking water guidance value = (2 x 70 x 0.1)/2 = 7 mg/L

B. Cancer

Methanol was not carcinogenic to rats or mice in chronic inhalation studies. Increased tumours from methanol in drinking water were reported by Soffritti et al. (2002); however, there are methodological problems with this study and questions have been raised about the validity of the results. No cancer reference value was derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Methanol is a highly flammable liquid.

Methanol does not exhibit the following physico-chemical properties:

- Explosivity
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Methanol exhibits a low toxicity concern for aquatic organisms, terrestrial invertebrates and plants.

B. Aquatic Toxicity

Acute Studies

Table 2 lists the results of acute aquatic toxicity studies conducted on methanol.

Table 2: Acute Aquatic Toxicity Studies on Methanol

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Bluegill	96-hour LC_{50}	15,400	1	Poirer et al. 1986
Salmo gairdneri	96-hour LC_{50}	20,100	1	Call et al., 1983
Pimphales promelas	96-hour LC_{50}	28,100	1	Call et al., 1983
Daphnia magna	96-hour EC_{50}	18,260	2	Dom et al., 2012; ECHA
Daphnia magna	48-hour EC_{50}	>10,000	2	Kuehn et al., 1989
Selenastrum capricornutum	96-hour EC_{50}	~22,000	2	Cho et al., 2008; ECHA
Chlorella pyrenoidosa	10 to 14-day EC_{50}	28,400	2	Stratton and Smith, 1988

Chronic Studies

No adequate chronic studies were identified. Reported studies were either invalid or their reliability was questionable. Methanol belongs to the category of organic chemicals exerting toxicity for aquatic organisms with a non-specific mode of action. The acute and chronic toxicity may be estimated for such kind of chemicals using QSAR methods. The ECOSAR model (version 1.11, US EPA, July 2012) predicts for methanol a chronic toxicity value of about 450 mg/L (equivalent to a NOEC) for *Pimephales promelas* and a value of 208 mg/L for *Daphnia magna* (REACH) [Kl. score = 1].

C. Terrestrial Toxicity

The terrestrial toxicity studies on methanol are listed in **Table 3**.

Table 3: Terrestrial Toxicity Studies on Methanol

Test Species (Method)	Endpoint	Results (mg/kg soil dw)	Klimisch score	Reference
Earthworm *Eisenia fetida* (OECD 222)	35-day EC_{50} 63-day EC_{50}	17,199 26,646	2	ECHA
Folsomia candida (OECD 232)	28-day EC_{25} 28-day NOEC* (reproduction)	2,842 1,000	1	ECHA
Hordeum vulgare (OECD 208)	14-day EC_{50} 14-day NOEC* (seedling emergence)	15,492 12,000	1	ECHA
	14-day EC_{25} 14-day NOEC* (shoot dry mass)	2,538 1,555		
	14-day EC_{25} 14-day NOEC* (root dry mass)	2,823 2,592		
	14-day EC_{25} 14-day NOEC* (shoot length)	4,885 2,592		
	14-day EC_{25} 14-day NOEC* (root length)	5,752 4,320		

* Since only EC_{25} values were available from the test results, NOECs were derived graphically from the representing treatment means.

D. Calculation of PNEC

The PNEC calculations for methanol follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute $E(L)C_{50}$ values are available for fish (15,400 mg/L), *Daphnia* (> 10,000 mg/L) and algae (22,000 mg/L). There are no well-conducted long-term studies on methanol. Therefore, an assessment of 1,000 has been applied to the lowest reported effect concentration of 10,000 mg/L for *Daphnia*. The $PNEC_{water}$ is 10 mg/L.

PNEC Sediment

There are no adequate toxicity studies on sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 6.3 mg/kg wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (0.81/1280) \times 1000 \times 10$
$= 6.3$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [0.2 \times Kp_{sed}/1000 \times BD_{soilid}]$
$= 0.8 + [0.2 \times 0.02/1000 \times 2400]$
$= 0.81\ m^3/m^3$

Where:
Kp_{sed} = solid-water partition coefficient (L/kg).
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]
$Kp_{sed} = K_{oc} \times f_{oc}$
$= 0.61 \times 0.04$
$= 0.02$ L/kg

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for methanol is 0.61 L/kg.
f_{oc} = fraction of organic carbon suspended sediment = 0.04 [default].

PNEC Soil

Experimental results from chronic studies are available for three trophic levels. The lowest NOEC is 1,000 mg/kg soil dry weight for the arthropod *Folsomia candida*. On the basis that the data consists of long-term results from three trophic levels, an assessment factor of 10 has been applied to the lowest reported long-term NOEC of 1,000 mg/kg soil dry weight. The $PNEC_{soil}$ is 100 mg/kg soil dry weight.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009 and ECHA, 2008).

Methanol is readily biodegradable and thus it does not meet the screening criteria for persistence.

Based on an experimental BCF of < 10 in fish, methanol does not meet the criteria for bioaccumulation.

There are no adequate chronic toxicity studies on methanol. Predicted toxicity based on QSAR methods indicates chronic values > 0.1 mg/L for fish and invertebrates. The acute EC_{50} values of methanol in fish, invertebrates and algae is >1 mg/L; thus, it does not meet the screening criteria for toxicity.

The overall conclusion is that methanol is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Flammable Liquid Category 2

Acute Toxicity Category 3 [Oral]

Acute Toxicity Category 3 [dermal]

Acute Toxicity Category 3 [inhalation]

STOT SE Category 1 [optic nerve, central nervous system]

In the EU, there are concentration limits for the STOT SE classification of methanol. This may or may not apply to GHS classifications for Australian SDS.

Concentration range (%):

>10	STOT SE Category 1
>3 and <10	STOT SE Category 2

B. Labelling

Danger

C. Pictograms

The health hazard pictogram is omitted if the STOT SE classification for methanol does not apply (i.e., concentration of methanol is below the concentration limits).

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

Note: Methanol is used in the drilling mud product ALDACIDE® G ANTIMICROBIAL at a concentration of 0.1% to 1%. The safety and handling of methanol at this concentration in ALDACIDE® G ANTIMICROBIAL will be provided in the dossier on glutaraldehyde, the major constituent of ALDACIDE® G ANTIMICROBIAL.

A. Occupational Exposure Standards

The workplace exposure standard for methanol in Australia is 200 ppm (262 mg/m^3) as an 8-hour TWA and 250 ppm (328 mg/m^3) as a 15-minute STEL. There is also a skin notation indicating that absorption through the skin may be a significant source of exposure.

B. Transport Information

Methanol is used in drilling mud product ALDACIDE® G ANTIMICROBIAL at a concentration of 0.1 to 1%. The transportation information for ALDACIDE® G ANTIMICROBIAL will be provided in the dossier on glutaraldehyde, the major constituent of ALDACIDE® G ANTIMICROBIAL.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

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POLYACRYLAMIDE

This dossier on polyacrylamide presents the most critical studies pertinent to the risk assessment of polyacrylamide in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained the Cosmetic Ingredient Review on polyacrylamide (CIR, 2005) and from the book titled Ecological Assessment of Polymers, Strategies for Product Stewardship and Regulatory Programs (Lyons and Vasconellos, 1997). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed polyacrylamide in an IMAP Tier 1 assessment and concluded that it is a polymer that poses no unreasonable risk to the environment[1]

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Copolymer of polyacrylamide (poly(2-propenamide)] and polyacrylate [poly(2-propenoic acid)]

CAS RN: 9003-05-8

Molecular formula: $(C_3H_5NO)_x$- and $(C_3H_3O_2)_x$-

Molecular weight: 1,000,000 to >50,000,000 g/mol for polyacrylamide copolymers used as flocculants (Lyons and Vasconcellos, 1997)

Synonyms: Polyacrylamide, anionic polyacrylamide, Copolymer of polyacrylamide (poly(2-propenamide)] and polyacrylate [poly(2-propenoic acid)]

SMILES: not applicable (polymer)

II. PHYSICO-CHEMICAL PROPERTIES

Polyacrylamide polymers can exist in cationic, anionic or non-ionic forms, depending on their ionic charge. The non-ionic form of polyacrylamide is generated from the basic polymerisation of acrylamide. Polyacrylamide polymer can then be formed from the hydrolysis of the acrylamide homopolymer either simultaneously during the polymerisation process or as a subsequent step (Zheng et al., 2013). Polyacrylamide polymer can also be formed from the copolymerisation of acrylamide and acrylic acid (Lyons and Vasconellos, 1997; Zheng et al., 2013).

[1] https://www.industrialchemicals.gov.au/chemical-information/search-assessments?assessmentcasnumber=9003-05-8++

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

There are no studies on the environmental fate of polyacrylamide available. As a high-molecular-weight, water-soluble polymer, it is not expected to biodegrade or bioaccumulate (Lyons and Vasconcellos, 1997). The environmental fate of polyacrylamide will be determined primarily by adsorption (Lyons and Vasconcellos, 1997).

The polyanions in this group are expected to partition onto natural colloids in surface waters and in soil and are not expected to undergo long-range transport in the environment (DoEE, 2017).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Polyacrylamide is not bioavailable when ingested. It is essentially non-toxic by the oral route, and it is not irritating to the skin or eyes. Lifetime dietary studies in rats showed no toxicity or carcinogenic effects. There were no indications of reproductive or developmental toxicity in rats given polyacrylamide in their feed over several generations.

B. Metabolism

Female rats were dosed by oral gavage with 140 mg/kg bw/day [^{14}C]-anionic polyacrylamide (molecular weight of 3,000,000). No radioactivity was observed in any of the animals. After 25 hours, the sum of the radioactivity recovered in the feces was 95.13% of the administered dose, and the gastrointestinal tract and contents accounted for 1.64% of the dose. The urine contained activity representing 0.82% of the dose and carbon dioxide in the expired air was 0.07%. Liver and kidney tissue contained about 0.05%. (McCollister et al., 1965).

C. Acute Toxicity

Oral

No deaths were observed in rats given either nonionic or anionic polyacrylamide at oral doses up to 4,000 mg/kg. The oral LD_{50} is >4,000 mg/kg bw/day (McCollister et al., 1965).

Inhalation

There are no studies available.

Dermal

There are no studies available.

D. Irritation

Application of a 5% solution of polyacrylamide to the skin of rabbits was “well tolerated” (CIR, 2005). Polyacrylamide is non-irritating to slightly irritating to the eyes (CIR, 2005).

E. Sensitisation

There are no studies are available.

F. Repeated Dose Toxicity

Oral

Male and female rats were given in their diet 0, 5, or 10% anionic polyacrylamide (molecular weight of 3,000,000) for two years. The animals in the 10% dose group showed significant retardation of growth. At the end of the study, there was a slight statistically significant increase in kidney weights in the 10% males and in the ≥5% females. Gross and microscopic examination of the tissues from the ≥5% groups at 12 months showed some slight diffuse cloudy swelling, areas of focal necrosis and mild replacement fibrosis in the liver. At 18 and 24 months, all the animals showed tissue changes indicate of old age. These changes involved the small arterioles of the heart, kidney, spleen, pancreas, and to a lesser degree, the liver. All groups of animals were affected including the controls, but the degree of severity was somewhat increased in the ≥5% animals. The authors of the study suggested that the effects seen in the ≥5% dietary groups are attributed indirectly to the large, hydrophilic, non-nutritive bulkiness of the polymer in the gastrointestinal tract. For instance, reduced caloric intake may be partially responsible for the growth retardation; there may also have been interference of the absorption of dietary nutrients. Moreover, the [C^{14}]polymer bioavailability studies no gastrointestinal absorption. The NOAEL for this study is 10% in the diet (McCollister et al., 1965).

Inhalation

There are no studies available.

Dermal

There are no studies available.

G. Genotoxicity

There are no *in vitro* or *in vivo* studies available for polyacrylamide.

H. Carcinogenicity

Oral

Male and female rats were fed 0, 5, or 10% anionic polyacrylamide (molecular weight of 3,000,000) in their diet for two years. The tumour incidences were similar between the treated and control animals (McCollister et al., 1965).

Inhalation

There are no studies available.

Dermal

There are no studies available.

I. Reproductive Toxicity and Developmental Toxicity

In an abstract, it was reported that rats fed up to 2,000 ppm polyacrylamide in a three-generation reproductive toxicity study showed no reproductive, developmental, or parental toxicity (CIR, 2005).

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for polyacrylamide follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

No adverse effects were reported in rats fed anionic polyacrylamide in their diet at doses up to 10% for two years (McCollister et al., 1965). Using 0.05 as the fraction of body weight that is consumed per day as food for the rat, the NOAEL for this study is 5,000 mg/kg bw/day-day. The NOAEL of 5,000 mg/kg bw/day-day will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:
UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1

Oral RfD = 5,000/(10 x 10 x 1 x 1 x 1) = 5000/100 = 50 mg/kg bw/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

where:
Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)

Drinking water guidance value = (50 x 70 x 0.1)/2 = 175 mg/L

B. Cancer

Polyacrylamide was not carcinogenic to rats when given in a two-year dietary study; thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Polyacrylamide does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Anionic polyacrylamide has a low acute toxicity concern to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

Table 1 lists the results of acute aquatic toxicity studies on the powder form of anionic polyacrylamides. The data were reported in a table as LC_{50} values with no details on the individual studies.

Table 1: Acute Aquatic Toxicity Studies on Polyacrylamide in powder form*

Test Species	Ionic charge	Results (mg/L)	Klimisch score	Reference
Fathead minnow	-31	LC_{50}: 810	-	Betz laboratories, Inc. (1995)
Rainbow trout	-31	LC_{50}: >100	-	Betz laboratories, Inc (1995)
Bluegill sunfish	-31	LC_{50}: >300	-	Betz laboratories, Inc (1995)
Rainbow trout	-22	LC_{50}: >100	-	Betz laboratories, Inc (1995)
Bluegill sunfish	-22	LC_{50}: >300	-	Betz laboratories, Inc (1995)
Rainbow trout	-12	LC_{50}: >100	-	Betz laboratories, Inc (1995)
Bluegill sunfish	-12	LC_{50}: >300	-	Betz laboratories, Inc (1995)
Daphnia magna	-39	LC_{50}: 470	-	Betz laboratories, Inc (1995)

*Acrylic acid-acrylamide copolymers with molecular weights of >1,000,000.

Chronic Studies

There are no studies available.

C. Terrestrial Toxicity

There are no studies available.

D. Calculation of PNEC

The PNEC calculations for polyacrylamide follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for two trophic levels. Acute $E(L)C_{50}$ values are available for fish (>100 mg/L) and *Daphnia* (470 mg/L). On the basis that the data consists of only short-term results from two trophic levels, an assessment factor of 1,000 has been applied to the lowest reported $E(L)C_{50}$ value of >100 mg/L for fish. The $PNEC_{aquatic}$ is 0.1 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. The K_{ow} and K_{oc} have not been experimentally derived for anionic polyacrylamide; these values cannot be estimated using QSAR models because of the high molecular weight of anionic polyacrylamide. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{sed}$.

PNEC Soil

There are no toxicity data for soil-dwelling organisms. The K_{ow} and K_{oc} have not been experimentally derived for anionic polyacrylamide; these values cannot be estimated using QSAR models because of the high molecular weight of anionic polyacrylamide. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{soil}$.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Polyacrylamide is a large molecular weight, water-soluble polymer. It is not expected to be readily biodegradable; thus, it meets the screening criteria for persistence.

Pharmacokinetic studies showed that polyacrylamide was not bioavailable to rats when ingested; this is most likely due to its large size (high molecular weight) and presumed resistance to breakdown in the gastrointestinal tract. Polyacrylamide is thus not expected to be bioavailable to aquatic or terrestrial organisms. It is not expected to meet the criteria for bioaccumulation.

There are no chronic aquatic toxicity data available for polyacrylamide. The acute $E(L)C_{50}$ values from the acute aquatic toxicity studies on polyacrylamide are > 1 mg/L. Thus, polyacrylamide does not meet the criteria for toxicity.

The overall conclusion is that polyacrylamide is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Polyacrylamide is not classified.

B. Labelling

None

A. Pictogram

None

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention if respiratory irritation develops or if breathing becomes difficult.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Avoid dust formation. Ensure adequate ventilation. Do not breathe dust. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace Australia has not established an occupational exposure standard for polyacrylamide.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Polyacrylamide is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

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POLYOXYETHYLENE NONYLPHENOL ETHER, [NONYLPHENOL, ETHOXYLATED] (CAS NO. 9016-45-9)
POLYOXYETHYLENE GLYCOL TRIMETHYLNONYL ETHER (CAS NO. 127087-87-0)
NONOXYNOL-9 (CAS NO. 26571-11-9)

This group contains polyoxyethylene nonylphenol ether (also referred to as nonylphenol, ethoxylated or NPE), and similar NPEs polyoxyethylene glycol trimethylnonyl ether (also referred to as branched p-nonylphenol ethoxylate) and nonoxynol-9. Information provided in this dossier is based on the group.

This dossier on NPE, branched p-nonylphenol ethoxylate and nonoxynol-9 presents the most critical studies pertinent to the risk assessment of these substances in their use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the NICNAS environmental and human health tier II assessments for the NPE group (NICNAS, 2018 and 2019) and from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): 26-(Nonylphenoxy)-3,6,9,12,15,18,21, octaoxahexacosan-1-ol

CAS RN: 26571-11-9

Molecular formula:C_{33}-H_6O-$O_1$0

Molecular weight: 616.827 g/mol

Synonyms: Nonoxynol-9; 3,6,9,12,15,18,21,24-Octaoxahexacosan-1-ol, 26-(nonylphenoxy)-; Nonaethylene glycol mono (nonylphenyl) ether; nonaethylene glycol nonylphenyl ether; nonylphenol octa(oxyethylene) ethanol

SMILES: CCCCCCCCCC1=CC=C(C=C1)OCCOCCOCCOCCOCCOCCOCCOCCOCCO;

Chemical Name (IUPAC): 2-(4-nonylphenoxy)ethanol

CAS RN: 127087-87-0

Molecular formula: $C_{17}H_{28}O_2$

Molecular weight: 264.4 g/mol

Synonyms: Polyoxyethylene glycol trimethylnonyl ether; Poly(oxy-1,2-ethanediyl), .alpha.-(4-nonylphenyl)-.omega.-hydroxy-branched; 2-(p-Nonylphenoxy)ethanol

SMILES: CCCCCCCCCC1=CC=C(C=C1)OCCO;

Chemical Name (IUPAC): Poly(oxy-1,2-ethanediyl), .alpha.-(nonylphenyl)-.omega.-hydroxy-

CAS RN: 9016-45-9

Molecular formula: $C_{39}H_{72}O_{13}$ (can vary based on length of ethoxy ether chain)

Molecular weight: 748.98 g/mol (can vary based on length of ethoxy ether chain)

Synonyms: polyoxyethylene nonylphenol ether; nonylphenol, ethoxylated; nonylphenol ethoxylate; 2-[2-(4-Nonylphenoxy)ethoxy]ethanol; ethoxylated nonylphenol

SMILES: CC(C)CC(C)CC(C)c1ccc(OCCOCCOCCOCCOCCOCCOCCOCCOCCOCCOCCOCCO)cc1

II. PHYSICO-CHEMICAL PROPERTIES

NPE is a non-ionic surfactant used as a detergent, emulsifier, wetting agent, or defoaming agents. The general formula of NPEs is C15H24(C2H4O) n; where 'n' is the number of ethylene oxide (EO) units attached to the phenol ring, and can vary from 1–120. The NPEs differ by the length of the EO chain, which also contributes to different physicochemical properties and the degree of toxicity (NICNAS, 2019).

Key physical and chemical properties for a representative nonylphenol ether (NPE) are shown in Table 1.

Table 1: Overview of the Physico-chemical Properties of Nonylphenol, Ethoxylated

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Liquid	2	ECHA
Melting Point	>42-<44°C (pressure not provided)	2	ECHA
Boiling Point	>295-<320°C (pressure not provided)	2	ECHA
Density	1050 kg/m3@ 50°C	2	ECHA
Vapour Pressure	140 Pa @ 25°C	2	ECHA
Partition Coefficient (log K_{ow})	3.70 @ 25°C	2	ECHA
Water Solubility	153 g/L @ 20°C	2	ECHA
Flash Point	Not available	-	ECHA
Auto flammability	383°C @ 101.7 kPa	2	ECHA
Viscosity	Not available	-	ECHA
Henry's Law Constant	Not available	-	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

NPE is readily biodegradable, and it is not expected to bioaccumulate. It has a low potential to adsorb to soil or sediment.

B. Biodegradation

NPE is readily biodegradable. There was 96% degradation of NPEs after 30 days, indicating substantial primary biodegradation. The biodegradation process generated degradants nonylphenol mono- and di-ethoxylates, nonylphenoxy acetate and nonylphenol mono-ethoxyacetate, some of which remained at the end of 30 days (ECHA). [Kl. Score = 2].

These degradants are expected to be ultimately biodegraded in the environment (NICNAS, 2018).

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

No sediment or soil partitioning data were found. Long chain nonylphenol ethoxylates are expected to remain in water as they have high water solubility and low volatility. Thus, it is expected that NPE has a low potential for adsorption to soil or sediment. Water soluble degradation products, nonylphenol ethoxyacetates, are also expected to remain in water (NICNAS, 2018).

D. Bioaccumulation

NPEs are surfactants and most surfactants tend to be retained on epithelial surfaces, rather than cross cellular membranes and bioaccumulate (de Oude, 1992; McWilliams and Payne, 2001). Hence, bioaccumulation for most classes of surfactants is generally below the level for concern (McWilliams and Payne, 2001). As a result, NPE is expected to have low bioaccumulation potential in aquatic organisms. The BCF in the fish Cyprinus carprio of nonylphenol ethoxylates was reported to be <0.2 L/kg at 2 mg/L and <1.4 L/kg at 0.2 mg/L (NICNAS, 2018).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Most of the human and animal data available were from studies conducted using NPEs with 1–50 EO units. The NPEs metabolise in the body and biodegrade in the environment to nonylphenols (NPs). Therefore, toxicity of NPs was considered acceptable to derive the toxicity of the ethoxylates when there were no hazard data available on specific systemic endpoints. It is noted that compared with NPEs, NPs are more toxic (NICNAS, 2019).

NPEs exhibits low to moderate oral acute toxicity and low dermal toxicity. Skin irritation studies in rabbits with some NPEs have shown moderate to severe irritation. It is not a skin sensitiser. The available studies with various NPEs indicate that the level of eye irritation generally increases with decreasing EO chain length. No dermal or inhalation repeat dose studies were available but oral repeat does studies do not suggest that NPEs cause serious damage to health. The substance is not genotoxic or carcinogenic. Based on the available data and considering the routes of exposure relevant for humans (excluding spermicide use), a conclusion on the reproductive and developmental toxicity of NPEs cannot be derived. However, NPs are classified for reproductive and developmental toxicity based on animal data.

B. Acute Toxicity

Oral

The acute oral toxicity of NPEs could range from low to moderate. The toxicity of NPEs is considered to increase with decreasing EO units (or chain length) (Health Canada, 2002). For NPE the oral LD50 was reported to be 1310 mg/kg bw in rats (HSDB). However, the CAS RN for NPE applies to many NPEs containing 1–120 EO units. The following LD50s were reported for NPEs of various EO chain lengths (NICNAS, 2019):

- 3500–4500 mg/kg bw in rats for NPEs with EO units 2, 5, 7 or 9;
- 2000–4290 mg/kg bw in mice, guinea pigs and rabbits for an NPE with 9 EO units;
- 1300 mg/kg bw in rats for an NPE with 10 EO units; and
- other NPEs with 30 EO units were reported as 'relatively harmless' in rats but no LD_{50}s were determined.

Reported signs of toxicity included diarrhoea, tremors, prostration and narcosis. Necropsy revealed congested lungs, gastrointestinal system, and kidneys (CIR, 1983 as cited in NICNAS, 2019).

Inhalation

The limited data available are not sufficient to derive a conclusion on the acute inhalation toxicity of the chemicals.

In an acute inhalation study, male rats were exposed (whole body) to undiluted or 1 % NPEs (with 4, 7 or 9 EO units) for either four or eight hours and observed for 14 days. The exposure concentrations were not reported. No toxic effects were observed (CIR, 1983 as cited in NICNAS, 2019).

In a 4-hour acute inhalation study, Sprague Dawley (SD) rats were exposed (whole body) to aerosolised detergent (containing NPE as the principal component) at concentrations of 0.50, 0.90 or 1.41 mg/L. Sub-lethal effects included eye and respiratory 4rritation, hypoactivity, laboured and audible breathing, unkempt fur, and distended abdomens. At two weeks post-exposure, the animals showed body weight loss or decreased weight gain, and perinasal encrustation. The LC_{50} was reported as 1.60 g/m3 (CalEPA, 2010 as cited in NICNAS, 2019).

Dermal

Based on the limited data available, the chemicals are expected to have low acute dermal toxicity. The dermal LD_{50} for NPE was reported to be 2000 mg/kg bw in rabbits (NICNAS, 2019).

C. Irritation

Skin

Skin irritation studies in rabbits with some NPEs have shown moderate to severe irritation. The degree of irritation changes with the number of EO units (NICNAS, 2019).

In a skin irritation study in New Zealand White (NZW) rabbits, 11 NPEs (with EO units 2, 4, 6, 7, 9, 10, 12, 13, 15, 30 or 40) were tested undiluted by applying occlusive patches of 0.01–0.50 mL. The NPEs with EO chains ≤6 caused moderate to severe irritation (CIR, 2015 as cited in NICNAS, 2019).

Severe skin irritation effects were observed in animals tested with NPEs containing five or six EO units. In a skin irritation study, an NPE containing six EO units (NPE-6) was applied (occlusively, 0.5 mL) to the clipped intact and abraded skin of six rabbits. The effects (erythema and oedema) were scored at 24 and 72 hours after application. The chemical was classified as severely irritating to the skin of rabbits, with a primary irritation index (PII) of 3.0. A PII of 6.6 was reported in another skin irritation study with NPE-6 (animal species and experimental details not stated) (CIR, 1999 as cited in NICNAS, 2019).

However, NPEs with an EO) chain of > 30 are slightly irritation or non-irritating (Talmage, 1994).

Eye

The available studies with various NPEs indicate that the level of irritation generally increases with decreasing EO chain length (NICNAS, 2019).

In a study conducted according to the Draize method, an NPE with six EO units caused severe eye irritation in rabbits. The average scores obtained on days one and seven post-exposure were 28.8 and 16.0, respectively (maximum score=110) (CIR, 1999 as cited in NICNAS, 2019).

In studies involving the instillation of 0.1 mL of an undiluted NPE solution to the eyes of rabbits, NPEs with chains of 2 to 15 were moderately to severely irritating. NPEs with EO chains of >30 were non-irritating (ECHA) [Kl. Score = 2].

D. Sensitisation

Based on the available data, NPEs and their anionic surfactant derivatives are generally not considered to have skin sensitisation potential.

In a guinea pig maximisation test, five albino guinea pigs were exposed intradermally to NPE containing six EO units (NPE-6) at concentrations of 0, 1.7, 3.0, 9.0 or 27 % (w/w) during the induction phase. After seven days, undiluted NPE-6 was applied topically, and the site occluded for 48 hours. The application site was later challenged topically with 2.7 % NPE-6. No dermal responses were observed after 48 hours following the challenge (ECHA) [Kl. Score = 2].

E. Repeated Dose Toxicity

Oral

In several 90-day repeated dose oral toxicity studies (individual test protocols), NPEs containing 4, 6, 15, 20, 30 or 40 EO units were orally administered to rats in the diet at 40–5000 mg/kg bw/day (0.01–1% of the diet). Growth retardation due to poor palatability of the diets was observed with NPEs containing 4, 6, 15 and 20 EO units at > 200 mg/kg bw/day. Increased absolute and relative liver weights were observed when animals were administered NPE-4 or NPE-6 at 200 mg/kg bw/day, but no histopathological changes were observed. No effects were observed in rats that ingested NPE-30. Slight hepatic necrosis and centrilobular granular degeneration were observed in rats administered NPE-40 at a 3 % dietary concentration (~700 mg/kg bw/day) (CIR, 1983; Danish EPA, 2000 as cited in NICNAS, 2019).

In 2-year repeated dose oral toxicity studies, NPE-4 and NPE-9 were administered to rats at doses of ~400–1000 mg/kg bw/day. In rats, reduced body weights and enlarged livers were observed at doses > 1000 mg/kg bw/day. The authors concluded that these NPEs had low chronic toxicity (CIR, 1983 as cited in NICNAS, 2019).

In another study using NPE-9 in rats, enlarged livers were accompanied by cloudy swelling and reduced polysaccharides at the 250 mg/kg bw/day dose, and focal hepatic cell necrosis at the 1250 mg/kg bw/day dose (Danish EPA, 2000 as cited in NICNAS, 2019).

In repeated dose oral toxicity study, mice were administered NPE-10 in the diet at doses of 0, 500, 1500 or 4500 ppm (0, 81.5, 254 or 873 mg/kg bw/day) for 104 weeks. At the highest dose, decreased body weight gain, decreased absolute liver and kidney weights, and increased relative brain, liver and kidney weights were observed. No other significant effects attributed to the chemical were observed (CIR, 2015 as cited in NICNAS, 2019). The no observed adverse effect level (NOAEL) for NPE-10 was determined as 254 mg/kg bw/day.

Inhalation

No data are available.

Dermal

No data are available.

F. Genotoxicity

Based on the available in vitro genotoxicity data, NPEs are not considered to be genotoxic. NPEs with EO chains of 9 and 30 were not mutagenic to S. typhimurium strains TA 1535, TA 1537, TA 98, and TA 100 in the absence or presence of metabolic activation (ECHA) [Kl. Score = 2].

No *in vivo* genotoxicity data are available for NPEs.

G. Carcinogenicity

Based on the available data, NPEs are not considered to be carcinogenic.

In a carcinogenicity study, female mice (n = 50/dose) were administered NPE-10 (NPE with 10 EO units) in the diet at doses of 0, 500, 1500 or 4500 ppm (0, 81.5, 254 or 873 mg/kg bw/day) for 104 weeks. No increase in the incidence of neoplastic or non-neoplastic lesions was observed at any dose level. The authors concluded that NPE-10 was not carcinogenic (CIR, 2015 as cited in NICNAS, 2019).

In another carcinogenicity study, rats were administered NPE-9 (NPE with 9 EO units) intravaginally at doses of 0, 6.7 or 33.6 mg/kg bw/day, 3 times a week for 24 months. The administered doses were equivalent to 4 or 20 times the clinical dose, respectively. No significant differences (including masses or mortalities) compared with controls were observed. Positive observations (details not available) in the experimental groups at necropsy were considered to be related to ageing. The authors concluded that NPE-9 was 'neither toxic nor carcinogenic in this lifetime exposure study, even at a dose that was 20 times that recommended for humans' for use as a spermicide (CIR, 1999 as cited in NICNAS, 2019).

In 2-year carcinogenicity studies, NPE-4 and NPE-9 were administered orally to rats at doses of 200 and 140 mg/kg bw/day. No increase in the frequency of tumours was reported (Danish EPA, 2000 as cited in NICNAS, 2019).

H. Reproductive Toxicity

Studies are available only for NPE-9, NPE-10, NPE-30. No data are available for the other chemicals in this group.

The chemical NPE-9 is a known spermicide and the studies available using NPE-9 have reported reproductive toxicity effects in rats from doses of 50 mg/kg bw/day, when administered intravaginally. However, oral studies in rats with NPE-9 showed reproductive and developmental effects only at a dose of 250 mg/kg bw/day. Based on the available data and considering the routes of exposure relevant for humans (excluding spermicide use), a conclusion on the reproductive and developmental toxicity of NPEs cannot be derived. However, NPs are classified for reproductive and developmental toxicity based on animal data (NICNAS, 2019).

In an in vivo sperm abnormality assay, male mice (n = 5/sex/dose) were injected intraperitoneally with NPE-9 in distilled water at doses of 0, 20, 40, 50 or 60 mg/kg bw/day for five days. No increase in the frequency of morphologically abnormal sperm was observed compared with controls (CIR, 1999 as cited in NICNAS, 2019).

In a reproductive toxicity study to evaluate embryotoxicity of NPE-9, nulliparous female Wistar rats were intravaginally administered the chemical at 5 mg/100 g bw (50 mg/kg bw) on gestation days (GD) three or seven. Ulcerative vaginitis and perivaginal oedema were observed in the dams, but were reversible by GD 15. Significant differences in the mean number of normal implantation sites and the number of resorption sites were observed in dams (NICNAS, 2019).

In another study, pregnant Wistar rats were intravaginally administered NPE-9 at 25 mg/kg bw/day on GD 1–10. Increased incidences of nonpregnancies and resorptions were observed in dams administered the chemical on GD 3–6, and a significantly reduced number of live foetuses in dams was observed when the chemical was administered on GD 4, 5, and 9. The chemical NPE-9 was reported to be embryolethal and foetocidal, but not teratogenic when administered intravaginally (CIR, 2015 as cited in NICNAS, 2019).

In an oral developmental toxicity study, female rats were administered NPE-9 at doses up to 500 mg/kg bw/day on GD 6–15. The no observed effect level (NOEL) was determined as 50 mg/kg bw/day based on reproductive and developmental effects (increased pre-implantation losses, skeletal anomalies in the litters) observed at doses > 250 mg/kg bw/day. The same authors conducted a dermal study in female mated rats with NPE-9 at doses of 50 or 500 mg/kg bw/day. No treatment-related effects on the skeletal or soft tissues were observed. However, an increased incidence of extra ribs was observed at 50 mg/kg bw/day (CIR, 1999 as cited in NICNAS, 2019).

In a developmental toxicity study, female mice were administered oral gavage doses of NPE-10 at 600 mg/kg bw/day on GD 6–13. No developmental toxicity effects were observed (CIR, 1999). Repeated subcutaneous administration of NPE-10 in female rats (from birth to day 21 after the birth of F1 offspring) at up to 80 mg/kg bw/day did not cause teratogenic effects. However, the treatment affected the growth of the offspring, e.g., decreased body weight or tendency to decrease body weight from day seven after birth (CIR, 2015 as cited in NICNAS, 2019).

Studies with NPE-30 have shown no treatment-related effects in female rats at oral doses up to 1000 mg/kg bw/day on GD 6–15 (HSDB as cited in NICNAS, 2019).

I. Developmental Toxicity

In an oral developmental toxicity study, female rats were administered NPE-9 at doses up to 500 mg/kg bw/day on GD 6–15. The NOEL was determined as 50 mg/kg bw/day based on reproductive and developmental effects (increased pre-implantation losses, skeletal anomalies in the litters) observed at doses $\geq$ 250 mg/kg bw/day. The same authors conducted a dermal study in female mated rats with NPE-9 at doses of 50 or 500 mg/kg bw/day. No treatment-related effects on the skeletal or soft tissues were observed. However, an increased incidence of extra ribs was observed at 50 mg/kg bw/day (CIR, 1999 as cited in NICNAS, 2019).

In a developmental toxicity study, female mice were administered oral gavage doses of NPE-10 at 600 mg/kg bw/day on GD 6–13. No developmental toxicity effects were observed (CIR, 1999 as cited in NICNAS, 2019). Repeated subcutaneous administration of NPE-10 in female rats (from birth to day 21 after the birth of F1 offspring) at up to 80 mg/kg bw/day did not cause teratogenic effects. However, the treatment affected the growth of the offspring, e.g., decreased body weight or tendency to decrease body weight from day seven after birth (CIR, 2015 as cited in NICNAS, 2019).

Studies with NPE-30 have shown no treatment-related effects in female rats at oral doses up to 1000 mg/kg bw/day on GD 6–15 (HSDB as cited in NICNAS, 2019).

The metabolites, NP and OP, have measured oestrogenic activity. Assessment of NP suggested that developmental effects may derive from antiandrogenic activity (NICNAS, 2019).

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for NPE follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

A two-year dietary study has been conducted in mice with NPE-10. At the highest dose, decreased body weight gain, decreased absolute liver and kidney weights, and increased relative brain, liver and kidney weights were observed. The NOAEL was determined as 254 mg/kg bw/day. The NOAEL from this repeat dose study will be used to derive an oral reference dose (RfD) and drinking water guideline value. This NOAEL was selected rather than NOELs reported for NPE-9 in reproductive/developmental toxicity studies as the conclusion on the reproductive and developmental toxicity of NPEs could not be derived.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

- UF_A (interspecies variability) = 10
- UF_H (intraspecies variability) = 10
- UF_L (LOAEL to NOAEL) = 1
- UF_{Sub} (subchronic to chronic) = 1
- UF_D (database uncertainty) = 1

Oral RfD = 254/ (10 x 10 x 1 x 1 x 1) = 254/100 = 2.54 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (2.54 x 70 x 0.1)/2 = 8.9 mg/L

B. Cancer

Based on the available data, NPEs are not considered to be carcinogenic. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

NPE does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

NPEs are of moderate toxicity concern to aquatic receptors.

B. Aquatic Toxicity

Acute Studies

Table 2 lists the results of acute aquatic toxicity studies on NPEs. NPEs rapidly degrade to more recalcitrant and toxic common degradants, some of which possess estrogenic activity. As a result, data for nonylphenol monoethoxylate (CAS RN 27986-36-3), which is a common degradant of the chemicals in this group and the most toxic member of the group, are also presented.

Table 2 lists the results of acute aquatic toxicity studies conducted on for a representative nonylphenol ether (NPE).

Table 2: Acute Aquatic Toxicity Studies on NPE

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Pimephales promelas (Fathead minnow0	95-hr LC_{50}	0.128*	-	NICNAS, 2018
Lepomis macrochirus (Bluegill)	96-hr LC_{50}	1.3	-	NICNAS, 2018
Ceriodaphnia dubia (Water flea)	48-hr EC_{50}	0.328*	-	NICNAS, 2019
Daphnia magna	48-hr LC_{50}	1.8	2	ECHA
Pseudokirchneriella subcapitata	48-hr EC_{50}	20-50	2	ECHA

* data for nonylphenol monoethoxylate (CAS RN 27986-36-3)

Chronic Studies

Based on chronic toxicity studies from degradant nonylphenol monoethoxylate (CAS RN 27986-36-3), the 21-day NOEC for Oncorhynchus mykiss (Rainbow trout) is 0.048 mg/L and the 7-day NOEC for Ceriodaphnia dubia is 0.285 mg/L.

The 6-d NOEC for NPE from a chronic study on invertebrates (Daphnia Magna) is 1.0 mg/L. The 96-hr NOEC from an algal (Pseudokirchneriella subcapitata) is 8 mg/L while a 120-hr (5-d) EC_{50} of 37.4 mg/L was determined for green algae (Scenedesmus Opoliensis) (NICNAS, 2018).

Both NPs and short-chain NPEs have been reported to have endocrine activity and cause toxic effects in the reproductive systems of organisms, with NPEs having less activity than NPs (NICNAS, 2018).

C. Sediment Toxicity

The 48-hr LC_{50} to the Gallery worm (Capitella capitata) is 3.26 mg/L (NICNAS).

D. Terrestrial Toxicity

No terrestrial toxicity data was identified for NPE.

E. Calculation of PNEC

The PNEC calculations for NPE follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Using data from the more toxic degradant, acute E(L)C50 values are available for fish (0.218mg/L), and invertebrates (0.328 mg/L). Results are also available from chronic studies on two trophic levels, with NOEC values for fish (0.048 mg/L) and invertebrates (0.285 mg/L). On the basis that the data consists of short-term results from two trophic levels and long-term results from two trophic levels, an assessment factor of 50 has been applied to the lowest reported NOEC value of 0.048 mg/L for fish. The $PNEC_{water}$ is 0.00096 mg/L.

Acute E(L)C_{50} values are available for fish (22,810 mg/L), *Daphnia* (>100 mg/L), and algae (10,940 mg/L). NOEC values from long-term studies are available for fish (15,380 mg/L), invertebrates (8,590 mg/L) and algae (10,000 mg/L). On the basis that the data consists of short-term and long-term results from three trophic levels, an assessment factor of 10 has been applied to the lowest reported E(L)C_{50} value of 100 mg/L for fish. The E(L)C_{50} value is used because the value for fish is lower than the NOEC values for all three trophic levels. The $PNEC_{aquatic}$ is 10 mg/L.

PNEC Sediment

There are limited toxicity data for sediment-dwelling organisms. In addition, no sediment or soil partitioning data were found. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{sediment}$. Based on its properties, NPE is not expected to significantly adsorb to sediment and is subject to rapid degradation. Some of the degradants are highly toxic to aquatic organisms. Therefore, the assessment of this compartment will be covered by the aquatic assessment.

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. In addition, no sediment or soil partitioning data were found. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{soil}$. Based on its properties, NPE is not expected to significantly adsorb to soil and is subject to rapid degradation. Some of the degradants are highly toxic to aquatic organisms. Therefore, the assessment of this compartment will be covered by the aquatic assessment.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

NPEs are readily biodegradable and thus do not meet the screening criteria for persistence.

The measured BCF values in fish for NPEs are <1.4 L/Kg; thus, NPEs do not meet the screening criteria for bioaccumulation.

The NOEC values from chronic aquatic toxicity studies are > 0.1 mg/L for NPE. The acute E(L)C50 values for NPE are > 1 mg/L. Thus, NPE does not meet the criteria for toxicity.

The overall conclusion is that NPEs are not PBT substances.

IX. CLASSIFICATION AND LABELLING

A. Classification

H315: Skin irritation-category 2
H302: Acute toxicity (ingestion)-category 4
H319: Eye irritation-category 2A

B. Labelling

Warning

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 20 to 30 minutes. IMMEDIATELY transport the victim after flushing eyes to a hospital even if no symptoms (such as redness or irritation) develop.

Skin Contact

IMMEDIATELY flood affected skin with water while removing and isolating all contaminated clothing. Wash thoroughly with soap and water.

Inhalation

IMMEDIATELY leave the contaminated area; take deep breaths of fresh air. If symptoms (such as wheezing, coughing, shortness of breath, or burning in the mouth, throat, or chest) develop, call a physician, and be prepared to transport the victim to a hospital.

Ingestion

Rinse mouth with water and then drink plenty of water and IMMEDIATELY call a hospital or poison control centre. Never give anything by mouth to an unconscious person. DO NOT INDUCE VOMITTING.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace Australia exposure standards have not been established for NPE.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

NPEs are considered Australian Dangerous Goods Class 9 for purposes of transportation by road or rail.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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POTASSIUM HYDROXIDE

This dossier on potassium hydroxide (CAS RN 1310-58-3) presents the most critical studies pertinent to the risk assessment of potassium hydroxide in its use in coal seam or shale gas extraction activities. It does not represent an exhaustive or critical review of all available data. The information presented in this dossier was obtained from the OECD-SIDS documents on potassium hydroxide (OECD, 2002) and the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed potassium hydroxide in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment.

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Potassium hydroxide

CAS RN: 1310-58-3

Molecular formula: KOH

Molecular weight: 56.1 g/mol

Synonyms: Potassium hydroxide; caustic potash; potash lye; potassium hydrate

SMILES: [OH-].[K+]

II. PHYSICO-CHEMICAL PROPERTIES

Key physical and chemical properties for the substance are shown in Table 1.

Table 1: Overview of the Physico-Chemical Properties of Potassium Hydroxide

Property	Value	Klimisch score	Reference
Physical state at 20°C and 101.3 kPa	White, crystalline solid	2	ECHA
Melting Point	406°C (pressure not provided) 250°C	2	ECHA
Boiling Point	1,327°C @ 1013 hPa	2	ECHA
Density	2044 kg/m^3 @ 20°C	2	ECHA
Partition Coefficient (log K_{ow})	Not applicable	-	-
Water Solubility	Very soluble	2	ECHA

Potassium hydroxide is a strong alkaline substance that dissociates completely in water to potassium (K^+) and hydroxyl (OH^-) ions.

III. ENVIRONMENTAL FATE PROPERTIES

Potassium hydroxide will be found predominantly in the aquatic environment where it dissociates completely to potassium (K^+) and hydroxyl (OH^-) ions as a result of its high water solubility and low vapour pressure. Both ions are ubiquitous in the environment (UNEP, 1995).

Potassium is an essential nutrient involved in fluid and electrolyte balance and is required for normal cellular function. The hazard of potassium hydroxide for aquatic organisms is caused by the hydroxyl ion (OH-), which has the potential to increase the pH of the aquatic environment, depending on the buffering capacity of the receiving water. In general, the buffer capacity is regulated by the equilibria between CO_2, HCO_3^- and CO_3^{2-}:

$CO_2 + H_2O \leftrightarrow HCO_3^- + H^+$ ($pKa_1 = 6.35$)
$HCO_3^- \leftrightarrow CO_3^{2-} + H^+$ ($pKa_2 = 10.33$)

A release of potassium hydroxide into the aquatic environment from the use of KOH could potentially increase the potassium concentration and the pH in the aquatic environment. Table 2 shows the concentration of potassium hydroxide needed to increase the pH to values of 9.0, 10.0, 11.0 and 12.0.

Table 2: Potassium Hydroxide Concentration (mg/L) Needed to Increase pH to a Value of 9 (OECD, 2002)

Buffer capacity	Concentration of KOH (mg/L)
0 mg/L HCO_3 (distilled water)	0.56
20 mg/L HCO_3 (10^{th} percentile of 77 rivers)	0.86
106 mg/L HCO_3^- (mean value of 77 rivers)	4.51
195 mg/L HCO_3^- (90^{th} percentile of 77 rivers)	8.30

K^+ and OH^- ions will not adsorb on the particulate matter or surfaces and will not accumulate in living tissues (OECD, 2002).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Limited toxicity data exist for potassium hydroxide. Depending on the concentration, solutions of potassium hydroxide are corrosive, irritating, or non-irritating. These solutions cause direct effects to the skin, eyes, respiratory tract, and gastrointestinal tract. Vapours from aqueous solutions of potassium hydroxide can cause respiratory irritation. Potassium hydroxide is not a skin sensitiser. There are no repeated dose, reproductive, and developmental toxicity studies on potassium hydroxide.

B. Metabolism

Potassium hydroxide dissociates completely in aqueous solutions to potassium (K^+) and hydroxide (OH-) ions. Potassium is an essential nutrient involved in fluid and electrolyte balance and is required for normal cellular function (OECD, 2002).

C. Acute Toxicity

Oral

The oral LD50 values in rats for potassium hydroxide have been reported to be 365 milligrams per kilogram (mg/kg) (Johnson et al., 1975; ECHA) and 273 mg/kg (Bruce, 1987; ECHA). [Kl. scores = 2].

Inhalation

No acute inhalation studies are available.

Dermal

No acute dermal toxicity studies are available

D. Irritation

Skin

Application of 0.5 millilitres (mL) of a 5% solution of potassium hydroxide to the skin of rabbits for 4 hours under semi-occlusive conditions was moderately irritating, with a primary dermal irritation indices (PII) score of 4.8 (OECD, 2002). A 10% solution was severely irritating (Nixon et al., 1990; OECD, 2002) [Kl. score = 2]. Application of 0.1 mL of a 5% solution of potassium hydroxide to the skin of rabbits for 24 hours under semi-occlusive conditions was mildly irritating to intact skin (Johnson *et al.*, 1975; OECD, 2002) [Kl. score = 2].

Eye

Instillation of 0.1 mL of a 5% potassium hydroxide solution into the eyes of rabbits for 5 minutes was extremely irritating to corrosive; a 1% KOH solution for 5 minutes or 24 hours was considered irritating; 0.5% potassium hydroxide solution for 24 hours was marginally irritating; and 0.1% potassium hydroxide solution for 24 hours was negative (Johnson et al., 1975; OECD, 2002) [Kl. score = 2].

E. Sensitisation

Potassium hydroxide was not a skin sensitiser in a guinea pig sensitisation test (Johnson et al., 1975; OECD, 2002) [Kl. score = 2]

F. Repeated Dose Toxicity

No studies are available

G. Genotoxicity

In Vitro Studies

Potassium hydroxide was not mutagenic to *S. typhimurium* strains TA 97 and TA 102 in the absence or presence of metabolic activation (ECHA). [Kl. score = 2]

In Vivo Studies

No studies are available.

H. Carcinogenicity

Oral

No studies are available.

Inhalation

No studies are available.

I. Reproductive Toxicity

No reliable studies have been conducted that address female fertility or reproductive toxicity by a relevant route of exposure.

J. Developmental Toxicity

No studies are available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for potassium hydroxide follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

There are no repeated dose, reproductive, and developmental toxicity studies available on potassium hydroxide. Potassium hydroxide dissociates to potassium and hydroxide ions in bodily fluids, and a significant amount of these ions are already ingested in foods. Furthermore, both ions are present in the body and are highly regulated by homeostatic mechanisms. Thus, a toxicological reference value was not derived for potassium hydroxide.

The Australian drinking water guideline value for pH is 6.5 to 8.5 (ADWG, 2011).

B. Cancer

There are no carcinogenicity studies on potassium hydroxide. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Potassium hydroxide does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Aquatic Toxicity

As noted in(OECD, 2002) toxicity tests with potassium hydroxide depend on the buffer capacity of the test medium. Thus, the pH change could influence the speciation of other chemicals and therefore increase and/or decrease the toxicity.

There are no guideline studies on potassium hydroxide; the studies summarised below have Klimisch scores of 3 or 4. Studies on sodium hydroxide (NaOH) have also been included, given its similarity to potassium hydroxide (KOH).

Acute Fish

KOH: The 96-hour LC_{50} to *Gambusia affinis* (mosquito fish) is 80 milligrams per litre (mg/L). At 56 mg/L, no mortality was observed.

NaOH: The 24-hour LC_{50} to *Carassius auratus* (goldfish) is 160 mg/L. At 100 mg/L, which was equivalent to a pH of 9.8, no mortality was observed. The 48-hour LC_{50} to *Leuciscus idus melanotus*, is 189 mg/L. The 96-hour LC_{50} of *Gambusia affinis* (mosquitofish) is 125 mg/L. At 84 mg/L, no effects on the fish were observed. The pH was 9 at 100 mg/L.

Acute Invertebrate

KOH: No studies are available.

NaOH: The 48-hour LC_{50} is 40 mg/L for *Ceriodaphnia cf. dubia*. The toxicity threshold concentration of NaOH for *Daphnia magna* was reported to range from 40 to 240 mg/L.

Acute Algae

No studies are available.

Chronic Toxicity

No studies are available.

B. Terrestrial Toxicity

No studies are available.

VIII. CALCULATION OF PNEC

Based on the available data it is not considered useful to derive a PNEC for potassium hydroxide (OECD, 2002) as:

- The natural pH of aquatic ecosystems can vary significantly between aquatic ecosystems;
- The sensitivity of the aquatic ecosystems to a change of the pH can vary significantly between aquatic ecosystems; and
- The change in pH due to an anthropogenic potassium hydroxide addition is influenced significantly by the buffer capacity of the receiving water.

Based on the information above, PNEC values for water, sediment, and soil were not derived for potassium hydroxide.

IX. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Potassium hydroxide is an inorganic salt that dissociates completely to potassium and hydroxide ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions; both potassium and hydroxide ions are also ubiquitous and are present in most water, soil and sediment. For the purposes of this PBT assessment, the persistent criteria are not considered applicable to this inorganic salt.

Potassium and hydroxide ions are essential to all living organisms, and their intracellular and extracellular concentrations are actively regulated. Thus, potassium hydroxide is not expected to bioaccumulate.

No chronic toxicity data exist on potassium hydroxide; however, the acute LC_{50} values are >1 mg/L in fish, invertebrates and algae. Thus, potassium hydroxide does not meet the screening criteria for toxicity.

The overall conclusion is that potassium hydroxide is not a PBT substance.

X. CLASSIFICATION AND LABELLING

A. Classification

Acute toxicity – category 4
Skin corrosion – category 1A

H302 (Harmful if swallowed)
H314 (Causes severe skin burns and eye damage)

B. Labelling

Danger

C. Pictogram

XI. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

In case of contact, immediately flush eyes with plenty of water for 15 minutes.

Skin Contact

IMMEDIATELY flood affected skin with water while removing and isolating all contaminated clothing. Wash thoroughly with soap and water.

Ingestion

Rinse mouth with water and then drink plenty of water and IMMEDIATELY call a hospital or poison control centre. Never give anything by mouth to an unconscious person. DO NOT INDUCE VOMITTING.

Inhalation

IMMEDIATELY leave the contaminated area; take deep breaths of fresh air. If symptoms (such as wheezing, coughing, shortness of breath, or burning in the mouth, throat, or chest) develop, seek medical attention.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May form explosive mixtures with strong acids. May emit toxic fumes under fire conditions including halogenated compounds, metal oxides/oxides, potassium monoxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment and avoid direct contact.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilled

Soak up with inert absorbent material.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace exposure standard for potassium hydroxide in Australia is 2 mg/m^3 as a peak limitation, with a sensitisation notation. A peak limitation is defined by Safe Work Australia as a maximum or peak airborne concentration of a substance determined over the shortest analytically practicable period of time which does not exceed 15 minutes.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is required. Use a mask or approved air purifying respirator.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Potassium hydroxide is considered Australian Dangerous Goods Class 8 for purposes of transportation by road or rail. Packing Group II or III

XII. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XIII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIV. REFERENCES

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POTASSIUM PERSULFATE

This dossier on potassium persulfate presents the most critical studies pertinent to the risk assessment of potassium persulfate in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed potassium persulfate in an IMAP Tier 1 assessment and considers it to be of low concern[1] .

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): dipotassium peroxodisulphate

CAS RN: 7727-21-1

Molecular formula: $H_2O_8S_2.2K$

Molecular weight: 270.33 g/mol

Synonyms: potassium persulfate; dipotassium peroxydisulfate; Anthion; Peroxydisulfuric acid ([(HO)S(O)2]2O2), potassium salt (1:2); Peroxydisulfuric acid ([(HO)S(O)2]2O2), dipotassium salt

SMILES: [K+].[K+].[O-]S(=O)(=O)OOS([O-])(=O)=O

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-chemical Properties of Potassium persulfate

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Inorganic, odourless, white, crystalline white solid	1	ECHA
Melting Point	Decomposes at 100 °C @ 100.8 kPa	1	ECHA
Boiling Point	Decomposes at 100 °C @ 100.8 kPa	1	ECHA
Density	1390 kg/m^3@ 20°C	1	ECHA
Vapour Pressure	0 Pa @ 25°C*	2	ECHA
Partition Coefficient (log K_{ow})	Not applicable (inorganic substance)	-	ECHA
Water Solubility	52.77 g/L @ 20°C	1	ECHA
Flash Point	Not available because this substance is a solid	-	ECHA
Auto flammability	>600°C** (substance is not expected to be auto flammable)	1	ECHA

[1] https://www.industrialchemicals.gov.au/chemical-information/search-assessments?assessmentcasnumber=7727-21-1

Property	Value	Klimisch Score	Reference
Viscosity	Not available because this substance is a solid	-	ECHA
Henry's Law Constant	Not available because this substance is readily oxidisable in water	-	ECHA

*Inorganic chemicals are outside of the EPIWIN (v.4.0) domain so no experimental determination for vapor pressure was carried out for this substance

**This value was determined using read across for a similar substance (diammonium persulfate, CAS RN 7727-54-0)

III. ENVIRONMENTAL FATE PROPERTIES

Potassium persulfate is known to dissociate completely to the potassium cation (K^{2+}) and persulfate anion ($S_2O_8^{2-}$) when dissolved in water. The persulfate anion, independent of the cation, undergoes further decomposition in normal water or acid conditions which readily oxidizes water to oxygen thus producing sulphate and hydrogen ions. All persulfate decomposition products are ubiquitous to the environment (ECHA).Biodegradation is not applicable to inorganic compounds.

Potassium persulfate has a low potential for bioaccumulation. Persulfates are very soluble in water and are not expected to bioaccumulate in soil or aqueous solutions. (ECHA).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Potassium persulfate is unlikely to become bioavailable in the body regardless of the exposure route. Potassium persulfate has moderate acute oral toxicity, and it has low acute dermal and inhalation toxicity. This substance was reported to be irritating to the skin and slightly irritating to the eyes. Potassium persulfate is a moderate-strong skin sensitiser in animals and humans. There is evidence from occupational studies that potassium persulfate may be a respiratory sensitiser. Potassium persulfate has low systemic toxicity. Potassium persulfate is not genotoxic or carcinogenic. Potassium persulfate is not a reproductive or developmental toxicant.

B. Metabolism

Potassium persulfate will hydrolyse upon contact with water, and it will degrade to eventually form corresponding cations (potassium) and persulfate anions. The persulfate anion, independent of the cation, undergoes further decomposition upon contact with water to form sulfate species. Given these properties, potassium persulfate is unlikely to become bioavailable neither by inhalation, ingestion, or dermal contact. In addition to this, all of potassium persulfate degradation products are physiologically essential to organisms. Therefore, bioaccumulation of potassium persulfate is unlikely in view of its rapid degradation and its high-water solubility (ECHA) [KI. score =1].

The persulfate ion is poorly absorbed from the gastro-intestinal tract, especially when administered in large doses, such that the capacity of specialised transport processes for this ion in the intestines is exceeded. No data were available on the distribution of the persulfate salts in the body. Based on the *in vitro* chemistry of persulfates, the persulfate anion is expected to decompose under *in vivo* conditions to form hydrogen peroxide and sulfate ions. Hydrogen peroxide is rapidly metabolised to oxygen and water by catalase and peroxidase enzymes in mammalian tissues and there is practically no potential for bioaccumulation (OECD, 2005). Sulfate ions are required by the body for the synthesis of sulfur-containing macromolecules. Physiological studies have demonstrated that

sodium, potassium, and ammonium ions are mainly excreted in the urine. Inorganic sulfate is also eliminated from the body, almost entirely by renal excretion (i.e., without biotransformation) (NICNAS, 2020).

C. Acute Toxicity

Oral

Potassium persulfate was tested for acute oral toxicity in male rats in who were administered Dipotassium persulfate by oral gavage as a suspension in corn oil in doses of 2500 mg/kg bw, 1000 mg/kg bw, and 500 mg/kg bw. The acute LD50 value for dipotassium persulfate was determined to be 1130 mg/kg bw. (ECHA)[KI. score =2].

An OECD Guideline 401 (Acute Oral Toxicity) study was conducted using 10 male and 10 female Sprague-Dawley rats exposed to 215, 464, 562, 681, 825, 1,000, 1,210 and 1470 mg/kg disodium peroxodisulphate (CAS RN 7775-27-1) via oral gavage. The rats were observed for four weeks following exposure to disodium peroxodisulphate (CAS RN 7775-27-1). No animal died in the lowest dose group (215 mg/kg bw), two rats (one male and one female rat) died in the intermediate dose group (681 mg/kg bw) and all rats died in the highest dose group (1470 mg/kg bw). Death occurred within 60 minutes until 6 days after application. Surviving animals had recovered after 48 hours after application. Clinical signs included sedation, dyspnoea, diarrhoea, muscular hypotension, reduced feed intake and face-down position. LD_{50}-values of 930 mg/kg bw (males) and 920 mg/kg bw (females) were determined after a 14 days observation period and corresponding LD_0 values of 464 mg/kg in male rats and 562 mg/kg in female rats were revealed (ECHA)[KI. score =2].

The acute oral median lethal dose (LD_{50}) values for the three persulfate salts (in rats) were reported as 495-820 mg/kg bw for ammonium persulfate (Smyth et al, 1969; FMC, 2001), 895-930 mg/kg bw for sodium persulfate (Degussa AG, 1979; as cited in NICNAS 2020) and 1130 mg/kg bw for potassium persulfate (FMC, 1979a as cited in NICNAS 2020). Clinical signs for all persulfates were ocular and oral discharge, irregular breathing, and loss of muscle control (NICNAS, 2020).

Inhalation

Male rats were exposed to 42.9 mg/L of potassium persulfate for one hour. None of the seven test animals died during the 14 days observation period. Thus, the LC_{50} and LC_0 values for inhalation toxicity for dipotassium persulfate were estimated to be greater than >42.9 mg/L and 42.9 mg/L, respectively (ECHA)[KI. score =2].

An EPA OPP 81-3 (Acute inhalation study) was conducted using male and female Sprague Dawley rats exposed to diammonium peroxodisulphate (CAS RN 7727-54-0) via whole body inhalation of dust for 240 minutes. The acute LC_{50} and LC_0 for the 4-hour whole body exposure were greater than >2.95 mg/L and >2.95 mg/L, respectively. The administered concentration was considered the maximum attainable concentration (ECHA) [KI. score =1].

Acute inhalation studies with ammonium, sodium and potassium persulfates performed according to OECD guidelines in rats, indicated median lethal concentration (LC_{50}) values of greater than the maximum attainable concentrations, 2.95 mg/L, 5.1 mg/L and 42.9 mg/L, respectively. Following exposure to high concentrations of persulfates, animals exhibited dyspnoea, respiratory distress and increased nasal, ocular, and oral secretion (FMC 1987, FMC, 1979b; FMC 1995; as cited in NICNAS, 2020).

Dermal

10,000 mg/kg bw of disodium peroxodisulphate (CAS RN 7775-27-1) was administered to male rabbits via a single dermal application. None of the four test animals died during the 14 days observation period. Based on the obtained results, LD_{50} and LD_0 values of >10,000 mg/kg bw and 10,000 mg/kg bw, respectively, were determined (ECHA)[KI. score =2].

As per an EPA OPP 81-2 (Acute dermal toxicity) study, male and female Sprague-Dawley rats were exposed to 2,000 mg/kg bw of diammonium peroxodisulphate (CAS RN 7727-54-0) via occulusive dressing for 24 hours. In this study, the acute LD_{50} and LD_0 values were > 2,000 mg/kg bw and 2,000 mg/kg bw, respectively, in both male and female rats. Under the conditions of this study, diammonium persulfate was considered as non-toxic to both male and female rats when topically applied (ECHA)[KI. score=1].

The acute dermal LD_{50} was >2000 mg/kg bw (rats) for ammonium persulfate (FMC, 1991b), and >10,000 mg/kg bw (rabbits) for sodium and potassium persulfates (FMC, 1979c). Ocular and nasal discharge and slight irritation were reported in animals dermally exposed to high levels of persulfates (FMC, 1979b; as cited in NICNAS, 2020).

D. Irritation

Skin

An OECD Guideline 404 (Acute dermal irritation/corrosion) study was conducted using three Albino-White Russian rabbits exposed to Diammonium persulfate (CAS RN 7727-54-0) via occlusive dressing for four hours. Diammonium persulfate showed formation of severe non-reversible erythema and slight oedema. Based on these results diammonium persulfate was considered irritating to the skin (ECHA)[KI. score =2].

The dermal irritation potential of ammonium persulfate was determined (according to OECD Test Guideline TG404) using six male and female New Zealand White rabbits (CTFA, 1994). No irritation was noted within 72 hours following application. In another study, ammonium persulfate, 0.5 g moistened with 0.1 mL of water was applied under an occlusive patch to the intact and abraded skin of three white Russian rabbits for 4 hours (BGChemie, 1994). Slight oedema, which disappeared within 24 hours, was observed on intact skin, while moderate to severe erythema, moderate oedema, and scab formation were observed at the abraded sites. Ammonium persulfate was considered non-irritating to intact skin. Three brief study reports submitted by industry on sodium persulfate showed at most a slight skin irritant potential in rabbits (FMC, 1979d; FMC, 1980; as cited in NICNAS, 2020).

Standard patch tests have shown 5 % ammonium persulfate to be irritating to human skin (Calnan & Shuster, 1963; Cronin, 1980; as cited in NICNAS, 2020), although a separate study found 1/20 people exhibited an equivocal response when tested with 5 % to 10 % persulfate (Forck, 1968; as cited in NICNAS, 2020). Application of 17.5 % solution of the persulfate salts under an occlusive wrap for four hours was found to cause irritation in 8/46 subjects (Jordan, 1998 cited in CIR, 2001; as cited in NICNAS, 2020).

Eye

An OECD Guideline 405 (Acute Eye Irritation/Corrosion) study was conducted using Albino rabbits exposed to 0.1 mL of diammonium peroxodisulphate (CAS RN 7727-54-0). Conjunctival redness,

obvious swelling with partial eversion of lids plus hypersecretion were observed (in one animal, one hour after application. 72 hours after application full recovery was observed. The irritating index was determined to be 10.5. Under the conditions of this study diammonium persulfate was considered to be slightly irritating to eyes. No systemic-toxic effects were observed, and the general state of the animals was good throughout the study period. (ECHA) [KI. score =1].

In one eye irritation study, ammonium persulfate (0.1 g) was instilled into the conjunctival sacs of the eyes of three white Russian rabbits (BG Chemie, 1996; as cited in NICNAS, 2020). Severe diffused reddening and swelling with hyper-secretion were noticed, and subsided within 72 hours, although clouding of the cornea was still present at this time. Ammonium persulfate was considered slightly irritating to the eye. No irritation scores were available (NICNAS, 2020).

In another study conducted according to the OECD TG 405 (details not available), ammonium persulfate was instilled in the eyes of nine New Zealand White rabbits. The eyes of six animals were not rinsed whereas the eyes of three animals were rinsed 30 seconds after instillation (CTFA 1994; as cited in NICNAS, 2020). Ammonium persulfate caused slight to mild conjunctivitis and iritis in the unrinsed eyes and was considered minimally irritating to these eyes. Ammonium persulfate was practically non-irritating to rinsed eyes. No irritation scores were available (NICNAS, 2020).

In a single unpublished study, sodium persulfate was instilled into the eyes of 8 rabbits. Eye irritation was scored by the Draize method at 24, 48 and 72 h. Slight conjunctivitis was noted at 48 h (FMC, 1979c; as cited in NICNAS, 2020).

E. Sensitisation

Skin

An OECD Guideline 406 (Skin sensitisation) study was conducted using male and female Pirbright white guinea pigs exposed to 0.1% diammonium peroxodisulphate (CAS RN 7727-54-0) via the intradermal route of exposure. After challenge, erythema and oedema were observed in 16 of 20 guinea pigs in the test group, compare to only 3 control animals that revealed slight erythema. All animals remained healthy and gained weight during the study. Under the conditions of this study, the test material diammonium persulfate was considered sensitising to the skin of Guinea pigs (ECHA) [KI. score =2].

There was evidence of delayed contact hypersensitivity in two maximisation tests (OECD TG 406) using ammonium and sodium persulfate in guinea pigs. All test animals reacted positively following challenge by intradermal injection of 0.1 % ammonium persulfate and 80 % of animals were positive following dermal challenge with 1 % ammonium persulfate 14 days later. The corresponding figures for sodium persulfate were 90 % positive for test animals positive following an (non-standard) intracutaneous challenge and 60 % of the test animals were positive following topical challenge (CIR, 2001; BIBRA International, 1997; as cited in NICNAS, 2020).

Sodium persulfate was not sensitising when applied to the skin of guinea pigs in an unpublished Buehler Test, conducted to guideline standards (FMC, 1990b). In a murine local lymph node assay (LLNA), investigators concluded that both ammonium and sodium persulfate were moderate to strong sensitisers with EC3 values (amount of chemical required to elicit a stimulation index of 3) calculated to be 1.9 % and 0.9 % respectively (Cruz et al., 2009 cited in HSDB; as cited in NICNAS, 2020).

Many patch-test studies in human volunteers gave positive response to sodium and ammonium persulfates (Fisher et al., 1976; Pepys et al, 1976; as cited in NICNAS, 2020).

There are strong indications that ammonium, sodium, and potassium persulfate are linked to a variety of skin complaints indicative of sensitisation in occupationally exposed human subjects. In general, persulfates are associated with immediate and delayed contact hypersensitivity, contact urticaria, eczema, dermatoses, and rashes (White et al., 1982; as cited in NICNAS, 2020).

The persulfates caused both delayed-type and immediate skin reactions. These reactions include irritant dermatitis, allergic eczematous dermatitis, localised contact urticaria, generalised urticaria, rhinitis, asthma, and syncope. The most common causes of allergic dermatitis in hairdressers are the active ingredients in hair dyes, and ammonium persulfate has been identified as a frequent allergen. Several occupational case studies document these types of reactions, but no incidence data were available (CIR, 2001; as cited in NICNAS, 2020).

Respiratory

Occupational asthma, rhinitis, bronchitis, and decreased lung function has been widely reported in hairdressers from bleaching powders and industrial workers exposed to persulfate salts. Several occupational studies have been reviewed in CIR (2001; as cited in NICNAS, 2020).

F. Repeated Dose Toxicity

Oral

An OECD Guideline 408 (Repeated Dose 90-Day Oral Toxicity Study in Rodents) subchronic study was conducted using male and female Charles River CR strain rats exposed to 0; 22; 91; 200 mg/kg bw/day disodium persulfate. Observations included body weight, food consumption, blood and urine parameters. Further ophthalmologic examinations and gross and microscopic examinations were carried out. All animals survived the study. Significant differences were seen among the groups in body weights and food consumption. No significant differences were seen among groups in Haematological blood chemical, and urine analytical parameters, and organ weight and body weight ratios. Organ weights, organ-to-body weight ratios and type and frequency of grossly observable lesions seen during necropsy were comparable among the four groups. Intestinal changes were noted in rats which received 3000 ppm of sodium persulfate for 13 weeks. These changes were seen more frequently among females than males. The former received 50 percent more test material than the latter on a dose per body weight basis. No significant changes were seen among the controls or the groups which received 300 ppm, or 1000 ppm in the diet for eight weeks, followed by 5000 ppm in the diet for the remainder of the study. No other microscopic changes were noted on comparison among these three groups. LOAEL and NOAEL values of 200 and 91 mg/kg bw /day (3000 and 1000 ppm), respectively were determined. (ECHA)[KI. score =2].

An OECD Guideline 407 (Repeated Dose 28-day oral toxicity) sub-chronic study was conducted using male Weanling CR-CD albino rats exposed to 0, 12.62, 41.15, and 131.50 mg/kg bw/day (0, 100, 316, and 1000 ppm) potassium persulfate in their feed for 28 days. All test animals showed normal body weight gain and survived the study period. No significant pathology was observed. The NOAEL was determined to be 131.5 mg/kg bw/day (ECHA)[KI. score =2].

The persulfates have low repeat dose toxicity. A 28-day repeated dose oral (dietary) toxicity studies were conducted in rats with all three persulfate salts. The oral doses for the three salts were 0, 100, 316, 1000 ppm (equivalent to 0, 12.6, 41.2, 131.5 mg/kg bw/day for the potassium salt). Tests were performed in male rats only. The NOAEL for sodium and potassium salts were 137 and 131.5 mg /kg

bw/day, respectively (the highest doses tested), while the NOAEL for ammonium persulfate was 41 mg/kg bw/day, based on decreased relative adrenal weight at the highest dose (FMC, 1979a; FMC, 1979b; FMC1979c; as cited in NICNAS, 2020).

Another oral (dietary) subchronic toxicity study using sodium persulfate was conducted in rats. Rats (20/sex/group; strain not provided) were fed rodent chow containing 0, 300, 1000 or 3000 ppm sodium persulfate (0, 23, 100 or 225 mg/kg bw/day) for 90 days. On day 48 of the study, the concentration of the group receiving 1000 ppm was increased to 5000 ppm for the remainder of the study. At the two high dose levels body weight was decreased during the last 6 weeks of treatment (FMC 1979e; as cited in NICNAS, 2020).

There were no treatment-related effects on urinalysis, clinical chemistry, or haematology parameters. Pathological findings were limited to the 3000 ppm group only and consisted of necrosis and atrophy of the gastrointestinal tract epithelial lining. The absence of the gastrointestinal lesions in the group receiving 1000 ppm for 8 weeks, followed by 5000 ppm for 5 weeks, indicates that the lesions are related both to concentration in diet (dose) and length of exposure. There were no treatment related pathological findings in reproductive organs or any other organ system or tissue. A lowest observed adverse effect level (LOAEL) of 3000 ppm (200-250 mg/kg bw/day) was established in this study (CIR, 2001; as cited in NICNAS, 2020)

Inhalation

No inhalation studies were available for potassium persulfate. However, studies were available for other persulfates.

