Appendix A Bushfire Management Plan

Exploration Permit 98

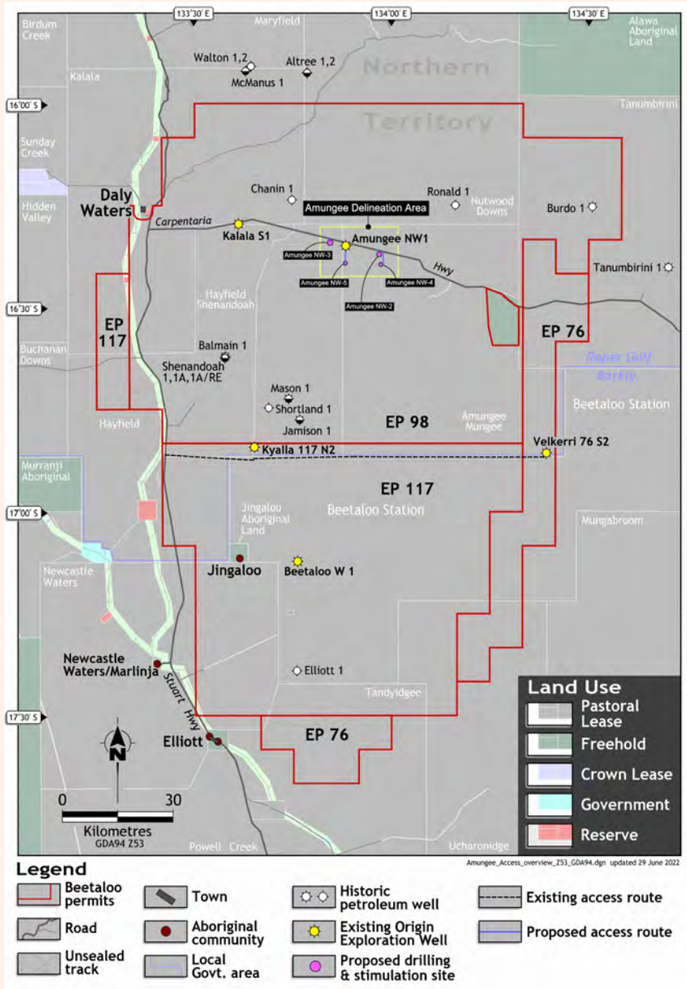
Bushfire Management Plan 2022/25 (Rev 1)

Amungee 2D Seismic Program

Location of Amungee NW-5 Lease	
Property land uses	Gas exploration and cattle grazing
Site fire management aim	To reduce the occurrence of, and minimise the impact of bushfires, thereby reducing the threat to life, property, cultural values and the environment.
Site fire management objectives	Mitigate the potential impact of unplanned fires on Origin's people, assets and operations and neighbouring land uses.

Fire Management Risks

- Ignitions (humans and lightening) on or off site resulting in harm to workers and loss of equipment.
- Fire scar mapping indicates the exploration area burns approximately every 3 to 5 years.
- Bullwaddy and Lancewood vegetation communities occur in areas across the permit and are fire sensitive. Hot fires have the ability to re-duce habitat quality for both flora and fauna species which utilise these vegetation communities.
- Spread of high fuel load grassy weeds could increase fire intensity, e.g. gamba, grader and buffel grass, adjacent infrastructure areas and access tracks.

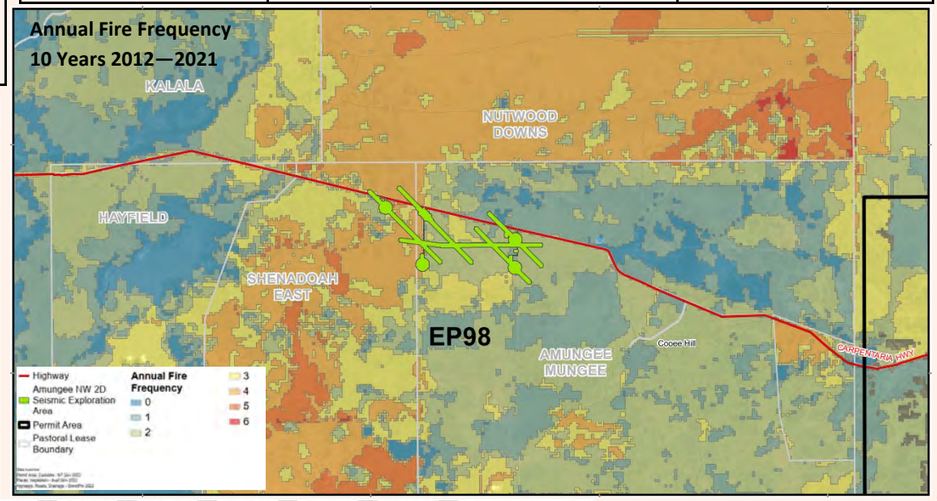


This Site Specific Bushfire Management Plan has been prepared for Origin to manage the risk from bushfire within the Amungee 2D Seismic Program (Rev 1). This Plan should be read in conjunction with the Overarching *Environmental Management Plan and Emergency Response Plans* for Origins operations in the Beetaloo Basin.

Bushfire Officer	Contact Details	Name
	[redacted]	Robert Wear

Properties	Contact Details		Name
Amungee Mungee Station	[redacted]	[redacted]	Adrian Brown Emma Brown
Hayfield Shenandoah Station	[redacted]	[redacted]	Justin Dyer Sally Dyer

Offsite Stakeholders	Contact Details	Name
National Response Centre	1800 076 251	24/7 contract line
Emergency	000 or 112 mobile	
Bushfire NT Katherine office (Savanna)	(08) 8973 8876	
Bushfire NT Alice Springs office (Barkly)	(08) 8952 3066	
NAFI North	https://www.firenorth.org.au/nafi3/	
Secure NT (Fire Bans)	https://securent.nt.gov.au/alerts	
Fire incident map	https://www.pfes.nt.gov.au/incidentmap/	



Amungee 2D Seismic Bushfire Management Actions

Seismic Acquisition, including line preparation	• Adequate fire protection equipment to be provided to prevent fires, the spread of fire, injury to personnel, and to ensure local bushfire and other fire regulations are observed. • Fire extinguishers to be fitted to all vehicles and key locations at camp. • Line preparation in grassed areas will be flattened to reduce the build-up of fuel within the vehicle's engine bays. Routine inspection of vehicles throughout day.
Neighbouring Property Fire Management Zone	• Fire management planning meeting with neighbouring properties prior to commencing activities, and reviewed annually. • Neighbour to advise proponent of planned burns.

Bushfire Preparedness

Preparedness Planning

Mandatory for all Severe, Extreme and Catastrophic FDI days

The following must be reviewed daily. If fire alerts are active or presenting with a know risk (fire in the area), personnel must execute their contingency plans which need to encompass the following:

- ☐ Procedure on identifying and notifying of a bushfire.
- ☐ Critical equipment to be removed / isolated/ shut down.
- ☐ Safe evacuation routes from site and muster points.
- ☐ Communication methods:
  - ✓ Team channels and / or phone numbers
  - ✓ Area channels and/or phone numbers
- ☐ Closest 'Safe Havens' .

Monitoring

- ☐ Provide timely advice on changes in level of fire risk as available.
- ☐ Monitor team and area common channels for bushfire early warning.

Bushfire First Responder Checklist

The following sequence must be followed by the first person responding to a fire:

1. Danger – Remove yourself and others from danger is safe to do so.

2. Alarm – Raise the alarm either on common radio channel or other agreed process.

3. Gather Information –

- ☐ **Location** – Direction from known reference points, (e.g. roads and Origin's infrastructure such as lease pad location).
- ☐ **Impacts (actual and potential)** – Life, property and the environment.
- ☐ **Fire Characteristics** – Grass or woodlands, flame height, fire front and direction of travel.
- ☐ **Weather** – Wind strength and direction.
- ☐ **Response in Progress** – What response is underway and by who (Origin Contractors, Pastoralist or Emergency Services).
- ☐ **Response required** – Origin Contractors and / or Pastoralist and / or Emergency Services.
- ☐ **Access** – Safe access and egress routes.

4. Notify Origin – Fire Officer/Supervisor

5. Notify Pastoralists – Refer to Property Contacts

6. Notify Emergency Services—Call 000 or 112 if Origin and Pastoralist unable to manage situation

7. Respond—If safe to do so in consultation with Pastoralist

8. Handover—To Pastoralist or Emergency Services as determined.

Annual Works Calendar

Month	Bushfire Risk	Action	Month	Bushfire Risk	Action
Jan	Low	• No fire management activity	July	High	• Manage vegetation onsite including weeds • Manage fire break and fire access trail • Monitor NAFI • Liaise with neighbour regarding bushfires
Feb	Low	• No fire management activity	Aug	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning require-ments
Mar	Low	• Weed survey • Planning meeting with neighbour • Annual fire mapping to monitor changes to fire frequency in the relevant area.	Sept	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning require-ments
Apr	Low	• No fire management activity	Oct	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning require-ments
May	Low	• No fire management activity	Nov	Medium	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning require-ments
June	Medium	• Manage vegetation onsite including weeds • Manage fire break and fire access trail • Monitor NAFI • Liaise with neighbour regarding bushfires	Dec	Low	• No fire management activity.

Exploration Permit 98

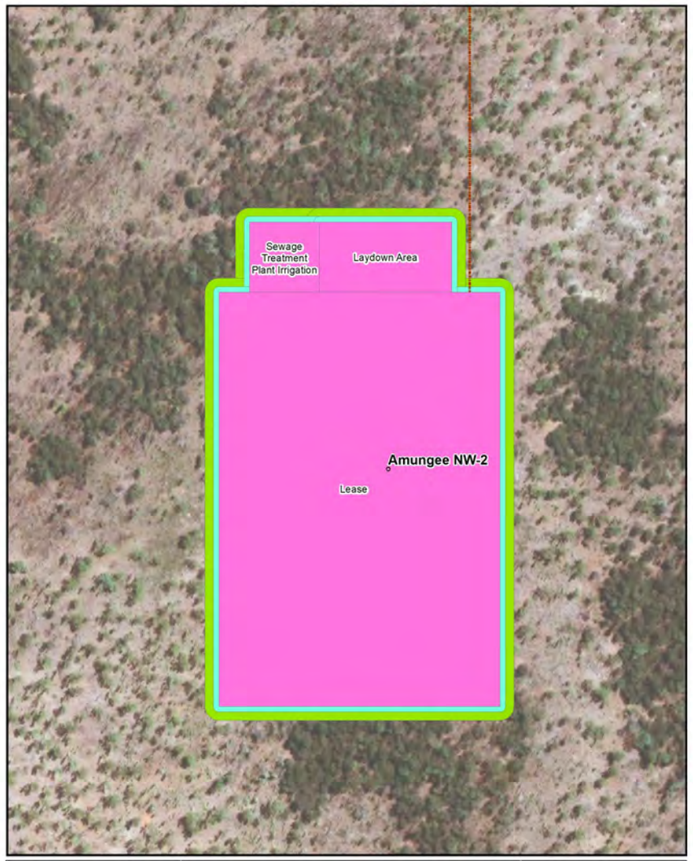
Bushfire Management Plan 2022/25 (Rev 1)

Amungee NW-2 Exploration Well

Location of Amungee NW-Lease	
Property land uses	Gas exploration and cattle grazing
Site fire management aim	To reduce the occurrence of, and minimise the impact of bushfires, thereby reducing the threat to life, property, cultural values and the environment.
Site fire management objectives	Mitigate the potential impact of unplanned fires on Origin's people, assets and operations and neighbouring land uses.

Fire Management Risks

- Ignitions (humans and lightening) on or off site resulting in harm to workers and loss of equipment.
- Fire scar mapping indicates the exploration area burns approximately every 3 to 5 years.
- Bullwaddy and Lancewood vegetation communities occur in areas across the permit and are fire sensitive. Hot fires have the ability to reduce habitat quality for both flora and fauna species which utilise these vegetation communities.
- Spread of high fuel load grassy weeds could increase fire intensity, e.g. gamba, grader and buffel grass, adjacent infrastructure areas and access tracks.

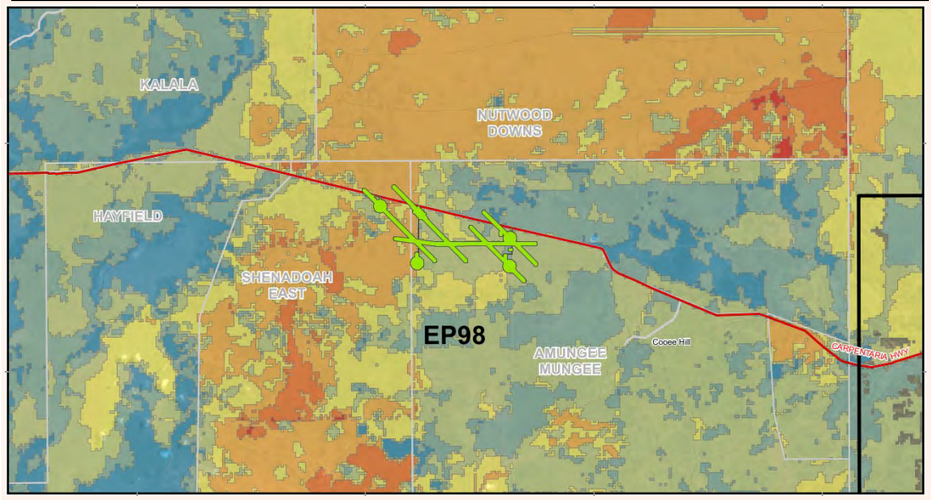


This Site Specific Bushfire Management Plan has been prepared for Origin to manage the risk from bushfire within the Amungee NW-2 (Rev 1) lease area. This Plan should be read in conjunction with the Overarching *Environmental Management Plan and Emergency Response Plans* for Origins operations in the Beetaloo Basin.

Bushfire Officer	Contact Details	Name
	[redacted]	Robert Wear

Properties	Contact Details		Name
Amungee Mungee Station	[redacted]	[redacted]	Adrian Brown Emma Brown
Hayfield Shenandoah Station	[redacted]	[redacted]	Justin Dyer Sally Dyer

Offsite Stakeholders	Contact Details	Name
National Response Centre	1800 076 251	24/7 contract line
Emergency	000 or 112 mobile	
Bushfire NT Katherine office (Savanna)	(08) 8973 8876	
Bushfire NT Alice Springs office (Barkly)	(08) 8952 3066	
NAFI North	https://www.firenorth.org.au/nafi3/	
Secure NT (Fire Bans)	https://securent.nt.gov.au/alerts	
Fire incident map	https://www.pfes.nt.gov.au/incidentmap/	



Amungee NW–2 Bushfire Management Actions

Well Pads and Tank Pads	• Remove all vegetation within the lease pad area and implement erosion and sediment control plan. • Treat emerging vegetation with herbicide. • Hot works are not permitted on total fire ban days without written approval from a fire control officer or fire warden.
Fire management break	• A 10 m wide cleared perimeter around well pads and tank pads • An additional 10 m wide bare earth fire break incorporating a 4 m wide fire access trail
Fire access trails	• Create and maintain 4 m wide access trail by grading or spraying
Asset Protection Zone (APZ)	• Site Manager to assess fuel load prior to camp establishment and again at end of wet season if infrastructure is still in place. • Establish a 20 m low fuel zone around well pads and lease pads. • Monitor for grassy weeds and control where appropriate. • If deemed necessary, conduct controlled burns where other controls are not effective and in consultation with neighbouring properties. • Ensure 4 m wide fire access trail around the perimeter of the asset protection zone is trafficable by fire fighting appliances.
Neighbouring Property Fire Management Zone	• Fire management planning meeting with neighbouring properties prior to commencing activities, and reviewed annually. • Neighbour to advise proponent of planned burns.

Bushfire Preparedness

Preparedness Planning

Mandatory for all Severe, Extreme and Catastrophic FDI days

The following must be reviewed daily. If fire alerts are active or presenting with a know risk (fire in the area), personnel must execute their contingency plans which need to encompass the following:

- ☐ Procedure on identifying and notifying of a bushfire.
- ☐ Critical equipment to be removed / isolated/ shut down.
- ☐ Safe evacuation routes from site and muster points.
- ☐ Communication methods:
  - ✓ Team channels and / or phone numbers
  - ✓ Area channels and/or phone numbers
- ☐ Closest 'Safe Havens' .

Monitoring

- ☐ Provide timely advice on changes in level of fire risk as available.
- ☐ Monitor team and area common channels for bushfire early warning.
- ☐ Update changes in work location.

Bushfire First Responder Checklist

The following sequence must be followed by the first person responding to a fire:

1. Danger – Remove yourself and others from danger is safe to do so.

2. Alarm – Raise the alarm either on common radio channel or other agreed process.

3. Gather Information –

- ☐ **Location** – Direction from known reference points, (e.g. roads and Origin's infrastructure such as lease pad location).
- ☐ **Impacts (actual and potential)** – Life, property and the environment.
- ☐ **Fire Characteristics** – Grass or woodlands, flame height, fire front and direction of travel.
- ☐ **Weather** – Wind strength and direction.
- ☐ **Response in Progress** – What response is underway and by who (Origin Contractors, Pastoralist or Emergency Services).
- ☐ **Response required** – Origin Contractors and / or Pastoralist and / or Emergency Services.
- ☐ **Access** – Safe access and egress routes.

4. Notify Origin – Fire Officer/Supervisor

5. Notify Pastoralists – Refer to Property Contacts

6. Notify Emergency Services—Call 000 or 112 if Origin and Pastoralist unable to manage situation

7. Respond—If safe to do so in consultation with Pastoralist

Annual Works Calendar

Month	Bushfire Risk	Action	Month	Bushfire Risk	Action
Jan	Low	• No fire management activity	July	High	• Manage vegetation onsite including weeds • Manage fire break and fire access trail • Monitor NAFI • Liaise with neighbour regarding bushfires
Feb	Low	• No fire management activity	Aug	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
Mar	Low	• Weed survey • Planning meeting with neighbour • Annual fire mapping to monitor changes to fire frequency in the relevant area.	Sept	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
Apr	Low	• No fire management activity	Oct	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
May	Low	• No fire management activity	Nov	Medium	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
June	Medium	• Manage vegetation onsite including weeds • Manage fire break and fire access trail • Monitor NAFI • Liaise with neighbour regarding bushfires	Dec	Low	• No fire management activity.

Exploration Permit 98

Bushfire Management Plan 2022/25 (Rev 1)

Amungee NW-3 Exploration Well

Location of Amungee NW-3 Lease	
Property land uses	Gas exploration and cattle grazing
Site fire management aim	To reduce the occurrence of, and minimise the impact of bushfires, thereby reducing the threat to life, property, cultural values and the environment.
Site fire management objectives	Mitigate the potential impact of unplanned fires on Origin's people, assets and operations and neighbouring land uses.

Fire Management Risks

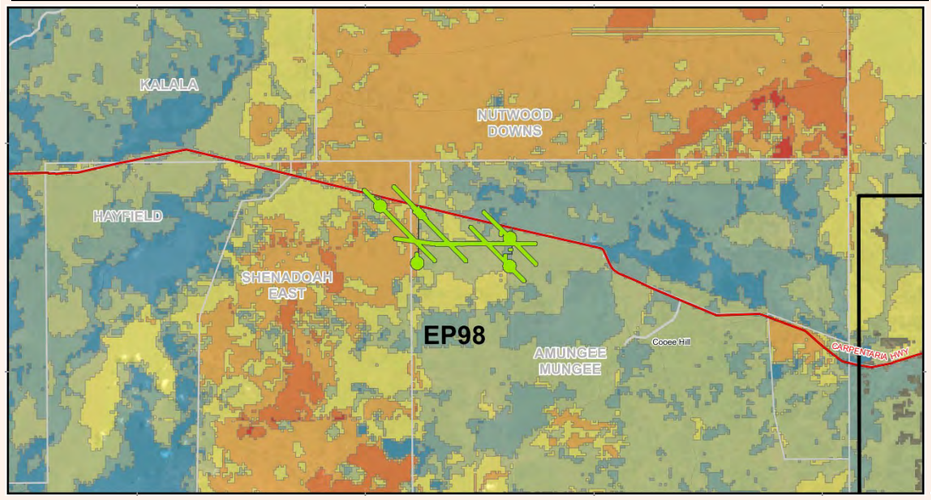
- Ignitions (humans and lightening) on or off site resulting in harm to workers and loss of equipment.
- Fire scar mapping indicates the exploration area burns approximately every 3 to 5 years.
- Bullwaddy and Lancewood vegetation communities occur in areas across the permit and are fire sensitive. Hot fires have the ability to reduce habitat quality for both flora and fauna species which utilise these vegetation communities.
- Spread of high fuel load grassy weeds could increase fire intensity, e.g. gamba, grader and buffel grass, adjacent infrastructure areas and access tracks.

This Site Specific Bushfire Management Plan has been prepared for Origin to manage the risk from bushfire within the Amungee NW-3 (Rev 1) lease area. This Plan should be read in conjunction with the Overarching *Environmental Management Plan and Emergency Response Plans* for Origins operations in the Beetaloo Basin.

Bushfire Officer	Contact Details	Name
	[redacted]	Robert Wear

Properties	Contact Details		Name
Amungee Mungee Station	[redacted]	[redacted]	Adrian Brown Emma Brown
Hayfield Shenandoah Station	[redacted]	[redacted]	Justin Dyer Sally Dyer

Offsite Stakeholders	Contact Details	Name
National Response Centre	1800 076 251	24/7 contract line
Emergency	000 or 112 mobile	
Bushfire NT Katherine office (Savanna)	(08) 8973 8876	
Bushfire NT Alice Springs office (Barkly)	(08) 8952 3066	
NAFI North	https://www.firenorth.org.au/nafi3/	
Secure NT (Fire Bans)	https://securent.nt.gov.au/alerts	
Fire incident map	https://www.pfes.nt.gov.au/incidentmap/	



Amungee NW–3 Bushfire Management Actions

Well Pads and Tank Pads	• Remove all vegetation within the lease pad area and implement erosion and sediment control plan. • Treat emerging vegetation with herbicide. • Hot works are not permitted on total fire ban days without written approval from a fire control officer or fire warden.
Fire management break	• A 10 m wide cleared perimeter around well pads and tank pads • An additional 10 m wide bare earth fire break incorporating a 4 m wide fire access trail
Fire access trails	• Create and maintain 4 m wide access trail by grading or spraying
Asset Protection Zone (APZ)	• Site Manager to assess fuel load prior to camp establishment and again at end of wet season if infrastructure is still in place. • Establish a 20 m low fuel zone around well pads and lease pads. • Monitor for grassy weeds and control where appropriate. • If deemed necessary, conduct controlled burns where other controls are not effective and in consultation with neighbouring properties. • Ensure 4 m wide fire access trail around the perimeter of the asset protection zone is trafficable by fire fighting appliances.
Neighbouring Property Fire Management Zone	• Fire management planning meeting with neighbouring properties prior to commencing activities, and reviewed annually. • Neighbour to advise proponent of planned burns.