A sub chronic inhalation study was conducted using male and female Sprague-Dawley rats exposed to 0, 5.0, 10.3, and 25 mg/m^3 ammonium persulfate (CAS RN 7727-54-0) via whole body inhalation of dust for 6 hours per day (5 days per week) for 13 weeks There were no exposure-related deaths during the study. Increased respiration rates were noted in both males and females in the 25 mg/m³ group, and in a few animals in the 10.3 mg/m³ group. The incidence of these clinical signs decreased to zero during the first weeks of the recovery period. Body weights for both males and females in the 25 mg/m³ group were significantly depressed during most of the exposure period compared to the control group. By the end of the recovery period, body weights for the exposed animals were similar to the control group values. Lung weights were elevated in the 25 mg/m³ group after 13 wk of exposure but were similar to controls at 6 wk post exposure. Irritation of the trachea and bronchi/bronchioles was noted microscopically after 13 weeks of exposure to 25 mg/m³. These lesions had recovered by 6 wk post exposure. Based on these results, the no-observed-adverse-effect concentration (NOAEC) was 10.3 mg/m³, while the no-observed-effect concentration (NOEC) for exposure of rats to a dust aerosol of ammonium persulfate was 5.0 mg/m³(ECHA) [KI. score =1].

A well conducted 90-day inhalation study using ammonium persulfate gave evidence of inflammation of the airways, reduced body weight gain, rales, increased respiratory rate and increased lung weights (FMC 1998; NICNAS, 2020). In the study, rats (10/sex/group, rat strain not specified) were exposed in whole body chambers to dust aerosol concentrations of 0, 5, 10 or 25 mg/m^3 ammonium persulfate, 6 hours/day, 5 days/week for 13 weeks. Additional groups of 5 animals/sex/group were exposed for 13 weeks followed by a 6-week or 13-week recovery periods. Rales and increased respiratory rates were noted in high dose males and females during the study, and sporadically in the mid-dose group. At 25 mg/m^3, inflammation of the trachea and bronchi/bronchioles, decreased body weights and increased lung weights were found after 13 weeks. These lesions had reversed to normal by the end of the 6-week recovery period. The no

observed adverse effect concentration (NOAEC) in this study was determined to be 10.3 mg/m^3 (NICNAS, 2020).

Pulmonary function tests conducted on employees of a persulfate production facility indicated no adverse effects on pulmonary function at workplace exposure levels, measured at 0.5 mg/m^3 (FMC, 1992; as cited in NICNAS, 2020). Follow-up of these same employees indicated that exposure at 0.5 mg/m^3 had no long-term effects on pulmonary function (Greaves, 1997; as cited in NICNAS, 2020).

Dermal

There are no studies available.

G. Genotoxicity

In vitro Studies

The *in vitro* genotoxicity studies on potassium persulfate are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Potassium Persulfate

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation assay (E. coli WP2 uvr A)	-	-	1	ECHA
Salmonella typhimurium (TA 1538, TA 1535, TA 1537, TA 98, TA 100) **	-	-	2	ECHA
Unscheduled DNA synthesis (rat liver hepatocytes) **	-	-	2	ECHA
Bacterial reverse mutation assay (Salmonella typhimurium TA 1538, TA 1535, TA 1537, TA98, TA 100)	-	-	2	ECHA
DNA damage and repair study (rat liver hepatocytes)	-	-	2	ECHA

*+, positive; -, negative
**Disodium peroxodisulphate (CAS RN 7775-27-1)

In vivo Studies

An OECD Guideline 474 (Mammalian Erythrocytes Micronucleus) test was conducted using male and female ICR mice exposed to 85, 169, 338 mg/kg of disodium peroxodisulphate (CAS RN 7775-27-1) via intraperitoneal exposure. No significant increases in micronucleated polychromatic erythrocytes were observed at 24, 48 or 72 hours after dose administration in males or females. The results of the assay indicated that under the conditions described disodium persulfate did not induce a significant increase in micronucleated polychromatic erythrocytes in male or female ICR mice. Disodium persulfate was concluded to be negative in the mouse micronucleus assay. Thus, disodium persulfate was considered to be not clastogenic (ECHA) [KI. score =2].

An *in vivo/in vitro* unscheduled DNA synthesis test was conducted using male Fischer 344 rats exposed to 41, 164, and 820 mg/kg bw/day disodium peroxodisulphate (CAS RN 7775-27-1) via oral gavage for 2-18 hours. The results of the *in vivo*/*in vitro* UDS assay indicated that under the test

conditions, the test substance did not cause a significant increase in the mean net nuclear grain counts (i.e., an increase of at least 5 counts over the vehicle control) in hepatocytes isolated from treated animals (a negative result). Therefore, disodium persulfate was considered not mutagenic (ECHA)[KI. score =2].

Sodium persulfate was negative in two in vivo genotoxicity studies. Doses of sodium persulfate up to 338 mg/kg injected into mice intraperitoneally did not increase the incidence of micronuclei in bone marrow polychromatic erythrocytes (FMC, 1990c; as cited in NICNAS). Sodium persulfate was found to be non-genotoxic when tested up to 820 mg/kg in an in vivo unscheduled DNA synthesis test in rats (FMC, 1991c; as cited in NICNAS).

H. Carcinogenicity

Oral

There are no studies available.

Inhalation

There are no studies available.

Dermal

An OECD Guideline 451 (Carcinogenicity) study was conducted using female Sencar mice exposed to 200 mg/mL to potassium persulfate twice weekly via dermal exposure (shaved dorsum) for 52 weeks. There was no significant difference observed between the treated group and the control group. Based on the obtained results potassium persulfate was considered neither a tumour promoter nor a carcinogen when applied to the skin (ECHA) [KI. score =2].

In a non-guideline study, female SENCAR mice were exposed dermally twice weekly to 0.2 mL of a 200 mg/mL solution of ammonium persulfate for 51 weeks. The investigators concluded that ammonium persulfate is neither a tumour promoter nor a complete carcinogen when applied to the skin (Kurokawa et al., 1984; as cited in NICNAS, 2020).

I. Reproductive Toxicity

Diammonium peroxodisulphate (APS) was examined for its possible prenatal developmental toxicity in accordance with subacute OECD guideline 414 study. Groups of 26 sperm-positive female Han: Wistar rats were treated with APS by oral administration daily at three dose levels of 10, 30 and 100 mg/kg bw/day respectively from day 5 up to and including day 19 post coitum. A control group of 26 sperm positive females was included and the animals were given the vehicle water. There were no test item related adverse effects on the foetal- and placental weight. There were no test item related external malformations and variations found. The visceral malformations were not attributed to the treatment. The skeletal malformations were found in three foetuses with a statistical significance. However, the incidence was low and the type of alterations less severe and partially different. There was no dose related increase seen in external and skeletal variations. Based on these observations the NOAELs were determined as follows: NOAEL (maternal toxicity): 30 mg/kg bw/day, NOAEL (developmental toxicity): 100 mg/kg bw/day, NOAEL (teratogenicity): 100 mg/kg bw/day (high dose) (ECHA)[KI. score =1].

Diammonium persulfate was tested for oral reproductive/developmental toxicity in a screening test with rats according to OECD guideline 421.The purpose of this study was to obtain initial information

on the possible effects of the test item on reproduction and development when administered orally in the diet to Crl:CD (SD)IGS BR rats at doses of 40, 100 and 250 mg/kg bw/day compared to control animals (plain diet only). There were no treatment-related clinical signs of toxicity observed in F0 parents of either sex or in F1 pups at any treatment level. Remarkable clinical signs in the F0 parents and F1 pups were not attributed to treatment with diammonium persulfate, as they occurred sporadically, were of short duration, and did not demonstrate a dose response. No significant changes were observed in male and female reproductive performance such as gonadal function, mating behaviour, conception, pregnancy, parturition and in development of the F1 offspring from conception to day 4 postpartum. In conclusion, under the conditions of this study, the NOAEL for male and female toxicity, the NOAEL for male and female fertility performance and the NOAEL for F1 viability and development was ≥250 mg/kg/day (ECHA) [KI. score =1].

A one-generation reproductive toxicity study was conducted using male and female rats exposed to 50, 100, 180, or 200 potassium persulfate in their diet. There were no effects on reproductive performance following exposure to the test substance. The NOAEL for systemic toxicity and female reproductive performance was reported to be 50 mg/kg bw/day. The NOAEL for male reproductive performance was reported to be 180 mg/kg bw/day (ECHA)[KI.score=1].

J. Developmental Toxicity

Oral

Diammonium peroxodisulphate (APS) was examined for its possible prenatal developmental toxicity. Groups of 25 (low and mid dose) and 26 (high dose) inseminated New Zealand White rabbits were treated with Diammonium peroxodisulphate (APS) by oral (gavage) administration daily at three dose levels of 10, 30 and 100 mg/kg bw/day respectively from day 6 up to and including day 27 post insemination. A control group of 25 inseminated females was included and the animals were given the vehicle water. There was no test item related mortality, moribund state or abortion observed. In total, on gestation day 28 there were 22, 23, 21 and 20 evaluated litters in the control, 10, 30 and 100 mg/kg bw/day group respectively. There were no test item related clinical signs and pathological macroscopic findings observed. Treatment with the test item at 100 mg/kg bw/day induced maternal toxicity manifest as an initial weight loss and subsequent reduction in body weight gain (77% between GD 6-28). Corrected body weight and corrected body weight gain clearly reflected the effect at 100 mg/kg bw/day. The reduction in body weight correlated with a reduction in food consumption, observed from the start of the treatment. Treatment with the test item at 30 mg/kg bw/day did not induce maternal toxicity. Variations in weight gain were not statistically significant during the study. Variations in the food consumption were not statistically significant at 30 mg/kg bw/day except for GD 18-21. This did not result in statistically lower body weight gain. There was no effect of 10 mg/kg bw/day on maternal body weight or food consumption. There was evidence of an increase in early embryonic death/post-implantation loss/total intrauterine mortality and a slightly lower mean number of viable foetuses (without a statistical significance) in the 100 mg/kg bw/day dose group. This outcome was considered to be related to the severity of the maternal toxicity induced. Significantly lower foetal weight and crown-rump length were observed in the 100 mg/kg bw/day dose group. These smaller foetuses showed evidence of delayed ossification (e.g., larger or slightly larger anterior fontanelle, reduced or asymmetric ossification of the bones of the digits (including pollex) or small hole in xiphoid cartilage. These effects were considered to be a consequence of the maternal toxicity induced. There was no evidence of treatment-related malformation at 100 mg/kg bw/day. There was no effect of treatment at 10 or 30 mg/kg bw/day on foetal growth or development. The severity of the maternal toxicity at 100 mg/kg bw/day was considered to impact foetal viability and growth and to slightly delay ossification. This dose of Diammonium peroxodisulphate (APS) did not induce foetal malformation. The NOAEL for developmental toxicity is 30 mg/kg bw/day (ECHA)[KI. score=1].

A developmental toxicity study was conducted using Wistar rats exposed to 10, 30, 100 mg//kg bw/day potassium persulfate via oral gavage. There were no treatment related effects observed in this study. The NOAEL for maternal toxicity was reported to be 30 mg/kg bw/day. The NOAEL for developmental toxicity was reported to be 100 mg/kg bw/day (ECHA) [KI. score =1].

A developmental toxicity study was conducted using New Zealand white rabbits exposed to 10,30,100 mg/kg bw/day potassium persulfate via oral gavage. The NOAEL for maternal toxicity was reported to be 30 mg/kg bw/day based on body weight gain (ECHA)[KI. score =1].

In a well conducted fertility/developmental study (OECD 421), groups of rats (Crl:CD (SD)IGS BR, 12/sex/group) were administered ammonium persulfate in the diet at doses of 0, 40, 100 and 250 mg/kg bw/day (Weaver, 2004). Animals (both sexes) were dosed two weeks prior to and during mating. Females were administered the substance following mating, throughout gestation and until lactation day 4. In the parental generation group, there were no treatment related clinical signs, effects on body and organ weights or gross lesions. There were no significant adverse effects on the gonads and progression of spermatogenesis, although a non-significant decrease in pregnancy rates was reported at = 100 mg /kg bw/day. On this basis, it was concluded that the NOAEL for fertility indices and reproductive performance was the top dose of 250 mg /kg bw/day. There were no treatment-related clinical signs, mortality or necropsy findings among pups (live birth and viability indices were similar across all groups). There was a slight transient depression in mean pup body weight; however, it was not considered adverse. The developmental toxicity NOAEL determined was the highest dose of 250 mg /kg bw/day (Weaver, 2004; as cited in NICNAS, 2020).

Inhalation

There are no studies available.

Dermal

There are no studies available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for potassium persulfate follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

An OECD Guideline 408 (Repeated Dose 90-Day Oral Toxicity Study in Rodents) sub chronic study was conducted using male and female Charles River CR strain rats exposed to 0; 22; 91; 200 mg/kg bw/day disodium persulfate. All animals survived the study. Significant differences were seen among the groups in body weights and food consumption. No significant differences were seen among groups in Haematological blood chemical, and urine analytical parameters, and organ weight and body weight ratios. Organ weights, organ-to-body weight ratios and type and frequency of grossly observable lesions seen during necropsy were comparable among the four groups. LOAEL and NOAEL values of 200 and 91 mg/kg bw /day (3000 and 1000 ppm), respectively were determined (ECHA)[KI. score =2].

A NOAEL of 91 mg/kg bw/day for repeated dose toxicity will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 10
UF_D (database uncertainty) = 1
Oral RfD = 91/(1 x 10 x 1 x 1 x 1) = 91/1000 = 0.091mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (0.091 x 70 x 0.1)/2 = 0.32 mg/L

Potassium persulfate readily dissociates in aqueous media to the potassium (K^{2+}) and persulfate ($S_2O_8^{2-}$) ions. The persulfate anion, independent of the cation, undergoes further decomposition in normal water or acid conditions which readily oxidizes water to oxygen thus producing sulphate and hydrogen ions. Therefore, the Australian drinking water guideline values for sulphate (250 mg/L) may also apply to potassium persulfate.

B. Cancer

There is limited data available and there is no evidence of carcinogenicity for any persulfate salt including potassium persulfate.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Potassium persulfate does exhibit the following physico-chemical properties:

- Explosivity
- Flammability

It is considered an oxidiser (ECHA).

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Potassium persulfate are of low toxicity concern to aquatic receptors.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on potassium persulfate.

Table 3: Acute Aquatic Toxicity Studies on Potassium Persulfate

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Oncorhynchus mykiss (Rainbow trout)	96-h LC_{50}	76.3 (mortality)*	1	ECHA
Oncorhynchus mykiss (Rainbow trout)	96-h LC_{50}	163 (mortality)*	1	ECHA
Oncorhynchus mykiss (Rainbow trout)	96-h LC_{50}	76.3 (mortality)**	1	ECHA
Daphnia magna	48-h EC_{50}	120 (mobility)*	1	ECHA
Daphnia magna	48-h EC_{50}	120 (mobility)**	1	ECHA
Phaeodactylum tricornutum	72-EC_{50}	320 (growth rate reduction) *	1	ECHA

*Disodium peroxodisulphate (CAS RN 7775-54-0)
** Dipotassium peroxodisulphate (CAS RN 7727-21-1)

Chronic Studies

Table 4 lists the results of chronic aquatic toxicity studies conducted on potassium persulfate.

Table 4: Chronic Aquatic Toxicity Studies on Potassium persulfate

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Daphnia magna	21-d NOEC	20.8 (reproduction)*	1	ECHA
Daphnia magna	21-d NOEC	20.8* (reproduction)**	1	ECHA
Phaeodactylum tricornutum	72-h NOEC	32 (cell growth inhibition and growth rate reduction) *	1	ECHA

*Diammonium peroxodisulphate (CAS RN 7727-54-0)
**Dipotassium peroxodisulphate (CAS RN 7727-21-1)

C. Terrestrial Toxicity

There are no studies available. Persulfates are not expected to be distributed into the terrestrial compartment and consequently not to cause toxicity to terrestrial organisms and plants (ECHA).

D. Calculation of PNEC

The PNEC calculations for Potassium persulfate follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E(L)C_{50} values are available for fish (76.3 mg/L), *Daphnia* (120 mg/L), and algae (320 mg/L). NOEC values from long-term studies are available for invertebrates (20.8 mg/L) and algae (32 mg/L). On the basis that the data consists of short-term results for three tropic levels and long-term results from two trophic levels, an assessment factor of 50 has been applied to the lowest reported E(L)C_{50} value of 20.8 mg/L for invertebrates. The $PNEC_{water}$ is 0.416 mg/L.

PNEC Sediment

There are limited toxicity data for sediment-dwelling organisms. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as potassium persulfate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{sediment}$. Based on its properties, no adsorption of potassium persulfate to sediment is expected and the assessment of this compartment will be covered by the aquatic assessment.

PNEC Soil

There are no toxicity data for terrestrial or soil organisms K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as potassium persulfate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{soil}$. Based on its properties, no adsorption of potassium persulfate to soil is expected. In addition, persulfates are not expected to be distributed into the terrestrial compartment and consequently not to cause toxicity to terrestrial organisms and plants.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Potassium persulfate is an inorganic compound that dissociates completely to ionic species. Biodegradation is not applicable to these compounds. For the purposes of this PBT assessment, the persistent criterion is not considered applicable to potassium persulfate or its dissociated compounds.

Persulfates are very soluble in water and are not expected to bioaccumulate in soil or aqueous solutions. Thus, potassium persulfate does not meet the criteria for bioaccumulation.

The NOECs from the chronic aquatic toxicity studies on potassium persulfate and read-across compounds are > 0.1 mg/L. The acute E(L)C_{50} values from the acute aquatic toxicity studies on potassium persulfate and read-across compounds are > 1 mg/L. Thus, potassium persulfate does not meet the criteria for toxicity.

The overall conclusion is that potassium persulfate is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H302 (Harmful if swallowed)
H315 (Causes skin irritation)
H319 (Causes serious eye irritation)
H334 (May cause allergy or asthma symptoms or breathing difficulties if inhaled)
H317 (May cause an allergic skin reaction)
H335 (May cause respiratory irritation)
H272 (May intensify fire, oxidizer)

B. Labelling

Danger

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace exposure standards for potassium persulfate in Australia is as follows: 0.1 mg/m^3 (peak limitation, time-weighted average).

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Potassium persulfate is considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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POTASSIUM SORBATE

This dossier on potassium sorbate presents the most critical studies pertinent to the risk assessment of potassium sorbate in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed potassium sorbate in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment.[1]

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): potassium (E, E)-hexa-2,4-dienoate

CAS RN:24634-61-5

Molecular formula: $C_6H_8O_2.K$

Molecular weight: 150.22 g/mol

Synonyms: potassium sorbate, potassium (E, E)-hexa-2,4-dienoate

SMILES: CC=CC=CC(=O) [O-]. [K+]

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-chemical Properties of Potassium Sorbate

Property	Value	Klimisch Score	Reference
Physical state at 20oC and 101.3 kPa	Organic, crystalline, white, odorless powder	1	ECHA
Melting Point	This chemical decomposes at temperatures ≥ 205 °C	1	ECHA
Boiling Point	≥ 205°C @ 101.3 kPa	1	ECHA
Density	1.36 (relative density) @ 23.5°C	1	ECHA
Vapour Pressure	< 0 Pa* @ 20°C	1	ECHA
Partition Coefficient (log K_{ow})	1.32 (@ pH 2.5) and -1.72 (@pH 6.5) @ 20°C	1	ECHA
Water Solubility	≥1.95-≤ 543 g/L @ 20°C	1	ECHA
Flash Point	Not applicable	-	-
Auto flammability	178°C	1	ECHA
Viscosity	≥ 17.4-≤ 19.3 mPa s @ 20°C	1	ECHA

[1] https://www.industrialchemicals.gov.au/chemical-information/search-assessments?assessmentcasnumber=24634-61-5+

Property	Value	Klimisch Score	Reference
Henry's Law Constant	2.77 x 10^{-9} Pa m^3/mol @ 20°C	1	ECHA

*Calculated based on conservative estimates using the Antoine equation

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Potassium sorbate is soluble in water, it is readily biodegradable, and it is expected to have negligible bioaccumulation potential. Potassium sorbate is expected to be mobile in soil and it has a high potential to leach into groundwater. However, potassium sorbate is not expected to volatize from water.

B. Biodegradation

In an OECD Guideline 301 D (Ready Biodegradability: Closed Bottle) test on sorbic acid, degradation was 74.9% after 28 days. Thus, potassium sorbate is expected to be readily biodegradable (ECHA) [KI. score =1]. If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

In an OECD Guide 121 (Estimation of the adsorption coefficient K_{oc} on soil and on sewage sludge using high performance liquid chromatography), the estimated log K_{oc} value for sorbic acid was reported to be -1.82 L/Kg at pH 6.0 and 20 °C. The K_{oc} value was reported to be 0.015 L/Kg at 20°C which suggests that potassium sorbate has high mobility in soil, and it has a high potential to leach into groundwater (ECHA) [KI. score =1].

D. Bioaccumulation

A bioconcentration factor (BCF) was estimated for sorbic acid based on its physio-chemical properties. The formula log BCF_{fish} =0.85 x logP_{ow}-0.7 was used to estimate the BCF value for sorbic acid. The BCF value for sorbic acid at pH 2.5 was reported to be 2.6. The BCF value for sorbic acid at pH 6.5 was reported to be 0.007. These values suggest that sorbic acid/potassium sorbate has negligible bioaccumulation potential (ECHA) [KI. score =1].

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Potassium sorbate has low acute oral, inhalation, and dermal toxicity. Potassium sorbate is not a skin irritant, and it is not a skin sensitizer. Potassium sorbate is moderately irritating to the eye of rabbits. Potassium sorbate is not expected to be genotoxic despite mixed findings reported in the *in vitro* studies. This substance is not expected to be carcinogenic nor is there any of evidence that potassium sorbate elicits reproductive toxicity or developmental toxicity.

B. Metabolism

In an EU method B.36 (Toxicokinetic) study 40 and 3000 mg/kg bw/day of 1-14 C radiolabelled surrogate sorbic acid was given to female mice by oral gavage. The mice were observed for four days

following exposure to sorbic acid. Within four days ~80% of the administered dose of sorbic acid was expired as radioactive carbon dioxide, only 2.6-5.4% of sorbic acid was excreted in the urine, and less than 1% was excreted in the faeces. In the urine, 0.7% of the administered dose was recovered as unchanged sorbic acid and 0.2-0.6% was recovered as muconic acid. In this study, the major metabolic pathway for sorbic acid was reported to be oxidation to CO_2 and water. The extrapolation from sorbic acid to potassium sorbate or vice versa is considered not to be restricted in any way, since the determinant of potential toxicity is on the "sorbate" anion (ECHA) [KI. score =2].

In an EU method B. 36 (Toxicokinetic) study 61, 130, 160, 261,287, 277, 500, 587, 825, 888, and 1213 mg/kg bw/day of 1-14 C radiolabelled sorbic acid was given to female Sprague-Dawley rats by oral gavage. The rats were observed for four to twenty hours after treatment. The total recovery of radioactivity was 100% in the low and high dose groups of mice. The major route of metabolism for sorbic acid was via expired CO2 with 85% of the administered dose being recovered as CO2 within 4-10 hours after administration (ECHA) [KI. score =2].

Potassium sorbate is expected to be metabolize rapidly and completely in the gastrointestinal tract (ECHA).

C. Acute Toxicity

Oral

Male and female Sherman rats were fed 0.2 g/ml of sorbic acid and they were observed for 14 days. The reported LD_{50} of 10,500 mg/kg bw/day (ECHA) [KI. score = 2].

Male and female Wistar rats were given 0, 3.8, 5.1, 6.9, 9.3, 12.5, and 16.9 g/kg of sorbic acid by oral gavage and they were observed for seven days. The LD_{50} in males was reported to be 12,500 mg/kg bw and the reported LD_{50} in females was reported to be 9,600 mg/kg bw/day (ECHA) [KI. score = 2].

Inhalation

No reliable inhalation studies available.

Dermal

An OECD guideline 402 (Acute dermal toxicity) test was conducted using male and female Sprague-Dawley rats exposed to sorbic acid via semi occlusive dressing. The LD_{50} was reported to be > 2000 mg/kg bw/day (ECHA) [KI. score =1].

D. Irritation

Skin

An OECD guideline 404 (Acute dermal irritation/corrosion) test was conducted using New Zealand White rabbits exposed to potassium sorbate by semi occlusive dressing. One rabbit had slight erythema and oedema and another rabbit had well defined erythema and oedema one hour after exposure to potassium sorbate. After 24 hours, the individual scores for erythema and oedema in all the rabbits was reported to be zero. Only one rabbits had dry skin 72 hours after exposure to potassium sorbate. The max score for erythema and oedema was reported to be 4 after 24, 48, and 72 hours. Potassium sorbate was reported to be non-irritating to the skin of rabbits (ECHA) [KI. score =1].

Eye

An OECD guideline 405 (Acute Eye irritation/corrosion) test was conducted using New Zealand White rabbits. Approximately 100 mg of potassium sorbate was instilled into the eyes of the rabbits and the other eye was used as a control. The rabbits were observed for 21 days at observation timepoints of 1, 24, 48, 72 hours and day 7, day 14, and day 21. The mean chemosis score was reported to be 2.11, the mean conjunctivae score was reported to be 1.66, and the mean iris score was reported to be 0.44, and the mean cornea opacity score was reported to be 0.44. The rabbits experienced discoloration, swelling and haemorrhage of the conjunctivae after exposure to potassium sorbate. All the observed effects were found to be fully reversible within 7- 21days. Potassium sorbate was reported to moderately irritating to the eyes of rabbits (ECHA) [KI. score =1].

E. Sensitisation

A guinea pig maximization test was conducted according to EU method B.6 (Skin sensitization) using male and female Pirbright-Hartley guinea pigs. There was no evidence of a positive reaction after intradermal injection of 0.1 or 1% sorbic acid. Based on this study sorbic acid is not expected to be sensitizing to the skin of guinea pigs (ECHA) [KI. score =2].

F. Repeated Dose Toxicity

Oral

An OECD guideline 407 (28-day repeated dose toxicity study in rodents) test was conducted using male and female Sprague-Dawley rats exposed to 0, 25,000, 50,000, and 100,000 ppm of sorbic acid in their feed for 28 days. There were no overt clinical signs of toxicity, no mortalities, no treatment related effects on food consumption, nor were there any changes in neurotoxicological measurements in this study. A NOAEL (males and females) of 100,000 ppm was reported in this study. A NOAEL of 9200 mg/kg bw/day was reported for male rats and a NOAEL of 8600 mg/kg bw/day was reported for female rats (ECHA) [KI. score = 1].

An OECD guideline 408 (90-day repeat dose oral toxicity study in rodents) test was conducted using male and female Sprague-Dawley rats exposed to 25,000, 50,000, and 100,000 ppm of sorbic acid in their feed for 90-92 days. There were no overt clinical signs of toxicity, no mortalities, no-treatment related effects on food consumption, and no ophthalmologic findings observed in this study. A NOAEL (males and females) of 100,000 was reported for this study. A NOAEL of 6800 mg/kg bw/day was reported for male rats and a NOAEL of 7200 mg/kg bw/day was reported for female rats (ECHA) [KI. score = 1].

A EU method B.27 (90-day oral repeated dose sub chronic toxicity test in rodents) study was conducted using male and female half cocker, mixed cocker + terrier dogs exposed to 0 and 400,000 ppm of sorbic acid in their feed for 88-91 days. There were no specific abnormalities reported upon gross and histopathological examination of the tissues and evaluation of haematological parameters. A NOAEL of > 40,000 ppm was reported for this study (ECHA) [KI. score = 2].

A chronic toxicity and carcinogenicity study was conducted using male and female Wistar rats exposed to concentrations of 1.5 or 10% sorbic acid in their feed for two years. In the high dose group, a decrease in body weight gain and a decrease of body weight value was observed. However, as the difference from the control was small, this was associated with some reduction in food intake. Food consumption and compound intake showed no consistent differences between treated and control rats, although there were some statistically significant decreases. Haematology examination showed a statistically significant reduction in the total leucocyte count in high dose females at week

27 (individual data not presented in the publication) and a statistically significant increase in the total red blood cells count in the low dose female group at week 52. As no similar changes were found in the males, these findings were determined to be incidental. Clinical chemistry analysis showed no relevant effect data. The high dose males showed a statistically significant increase of urea when compared to the control group. This was related to the normal ageing changes in the rat kidney. The urinary volume of the high dose females showed a slight statistically significant increase at week 13 and 52 when compared to the control. The absolute and relative organ weight of the thyroid in the high dose male group was increased. The animals with increased thyroid showed some signs of advanced renal changes. It is concluded that the heavier thyroids do not represent an effect of sorbic acid on the thyroid but rather an indirect effect of renal damage on the parathyroid. In the relative organ weight analysis, the liver was statistically significantly increased in the high dose male and female group. As demonstrated by the histopathological examination these effects are not definitely hepatotoxic. The kidneys, small intestine and gonads of the high dose females showed a statistically significant increase when compared to the control. In the microscopic pathology the liver of the high dose females showed an increase in focal fatty change, a statistically significantly decreased incidence of bile-duct hyperplasia and a statistically significantly increased incidence of focal necrosis. The fatty change could have resulted from an increased intake of fatty acids. The focal necrosis may have been an indication of an incidental infection, probably of viral origin. In high dose males a statistically significantly decreased incidence of increased extramedullary haematopoiesis in spleen and a decrease of haemosiderin deposition in spleen was observed. Dietary levels up to 10 % sorbic acid caused no carcinogenic effect. Thus, the study failed to detect carcinogenic potential of sorbic acid. A NOAEL of 750 mg/kg bw/day/day and a LOAEL of 5,000 mg/kg/day was reported for this study (ECHA) [KI. score = 2].

A chronic oral toxicity and carcinogenicity study was conducted using male and female ASH/CS1 mice exposed to concentrations of1400, 7000, and 14, 000 mg/kg bw/day bw sorbic acid in their feed for 80 weeks. There were no adverse effects on mortality or the incidence of histological lesions including tumours. The mice that were exposed to 10% sorbic acid experienced a decrease in body weight when compared to control mice. The mice exposed to 5% and 10% sorbic acid had increased kidney weights and increased relative liver weights A NOEL of 1400 mg/kg bw/day was reported for this study (ECHA) [KI. score = 2].

Inhalation

There are no studies available.

Dermal

There are no studies available.

G. Genotoxicity

In vitro Studies

The *in vitro* genotoxicity studies on potassium sorbate are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Potassium Sorbate

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacteria Reverse Mutation Assay (Salmonella typhimurium TA 1535, TA 1537, TA98, and TA 100)	-	-	2	ECHA
Bacteria Reverse Mutation Assay (Salmonella typhimurium TA97a and TA102)	-	-	2	ECHA
In vitro mammalian chromosome aberration test (Chinese hamsters lung fibroblasts or CHL cells)	+	-	2	ECHA
Mammalian cell gene mutation assay (Chinese hamster ovary or CHO cells)	-	-	2	ECHA
DNA damage and repair assay, unscheduled DNA synthesis in mammalian cells (human cell line A 549 American type culture collection no. CCL 185) *	-	-	2	ECHA

*+, positive; -, negative
*sorbic acid

In Vivo Studies

An OECD guideline 474 (mammalian erythrocyte micronucleus test) study was conducted using male and female NMRI mice exposed to 0, 500, 1500, or 5000 mg/kg bw/day of sorbic acid by oral gavage for 72 hours. The mice were observed after 24 hours, 48 hours, and 72 hours post treatment. There were no increases in the number of micro nucleated polychromatic erythrocytes and micro-nucleated normo-chromatic erythrocytes at any of the observation time points. Sorbic acid was reported to be non-genotoxic under the condition of this test (ECHA) [KI. Score =2].

The genotoxic potential of sorbic acid was evaluated in a sister chromatid exchange assay using male and female NMRI mice exposed to 500, 1500, and 5000 mg/kg bw/day of sorbic acid for 24 hours. The mice were observed 24 hours post treatment. Sorbic acid did not induce any sister chromatid exchanges in bone marrow cells at any of the dose levels evaluated. Sorbic acid was non genotoxic under the conditions of this test (ECHA) [KI. Score =2].

H. Carcinogenicity

Oral

A chronic oral toxicity and carcinogenicity study was conducted using male and female ASH/CS1 mice exposed to concentrations of 0,1,5 or 10%, sorbic acid in their feed for 80 weeks. There were no adverse effects on mortality or the incidence of histological lesions including tumours. The mice that were exposed to 10% sorbic acid experienced a decrease in body weight when compared to control mice. Compared with the control this decrease was statistically significant in high dose females. The lower weight was considered to represent only a mildly unfavourable response, since only the mice treated with the highest level of test substance weighed significantly less. Haematology examination showed a statistically significant reduction in the haemoglobin concentration of treated male mice after 13 weeks of administration, for medium dose males also

after 26 weeks and a statistically significant decrease of red blood cells (RBC) for low dose males after 26 weeks. As there were no parallel reductions in the other erythrocyte measurements and the differences were confined to one sex, this incidence did not appear to be treatment related. The higher values for the relative weights of brain, spleen, stomach and small intestine were seen in the absence of any significant differences in the absolute weights and with no indication of any histological change. The increased values for relative heart weights in females are anomalous as there were no comparable changes in the males. It is possible that the increased value at the highest level may reflect the lower body weight. In addition to this, the weight of the hearts in the female controls was slightly less than expected for mice of this size. The increase of relative liver weights cannot be attributed to differences in body weight since some higher values were found in the absolute weights despite the lower body weights. This increase is a reflection of an increase in metabolic demand rather than a toxic effect of Sorbic acid. The increased relative kidney weight does not represent any marked toxic effect of Sorbic acid, as the histological examination found significantly fewer incidences of lesions in the kidney in treated mice than in the control. In the kidneys a statistically significant reduction of perivascular lymphocytes occurred in the treated male and female groups compared to the controls. Also, the kidneys of the treated female groups showed degenerative changes, the low dose group with a statistically significant increase. Early degenerative changes in the liver occurred more frequently in the control than in the treated males. High dose females showed more incidence of early degenerative change in liver than the control. Hyperplastic nodules and amyloids in liver and spleen occurred once in a female mouse administered the 10 % dose. Treated females showed a reduction of follicular cysts in the ovary, with a statistically significant decrease in the medium dose group compared to the control. Chronic inflammation in the lung was found in treated as well as in control females. Most of the types of tumours encountered occurred with a similar or higher frequency in control than in treated mice. One case of a malignant squamous skin epithelioma, although found in a high dose female mouse was a singular observation among 264 treated animals and cannot be construed as a carcinogenic effect since such tumours are known to occur spontaneously. The single mammary adenocarcinoma in a high dose female mouse represents an incidence, which lies in the overall incidence range recorded in females of the same strain of mice at the end of other studies in these laboratories. The squamous-cell carcinoma of the stomach in a low dose male mouse is not considered as an indication for a carcinogenic effect. Overall, dietary levels up to 10 % of sorbic acid for 80 weeks caused no carcinogenic effects in mice. A NOEL of 1400 mg/kg bw/day and a LOAEL of 3750 mg/kg bw/day was reported for this study (ECHA) [KI.score =2].

A chronic toxicity and carcinogenicity study was conducted using male and female Wistar rats exposed to concentrations of 1.5 or 10% sorbic acid in their feed for two years. In the high dose group, a decrease in body weight gain and a decrease of body weight value was observed. However, as the difference from the control was small, this was associated with some reduction in food intake. Food consumption and compound intake showed no consistent differences between treated and control rats, although there were some statistically significant decreases. Haematology examination showed a statistically significant reduction in the total leucocyte count in high dose females at week 27 (individual data not presented in the publication) and a statistically significant increase in the total red blood cells count in the low dose female group at week 52. As no similar changes were found in the males, these findings were determined to be incidental. Clinical chemistry analysis showed no relevant effect data. The high dose males showed a statistically significant increase of urea when compared to the control group. This was related to the normal ageing changes in the rat kidney. The urinary volume of the high dose females showed a slight statistically significant increase at week 13 and 52 when compared to the control. The absolute and relative organ weight of the thyroid in the high dose male group was increased. The animals with increased thyroid showed some signs of advanced renal changes. It is concluded that the heavier thyroids do not represent an effect of sorbic acid on the thyroid but rather an indirect effect of renal damage on the parathyroid. In the

relative organ weight analysis, the liver was statistically significantly increased in the high dose male and female group. As demonstrated by the histopathological examination these effects are not definitely hepatotoxic. The kidneys, small intestine and gonads of the high dose females showed a statistically significant increase when compared to the control. In the microscopic pathology the liver of the high dose females showed an increase in focal fatty change, a statistically significantly decreased incidence of bile-duct hyperplasia and a statistically significantly increased incidence of focal necrosis. The fatty change could have resulted from an increased intake of fatty acids. The focal necrosis may have been an indication of an incidental infection, probably of viral origin. In high dose males a statistically significantly decreased incidence of increased extramedullary haematopoiesis in spleen and a decrease of haemosiderin deposition in spleen was observed. Dietary levels up to 10 % sorbic acid caused no carcinogenic effect. Thus, the study failed to detect carcinogenic potential of sorbic acid. A NOAEL of 750 mg/kg bw/day and a LOAEL of 5,000 mg/kg bw/day was reported for this study (ECHA) [KI. score =2].

Inhalation

There are no studies available.

Dermal

There are no studies available.

I. Reproductive Toxicity

An OECD guideline 416 (two generation reproductive toxicity study) test was conducted using male and female Crj: CD (SD) rats exposed to 0, 300, 1000, or 3000 mg/kg bw/day of sorbic acid by oral gavage. The NOAEL for male and female animals of the P-generation was established at 3000 mg/kg bw/day . The statistically significant reduction of food intake for F0 and F1 dams at 1000 and 3000 mg/kg bw/day , in the presence of caloric substitution by sorbic acid was considered to be the cause of reduced body weight development and slight developmental disturbances (morphological landmarks, learning and memory) of the F1/F2 offspring of the mid and high dose group during lactation. The reason for this effect remains unknown, however nutritional deficiencies in the pups masked by caloric overcompensation in lactating females might be an explanation. The NOAEL concerning effects on development of the conceptus and the offspring (F1-generation) through sexual maturity was established at 1000 mg/kg bw/day . Unscheduled deaths and clinical signs in F1 weanlings selected for mating (observed for 5 juveniles at 3000 mg/kg bw/day and one juvenile at 1000 mg/kg bw/day) during early pre-mating period is not uncommon in oral (gavage) reproduction toxicity studies. When administering excessive dose levels to juveniles as in this case, intolerance to oral gavage treatment often is a more important aspect rather than toxic effects induced by sorbic acid itself - findings that would not be necessarily seen in a corresponding dietary (feeding) study. Hence, these deaths may also be considered as incidental and not treatment related, and therefore are of no toxicological relevance (ECHA) [KI. score = 1]

J. Developmental Toxicity

Oral

An EU method B.31 (Prenatal Developmental Toxicity study) test was conducted using Wistar rats exposed to 3.4-340.0 mg/kg bw/day of potassium sorbate by oral gavage on day 6-16 of mating. The administration of up to 340 mg/kg bw/day of potassium sorbate had no clearly discernible effect on nidation or on maternal or foetal survival. The number of abnormalities seen in either soft or skeletal tissues of the test groups did not differ from the number occurring spontaneously in the

sham-treated controls. A LOAEL and NOAEL of 340 mg/kg bw/day was reported for maternal toxic effects. A LOAEL and NOAEL of 340 mg/kg bw/day was reported for embryotoxic and teratogenic effects (ECHA) [KI. score = 2].

An OECD guideline 414 (prenatal developmental toxicity study) test was conducted using Himalayan rabbits exposed to 10 mL/kg bw/day of sorbic acid by oral gavage from day 6-29 of gestation. There were no teratogenic properties observed up to a dose level of 1000 mg/kg bw/day . There were no treatment-related maternal or developmental effects were observed at 300 mg/kg bw/day . Maternal findings in the mid dose group included increased respiratory rate following administration, decreased body weight gain and rough surface of the spleen. Maternal findings in high dose females included increased respiratory rate following administration, death, abortion, decreased body weight and body weight gain, marked decrease in food consumption and pathological findings upon necropsy (rough surface and reduced size of the spleen). Statistically significant reductions in mean foetal and placental weights and the viability of the foetuses were observed at the mid and high dose levels. At 1000 mg/kg bw/day , marginal statistically significantly increased incidences of unclassified macroscopic variations (abdominal distension caused by an inflated gastric tract) and skeletal variations (less than 7 lumber vertebral bodies ossified) occurred. Abdominal distension was noted in two dams where all foetuses were affected and was regarded as not related to sorbic acid exposure. At the high dose level, causing severe maternal toxicity, increased post-implantation loss, severely reduced viability of foetuses, increased incidences of malrotation of fore paws, domed head, accessory 13th ribs, skeletal retardations according to Dawson and soft tissue variations of the head according to Wilson were recorded. However, it did not appear justified to draw any valid conclusion on teratogenic properties at the highest dose level of this study. Slight or severe maternal toxicity observed at the mid and high dose level, respectively, and severely reduced food consumption at both dose levels indicated malnutrition. This normally results in inadequate intake of calcium and micronutrients like trace elements and vitamins, which are required for normal development of the foetuses. The malnutrition resulted in premature death of dams, retarded development of the foetuses and reduced viability of foetuses in the highest dose group. As a reason, it was considered that sorbic acid was administered over the test period by single gastric intubations per day. Necropsy revealed gastric lesions in all deceased animals. Since sorbic acid is known to have irritant properties, such lesions are probably attributable to administration of a large quantity of the irritant test article by gastric intubation. Furthermore, it cannot be excluded that administration of large quantities of an antimicrobial substance resulted in disturbance of the intestinal microflora which, in turn, would result in deficiencies in nutrients, in particular for the rabbit species. It should, in contrast, be noted that in feeding studies for rodents and dogs (with human-like intestinal function), high doses of sorbic acid are well tolerated, which supports these conclusions. The LOAEL for maternal effects was reported to be 1000 mg/kg bw/day and the NOAEL for maternal effects was reported to be 300 mg/kg bw/day . A LOAEL of 1000 mg/kg bw/day was reported for embryotoxic or teratogenic effects and a NOAEL of 300 mg/kg bw/day was reported for embryotoxic or teratogenic effects (ECHA) [KI. score = 1].

Inhalation

There are no studies available.

Dermal

There are no studies available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for potassium sorbate follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

A prenatal developmental toxicity study provided the basis for the NOAEL of 300 mg/kg bw/day bw/day reported in rabbits. The NOAEL of 300 mg/kg bw/day will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 10
UF_D (database uncertainty) = 1
Oral RfD = 300/ (1 x 10 x 1 x 1 x 1) = 300/1000 = 0.30 mg/kg bw/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (0.30 x 70 x 0.1)/2 = 1.05 mg/L

B. Cancer

There is no evidence that potassium sorbate is carcinogenic. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Potassium sorbate does not exhibit the following physico-chemical properties:

- Explosivity

- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Potassium sorbate has low toxicity to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on potassium sorbate.

Table 3: Acute Aquatic Toxicity Studies on Potassium Sorbate

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Danio rerio	96-hour LC_{50}	>500 (mortality)	1	ECHA
Oncorhynchus mykiss	96-hour LC_{50}	>1,000 (mortality)	1	ECHA
Danio rerio	96-hour LC_{50}	1,250 (mortality)	2	ECHA
Daphnia magna	48-hour EC_{50}	982 (mobility)	1	ECHA
Daphnia magna	48-hour EC_{50}	750 (mobility)	2	ECHA
Desmodesmus subspicatus	48-hour EC_{50}	480 (growth rate)	3	ECHA

Chronic Studies

Table 4 lists the results of chronic aquatic toxicity studies conducted on sorbic acid.

Table 4: Chronic Aquatic Toxicity Studies on Sorbic Acid

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
*Desmodesmus subspicatus**	72-hour NOEC	8.46	3	ECHA
Daphnia magna	21-day NOEC	50	1	ECHA

C. Terrestrial Toxicity

An OECD guideline 207 (Earthworm, Acute Toxicity) test was conducted using *Eisenia fetida* exposed to sorbic acid in their soil for 14 days. Sorbic acid did not cause any adverse effects on mortality or body weight. The 14-day LC_{50} was reported to be 675 mg/kg soil dw and the 14-day NOEC was reported to be 455 mg/kg soil dw. However, these endpoint values were recalculated and the resulting 14-day LC_{50} was reported to be 864 mg/kg soil dry weight and the 14-day NOEC was reported to be 582 mg/kg soil dry weight (ECHA) [KI. score=1].

Guideline ISO 22030 (2005) was used to evaluate the toxicity of potassium sorbate to terrestrial plants Brassica rapa and Avena sativa for 44 days using natural soil. The 31-day NOEC for Brassica

rapa was reported to be ≥ 100 mg/kg soil dw. The 39-day NOEC for Avena sativa was reported to be ≥ 100 mg/kg soil dw (ECHA) [KI. score =1].

D. Calculation of PNEC

The PNEC calculations for potassium sorbate follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E(L)C_{50} values are available for fish (>500 mg/L), *Daphnia* (750 mg/L), and algae (480 mg/L). NOEC values from long-term studies are available for invertebrates (50 mg/L) and algae (8.46 mg/L). On the basis that the data consists of short-term results from three tropic levels and long-term results from two trophic levels, an assessment factor of 50 has been applied to the lowest reported NOEC value of 8.46 mg/L for algae. The NOEC value is used because the value for algae is lower than the NOEC values for both trophic levels. The $PNEC_{water}$ is 0.169mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 0.106 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (0.8/1280) \times 1000 \times 0.169$
$= 0.106$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1000 \times BD_{solid}]$
$= 0.8 + [(0.2 \times 0.0006/1000 \times 2400]$
$= 0.8\ m^3/m^3$

Where:
Kp_{sed} = solid-water partition coefficient (L/kg)
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]
$Kp_{sed} = K_{oc} \times f_{oc}$
$= 0.015 \times 0.04$
$= 0.0006$ L/kg

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for potassium sorbate was estimated to be 0.015 L/Kg using an OECD Guide 121 (Estimation of the adsorption coefficient K_{oc} on soil and on sewage sludge using high performance/ liquid chromatography) test (ECHA)[KI. score =1].
F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are only two toxicity studies using terrestrial receptors or soil organisms. The NOEC for earthworms is 582 mg/kg soil dw and the NOEC for plants is ≥ 100 mg/kg soil dw. Given the limited data for the soil compartment, an assessment factor of 100 was applied to derive the $PNEC_{soil}$ value of 1 mg/kg soil dry weight.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Potassium sorbate is readily biodegradable and thus does not meet the screening criteria for persistence.

The measured BCF in fish is 0.007 at pH 6.5. Thus, potassium sorbate does not meet the criteria for bioaccumulation.

The NOECs from the chronic aquatic toxicity studies on potassium sorbate are > 0.1 mg/L. The acute $E(L)C_{50}$ values from the acute aquatic toxicity studies on potassium sorbate are > 1 mg/L. Thus, potassium sorbate does not meet the criteria for toxicity.

The overall conclusion is that potassium sorbate is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H319: Causes serious eye irritation (Eye irritation-category 2)

B. Labelling

Warning

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

There are no workplace exposure standards for potassium sorbate in Australia.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Potassium sorbate is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

ADWG. (2011). National Water Quality Management Strategy. Australian Drinking Water Guidelines, Section 6, Australian Government, National Health and Medical Research Council, Natural Resource Management Ministerial Council. Updated January 2022. Available: https://www.nhmrc.gov.au/about-us/publications/australian-drinking-water-guidelines

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QUATERNARY AMMONIUM COMPOUNDS, BIS (HYDROGENATED TALLOW ALKYL), DIMETHYL, SALTS WITH BENTONITE

This dossier is for quaternary ammonium compounds, bis (hydrogenated tallow alkyl), dimethyl, salts with bentonite (CAS RN 68953-58-2). For the purposes of this dossier, this substance will be referred to as dialkyl chain quaternary ammonium compound [2M(2Alk)] bentonite.

This dossier presents the most critical studies pertinent to the risk assessment of 2M(2Alk) bentonite in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the OECD-SIDS Initial Assessment Profile on the Organoclays Category (OECD, 2007). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): quaternary ammonium compounds, bis (hydrogenated tallow alkyl), dimethyl, salts with bentonite

CAS RN: 68953-58-2

Molecular formula: Unspecified

Molecular weight: Unspecified. Substance is a UVCB.

Synonyms: Quaternium-18 Bentonite; dialkyl chain quaternary ammonium compound [2M(2Alk)] bentonite; bis (hydrogenated tallow alkyl) dimethylammonium bentonite

SMILES: Not applicable. Substance is a UVCB.

II. PHYSICO-CHEMICAL PROPERTIES

2M(2Alk) bentonite is one of a group of organoclays composed of quaternary ammonium compounds (cations) that have the following general formula:

N+R1, R2, R3, R4

Where R1, R2, R3, and R4 are substitutions on the N (nitrogen atom) of the quaternary compound (salt) as follows:

- Methyl – 1 or 2 substitutions
- Benzyl – 0 or 1 substitutions
- Alkyl (C14-22) – 1, 2 or 3 substitutions

The organoclays discussed in this dossier are hydrogenated tallowalkonium bentonites and are the product of the reaction of hydrogenated tallowalkonium chloride and bentonite. Bentonite is a widely distributed natural material consisting predominantly of the clay montmorillonite, a smectite clay. Bentonite is formed of highly colloidal and plastic clays and is produced by in-situ devitrification of volcanic ash (CIR, 2016).

Organoclays, such as 2M(2Alk) bentonite, are free flowing solid powders that are essentially insoluble in water, in organic solvents and in lipids. They are not volatile under ambient conditions. The organoclays do not melt or boil, although some degradation may occur when subjected to extreme heat at about 180^0C to 600^oC. The densities range from 1,400 to 1,800 kg/m^3 (temperature not provided) (OECD, 2007).

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

The clay component of 2M(2Alk) bentonite is not biodegradable, and the organic component is not readily biodegradable. Bioaccumulation is not expected due to the insolubile nature of 2M(2Alk) bentonite. Quaternary ammonium ions are tightly held to the clay, resulting in organoclay compounds ("salts") that are very hydrophobic in nature (OECD, 2007).

B. Biodegradation

No biodegradation studies were located for 2M(2Alk) bentonite. Biodegradation studies are available for quaternary ammonium compounds, benzylbis (hydrogenated tallow alkyl)methyl, chlorides, compounds with bentonite [also referred to as B(2Alk)M bentonite].

In three separate OECD TG 306 biodegradation tests using B(2Alk)M bentonite, biodegradation ranged from 4.7 to 33.4% in 28 days (OECD, 2007). Based on these data as well as the structural and chemical properties of these compounds, it is assumed that other organoclay category members will also show limited biodegradation. It should be noted that biodegradation relates only to the organic component of the organoclays (i.e., the alkyl quaternary ammonium salts).

C. Environmental Distribution

Quaternary ammonium ions are tightly held to the clay, resulting in organoclay compounds ("salts") that are very hydrophobic in nature (OECD, 2007).

D. Bioaccumulation

Bioaccumulation is not expected due to the insolubility of 2M(2Alk) bentonite (OECD, 2007).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

2M(2Alk) bentonite has low acute toxicity and is excreted rapidly. This substance is not irritating nor is it a skin sensitiser.

No systemic effects were observed in repeat dose oral or dermal toxicity studies. It is not genotoxic, carcinogenic, nor is it a reproductive or developmental toxicant.

B. Metabolism

2M(2Alk) bentonite are not expected to be absorbed following oral (gavage) and it will be excreted rapidly via faeces with negligible elimination from the urine and bile. There is no evidence of any tissue retention or systemic uptake of this substance. This substance is not expected to be respirable based on the particle size nor is it is expected to be absorbed through the skin.

C. Acute Toxicity

Acute toxicity studies demonstrate a low order of toxicity with an inhalation 4-hr LC_{50}S and an oral gavage LD_{50}S greater than 5.0 milligrams per litre (mg/L) and 5,000 milligrams per kilogram body weight (mg/kg/bw) respectively.

The oral LD_{50} for 2M(2Alk) bentonite is >8,000 mg/kg in rats (CIR, 1982) [Kl. score = 4]. The inhalation LC_{50} for 2M(2Alk) bentonite is >5.7 mg/L in rats for a 4-hr. 22-min. exposure. There were no mortalities, and the particle size was $\geq$ 10 micrometre (μm), 30% $\leq$ μm (CIR, 2016) [Kl. score = 4].

No acute dermal toxicity studies are available.

D. Irritation

2M(2Alk) bentonite is not irritating to the skin. Eye irritation is generally minimal in human and moderate in animals.

Application of 0.5 g. 2M(2Alk) bentonite to the skin of rabbits for 6 hours/day for five consecutive days, followed by 10 days of rest and then five more days of exposure, did not result in any signs of irritation (CIR, 1982) [Kl. score = 4].

Instillation of 0.1 mL of a 10% suspension of 2M(2Alk) bentonite in physiological saline produced no signs of irritation (CIR, 1982) [Kl. score = 4].

E. Sensitisation

2M(2Alk) bentonite was not considered a skin sensitiser when tested in a guinea pig sensitisation test (CIR, 1982) [Kl. score = 4].

F. Repeated Dose Toxicity

Oral

Rats were fed diets containing 0, 1%, 5%, or 25% 2M(2Alk) bentonite for 12 weeks. There was a depression of growth rate at the 25% test substance dose level, being somewhat more marked in males. There were no treatment-related effects seen in the hematology parameters, organ weights, gross pathology, or histopathology. Assuming 1% in the diet translated to about 500-1,000 mg/kg-bw/day, the no observed adverse effect level (NOAEL) (25% in the diet) was determined to be approximately 12,500 to 25,000 mg/kg-bw/day, the highest dose tested (OECD, 2007). [Kl. Score = 4].

Inhalation

No studies available.

Dermal

Rabbits were administered to the skin under occlusive conditions 0.5 g 2M(2Alk) bentonite for 6 hours/day for 90 days. There was no evidence of local or systemic toxicity (CIR, 1982) [Kl. score = 4].

G. Genotoxicity

In vitro Studies

The *in vitro* genotoxicity studies on 2M(2Alk) bentonite are presented in Table 1.

Table 1: *In vitro* Genotoxicity Studies on 2M(2Alk) bentonite

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Mouse lymphoma cells	-	-	-	OECD SIDS 2007
Bacterial reverse mutation assay	-	-	-	OECD SIDS 2007

*+, positive; -, negative

In vivo Studies

No studies are available for 2M(2Alk) bentonite. However, this substance is not expected to be mutagenic based on read across to B(Alk)2M bentonite (OECD, 2007).

H. Carcinogenicity

There are no data regarding the carcinogenicity of 2M(2Alk) bentonite. However, the impurity, respirable crystalline silica which may be present at 0.1-5% is considered a known human carcinogen (Group 1 according to IARC) (OECD, 2007).

I. Reproductive Toxicity

2M(2Alk) bentonite is not expected to be a reproductive toxicant based on results from a one-generation reproduction study using another organoclay substance [B(2Alk)M hectorite] at dose levels of 0, 50, 225, and 1,000 mg/kg bw/day in rats. There were no treatment-related effects on adults or litters at any dose level. The parental and F1 offspring NOAEL was 1,000 mg/kg bw/d, the highest dose tested (OECD, 2007).

J. Developmental Toxicity

2M(2Alk) bentonite is not expected to be a developmental toxicant based on results from a one-generation reproduction/developmental toxicity study using another organoclay substance [B(2Alk)M hectorite] at dose levels of 0, 50, 225, and 1,000 mg/kg bw/day in rats. There were no treatment-related effects on litters at any dose level. The only statistically significant effect was a reduction in group mean litter weight from day 7 to 21 of lactation caused by a slightly reduced group mean litter size at 1,000 mg/kg bw/d. This effect was not considered to be of toxicological significance. There were no effects on mean individual offspring weights. There were no toxicologically significant findings for all parameters evaluated; the NOAEL was 1,000 mg/kg bw/d, the highest dose tested (OECD, 2007).

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKIN WATER GUIDANCE VALUES

The toxicological reference values developed for 2M(2Alk) bentonite follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

2M(2Alk) bentonite has been tested in a rat 12-week dietary study. The NOAEL was 12,500 mg/kg bw/day, the highest dose tested. This NOAEL will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 3
UF_D (database uncertainty) = 1
Oral RfD = 12500/(10 x 10 x 1 x 3 x 10) = 12500/300 = 41.67 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (41.67 x 70 x 0.1)/2 = 146 mg/L

B. Cancer

There are no carcinogenicity studies on 2M(2Alk) bentonite. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

2M(2Alk) bentonite does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

2M(2Alk) bentonite displays low acute aquatic toxicity.

B(Alk)2M bentonite has low acute toxicity to fish and invertebrates, with likely low acute toxicity to algae. A chronic *Daphnia* study conducted on an organoclay similar to B(Alk)2M bentonite suggests that these compounds may have moderate chronic toxicity concerns for aquatic organisms. However, the toxicity observed in the study has been due, in part, to the physical effects of the organoclay test material. B(Alk)2M bentonite is virtually non-toxic to terrestrial organisms.

B. Aquatic Toxicity

Acute Studies

Table 2 lists the results of acute aquatic toxicity studies conducted on 2M(2Alk) bentonite and similar organoclays.

Table 2: Acute Aquatic Toxicity Studies on 2M(2Alk) bentonite and Similar Organoclays

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Freshwater rainbow trout	96-hour LC_{50}	>ca. 500**	4	OECD SIDS, 2007
Daphnia magna	48-hour EC_{50}	>100	4	OECD SIDS, 2007
Daphnia magna	96-hour EC_{50}	300 **	4	OECD SIDS, 2007
Daphnia magna	48-hour EC_{50}	<500**	4	OECD SIDS, 2007
Skeletonema costatum	72-hour EC_{50}	23.8** (growth rate)	4	OECD SIDS, 2007
Skeletonema costatum	72-hour EC_{50}	82.3 (growth rate)	4	OECD SIDS, 2007
Skeletonema costatum	72-hour EC_{50}	>1,000** (growth rate)	4	OECD SIDS, 2007
Scenedesmus subspicatus	72-hour EC_{50}	>100 (growth rate)***	4	OECD SIDS, 2007

*Only one concentration was used
** Test material was B(2Alk)M bentonite (CAS No. 68153-30-0)
*** Test material was B(2Alk)M hectorite (CAS RN 121888-67-3).

Chronic Studies

No chronic studies are available on 2M(2Alk) bentonite. The 21-day no observed effect concentration (NOEC) in a *Daphnia* reproduction test on B(2Alk)M hectorite was 3.2 mg/L (OECD, 2007). The mortality of *Daphnia* seen at the LOEC of 32 mg/L was considered to be due, in part, to physical effects of the test material.

The 72-hour NOEC in a *Scenedesmus subspicatus* OECD TG201 toxicity test on B(2Alk)M hectorite was 100 mg/L, based on growth rate (OECD, 2007).

C. Terrestrial Toxicity

The 14-day NOEC of another organoclay substance [B(Alk)2M bentonite] to earthworms is 1,000 mg/kg. Since 1,000 mg/kg is the limit dose, it is assumed that the LC_{50} is >1,000 mg/kg (OECD, 2007).

Terrestrial plant toxicity are available for B(2Alk)M hectorite (CAS No. 12188-67-3). The EC_{50} values of B(2Alk)M hectorite for the emergence and early growth stages of wheat and radish seedlings (*Tritium aestivum* and *Raphanus sativus*, respectively) are >100 mg/kg; the NOEC are 100 mg/kg, the

highest dose tested (OECD, 2007). The LC_{50} of B(2Alk)M hectorite was 9 mg/kg for the emergence and early growth stages of cress seedling (*Lepidum sativum*); the LOEC was 1 mg/kg, and a NOEC was not established (OECD, 2007).

D. Calculation of PNEC

The PNEC calculations for 2M(2Alk) bentonite follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E(L)C_{50} values are available for fish (500 mg/L), invertebrates (100 mg/L), and algae (100 mg/L). Chronic NOEC values are available for invertebrates (3.2 mg/L) and algae (100 mg/L). On the basis that the data consists of short-term results from three trophic levels and long-term results from two trophic levels, an assessment factor of 50 has been applied to the lowest reported E(L)C_{50} value of 3.2 mg/L for invertebrates (daphnia). The $PNEC_{aquatic}$ is 0.064 mg/L.

PNEC Sediment

No experimental toxicity data on sediment organisms are available. The K_{ow} of 2M(2Alk) bentonite cannot be calculated because it is essentially insoluble in water. Thus, the equilibrium partition method cannot be used to determine a $PNEC_{sediment}$.

PNEC Soil

No experimental toxicity data on terrestrial or soil organisms are available for 2M(2Alk) bentonite. Experimental results are available for two trophic levels for other organoclay substances in the group. An acute LC_{50} value is available for earthworms (>1,000 mg/kg). Results from the long-term studies are only available for terrestrial plants, which give widely divergent results. On the basis that the data consist of one short-term result from one trophic level, an assessment factor of 1,000 has been applied to the acute LC_{50} value of 1,000 mg/kg for earthworms. The $PNEC_{soil}$ is 1.0 mg/kg soil dry weight.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

2M(2Alk) bentonite is not readily biodegradable and thus does not meet the screening criteria for persistence.

2M(2Alk) bentonite is insoluble in water and is not bioavailable. Thus, it is not expected to meet the screening criteria for bioaccumulation.

There are no chronic aquatic toxicity studies available on 2M(2Alk) bentonite; however, the NOEC from a chronic *Daphnia* study on a similar organoclay is >0.1 mg/L. The acute EC_{50} values for 2M(2Alk) bentonite and similar organoclays are >1 mg/L in fish, invertebrates and algae. Thus, it does not meet the criteria for toxicity.

The overall conclusion is that 2M(2Alk) bentonite is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Not Classified

B. Labelling

None

C. Pictogram

None

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established a value for this substance.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

2M(2Alk) bentonite is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

Australian Drinking Water Guidelines (ADWG). (2011). National Water Quality Management Strategy. Australian Drinking Water Guidelines, Section 6, Australian Government, National Health and Medical Research Council, Natural Resource Management Ministerial Council. Updated January 2022. Available: https://www.nhmrc.gov.au/about-us/publications/australian-drinking-water-guidelines

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SODIUM (C14-16) OLEFIN SULFONATE

This dossier on sodium (C14-16) olefin sulfonate (CAS RN 68439-57-6) presents the most critical studies pertinent to the risk assessment of the substance in its use in coal seam gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Sulfonic acids, C14-16 alkane hydroxy and C14-16-alkene, sodium salts

CAS RN: 68439-57-6

Molecular formula: C(4+2n)H(9+4n)SO4Na C(4+2n)H(7+4n)SO4Na n = 5-6

Molecular weight: 298.42 – 344.49 g/mol (Substance is a UVCB)

Synonyms: Sodium C14-16 olefin sulfonate; sodium C14-16-alkane hydroxy and C14-16-olefin sulfonates; alkenes, C14-16 alpha-, sulfonated, sodium salts; sodium tetradecenesulfonate; sodium α-olefin sulfonate sodium (C14-16) olefin sulfonate

Smiles: CCCCCCCCCCCC=CCS(=O)(=O)[O-].[Na+]

II. PHYSICO-CHEMICAL PROPERTIES

Sodium (C14-16) olefin sulfonate is an anionic surfactant. It is a mixture of long chain sulfonate salts prepared by sulfonation of C14-16 alpha olefins. Key physical and chemical properties for the substance are shown in Table 1.

Table 1: Overview of the Physico-Chemical Properties of Sodium (C14-16) Olefin Sulfonate

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Solid white powder	-	ECHA
Melting Point	≥240 °C @ 101.3 kPa		ECHA
Boiling Point	Not applicable		ECHA
Density	1054 kg/m^3 @ 20°C		ECHA
Vapour Pressure	≤5.87 x 10^{-6} Pa @ 25°C	-	ECHA
Partition Coefficient (log K_{ow})	-1.3 @ 20°C	-	ECHA
Water Solubility	292 g/L @ 20°C		ECHA
Flash Point	Not applicable as substance is solid	-	ECHA
Auto flammability	372.9°C @ 101.3 kPa	-	ECHA
Viscosity	Not applicable as substance is solid	-	ECHA

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Sodium (C14-16) olefin sulfonate is readily biodegradable. It is not expected to bioaccumulate. It has a low potential to adsorb to soil or sediment.

B. Biodegradation

Sodium (C14-16) olefin sulfonate is readily biodegradable. Several biodegradation tests are available for alpha olefin sulfonates. The key study investigates the biodegradation of sulfonic acids, C14-16 (even numbered)-alkane hydroxy and C14-16 (even numbered)-alkene, sodium salts in a modified Sturm test according to OECD guideline 301B using domestic activated sludge as inoculum. After 28 days the test substance was degraded by 80 % (ECHA) [Kl. Score = 2]. Hence, the test substance is readily biodegradable according to OECD criteria (ECHA).

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

No experimental data are available for sodium (C14-C16) olefin sulfonate. It was determined that an adsorption / desorption test was not required because the test substance decomposes rapidly (ECHA). Using KOCWIN in EPISUITE™ (EPA, 2019), the estimated K_{oc} value for sodium (C14-C16) olefin sulfonate is 1.607 (if the experimental log $K_{ow}$of -1.3 is entered into the program) (ECHA) [Kl. Score = 2]. However, one should keep in mind that surfactancy (the fact that surfactants tend to stay in the boundary layer between the phases) and dissociation is not considered in the EPISUITE™ estimations. Therefore, calculated K_{oc} values should be used with caution (ECHA).

If released to soil, based on this K_{oc} value, the substance is expected to have very high mobility. If released to water, based on the K_{oc} value and its water solubility, the substance is not expected to adsorb to suspended solids and sediment.

D. Bioaccumulation

Sodium (C14-16) olefin sulfonate has a low potential for bioaccumulation as indicative of a log K_{ow} of -1.3 (ECHA).

A bioconcentration test with aquatic organisms is not available for the test substance. Based on the experimental log K_{ow} of -1.3, the test substance has a low bioaccumulation potential. This assumption is confirmed by the SIDS Initial Assessment Report for Alkyl Sulfates, Alkane Sulfonates, Alpha-Olefin Sulfonates (SIAM 25) (OECD, 2007). The document summarizes the data for the category consisting of the mentioned groups and concluded that the bioconcentration tendency for α-olefin sulfonates (AOS) is low (BCF < 100) for chain lengths up to C16 and due to similar chemistry and physical properties, bioaccumulation potential of AOS is expected to be similar to that of the Alkyl Sulfates. Hence, bioconcentration factors for AOS are expected to be like those of the Alkyl Sulfates. Experimental data from a fish study (Wakabayashi et al., 1980) show that the BCF of Alkyl Sulfates in aquatic species is < 100. Both BCF and depuration time (the latter at least for 12 and 14 carbons in the alkyl chain) indicate that the substances are not bioaccumulative up to 16 carbons in the alkyl chain (SIDS, 2007).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Sodium (C14-16) olefin sulfonate exhibits low acute toxicity by the oral, inhalation and dermal routes. Sodium (C14-16) olefin sulfonate is irritating to the skin (≥5%) and serious irritation to the eyes (≥1%). Sodium (C14-16) olefin sulfonate was not a skin sensitiser. No systemic effects were observed in chronic repeated dose toxicity studies up to 259 mg/kg/day. The substance was not genotoxic in *in vitro* and *in vivo* models and is not carcinogenic. It is not a reproductive or developmental toxicant.

B. Acute Toxicity

Oral

In an OECD 401 (Acute Oral Toxicity) study, an LD_{50} of 2079 mg/kg was established for sodium (14-16) olefin sulfonate (ECHA). [KI score = 1].

Inhalation

In an OECD 403 (Acute Inhalation Toxicity) study, an LD_{50} >52 mg/L was established for sodium (14-16) olefin sulfonate (ECHA). [KI score = 2].

Dermal

In an OECD 402 (Acute Dermal Toxicity) study, an LD_{50} of 6300 mg/kg was established for sodium (14-16) olefin sulfonate (ECHA). [KI score = 2].

C. Irritation

Skin

An OECD Guideline 404 (Acute Dermal Irritation / Corrosion) was conducted to determine the skin irritation potential of sodium (C14-16) olefin sulfonate using New Zealand White rabbits. sodium (C14-16) olefin sulfonate was irritating following semi-occlusive exposure for 4 hr at 95% and following occlusive exposure for 24 hr at 5%. (ECHA) [KI score = 2].

Eye

An OECD Guideline 405 (Acute Eye Irritation / Corrosion) was conducted to determine the eye irritation potential of sodium (C14-16) olefin sulfonate using New Zealand White rabbits. Sodium (C14-16) olefin sulfonate was irritating at concentrations ≥5%. (ECHA) [KI score = 1].

D. Sensitisation

An OECD Guideline 406 (Skin Sensitisation) study (i.e., Buehler test) was performed on Pirbright-Hartley guinea pigs. Sodium (C14-16) olefin sulfonate did not induce skin sensitisation in this study (ECHA) [Kl score = 1].

E. Repeated Dose Toxicity

Oral

A chronic oral toxicity study in rodents was performed using male and female rats. Sodium (C14-16) olefin sulfonate was administered orally via feed for 104 weeks at a dose of 0, 39, 96, 195 mg/kg/day for males and 0, 57, 132, 259 mg/kg/day for females. A no observed adverse effect level (NOAEL) of 259 mg/kg/day was established based on the absence of effects at all doses up to 259 mg/kg/day. (ECHA) [KI score = 2].

Inhalation

No data were available.

Dermal

An OECD Guideline 411 study (Subchronic Dermal Toxicity: 90-Day study) was performed using rabbits. Sodium (C14-16) olefin sulfonate was administered in accordance with the OECD Guideline 411. At necroscopy, hematology, organ weights and organ to body weight data were all normal. Skin irritation was rated to mild to moderate as there was non-suppurative dermatitis, parakeratosis and hyperkeratosis observed. One of the animals had a firm, swollen salivary gland which upon microscopic examination exhibited inflammation and hyperplastic changes. The NOAEL was determined to be 35.7 mg/kg/day. (ECHA) [KI score = 1].

F. Genotoxicity

In vitro Studies

The results of the *in vitro* genotoxicity studies on sodium (C14-16) olefin sulfonate are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Copper (II) Sulfate

Test System[1]	Results*		Klimisch Score	Reference
	-S9	+S9		
OECD Guideline 471 (Bacterial Reverse Mutation Assay) Salmonella typhimurium strains TA98, TA100, TA1535, TA1537, TA102	-	-	1	ECHA

*+, positive; -, negative.

In vivo Studies

In an In Vivo Mammalian Germ Cell study was performed using CD-1 mice exposed intramuscularly to sodium (C14-16) olefin sulfonate (5,000 mg/kg). The injected bacteria or yeast cells were recovered and investigated. Sodium (C14-16) olefin sulfonate was not mutagenic in bacteria and yeast when metabolized by mice. (ECHA) [KI score = 4].

G. Carcinogenicity

A chronic oral toxicity study in rodents was performed using male and female rats. Sodium (C14-16) olefin sulfonate was administered orally via feed for 104 weeks at a dose of 0, 39, 96, 195 mg/kg/day

for males and 0, 57, 132, 259 mg/kg/day for females. The NOAEL for the test substance for carcinogenic effects was determined to be 259 mg/kg bw/day for the oral and 157.5 mg/kg bw/day for the dermal route. Sulfonic acids, C14-16 (even numbered)-alkane hydroxy and C14-16 (even numbered)-alkene, sodium salts does not have to be classified for carcinogenicity according to the criteria of EU Directive 67/548/EEC or Regulation (EC) No 1272/2008. (ECHA) [KI score = 2].

H. Reproductive Toxicity

Swiss albino male mice were fed with SLS either at 1 % (corresponds to 1000 mg/kg bw/day) for two weeks, or with 0.1% for six weeks (corresponds to 100 mg/kg bw/day). The study concluded that SLS has no adverse effects on fertility when administered at concentrations sufficient to cause a significant reduction in body weight (parental toxicity). A NOAEL of 1,000 mg/kg bw/day (in males) for fertility was reported for the study (NICNAS).

I. Developmental Toxicity

Sodium (C14-16) olefin sulfonate was analysed in accordance with OECD Guideline 414: Prenatal Developmental Toxicity Study. Pregnant CD-1 mice were exposed to sodium (C14-16) olefin sulfonate via oral gavage (0, 0.2, 2, 300, 600 mg/kg/day from gestational day 6 through 15. Embryotoxic effects have been observed from 300 mg/kg bw/d on. However, as these observations were accompanied by marked maternal toxicity (even maternal death at the highest dose level of 600 mg/kg bw/d) and were not significantly different from historic controls at 300 mg/kg bw/d, they were considered secondary to the toxicity of the test substance on the dam and are therefore insufficient for a classification as embryotoxic. The test item induced embryotoxic effects only in the presence of maternal toxicity. These effects were therefore considered to be secondary to maternal toxicity. (ECHA). [Kl. Score = 2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for sodium (C14-16) olefin sulfonate follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

A two-year dietary study in rats has been conducted on sodium (C14-16) olefin sulfonate. A NOAEL of 259 mg/kg/day was established based on the absence of effects at all doses up to the highest dose tested. The NOAEL of 259 mg/kg/day will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 1
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1
Oral RfD = 259/(1 x 10 x 1 x 1 x 1) = 259/10 = <u>25.9 mg/kg/day</u>

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:
Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)

Drinking water guidance value = (25.9 x 70 x 0.1)/2 = 90.65 mg/L

B. Cancer

Sodium (C14-16) olefin sulfonate is not considered a carcinogen. Thus, a cancer reference value will not be calculated for this substance.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Sodium (C14-16) olefin sulfonate does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Sodium (C14-16) olefin sulfonate has moderate toxicity to aquatic organisms and low toxicity to terrestrial organisms.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on sodium (C14-16) olefin sulfonate.

Table 3: Acute Aquatic Toxicity Studies on Sodium (C14-16) olefin sulfonate

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Danio rerio (Zebra Fish)	96-hr LC_{50}	4.2	1	ECHA
Ceriodaphnia dubia	48-hr EC_{50}	4.53	2	ECHA
Skeletonema costatum	72-hr EC_{50}	5.2	1	ECHA

Chronic Studies

Long-term aquatic toxicity test of sodium (C14-16) olefin sulfonate was conducted in invertebrates. The chronic toxicity to Daphnia magna (OECD 211) was studied with a 21-d reproduction test in a semistatic system. The test solution was renewed 3 times per week. The 21-day no observed effect concentration (NOEC) was determined to be 6.3 mg/L for the tested substance at 38.5% sodium (C14-16) olefin sulfonate and 2.42 mg/L at 100% for reproduction and survival of the adult test animals (ECHA) [Kl. Score = 1].

A long-term fish study was deemed not necessary based on short-term fish study and the above long-term invertebrate results (ECHA).

C. Terrestrial Toxicity

Based on the available data, no toxicity of sodium (C14-16) olefin sulfonate to terrestrial organisms is expected. Additionally, the substance is not expected to remain in the terrestrial environment, due to ready biodegradability and low adsorption potential, reducing the potential for chronic exposure (ECHA).

D. Calculation of PNEC

The PNEC calculations for sodium (C14-16) olefin sulfonate follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E(L)C_{50} values are available for fish (4.20 mg/L), invertebrates (4.53 mg/L) and algae (5.20 mg/L). Results from a chronic study is available for invertebrates (6.3 mg/L). On the basis that the data consists of short-term studies for three trophic levels and long-term results studies for two trophic levels, an assessment factor of 50 has been applied to the lowest reported NOEC of 4.20 mg/L for fish. The $PNEC_{water}$ is 0.08 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 0.05 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1{,}000 \times PNEC_{water}$

$= (0.83/1{,}280) \times 1{,}000 \times 0.08$

$= 0.05$ mg/kg

Where:

$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (cubic metre per cubic metre [m^3/m^3])

BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]

$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1{,}000 \times BD_{solid}]$

$= 0.8 + [(0.2 \times 0.064/1{,}000 \times 2{,}400]$

$= 0.83$ m^3/m^3

Where:

Kp_{sed} = solid-water partition coefficient (L/kg).

BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]

$Kp_{sed} = K_{oc} \times f_{oc}$
= 1.607 x 0.04
= 0.064 L/kg

Where:
K_{oc} = organic carbon normalized distribution coefficient (L/kg). The K_{oc} for sodium (C14-C16) olefin sulfonate calculated from EPISUITE™ 1.607 L/kg .
F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There is no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.002 mg/kg soil dry weight.

The calculations are as follows:
$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
= (0.03/1500) x 1000 x 0.08
= 0.002 mg/kg

Where:
Kp_{soil} = soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]
$Kp_{soil} = K_{oc} \times f_{oc}$
= 1.607 x 0.02
= 0.03 m^3/m^3

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for sodium (C14-C16) olefin sulfonate calculated from EPISUITE™ is 1.607 L/kg .
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Sodium (C14-16) olefin sulfonate is readily biodegradable and thus does not meet the screening criteria for persistence.

Based on a measured log K_{ow} of -1.3, sodium (C14-16) olefin sulfonate does not meet the screening criteria for bioaccumulation.

The lowest chronic NOEC for sodium (C14-16) olefin sulfonate is >0.1 mg/L. The acute $E(L)C_{50}$ values are >1 mg/L. Thus, sodium (C14-16) olefin sulfonate does not meet the screening criteria for toxicity.

The overall conclusion is that sodium (C14-16) olefin sulfonate is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Irritation-Eye category 1:H318: Causes serious eye damage.

Irritation-Skin category 2: H315: Causes skin irritation.

B. Labelling

Danger! According to the classification provided by companies to ECHA in REACH registrations this substance causes serious eye damage and causes skin irritation.

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. Get medical attention immediately, preferably a physician for an ophthalmologic examination.

Skin Contact

For minor skin contact, avoid spreading material on unaffected skin. Remove and isolate contaminated clothing. Wash the contaminated area of body with soap and fresh water. Get medical attention. Launder contaminated clothing before reuse.

Inhalation

Move person to fresh air. Administer oxygen if breathing is difficult. Do not use mouth-to-mouth method if victim inhaled the substance; give artificial respiration with the air of a pocket mask equipped with a one-way valve or other proper respiratory medical device. Give artificial respiration if victim is not breathing. Get medical attention immediately.

Ingestion

Do not induce vomiting. Get medical attention immediately.

B. Fire Fighting Information

Extinguishing Media

Use water spray or fog, foam, dry chemical or carbon dioxide.

Specific Exposure Hazards

Emits toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon dioxide, carbon monoxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Isolate area. Keep unnecessary and unprotected personnel from entering the area. Use personal protective clothing. Ensure adequate ventilation. Wear respiratory protection if ventilation is inadequate. Do not breath mist, vapours or spray. Avoid contact with skin, eyes and clothing. Eliminate all sources of ignition.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

Eliminate all sources of ignition. Pick up with suitable absorbent material and transfer to a container for chemical waste. For large amounts: dike spillage and pump off product into container for chemical waste. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep away from heat, sparks and flame. Avoid contact with eyes, skin and clothing. Avoid breathing vapour. Wash thoroughly after handling. Keep container closed. Use with adequate ventilation.

Storage

Keep container tightly closed. Store away from heat and light.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for sodium (C14-16) olefin sulfonate.

Engineering Controls

Good general ventilation should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation, or other engineering controls to maintain airborne levels below recommended exposure limits. If exposure limits have not been established, maintain airborne levels to an acceptable level.

Personal Protection Equipment

Respiratory Protection: If workers are exposed to concentrations above the exposure limit, they must use appropriate, certified respirators. If there are no applicable exposure limit requirements or guidelines, use an approved respirator. Selection of air-purifying or positive pressure supplied-air will depend on the specific operation and the potential airborne concentration of the product. For emergency conditions, use an approved positive-pressure self-contained breathing apparatus. The following should be effective types of air-purifying respirators: organic vapour cartridge with a particulate pre-filter.

Hand Protection: Use gloves chemically resistant to this material. Consult the SDS for appropriate glove barrier materials.

Skin Protection: Use protective clothing chemically resistant to the material. Selection of specific items such as face shield, boots, apron or full body suit will depend on the task.

Eye Protection: Use chemical goggles.

Other Precautions: Wash hands, forearms and face thoroughly after handling chemical products; before eating, smoking, and using the lavatory; and at the end of the working period. Appropriate techniques should be used to remove potentially contaminated clothing. Wash contaminated clothing before reusing. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Sodium (C14-16) olefin sulfonate is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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SODIUM BENZOATE

This dossier on sodium benzoate presents the most critical studies pertinent to the risk assessment of sodium benzoate in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed sodium benzoate in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment.[1]

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Sodium benzoate

CAS RN: 532-32-1

Molecular formula: $C_7H_6O_2.Na$

Molecular weight: 144.105 g/mol

Synonyms: benzoate, sodium, benzoic acid, sodium salt,

SMILES: [Na+].[O-]C(=O)C1=CC=CC=C1

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-Chemical Properties of Sodium Benzoate

Property	Value	Klimisch Score	Reference
Physical state at 20oC and 101.3 kPa	White, odorless granules or crystalline powder with a sweet astringent taste	2	ECHA
Melting Point	436°C @ 101.3 kPa	1	ECHA
Boiling Point	Decomposes at 450-475°C without boiling point	1	ECHA
Density	1500 kg/m3@ 20°C	1	ECHA
Vapour Pressure	0 Pa @ 20°C	2	ECHA
Partition Coefficient (log K_{ow})	1.88 (temperature not reported) *	2	ECHA
Water Solubility	556 g/L @ 20°C	2	ECHA
Flash Point	Not applicable	2	ECHA
Auto flammability	Not applicable	2	ECHA
Viscosity	Not applicable	2	ECHA
Dissociation constant (pKa)	4.03 @ 20°C *	2	ECHA

*Based on read across from benzoic acid

[1] https://www.industrialchemicals.gov.au/chemical-information/search-assessments?assessmentcasnumber=532-32-1+

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Sodium benzoate is water soluble, readily biodegradable, and it is not expected to bioaccumulate.

B. Biodegradation

A biotic degradation CO_2 evolution study reported that there was slightly lower degradation (75% of ThOD) recorded over 30 days in a closed bottle test. This study showed that there was 85-92% degradation for sodium benzoate even though there is no information on the 10-day window. It can be concluded that sodium benzoate is readily biodegradable (ECHA) [Kl. score = 2].

In an OECD guideline 301 CO_2 evolution test, degradation ranged from 85 to 94% after 28 days (ECHA) [Kl. score = 2].

The biodegradability of sodium benzoate was evaluated in using an ECETOC (1988) method. Concentrations of 50, 60, and mg/L of sodium benzoate were used, and the fermentation periods were 28-61 days. The biodegradation of sodium benzoate was reported to be 50-97% over a period of 60 days (ECHA) [KI. score = 2].

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

A quantitative structure activity relationship model (EPISUITE v4.11 KOCWIN 2017 program) was used to estimate the soil adsorption coefficient (K_{oc}) for sodium benzoate at or above 0.1 % w/w). The K_{oc} was predicted to be 7.033 L/kg at neutral pH (ECHA)[KI. score =2).

D. Bioaccumulation

There are no bioconcentration studies available for sodium benzoate. Sodium benzoate is not expected to bioaccumulate based on a log K_{ow} of 1.88 based on read across from benzoic acid (ECHA) [Kl. score = 2].

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Sodium benzoate is the sodium salt of benzoic acid, and it is completely metabolized to benzoic acid and ultimately hippuric acid in the body. Sodium benzoate is absorbed rapidly, and it is rapidly excreted as hippuric acid through urine. This substance is not acutely toxic through any route of exposure (i.e., oral, inhalation, dermal). Sodium benzoate is slightly irritating to the eye of rabbits and non-irritating to the skin of rabbits. This substance is not a skin sensitizer. It has low repeated oral, dermal, and inhalation toxicity. Sodium benzoate is not genotoxic or carcinogenic and does not induce reproductive or developmental toxicity.

B. Metabolism

Sodium benzoate is metabolized to benzoic acid and ultimately hippuric acid by conjugation with glycine. Sodium benzoate is not expected to accumulate in the body. Sodium benzoate and its metabolites are excreted through urine (ECHA) [KI. score =2].

Upon oral ingestion sodium benzoate is rapidly absorbed (100%, assumed). Dermal absorption is less effective and is inversely proportional to the administered dose (14-43%). There are no update data available regarding the inhalation route of exposure (ECHA) [KI. score = 2].

C. Acute Toxicity

Oral

In an acute oral toxicity study, sodium benzoate was given to male and female Sherman rats through their feed. The rats were observed for 14 days after dosing. The acute oral LD_{50} was reported to be 3,450 mg/kg bw (ECHA) [KI. score = 2].

In an acute oral toxicity study, 5000 mg/kg of sodium benzoate was given to male rats. The rats were observed for 10 days post treatment. No mortality or abnormal gross pathology findings were reported in this study. The acute oral LD_{50} was reported to be > 5000 mg/kg (ECHA) [KI. score = 2].

Inhalation

In an acute inhalation toxicity study, male and female Spartan rats were given 12,200 mg/m^3 of benzoic acid dust through whole body inhalation for four hours. The rats were observed for 14 days after treatment. There were no deaths following a single inhalation dose of 12,200 mg/m^3 of benzoic acid dust. Increased motor activity and slight erythema was observed during the four-hour exposure period. The rats appeared to be normal after 24 hours and after the 14-day observation period. The LC_{50} was reported to be >12,200 mg/m^3 air (ECHA) [KI. score = 2].

Dermal

In an acute dermal toxicity study, male and female New Zealand white rabbits were exposed to 2000 mg/kg of benzoic acid via semi occlusive dressing for 24 hours. The rabbits were observed for 14 days following exposure to benzoic acid. The LD50 was reported to be > 2,000 mg/kg bw (ECHA) [KI. score = 2].

D. Irritation

Skin

In an OECD guideline 404 (acute dermal irritation/corrosion) study, New Zealand White rabbits were exposed 0.5 grams of sodium benzoate via semi occlusive dressing (test are of skin =100 cm^2) for four hours. The rabbits were observed for 1,24,48, and 72 hours after treatment. One of the rabbits had slight erythema but it was resolved within 24 hours after exposure. A primary irritation index (PII) score of zero (max score for erythema = 1 and max score for oedema =0) was reported for sodium benzoate in this study. Sodium benzoate is reported to be non-irritating to the skin of rabbits (ECHA) [KI. score = 1].

Eye

An OECD guideline 405 (Acute eye irritation/corrosion) test was conducted using female New Zealand white rabbits exposed to ± 60 mg of sodium benzoate (instilled into one eye) for 24 hours. The rabbits were observed for 1,24,48,72 hours and 7-14 days following treatment. The mean cornea opacity score was reported to be 0, the mean iris score was reported to be 0, the mean conjunctivae score was reported to be 2.44, and the mean chemosis score was reported to be 0.67. All of these effects were determined to be fully reversible after 14 days. Sodium benzoate was reported to be slightly irritating to the eye (ECHA) [KI. score = 1].

E. Sensitisation

An OECD guideline 429 (Skin sensitisation: Local Lymph Node Assay) was conducted using female CBA mice exposed to 5,10, and 20% benzoic acid. The stimulation index (SI) values for each administered dose (5, 10, 20%) of benzoic acid were reported to be 0.8, 0.9, and 0.8 respectively. Based on this data, benzoic acid is not a skin sensitizer to female mice (ECHA) [KI. score =2].

An OECD guideline 429 (Skin sensitisation: Local Lymph Node Assay) was conducted using female CBA mice exposed to 5,10, and 20% sodium benzoate. The stimulation index (SI) values for each administered dose (5, 10, 20%) of benzoic acid were reported to be 0.8, 0.9, and 0.8 respectively. Based on this data, sodium benzoate is not a skin sensitizer to female mice (ECHA) [KI. score =2].

F. Repeated Dose Toxicity

Oral

A chronic oral repeated dose toxicity study was conducted using male and female Fischer 344 rats exposed to a daily dose of 1 or 2% sodium benzoate in their feed for 18-24 months. There were no adverse clinical signs identified in this study. The difference in average body weight and mortality rates between the treated and control groups were negligible. A variety of tumors occurred in the test animals and the controls rats for each sex. However, there was no evidence of carcinogenicity in the reported in the rats exposed to sodium benzoate. A NOAEL of 1,000 mg/kg bw/day was reported for this study (ECHA) [KI. score =2].

Inhalation

An OECD guideline 412 (28-day sub-acute inhalation toxicity) study was conducted using male and female Sprague-Dawley rats exposed to 25, 250, or 1,200 mg/m^3 of benzoic acid dust by whole body inhalation exposure for 28 days (6 hours per day for 5 days per week for four consecutive weeks). A mean equivalent aerodynamic diameter of 4.7 µm was defined for this study. All of the test concentrations induced local effects which consisted of nasal redness, nasal discharge, pulmonary fibrosis, and inflammatory cell infiltrates in the lungs. There were no systemic effects reported in the animals exposed to 25 mg/m^3 of benzoic acid. The female rats exposed to 250 mg/m^3 of benzoic acid developed a slight decrease in absolute kidney weight and their body weights were slightly (not statistically significant) lower than the control rats. The rats exposed to 1,200 mg/m^3 of benzoic acid developed a decrease in body weight and a decrease in liver, kidney, and lung weights. There were no histopathological findings except for the lungs. A NOEC of ≤ 25 mg/m^3 air was reported for local effects and a NOAEL of 250 mg/m3 was reported for systemic effects (ECHA) [KI. score = 1].

Dermal

An EPA OPP 82-2 (Repeated dose dermal toxicity-21/28 days) study was conducted using male and female New Zealand White rabbits exposed to 100, 500, and 2500 mg/kg of benzoic acid for six hours (once a day, 5 days per week, for three consecutive weeks). Slight dermal irritation was reported for one rabbit exposed to 2500 mg/kg of benzoic acid. There was no compound related systemic effects reported in this study. Thus, a NOAEL of > 2,500 was reported for this study (ECHA) [KI. score = 1].

An EPA OPP 82-2 (Repeated dose dermal toxicity-21/28 days) study was conducted using male and female New Zealand White rabbits exposed to 100, 500, and 2500 mg/kg of sodium benzoate for six hours (once a day, 5 days per week, for three consecutive weeks). Slight dermal irritation was reported for one rabbit exposed to 2500 mg/kg of sodium benzoate. There was no compound related systemic effects reported in this study. Thus, a NOAEL of > 2,500 was reported for this study (ECHA) [KI. score = 1].

G. Genotoxicity

In vitro Studies

The *in vitro* genotoxicity studies on sodium benzoate are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Sodium Benzoate

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
OECD guideline 471 (Bacterial Reverse Mutation Assay) -Salmonella typhimurium TA 1535, TA 1537, TA 98, TA100, TA 1538, and E. coli WP2	-	-	2	ECHA
In vitro chromosome aberration study (human embryonic lung cultures)	-	-	2	ECHA

*+, positive; -, negative

In vivo Studies

An OECD guideline 475 (Mammalian Bone Marrow Chromosome Aberration Test) was conducted using male Sprague-Dawley rats exposed to 50, 500, and 5,000 mg/kg of sodium benzoate for 96 hours. The rats were euthanized 6 hours, 24 hours, and 48 hours after treatment. Sodium benzoate did not product a significant increase in the number of aberrations in bone marrow metaphase chromosomes in rats. Thus, sodium benzoate is reported to be non-genotoxic in this study (ECHA) [KI. score = 2].

H. Carcinogenicity

Oral

Male and female Fischer 344 rats were given 1 or 2% of sodium benzoate in their feed for 18-24 months. There were no adverse clinical signs associated with exposure to sodium benzoate when compared to control rats. The differences in average body weight and mortality rates between treated and control groups were negligible. A variety of tumours were identified in the test and

control rats for each sex. However, the number of tumours in the treated and control mice were not statistically significant. A NOAEL of >1,000 mg/kg bw/day was reported and there was no evidence of carcinogenicity reported in this study (ECHA) [KI. score = 2].

Male and female Swiss mice were exposed to 2% sodium benzoate in their drinking water from five weeks of age through death. Consumption of sodium benzoate did not cause any detectable tumorigenic effects in the treated mice. A NOAEL of >4,000 mg/kg bw/day was reported in this study (ECHA) [KI. score = 2].

Inhalation

There are no studies available.

Dermal

There are no studies available.

I. Reproductive Toxicity

An OECD guideline 416 (two-generation toxicity) study was conducted using rats exposed to 0,0.5, or 1% benzoic acid in their feed for 11-12 weeks prior to mating and through four generations. There were no adverse effects on reproductive parameters including fertility measures, delayed sexual maturity, total number of pups born, pup survival, onset of reproductive senescence or litter size. In addition to this, organ weights and histopathologic findings were similar for all dose groups. There was an unexplained statistically significant increase in the lifespan of rats in the 0.5% dose group (i.e., a higher percentage of the rats lived longer). There were no dose-related adverse effects on either reproductive or developmental parameters over four generations. Thus, benzoic acid is not a reproductive or developmental toxicant. A NOEL was not established in this study (ECHA) [KI. score = 2].

J. Developmental Toxicity

Oral

An OECD guideline 414 (prenatal developmental toxicity) study was conducted using Wistar rats exposed to 0,699, 965,1,306, or 1,874, mg/kg bw/day (0,1,2, 4, 8 %) of sodium benzoate through their feed throughout the entire gestation period, delivery, and weaning period. The rats were euthanized on gestation day 20. Maternal feed consumption values in the 4% and 8% groups were decreased 58% and 87%, respectively, when compared to the control group and resulted in body weight loses in both treatment groups during the entire gestation period. The study authors considered these to reflect a palatability issue with the test diet rather than a consequence of the toxicity of sodium benzoate. No developmental toxicity was observed in the maternal or foetal animals exposed to up to 2% sodium benzoate. The number of abnormalities observed in either soft or skeletal tissues of the foetuses and weanlings in the 1% and 2% dose groups did not differ significantly from the control group. The NOEL for this study was reported to be 965 mg/kg bw/day (2%) (ECHA)[KI. score = 2].

In a developmental toxicity study, pregnant Wistar rats were administered 0, 1.75, 8.0, 38.0, or 175.0 mg/kg sodium benzoate by oral gavage once daily on gestation days (GD) 6 through 15 while positive control animals received 250 mg/kg aspirin. On GD 20, all surviving dams were subjected to Caesarean section under anaesthesia, and the numbers of corpora lutea, implantation sites, resorptions sites, and live and dead foetuses were recorded. Under conditions of this study, no dose-

related adverse effects were observed in the dams or foetuses in any of the groups receiving up to 175.0 mg/kg sodium benzoate during gestation days 6 through 15. The NOEL for maternal toxicity and developmental toxicity was reported to be > 175 mg/kg bw/day (ECHA) [KI. score = 2].

In a developmental toxicity study, pregnant Dutch-belted female rabbits were administered 0, 2.5, 12.0, 54.0, or 250.0 mg/kg sodium benzoate by oral gavage once daily on gestation days (GD) 6 through 18. On GD 29, all surviving does were subjected to Caesarean section under anaesthesia, and the numbers of corpora lutea, implantation sites, resorptions sites, and live and dead foetuses were recorded. Under conditions of this study, no dose-related adverse effects were observed in the does or foetuses in any of the sodium benzoate-treated groups. The NOEL for developmental toxicity was reported to be 250 mg/kg bw/day and the NOEL for maternal toxicity was reported to be >250 mg/kg bw/day (ECHA) [KI. score = 2].

Inhalation

There are no studies available.

Dermal

There are no studies available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

A chronic oral repeated dose toxicity study was conducted using male and female Fischer 344 rats exposed to a daily dose of 1 or 2% sodium benzoate in their feed for 18-24 months. There was no evidence of carcinogenicity in the reported in the rats exposed to sodium benzoate. A NOAEL of 1,000 mg/kg bw/day was reported for this study (ECHA) [KI. score =2].

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1
Oral RfD = 1000/(10 x 10 x 1 x 1 x 1) = 1000/100 = 10 mg/kg bw/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (10 x 70 x 0.1)/2 = 35 mg/L

B. Cancer

There is no evidence that sodium benzoate is carcinogenic. Thus, a value for cancer was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Sodium benzoate does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

The sodium benzoate is of low toxicological concern to environmental receptors.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on sodium benzoate.

Table 3: Acute Aquatic Toxicity Studies on Sodium Benzoate

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Pimephales promelas	96-hour LC_{50}	484 (mortality)	2	ECHA
Pimephales promelas	96-hour LC_{50}	>100 (mortality)	2	ECHA
Daphnia magna	96-hour LC_{50}	>100 (mortality)	2	ECHA
Raphidocelis subcapitata (formerly known as *Pseudokirchneriella subcapitata*)	72-hour EC_{50}	>30.5 (growth rate)	1	ECHA

Chronic Studies

Table 4 lists the results of chronic aquatic toxicity studies conducted on sodium benzoate.

Table 4: Chronic Aquatic Toxicity Studies on Sodium Benzoate

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Raphidocelis subcapitata	72-hour EC_{10}	6.5 (growth rate)	1	ECHA
Danio rerio	144-hour NOEC	10	2	ECHA

C. Terrestrial Toxicity

There are no studies available.

D. Calculation of PNEC

The PNEC calculations for sodium benzoate follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E(L)C_{50} values are available for fish (>100 mg/L), *Daphnia* (>100 mg/L), and algae (>30.5 mg/L). NOEC values from long-term studies are available for fish (10 mg/L) and algae (6.5 mg/L). On the basis that the data consists of short-term results from three trophic levels and long-term results from two trophic levels, an assessment factor of 10 has been applied to the lowest reported NOEC value of 6.5 mg/L for fish. The NOEC value is used because the value for algae is lower than the NOEC values for the other trophic level. The $PNEC_{water}$ is 0.65 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 0.475 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (1.11/1280) \times 1000 \times 0.65$
$= 0.475$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]
$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1000 \times BD_{solid}]$
$= 0.8 + [(0.2 \times 0.66/1000 \times 2400]$
$= 0.935\ m^3/m^3$

Where:
Kp_{sed} = solid-water partition coefficient (L/kg)
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]
$Kp_{sed} = K_{oc} \times f_{oc}$
$= 7.03 \times 0.04$
$= 0.28$ L/kg

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for sodium benzoate calculated from EPISUITE™ using QSAR is 7.03 L/kg.
F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.06 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
$= (0.33/1500) \times 1000 \times 10$
$=0.06$ mg/kg

Where:
Kp_{soil} = soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]
$Kp_{soil} = K_{oc} \times f_{oc}$
$= 7.03 \times 0.02$
$= 0.14\ m^3/m^3$

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for sodium benzoate calculated from EPISUITE™ using the QSAR is 7.03 L/kg.
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Sodium benzoate is readily biodegradable and thus does not meet the screening criteria for persistence.

There are no bioconcentration studies available for sodium benzoate. The measure log K_{ow} for benzoic acid is reported to be 1.88. Therefore, sodium benzoate does not meet the screening criteria for bioaccumulation.

The NOEC and EC_{10} values from the chronic aquatic toxicity studies on sodium benzoate are > 0.1 mg/L. The acute $E(L)C_{50}$ values from the acute aquatic toxicity studies on sodium benzoate are > 1 mg/L. Thus, sodium benzoate does not meet the criteria for toxicity.

The overall conclusion is that sodium benzoate is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H319: Causes serious eye irritation

B. Labelling

Warning

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

There are no workplace exposure standards for sodium benzoate in Australia.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Sodium benzoate is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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SODIUM BISULFITE

This dossier on sodium bisulfite presents the most critical studies pertinent to the risk assessment of sodium bisulfite in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed sodium bisulfite in an IMAP Tier 1 assessment and considers it an inorganic substance comprising ions of low ecological concern[1] .

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Sodium hydrogen sulfite

CAS RN: 7631-90-5

Molecular formula: $NaHSO_3$

Molecular weight: 104.06 g/mol

Synonyms: sodium bisulfite; sodium acid sulfite; sulfurous acid, monosodium salt; sodium bisulphite

SMILES: H-O3-S. Na

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-chemical Properties of Sodium Bisulfite

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	White, crystalline, solid	-	PubChem
Melting Point	104 °C , Decomposes	-	Pubchem
Boiling Point	Decomposes	-	Pubchem
Density	1348 kg/m^3@ 20°C	1	ECHA
Vapour Pressure	Not applicable	-	-
Partition Coefficient (log K_{ow})	Not applicable (inorganic substance)	-	-
Water Solubility	724 g/L @ 20°C	2	ECHA
Flash Point	Not available	-	-
Auto flammability	Not available	-	-
Viscosity	3.64 mPa s @ 20°C	-	PubChem
Henry's Law Constant	Not applicable	-	-

[1] https://services.industrialchemicals.gov.au/assessment-detail/?id=96e2433e-f36b-1410-8e4e-00f1fcf8411a

Sodium bisulfite is a weak acid with a pK_a of 6.97. Its conjugate base is the sulfite ion (SO_3^{2-}).

$NaHSO_3 \leftrightarrow Na^+ + HSO_3^-$

$HSO_3^- \leftrightarrow H^+ + SO_3^{2-}$

At neutral pH, a mixture of 50% sulfite (SO_3^{2-}) and 50% bisulfite (HSO_3^{2-}) is present.

In surface waters, sulfite is oxidised to sulfate either catalytically by air oxygen or by microbial action (OECD, 2008). The presence of cations like iron, copper or manganese in the environment accelerates the oxidation rate significantly.

Dissociation of sodium bisulfite in aqueous solutions can also liberate sulfur dioxide (SO_2), which is a gas.

III. ENVIRONMENTAL FATE PROPERTIES

At environmental pHs, sodium bisulfite dissociates in water to form sodium (Na^+) ions, bisulfite ions (HSO_3^-), sulfite (SO_2^{3-}) ions, and sulfur dioxide (SO_2) which is a gas.

Sodium bisulfite is not expected to bioaccumulate in the environment because of its dissociation to ionic species and a gas. Furthermore, sulfite will oxidise to sulfate, which is ubiquitous in the environment.

Sodium bisulfite and its dissociated species are expected to have a low potential to adsorb to soil and sediment.

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Limited toxicity data are available on sodium bisulfite; therefore, structural analogues have been used to read across to sodium bisulfite. Sodium bisulfite has low acute toxicity by the oral, inhalation, and dermal routes. Sodium bisulfite is minimally irritating to the skin and slightly irritating to the eyes. It is not a skin sensitiser. No systemic toxicity was seen in rats when given read across substance sodium metabisulfite in their diet over a lifetime. There were, however, indications of stomach lesions as a result of localized irritation from the ingestion of sodium metabisulfite. Sodium bisulfite is not expected to be genotoxic or carcinogenic. No reproductive or developmental toxicity was observed in any of the animal studies on sodium bisulfite or its structural analogues.

B. Acute Toxicity

<u>Oral</u>

No acute toxicity studies are available for sodium bisulfite.

The oral LD_{50} value in rats for sodium sulfite is 2,610 mg/kg (ECHA) [Kl. score = 2]. The oral LD_{50} values in rats for sodium metabisulfite are 1,420 mg/kg (males), 1,630 mg/kg (females), and 1,540 mg/kg (combined sexes) (ECHA) [Kl. score = 2].

Inhalation

The 4-hour inhalation LC_{50} in rats for sodium sulfite is >5.5 mg/L (ECHA)[Kl. score = 2]

Dermal

The dermal LD_{50} in rats for sodium sulfite is >2,000 mg/kg (ECHA)[Kl. score = 2]

C. Irritation

There are no studies are available for sodium bisulfite.

Application of 0.5 mL of sodium sulfite to the skin of rabbits for 4 hours under occlusive conditions was minimally irritating. The mean of the 24, 48, and 72 scores were: 0.5 for erythema and 0.0 for oedema (ECHA). [Kl. score = 2]

Instillation of 0.1 mL of sodium sulfite (with 0.5% cobalt sulfate) into the eyes of rabbits produced slight irritation. The mean of the 24-, 48- and 72-hour scores are as follows: 0.5 for conjunctival redness; 0.5 for conjunctival chemosis; 0.0 for corneal lesions; and 0.0 for iridial lesions (ECHA)[Kl. score = 2]

D. Sensitisation

Sodium bisulfite was not considered a skin sensitiser in a mouse local lymph node assay (ECHA)[K. score = 1].

E. Repeated Dose Toxicity

Oral

There are no studies available for sodium bisulfite.

A study is available on sodium metabisulfite. Sodium metabisulfite dissociates in water to form sodium (Na^+) ions, disulfite ($S_2O_5^{2-}$) ions, and sulfur dioxide (SO_2). The disulfite ions can form bisulfite (HSO_3^-) and sulfite ions (SO_2^{3-}); at neutral pH, a mixture of 50% sulfite (SO_3^{2-}) and 50% bisulfite (HSO_3^{2-}) is present.

Male and female Wistar rats were fed in their diet 0, 0.125, 0.25, 0.5, 1.0, or 2.0% sodium metabisulfite for up to two years and over three generations. The diet was enriched with thiamine to prevent thiamine deficiency as a result of sulfite-induced destruction of this vitamin. During storage up to the time of consumption, the losses of sulfite from the feed containing sodium metabisulfite at levels of 0.125, 0.25, 0.5, 1.0, and 2.0% averaged 22, 14,12, 8, and 4.5%, respectively, while the decrease in thiamine was 2.7, 1.7, 8.3, 14.5, and 15.4%, respectively. Addition of thiamine to the diet prevented thiamine deficiency in rats at all dose levels based on measurements of thiamine levels in the urine and liver. The general condition of the rats was good during the first 72 weeks in the F0 generation, as well as the other two generations. After 72 weeks, there was a rapid increase in mortality in all groups. Survival in the treated groups were generally higher than the controls, except for the 2% F1 males; no deaths occurred in the 2% F2 females. A marginal reduction in body weight gain was observed in the 2% dose group (both sexes) in the F1 and F2 generations. Feed consumption was similar between treated and control groups. There were no changes in hematology and clinical chemistry parameters and urinalysis that were considered toxicologically significant. The >1% dietary groups had occult blood in their feces. Relative kidney weights were

increased in the 2% F2 females, but there were no pathological changes noted in the kidneys from this group. Hyperplastic changes in the fore- and glandular stomachs were noted in the >1% groups in all three generations. Some slight alterations were also noted in stomachs of the 0.5% F2 rats. The NOAEL for systemic toxicity is 1.91% in the diet. This was estimated to be 955 mg/kg bw/day based on a rat body weight of 400 g and a daily feed intake of 20 g. The histopathologic effects on the stomach and the occult blood in faeces are considered to be the result of localized irritation (a site-of-contact effect) from the ingestion of sodium metabisulfite. Because there was no evidence of systemic toxicity following chronic treatment, the NOAEL for systemic effects can be expected to be above the highest dose of 2% sodium metabisulfite which corresponds to 955 mg/kg bw/day of $Na_2S_2O_5$ or 1045 mg/kg bw/day of sodium hydrogensulfite (Til et al., 1972; as cited in ECHA). [Kl. score = 2]

Inhalation

There are no studies available.

Dermal

There are no studies available.

F. Genotoxicity

In vitro Studies

There are no *in vitro* genotoxicity studies were located for sodium bisulfite. Table 2 presents the findings from *in vitro* genotoxicity studies conducted on structural analogues of sodium bisulfite.

Table 2: *In vitro* Genotoxicity Studies on Structural Analogues to Sodium Bisulfite

Test System	Test Substance	Results*		Klimisch Score	Reference
		-S9	+S9		
Bacterial reverse mutation (*S. typhimurium* strains)	Sodium metabisulfite	-	-	2	ECHA
Bacterial reverse mutation (*S. typhimurium* strains)	Potassium metabisulfite	-	-	2	ECHA
Bacterial reverse mutation (*S. typhimurium* strains)	Potassium metabisulfite	-	-	2	ECHA
Bacterial reverse mutation (*S. typhimurium* and *E. coli* strains)	Sodium metabisulfite	-	-	2	ECHA
Mammalian cell gene mutation (mouse lymphoma L5178Y cells)	Sodium metabisulfite	-	-	2	ECHA
Chromosomal aberration (human lymphocytes)	Sodium metabisulfite	-	-	1	ECHA

*+, positive; -, negative

In vivo Studies

Sodium bisulfite did not show a mutagenic response in a rat dominant lethal assay when given in feed at doses of 0, 4.5, 15, or 45 mg/kg/day (ECHA). [Kl. score = 2].

Sodium sulfite was not genotoxic in a bone marrow micronucleus test in rats. Male NMRI rats were given a single subcutaneous injection of 0, 250, 500, or 1,000 mg/kg sodium sulfite (ECHA). [Kl. score = 1].

G. Carcinogenicity

Oral

There are no studies are available for sodium bisulfite.

Male and female Wistar rats were fed in their diet 0, 0.125, 0.25, 0.5, 1.0, or 2.0% sodium metabisulfite for up to two years and over three generations. There was no increased incidence of tumours in the treated groups compared to the controls (Til et al., 1972; as cited in ECHA). [Kl. score = 2].

Male and female ICR/JCL mice were given in their drinking water 0, 1, or 2% potassium metabisulfite for two years. There was no increased incidence of tumours in the treated groups compared to the controls (Tanaka et al., 1979; as cited in ECHA) [Kl. score = 2].

Inhalation

There are no studies available.

Dermal

There are no studies available.

H. Reproductive Toxicity

There are no studies are available for sodium bisulfite.

Male and female Wistar rats were fed in their diet 0, 0.125, 0.25, 0.5, 1.0, or 2.0% sodium metabisulfite for up to two years and over three generations. The diet was enriched with thiamine to prevent thiamine deficiency as a result of sulfite-induced destruction of this vitamin. During storage up to the time of consumption, the losses of sulfite from the feed containing sodium metabisulfite at levels of 0.125, 0.25, 0.5, 1.0, and 2.0% averaged 22, 14,12, 8, and 4.5%, respectively, while the decrease in thiamine was 2.7, 1.7, 8.3, 14.5, and 15.4%, respectively. Addition of thiamine to the diet prevented thiamine deficiency in rats at all dose levels based on measurements of thiamine levels in the urine and liver. The effects other than reproductive and developmental toxicity are discussed above in the Repeated Dose Toxicity section. There were no treatment-related effects on female fertility, the number of young per litter, or birth weight or mortality of the offspring. The number of F2a pups was significantly reduced in the >0.5% groups during the first breeding cycle, but there was no dose-response, and the reduction did not occur during the second breeding cycle. Slight growth retardation was observed in the F1 and F2 generation rats both before and after weaning. The NOAEL for reproductive toxicity is 1.91% in the diet. This was estimated to be 955 mg/kg bw/day based on a rat body weight of 400 g and a daily feed intake of 20 g (Til et al., 1972; as cited in ECHA) [Kl. score = 2].

Male and female rats were given sodium metabisulfite in their drinking water for up to 2.5 years and in three successive generations. The doses were 375 and 750 ppm as sulfur dioxide (SO_2). There was no evidence of systemic toxicity in either dose group. The number of offspring of either the F1 and F2 generation and the proportion surviving to the end of lactation were similar between treated and

control groups. The NOAEL for reproductive toxicity is 750 ppm (as SO2) in drinking water. Assuming an average rat body weight of 400 g and a daily water intake of 28 mL, 750 ppm (as SO_2) corresponds to 53 mg/kg bw/day sodium metabisulfite (Lockett and Natoff, 1960; as cited in ECHA) [Kl. score = 2].

I. Developmental Toxicity

Oral

Pregnant female Wistar rats were dosed by oral gavage with up to 110 mg/kg-day sodium bisulfite during GD 6-15. There was no maternal or developmental toxicity. The NOAEL for maternal and developmental toxicity for this study is 110 mg/kg bw/day (ECHA) [Kl. score = 2].

Pregnant female CD-1 mice were dosed by oral gavage with up to 150 mg/kg-day sodium bisulfite during GD 6-15. There was no maternal or developmental toxicity. The NOAEL for maternal and developmental toxicity for this study is 150 mg/kg bw/day (ECHA) [Kl. score = 2].

Pregnant female Dutch-belted were dosed by oral gavage with up to 100 mg/kg-day sodium bisulfite during GD 6-18. There was no maternal or developmental toxicity. The NOAEL for maternal and developmental toxicity for this study is 100 mg/kg bw/day (ECHA) [Kl. score = 2].

Inhalation

There are no studies available.

Dermal

There are no studies available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for sodium bisulfite follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

No repeated dose toxicity studies have been conducted on sodium bisulfite. In a study conducted on sodium metabisulfite, there was no evidence of systemic toxicity in rats fed up to 2% for two years (Til et al., 1972; as cited in ECHA). The NOAEL for this study is 2% or 955 mg/kg bw/day Na_2S2O5 or 1,045 mg/kg bw/day sodium bisulfite. The NOAEL of 1,045 mg/kg bw/day will be used for determining the oral reference dose (RfD) and the drinking water guidance value for sodium bisulfite.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10

UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 10
UF_D (database uncertainty) = 1
Oral RfD = 1045/(10 x 10 x 1 x 10 x 1) = 1045/1000 = 1.045 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:
Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (1.045 x 70 x 0.1)/2 = 3.66 mg/L

B. Cancer

There are no carcinogenicity studies for sodium bisulfite. No carcinogenic effects were reported for sodium metabisulfite in rat and mouse chronic studies. The available data on long-term oral exposure of experimental animals to sodium and potassium metabisulphite allow an evaluation of the carcinogenic risks of sulphite compounds for humans exposed via the oral route. There was no indication that metabisulphite had any carcinogenic effect itself (ECHA), therefore a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Sodium bisulfite does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

No aquatic toxicity studies have been conducted on sodium bisulfite. Other inorganic sulfite compounds show low to moderate toxicity concern to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

No acute aquatic studies are available on sodium bisulfite; however, studies are available on other inorganic sulfite compounds. The studies on these inorganic sulfite compounds can be used to read across to sodium bisulfite since sulfite ions are formed in water upon dissociation of sodium

bisulfite. Table 3 lists the results of acute aquatic toxicity studies on the structural analogues of sodium bisulfite.

Table 3 lists the results of acute aquatic toxicity studies conducted on the structural analogues of sodium bisulfite.

Table 3: Acute Aquatic Toxicity Studies on the structural analogues of sodium bisulfite

Test Species	Test Substance	Endpoint	Results (mg/L)	Klimisch score	Reference
Oncorhynchus mykiss(Salmo gairdneri)	Disodium disulphite	96-hour LC_{50}	147-215 (177.8*)	2	ECHA
Leuciscus idus	Potassium sulphite	96-hour LC_{50}	>220-460 (316*)	2	ECHA
Leuciscus idus	Disodium sulfite	96-hour LC_{50}	316	2	ECHA
Oncorhynchus mykiss	Diammonium thiosulfate	96-hour LC_{50}	770	1	ECHA
Lepomis macrochirus	Diammonium thiosulfate	96-hour LC_{50}	510	1	ECHA
Brachydanio rerio	Potassium metabisulfite	96-hour LC_{50}	464-1,000 (681.2*)	1	ECHA
Daphnia magna	Sodium disulphite	48-hour EC_{50}	88.8	2	ECHA
Daphnia magna	Sodium dithionite	48-hour EC_{50}	98.31	2	ECHA
Daphnia magna	Diammonium thiosulfate	48-hour EC_{50}	230	1	ECHA
S. subspicatus	Sodium disulfite	96-hour EC_{50} 72-hour EC_{10}	43.9 (36.8**) 33.3	2	ECHA
Desmodesmus subspicatus (S. subpicatus)	Disodium disulphite	72-hour EC_{50}	43.8	2	ECHA
Scenedesmus brasiliensis	Disodium sulfite	96-hour EC_{50}	37.8	2	ECHA
Desmodesmus subspicatus (S. subspicatus)	Disodium dithionite	72-hour EC_{50}	206.2 (189**)	2	ECHA
Raphidocelis subcapitata	Ammonium thiosulfate	72-hour EC_{50}	>100	1	ECHA

*Geometric mean.
** sulfite ion (SO_3^{2-})

Chronic Studies

No chronic studies are available on sodium bisulfite; however, studies are available on sodium sulfite.

Table 4 lists the results of chronic aquatic toxicity studies conducted on sodium sulfite.

Table 4: Chronic Aquatic Toxicity Studies on Structural Analogues of Sodium Sulfite (CAS No. 7757-83-7)

Test Species	Test substance	Endpoint	Results (mg/L)	Klimisch score	Reference
Danio rerio	Sodium sulfite	34-day NOEC	≥316 (200.5*)	1	ECHA
Daphnia magna	Disodium disulphite	21-day NOEC	>10	2	ECHA
Dophnia magna	Sodium dithionite	21-day NOEC	>10	1	ECHA
Desmodesmus subspicatus	Disodium disulphite	72-hour EC_{10}	33.3 (28*)	2	ECHA
Scenedesmus brasiliensis	Sodium sulphite	96-hour NOEC	37.8	2	ECHA
Desmodesmus subspicatus	Disodium dithionite	72-hour EC_{10}	81.7 (75*)	2	ECHA
Raphidocelis subcapitata	Ammonium thiosulfate	72-hour NOEC	≥100	1	ECHA

*sulfite ion (SO_3^{2-})

C. Terrestrial Toxicity

There are no studies available.

D. Calculation of PNEC

The PNEC calculations for sodium bisulfite follow the methodology discussed in DEWHA (2009).

PNEC Water

There are no studies available for sodium bisulfite. However, the results from studies conducted on other inorganic sulphite compounds can be used to read across to sodium bisulfite. Hence, experimental acute and chronic results are available for three trophic levels. Acute E(L)C50 values are available for fish (177.8 mg/L for sodium pyrosulfite), invertebrates (88.8 mg/L for sodium sulfite), and algae (36.8 mg/L for sulfite ion)). Results from chronic studies on sodium sulfite or other inorganic sulphite compounds are also available for all three trophic levels. NOEC or EC_{10} values from long-term studies are available for fish (200.5 mg/L for sulfite ion), invertebrates (>10 mg/L), and algae (28 mg/L, for sulfite ion) , with the lowest NOEC being >10 mg/L for invertebrates. Using the molecular weights of sodium sulfite (126 g/mol) and sodium bisulfite (104.1 g/mol, the NOEC of 10 mg/L for sodium sulfite is converted to 8.3 mg/L. On the basis that the data consist of short-term and long-term results from three trophic levels, an assessment factor of 10 has been applied to the lowest reported NOEC of 8.3 mg/L for invertebrates. The $PNEC_{water}$ is 0.8 mg/L.

PNEC Sediment

No experimental toxicity data on sediment organisms are available. Sodium bisulfite dissociates completely in water with its environmental distribution is dominated by its high water solubility. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as sodium bisulfite. Thus, the equilibrium

partitioning method cannot be used to calculate the $PNEC_{sed}$. Based on its properties, no adsorption of sodium bisulfite to sediment is to be expected, and the assessment of this compartment will be covered by the aquatic assessment.

PNEC Soil

No experimental toxicity data on soil organisms are available. Sodium bisulfite dissociates completely in water with its environmental distribution is dominated by its high water solubility. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as sodium bisulfite. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{soil}$. Based on its properties, no adsorption of sodium bisulfite to soil is to be expected, and the assessment of this compartment will be covered by the aquatic assessment.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Sodium bisulfite is an inorganic compound that dissociates completely to ionic species and sulfur dioxide gas. Biodegradation is not applicable to these compounds. For the purposes of this PBT assessment, the persistent criterion is not considered applicable to sodium bisulfite or its dissociated compounds.

Sodium bisulfite is not expected to bioaccumulate because its dissociated species are inorganic ions and a gas.

There are no aquatic toxicity data on sodium bisulfite. The lowest NOEC from chronic aquatic toxicity studies on sodium sulfite, a structural analogue of sodium bisulfite, is >0.1 mg/L. Thus, sodium bisulfite is not expected to meet the criteria for toxicity.

The overall conclusion is that sodium bisulfite is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H302-Acute Toxicity category 4: Harmful if swallowed

H318-Eye damage-category 1: Causes serious eye damage

B. Labelling

Danger

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The workplace exposure standards for sodium bisulphite in Australia is as follows: 5 mg/m^3 (Time weighted average, TWA).

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Sodium bisulfite is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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SODIUM BROMATE

This dossier on sodium bromate presents the most critical studies pertinent to the risk assessment of sodium bromate in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA), the 2020 National Industrial Chemical Notification and Assessment Scheme Inventory [NICNAS] human health tier II assessment for bromates, and the 1994 cosmetic ingredient review (CIR) for sodium bromate and potassium bromate. Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Sodium bromate

CAS RN:7789-38-0

Molecular formula: $NaBrO_3$ or $BrNaO_3$

Molecular weight: 150.89 g/mol

Synonyms: bromic acid sodium salt

SMILES: [O-]Br(=O) =O. [Na+]

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-chemical Properties of Sodium Bromate

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	White, odorless, crystalline powder	2	ECHA
Melting Point	381°C (pressure not indicated)	2	ECHA
Boiling Point	Not applicable, substance is a solid which melts above 300 °C	-	-
Density	3339 kg/m^3 @ 20°C	2	ECHA
Vapour Pressure	Not applicable	-	-
Partition Coefficient (log K_{ow})	Not applicable (inorganic substance)	-	-
Water Solubility	364 g/L @ 20°C	2	ECHA
Flash Point	Not applicable	-	-
Auto flammability	Not applicable	-	-
Viscosity	Not applicable	-	-
Henry's Law Constant	Not applicable	-	-

Sodium bromate is the sodium salt of bromic acid that is highly soluble in water. Sodium bromate is formed by passing bromine through a solution of sodium carbonate and it can also be created by

oxidation of bromine with chlorine to sodium hydroxide (CIR 1994). It dissociates to form sodium (Na+) and bromate (BrO_3^-) ions. It has strong oxidizing properties, and it reacts vigorously with organic matter and is reduced to bromide. (ECHA).

III. ENVIRONMENTAL FATE PROPERTIES

Sodium bromate dissociates in aqueous media to form sodium (Na+) and bromate (BrO_3^-) ions. Biodegradation is not applicable to inorganic compounds. Sodium bromate is not expected to bioaccumulate in the environment because of its dissociation to ionic species. Sodium bromate is not expected to adsorb to soil or sediment because of its high water solubility of 364 g/L (ECHA).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Sodium bromate is reduced in the body to bromide and it is ultimately excreted in the urine and feces. Sodium bromate has high acute oral toxicity in rats and it is expected to be irritating to the eyes and the skin. Sodium bromate is a mild skin sensitiser. Sodium bromate is genotoxic and the bromate moiety is possibly carcinogenic . It is not a reproductive or developmental toxicant.

B. Metabolism

Sodium bromate is rapidly absorbed from the gastrointestinal tract and it remains largely unchanged. It is then distributed throughout the body where it will appear in the plasma, urine, and unchanged in other tissues as bromide. Sodium bromate is reduced to bromide in several body tissues, most likely by glutathione (GSH) or other sulfhydryl-containing compounds. Sodium bromate is mostly excreted in the urine as either bromate or bromide and it can also be excreted in the faeces (ECHA) [KI. score =2].

Sodium bromate will dissociate in water and the bromate ion is rapidly absorbed from the gastrointestinal tract. Bromine has been detected in the adipose tissue of mice following long-term treatment with bromate (NICNAS, 2020).

C. Acute Toxicity

An acute oral toxicity study in rats was reported for sodium bromate. An oral LD_{50} value of 301 mg/kg bw was reported for this study (ECHA) [KI. score =4].

Several cases of acute bromate toxicity have been reported in humans following accidental or intentional ingestion of permanent hair wave neutralising solution. These products usually contain either 2 % potassium bromate, or 10 % sodium bromate. Bromate intoxication leads to gastrointestinal symptoms (abdominal pain, nausea, vomiting, diarrhoea), central nervous system depression, renal failure, and hearing loss. Although these effects are usually reversible, death from renal failure may ensue if medical intervention is not successful. Hearing loss is usually irreversible (NICNAS, 2020).

D. Irritation

<u>Skin</u>

There are no adequate studies available to evaluate the skin irritancy potential of sodium bromate. However, sodium bromate is reported to have skin irritating properties (ECHA) [KI. score =4].

An *in vitro* study was conducted according to Organisation for Economic Co-operation and Development (OECD) Test Guideline (TG) 431, using a human skin model. The study consisted of a topical exposure of potassium bromate to a human reconstructed model followed by a cell viability test. Potassium bromate was not considered to possess a corrosive potential (NICNAS,2020).

Eye

There are no adequate studies available to evaluate the eye irritancy potential of sodium bromate. However, sodium bromate is reported to have eye irritating properties (ECHA) [KI. score =4].

An eye irritation study was conducted according to OECD TG 437: Bovine Corneal Opacity and Permeability Test Method for Identifying Ocular Corrosives and Severe Irritants. In this test, the damage is assessed by quantitative measurements of changes in corneal opacity and permeability with an opacitometer and a visible light spectrophotometer, respectively. Potassium bromate caused weak opacity but no permeability of the cornea compared with the results of the negative control group. The chemical was considered to be a mild eye irritant (NICNAS, 2020).

E. Sensitisation

There are no adequate studies available to evaluate if sodium bromate is a skin sensitiser.

The Buehler method (No. 406 Skin sensitization Buehler Test, Method B6) study was used to evaluate the sensitisation potential of sodium bromate in guinea pigs. An undiluted dose of 0.5 ml of sodium bromate was applied to the flank of four guinea pigs using occlusive dressing for six hours and the treated site was scored at 24 and 48 hours post treatment. Two of the guinea pigs who received the undiluted dose of sodium bromate developed mild irritation after 24 hours, one guinea pig did not have any observable effects, and the fourth guinea pig also developed mild irritation that ultimately resolved after 48 hours. Next, a 75% dilution of sodium bromate was used and resulted in a mild irritant reaction after 48 hours. Based on these results in guinea pigs, sodium bromate was reported to be a mild sensitizer (CIR, 1994).

A skin sensitisation study conducted according to OECD TG 429 (local lymph node assay—LLNA), potassium bromate (CAS No. 7758-01-2) at 1.25 %, 2.5 %, and 7.5 % (w/v) concentration was applied topically at the dorsum of each ear of female CBA mice once daily on three consecutive days. A further group of mice was treated with the positive control item and a control group of mice was also treated with the vehicle only. Stimulation Indices (S.I.) of 0.90, 0.53, and 0.64 were determined with the test item at concentrations of 1.25, 2.5, and 7.5 % (w/v), respectively. The EC3 value could not be calculated since none of the tested concentrations induced an S.I. of greater than three. Potassium bromate was not considered to be a skin sensitiser (NICNAS, 2020).

F. Repeated Dose Toxicity

Oral

Several repeated dose oral toxicity studies in animals indicate that the kidney is the major target organ of bromate associated toxicity, leading to carcinogenicity. Specific non-cancer effects included degenerative, necrotic, nephropathic, and regenerative changes in the kidney (NICNAS, 2020).

A 13-week (sub-chronic) oral drinking water repeated dose toxicity study was performed using male and female F344 rats exposed to 0, 150, 300, 600, 1,250, 5,000, and 10,000 ppm potassium bromate. All the rats in the 1,250-ppm group died within seven weeks. The observed signs of toxicity included a significant reduction in body weight gain in the male rats treated with the 600, 1,250, 5,000, and

10,000 ppm potassium bromate There was also a significant increase the following serum parameter: glutamate oxaloacetate transaminase, glutamate pyruvate transaminase, lactate dehydrogenase, alkaline phosphatase, blood urea nitrogen [BUN], Na+, and cholinesterase. There was also a decrease in serum potassium levels in both sexes of rats treated with 600 ppm of potassium bromate. Droplets of various sizes and regenerative changes in the renal tubules were also observed in rats exposed to potassium bromate. Ultimately, a LOAEL of ≤ 63 mg/kg bw/day was established for this study (ECHA) [KI. score =2]. A NOAEL of 300 mg/L was determined (NICNAS, 2020).

A 15-month oral drinking water repeated dose toxicity study was performed using male Wistar rats exposed to 0.04% potassium bromate. All the rats exposed to potassium bromate experienced a reduction in body weight. Histological examination of the kidneys of each rat at 7-11 weeks revealed karyopknotic foci (a necrotic change characterized by shrinking of the nucleus and condensation of the chromatin) in the tubules of the inner medulla. There was also in an increase in the blood urea nitrogen (BUN) levels and marked structural abnormalities of the cortical tubules in the rats exposed to potassium bromate after 15 months. A NOAEL was not established for this study, but a LOAEL of 30 mg/kg bw/day was established based on a decrease in body weight and the reported renal effects (ECHA) [KI. score =2].

An 18-month chronic toxicity study was conducted using five groups of Wistar rats (60 male and 60 females in each group) that were fed 1) 0 (control); 2) 50 ppm potassium bromate; 3) 75 ppm potassium bromate; 4) 50 ppm potassium bromate with 30 ppm ascorbic acid and 50 ppm benzoyl peroxide; 5) 50 ppm potassium bromate with 30 ppm ascorbic acid and 50 ppm benzoyl peroxide and 15 ppm chlorine dioxide bread base diets. The cumulative mortality of the treatment groups and the mean body weights of the rats were not altered significantly, for majority of the treatment groups, when compared to the rats in the control group. However, the male rats exposed to 50 ppm potassium bromate had significantly increased body weights between week 12 and 72 of treatment when compared to the control group (CIR, 1994).

In a chronic toxicity/carcinogenicity study, potassium bromate was administered at 0, 250, and 500 ppm concentrations to F344 rats (53/sex/group) for 110 weeks. Daily intake of potassium bromate was equivalent to 12.5 and 27.5 mg/kg bw/day in males and 12.5 and 25.5 mg/kg bw/day in females, respectively. As the growth of males in the high dose group was severely inhibited, the concentration in this group was reduced to 400 ppm at week 60. Body weight gain was significantly reduced in high-dose males, but not in the other treated groups. Survival was reduced in high-dose males by about week 60 and in low-dose males by about week 100. No effect on survival was observed in treated female rats. A variety of non-cancer effects were reported, including degenerative, necrotic, and regenerative changes in renal tubules; formation of hyaline droplets; thickening of transitional epithelium of the renal pelvis; papillary hyperplasia; and papillary growth. It was noted that the lesions were more extensive in degree and distribution in treated rats compared with controls, especially males. However, in the absence of information on the incidence of these lesions or on the statistical significance of these findings, a NOAEL for non-cancer effects could not be determined (NICNAS, 2020).

In another chronic study, potassium bromate was administered to male F344 rats and male B6C3F1 mice in drinking water at concentrations of 0, 0.02, 0.1, 0.2, and 0.4 g/L and 0, 0.08, 0.4, and 0.8 g/L, respectively, for 100 weeks. The doses were equal to 0, 1.5, 7.9, 16.9, and 37.5 mg/kg bw/day and 0, 9.1, 42.4, and 77.8 mg/kg bw/day, respectively, for rats and mice. In male rats, a statistically significant decrease in the mean body weight and survival was noted at the termination of the study at 0.4 g/L. The decrease in survival and body weight was attributed to an excessive mesothelioma burden. The effects on survival and body weight in rats indicate that the maximum tolerated dose

(MTD) was reached in this study. A significant dose-dependent increase in the incidence of urothelial hyperplasia was noted in rats in the 0.1 g/L and higher dose groups. Foci of mineralisation of the renal papilla and eosinophilic droplets in the proximal tubule epithelium were also noted, without any information on dose levels. There were no other treatment-related non-neoplastic effects observed in any other tissue examined. Based on kidney effects in male rats, a NOAEL of 0.02 g/L (20 ppm; 1.5 mg/kg bw/day) was determined (NICNAS, 2020).

These results also indicate that male B6C3F1 mice are potentially less sensitive to the effects of bromate exposure than rats. Bromate in drinking water had no effect on the body weights and survival of male mice. There was no increased incidence of non-neoplastic lesion in any tissue examined. Therefore, the highest tested dose of 0.8 g/L (77.8 mg/kg bw/day) is a NOAEL for male mice (NICNAS, 2020).

Inhalation

There are no studies available.

Dermal

There are no studies available.

G. Genotoxicity

In vitro Studies

The *in vitro* genotoxicity studies on sodium bromate and potassium bromate are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Sodium Bromate and Potassium Bromate

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial Reverse Mutation Assay (S. typhimurium TA 97, TA98, TA 100, TA 102)	-	-	2	ECHA
Chromosome aberration (Chinese hamster fibroblast cells) *	+	-	-	CIR, 1994
Ames assay (Salmonella typhimurium TA92, TA94, TA98, TA1535, and TA1537)*	-	-	-	CIR, 1994
Ames assay (Salmonella typhimurium TA97, TA98, TA100, TA1535, and TA1537)*	+	+		NICNAS, 2020
Ames assay (Salmonella typhimurium TA100, TA102, TA104)*	-	+	-	CIR, 1994
Bacterial Reverse Mutation Assay (Escherichia coli WP2try$^-$ and WP2try$^-$ his$^-$)*	-	-	-	CIR, 1994
Rec mutagenic assay (Bacillus subtilis)*	-	-	-	CIR, 1994

*+, positive; -, negative
*Potassium bromate

Induction of oxidative DNA modifications in isolated perfused kidneys or calf thymus DNA was not observed after potassium bromate administration. Dose-dependent increases in the number of aberrant metaphase cells in rat bone marrow cells were reported in all treated animals as acute cytogenetic effects of potassium bromate (NICNAS,2020).

In assays using V79 Chinese hamster ovary cells, potassium bromate increased the frequency of cells with micronuclei, the number of chromosomal aberrations and the number of DNA strand breaks and induced gene mutations at the HPRT locus. Many chromosome aberrations observed were chromatid breaks and chromatid exchanges. Significantly increased levels of 8-oxodeoxyguanosine were also detected (Health Canada, 2010; ECHA). The result of a chromosomal aberration assay (Chinese hamster fibroblasts) using potassium bromate indicated a dose-related increase in the frequency of exchange-type aberrations (including gaps) (NICNAS,2020).

Potassium bromate induced deoxyribonucleic acid (DNA) damage in cultured mammalian cells and primary human thyroid, white blood and kidney cells as measured by the in vitro comet assay; micronuclei in cultured mammalian cells and primary human lymphocytes and kidney cells; chromosomal aberrations, DNA repair, sister chromatid exchange, and DNA modifications (increased oxidation of DNA) in mammalian cell lines, primary human cultured cells and cell-free systems; and weak chromosomal aberration induction in cultured mammalian cells (NICNAS,2020).

In Vivo Studies

An OECD guideline 474 (Mammalian Erythrocyte Micronucleus) test was performed in male and female mice (genetically modified: Tg.AC hemizygous, p53 haploinsufficient; n=15 per sex per dose) exposed to a daily dose 0, 64, 128, 256 mg/kg bw/day of sodium bromate via dermal exposure for 26 weeks. Sodium bromate induced a dose response statistically significant increase in the frequency of micronucleated erythrocytes in all of the treated mice which indicates that sodium bromate is mutagenic in this study. There were also significant increases in the percentage of polychromatic erythrocytes among total erythrocytes in the male mice exposed to sodium bromate (ECHA) [KI. score = 1].

An OECD guideline 474 (Mammalian Erythrocyte Micronucleus) test was performed in male and female mice (genetically modified: Tg.AC hemizygous; n=15 per sex per dose) exposed to a daily dose 0, 80, 400, 800 mg/L of sodium bromate in their drinking water for 26 weeks. Sodium bromate induced a dose response statistically significant increase in the frequency of micronucleated erythrocytes in all of the treated mice which indicates that sodium bromate is mutagenic in this study. There were also significant increases in the percentage of polychromatic erythrocytes among total erythrocytes in the male and female mice exposed to sodium bromate (ECHA) [KI. score = 1].

An OECD guideline 474 (Mammalian Erythrocyte Micronucleus) test was performed in male and female mice (p53 haploinsufficient; n=15 per sex/dose) exposed to a daily dose of 0, 80, 400, and 800 mg/L of sodium bromate in their drinking water for 27 weeks. Sodium bromate induced a, dose response, statistically significant increase in the frequency of micronucleated erythrocytes in all of the treated mice which indicates that sodium bromate is mutagenic in this study (ECHA) [KI. score =1].

However, in the carcinogenicity study reported in this publication, sodium bromate, did not show evidence of any carcinogenic activity. Thus, in an overall assessment of the entire data set, it is not possible to conclude on the classification for genotoxicity of sodium bromate (inconclusive).

Potassium bromate induced micronuclei *in vivo* in multiple organs in rats and mice: micronucleated reticulocytes in CD-1 mice following intraperitoneal (IP) injection; peripheral blood cell micronuclei (micronuclei reticulocytes) in male F344 rats following IP injection; micronuclei in femoral bone marrow cells of mice following intraperitoneal injections; and micronucleated polychromatic erythrocytes in two strains of mice following gavage administration (NICNAS, 2020).

Potassium bromate was negative with respect to *in vivo* genotoxicity assays: induction of micronuclei was not observed in spermatids, and no induction of DNA damage was observed in the lung, spleen or bone marrow of mice (NICNAS,2020).

H. Carcinogenicity

Considering that potassium bromate and sodium bromate will produce similar effects through bromate ions, an International Agency for Research on Cancer (IARC) classification of “probably carcinogenic to humans” has been recommended for sodium bromate. This is based on sufficient evidence of carcinogenicity in animal studies for potassium bromate and no data in humans. This is supported by the classification of 'bromate moiety' as a carcinogen by other regulatory agencies. The US EPA has also classified the bromate moiety as a 'probable human carcinogen based on no evidence in humans, but adequate evidence of carcinogenicity in male and female rats' (Group B2 carcinogen) under previous guidelines and as a 'likely human carcinogen by the oral route of exposure, insufficient data for evaluation by the inhalation route' under current guidelines. The World Health Organization (WHO) evaluated the bromate moiety under the WHO Guidelines for Drinking-water Quality and stated that 'the weight of evidence from rat bioassays clearly indicates that bromate has the potential to be a human carcinogen' (NICNAS,2020).

<u>Oral</u>

In a 27-week NTP carcinogenicity study, sodium bromate was administered to genetically modified male and female mice (953 deficient and Tg.AC hemizygous) in water at dose levels of 0, 80, 400 and 800 mg/L. The mice in the 800 mg/L group developed a decrease body weight. There were no increases in tumour incidence nor was there evidence of carcinogenicity in this study (ECHA) [KI.score =1].

In a 43-week NTP carcinogenicity study, sodium bromate was administered to genetically modified male and female mice (p53 deficient and Tg. AC mice) in water at dose levels of 0, 80, 400 and 800 mg/L. The mice in the 800 mg/L group developed a decrease body weight. There were no increases in tumour incidence nor was there evidence of carcinogenicity in this study (ECHA) [KI.score =1].

In a 111-week study in F344 rats (53 male and 53 female) were fed 250 or 500 ppm potassium bromate. All the animals survived the 111-week treatment, but the first renal cell neoplasm was found in a male rat exposed to 500 ppm of potassium bromate during week 14 of treatment. The animals treated with 500 ppm potassium bromate had a decrease in body weight so the concentration of potassium bromate was reduced to 400 ppm at week 60. Neoplasms were identified in the kidneys, testis, peritoneum, thyroid, pituitary, mammary glands, and the spleen in both the treated and control rats. Renal cell neoplasms developed in 0% (control), 56% (250ppm), and 80% (500 ppm) of female rats, and 6% (control), 60% (250 ppm), and 88% (500 ppm) of the male rats. The male rats that survived beyond week 14 and the female rats that survived beyond week 58 were included in the effective number of rats. In the treated rats, the other neoplasms found in the kidneys included two transitional cell carcinomas and one angiosarcoma. One liposarcoma was found in a control rat. More than 80% of the renal cell neoplasms were diagnosed as carcinomas. The mean survival time (88.1 ± 18.1 weeks) was the shortest in male rats fed 500 ppm potassium

bromate in their diet. The mean survival times for the other treated groups were 101-104 weeks. The survival of the controls in week 104 for the female rats was 66% compared with 77.4% for the male rats. Under the conditions of this bioassay, potassium bromate was reported to be carcinogenic and induced renal cell carcinomas in high incidences in a dose-response relationship in both male and female F344 rats (CIR, 1994).

A two-stage, 26-week carcinogenesis study was conducted in F344 rats (128 males) who received 500 or 1000 ppm of potassium bromate in their diet. N-ethyl-N-hydroxyethylnitrosamine (EHEN) was used as an initiator. Ten out of 20 rats developed renal tumours in the 500 ppm EHEN dose group (plus potassium bromate in drinking water) after 24 weeks. Four of the 23 rats who received EHEN, for only two weeks, developed renal cell tumours. Although, potassium bromate induced cancer at two years in other studies, none of the rats who received potassium bromate for 24 weeks developed cancer. The authors concluded that potassium bromate can be classified as a carcinogen that has both initiating and enhancing activities in the kidneys of rats. The initiating activity was not observed in a 104-week study, in which F344 rats (6 weeks old) were given an intragastrical dose of potassium bromate followed by being maintained on a diet containing 4000 ppm sodium barbital as a promoting agent (CIR, 1994).

In another study, male F344 rats (180 male) were divided into twelve groups followed by 500 ppm of EHEN in their drinking water or distilled water for two weeks followed by potassium bromate, potassium bromide, or distilled water for the next 24 weeks. The male rats in groups 1-9 were given EHEN at 500 ppm three times per week for two weeks at the initiation stage. The male rats in groups of 1-6 were given potassium bromate in their drinking water at concentrations of 15, 30, 60, 125, 250, or 500 ppm for 24 weeks. The male rats in groups of 7 and 8 were given potassium bromate for 24 weeks at concentrations of 350 and 1,750 ppm. The rats in group 9 were given distilled water initiation with EHEN. The male rats in group 10-12 were given distilled water for the first two weeks followed by 500 ppm potassium bromate, 1,750 potassium bromide, or distilled water for 24 weeks. The number of dysplastic hepatic foci per cm_2 were significantly increased in a dose-related manner from 15-500 ppm of potassium bromate in their drinking water. The number of renal cell neoplasms per cm^2 were significantly higher in the 500-ppm group. The incidence of dysplastic hepatic foci and renal cell neoplasms did not significantly increase with increasing levels of potassium bromate in the drinking water. The threshold concentration of potassium bromate in the drinking water of the rats, for the enhancement of renal carcinogenesis, was between 15 and 30 ppm. There was no evidence of renal carcinogenesis observed with exposure to potassium bromate in this study (CIR, 1994).

Twenty male Syrian golden hamsters, a species that rarely develop spontaneous renal neoplasms, were administered 125, 250, 500, or 2,000 ppm potassium bromate in their drinking for 89 weeks. There were no apparent differences in the survival time between the control and the treated groups. There was a significant difference in the body weight gain between the control and the high-dose groups. The authors concluded that although the incidence of renal cell tumours in the test group was not statistically significant, the fact that these tumours were not seen in the controls suggests that potassium bromate has the potential to produce tumours in Syrian golden hamsters (CIR, 1994).

Dose response studies were used to evaluate the potential for potassium bromate to induce carcinogenesis in 149 male F344 rats. The rats were given potassium bromate in their drinking water at concentrations of 15, 30, 60, 125, 250, or 500 ppm for a period of 104 weeks. Potassium bromate was dissolved in distilled water at a concentration of one percent as a stock solution refrigerated at 4 ^{o}C and diluted twice weekly before use. Renal cell carcinomas were identified in three of the twenty rats that were exposed to potassium bromate. The combined incidences of renal cell adenocarcinomas and adenomas were significantly increased in rats treated with doses of 125, 250, or 500 pm potassium bromate. Mesotheliomas of the peritoneum were observed in rats fed doses >

300 ppm potassium bromate and the rate was significantly increased in the 500-ppm group. The incidence of interstitial cell adenomas of the testis was very high in both the potassium bromate treated rats and the control rats. Papillomas of the urinary bladder were identified in the rats given water containing 15 or 250 ppm potassium bromate. In this study, renal carcinomas were observed in 20 (15%) of the rats exposed to 500 potassium bromate (CIR, 1994).

Male and female Theiller mice were fed diets containing 79% breadcrumbs made from flour treated with 75 (Group I), 50 (Group II), and 0 mg (Group III) mg/kg of potassium bromate for 80 weeks. Of groups I, II, III, 53, 46, and 35 male mice and 52, 54, and 53 female mice respectively underwent necropsy for detailed histopathological examination. There were no carcinogenic effects produced in mice that were fed bread made from flour that had been treated with potassium bromate before baking (CIR, 1994). A similar feeding studying using male (60) and female (60) Wistar rats was conducted and no carcinogenic effects were produced in any of the treated rats when they were maintained on the treated-bread diet for 104 weeks (CIR, 1994).

Based on these animal feeding studies, IARC classified potassium bromate as an animal carcinogen and a possible carcinogen to humans (given the lack of adequate data). The IARC working group indicated that ionic compounds such as potassium bromate are poorly absorbed through the skin and there is negative data for skin application studies using potassium bromate (CIR, 1994).

Inhalation

There are no studies available.

Dermal

In a 26-week NTP carcinogenicity study, solutions containing sodium bromate were also applied to the backs of male and female Tg.AC mice at dose levels of 0, 64, 128, or 256 mg/kg. The mice in the 256 mg/kg group had a decrease in body weight. There were no increases in tumour incidence nor was there evidence of carcinogenicity in this study (ECHA) [KI. score =1].