Bushfire Preparedness

Preparedness Planning

Mandatory for all Severe, Extreme and Catastrophic FDI days

The following must be reviewed daily. If fire alerts are active or presenting with a know risk (fire in the area), personnel must execute their contingency plans which need to encompass the following:

- ☐ Procedure on identifying and notifying of a bushfire.
- ☐ Critical equipment to be removed / isolated/ shut down.
- ☐ Safe evacuation routes from site and muster points.
- ☐ Communication methods:
  - ✓ Team channels and / or phone numbers
  - ✓ Area channels and/or phone numbers
- ☐ Closest 'Safe Havens' .

Monitoring

- ☐ Provide timely advice on changes in level of fire risk as available.
- ☐ Monitor team and area common channels for bushfire early warning.
- ☐ Update changes in work location.

Bushfire First Responder Checklist

The following sequence must be followed by the first person responding to a fire:

1. Danger – Remove yourself and others from danger is safe to do so.

2. Alarm – Raise the alarm either on common radio channel or other agreed process.

3. Gather Information –

- ☐ **Location** – Direction from known reference points, (e.g. roads and Origin's infrastructure such as lease pad location).
- ☐ **Impacts (actual and potential)** – Life, property and the environment.
- ☐ **Fire Characteristics** – Grass or woodlands, flame height, fire front and direction of travel.
- ☐ **Weather** – Wind strength and direction.
- ☐ **Response in Progress** – What response is underway and by who (Origin Contractors, Pastoralist or Emergency Services).
- ☐ **Response required** – Origin Contractors and / or Pastoralist and / or Emergency Services.
- ☐ **Access** – Safe access and egress routes.

4. Notify Origin – Fire Officer/Supervisor

5. Notify Pastoralists – Refer to Property Contacts

6. Notify Emergency Services—Call 000 or 112 if Origin and Pastoralist unable to manage situation

7. Respond—If safe to do so in consultation with Pastoralist

8. Handover—To Pastoralist or Emergency Services as determined.

Annual Works Calendar

Month	Bushfire Risk	Action	Month	Bushfire Risk	Action
Jan	Low	• No fire management activity	July	High	• Manage vegetation onsite including weeds • Manage fire break and fire access trail • Monitor NAFI • Liaise with neighbour regarding bushfires
Feb	Low	• No fire management activity	Aug	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
Mar	Low	• Weed survey • Planning meeting with neighbour • Annual fire mapping to monitor changes to fire frequency in the relevant area.	Sept	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
Apr	Low	• No fire management activity	Oct	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
May	Low	• No fire management activity	Nov	Medium	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
June	Medium	• Manage vegetation onsite including weeds • Manage fire break and fire access trail • Monitor NAFI • Liaise with neighbour regarding bushfires	Dec	Low	• No fire management activity.

Exploration Permit 98

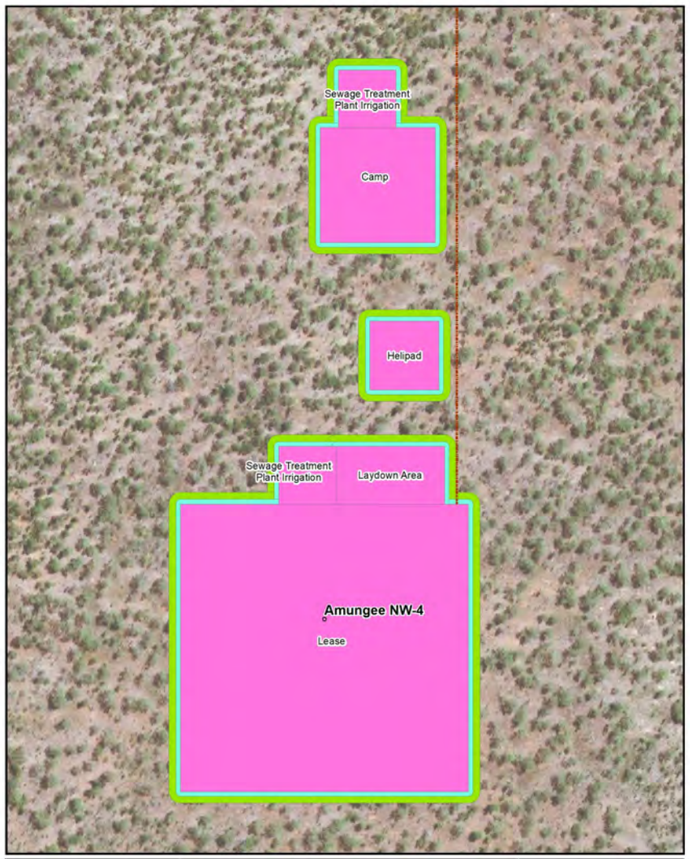
Bushfire Management Plan 2022/25 (Rev 1)

Amungee NW-4 Exploration Well

Location of Amungee NW-4 Lease	
Property land uses	Gas exploration and cattle grazing
Site fire management aim	To reduce the occurrence of, and minimise the impact of bushfires, thereby reducing the threat to life, property, cultural values and the environment.
Site fire management objectives	Mitigate the potential impact of unplanned fires on Origin's people, assets and operations and neighbouring land uses.

Fire Management Risks

- Ignitions (humans and lightening) on or off site resulting in harm to workers and loss of equipment.
- Fire scar mapping indicates the exploration area burns approximately every 3 to 5 years.
- Bullwaddy and Lancewood vegetation communities occur in areas across the permit and are fire sensitive. Hot fires have the ability to reduce habitat quality for both flora and fauna species which utilise these vegetation communities.
- Spread of high fuel load grassy weeds could increase fire intensity, e.g. gamba, grader and buffel grass, adjacent infrastructure areas and access tracks.

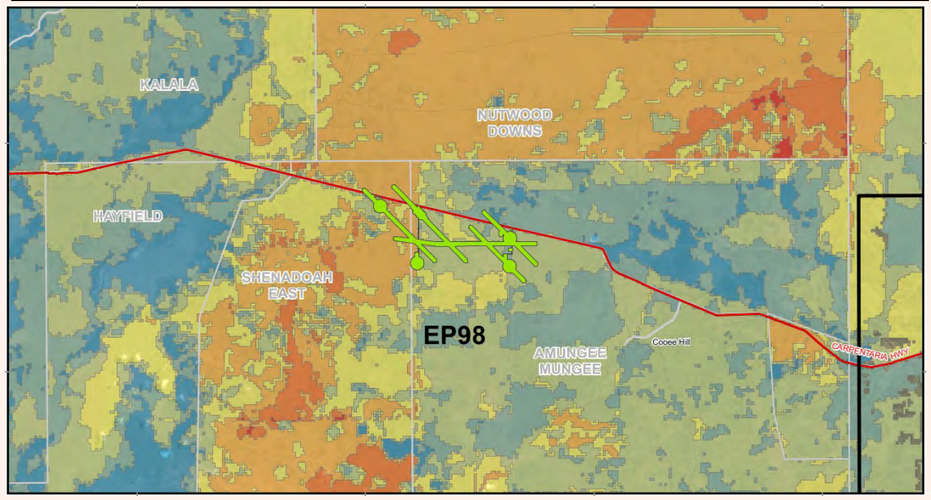


This Site Specific Bushfire Management Plan has been prepared for Origin to manage the risk from bushfire within the Amungee NW-4 (Rev 1) lease area. This Plan should be read in conjunction with the Overarching *Environmental Management Plan and Emergency Response Plans* for Origins operations in the Beetaloo Basin.

Bushfire Officer	Contact Details	Name
	[redacted]	Robert Wear

Properties	Contact Details		Name
Amungee Mungee Station	[redacted]	[redacted]	Adrian Brown Emma Brown
Hayfield Shenandoah Station	[redacted]	[redacted]	Justin Dyer Sally Dyer

Offsite Stakeholders	Contact Details	Name
National Response Centre	1800 076 251	24/7 contract line
Emergency	000 or 112 mobile	
Bushfire NT Katherine office (Savanna)	(08) 8973 8876	
Bushfire NT Alice Springs office (Barkly)	(08) 8952 3066	
NAFI North	https://www.firenorth.org.au/nafi3/	
Secure NT (Fire Bans)	https://securent.nt.gov.au/alerts	
Fire incident map	https://www.pfes.nt.gov.au/incidentmap/	



Amungee NW–4 Bushfire Management Actions

Well Pads and Tank Pads	• Remove all vegetation within the lease pad area and implement erosion and sediment control plan. • Treat emerging vegetation with herbicide. • Hot works are not permitted on total fire ban days without written approval from a fire control officer or fire warden.
Fire management break	• A 10 m wide cleared perimeter around well pads and tank pads • An additional 10 m wide bare earth fire break incorporating a 4 m wide fire access trail
Fire access trails	• Create and maintain 4 m wide access trail by grading or spraying
Asset Protection Zone (APZ)	• Site Manager to assess fuel load prior to camp establishment and again at end of wet season if infrastructure is still in place. • Establish a 20 m low fuel zone around well pads and lease pads. • Monitor for grassy weeds and control where appropriate. • If deemed necessary, conduct controlled burns where other controls are not effective and in consultation with neighbouring properties. • Ensure 4 m wide fire access trail around the perimeter of the asset protection zone is trafficable by fire fighting appliances.
Neighbouring Property Fire Management Zone	• Fire management planning meeting with neighbouring properties prior to commencing activities, and reviewed annually. • Neighbour to advise proponent of planned burns.

Bushfire Preparedness

Preparedness Planning

Mandatory for all Severe, Extreme and Catastrophic FDI days

The following must be reviewed daily. If fire alerts are active or presenting with a know risk (fire in the area), personnel must execute their contingency plans which need to encompass the following:

- ☐ Procedure on identifying and notifying of a bushfire.
- ☐ Critical equipment to be removed / isolated/ shut down.
- ☐ Safe evacuation routes from site and muster points.
- ☐ Communication methods:
  - ✓ Team channels and / or phone numbers
  - ✓ Area channels and/or phone numbers
- ☐ Closest 'Safe Havens' .

Monitoring

- ☐ Provide timely advice on changes in level of fire risk as available.
- ☐ Monitor team and area common channels for bushfire early warning.

Bushfire First Responder Checklist

The following sequence must be followed by the first person responding to a fire:

1. Danger – Remove yourself and others from danger is safe to do so.

2. Alarm – Raise the alarm either on common radio channel or other agreed process.

3. Gather Information –

- ☐ **Location** – Direction from known reference points, (e.g. roads and Origin's infrastructure such as lease pad location).
- ☐ **Impacts (actual and potential)** – Life, property and the environment.
- ☐ **Fire Characteristics** – Grass or woodlands, flame height, fire front and direction of travel.
- ☐ **Weather** – Wind strength and direction.
- ☐ **Response in Progress** – What response is underway and by who (Origin Contractors, Pastoralist or Emergency Services).
- ☐ **Response required** – Origin Contractors and / or Pastoralist and / or Emergency Services.
- ☐ **Access** – Safe access and egress routes.

4. Notify Origin – Fire Officer/Supervisor

5. Notify Pastoralists – Refer to Property Contacts

6. Notify Emergency Services—Call 000 or 112 if Origin and Pastoralist unable to manage situation

7. Respond—If safe to do so in consultation with Pastoralist

8. Handover—To Pastoralist or Emergency Services as determined.

Annual Works Calendar

Month	Bushfire Risk	Action	Month	Bushfire Risk	Action
Jan	Low	• No fire management activity	July	High	• Manage vegetation onsite including weeds • Manage fire break and fire access trail • Monitor NAFI • Liaise with neighbour regarding bushfires
Feb	Low	• No fire management activity	Aug	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
Mar	Low	• Weed survey • Planning meeting with neighbour • Annual fire mapping to monitor changes to fire frequency in the relevant area.	Sept	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
Apr	Low	• No fire management activity	Oct	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
May	Low	• No fire management activity	Nov	Medium	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
June	Medium	• Manage vegetation onsite including weeds • Manage fire break and fire access trail • Monitor NAFI • Liaise with neighbour regarding bushfires	Dec	Low	• No fire management activity.

Exploration Permit 98

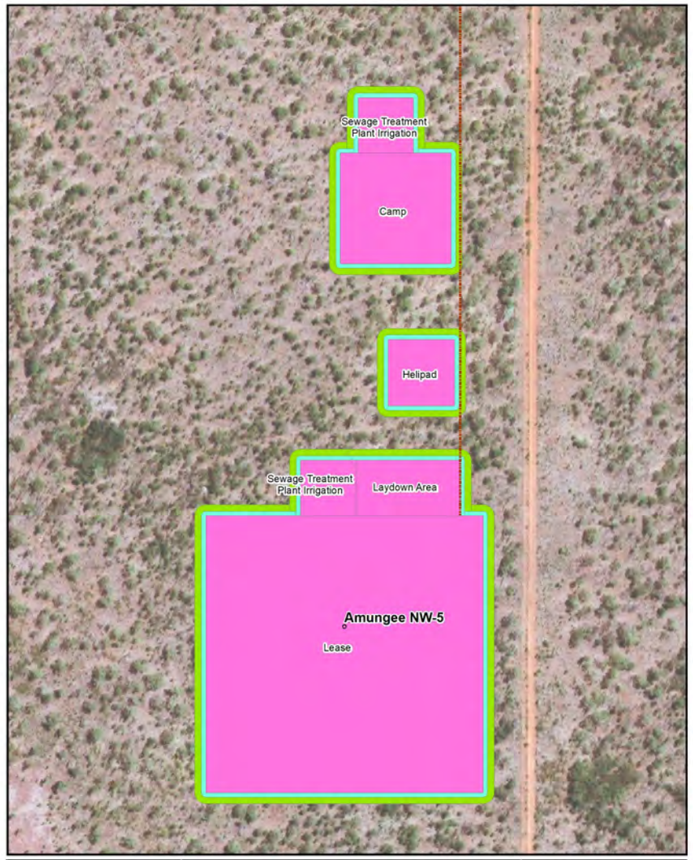
Bushfire Management Plan 2022/25 (Rev 1)

Amungee NW-5 Exploration Well

Location of Amungee NW-5 Lease	
Property land uses	Gas exploration and cattle grazing
Site fire management aim	To reduce the occurrence of, and minimise the impact of bushfires, thereby reducing the threat to life, property, cultural values and the environment.
Site fire management objectives	Mitigate the potential impact of unplanned fires on Origin's people, assets and operations and neighbouring land uses.

Fire Management Risks

- Ignitions (humans and lightening) on or off site resulting in harm to workers and loss of equipment.
- Fire scar mapping indicates the exploration area burns approximately every 3 to 5 years.
- Bullwaddy and Lancewood vegetation communities occur in areas across the permit and are fire sensitive. Hot fires have the ability to reduce habitat quality for both flora and fauna species which utilise these vegetation communities.
- Spread of high fuel load grassy weeds could increase fire intensity, e.g. gamba, grader and buffel grass, adjacent infrastructure areas and access tracks.

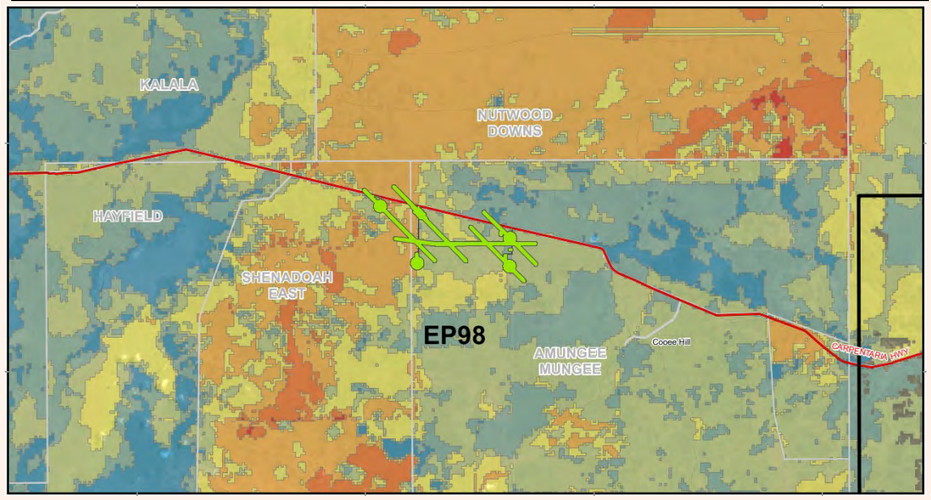


This Site Specific Bushfire Management Plan has been prepared for Origin to manage the risk from bushfire within the Amungee NW-5 (Rev 1) lease area. This Plan should be read in conjunction with the Overarching *Environmental Management Plan and Emergency Response Plans* for Origins operations in the Beetaloo Basin.

Bushfire Officer	Contact Details	Name
	[redacted]	Robert Wear

Properties	Contact Details		Name
Amungee Mungee Station	[redacted]	[redacted]	Adrian Brown Emma Brown
Hayfield Shenandoah Station	[redacted]	[redacted]	Justin Dyer Sally Dyer

Offsite Stakeholders	Contact Details	Name
National Response Centre	1800 076 251	24/7 contract line
Emergency	000 or 112 mobile	
Bushfire NT Katherine office (Savanna)	(08) 8973 8876	
Bushfire NT Alice Springs office (Barkly)	(08) 8952 3066	
NAFI North	https://www.firenorth.org.au/nafi3/	
Secure NT (Fire Bans)	https://securent.nt.gov.au/alerts	
Fire incident map	https://www.pfes.nt.gov.au/incidentmap/	



Amungee NW–5 Bushfire Management Actions

Well Pads and Tank Pads	• Remove all vegetation within the lease pad area and implement erosion and sediment control plan. • Treat emerging vegetation with herbicide. • Hot works are not permitted on total fire ban days without written approval from a fire control officer or fire warden.
Fire management break	• A 10 m wide cleared perimeter around well pads and tank pads • An additional 10 m wide bare earth fire break incorporating a 4 m wide fire access trail
Fire access trails	• Create and maintain 4 m wide access trail by grading or spraying
Asset Protection Zone (APZ)	• Site Manager to assess fuel load prior to camp establishment and again at end of wet season if infrastructure is still in place. • Establish a 20 m low fuel zone around well pads and lease pads. • Monitor for grassy weeds and control where appropriate. • If deemed necessary, conduct controlled burns where other controls are not effective and in consultation with neighbouring properties. • Ensure 4 m wide fire access trail around the perimeter of the asset protection zone is trafficable by fire fighting appliances.
Neighbouring Property Fire Management Zone	• Fire management planning meeting with neighbouring properties prior to commencing activities, and reviewed annually. • Neighbour to advise proponent of planned burns.

Bushfire Preparedness

Preparedness Planning

Mandatory for all Severe, Extreme and Catastrophic FDI days

The following must be reviewed daily. If fire alerts are active or presenting with a know risk (fire in the area), personnel must execute their contingency plans which need to encompass the following:

- ☐ Procedure on identifying and notifying of a bushfire.
- ☐ Critical equipment to be removed / isolated/ shut down.
- ☐ Safe evacuation routes from site and muster points.
- ☐ Communication methods:
  - ✓ Team channels and / or phone numbers
  - ✓ Area channels and/or phone numbers
- ☐ Closest 'Safe Havens' .

Monitoring

- ☐ Provide timely advice on changes in level of fire risk as available.
- ☐ Monitor team and area common channels for bushfire early warning.

Bushfire First Responder Checklist

The following sequence must be followed by the first person responding to a fire:

1. Danger – Remove yourself and others from danger is safe to do so.

2. Alarm – Raise the alarm either on common radio channel or other agreed process.

3. Gather Information –

- ☐ **Location** – Direction from known reference points, (e.g. roads and Origin's infrastructure such as lease pad location).
- ☐ **Impacts (actual and potential)** – Life, property and the environment.
- ☐ **Fire Characteristics** – Grass or woodlands, flame height, fire front and direction of travel.
- ☐ **Weather** – Wind strength and direction.
- ☐ **Response in Progress** – What response is underway and by who (Origin Contractors, Pastoralist or Emergency Services).
- ☐ **Response required** – Origin Contractors and / or Pastoralist and / or Emergency Services.
- ☐ **Access** – Safe access and egress routes.

4. Notify Origin – Fire Officer/Supervisor

5. Notify Pastoralists – Refer to Property Contacts

6. Notify Emergency Services—Call 000 or 112 if Origin and Pastoralist unable to manage situation

7. Respond—If safe to do so in consultation with Pastoralist

8. Handover—To Pastoralist or Emergency Services as determined.

Annual Works Calendar

Month	Bushfire Risk	Action	Month	Bushfire Risk	Action
Jan	Low	• No fire management activity	July	High	• Manage vegetation onsite including weeds • Manage fire break and fire access trail • Monitor NAFI • Liaise with neighbour regarding bushfires
Feb	Low	• No fire management activity	Aug	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
Mar	Low	• Weed survey • Planning meeting with neighbour • Annual fire mapping to monitor changes to fire frequency in the relevant area.	Sept	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
Apr	Low	• No fire management activity	Oct	High	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
May	Low	• No fire management activity	Nov	Medium	• Monitor NAFI and visual scan horizon for smoke • Liaise with neighbour regarding bushfires • Review the preparedness planning requirements
June	Medium	• Manage vegetation onsite including weeds • Manage fire break and fire access trail • Monitor NAFI • Liaise with neighbour regarding bushfires	Dec	Low	• No fire management activity.

Appendix B Weed Management Plan

BEETALOO BASIN EXPLORATION PROJECT

Weed Management Plan

Review record

Rev	Date	Reason for issue	Author	Reviewer	Approver
0	05/10/2018	Issue for release	A Court	M Kernke	M Hanson
1	29/03/2019	Issue for release	A Court	M Kernke	M Hanson
2	20/05/2019	Minor Update	A Court	M Kernke	M Hanson
2.1	10/09/2019	Minor update	M Kernke		M Hanson
2.2	10/09/2019	Minor update to include feedback from Amungee NW-1H EMP review			M Kernke
2.3	25/08/2021	Minor update to content based on DEPWS feedback	M Kernke		M Kernke
2.4	10/11/2021	Update to include 2021 weed survey	M Kernke		M Kernke
2.5	18/01/2022	Update to include revised RWMP			M Kernke
2.6	27/02/2022	Update to include DEPWs comments			M Kernke
2.7	30/08/2022	Update to reference the *Tennant Creek Weeds Strategy 2021-2026* and Gamba Grass eradication in Zone A	L Pugh		M Kernke

Review due: 29/03/2024

Weed Management Plan

NT-2050-15-MP-0016

Table of contents

Table of figures

Review due: 29/03/2024

List of tables

List of appendices

1. Introduction

1.1 Objectives of the WMP

This WMP has been developed to ensure that the risk of weed introduction and spread, resulting from activities associated with Origin Exploration activities are mitigated to protect the economic, community, industry and environmental interests of the Territory.