In a 39-week NTP carcinogenicity study, solutions containing sodium bromate were also applied to the backs of male and female Tg.AC mice at dose levels of 0, 64, 128, or 256 mg/kg. The mice in the 256 mg/kg group had a decrease in body weight. There were no increases in tumour incidence nor was there evidence of carcinogenicity in this study (ECHA) [KI. score =1].

I. Reproductive Toxicity

Male and female rats were exposed to sodium bromate in a reproductive toxicity study. Sodium bromate did not induce any adverse signs of general toxicity at any dose levels (a maximum tolerated dose was not achieved in this study). Reproductive function in female rats was not adversely impacted and there were no treatment related gross or microscopic changes in the kidney, liver, spleen, testis, or epididymis. Treated male rats in the 250-ppm dose group developed a statistically significant decrease (18%) in epididymal sperm density. However, all other endpoints in the male rats were comparable to controls. A NOAEL of 80 ppm (7.7 mg/kg bw/day) and a LOAEL of 250 ppm (22 mg/kg bw/day) was established for sodium bromate based on changes in sperm density in male rats. (ECHA) [KI. score =2].

J. Developmental Toxicity

Oral

In a multigeneration, continuous-breeding paradigm, sodium bromate was administered to male and female Sprague-Dawley (SD) rats in drinking water at concentrations of 0, 30, 100, and 300 mg/L. The chemical produced general toxicity in male and female SD rats at 100 and 300 mg/L as noted by chronic progressive nephropathy and hyaline droplets in males and renal cell proliferative changes in females. Even though the chemical produced a 16 % decrease in sperm density in the F0 generation, the chemical is not considered a reproductive toxicant as no treatment-related changes were observed in the reproductive litter parameters. Although the sperm density was also decreased by 8 % in the F1 generation, the change was not statistically significant (NICNAS,2020).

Inhalation

There are no studies available.

Dermal

There are no studies available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for sodium bromate follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

An oral reference dose was not derived for sodium bromate. Sodium bromate will dissociate in water to form sodium (Na+) and bromate (BrO_3^-) ions.

The Australian drinking water guideline (DWG) value for bromate is 0.02 mg/L based on health considerations. Drinking water that contains 2-10% bromate can cause toxic effects including nausea, abdominal pain, diarrhea, central nervous system depression, and pulmonary oedema which are mostly reversible. However, irreversible effects include kidney failure and deafness (ADWG, 2011).

There is also an Australian drinking water guideline value of 180 mg/L for sodium based on aesthetic considerations (taste). Excessive sodium intake can severely aggravate chronic congestive heart failure (ADWG, 2011).

B. Cancer

There is no evidence that sodium bromate is carcinogenic. However, the bromate moiety is classified as a possible carcinogen by regulatory agencies. There are animal studies that suggest that potassium bromate (surrogate chemical for sodium bromate) is a possible carcinogen to humans (CIR 1994, ECHA, NICNAS,2020).Therefore, under considerations of the classification from the structural analogue potassium bromate, sodium bromate is also suspected to be a carcinogen

(ECHA). However, a cancer reference value was not derived. As described above, an Australian DWG value is available for bromate.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Sodium bromate is not combustible but enhances combustion of other substances. It is a strong oxidizer. The substance gives off irritating or toxic fumes (or gases) in a fire. Further, there is a risk of fire and explosion on contact with combustible substances or reducing agents (PubChem).

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

There are limited environmental studies evaluating the ecotoxicological effects of sodium bromate. Based on read across using surrogate chemical potassium bromate, sodium bromate is of low acute and chronic toxicity concern to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on potassium bromate.

Table 3: Acute Aquatic Toxicity Studies on Potassium Bromate

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
*Morone saxatillis (striped bass)**	96-hour-LC_{50}	30.8	2	ECHA
*Morone saxatillis (striped bass)***	48-hour LC_{50}	605.0	2	ECHA
Leiostomus xanthurus	24-hour-LC_{50}	698.0	2	ECHA
Daphnia magna	48-hour EC_{50}	>100	1	ECHA
Desmodesmus subpicatus	72-hour EC_{50}	>100	1	ECHA

*Newly hatched
**four-day old

Chronic Studies

Table 4 lists the results of chronic aquatic toxicity studies conducted on potassium bromate

Table 4: Chronic Aquatic Toxicity Studies on Potassium Bromate

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Morone saxatillis (striped bass)	10-day LC_{50}	92.6	2	ECHA
Leiostomus xanthurus	10-day LC_{50}	278.6	2	ECHA
Desmodesmus subspicatus	72-hour NOEC	31.6	1	ECHA

C. Terrestrial Toxicity

There are no studies available.

D. Calculation of PNEC

The PNEC calculations for sodium bromate follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic level. Acute $E(L)C_{50}$ values are available for fish (30.8 mg/L), invertebrates (>100), and algae (>100). Chronic LC_{50} values from long-term studies are available for two tropic levels including fish (92.6 mg/L) and algae (31.6). On the basis that the data consists of short-term results from three trophic levels and long-term results from two trophic levels, an assessment factor of 100 has been applied to the lowest reported $E(L)C_{50}$ value of 30.8 mg/L for fish. The $E(L)C_{50}$ value is used because the value for acute toxicity value fish is lower than the chronic values for this trophic level. The $PNEC_{water}$ is 0.308 mg/L.

PNEC Sediment

No experimental toxicity data on sediment organisms are available. Sodium bromate's environmental distribution is dominated by its high-water solubility. K_{oc} parameter do not readily apply to inorganics, such as sodium bromate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{sediment}$. Based on its properties, no adsorption of sodium bromate to sediment is to be expected, and the assessment of this compartment will be covered by the aquatic assessment.

PNEC Soil

No experimental toxicity data on terrestrial organisms are available. The environmental distribution of sodium bromate is dominated by its water solubility. Sorption of sodium bromate should probably be regarded as a reversible situation, i.e., the substance is not tightly nor permanently bound. K_{oc} parameters do not readily apply to inorganics, such as sodium bromate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{soil}$. Based on its properties, sodium bromate is not expected to significantly adsorb to soil, and the assessment of this compartment will be covered by the aquatic assessment.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Sodium bromate is an inorganic salt that will dissociate to sodium and bromate ions. Biodegradation is not applicable to this inorganic chemical. For the purpose of this PBT assessment, the persistent criteria are not considered applicable to this inorganic salt.

Sodium bromate is not expected to bioaccumulate because its dissociated species are inorganic ions. Thus, the substance does not meet the criteria for bioaccumulation.

There are no aquatic toxicity data on sodium bromate. The lowest NOEC from chronic aquatic toxicity studies on potassium bromate, a structural analogue, are >0.1 mg/L. The acute $E(L)C_{50}$ values

from the acute aquatic toxicity studies on potassium bromate are > 1 mg/L. Thus, sodium bromate does not meet the criteria for toxicity.

The overall conclusion is that sodium bromate is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H272: May intensify fire; oxidiser

H302: Harmful if swallowed

H315: Causes skin irritation

H319: Causes serious eye irritation

H335: May cause respiratory irritation.

H351: Suspected of causing cancer

Acute toxicity-category 3

Carcinogenicity-category 1B

B. Labelling

Danger

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

Eye Contact

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use personal protective clothing. Avoid dust formation. Ensure adequate ventilation. Do not breathe dust. Wear respiratory protection if ventilation is inadequate. Avoid contact with skin, eye, and clothing.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Good general ventilation should be used. Ventilation rates should be matched to conditions. If applicable, use process enclosures, local exhaust ventilation, or other engineering controls to maintain airborne levels below recommended exposure limits

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

There are no workplace exposure standards established for sodium bromate in Australia.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Use respiratory protection when airborne concentrations are expected to be high.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

The transport classification of sodium bromate is UN1494 class 51 II 02 (ECHA) [KI. score =2].

UN 1494

Class: 5.1

Packaging Group: II

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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SODIUM ERYTHORBATE

This dossier on sodium erythorbate presents the most critical studies pertinent to the risk assessment of sodium erythorbate in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): sodium;(2R)-2-[(1R)-1,2-dihydroxyethyl]-4-hydroxy-5-oxo-2H-furan-3-olate

CAS RN: 6381-77-7

Molecular formula: $C_6H_7NaO_6$

Molecular weight: 198.11 g/mol

Synonyms: D-araboascorbic acid, erythorbic acid, erythroascorbic acid, isoascorbic acid, isoascorbic acid, disodium salt, isoascorbic acid, monosodium salt, isoascorbic acid, sodium salt, 2,3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone.

SMILES: C(C(C1C(=C(C(=O)O1)O)[O-])O)O.[Na+]

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-chemical Properties of Sodium Erythorbate

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Crystalline, odourless solid	2	ECHA
Melting Point	>160°C (decomposes at 180°C) @ 101.3 kPa	2	ECHA
Boiling Point	-	-	-
Density	1702 kg/m^3 @ 20°C	2	ECHA
Vapor Pressure	0 Pa at 25°C	2	ECHA
Partition Coefficient (log K_{ow})	-3.29 (estimated) @ 25°C	2	ECHA
Water Solubility	146 g/L at 20°C	1	ECHA
Flash Point[a]	Study scientifically not necessary	-	ECHA
Auto flammability[a]	Study scientifically not necessary	-	ECHA
Flammablility[a]	Non-flammable	2	ECHA
Viscosity	As solid, study scientifically not necessary	-	ECHA
Henry's Law Constant	Not available	-	-

a - The substance has no pyrophoric properties and does not liberate flammable gases on contact with water. It is not highly flammable solid derived from preliminary screening test result of flammable solids.

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Sodium erythorbate is highly soluble in water and it has a low potential to bind to soil or sediment. It is ultimately biodegradable. Sodium erythorbate is not expected to bioaccumulate.

B. Biodegradation

In an OECD 301E compliant test, the degradation after the 28-day plateau was not yet visible in the degradation curve. The DOC elimination was 56% after 28 days. Thus, the substance can't be considered as readily degradable. However, under strict test conditions, the substance appears to be ultimately biodegradable (under the subclassification of inherent biodegradability) (ECHA) [Kl. score = 2].

If a chemical is found to be readily or inherently biodegradable, it is categorized as Not Persistent since its half-life is substantially less than 60 days.

C. Environmental Distribution

No experimental data are available for sodium erythorbate. Based on its log K_{ow} and high-water solubility values, if released to the soil, sodium erythorbate is expected to have a low potential for adsorption and a high potential for mobility. If released to water, it is likely to remain in water and it is not expected to adsorb to sediment.

D. Bioaccumulation

There are no bioaccumulation studies available for sodium erythorbate. The bioconcentration factor (BCF) was estimated to be 0.8933 based on the Arnot-Gobas method (for the upper trophic level) (USEPA, 2020). Based on the estimated BCF and the low log K_{ow} value of – 3.29, bioaccumulation is not expected.

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Sodium erythorbate is absorbed orally and dermally. However, the acute toxicity of sodium erythorbate is low by oral and dermal routes of exposure. Sodium erythorbate is not irritating to the eyes or the skin and it is not a skin sensitizer. Sodium erythorbate is not genotoxic or carcinogenic. There is no evidence to suggest that sodium erythorbate elicits reproductive toxicity or developmental toxicity.

B. Pharmacokinetics/Metabolism

<u>Absorption - Oral</u>

In accordance with the ECHA Guidance on Information Requirements and Chemical Safety Assessment, Chapter R.7C Section R.7.12 (Endpoint Specific Guidance; ECHA, 2008), the physico-chemical properties can provide an insight into the potential behaviour of 2,3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone in the body.

The molecular weight (199.12 g/mol) and water solubility of 146 g/L at 20°C are favourable for oral absorption of 2,3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone. The log P of -3.29 (estimated) suggests that 2,3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone is considerably hydrophilic and is not in the favourable range for passive diffusion (log P: -1 to 4) or absorption via the lymphatic system (log >5). As 2,3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone is very hydrophilic and the molecular weight is <200 g/mol, it may pass through aqueous pore, be carried through the epithelial barrier by the bulk passage of water, or an active transport mechanism may be involved (ECHA).

Absorption – Dermal

The molecular weight of 199.12 g/mol is above the range for favourable dermal absorption (<100 g/mol). The water solubility of 149 g/L at 20°C and poor lipophilicity (log P of -3.29, estimated) indicate that 2,3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone is likely to be too hydrophilic to cross the stratum corneum, therefore dermal absorption is likely to be low (ECHA).

Absorption – Inhalation

The particle size distribution report for 2,3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone indicates ranges from 4.365 µm - 1096.478 µm. The % of particles available in the inhalable fractions of air (<100 µm) is likely to be negligible. Based on the molecular weight (199.12 g/mol), water solubility (149 g/L) and particle size, 2,3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone may readily diffuse/dissolve into the mucus lining of the respiratory tract. As it is very hydrophilic (log P: -3.29) it may be absorbed through aqueous pores (molecular weight <200 g/mol) or be retained in the mucus and transported out of the respiratory tract. Therefore, there is potential for absorption via the inhalation route (ECHA).

Distribution/Metabolism/Excretion

The molecular weight (199.12 g/mol) and water solubility (149 g/L at 20°C) of 2,3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone are favourable for wide distribution, but the very low log P (-3.29, estimated) indicates it is not likely to accumulate in fat during intermittent exposure (ECHA).

In a dietary study, 3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone was administered to Male F344 rats (five per group) at dose levels of 5% for 22 weeks. The rats eliminated totals of 203.3 ± 33.2 mg/100 mL erythorbic acid and 9.0 ± 5.1 mg/100 mL dehydroerythorbic acid during the study. Ascorbic acid and dehydroascorbic acid were not detected. Urine pH was 6.98 ±0.31, which was significantly different from that of rats given basal diet alone (6.31 ± 0.18; $p < 0.05$). Urine osmolarity also differed significantly from controls; osmolarity was 1378 ± 277 mOsmol/kg H20 in rats given Sodium Erythorbate and 1756 ± 200 mOsmol/kg H20 in rats of the control group. Crystals were detected in urine of rats given basal diet and sodium erythorbate or basal diet alone. This study indicated that erythorbic acid is the major metabolite and dehydroerythorbic acid is the minor metabolite of 3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone and it is expected to be excreted in the urine (ECHA).

Based upon the molecular weight of 199.12 g/mol and water solubility of 149 g/L at 20°C, it is likely that 2,3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone is excreted mainly in the urine (ECHA).

C. Acute Toxicity

Oral

Ten fasted albino rats were administered 5,000 mg/kg of sodium erythorbate in a 50% aqueous suspension. Clinical observations were noted at 3, 5, and 24 hrs. post-dosing using an unspecified standard acute toxicity test. The treated rats had soft, pasty stools within 3 hours of dosing, followed in 2 hours by marked diarrhea that persisted for 24 hrs. The LD_{50} was determined to be > 5,000 mg/kg bw (ECHA) [Kl. score=2].

Inhalation

There are no inhalation toxicity studies available.

Dermal

Sodium erythorbate (2,000 mg/kg) was applied to the intact and abraded skin of six rabbits. Each test site was moistened with physiological saline just prior to dosing. After application of the test material, the exposure area was covered with a double layer of surgical gauze and a piece of rubber dam (occlusive dressing). The trunk of each rabbit was wrapped in a stockinette, which was secured to the body with tape. The dressings were removed after 24 hours, and the amount of residual sample and signs of localized irritation were noted. The exposure area was cleaned by thorough wiping, and the rabbits were observed for signs of toxicity for signs of toxicity for 48 hours, 72 hours, and 14 days.

The behaviour, body weight gain, and consumption of feed and water were normal for all of the animals, and no signs of toxicity were observed. No erythema, oedema, or other signs of dermal irritation were observed at five of six test sites. One rabbit (abraded skin) had slight (1+) erythema at 24 hours that cleared by 48 hours.

The dermal LD_{50} was determined to be > 2000 mg/kg bw (ECHA)[Kl. score=2].

D. Irritation

Skin

Sodium erythorbate powder (2,000 mg/kg) was applied to the intact and abraded skin of six albino rabbits. Each test site was moistened with physiological saline just prior to dosing. After application of the test material, the exposure area was covered with a double layer of surgical gauze and a piece of rubber dam. The trunk of each rabbit was wrapped in a stockinette, which was secured to the body with tape. The dressings were removed after 24 hours, and the amount of residual sample and signs of localized irritation were noted. The exposure area was cleaned by thorough wiping, and the rabbits were observed for signs of toxicity for 48 hours, 72 hours, and 14 days.

No erythema, oedema, or other signs of dermal irritation were observed at five of six test sites. One rabbit (abraded skin) had slight erythema at 24 hours that cleared by 48 hours. In this study, sodium erythorbate is not a dermal irritant (ECHA) [Kl. score=2].

Eye

Sodium erythorbate powder (100 mg) was instilled into the conjunctival sac of albino rabbits (10 male and 2 female). The eyes of half of the treated rabbits were rinsed after 5 seconds and the

rabbits were observed for two days. The reactions were compared between rinsed and unrinsed eyes and the following irritation parameters were noted: iris, conjunctival redness. The reactions were comparable in rinsed and unrinsed eyes and were slight and transient in nature.

One hour after dosing, two of six unrinsed eyes had congestion of the iris, but the iris reacted normally to light. Varying degrees of redness were observed in the lids of all unrinsed eyes. Slight redness of the nictitating membrane or palpebral conjunctiva at the medial canthus was observed in two unrinsed eyes.

At one hour, 1+ iritis was observed in one rinsed eye. Five of six rinsed eyes had slight redness that was limited to only the nictitating membrane in three cases. At 24 hours, all eyes were normal, with the exception of one that had slight reddening of the conjunctiva at the medial canthus. All eyes, rinsed and unrinsed, were normal at 48 hours.

The mean ocular irritation scores after 48 hours (2 days) were 0.33/110 (unrinsed eyes) and 0.17/110 (rinsed eyes). Therefore, in this study, sodium erythorbate is not an eye irritant (ECHA) [Kl. score=2].

E. Sensitisation

An OECD Guideline 429 (Skin Sensitization: Local Lymph Node Assay) was performed.

In the dermal sensitisation study with sodium erythorbate (5, 10, 25% w/w in propylene glycol), young adult female CBA/Ca (CBA/CaOlaHsd) mice (4/group) were tested using the local lymph node assay (LLNA). The reliability of the test system was confirmed by the most recent positive control assay (Phenylacetaldehyde [>90%] in propylene glycol; January 2013).

There was no mortality and all animals appeared normal throughout the study. There were no statistically significant differences observed between any treatment groups with respect to body weight. Treatment with sodium erythorbate at 5, 10, or 25% (w/w) resulted in stimulation indices (SI) of 1.13, 0.91, and 1.29 respectively.

In this study, sodium erythorbate is not a potential skin sensitiser (ECHA) [Kl. score=1].

F. Combined Repeated Dose and Carcinogenicity Evaluation

<u>Oral</u>

A combined repeated dose and carcinogenicity study was conducted (Inai et. al. 1989; as cited in ECHA) [KI. score = 2].

In a preliminary test male and female B6C3F1 mice (10 per sex per group) were given drinking water containing 0.625%, 1.25%, 2.5%, 5.0%, or 10% sodium erythrobate for 10 weeks. Water and feed were available ad libitum. The untreated control group consisted of 20 male and 20 female mice. Mortality, bodyweight gain, gross pathology, and histopathology were noted (ECHA).

In the main test, sodium erythorbate was administered in drinking water to male B6C3F1 mice at concentrations of 1.25% and 2.5%. Female mice received 2.5% and 5% (maximum tolerated dose [MTD]). Each group contained 50 mice. Treatment continued for 96 weeks; the study was terminated at week 110. Feed and water were available ad libitum. Mortality, body weight, organ weights and neoplastic histopathology were noted.

In the preliminary study, six male mice and one female mouse of the 10% dosing group had died by the end of week 1. In male mice given 5.0% sodium erythrobate, the average weekly body weight gain was slightly less than 90% that of the control female mice. Body weight gain was increased in female mice given sodium erythrobate at a concentration of 5.0%, compared to that of control mice. No significant changes were observed in the visceral organs of untreated mice or mice given the dose less than or equal to the MTD of sodium erythrobate. Mice given doses greater than the MTD had marked atrophy of both hepatocytes and splenic lymphoid follicles, as well as hydropic degeneration of the renal tubular epithelium. The MTD of sodium erythrobate in drinking water was 2.5% (25,000 mg/L) for male mice (2,400 mg/kg-day)[1] and 5.0% (50,000 mg/L) for female mice (2456 mg/kg -day)[1], respectively.

In the main study, the average body weights of the treated mice were similar to controls. Of the male mice (without tumours) that survived beyond week 43, dose-dependent reductions in the heart and brain weights were observed. The weights of the heart, lungs, kidneys, and brain of female mice (without tumours) were significantly different between the high dose group and the control group. At the doses tested, there was not a treatment-related increase in tumour incidence when compared to controls. Overall, tumour incidence, time to death with tumours, and the distribution of tumours in treated mice did not differ significantly from mice of the control group. The data from the main study indicates that sodium erythrobate is not a carcinogen under the conditions of the study (ECHA) [Kl Score=2].

For the purposes of this dossier, the MTD of 2,400 mg/kg-day for male mice was considered the NOAEL.

In a repeated dose and carcinogenicity study, sodium erythorbate was administered to 10 Fischer 344/DuCrj rats/sex/dose in water at dose levels of 0, 0.625, 1.25, 2.5, 5 and 10% for 13 weeks (preliminary study) and then to 52 male/50 female Fischer 344/DuCrj rats in water at dose levels of 1.25 and 2.5% for 104 weeks (main study) (ECHA)[KI. score =2].

In the preliminary study, all the rats given the 10% solution refused to drink and died in 2 to 5 weeks. Three males and one female out of the 10 given the 5% solution died during the first 4 days. All the rats given the 2.5% and lower concentrations survived to the end of 13 weeks. The 2.5% solution suppressed body weight gains by 12% in males and by 6% in females as compared with nontreated controls.

In the main study, body weight gain was normal in low dose group rats and was reduced by 8.5% for males and 15.5% for females at weeks 88 and 85, respectively in rats given 2.5% sodium erythorbate. At the doses tested, there was not a treatment related increase in tumour incidence when compared to controls. The pattern of occurrence of the various types of tumours was similar among the groups. A NOAEL was not established for this study (ECHA)[KI. score =2].

Spontaneous testicular interstitial-cell tumours, endometrial stromal polyps, mammary fibroadenomas, adrenal pheochromocytomas, and other endocrine tumours in control rats showed a pattern of incidence similar to that of earlier reports by others. The incidence of leukemias in female controls, however, was higher at 37.8% than in prior studies by others, (9.9%, 11.0% and 21.9%). The authors had no specific explanation for the difference (ECHA)[KI. score =2].

[1] NTP 2009. Converted using mean mouse water ingestion rates and body weights at 53-101 week old animals Tables J3 and J from NTP 2009. NTP TECHNICAL REPORT ON THE TOXICOLOGY AND CARCINOGENESIS STUDIES OF BROMOCHLOROACETIC ACID (CAS NO. 5589-96-8) IN F344/N RATS AND B6C3F1 MICE (DRINKING WATER STUDIES) NATIONAL TOXICOLOGY PROGRAM Research Triangle Park, NC 27709 NTP TR 549 NIH Publication No. 09-5890

Inhalation

There are no repeat dose inhalation data available.

Dermal

There are no repeat dose dermal data available.

G. Genotoxicity

In vitro Studies

Table 2 presents the results of the *in vitro* genotoxicity studies on sodium erythorbate.

Table 2: *In vitro* Genotoxicity Studies on sodium erythorbate

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Chromosomal aberration Chinese hamster lung (CHL))	-	-	2	Matsuoka et al 1979. Andersen 1999. (as cited in ECHA).
Bacterial Reverse Mutation Assay (S. typhimurium TA 1535, TA 1537, TA 98, TA 100, TA 92, and TA 94)	-	-	2	ECHA

*+, positive; -, negative; NA, not applicable

In vivo Studies

A chromosome aberration (rodent dominant lethal assay) was performed (Jorgenson et. al. 1978; as cited in ECHA). Proven breeder male rats were distributed into groups of 10 each. Treatments were by oral gavage as a single dose and with 5 consecutive daily doses; 3 dosage levels were used for each regimen. Untreated reference controls and positive controls receiving a single intraperitoneal injection of triethylenemelamine were used with each compound studied. Following treatment, each single-dose male was mated to two adult females weekly for 8 weeks; each multiple-dosed male was mated to two adult females weekly for 7 weeks.

The positive control induced the appropriate mutagenic response. No consistent responses occurred to suggest that sodium erythorbate was not mutagenic to the rat by the dominant lethal procedure (ECHA) [Kl. score = 2].

Male and female mice were treated with two dose levels of sodium erythorbate for seven weeks in a chromosome aberration (mouse heritable translocation assay). The positive control (triethylenemelamine) induced the appropriate response (positive translocations). Cytogenetic examinations were made on meiotic cells from the males considered as the presumptive positives following two successive breeding. All the breeding data were evaluated and correlated with the cytogenetic examinations. There were no positive reciprocal translocations observed in the control and sodium erythorbate treated groups. Sodium erythorbate did not induce heritable translocation heterozygosity.

H. Carcinogenicity

See combined repeated dose and carcinogenicity study above (Section F).

I. Reproductive Toxicity

There are no specific reproductive toxicity studies have been conducted on sodium erythorbate by any route of exposure.

J. Developmental Toxicity

Oral

An OECD Guideline 414 (Prenatal Developmental Toxicity) study was performed in Wistar rats. (Andersen 1999; as cited in ECHA) [KI. score =2]. The female rats were mated with young adult males and observation of the vaginal sperm plug was considered Day 0 of gestation. (One male was not permitted to impregnate more than one female per group). Pregnant females were dosed orally in a water carrier via oral gavage at doses of 9.0, 41.8, 194.0, or 900.0 mg/kg bw/day of sodium erythorbate on days 6-15 of gestation. All dams were subjected to caesarean section on day 20.

The number of animals for each dosage group were as follows.

- Positive control: 22 animals
- 0, 900 mg/kg bw/day: 24 animals
- 9, 41.8 mg/kg bw/day: 20 animals
- 194 mg/kg bw/day: 21 animals

Maternal Effects: No statistically significant differences were observed in number of pregnancies, corpus lutea, implantation rates, live births, resorptions, dams with >1 site resorbed, dams with all sites resorbed, % partial resorptions, complete resorptions, number live foetuses (average/dam) between treated and control groups. The NOAEL for maternal effects was determined to be 900 mg/kg bw/day (ECHA) [KI. score =2].

Foetal Effects: No statistically significant differences in average foetus weight or number of live foetuses examined at term in rats of the negative control group or in rats given sodium erythorbate. No gross, skeletal or soft tissue morphological abnormalities were observed in rats of the negative control group or in rats given sodium erythorbate. The NOAEL for developmental effects was determined to be 900 mg/kg bw/day (ECHA) [KI. score= 2].

In a developmental toxicity study, sodium erythorbate was administered to CD-1 mice by oral gavage at dose levels of 0, 10.3, 47.8, 221.9, 1030 mg/kg bw/day from day 6-15 of gestation. All of the dams were subjected to caesarean section on gestation day 17). There were no deaths or premature deliveries recorded in this study. The highest dose tested (1030 mg/kg bw/day) did not produce any discernible effects on maternal or foetal survival. Therefore, the NOAEL for maternal toxicity was determined to be 1030 mg/kg bw/day.

One pup of a dam from the positive control group developed exophthalmos, encephalomeningocele, and gastroschisis. A cleft palate was also observed in one of the pups from the group treated with 1030 mg/kg bw/day of sodium erythorbate. The number of abnormalities observed in either soft or skeletal tissues of the mice treated with sodium erythorbate did not differ from the number of spontaneously occurring abnormalities in the sham treated controls. Therefore, the NOAEL for developmental toxicity was determined to be 1030 mg/kg bw/day (ECHA) [KI. score= 2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for sodium erythorbate follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

A prenatal developmental toxicity study discussed in Section J provided the basis for the NOAEL of 900 mg/kg bw/day in rats. The NOAEL of 900 mg/kg bw/day will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:
UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 10
UF_D (database uncertainty) = 1

Oral RfD = 900/1000 = 0.9 mg/kg-day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,
Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:
Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)

Drinking water guidance value = (0.9 x 70 x 0.1)/2 = 3.15 mg/L

B. Cancer

Sodium erythorbate was not carcinogenic to mice in a combined repeated dose and carcinogenicity study (Inai et. al 1989; as cited in ECHA). Thus, a cancer reference value for sodium erythorbate was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Sodium erythorbate does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Sodium erythorbate exhibits low acute toxicity to aquatic organisms.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies on sodium erythorbate.

Table 3: Acute Aquatic Toxicity Studies on Sodium erythorbate

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Oncorhynchus mykiss	96-h LC_{50}	>100	1	ECHA
Daphnia magna	48-h EC_{50}	>100	1	ECHA
Raphidocelis subcapitata	72-h EC_{50}	>160	1	ECHA

Chronic Studies

There are no chronic aquatic toxicity studies for fish and invertebrates. However, there is a 72-hour NOEC value of 20 mg/L reported for *Raphidocelis subcapitata* (previous names: *Pseudokirchneriella subcapitata, Selenastrum capricornutum*). (ECHA) [Kl. score = 1].

C. Terrestrial Toxicity

There are no terrestrial toxicity data available for sodium erythorbate.

D. Calculation of PNEC

The PNEC calculations for sodium erythorbate follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E (L)C_{50} values are available for fish (>100 mg/L), Daphnia (>100 mg/L), and algae (>160 mg/L) . A chronic NOEC value is also available for algae (20 mg/L). On the basis that the data consists of short-term results from three trophic levels and long-term results from one trophic level, an assessment factor of 100 has been applied to the NOEC of 20 mg/L for algae. The $PNEC_{water}$ is 0.2 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Moreover, the substance is not expected to substantially partition to sediments. Nonetheless, a $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 0.16 mg/kg sediment wet weight.

The calculations are as follows:

$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= 0.922/1280 \times 1000 \times 0.2$
$= 0.155$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]

$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1000 \times BD_{solid}]$
$= 0.8 + [(0.2 \times 0.4/1000 \times 2400]$
$= 0.992\ m^3/m^3$

Where:
Kp_{sed} = solid-water partition coefficient (L/kg).
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]

$Kp_{sed} = K_{oc} \times f_{oc}$
$= 10 \times 0.04$
$= 0.4$ L/kg

Where:
K_{oc} = organic carbon normalized distribution coefficient (L/kg). The K_{oc} was calculated using EPISUITE via the molecular connectivity index (MCI) method to be 10 L/kg (USEPA, 2020).
f_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 0.027 mg/kg soil dry weight.

The calculations are as follows:

$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
$= (0.2/1500) \times 1000 \times 0.2$
$= 0.0267$ mg/kg soil dry weight

Where:
Kp_{soil} = soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]

$Kp_{soil} = K_{oc} \times f_{oc}$
$= 10 \times 0.02$

= 0.2 m^3/m^3

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} was calculated using EPISUITE via the MCI method to be 10 L/kg (USEPA, 2020).
f_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Sodium erythorbate is ultimately biodegradable; thus, it does not meet the screening criteria for persistence.

Based on a log K_{ow} value of – 3.29, sodium erythorbate does not meet the criteria for bioaccumulation.

The lowest chronic NOEC value for sodium erythorbate is >0.1 mg/L. The $E(L)C_{50}$ values from acute aquatic toxicity studies on sodium erythorbate are >1 mg/L. Thus, this substance does not meet the screening criteria for toxicity.

Therefore, sodium erythorbate is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Not classified

B. Labelling

No label

C. Pictogram

None

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. Get medical attention immediately, preferably a physician for an ophthalmologic examination.

Skin Contact

Remove contaminated clothing. Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Give artificial respiration if victim is not breathing. Get medical attention.

Ingestion

Rinse mouth with water and then drink plenty of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: nitrogen oxides, carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice. Avoid contact with skin, eyes, and clothing.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage and Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for sodium erythorbate.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection:

Use respiratory protection in case of vapor or aerosol release.

Hand Protection:
Chemical resistant protective gloves.

Skin Protection:

Body protection must be chosen depending on activity and possible exposure.

Eye protection:
Safety glasses with side-shields.

Other Precautions:
Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Sodium erythorbate is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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SODIUM GLUCONATE

This dossier on sodium gluconate presents the most critical studies pertinent to the risk assessment of this substance in its use in coal seam and shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from Organization for Economic Cooperation and Development Screening Information Dataset (OECD SIDS) (OECD, 2004). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed sodium gluconate in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment.

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Sodium D-gluconate

CAS RN: 527-07-1

Molecular formula: $C_6H_{11}NaO_7$

Molecular weight: 218.14 g/mol

Synonyms: Sodium gluconate; Sodium D-gluconate 527-07-1; D-Gluconic acid, monosodium salt;D-Gluconic acid sodium salt

SMILES: C(C(C(C(C(C(=O)[O-])O)O)O)O)O.[Na+]

II. PHYSICO-CHEMICAL PROPERTIES

Key physical and chemical properties for the substance are shown in Table 1.

Table 1: Overview of the Physico-Chemical Properties of Sodium Gluconate

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Dry, white, crystalline powder	-	PubChem
Melting Point	205-209 °C (pressure not provided)	-	OECD, 2004
Boiling Point	613.1 °C (pressure not provided)	-	OECD, 2004
Density	1790 kg/m^3 @ 20 °C	-	PubChem
Vapor Pressure	Negligible @ 25 °C	-	OECD, 2004
Partition Coefficient (log K_{ow})	-5.99	-	OECD, 2004
Water Solubility	590 g/L @ 25 °C	-	OECD, 2004
Dissociation constant (pKa)	3.70	-	OECD, 2004

Sodium gluconate is the sodium salt of gluconic acid. Gluconic acid is a naturally occurring weak acid and its dissociation in water is expected to be complete. Sodium gluconate is a chelator that forms stable complexes with various ions and ultimately prevents these ions from engaging in chemical reactions. Gluconates are naturally occurring substances that freely dissociate to the gluconate anion and its respective cations. Gluconate is used as a chelating agent in many cleaning products, industrial applications, and foodstuffs.

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Sodium gluconate is readily biodegradable. It is not expected to bioaccumulate, and it has low potential to adsorb to sediment and soil.

B. Partitioning

Sodium gluconate is highly soluble in water. Volatilisation from water or moist soil surfaces is not expected to be an important fate process based upon its water solubility and that it is a salt. It is not expected to volatilise from dry soil surfaces based upon its estimated negligible vapour pressure.

C. Biodegradation

Sodium gluconate is readily biodegradable under both aerobic and anaerobic conditions. In an aerobic closed bottle test of sodium gluconate, the biodegradation was 89% expressed as the Theoretical Oxygen Demand after 28 days; while under anaerobic conditions, 100% of sodium gluconate was determined as degraded after 35 days. These data demonstrate that gluconates are readily biodegradable both under aerobic and anaerobic test conditions (OECD, 2004).

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

D. Environmental Distribution

No experimental data are available for sodium gluconate. Using KOCWIN in EPISuite™ (USEPA, 2018), the estimated K_{oc} value from log K_{ow} is 0.0001046 litres per kilogram (L/kg). The estimated K_{oc} value from the molecular connectivity index (MCI) is 10 L/kg. Based on these values, sodium gluconate has a low potential for adsorption to soil and sediment and is expected to have very high mobility in soil.

E. Bioaccumulation

Based on a log K_{ow} value of -5.99, sodium gluconate has a very low potential for bioaccumulation. This is further supported by metabolic in vivo studies showing that gluconate is readily catabolized or utilized for glucose synthesis (OECD, 2004).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Gluconic acid, the anion of sodium gluconate, is a normal metabolic product of glucose metabolism in mammals. It exhibits low acute toxicity by the oral route. No irritation or skin sensitisation studies

are available. However, it is not expected to be a skin sensitiser. None of the repeated dose toxicity studies showed any significant toxicological effects. Sodium gluconate is not genotoxic.

B. Metabolism

Gluconic acid, the anion of sodium gluconate, is a normal metabolic product of glucose metabolism in mammals. Orally administered gluconate is absorbed rapidly in mammals, A major part is excreted in the urine and the remainder is metabolized (OECD, 2004)

C. Acute toxicity

Oral

Data on acute oral toxicity for sodium gluconate in rat (Mochizuki, M, Bozo Research Center 1995) (doses: 500, 1000, 2000 milligrams per kilogram [mg/kg]) and dog (Okamoto M., 1995) (doses: 1000 and 2000 mg/kg) fed by gavage showed no death at any dose, hence the minimum lethal dose was estimated > 2000 mg/kg for both species.

Inhalation

No acute studies are available.

Dermal

No acute studies are available.

D. Irritation

No studies are available. It is not considered a skin or eye irritant based on studies conducted on similar substance gluconic acid (OECD, 2004).

E. Sensitisation

No studies are available.

F. Repeat Dose Toxicity

Oral

A 28-day study was conducted by feeding rats by gavage with sodium gluconate at doses of 0, 500, 1,000, 2,000 mg/kg body weight in water at a volume of 1 millilitre (mL)/ 100 grams (g) bw. No death or clinical signs of abnormality were observed in any of the groups. Histopathological examination showed a thickening of the limiting ridge of the stomach in 5 out of 12 males at 2,000 mg/kg bw per day dose. No toxic changes associated with the test article were detected. As the limiting ridge is a tissue specific to rodents, this lesion is not toxicologically relevant for humans. Other lesions occurred incidentally and were not treatment related. The NOAEL was estimated to be 1,000 mg/kg bw/day for males and 2000 mg/kg bw/day for females (Mochizuki, M, Bozo Research Centre, 1995).

Another 28-day toxicity study in rats fed with a diet containing up to 5% w/w sodium gluconate (max. 4,100 mg/kg bw for males and 4,400 mg/kg bw for females) was conducted using a control group receiving equivalent concentration of sodium in the form of NaCl to differentiate the potential effects of high doses of sodium intake. No deaths occurred during the study period. No revisions in the general condition, body weight, or food and water intake were observed in the animals over the

study period. No changes were observed in the investigated ophthalmologic tests, urinalysis, hematology, and blood chemistry over the study period. In addition, histopathological examination indicated no adverse effects as a result of the treatment regime. Statistically significant differences in some urinary parameters reported in animals receiving 2.5 or 5% sodium gluconate were comparable to those observed in the NaCl control group and were interpreted as related to the high sodium concentration of the diet.

The authors concluded that the no observed adverse effect level (NOAEL) was 5% (equal to 4100 mg/kg bw per day). However, the Joint Food and Agriculture Organization (FAO)/World Health Organization (WHO) Expert Committee on Food Additives (JECFA) who evaluated this report has concluded that the study was not suitable for identifying a NOAEL because of the small group sizes and the positive findings in the qualitative analysis, even if they have acknowledged that the effects shown in the qualitative urine analyses were related to the high sodium intake (Mochizuki, M. Bozo Research Center, 1997, cited in OECD SIDS, 2004). Nonetheless, this study demonstrates the lack of effects of the gluconate anion even in large doses as the urinary effects were attributed to the high sodium intake and was therefore considered as critical for this endpoint.

None of the repeated dose toxicity studies of any duration (4 weeks, 6 months, or 24 months) showed any significant toxicological effects of gluconates. Potential side effects were attributed to high doses of cation intake, evidenced by results from assays designed for the gluconate anion effect specifically. The NOAEL of sodium gluconate determined from the 28 days studies on rats was equal to 1,000 mg/kg bw for males and 2,000 mg/kg bw for females. On the basis of these data and considering that gluconates are used as food additives permitted in the EU following the Quantum Satis principle (no maximum level specified), further chronic toxicity tests are considered unnecessary (SIDS OECD, 2004).

Inhalation

No adequate or reliable studies are available

Dermal

No adequate or reliable studies are available

G. Genotoxicity

Two *in vitro* studies on bacteria indicated negative results for gene mutation by sodium gluconate with and without metabolic activation (OECD, 2004). The bone marrow of mice exposed orally to sodium gluconate as either a single dose or repeated dose over four consecutive days was examined for evidence of chromosomal aberrations (OECD, 2004). The results from both the single and repeated dose exposures indicated sodium gluconate did not induce chromosomal aberrations and was considered non-genotoxic. These negative results provide sufficient information to indicate low concern for genotoxicity by sodium gluconate.

H. Carcinogenicity

Oral

No studies are available

Inhalation

No studies are available

I. Reproductive Toxicity

No studies are available

J. Developmental Toxicity

No studies are available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for sodium gluconate follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Toxicological reference values were not derived. Sodium gluconate dissociates in water to sodium and gluconate ions.

The Australian drinking water guideline value for sodium is 180 mg/L based on aesthetics (ADWG, 2011).

B. Cancer

There are no carcinogenicity studies on sodium gluconate. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Sodium gluconate does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Sodium gluconate has low toxicity to aquatic and terrestrial organisms.

B. Aquatic Toxicity

Acute Studies

Table 3 presents the results of acute aquatic toxicity studies conducted on sodium gluconate.

Table 3: Acute Aquatic Toxicity Studies on Sodium Gluconate

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
*Oryzias latipes (*Fish, freshwater)	96-hr LC_{50}	>100	-	OECD, 2004
Daphnids magna (Crustacea)	48-hr EC_{50}	>1000	-	OECD, 2004
Selenastrum capricornutum (Algae)	72-hr E_rC_{50}	>1000	-	OECD, 2004

Chronic Studies

Table 4 presents the results of chronic aquatic toxicity studies conducted on sodium gluconate.

Table 4: Chronic Aquatic Toxicity Studies on Sodium Gluconate

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Selenastrum capricornutum (Algae)	72-hr NOEC	560	-	OECD, 2004

C. Terrestrial Toxicity

No terrestrial toxicity data for gluconates are available. However, the demonstrated biodegradability and the low intrinsic toxicity of gluconates that was observed for aquatic organisms, data on animal toxicokinetic and metabolism (cfr. human toxicology) and their role in mammalian carbohydrate metabolism may also predict a low effect on terrestrial organisms. Therefore, no terrestrial toxicity studies would be required (OECD, 2004).

D. Calculation of PNEC

The PNEC calculations for sodium gluconate follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute $E(L)C_{50}$ values are available for fish (100 mg/L), invertebrates (1,000 mg/L) and for algae (1,000 mg/L). Chronic NOECs are available for algae (560 mg/L). On the basis that the data consists of results from short-term studies from three trophic levels ang long-term studies from one trophic level, an assessment factor of 100 has been applied to the chronic NOEC value of 560 mg/L for algae. The $PNEC_{water}$ is 5.6 mg/L.

PNEC Sediment

No reliable experimental toxicity data on sediment organisms are available. Sodium gluconate dissociates in water with its environmental distribution is dominated by its high water solubility. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as sodium gluconate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{sediment}$. Based on its properties, no adsorption of sodium gluconate to sediment is to be expected, and the assessment of this compartment will be covered by the aquatic assessment.

PNEC Soil

No reliable experimental toxicity data on terrestrial organisms are available. The environmental distribution of sodium gluconate is dominated by its water solubility. Sorption of sodium gluconate should probably be regarded as a reversible situation (i.e., the substance is not tightly nor permanently bound). K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as sodium gluconate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{soil}$. Based on its properties, sodium gluconate is not expected to significantly adsorb to soil, and the assessment of this compartment will be covered by the aquatic assessment.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Sodium gluconate is readily biodegradable; thus, it does not meet the screening criteria for persistence.

The estimated log K_{ow} for sodium gluconate is -5.99. Thus, sodium gluconate does not meet the criteria for bioaccumulation.

The chronic toxicity data on sodium gluconate has NOEC values > 0.1 mg/L.The acute $E(L)C_{50}$ values are >1 mg/L. Thus, sodium gluconate does not meet the screening criteria for toxicity.

Therefore, sodium gluconate is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Not classified.

B. Labelling

No signal words.

C. Pictogram

None

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Immediately flush eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. If symptoms persist, seek medical attention.

Skin Contact

Wash with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention if respiratory irritation develops or if breathing becomes difficult.

Ingestion

Do not induce vomiting. Rinse mouth with water and then drink a small amount of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: sodium oxides.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Avoid creating and breathing dust.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

Scoop and remove.

D. Storage And Handling

General Handling

Avoid creating or inhaling dust.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational standard for sodium gluconate.

Engineering Controls

Use in a well-ventilated area.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye Protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Eyewash fountains and safety showers must be easily accessible.

F. Transport Information

Sodium gluconate is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods Code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

ADWG. (2011). National Water Quality Management Strategy. Australian Drinking Water Guidelines, Section 6, Australian Government, National Health and Medical Research Council, Natural Resource Management Ministerial Council. Updated January 2022. Available: https://www.nhmrc.gov.au/about-us/publications/australian-drinking-water-guidelines

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United States Environmental Protection Agency (USEPA). 2018. EPISuite™ v. 4.11, United States Environmental Protection Agency, Office of Pollution Prevention and Toxics and Syracuse Research Corporation. Available at: https://www.epa.gov/tsca-screening-tools/epi-suitetm-estimation-program-interface.

SODIUM POLYACRYLATE (CAS NO. 9003-04-7)
2-PROPENOIC ACID, HOMOPOLYMER, AMMONIUM SALT (CAS NO. 9003-03-6)

This group contains a sodium salt and ammonium salt of polyacrylic acid homopolymers. They are expected to have similar environmental concerns and have consequently been assessed as a group. Information provided in this dossier is based on sodium polyacrylate (CAS No. 9003-04-7).

This dossier on sodium polyacrylate and similar polymers presents the most critical studies pertinent to the risk assessment of these polymers in their use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed sodium polyacrylate in an IMAP Tier 1 assessment and considers it a polymer of low concern[1] .

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): 1-Propenoic acid, homopolymer, sodium salt

CAS RN: 9003-04-7

Molecular formula: $(C_3H_4O_2)$ x-.x-Na

Molecular weight: 94.0447 g/mol (monomer); Variable (polymer)

Synonyms: 2-Propenoic acid, homopolymer, sodium salt; polyacrylic acid, sodium salt, sodium polyacrylate; acrylic acid, polymers, sodium salt; poly (acrylic acid), sodium salt; polyacrylate sodium salt

SMILES: Not available

Chemical Name (IUPAC): 2-Propenoic acid, homopolymer, ammonium salt

CAS RN: 9003-03-6

Molecular formula: $(C_3\text{-}H_4\text{-}O_2)$x-.x-$H_3$-N

Molecular weight: 89.0933 g/mol (monomer); Variable (polymer)

Synonyms: 2-Propenoic acid, homopolymer, ammonium salt;2-Propenoic acid, homopolymer, sodium salt; ammonium polyacrylate; poly(acrylic acid),ammonium salt; ammonium acrylate

SMILES: Not available; C=CC(=O)[O-].[Na]

[1] https://www.nicnas.gov.au/chemical-information/imap-assessments/how-chemicals-are-assessed/Low-concern-polymers.

II. PHYSICO-CHEMICAL PROPERTIES

Sodium polyacrylates are polymers that range in molecular weight (MW) from 1,000 to 78,000 g/mol (HERA, 2014). The sodium polyacrylates mostly used in detergents have a typical molecular weight of approximately 4,500 g/mol (HERA, 2014). For sodium polyacrylate (MW 4,500), the melting point is >150oC, where it decomposes; and the water solubility is >400 g/L (HERA, 2014).

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Sodium polyacrylates are not readily biodegradable. Due to their high molecular weights, sodium polyacrylates are not expected to bioaccumulate. In addition, these water-soluble polymers can form insoluble calcium salts in natural waters, suggesting that bioaccumulation is unlikely.

B. Partitioning

Abiotic degradation mechanisms like photolytic and hydrolytic processes do not significantly influence the environmental fate of sodium polyacrylates (HERA, 2014).

C. Biodegradation

Sodium polyacrylates are not readily biodegradable but are partly accessible to ultimate biodegradation particularly under long incubation conditions. Sodium polyacrylates with MW of <2,000 g/mol are partly biodegradable under the conditions of soil and sediment inoculation. Test results with activated sludge inoculum indicate different elimination degrees, apparently due to adsorption and precipitation processes. The removal degrees of different sodium polyacrylates show no clear relationship between elimination extent and molecular weight (HERA, 2014).

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

D. Environmental Distribution

Adsorption onto solids and precipitation are the principal mechanisms of abiotic elimination for this type of polymer, the degree of elimination differs and is strongly influenced by test concentration and water hardness (HERA, 2014).

E. Bioaccumulation

No experimental studies are available on sodium polyacrylates. Estimated bioconcentration factors based on octanol-water coefficients are not appropriate since the molecular weights of these polymers are higher than the molecular weight range for the QSAR models. Due to their high molecular weights, sodium polyacrylates are not expected to bioaccumulate. In addition, these water-soluble polymers can form insoluble calcium salts in natural waters, suggesting that bioaccumulation is unlikely (HERA, 2014).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

The acute toxicity of sodium polyacrylates are very low by the oral and dermal routes. These polymers are not irritating to the skin and eyes; nor are they skin sensitisers. No systemic toxicity was observed in rats given high oral doses of a sodium polyacrylate for four weeks; pulmonary irritation was seen in rats that inhaled an aerosol or dust of a sodium polyacrylate for 13 weeks, but there was no systemic toxicity. No developmental toxicity was seen in rats when given high oral doses of sodium polyacrylates. Sodium polyacrylates are not genotoxic or mutagenic.

B. Acute Toxicity

Oral

Acute oral toxicity studies have been conducted in rats on sodium polyacrylates with molecular weights (MW) of 1,000 to 78,000. The oral LD50 values are >5,000 or >10,000 mg/kg (the highest doses tested), except for one study on a 3,500 MW sodium polyacrylate, which was reported to be >1,000 mg/kg (the attainable limit dose of a 10% aqueous solution) (HERA, 2014). [Kl. scores = 2].

Inhalation

There are no acute inhalation studies available.

Dermal

The dermal LD50 values in rabbits for sodium polyacrylates with MW of 1,000 or 4,500 are >5,000 mg/kg (HERA, 2014). [Kl. scores = 2].

C. Irritation

According to (HERA, 2014) sodium polyacrylates with MW of 1,000 to 78,000 are not irritating to the skin or eyes [Kl. scores = 2]. However, as per ECHA current classification, the substance 2-Propenoic acid, homopolymer, sodium is considered a skin and eye irritant. Thus, this classification will be retained for purposes of this dossier.

D. Sensitisation

Sodium polyacrylates with MW of 4,500 or 78,000 were not dermal sensitisers in the guinea pig maximisation test (HERA, 2014). [Kl. scores = 2 and 4, respectively].

E. Repeated Dose Toxicity

Oral

Male rats were fed diets containing 0 or 2.5% sodium polyacrylate (MW 2,500) for four weeks. Body weight, body weight gain, and appearance of the animals were similar between treated and control animals. In the fourth week of the study, a small, but significant, decrease in total weight of bone minerals was detected and confirmed by radiographic and histological examination. There was a significant reduction in the concentration of magnesium in the bones and plasma of the treated animals. Calcium loss was slight and not statistically significant. Urinary excretion of sodium and phosphorus was markedly increased, calcium only slightly increased. The authors of the study

interpreted the finding as a metabolic imbalance rather than systemic toxicity. Sodium excretion could have been increased by the high intake of the sodium-neutralised test substance. The NOAEL for the study was considered to be 2.5% sodium polyacrylate in the diet, which was estimated to be 1,136 mg/kg-day (HERA, 2014). [Kl. score = 2].

Inhalation

Male and female rats were exposed by inhalation to 0, 0.2, 1.0, or 5.0 mg/m3 sodium polyacrylate (MW 4,500) as an aerosol for 6 hours/day, 5 days/week for 13 weeks. Additional groups of animals were exposed for 13 weeks followed by a 91-day recovery period. There were no treatment-related effects on body weights, organ weights, feed and water consumption, clinical observations, and blood chemistry. In the histopathologic examination, the lungs of the mid- and high-dose animals showed signs of mild pulmonary irritation increases in polymorphonuclear granulocytes or alveolar macrophages, pneumocyte hyperplasia, alveolar wall thickening and focal alveolitis. The lung effects were reversible and were not seen in the recovery group animals. The NOEC for systemic effects in this study was considered to be 5 mg/m3, and the NOEC for localised irritation is 0.2 mg/m3 (HERA, 2014). [Kl. score = 2].

Dermal

There are no studies available.

F. Genotoxicity

In vitro Studies

The results of the *in vitro* studies on sodium polyacrylates are presented below in Table 1. All the studies show that sodium polyacrylates are not mutagenic or genotoxic.

The *in vitro* genotoxicity studies on sodium polyacrylates are presented in Table 1.

Table 1: *In vitro* Genotoxicity Studies on Sodium Polyacrylates (HERA, 2014)

Mean MW	Test System	Results*	Klimisch Score	Reference
2,000	Bacterial reverse mutation	-	2	HERA (2014)
2,000	Mouse lymphoma	-	2	HERA (2014)
2,000	Unscheduled DNA synthesis	-	2	HERA (2014)
4,500	Bacterial reverse mutation	-	2	HERA (2014)
4,500	Mouse lymphoma	-	2	HERA (2014)
4,500	Unscheduled DNA synthesis	-	2	HERA (2014)
4,500	Cytogenetic (CHO cells)	-	2	HERA (2014)
4,500	Bacterial reverse mutation	-	2	HERA (2014)
4,500	Mammalian cell gene mutation	-	2	HERA (2014)
4,500	Unscheduled DNA synthesis	-	2	HERA (2014)

*+, positive; -, negative

In vivo Studies

There was no increase in micronuclei in polychromatic erythrocytes from the bone marrow of mice given a single oral gavage dose of 13,850 mg/kg sodium polyacrylate with a MW of 2,000 (HERA, 2014).

G. Carcinogenicity

Oral

There are no studies available.

Inhalation

There are no studies available.

Dermal

There are no studies available.

H. Reproductive Toxicity

There are no studies available.

I. Developmental Toxicity

Oral

Pregnant female rats were dosed by oral gavage with 0, 500, 1,000, or 3,000 mg/kg sodium polyacrylate (MW 4,500) on GD 6 to 15. At 3,000 mg/kg, the dams had soft or liquid stools during the treatment period. There was no maternal or developmental toxicity observed in this study. The NOAEL for maternal and developmental toxicity is 3,000 mg/kg-day (HERA, 2014). [Kl. score = 2]

Pregnant female rats were dosed by oral gavage with 0, 125, 375, or 1,125 mg/kg sodium polyacrylate (MW 90,000 as a 77.5% aq. solution) during GD 6 to 13. Some of the dams were sacrificed on GD 13 and the remaining on GD 19. One mid-dose dam and 6 high-dose dams died during the study; of these, three of the high-dose deaths were treatment-related and the remaining were considered the result of gavage errors. There was a transient decrease in feed consumption in the high-dose dams during GD 7-9, but not other indications of maternal toxicity. There was no developmental toxicity. The NOAELs for maternal and developmental toxicity are 375 and 1,125 mg/kg-day (HERA, 2014). [Kl. score = 2]

Inhalation

There are no studies available.

Dermal

There are no studies available.

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for sodium polyacrylate follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

A 4-week dietary study showed no systemic toxicity in rats given 2.5% sodium polyacrylate (MW 2,500) in their feed. The estimated dose is 1,136 mg/kg-day. Two pre-natal developmental toxicity studies showed no effects at the highest dose tested: 3,000 and 1,125 mg/kg-day for sodium polyacrylates with MW of 4,500 and 90,000, respectively. The NOAEL of 1,136 mg/kg-day from the 4-week dietary study will be used for determining the oral Reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:

UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 10
UF_D (database uncertainty) = 1
Oral RfD = 1,136/ (1 x 10 x 1 x 1 x 1) = 1,136/1,000 = 1.1 mg/kg/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,

Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

Where:

Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)
Drinking water guidance value = (1.1 x 70 x 0.1)/2 = 3.85 mg/L

B. Cancer

No carcinogenicity studies have been conducted on sodium polyacrylates. Therefore, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Sodium polyacrylates does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Sodium polyacrylates are a low toxicity concern for aquatic organisms, terrestrial invertebrates, and plants.

B. Aquatic Toxicity

Acute Studies

Table 2 lists the results of acute aquatic toxicity studies conducted on sodium polyacrylates.

Table 2: Acute Aquatic Toxicity Studies on Sodium Polyacrylates

Mean MW	Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
1,000	*Brachydanio rerio*	96-hour LC_{50}	>200	1	HERA, 2014
1,000	*Salmo gairdneri*	96-hour LC_{50}	>1,000	1	HERA, 2014
1,200	*Leuciscus idus*	96-hour LC_{50}	>500	1	HERA, 2014
2,000	*Brachydanio rerio*	96-hour LC_{50}	>200	1	HERA, 2014
2,500	*Leuciscus idus*	96-hour LC_{50}	>500	1	HERA, 2014
4,500	*Lepomis macrochirus*	96-hour LC_{50}	>1,000	1	HERA, 2014
4,500	*Lepomis macrochirus*	96-hour LC_{50}	>1,000	1	HERA, 2014
8,000	*Leuciscus idus*	96-hour LC_{50}	>500	1	HERA, 2014
10,000	*Lepomis macrochirus*	96-hour LC_{50}	>1,000	1	HERA, 2014
15,000	*Leuciscus idus*	96-hour LC_{50}	>10,000	1	HERA, 2014
78,000	*Brachydanio rerio*	96-hour LC_{50}	>400	2	HERA, 2014
1,000	*Daphnia magna*	48-hour EC_{50}	>200	1	HERA, 2014
1,000	*Daphnia magna*	48-hour EC_{50}	>1,000	1	HERA, 2014
2,000	*Daphnia magna*	48-hour EC_{50}	>200	1	HERA, 2014
4,500	*Daphnia magna*	48-hour EC_{50}	>200	1	HERA, 2014
4,500	*Daphnia magna*	48-hour EC_{50}	>1,000	1	HERA, 2014
78,000	*Daphnia magna*	24-hour EC_{50}	276	2	HERA, 2014

Mean MW	Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
8,000	*Selenastrum capricornutum*	72-hour EC_{50}	40	1	HERA, 2014
78,000	*Scenedesmus subspicatus*	96-hour EC_{50}	44	2	HERA, 2014

Chronic Studies

Table 3 lists the results of chronic aquatic toxicity studies conducted on sodium polyacrylates.

Table 3: Chronic Aquatic Toxicity Studies on Sodium Polyacrylates (HERA, 2014)

Mean MW	Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
4,500	*Pimephales promelas*	32-day NOEC	56	2	HERA, 2014
4,500	*Brachydanio rerio*	28-day NOEC	>450	1	HERA, 2014
78,000	*Brachydanio rerio*	14-day NOEC	>400	2	HERA, 2014
4,500	*Daphnia magna*	21-day NOEC	450	1	HERA, 2014
4,500	*Daphnia magna*	21-day NOEC	58	1	HERA, 2014
4,500	*Daphnia magna*	21-day NOEC	12	2	HERA, 2014
78,000	*Daphnia magna*	21-day NOEC	100	2	HERA, 2014
4,500	*Scenedesmus subspicatus*	96-hour NOEC	180	2	HERA, 2014
78,000	*Scenedesmus subspicatus*	96-hour NOEC	32.8	2	HERA, 2014

There is considerable variability in the chronic aquatic toxicity results for Daphnia magna for sodium polyacrylates with the same molecular weight of 4,500. This was discussed in HERA (2014) and was explained by the solubility of sodium polyacrylates in water. In distilled water, the solubility of sodium polyacrylates with the molecular weight of 4,500 is >400 mg/L; however, under test conditions water solubility will decrease due to the presence of Ca++ and Mg++ (as measured by water hardness). In a study by BASF (reviewed in HERA, 2014), the water solubility of sodium polyacrylate (MW 4,500) was determined with radiolabelled compounds in a test system with a calcium concentration of 70 mg/L, which corresponds to the mean water hardness to the media used in an OECD TG 202 test. Under these conditions, the water solubility of sodium polyacrylate was 1.3 mg/L after 24 hours. So, one explanation for the variability of the chronic Daphnia studies may be due to differences in water hardness.

C. Toxicity to Sediment Organisms

The 96-hour EC0 to Chironomus riparius (larvae) is >4,500 mg/kg sediment dry weight (HERA, 2014).

D. Terrestrial Toxicity

Table 4 lists the results of terrestrial toxicity studies on sodium polyacrylates polymers.

Table 4: Terrestrial Toxicity Studies on Sodium Polyacrylates (HERA, 2014)

Mean MW	Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
4,500	*Eisenia foetida foetida*	14-day EC_0	1,000	1	HERA, 2014
78,000	*Eisenia foetida andrei*	14-day EC_0	1,000	2	HERA, 2014
78,000	*Brassica rapa*	21-day NOEC	1,000	2	HERA, 2014
4,500	Nitrogen transformation*	28-day EC_{10}	>2,500	1	HERA, 2014
4,500	Carbon transformation*	28-day EC_{10}	>2,500	1	HERA, 2014

*Soil organisms

E. Calculation of PNEC

The PNEC calculations for sodium polyacrylate follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E(L)C_{50} values are available for fish (>200mg/L), *Daphnia* (>200 mg/L), and algae (40 mg/L). NOEC values from long-term studies are available for fish (56 mg/L), invertebrates (12 mg/L) and algae (32.8 mg/L). On the basis that the data consists of short-term and long-term results from three trophic levels, an assessment factor of 10 has been applied to the lowest reported NOEC value of 12 mg/L for invertebrates. The E(L)C_{50} value is used because the value for fish is lower than the NOEC values for all three trophic levels. The $PNEC_{water}$ is 1.2 mg/L.

PNEC Sediment

Experimental results are available for one trophic level. There were no visual signs of toxicity to Chironomus riparius (larvae) at the highest concentration tested (>4,500 mg/kg sediment dry weight) (HERA) 2014). The EC_0 is considered to be above 4,500 mg/kg and an assessment factor cannot apply. Thus, the equilibrium partitioning method will be used to determine the $PNEC_{sed}$. The HERA (2014) risk assessment calculated a $PNEC_{sed}$ of 130 mg/kg sediment wet weight using the default of 0.05 as the weight fraction of organic carbon in sediment according to the EU Technical Guidance Document (TGD) (EU 2003).

PNEC Soil

Experimental results are available for three trophic levels. An acute LC_{50} value is available for earthworms (1,000 mg/kg soil dry weight). A 21-day NOEC for Brassica rapa was reported to be 1,000 mg/kg soil dry weight. Results from two long-term studies are available for soil microorganisms, with the NOECs for nitrogen and carbon transformation being >2,500 mg/kg soil dry weight. On the basis that the data consists of short-term tests, as well as one long-term test from

one trophic level, an assessment factor of 100 has been applied to the lowest reported long-term NOEC of >2,500 mg/kg soil dry weight. The $PNEC_{soil}$ is 25 mg/kg soil dry weight.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2017).

Sodium polyacrylates are not readily biodegradable, thus does not meet the screening criteria for persistence.

The sodium polyacrylates are expected to have high molecular weights and are not expected to be bioavailable. Thus, these polymers do not meet the criteria for bioaccumulation.

Chronic NOECs for fish, daphnia and algae are available for sodium polyacrylates, and the NOEC values are >0.1 mg/L. Thus, sodium polyacrylates do not meet the screening criteria for toxicity.

The overall conclusion is that sodium polyacrylates are not PBT substances.

IX. CLASSIFICATION AND LABELLING

A. Classification

Aquatic Acute Toxicity Category 3

B. Labelling

Warning

According to the classification provided by companies to ECHA in CLP notifications this substance causes serious eye irritation and causes skin irritation.

A. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Please refer to the product SDS for additional information and confirmation of the information provided herein.

<u>Eye Contact</u>

In case of contact, immediately flush eyes with plenty of water for at least 15 minutes.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention.

Ingestion

Rinse mouth with water and then drink a glass of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

Keep product and empty container away from heat and sources of ignition. May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Handle in accordance with good industrial hygiene and safety practice.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

For large amounts: dike spillage and pump off product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Keep product and empty container away from heat and sources of ignition. Ensure adequate ventilation, especially in confined areas.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

The are no workplace exposure standards for sodium polyacrylates in Australia.

Engineering Controls

Good general ventilation should be used.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Wearing of closed work clothing is recommended. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Sodium polyacrylate is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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SODIUM SULPHATE

This dossier on sodium sulphate presents the most critical studies pertinent to the risk assessment of sulphate in its use in coal seam gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained the OECD-SIDS documents on sodium sulphate (OECD, 2005a,b), and from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed sodium sulphate in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment.

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Disodium sulphate

CAS RN: 7757-82-6

Molecular formula: Na_2SO_4

Molecular weight: 142.04 g/mol

Synonyms: Sodium sulphate; disodium sulphate; sodium bisulphate; sulphuric acid, disodium salt

SMILES: [O-]S(=O)(=O)[O-].[Na+].[Na+]

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-chemical Properties of Sodium Sulphate

Property	Value	Klimisch score	Reference
Physical state at 20°C and 101.3 kPa	White crystalline solid	2	ECHA
Melting Point	ca. 884°C (pressure not reported)	2	ECHA
Density	2700 kg/m^3 @ 20°C	2	ECHA
Partition Coefficient (Log K_{ow})	-4.38 (temperature not provided)	2	ECHA
Water Solubility	445.5 g/L @ 20°C	1	ECHA
Auto flammability	Not auto flammable	1	ECHA

III. ENVIRONMENTAL FATE SUMMARY

Sodium sulphate dissociates in aqueous media to sodium (Na^+) and sulphate (SO_4^{2-}) ions. Biodegradation is not applicable to inorganic compounds. Sodium sulphate is not expected to bioaccumulate; it will dissociate to ions that are ubiquitous in the environment. Sodium sulphate is not expected to adsorb to soil or sediment because of its dissociation properties and high water solubility.

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Sodium sulphate exhibits low acute toxicity by the oral and inhalation routes. It is not irritating to the skin and eyes; and it is not a skin sensitiser. In a reproductive and developmental toxicity screening study, there was no indication of any toxicity in rats given oral doses as high as 1,000 mg/kg/day. Sodium sulphate is not genotoxic.

B. Acute Toxicity

Oral

The oral LD_{50} in rats is > 2,000 mg/kg (ECHA) [KI score = 1].

Human data indicate a very low acute toxicity of sodium sulphate. High oral doses of sodium sulphate, from 300 mg/kg up to 20 grams for an adult, are well tolerated, except from (intentionally) causing severe diarrhea (OECD, 2005a,b).

Inhalation

The 4-hour inhalation LC_{50} for an aerosol of sodium sulphate is > 2.4 mg/L, which was the highest technically feasible aerosol concentration. The mass median aerodynamic diameters (MMAD) were 2.65 to 2.71 µm (ECHA) [KI score = 1].

Dermal

There is no data on acute dermal toxicity.

C. Irritation

Application of 0.5 g sodium sulphate (in PEG 400) to the skin of rabbits for 4 hours was not irritating (ECHA) [KI score = 1].

Instillation of 90 mg sodium sulphate to the eyes of rabbits was not irritating (ECHA) [KI score = 1].

D. Sensitisation

Sodium sulphate was not considered a skin sensitiser in a mouse local lymph node assay (ECHA) [KI score = 1].

E. Repeated Dose Toxicity

Oral

In a reproductive and developmental toxicity screening (OECD 421) study, male and female Wistar rats were dosed by oral gavage with 0, 100, 300 or 1,000 mg/kg sodium sulphate for a total of 4 weeks for males and 7 weeks for females. There was no evidence of toxicity at any dose level. The NOAEL for systemic toxicity is 1,000 mg/kg/day, the highest dose tested.

Inhalation

No studies are available.

Dermal

No studies are available.

F. Genotoxicity

In Vitro Studies

The *in vitro* genotoxicity studies on sodium sulphate are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Sodium Sulphate

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (S. typhimurium and E. coli strains)	-	-	1	ECHA
Mammalian cell gene mutation (mouse lymphoma L5178Y cells)	-	-	1	ECHA
Chromosomal aberration (Chinese hamster lung fibroblasts)	-	-	1	ECHA

*+, positive; -, negative

In Vivo Studies

No studies are available.

G. Carcinogenicity

No valid studies are available.

H. Reproductive/Developmental Toxicity

A reproductive and developmental toxicity screening (OECD 421) study has been conducted on sodium sulphate. Male and female Wistar rats were dosed by oral gavage with 0, 100, 300 or 1,000 mg/kg sodium sulphate. There were no deaths during the study and no clinical signs of reproductive or developmental toxicity at any dose level. Body weights, body weight gain and feed consumption were similar across all groups. The NOAEL for systemic, reproductive and developmental toxicity is 1,000 mg/kg/day, the highest dose tested (ECHA) [KI score = 1].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

Toxicological reference values were not derived. Sodium sulphate dissociates in water to sodium and sulphate ions.

The Australian drinking water guideline value for sodium is 180 mg/L based on aesthetics (ADWG, 2021).

The Australian drinking water guideline value for sulphate is 500 mg/L based on health. Concentrations of > 500 mg/L can have purgative effects. There is also an Australian drinking water guideline value for sulphate of 250 mg/L based on aesthetics; it is the taste threshold (ADWG, 2021).

A. Cancer

There are no valid carcinogenicity studies on sodium sulphate. Thus, a cancer reference value was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Sodium sulphate does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Sodium sulphate is of low acute concern to aquatic life.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on sodium sulphate.

Table 3: Acute Aquatic Toxicity Studies on Sodium Sulphate

Test Species	Endpoint	Results (mg/L)	Klimisch score	Reference
Pimephales promelas	96-hour LC_{50}	7,960	2	Mount et al. (1997)
Daphnia magna	48-hour EC_{50}	4,736*	2	Davies and Hall (2007)

* Standard test conditions: 100 mg $CaCO_3$/L and Ca:Mg ratio of 0.7.

Chronic Studies

The 7-day LOEC from a *Ceriodapnia dubia* reproduction study, in which the test media contained varying degrees of water hardness, was 1,329 mg/L. The NOEC was extrapolated to be approximately 1,109 mg/L (Soucek, 2007).

C. Sediment Toxicity

The lowest 96-hour LC_{50} value to *Hyalella azteca* in a series of studies involving different hardnesses of water was 757 mg/L (Soucek and Kennedy, 2005). In another study with *Hyalella azteca*, the lowest 96-hour LC_{50} value (in water with the lowest hardness) was 841 mg/L (Davies and Hall, 2007). The lowest 96-hour LC_{50} value to *Chironomus tentans* in a series of studies involving different hardnesses of water was 20,899 mg/L (Soucek and Kennedy, 2005).

D. Terrestrial Toxicity

No adequate studies were located.

E. Calculation of PNEC

The PNEC calculations for sodium sulphate follow the methodology discussed in DEWHA (2009).

PNEC water

Experimental results are available for two trophic levels. Acute E(L)C50 values are available for fish (7,960 mg/L) and *Daphnia* (4,736 mg/L). The NOEC from a chronic study on invertebrates was 1,109 mg/L. On the basis that the data consists of results from short-term studies from two trophic levels and a single long-term study, an assessment factor of 100 has been applied to the chronic NOEC value of 1,109 mg/L for invertebrates. The $PNEC_{water}$ is 11 mg/L.

PNEC sediment

No reliable experimental toxicity data on sediment organisms are available. Sodium sulphate dissociates completely in water with its environmental distribution is dominated by its high water solubility. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as sodium sulphate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{sediment}$. Based on its properties, no adsorption of sodium sulphate to sediment is to be expected, and the assessment of this compartment will be covered by the aquatic assessment.

PNEC soil

No reliable experimental toxicity data on terrestrial organisms are available. The environmental distribution of sodium sulphate is dominated by its water solubility. Sorption of sodium sulphate should probably be regarded as a reversible situation, i.e., the substance is not tightly nor permanently bound. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as sodium sulphate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{soil}$. Based on its properties, sodium sulphate is not expected to significantly adsorb to soil, and the assessment of this compartment will be covered by the aquatic assessment.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU REACH Criteria methodology (DEWHA, 2009; ECHA, 2008).

Sodium sulphate is an inorganic salt that dissociates completely to sodium and sulphate ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions; both sodium and sulphate ions are also ubiquitous and are present in most water, soil and sediment. For the purposes of this PBT assessment, the persistent criteria are not considered applicable to sodium sulphate or its dissociated ions.

Sodium and sulphate ions are essential to all living organisms and their intracellular and extracellular concentrations are actively regulated. Thus, sodium sulphate is not expected to bioaccumulate.

The NOEC from a chronic toxicity study with *Ceriodaphnoa rerio* is > 0.1 mg/L. The acute $E(L)C_{50}$ values for fish and *Daphnia* are > 1 mg/L. Thus, sodium sulphate does not meet the criteria for toxicity.

Therefore, sodium sulphate is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

Not classified.

B. Labelling

No signal words.

C. Pictogram

None

X. SAFETY AND HANDLING

A. First Aid

Eye Contact

Immediately flush eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. If symptoms persist, seek medical attention.

Skin Contact

Wash with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention if respiratory irritation develops or if breathing becomes difficult.

Ingestion

Do not induce vomiting. Rinse mouth with water and then drink a small amount of water. Get medical attention. Never give anything by mouth to an unconscious person.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: sodium and sulfur oxides.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment. Avoid creating and breathing dust.

Environmental Precautions

Prevent from entering sewers, waterways or low areas.

Steps to be Taken if Material is Released or Spilled

Scoop and remove.

D. Storage And Handling

General Handling

Avoid creating or inhaling dust.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational standard for sodium sulphate.

Engineering Controls

Use in a well-ventilated area.

Personal Protection Equipment

Respiratory Protection: Respiratory protection is not required.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye Protection: Safety glasses with side-shields.

Other Precautions: Handle in accordance with good industrial hygiene and safety practice. Eyewash fountains and safety showers must be easily accessible.

F. Transport Information

Sodium sulphate is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods Code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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SODIUM SULPHITE

This dossier on sodium sulphite presents the most critical studies pertinent to the risk assessment of sodium sulphite in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The information presented in this dossier was obtained primarily from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed sodium sulfite in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment[1]

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Disodium sulphate

CAS RN: 7757-83-7

Molecular formula: Na_2SO_3

Molecular weight: 126.04 g/mol

Synonyms: Sodium sulphite, disodium sulphite, sodium bisulphite anhydrous, sodium sulfite

SMILES: [O-]S(=O)[O-].[Na+].[Na+]

II. PHYSICO-CHEMICAL PROPERTIES

Table 1: Overview of the Physico-Chemical Properties of Sodium Sulphite

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	White, hexagonal, crystalline solid	2	ECHA
Melting Point	911°C (pressure not provided)	2	ECHA
Boiling Point	No data	-	-
Density	2630 kg/m^3 @ 20°C	2	ECHA
Vapor Pressure	Not applicable	-	-
Partition Coefficient (log K_{ow})	Not applicable	-	-
Water Solubility	307 g/L @ 25°C	2	ECHA
Flash Point			
Auto flammability	Not applicable	-	-
Viscosity	Not applicable	-	-

[1] https://www.industrialchemicals.gov.au/chemical-information/search-assessments?assessmentcasnumber=7757-83-7+

Property	Value	Klimisch Score	Reference
Henry's Law Constant	Not applicable	-	-

Sodium sulphite readily dissociates in aqueous media to the sodium (Na^+) and sulphite (SO_3^{2-}) ions. At neutral pH, a mixture of 50% sulphite (SO_3^{2-}) and 50% bisulphite (HSO_3^{2-}) is present.

In surface waters, sulphite is oxidized to sulfate either catalytically by air oxygen or by microbial action. The presence of cations like iron, copper or manganese in the environment accelerates the oxidation rate significantly.

III. ENVIRONMENTAL FATE PROPERTIES

At environmental pHs, sodium sulphite dissociates in water to form sodium (Na^+) ions, sulphite (SO_2^{3-}) ions, and bisulphite ions (HSO_3^-). In acidic solutions, sulfur dioxide (SO_2) gas may be formed.

Sodium sulphite is not expected to bioaccumulate in the environment because of the resulting strong anionic nature of the substance, as well as its rapid oxidative transformation to sulphates under physiological and environmental circumstances. Due to its anionic nature, any quantitatively relevant adsorption onto soil, or sediments, or suspended matter for sodium sulfite as well as its dissociation products is not to be expected. Furthermore, sulphite will oxidize to sulfate, which is ubiquitous in the environment (ECHA).

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Sodium sulphite has low acute toxicity by the oral, inhalation and dermal routes. It is not irritating to the skin or eyes; it is not a skin sensitiser. No systemic toxicity was seen in rats when given sodium metabisulphite (which dissociates to the sulphite ion) in their diet over a lifetime. There were, however, indications of stomach lesions as a result of localized irritation from the ingestion of sodium metabisulphite. Genetic toxicity studies were negative. Lifetime oral feeding studies on sodium metabisulphite in rats and mice showed no evidence of carcinogenicity. No reproductive or developmental toxicity was observed in any of the animal studies on sodium metabisulphite.

B. Pharmacokinetics and Metabolism

Sodium sulphite is rapidly absorbed from the gastro-intestinal tract. Sulfate is the main metabolite formed by the action of sulphite oxidase in many tissues. Tissue accumulation of sulphite-derived S is highest in stomach, skin and hair, intestine, and kidney. Excretion is rapid, mainly in the urine (OECD, 2008).

C. Acute Toxicity

The oral LD_{50} of sodium sulphite in male and female Sprague-Dawley rats is 2,610 mg/kg bw (ECHA) [KI. score = 2].

The 4-hour inhalation LC_{50} in male and female Sprague-Dawley rats by nose/head-only dust/aerosol exposure to sodium sulphite is >5.5 mg/L. The mass median aerodynamic diameter (MMAD) was 3.0 μm, with 90.7% of the dust being respirable (ECHA) [Kl. score = 2].

The acute dermal LD_{50} in male and female Wistar rats exposed to disodium sulfate via semi occlusive dressing is >2,000 mg/kg bw (ECHA) [Kl. score = 1].

D. Irritation

Application of 0.5 g disodium sulfate to the skin of Vienna white rabbits for 4 hours under occlusive conditions was non-irritating. The mean erythema score was 0.5 and the mean oedema score was 0. In addition to this, oedema and erythema was not observed at the 8th day reading (ECHA) [Kl. score = 2].

Instillation of 162 mg disodium sulfate (equivalent to 0.1 mL bulk volume) into the eyes of Vienna white rabbits was not irritating. The mean of the 24, 48, and, 72-hour scores were: 0.00 for corneal lesions; 0.00 for iridial lesions; 0.9 for conjunctival redness; and 0.5 for chemosis (ECHA) [Kl. score = 2].

E. Sensitisation

Sodium sulfite was not considered to be a skin sensitiser at concentrations of 10%, 25%, and 50% w/w in a mouse local lymph node assay (ECHA) [Kl. score = 1].

F. Repeated Dose Toxicity

<u>Oral</u>

There are no studies available on sodium sulphite.

Male and female Wistar rats were given in their diet 0, 0.125, 0.25, 0.5, 1.0, or 2.0% sodium metabisulphite for up to two years and over three generations. The diet was enriched with thiamine to prevent thiamine deficiency as a result of sulphite-induced destruction of this vitamin. During storage up to the time of consumption, the losses of sulphite from the feed containing sodium metabisulphite at levels of 0.125, 0.25, 0.5, 1.0, and 2.0% averaged 22, 14,12, 8, and 4.5%, respectively, while the decrease in thiamine was 2.7, 1.7, 8.3, 14.5, and 15.4%, respectively. Addition of thiamine to the diet prevented thiamine deficiency in rats at all dose levels based on measurements of thiamine levels in the urine and liver. The general condition of the rats was good during the first 72 weeks in the F_0 generation, as well as the other two generations. After 72 weeks, there was a rapid increase in mortality in all groups. Survival in the treated groups were generally higher than the controls, except for the 2% F_1 males; no deaths occurred in the 2% F_2 females. A marginal reduction in body weight gain was observed in the 2% dose group (both sexes) in the F_1 and F_2 generations. Feed consumption was similar between treated and control groups. There were no changes in hematology and clinical chemistry parameters and urinalysis that were considered toxicologically significant. The $\geq$1% dietary groups had occult blood in their feces. Relative kidney weights were increased in the 2% F_2 females, but there were no pathological changes noted in the kidneys from this group. Hyperplastic changes in the fore- and glandular stomachs were noted in the $\geq$1% groups in all three generations. Some slight alterations were also noted in stomachs of the 0.5% F_2 rats. The NOAEL for systemic toxicity is 1.91% in the diet. This was estimated to be >955 mg/kg bw/day based on a rat body weight of 400 g and a daily feed intake of 20 g. The histopathologic

effects on the stomach and the occult blood in faeces are considered to be the result of localized irritation (a site-of-contact effect) from the ingestion of sodium metabisulphite (Til et al., 1972 as cited in ECHA). [Kl. score = 2]

Inhalation

There are no adequate studies are available.

Dermal

There are no studies available.

G. Genotoxicity

In vitro Studies

The in vitro genotoxicity studies conducted on sodium sulphite and sodium metasulphite are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Sodium Sulphite and Sodium Metabisulphite

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (*S. typhimurium* strains)	-	-	2	ECHA
Mammalian cell gene mutation (mouse lymphoma L5178Y cells)**	-	-	1	ECHA
Bacterial reverse mutation (S. typhimurium strains) ***	-	-	2	ECHA

*+, positive; -, negative
**Sodium metasulphite
***sodium disulphite

In Vivo Studies

Male Sprague-Dawley rats were fed in their diet 0, 4.5, 15, or 45 mg/kg-day sodium bisulfite Sodium bisulfite was negative in a rodent dominant lethal mutation assay. The dominant lethal test did not produce any consistent responses that would suggest that sodium bisulfite is mutagenic to Sprague-Dawley rats (ECHA) [KI. score =2].

Male NMRI mice were given a single subcutaneous dose of disodium sulfate at the following concentrations: 0, 250, 500, or 1,000 mg/kg in a chromosome aberration assay. There were no increases in chromosomal aberrations in the bone marrow cells of treated rats compared to the those in the control animals. Under the experimental conditions, disodium sulfate did not induce any chromosome-damaging (clastogenic) effects nor were there any indications of impairment of chromosome distribution during mitosis (aneugenic activity) in bone marrow cells *in vivo* (ECHA) [Kl. score = 1].

H. Carcinogenicity

Oral

There are no carcinogenicity studies available sodium sulphite.

Male and female Wistar rats were fed in their diet 0, 0.125, 0.25, 0.5, 1.0, or 2.0% sodium metabisulphite for up to two years and over three generations. There was no increased incidence of tumours in the treated groups compared to the controls and there was no evidence of carcinogenic activity (Til et al., 1972; as cited in ECHA). [Kl. score = 2]

Male and female ICR/JCL mice were given 0, 1, or 2% potassium metabisulphite continuously in their drinking water for 24 months (2 years). There were no increased incidences of tumours in the treated mice compared to controls and there was no evidence for carcinogenicity (Taneka et al., 1994; as cited in ECHA) [Kl. score = 2].

Male and female Wistar rats were continuously exposed to sodium metabisulfite in their diet/feed for 56 days (short-term study) and up to 24 months (long-term study) at the following concentrations: 0.125%, 0.25%, 0.5%, 1.0%, and 2.0%. Sodium metabisulfite induced hyperplastic changes in the forestomach at dietary levels of 0.5% and higher. The NOAEL for local toxicity was determined to be 0.25% (corrected to 0.215% based on analytical verifications). The lesions induced by sodium metabisulfite in the glandular stomach consisted of microerosions, necrosis of epithelial cells, cellular infiltrations, and atypical glandular hyperplasia. However, upon microscopic examination there was no evidence for the formation of tumour (ECHA) [KI. score = 2].

Male and female rats were exposed to 750 ppm and 275 ppm of sodium metabisulphite via their drinking water for up to 2.5 years (over 3 generations). The incidence of tumours was unaffected by the addition of sodium metabisulphite to rats drinking water and sodium metabisulphite was proven to be non-toxic (ECHA) [KI. score = 2].

Male Fischer 344/DuCrj were exposed to potassium sulfite and potassium metabisulfite via a single dose oral gavage at the following concentrations: 0.45, 0.89, 1.34 g/kg bw (potassium sulfite) and 0.5,0.8,1.1, and 1.4 g/kg bw (potassium metabisulfite). The results from this study suggest that potassium sulfite and potassium metabisulfite may have tumour promoting activities in glandular stomach carcinogenesis (ECHA) [KI. score = 2].

Male Wistar rats were exposed to 1% potassium metabisulfite via their drinking water for up to 40 weeks. The findings from this study suggest that potassium metabisulfite could be considered to exert tumour promoting activity in the rat glandular stomach (ECHA) [KI. score = 2].

Inhalation

There were no adequate studies available.

I. Reproductive Toxicity

Male and female Wistar rats were continuously fed in their diet 0, 0.125, 0.25, 0.5, 1.0, or 2.0% sodium metabisulphite for up to two years and over three generations. The diet was enriched with thiamine to prevent thiamine deficiency as a result of sulphite-induced destruction of this vitamin. During storage up to the time of consumption, the losses of sulphite from the feed containing sodium metabisulphite at levels of 0.125, 0.25, 0.5, 1.0, and 2.0% averaged 22, 14,12, 8, and 4.5%, respectively, while the decrease in thiamine was 2.7, 1.7, 8.3, 14.5, and 15.4%, respectively. Addition of thiamine to the diet prevented thiamine deficiency in rats at all dose levels based on measurements of thiamine levels in the urine and liver. The effects other than reproductive and developmental toxicity are discussed above in the Repeated Dose Toxicity section. There were no treatment-related effects on female fertility, the number of young per litter, or birth weight or mortality of the offspring. The number of F_{2a} pups was significantly reduced in the $\geq$0.5% groups during the first breeding cycle, but there was no dose-response, and the reduction did not occur during the second breeding cycle. Slight growth retardation was observed in the F_1 and F_2 generation rats both before and after weaning. The NOAEL for reproductive toxicity is 1.91% in the diet. This was estimated to be >955 mg/kg bw/day based on a rat body weight of 400 g and a daily feed intake of 20 g (Til et al., 1972; as cited in ECHA). [Kl. score = 2]

Male and female rats were given sodium metabisulphite in their drinking water for up to 2.5 years and in three successive generations. The doses were 375 and 750 ppm as sulfur dioxide (SO_2). There was no evidence of systemic toxicity in either dose group. The number of offspring of either the F_1 and F_2 generation and the proportion surviving to the end of lactation were similar between treated and control groups. The NOAEL for reproductive toxicity is 750 ppm (as SO_2) in drinking water. Assuming an average rat body weight of 400 g and a daily water intake of 28 mL, 750 ppm (as SO_2) corresponds to 53 mg/kg bw/day sodium metabisulphite (Lockett and Natoff, 1960; as cited in ECHA). [Kl. score = 2]

J. Developmental Toxicity

Pregnant female Wistar rats were fed in the diet 0, 0.32, 0.63, 1.25, 2.5, or 5% sodium sulfite heptahydrate($Na_2SO_3 \bullet 7H_2O$) during GD 8 to 20. Maternal body weight gain and feed consumption were reduced in the 5% dose group. There was some evidence of reduced body weight gain in all treated groups, but there was no dose-response relationship, and these effects were not observed in the live birth component of the study. The live birth component showed no treatment-related changes in the pups at three weeks after birth. There was no evidence of teratogenicity. The NOAELs for maternal and developmental toxicity are 2.5% and 5% in the diet, respectively. The calculated daily doses are approximately 850 and 1,450 mg/kg-day, respectively (ECHA). [Kl. score = 2]

Dutch rabbits were exposed to 1.23, 5.71, 26.5, and 123 mg/kg bw of sodium metabisulfite daily via oral gavage from gestation day 6-18 until gestation day 29. The highest tested dose of 123 mg/kg bw/day of sodium metabisulfite did not produce any clearly discernible effects on maternal or foetal survival. The number of abnormalities seen in either soft or skeletal tissues of the test groups did not differ from the number occurring spontaneously in the sham treated controls. The NOAEL for maternal and developmental toxicity is expected to be above the highest dose of 123 mg/kg bw/day sodium metabisulfite in this study (ECHA) [KI. score = 2].

Wistar rats were fed 0.1%, 1.0% and 10% potassium metabisulfite from gestation days 7-14 up to day 20 of gestation (two-thirds of animals) or until week 15 after birth (one third of animals). Exposure to 10%

potassium metabisulfite caused a slight decrease in the postnatal survival rate of the offspring (most likely due to maternal malnutrition) and a reduction in maternal body weight gain during pregnancy and food intake. There was no evidence of teratogenesis of the foetuses in this study. There were several types of skeletal variations as well as delayed ossification in some treatment groups, but these findings were not significantly different from the control group. There were no adverse effects on the pre and postnatal development of the offspring in the rats exposed to 0.1% and 1.0% potassium metabisulfite. The findings from this study helped to conclude that potassium metabisulfite does not have teratogenic effects in rats. The NOAEL for maternal and fetotoxicity in this study was established at the 1.0% dose level (1,320 mg/kg bw/day or 766 mg/kg bw/day SO2 equivalents) (ECHA) [KI. score = 2].

Wistar rats were administered 1.55, 7.19, 33.4, and 155 mg/kg bw potassium metabisulfite daily via oral gavage from day 6 to 15 of gestation until day 20 of gestation. The highest dose tested (155 mg/kg bw) did not induce any discernible effects on maternal or foetal survival. The NOAEL for maternal and developmental toxicity is expected to be above the 155 mg/kg bw/day (ECHA) [KI. score = 2].

CD-1 mice were exposed to 1.25, 5.47, 26.9, and 125 mg/kg bw potassium metabisulfite daily via oral gavage from day 6 to 15 of gestation until day 17 of gestation. The highest dose tested did not cause any discernible effects on maternal or foetal survival. The NOAEL for maternal and developmental toxicity is expected to be above 125 mg/kg bw/day (ECHA) [KI. score = 2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for sodium sulphite follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

Local effects in the stomach were the most predominant finding of repeated dose toxicity. The NOAEL for local chronic effects in the study described by Til et al. (1972) reported in ECHA is represented by the dose of 0.25% metabisulfite. The corrected dose level corresponded to a dose of 108 mg/kg bw/day $Na_2S_2O_5$. All observed effects (occurrence of occult blood in faeces and changes in gastric morphology) were detected at higher dose levels at and above 0.5% in the diet (220 mg/kg bw/day Na_2S2O_5). There was no evidence of systemic toxicity following chronic treatment with sodium metabisulfite. Therefore, the NOAEL for systemic effects can be expected above the highest dose of 2% metabisulfite in the diet corresponding to 955 mg/kg bw/day of $Na_2S_2O_5$. The NOAEL of 955 mg/kg bw/day from this study will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:
UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10

UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 1
UF_D (database uncertainty) = 1

Oral RfD = 955/(10 x 10 x 1 x 1 x 1) = 955/100 = 9.55 mg/kg bw/day

Drinking water guidance value

Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,
Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

where:
Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)

Drinking water guidance value = (9.55 x 70 x 0.1)/2 = 33.4 mg/L

Sodium sulphite readily dissociates in aqueous media to the sodium (Na^+) and sulphite (SO_3^{2-}) ions. The Australian drinking water guideline values for sodium (180 mg/L) and sulphate (250 mg/L) may also apply to sodium sulphite.

B. Cancer

No carcinogenic effects were reported for sodium metabisulphite in rat and mouse chronic studies. Thus, a cancer reference value for sodium sulphite was not derived.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Sodium sulphite does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidizing potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Sodium sulphite is low toxicity to aquatic life.

B. Aquatic Toxicity

Acute Studies

Table 3 lists the results of acute aquatic toxicity studies conducted on sodium sulphite and sodium disulphite.

Table 3: Acute Aquatic Toxicity Studies on Sodium Sulphite and Sodium Disulphite

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Leuciscus idus (Golden orfe)	96-hr LC_{50}	316	2	ECHA
Daphnia magna	48-hr EC_{50}	89* (59)	2	ECHA
Desmodesmus subspicatus	72-hr EC_{50}	43.8* (29)	2	ECHA

*Test substance: sodium disulphite

Chronic Studies

Table 4 lists the results of chronic aquatic toxicity studies conducted on sodium sulphite and sodium disulphite.

Table 4: Chronic Aquatic Toxicity Studies on Sodium Sulphite and Sodium Disulphite

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Zebrafish	34-d NOEC	>316	1	ECHA
Daphnia magna	21-d NOEC	>10* (6.6)	1	ECHA
Desmodesmus subspicatus	EC_{10}	33.3* (22)	2	ECHA

*Test substance: sodium disulphite; adjusted concentration for sodium sulphite in parentheses.

C. Terrestrial Toxicity

No data are available.

D. Calculation of PNEC

The PNEC calculations for sodium sulphite follow the methodology discussed in DEWHA (2009).

The PNEC calculations for sodium metabisulphite follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute $E(L)C_{50}$ values are available for fish (316 mg/L), *Daphnia* (59 mg/L), and algae (29 mg/L). Results from chronic studies are also available for all three trophic levels, with the lowest NOEC or EC_{10} being 6.6 mg/L for invertebrates. On the basis that the data consists of short-term and long-term results from three trophic levels, an assessment factor of 10 has been applied to the lowest reported NOEC of 6.6 mg/L for invertebrates. The $PNEC_{water}$ is 0.7 mg/L.

PNEC Sediment

No experimental toxicity data on sediment organisms are available. Sodium sulphite dissociates completely in water with its environmental distribution is dominated by its high water solubility. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as sodium sulphite. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{sed}$. Based on its properties, no adsorption of sodium sulphite to sediment is to be expected, and the assessment of this compartment will be covered by the aquatic assessment.

PNEC Soil

No experimental toxicity data on soil organisms are available. Sodium sulphite dissociates completely in water with its environmental distribution is dominated by its high-water solubility. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as sodium sulphite. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{soil}$. Based on its properties, no adsorption of sodium sulphite to soil is to be expected, and the assessment of this compartment will be covered by the aquatic assessment.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU Reach Criteria methodology (DEWHA, 2009; ECHA, 2008).

Sodium sulphite is an inorganic compound that dissociates completely to sodium ions, sulphite and bisulphite ions, and sulfur dioxide in aqueous solutions. Biodegradation is not applicable to these compounds. For the purposes of this PBT assessment, the persistent criteria are not considered applicable to sodium sulphite or its dissociated compounds.

Bioaccumulation is not to be expected because of the resulting strong anionic nature of the substance, as well as its rapid oxidative transformation to sulphates under physiological and environmental circumstances. Thus, sodium sulphite does not meet the screening criteria for bioaccumulation.

The NOEC or EC_{10} values from chronic aquatic toxicity studies on sodium sulphite is >0.1 mg/L. Thus, sodium sulphite does not meet the criteria for toxicity.

Therefore, sodium sulphite is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H302: Harmful if swallowed
H314: Causes severe skin burns and eye damage
H315: Causes skin irritation
H319: Causes serious eye irritation
Acute toxicity-category 4
Eye damage- category 1

B. Labelling

Danger

C. Pictogram

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. Get medical attention immediately, preferably a physician for an ophthalmologic examination.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air. Get medical attention if respiratory irritation develops or if breathing becomes difficult.

Ingestion

Rinse mouth with water and then drink plenty of water. Never give anything by mouth to an unconscious person. If symptoms develop, seek medical advice.

B. Fire Fighting Information

Extinguishing Media

Water spray, carbon dioxide, foam, dry chemical.

Specific Exposure Hazards

When contacted by water, sodium metabisulphite releases sulfur dioxide (SO_2), a poisonous gas. In the case of fire, the following may be liberated: Sulfur oxides and sulfur dioxide.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

Use appropriate protective equipment.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas. When contacted by water, sodium metabisulphite releases sulfur dioxide (SO_2), a poisonous gas.

Steps to be Taken if Material is Released or Spilled

Scoop up and remove.

D. Storage And Handling

General Handling

When sodium metabisulphite gets wet or moist, it liberates sulfur dioxide (SO_2), a poisonous gas. Use proper protective equipment and exposure controls to prevent exposure to this toxic gas.

Other Handling Precautions

Avoid eye and skin contact. Avoid creating or inhaling dust. Keep away from acids and oxidizing agents.

Storage

Keep container tightly closed and in a dry and well-ventilated place. Keep in a cool place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

A workplace exposure standard is not available in Australia for sodium sulphite. However, the workplace exposure standards for sodium metabisulphite (disulphite) and sodium bisulphite in Australia is 5 mg/m^3 as an 8-hr TWA.

Engineering Controls

None

Personal Protection Equipment

Respiratory Protection:
Respiratory protection is not required.

Hand Protection:
Chemical resistant protective gloves.

Skin Protection:
Body protection must be chosen depending on activity and possible exposure.

Eye protection:
Safety glasses with side-shields.

Other Precautions:
Handle in accordance with good industrial hygiene and safety practice. Eyewash fountains and safety showers must be easily accessible.

F. Transport Information

Sodium sulphite is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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SODIUM THIOSULPHATE

This dossier on sodium thiosulphate presents the most critical studies pertinent to the risk assessment of sodium thiosulphate in its use in coal seam or shale gas extraction activities. This dossier does not represent an exhaustive or critical review of all available data. The information presented in this dossier was obtained from the ECHA database that provides information on chemicals that have been registered under the EU REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

NICNAS has assessed sodium thiosulphate in an IMAP Tier 1 assessment and concluded that it poses no unreasonable risk to the environment[1].

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Disodium sulfanidesulphonate

CAS RN: 7772-98-7

Molecular formula: $Na_2S_2O_3$

Molecular weight: 158.1 g/mol

Synonyms: Sodium thiosulphate; disodium sulphanidesulphonate; sodium thiosulphate; thiosulfuric acid, disodium salt; disodium sulphurothioate

SMILES: [O-]S(=O)(=S)[O-].[Na+].[Na+]

II. Physico-Chemical Properties

Table 1: Overview of the Physico-Chemical Properties of Sodium Thiosulphate

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Colourless crystalline solid	2	ECHA
Melting point	<500°C (decomposition occurs) (pressure not indicated)	1	ECHA
Boiling Point	Not available	-	-
Density	1690 kg/m^3 @ 20°C	2	ECHA
Vapor Pressure	Not applicable	-	-
Partition Coefficient (log Kow)	Not applicable	-	-
Water solubility	764 g/L @ 25°C	2	ECHA
Flash Point	Not applicable	-	-
Auto flammability	Not applicable	-	-
Viscosity	Not applicable	-	-

[1]https://www.industrialchemicals.gov.au/chemical-information/search-assessments?assessmentcasnumber=7772-98-7++

Property	Value	Klimisch Score	Reference
Henry's Law Constant	Not applicable	-	-

III. ENVIRONMENTAL FATE PROPERTIES

Sodium thiosulphate dissociates in aqueous media to sodium (Na^+) and thiosulphate ($S_2O_3^{2-}$) ions. The thiosulphate anion is stable in neutral or alkaline media, but not in acidic media (EPA, 2007). In aqueous media, thiosulphate irreversibly disproportionates to sulphide and sulphate (EPA, 2007).

Biodegradation is not applicable to inorganic compounds. Sodium thiosulphate is not expected to bioaccumulate; it will dissociate to ions that are ubiquitous in the environment. Sodium thiosulphate is not expected to adsorb to soil or sediment because of its dissociation properties and high water solubility.

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Sodium thiosulphate is of low acute and chronic toxicity via oral dosing. It is not an eye or skin irritant nor does it illicit skin sensitisation effects. The substance does not exhibit genotoxicity, mutagenicity, carcinogenicity, reproductive or developmental toxicity.

B. Acute Toxicity

There are no acute toxicity studies available for sodium thiosulphate.

The oral LD_{50} of potassium thiosulphate in rats is >2,500 mg/kg (ECHA) [Kl. score = 2]. The oral LD_{50} of calcium thiosulphate in rats is >2,000 mg/kg (ECHA) [Kl. score = 1].

The inhalation 4-hr LC_{50} of potassium thiosulphate in rats is >2.6 mg/L aerosol, whole body. (ECHA) [Kl. score = 1]. The mass median aerodynamic diameter was 2.1 µm (ECHA) [KI. score =1].

The inhalation 4-hr LC_{50} for sodium sulphite in rats is >5.5 mg/L dust/aerosol test, nose/head only (ECHA) [KI. Score =2]. The mass median aerodynamic diameter was 2.7µm (ECHA) [KI. score =2].

The dermal LD_{50} of potassium thiosulphate in rabbits is >2000 mg/kg bw. The dermal LD_{50} of ammonium thiosulphate in rabbits is >2000 mg/kg bw (ECHA) [KI. score =2].

C. Irritation

No reliable skin irritation studies are available for sodium thiosulphate or other thiosulphate salts.

Sodium sulphite in the amount of 0.5 grams was administered to Vienna white rabbits via occlusive dressing for four hours. The rabbits were observed for 8 days with readings at 30-60 minutes after application of test material and 24 hours, 48 hours, and 8 days after the start of application. The mean score for after application of the test substance was 0.33 for erythema and 0 for oedema. On the 8th day of observation, there was no evidence of erythema or oedema, which suggests that all the observed effects were fully reversible (ECHA) [KI. score =2].

Instillation of 0.1 mL or 75 mg of ammonium thiosulphate into the left eyes of rabbits was determined to be non-irritating. The mean of the 1,24-, 48-, and 72-hour scores were: 0.00 for corneal opacity; 0.00 for iridial lesions; 0.56 for conjunctival redness; and 0.11 for chemosis (ECHA) [Kl. score = 2]. All signs of eye irritation in all of the treated animals were cleared by the 72-hour observation period (ECHA) [KI. score =2].

D. Sensitisation

Ammonium thiosulphate was not considered to be a skin sensitiser to mice based on reported findings from a mouse local lymph node assay (ECHA) [Kl. score = 1]. Treatment with concentrations of 10%, 25% or 50% ammonium thiosulphate did not induce a stimulation index for lymph node cell count above 1.4 and lymph node weight was not increased. In addition to this, there were no signs of local or systemic intolerance and the animal body weight was not impacted by exposure to ammonium thiosulphate (ECHA) [KI. score =1].

E. Repeated Dose Toxicity

Oral

No studies are available on the thiosulphate salts. Under acidic conditions, thiosulphates will disproportionate in aqueous mediate to form polythionic acids and bisulphite (HSO_3^-) ions plus sulfur dioxide gas (SO_2) (ECHA). A 2-year three-generation rat study on sodium metabisulfite will be used to read-across to sodium thiosulphate because sodium metabisulfite dissociates in water to form sodium (Na^+) ions, disulphite ($S_2O_5^{2-}$) ions, and sulfur dioxide (SO_2). The disulfite ions can form bisulphite (HSO_3^-) and sulfite ions (SO_2^{3-}) in varying proportions dependent on the pH of the solution (OECD, 2001).

Male and female Wistar rats were fed in their diet 0, 0.125, 0.25, 0.5, 1.0, or 2.0% sodium metabisulfite in a thiamine-containing diet (50 ppm) for up to two years and over three generations. The diet was enriched with thiamine to prevent thiamine deficiency as a result of the sulphite-induced destruction of this vitamin. During storage up to the time of consumption, the losses of sulphite from the feed containing sodium metabisulfite at levels of 0.125, 0.25, 0.5, 1.0, and 2.0% averaged 22, 14,12, 8, and 4.5%, respectively, while the decrease in thiamine was 2.7, 1.7, 8.3, 14.5, and 15.4%, respectively. The addition of thiamine to the diet prevented thiamine deficiency in rats at all dose levels based on measurements of thiamine levels in the urine and liver. The general condition of the rats were good during the first 72 weeks of the F0 generation, as well as the other two generations. After 72 weeks, there was a rapid increase in mortality in all groups. Survival in the treated groups was higher than the controls, except for the 2% F_1 males; no deaths occurred in the 2% F_2 females. A marginal reduction in body weight gain was observed in the 2% dose group (both sexes) in the F_1 and F_2 generations. Feed consumption was similar was between treated and control groups. There were no changes in haematology and clinical chemistry parameters and urinalysis that were considered toxicologically significant. The $\geq$1% dietary groups had occult blood in their feces. Relative kidney weights were increased in the 2% F_2 females, but there were no pathological changes noted in the kidneys from this group. Hyperplastic changes in the fore- and glandular stomachs were noted in the $\geq$1% groups in all three generations. Some slight alterations were also noted in stomachs of the 0.5% F_2 rats. Based on the occurrence of occult blood in faeces and changes in gastric morphology at dose levels of 0.5% or more, the NOAEL for local chronic toxicity in this study is represented by the dose of 0.25% metabisulfite (0.215% accounting for the loss of metabisulfite). The corrected dose level corresponds to a dose of 108 mg/kg bw/day of sodium thiosulphate. The NOAEL for systemic toxicity is 1.91% in the diet. This was estimated to be >955 mg/kg-day (1589 mg/kg bw/day sodium thiosulphate) based on a rat body weight of 400 g and a

daily feed intake of 20 g. The histopathologic effects on the stomach and the occult blood in feces are considered to be the result of localised irritation (a site-of-contact effect) from the ingestion of sodium metabisulfite (Til et al., 1972; as cited in ECHA). [Kl. score = 2]

Inhalation

There are no adequate studies available to determine a NOAEC for sodium thiosulphate.

Dermal

No studies are available given the fact that there is no evidence for significant absorption through the skin

F. Genotoxicity

In vitro Studies

No studies are available on sodium thiosulphate. The *in vitro* genotoxicity studies on ammonium thiosulphate are presented below in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Ammonium Thiosulphate

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
OECD Guideline 471 Bacterial reverse mutation (*S. typhimurium* and *E. coli* strains)	-	-	1	ECHA
OECD Guideline 476 (*In Vitro* Mammalian Cell Gene Mutation Test)	-	-	1	ECHA
OECD Guideline 473 *In Vitro* Mammalian Chromosomal aberration Test (Chinese hamster ovary cells)	-	-	1	ECHA

*+, positive; -, negative

In vivo Studies

No studies are available.

G. Carcinogenicity

Oral

No studies are available on the thiosulphate salts. Under acidic conditions, thiosulphates will disproportionate in aqueous mediate to form polythionic acids and bisulphite (HSO_3^-) ions plus sulfur dioxide gas (SO_2) (ECHA). A 2-year three-generation rat study on sodium metabisulfite will be used to read-across to sodium thiosulphate because sodium metabisulfite dissociates in water to form sodium (Na^+) ions, disulphite ($S_2O_5^{2-}$) ions, and sulfur dioxide (SO_2). The disulfite ions can form bisulphite (HSO_3^-) and sulfite ions (SO_2^{3-}) in varying proportions dependent on the pH of the solution (OECD, 2001).

Male and female Wistar rats were fed in their diet 0, 0.125, 0.25, 0.5, 1.0, or 2.0% sodium metabisulfite for up to two years and over three generations. There was no increased incidence of tumours in the treated groups compared to the controls (Til et al., 1972 as cited in ECHA). [Kl. score = 2].

Male and female ICR/JCL mice were given in their drinking water 0, 1, or 2% potassium metabisulfite for two years. There was no increased incidence of tumours in the treated groups compared to the controls (Tanaka et al., 1994 as cited in ECHA). [Kl. score = 2].

Male and female Wistar rats were continuously fed in their diet 0%, 0.5%,1%, 2%, 4%, 6%, and 8% sodium metabisulphite for 10-56 days. Microscopic examinations gave no evidence of the formation of tumours (ECHA) [KI. score =2].

Male and female rats were exposed to 375 and 750 ppm sodium metabisulphite continuously via their drinking water for 2.5 years/over 3 generations. The incidence of tumours was unaffected by the addition of disodium disulphate (ECHA) [KI. Score =2].

Male Fischer 344/DuCrj rats were exposed to potassium sulphite or potassium metabisulfite via oral gavage (single dose). The results from this study indicate that potassium sulphite and potassium metabisulfite may have tumour promoting activity in glandular stomach carcinogenesis (ECHA) [KI. score =2].

Male Wistar rats were exposed to 1% potassium metabisulfite continuously via their drinking water for up to 40 weeks. It was concluded that potassium metabisulfite could be considered to exert tumour promoting activity in the rat glandular stomach (ECHA) [KI. score =2].

Inhalation

Male Sprague-Dawley rats were exposed to 10 and 30 ppm sulfur dioxide via whole body inhalation (gas) for 6 hours per day for 5 days per week for a total of 21 weeks/ 101 exposure days. There were no adverse effects reported from sulfur dioxide exposure (ECHA) [KI. score =2].

H. Reproductive Toxicity

No studies are available on the thiosulphate salts. Under acidic conditions, thiosulphates will disproportionate in aqueous mediate to form polythionic acids and bisulphite (HSO_3^-) ions plus sulfur dioxide gas (SO_2) (ECHA). A 2-year three-generation rat study on sodium metabisulfite will be used to read-across to sodium thiosulphate because sodium metabisulfite dissociates in water to form sodium (Na^+) ions, disulfate ($S_2O_5^{2-}$) ions, and sulfur dioxide (SO_2). The disulfite ions can form bisulfited (HSO_3^-) and sulfite ions (SO_2^{3-}) in varying proportions dependent on the pH of the solution (OECD, 2001).

Male and female Wistar rats were fed in their diet 0, 0.125, 0.25, 0.5, 1.0, or 2.0% sodium metabisulfite for up to two years and over three generations. The diet was enriched with thiamine to prevent thiamine deficiency as a result of the sulfite-induced destruction of this vitamin. During storage up to the time of consumption, the losses of sulfite from the feed containing sodium metabisulfite at levels of 0.125, 0.25, 0.5, 1.0, and 2.0% averaged 22, 14,12, 8, and 4.5%, respectively, while the decrease in thiamine was 2.7, 1.7, 8.3, 14.5, and 15.4%, respectively. The addition of thiamine to the diet prevented thiamine deficiency in rats at all dose levels based on measurements of thiamine levels in the urine and liver. The effects other than reproductive and developmental toxicity are discussed above in the Repeated Dose Toxicity section. There were no

treatment-related effects on female fertility, the number of young per litter, or birth weight or mortality of the offspring. The number of F_{2a} pups was significantly reduced in the ≥0.5% groups during the first breeding cycle, but there was no dose-response, and the reduction did not occur during the second breeding cycle. Slight growth retardation was observed in the F_1 and F_2 generation rats both before and after weaning. The NOAEL for reproductive toxicity is 1.91% in the diet. This was estimated to be 955 mg/kg-day (640 mg sulfur dioxide/kg bw/day) based on a rat body weight of 400 g and a daily feed intake of 20 g (Til et al., 1972 as cited in ECHA). [Kl. score = 2]

Male and female rats were given sodium metabisulfite in their drinking water for up to 2.5 years and three successive generations. The doses were 375 and 750 ppm as sulfur dioxide (SO2). There was no evidence of systemic toxicity in either dose group. The number of offspring of either the F1 and F2 generation and the proportion surviving to the end of lactation were similar between treated and control groups. The NOAEL for reproductive toxicity is 750 ppm (as SO2) in drinking water. Assuming an average rat body weight of 400 g and a daily water intake of 28 mL, 750 ppm (as SO2) corresponds to 53 mg/kg-day sodium metabisulfite (Lockett, 1960 as cited in ECHA). [Kl. score = 2]

I. Developmental Toxicity

Pregnant female Wistar rats were dosed by oral gavage with 0, 4, 19, 86, or 400 mg/kg sodium thiosulphate on GD 6 to 15. There was no maternal or developmental toxicity. The NOAEL for maternal and developmental toxicity is 400 mg/kg-day, the highest dose tested (ECHA) [Kl. score = 2].

Pregnant female CD-1 mice were dosed by oral gavage with 0, 5.5, 25.5, 118, or 555 mg/kg sodium thiosulphate on GD 6 to 15. There was no maternal or developmental toxicity. The NOAEL for maternal and developmental toxicity is 555 mg/kg-day, the highest dose tested (ECHA) [Kl. score = 2].

Pregnant female Dutch-belted rabbits were dosed by oral gavage with 0, 2.5, 5.8, 27, 125.4, or 580 mg/kg sodium thiosulphate on GD 6 to 18. There was no maternal or developmental toxicity. The NOAEL for maternal and developmental toxicity is 580 mg/kg-day, the highest dose tested (ECHA) [Kl. score = 2].

Pregnant female golden hamsters were dosed by oral gavage with 4.0, 19.0, 86.0, and 400 mg/kg bw sodium thiosulphate from gestation day 6 until gestation day 14. The NOAEL for maternal and developmental toxicity is 400 mg/kg-day, the highest dose tested (ECHA) [Kl. score = 2].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

A. Non-Cancer

Oral

Sodium thiosulphate dissociates in aqueous media to sodium (Na^+) and thiosulphate ($S_2O_3^{2-}$) ions. In addition, NICNAS does not consider sodium thiosulphate to pose an unreasonable risk to the health of workers and public health on the basis of the Tier I IMAP assessment.[2] Therefore, an oral reference dose and drinking water guidance value was not derived for sodium thiosulphate.

[2] https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-assessments/human-health-assessments#cas-A_7772-98-7.

The Australian drinking water guideline values for sodium (180 mg/L) and sulphate (250 mg/L) may apply to sodium thiosulphate.

B. Cancer

Sodium or potassium metasulphite were not carcinogenic to rodents in two-year dietary studies. Thus, a cancer reference value was not derived for sodium thiosulphate.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

Sodium thiosulphate does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidizing potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

The substance does not appear to exhibit significant acute aquatic toxicity. No data are available for chronic toxicity studies.

B. Aquatic Toxicity

Acute Studies

No data are available on sodium thiosulphate. Table 3 lists the results of acute aquatic toxicity studies conducted on ammonium thiosulphate (CAS No. 7783-18-8).

Table 3: Acute Aquatic Toxicity Studies on Ammonium Thiosulphate[1]

Test Species	Endpoint	Results (mg/L)	Klimisch Score	Reference
Lepomis macrochirus	96-hr LC_{50}	510	1	ECHA
Salmo gairdneri	96-hr LC_{50}	770 (583)	1	ECHA
Daphnia magna	48-hr EC_{50}	230 (174)	1	ECHA
Pseudokirchneriella subcapitata	72-hr EC_{50}	>100 (>75.7)	1	ECHA

1 Where provided in ECHA, value in parenthetical is expressed as thiosulphate.

Chronic Studies

No studies were identified for sodium thiosulphate or ammonium thiosulphate. However, reliable chronic toxicity data were available for sodium sulphite (CAS No. 7757-83-7) and sodium disulphite (CAS No. 7757-74-6). **Table 4** lists the results of chronic aquatic toxicity studies conducted on sodium sulphite and sodium disulphite.

Table 4: Chronic Aquatic Toxicity Studies on Sodium Sulphite and Sodium Disulphite

Test Species	Endpoint	Results (mg/L)[1]	Klimisch Score	Reference
Danio rerio (zebrafish)	34-d NOEC	>316 (140.6)	1	ECHA
Daphnia magna	21-d NOEC	>10 (>8)	2	ECHA
Pseudokirchneriella subcapitata	EC_{10}	>75.7	2	ECHA

1 Value in parenthetical indicates data translated to sodium thiosulphate, assuming that all S is converted to sulphite when thiosulphate oxidizes

C. Terrestrial Toxicity

No terrestrial toxicity data are available for this substance.

D. Calculation of PNEC

The PNEC calculations for sodium thiosulphate follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available on ammonium thiosulphate for three trophic levels. Acute $E(L)C_{50}$ values are available for fish (583 mg/L), Daphnia (174 mg/L), and algae (>75.7 mg/L). NOEC values from long-term studies are available for fish (140.6 mg/L), invertebrates (>8 mg/L), and algae (>75.7 mg/L). On the basis that the data consists of short-term and long-term results from three trophic levels, an assessment factor of 10 has been applied to the lowest reported NOEC value of 8 mg/L for invertebrates. The $PNEC_{water}$ for sodium thiosulphate is 0.8 mg/L.

PNEC Sediment

No experimental toxicity data on sediment organisms are available. Sodium thiosulphate dissociates completely in water with its environmental distribution is dominated by its high water solubility. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as sodium thiosulphate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{sediment}$. Based on its properties, no adsorption of sodium thiosulphate to sediment is to be expected, and the assessment of this compartment will be covered by the aquatic assessment.

PNEC Soil

No reliable experimental toxicity data on terrestrial organisms are available. The environmental distribution of sodium thiosulphate is dominated by its water solubility. Sorption of sodium thiosulphate should probably be regarded as a reversible situation, i.e., the substance is not tightly nor permanently bound. K_{ow} and K_{oc} parameters do not readily apply to inorganics, such as sodium thiosulphate. Thus, the equilibrium partitioning method cannot be used to calculate the $PNEC_{soil}$. Based on its properties, sodium thiosulphate is not expected to significantly adsorb to soil, and the assessment of this compartment will be covered by the aquatic assessment.

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU Reach Criteria methodology (DEWHA, 2009; ECHA, 2008).

Sodium thiosulphate is an organic salt that dissociates completely to sodium, sulphide, and sulphate ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions; these ionic species are also ubiquitous and are present in most water, soil and sediment. For the purposes of this PBT assessment, the persistent criteria are not considered applicable to sodium thiosulphate or its dissociated ions.

Sodium thiosulphate dissociates to ionic species. The sulphide ion can be oxidized by bacteria to sulphate. The sodium and sulphate ions are essential to all living organisms and their intracellular and extracellular concentrations are actively regulated. Thus, sodium thiosulphate is not expected to bioaccumulate.

There are no chronic toxicity studies on sodium thiosulphate. However, the NOEC or EC10 values from chronic aquatic toxicity studies on read-across sodium sulphite are >0.1 mg/L. The acute $EC(L)_{50}$ values on read-across ammonium thiosulphate are >1 mg/L in fish, invertebrates and algae. Thus, sodium thiosulphate does not meet the screening criteria for toxicity.

The overall conclusion is that sodium thiosulphate is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H315: Causes skin irritation
H319: Causes serious eye irritation
H335: May cause respiratory irritation

B. Labelling

Warning

C. Pictogram

X. HANDLING AND SAFETY (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

In the case of contact, immediately flush eyes with plenty of water for at least 15 minutes. If symptoms persist, seek medical advice.

Skin Contact

Wash thoroughly with soap and water.

Inhalation

If inhaled, remove from area to fresh air.

Ingestion

Rinse mouth with water and then drink plenty of water. Do not induce vomiting. Never give anything by mouth to an unconscious person. Seek medical attention.

B. Fire Fighting Information

Extinguishing Media

Water spray or fog, carbon dioxide, dry powder.

Specific Exposure Hazards

Burning produces harmful and toxic fumes.

Special Protective Equipment for Firefighters

Wear a self-contained breathing apparatus.

C. Accidental Release Measures

Personal Precautions

No special precautions are necessary. Ensure adequate ventilation.

Environmental Precautions

Do not discharge into drains, sewers, or waterways.

Steps to be Taken if Material is Released or Spilt

For large amounts: dike spillage and pump off the product. For residues: pick up with suitable absorbent material. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Handle in accordance with good industrial hygiene and safety practice.

Other Handling Precautions

Protect against fire and explosion: prevent electrostatic charge; sources of ignition should be kept well clear, and fire extinguishers should be kept handy.

Storage

Keep container tightly closed and dry. Protect against heat. Store below 25^{o}C.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Occupational exposure standards for the low molecular weight PEGs have not been established.

Engineering Controls

Provide local exhaust ventilation to control vapours and mists.

Personal Protection Equipment

Respiratory Protection: Respiratory protection in case of vapours/aerosol release. Wear a certified organic vapour/particulate respirator.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Body protection must be chosen depending on activity and possible exposure. Safety glasses with side-shields.

Other Precautions: Wash hands, forearms, and face thoroughly after handling chemical products, before eating, smoking, and using the lavatory and at the end of the working period. Appropriate techniques should be used to remove potentially contaminated clothing. Wash contaminated clothing before reusing. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for sodium thiosulphate.

Engineering Controls

Provide local exhaust ventilation to control vapours and mists.

Personal Protection Equipment

Respiratory Protection: Respiratory protection in case of vapours/aerosol release. Wear a certified organic vapour/particulate respirator.

Hand Protection: Chemical resistant protective gloves.

Skin Protection: Body protection must be chosen depending on activity and possible exposure.

Eye protection: Body protection must be chosen depending on activity and possible exposure. Safety glasses with side-shields.

Other Precautions: Wash hands, forearms, and face thoroughly after handling chemical products, before eating, smoking, and using the lavatory and at the end of the working period. Appropriate techniques should be used to remove potentially contaminated clothing. Wash contaminated clothing before reusing. Ensure that eyewash stations and safety showers are close to the workstation location.

G. Transport Information

Sodium thiosulphate is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

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ETHOXYLATED TALLOW ALKYL AMINE

This dossier on tallow alkyl amines ethoxylated presents the most critical studies pertinent to the risk assessment of glutaraldehyde in its use in coal seam or shale gas extraction activities. There is no sufficient data available for this particular substance. This dossier does not represent an exhaustive or critical review of all available data. The majority of information presented in this dossier was obtained from the National Industrial Chemicals Notification and Assessment Scheme (NICNAS, 1994) and the ECHA database that provides information on chemicals that have been registered under the European Union (EU) REACH (ECHA). Where possible, study quality was evaluated using the Klimisch scoring system (Klimisch et al., 1997).

For the purpose of this dossier, fatty acids tall oil ethoxylated (CAS RN 61791-00-2), fatty acids, tall oil, ethoxylated (EO 5) (CAS No. 9004-96-0), or fatty acids, tall-oil, 2-hydroxyethyl esters (CAS RN 97281-31-7) has been reviewed as surrogate chemicals for ethoxylated tallow alkyl amine, where appropriate.

I. SUBSTANCE IDENTIFICATION

Chemical Name (IUPAC): Tallow alkyl amines ethoxylated

CAS RN: 61791-26-2

Molecular formula: Not applicable. This substance is a UVCB.

Molecular weight: Not applicable. This substance is a UVCB.

Synonyms: Ethoxylated tallow alkyl amine; amines, tallow alkyl, ethoxylated; Polyoxyethylene, tallow amine; Primary tallow amine, ethylene oxide adduct

SMILES: Not applicable. This substance is a UVCB.

II. PHYSICO-CHEMICAL PROPERTIES

There are no physical or chemical data for tallow alkyl amines ethoxylated. The data presented below are abstracted from data on a similar substance, fatty acids tall oil ethoxylated (CAS RN 61791-00-2).

Table 1: Physico-Chemical Properties of Tallow Alkyl Amines Ethoxylated[1]

Property	Value	Klimisch Score	Reference
Physical state at 20°C and 101.3 kPa	Liquid.	1	ECHA
Melting point	≥-85 ≤ -5°C @ 101.3 kPa	1	ECHA
Boiling point	Not available. During the heating process the test item began to change its state at approximately 172 °C from liquid to highly viscous. This indicates a thermally caused change of the test item.	1	ECHA

Property	Value	Klimisch Score	Reference
Density	0.958 (relative density) @ 20°C	1	ECHA
Vapour pressure	The vapour pressure could not be determined.	1	ECHA
Partition coefficient (log K_{ow})	5.94 @ 25 °C	1	ECHA
Water solubility	The test item can be mixed with water up to a ratio of 3:7 (m (test item): :m (water). It is not possible to determine a concrete value for the water solubility	1	ECHA
Flash point	Flash point @102.2 kPa 138 °C	1	ECHA
Auto flammability	377 °C @103.1 kPa	1	ECHA
Viscosity	58.0 mPa*s at 20 °C	1	ECHA

[1] = data from fatty acids tall-oil ethoxylated (CAS RN 61791-00-2)

III. ENVIRONMENTAL FATE PROPERTIES

A. Summary

Tallow alkyl amines ethoxylated are expected to biodegrade and show some degree of sorption to sediments and soils. They are not expected to bioaccumulate.

B. Biodegradation

Data on the ready biodegradability of tallow alkyl amines ethoxylated (EO > 1 < 2.5) (CAS 61791-00-2) are not available. Therefore, data on the ready biodegradability of the structurally related analogue substance fatty acids, tall oil, ethoxylated (EO 5) (CAS No. 9004-96-0) is used as read across substance.

This read-across is justified because both, target, and source substance, are structurally identical (ethoxylated oleic acid) except for the fact that the source substance is slightly higher ethoxylated (5 EO) than the target substance (1-2.5EO). This difference might lead to a slightly lower water solubility of the target substance; however, since the solubility of both substances is rather high and not limiting the bio accessibility of the substances to aquatic microorganisms this is not considered to influence the identical biodegradation behaviour of both substances. Both substances share the same functional groups and the same mode of action (baseline toxicity caused by the long lipophilic fatty acid chain). Thus, biotransformation can with very high certainty assumed to be identical.

The biodegradation of fatty acids, tall oil, ethoxylated (EO 5) (CAS No. 9004-96-0) was evaluated test in accordance with OECD Guideline 301 B (Ready Biodegradability: CO_2 Evolution Test), under GLP conditions. Domestic, non-adapted activated sludge was exposed to fatty acids, tall oil, ethoxylated (EO 5) (CAS No. 9004-96-0) for 28 days at 22°C, and biodegradation was measured by CO_2 consumption. After 28 days, fatty acids, tall oil, ethoxylated (EO 5) (CAS No. 9004-96-0) reached a biodegradation of 90 - 100 %. Based on the results for the read-across substance, fatty acids, tall-oil, ethoxylated (EO > 1 < 2.5) (CAS 61791-00-2) is considered to be readily biodegradable (ECHA) [KI. score = 1].

If a chemical is found to be readily biodegradable, it is categorised as Not Persistent since its half-life is substantially less than 60 days (DoEE, 2017).

C. Environmental Distribution

One study investigating the adsorption/desorption behaviour of fatty acids, tall-oil, ethoxylated (CAS RN 61791-00-2) is available. The study was performed according to GLP and OECD guideline 121 (Estimation of Adsorption Coefficient K_{oc} on Soil and on Sewage Sludge using high performance liquid Chromatography or HPLC). Six different peaks were observed with log K_{oc} values ranging from < 1.8 to > 5.63. The two main components (> 85%) show log K_{oc} values > 4. (ECHA) [KI.score =1]. Based on these values and its limited water solubility, fatty acids, tall-oil, ethoxylated will be slightly to hardly mobile in soil as adsorption to soil is expected.

D. Bioaccumulation

Fatty acids, tall-oil, 2-hydroxyethyl esters (CAS RN 97281-31-7) consists of components with log K_{ow} values in the range of 5 to > 10 (KOWWIN v1.68) indicating a potential for bioaccumulation. But due to rapid environmental biodegradation, metabolization via enzymatic hydrolysis (monoesters and diesters) as well as steric hindrance of crossing biological membranes (high molecular weight of diesters) a relevant uptake and bioaccumulation in aquatic organisms is not expected. This is supported by low bioconcentration factor (BCF values of < 100 litres per kilogram of water weight (L/kg ww) (BCFBAF v3.01, Arnot-Gobas, including biotransformation, upper trophic) calculated for different components of the UVCB (mono- and diester EO1 to EO5).

Thus, taking all information into account, fatty acids, tall-oil, 2-hydroxyethyl esters (CAS RN 97281-31-7) is not considered to be bioaccumulative.

IV. HUMAN HEALTH HAZARD ASSESSMENT

A. Summary

Tallow alkyl amines ethoxylated are not acutely toxic. The substance is not expected to be irritating to the eyes or the skin, but it is expected to be a skin sensitiser. Tallow alkyl amines ethoxylated are not genotoxic, mutagenic, or carcinogenic. There is no evidence that tallow alkyl amines ethoxylated cause reproductive or developmental toxicity.

B. Toxicokinetics

There are no data available for tallow alkyl amines ethoxylated.

C. Acute Toxicity

In an acute oral toxicity study performed similar to OECD guideline 401, three groups of Gassner rats consisting of 10 animals/sex/dose were treated by single gavage application with an aqueous solution of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) (10,000, 8,000, 6,400 milligrams per kilogram of body weight [mg/kg bw]). The animals were observed for mortality and for clinical symptoms of toxicity over a period of 7 days. At the end of the observation period, the surviving animals were sacrificed for the purpose of necropsy. No mortality was observed for any of the tested concentrations. At all doses mastication, irregular breathing, redness of the eyes and closed eyes were seen immediately after dosing. The next morning mastication and irregular breathing was observed. On the following days, no clinical sings were observed. Pathological examination revealed hydrometra in 3 animals exposed to 10000 mg/kg bw, 2 animals exposed to 8000 mg/kg bw, and 3

animals exposed to 6,400 mg/kg bw. Based on the results obtained under the test conditions of this study, the acute oral LD_{50} was determined to be > 10,000 mg/kg bw.

In another acute oral toxicity study of similar design four groups of rats consisting of 5 animals/sex/dose were treated by single gavage application with an aqueous solution of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) (200, 6,400, 3,200, 1,600microlitres per kilogram [µL/kg]). The animals were observed for mortality and for clinical symptoms of toxicity. At the end of the observation period, the surviving animals were sacrificed for the purpose of necropsy. No mortality occurred at the tested concentrations. At all doses on the day of the experiment, restless behaviour was observed after application. The animals had slightly accelerated breathing as well as ruffled fur. Four days after the application all animals were without clinical signs. In this study no pathological changes in the organs were observed. One animal showed bronchitis and bronchiectasis on both sides. The LD_{50} was reported to be > 6.4 ml/kg bw (ECHA) [KI. score = 2].

In an additional study a limit test was performed using male and female Wistar rats were treated by single oral administration 2,000 milligrams per kilogram (mg/kg) of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) (2 animals/sex/dose). During the observation period of 14 days, no clinical symptoms of toxicity or mortality were observed. The LD_{50} was reported to be >2,000 mg/kg (ECHA) [KI. score =2].

Inhalation

Based on the inhalation studies, no conclusion on LC_{50} can be drawn, because the tested concentrations of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) are too low in relation to the classification criteria (ECHA)[KI. score =2].

Dermal

There are no data to evaluate dermal toxicity of tallow alkyl amines ethoxylated .

D. Irritation

Skin

By using the currently available methods a single *in vitro* assay is not sufficient to cover the full range of skin irritating/corrosion potential. Therefore, two *in vitro* assays were part of an *in vitro* skin irritation and corrosion test strategy (BASF 2017): The Skin Corrosion Test (SCT) and Skin Irritation Test (SIT). However, the results derived with SIT (performed in a GLP compliant study according to OECD 431, OECD 439, EU method B.40 BIS. And EU method B.46) alone were sufficient for a final assessment. Therefore, further testing in SCT was waived.

The potential of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) to cause dermal irritation was assessed by a single topical application of 30 microlitres (µL) of the undiluted test substance to a reconstructed three-dimensional human epidermis model (EpiDerm™). The irritation test was performed with three EpiDerm™ tissues which were incubated with the test substance for 1 hour followed by a 42-hour post-incubation period. Tissue destruction was determined by measuring the metabolic activity of the tissue after exposure/post-incubation by using a colorimetric test. The reduction of mitochondrial dehydrogenase activity measured by reduced formazan production after incubation with a tetrazolium salt (MTT) was chosen as endpoint. The formazan production of the epidermal tissues treated with the test substance is compared to that of negative control tissues. The quotient of the values indicates the relative tissue viability.

The following results were obtained in the EpiDerm™ skin irritation test: 1) The test substance is able to directly reduce MTT. Therefore, an additional MTT reduction control KC (freeze-killed control tissues) was introduced. 2) The final mean viability of the tissues treated with the fatty acids tall oil ethoxylated (CAS RN 61791-00-2) determined after an exposure period of 1 hour with an about 42-hour post-incubation was 100.7%.

Based on the results observed and by applying the evaluation criteria, it was concluded that the fatty acids tall oil ethoxylated (CAS RN 61791-00-2) does not show a skin irritation potential in the EpiDerm™ *in vitro* skin irritation and corrosion test strategy under the test conditions chosen (ECHA) [KI. score = 1].

In a supporting skin irritation test two Vienna white rabbits were treated with fatty acids tall oil ethoxylated (CAS RN 61791-00-2) for 1, 5, 15 min and 20 hours under occlusive conditions (BASF 1971). An application site of 2.5 x 2.5 cm was covered with the liquid test substance. After the application time (1, 5, 15 min and 20 hours) the skin was washed with Lutrol (50%). The animals were observed for 8 days, and skin changes were recorded daily. The report describes findings after 24 hours and at the end of the observation period (8 days). After 20 hours exposure to the test-substance one animals showed slight erythema after 24 hours (Draize score 2). The observed redness was resolved by the end of the observation period, but a slight scaling was still present. The other animal exposed for 20 hours showed only some questionable erythema effect after 24 hours (score 1) which was fully reversible within 72 hours. No other effects were noted in the animals exposed for 20 hours. Of the animals exposed for shorter periods (1, 5 or 15 minutes) only one animal exposed for 15 minutes showed some questionable erythema which was fully reversible (ECHA) [KI. score =2].

In another similar performed skin irritation test showed stronger effects. The Vienna white rabbits were exposed to fatty acids tall oil ethoxylated (CAS RN 61791-00-2) for 20 hours showed strong to very strong erythema across the whole exposed area. After 8 days the redness in one animal was decreased to slight and had disappeared in the other. However, strong scaling was observed in both animals. In addition to the erythema a slight swelling was seen at 24 hours which also had disappeared after 8 days. The animals exposed for 15 minutes showed questionable erythema which was fully reversible. No ulcers, bleeding, or bloody scabs were observed. Animals exposed for shorter period did not show any signs of irritation. The OECD guideline 404 (Acute Dermal Irritation/Corrosion) states a typical exposure duration of 4 hour under open or semi-occlusive conditions. Therefore, the test employing 20 hours exposure under occlusive conditions is considered a worst-case situation (ECHA) [KI. score = 2].

Severe skin irritating effects were only seen in one of the studies, however, considering the worst-case conditions these effects are questionable. In contrast, the *in vitro* guideline study fatty acids tall oil ethoxylated (CAS RN 61791-00-2) was considered not to be skin irritant, which is supported by the other *in vivo* study (ECHA)

Based on these data, fatty acids tall oil ethoxylated (CAS RN 61791-00-2) is not considered a skin irritant.

<u>Eye</u>

The eye irritating potential of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) was tested *in vitro*. By using the methods currently available a single *in vitro* assay is not sufficient to cover the full range of eye irritating potential. Therefore, two *in vitro* assays were part of this *in vitro* eye irritation test strategy: The Bovine Corneal Opacity and Permeability Test (BCOP Test) and EpiOcular Eye Irritation

Test. However, in the current case the results derived with the EpiOcular test alone (which was applied conforming GLP and in accordance with OECD 492) were sufficient for a final assessment. Therefore, further testing in BCOP was waived.

The potential of the fatty acids tall oil ethoxylated (CAS RN 61791-00-2) to cause ocular irritation was assessed by a single topical application of 50 µL undiluted fatty acids tall oil ethoxylated (CAS RN 61791-00-2) to a reconstructed three-dimensional, human cornea model (EpiOcular™). Two EpiOcular™ tissues were incubated with the test substance for 30 minutes followed by a 2-hour post-incubation period. Tissue destruction was determined by measuring the metabolic activity of the tissue after exposure/post-incubation by using a colorimetric test. The reduction of mitochondrial dehydrogenase activity measured by reduced formazan production after incubation with MTT was chosen as endpoint. The formazan production of the epidermal tissues treated with the test substance is compared to that of negative control tissues. The ratio of the values indicates the relative tissue viability. The following results were obtained in the EpiOcular™ eye irritation assay: 1) Fatty acids tall oil ethoxylated (CAS RN 61791-00-2) is able to directly reduce MTT. Therefore, an additional MTT reduction control (freeze-killed control tissues (KC)) was introduced. 2) The final mean viability of the tissues treated with the test substance was 109.3% (ECHA) [KI. score =1].

Based on the results observed in the EpiOcular Test alone and by applying the evaluation criteria, it was concluded that fatty acids tall oil ethoxylated (CAS RN 61791-00-2) does not show an eye irritation potential in the *in vitro* eye irritation test strategy under the test conditions chosen (ECHA).

In a supporting eye irritation test (BASF 1971) 50 µL of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) was applied to the conjunctival sac of one eye in of two Vienna white rabbits. The adjacent eye served as saline-control. The animals were observed after 1 and 24 hours on the day of treatment and up to 8 days afterwards. The eyes were not washed out after 24 hours as specified in OECD Guideline 405. One hour after application of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) slight redness of the conjunctivae was observed in both animals. After 24 hours one animal still showed slight redness of the conjunctivae while the effects in the other animal were completely reversed. After 8 days both animals were without eye irritating effects (ECHA) [KI. score =2].

In another supporting eye irritation test (BASF 1966) of the same design and exposure regime similar results were obtained. One hour after application of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) slight redness of the conjunctivae was observed in both animals. After 24 hours no eye irritation effects were observed until the end of the observation period. Based on these results, the test substance is considered to be not irritating to the eyes (ECHA) [KI. score =2].

E. Sensitisation

Fatty acids tall oil ethoxylated (CAS RN 61791-00-2) is not considered to be a sensitiser based on results obtained via the Buehler test.

<u>Local Lymph Node Assay (LLNA)</u>

The skin sensitising potential of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) was assessed using the radioactive Murine Local Lymph Node Assay in a GLP compliant study according to OECD no. 429, Commission Regulation (EC) No 440/2008 Part B, and EPA OPPTS 870.2600. The assay simulates the induction phase for skin sensitisation in mice. It determines the response of the auricular lymph nodes on repeated application of the test substance to the dorsal skin of the ears. Groups of 5 female CBA/J mice each were treated with 3%, 10% and 30% w/w preparations of the

test substance in methyl ethyl ketone (MEK) or with the vehicle alone. The high concentration was selected based on the presence of ear irritation in a pretest using a 60% preparation. The study used 3 test groups and 1 control group. Each test animal was applied with 25 µL per ear of the respective test-substance preparation to the dorsum of both ears for three consecutive days. The control group was treated with 25 µL per ear of the vehicle alone. Three days after the last application the mice were injected intravenously with 20 µCi of 3H-thymidine in 250 µL of sterile saline into a tail vein. About 5 hours after the 3H-thymidine injection, the mice were sacrificed, and the auricular lymph nodes were removed. The weights of each animal's pooled lymph nodes were determined. Thereafter lymph nodes were pooled group wise and further evaluated by measuring their cellular content and 3H-thymidine incorporation into the lymph node cells (indicators of cell proliferation). Moreover, a defined area with a diameter of 0.8 cm was punched out of the apical part of each ear and for each test group the weight of the pooled punches was determined in order to obtain an indication of possible skin irritation. The stimulation indices (fold of change as compared to the vehicle control) for cell count, 3H-thymidine incorporation, lymph node weight and ear weight were determined. No signs of systemic toxicity were noticed. When applied as 3%, 10% and 30% preparations in MEK, the test substance did not induce a biologically relevant response (no increase to 1.5-fold or above of control value = stimulation index (SI) ≥ 1.5) in the auricular lymph node cell counts. There was no relevant increase in lymph node weights as well. Concomitantly, the increase of 3H-thymidine incorporation into the cells was not biologically relevant (no increase above the cut off stimulation index of 3) at this concentration. The 30% test-substance preparation caused a minimal increase in ear weights as indication of ear skin irritation. Thus, it is concluded that fatty acids tall oil ethoxylated (CAS RN 61791-00-2) does not show a skin sensitising effect in the Murine Local Lymph Node Assay under the test conditions chosen (ECHA) [KI. score=1].

<u>Buehler test</u>

The dermal sensitising potential of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) was investigated according to one of the methods recommended in the OECD Guideline No. 406, "Skin Sensitisation", 1992 and the EEC Guideline "EEC 92/69 part B6", 1992. The test used was the Buehler test.

The experiment was performed on 30 guinea pigs divided into a test group of 20 animals, and a control group of 10 animals. The study included an induction and a challenge phase. The animals in the test group were induced with the test article and the animals in the control group were induced with sterile distilled water. The induction procedure included a closed patch topical application for 6 hours once a week for 3 weeks.

The challenge procedure included a closed patch topical treatment of the test article on the flank 4 weeks after the first induction. All animals were challenged for 6 hours. The skin reactions were evaluated 24 and 48 hours after termination of the challenge application. The undiluted fatty acids tall oil ethoxylated (CAS RN 61791-00-2) was used for the inductions as well as for the challenge application.

Slight erythema was observed in 8 and 6 animals after 24 and 48 hours, respectively. However, slight erythema was considered a marginal skin change due to other factors than skin sensitisation. After 24 hours a moderate erythema was seen in 1 animal and after 48 hours a moderate erythema was seen in 5 animals. Based on these results, fatty acids tall oil ethoxylated (CAS RN 61791-00-2) is considered to be sensitising to the skin in the Buehler test (ECHA) [KI. score =1].

F. Repeated Dose Toxicity

Oral

An OECD Guideline 422 (Combined Repeated Dose Toxicity Study with the Reproduction / Developmental Toxicity Screening Test) was performed in 2015. The rat is the preferred animal species for reproduction studies according to the various test guidelines and the Wistar strain was selected. This Wistar rat strain (Crl:WI[Han]) was selected since extensive historical control data were available for this strain.

Male and female Wistar rats were dosed with fatty acids tall oil ethoxylated (CAS RN 61791-00-2) by oral gavage with 0, 100, 300, 1000 milligrams per kilogram per day (mg/kg/day). The duration of treatment covered a 2-week premating period and mating in both sexes (mating pairs were from the same test group) as well as entire gestation and lactation period in females up to one day prior to the day of schedule sacrifice of the animals (a total of 28 days).

No clinical effects were observed, no mortality was observed, and body weight changes were not significantly different from controls. There were no treatment related changes in food consumption during the entire study. Water consumption was not affected. There were no haematological effects nor effects on clinical biochemistry parameters. An assessment of functional observation battery indicated no effects no test substance related deviations relative to motor activity were noted. Organ weights were not affected by exposure to the substance at any dose level. Gross pathological and histopathological findings did not indicate any adverse effects.

The no observed adverse effect level (NOAEL) for general systemic toxicity was determined to be 1,000 milligram per kilogram body weight per day (mg/kg bw/day) (ECHA) [KI. score =1].

Inhalation

There are no studies are available.

Dermal

There are no studies are available.

G. Genotoxicity

In vitro Studies

The key *in vitro* genotoxicity studies are presented in Table 2.

Table 2: *In vitro* Genotoxicity Studies on Fatty Acids Tall Oil Ethoxylated (CAS RN 61791-00-2)

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
Bacterial reverse mutation (*S. typhimurium* TA98, TA100, TA1535, TA1537 and E.coli WP2))	-	-	1	ECHA
Mammalian cell gene mutation (Chinese hamster lung fibroblasts (V 79) cells	-	-	1	ECHA

Test System	Results*		Klimisch Score	Reference
	-S9	+S9		
In vitro mammalian cell micronucleus test (human lymphocytes)	-	-	1	ECHA

*+, positive; -, negative

In vivo Studies

There are no studies were available.

H. Carcinogenicity

There are no studies are available.

I. Reproductive Toxicity

The reproductive toxicity potential of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) was evaluated in a combined repeated dose toxicity study with a reproductive/developmental toxicity screening test (OECD 422). Male and female Wistar rat strain (Crl:WI[Han]) rats were given oral gavage doses of 0, 100, 300, or 1,000 mg/kg-day. There was no indication of reproductive toxicity or any effects on tested endocrine system related parameters (T4 and TSH levels) at any dose level. The NOAEL for reproductive toxicity is 1,000 mg/kg bw/day, the highest dose tested (ECHA) [Kl. score = 1].

J. Developmental Toxicity

The developmental toxicity potential of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) was evaluated in a combined repeated dose toxicity study with a reproductive/developmental toxicity screening test (OECD 422). Male and female Wistar rat strain (Crl:WI[Han]) SD rats were given oral gavage doses of 0, 100, 300, or 1,000 mg/kg bw/day. There was no indication of teratogenic toxicity at any dose level. The NOAEL for developmental toxicity (systemic toxicity and fertility) is 1,000 mg/kg-day, the highest dose tested (ECHA) [Kl. score = 1].

V. DERIVATION OF TOXICOLOGICAL REFERENCE AND DRINKING WATER GUIDANCE VALUES

The toxicological reference values developed for tallow alkyl amines ethoxylated follow the methodology discussed in enHealth (2012). The approach used to develop drinking water guidance values is described in the Australian Drinking Water Guidelines (ADWG, 2011).

A. Non-Cancer

Oral

Under the conditions of a Combined Repeated Dose Toxicity Study with the Reproduction/Developmental Toxicity Screening Test, the oral administration by gavage of fatty acids tall oil ethoxylated (CAS RN 61791-00-2) to Wistar rats revealed no adverse signs of toxicity in male and female animals at a dose level of 1,000 mg/kg bw/day. Thus, the NOAEL for general systemic toxicity was 1,000 mg/kg bw/day, the highest dose tested, for male and female Wistar rats. The NOAEL of 1,000 mg/kg bw/day will be used for determining the oral reference dose (RfD) and the drinking water guidance value.