The plan provides an overview of:

- The project context (Section 2)
- Legal requirements in relation to weed management (Section 3)
- The appointment of a Dedicated Weed Officer (Section 4)
- Identified risks and proposed mitigation measures and management objectives (Section 5 and 6)
- The weed species that are considered likely or known to occur within the Permit Area (Section 6 and 7)
- The Annual Action Plan for those species that are known to occur with the Permit Area (Section 8)
- Control options for species known to occur within the Permit Area (Section 8)
- The monitoring, notification, recording and reporting requirements for the WMP (Sections 9 – 12)

This plan is supported by Appendices that provide guidance on how to identify weed species in the field and collect the necessary data to support the monitoring and reporting requirements of this WMP.

The location of the proposed exploration activities are shown on Figure 1.

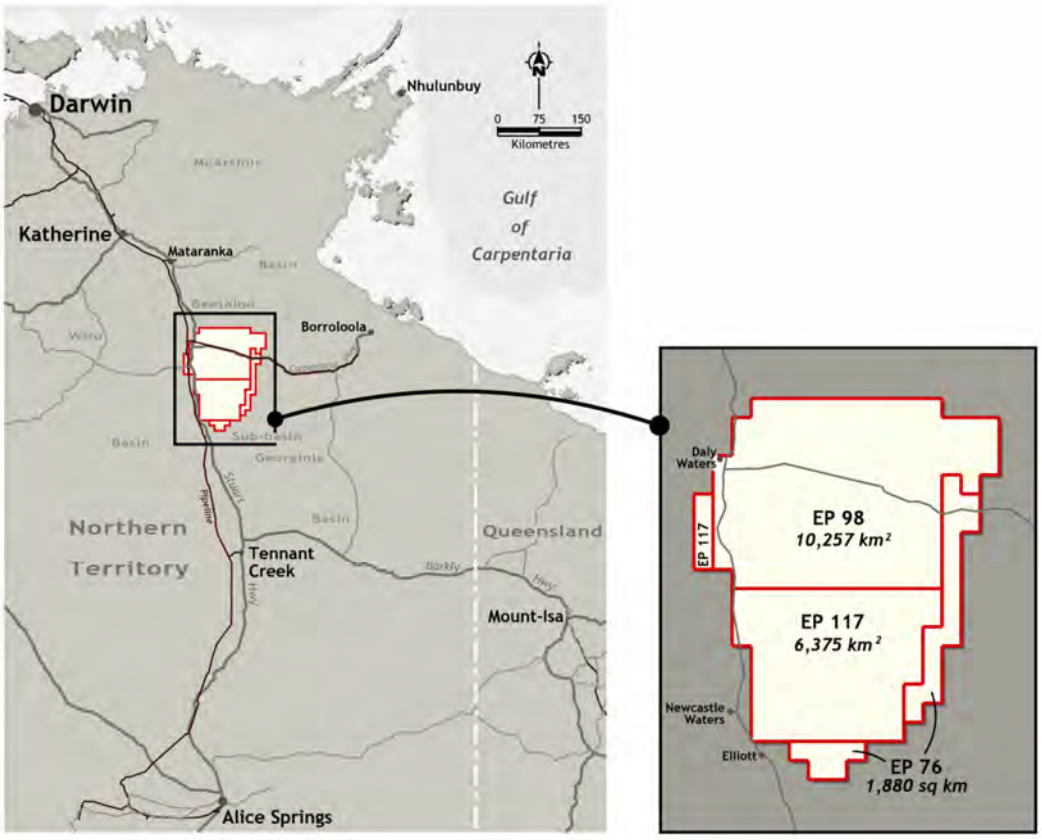


Figure 1 Location of Origin Permit Area

1.2 Intent of the WMP

Weed control is considered to be a significant land management issue in the Northern Territory. This Weed Management Plan (WMP) forms a core component of Origin's overarching environmental management strategy and supports the various project Environment Management Plan (EMPs).

The movement of rigs, vehicles, machinery and other materials to, from and within the exploration permit area may result in weeds being moved around the pastoral lease, into the lease from surrounding areas or interstate, depending on where the vehicles and materials are sourced from or returned to.

The focus of this WMP is therefore to ensure that infestations are eradicated, or at the very least that existing weed infestations are controlled such that no further weed species colonise the permit area as a result of Origin's activities.

This document is based upon the Weed Management Planning Guide - Onshore Petroleum Projects produced by the Department of Environment, Parks and Water Security (DEPWS 2019).

2. Project Context

This plan covers all civil, drilling, stimulating, rehabilitation and routine maintenance/monitoring activities undertaken by Origin within permit EP76, EP98 and EP117 as detailed in Table 1. The proposed activities for the forward exploration program are highlighted within the table.

Table 1 Coordinates of centroid of proposed exploration lease areas

Exploration Permit	Lease Name	Zone*	Easting	Northing
EP98	Velkerri 98 E1	53	415515	8180683
EP98	Velkerri 98 N1	53	392292	8189891
EP98	Kyalla 98 W1	53	364955	8177458
EP98	Amungee NW	53	380859	8192299
EP98	Amungee NW 2	53	389841.38	8190092.63
EP98	Amungee NW 3	53	376611.28	8193100.37
EP98	Amungee NW 4	53	390313.59	8187337.06
EP98	Amungee NW 5	53	380597.38	8187469.62
EP76	Velkerri 76 S1	53	424362	8113273
EP76	Velkerri 76 S2	53	435488	8136321
EP117	Kyalla 117 N2	53	356175	8137500
EP117	Stuart Highway Intersection	53	332371	8135170
EP117	Velkerri 117 E1	53	428861	8120782
EP117	Kyalla 117 W1	53	368079	8106696

Grey shading are planned sites for 2019/200

** Universal Transverse Mercator (UTM) geographic coordinate system is Geocentric Datum of Australia (GDA) 94.*

The primary activities subject to this WMP are:

- Access track construction, use and maintenance
- Exploration lease pad construction, use and maintenance
- Gravel pit construction and maintenance

- Drilling, stimulating, completing and maintaining petroleum exploration wells
- Routine access, maintenance and monitoring of all exploration areas subject to this plan.

3. Legal Requirements

The following presents the relevant legislation and statutory obligations for the project.

3.1 Northern Territory Petroleum (Environment) Regulations

Petroleum Act 1984,* Petroleum (Environment) Regulations 2016 *and Code of Practice for Petroleum Activities within the Northern Territory

The *Petroleum Act 1984* provides legal framework within which persons are encouraged to undertake effective exploration for petroleum and to develop petroleum production so that the optimum value of the resource is returned to the Territory. It regulates the exploration for, and production of petroleum, including environmental protection measures which should be employed during exploration and production activities, including protection of parks and reserves and rehabilitation.

In addition, the Act is supported by the Petroleum (Environment) Regulations 2016.

The Petroleum (Environment) Regulations 2016 requires that regulated activities are carried out in a manner consistent with the principles of ecologically sustainable development, and by which the environmental impacts and environmental risks of the activities are identified and reduced to an acceptable level.

The Code of Practice for Petroleum Activities in the Northern Territory is a mandatory code of practice for the petroleum industry to ensure that petroleum activities in the Northern Territory are managed according to minimum acceptable standards to ensure that risks to the environment can be managed to a level that is as low as reasonably practical (ALARP) and acceptable.

Under these regulations Origin is required to submit an EMP prior to any petroleum exploration or production activity.

EMP's must include:

- potential environmental risks or impacts (in this instance relating to the introduction and spread of weeds);
- appropriate environmental outcomes, environmental performance standards and measurement criteria;
- appropriate implementation strategy and monitoring, recording and reporting arrangements; and
- demonstrate that there has been an appropriate level of engagement with directly affected stakeholders in developing the plan.

This WMP is designed to support and implement the requirements of Origins Project Specific Environmental Management Plans.

3.2 Northern Territory Weeds Management Act

The aim of the *Weeds Management Act 2001* is 'to protect the Territory's economy, community, industry and environment from the adverse impact of weeds.

The purpose of the Act, as defined in section 3, is:

- To prevent the spread of weeds in, into and out of the Territory and to ensure that the management of weeds is an integral component of land management in accordance with the Northern Territory Weeds Management Strategy 1996 – 2005 or any other strategy adopted to control weeds in the Territory.
- To ensure there is community consultation in the creation of weed management plans.
- To ensure that there is community responsibility in implementing weed management plans.

The Act identifies declared weeds (those which must be controlled) and provides a framework for weed management. It includes the following weed declaration classes:

Class A – to be eradicated
Class B – growth and spread to be controlled
Class C* – Not to be introduced into the Northern Territory
** All Class A and B weeds are also Class C.*

The Act enables the relevant Minister to approve statutory weed management plans. Management obligations in these plans must be adhered to.

Currently there are statutory management plans for 10 high priority weed species in the Northern Territory.

The WMP must address weeds in accordance with their declaration status and the statutory requirements of any relevant weed management plans.

3.3 Regional Weed Strategies

Regional Weed Strategies (RWS) have been developed for areas of the NT, with the Tennant Creek regional weeds strategy 2021 – 2026 and the Katherine regional weeds strategy 2021 – 2026, overlapping Origin's Beetaloo exploration tenure. the aim of these regional plans is to assist in prioritising weed management by:

- identifying the region's priority weeds and associated pathways of spread to inform management priorities
- identifying landscapes that may need prioritised protection from weed impacts like river corridors or sacred Aboriginal sites
- containing information on alert weeds that are not yet found in the region, but could become major issues if they establish

3.4 Commonwealth Environment Protection Biodiversity Conservation Act

The objectives of the *Environment Protection and Biodiversity Conservation Act 1999* (EPBC Act) are, among other things:

- provide for the protection of the environment, especially those aspects of the environment that are matters of national environmental significance; and
- promote ecologically sustainable development through the conservation and ecologically sustainable use of natural resources; and
- promote the conservation of biodiversity; and
- promote a co-operative approach to the protection and management of the environment involving governments, the community, land-holders and indigenous peoples; and
- assist in the co-operative implementation of Australia's international environmental responsibilities.

The *EPBC Act* provides for the identification and listing of key threatening processes on matters of national environmental significance (MNES). A threatening process is defined as a key threatening process if it threatens or may threaten the survival, abundance or evolutionary development of a native species or ecological community. Key threatening processes include invasive species, such as weeds, which have a major impact on Australia's environment, threatening our unique biodiversity and reducing overall species abundance and diversity (DOTEE 2018).

Threat abatement plans (TAP) are developed to address key threatening processes. A TAP has been developed covering 5 listed grass species present within the NT (The Threat abatement plan to reduce the impacts on northern Australia's biodiversity by the five listed grasses (2012)). This TAP covers a range of grasses originally introduced to support pastoralism, and includes Gamba Grass, Para grass, olive hymenachne, mission grass and annual mission grass. The controls in this WMP are designed to align with the TAP.

4. Dedicated Weed Officer

As per recommendation 8.3 of the Scientific Inquiry into Hydraulic Fracturing Stimulation there must be a dedicated Weed Officer for each gas field.

The Weed Officer must have relevant skills and experience and availability to successfully manage weed related issues for the project, including:

- Knowledge of the biology/ecology of local weeds.
- Knowledge of relevant weed management frameworks including Northern Territory legislation and plans, the EPBC Act.
- Understanding of existing weed management arrangements being undertaken by landholders.

The Weed Officer is responsible and accountable for delivery of all weed related requirements of the project in accordance with the WMP and the overarching Environmental Management Plan, including:

- Planning and execution of weed monitoring requirements, including baseline weed assessments and ongoing monitoring both during periods of gas related activities as well as during the target identification period of February to May.
- Facilitate training all workers (including contractors) in weed management requirements, with support from the Northern Territory Government Onshore Petroleum Weed Management Officer.
- Oversight of implementation of weed control mechanisms including but not limited to wash-downs and proactive weed control programs.
- Ensuring all reporting requirements are met.
- Act as the designated point of contact for and rapidly responding to any weed related complaints and incidents in accordance with the pre-determined strategies in this WMP and additional strategies as required developed in consultation with the Onshore Petroleum Weed Management Officer and affected landholders.
- Review and update of WMP's to remain effective in communication with relevant landholders and Onshore Petroleum Weed Management Officer in consideration of monitoring results and emerging weed issues for both gas and pastoral operations.

Origin has appointed **Robert Wear, Construction Superintendent** as the dedicated Weed Officer of the Beetaloo Exploration Activities. This role is supported by Origin's Approvals and HSE personnel.

5. Weed Species Information

Weed surveys completed across the proposed and existing exploration areas indicate the abundance of weeds within the proposed and existing project area is low. *Hyptis suaveolens* (Hyptis), has been identified at the Kalala S1 and Amungee NW site (access tracks, camp pad and lease pad). Hyptis has also recently been observed at the Velkerri 76 S2 site camp pad and irrigation area. Rubber Bush and *Parkinsonia aculeata* (Parkinsonia) have been previously identified along/in close proximity to the Beetaloo W access track, with rubber bush also found along the Kyalla 117 N2 access track. Parkinsonia is considered a Weed of National Significance (WoNS), which are weed species that are the focus of national management programs for the purpose of restricting their spread and/or eradicating them from parts of Australia. These species are specifically presented in Table 2 and Section 9.

Gamba Grass (*Andropogon gayanus*) is a Declared Class A (to be eradicated) weed within the Beetaloo Sub-basin.[1] Gamba grass is grown within the Class A zone on two pastoral leases under strict permit conditions. These pastoral leases are subject to annual audits and regular inspections to ensure it is not spread outside the permitted areas. An active compliance program is in place to ensure eradication is being achieved in Zone A, as per the requirements of the statutory weed management plan for gamba grass (DEPWS 2020). Gamba Grass has

[1] The Beetaloo lies within the NT Statutory Weed Management Zone Class A (for eradication).

not been identified within Origin's exploration permits. Origin is committed to preventing the spread of Gamba Grass into the project area from known Gamba locations. Should Gamba Grass be identified within Origin's EPs, it would need to be identified, recorded and treated in accordance with the Threat Abatement Advice released by the Commonwealth Government.

Figure 2 illustrates the weeds species confirmed in the region during field surveys, along with other weed species that are known to occur or likely to occur within the wider exploration Permit Areas. This information is based on:

- Origin exploration program weed survey data (2014-2020 results).
- Mapping data provided by the Weed Management Branch, DEPWS.
- Guidelines for the *Management of the Weeds of Beetaloo 2018* (DLRM et al 2018).
- Tennant Creek weeds strategy 2021 – 2026 and Katherine weeds strategy 2021 – 2026
- Department of Climate Change, Energy, the Environment and Water (DCCEEW)[2] EPBC Act Protected Matters Report database.

Table 3 has been separated into priority weeds, which are broken down into 5 distinct categories:

- Category 1: These species are present in the region and are widely considered feasible to eradicate from the Region. They are typically evaluated as very high risk and have isolated and restricted distributions.
- Category 2 These species warrant strategic control across the landscape due to the high impact they have on land managers and on broader economic and environmental values. These species have outlier populations that may be practical to locally eradicate, and core infestations that are too large for eradication to be considered an option.
- Category 3: These species have been assessed by the weed risk management system as a medium to high risk (or have not been assessed) and have been identified by stakeholders as posing a threat to the values of the Region
- Category 4: These species are typically evaluated as low risk; however, they do still have local impacts. T
- Category 5: The Weed Management Branch uses a working definition of an 'alert' weed as a species:
    - not yet naturalised in a Region
    - with the potential to have a high level of impact should it become established
    - having a reasonable likelihood of arriving in the Region (or of being present undetected)

It is noted that Parthenium (*Parthenium hysterophorus*) is a major problem in rangelands and cropping areas of" Queensland and is estimated to cost farmers and graziers more than $22 million a year in reduced production and increased management costs. Vehicle, machinery and material movements from Queensland into the project area present a risk of spread of Parthenium if not managed correctly (Department of Primary Industry and Resources 2016).

Additional mapped locations of weeds within the Tennant and Katherine RWS are provided in Figure 3, Figure 4 and Figure 5.

[2] Formerly the Commonwealth Department of the Environment and Energy (DOTEE).

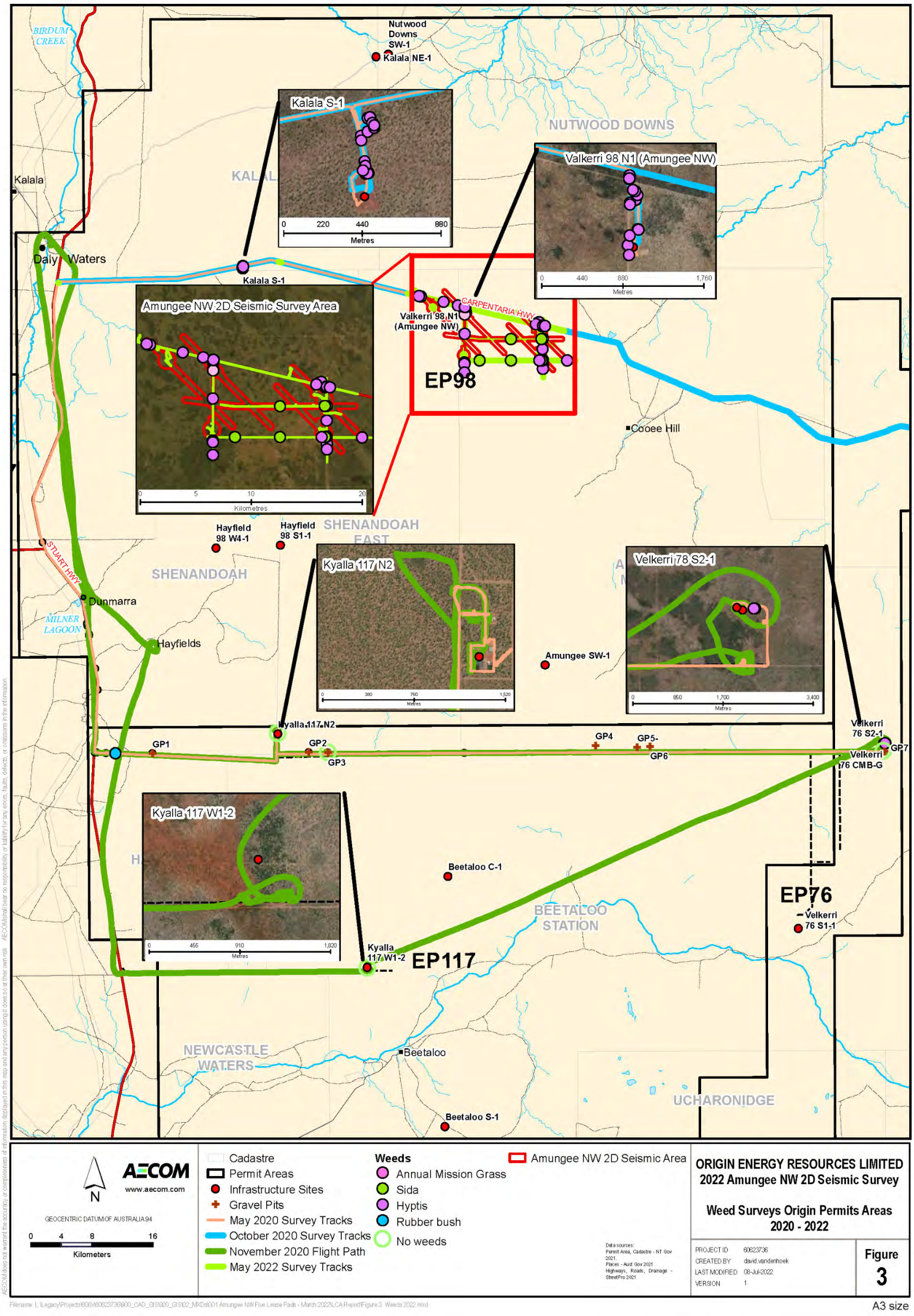


Figure 2 Location of Weeds Species in Permit Areas

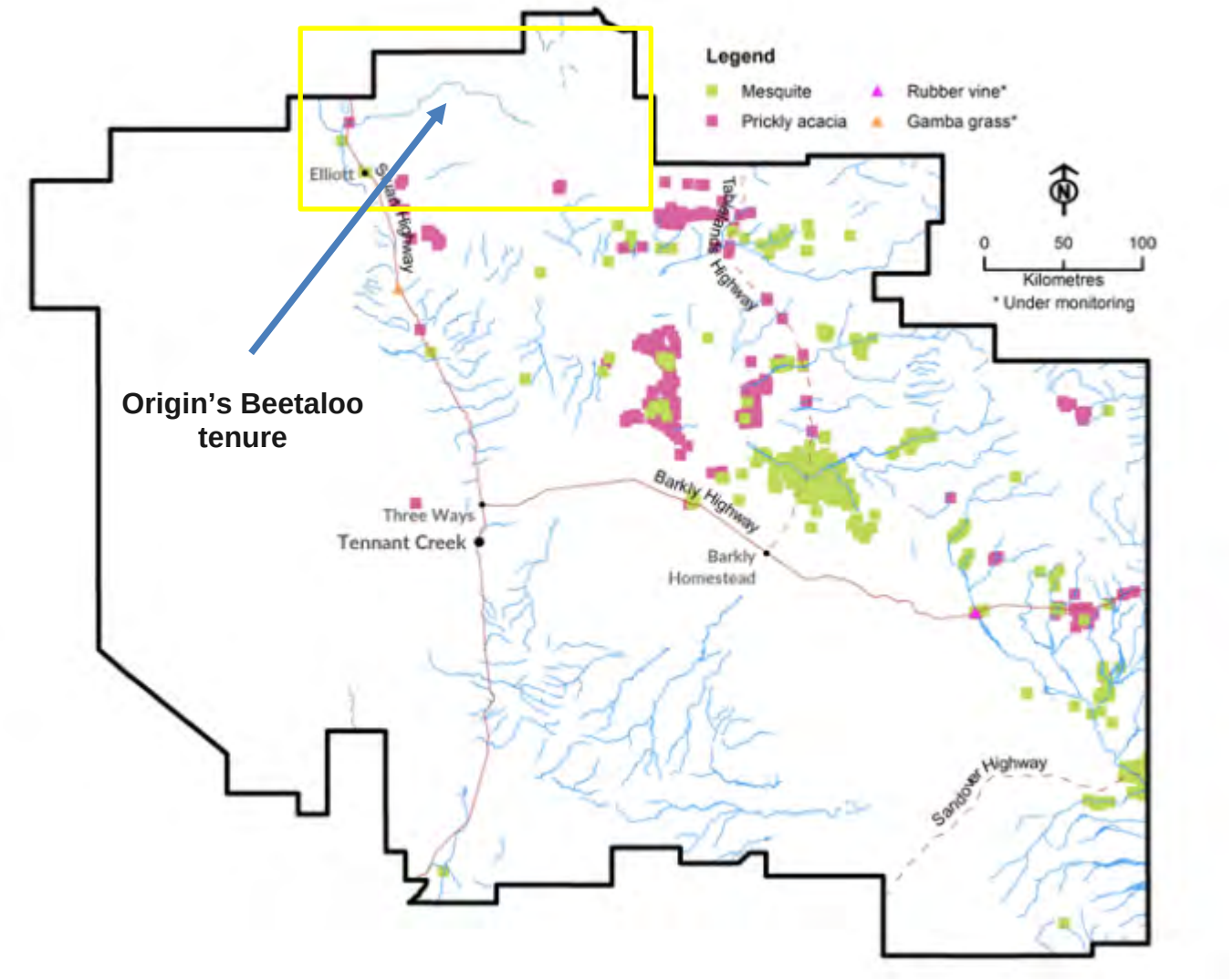


Figure 3 Tennant Creek RWS mapped priority weed for eradication locations (DEPWS 2021)

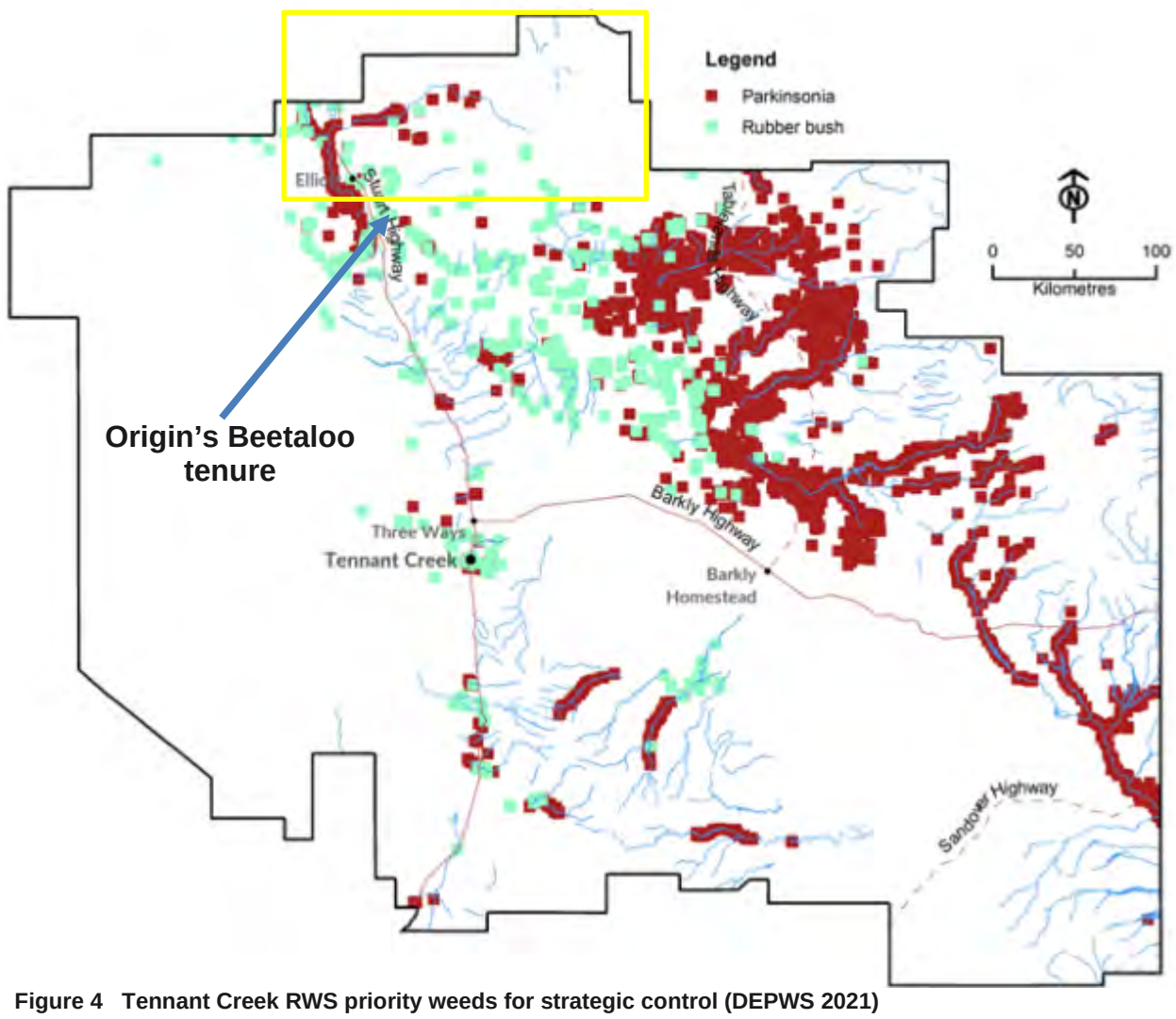


Figure 4 Tennant Creek RWS priority weeds for strategic control (DEPWS 2021)

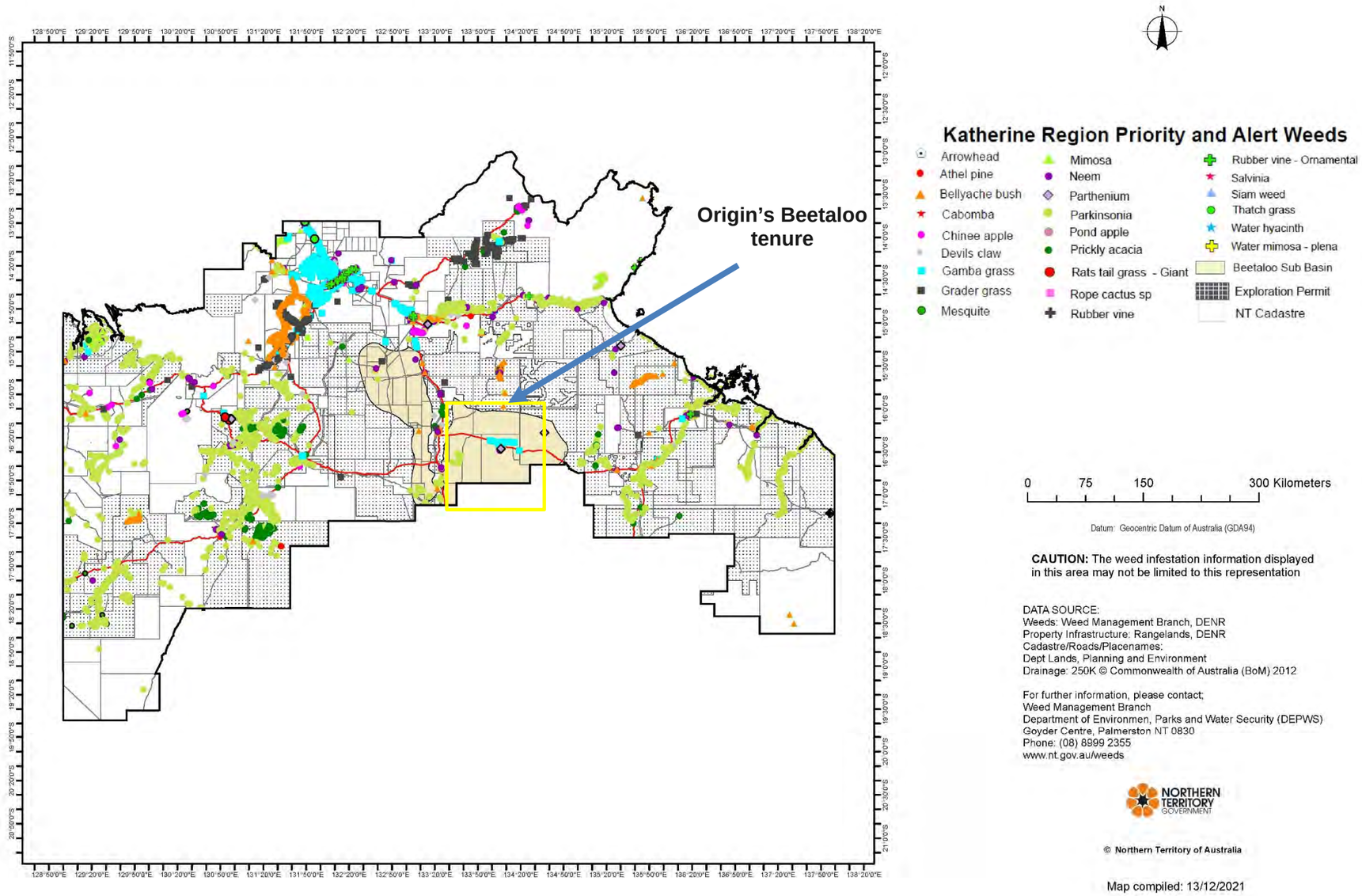


Figure 5 Katherine RWS mapped priority and alert weeds

Table 2 NT listed weeds known of likely to occur within the Permit Area

Scientific Name	Common Name	Status	Category	Data Source
Acacia nilotica	Prickly Acacia	Class A, WoNS	1	Mapped in the exploration lease within the Katherine RWS
Andropogon gayanus	Gamba Grass	Class A WoNS	1	Confirmed within exploration lease. High potential introduction through sourcing of equipment from Katherine and Darwin area.
Calotropis procera	Rubber Bush	Class B and C	2	Mapped in the exploration lease within the Tennant Creek RWS
Cryptostegia grandiflora	Rubber Vine	Class A	1	Mapped in the exploration lease within the Katherine RWS
Hyptis suaveolens	Hyptis	Class B and C	4	Confirmed within exploration lease during previous weed Origin surveys
Jatropha gossypiifolia	Bellyache Bush	Class A, WoNS	1	Mapped in the exploration lease within the Katherine RWS. Potential introduction through sourcing of equipment from Katherine area.
Parkinsonia aculeata	Parkinsonia	Class B and C, WONS	2	Confirmed within exploration lease during previous weed Origin surveys and Mapped in the exploration lease within the Katherine RWS. Potential introduction through sourcing of equipment from Katherine area.
Prosopis pallida	Mesquite	Class A and C, WONS	1	Mapped in the area surrounding exploration lease within the Katherine and Tennant Creek RWS
Themeda quadrivalvis	Grader Grass	Class B and C, WoNs	5	Confirmed within the exploration lease and mapped in the area within the Katherine RWS. High potential introduction through sourcing of equipment from Katherine area.
Parthenium hysterophorus	Parthenium	Class A and Class C, WoNS	1/ 5	Confirmed by DEPWS to occur within the exploration lease. Potential introduction through equipment sourced from QLD.
Cryptostegia grandiflora	Rubber vine	Class A and C, WONS	1	Alert Species within the Tennant Creek and Katherine RWS
Chromolaena odorata	Siam Weed	Class C	5	Alert Species Katherine RWS
Azadirachta indica	Neem	Class B and C	2	Weed Management Branch – Mapping data
Cenchrus ciliaris	Buffel Grass	Not declared in NT	3	DOTEE Protected Matters Report

Scientific Name	Common Name	Status	Category	Data Source
Cenchrus echinatus	Mossman River Grass	Class B and C	3	DLRM databases (DLRM *et al* 2018)
Datura ferox	Fierce Thornapple	Class A and C	3	DLRM databases (DLRM *et al* 2018)
Sida acuta	Spinyhead sida	Class B and C	4	Weed Management Branch – Mapping data
Sida cordifolia	Flannel Weed	Class B and C	4	Weed Management Branch – Mapping data DLRM databases (DLRM *et al* 2018)
Sida rhombifolia	Paddy's Lucerne	Class B and C	4	DLRM databases (DLRM *et al* 2018)
Xanthium occidentale	Noogoora Burr	Class B and C	3	Weed Management Branch – Mapping data DLRM databases (DLRM *et al* 2018)

Note: Declarations under the Northern Territory Weeds *Management Act 2001*:

6. Weed Management Mandatory Requirements

6.1 Weed hygiene declarations for vehicles and equipment

1. All vehicles, equipment and loads are to be clean (free of plant matter, seeds, dirt and mud) and have a valid weed hygiene declaration form prior to accessing any pastoralist property
2. Weed hygiene certificates are only to be issued by an authorised inspector that is satisfied that the vehicle is free of plant matter, seeds, dirt, mud animal wastes and any other time that could potentially represent a biosecurity or weeds risk.
3. An authorised inspector is someone who has successfully completed the nationally recognised "AHCBIO201- Inspect and clean machinery for plan, animal and soil material" training course
4. Weed hygiene declarations shall contain:
   a) The identification details of the vehicle or thing inspected.
   b) Odometer reading (where applicable)
   c) Date and location inspected
   d) Name and signature of the authorised inspector issuing the declaration
   e) The organisation with which the inspector issuing the declaration is affiliated
   f) Name and signature of the driver (where applicable)
5. A biosecurity hygiene declaration for a vehicle/equipment remains valid when the vehicle/equipment:
   a) does not travel off sealed/formed roads, or
   b) clean (i.e. free of biosecurity matter including weeds, pests and diseases, and biosecurity carriers) or
   c) is located on the same or adjacent property and has not come in contact with any areas with weeds. Areas where it is reasonably expected to come in contact with weeds include the unsealed shoulders of road corridors and known infestation areas as provided in Figure 2.
6. A biosecurity declaration becomes invalid when:
   a) The vehicle or equipment has come into contact with known areas of weed infestations.

b) The vehicle or equipment has come from a property that is not adjacent to the property to be accessed
c) It is not known where the vehicle/ equipment has been previously used.

7. Where a vehicle or piece of equipment arrives at site dirty, they shall be refused entry. The vehicle/ equipment must be directed to the closest washdown facility (Daly Waters), recertified and inspected prior to accessing the site.

6.2 Weed hygiene declarations for loads and materials.

1. Weed hygiene declarations are to be utilised to satisfy that a load of materials (including hay, seed, sand, gravel, topsoil) is free of or containing a biosecurity matter and carriers. Anyone who is either the seller, supplier or the driver may issue a Weed Hygiene Declaration for a load just as long as they have direct knowledge of the product and the status as weed free or containing a biosecurity matter.
2. Weed declarations are not required for loads moved within areas within the same or adjacent properties that have been determined through baseline weed studies as being weed free.
3. Where loads of material cannot be determined to be weed free, they shall be returned to the supplier and an alternative clean source utilised.

6.3 Weed washdown facility requirements

1. Cleaning activities should be undertaken at facilities with effective environmental controls to prevent the spread of biosecurity matter.
2. Wash water, mud/ silt, weed material and other contaminants must be bagged and disposed of at a licenced landfill.
3. Where possible, high pressure water spray should be used. This is the preferred method. If this is impractical, (such as at a site location) the minimum requirement is to use a suitable bar or shovel, brooms/ brushes and compressed air to remove contaminants (dry cleaning).

6.4 Equipment sourcing and selection

1. Equipment shall be sourced based on the following prioritisation:
   a) Local equipment, particularly civil construction equipment, shall be sourced as a priority.
   b) Regional equipment (NT) shale be sourced where no local equipment supplier exists
   c) Interstate equipment should be sourced only where local/regional equipment is not available (due to availability or cost constraints). In such cases, additional inspections may be required to ensure vehicles/ equipment are free of weed containing material prior to accessing site.

6.5 Interstate Transportation

All vehicles, equipment and loads moved interstate/territory shall be free of weeds and weed containing material (vegetation, seed, grass, soil, mud etc.) prior to entry into the NT.

All vehicles, equipment and loads travelling from interstate shall have a further inspection prior to access to any pastoral property. If required, additional cleaning shall be undertaken to remove any weeds or weed carrying material.

Where a load/equipment/ vehicle is unclean and is suspected of not being washed prior to entry into the NT, a load must be refused entry into a pastoralist property. The vehicle will require a washdown at an appropriate facility within the state/territory the equipment/vehicle/load originated from.

6.6 Weed management awareness

All staff and contractors shall be made aware of their weed management obligations. This shall be undertaken through:

- Building weed prevention and management requirements into contracts and assessed as a part of work readiness reviews and ongoing assurance activities.
- Inclusion of weed management requirements within site inductions and toolbox talks

7. Weed Introduction and Spread Risks Assessment

As part of the development of the EMP for this project, Origin has undertaken an assessment of the risk of introducing or spreading weeds in the project area. This assessment and the corresponding proposed mitigation measures and management objectives are presented in Table 3 below. Due to the low abundance of weeds within the proposed project area, management controls will primarily focus on preventing the introduction of weed species through appropriate equipment sourcing cleaning and inspection.

Table 3 Risk of weed introduction and spread and corresponding mitigation measures

Environmental Values	Maintain the integrity of significant ecosystems and agricultural productivity		
Management Objectives	Avoid the introduction of weeds Avoid the spread of existing weeds		
Measures Criteria	No introduction or spread of declared weeds resulting from Origin's activities.		
Activity	**Potential Risks**		**Management Controls**
	Introduction of new weeds	**Spread of existing weeds**	
Vehicle and equipment movements	Vehicles and equipment sourced from other locations infested with weed species not found in or around Project Area	Traversing of weed infested areas with machinery	- Code of Practice for Petroleum Activities in the Northern Territory Part A- Surface Activities. - Activities will adhere to the guidelines within the NT Weed Management Handbook. - Weed management and control measures to be implemented in alignment with existing landholder biosecurity requirements. - All equipment will have certified equipment wash-down completed prior to entry to the field. Wash-down would occur at Contractors deport or a commercial wash facility prior to mobilisation in a manner that prevents pollution of the surrounding environment. - Machinery to be preferentially sourced locally, with machinery sourced from surrounding areas or Queensland being the 2nd and 3rd preferred option respectively. - Weeds will be actively controlled in cleared/ hardstand areas. - Major equipment moves will be planned from weed-free areas to infested areas and not the other way around.

Weed Management Plan

NT-2050-15-MP-0016

Environmental Values	Maintain the integrity of significant ecosystems and agricultural productivity		
Management Objectives	Avoid the introduction of weeds Avoid the spread of existing weeds		
Measures Criteria	No introduction or spread of declared weeds resulting from Origin's activities.		
Activity	**Potential Risks**		**Management Controls**
	Introduction of new weeds	**Spread of existing weeds**	
			- Ensuring all material imported to or between sites is free of weeds.
Construction of access tracks and monitoring bore pads	Importing materials from areas where weeds are present and creating opportunities for weed species to colonise disturbed areas	Traversing of weed infested areas and creating opportunities for weed species to colonise disturbed areas	- Code of Practice for Petroleum Activities in the Northern Territory Part A- Surface Activities. - Activities will adhere to the guidelines within the NT Weed Management Handbook. - Weed management and control measures to be implemented in alignment with existing landholder biosecurity requirements. - All equipment will have certified equipment wash-down completed prior to entry to the field. - Ensure field staff, contractors and machinery operators are familiar with hygiene protocols and weed identification. - Machinery to be preferentially sourced locally, with machinery sourced from surrounding areas or Queensland being the 2nd and 3rd preferred option respectively. - Weeds will be actively controlled in cleared/ hardstand areas. - Stabilise disturbed areas.
Drilling, stimulation and well testing	Introduction of weed species not found in or around EP area.	Traversing of weed infested areas with machinery	- Code of Practice for Petroleum Activities in the Northern Territory Part A- Surface Activities. - Activities will adhere to the guidelines within the NT Weed Management Handbook. - Weed management and control measures to be implemented in alignment with existing landholder biosecurity requirements. - All equipment will have certified equipment wash-down completed prior to entry to the field. Wash-down would occur at Contractors deport or a commercial wash facility prior to mobilisation in a manner that prevents pollution of the surrounding environment. - Ensure field staff, contractors and machinery operators are familiar with hygiene protocols and weed identification. - Weeds will be actively controlled in cleared/ hardstand areas.

Review due: 29/03/2024

Weed Management Plan

NT-2050-15-MP-0016

Environmental Values	Maintain the integrity of significant ecosystems and agricultural productivity		
Management Objectives	Avoid the introduction of weeds Avoid the spread of existing weeds		
Measures Criteria	No introduction or spread of declared weeds resulting from Origin's activities.		
Activity	**Potential Risks**		**Management Controls**
	Introduction of new weeds	**Spread of existing weeds**	
			- Major equipment moves will be planned from weed-free areas to infested areas and not the other way around. - Drilling and stimulation equipment will be restricted to cleared lease areas. - Ensuring all material imported to or between sites is free of weeds.
Operational/ site management	Personnel unable to identify weeds or unaware of weed species present in areas where machinery and equipment is sourced from	Existing weed distribution not known due to: insufficient survey effort, surveys conducted at wrong time of year, surveyors not familiar with / unable to identify declared weed species	- Code of Practice for Petroleum Activities in the Northern Territory Part A- Surface Activities. - Staff members responsible for preventing, identifying and managing weeds to be appropriately trained. - Weed desktop and field-based surveys to be provided to identify existing weed areas. - Pre-and post wet (February to May) inspections and periodic audits will be conducted to identify and report weed outbreaks.
	Insufficient management control to prevent the introduction of weeds	Insufficient management control to prevent the spread of weeds	- Staff members responsible for preventing, identifying and managing weeds to be appropriately trained. - Ensure field staff, contractors and machinery operators are familiar with hygiene protocols and weed identification (Weed identification posters and the NTG Weed Deck will be made available) - Weeds will be actively controlled in cleared/ hardstand areas. - Weed management and control measures to be implemented in alignment with existing landholder biosecurity requirements. - New activities will be planned to address prevention of weed or non-indigenous plant spread.

8. Statutory Weed Management Plans

No statutory weeds have been identified during surveys of the Project Area; however the following plans apply to species that have been found/ could be potential found in the broader region:

- Weed Management Plan for Athel pine (*Tamarix aphylla*)
- Weed Management Plan for Mesquite (*Prosopis* spp.)
- Weed Management Plan for Prickly Acacia (*Acacia nilotica*)
- Weed Management Plan for Bellyache Bush (*Jatropha gossypiifolia*)
- Weed Management Plan for Neem (*Azadirachta indica*)
- Weed Management Plan for Gamba Grass (*Andropogon gayanus*)
- Weed Management Plan for Grader Grass (*Themeda quadrivalvis*).

The weed management plans detail the legislated obligations of all landowners, land managers and land users in the Northern Territory to eradicate or manage and avoid further spread of the weed species. Conducting land management practices in accordance with the weed management plans will secure compliance with the requirements of the Act (DEPWS 2021).

9. Annual Action Plan

An action plan for each of the weed species identified in the Project Area is presented in Table 4. Treatment options as contained in the Northern Territory Weed Management Handbook are presented in Section 9.1 to Section 9.3.

This section will be undated if new weed species are discovered over the life of the program to ensure that statutory requirements with relation to declaration status and relevant weed management plans are addressed (refer to Section 0).