Oral Reference Dose (oral RfD)

Oral RfD = NOAEL / (UF_A x UF_H x UF_L x UF_{Sub} x UF_D)

Where:
UF_A (interspecies variability) = 10
UF_H (intraspecies variability) = 10
UF_L (LOAEL to NOAEL) = 1
UF_{Sub} (subchronic to chronic) = 3
UF_D (database uncertainty) = 1

Oral RfD = 1,000/(10 x 10 x 1 x 3 x 1) = 1,000/300 = 3.33 mg/kg-day

Drinking water guidance value
Drinking water guidance value = (animal dose) x (human weight) x (proportion of intake from water) / (volume of water consumed) x (safety factor)

Using the oral RfD,
Drinking water guidance value = (oral RfD) x (human weight) x (proportion of water consumed) / (volume of water consumed)

where:
Human weight = 70 kg (ADWG, 2011)
Proportion of water consumed = 10% (ADWG, 2011)
Volume of water consumed = 2L (ADWG, 2011)

Drinking water guidance value = (3.33 x 70 x 0.1)/2 = 11.65 mg/L

B. Cancer

There are no carcinogenicity studies are available for tallow alkyl amines ethoxylated. Thus, a cancer reference value was not derived for tallow alkyl amines ethoxylated.

VI. HUMAN HEALTH HAZARD ASSESSMENT OF PHYSICO-CHEMICAL PROPERTIES

The ethoxylated tallow alkyl amine does not exhibit the following physico-chemical properties:

- Explosivity
- Flammability
- Oxidising potential

VII. ENVIRONMENTAL HAZARD ASSESSMENT

A. Summary

Tallow alkyl amines ethoxylated are of low aquatic toxicity concern based on data from analogous substances.

B. Aquatic Toxicity

Table 3 lists the results of acute aquatic toxicity studies on of tallow alkyl amines ethoxylated.

Table 3: Acute Aquatic Toxicity Studies Tallow Alkyl Amines Ethoxylated *

Test Substance	Test Species	Endpoint	Results (mg/L) [WAF]	Kl. score
fatty acids, tall-oil, ethoxylated	*Danio rerio*	96-h LL_{50}	>100 (mortality)	1
fatty acids, tall-oil, ethoxylated	*Daphnia magna*	48-h EL_{50}	12.41 (mobility)	1
fatty acids, tall-oil, ethoxylated	*Pseudokirchnerella subcapitata*	72-h EL_{50}	39.7 (growth rate)	1

* Based on acute aquatic toxicity studies on fatty acids, tall-oil, ethoxylated (CAS RN 61791-00-2)
LL_{50} – median lethal loading rate
EL_{50} – median effective loading rate
The statistical methods used to determine LL_{50} and EL_{50} values are the same as those used to determine LC_{50}, EC_{50} and NOEC values.

All studies used the water accommodated fractions (WAFs) of the test substance.

Chronic Studies

Long-term toxicity data with fatty acids, tall-oil, ethoxylated (CAS RN 61791-00-2) are only available for algae. The algal test revealed the substance to be of low toxicity to algae (72h-EL_{10} = 7.08 mg/L) (ECHA) [Kl. score = 1].

C. Terrestrial Toxicity

There are no studies are available.

D. Calculation of Predicted No Effect Concentrations (PNECs)

The PNEC calculations for the substance follow the methodology discussed in DEWHA (2009).

PNEC Water

Experimental results are available for three trophic levels. Acute E (L)L_{50} values are available for fish (>100 mg/L), invertebrates (12.41 mg/L), and algae (39.7 mg/L). Chronic EL_{10} values are available for algae (7.08 mg/L). On the basis that the data consists of short-term studies from three trophic levels and chronic studies from one trophic level, an assessment factor of 100 has been applied to the lowest reported chronic value (E(L)L_{10} value of 7.08 mg/L for algae. The derived $PNEC_{water}$ for the substance is 0.071 mg/L.

PNEC Sediment

There are no toxicity data for sediment-dwelling organisms. Therefore, the $PNEC_{sed}$ was calculated using the equilibrium partitioning method. The $PNEC_{sed}$ is 3.6 mg/kg sediment wet weight.

The calculations are as follows:
$PNEC_{sed} = (K_{sed\text{-}water}/BD_{sed}) \times 1000 \times PNEC_{water}$
$= (65/1280) \times 1000 \times 0.71$
$= 3.61$ mg/kg

Where:
$K_{sed\text{-}water}$ = suspended matter-water partition coefficient (m^3/m^3)
BD_{sed} = bulk density of sediment (kg/m^3) = 1,280 [default]

$K_{sed\text{-}water} = 0.8 + [(0.2 \times Kp_{sed})/1000 \times BD_{solid}]$
$= 0.8 + [(0.2 \times 133/1000 \times 2400]$
$= 65\ m^3/m^3$

Where:
Kp_{sed} = solid-water partition coefficient (L/kg).
BD_{solid} = bulk density of the solid phase (kg/m^3) = 2,400 [default]

$Kp_{sed} = K_{oc} \times f_{oc}$
$= 3321 \times 0.04$
$= 133$ L/kg

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for read-across substance (CAS RN 61791-00-2) calculated from EPISUITE™ using the MCI method is 3321 L/kg .
F_{oc} = fraction of organic carbon in sediment = 0.04 [default].

PNEC Soil

There are no toxicity data for terrestrial or soil organisms. Therefore, the $PNEC_{soil}$ was calculated using the equilibrium partitioning method. The $PNEC_{soil}$ is 3.14 mg/kg soil dry weight.

The calculations are as follows:
$PNEC_{soil} = (Kp_{soil}/BD_{soil}) \times 1000 \times PNEC_{water}$
$=(66/1500) \times 1000 \times 0.071$
$= = 3.14$ mg/kg

Where:
Kp_{soil} = soil-water partition coefficient (m^3/m^3)
BD_{soil} = bulk density of soil (kg/m^3) = 1,500 [default]

$Kp_{soil} = K_{oc} \times f_{oc}$
$= 3321 \times 0.02$
$= 66.42\ m^3/m^3$

Where:
K_{oc} = organic carbon normalised distribution coefficient (L/kg). The K_{oc} for the read-across substance (CAS RN 61791-00-2) calculated from EPISUITE™ using the MCI method is 3321 L/kg.
F_{oc} = fraction of organic carbon in soil = 0.02 [default].

VIII. PERSISTENCE, BIOACCUMULATION AND TOXICITY (PBT) ASSESSMENT

The methodology for the Persistent, Bioaccumulative and Toxic (PBT) substances assessment is based on the Australian and EU Reach Criteria methodology (DEWHA, 2009; ECHA, 2008).

Tallow alkyl amines ethoxylated was noted to be readily biodegradable. Thus, the substance is not expected to meet the screening criteria for persistence.

Modelling of a representative structure indicates tallow alkyl amines ethoxylated does not have the potential to bioaccumulate. Thus, it does not meet the screening criteria for bioaccumulation.

Tallow alkyl amines ethoxylated did not exhibit substantial acute toxicity to fish, invertebrates, or algae. Thus, tallow alkyl amines ethoxylated is not expected to meet the screening criteria for toxicity.

The overall conclusion is that ethoxylated tallow alkyl amine is not a PBT substance.

IX. CLASSIFICATION AND LABELLING

A. Classification

H315-Skin Irrit. 2

H319-Eye Irrit. 2

H317-Skin Sens. 1B

B. Labelling

Warning

C. Pictograms

X. HANDLING AND SAFETY INFORMATION (OCCUPATIONAL LIMITS AND TRANSPORTATION REQUIREMENTS)

A. First Aid

Eye Contact

Immediately flush open eyes with plenty of water for at least 15 minutes. Remove contacts, if present and easy to do. If irritation occurs, get medical attention.

Skin Contact

Wash the contaminated area of with soap and water. Remove and isolate contaminated clothing. Launder contaminated clothing before reuse.

Inhalation

Move person to fresh air. If respiratory irritation, dizziness, nausea, or unconsciousness occurs, seek immediate medical assistance. Give artificial respiration if victim is not breathing.

Ingestion

Do not induce vomiting. Get medical attention immediately.

Notes to Physician

If ingested, material may be aspirated into the lungs and may cause chemical pneumonitis. Treat appropriately.

B. Fire Fighting Information

Extinguishing Media

Use water spray or fog, foam, dry chemical or carbon dioxide. Do not use straight streams of water.

Specific Exposure Hazards

May emit toxic fumes under fire conditions. Depending on conditions, decomposition products may include the following: carbon monoxide, carbon oxides.

Special Protective Equipment for Firefighters

Wear self-contained breathing apparatus and chemical-protective clothing.

C. Accidental Release Measures

Personal Precautions

Isolate area. Keep unnecessary and unprotected personnel from entering the area. Use personal protective clothing. Ensure adequate ventilation. Wear respiratory protection if ventilation is inadequate. Do not breath mist, vapours or spray Avoid contact with skin, eye, and clothing.

Environmental Precautions

Prevent from entering sewers, waterways, or low areas.

Steps to be Taken if Material is Released or Spilled

Pick up with non-combustible absorbent material and transfer to a container for chemical waste. For large amounts: dike spillage and pump off product into container for chemical waste. Dispose of contaminated material as prescribed.

D. Storage And Handling

General Handling

Avoid breathing vapour or aerosol. Keep away from open flames, hot surfaces and sources of ignition. Provide sufficient ventilation in work area.

Storage

Keep container tightly closed and in a dry, well-ventilated place.

E. Exposure Controls / Personal Protection

Occupational Exposure Standards

Workplace Australia has not established an occupational exposure standard for ethoxylated tallow alkyl amine.

Engineering Controls

Use adequate ventilation to control air-borne concentrations.

Personal Protection Equipment

Respiratory Protection: If workers are exposed to concentrations at a level that is not adequate to protect work health, they must use appropriate, certified respirators. The following type of respirator should be considered for this material: particulate, dust or mists. For high airborne concentrations, use an approved supplied-air respirator, operated in positive pressure mode.

Hand Protection: Use gloves chemically resistant to this material. Consult the safety data sheet for appropriate glove barrier materials.

Skin Protection: Use protective clothing chemically resistant to this material. Selection of specific items such as face shield, boots, apron, or full body suit will depend on the task.

Eye protection: Use chemical goggles.

Other Precautions: Wash hands, forearms, and face thoroughly after handling chemical products, before eating, smoking, and using the lavatory and at the end of the working period. Appropriate techniques should be used to remove potentially contaminated clothing. Wash contaminated clothing before reusing. Ensure that eyewash stations and safety showers are close to the workstation location.

F. Transport Information

Ethoxylated tallow alkyl amine) is not considered hazardous for purposes of transportation by road or rail. An Australian Dangerous Goods code is not required.

UN 1993

Class: 3

Packaging Group: II

XI. DISPOSAL MANAGEMENT

Disposal should be in accordance with all local, state, and federal regulations.

XII. REGULATORY STATUS

Australian AICS Inventory: Listed.

XIII. REFERENCES

ADWG. (2011). National Water Quality Management Strategy. Australian Drinking Water Guidelines, Section 6, Australian Government, National Health and Medical Research Council, Natural Resource Management Ministerial Council. Updated January 2022. Available: https://www.nhmrc.gov.au/about-us/publications/australian-drinking-water-guidelinesDepartment of the Environment, Water, Heritage and the Arts [DEWHA]. (2009). Environmental risk assessment guidance manual for industrial chemicals, Department of the Environment, Water, Heritage and the Arts, Commonwealth of Australia. Available: http://www.nepc.gov.au/resource/chemical-risk-assessment-guidance-manuals

ECHA. ECHA REACH database: http://echa.europa.eu/information-on-chemicals/registered-substances

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European Chemicals Agency [ECHA]. (2017). Guidance on Information Requirements and Chemical Safety Assessment, Chapter R11: PBT Assessment, European Chemicals Agency, Helsinki, Finland. Available: https://echa.europa.eu/guidance-documents/guidance-on-information-requirements-and-chemical-safety-assessment

Klimisch, H.J., Andreae, M., and Tillmann, U. (1997). A systematic approach for evaluating the quality of experimental and toxicological and ecotoxicological data. Regul. Toxicol. Pharmacol. 25:1-5.

NICNAS (1994). National Industrial Chemicals Notification and Assessment Scheme (NICNAS), AGPS, Canberra, Australia.

Appendix D Safety Data Sheets

SAFETY DATA SHEET

Revision date: 02-Dec-2021

Revision Number 1

1. IDENTIFICATION OF THE MATERIAL AND SUPPLIER

Product identifier

Product Name CF550KCB

Product Code(s) 000000069045

Other means of identification

Synonyms Manufactured exclusively for Condor Energy Services by Fusion Technologies (Australia) Pty Ltd

Recommended use of the chemical and restrictions on use

Recommended use Friction reducer.

Uses advised against No information available.

Supplier
Fusion Technologies Australia Pty Ltd
ABN: 50 636 538 960
Street Address: 7 Noble Street
Bridgeman Downs QLD 4035
Australia

Telephone number: +61 (0)460 047 656
Website: www.fusiontechinc.net

Emergency telephone number

Emergency telephone number **1800 033 111 (ALL HOURS)**

Please ensure you refer to the limitations of this Safety Data Sheet as set out in the "Other Information" section at the end of this Data Sheet.

2. HAZARDS IDENTIFICATION

GHS Classification

Classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS).

Not classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG)

Flammable liquids	Category 4
Acute toxicity - Oral	Category 4

SIGNAL WORD
Warning

Label elements

Exclamation mark

Hazard statements
H227 - Combustible liquid
H302 - Harmful if swallowed

Precautionary Statements - Prevention
Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking
Wear protective gloves / protective clothing / eye protection / face protection
Wash hands and face thoroughly after handling
Do not eat, drink or smoke when using this product
Precautionary Statements - Response
IF exposed: Get medical advice/attention if you feel unwell
IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell
Rinse mouth
In case of fire: Use extinguishing media as outlined in Section 5 of this Safety Data Sheet to extinguish.
Precautionary Statements - Storage
Store in a well-ventilated place
Precautionary Statements - Disposal
Dispose of contents/container in accordance with local, regional, national, and international regulations as applicable

Other hazards which do not result in classification

General Hazards Repeated exposure may cause skin dryness or cracking

Poisons Schedule (SUSMP) 5

3. COMPOSITION/INFORMATION ON INGREDIENTS

Chemical name	CAS No.	Weight-%
Polyacrylamide	9003-05-8	30-60%
Aliphatic hydrocarbons	-	30-60%
Glycol ether derivative	-	<5%
Organophillic silicate	-	<5%

4. FIRST AID MEASURES

Description of first aid measures

Emergency telephone number Poisons Information Center, Australia: 13 11 26
Poisons Information Center, New Zealand: 0800 764 766

Inhalation Remove to fresh air and keep at rest in a position comfortable for breathing. If exposed or concerned: Get medical advice/attention.

Eye contact In case of eye contact, remove contact lens and rinse immediately with plenty of water, also under the eyelids, for at least 15 minutes. If symptoms persist, call a physician.

Skin contact Wash off immediately with soap and plenty of water. If skin irritation persists, call a physician. Take off contaminated clothing and wash before reuse.

Ingestion Rinse mouth. Drink 1 or 2 glasses of water. Get medical attention.

Most important symptoms and effects, both acute and delayed

Symptoms No information available.

Indication of any immediate medical attention and special treatment needed

Note to physicians Treat symptomatically. Material swells on contact with water.

5. FIRE FIGHTING MEASURES

Suitable Extinguishing Media

Suitable Extinguishing Media Use extinguishing measures that are appropriate to local circumstances and the surrounding environment. Dry chemical, CO2, sand, earth, water spray or regular foam.

Unsuitable extinguishing media High volume water jet.

Specific hazards arising from the chemical

Specific hazards arising from the chemical Extremely slippery when spilled. Combustible liquid.

Hazardous combustion products Carbon oxides. Nitrogen oxides.

Special protective actions for fire-fighters

Special protective equipment for fire-fighters Firefighters should wear self-contained breathing apparatus and full firefighting turnout gear. Use personal protection equipment.

6. ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Personal precautions Ensure adequate ventilation. Remove all sources of ignition. Special danger of slipping by leaking/spilling product.

For emergency responders Use personal protection recommended in Section 8.

Environmental precautions

Environmental precautions See Section 12 for additional Ecological Information.

Methods and material for containment and cleaning up

Methods for containment Prevent further leakage or spillage if safe to do so. Dike far ahead of spill to collect runoff water. Contain and collect spillage with non-combustible absorbent material, (e.g. sand, earth, diatomaceous earth, vermiculite) and place in container for disposal according to

local / national regulations (see Section 13).

Methods for cleaning up Collect in properly labelled drums or other suitable containers, with loose fitting lids. Use clean non-sparking tools to collect absorbed material. Prevent product from entering drains. After cleaning, flush away traces with water and detergent.

7. HANDLING AND STORAGE

Precautions for safe handling

Advice on safe handling Handle in accordance with good industrial hygiene and safety practice. Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking. Use grounding and bonding connection when transferring this material to prevent static discharge, fire or explosion.

General hygiene considerations Avoid contact with skin, eyes, and clothing.

Conditions for safe storage, including any incompatibilities

Storage Conditions Keep containers tightly closed in a dry, cool and well-ventilated place.

Incompatible materials Strong oxidizing agents.

Poisons Schedule (SUSMP) 5

8. EXPOSURE CONTROLS/PERSONAL PROTECTION

Control parameters

Exposure Limits No value assigned for this specific material by Safe Work Australia. However, supplier recommended Workplace Exposure Standard(s) for constituent(s):

Chemical name	Australia	ACGIH TLV
Aliphatic hydrocarbons		TWA: 200 mg/m³, Sk (as total hydrocarbon vapour)

As published by Safe Work Australia Workplace Exposure Standards for Airborne Contaminants.

TWA - The time-weighted average airborne concentration of a particular substance when calculated over an eight-hour working day, for a five-day working week.

`Sk' (skin) Notice - absorption through the skin may be a significant source of exposure. The exposure standard is invalidated if such contact should occur.

These Workplace Exposure Standards are guides to be used in the control of occupational health hazards. All atmospheric contamination should be kept to as low a level as is workable. These workplace exposure standards should not be used as fine dividing lines between safe and dangerous concentrations of chemicals. They are not a measure of relative toxicity.

Appropriate engineering controls

Engineering controls Apply technical measures to comply with the occupational exposure limits.

If in the handling and application of this material, safe exposure levels could be exceeded, the use of engineering controls such as local exhaust ventilation must be considered and

the results documented. If achieving safe exposure levels does not require engineering controls, then a detailed and documented risk assessment using the relevant Personal Protective Equipment (PPE) (refer to PPE section below) as a basis must be carried out to determine the minimum PPE requirements.

Individual protection measures, such as personal protective equipment

The selection of PPE is dependent on a detailed risk assessment. The risk assessment should consider the work situation, the physical form of the chemical, the handling methods, and environmental factors.

OVERALLS, SAFETY SHOES, CHEMICAL GOGGLES, GLOVES.

Eye/face protection	Wear safety glasses with side shields (or goggles).
Skin and body protection	Wear suitable protective clothing.
Hand protection	Impervious gloves.
Respiratory protection	No protective equipment is needed under normal use conditions. If exposure limits are exceeded or irritation is experienced, ventilation and evacuation may be required. If determined by a risk assessment an inhalation risk exists, wear an organic vapour respirator meeting the requirements of AS/NZS 1715 and AS/NZS 1716.
Environmental exposure controls	Local authorities should be advised if significant spillages cannot be contained.

9. PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Physical state	Liquid
Appearance	Slurry
Color	Light brown
Odor	Hydrocarbon
Odor threshold	No information available.

Property	**Values**	**Remarks • Method**
pH	6.0 - 8.0	None known
pH (as aqueous solution)	No data available	None known
Melting point / freezing point	-5°C	None known
Boiling point / boiling range	No data available	None known
Flash point	75.5°C	Pensky-Martens Closed Cup (PMCC)
Evaporation rate	No data available	None known
Flammability (solid, gas)	No data available	None known
Flammability Limit in Air		None known
Upper flammability or explosive limits	No data available	
Lower flammability or explosive limits	No data available	
Vapor pressure	No data available	None known
Vapor density	No data available	None known
Relative density	1.1	None known
Water solubility	Dispersible	None known
Solubility(ies)	No data available	None known

Partition coefficient	No data available	None known
Autoignition temperature	No data available	None known
Decomposition temperature	No data available	None known
Kinematic viscosity	No data available	None known
Dynamic viscosity	No data available	None known

Other information

10. STABILITY AND REACTIVITY

Reactivity

Reactivity No information available.

Chemical stability

Stability Stable under normal conditions.

Explosion data

Sensitivity to mechanical impact None.

Sensitivity to static discharge None.

Possibility of hazardous reactions

Possibility of hazardous reactions None under normal processing.

Conditions to avoid

Conditions to avoid Heat, flames and sparks.

Incompatible materials

Incompatible materials Strong oxidizing agents.

Hazardous decomposition products

Hazardous decomposition products Carbon oxides. Nitrogen oxides. Ammonia.

11. TOXICOLOGICAL INFORMATION

Acute toxicity

Information on likely routes of exposure

Product Information No adverse health effects expected if the chemical is handled in accordance with this Safety Data Sheet and the chemical label. Symptoms or effects that may arise if the chemical is mishandled and overexposure occurs are:

Inhalation May cause central nervous system depression with nausea, headache, dizziness, vomiting, and incoordination.

Eye contact May cause irritation.

Skin contact May cause irritation. Repeated exposure may cause skin dryness or cracking.

Ingestion Ingestion may cause gastrointestinal irritation, nausea, vomiting and diarrhoea. Product swells when exposed to moisture and may cause choking if large quantities are involved.

Symptoms No information available.

Numerical measures of toxicity - Product Information

No information available.

Numerical measures of toxicity - Component Information

Component Information

Chemical name	Oral LD50	Dermal LD50	Inhalation LC50
Polyacrylamide	> 1 g/kg (Rat)	-	-
Aliphatic hydrocarbons	> 5000 mg/kg (Rat)	> 2000 mg/kg (Rabbit)	-
Glycol ether derivative	= 1310 mg/kg (Rat)	-	-

See section 16 for terms and abbreviations

Delayed and immediate effects as well as chronic effects from short and long-term exposure

Skin corrosion/irritation May cause skin irritation. Classification based on data available for ingredients.

Serious eye damage/eye irritation May cause slight irritation. Classification based on data available for ingredients.

Respiratory or skin sensitization No information available.

Germ cell mutagenicity No information available.

Carcinogenicity The table below indicates whether each agency has listed any ingredient as a carcinogen.

Chemical name	Australia
Organophillic silicate -	Carc. 1A

Reproductive toxicity No information available.

STOT - single exposure No information available.

STOT - repeated exposure No information available.

Aspiration hazard No information available.

12. ECOLOGICAL INFORMATION

Ecotoxicity

Ecotoxicity The environmental impact of this product has not been fully investigated.

Persistence and degradability

Persistence and degradability For the major component: Expected to be biodegradable.

Bioaccumulative potential

Bioaccumulation No information available.

Mobility

Mobility in soil No information available.

Other adverse effects

13. DISPOSAL CONSIDERATIONS

Waste treatment methods

Waste from residues/unused products Dispose of in accordance with local regulations. Dispose of waste in accordance with environmental legislation.

Contaminated packaging Dispose of contents/containers in accordance with local regulations.

14. TRANSPORT INFORMATION

ADG
Not classified as Dangerous Goods by the criteria of the Australian Dangerous Goods Code (ADG Code) for transport by Road and Rail; NON-DANGEROUS GOODS.

IATA
Not classified as Dangerous Goods by the criteria of the International Air Transport Association (IATA) Dangerous Goods Regulations for transport by air; NON-DANGEROUS GOODS.

IMDG
Not classified as Dangerous Goods by the criteria of the International Maritime Dangerous Goods Code (IMDG Code) for transport by sea; NON-DANGEROUS GOODS.

15. REGULATORY INFORMATION

Safety, health and environmental regulations/legislation specific for the substance or mixture

National regulations

Australia
Classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS).

Not classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG)

See section 8 for national exposure control parameters

Poisons Schedule (SUSMP) 5

International Inventories

AIIC	All the constituents of this material are listed on the Australian Inventory of Industrial Chemicals.
NZIoC	All the constituents of this material are listed on the New Zealand Inventory of Chemicals.

Legend:
- Australian Inventory of Industrial Chemicals

International Regulations

The Montreal Protocol on Substances that Deplete the Ozone Layer Not applicable

The Stockholm Convention on Persistent Organic Pollutants Not applicable

The Rotterdam Convention Not applicable

16. OTHER INFORMATION

Supplier Safety Data Sheet

Reason(s) For Issue: First Issue Primary SDS

Issuing Date: 02-Dec-2021

This Safety Data Sheet has been prepared by Ixom Operations Pty Ltd (Toxicology and SDS Services).

Revision Note:
The symbol (*) in the margin of this SDS indicates that this line has been revised.

Key or legend to abbreviations and acronyms used in the safety data sheet

Legend Section 8: EXPOSURE CONTROLS/PERSONAL PROTECTION

TWA	TWA (time-weighted average)	STEL	STEL (Short Term Exposure Limit)
Ceiling	Maximum limit value	*	Skin designation
C	Carcinogen		

Key literature references and sources for data used to compile the SDS

EPA (Environmental Protection Agency)
Acute Exposure Guideline Level(s) (AEGL(s))
U.S. Environmental Protection Agency Federal Insecticide, Fungicide, and Rodenticide Act
U.S. Environmental Protection Agency High Production Volume Chemicals
Food Research Journal
Hazardous Substance Database
International Uniform Chemical Information Database (IUCLID)
Japan GHS Classification
Australian Industrial Chemicals Introduction Scheme (AICIS)
NIOSH (National Institute for Occupational Safety and Health)
National Library of Medicine's ChemID Plus (NLM CIP)
National Library of Medicine's PubMed database (NLM PUBMED)
National Toxicology Program (NTP)
New Zealand's Chemical Classification and Information Database (CCID)
Organization for Economic Co-operation and Development Environment, Health, and Safety Publications
Organization for Economic Co-operation and Development High Production Volume Chemicals Program
Organization for Economic Co-operation and Development Screening Information Data Set
RTECS (Registry of Toxic Effects of Chemical Substances)
World Health Organization

Disclaimer

This SDS summarises to our best knowledge at the date of issue, the chemical health and safety hazards of the material and general guidance on how to safely handle the material in the workplace. Since The Supplier cannot anticipate or control the conditions under which the product may be used, each user must, prior to usage, assess and control the risks arising from its use of the material.

If clarification or further information is needed, the user should contact their Supplier representative or The Supplier at the contact details on page 1.

The Supplier's responsibility for the material as sold is subject to the terms and conditions of sale, a copy of which is

available upon request.

End of Safety Data Sheet

SAFETY DATA SHEET

Revision date: 12-Nov-2021

Revision Number 1

1. IDENTIFICATION OF THE MATERIAL AND SUPPLIER

Product identifier

Product Name CF10GGC

Product Code(s) 000000069033

Other means of identification

Synonyms Manufactured by Condor Energy Services Ltd

Recommended use of the chemical and restrictions on use

Recommended use Completion fluid.

Uses advised against No information available.

Supplier
Condor Energy Services Ltd
ABN: 35 153 250 670
Brisbane Head Office: Level 11, 333 Ann Street
Brisbane QLD 4000
Australia

Telephone number: 07 3999 9044
Website: www.CondorEnergy.com.au

Emergency telephone number

Emergency telephone number **1800 033 111 (ALL HOURS)**

Please ensure you refer to the limitations of this Safety Data Sheet as set out in the "Other Information" section at the end of this Data Sheet.

2. HAZARDS IDENTIFICATION

GHS Classification

Classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS).

Not classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG)

Flammable liquids	Category 4

Acute toxicity - Oral	Category 4
Skin corrosion/irritation	Category 2
Serious eye damage/eye irritation	Category 2A

SIGNAL WORD
Warning

Label elements

Exclamation mark

Hazard statements
H227 - Combustible liquid
H302 - Harmful if swallowed
H315 - Causes skin irritation
H319 - Causes serious eye irritation

Precautionary Statements - Prevention
Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking
Wear protective gloves / protective clothing / eye protection / face protection
Wash hands and face thoroughly after handling
Do not eat, drink or smoke when using this product
IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing
If eye irritation persists: Get medical advice/attention
IF ON SKIN: Wash with plenty of soap and water
If skin irritation occurs: Get medical advice/attention
Take off immediately all contaminated clothing and wash it before reuse
IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell
Rinse mouth
In case of fire: Use extinguishing media as outlined in Section 5 of this Safety Data Sheet to extinguish.
Precautionary Statements - Storage
Store in a well-ventilated place
Precautionary Statements - Disposal
Dispose of contents/container in accordance with local, regional, national, and international regulations as applicable

Other hazards which do not result in classification
May be harmful in contact with skin
May be harmful if swallowed and enters airways
Combustible liquid

Poisons Schedule (SUSMP) 5

3. COMPOSITION/INFORMATION ON INGREDIENTS

Chemical name	CAS No.	Weight-%
Guar gum	9000-30-0	30-60%
Aliphatic hydrocarbons	-	30-60%
Glycol ether derivative	-	< 10%
Organophillic silicate	-	< 5%

4. FIRST AID MEASURES

Description of first aid measures

Emergency telephone number	Poisons Information Center, Australia: 13 11 26 Poisons Information Center, New Zealand: 0800 764 766
Inhalation	Remove to fresh air and keep at rest in a position comfortable for breathing. If exposed or concerned: Get medical advice/attention.
Eye contact	In case of eye contact, remove contact lens and rinse immediately with plenty of water, also under the eyelids, for at least 15 minutes. If symptoms persist, call a physician.
Skin contact	Wash off immediately with soap and plenty of water. If skin irritation persists, call a physician. Take off contaminated clothing and wash before reuse.
Ingestion	Rinse mouth. Drink 1 or 2 glasses of water. Get medical attention.

Most important symptoms and effects, both acute and delayed

Symptoms	No information available.

Indication of any immediate medical attention and special treatment needed

Note to physicians	Treat symptomatically.

5. FIRE FIGHTING MEASURES

Suitable Extinguishing Media

Suitable Extinguishing Media	Use extinguishing measures that are appropriate to local circumstances and the surrounding environment. Dry chemical, CO2, sand, earth, water spray or regular foam.
Unsuitable extinguishing media	High volume water jet.

Specific hazards arising from the chemical

Specific hazards arising from the chemical	Extremely slippery when spilled. Combustible liquid.
Hazardous combustion products	Carbon oxides.

Special protective actions for fire-fighters

Special protective equipment for fire-fighters	Firefighters should wear self-contained breathing apparatus and full firefighting turnout gear. Use personal protection equipment.

6. ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Personal precautions	Ensure adequate ventilation. Remove all sources of ignition. Special danger of slipping by leaking/spilling product.
For emergency responders	Use personal protection recommended in Section 8.

Environmental precautions

Environmental precautions See Section 12 for additional Ecological Information.

Methods and material for containment and cleaning up

Methods for containment Contain and collect spillage with non-combustible absorbent material, (e.g. sand, earth, diatomaceous earth, vermiculite) and place in container for disposal according to local / national regulations (see Section 13).

Methods for cleaning up After cleaning, flush away traces with water and detergent. Collect in properly labelled drums or other suitable containers, with loose fitting lids. Use clean non-sparking tools to collect absorbed material.

7. HANDLING AND STORAGE

Precautions for safe handling

Advice on safe handling Handle in accordance with good industrial hygiene and safety practice. Avoid contact with skin, eyes, and clothing. Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking. Use grounding and bonding connection when transferring this material to prevent static discharge, fire or explosion.

Conditions for safe storage, including any incompatibilities

Storage Conditions Keep containers tightly closed in a dry, cool and well-ventilated place.

Incompatible materials Strong oxidizing agents.

Poisons Schedule (SUSMP) 5

8. EXPOSURE CONTROLS/PERSONAL PROTECTION

Control parameters

Exposure Limits No value assigned for this specific material by Safe Work Australia. However, supplier recommended Workplace Exposure Standard(s) for constituent(s):

Chemical name	Australia	ACGIH TLV
Aliphatic hydrocarbons		TWA: 200 mg/m³, Sk (as total hydrocarbon vapour)

As published by Safe Work Australia Workplace Exposure Standards for Airborne Contaminants.

TWA - The time-weighted average airborne concentration of a particular substance when calculated over an eight-hour working day, for a five-day working week.

`Sk' (skin) Notice - absorption through the skin may be a significant source of exposure. The exposure standard is invalidated if such contact should occur.

These Workplace Exposure Standards are guides to be used in the control of occupational health hazards. All atmospheric contamination should be kept to as low a level as is workable. These workplace exposure standards should not be used as fine dividing lines between safe and dangerous concentrations of chemicals. They are not a measure of relative toxicity.

Appropriate engineering controls

Engineering controls Apply technical measures to comply with the occupational exposure limits.

If in the handling and application of this material, safe exposure levels could be exceeded, the use of engineering controls such as local exhaust ventilation must be considered and the results documented. If achieving safe exposure levels does not require engineering controls, then a detailed and documented risk assessment using the relevant Personal Protective Equipment (PPE) (refer to PPE section below) as a basis must be carried out to determine the minimum PPE requirements.

Individual protection measures, such as personal protective equipment

The selection of PPE is dependent on a detailed risk assessment. The risk assessment should consider the work situation, the physical form of the chemical, the handling methods, and environmental factors.

OVERALLS, SAFETY SHOES, CHEMICAL GOGGLES, GLOVES.

Eye/face protection Wear safety glasses with side shields (or goggles).

Skin and body protection Wear suitable protective clothing.

Hand protection Impervious gloves.

Respiratory protection No protective equipment is needed under normal use conditions. If exposure limits are exceeded or irritation is experienced, ventilation and evacuation may be required. If determined by a risk assessment an inhalation risk exists, wear an organic vapour respirator meeting the requirements of AS/NZS 1715 and AS/NZS 1716.

Environmental exposure controls Local authorities should be advised if significant spillages cannot be contained.

9. PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Physical state	Liquid
Appearance	Slurry
Color	Light brown
Odor	Mild Hydrocarbon
Odor threshold	No information available.

Property	**Values**	**Remarks • Method**
pH	No data available	None known
pH (as aqueous solution)	No data available	None known
Melting point / freezing point	No data available	None known
Boiling point / boiling range	No data available	None known
Flash point	76.7°C	Pensky-Martens Closed Cup (PMCC)
Evaporation rate	No data available	None known
Flammability (solid, gas)	No data available	None known
Flammability Limit in Air		None known
Upper flammability or explosive limits	No data available	
Lower flammability or explosive	No data available	

limits		
Vapor pressure	No data available	None known
Vapor density	No data available	None known
Relative density	1.02 - 1.09	
Water solubility	Emulsifiable	
Solubility(ies)	No data available	None known
Partition coefficient	No data available	None known
Autoignition temperature	No data available	None known
Decomposition temperature	No data available	None known
Kinematic viscosity	28 mm2/s	
Dynamic viscosity	No data available	None known

Other information

10. STABILITY AND REACTIVITY

Reactivity

Reactivity No information available.

Chemical stability

Stability Stable under normal conditions.

Explosion data

Sensitivity to mechanical impact None.

Sensitivity to static discharge None.

Possibility of hazardous reactions

Possibility of hazardous reactions None under normal processing.

Conditions to avoid

Conditions to avoid Heat, flames and sparks.

Incompatible materials

Incompatible materials Strong oxidizing agents.

Hazardous decomposition products

Hazardous decomposition products None known based on information supplied.

11. TOXICOLOGICAL INFORMATION

Acute toxicity

Information on likely routes of exposure

Product Information No adverse health effects expected if the chemical is handled in accordance with this Safety Data Sheet and the chemical label. Symptoms or effects that may arise if the chemical is mishandled and overexposure occurs are:

Inhalation May cause central nervous system depression with nausea, headache, dizziness, vomiting, and incoordination.

Eye contact Causes serious eye irritation.

Skin contact Causes skin irritation. Repeated exposure may cause skin dryness or cracking. Repeated or prolonged skin contact may cause allergic reactions with susceptible persons.

Ingestion Ingestion may cause gastrointestinal irritation, nausea, vomiting and diarrhoea.

Symptoms No information available.

Numerical measures of toxicity - Product Information

The following values are calculated based on chapter 3.1 of the GHS document

ATEmix (oral) 4,967.20
ATEmix (dermal) 4,448.90

Component Information

Chemical name	Oral LD50	Dermal LD50	Inhalation LC50
Guar gum	= 6770 mg/kg (Rat)	-	-
Aliphatic hydrocarbons	> 5000 mg/kg (Rat)	> 2000 mg/kg (Rabbit)	-
Glycol ether derivative	= 1310 mg/kg (Rat)	-	-

See section 16 for terms and abbreviations

Delayed and immediate effects as well as chronic effects from short and long-term exposure

Skin corrosion/irritation Irritating to skin. Classification based on data available for ingredients.

Serious eye damage/eye irritation Causes serious eye irritation. Classification based on data available for ingredients.

Respiratory or skin sensitization No information available.

Germ cell mutagenicity No information available.

Carcinogenicity The table below indicates whether each agency has listed any ingredient as a carcinogen.

Chemical name	Australia
Organophillic silicate -	Carc. 1A

Reproductive toxicity No information available.

STOT - single exposure No information available.

STOT - repeated exposure No information available.

Aspiration hazard No information available.

12. ECOLOGICAL INFORMATION

Ecotoxicity

Ecotoxicity The environmental impact of this product has not been fully investigated.

Persistence and degradability

Persistence and degradability For the major component: Biodegradable.

Bioaccumulative potential

Bioaccumulation No information available.

Mobility

Mobility in soil No information available.

Other adverse effects

13. DISPOSAL CONSIDERATIONS

Waste treatment methods

Waste from residues/unused products Dispose of in accordance with local regulations. Dispose of waste in accordance with environmental legislation.

Contaminated packaging Dispose of contents/containers in accordance with local regulations.

14. TRANSPORT INFORMATION

ADG
Not classified as Dangerous Goods by the criteria of the Australian Dangerous Goods Code (ADG Code) for transport by Road and Rail; NON-DANGEROUS GOODS.

IATA
Not classified as Dangerous Goods by the criteria of the International Air Transport Association (IATA) Dangerous Goods Regulations for transport by air; NON-DANGEROUS GOODS. Not regulated

IMDG
Not classified as Dangerous Goods by the criteria of the International Maritime Dangerous Goods Code (IMDG Code) for transport by sea; NON-DANGEROUS GOODS. Not regulated

15. REGULATORY INFORMATION

Safety, health and environmental regulations/legislation specific for the substance or mixture

National regulations

Australia
Classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS).

Not classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG)

See section 8 for national exposure control parameters

Poisons Schedule (SUSMP) 5

International Inventories

AIIC	All the constituents of this material are listed on the Australian Inventory of Industrial Chemicals.
NZIoC	All the constituents of this material are listed on the New Zealand Inventory of Chemicals.

Legend:
- Australian Inventory of Industrial Chemicals

International Regulations

The Montreal Protocol on Substances that Deplete the Ozone Layer Not applicable

The Stockholm Convention on Persistent Organic Pollutants Not applicable

The Rotterdam Convention Not applicable

16. OTHER INFORMATION

Supplier Safety Data Sheet

Reason(s) For Issue: First Issue Primary SDS

Issuing Date: 11-Nov-2021

This Safety Data Sheet has been prepared by Ixom Operations Pty Ltd (Toxicology and SDS Services).

Revision Note:
The symbol (*) in the margin of this SDS indicates that this line has been revised.

Key or legend to abbreviations and acronyms used in the safety data sheet

Legend Section 8: EXPOSURE CONTROLS/PERSONAL PROTECTION

TWA	TWA (time-weighted average)	STEL	STEL (Short Term Exposure Limit)
Ceiling	Maximum limit value	*	Skin designation
C	Carcinogen		

Key literature references and sources for data used to compile the SDS

EPA (Environmental Protection Agency)
Acute Exposure Guideline Level(s) (AEGL(s))
U.S. Environmental Protection Agency Federal Insecticide, Fungicide, and Rodenticide Act
U.S. Environmental Protection Agency High Production Volume Chemicals
Food Research Journal
Hazardous Substance Database
International Uniform Chemical Information Database (IUCLID)
Japan GHS Classification
Australian Industrial Chemicals Introduction Scheme (AICIS)
NIOSH (National Institute for Occupational Safety and Health)
National Library of Medicine's ChemID Plus (NLM CIP)
National Library of Medicine's PubMed database (NLM PUBMED)
National Toxicology Program (NTP)
New Zealand's Chemical Classification and Information Database (CCID)
Organization for Economic Co-operation and Development Environment, Health, and Safety Publications
Organization for Economic Co-operation and Development High Production Volume Chemicals Program
Organization for Economic Co-operation and Development Screening Information Data Set
RTECS (Registry of Toxic Effects of Chemical Substances)
World Health Organization

Disclaimer
The information provided in this Safety Data Sheet is correct to the best of our knowledge, information and belief at the

date of its publication. The information given is designed only as a guidance for safe handling, use, processing, storage, transportation, disposal and release and is not to be considered a warranty or quality specification. The information relates only to the specific material designated and may not be valid for such material used in combination with any other materials or in any process, unless specified in the text

End of Safety Data Sheet

SAFETY DATA SHEET

Revision date: 30-Nov-2021

Revision Number 1

1. IDENTIFICATION OF THE MATERIAL AND SUPPLIER

Product identifier

Product Name CF150FBS

Product Code(s) 000000069041

Other means of identification

Synonyms Manufactured exclusively for Condor Energy Services by Fusion Technologies (Australia) Pty Ltd

Recommended use of the chemical and restrictions on use

Recommended use Hydraulic fracturing additive.

Uses advised against No information available.

Supplier
Fusion Technologies Australia Pty Ltd
ABN: 50 636 538 960
Street Address: 7 Noble Street
Bridgeman Downs QLD 4035
Australia

Telephone number: +61 (0)460 047 656
Website: www.fusiontechinc.net

Emergency telephone number

Emergency telephone number **1800 033 111 (ALL HOURS)**

Please ensure you refer to the limitations of this Safety Data Sheet as set out in the "Other Information" section at the end of this Data Sheet.

2. HAZARDS IDENTIFICATION

GHS Classification

Classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS).

Not classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG)

Acute toxicity - Oral	Category 4
Skin corrosion/irritation	Category 2
Serious eye damage/eye irritation	Category 1
Specific target organ toxicity (single exposure)	Category 3
Specific target organ toxicity (repeated exposure)	Category 2
Acute aquatic toxicity	Category 2
Chronic aquatic toxicity	Category 3

SIGNAL WORD
Warning

Label elements

Corrosion
Health hazard
Exclamation mark

Hazard statements
H302 - Harmful if swallowed
H315 - Causes skin irritation
H318 - Causes serious eye damage
H371 - May cause damage to kidneys if swallowed
H373 - May cause damage to organs through prolonged or repeated exposure if swallowed
H401 - Toxic to aquatic life
H412 - Harmful to aquatic life with long lasting effects

Precautionary Statements - Prevention
Wash face, hands and any exposed skin thoroughly after handling
Do not eat, drink or smoke when using this product
Wear protective gloves/eye protection/face protection
Do not breathe mist, vapours, spray.
Avoid release to the environment
Precautionary Statements - Response
IF exposed or concerned: Call a POISON CENTER or doctor if you feel unwell
IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing
Immediately call a POISON CENTER or doctor
IF ON SKIN: Wash with plenty of soap and water
If skin irritation occurs: Get medical advice/attention
Take off contaminated clothing and wash it before reuse
IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell
Rinse mouth
Precautionary Statements - Storage
No storage statements
Precautionary Statements - Disposal
Dispose of contents/container in accordance with local, regional, national, and international regulations as applicable

Other hazards which do not result in classification
Poisons Schedule (SUSMP) 6

3. COMPOSITION/INFORMATION ON INGREDIENTS

Chemical name	CAS No.	Weight-%
Ethylene glycol	107-21-1	10-30%

Nonionic surfactant	-	10-30%
Anionic surfactant	-	10-30%
Non-hazardous ingredients	Proprietary	Balance

4. FIRST AID MEASURES

Description of first aid measures

General advice If swallowed, seek medical advice immediately and show this container or label.

Emergency telephone number Poisons Information Center, Australia: 13 11 26
Poisons Information Center, New Zealand: 0800 764 766

Inhalation Remove to fresh air and keep at rest in a position comfortable for breathing. Call a physician if symptoms occur.

Eye contact Rinse immediately with plenty of water, also under the eyelids, for at least 15 minutes. Keep eye wide open while rinsing. Remove contact lenses, if present and easy to do. Continue rinsing. Get immediate medical advice/attention.

Skin contact Wash off immediately with soap and plenty of water. Get medical attention if irritation develops and persists. Take off contaminated clothing and wash before reuse.

Ingestion Rinse mouth immediately and drink plenty of water. Get medical attention. Do NOT induce vomiting.

Self-protection of the first aider Avoid breathing vapors or mists. Avoid contact with skin, eyes, and clothing. See section 8 for more information.

Most important symptoms and effects, both acute and delayed

Symptoms No information available.

Indication of any immediate medical attention and special treatment needed

Note to physicians Treat symptomatically.

5. FIRE FIGHTING MEASURES

Suitable Extinguishing Media

Suitable Extinguishing Media Dry chemical, CO2, alcohol-resistant foam or water spray.

Unsuitable extinguishing media Do not use a solid water stream as it may scatter and spread fire.

Specific hazards arising from the chemical

Specific hazards arising from the chemical No information available.

Hazardous combustion products Carbon oxides. Oxides of sulfur.

Special protective actions for fire-fighters

Special protective equipment for fire-fighters Firefighters should wear self-contained breathing apparatus and full firefighting turnout gear. Use personal protection equipment.

6. ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Personal precautions Ensure adequate ventilation. Avoid breathing vapors or mists. Avoid contact with skin, eyes, and clothing. Do not touch or walk through spilled material. Extremely slippery when spilled.

For emergency responders Use personal protection recommended in Section 8.

Environmental precautions

Environmental precautions Keep out of drains, sewers, ditches and waterways. Local authorities should be advised if significant spillages cannot be contained.

Methods and material for containment and cleaning up

Methods for containment Prevent further leakage or spillage if safe to do so. Dike to collect large liquid spills. Contain and collect spillage with non-combustible absorbent material, (e.g. sand, earth, diatomaceous earth, vermiculite) and place in container for disposal according to local / national regulations (see Section 13).

Methods for cleaning up Avoid breathing dust or spray mist. Soak up with inert absorbent material (e.g. sand, silica gel, acid binder, universal binder, sawdust). Collect in properly labelled drums or other suitable containers, with loose fitting lids. After cleaning, flush away traces with water and detergent.

7. HANDLING AND STORAGE

Precautions for safe handling

Advice on safe handling Handle in accordance with good industrial hygiene and safety practice. Solutions extremely slippery when spilled.

General hygiene considerations Avoid breathing vapors or mists. Avoid contact with skin, eyes, and clothing. Do not eat, drink or smoke when using this product. Wear suitable gloves and eye/face protection. Remove and wash contaminated clothing and gloves, including the inside, before re-use.

Conditions for safe storage, including any incompatibilities

Storage Conditions Keep containers tightly closed in a dry, cool and well-ventilated place.

Incompatible materials Strong oxidizing agents.

Poisons Schedule (SUSMP) 6

8. EXPOSURE CONTROLS/PERSONAL PROTECTION

Control parameters

Exposure Limits No value assigned for this specific material by Safe Work Australia. However, Workplace Exposure Standard(s) for constituent(s):

Ethylene glycol (vapour): 8hr TWA = 52 mg/m^3 (20 ppm), 15 min STEL = 104 mg/m^3 (40 ppm), Sk

As published by Safe Work Australia Workplace Exposure Standards for Airborne Contaminants.

TWA - The time-weighted average airborne concentration of a particular substance when calculated over an eight-hour working day, for a five-day working week.

STEL (Short Term Exposure Limit) - the airborne concentration of a particular substance calculated as a time-weighted average over 15 minutes, which should not be exceeded at any time during a normal eight hour work day. According to current knowledge this concentration should neither impair the health of, nor cause undue discomfort to, nearly all workers.

`Sk' (skin) Notice - absorption through the skin may be a significant source of exposure. The exposure standard is invalidated if such contact should occur.

These Workplace Exposure Standards are guides to be used in the control of occupational health hazards. All atmospheric contamination should be kept to as low a level as is workable. These workplace exposure standards should not be used as fine dividing lines between safe and dangerous concentrations of chemicals. They are not a measure of relative toxicity.

Appropriate engineering controls

Engineering controls Apply technical measures to comply with the occupational exposure limits.

If in the handling and application of this material, safe exposure levels could be exceeded, the use of engineering controls such as local exhaust ventilation must be considered and the results documented. If achieving safe exposure levels does not require engineering controls, then a detailed and documented risk assessment using the relevant Personal Protective Equipment (PPE) (refer to PPE section below) as a basis must be carried out to determine the minimum PPE requirements.

Individual protection measures, such as personal protective equipment

The selection of PPE is dependent on a detailed risk assessment. The risk assessment should consider the work situation, the physical form of the chemical, the handling methods, and environmental factors.

OVERALLS, SAFETY SHOES, SAFETY GLASSES, GLOVES.

Eye/face protection Wear safety glasses with side shields (or goggles).

Skin and body protection Wear suitable protective clothing. Long sleeved clothing. Protective shoes or boots.

Hand protection Wear suitable gloves.

Respiratory protection No protective equipment is needed under normal use conditions. If exposure limits are exceeded or irritation is experienced, ventilation and evacuation may be required. If determined by a risk assessment an inhalation risk exists, wear a suitable mist respirator meeting the requirements of AS/NZS 1715 and AS/NZS 1716.

Environmental exposure controls Do not allow into any sewer, on the ground or into any body of water. Local authorities should be advised if significant spillages cannot be contained.

9. PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Physical state Liquid

Appearance	Clear
Color	Pale Yellow
Odor	Slight Ester
Odor threshold	No information available.

Property	Values	Remarks • Method
pH	7.0 - 8.5	None known
pH (as aqueous solution)	No data available	None known
Melting point / freezing point	-10°C	None known
Boiling point / boiling range	>100°C	None known
Flash point	No data available	None known
Evaporation rate	No data available	None known
Flammability (solid, gas)	No data available	None known
Flammability Limit in Air		None known
Upper flammability or explosive limits	No data available	
Lower flammability or explosive limits	No data available	
Vapor pressure	No data available	None known
Vapor density	No data available	None known
Relative density	0.99 - 1.01	None known
Water solubility	Soluble in water	None known
Solubility(ies)	Soluble in ethanol	None known
Partition coefficient	No data available	None known
Autoignition temperature	No data available	None known
Decomposition temperature	No data available	None known
Kinematic viscosity	No data available	None known
Dynamic viscosity	No data available	None known

Other information

10. STABILITY AND REACTIVITY

Reactivity

Reactivity No information available.

Chemical stability

Stability Stable under normal conditions.

Explosion data

Sensitivity to mechanical impact None.

Sensitivity to static discharge None.

Possibility of hazardous reactions

Possibility of hazardous reactions None under normal processing.

Conditions to avoid

Conditions to avoid Keep away from open flames, hot surfaces and sources of ignition.

Incompatible materials

Incompatible materials Strong oxidizing agents.

Hazardous decomposition products

Hazardous decomposition products Carbon oxides. Oxides of sulfur.

11. TOXICOLOGICAL INFORMATION

Acute toxicity

Information on likely routes of exposure

Product Information No adverse health effects expected if the chemical is handled in accordance with this Safety Data Sheet and the chemical label. Symptoms or effects that may arise if the chemical is mishandled and overexposure occurs are:

Inhalation Inhalation of vapors in high concentration may cause irritation of respiratory system.

Eye contact Causes serious eye damage.

Skin contact Causes skin irritation.

Ingestion Ingestion may cause gastrointestinal irritation, nausea, vomiting and diarrhoea. Harmful if swallowed. May cause adverse kidney effects.

Symptoms No information available.

Numerical measures of toxicity - Product Information
No information available.

Component Information

Chemical name	Oral LD50	Dermal LD50	Inhalation LC50
Ethylene glycol	= 1700 mg/kg (Rat)	= 10600 mg/kg (Rat) = 9530 µL/kg (Rabbit)	-

See section 16 for terms and abbreviations

Delayed and immediate effects as well as chronic effects from short and long-term exposure

Skin corrosion/irritation Causes skin irritation. Classification based on data available for ingredients.

Serious eye damage/eye irritation Causes serious eye damage. Classification based on data available for ingredients.

Respiratory or skin sensitization No information available.

Germ cell mutagenicity No information available.

Carcinogenicity No information available.

Reproductive toxicity No information available.

STOT - single exposure Causes damage to organs if swallowed.

STOT - repeated exposure Causes damage to organs through prolonged or repeated exposure if swallowed.

Aspiration hazard No information available.

12. ECOLOGICAL INFORMATION

Ecotoxicity

Ecotoxicity Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Chemical name	Algae/aquatic plants	Fish	Toxicity to microorganisms	Crustacea
Ethylene glycol	EC50: 6500 - 13000mg/L (96h, Pseudokirchneriella subcapitata)	LC50: =41000mg/L (96h, Oncorhynchus mykiss) LC50: 14 - 18mL/L (96h, Oncorhynchus mykiss) LC50: =27540mg/L (96h, Lepomis macrochirus) LC50: =40761mg/L (96h, Oncorhynchus mykiss) LC50: 40000 - 60000mg/L (96h, Pimephales promelas) LC50: =16000mg/L (96h, Poecilia reticulata)	-	EC50: =46300mg/L (48h, Daphnia magna)

Persistence and degradability

Persistence and degradability Expected to be biodegradable.

Bioaccumulative potential

Bioaccumulation Bioaccumulation is not expected.

Chemical name	Partition coefficient
Ethylene glycol	-1.93

Mobility

Mobility in soil No information available.

Other adverse effects

13. DISPOSAL CONSIDERATIONS

Waste treatment methods

Waste from residues/unused products Dispose of in accordance with local regulations. Dispose of waste in accordance with environmental legislation.

Contaminated packaging Dispose of in accordance with federal, state and local regulations.

14. TRANSPORT INFORMATION

ADG
Not classified as Dangerous Goods by the criteria of the Australian Dangerous Goods Code (ADG Code) for transport by Road and Rail; NON-DANGEROUS GOODS.

IATA
Not classified as Dangerous Goods by the criteria of the International Air Transport Association (IATA) Dangerous Goods Regulations for transport by air; NON-DANGEROUS GOODS.

IMDG
Not regulated Not classified as Dangerous Goods by the criteria of the International Maritime Dangerous Goods Code (IMDG Code) for transport by sea; NON-DANGEROUS GOODS.

15. REGULATORY INFORMATION

Safety, health and environmental regulations/legislation specific for the substance or mixture

National regulations

Australia
Classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS).

Not classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG)

See section 8 for national exposure control parameters

Poisons Schedule (SUSMP) 6

Chemical name	National pollutant inventory
Ethylene glycol - 107-21-1	10 tonne/yr Threshold category 1

International Inventories

AIIC	All the constituents of this material are listed on the Australian Inventory of Industrial Chemicals.
NZIoC	All the constituents of this material are listed on the New Zealand Inventory of Chemicals.

Legend:
- Australian Inventory of Industrial Chemicals

International Regulations

The Montreal Protocol on Substances that Deplete the Ozone Layer Not applicable

The Stockholm Convention on Persistent Organic Pollutants Not applicable

The Rotterdam Convention Not applicable

16. OTHER INFORMATION

Supplier Safety Data Sheet 08/ 2016

Reason(s) For Issue: First Issue Primary SDS

Issuing Date: 30-Nov-2021

This Safety Data Sheet has been prepared by Ixom Operations Pty Ltd (Toxicology and SDS Services).

Revision Note:
The symbol (*) in the margin of this SDS indicates that this line has been revised.

Key or legend to abbreviations and acronyms used in the safety data sheet
Legend Section 8: EXPOSURE CONTROLS/PERSONAL PROTECTION

TWA	TWA (time-weighted average)	STEL	STEL (Short Term Exposure Limit)
Ceiling	Maximum limit value	*	Skin designation
C	Carcinogen		

Key literature references and sources for data used to compile the SDS

EPA (Environmental Protection Agency)
Acute Exposure Guideline Level(s) (AEGL(s))
U.S. Environmental Protection Agency Federal Insecticide, Fungicide, and Rodenticide Act
U.S. Environmental Protection Agency High Production Volume Chemicals
Food Research Journal
Hazardous Substance Database
International Uniform Chemical Information Database (IUCLID)
Japan GHS Classification
Australian Industrial Chemicals Introduction Scheme (AICIS)
NIOSH (National Institute for Occupational Safety and Health)
National Library of Medicine's ChemID Plus (NLM CIP)
National Library of Medicine's PubMed database (NLM PUBMED)
National Toxicology Program (NTP)
New Zealand's Chemical Classification and Information Database (CCID)
Organization for Economic Co-operation and Development Environment, Health, and Safety Publications
Organization for Economic Co-operation and Development High Production Volume Chemicals Program
Organization for Economic Co-operation and Development Screening Information Data Set
RTECS (Registry of Toxic Effects of Chemical Substances)
World Health Organization

Disclaimer

This SDS summarises to our best knowledge at the date of issue, the chemical health and safety hazards of the material and general guidance on how to safely handle the material in the workplace. Since The Supplier cannot anticipate or control the conditions under which the product may be used, each user must, prior to usage, assess and control the risks arising from its use of the material.

If clarification or further information is needed, the user should contact their Supplier representative or The Supplier at the contact details on page 1.

The Supplier's responsibility for the material as sold is subject to the terms and conditions of sale, a copy of which is available upon request.

End of Safety Data Sheet

CF380DXL	

SAFETY DATA SHEET

EMERGENCY TELEPHONE NUMBER: + 1 - 587 - 353 - 2940 (24 Hrs)

Product Name: CF380DXL
Date Issued: April 29, 2021

Prepared by: HSE Dept
& Version: A7-1.0

1. PRODUCT IDENTIFICATION AND COMPANY IDENTIFICATION

Product Name:	CF380DXL
Product Purpose:	Fracturing Additive
Supplier Identification:	Fusion Technologies (Australia) Pty Ltd. 7 Noble Street Bridgeman Downs QLD, 4035 Australia

PREPARER'S TELEPHONE NUMBER: + 1 - 587 - 353 - 2940

2. HAZARDS IDENTIFICATION

HSNO Hazard classification
Respiratory sensitization : Category 1

Hazard Pictograms:

Signal word: Danger

Primary Routes of Exposure: Inhalation and skin

GHS Classification in accordance with 29 CFR 1910 (OSHA HCS)
Acute Toxicity Oral (Category 4), H302
Skin Corrosion /Irritation (Category 1), H314
Reproductive Toxicity (Category 1), H360

Hazard statements:

H302 - Harmful if swallowed
H314 - Causes severe skin burns and eye damage
H360 - May damage fertility or the unborn child

Precautionary statements:

P260 - Do not breathe mist, vapours, spray
P264 - Wash exposed skin thoroughly after handling
P270 - Do not eat, drink or smoke when using this product
P280 - Wear protective gloves, protective clothing, eye protection, face protection
P301 + P330 + P331 - If Swallowed: rinse mouth. Do NOT induce vomiting
P303 + P361 + P353 – If on skin (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower P304 + P340 – In inhaled: remove victim to fresh air and keep at rest in a position comfortable for breathing
P305 + P351 + P338 - If in eyes: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing
P310 - Immediately call a POISON CENTER or doctor/physician
P363 - Wash contaminated clothing before reuse
P405 - Store locked up
P501 - Dispose of contents/container to comply with local, state and federal regulations

Human health effects:

Eye: Corrosive. May cause severe irritation with corneal injury which may result in permanent impairment of vision, even blindness.
Skin: Corrosive. Initial contact may result in itching with increasing irritation if not removed. Causes severe skin irritation with tissue destruction. Prolonged contact and badly damaged skin may result in absorption causing redness and peeling of skin.
Ingestion: Maybe fatal if swallowed. Causes burns to the mouth, throat and stomach. Symptoms may include nausea, headache, and vomiting. Cardiac failure, pulmonary edema, and severe kidney

damage may develop. Potassium carbonate is high caustic, and ingestion of either the granular or liquid forms will cause severe burning and pain in lips, mouth, tongue, throat and stomach.
Inhalation: Inhalation of mist may cause damage to nasal and respiratory passages. Inhalation of large amounts may cause nausea, vomiting and diarrhea. Irritation may lead to chemical pneumonitis and pulmonary edema.
Chronic: May cause asthma, lung diseases and skin diseases.

3. PRODUCT COMPOSITION/INGREDIENTS

Chemical Name	CAS #	% by Weight
Sodium Gluconate	527-07-1	15 to 40
Boric Acid	10043-35-3	7 to 13
Potassium Hydroxide	1310-58-3	15 to 40

4. FIRST AID MEASURES

Eye Contact: Rinse eyes immediately with copious amounts of water and under the eyelids for at least 30 minutes. If symptoms persist seek medical advice.

Skin Contact: Remove contaminated clothing. Immediately wash off all material with soap and copious amounts of water. Remove all contaminated clothing and footwear. Discard contaminated leather articles such as shoes and belt.

Ingestion: Do not induce vomiting without medical advice. Seek medical advice. If the victim is not breathing, perform resuscitation using an approved respiratory barrier.

Inhalation: Remove to fresh air, treat symptomatically. If symptoms persist, seek medical advice. If person is not breathing and heart has stopped, begin performing cardiopulmonary resuscitation immediately.

5. FIRE FIGHTING MEASURES

Flashpoint: Not determined
Auto-Ignition Temperature: Not determined

Lower Explosion Limit: Not determined
Upper Explosion Limit: Not determined

Extinguishing Media: Water fog or fine spray, carbon dioxide or dry chemical foam. Water spray or fog for larger fires is acceptable.

Special Fire Fighting Procedures: Cool tanks and containers with water spray. Do not flush into surface water or sanitary sewer system. Keep product and empty containers away from heat and ignition sources.

Unusual Fire & Explosion Hazard: Heating can release hazardous gases

Hazardous Combustion Products: May evolve oxides of nitrogen, potassium and carbon under fire conditions.

Protective Equipment for Firefighters: Self contained breathing apparatus

6. ACCIDENTAL RELEASE MEASURES

Personal Precautions: Avoid contact with skin, eyes and clothing. Evacuate personnel to safe areas. Keep people away from and upwind of spill or leak. PPE: see section 8.

Environmental Precautions: Do not contaminate surface water. Do not release into the environment. Prevent product from entering any drains. Do not flush product into surface water or sanitary sewer systems.

Methods For Cleaning Up: Sweep up and shovel and then place into an appropriate waste container. Remove soiled refuse and place in a suitable disposal container.

Disposal: Dispose of material in compliance with local, provincial and Federal regulations. See Section 13.

7. HANDLING AND STORAGE

Handling Precautions: Handle wearing appropriate PPE as per section 8. Ensure adequate ventilation is available to avoid breathing vapors. Avoid contact with

	eyes, skin and clothing. Do not ingest. Empty containers may contain product residues. Keep the containers closed when not in use. Protect against physical damage. Do not consume food, drink or smoke when handling this material. When mixing, slowly add to water to minimize heat generation and spattering.
Storage Precautions:	Store according to State and Federal regulations. Store in a cool, dry, well-ventilated area. Place away from incompatible materials. Keep containers tightly closed. Store at ambient temperatures. Tanks must be diked.

8. EXPOSURE CONTROLS/PERSONAL PROTECTION

Components with workplace control parameters:

Ingredient	Exposure Limits
Boric Acid	6 mg/m³ STEL 2 mg/m³ TLV-TWA
Sodium Hydroxide	2 mg/m³ Ceiling

Personal protective equipment:

Eye protection	Wear safety glasses with side shields or chemical goggles. Wear a face shield if splashing hazard exists.
Hand protection	Wear PVC, rubber or nitrile gloves.
Skin protection	Wear standard protective clothing – consider selecting type of protective clothing depending on quantity of chemical to be handled.
Respiratory protection	If exposure exceeds occupational exposure limits, use an appropriate NIOSH-approved respirator. For most conditions, no respirator protection is needed; however, if handling at elevated temperatures without sufficient ventilation, use an approved air-purifying respirator. Organic vapor cartridge with a particulate pre-filter.
Hygiene measures	Keep an eye wash fountain and safety shower available

9. PHYSICAL AND CHEMICAL PROPERTIES

Form:	Liquid
Color:	Colourless
Odor:	Characteristic
pH:	> 14
Density:	1.44
Solubility:	Soluble
Freezing Point:	-20°C

10. STABILITY AND REACTIVITY

Stability:	Stable under normal conditions.
Conditions to Avoid:	Avoid excessive heat, open flames and all ignition sources. Incompatible materials.
Materials to Avoid:	Strong oxidizing agents, strong acids and bases. Contact with reactive metals may produce flammable hydrogen gas.
Hazardous Polymerization:	Will not occur
Hazardous Decomposition Products:	Oxides of nitrogen, potassium and carbon.
Under Fire Conditions:	Heating can release hazardous gases

11. TOXICOLOGICAL INFORMATION

Ingredients	Acute Oral Toxicity	LD50/oral/rat	LC50/inhalation/rat	LD50/dermal/4hr/rabbit
Sodium Gluconate	No data available	No data available	No data available	No data available
Boric Acid	No data available	2,660 mg/kg	>0.16 mg/L in 4 hr	>2,000 mg/kg
Sodium Hydroxide	No data available	No data available	No data available	1350 mg/kg

Sensitization: Possible and may cause allergic reaction

Mutagenic Effects: Possible

Reproductive Toxicity: Boric acid studies in rat, mouse and dog at high doses, have demonstrated effects on fertility and testes. Boric acid studies in rat, mouse, and rabbit demonstrate developmental effects on the fetus, including fetal weight loss and minor skeletal variations. The doses administered were many times in excess of those to which humans would normally be exposed

Carcinogenic Effects: Boric acid is listed as A4 Carcinogens by the International Agency for Research on Cancer (IARC), the National Toxicology Program (NTP) or the American Conference of Governmental Industrial Hygienists (ACGIH).

Teratogenicity and Embryo Toxicity: See information listed above in reproductive category.

Human Experience: High

Other Toxicity Information: Toxicological Synergistic products: none known.

12. ECOLOGICAL INFORMATION

Ingredients	Ecotoxicity – Fish Species Data	Acute Crustaceans Toxicity	Ecotoxicity – Fresh water Algae
Sodium Gluconate	Not available	Not available	Not available
Boric Acid	1,020 mg/L LC50 (Carassius auratus) 72 h flow through	Not available	Not available
Sodium Hydroxide	Not available	Not available	Not available

Persistence and Degradability: Material is not readily biodegradable
Mobility: Product is liquid and therefore readily mobile.

13. DISPOSAL INFORMATION

Waste Residues/Unused Product and Package Dispose of waste in an approved incinerator or waste treatment site, in accordance with all applicable regulations. Do not dispose of wastes in local sewer or with normal garbage. Empty containers should be recycled locally or taken away for waste disposal.

14. TRANSPORT INFORMATION

The shipper/consignor/sender is responsible to ensure that the packaging, labeling, and markings are in compliance with the selected mode of transport.

Typical proper shipping name for this product are as follows:

SODIUM HYDROXIDE, SOLUTION	CLASS 8	UN 1824	PKG GRP: II

Important Note: This information does not take the place of shipping paper (Bill of Lading or BOL)

15. REGULATORY INFORMATION

None available.

16. OTHER INFORMATION

NFPA 704M RATING

Health: 3	Flammability: 0	Instability: 1	Other: n/a

HMIS

Health: 3	Flammability: 0	Instability: 1	Other: n/a

0= insignificant 1= slight 2= moderate 3= high 4= Extreme * = Chronic Hazard

Label Hazard Warning: Corrosive

Label Precautions: Inhalation of mist may cause damage to nasal and respiratory passages. Inhalation of large amounts may cause nausea, vomiting and diarrhea. Irritation may lead to chemical pneumonitis and pulmonary edema.
Corrosive. May cause severe irritation with corneal injury which may result in permanent impairment of vision, even blindness.
Corrosive. Initial contact may result in itching with increasing irritation if not removed. Causes severe skin irritation with tissue destruction. Prolonged contact and badly damaged skin may result in absorption causing redness and peeling of skin.

Label First Aid: Wash product off of skin or out of eyes. If swallowed, do not induce vomiting without medical advice. If irritation develops, seek medical attention.

This material safety data sheet provides health and safety information for the safe use of this product provided it is used as recommended per the associated product literature. Users of this product should be aware of the recommended safety precautions. For any other use, exposures must be evaluated so

that appropriate handling and training programs can be created and implemented to insure safe workplace operations. Consult with Fusion Technologies for any additional information.

This material safety data sheet provides health and safety information for the safe use of this product provided it is used as recommended per the associated product literature. Users of this product should be aware of the recommended safety precautions. For any other use, exposures must be evaluated so that appropriate handling and training programs can be created and implemented to insure safe workplace operations. Consult with Fusion Technologies for any additional information.

SAFETY DATA SHEET

CONDOR ENERGY SERVICES CF8800

Section: 1. PRODUCT AND COMPANY IDENTIFICATION

Product name	:	CONDOR ENERGY SERVICES CF8800
Other means of identification	:	Manufactured exclusively for Condor Energy Services by NALCO Champion
Recommended use	:	EMULSION BREAKER
Restrictions on use	:	Refer to available product literature or ask your local Sales Representative for restrictions on use and dose limits.
Company	:	ChampionX Australia Pty Ltd Suite 1/5 Brodie-Hall Drive, Technology Park Bentley WA 6102 Australia TEL: +61 8 9473 9000
Emergency telephone number	:	CHEMCALL 1800 127 406, International: +64 4 917 8888
Issuing date	:	08.01.2020

Section: 2. HAZARDS IDENTIFICATION

GHS Classification

Oxidizing solids	:	Category 1
Acute toxicity (Oral)	:	Category 4
Skin corrosion/irritation	:	Category 2
Serious eye damage/eye irritation	:	Category 2A
Germ cell mutagenicity	:	Category 2
Carcinogenicity	:	Category 2
Specific target organ toxicity - single exposure	:	Category 3 (Respiratory system)

GHS Label element

Hazard pictograms :

Signal Word : Danger

Hazard Statements : May cause fire or explosion; strong oxidiser.
Harmful if swallowed.
Causes skin irritation.
Causes serious eye irritation.
May cause respiratory irritation.
Suspected of causing genetic defects.
Suspected of causing cancer.

Precautionary Statements : **Prevention:**
Obtain special instructions before use. Do not handle until all safety precautions have been read and understood. Keep away from heat/sparks/open flames/hot surfaces. - No smoking. Keep/Store away from clothing and other combustible materials. Avoid breathing dust/ fume/ gas/ mist/ vapours/ spray. Wear fire resistant or flame retardant clothing. Do not eat, drink or smoke when using this product. Wear protective gloves/ protective clothing/ eye protection/ face

protection.
Response:
IF SWALLOWED: Call a POISON CENTER or doctor/ physician if you feel unwell. Rinse mouth. IF ON SKIN: Wash with plenty of soap and water. IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing. Call a POISON CENTER or doctor/ physician if you feel unwell. IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. IF ON CLOTHING: rinse immediately contaminated clothing and skin with plenty of water before removing clothes. In case of major fire and large quantities: Evacuate area. Fight fire remotely due to the risk of explosion. If eye irritation persists: Get medical advice/ attention. Take off contaminated clothing and wash it before reuse. Call a POISON CENTER or doctor/ physician if you feel unwell.
Disposal:
Dispose of contents/container to an approved facility in accordance with local, regional, national and international regulations.

Other hazards : None known.

Section: 3. COMPOSITION/INFORMATION ON INGREDIENTS

Pure substance/mixture : Substance

Chemical Name	CAS-No.	Concentration: (%)
Sodium Bromate	7789-38-0	60 - 100

Section: 4. FIRST AID MEASURES

In case of eye contact : Rinse immediately with plenty of water, also under the eyelids, for at least 15 minutes. Remove contact lenses, if present and easy to do. Continue rinsing. Get medical attention.