As part of the 2019 Annual Weed Management Action Plan, Origin also commits to undertaking finer detailed weed mapping of all permit area, lease pads, access tracks and gravel pits, as well as any other areas disturbed as part activity.

.

Table 4 Annual Weed Management Action Plan

Management objective	- Avoid the introduction of weeds - Avoid the spread of existing weeds			
Weed species	**Survey time/s**	**Treatment time/s**	**Control options**	**Where located**
Hyptis *Hyptis suaveolens*	6 monthly- pre-and post wet season	- Preferred Dec – Mar - Also Nov and April	Refer to section 9.1.	Beetaloo access track Access track to Amungee Nw Kalala S1 site Velkerri 76 S2 camp pad
Parkinsonia *Parkinsonia aculeata*	6 monthly- pre-and post wet season	- Preferred Mar – May - Also all year round	Refer to section 9.2.	Beetaloo access track
Rubber Bush *Calotropis procera*	6 monthly- pre-and post wet season	- Preferred October – March - April - July	Refer to section 9.3.	Close proximity to the Beetaloo access track Kyalla 117 N2 access track and Stuart Highway intersection

9.1 Hyptis (*Hyptis suaveolens*) treatment options

Table 5 includes herbicide and non-chemical treatment options for Hyptis *(Hyptis suaveolens) (*Northern Territory Government 2021).

Table 5 Hyptis *(Hyptis suaveolens)* treatment options

Weed Species	Hyptis *(Hyptis suaveolens)*		
Control Methods	**Chemical and concentration**	**Rates**	**Weed growth stage, method and comments**
Herbicides	**2, 4-D amine 625 g/L** Various trade names	320 mL / 100 L	**Seedling or adult (individuals or infestation):** Foliar spray – apply when actively growing.
	Glyphosate 360 g/L Various trade names and formulations	15 mL / 1 L	**Seedling or adult (individuals or infestation):** Foliar spray – apply when actively growing.

Weed Species	Hyptis *(Hyptis suaveolens)*		
Control Methods	**Chemical and concentration**	**Rates**	**Weed growth stage, method and comments**
Non-chemical applications	- Manually remove all plant material; slash to encourage competition from desirable species.		

Source: Northern Territory Weed Management Handbook (Northern Territory Government 2021).

9.2 Parkinsonia (*Parkinsonia aculeata*) treatment options

Table 6 includes herbicide and non-chemical treatment options for Parkinsonia *(Parkinsonia aculeata) (*Northern Territory Government 2021).

Table 6 Parkinsonia *(Parkinsonia aculeata)* treatment options

Weed Species	Parkinsonia *(Parkinsonia aculeata)*		
Control Methods	**Chemical and concentration**	**Rate**	**Weed growth stage, method and comments**
Herbicides	**Aminopyralid 8 g/L + Triclopyr 300 g/L + Picloram 100 g/L** Grazon™ Extra	350 mL / 100 L or 3 L / ha	**Seedling (individuals and infestation)** Foliar spray – avoid spraying if plants are stressed or bearing pods – Uptake Spraying Oil required Foliar spray – plants up to 2 m or 2 years old - Uptake Spraying Oil required.
	Triclopyr 240 g/L + Picloram 120 g/L Access™	1 L / 60 L (diesel) 1 L / 60 L (diesel)	**Seedling or adult (individuals or infestation)** Basal bark < 5 cm stem diameter Cut stump > 5 cm stem diameter
	Tebuthiuron 200 g/kg	1.5 g / m^2	**Seedling or adult (individuals or infestation)** Granulated herbicide - ground applied Do not use within 30 m of desirable trees or apply to continuous area > 0.5 ha. Do not use if fire is eminent. Apply when there is soil moisture or prior to rain.
Non-chemical applications	- Blade-ploughing, stick-raking, bulldozing and chaining can be effective if the root layer is removed from the soil. - Cultivation of pasture or native vegetation after mechanical control will help to prevent re-sprouting and seedling establishment. - Fire destroys seed in the soil surface and can be used as a follow-up to remove seedlings after other control efforts.		

Weed Species	Parkinsonia *(Parkinsonia aculeata)*		
Control Methods	**Chemical and concentration**	**Rate**	**Weed growth stage, method and comments**
	- Fire may also be used to manage mature trees. Hand grubbing for single plants or small outbreaks, ensure removal of the root system. - Biocontrol options are available with Uu establishing slowly in some areas.		

Source: Northern Territory Weed Management Handbook (Northern Territory Government 2021).

9.3 Rubber bush (*Calotropis procera*) treatment options

Table 7 includes herbicide and non-chemical treatment options for Rubber bush *(Calotropis procera) (*Northern Territory Government 2021).

Table 7 Rubber bush *(Calotropis procera)* treatment options

Weed Species	Rubber bush *(Calotropis procera)*		
Control Methods	**Chemical and concentration**	**Rate**	**Weed growth stage, method and comments**
Herbicides	**Triclopyr 300 g/L + Picloram 100 g/L** **Conqueror®** **+ Aminopyralid 8 g/L** Grazon™ Extra	750 mL / 100 L (water) 500-750mL / 100 L (water)	**Seedling (individuals or infestation):** Foliar spray. Check label for recommended adjuvant product. More effective on plants <2m as thorough coverage on all leaves is required
	Triclopyr 240 g/L + Picloram 120 g/L Access™	 1 L / 60 L (diesel) 1 L / 10 L (diesel) 1 L / 60 L (diesel)	**Adult (individuals and infestation):** Basal bark < 5cm stem diameter. Spray all stems. Spray to point of runoff. Thin Line up to 5cm stem diameter. Cut stump > 5cm stem diameter.
	Tebuthiuron (200g/kg) Graslan Pending registration. Please check with Weed Management Branch for status confirmation.	1.5-2g/m^2	**Seedling or adult:** Application to black clay soils in conjunction with seasonal rainfall. Spread granules according to density of the infestation.
	Fluroxypyr (333g/L)	3 L / 100 L	**Adult:** Cut stump method for plants up to 10cm diameter and 3m high.

Weed Species	Rubber bush *(Calotropis procera)*		
Control Methods	**Chemical and concentration**	**Rate**	**Weed growth stage, method and comments**
	Starane™ Advanced	(diesel)	
Non-chemical applications	- This plant is difficult to eradicate as the deep roots survive almost any treatment. - Maintenance of a dense pasture sward will assist in preventing invasion.		

Source: Northern Territory Weed Management Handbook (Northern Territory Government 2021).

10. Notification Procedure

The Onshore Petroleum Weed Management Officer at the Weed Management Branch of the DEPWS should be notified within 48 hours of the discovery of a new weed species in the Project Area.

Initial notification may be verbal, with follow-up written notification provided within seven working days. The notification should include a preliminary species identification and location information. The Regional Weed Officer will advise what further action is required.

It is noted that some species spread rapidly so immediate action may be required to control spread. For example, as stated above *Parthenium (Parthenium hysterophorus) is a Class A (to be eradicated) and Class C (not to be introduced) weed in the Northern Territory as well as being classified as a Weed of National Significance. Early detection is crucial in not allowing this species to spread in the Northern Territory* (Department of Primary Industry and Resources 2016).

In addition, it is noted that under the *Weeds Management Act* that:

'The owner and occupier of land must… within 14 days after becoming aware of a declared weed that has not previously been, or known to have been, present on the land, notify an officer of the presence of the declared weed'.

All weed outbreak incidents will be reported in Origin's OCIS and corrective action initiated.

11. Recording

Records of weed inspections will be maintained by Origin.

Data on weed distribution will be maintained within Origin's GIS and provided to the Weeds Officer at DEPWS as part of the annual report on performance against the Weed Management Plan, or as requested.

Data will be collected as per the requirements of the Northern Territory Weed Data Collection Manual - Section One Technical Data Description (Weed Management Branch, 2015).

Data will be recorded using the guidelines provided in Appendix A using the data sheet provided in Appendix B (Weed Management Branch, 2015).

The Northern Territory Weed ID Deck (Northern Territory Government 2021) will be referenced to assist with identification of species that have been identified as likely or know to occur in the Permit Area.

Field data will be submitted directly to the Weed Management Branch in a shapefile format or as an Excel spreadsheet, including incidental identification of weeds and following completion of field surveys.

12. Reporting

All weed outbreak incidents will be reported in Origin's OCIS and corrective action initiated.

A report on the performance against this Weed Management Plan will be submitted to DEPWS on an annual basis.

At a minimum, this should include:

a) Details of activities implemented to address weed spread and introduction risks (e.g. vehicle wash down/ blow down locations, examples of track construction from working from weed free areas into weed infested areas to reduce spread).

b) Details of survey and monitoring events, including dates, personnel, maps and track data.

c) Submission of all weed data collected.

d) Overview of weed control events and success rates (weed control should be captured in detail through the data collection process and submitted as a component of (a)).

13. References

Department of Climate Change, Energy, the Environment and Water. 2018. *Key threatening processes under the EPBC Act.* http://www.environment.gov.au/biodiversity/threatened/key-threatening-processes accessed14 September 2018.

Department of Environment, Parks and Water Security. 2020. *Weed Management Plan. Gamba Grass 2020-2030.* https://nt.gov.au/__data/assets/pdf_file/0006/954789/weed-management-plan-for-gamba-grass-2020-2030.PDF.

Department of Environment, Parks and Water Security. 2021. *Katherine regional weeds strategy 2021 – 2026. https://depws.nt.gov.au/__data/assets/pdf_file/0006/269286/Katherine-Regional-Weeds-Strategy-2021-2026.pdf.*

Department of Environment, Parks and Water Security. 2021. *Tennant Creek Regional Weeds Strategy 2021-2026.* https://depws.nt.gov.au/__data/assets/pdf_file/0006/258099/tennant-creek-regional-weeds-strategy.pdf, accessed 18 January 2022.

Department of Environment, Parks and Water Security. 2021. *Weed Management Act: Compliance Policy, December 2021*. https://nt.gov.au/__data/assets/pdf_file/0011/668387/weeds-management-act-compliance-policy.pdf accessed 30 August 2022.

Department of Land Resource Management and Charles Darwin University. 2018, *Guidelines for the Management of the Weeds of Beetaloo 2018.*

Department of Natural Resources and Environment. 2019. *Weed Management Planning Guide - Onshore Petroleum Projects,* June 2019*.* https://denr.nt.gov.au/__data/assets/pdf_file/0006/708558/weed-management-planning-guide-onshore-petroleum-projects.pdf.

Department of Primary Industry and Resources. 2016. Partheneum found in the NT." https://dpir.nt.gov.au/news/2016/december/parthenium-found-in-the-nt accessed 14 September 2018.

Northern Territory Government. 2000. *Information Sheet Gamba Grass*. http://www.drytropics.org.au/weeds_gamba_control.htm accessed 29 March 2019.

Northern Territory Government. 2021. *Northern Territory Weed Management Handbook*. https://nt.gov.au/__data/assets/pdf_file/0004/233833/nt-weed-management-handbook.pdf.

Northern Territory of Australia. 2021. *Northern Territory Weed ID Deck.* https://depws.nt.gov.au/__data/assets/pdf_file/0012/257988/2021-weed-id-desk.pdf.

Northern Territory Government. 2018. *A – Z List of Weeds in the Northern Territory*. https://nt.gov.au/environment/weeds/weeds-in-the-nt/A-Z-list-of-weeds-in-the-NT accessed 13 September 2018.

Scientific Inquiry into Hydraulic Fracturing in the Northern Territory. 2018. *Scientific Inquiry into Hydraulic Fracturing in the Northern Territory – Final Report.*

Weed Management Branch, Northern Territory Government. 2015. *Northern Territory Weed Data Collection Manual - Section One Technical Data Description.*https://nt.gov.au/__data/assets/pdf_file/0007/233854/nt-weed-data-collection-manual-section-1.pdf.

Appendix A Weed Data Collection Methodology

Field data collection for weed infestations

The following is a guide to efficiently evaluating and recording a weed site in the field.

Each record must identify the person or organisation taking the record, as well as the details explained below.

How to record weed area as a point record

1. Record the species.

When a weed is sighted, move to the area and confirm identification of the weed. If you cannot positively identify the weed record it as "Unknown weed" and take a sample or photograph, do not try to guess. If more than one weed species is present then repeat the process with separate records for each species.

2. Assess the size of the weed patch.

Look across the area of weeds to the furthest weed plant and decide the diameter. Decide if the area is best fits in a circle of either 20, 50 or 100 metres. If it is a single plant or small patch you would choose 20 metres. The size 100 metres extends about as far as you can see on the ground, if the weeds extend out of sight you will need to make another point further on. You may place overlapping circle areas to reflect different densities.

3. Assess the density of weeds within the circle.

Decide how much of the area is covered by weeds. Assign a score from 2 to 5 based on the percentage table below. It will be useful (if possible) to move into the centre of the weed circle. Consider the whole circle size chosen in step 2 deciding on the density score. Area covered should be determined by a 'projected canopy' method.

Density categories

1 = Absent, no weeds of this species in this area.
2 = < 1%, Very few, not many weeds eg: single plant, perhaps with seedlings.
3 = 1 -10%, More than one or two isolated plants but not a lot eg: a few small plants.
4 = 11-50%, A lot, up to half the area covered eg: a tree, dense patches of weeds.
5 = > 50%, Dominant cover is weed, more than half covered eg: thickets, monocultures.

4. Record the location.

Take the GPS location (ideally) from the centre of the circle. If weed seeds may be spread or it is difficult to access the centre it is acceptable to take the reading from the location as close to the centre as practical.

5. Record the treatment.

Record the method you apply a treatment to the weeds, or record 'No Treatment'.
Choose from the list of treatment methods
i.e: No treatment, Unknown, Treated, Foliar spray etc.

How to record weed area as a line (polyline) record

1. Record the species.

When a weed is sighted, move to the area and confirm identification of the weed. If you cannot positively identify the weed record it as "Unknown weed" and take a sample or photograph, do not try to guess. If more than one weed species is present then repeat the process with separate records for each species.

2. Assess the 'best fit' width in metres of the linear weed area.

Look along the area of weeds to the furthest weed plant and decide a width that best sums up the width of the infestation from values of 5, 20, 50 or 100 metres. If the width is too variable you may need to make more than one line or consider recording as points or as a polygon.

3. Assess the density of weeds within the line.

For the area of the line, being from start to finish at the designated width, decide the area covered by weeds. Assign a score from 2 to 5 based on the percentage table below. Consider the whole line area when deciding on the density score. Area covered should be determined by a 'projected canopy' method.

Density categories
1 = Absent, no weeds of this species in this area.
2 = < 1%, Very few, not many weeds eg: single plant, perhaps with seedlings.
3 = 1 -10%, More than one or two isolated plants but not a lot eg: a few small plants.
4 = 11-50%, A lot, up to half the area covered eg: a tree, dense patches of weeds.
5 = > 50%, Dominant cover is weed, more than half covered eg: thickets, monocultures.

4. Record the location.

Start the GPS track, or line sketch from one end of the linear weed area. Walk or sketch a line as best fit through the middle of the linear weed area and finish at the end point.

5. Record the treatment.

Record the method you apply a treatment to the weeds, or record 'No Treatment'.
Choose from the list of treatment methods
ie: No treatment, Unknown, Treated, Foliar spray etc.

How to record weed area as a polygon record

1. Record the species.

When a weed is sighted, move to the area and confirm identification of the weed. If you cannot positively identify the weed record it as "Unknown weed" and take a sample or photograph, do not try to guess. If more than one weed species is present then repeat the process with separate records for each species.

2. Assess the extent of the weed area an ensure it can be practically enclosed.

Polygons are good for clearly delineated areas of weeds, you should be able to walk around the edge of the weed area with confidence. Ensure the defined area of weed at a similar density can be delineated before attempting to create the area, you may need more than one polygon. If the area is poorly defined then the point method may be a more useful.

3. Assess the density of weeds within the polygon.

Assess the area covered by weeds for density, you may need to move to several vantage points to get a clear picture. Assign a score from 2 to 5 based on the percentage table below. Consider the whole area within the polygon when deciding on the density score. Area covered should be determined by a 'projected canopy' method.

Density categories
1 = Absent, no weeds of this species in this area.
2 = < 1%, Very few, not many weeds eg: single plant, perhaps with seedlings.
3 = 1 -10%, More than one or two isolated plants but not a lot eg: a few small plants.
4 = 11-50%, A lot, up to half the area covered eg: a tree, dense patches of weeds.
5 = > 50%, Dominant cover is weed, more than half covered eg: thickets, monocultures.

4. Record the location.

Start the GPS track, or polygon sketch from one point of the polygon weed area. It is useful to start from a landmark or flagging tape. Create the polygon edge line by walk a path or sketching along the outer edge of the weed area until you return to the start point. If using a GPS track to create the polygon ensure that you cross your start point so as to close the polygon.

5. Record the treatment.

Record the method you apply a treatment to the weeds in the area, or record 'No Treatment'. Choose from the list of treatment methods

ie: No treatment, Unknown, Treated, Foliar spray etc.

Appendix B Example Weed Data Collection Sheet

Weed Management Plan

NT-2050-15-MP-0016

RECORDER:					PROJECT:							LOCALITY:		
ORG_NAME:					GPS NAME/MODEL:							RECORDING METHOD :		
SITE_ID	DATE_REC	LAT_G94	LONG_G94	WEED_NAME	SIZE_DIA_M	DENS_CAT	SEEDLINGS	JUVENILES	ADULTS	SEED_PRES	PAST_TREAT	TREATMENT	HERBICIDE	COMMENTS

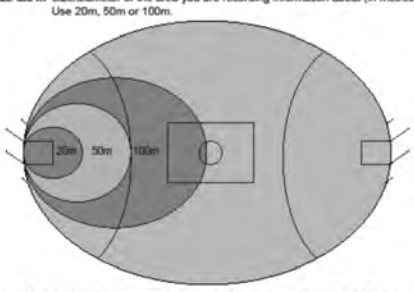
Notes:

Treatment method
Control method applied today as per below.
If none, record 'No treatment'

- Foliar spray
- Residual application
- Basal bark
- Cut stump
- Stem injection
- Aerial spray
- Slashed or cut
- Hand pull

Herbicide The active ingredient(s) of the herbicide applied today (if any)
GPS waypt Waypoint ID as entered in the GPS
Weed name Common name or scientific name for the weed recorded
S (y/n) Seedlings: Are seedlings visible?
J (y/n) Juveniles: Are juvenile plants visible?
A (y/n) Adults: Are there adult plants, or seeds, or evidence of past seeding present?
Seed (y/n) Seeds: Are seeds visible today? Or plants with seeds or pods?
Treat (y/n) Treatment: Did you apply treatment to this site?
Comment Record any notes for yourself here.

Size dia m Size/diameter of the area you are recording information about (in metres). Use 20m, 50m or 100m.

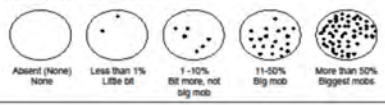


Example of size/diameter compared to a football oval. (Sizes 20m, 50m, 100m)

Dens cat Density of weeds in the assessed area using categories described below

1 = No weeds (absent)	2 = Single plant or very few (<1%)	3 = A few plants (1-10%)
4 = Many weeds, up to half (11 – 50%)	5 = Mostly weeds, more than 50%	6 = Density not assessed

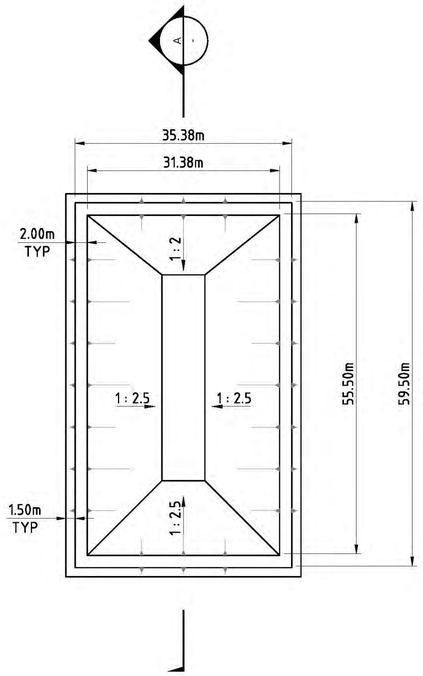
Density category (Dens cat) examples



(extracted from *Northern Territory Weed Data Collection Manual - Section One Technical Data Description*

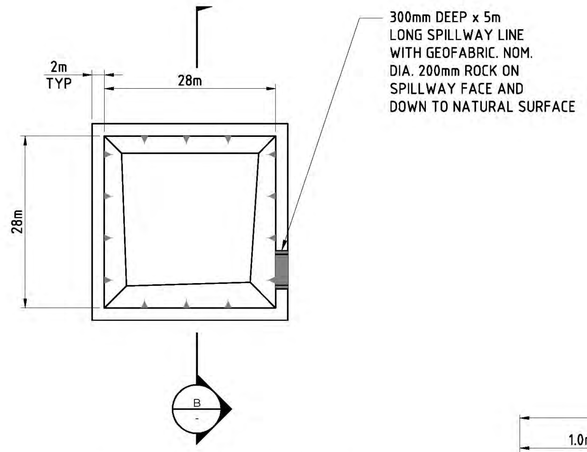
Review due: 29/03/2024

Appendix C Engineering drawing, layouts and specifications



PLAN - MUD SUMP

Scale N.T.S.



PLAN - SECONDARY CONTAINMENT

Scale N.T.S.

NOTES:

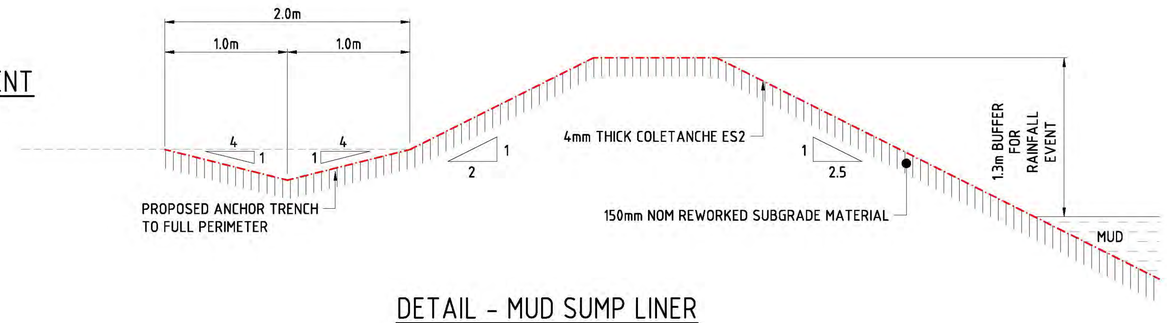
1. ALL UNITS ARE IN METRES (m) UNLESS NOTED OTHERWISE.
2. GROUND CONDITIONS AND SUITABLE MATERIALS TO BE CONFIRMED ON SITE BY SUPERINTENDENT.
3. REFER TO SPECIFICATION FOR COMPACTION REQUIREMENTS.
4. REFER TO SPECIFICATION FOR GENERAL REQUIREMENTS.
5. THE CONTRACTOR IS TO NOTIFY THE ORIGIN SUPERVISOR IF AREAS OF SOFT GROUND ARE ENCOUNTERED OR IF THE SPECIFICATION CANNOT BE ACHIEVED.
6. REFER TO DRAWING NT-2050-20-DA-0022 FOR SETOUT DETAILS OF MUD SUMP.
7. FAUNA LADDERS TO BE INSTALLED TO MUD SUMP.
8. CONTRACTOR TO PROVIDE CATTLE PANELS FOR FENCING AROUND SUMP WHERE DEPTH IS GREATER THAN 1.5m.
9. CATTLE PANEL FENCING TO BE INSTALLED AROUND MUD SUMP AND SEDIMENT BASIN INCLUDING CRITTER FENCING.
10. MUD SUMP VEHICLE OFFLOAD AREA TO BE COMPACTED TO 200 KPa

CERTIFICATION

DISCIPLINE	CERTIFIER	RPEQ No.
CIVIL	J.JENTZ	5583

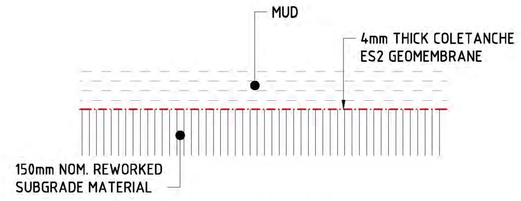
NOTES:

1. GEOSYNTHETICS ARE DRAWN EXAGGERATED FOR CLARITY.



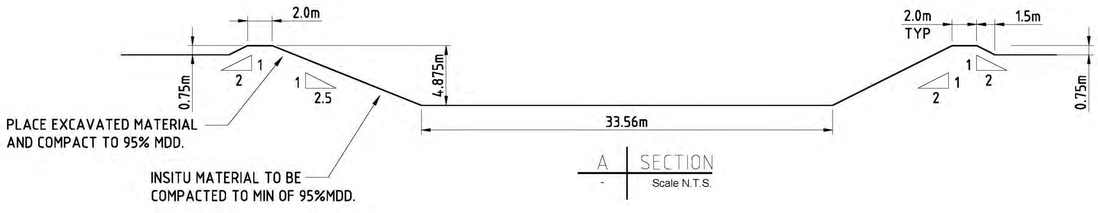
DETAIL - MUD SUMP LINER

Scale N.T.S.

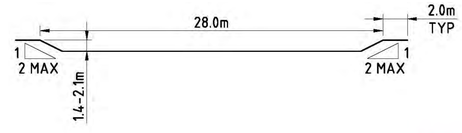


DETAIL - BASE LINER

Scale N.T.S.



A SECTION
Scale N.T.S.



B SECTION
Scale N.T.S.

ISSUED FOR CONSTRUCTION

DRAWING NO.	REFERENCE DRAWING TITLE	REV	DATE	REVISION DESCRIPTION	DRAWN	DWG CHECK	DESIGN	ENG DES CHECK	DE DRAFT SUPER	DE PROJECT APPROVAL
		3	04.10.19	REISSUED FOR CONSTRUCTION	CD	PM	CD	JJ		
		2	25.09.19	LAYOUT UPDATES FOR REVIEW	CD	PM	CD	JJ		
		1	24.05.19	REISSUED FOR CONSTRUCTION	CD	PM	CD	JJ	MR	MR
NT-2050-20-DA-0023	GENERAL ARRANGEMENT PLAN	0	24.04.19	ISSUED FOR CONSTRUCTION	CRD	PCM	PCM	JMJ		

APPROVALS | PROJECT APPROVALS - OTHERS

PREPARED FOR ORIGIN BY:
AECOM
AECOM Australia Pty Ltd
A.B.N 20 093 846 925
www.aecom.com

PROJECT: 60480548 BEETALOO EXPLORATION
DRAWING OFFICE: DARWIN

origin

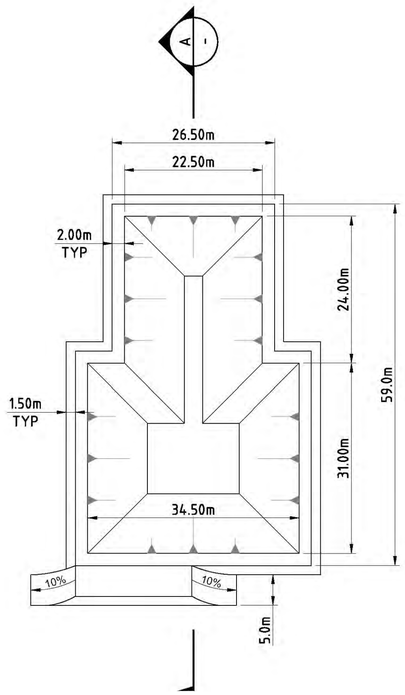
Origin Energy Ltd
ABN 30 000 051 696
GPO Box 148
Brisbane Qld. 4001
Ph: (07) 3858 0600
Fax: (07) 3369 7840

ORIGIN		BY	DATE
	DRAWN	CD	23.04.2019
	DWG CHECK	PM	23.04.2019
	DESIGN	CD	23.04.2019
	ENG DES CHECK	JJ	23.04.2019
	DRAFT. SUPER.	MR	30.05.2019
	PROJECT APPROVAL	MR	30.05.2019

TITLE
BEETALOO EXPLORATION DRILL CAMPAIGN
INDICATIVE LAYOUT
MUD SUMP DETAILS

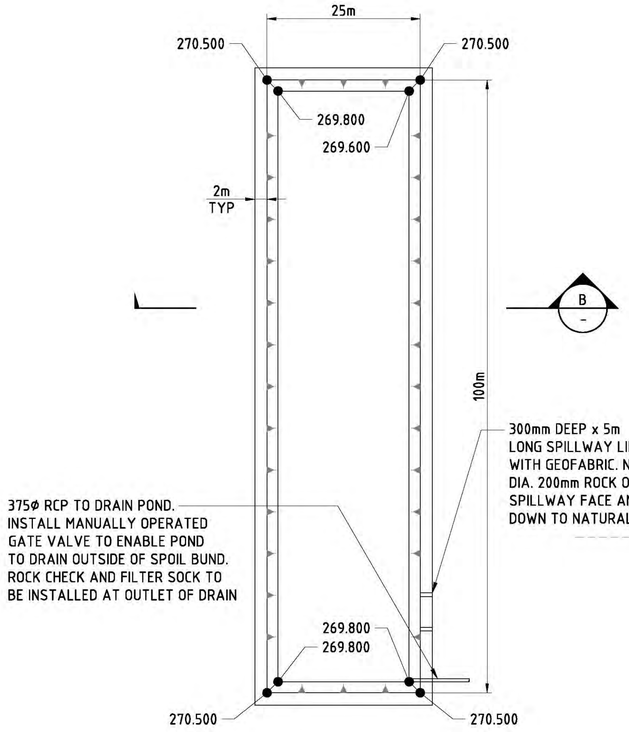
PROJECT NO.
MOD NO.
SCALE: 1:1000 @ A1
DRAWING NO.: NT-2050-20-DD-0031
REVISION: 3
CADFILE: NT-2050-20-DD-0031.dwg
A1

Mon, 28 Oct 2019 - 10:47am P:\604X\604895&NS CAD\20-SHEETS\VELKERRI 76 S2-1\NT-2050-20-DD-0031.DWG Dudley, Calvin



PLAN - MUD SUMP

Scale N.T.S.



PLAN - SECONDARY CONTAINMENT

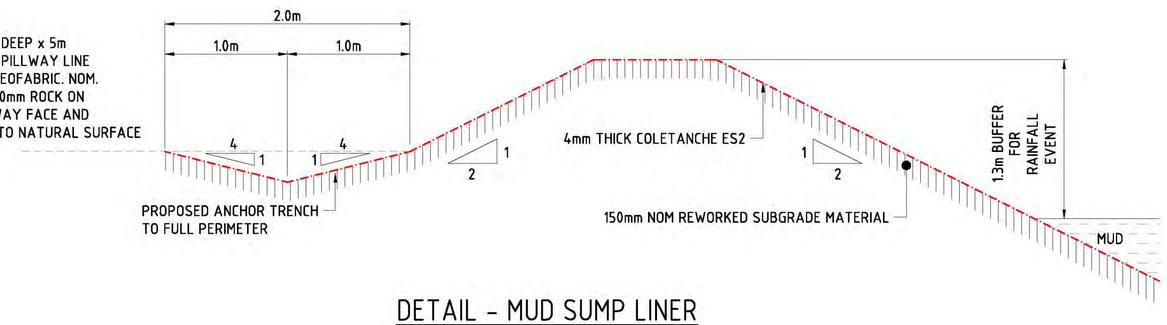
Scale N.T.S.

NOTES:

1. ALL UNITS ARE IN MILLIMETRES (mm) UNLESS NOTED OTHERWISE.
2. GROUND CONDITIONS AND SUITABLE MATERIALS TO BE CONFIRMED ON SITE BY SUPERINTENDENT.
3. REFER TO SPECIFICATION FOR COMPACTION REQUIREMENTS.
4. REFER TO SPECIFICATION FOR GENERAL REQUIREMENTS.
5. THE CONTRACTOR IS TO NOTIFY THE ORIGIN SUPERVISOR IF AREAS OF SOFT GROUND ARE ENCOUNTERED OR IF THE SPECIFICATION CANNOT BE ACHIEVED.
6. REFER TO DRAWING NT-2050-20-DA-0023 FOR SETOUT DETAILS OF MUD SUMP.
7. FAUNA LADDERS TO BE INSTALLED TO MUD SUMP.
8. CONTRACTOR TO PROVIDE CATTLE PANELS FOR FENCING AROUND SUMP WHERE DEPTH IS GREATER THAN 1.5m.
9. FENCE OR BARRICADE TO BE INSTALLED AT SUMP LOAD OUT LOCATION TO ENSURE VEHICLES DO NOT COMPROMISE SUMP WALL/ENTER SUM.
10. MUD SUMP VEHICLE OFFLOAD AREA TO BE COMPACTED TO 200 KPa

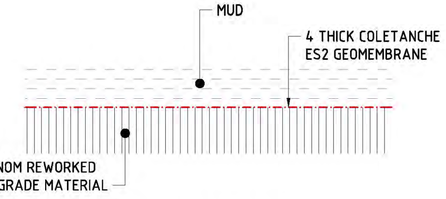
NOTES:

1. GEOSYNTHETICS ARE DRAWN EXAGGERATED FOR CLARITY.



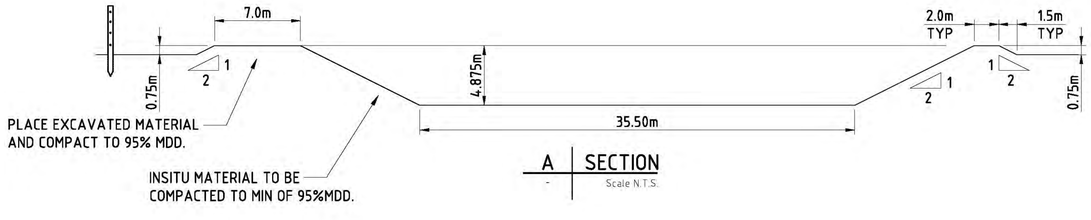
DETAIL - MUD SUMP LINER

Scale N.T.S.

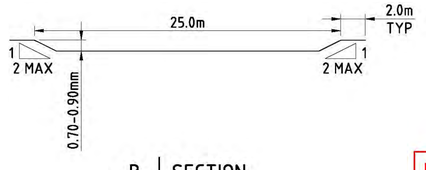


DETAIL - BASE LINER

Scale N.T.S.



A | SECTION
- | Scale N.T.S.



B | SECTION
- | Scale N.T.S.

ISSUED FOR CONSTRUCTION

DRAWING NO.	REFERENCE DRAWING TITLE	REV	DATE	REVISION DESCRIPTION	DRAWN	DWG CHECK	DESIGN	ENG DES CHECK	DE DRAFT SUPER	DE PROJECT APPROVAL
		1	16.04.19	REISSUED FOR CONSTRUCTION	CRD	PCM	PCM	JMJ		
NT-2050-20-DA-0022	GENERAL ARRANGEMENT PLAN	0	11.04.19	ISSUED FOR CONSTRUCTION	CRD	PCM	PCM	JMJ		

APPROVALS

PREPARED FOR ORIGIN BY:

AECOM

AECOM Australia Pty Ltd
A.B.N 20 093 846 925
www.aecom.com

PROJECT: 60480548 BEETALOO EXPLORATION
DRAWING OFFICE: DARWIN
PROJECT APPROVALS - OTHERS

origin

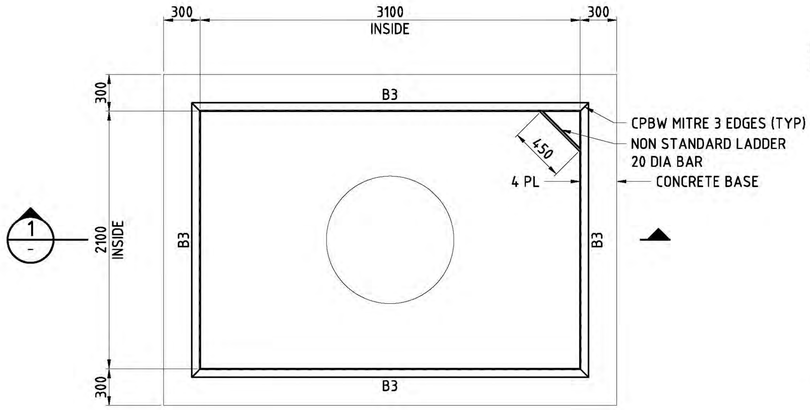
Origin Energy Ltd
ABN 30 000 051 696
GPO Box 148
Brisbane Qld. 4001
Ph: (07) 3858 0600
Fax: (07) 3369 7840

	BY	DATE
DRAWN	CRD	20.03.2019
DWG CHECK	PCM	20.03.2019
DESIGN	PCM	20.03.2019
ENG DES CHECK	JMJ	20.03.2019
DE DRAFT SUPER		
DE PROJECT APPROVAL		

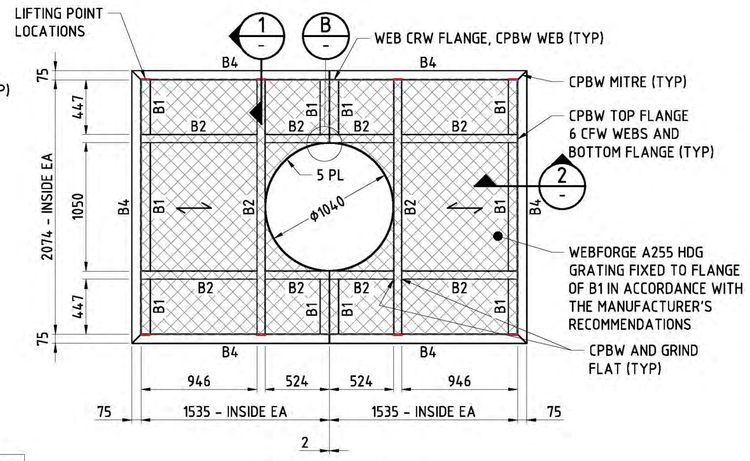
TITLE:
BEETALOO EXPLORATION DRILL CAMPAIGN
INDICATIVE LAYOUT
MUD SUMP DETAILS

PROJECT NO.
MOD NO.
SCALE: 1:1000 @ A1
DRAWING NO.: NT-2050-20-DD-0031
REVISION: 1
CADFILE: NT-2050-20-DD-0031.dwg
A1

Thu, 18 Apr 2019 - 1:51pm P:\60\AX\604805\AIS\CAD\20-SHEETS\VELK1RR1.76.S2-1\NT-2050-20-DD-0031.DWG Dudley, Calvin

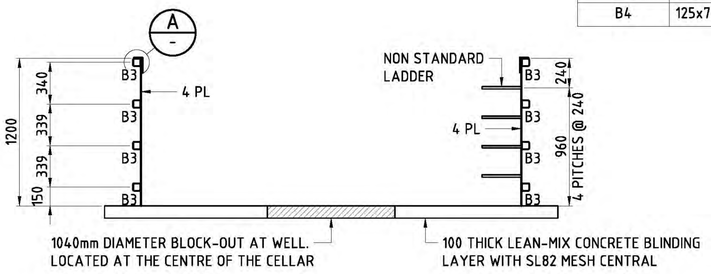


CELLAR PLAN
SCALE 1:25

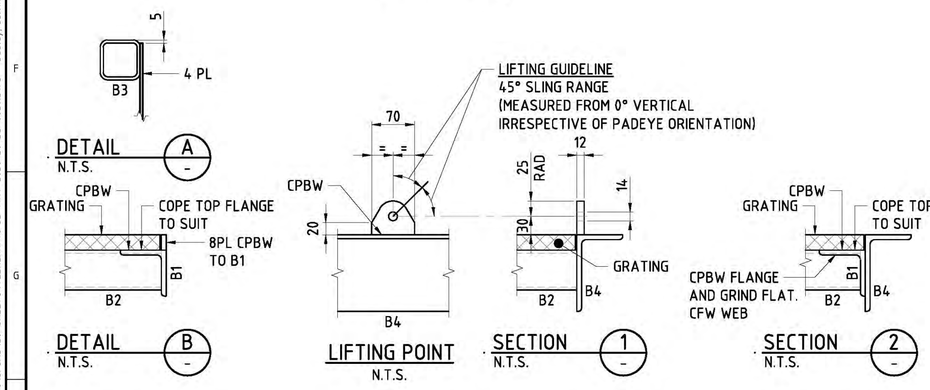


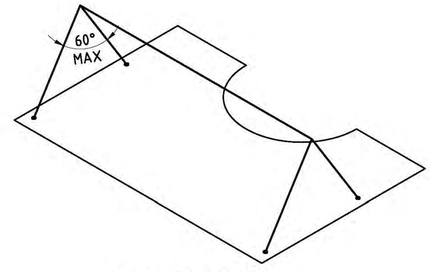
CELLAR COVER PLAN
SCALE 1:25
GRATING SPAN DIRECTION

MEMBER SCHEDULE	
MARK	SIZE
B1	75x8 EA
B2	65x4 SHS
B3	65x4 SHS
B4	125x75x8 UA



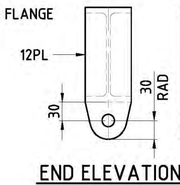
SECTION 1
SCALE 1:25

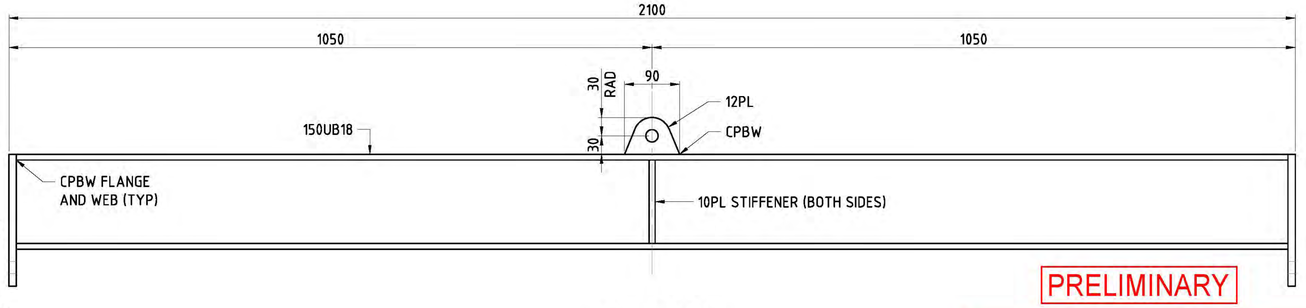




LIFTING DIAGRAM

LIFTING NOTES
- DESIGN ASSUMES A DYNAMIC IMPACT FACTOR Kd=1.2 AND FACTOR OF SAFETY (FOS) OF 3.0
- SLING ANGLE MUST BE 60 DEGREES MAXIMUM AT THE HOOK.
- LIFTING BEAM SHALL HAVE WWL200kg STAMPED INTO FLANGE





LIFTING BEAM
N.T.S.

GENERAL NOTES
- THE FOOTINGS HAVE BEEN DESIGNED FOR A SAFE BEARING PRESSURE OF 50 kPa.
- THE FOOTING DEPTH SHALL BE CHECKED BY THE SUPERINTENDENT AFTER EXCAVATION, AND THE DEPTH MAY BE VARIED FROM THAT DETAILED ON THE DRAWINGS DEPENDING ON THE EXACT LOCATION OF THE LOAD BEARING STRATA.
- FILL UNDER BUILDING FLOOR SLABS: GRANULAR MATERIAL COMPACTED TO A DRY DENSITY RATIO NOT LESS THAN 98% (STANDARD COMPACTION).
- FILL UNDER PAVEMENT SLABS: GRANULAR MATERIAL TO A MIN. THICKNESS OF 50mm UNO ALL FILL AND TOP 150mm OF SUBGRADE COMPACTED TO A DRY DENSITY RATIO NOT LESS THAN 98% (STANDARD COMPACTION).
- DESIGN LIVE LOADS HAVE BEEN CALCULATED IN ACCORDANCE WITH A.S. 1170 PART 1 & A.S. 1657.
- DESIGN LIVE LOAD SURCHARGE ON SOIL 5 kPa
- THESE DRAWINGS SHALL BE READ IN CONJUNCTION WITH THE ARCHITECTS AND OTHER ENGINEERS DRAWINGS, AND ALL DIMENSIONS AND DETAILS SHALL BE CHECKED WITH THEIR DRAWINGS AND ON SITE.
- TRADE NAMES HAVE BEEN USED ONLY TO ESTABLISH A BASIC REQUIREMENT AND ANY SATISFACTORY EQUIVALENT MAY BE SUBMITTED FOR REVIEW.
- ALL DIMENSIONS OF EXISTING STRUCTURES SHALL BE CHECKED PRIOR TO FABRICATION AND ERECTION.
- DURING CONSTRUCTION THE CONTRACTOR SHALL BE RESPONSIBLE FOR MAINTAINING THE STRUCTURE AND EXCAVATIONS IN A STABLE CONDITION, AND ENSURING NO PART SHALL BE OVERSTRESSED UNDER CONSTRUCTION ACTIVITY.