In case of skin contact : Wash off immediately with plenty of water for at least 15 minutes. Use a mild soap if available. Get medical attention if irritation develops and persists.

If swallowed : Rinse mouth. Get medical attention if symptoms occur.

Contact the Poison's Information Centre (eg Australia 13 1126; New Zealand 0800 764 766).

If inhaled : Remove to fresh air. Treat symptomatically. Get medical attention if symptoms occur.

Protection of first-aiders : In event of emergency assess the danger before taking action. Do not put yourself at risk of injury. If in doubt, contact emergency responders. Use personal protective equipment as required.

Notes to physician : Treat symptomatically.

Most important symptoms and effects, both acute and delayed : See Section 11 for more detailed information on health effects and symptoms.

Section: 5. FIREFIGHTING MEASURES

Suitable extinguishing media : Use extinguishing measures that are appropriate to local circumstances and the surrounding environment.

Unsuitable extinguishing : None known.

media

Specific hazards during firefighting	:	Oxidizer. Contact with other material may cause fire.
Hazardous combustion products	:	Hydrogen halides metal oxides
Special protective equipment for firefighters	:	Use personal protective equipment.
Specific extinguishing methods	:	Fire residues and contaminated fire extinguishing water must be disposed of in accordance with local regulations.
Hazchem Code	:	1Y

Section: 6. ACCIDENTAL RELEASE MEASURES

Initial Emergency Response Guide No	:	31
Personal precautions, protective equipment and emergency procedures	:	Ensure adequate ventilation. Ensure clean-up is conducted by trained personnel only. Refer to protective measures listed in sections 7 and 8.
Environmental precautions	:	Do not allow contact with soil, surface or ground water.
Methods and materials for containment and cleaning up	:	Sweep up and shovel into suitable containers for disposal.

Section: 7. HANDLING AND STORAGE

Advice on safe handling	:	Avoid contact with skin and eyes. Do not ingest. Do not breathe dust/fume/gas/mist/vapours/spray. Wash hands thoroughly after handling. Use only with adequate ventilation.
Conditions for safe storage	:	Keep in a cool, well-ventilated place. Keep away from reducing agents. Keep away from combustible material. Keep out of reach of children. Keep container tightly closed. Store in suitable labelled containers.
Suitable material	:	Keep in properly labelled containers.
Unsuitable material	:	not determined

Section: 8. EXPOSURE CONTROLS/PERSONAL PROTECTION

Components with workplace control parameters

Contains no substances with occupational exposure limit values.

Engineering measures	:	Effective exhaust ventilation system. Maintain air concentrations below occupational exposure standards.

Personal protective equipment

Eye protection	:	Safety goggles Face-shield
Hand protection	:	Wear the following personal protective equipment:

		NEOPRENE, NITRILE, OR NATURAL RUBBER GLOVES Standard glove type. Gloves should be discarded and replaced if there is any indication of degradation or chemical breakthrough.
Skin protection	:	Flame retardant protective clothing
Respiratory protection	:	When workers are facing concentrations above the exposure limit they must use appropriate certified respirators.
Hygiene measures	:	Handle in accordance with good industrial hygiene and safety practice. Remove and wash contaminated clothing before re-use. Wash face, hands and any exposed skin thoroughly after handling.

The Personal Protective Equipment (PPE) recommendations provided above have been made in good faith based on typical expected conditions of use. PPE selection should always be completed in conjunction with a proper risk assessment and in accordance with a PPE management program.

Section: 9. PHYSICAL AND CHEMICAL PROPERTIES

Appearance	:	Granular
Colour	:	white
Odour	:	odourless
Flash point	:	Not applicable.
pH	:	no data available
Odour Threshold	:	no data available
Melting point/freezing point	:	no data available
Initial boiling point and boiling range	:	380 °C, Decomposes on heating.
Evaporation rate	:	no data available
Flammability (solid, gas)	:	no data available
Upper explosion limit	:	no data available
Lower explosion limit	:	no data available
Vapour pressure	:	no data available
Relative vapour density	:	no data available
Relative density	:	3.34, (20 °C),
Density	:	no data available
Water solubility	:	360 g/l completely soluble (20 °C)
Solubility in other solvents	:	no data available
Partition coefficient: n-octanol/water	:	no data available
Auto-ignition temperature	:	no data available
Thermal decomposition	:	no data available
Viscosity, dynamic	:	no data available
Viscosity, kinematic	:	no data available
Molecular weight	:	no data available
VOC	:	no data available

Section: 10. STABILITY AND REACTIVITY

Reactivity	:	No dangerous reaction known under conditions of normal use.
Chemical stability	:	Stable under normal conditions.
Possibility of hazardous reactions	:	No dangerous reaction known under conditions of normal use.
Conditions to avoid	:	None known.
Incompatible materials	:	None known.
Hazardous decomposition products	:	In case of fire, hazardous decomposition products may be produced such as: Hydrogen halides metal oxides

Section: 11. TOXICOLOGICAL INFORMATION

Information on likely routes of exposure	:	Eye contact, Skin contact

Potential Health Effects

Eyes	:	Causes serious eye irritation.
Skin	:	Causes skin irritation.
Ingestion	:	Harmful if swallowed.
Inhalation	:	May cause respiratory tract irritation.
Chronic Exposure	:	Suspected of causing genetic defects. Suspected of causing cancer.

Experience with human exposure

Eye contact	:	Redness, Pain, Irritation
Skin contact	:	Redness, Irritation
Ingestion	:	Vomiting
Inhalation	:	Respiratory irritation, Cough

Toxicity

Product

Acute oral toxicity	:	Acute toxicity estimate: 385 mg/kg
Acute inhalation toxicity	:	no data available
Acute dermal toxicity	:	no data available
Skin corrosion/irritation	:	no data available
Serious eye damage/eye irritation	:	no data available
Respiratory or skin sensitization	:	no data available

Carcinogenicity	:	Suspected of causing cancer.
Reproductive effects	:	No toxicity to reproduction
Germ cell mutagenicity	:	Suspected of causing genetic defects.
Teratogenicity	:	no data available
STOT - single exposure	:	no data available
STOT - repeated exposure	:	no data available
Aspiration toxicity	:	No aspiration toxicity classification

Human Hazard Characterization

Based on our hazard characterization, the potential human hazard is: High

Section: 12. ECOLOGICAL INFORMATION

Ecotoxicity

Environmental Effects	:	This product has no known ecotoxicological effects.

Product

Toxicity to fish	:	no data available
Toxicity to daphnia and other aquatic invertebrates	:	no data available
Toxicity to algae	:	no data available

Persistence and degradability

no data available

Mobility

The environmental fate was estimated using a level III fugacity model embedded in the EPI (estimation program interface) Suite TM, provided by the US EPA. The model assumes a steady state condition between the total input and output. The level III model does not require equilibrium between the defined media. The information provided is intended to give the user a general estimate of the environmental fate of this product under the defined conditions of the models.
If released into the environment this material is expected to distribute to the air, water and soil/sediment in the approximate respective percentages;

Air	:	<5%
Water	:	30 - 50%
Soil	:	50 - 70%

Bioaccumulative potential

no data available

Other information

no data available

ENVIRONMENTAL HAZARD AND EXPOSURE CHARACTERIZATION
Based on our hazard characterization, the potential environmental hazard is: Low

Section: 13. DISPOSAL CONSIDERATIONS

Disposal methods	:	Where possible recycling is preferred to disposal or incineration. If recycling is not practicable, dispose of in compliance with local regulations. Dispose of wastes in an approved waste disposal facility.
Disposal considerations	:	Dispose of as unused product. Empty containers should be taken to an approved waste handling site for recycling or disposal. Do not re-use empty containers.

Section: 14. TRANSPORT INFORMATION

The shipper/consignor/sender is responsible to ensure that the packaging, labeling, and markings are in compliance with the selected mode of transport.

Land transport

Proper shipping name	:	SODIUM BROMATE
UN/ID No.	:	UN 1494
Transport hazard class(es)	:	5.1
Packing group	:	II
IERG No	:	31
Hazchem Code	:	1Y
Special precautions for user	:	Dangerous goods of Class 5.1 (Oxidising Agent) are incompatible in a placard load with any of the following: Class 1 Explosives Class 2.1 Flammable gases Class 2.3 Poisonous gases Class 3 Flammable liquids Class 4.1 Flammable solids Class 4.2 Spontaneously combustible substances Class 4.3 Dangerous when wet substances Class 5.2 Organic peroxides Class 7 Radioactive substances Class 8 Corrosives

Air transport (IATA)

UN/ID No.	:	UN 1494
Proper shipping name	:	SODIUM BROMATE
Technical name(s)	:	
Transport hazard class(es)	:	5.1
Packing group	:	II

Sea transport (IMDG/IMO)

UN/ID No.	:	UN 1494
Proper shipping name	:	SODIUM BROMATE
Technical name(s)	:	
Transport hazard class(es)	:	5.1
Packing group	:	II

Section: 15. REGULATORY INFORMATION

Standard for the Uniform Scheduling of Medicines and Poisons	:	Schedule 6

INTERNATIONAL CHEMICAL CONTROL LAWS :

United States TSCA Inventory
The substances in this preparation are included on or exempted from the TSCA 8(b) Inventory (40 CFR 710)

Canadian Domestic Substances List (DSL)
The substances in this preparation are listed on the Domestic Substances List (DSL), are exempt, or have been reported in accordance with the New Substances Notification Regulations.

Taiwan Chemical Substance Inventory
All substances in this product comply with the Taiwan Existing Chemical Substances Inventory (ECSI).

Australia. Industrial Chemical (Notification and Assessment) Act
All substances in this product comply with the National Industrial Chemicals Notification & Assessment Scheme (NICNAS).

Korea. Korean Existing Chemicals Inventory (KECI)
All substances in this product comply with the Chemical Control Act (CCA) and are listed on the Existing Chemicals List (ECL)

Japan. ENCS - Existing and New Chemical Substances Inventory
All substances in this product comply with the Law Regulating the Manufacture and Importation Of Chemical Substances and are listed on the Existing and New Chemical Substances list (ENCS).

Philippines Inventory of Chemicals and Chemical Substances (PICCS)
All substances in this product comply with the Republic Act 6969 (RA 6969) and are listed on the Philippines Inventory of Chemicals & Chemical Substances (PICCS).

China Inventory of Existing Chemical Substances
All substances in this product comply with the Provisions on the Environmental Administration of New Chemical Substances and are listed on or exempt from the Inventory of Existing Chemical Substances China (IECSC).

New Zealand. Inventory of Chemicals (NZIoC), as published by ERMA New Zealand
All substances in this product comply with the Hazardous Substances and New Organisms (HSNO) Act 1996,and are listed on or are exempt from the New Zealand Inventory of Chemicals.

Section: 16. OTHER INFORMATION

REFERENCES

Hazardous Substances Data Bank, National Library of Medicine, Bethesda, Maryland (TOMES CPS™ CD-ROM Version), Micromedex, Inc., Englewood, CO.

IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man, Geneva: World Health Organization, International Agency for Research on Cancer.

Integrated Risk Information System, U.S. Environmental Protection Agency, Washington, D.C. (TOMES CPS™ CD-ROM Version),
Micromedex, Inc., Englewood, CO.

Annual Report on Carcinogens, National Toxicology Program, U.S. Department of Health and Human Services, Public Health Service.

Registry of Toxic Effects of Chemical Substances, National Institute for Occupational Safety and Health, Cincinnati, OH,
(TOMES CPS™ CD-ROM Version), Micromedex, Inc., Englewood, CO.

The Teratogen Information System, University of Washington, Seattle, WA (TOMES CPS™ CD-ROM Version), Micromedex, Inc., Englewood, CO.

Revision Date	:	08.01.2020
Date of first issue	:	09.06.2016
Version Number	:	1.2
Prepared By	:	Regulatory Affairs

REVISED INFORMATION: Significant changes to regulatory or health information for this revision is indicated by a bar in the left-hand margin of the SDS.

The information provided in this Safety Data Sheet is correct to the best of our knowledge, information and belief at the date of its publication. The information given is designed only as a guidance for safe handling, use, processing, storage, transportation, disposal and release and is not to be considered a warranty or quality specification. The information relates only to the specific material designated and may not be valid for such material used in combination with any other materials or in any process, unless specified in the text.

SAFETY DATA SHEET

Revision date: 24-Jan-2022

Revision Number 1

1. IDENTIFICATION OF THE MATERIAL AND SUPPLIER

Product identifier

Product Name CF8550EA

Product Code(s) 000000069052

Other means of identification

UN number 1444

Synonyms Manufactured exclusively for Condor Energy Services by Fusion Technologies (Australia) Pty Ltd

Recommended use of the chemical and restrictions on use

Recommended use Hydraulic fracturing additive.

Uses advised against No information available.

Supplier
Fusion Technologies Australia Pty Ltd
ABN: 50 636 538 960
Street Address: 7 Noble Street
Bridgeman Downs QLD 4035
Australia

Telephone number: +61 (0)460 047 656
Website: www.fusiontechinc.net

Emergency telephone number

Emergency telephone number **1800 033 111 (ALL HOURS)**

Please ensure you refer to the limitations of this Safety Data Sheet as set out in the "Other Information" section at the end of this Data Sheet.

2. HAZARDS IDENTIFICATION

GHS Classification

Classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS).

Classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG).

Oxidizing solids	Category 3
Acute toxicity - Oral	Category 4
Respiratory sensitization	Category 1
Skin sensitization	Category 1
Specific target organ toxicity (single exposure)	Category 3

SIGNAL WORD
Danger

Label elements

Flame over circle
Exclamation mark
Health hazard

Hazard statements
H302 - Harmful if swallowed
H334 - May cause allergy or asthma symptoms or breathing difficulties if inhaled
H317 - May cause an allergic skin reaction
H335 - May cause respiratory irritation
H272 - May intensify fire; oxidizer

Precautionary Statements - Prevention
Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking
Keep/Store away from clothing/ combustible materials
Do not eat, drink or smoke when using this product
Wear protective gloves / protective clothing / eye protection / face protection
Avoid breathing dust / fume / gas / mist / vapours / spray
Use only outdoors or in a well-ventilated area
In case of inadequate ventilation wear respiratory protection
Wash face, hands and any exposed skin thoroughly after handling
Contaminated work clothing should not be allowed out of the workplace
Precautionary Statements - Response
IF exposed:
IF IN EYES If eye irritation persists: Get medical advice/attention
IF ON SKIN: Gently wash with plenty of soap and water If skin irritation or rash occurs: Get medical advice/attention Take off contaminated clothing and wash before reuse
IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing
If experiencing respiratory symptoms: Call a POISON CENTER or doctor
IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell
Rinse mouth
In case of fire: Use extinguishing media as outlined in Section 5 of this Safety Data Sheet to extinguish.
Precautionary Statements - Storage
Store in a well-ventilated place. Keep container tightly closed
Store locked up
Precautionary Statements - Disposal
Dispose of contents/container in accordance with local, regional, national, and international regulations as applicable

Other hazards which do not result in classification
Poisons Schedule (SUSMP) 6

3. COMPOSITION/INFORMATION ON INGREDIENTS

Chemical name	CAS No.	Weight-%
Ammonium persulfate	7727-54-0	60-90%
Talc	14807-96-6	<5%
Non-hazardous ingredients	Proprietary	Balance

4. FIRST AID MEASURES

Description of first aid measures

General advice Take a copy of the Safety Data Sheet when going for medical treatment.

Inhalation Remove to fresh air and keep at rest in a position comfortable for breathing. If breathing is difficult, (trained personnel should) give oxygen. Give artificial respiration if victim is not breathing. Get immediate medical advice/attention.

Eye contact Rinse immediately with plenty of water, also under the eyelids, for at least 15 minutes. Remove contact lenses, if present and easy to do. Continue rinsing. Do not rub affected area. Seek immediate medical attention/advice.

Skin contact Remove and isolate contaminated clothing and shoes. Wash off immediately with plenty of water. Get medical attention if symptoms occur. Allergic symptoms may be delayed.

Ingestion Rinse mouth thoroughly with water. Do NOT induce vomiting. Drink 1 or 2 glasses of water. Get immediate medical advice/attention.

Most important symptoms and effects, both acute and delayed

Symptoms May cause allergic skin reaction. May cause allergy or asthma symptoms or breathing difficulties if inhaled.

Indication of any immediate medical attention and special treatment needed

Note to physicians Treat symptomatically.

5. FIRE FIGHTING MEASURES

Suitable Extinguishing Media

Suitable Extinguishing Media Water spray or fog is preferred; if water not available use dry chemical, CO2 or regular foam.

Unsuitable extinguishing media No information available.

Specific hazards arising from the chemical

Specific hazards arising from the chemical Oxidizer. Non-combustible, substance itself does not burn but may decompose upon heating to produce corrosive and/or toxic fumes. Promotes the combustion (oxidizer). Can cause fire and explosion when in contact with flammable substances. Any material contaminated with the product (e.g. clothes) ignites easily and burns vigorously - increased fire hazard. Containers may explode when heated.

Hazardous combustion products Carbon oxides.

Special protective actions for fire-fighters

Special protective equipment for fire-fighters	Firefighters should wear self-contained breathing apparatus and full firefighting turnout gear. Cool containers with flooding quantities of water until well after fire is out.
Hazchem code	1Z

6. ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Personal precautions	Ensure adequate ventilation. Evacuate personnel to safe areas. Stop leak if you can do it without risk. Avoid breathing dust / fume / gas / mist / vapours / spray. Avoid generation of dust.
Other information	ELIMINATE all ignition sources (no smoking, flares, sparks or flames in immediate area).
For emergency responders	Use personal protection recommended in Section 8.

Environmental precautions

Environmental precautions	See Section 12 for additional Ecological Information.

Methods and material for containment and cleaning up

Methods for containment	Cover powder spill with plastic sheet or tarp to minimize spreading. Prevent dust cloud.
Methods for cleaning up	Take up with inert, damp, non-combustible material using clean non-sparking tools and place into loosely covered plastic containers for later disposal. Do not dry sweep dust. Wet dust with water before sweeping or use a vacuum to collect dust. Keep in suitable, closed containers for disposal. Prevent product from entering drains.

7. HANDLING AND STORAGE

Precautions for safe handling

Advice on safe handling	Handle in accordance with good industrial hygiene and safety practice. Avoid contact with skin, eyes, and clothing. Avoid breathing dust or spray mist. Take precautionary measures against static discharges.
General hygiene considerations	Take off contaminated clothing and wash it before reuse. Wash hands and face before breaks and immediately after handling the product. Wear suitable gloves and eye/face protection. When using do not eat, drink or smoke.

Conditions for safe storage, including any incompatibilities

Storage Conditions	Keep containers tightly closed in a dry, cool and well-ventilated place.
Incompatible materials	Acids. Alkalis. Combustible material. Halogenated compounds. Organic compounds.
Poisons Schedule (SUSMP)	6

8. EXPOSURE CONTROLS/PERSONAL PROTECTION

Control parameters

Exposure Limits	No value assigned for this specific material by Safe Work Australia. However, Workplace Exposure Standard(s) for constituent(s):

Chemical name	Australia	ACGIH TLV
Ammonium persulfate 7727-54-0	0.1 mg/m³ Peak	TWA: 0.1 mg/m³ persulfate

Talc (containing no asbestos fibres): 8hr TWA = 2.5 mg/m³

As published by Safe Work Australia Workplace Exposure Standards for Airborne Contaminants.

Peak Limitation - a maximum or peak airborne concentration of a particular substance determined over the shortest analytically practicable period of time which does not exceed 15 minutes.

TWA - The time-weighted average airborne concentration of a particular substance when calculated over an eight-hour working day, for a five-day working week.

These Workplace Exposure Standards are guides to be used in the control of occupational health hazards. All atmospheric contamination should be kept to as low a level as is workable. These workplace exposure standards should not be used as fine dividing lines between safe and dangerous concentrations of chemicals. They are not a measure of relative toxicity.

Appropriate engineering controls

Engineering controls

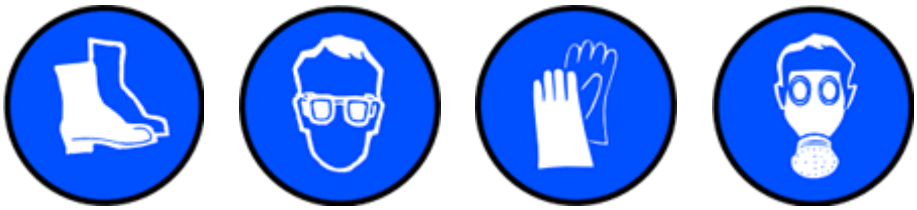
Apply technical measures to comply with the occupational exposure limits.

If in the handling and application of this material, safe exposure levels could be exceeded, the use of engineering controls such as local exhaust ventilation must be considered and the results documented. If achieving safe exposure levels does not require engineering controls, then a detailed and documented risk assessment using the relevant Personal Protective Equipment (PPE) (refer to PPE section below) as a basis must be carried out to determine the minimum PPE requirements.

Individual protection measures, such as personal protective equipment

The selection of PPE is dependent on a detailed risk assessment. The risk assessment should consider the work situation, the physical form of the chemical, the handling methods, and environmental factors.

OVERALLS, SAFETY SHOES, SAFETY GLASSES, GLOVES, DUST MASK.



Eye/face protection

Wear safety glasses with side shields (or goggles).

Skin and body protection

Wear suitable protective clothing. Long sleeved clothing.

Hand protection

Wear suitable gloves.

Respiratory protection

No protective equipment is needed under normal use conditions. If exposure limits are exceeded or irritation is experienced, ventilation and evacuation may be required. If determined by a risk assessment an inhalation risk exists, wear a dust mask/respirator meeting the requirements of AS/NZS 1715 and AS/NZS 1716.

Environmental exposure controls

Avoid creating dust.

9. PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Physical state	Solid
Appearance	Crystalline Powder
Color	Beige
Odor	Faint Organic
Odor threshold	No information available.

Property	**Values**	**Remarks • Method**
pH	7.2	
pH (as aqueous solution)	No data available	None known
Melting point / freezing point	121°C (Decomposes on heating)	
Boiling point / boiling range	No data available	None known
Flash point	121°C	None known
Evaporation rate	No data available	None known
Flammability (solid, gas)	No data available	None known
Flammability Limit in Air		None known
Upper flammability or explosive limits	No data available	
Lower flammability or explosive limits	No data available	
Vapor pressure	No data available	None known
Vapor density	No data available	None known
Relative density	1.8	
Water solubility	Insoluble in water	
Solubility(ies)	No data available	None known
Partition coefficient	No data available	None known
Autoignition temperature	No data available	None known
Decomposition temperature	>120°C	
Kinematic viscosity	No data available	None known
Dynamic viscosity	No data available	None known

Other information

10. STABILITY AND REACTIVITY

Reactivity

Reactivity Oxidizer.

Chemical stability

Stability Stable under normal conditions. Unstable if heated.

Explosion data

Sensitivity to mechanical impact None.

Sensitivity to static discharge None.

Possibility of hazardous reactions

Possibility of hazardous reactions Can react violently with reducing agents. Contact with combustible material may cause fire.

Hazardous polymerization Hazardous polymerization does not occur.

Conditions to avoid

Conditions to avoid Dust formation. Extremes of temperature and direct sunlight.

Incompatible materials

Incompatible materials Acids. Alkalis. Combustible material. Halogenated compounds. Organic compounds.

Hazardous decomposition products

Hazardous decomposition products Carbon oxides. Nitrogen oxides. Oxides of sulfur.

11. TOXICOLOGICAL INFORMATION

Acute toxicity

Information on likely routes of exposure

Product Information No adverse health effects expected if the chemical is handled in accordance with this Safety Data Sheet and the chemical label. Symptoms or effects that may arise if the chemical is mishandled and overexposure occurs are:

Inhalation Irritating to respiratory system. May cause sensitization by inhalation. May cause allergy or asthma symptoms or breathing difficulties if inhaled.

Eye contact May cause irritation.

Skin contact May cause irritation. May cause sensitization by skin contact. Repeated or prolonged skin contact may cause allergic reactions with susceptible persons.

Ingestion Ingestion may cause gastrointestinal irritation, nausea, vomiting and diarrhoea.

Symptoms Irritating. Asthma-like and/ or skin allergy-like symptoms. May cause sensitization by inhalation and skin contact.

Numerical measures of toxicity - Product Information
No information available.

Component Information

Chemical name	Oral LD50	Dermal LD50	Inhalation LC50
Ammonium persulfate	= 495 mg/kg (Rat)	> 10000 mg/kg (Rabbit)	= 520 mg/L (Rat) 1 h

See section 16 for terms and abbreviations

Delayed and immediate effects as well as chronic effects from short and long-term exposure

Skin corrosion/irritation May cause skin irritation. Classification based on data available for ingredients.

Serious eye damage/eye irritation Mild eye irritation. Classification based on data available for ingredients.

Respiratory or skin sensitization May cause sensitization by inhalation and skin contact.

Germ cell mutagenicity No information available.

Carcinogenicity No information available.

Reproductive toxicity No information available.

STOT - single exposure May cause respiratory irritation.

STOT - repeated exposure No information available.

Aspiration hazard Not applicable.

12. ECOLOGICAL INFORMATION

Ecotoxicity

Ecotoxicity Keep out of waterways.

Chemical name	Algae/aquatic plants	Fish	Toxicity to microorganisms	Crustacea
Ammonium persulfate	-	LC50: =103mg/L (96h, Lepomis macrochirus) LC50: =76.3mg/L (96h, Oncorhynchus mykiss) LC50: =323mg/L (96h, Poecilia reticulata)	-	EC50: =120mg/L (48h, Daphnia magna)
Talc	-	LC50: >100g/L (96h, Brachydanio rerio)	-	-

Persistence and degradability

Persistence and degradability Not readily biodegradable.

Bioaccumulative potential

Bioaccumulation Bioaccumulation is not expected.

Mobility

Mobility in soil No information available.

Other adverse effects

13. DISPOSAL CONSIDERATIONS

Waste treatment methods

Waste from residues/unused products Dispose of in accordance with local regulations. Dispose of waste in accordance with environmental legislation.

Contaminated packaging Dispose of contents/containers in accordance with local regulations.

14. TRANSPORT INFORMATION

ADG
Classified as Dangerous Goods by the criteria of the Australian Dangerous Goods Code (ADG Code) for Transport by Road and Rail; DANGEROUS GOODS.

UN number 1444
Proper shipping name AMMONIUM PERSULPHATE
Hazard class 5.1
Packing group III
Hazchem code 1Z

IATA
Classified as Dangerous Goods by the criteria of the International Air Transport Association (IATA) Dangerous Goods Regulations

for transport by air; DANGEROUS GOODS.

UN number	1444
UN proper shipping name	AMMONIUM PERSULPHATE
Transport hazard class(es)	5.1
Packing group	III

IMDG
Classified as Dangerous Goods by the criteria of the International Maritime Dangerous Goods Code (IMDG Code) for transport by sea; DANGEROUS GOODS.

UN number	1444
UN proper shipping name	AMMONIUM PERSULPHATE
Transport hazard class(es)	5.1
Packing group	III
IMDG EMS Fire	F-A
IMDG EMS Spill	S-Q

15. REGULATORY INFORMATION

Safety, health and environmental regulations/legislation specific for the substance or mixture

National regulations

Australia
Classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS).

Classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG).

See section 8 for national exposure control parameters

Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP)
Classified as a scheduled poison according to the Standard for Uniform Scheduling of Medicines and Poisons (SUSMP)
Poisons Schedule (SUSMP) 6

International Inventories

AIIC	This material is listed on the Australian Inventory of Industrial Chemicals.
NZIoC	All the constituents of this material are listed on the New Zealand Inventory of Chemicals.

Legend:
- Australian Inventory of Industrial Chemicals

International Regulations

The Montreal Protocol on Substances that Deplete the Ozone Layer Not applicable

The Stockholm Convention on Persistent Organic Pollutants Not applicable

The Rotterdam Convention Not applicable

16. OTHER INFORMATION

Supplier Safety Data Sheet 06/ 2020

Reason(s) For Issue: First Issue Primary SDS

Issuing Date: 24-Jan-2022

This Safety Data Sheet has been prepared by Ixom Operations Pty Ltd (Toxicology and SDS Services).

Revision Note:
The symbol (*) in the margin of this SDS indicates that this line has been revised.

Key or legend to abbreviations and acronyms used in the safety data sheet

Legend Section 8: EXPOSURE CONTROLS/PERSONAL PROTECTION

TWA	TWA (time-weighted average)	STEL	STEL (Short Term Exposure Limit)
Ceiling	Maximum limit value	*	Skin designation
C	Carcinogen		

Key literature references and sources for data used to compile the SDS

EPA (Environmental Protection Agency)
Acute Exposure Guideline Level(s) (AEGL(s))
U.S. Environmental Protection Agency Federal Insecticide, Fungicide, and Rodenticide Act
U.S. Environmental Protection Agency High Production Volume Chemicals
Food Research Journal
Hazardous Substance Database
International Uniform Chemical Information Database (IUCLID)
Japan GHS Classification
Australian Industrial Chemicals Introduction Scheme (AICIS)
NIOSH (National Institute for Occupational Safety and Health)
National Library of Medicine's ChemID Plus (NLM CIP)
National Library of Medicine's PubMed database (NLM PUBMED)
National Toxicology Program (NTP)
New Zealand's Chemical Classification and Information Database (CCID)
Organization for Economic Co-operation and Development Environment, Health, and Safety Publications
Organization for Economic Co-operation and Development High Production Volume Chemicals Program
Organization for Economic Co-operation and Development Screening Information Data Set
RTECS (Registry of Toxic Effects of Chemical Substances)
World Health Organization

Disclaimer

This SDS summarises to our best knowledge at the date of issue, the chemical health and safety hazards of the material and general guidance on how to safely handle the material in the workplace. Since The Supplier cannot anticipate or control the conditions under which the product may be used, each user must, prior to usage, assess and control the risks arising from its use of the material.

If clarification or further information is needed, the user should contact their Supplier representative or The Supplier at the contact details on page 1.

The Supplier's responsibility for the material as sold is subject to the terms and conditions of sale, a copy of which is available upon request.

End of Safety Data Sheet

SAFETY DATA SHEET

Revision date: 20-Apr-2022

Revision Number 1

1. IDENTIFICATION OF THE MATERIAL AND SUPPLIER

Product identifier

Product Name CF8200E

Product Code(s) 000000069059

Other means of identification

Recommended use of the chemical and restrictions on use

Recommended use Hydraulic fracturing fluid.

Uses advised against No information available.

Supplier
Condor Energy Services Ltd
ABN: 35 153 250 670
Brisbane Head Office: Level 11, 333 Ann Street
Brisbane QLD 4000
Australia

Telephone number: 07 3999 9044
Website: www.CondorEnergy.com.au

Emergency telephone number

Emergency telephone number **1800 033 111 (ALL HOURS)**

Please ensure you refer to the limitations of this Safety Data Sheet as set out in the "Other Information" section at the end of this Data Sheet.

2. HAZARDS IDENTIFICATION

GHS Classification

Classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS).

Not classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG)

Respiratory sensitization	Category 1

SIGNAL WORD

Danger

Label elements

Health hazard

Hazard statements
H334 - May cause allergy or asthma symptoms or breathing difficulties if inhaled

Precautionary Statements - Prevention
Avoid breathing dust / fume / gas / mist / vapours / spray
In case of inadequate ventilation wear respiratory protection
Precautionary Statements - Response
IF exposed or concerned
IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing
If experiencing respiratory symptoms: Call a POISON CENTER or doctor/physician
Precautionary Statements - Storage
No storage statements
Precautionary Statements - Disposal
Dispose of contents/container in accordance with local, regional, national, and international regulations as applicable

Other hazards which do not result in classification
Poisons Schedule (SUSMP) 5

3. COMPOSITION/INFORMATION ON INGREDIENTS

Chemical name	CAS No.	Weight-%
Mannanase (Mannan endo-1,4-beta-mannosidase)	37288-54-3	<5
Non-hazardous ingredients	Proprietary	Balance

4. FIRST AID MEASURES

Description of first aid measures

Emergency telephone number Poisons Information Center, Australia: 13 11 26

Inhalation Move victim to fresh air. Treatment should be symptomatic and supportive. Get immediate medical advice/attention. If breathing is difficult, (trained personnel should) give oxygen. Do not use mouth-to-mouth method if victim ingested or inhaled the substance; give artificial respiration with the aid of a pocket mask equipped with a one-way valve or other proper respiratory medical device.

Eye contact In case of eye contact, immediately flush eyes with plenty of water for at least 15 minutes. Get medical attention if symptoms occur.

Skin contact Wash off immediately with soap and plenty of water. Get medical attention if symptoms occur.

Ingestion Rinse mouth thoroughly with water. Get medical attention if symptoms occur.

Self-protection of the first aider Do not breathe fume, gas, mist, vapours, spray. Use personal protective equipment as

required.

Most important symptoms and effects, both acute and delayed

Symptoms May cause allergy or asthma symptoms or breathing difficulties if inhaled.

Indication of any immediate medical attention and special treatment needed

Note to physicians Treat symptomatically.

5. FIRE FIGHTING MEASURES

Suitable Extinguishing Media

Suitable Extinguishing Media Use extinguishing measures that are appropriate to local circumstances and the surrounding environment.

Unsuitable extinguishing media None known.

Specific hazards arising from the chemical

Specific hazards arising from the chemical Non-combustible, substance itself does not burn but may decompose upon heating to produce corrosive and/or toxic fumes.

Hazardous combustion products Carbon oxides.

Special protective actions for fire-fighters

Special protective equipment for fire-fighters Firefighters should wear self-contained breathing apparatus and full firefighting turnout gear. Use personal protection equipment.

6. ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Personal precautions Avoid breathing vapors or mists. Ensure adequate ventilation. Use personal protective equipment as required.

For emergency responders Use personal protection recommended in Section 8.

Environmental precautions

Environmental precautions Do not allow to enter into soil/subsoil. Keep out of waterways.

Methods and material for containment and cleaning up

Methods for containment Contain and collect spillage with non-combustible absorbent material, (e.g. sand, earth, diatomaceous earth, vermiculite) and place in container for disposal according to local / national regulations (see Section 13).

Methods for cleaning up Take up with sand or other non-combustible absorbent material and place into containers for later disposal. Dike to collect large liquid spills. Prevent product from entering drains.

7. HANDLING AND STORAGE

Precautions for safe handling

Advice on safe handling Handle in accordance with good industrial hygiene and safety practice. Avoid breathing dust / fume / gas / mist / vapours / spray. Ensure adequate ventilation.

General hygiene considerations Do not eat, drink or smoke when using this product.

Conditions for safe storage, including any incompatibilities

Storage Conditions Keep out of the reach of children. Keep container closed when not in use. Store in accordance with local regulations.

Incompatible materials None known.

Poisons Schedule (SUSMP) 5

8. EXPOSURE CONTROLS/PERSONAL PROTECTION

Control parameters

Exposure Limits No value assigned for this specific material by Safe Work Australia.

Appropriate engineering controls

Engineering controls Apply technical measures to comply with the occupational exposure limits.

If in the handling and application of this material, safe exposure levels could be exceeded, the use of engineering controls such as local exhaust ventilation must be considered and the results documented. If achieving safe exposure levels does not require engineering controls, then a detailed and documented risk assessment using the relevant Personal Protective Equipment (PPE) (refer to PPE section below) as a basis must be carried out to determine the minimum PPE requirements.

Individual protection measures, such as personal protective equipment

The selection of PPE is dependent on a detailed risk assessment. The risk assessment should consider the work situation, the physical form of the chemical, the handling methods, and environmental factors.

OVERALLS, SAFETY SHOES, SAFETY GLASSES, GLOVES.

Eye/face protection Wear safety glasses with side shields (or goggles).

Skin and body protection Wear suitable protective clothing.

Hand protection Protective gloves. Nitrile rubber.

Respiratory protection If determined by a risk assessment an inhalation risk exists, wear a suitable mist respirator meeting the requirements of AS/NZS 1715 and AS/NZS 1716.

Environmental exposure controls No information available.

9. PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Physical state	Liquid
Appearance	Clear
Color	Amber
Odor	Slight Fermentation
Odor threshold	No information available.

Property	**Values**	**Remarks • Method**
pH	4.8 - 6.5	None known
pH (as aqueous solution)	No data available	None known
Melting point / freezing point	0 °C	Pour Point
Boiling point / boiling range	100 °C	None known
Flash point	No data available	None known
Evaporation rate	No data available	None known
Flammability (solid, gas)	No data available	None known
Flammability Limit in Air		None known
Upper flammability or explosive limits	No data available	
Lower flammability or explosive limits	No data available	
Vapor pressure	No data available	None known
Vapor density	No data available	None known
Relative density	1.000 - 1.050	None known
Water solubility	No data available	None known
Solubility(ies)	No data available	None known
Partition coefficient	No data available	None known
Autoignition temperature	No data available	None known
Decomposition temperature	No data available	None known
Kinematic viscosity	No data available	None known
Dynamic viscosity	1 mPa s	None known

Other information

10. STABILITY AND REACTIVITY

Reactivity

Reactivity Non-reactive under normal conditions of use, storage and transport.

Chemical stability

Stability Stable under normal conditions.

Explosion data

Sensitivity to mechanical impact None.

Sensitivity to static discharge None.

Possibility of hazardous reactions

Possibility of hazardous reactions None under normal processing.

Conditions to avoid

Conditions to avoid None known based on information supplied.

Incompatible materials

Incompatible materials None known.

Hazardous decomposition products

Hazardous decomposition products Carbon oxides.

11. TOXICOLOGICAL INFORMATION

Acute toxicity

Information on likely routes of exposure

Product Information No adverse health effects expected if the chemical is handled in accordance with this Safety Data Sheet and the chemical label. Symptoms or effects that may arise if the chemical is mishandled and overexposure occurs are:

Inhalation May cause allergy or asthma symptoms or breathing difficulties if inhaled.

Eye contact May cause slight irritation.

Skin contact May cause irritation.

Ingestion Ingestion may cause gastrointestinal irritation, nausea, vomiting and diarrhoea.

Symptoms May cause allergy or asthma symptoms or breathing difficulties if inhaled.

Numerical measures of toxicity - Product Information
No information available.

See section 16 for terms and abbreviations

Delayed and immediate effects as well as chronic effects from short and long-term exposure

Skin corrosion/irritation No information available.

Serious eye damage/eye irritation No information available.

Respiratory or skin sensitization May cause sensitization by inhalation. Classification based on individual ingredients of the mixture.

Germ cell mutagenicity No information available.

Carcinogenicity No information available.

Reproductive toxicity No information available.

Developmental toxicity None known

STOT - single exposure No information available.

STOT - repeated exposure No information available.

Aspiration hazard No information available.

Chronic effects: No long term risks to humans are associated with this material when handled and used as directed on the label.

12. ECOLOGICAL INFORMATION

Ecotoxicity

Ecotoxicity Not considered to be harmful to aquatic life.

Persistence and degradability

Persistence and degradability Readily biodegradable.

Bioaccumulative potential

Bioaccumulation Bioaccumulation is not expected.

Mobility

Mobility in soil Expected to be mobile in soil.

Mobility Soluble in water.

Other adverse effects

Other adverse effects No information available.

13. DISPOSAL CONSIDERATIONS

Waste treatment methods

Waste from residues/unused products Dispose of in accordance with local regulations.

Contaminated packaging Dispose of in accordance with federal, state and local regulations. Dispose of wastes in an approved waste disposal facility. Empty containers should be taken to an approved waste handling site for recycling or disposal.

14. TRANSPORT INFORMATION

ADG
Not classified as Dangerous Goods by the criteria of the Australian Dangerous Goods Code (ADG Code) for transport by Road and Rail; NON-DANGEROUS GOODS.

IATA
Not classified as Dangerous Goods by the criteria of the International Air Transport Association (IATA) Dangerous Goods Regulations for transport by air; NON-DANGEROUS GOODS.

IMDG
Not classified as Dangerous Goods by the criteria of the International Maritime Dangerous Goods Code (IMDG Code) for transport by sea; NON-DANGEROUS GOODS.

15. REGULATORY INFORMATION

Safety, health and environmental regulations/legislation specific for the substance or mixture

National regulations

Australia
Classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS).

Not classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG)

See section 8 for national exposure control parameters

Poisons Schedule (SUSMP) 5

International Inventories

AIIC	All the constituents of this material are listed on the Australian Inventory of Industrial Chemicals.
NZIoC	All the constituents of this material are listed on the New Zealand Inventory of Chemicals.

Legend:
- Australian Inventory of Industrial Chemicals

International Regulations

The Montreal Protocol on Substances that Deplete the Ozone Layer Not applicable

The Stockholm Convention on Persistent Organic Pollutants Not applicable

The Rotterdam Convention Not applicable

16. OTHER INFORMATION

Supplier Safety Data Sheet 06/ 2020

Reason(s) For Issue: First Issue Primary SDS

Issuing Date: 20-Apr-2022

This Safety Data Sheet has been prepared by Ixom Operations Pty Ltd (Toxicology and SDS Services).

Revision Note:
1.

Key or legend to abbreviations and acronyms used in the safety data sheet

Legend Section 8: EXPOSURE CONTROLS/PERSONAL PROTECTION

TWA	TWA (time-weighted average)	STEL	STEL (Short Term Exposure Limit)
Ceiling	Maximum limit value	*	Skin designation
C	Carcinogen		

Key literature references and sources for data used to compile the SDS

EPA (Environmental Protection Agency)

Acute Exposure Guideline Level(s) (AEGL(s))
U.S. Environmental Protection Agency Federal Insecticide, Fungicide, and Rodenticide Act
U.S. Environmental Protection Agency High Production Volume Chemicals
Food Research Journal
Hazardous Substance Database
International Uniform Chemical Information Database (IUCLID)
Japan GHS Classification
Australian Industrial Chemicals Introduction Scheme (AICIS)
NIOSH (National Institute for Occupational Safety and Health)
National Library of Medicine's ChemID Plus (NLM CIP)
National Library of Medicine's PubMed database (NLM PUBMED)
National Toxicology Program (NTP)
New Zealand's Chemical Classification and Information Database (CCID)
Organization for Economic Co-operation and Development Environment, Health, and Safety Publications
Organization for Economic Co-operation and Development High Production Volume Chemicals Program
Organization for Economic Co-operation and Development Screening Information Data Set
RTECS (Registry of Toxic Effects of Chemical Substances)
World Health Organization

Disclaimer

This SDS summarises to our best knowledge at the date of issue, the chemical health and safety hazards of the material and general guidance on how to safely handle the material in the workplace. Since The Supplier cannot anticipate or control the conditions under which the product may be used, each user must, prior to usage, assess and control the risks arising from its use of the material.

If clarification or further information is needed, the user should contact their Supplier representative or The Supplier at the contact details on page 1.

The Supplier's responsibility for the material as sold is subject to the terms and conditions of sale, a copy of which is available upon request.

End of Safety Data Sheet

SAFETY DATA SHEET

Revision date: 16-Jul-2021

Revision Number 1

1. IDENTIFICATION OF THE MATERIAL AND SUPPLIER

Product identifier

Product Name CFBE5

Product Code(s) 000000069016

Other means of identification

UN number 2922

Synonyms Manufactured exclusively for Condor Energy Services by Fusion Technologies (Australia) Pty Ltd

Recommended use of the chemical and restrictions on use

Recommended use Biocidal product.

Uses advised against No information available.

Supplier
Fusion Technologies Australia Pty Ltd
ABN: 50 636 538 960
Street Address: 7 Noble Street
Bridgeman Downs QLD 4035
Australia

Telephone number: +61 (0)460 047 656
Website: www.fusiontechinc.net

Emergency telephone number

Emergency telephone number **1800 033 111 (ALL HOURS)**

Please ensure you refer to the limitations of this Safety Data Sheet as set out in the "Other Information" section at the end of this Data Sheet.

2. HAZARDS IDENTIFICATION

GHS Classification

Classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS).

Classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG).

Acute toxicity - Oral	Category 4
Acute toxicity - Inhalation (Vapors)	Category 3
Skin corrosion/irritation	Category 1 Sub-category B
Respiratory sensitization	Category 1
Skin sensitization	Category 1A
Specific target organ toxicity (single exposure)	Category 3
Acute aquatic toxicity	Category 1
Chronic aquatic toxicity	Category 2

SIGNAL WORD
Danger

Label elements

Skull and crossbones
Corrosion
Health hazard
Environment

Hazard statements
H331 - Toxic if inhaled
H302 - Harmful if swallowed
H335 - May cause respiratory irritation
H314 - Causes severe skin burns and eye damage
H334 - May cause allergy or asthma symptoms or breathing difficulties if inhaled
H317 - May cause an allergic skin reaction
H400 - Very toxic to aquatic life
H411 - Toxic to aquatic life with long lasting effects

Precautionary Statements - Prevention
Do not breathe mist, vapours, spray.
Use only outdoors or in a well-ventilated area
In case of inadequate ventilation wear respiratory protection
Wear protective gloves / protective clothing / eye protection / face protection
Wash face, hands and any exposed skin thoroughly after handling
Contaminated work clothing should not be allowed out of the workplace
Do not eat, drink or smoke when using this product
Avoid release to the environment
Precautionary Statements - Response

IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing
IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower
Wash contaminated clothing before reuse
If skin irritation or rash occurs: Get medical advice/attention
IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing
Immediately call a POISON CENTER or doctor/physician
IF SWALLOWED: Call a POISON CENTER or doctor/physician if you feel unwell
Rinse mouth
Collect spillage
Precautionary Statements - Storage
Store in a well-ventilated place. Keep container tightly closed

Store locked up

Precautionary Statements - Disposal

Dispose of contents/container in accordance with local, regional, national, and international regulations as applicable

Other hazards which do not result in classification

Poisons Schedule (SUSMP) 6

3. COMPOSITION/INFORMATION ON INGREDIENTS

Chemical name	CAS No.	Weight-%
Glutaraldehyde	111-30-8	20-50%
Methanol (methyl alcohol)	67-56-1	1-5%
Non-hazardous ingredients	Proprietary	Balance

4. FIRST AID MEASURES

Description of first aid measures

General advice Take a copy of the Safety Data Sheet when going for medical treatment.

Emergency telephone number Poisons Information Center, Australia: 13 11 26
Poisons Information Center, New Zealand: 0800 764 766

Inhalation Move to fresh air in case of accidental inhalation of vapors. Seek immediate medical attention/advice. If not breathing, give artificial respiration. Do not use mouth-to-mouth method if victim ingested or inhaled the substance; give artificial respiration with the aid of a pocket mask equipped with a one-way valve or other proper respiratory medical device.

Eye contact Rinse immediately with plenty of water, also under the eyelids, for at least 15 minutes. Get immediate medical advice/attention.

Skin contact Wash off immediately with soap and plenty of water for at least 15 minutes. Remove and isolate contaminated clothing and shoes. Get immediate medical advice/attention. Wash contaminated clothing before reuse.

Ingestion Rinse mouth thoroughly with water. Never give anything by mouth to an unconscious person. Do NOT induce vomiting. Immediate medical attention is required.

Self-protection of the first aider Do not breathe vapor or mist. Do not use mouth-to-mouth method if victim ingested or inhaled the substance; give artificial respiration with the aid of a pocket mask equipped with a one-way valve or other proper respiratory medical device. Avoid contact with skin, eyes, and clothing.

Most important symptoms and effects, both acute and delayed

Symptoms Asthma-like and/ or skin allergy-like symptoms. May cause redness and tearing of the eyes. Burning sensation.

Indication of any immediate medical attention and special treatment needed

Note to physicians Treat symptomatically.

5. FIRE FIGHTING MEASURES

Suitable Extinguishing Media

Suitable Extinguishing Media Use extinguishing measures that are appropriate to local circumstances and the surrounding environment.

Unsuitable extinguishing media High volume water jet.

Specific hazards arising from the chemical

Specific hazards arising from the chemical Non-combustible. Environmentally hazardous.

Hazardous combustion products Carbon oxides.

Special protective actions for fire-fighters

Special protective equipment for fire-fighters Firefighters should wear self-contained breathing apparatus and full firefighting turnout gear. Use personal protection equipment.

Hazchem code 2X

6. ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Personal precautions Ensure adequate ventilation. Keep people away from and upwind of spill/leak. Avoid contact with skin, eyes and inhalation of vapors.

For emergency responders Use personal protection recommended in Section 8.

Environmental precautions

Environmental precautions Do not allow to enter into soil/subsoil. Keep out of waterways. See Section 12 for additional Ecological Information.

Methods and material for containment and cleaning up

Methods for containment Stop leak if you can do it without risk. Contain and collect spillage with non-combustible absorbent material, (e.g. sand, earth, diatomaceous earth, vermiculite) and place in container for disposal according to local / national regulations (see Section 13). Dike to collect large liquid spills.

Methods for cleaning up Soak up with inert absorbent material (e.g. sand, silica gel, acid binder, universal binder, sawdust). Sweep up and shovel into suitable containers for disposal. Avoid breathing dust or spray mist. Clean contaminated surface thoroughly.

7. HANDLING AND STORAGE

Precautions for safe handling

Advice on safe handling Handle in accordance with good industrial hygiene and safety practice. Do not breathe vapor or mist. Do not get in eyes. Avoid contact with skin. Wash thoroughly after handling. Ensure adequate ventilation. In case of insufficient ventilation, wear suitable respiratory equipment.

General hygiene considerations Remove and wash contaminated clothing and gloves, including the inside, before re-use. Take off contaminated clothing and wash it before reuse. When using do not eat, drink or smoke.

Conditions for safe storage, including any incompatibilities

Storage Conditions Keep container tightly closed in a dry and well-ventilated place. Store locked up. Keep in properly labelled containers.

This material is a Scheduled Poison and must be stored, maintained and used in accordance with the relevant regulations.

Incompatible materials None known based on information supplied.

Poisons Schedule (SUSMP) 6

8. EXPOSURE CONTROLS/PERSONAL PROTECTION

Control parameters

Exposure Limits No value assigned for this specific material by Safe Work Australia. However, Workplace Exposure Standard(s) for constituent(s):

Chemical name	Australia	ACGIH TLV
Glutaraldehyde 111-30-8	0.1 ppm Peak 0.41 mg/m³ Peak	Ceiling: 0.05 ppm activated and inactivated

Glutaraldehyde: Peak Limitation = 0.41 mg/m³ (0.1 ppm), Sen
Methyl alcohol (Methanol): 8hr TWA = 262 mg/m³ (200 ppm), 15 min STEL = 328 mg/m³ (250 ppm), Sk

As published by Safe Work Australia Workplace Exposure Standards for Airborne Contaminants.

TWA - The time-weighted average airborne concentration of a particular substance when calculated over an eight-hour working day, for a five-day working week.

STEL (Short Term Exposure Limit) - the airborne concentration of a particular substance calculated as a time-weighted average over 15 minutes, which should not be exceeded at any time during a normal eight hour work day. According to current knowledge this concentration should neither impair the health of, nor cause undue discomfort to, nearly all workers.

`Sen' Notice - sensitiser. The substance can cause a specific immune response in some people. An affected individual may subsequently react to exposure to minute levels of that substance and should not be further exposed to the substance.

`Sk' (skin) Notice - absorption through the skin may be a significant source of exposure. The exposure standard is invalidated if such contact should occur.

These Workplace Exposure Standards are guides to be used in the control of occupational health hazards. All atmospheric contamination should be kept to as low a level as is workable. These workplace exposure standards should not be used as fine dividing lines between safe and dangerous concentrations of chemicals. They are not a measure of relative toxicity.

Appropriate engineering controls

Engineering controls Apply technical measures to comply with the occupational exposure limits.

If in the handling and application of this material, safe exposure levels could be exceeded, the use of engineering controls such as local exhaust ventilation must be considered and the results documented. If achieving safe exposure levels does not require engineering controls, then a detailed and documented risk assessment using the relevant Personal Protective Equipment (PPE) (refer to PPE section below) as a basis must be carried out to determine the minimum PPE requirements.

Individual protection measures, such as personal protective equipment

The selection of PPE is dependent on a detailed risk assessment. The risk assessment should consider the work situation, the physical form of the chemical, the handling methods, and environmental factors.

OVERALLS, SAFETY SHOES, SAFETY GLASSES, GLOVES, RESPIRATOR.

Eye/face protection	Wear safety glasses with side shields (or goggles). If splashes are likely to occur:. Face protection shield.
Skin and body protection	Wear suitable protective clothing. Long sleeved clothing.
Hand protection	Wear suitable gloves. Nitrile rubber. Neoprene gloves. Impervious gloves.
Respiratory protection	No protective equipment is needed under normal use conditions. If exposure limits are exceeded or irritation is experienced, ventilation and evacuation may be required. If determined by a risk assessment an inhalation risk exists, wear an organic vapour respirator meeting the requirements of AS/NZS 1715 and AS/NZS 1716.
Environmental exposure controls	Do not allow into any sewer, on the ground or into any body of water. Local authorities should be advised if significant spillages cannot be contained.

9. PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Physical state	Liquid
Appearance	Clear
Color	Colourless
Odor	Pungent
Odor threshold	No information available.

Property	**Values**	**Remarks • Method**
pH	3 - 5	
pH (as aqueous solution)	No data available	None known
Melting point / freezing point	No data available	None known
Boiling point / boiling range	No data available	None known
Flash point	> 100°C	None known
Evaporation rate	No data available	None known
Flammability (solid, gas)	No data available	None known
Flammability Limit in Air		None known
Upper flammability or explosive limits	No data available	
Lower flammability or explosive limits	No data available	
Vapor pressure	No data available	None known
Vapor density	No data available	None known
Relative density	1.063	
Water solubility	Miscible in water	
Solubility(ies)	No data available	None known
Partition coefficient	No data available	None known
Autoignition temperature	No data available	None known
Decomposition temperature	No data available	None known
Kinematic viscosity	No data available	None known
Dynamic viscosity	No data available	None known

Other information

10. STABILITY AND REACTIVITY

Reactivity

Reactivity No information available.

Chemical stability

Stability Stable under normal conditions.

Explosion data

Sensitivity to mechanical impact None.

Sensitivity to static discharge None.

Possibility of hazardous reactions

Possibility of hazardous reactions None under normal processing.

Conditions to avoid

Conditions to avoid None known based on information supplied.

Incompatible materials

Incompatible materials None known based on information supplied.

Hazardous decomposition products

Hazardous decomposition products Carbon oxides.

11. TOXICOLOGICAL INFORMATION

Acute toxicity

Information on likely routes of exposure

Product Information No adverse health effects expected if the chemical is handled in accordance with this Safety Data Sheet and the chemical label. Symptoms or effects that may arise if the chemical is mishandled and overexposure occurs are:

Inhalation Toxic by inhalation. Vapors may be irritating to eyes, nose, throat, and lungs. May cause allergy or asthma symptoms or breathing difficulties if inhaled. May cause sensitization by inhalation.

Eye contact Causes serious eye irritation.

Skin contact Harmful in contact with skin. Causes severe burns. Repeated or prolonged skin contact may cause allergic reactions with susceptible persons.

Ingestion Harmful if swallowed. Can burn mouth, throat, and stomach. Ingestion may cause gastrointestinal irritation, nausea, vomiting and diarrhoea.

Symptoms Asthma-like and/ or skin allergy-like symptoms. May cause sensitization by inhalation and skin contact. Irritation/Corrosion. May cause redness and tearing of the eyes. Rashes. Coughing and/ or wheezing.

Numerical measures of toxicity - Product Information
No information available.

Numerical measures of toxicity - Component Information

Component Information

Chemical name	Oral LD50	Dermal LD50	Inhalation LC50
Glutaraldehyde	= 252 mg/kg (Rat)	= 1800 mg/kg (Rabbit) = 560 µL/kg (Rabbit)	= 40.1 ppm (Rat) 4 h = 23.5 ppm (Rat) 4 h
Methanol (methyl alcohol)	= 6200 mg/kg (Rat)	= 15840 mg/kg (Rabbit) = 15800 mg/kg (Rabbit)	= 64000 ppm (Rat) 4 h = 22500 ppm (Rat) 8 h

See section 16 for terms and abbreviations

Delayed and immediate effects as well as chronic effects from short and long-term exposure

Skin corrosion/irritation Causes burns.

Serious eye damage/eye irritation Causes serious eye irritation.

Respiratory or skin sensitization May cause sensitization by inhalation and skin contact.

Germ cell mutagenicity No information available.

Carcinogenicity No information available.

Reproductive toxicity No information available.

STOT - single exposure May cause respiratory irritation.

STOT - repeated exposure No information available.

Aspiration hazard No information available.

12. ECOLOGICAL INFORMATION

Ecotoxicity

Ecotoxicity Toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Chemical name	Algae/aquatic plants	Fish	Toxicity to microorganisms	Crustacea
Glutaraldehyde	EC50: =0.61mg/L (72h, Desmodesmus subspicatus) EC50: =0.84mg/L (96h, Desmodesmus subspicatus)	LC50: 7.8 - 22mg/L (96h, Lepomis macrochirus) LC50: 2.6 - 4.8mg/L (96h, Oncorhynchus mykiss) LC50: 7.8 - 13mg/L (96h, Oncorhynchus mykiss) LC50: =5.4mg/L (96h, Pimephales promelas)	-	EC50: =14mg/L (48h, Daphnia magna) EC50: 0.56 - 1.0mg/L (48h, Daphnia magna)
Methanol (methyl alcohol)	-	LC50: =28200mg/L (96h, Pimephales promelas) LC50: >100mg/L (96h, Pimephales promelas) LC50: 19500 - 20700mg/L (96h, Oncorhynchus mykiss) LC50: 18 - 20mL/L (96h,	-	-

		Oncorhynchus mykiss) LC50: 13500 - 17600mg/L (96h, Lepomis macrochirus)		

Persistence and degradability

Persistence and degradability No information available.

Bioaccumulative potential

Bioaccumulation There is no data for this product.

Component Information

Chemical name	Partition coefficient
Glutaraldehyde	0.22
Methanol (methyl alcohol)	-0.77

Mobility

Mobility in soil No information available.

Other adverse effects

13. DISPOSAL CONSIDERATIONS

Waste treatment methods

Waste from residues/unused products Dispose of in accordance with local regulations. Dispose of waste in accordance with environmental legislation.

Contaminated packaging Dispose of contents/containers in accordance with local regulations.

14. TRANSPORT INFORMATION

ADG

Classified as Dangerous Goods by the criteria of the Australian Dangerous Goods Code (ADG Code) for Transport by Road and Rail; DANGEROUS GOODS.

UN number 2922
Proper shipping name CORROSIVE LIQUID, TOXIC, N.O.S. (CONTAINS GLUTARALDEHYDE)
Hazard class 8
Subsidiary hazard class 6.1
Packing group II
Hazchem code 2X

IATA

Classified as Dangerous Goods by the criteria of the International Air Transport Association (IATA) Dangerous Goods Regulations for transport by air; DANGEROUS GOODS.

UN number 2922
UN proper shipping name CORROSIVE LIQUID, TOXIC, N.O.S. (CONTAINS GLUTARALDEHYDE)
Transport hazard class(es) 8
Subsidiary hazard class 6.1
Packing group II

IMDG

Classified as Dangerous Goods by the criteria of the International Maritime Dangerous Goods Code (IMDG Code) for transport by sea; DANGEROUS GOODS.

UN number	2922
UN proper shipping name	CORROSIVE LIQUID, TOXIC, N.O.S. (CONTAINS GLUTARALDEHYDE)
Transport hazard class(es)	8
Subsidiary hazard class	6.1
Packing group	II

15. REGULATORY INFORMATION

Safety, health and environmental regulations/legislation specific for the substance or mixture

National regulations

Australia
Classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS).

Classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG).

See section 8 for national exposure control parameters

Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP)
Classified as a scheduled poison according to the Standard for Uniform Scheduling of Medicines and Poisons (SUSMP)
Poisons Schedule (SUSMP) 6

National pollutant inventory
Subject to reporting requirement

Chemical name	National pollutant inventory
Glutaraldehyde - 111-30-8	10 tonne/yr Threshold category 1
Methanol (methyl alcohol) - 67-56-1	10 tonne/yr Threshold category 1

Banned and/or restricted
This product contains one or more substance(s) subject to prohibition, authorization or restriction. Verify that requirements related to using, handling, and storing substances subject to prohibition, authorization or restriction are met.

Chemical name	Carcinogen	Restricted substance
Methanol (methyl alcohol) - 67-56-1		For spray painting at a concentration of >1% by volume

International Inventories

AICS	All the constituents of this material are listed on the Australian Inventory of Industrial Chemicals.

Legend:
- Australian Inventory of Industrial Chemicals

International Regulations

The Montreal Protocol on Substances that Deplete the Ozone Layer Not applicable

The Stockholm Convention on Persistent Organic Pollutants Not applicable

The Rotterdam Convention Not applicable

16. OTHER INFORMATION

Supplier Safety Data Sheet 05/ 2020

Reason(s) For Issue: First Issue Primary SDS

Issuing Date: 16-Jul-2021

This Safety Data Sheet has been prepared by Ixom Operations Pty Ltd (Toxicology and SDS Services).

Revision Note:
The symbol (*) in the margin of this SDS indicates that this line has been revised.

Key or legend to abbreviations and acronyms used in the safety data sheet

Legend Section 8: EXPOSURE CONTROLS/PERSONAL PROTECTION

TWA	TWA (time-weighted average)	STEL	STEL (Short Term Exposure Limit)
Ceiling	Maximum limit value	*	Skin designation
C	Carcinogen		

Key literature references and sources for data used to compile the SDS

EPA (Environmental Protection Agency)
Acute Exposure Guideline Level(s) (AEGL(s))
U.S. Environmental Protection Agency Federal Insecticide, Fungicide, and Rodenticide Act
U.S. Environmental Protection Agency High Production Volume Chemicals
Food Research Journal
Hazardous Substance Database
International Uniform Chemical Information Database (IUCLID)
Japan GHS Classification
Australian Industrial Chemicals Introduction Scheme (AICIS)
NIOSH (National Institute for Occupational Safety and Health)
National Library of Medicine's ChemID Plus (NLM CIP)
National Library of Medicine's PubMed database (NLM PUBMED)
National Toxicology Program (NTP)
New Zealand's Chemical Classification and Information Database (CCID)
Organization for Economic Co-operation and Development Environment, Health, and Safety Publications
Organization for Economic Co-operation and Development High Production Volume Chemicals Program
Organization for Economic Co-operation and Development Screening Information Data Set
RTECS (Registry of Toxic Effects of Chemical Substances)
World Health Organization

Disclaimer

This SDS summarises to our best knowledge at the date of issue, the chemical health and safety hazards of the material and general guidance on how to safely handle the material in the workplace. Since The Supplier cannot anticipate or control the conditions under which the product may be used, each user must, prior to usage, assess and control the risks arising from its use of the material.

If clarification or further information is needed, the user should contact their Supplier representative or The Supplier at the contact details on page 1.

The Supplier's responsibility for the material as sold is subject to the terms and conditions of sale, a copy of which is available upon request.

End of Safety Data Sheet

SAFETY DATA SHEET
HYDROCHLORIC ACID SOLUTION

SECTION 1: Identification of the substance/mixture and of the company/undertaking

1.1. Product identifier

Product name HYDROCHLORIC ACID SOLUTION

Product No. H27

CAS number 7647-01-0

EC number 231-595-7

1.2. Relevant identified uses of the substance or mixture and uses advised against

Application Acidifier. Chemical intermediate. Laboratory reagent. Pickling and anodising metals, scale remover.

1.3. Details of the supplier of the safety data sheet

Supplier

Norkem Limited Australia
G19, Wheelers Hill Business Centre,
202 Jells Road, Wheelers Hill,
Vic 3150, Australia
T: +61 (0) 3 9560 0158
F: +61 (0) 3 9561 3935
datasheet@norkem.com

1.4. Emergency telephone number

Emergency telephone Australian Transport Contact Number: +61 (0) 2801 44558. New Zealand Transport Contact Number: +64 (0) 9929 1483. National Poison Information Number: 131126

SECTION 2: Hazards identification

2.1. Classification of the substance or mixture

Classification

Physical hazards Met. Corr. 1 - H290

Health hazards Skin Corr. 1B - H314 Eye Dam. 1 - H318 STOT SE 3 - H335

Environmental hazards Not classified.

2.2. Label elements

EC number 231-595-7

Pictogram

Signal word Danger

Hazard statements

H290 May be corrosive to metals.
H314 Causes severe skin burns and eye damage.
H335 May cause respiratory irritation.

HYDROCHLORIC ACID SOLUTION

Precautionary statements	P280 Wear protective gloves/protective clothing/eye protection/face protection. P301+P330+P331 IF SWALLOWED: rinse mouth. Do NOT induce vomiting. P303+P361+P353 IF ON SKIN (or hair): Remove/Take off immediately all contaminated clothing. Rinse skin with water/shower. P305+P351+P338 IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing. P310 Immediately call a POISON CENTER or doctor/physician. P403+P233 Store in a well-ventilated place. Keep container tightly closed.
Contains	HYDROCHLORIC ACID
Supplementary precautionary statements	P234 Keep only in original container. P260 Do not breathe vapour/spray. P261 Avoid breathing vapour/spray. P264 Wash contaminated skin thoroughly after handling. P271 Use only outdoors or in a well-ventilated area. P304+P340 IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing. P312 Call a POISON CENTER or doctor/physician if you feel unwell. P321 Specific treatment (see medical advice on this label). P363 Wash contaminated clothing before reuse. P390 Absorb spillage to prevent material damage. P405 Store locked up. P406 Store in corrosive resistant container with a resistant inner liner. P501 Dispose of contents/container in accordance with national regulations.

2.3. Other hazards

In contact with some metals can generate hydrogen gas, which can form explosive mixtures with air. Reacts with alkalis and generates heat.

SECTION 3: Composition/information on ingredients

3.2. Mixtures

HYDROCHLORIC ACID **> 25%**

CAS number: 7647-01-0 EC number: 231-595-7

Classification
Met. Corr. 1 - H290
Skin Corr. 1B - H314
Eye Dam. 1 - H318
STOT SE 3 - H335

The Full Text for all R-Phrases and Hazard Statements is Displayed in Section 16.

SECTION 4: First aid measures

4.1. Description of first aid measures

Inhalation	Move affected person to fresh air at once. Rinse nose and mouth with water. Get medical attention if any discomfort continues.
Ingestion	Rinse mouth thoroughly with water. Do not induce vomiting. Get medical attention immediately.
Skin Contact	Remove affected person from source of contamination. Remove contaminated clothing. Wash skin thoroughly with soap and water. Get medical attention immediately.

HYDROCHLORIC ACID SOLUTION

Eye contact Remove any contact lenses and open eyelids wide apart. Rinse with water. Continue to rinse for at least 15 minutes. Get medical attention.

4.2. Most important symptoms and effects, both acute and delayed

General information For further information, please refer to section 11.

Inhalation Irritating to respiratory system.

Skin contact Burning pain and severe corrosive skin damage. Corrosive to the respiratory tract.

Eye contact Causes serious eye damage. Corneal damage.

4.3. Indication of any immediate medical attention and special treatment needed

Notes for the doctor Treat symptomatically.

SECTION 5: Firefighting measures

5.1. Extinguishing media

Suitable extinguishing media The product is not flammable. Use fire-extinguishing media suitable for the surrounding fire.

5.2. Special hazards arising from the substance or mixture

Specific hazards In contact with some metals can generate hydrogen gas, which can form explosive mixtures with air.

5.3. Advice for firefighters

Special protective equipment for firefighters Wear positive-pressure self-contained breathing apparatus (SCBA) and appropriate protective clothing.

SECTION 6: Accidental release measures

6.1. Personal precautions, protective equipment and emergency procedures

Personal precautions Provide adequate ventilation. Do not touch or walk into spilled material. Avoid contact with skin, eyes and clothing. Wear protective clothing as described in Section 8 of this safety data sheet.

For non-emergency personnel Keep unnecessary and unprotected personnel away from the spillage.

6.2. Environmental precautions

Environmental precautions Do not discharge into drains or watercourses or onto the ground. If risk of water pollution occurs, notify appropriate authorities. The product may affect the acidity (pH) of water which may have hazardous effects on aquatic organisms.

6.3. Methods and material for containment and cleaning up

Methods for cleaning up Small Spillages: Neutralise spilled material with crushed limestone, slaked lime (calcium hydroxide), soda ash (sodium carbonate) or sodium bicarbonate. Absorb spillage with non-combustible, absorbent material. Collect and place in suitable waste disposal containers and seal securely. Flush contaminated area with plenty of water. Large Spillages: Contain and absorb spillage with sand, earth or other non-combustible material. Inform authorities if large amounts are involved. Collect and place in suitable waste disposal containers and seal securely.

6.4. Reference to other sections

Reference to other sections Wear protective clothing as described in Section 8 of this safety data sheet. Collect and dispose of spillage as indicated in Section 13.

SECTION 7: Handling and storage

7.1. Precautions for safe handling

HYDROCHLORIC ACID SOLUTION

Usage precautions Avoid spilling. Avoid contact with skin and eyes. Use personal protective equipment as required. Wear appropriate clothing to prevent any possibility of skin contact. Provide adequate ventilation.

7.2. Conditions for safe storage, including any incompatibilities

Storage precautions Store in tightly-closed, original container in a dry, cool and well-ventilated place. Store away from incompatible materials (see Section 10). Unsuitable container materials: Metals.

7.3. Specific end use(s)

Specific end use(s) The identified uses for this product are detailed in Section 1.2.

SECTION 8: Exposure Controls/personal protection

8.1. Control parameters

Occupational exposure limits

HYDROCHLORIC ACID

Ceiling value: 5 ppm 7.5 mg/m³

8.2. Exposure controls

Protective equipment

Appropriate engineering controls Provide adequate ventilation. Observe any occupational exposure limits for the product or ingredients. Use process enclosures, local exhaust ventilation or other engineering controls as the primary means to minimise worker exposure.

Eye/face protection Wear tight-fitting, chemical splash goggles or face shield. Personal protective equipment for eye and face protection should comply with European Standard EN166.

Hand protection Wear protective gloves. To protect hands from chemicals, gloves should comply with European Standard EN374. The most suitable glove should be chosen in consultation with the glove supplier/manufacturer, who can provide information about the breakthrough time of the glove material.

Other skin and body protection Provide eyewash station and safety shower. Wear appropriate clothing to prevent any possibility of skin contact.

Hygiene measures Do not smoke in work area. Wash hands at the end of each work shift and before eating, smoking and using the toilet. Wash promptly if skin becomes contaminated. Promptly remove any clothing that becomes contaminated. When using do not eat, drink or smoke.

Respiratory protection If ventilation is inadequate, suitable respiratory protection must be worn. Wear a respirator fitted with the following cartridge: Combination filter, type B+E/P3. Gas and combination filter cartridges should comply with European Standard EN14387.

Environmental exposure controls Emissions from ventilation or work process equipment should be checked to ensure they comply with the requirements of environmental protection legislation. In some cases, fume scrubbers, filters or engineering modifications to the process equipment will be necessary to reduce emissions to acceptable levels.

SECTION 9: Physical and Chemical Properties

9.1. Information on basic physical and chemical properties

Appearance Clear liquid.

Colour Colourless.

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Odour	Odourless.
Odour threshold	Not applicable.
pH	pH (concentrated solution):
Melting point	<-20°C
Initial boiling point and range	109°C
Flash point	Not applicable.
Evaporation rate	No information available.
Flammability Limit - Lower(%)	Not applicable.
Other flammability	No information available.
Vapour pressure	No information available.
Vapour density	No information available.
Relative density	1.161
Partition coefficient	No information available.
Auto-ignition temperature	Not applicable.
Decomposition Temperature	No information available.
Viscosity	Not applicable.
Explosive properties	There are no chemical groups present in the product that are associated with explosive properties.
Oxidising properties	There are no chemical groups present in the product that are associated with oxidising properties.

9.2. Other information

Other information	Not available.

SECTION 10: Stability and reactivity

10.1. Reactivity

Reactivity	The following materials may react violently with the product: Alkalis.

10.2. Chemical stability

Stability	Stable at normal ambient temperatures and when used as recommended.

10.3. Possibility of hazardous reactions

Possibility of hazardous reactions	In contact with some metals can generate hydrogen gas, which can form explosive mixtures with air. Reacts with alkalis and generates heat.

10.4. Conditions to avoid

Conditions to avoid	Avoid excessive heat for prolonged periods of time.

10.5. Incompatible materials

Materials to avoid	Alkalis. Oxidising agents. Metals.