CONCRETE NOTES
- CONCRETE, CONCRETE WORK, AND STEEL REINFORCEMENT SHALL CONFORM TO A.S. 3600, CONCRETE STRUCTURES.
- THE PROPORTION OF FLY ASH SHALL BE 25% BY WEIGHT OF THE TOTAL COMBINED WEIGHT OF FLY ASH AND CEMENT.
- MAXIMUM SLUMP OF CONCRETE = 80±15mm UNO.
- MAXIMUM NOMINAL SIZE OF AGGREGATE = 20mm.
- PLACE 2.N12 REINFORCING BARS X 1000 LONG IN TOP REINF. LAYER DIAGONALLY ACROSS ALL RE-ENTRANT CORNERS AND OPENINGS.
- REINFORCEMENT SHOWN ON DRAWINGS IS REPRESENTED DIAGRAMMATICALLY AND NOT NECESSARILY IN TRUE PROJECTION.
- ALL REINFORCEMENT HOOKS AND COGS SHALL BE STANDARD UNLESS SHOWN OR NOTED OTHERWISE ON THE DRAWINGS.
- ALL REINFORCING STEEL SHALL BE ACRS (AUSTRALIAN CERTIFICATION AUTHORITY FOR REINFORCING AND STRUCTURAL STEEL) CERTIFIED.

STEELWORK NOTES
- ALL STEELWORK SHALL CONFORM TO A.S. 4100, STEEL STRUCTURES.
- STEEL GRADES: 300 UNO. SHS - 350. PLATES - 250.
- FABRICATION SHALL BE CARRIED OUT BY WELDERS WHO ARE QUALIFIED IN ACCORDANCE WITH THE REQUIREMENTS OF A.S./NZS 1554.1.
- THE BOLTING PROCEDURE ADOPTED UNLESS NOTED OTHERWISE IS 8.8/S IN ACCORDANCE WITH THE AUSTRALIAN INSTITUTE OF STEEL CONSTRUCTION - MANUAL OF STANDARDISED STRUCTURAL CONNECTIONS.
- ALL BOLTS OR FASTENERS SHALL BE MARKED WITH THE MANUFACTURERS BRAND, AND COMPLY WITH THE FOLLOWING :
  - BOLTS DENOTED 4.6/S ARE COMMERCIAL BOLTS OF STRENGTH GRADE 4.6 TO A.S. 1111 - SNUG TIGHT.
  - BOLTS DENOTED 8.8/S, 8.8/TF, OR 8.8/TB ARE HIGH STRENGTH STRUCTURAL BOLTS OF STRENGTH GRADE 8.8 TO A.S. 1252.
- BOLTS SHALL BE M20 8.8/S UNO.
- GRADE 8.8 BOLTS SHALL NOT BE WELDED, INCLUDING TACK WELDS FOR CAGING.
- PLATES IN STRUCTURAL STEEL CONNECTIONS SHALL BE 10mm THICK UNO
- MINIMUM CONNECTIONS SHALL BE 10Pl. 2/M20 8.8/S BOLTS UNO.
- ALL BOLTS (INCLUDING MASONRY ANCHORS) SHALL BE HOT DIPPED GALVANISED.
- ALL WELDS SHALL BE 6 CFW UNLESS NOTED OTHERWISE.
- ALL WELDS SHALL BE CATEGORY S.P. AS SPECIFIED IN A.S. 1554, UNO.
- ELECTRODES SHALL BE B-G49X OR B-T49X UNO.
- ALL RHS AND CHS MEMBERS SHALL HAVE 5 PL. SEAL PLATES AT ENDS UNO.
- PROTECTIVE COATING SHALL BE EITHER
  - ALL STEELWORK SSHALL BE BLAST CLEANED TO CLASS 2½ AS SPECIFIED IN AS 1627.4 WITH SURFACE ROUGHNESS OF 75 MICRONS
  - 2 COATS OF IMMERSION GRADE EPOXY TO DRY FILM THICKNESS 400 MICRONS (EACH COAT).

  OR
  - HOT DIP GALVANISED TO AS4680
- SHOP DRAWINGS SHALL BE SUBMITTED BEFORE COMMENCEMENT OF FABRICATION. GRADE OF SANDBLASTING, PAINT BRAND, TYPE, FILM THICKNESS AND WELD CATEGORY SHALL BE NOTED ON THESE DRAWINGS.
- ALL MEMBER CENTROIDS AT CONNECTIONS SHALL INTERSECT AT A POINT UNO GAUGE LINES MAY BE USED IN LIEU OF CENTROIDS FOR BOLTED END CONNECTIONS OF ANGLES.
- ALL STRUCTURAL STEEL SHALL BE ACRS (AUSTRALASIAN CERTIFICATION AUTHORITY FOR REINFORCING AND STRUCTURAL STEEL) CERTIFIED.

PRELIMINARY

NOT FOR CONSTRUCTION

DRAWING NO.	REFERENCE DRAWING TITLE	REV	DATE	REVISION DESCRIPTION
		A	21.03.2019	PRELIMINARY ISSUE

	DRAWN	DWG CHECK	DESIGN	ENG DES CHECK	DE DRAFT SUPER APPROVALS	DE PROJECT APPROVAL
	–	–	–	–	–	–

PROJECT APPROVALS - OTHERS

PREPARED FOR ORIGIN BY:
AECOM
AECOM Australia Pty Ltd
A.B.N 20 093 846 925
www.aecom.com

PROJECT 60480548 BEETALOO EXPLORATION
DRAWING OFFICE DARWIN

Origin Energy Ltd
ABN 30 000 051 696
GPO Box 148
Brisbane Qld. 4001
Ph: (07) 3858 0600
Fax: (07) 3369 7840

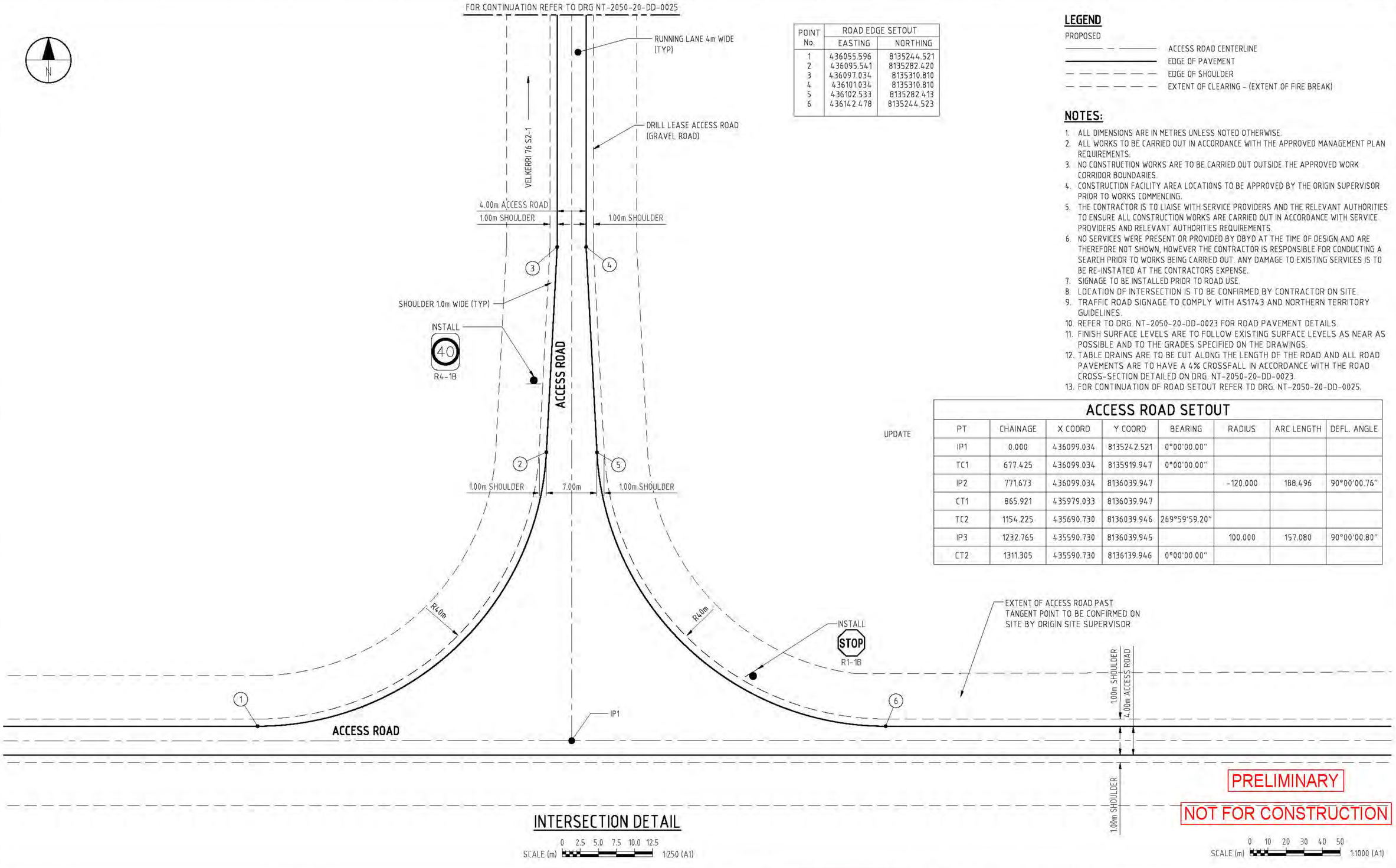
NOTE: THIS DRAWING IS SOLELY THE PROPERTY OF ORIGIN. THE INFORMATION IT CONTAINS IS NOT TO BE DISCLOSED, REPRODUCED OR COPIED IN WHOLE OR PART WITHOUT WRITTEN APPROVAL FROM ORIGIN.

ORIGIN APPROVALS	BY	DATE
DRAWN	–	– – –
DWG CHECK	–	– – –
DESIGN	–	– – –
ENG DES CHECK	–	– – –
DRAFT SUPER	–	– – –
PROJECT APPROVAL	–	– – –

TITLE
BEETALOO EXPLORATION DRILL CAMPAIGN
INDICATIVE LAYOUT
DRILLING CELLAR DETAILS

PROJECT NO.
MOD NO.
SCALE 1:25 @ A1
DRAWING NO. NT-2050-20-DD-0027
REVISION A
CADFILE NT-2050-20-DD-0027.dwg
A1

Thu, 18 Apr 2019 - 1:51pm P:\60\X\60480548\S_CAD\20-SHEETS\VIEL KERR\16 S2-1NAT-2050-20-DD-0027.DWG Dudley, Calvin



POINT No.	ROAD EDGE SETOUT	
	EASTING	NORTHING
1	436055.596	8135244.521
2	436095.541	8135282.420
3	436097.034	8135310.810
4	436101.034	8135310.810
5	436102.533	8135282.413
6	436142.478	8135244.523

LEGEND

PROPOSED

- ACCESS ROAD CENTERLINE
- EDGE OF PAVEMENT
- EDGE OF SHOULDER
- EXTENT OF CLEARING - (EXTENT OF FIRE BREAK)

NOTES:

1. ALL DIMENSIONS ARE IN METRES UNLESS NOTED OTHERWISE.
2. ALL WORKS TO BE CARRIED OUT IN ACCORDANCE WITH THE APPROVED MANAGEMENT PLAN REQUIREMENTS.
3. NO CONSTRUCTION WORKS ARE TO BE CARRIED OUT OUTSIDE THE APPROVED WORK CORRIDOR BOUNDARIES.
4. CONSTRUCTION FACILITY AREA LOCATIONS TO BE APPROVED BY THE ORIGIN SUPERVISOR PRIOR TO WORKS COMMENCING.
5. THE CONTRACTOR IS TO LIAISE WITH SERVICE PROVIDERS AND THE RELEVANT AUTHORITIES TO ENSURE ALL CONSTRUCTION WORKS ARE CARRIED OUT IN ACCORDANCE WITH SERVICE PROVIDERS AND RELEVANT AUTHORITIES REQUIREMENTS.
6. NO SERVICES WERE PRESENT OR PROVIDED BY DBYD AT THE TIME OF DESIGN AND ARE THEREFORE NOT SHOWN, HOWEVER THE CONTRACTOR IS RESPONSIBLE FOR CONDUCTING A SEARCH PRIOR TO WORKS BEING CARRIED OUT. ANY DAMAGE TO EXISTING SERVICES IS TO BE RE-INSTATED AT THE CONTRACTORS EXPENSE.
7. SIGNAGE TO BE INSTALLED PRIOR TO ROAD USE.
8. LOCATION OF INTERSECTION IS TO BE CONFIRMED BY CONTRACTOR ON SITE.
9. TRAFFIC ROAD SIGNAGE TO COMPLY WITH AS1743 AND NORTHERN TERRITORY GUIDELINES.
10. REFER TO DRG. NT-2050-20-DD-0023 FOR ROAD PAVEMENT DETAILS.
11. FINISH SURFACE LEVELS ARE TO FOLLOW EXISTING SURFACE LEVELS AS NEAR AS POSSIBLE AND TO THE GRADES SPECIFIED ON THE DRAWINGS.
12. TABLE DRAINS ARE TO BE CUT ALONG THE LENGTH OF THE ROAD AND ALL ROAD PAVEMENTS ARE TO HAVE A 4% CROSSFALL IN ACCORDANCE WITH THE ROAD CROSS-SECTION DETAILED ON DRG. NT-2050-20-DD-0023.
13. FOR CONTINUATION OF ROAD SETOUT REFER TO DRG. NT-2050-20-DD-0025.

UPDATE

ACCESS ROAD SETOUT

PT	CHAINAGE	X COORD	Y COORD	BEARING	RADIUS	ARC LENGTH	DEFL. ANGLE
IP1	0.000	436099.034	8135242.521	0°00'00.00"			
TC1	677.425	436099.034	8135919.947	0°00'00.00"			
IP2	771.673	436099.034	8136039.947		-120.000	188.496	90°00'00.76"
CT1	865.921	435979.033	8136039.947				
TC2	1154.225	435690.730	8136039.946	269°59'59.20"			
IP3	1232.765	435590.730	8136039.945		100.000	157.080	90°00'00.80"
CT2	1311.305	435590.730	8136139.946	0°00'00.00"			

INTERSECTION DETAIL

SCALE (m) 0 2.5 5.0 7.5 10.0 12.5 1:250 (A1)

SCALE (m) 0 10 20 30 40 50 1:1000 (A1)

PRELIMINARY

NOT FOR CONSTRUCTION

DRAWING NO.	REFERENCE DRAWING TITLE
NT-2050-20-DD-0023	DESIGN DETAILS
NT-2050-20-DD-0025	ACCESS ROAD

REV	DATE	REVISION DESCRIPTION	DRAWN	DWG CHECK	DESIGN	ENG DES CHECK	DE DRAFT SUPER APPROVAL	DE PROJECT APPROVAL
A	21.03.2019	PRELIMINARY ISSUE	CD	-	-	-	-	-

APPROVALS

PREPARED FOR ORIGIN BY:

AECOM

AECOM Australia Pty Ltd
A.B.N 20 093 846 925
www.aecom.com

PROJECT 60480548 BEETALOO EXPLORATION

DRAWING OFFICE DARWIN

PROJECT APPROVALS - OTHERS

origin

Origin Energy Ltd
ABN 30 000 051 696
GPO Box 148
Brisbane Qld. 4001
Ph: (07) 3858 0600
Fax: (07) 3369 7840

NOTE: THIS DRAWING IS SOLELY THE PROPERTY OF ORIGIN. THE INFORMATION IT CONTAINS IS NOT TO BE DISCLOSED, REPRODUCED OR COPIED IN WHOLE OR PART WITHOUT WRITTEN APPROVAL FROM ORIGIN.

	BY	DATE
DRAWN	CD	- . - . -
DWG CHECK	-	- . - . -
DESIGN	-	- . - . -
ENG DES CHECK	-	- . - . -
DRAFT. SUPER.	-	- . - . -
PROJECT APPROVAL	-	- . - . -

TITLE:
BEETALOO EXPLORATION DRILL CAMPAIGN
INDICATIVE LAYOUT
DRILL PAD, ACCOMODATION CAMP AND WATER TANK
PAD INTERSECTION DETAIL

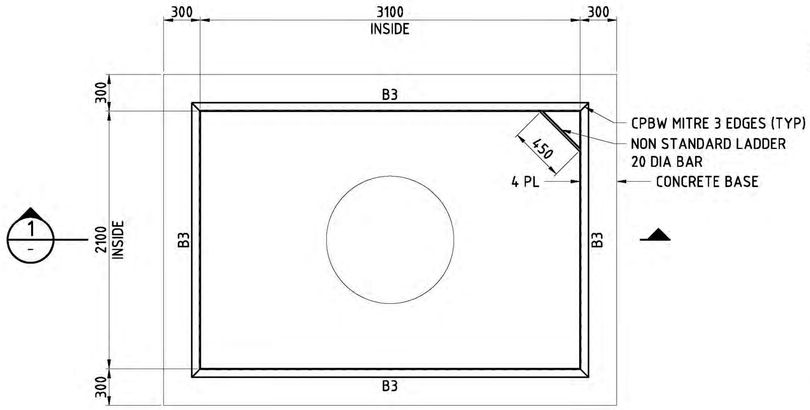
PROJECT NO.

MOD NO.

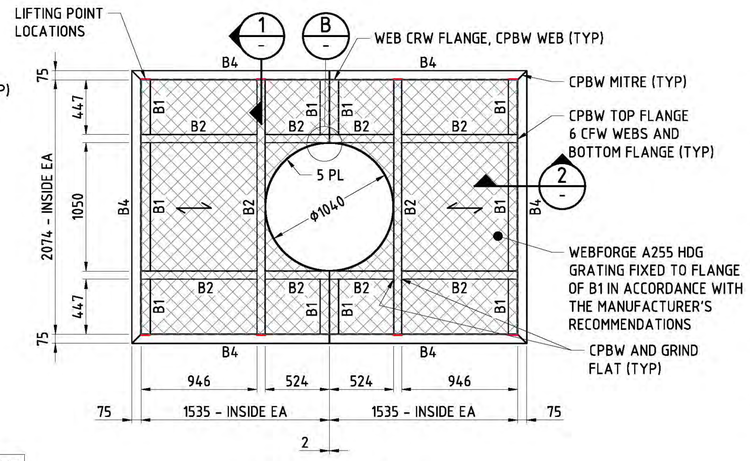
SCALE: 1:1000 @ A1

DRAWING NO. NT-2050-20-DD-0032 REVISION A

CADFILE NT-2050-20-DD-0032.dwg A1

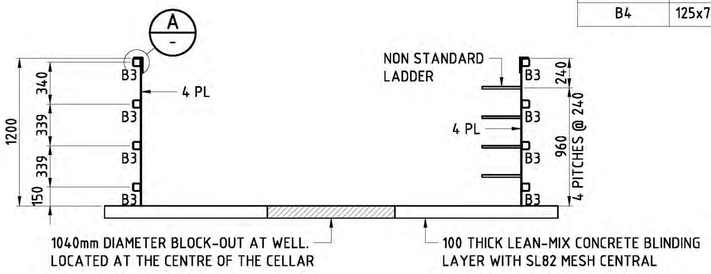


CELLAR PLAN
SCALE 1:25

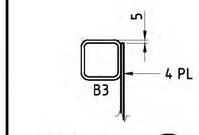


CELLAR COVER PLAN
SCALE 1:25
GRATING SPAN DIRECTION

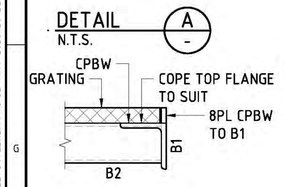
MEMBER SCHEDULE	
MARK	SIZE
B1	75x8 EA
B2	65x4 SHS
B3	65x4 SHS
B4	125x75x8 UA



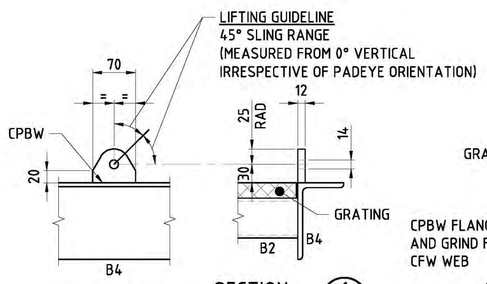
SECTION 1
SCALE 1:25



DETAIL A
N.T.S.

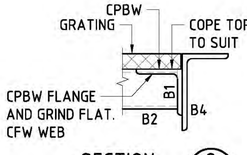


DETAIL B
N.T.S.

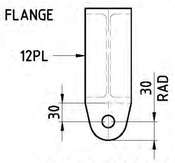


LIFTING POINT
N.T.S.

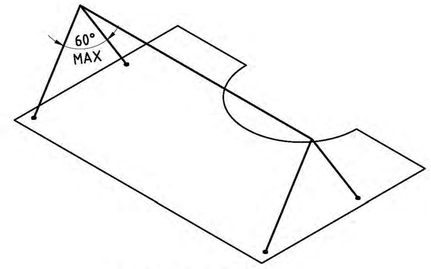
SECTION 1
N.T.S.



SECTION 2
N.T.S.



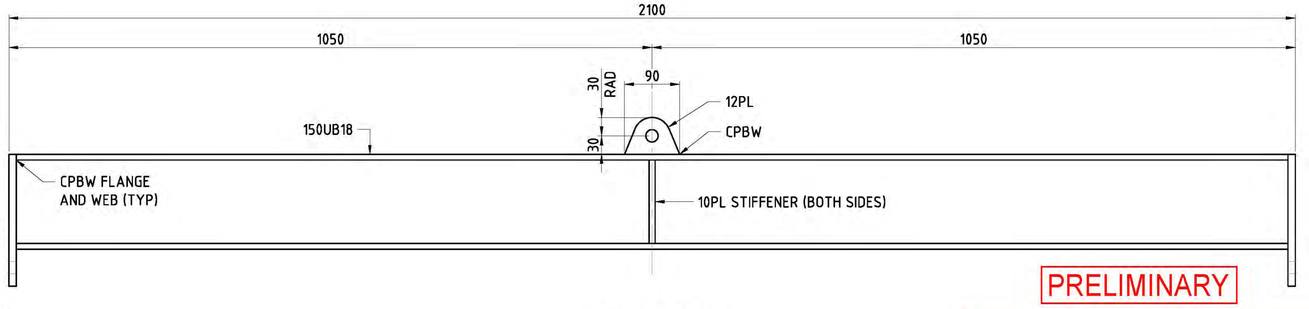
END ELEVATION
LIFTING BEAM
N.T.S.



LIFTING DIAGRAM

LIFTING NOTES

- DESIGN ASSUMES A DYNAMIC IMPACT FACTOR Kd=1.2 AND FACTOR OF SAFETY (FOS) OF 3.0
- SLING ANGLE MUST BE 60 DEGREES MAXIMUM AT THE HOOK.
- LIFTING BEAM SHALL HAVE WWL200kg STAMPED INTO FLANGE



LIFTING BEAM
N.T.S.