10.6. Hazardous decomposition products

Hazardous decomposition products	Hydrogen chloride (HCl). Hydrogen. Chlorine.

HYDROCHLORIC ACID SOLUTION

SECTION 11: Toxicological information

11.1. Information on toxicological effects

Acute toxicity - oral

Notes (oral LD_{50}) Data lacking.

Acute toxicity - dermal

Notes (dermal LD_{50}) Scientifically unjustified. Corrosive to skin.

Skin corrosion/irritation

Animal data Causes severe skin burns and eye damage.

Serious eye damage/irritation

Serious eye damage/irritation Causes serious eye damage.

Skin sensitisation

Skin sensitisation Not sensitising.

Germ cell mutagenicity

Genotoxicity - in vitro Does not contain any substances known to be mutagenic. Based on available data the classification criteria are not met.

Carcinogenicity

Carcinogenicity Does not contain any substances known to be carcinogenic. Based on available data the classification criteria are not met.

Reproductive toxicity

Reproductive toxicity - development Does not contain any substances known to be toxic to reproduction. Based on available data the classification criteria are not met.

Specific target organ toxicity - repeated exposure

STOT - repeated exposure Based on available data the classification criteria are not met.

SECTION 12: Ecological Information

Ecotoxicity Not regarded as dangerous for the environment.

12.1. Toxicity

12.2. Persistence and degradability

Persistence and degradability The product contains inorganic substances which are not biodegradable.

12.3. Bioaccumulative potential

Bioaccumulative Potential No data available on bioaccumulation.

Partition coefficient No information available.

12.4. Mobility in soil

Mobility Not known.

12.5. Results of PBT and vPvB assessment

Results of PBT and vPvB assessment This product does not contain any substances classified as PBT or vPvB.

12.6. Other adverse effects

Other adverse effects The product may affect the acidity (pH) of water which may have hazardous effects on aquatic organisms.

HYDROCHLORIC ACID SOLUTION

SECTION 13: Disposal considerations

13.1. Waste treatment methods

Disposal methods Dispose of waste to licensed waste disposal site in accordance with the requirements of the local Waste Disposal Authority.

SECTION 14: Transport information

14.1. UN number

UN No. Road 1789

UN No. Sea 1789

UN No., Air 1789

UN No. (ADN) 1789

14.2. UN proper shipping name

UN 1789 HYDROCHLORIC ACID, 8, II, (E)

Proper shipping name (ADR/RID) HYDROCHLORIC ACID

Proper shipping name (IMDG) HYDROCHLORIC ACID

Proper shipping name (ICAO) HYDROCHLORIC ACID

Proper shipping name (ADN) HYDROCHLORIC ACID

14.3. Transport hazard class(es)

ADR Class No. 8

ADR/RID classification code C1

ADR/RID label 8

IMDG Class 8

ICAO Class 8

ADN class 8

Transport labels

14.4. Packing group

ADR Pack Group II

IMDG packing group II

ADN packing group II

Air Pack Gr. II

14.5. Environmental hazards

Environmentally hazardous substance/marine pollutant

No.

14.6. Special precautions for user

HYDROCHLORIC ACID SOLUTION

EmS	F-A, S-B
ADR transport category	2
Emergency Action Code	2R
Hazard Identification Number (ADR/RID)	80
Tunnel restriction code	(E)

14.7. Transport in bulk according to Annex II of MARPOL73/78 and the IBC Code

Transport in bulk according to Annex II of MARPOL 73/78 and the IBC Code	Cat Z

SECTION 15: Regulatory information

15.1. Safety, health and environmental regulations/legislation specific for the substance or mixture

National regulations	Australian Inventory of Chemical Substances (AICS). Listed.
EU legislation	Regulation (EC) No 1272/2008 of the European Parliament and of the Council of 16 December 2008 on classification, labelling and packaging of substances and mixtures (as amended). Regulation (EC) No 1907/2006 of the European Parliament and of the Council of 18 December 2006 concerning the Registration, Evaluation, Authorisation and Restriction of Chemicals (REACH) (as amended).

15.2. Chemical safety assessment

No chemical safety assessment has been carried out.

SECTION 16: Other information

General information	The following information is provided to conform with article 13 of the EC Directive on Packaging and Packaging Waste 94/62/EC: • Wherever possible we use returnable packaging and pallets. Details of these are on our Sales Contracts • For any non-returnable packaging the cost of disposal is at your expense, but we do have a list of reprocessors available • In most cases, but not all, we are able to supply products in returnable packaging but the additional cost of this will be for the customer's expense. Please ask for details with your specific requirements • Any products supplied in returnable packaging is clearly marked to this effect.
Revision date	28/09/2015
Revision	1
SDS No.	20833
Hazard statements in full	H290 May be corrosive to metals. H314 Causes severe skin burns and eye damage. H318 Causes serious eye damage. H335 May cause respiratory irritation.

This information relates only to the specific material designated and may not be valid for such material used in combination with any other materials or in any process. Such information is, to the best of the company's knowledge and belief, accurate and reliable as of the date indicated. However, no warranty, guarantee or representation is made to its accuracy, reliability or completeness. It is the user's responsibility to satisfy himself as to the suitability of such information for his own particular use.

NALCO Champion
An Ecolab Company

SAFETY DATA SHEET

Condor Energy Services CAI500LT

Section: 1. PRODUCT AND COMPANY IDENTIFICATION

Product name	:	Condor Energy Services CAI500LT
Other means of identification	:	Manufactured exclusively for Condor Energy Services by NALCO Champion
Recommended use	:	CORROSION INHIBITOR
Restrictions on use	:	Refer to available product literature or ask your local Sales Representative for restrictions on use and dose limits.
Company	:	ECOLAB PTY LTD 2 Drake Avenue Macquarie Park NSW 2113 Australia A.B.N. 59 000 449 990 TEL: 1300 654 224 FAX: +61 2 8870 8680
Emergency telephone number	:	1800 205 506 International: +64 7 958 2372
Issuing date	:	04.06.2019

Section: 2. HAZARDS IDENTIFICATION

GHS Classification

Flammable liquids	:	Category 2
Skin corrosion/irritation	:	Category 2
Serious eye damage/eye irritation	:	Category 1
Skin sensitization	:	Category 1
Specific target organ toxicity - single exposure	:	Category 3 (Central Nervous System)

GHS Label element

Hazard pictograms :

Signal Word : Danger

Hazard Statements :
Highly flammable liquid and vapour.
Causes skin irritation.
May cause an allergic skin reaction.
Causes serious eye damage.
May cause drowsiness or dizziness.

Precautionary Statements :
Prevention:
Keep away from heat/sparks/open flames/hot surfaces. - No smoking. Avoid breathing dust/ fume/ gas/ mist/ vapours/ spray. Wear protective gloves/ eye protection/ face protection.
Response:
IF ON SKIN (or hair): Remove/ Take off immediately all contaminated clothing. Rinse skin with water/ shower. IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing. Call a POISON CENTER or doctor/ physician if you feel unwell. IF IN EYES: Rinse cautiously with water for

several minutes. Remove contact lenses, if present and easy to do so. Continue rinsing.
Storage:
Store in a well-ventilated place.
Disposal:
Dispose of contents/ container to an approved waste disposal plant.

Other hazards : None known.

Section: 3. COMPOSITION/INFORMATION ON INGREDIENTS

Pure substance/mixture : Mixture

Chemical Name	CAS-No.	Concentration: (%)
Isopropanol	67-63-0	30 - 60
Ethoxylated C12-C16 Alcohol	68551-12-2	10 - 30
Ethoxylated Decanol	26183-52-8	5 - 10
Cinnamaldehyde	104-55-2	5 - 10
Ethoxylated Tallow Alkyl Amine	61791-26-2	1 - 5
Methanol	67-56-1	0.1 - 1

Section: 4. FIRST AID MEASURES

In case of eye contact : Rinse immediately with plenty of water, also under the eyelids, for at least 15 minutes. Remove contact lenses, if present and easy to do. Continue rinsing. Get medical attention immediately.

In case of skin contact : Wash off immediately with plenty of water for at least 15 minutes. Use a mild soap if available. Wash clothing before reuse. Thoroughly clean shoes before reuse. Get medical attention immediately.

If swallowed : Rinse mouth with water. Do NOT induce vomiting. Never give anything by mouth to an unconscious person. Aspiration hazard if swallowed - can enter lungs and cause damage. Get medical attention immediately.

Contact the Poison's Information Centre (eg Australia 13 1126; New Zealand 0800 764 766).

If inhaled : Remove to fresh air. Treat symptomatically. Get medical attention if symptoms occur.

Protection of first-aiders : In event of emergency assess the danger before taking action. Do not put yourself at risk of injury. If in doubt, contact emergency responders. Use personal protective equipment as required.

Notes to physician : Treat symptomatically.

Most important symptoms and effects, both acute and delayed : See Section 11 for more detailed information on health effects and symptoms.

Section: 5. FIREFIGHTING MEASURES

Suitable extinguishing media : Use extinguishing measures that are appropriate to local circumstances and the surrounding environment.

Unsuitable extinguishing media : High volume water jet

Specific hazards during firefighting	:	Fire Hazard Keep away from heat and sources of ignition. Flash back possible over considerable distance. Beware of vapours accumulating to form explosive concentrations. Vapours can accumulate in low areas.
Hazardous combustion products	:	Carbon oxides nitrogen oxides (NOx) Sulphur oxides Hydrogen chloride
Special protective equipment for firefighters	:	Use personal protective equipment.
Specific extinguishing methods	:	Use water spray to cool unopened containers. Collect contaminated fire extinguishing water separately. This must not be discharged into drains. Fire residues and contaminated fire extinguishing water must be disposed of in accordance with local regulations. In the event of fire and/or explosion do not breathe fumes.
Hazchem Code	:	•3YE

Section: 6. ACCIDENTAL RELEASE MEASURES

Initial Emergency Response Guide No	:	14
Personal precautions, protective equipment and emergency procedures	:	Ensure adequate ventilation. Remove all sources of ignition. Keep people away from and upwind of spill/leak. Avoid inhalation, ingestion and contact with skin and eyes. When workers are facing concentrations above the exposure limit they must use appropriate certified respirators. Ensure clean-up is conducted by trained personnel only. Refer to protective measures listed in sections 7 and 8.
Environmental precautions	:	Do not allow contact with soil, surface or ground water.

Section: 7. HANDLING AND STORAGE

Advice on safe handling	:	Avoid contact with skin and eyes. Take necessary action to avoid static electricity discharge (which might cause ignition of organic vapours). Do not ingest. Keep away from fire, sparks and heated surfaces. Do not breathe dust/fume/gas/mist/vapours/spray. Do not get in eyes, on skin, or on clothing. Wash hands thoroughly after handling. Use only with adequate ventilation.
Conditions for safe storage	:	Keep away from heat and sources of ignition. Keep in a cool, well-ventilated place. Keep away from oxidizing agents. Keep out of reach of children. Keep container tightly closed. Store in suitable labelled containers.
Suitable material	:	Keep in properly labelled containers.
Unsuitable material	:	not determined

Section: 8. EXPOSURE CONTROLS/PERSONAL PROTECTION

Components with workplace control parameters

Components	CAS-No.	Form of exposure	Permissible concentration	Basis
Isopropanol	67-63-0	TWA	400 ppm 983 mg/m3	AU OEL

		VLE	500 ppm 1,230 mg/m3	AU OEL
Isopropanol	67-63-0	WES-TWA	400 ppm 983 mg/m3	NZ OEL
		WES-STEL	500 ppm 1,230 mg/m3	NZ OEL
Isopropanol	67-63-0	TWA	200 ppm	ACGIH
		STEL	400 ppm	ACGIH
		TWA	400 ppm 980 mg/m3	NIOSH REL
		STEL	500 ppm 1,225 mg/m3	NIOSH REL
		TWA	400 ppm 980 mg/m3	OSHA Z1
Methanol	67-56-1	TWA	200 ppm 262 mg/m3	AU OEL
		VLE	250 ppm 328 mg/m3	AU OEL
Methanol	67-56-1	WES-TWA	200 ppm 262 mg/m3	NZ OEL
		WES-STEL	250 ppm 328 mg/m3	NZ OEL
Methanol	67-56-1	TWA	200 ppm	ACGIH
		STEL	250 ppm	ACGIH
		TWA	200 ppm 260 mg/m3	NIOSH REL
		STEL	250 ppm 325 mg/m3	NIOSH REL
		TWA	200 ppm 260 mg/m3	OSHA Z1

Engineering measures : Effective exhaust ventilation system. Maintain air concentrations below occupational exposure standards.

Personal protective equipment

Eye protection : Safety goggles
Face-shield

Hand protection : Wear the following personal protective equipment:
Nitrile rubber
butyl-rubber
Gloves should be discarded and replaced if there is any indication of degradation or chemical breakthrough.

Skin protection : Personal protective equipment comprising: suitable protective gloves, safety goggles and protective clothing

Respiratory protection : When workers are facing concentrations above the exposure limit they must use appropriate certified respirators.

Hygiene measures : Handle in accordance with good industrial hygiene and safety practice. Remove and wash contaminated clothing before re-use. Wash face, hands and any exposed skin thoroughly after handling. Provide suitable facilities for quick drenching or flushing of the eyes and body in case of contact or splash hazard.

Section: 9. PHYSICAL AND CHEMICAL PROPERTIES

Appearance	:	liquid
Colour	:	clear amber
Odour	:	solvent-like, cinnamon-like
Flash point	:	22.2 °C, Method: Pensky-Martens closed cup
pH	:	4.0 - 6.0,(10 %), (25 °C), 75/25:IPA/H2O
Odour Threshold	:	no data available
Melting point/freezing point	:	Pour point: -34.4 °C
Initial boiling point and boiling range	:	79.5 °C, Method: ASTM D 86
Evaporation rate	:	no data available
Flammability (solid, gas)	:	no data available
Upper explosion limit	:	no data available
Lower explosion limit	:	no data available
Vapour pressure	:	23.4 hPa, (24 °C), ASTM D 5191, 135.8 hPa, (37.8 °C), ASTM D 5191,
Relative vapour density	:	no data available
Relative density	:	0.8856 - 0.9447, (20 °C),
Density	:	no data available
Water solubility	:	dispersible
Solubility in other solvents	:	no data available
Partition coefficient: n-octanol/water	:	no data available
Auto-ignition temperature	:	no data available
Thermal decomposition	:	no data available
Viscosity, dynamic	:	11.4 mPa.s (22 °C)
Viscosity, kinematic	:	no data available
Molecular weight	:	no data available
VOC	:	no data available

Section: 10. STABILITY AND REACTIVITY

Reactivity	:	No dangerous reaction known under conditions of normal use.
Chemical stability	:	Stable under normal conditions.
Possibility of hazardous reactions	:	No dangerous reaction known under conditions of normal use.
Conditions to avoid	:	Heat, flames and sparks.
Incompatible materials	:	Contact with strong oxidizers (e.g. chlorine, peroxides, chromates, nitric acid, perchlorate, concentrated oxygen, permanganate) may generate heat, fires, explosions and/or toxic vapors.
Hazardous decomposition products	:	In case of fire, hazardous decomposition products may be produced such as: Carbon oxides

nitrogen oxides (NOx)
Sulphur oxides
Hydrogen chloride

Section: 11. TOXICOLOGICAL INFORMATION

Information on likely routes of exposure : Inhalation, Eye contact, Skin contact

Potential Health Effects

Eyes : Causes serious eye damage.

Skin : Causes skin irritation. May cause allergic skin reaction.

Ingestion : Health injuries are not known or expected under normal use.

Inhalation : May cause drowsiness or dizziness.

Experience with human exposure

Eye contact : Redness, Pain, Corrosion

Skin contact : Redness, Pain, Irritation, Corrosion, Allergic reactions

Ingestion : Corrosion, Vomiting, Abdominal pain

Inhalation : Respiratory irritation, Cough, Dizziness, Drowsiness

Toxicity

Product

Acute oral toxicity	:	Acute toxicity estimate: > 2,000 mg/kg
Acute inhalation toxicity	:	Acute toxicity estimate: > 20 mg/l Exposure time: 4 h Test atmosphere: vapour
Acute dermal toxicity	:	Acute toxicity estimate: > 2,000 mg/kg
Skin corrosion/irritation	:	Result: Skin irritation
Serious eye damage/eye irritation	:	Result: Causes serious eye damage.
Respiratory or skin sensitization	:	no data available
Carcinogenicity	:	No component of this product present at levels greater than or equal to 0.1% is identified as probable, possible or confirmed human carcinogen by IARC.
Reproductive effects	:	No toxicity to reproduction
Germ cell mutagenicity	:	Based on available data, the classification criteria are not met.
Teratogenicity	:	no data available
STOT - single exposure	:	May cause drowsiness or dizziness.
STOT - repeated exposure	:	no data available
Aspiration toxicity	:	No aspiration toxicity classification

Human Hazard Characterization

Based on our hazard characterization, the potential human hazard is: High

Section: 12. ECOLOGICAL INFORMATION

Ecotoxicity

Environmental Effects : Harmful to aquatic life.

Product

Toxicity to fish : no data available

Toxicity to daphnia and other aquatic invertebrates : no data available

Toxicity to algae : no data available

Components

Toxicity to fish :

Isopropanol
LC50 Pimephales promelas (fathead minnow): 9,640 mg/l
Exposure time: 96 h

Ethoxylated C12-C16 Alcohol
LC50 : 1.5 mg/l
Exposure time: 96 h

Cinnamaldehyde
LC50 : 103.085 mg/l
Exposure time: 96 h

Ethoxylated Tallow Alkyl Amine
LC50 Fish: 1.1 mg/l
Exposure time: 96 h

Methanol
LC50 : 15,400 mg/l
Exposure time: 96 h

Components

Toxicity to daphnia and other aquatic invertebrates :

Isopropanol
LC50 Daphnia magna (Water flea): > 10,000 mg/l

Cinnamaldehyde
EC50 Daphnia magna (Water flea): 119.56 mg/l
Exposure time: 48 h

Methanol
EC50 : > 10,000 mg/l
Exposure time: 48 h

Components

Toxicity to algae :

Cinnamaldehyde
NOEC : 37.2314 mg/l
Exposure time: 72 h

Methanol
EC50 : 22,000 mg/l
Exposure time: 72 h

Components

Toxicity to bacteria	:	Isopropanol 1,050 mg/l Cinnamaldehyde 8.612 mg/l Methanol > 1,000 mg/l

Components

Toxicity to fish (Chronic toxicity)	:	Methanol NOEC: 7,900 mg/l Exposure time: 8.3 d

Persistence and degradability

no data available

Mobility

no data available

Bioaccumulative potential

no data available

Other information

no data available

ENVIRONMENTAL HAZARD AND EXPOSURE CHARACTERIZATION
Based on our hazard characterization, the potential environmental hazard is: Low

Section: 13. DISPOSAL CONSIDERATIONS

Disposal methods	:	The product should not be allowed to enter drains, water courses or the soil. Where possible recycling is preferred to disposal or incineration. If recycling is not practicable, dispose of in compliance with local regulations. Dispose of wastes in an approved waste disposal facility.
Disposal considerations	:	Dispose of as unused product. Empty containers should be taken to an approved waste handling site for recycling or disposal. Do not re-use empty containers.

Section: 14. TRANSPORT INFORMATION

The shipper/consignor/sender is responsible to ensure that the packaging, labeling, and markings are in compliance with the selected mode of transport.

Land transport

Proper shipping name	:	FLAMMABLE LIQUID, N.O.S.
Technical name(s):	:	Isopropanol
UN/ID No.	:	UN 1993
Transport hazard class(es)	:	3

Packing group	:	II
IERG No	:	14
Hazchem Code	:	•3YE

Air transport (IATA)

UN/ID No.	:	UN 1993
Proper shipping name	:	FLAMMABLE LIQUID, N.O.S.
Technical name(s)	:	Isopropanol
Transport hazard class(es)	:	3
Packing group	:	II

Sea transport (IMDG/IMO)

UN/ID No.	:	UN 1993
Proper shipping name	:	FLAMMABLE LIQUID, N.O.S.
Technical name(s)	:	Isopropanol
Transport hazard class(es)	:	3
Packing group	:	II

Section: 15. REGULATORY INFORMATION

Standard for the Uniform Scheduling of Medicines and Poisons : Schedule 5

INTERNATIONAL CHEMICAL CONTROL LAWS :

Australia. Industrial Chemical (Notification and Assessment) Act
not determined

Section: 16. OTHER INFORMATION

REFERENCES

Hazardous Substances Data Bank, National Library of Medicine, Bethesda, Maryland (TOMES CPS™ CD-ROM Version), Micromedex, Inc., Englewood, CO.

IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man, Geneva: World Health Organization, International Agency for Research on Cancer.

Integrated Risk Information System, U.S. Environmental Protection Agency, Washington, D.C. (TOMES CPS™ CD-ROM Version),
Micromedex, Inc., Englewood, CO.

Annual Report on Carcinogens, National Toxicology Program, U.S. Department of Health and Human Services, Public Health Service.

Registry of Toxic Effects of Chemical Substances, National Institute for Occupational Safety and Health, Cincinnati, OH,
(TOMES CPS™ CD-ROM Version), Micromedex, Inc., Englewood, CO.

The Teratogen Information System, University of Washington, Seattle, WA (TOMES CPS™ CD-ROM Version), Micromedex, Inc., Englewood, CO.

Revision Date	:	04.06.2019
Version Number	:	1.0
Prepared By	:	Regulatory Affairs

REVISED INFORMATION: Significant changes to regulatory or health information for this revision is indicated by a bar in the left-hand margin of the SDS.

The information provided in this Safety Data Sheet is correct to the best of our knowledge, information and belief at the date of its publication. The information given is designed only as a guidance for safe handling, use, processing, storage, transportation, disposal and release and is not to be considered a warranty or quality specification. The information relates only to the specific material designated and may not be valid for such material used in combination with any other materials or in any process, unless specified in the text.
For additional copies of an SDS visit www.nalco.com and request access.

SAFETY DATA SHEET

Revision date: 19-Jul-2021

Revision Number 1

1. IDENTIFICATION OF THE MATERIAL AND SUPPLIER

Product identifier

Product Name CA370FE

Product Code(s) 000000069021

Other means of identification

Synonyms Manufactured exclusively for Condor Energy Services by Fusion Technologies (Australia) Pty Ltd

Recommended use of the chemical and restrictions on use

Recommended use Iron control additive.

Uses advised against No information available.

Supplier
Fusion Technologies Australia Pty Ltd
ABN: 50 636 538 960
Street Address: 7 Noble Street
Bridgeman Downs QLD 4035
Australia

Telephone number: +61 (0)460 047 656
Website: www.fusiontechinc.net

Emergency telephone number

Emergency telephone number **1800 033 111 (ALL HOURS)**

Please ensure you refer to the limitations of this Safety Data Sheet as set out in the "Other Information" section at the end of this Data Sheet.

2. HAZARDS IDENTIFICATION

GHS Classification

Not classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS)

Not classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG)

SIGNAL WORD
Not Hazardous

Label elements

Hazard statements
None

Other hazards which do not result in classification
Poisons Schedule (SUSMP) None allocated

3. COMPOSITION/INFORMATION ON INGREDIENTS

Chemical name	CAS No.	Weight-%
Sodium salt of organic acid	-	70-100%
Non-hazardous ingredients	Proprietary	Balance

4. FIRST AID MEASURES

Description of first aid measures

Emergency telephone number Poisons Information Center, Australia: 13 11 26
Poisons Information Center, New Zealand: 0800 764 766

Inhalation Remove to fresh air and keep at rest in a position comfortable for breathing. If symptoms persist, call a physician.

Eye contact Rinse thoroughly with plenty of water, also under the eyelids. If symptoms persist, call a physician.

Skin contact Take off contaminated clothing. Wash skin with soap and water. Get medical attention if irritation develops and persists.

Ingestion Rinse mouth. Do NOT induce vomiting. Drink 1 or 2 glasses of water. Get medical attention.

Most important symptoms and effects, both acute and delayed

Symptoms No information available.

Indication of any immediate medical attention and special treatment needed

Note to physicians Treat symptomatically.

5. FIRE FIGHTING MEASURES

Suitable Extinguishing Media

Suitable Extinguishing Media Water spray or fog is preferred; if water not available use dry chemical, CO2 or regular foam.

Unsuitable extinguishing media No information available.

Specific hazards arising from the chemical

Specific hazards arising from the chemical	Fine dust dispersed in air may ignite.

Special protective actions for fire-fighters

Special protective equipment for fire-fighters	Firefighters should wear self-contained breathing apparatus and full firefighting turnout gear. Use personal protection equipment.

6. ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Personal precautions	Ensure adequate ventilation. Remove all sources of ignition.
For emergency responders	Use personal protection recommended in Section 8.

Environmental precautions

Environmental precautions	See Section 12 for additional Ecological Information.

Methods and material for containment and cleaning up

Methods for containment	Stop leak if you can do it without risk. Cover powder spill with plastic sheet or tarp to minimize spreading. Keep out of drains, sewers, ditches and waterways.
Methods for cleaning up	Take up with inert, damp, non-combustible material using clean non-sparking tools and place into loosely covered plastic containers for later disposal. Avoid generation of dust. Vacuum or sweep material and place in a disposal container. Do not dry sweep dust. Wet dust with water before sweeping or use a vacuum to collect dust.

7. HANDLING AND STORAGE

Precautions for safe handling

Advice on safe handling	Handle in accordance with good industrial hygiene and safety practice. Avoid contact with skin and eyes. Avoid breathing dust or spray mist. Avoid generation of dust.

Conditions for safe storage, including any incompatibilities

Storage Conditions	Keep in a dry, cool and well-ventilated place. Store away from sources of heat or ignition.
Incompatible materials	Strong oxidizing agents.
Poisons Schedule (SUSMP)	None allocated

8. EXPOSURE CONTROLS/PERSONAL PROTECTION

Control parameters

Exposure Limits	No value assigned for this specific material by Safe Work Australia. However, Workplace Exposure Standard(s) for particulates:

Dusts not otherwise classified: 8hr TWA = 10 mg/m^3

As published by Safe Work Australia Workplace Exposure Standards for Airborne Contaminants.

TWA - The time-weighted average airborne concentration of a particular substance when calculated over an eight-hour working day, for a five-day working week.

These Workplace Exposure Standards are guides to be used in the control of occupational health hazards. All atmospheric contamination should be kept to as low a level as is workable. These workplace exposure standards should not be used as fine dividing lines between safe and dangerous concentrations of chemicals. They are not a measure of relative toxicity.

Appropriate engineering controls

Engineering controls Apply technical measures to comply with the occupational exposure limits.

If in the handling and application of this material, safe exposure levels could be exceeded, the use of engineering controls such as local exhaust ventilation must be considered and the results documented. If achieving safe exposure levels does not require engineering controls, then a detailed and documented risk assessment using the relevant Personal Protective Equipment (PPE) (refer to PPE section below) as a basis must be carried out to determine the minimum PPE requirements.

Individual protection measures, such as personal protective equipment

The selection of PPE is dependent on a detailed risk assessment. The risk assessment should consider the work situation, the physical form of the chemical, the handling methods, and environmental factors.

OVERALLS, SAFETY SHOES, CHEMICAL GOGGLES, GLOVES, DUST MASK.

Eye/face protection Wear safety glasses with side shields (or goggles).

Skin and body protection Wear suitable protective clothing.

Hand protection Wear suitable gloves.

Respiratory protection No protective equipment is needed under normal use conditions. If exposure limits are exceeded or irritation is experienced, ventilation and evacuation may be required. If determined by a risk assessment an inhalation risk exists, wear a dust mask/respirator meeting the requirements of AS/NZS 1715 and AS/NZS 1716.

Environmental exposure controls No information available.

9. PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Physical state	Solid
Appearance	Crystalline Powder
Color	White
Odor	Odourless
Odor threshold	No information available.

Property	Values	Remarks • Method
pH	5.5 - 8.0	None known
pH (as aqueous solution)	No data available	None known
Melting point / freezing point	169 - 172°C	None known
Boiling point / boiling range	No data available	None known
Flash point	No data available	None known
Evaporation rate	No data available	None known
Flammability (solid, gas)	No data available	None known
Flammability Limit in Air		None known
Upper flammability or explosive limits	No data available	
Lower flammability or explosive limits	No data available	
Vapor pressure	No data available	None known
Vapor density	No data available	None known
Relative density	No data available	None known
Water solubility	Soluble in water 160 g/L at 20°C	None known
Solubility(ies)	No data available	None known
Partition coefficient	No data available	None known
Autoignition temperature	No data available	None known
Decomposition temperature	No data available	None known
Kinematic viscosity	No data available	None known
Dynamic viscosity	No data available	None known

Other information

10. STABILITY AND REACTIVITY

Reactivity

Reactivity No information available.

Chemical stability

Stability Stable under normal conditions.

Explosion data

Sensitivity to mechanical impact None.

Sensitivity to static discharge None.

Possibility of hazardous reactions

Possibility of hazardous reactions None under normal processing.

Hazardous polymerization Hazardous polymerization does not occur.

Conditions to avoid

Conditions to avoid Heat, flames and sparks. Dust formation.

Incompatible materials

Incompatible materials Strong oxidizing agents.

Hazardous decomposition products

Hazardous decomposition products Carbon oxides.

11. TOXICOLOGICAL INFORMATION

Acute toxicity

Information on likely routes of exposure

Product Information No adverse health effects expected if the chemical is handled in accordance with this Safety Data Sheet and the chemical label. Symptoms or effects that may arise if the chemical is mishandled and overexposure occurs are:

Inhalation Inhalation of dust in high concentration may cause irritation of respiratory system.

Eye contact Mild eye irritation. Dust contact with the eyes can lead to mechanical irritation.

Skin contact May cause irritation.

Ingestion May cause gastrointestinal discomfort if consumed in large amounts.

Symptoms No information available.

Numerical measures of toxicity - Product Information

Numerical measures of toxicity - Component Information

Chemical name	Oral LD50	Dermal LD50	Inhalation LC50
Sodium salt of organic acid	> 5 g/kg (Rat)	-	-

See section 16 for terms and abbreviations

Delayed and immediate effects as well as chronic effects from short and long-term exposure

Skin corrosion/irritation No information available.

Serious eye damage/eye irritation No information available.

Respiratory or skin sensitization No information available.

Germ cell mutagenicity No information available.

Carcinogenicity No information available.

Reproductive toxicity No information available.

STOT - single exposure No information available.

STOT - repeated exposure No information available.

Aspiration hazard No information available.

12. ECOLOGICAL INFORMATION

Ecotoxicity

Ecotoxicity The environmental impact of this product has not been fully investigated. Keep out of waterways.

Persistence and degradability

Persistence and degradability No information available.

Bioaccumulative potential

Bioaccumulation No information available.

Mobility

Mobility in soil No information available.

Other adverse effects

13. DISPOSAL CONSIDERATIONS

Waste treatment methods

Waste from residues/unused products Dispose of in accordance with local regulations. Dispose of waste in accordance with environmental legislation.

Contaminated packaging Dispose of contents/containers in accordance with local regulations.

14. TRANSPORT INFORMATION

ADG
Not classified as Dangerous Goods by the criteria of the Australian Dangerous Goods Code (ADG Code) for transport by Road and Rail; NON-DANGEROUS GOODS.

IATA
Not classified as Dangerous Goods by the criteria of the International Air Transport Association (IATA) Dangerous Goods Regulations for transport by air; NON-DANGEROUS GOODS.

IMDG
Not classified as Dangerous Goods by the criteria of the International Maritime Dangerous Goods Code (IMDG Code) for transport by sea; NON-DANGEROUS GOODS.

15. REGULATORY INFORMATION

Safety, health and environmental regulations/legislation specific for the substance or mixture

National regulations

Australia
Not classified as a hazardous chemical in accordance with the criteria of Safe Work Australia - Globally Harmonized System (GHS)

Not classified as dangerous goods in accordance with the Australian Code for the Transport of Dangerous Goods by Road and Rail (ADG)

See section 8 for national exposure control parameters

Poisons Schedule (SUSMP) None allocated

International Inventories
AICS Complies.

Legend:
- Australian Inventory of Industrial Chemicals

International Regulations

The Montreal Protocol on Substances that Deplete the Ozone Layer Not applicable

The Stockholm Convention on Persistent Organic Pollutants Not applicable

The Rotterdam Convention Not applicable

16. OTHER INFORMATION

Reason(s) For Issue: First Issue Primary SDS

Issuing Date: 19-Jul-2021

This Safety Data Sheet has been prepared by Ixom Operations Pty Ltd (Toxicology and SDS Services).

Revision Note:
The symbol (*) in the margin of this SDS indicates that this line has been revised.

Key or legend to abbreviations and acronyms used in the safety data sheet

Legend Section 8: EXPOSURE CONTROLS/PERSONAL PROTECTION

TWA	TWA (time-weighted average)	STEL	STEL (Short Term Exposure Limit)
Ceiling	Maximum limit value	*	Skin designation
C	Carcinogen		

Key literature references and sources for data used to compile the SDS

EPA (Environmental Protection Agency)
Acute Exposure Guideline Level(s) (AEGL(s))
U.S. Environmental Protection Agency Federal Insecticide, Fungicide, and Rodenticide Act
U.S. Environmental Protection Agency High Production Volume Chemicals
Food Research Journal
Hazardous Substance Database
International Uniform Chemical Information Database (IUCLID)
Japan GHS Classification
Australian Industrial Chemicals Introduction Scheme (AICIS)
NIOSH (National Institute for Occupational Safety and Health)
National Library of Medicine's ChemID Plus (NLM CIP)
National Library of Medicine's PubMed database (NLM PUBMED)
National Toxicology Program (NTP)
New Zealand's Chemical Classification and Information Database (CCID)
Organization for Economic Co-operation and Development Environment, Health, and Safety Publications
Organization for Economic Co-operation and Development High Production Volume Chemicals Program
Organization for Economic Co-operation and Development Screening Information Data Set
RTECS (Registry of Toxic Effects of Chemical Substances)
World Health Organization

Disclaimer

This SDS summarises to our best knowledge at the date of issue, the chemical health and safety hazards of the material and general guidance on how to safely handle the material in the workplace. Since The Supplier cannot anticipate or control the conditions under which the product may be used, each user must, prior to usage, assess and control the risks arising from its use of the material.

If clarification or further information is needed, the user should contact their Supplier representative or The Supplier at the contact details on page 1.

The Supplier's responsibility for the material as sold is subject to the terms and conditions of sale, a copy of which is available upon request.

End of Safety Data Sheet

CAI401HT

SAFETY DATA SHEET

EMERGENCY TELEPHONE NUMBER: 1800 033 111 (ALL HOURS)

Product Name: CAI401HT
Date Issued: Nov 10th, 2022

Prepared by: Fusion Australia
& Version: A21-1.0

1. PRODUCT IDENTIFICATION AND COMPANY IDENTIFICATION

Product Name:	CAI401HT
Product Purpose:	Acid Corrosion Inhibitor
Supplier Identification:	Fusion Technologies (Australia) Pty Ltd. 7 Noble Street Bridgeman Downs QLD, 4035 Australia

PREPARER'S TELEPHONE NUMBER: +61 4600 47 656

2. HAZARDS IDENTIFICATION

Hazard Pictograms:

Signal word: Danger

Primary Routes of Exposure: Inhalation, Skin contact, eye contact

GHS Classification in accordance with WHMIS 2015
Serious eye damage (Category 1), H318
Acute toxicity, Dermal (Category 4), H311
Acute toxicity, Oral (Category 3), H302
Specific target organ toxicity - single exposure - Kidneys (Category 2), H370

Hazard Statements: H318 – Causes serious eye damage

H302+H312 – Harmful if swallowed or in contact with skin
H370 - Causes damage to organs

Precautionary Statements:

P210 - Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260 - Do not breathe dust/ fume/ gas/ mist/ vapours/ spray.
P264 - Wash thoroughly after handling
P280 + P361 + P364 - Wear protective gloves/ protective clothing/ eye protection. Rinse immediately contaminated clothing and skin with plenty of water before removing clothes and wash before re-use.
P302 + P352 + P312 - If on skin: Wash with plenty of water. Call a POISON CENTER or doctor/ physician if you feel unwell.
P304 - If inhaled: Remove person to fresh air
P308 + P311 - If exposed or concerned: Call a POISON CENTER or doctor/ physician.
P403 + P233 – Store in a well-ventilated place. Keep container tightly closed.

Hazards not otherwise classified (HNOC) or not covered by GHS – *none*

Acute Effects: None known

3. PRODUCT COMPOSITION/INGREDIENTS

Chemical Name	CAS #	% by Weight
Ethylene Glycol	107-21-1	40 to 70
Cinnamaldehyde	104-55-2	10 to 30
Formic Acid	64-18-6	10 to 30
Alkylpyridine Quat	68909-18-2	5 to 20
2-Ethylhexanol PO/EO polymer	64366-70-7	5 to 20

4. FIRST AID MEASURES

Eye Contact: Rinse eyes immediately with copious amounts of water and under the eyelids for at least 15 minutes. If symptoms persist seek medical advice.

Skin Contact: Remove contaminated clothing and footwear. Immediately wash off all material with soap and copious amounts of water for at least 15 minutes. Contaminated clothing must be washed before reuse. Thoroughly clean contaminated shoes.

Ingestion: If swallowed, do not induce vomiting. Never give anything by mouth to an unconscious person. Obtain medical advice.

Inhalation: Remove victim to fresh air, treat symptomatically. If symptoms develop, seek medical advice.

5. FIRE FIGHTING MEASURES

Suitable extinguishing media: Use DRY chemicals, carbon dioxide, and dry powder. Water spray for larger fires is acceptable. NEVER use a water jet directly on the fire because it may spread to a larger area.

Unsuitable extinguishing media: High volume water jet

Specific hazards during firefighting: May evolve toxic fumes of oxides of carbon, nitrogen and/or sulphur under fire conditions. Formaldehyde.

	Vapors may travel to ignition source and flash back. Empty containers may contain product residue. Heating can release hazardous gases. Vapors may be ignited by static discharge.
Hazardous combustion products:	Decomposition products may include the following materials: Carbon oxides
Special protective equipment for firefighters:	Use personal protective equipment.
Specific extinguishing methods:	Fire residues and contaminated fire extinguishing water must be disposed of in accordance with local regulations.

6. ACCIDENTAL RELEASE MEASURES

Personal Precautions:	Avoid contact with skin, eyes and clothing. Evacuate personnel to safe areas. Keep people away from and upwind of spill or leak. PPE: see section 8.
Environmental Precautions:	Do not contaminate surface water. Do not release into the environment. Prevent product from entering any drains. Do not flush product into surface water or sanitary sewer systems. Harmful to aquatic organisms.
Emergency Procedures:	Prevent further leakage or spillage if safe to do so.
Methods For Cleaning Up:	Soak up spill with absorbent material and then place into an appropriate waste container. Remove soiled refuse and place in a suitable disposal container. Use non-sparking tools.
Disposal:	Dispose of material in compliance with local, Provincial and Federal regulations. See Section 13.

7. HANDLING AND STORAGE

Handling Precautions:	Handle wearing appropriate PPE as per section 8. Ensure adequate ventilation is available.
Storage Precautions:	Store in a cool, dry, well-ventilated area, away from heat and ignition sources. Tanks must be grounded and vented and should have vapor

	emission controls. Tanks must be diked. Place away from incompatible materials. All equipment must be grounded - bonded when transferring product in order to avoid static discharge from the equipment, and subsequent possible fire.
Suitable material:	Some attack: Polyethylene Satisfactory: Neoprene, phenolic resins, polyesters, natural rubber, butyl rubber Resistant: Polyvinyl chloride, unplasticized. Fixed storage containers, transfer containers and associated equipment should be grounded and bonded to prevent accumulation of static charge. Store in accordance with good industrial practices.Keep in properly labelled containers.

8. EXPOSURE CONTROLS/PERSONAL PROTECTION

Occupational Exposure Limits:	*This product does contain substances that have an established exposure limit.* Ethylene Glycol: ACGIH – Aerosol (100mg/m³) CA OEL – TWA Particulate (10mg/m³) Formic acid: ACGIH – TWA (5 ppm) ACGIH – STEL (10 ppm)
Engineering Measures:	Use process enclosure, local exhaust ventilation, or other engineering controls to keep airborne levels below recommended exposure limits. Use explosion proof equipment.
Hygiene Recommendations:	Keep an eye wash fountain and safety shower available

Eye Protection:	Wear safety glasses with side shields and goggles/side shield where splashing hazard exists.
Hand Protection:	Appropriate chemical resistant gloves should be worn. Viton gloves. Polyvinyl alcohol gloves. Nitrile gloves. Butyl rubber gloves.
Respiratory Protection:	Respirator selection must be done by a qualified person and be based upon a risk assessment of the work activities and exposure levels. Respirators must be fit tested and users must be clean shaven where the respirator seals to the face. Exposure must be kept at or below the applicable exposure limits and the maximum use concentration of the respirator must not be exceeded. Positive pressure, full-face piece self-contained breathing apparatus; or Positive pressure, full-face piece supplied air respirator with an auxiliary positive pressure self-contained breathing apparatus.
Skin and Body Protection:	Wear chemical resistant pants and jackets, preferably butyl or nitrile rubber.

9. PHYSICAL AND CHEMICAL PROPERTIES

Appearance:	Liquid
Colour:	Brown
Odour:	Characteristic
Flash point:	>100°C
pH:	1-3
Odour Threshold:	No data available
Melting point/freezing point:	-25°C
Initial boiling point and boiling range:	No data available
Evaporation rate:	No data available
Flammability (solid, gas):	No data available
Upper explosion limit:	No data available
Lower explosion limit:	No data available
Vapour pressure:	No data available

Relative vapour density:	No data available
Relative density:	1.12
Water solubility:	Soluble
Solubility in other solvents:	No data available
Partition coefficient: n-octanol/water:	No data available
Auto-ignition temperature:	No data available
Thermal decomposition temperature:	No data available
Viscosity, dynamic:	No data available
Viscosity, kinematic:	No data available
Molecular weight:	No data available
VOC:	No data available

10. STABILITY AND REACTIVITY

Stability:	Stable under normal conditions
Conditions to Avoid:	Temperature extremes, sources of heat and ignition and static discharge.
Materials to Avoid:	Contact with strong oxidizers as they may generate heat, fires, explosions and/or toxic vapors. Strong bases, organic acids, acetyl bromide, magnesium, strong mineral acids, aluminum powder, aluminum alkyl compounds. May attack some forms of plastic, rubber and coatings.
Hazardous Polymerization:	Will not occur
Hazardous Decomposition Products:	Oxides of carbon, nitrogen. Formaldehyde.

11. TOXICOLOGICAL INFORMATION

Acute LD50/oral:	7712 mg/kg (rat) (Ethylene Glycol)
Acute LC50/inhalation:	>2.5 mg/L (rat) (Ethylene Glycol), 6hr
Acute LD50/dermal:	>10,600 mg/kg (rabbit) (Ethylene Glycol)

Mutagenic Effects:	Not expected
Reproductive Toxicity:	Ingestion of large amounts of ethylene glycol has been shown to interfere with reproduction in animals.
Teratogenicity and Embryo Toxicity:	Ingestion of large amounts of ethylene glycol may produce birth defects.
Human Experience:	Based on hazard characterization, the potential human hazard is moderate.
Other Toxicity Information:	Ingestion of ethylene glycol can cause damage to liver and other organs.

12. ECOLOGICAL INFORMATION

Ingredients	Ecotoxicity - Fish Species Data	Acute Crustaceans Toxicity:	Ecotoxicity - Freshwater Algae Data
Ethylene Glycol	LC50, Pimephales promelas, static test, 96hr, 72,850mg/L	EC50, Daphnia magna, static test, 48hr, >100mg/l	EC50, activated sludge, 30min, 225mg/l
Formic Acid	LEC50, Danio rerio, static test, 96hr, 130mg/l	EC50, Daphnia magna, static test, 48hr, 365mg/l	ErC50, Psueodkirchneriella subcapitata, 72hr, 1240 mg/l

Other Information:
Do not allow product or runoff from fire control to enter storm or sanitary sewers, lakes, rivers, streams or public waterways. Block off drains and ditches. Spill areas must be cleaned and restored to original condition or to the satisfaction of authorities.

13. DISPOSAL INFORMATION

Waste Residues/Unused Product and Package	Dispose of waste containers in accordance with all applicable regulations.

14. TRANSPORT INFORMATION

Typical proper shipping name for this product are as follows:

Corrosive Liquid, Acidic, Organic, N.O.S	**CLASS 8**	**UN3265**	**PKG GRP: III**

Important Note: This information does not take the place of shipping paper (Bill of Lading or BOL)

15. REGULATORY INFORMATION

Australian Inventory of Chemical Substances (AICS)
May require notification before sale under Australian regulations.

All substances in this product comply with the National Industrial Chemicals Notification & Assessment Scheme (NICNAS).

CANADA: Workplace Hazardous Material Information System (WHMIS)
This product has been classified in accordance with the hazard criteria of the Hazardous Products Regulations (HPR) and is a WHMIS controlled product.

Canadian Environmental Protection Act (CEPA): The substance(s) in this SDS are included in or exempted from the Domestic Substance List (DSL)

National Pollutant Release Inventory (NPRI): This product contains the following substances listed in Part 1A (Core Substances) of the NPRI at a concentration of one percent or more by weight.

U.S. Regulatory Rules

Toxic Substances Control Act (TSCA): The substances in this SDS are included in or exempted from the TSCA 8(b) Inventory (40 CFR 710)

This section contains additional information that may have relevance to regulatory compliance. The information contained in this section is for reference only. Hybrid Chemical Technologies accepts no liability for the use of this information.

This section contains additional information that may have relevance to regulatory compliance. The information contained in this section is for reference only. Fusion Australia accepts no liability for the use of this information.

16. OTHER INFORMATION

NFPA 704M RATING

Health: 3	Flammability: 1	Reactivity: 0	Other: n/a

HMIS

Health: 3	Flammability: 1	Instability: 0	Other: n/a

0= insignificant 1= slight 2= moderate 3= high 4= Extreme * = Chronic Hazard

This is the Last Page of SDS

Disclaimer

This material safety data sheet provides health and safety information for the safe use of this product provided it is used as recommended per the associated product literature. Users of this product should be aware of the recommended safety precautions. For any other use, exposures must be evaluated so that appropriate handling and training programs can be created and implemented to insure safe workplace operations. Consult with Fusion Technologies for any additional information.

Condor Energy Services – Safety Data Sheet

CF 200

1. CHEMICAL PRODUCT AND COMPANY IDENTIFICATION

PRODUCT NAME: **CF 200**

APPLICATION: Friction Reducer

IMPORTER IDENTIFICATION:
Condor Energy Services Ltd
Level 4, 15 Ogilvie Road
Applecross WA 6153
Australia
+61 8 9315 5986

EMERGENCY TELEPHONE NUMBER(S): +61 430 138 290 (24 Hours)

+65 6542 9595

2. HAZARDS IDENTIFICATION

HAZARD CLASSIFICATION :

Not classified as hazardous according to Safe Work Australia. This product is not classified as a dangerous good according to national or international regulations.

SAFETY PHRASES
S24/25 - Avoid contact with skin and eyes.
S36/37/39 - Wear suitable protective clothing, gloves and eye/face protection.

3. COMPOSITION/INFORMATION ON INGREDIENTS

CHEMICAL NAME	CAS NO	% (w/w)
Ingredients determined not to be hazardous		100

4. FIRST AID MEASURES

EYE CONTACT :
Flush affected area with water. If symptoms develop, seek medical advice.

SKIN CONTACT :
Flush affected area with water. If symptoms develop, seek medical advice.

INGESTION :
DO NOT INDUCE VOMITING. If conscious, washout mouth and give water to drink. If symptoms develop, seek medical advice.

INHALATION :
Remove to fresh air, treat symptomatically. If symptoms develop, seek medical advice.

NOTE TO PHYSICIAN :
Based on the individual reactions of the patient, the physician's judgement should be used to control symptoms and clinical condition.

5. FIRE FIGHTING MEASURES

FLASH POINT : Not flammable

EXTINGUISHING MEDIA :
This product would not be expected to burn unless all the water is boiled away. The remaining organics may be ignitable. Use extinguishing media appropriate for surrounding fire.

FIRE AND EXPLOSION HAZARD :
May evolve oxides of carbon (COx) under fire conditions. May evolve oxides of nitrogen (NOx) and sulfur (SOx) under fire conditions.

SPECIAL PROTECTIVE EQUIPMENT FOR FIRE FIGHTING :
In case of fire, wear a full face positive-pressure self contained breathing apparatus and protective suit.

SENSITIVITY TO STATIC DISCHARGE :
Not expected to be sensitive to static discharge.

6. ACCIDENTAL RELEASE MEASURES

PERSONAL PRECAUTIONS :
Restrict access to area as appropriate until clean-up operations are complete. Use personal protective equipment recommended in Section 8 (Exposure Controls/Personal Protection). Stop or reduce any leaks if it is safe to do so. Ventilate spill area if possible. Notify appropriate government, occupational health and safety and environmental authorities.

METHODS FOR CLEANING UP :
SMALL SPILLS: Soak up spill with absorbent material. Place residues in a suitable, covered, properly labeled container. Wash affected area. LARGE SPILLS: Contain liquid using absorbent material, by digging trenches or by diking. Reclaim into recovery or salvage drums or tank truck for proper disposal. Clean contaminated surfaces with water or aqueous cleaning agents. Contact an approved waste hauler for disposal of contaminated recovered material. Dispose of material in compliance with regulations indicated in Section 13 (Disposal Considerations).

ENVIRONMENTAL PRECAUTIONS :
Do not contaminate surface water.

7. HANDLING AND STORAGE

HANDLING :
Do not get in eyes, on skin, on clothing. Do not take internally. Use with adequate ventilation. Keep the containers closed when not in use. Ensure all containers are labeled.

STORAGE CONDITIONS :
Store in suitable labeled containers. Store the containers tightly closed. Store separately from oxidizers.

SUITABLE CONSTRUCTION MATERIAL :
Stainless Steel 304, Neoprene, Viton, Buna-N, Polypropylene, Polyethylene, Polyurethane, EPDM, Epoxy phenolic resin, HDPE (high density polyethylene), PVC

UNSUITABLE CONSTRUCTION MATERIAL :
Brass, Hypalon, Mild steel

8. EXPOSURE CONTROLS/PERSONAL PROTECTION

OCCUPATIONAL EXPOSURE LIMITS

None of the components have been assigned an exposure standard by Safe Work Australia (Australia) or EPA (New Zealand).

ENGINEERING MEASURES :
General ventilation is recommended.

PERSONAL PROTECTION

RESPIRATORY PROTECTION :
Respiratory protection is not normally needed.

HAND PROTECTION :
NEOPRENE, NITRILE, OR PVC GLOVES Breakthrough time not determined as preparation, consult PPE manufacturers.

SKIN PROTECTION :
Wear standard protective clothing.

EYE PROTECTION :
Wear safety glasses with side-shields.

HYGIENE RECOMMENDATIONS :
Use good work and personal hygiene practices to avoid exposure. Keep an eye wash fountain available. Keep a safety shower available. If clothing is contaminated, remove clothing and thoroughly wash the affected area. Launder contaminated clothing before reuse. Always wash thoroughly after handling chemicals. When handling this product never eat, drink or smoke.

ENVIRONMENTAL EXPOSURE CONTROL PRECAUTIONS :
Consider the provision of containment around storage vessels.

9. PHYSICAL AND CHEMICAL PROPERTIES

PHYSICAL STATE	Liquid
APPEARANCE	Milky White
ODOR	Mild
pH	No data available.
VAPOR PRESSURE	No data available.
VAPOR DENSITY	No data available.
SPECIFIC GRAVITY	1.198 - 1.225 (23.88 °C)
DENSITY	No data available.
SOLUBILITY IN WATER	Complete
OCTANOL/WATER COEFFICIENT (log Kow)	-0.9 Product (estimated) OECD 117
MELTING POINT	No data available.
BOILING POINT	No data available.
FLASH POINT	Not flammable
LOWER EXPLOSION LIMIT	No data available.
UPPER EXPLOSION LIMIT	No data available.
AUTOIGNITION TEMPERATURE	No data available.

Note: These physical properties are typical values for this product and are subject to change.

10. STABILITY AND REACTIVITY

STABILITY :
Stable under normal conditions.

CONDITIONS TO AVOID
: Extremes of
temperature

INCOMPATIBLE MATERIALS :
Contact with strong oxidizers (e.g. chlorine, peroxides, chromates, nitric acid, perchlorate, concentrated oxygen, permanganate) may generate heat, fires, explosions and/or toxic vapors. SO2 may react with vapors from neutralizing amines and may produce a visible cloud of amine salt particles.

HAZARDOUS DECOMPOSITION PRODUCTS :
Under fire conditions: Oxides of carbon, Oxides of nitrogen, Oxides of sulfur

HAZARDOUS REACTIONS :
Hazardous polymerization will not occur.

11. TOXICOLOGICAL INFORMATION

OVERVIEW OF HEALTH HAZARDS

ACUTE HAZARDS - EYE CONTACT
May cause irritation with prolonged contact.

ACUTE HAZARDS - SKIN CONTACT
May cause irritation with prolonged contact.

ACUTE HAZARDS - INGESTION
Not a likely route of exposure. No adverse effects expected.

ACUTE HAZARDS - INHALATION
Not a likely route of exposure. No adverse effects expected.

CHRONIC HAZARDS :
No adverse effects expected other than those mentioned above.

SUMMARY OF TOXICITY INFORMATION

ACUTE TOXICITY DATA :
No toxicity studies have been conducted on this product.

SENSITIZATION :
This product is not expected to be a sensitizer.

CARCINOGENICITY :
None of the substances in this product are listed as carcinogens by the International Agency for Research on Cancer (IARC), the National Toxicology Program (NTP) or the American Conference of Governmental Industrial Hygienists (ACGIH).

For additional information on the hazard of the preparation, please consult section 2 and 12.

HUMAN HAZARD CHARACTERIZATION
Based on our hazard characterization, the potential human hazard is: Low

12. ECOLOGICAL INFORMATION

ECOTOXICOLOGICAL EFFECTS:

The following results are for the product.

AQUATIC PLANT RESULTS :

Species	Exposure	Test Type	Value	Test Descriptor
Marine Algae (Skeletonema costatum)	72 hrs	LC50	165.54 mg/l	Product
Marine Algae (Skeletonema costatum)	72 hrs	NOEC	10 mg/l	Product

MOBILITY AND BIOACCUMULATION POTENTIAL :

The environmental fate was estimated using a level III fugacity model embedded in the EPI (estimation program interface) Suite TM, provided by the US EPA. The model assumes a steady state condition between the total input and output. The level III model does not require equilibrium between the defined media. The information provided is intended to give the user a general estimate of the environmental fate of this product under the defined conditions of the models.

If released into the environment this material is expected to distribute to the air, water and soil/sediment in the approximate respective percentages;

Air	Water	Soil/Sediment
<5%	10 - 30%	70 - 90%

The portion in water is expected to be soluble or dispersible.

This preparation or material is not expected to bioaccumulate.

PERSISTENCY AND DEGRADATION :

The organic portion of this preparation is expected to be inherently biodegradable.

ENVIRONMENTAL HAZARD AND EXPOSURE CHARACTERIZATION
Based on our hazard characterization, the potential environmental hazard is: Moderate

13. DISPOSAL CONSIDERATIONS

Dispose of wastes in an approved waste treatment / disposal site, in accordance with all applicable regulations. Do not dispose of wastes in local sewer or with normal garbage.

Triple rinse (or equivalent) all containers and offer for recycling or reconditioning, or puncture and dispose of in a sanitary landfill, or by other procedures approved by state and local authorities.

14. TRANSPORT INFORMATION

The information in this section is for reference only and should not take the place of a shipping paper (bill of lading) specific to an order. Please note that the proper Shipping Name / Hazard Class may vary by packaging, properties, and mode of transportation. Typical Proper Shipping Names for this product are as follows.

LAND TRANSPORT
Proper Shipping Name : PRODUCT IS NOT REGULATED DURING TRANSPORTATION

AIR TRANSPORT (ICAO/IATA)
Proper Shipping Name : PRODUCT IS NOT REGULATED DURING TRANSPORTATION

MARINE TRANSPORT (IMDG/IMO)
Proper Shipping Name : PRODUCT IS NOT REGULATED DURING TRANSPORTATION

15. REGULATORY INFORMATION

AUSTRALIA :

NICNAS
All substances in this product comply with the National Industrial Chemicals Notification & Assessment Scheme (NICNAS).

SUSDP SCHEDULE : Not Listed

16. OTHER INFORMATION

This product material safety data sheet provides health and safety information. The product is to be used in applications consistent with our product literature. Individuals handling this product should be informed of the recommended safety precautions and should have access to this information. For any other uses, exposures should be evaluated so that appropriate handling practices and training programs can be established to insure safe workplace operations. Please consult your local sales representative for any further information.

REFERENCES

Hazardous Substances Data Bank, National Library of Medicine, Bethesda, Maryland (TOMES CPS™ CD-ROM Version), Micromedex, Inc., Englewood, CO.

IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man, Geneva: World Health Organization, International Agency for Research on Cancer.

Integrated Risk Information System, U.S. Environmental Protection Agency, Washington, D.C. (TOMES CPS™ CD-ROM Version),
Micromedex, Inc., Englewood, CO.

Annual Report on Carcinogens, National Toxicology Program, U.S. Department of Health and Human Services, Public Health Service.

Registry of Toxic Effects of Chemical Substances, National Institute for Occupational Safety and Health, Cincinnati, OH,
(TOMES CPS™ CD-ROM Version), Micromedex, Inc., Englewood, CO.

The Teratogen Information System, University of Washington, Seattle, WA (TOMES CPS™ CD-ROM Version), Micromedex, Inc., Englewood, CO.

Prepared By:	Condor Energy HSEQ Department
Date issued:	27 March 2014
Version Number:	1.0

Appendix E Tier 2 Assessment – Avian Wildlife

Table E-1
Tier 2 Assessment - Summary
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Common Name	Scientific Name	Body Mass (Kg)								Drinking WIR (L/day)[3,4]
		Sex[1]	N	Mean	Standard Deviation	Min	Max	Location	Source ID[2]	Mean
Crested Pigeon	*Ocyphaps lophotes*	**B**	**21**	**0.204**	**---**	**0.142**	**0.26**	**Australia**	**515a**	**0.020**
Willie Wagtail	*Rhipidura leucophrys picata*	**B**	**13**	**0.0201**	**---**	**0.0145**	**0.0255**	**Australia**	**518a**	**0.004**
Peaceful Dove	*Geopelia placida*	**B**	**38**	**0.0478**	**---**	**0.035**	**0.065**	**Australia**	**515a**	**0.008**
Cattle Egret	*Bubulcus ibis*	M	27	0.372	---	0.296	0.46	FL, USA	1207	0.0304
Cattle Egret	*Bubulcus ibis*	**F**	**59**	**0.36**	**---**	**0.27**	**0.512**	**FL, USA**	**1207**	**0.0298**
Brown Honeyeater	*Lichmera indistincta*	M	37	0.0118	0.0015	0.009	0.015	Australia	517	0.0030
Brown Honeyeater	*Lichmera indistincta*	**F**	**15**	**0.0106**	**0.0021**	**0.008**	**0.014**	**Australia**	**517**	**0.0028**

Notes:

1, Sex: M, Male; F, Female; B, Both

2, Body mass statistics compiled in Dunning (2008); Original source documents based on Source ID in Dunning (2008) include:

515a, *Higgins, P J and S J J F Davies 1996 Handbook of Australian, New Zealand and Antarctic birds Oxford University Press, Mel-bourne, Australia Volume 3*

518a, *Higgins, P J , J M Peter, and S J Cowling 2006 Handbook of Australian, New Zealand and Antarctic birds Oxford University Press, Melbourne, Australia Volume 7*

1207, *Telfair, R C 1994 Cattle Egret (Bubulcus ibis) In The Birds of North America, A Poole and F Gill (editors) The Birds of North America, Inc , Philadelphia, PA,* and The American Ornithologists' Union, Washington, DC Number 113

517, *Higgins, P J , J M Peter, and W K Steele 2001 Handbook of Australian, New Zealand and Antarctic birds Oxford University Press, Melbourne, Australia Volume 5*

3, Drinking water ingestion rate (WIR) based on the allometric relationship developed by Calder and Braun (1983), where WIR (L/day) = 0.059 x BW $(Kg)^{0.67}$

4, Proposed WIR shown in bold, estimated based on the arithmetic mean of female or combined body mass; WIR may be estimated based on other body mass statistics depending on the appropriate exposure scenario.

kg = kilogram

Table E-2
Tier 2 Assessment - Crested Pigeon
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Constituent Name	CAS No.	Mammal NOAELt	Mammal NOAEL		Avian NOAELt [1]	Avian NOAEL		Avian Receptor	
			Test Animal			Test Animal		Crested Pigeon	
			Animal	Body Weight (kg)		Animal	Body Weight (kg)	Body Weight (kg)	Derived TRV
Glutaraldehyde	111-30-8	4	Rat	0.35	206	Mallard Duck	1.58	0.204	3.4E+02

Notes:
NOAELt = No observed adverse effect level test animal
kg = kilogram
NA = not applicable
TRV = toxicity reference value
1/ If an avian NOAEL was not available, the mammal NOAEL was used to derive the TRV for the avian receptor.

$$Derived\ TRV = NOAEL_{test} * \left(\frac{Body\ Weighttest}{Body\ WeightAvian}\right)^{(1/4)}$$

Exposure Route	Parameter Code	Parameter Definition	Units (a)	Parameter Value	Source (b)
Ingestion	IR	Ingestion rate	l/day	0.020	Table E-1
	EF	Exposure frequency	day/yr	21	BPJ
	ED	Exposure duration	yr	1	BPJ
	BW	Body weight	kg	0.204	Table E-1
	AT-NC	Averaging time - noncancer	days	365	BPJ

Notes:
a/ Units:
l/day = litres per day
day/yr = days per year
yr = year
kg = kilogram
b/ References:
BPJ - Best Professional Judgement

Constituent Name	CAS No.	EPC [1]	Toxicity	Total Intake (mg/kg/day)	Hazard Quotient
		CW (mg/l)	TRVs		Ingestion
Glutaraldehyde	111-30-8	470	3.4E+02	2.7E+00	7.8E-03
				Cumulative:	**7.8E-03**

Notes:
CW = concentration in water
EPC = exposure point concentration
mg/kg/day = milligrams per kilograms per day
mg/l = milligrams per litre
NA = not available/applicable
TRV = toxicity reference value
1/ EPC is injected concentration presented on Table 1.

$$Total\ Intake = \frac{EPC\ \times IR\ \times EF\ \times ED}{BW\ \times ED\ x\ 365\ ^{days}/_{year}}$$

$$Hazard\ Quotient = \frac{Total\ Intake\ \left(\frac{mg}{kg-day}\right)}{TRV\ \left(\frac{mg}{kg-day}\right)}$$

Table E-3
Tier 2 Assessment - Willie Wagtail
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Constituent Name	CAS No.	Mammal NOAELt	Mammal NOAEL		Avian NOAELt [1]	Avian NOAEL		Avian Receptor	
			Test Animal			Test Animal		Willie Wagtail	
			Animal	Body Weight (kg)		Animal	Body Weight (kg)	Body Weight (kg)	Derived TRV
Glutaraldehyde	111-30-8	4	Rat	0.35	206	Mallard Duck	1.58	0.0201	6.1E+02

Notes:
NOAELt = No observed adverse effect level test animal
kg = kilogram
NA = not applicable
TRV = toxicity reference value
1/ If an avian NOAEL was not available, the mammal NOAEL was used to derive the TRV for the avian receptor.

$$Derived\ TRV = NOAEL_{test} * \left(\frac{Body\ Weighttest}{Body\ WeightAvian}\right)^{(1/4)}$$

Exposure Route	Parameter Code	Parameter Definition	Units (a)	Parameter Value	Source (b)
Ingestion	IR	Ingestion rate	l/day	0.004	Table E-1
	EF	Exposure frequency	day/yr	21	BPJ
	ED	Exposure duration	yr	1	BPJ
	BW	Body weight	kg	0.0201	Table E-1
	AT-NC	Averaging time - noncancer	days	365	BPJ

Notes:
a/ Units:
l/day = litres per day
day/yr = days per year
yr = year
kg = kilogram
b/ References:
BPJ - Best Professional Judgement

Constituent Name	CAS No.	EPC [1]	Toxicity	Total Intake (mg/kg/day)	Hazard Quotient
		CW (mg/l)	TRVs		Ingestion
Glutaraldehyde	111-30-8	470	6.1E+02	5.8E+00	9.4E-03
				Cumulative:	**9.4E-03**

Notes:
CW = concentration in water
EPC = exposure point concentration
mg/kg/day = milligrams per kilograms per day
mg/l = milligrams per liter
NA = not available/applicable
TRV = toxicity reference value
1/ EPC is injected concentration presented on Table 1.

$$Total\ Intake = \frac{EPC\ \times IR\ \times EF\ \times ED}{BW\ \times ED\ x\ 365\ ^{days}/_{year}}$$

$$Hazard\ Quotient = \frac{Total\ Intake\ \left(\frac{mg}{kg-day}\right)}{TRV\ \left(\frac{mg}{kg-day}\right)}$$

Table E-4
Tier 2 Assessment - Peaceful Dove
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Constituent Name	CAS No.	Mammal NOAELt	Mammal NOAEL		Avian NOAELt [1]	Avian NOAEL		Avian Receptor	
			Test Animal			Test Animal		Peaceful Dove	
			Animal	Body Weight (kg)		Animal	Body Weight (kg)	Body Weight (kg)	Derived TRV
Glutaraldehyde	111-30-8	4	Rat	0.35	206	Mallard Duck	1.58	0.0478	4.9E+02

Notes:
NOAELt = No observed adverse effect level test animal
kg = kilogram
NA = not applicable
TRV = toxicity reference value
1/ If an avian NOAEL was not available, the mammal NOAEL was used to derive the TRV for the avian receptor.

$$Derived\ TRV = NOAEL_{test} * \left(\frac{Body\ Weighttest}{Body\ WeightAvian}\right)^{(1/4)}$$

Exposure Route	Parameter Code	Parameter Definition	Units (a)	Parameter Value	Source (b)
Ingestion	IR	Ingestion rate	l/day	0.008	Table E-1
	EF	Exposure frequency	day/yr	21	BPJ
	ED	Exposure duration	yr	1	BPJ
	BW	Body weight	kg	0.0478	Table E-1
	AT-NC	Averaging time - noncancer	days	365	BPJ

Notes:
a/ Units:
l/day = litres per day
day/yr = days per year
yr = year
kg = kilogram
b/ References:
BPJ - Best Professional Judgement

Constituent Name	CAS No.	EPC [1]	Toxicity	Total Intake (mg/kg/day)	Hazard Quotient
		CW (mg/l)	TRVs		Ingestion
Glutaraldehyde	111-30-8	470.14	4.9E+02	4.4E+00	8.8E-03
				Cumulative:	**8.8E-03**

Notes:
CW = concentration in water
EPC = exposure point concentration
mg/kg/day = milligrams per kilograms per day
mg/l = milligrams per liter
NA = not available/applicable
TRV = toxicity reference value
1/ EPC is injected concentration presented on Table 1.

$$Total\ Intake = \frac{EPC\ \times IR\ \times EF\ \times ED}{BW\ \times ED\ x\ 365\ ^{days}/_{year}}$$

$$Hazard\ Quotient = \frac{Total\ Intake\ \left(\frac{mg}{kg-day}\right)}{TRV\ \left(\frac{mg}{kg-day}\right)}$$

Table E-5
Tier 2 Assessment - Cattle Egret
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Constituent Name	CAS No.	Mammal NOAELt	Mammal NOAEL		Avian NOAELt [1]	Avian NOAEL		Avian Receptor	
			Test Animal			Test Animal		Cattle Egret	
			Animal	Body Weight (kg)		Animal	Body Weight (kg)	Body Weight (kg)	Derived TRV
Glutaraldehyde	111-30-8	4	Rat	0.35	206	Mallard Duck	1.58	0.36	3.0E+02

Notes:
NOAELt = No observed adverse effect level test animal
kg = kilogram
NA = not applicable
TRV = toxicity reference value
1/ If an avian NOAEL was not available, the mammal NOAEL was used to derive the TRV for the avian receptor.

$$Derived\ TRV = NOAEL_{test} * \left(\frac{Body\ Weighttest}{Body\ WeightAvian}\right)^{(1/4)}$$

Exposure Route	Parameter Code	Parameter Definition	Units (a)	Parameter Value	Source (b)
Ingestion	IR	Ingestion rate	l/day	0.030	Table E-1
	EF	Exposure frequency	day/yr	21	BPJ
	ED	Exposure duration	yr	1	BPJ
	BW	Body weight	kg	0.36	Table E-1
	AT-NC	Averaging time - noncancer	days	365	BPJ

Notes:
a/ Units:
l/day = litres per day
day/yr = days per year
yr = year
kg = kilogram
b/ References:
BPJ - Best Professional Judgement

Constituent Name	CAS No.	EPC [1]	Toxicity	Total Intake (mg/kg/day)	Hazard Quotient
		CW (mg/l)	TRVs		Ingestion
Glutaraldehyde	111-30-8	470.1400	3.0E+02	2.2E+00	7.5E-03
				Cumulative:	**7.5E-03**

Notes:
CW = concentration in water
EPC = exposure point concentration
mg/kg/day = milligrams per kilograms per day
mg/l = milligrams per liter
NA = not available/applicable
TRV = toxicity reference value
1/ EPC is injected concentration presented on Table 1.

$$Total\ Intake = \frac{EPC\ \times IR\ \times EF\ \times ED}{BW\ \times ED\ x\ 365\ ^{days}/_{year}}$$

$$Hazard\ Quotient = \frac{Total\ Intake\ \left(\frac{mg}{kg-day}\right)}{TRV\ \left(\frac{mg}{kg-day}\right)}$$

Table E-6
Tier 2 Assessment - Brown Honeyeater
Condor Energy Northern Territory Tenement - Chemical Risk Assessment

Constituent Name	CAS No.	Mammal NOAELt	Mammal NOAEL		Avian NOAELt [1]	Avian NOAEL		Avian Receptor	
			Test Animal			Test Animal		Brown Honeyeater	
			Animal	Body Weight (kg)		Animal	Body Weight (kg)	Body Weight (kg)	Derived TRV
Glutaraldehyde	111-30-8	4	Rat	0.35	206	Mallard Duck	1.58	0.0106	7.2E+02

Notes:
NOAELt = No observed adverse effect level test animal
kg = kilogram
NA = not applicable
TRV = toxicity reference value
1/ If an avian NOAEL was not available, the mammal NOAEL was used to derive the TRV for the avian receptor.

$$Derived\ TRV = NOAEL_{test} * \left(\frac{Body\ Weighttest}{Body\ WeightAvian}\right)^{(1/4)}$$

Exposure Route	Parameter Code	Parameter Definition	Units (a)	Parameter Value	Source (b)
Ingestion	IR	Ingestion rate	l/day	0.0028	Table E-1
	EF	Exposure frequency	day/yr	21	BPJ
	ED	Exposure duration	yr	1	BPJ
	BW	Body weight	kg	0.0106	Table E-1
	AT-NC	Averaging time - noncancer	days	365	BPJ

Notes:
a/ Units:
l/day = litres per day
day/yr = days per year
yr = year
kg = kilogram
b/ References:
BPJ - Best Professional Judgement

Constituent Name	CAS No.	EPC [1]	Toxicity	Total Intake (mg/kg/day)	Hazard Quotient
		CW (mg/l)	TRVs		Ingestion
Glutaraldehyde	111-30-8	470.14000	7.2E+02	7.2E+00	9.9E-03
				Cumulative:	**9.9E-03**

Notes:
CW = concentration in water
EPC = exposure point concentration
mg/kg/day = milligrams per kilograms per day
mg/l = milligrams per liter
NA = not available/applicable
TRV = toxicity reference value
1/ EPC is injected concentration presented on Table 1.

$$Total\ Intake = \frac{EPC\ \times IR\ \times EF\ \times ED}{BW\ \times ED\ x\ 365\ ^{days}/_{year}}$$

$$Hazard\ Quotient = \frac{Total\ Intake\ \left(\frac{mg}{kg-day}\right)}{TRV\ \left(\frac{mg}{kg-day}\right)}$$