GENERAL NOTES

- THE FOOTINGS HAVE BEEN DESIGNED FOR A SAFE BEARING PRESSURE OF 50 kPa.
- THE FOOTING DEPTH SHALL BE CHECKED BY THE SUPERINTENDENT AFTER EXCAVATION, AND THE DEPTH MAY BE VARIED FROM THAT DETAILED ON THE DRAWINGS DEPENDING ON THE EXACT LOCATION OF THE LOAD BEARING STRATA.
- FILL UNDER BUILDING FLOOR SLABS: GRANULAR MATERIAL COMPACTED TO A DRY DENSITY RATIO NOT LESS THAN 98% (STANDARD COMPACTION).
- FILL UNDER PAVEMENT SLABS: GRANULAR MATERIAL TO A MIN. THICKNESS OF 50mm UNO ALL FILL AND TOP 150mm OF SUBGRADE COMPACTED TO A DRY DENSITY RATIO NOT LESS THAN 98% (STANDARD COMPACTION).
- DESIGN LIVE LOADS HAVE BEEN CALCULATED IN ACCORDANCE WITH A.S. 1170 PART 1 & A.S. 1657.
- DESIGN LIVE LOAD SURCHARGE ON SOIL 5 kPa
- THESE DRAWINGS SHALL BE READ IN CONJUNCTION WITH THE ARCHITECTS AND OTHER ENGINEERS DRAWINGS, AND ALL DIMENSIONS AND DETAILS SHALL BE CHECKED WITH THEIR DRAWINGS AND ON SITE.
- TRADE NAMES HAVE BEEN USED ONLY TO ESTABLISH A BASIC REQUIREMENT AND ANY SATISFACTORY EQUIVALENT MAY BE SUBMITTED FOR REVIEW.
- ALL DIMENSIONS OF EXISTING STRUCTURES SHALL BE CHECKED PRIOR TO FABRICATION AND ERECTION.
- DURING CONSTRUCTION THE CONTRACTOR SHALL BE RESPONSIBLE FOR MAINTAINING THE STRUCTURE AND EXCAVATIONS IN A STABLE CONDITION, AND ENSURING NO PART SHALL BE OVERSTRESSED UNDER CONSTRUCTION ACTIVITY.

CONCRETE NOTES

- CONCRETE, CONCRETE WORK, AND STEEL REINFORCEMENT SHALL CONFORM TO A.S. 3600, CONCRETE STRUCTURES.
- THE PROPORTION OF FLY ASH SHALL BE 25% BY WEIGHT OF THE TOTAL COMBINED WEIGHT OF FLY ASH AND CEMENT.
- MAXIMUM SLUMP OF CONCRETE = 80±15mm UNO.
- MAXIMUM NOMINAL SIZE OF AGGREGATE = 20mm.
- PLACE 2.N12 REINFORCING BARS X 1000 LONG IN TOP REINF. LAYER DIAGONALLY ACROSS ALL RE-ENTRANT CORNERS AND OPENINGS.
- REINFORCEMENT SHOWN ON DRAWINGS IS REPRESENTED DIAGRAMMATICALLY AND NOT NECESSARILY IN TRUE PROJECTION.
- ALL REINFORCEMENT HOOKS AND COGS SHALL BE STANDARD UNLESS SHOWN OR NOTED OTHERWISE ON THE DRAWINGS.
- ALL REINFORCING STEEL SHALL BE ACRS (AUSTRALIAN CERTIFICATION AUTHORITY FOR REINFORCING AND STRUCTURAL STEEL) CERTIFIED.

STEELWORK NOTES

- ALL STEELWORK SHALL CONFORM TO A.S. 4100, STEEL STRUCTURES.
- STEEL GRADES: 300 UNO.
  SHS - 350.
  PLATES - 250.
- FABRICATION SHALL BE CARRIED OUT BY WELDERS WHO ARE QUALIFIED IN ACCORDANCE WITH THE REQUIREMENTS OF A.S./NZS 1554.1.
- THE BOLTING PROCEDURE ADOPTED UNLESS NOTED OTHERWISE IS 8.8/S IN ACCORDANCE WITH THE AUSTRALIAN INSTITUTE OF STEEL CONSTRUCTION - MANUAL OF STANDARDISED STRUCTURAL CONNECTIONS.
- ALL BOLTS OR FASTENERS SHALL BE MARKED WITH THE MANUFACTURERS BRAND, AND COMPLY WITH THE FOLLOWING :
  - BOLTS DENOTED 4.6/S ARE COMMERCIAL BOLTS OF STRENGTH GRADE 4.6 TO A.S. 1111 - SNUG TIGHT.
  - BOLTS DENOTED 8.8/S, 8.8/TF, OR 8.8/TB ARE HIGH STRENGTH STRUCTURAL BOLTS OF STRENGTH GRADE 8.8 TO A.S. 1252.
- BOLTS SHALL BE M20 8.8/S UNO.
- GRADE 8.8 BOLTS SHALL NOT BE WELDED, INCLUDING TACK WELDS FOR CAGING.
- PLATES IN STRUCTURAL STEEL CONNECTIONS SHALL BE 10mm THICK UNO
- MINIMUM CONNECTIONS SHALL BE 10Pl. 2/M20 8.8/S BOLTS UNO.
- ALL BOLTS (INCLUDING MASONRY ANCHORS) SHALL BE HOT DIPPED GALVANISED.
- ALL WELDS SHALL BE 6 CFW UNLESS NOTED OTHERWISE.
- ALL WELDS SHALL BE CATEGORY S.P. AS SPECIFIED IN A.S. 1554, UNO.
- ELECTRODES SHALL BE B-G49X OR B-T49X UNO.
- ALL RHS AND CHS MEMBERS SHALL HAVE 5 PL. SEAL PLATES AT ENDS UNO.
- PROTECTIVE COATING SHALL BE EITHER
  - ALL STEELWORK SSHALL BE BLAST CLEANED TO CLASS 2½ AS SPECIFIED IN AS 1627.4 WITH SURFACE ROUGHNESS OF 75 MICRONS
  - 2 COATS OF IMMERSION GRADE EPOXY TO DRY FILM THICKNESS 400 MICRONS (EACH COAT).

  OR
  - HOT DIP GALVANISED TO AS4680
- SHOP DRAWINGS SHALL BE SUBMITTED BEFORE COMMENCEMENT OF FABRICATION. GRADE OF SANDBLASTING, PAINT BRAND, TYPE, FILM THICKNESS AND WELD CATEGORY SHALL BE NOTED ON THESE DRAWINGS.
- ALL MEMBER CENTROIDS AT CONNECTIONS SHALL INTERSECT AT A POINT UNO GAUGE LINES MAY BE USED IN LIEU OF CENTROIDS FOR BOLTED END CONNECTIONS OF ANGLES.
- ALL STRUCTURAL STEEL SHALL BE ACRS (AUSTRALASIAN CERTIFICATION AUTHORITY FOR REINFORCING AND STRUCTURAL STEEL) CERTIFIED.

PRELIMINARY

NOT FOR CONSTRUCTION

DRAWING NO.	REFERENCE DRAWING TITLE	REV	DATE	REVISION DESCRIPTION
		A	21.03.2019	PRELIMINARY ISSUE

	DRAWN	DWG CHECK	DESIGN	ENG DES CHECK	DE DRAFT SUPER APPROVALS	DE PROJECT APPROVAL
	–	–	–	–	–	–

PREPARED FOR ORIGIN BY:
AECOM
AECOM Australia Pty Ltd
A.B.N 20 093 846 925
www.aecom.com

PROJECT 60480548 BEETALOO EXPLORATION
DRAWING OFFICE DARWIN
PROJECT APPROVALS - OTHERS

Origin Energy Ltd
ABN 30 000 051 696
GPO Box 148
Brisbane Qld. 4001
Ph: (07) 3858 0600
Fax: (07) 3369 7840

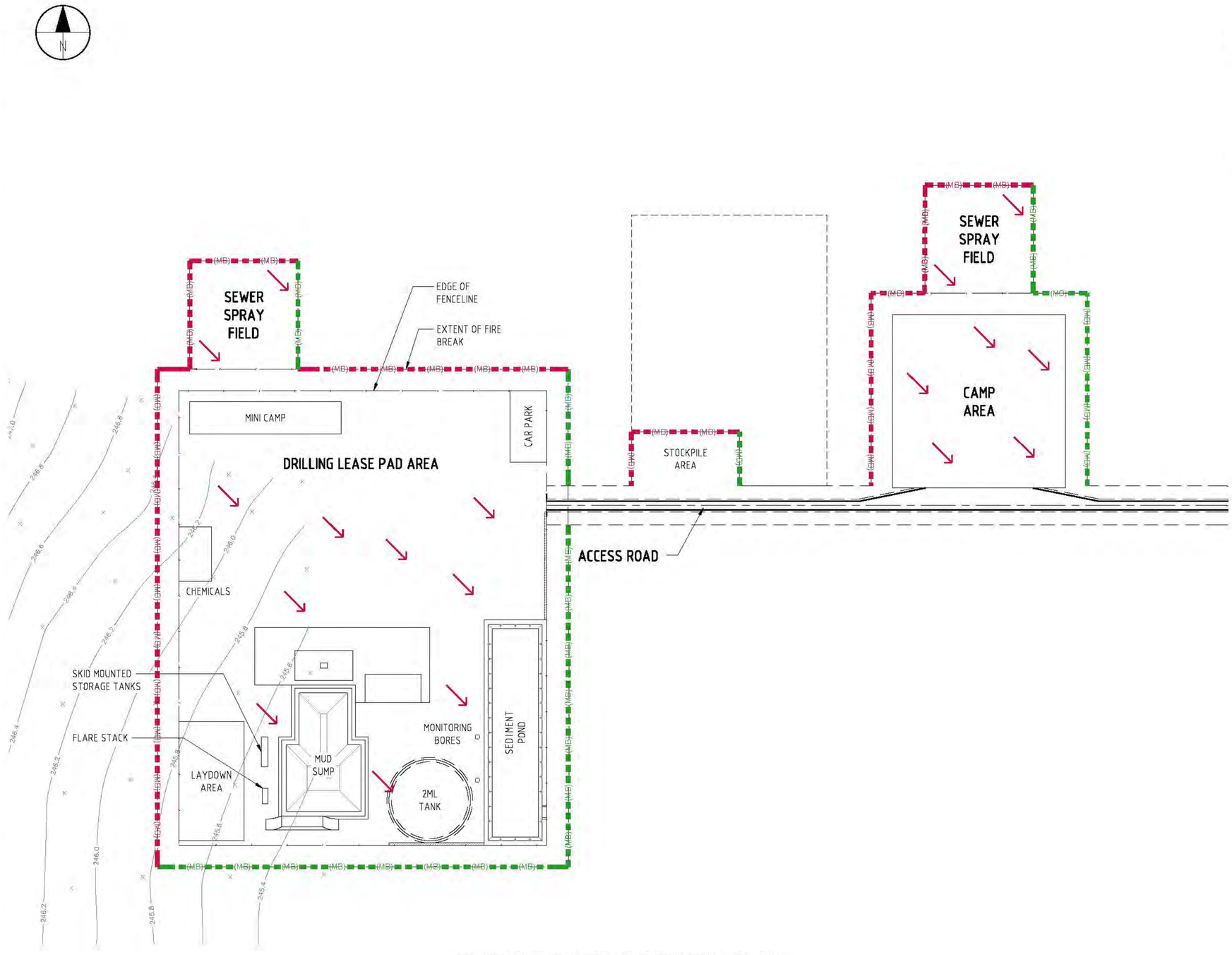
NOTE: THIS DRAWING IS SOLELY THE PROPERTY OF ORIGIN. THE INFORMATION IT CONTAINS IS NOT TO BE DISCLOSED, REPRODUCED OR COPIED IN WHOLE OR PART WITHOUT WRITTEN APPROVAL FROM ORIGIN.

ORIGIN APPROVALS	BY	DATE
DRAWN	–	– – –
DWG CHECK	–	– – –
DESIGN	–	– – –
ENG DES CHECK	–	– – –
DRAFT SUPER	–	– – –
PROJECT APPROVAL	–	– – –

TITLE
BEETALOO EXPLORATION DRILL CAMPAIGN
INDICATIVE LAYOUT
DRILLING CELLAR DETAILS

PROJECT NO.
MOD NO.
SCALE 1:25 @ A1
DRAWING NO. NT-2050-20-DD-0027
REVISION A
CADFILE NT-2050-20-DD-0027.dwg
A1

P:\60\X\60480548\S_CAD\20-SHEETS\VIEL KERR\16 S2-1NT-2050-20-DD-0027.DWG Dudley, Calvin
Thu, 18 Apr 2019 - 1:51pm



LEGEND

EXISTING

CONTOURS

PROPOSED

(MB) TOPSOIL BERM (DIRTY WATER)

(MB) TOPSOIL BERM (CLEAN WATER)

FLOW DIRETION

U-SHAPED SEDIMENT TRAP

NOTES:

1. ALL WORKS TO BE IN ACCORDANCE WITH THE INTERNATIONAL EROSION CONTROL ASSOCIATION (IECA) GUIDELINES.
2. TOPSOIL STRIPPING DEPTH ASSESSMENT MUST BE UNDERTAKEN IN ACCORDANCE WITH THE BEETALOO TOPSOIL STRIPPING TECHNICAL INSTRUCTION (NT-2050-15-TI-0001) AND AMELIORATION RATES AGREED WITH THE ORIGIN SUPERVISOR. THE EXPECTED NOMINAL TOPSOIL DEPTH FOR THE SITE IS 150mm, FINAL STRIP DEPTH TO BE CONFIRMED IN THE FIELD.
3. MAINTENANCE OF ESC DEVICES:
   - THE CONTRACTOR SHALL INSPECT ALL ENVIRONMENTAL DEVICES ON A REGULAR BASIS. ANY RECTIFICATION OF DAMAGE TO THE ENVIRONMENTAL CONTROL DEVICES OR CLEANING OUT OF THE DEVICES IS TO BE CARRIED OUT BY THE CONTRACTOR AS REQUIRED.
   - REGULAR MAINTENANCE SHALL BE UNDERTAKEN UNTIL SUFFICIENT GROUND COVER IS ESTABLISHED TO PROVIDE STABILISATION TO ALL DISTURBED AREAS.
4. TOPSOIL BERM DIMENSIONS IN ACCORDANCE WITH IECA FIGURE 1 ON STANDARD DRAWING MB-01.
5. ALL ESC DEVICES WILL BE DESIGN AND INSTALLED IN ACCORDANCE WITH THE NT LAND CLEARANCE GUIDELINES.
6. THE CONTRACTOR MUST SUBMIT AN EROSION AND SEDIMENT CONTROL PLAN WHICH WILL BE APPROVED BY ORIGIN PRIOR TO MOBILISATION.

EROSION AND SEDIMENT CONTROL PLAN

PRELIMINARY

NOT FOR CONSTRUCTION

SCALE (m) 0 10 20 30 40 50 1:1000 (A1)

DRAWING NO.	REFERENCE DRAWING TITLE	REV	DATE	REVISION DESCRIPTION	DRAWN	DWG CHECK	DESIGN	ENG DES CHECK	DE DRAFT SUPER	DE PROJECT APPROVAL
-	-									
-	-									
-	-	A	21.03.2019	PRELIMINARY ISSUE	CD	–	–	–	–	–

APPROVALS

PREPARED FOR ORIGIN BY:

AECOM

AECOM Australia Pty Ltd
A.B.N 20 093 846 925
www.aecom.com

PROJECT 60480548 BEETALOO EXPLORATION

DRAWING OFFICE DARWIN

PROJECT APPROVALS - OTHERS

origin

Origin Energy Ltd
ABN 30 000 051 696
GPO Box 148
Brisbane Qld. 4001
Ph: (07) 3858 0600
Fax: (07) 3369 7840

	BY	DATE
DRAWN	CD	— . — . —
DWG CHECK	–	— . — . —
DESIGN	–	— . — . —
ENG DES CHECK	–	— . — . —
DRAFT. SUPER	–	— . — . —
PROJECT APPROVAL	–	— . — . —

TITLE
BEETALOO EXPLORATION DRILL CAMPAIGN
INDICATIVE LAYOUT
DRILL PAD, ACCOMODATION CAMP AND WATER TANK
EROSION AND SEDIMENT CONTROL PLAN

PROJECT NO.

MOD NO.

SCALE 1:1000 @ A1

DRAWING NO. NT-2050-15-MP-0022

REVISION A

CADFILE NT-2050-15-MP-0022.dwg

A1

Liner and wastewater tank storage specifications for existing and new wastewater tanks

13 Oct 2016	Enviro Liner® 1000 Properties				
Style	**ASTM**	**EL 1020**	**EL 1030**	**EL 1040N**	**EL 1040**
Thickness	D5199	20 mil 0.5 mm	30 mil 0.75 mm	36 mil 0.91 mm	40 mil 1.0 mm
Density (Typical)	D792	0.93	0.93	0.93	0.93
Tensile Strength at Break	D6693	76 ppi 13 N/mm	114 ppi 20 N/mm	136 ppi 24 N/mm	152 ppi 27 N/mm
Elongation	D6693	800%	800%	700%	800%
Tear Resistance	D1004	11 lbs 49 N	16 lbs 70 N	19 lbs 84 N	22 lbs 100 N
Puncture Resistance	D4833	28 lbs 120 N	42 lbs 190 N	54 lbs 240 N	56 lbs 250 N
Carbon Black Content	D6370	≥ 2.0%	≥ 2.0%	2.0%	≥ 2.0%
High Pressure OIT	D5885	400 min	400 min	N/A	400 min
Low Temperature Impact Resistance	D746	-69°F -56°C	-69°F -56°C	-40°F -40°C	-69°F -56°C
Service Temperatures	Max Continuous Use	140°F 60°C	140°F 60°C	140°F 60°C	140°F 60°C

18 Oct 2016	Enviro Liner® 1000 Shop Seam Strengths				
Style	**ASTM D6392**	**Enviro Liner® 1020**	**Enviro Liner® 1030**	**Enviro Liner® 1040N**	**Enviro Liner® 1040**
Heat Bonded Seam Strength	25.4 mm (1") Strip	30 ppi 5.2 N/mm	45 ppi 7.7 N/mm	50 ppi 8.7 N/mm	60 ppi 10.3 N/mm
Peel Adhesion Strength (Wedge Weld)	25.4 mm (1") Strip	25 ppi 4.3 N/mm	38 ppi 6.5 N/mm	45 ppi 7.9 N/mm	50 ppi 8.7 N/mm

FlexiPond Wind Loading

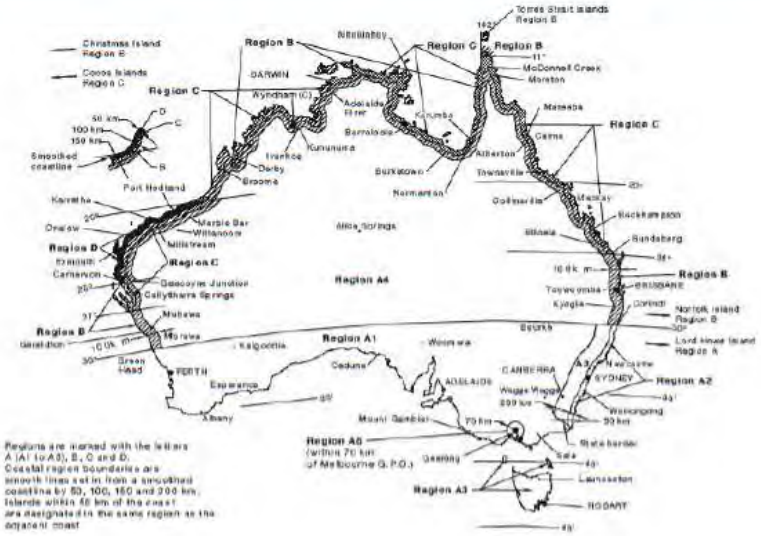


FIGURE 3.1(A) WIND REGIONS

(g) *2 effective surfaces* Lateral pressure on windward and leeward walls		0.9	1.0 (not an effective surface)
(h) *2 effective surfaces* Lateral pressure on external and internal surfaces	HINGE	0.9	0.9

Wind Rating as per AS1170.2:2011(R2016)

- Individual panel (or a straight line section of assembled panels)
- Overturning/Sliding rating (e.g. in storage or prior to installation) **rated to a maximum of 100km/hr**

- Fully Assembled (with corner pieces, locking pins and cables)
- **Stable to all wind conditions in all wind regions (includes cyclonic)**

Support Slide: Certified Working Parameters

- Wind Rating as per AS1170.2:2011(R2016), Structural Design Actions Part2 : Wind Actions
  - Individual panel (or a straight line section of assembled panels)
    - Overturning/Sliding rating (e.g. in storage or prior to installation) rated to a maximum of 100km/hr
  - Fully Assembled (with corner pieces, locking pins and cables)
    - Stable to all wind conditions in all wind regions (includes cyclonic)
    - However, recommend minimum 100mm of fluid in tank at shallow end to stop liner from blowing away/tearing

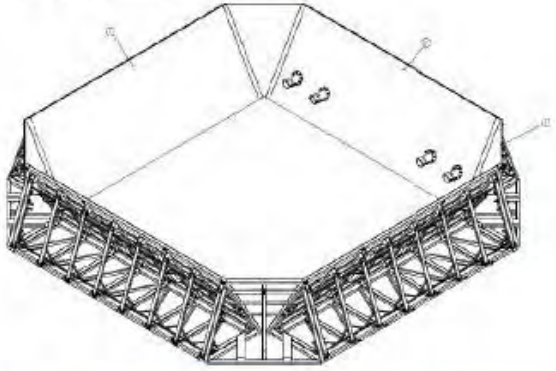
- Hydrostatic Loading as per AS NZS 1170.1-2011(R2016) and AS3990-1993(R2016)
  - Assembled with cables and pins
    - Fluid maximum specific gravity of 1.2 in conjunction with wind loading
    - Structurally able to handle erosion under 70% of the frame (although not recommended as the liner would be removed under the panel by the hydrostatic fluid
    - Cables to be capable of being safely loaded to 160kN

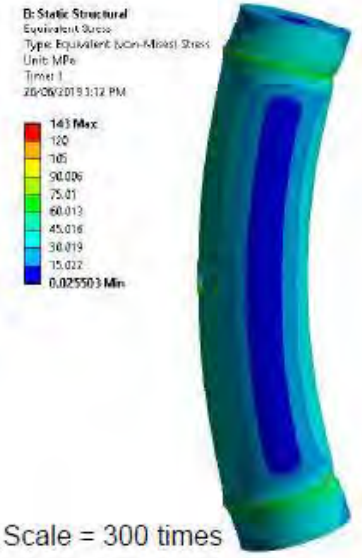
- Installation conditions
  - Site Slope
    - See chart for numbers of panels versus slope
  - Ground Bearing Pressures
    - Uniformly supported panels exert a pressure of 35 kPa beneath the frame members when filled with 1.2 SG fluid
    - The liner exerts a pressure on the ground of 25 kPa when filled with 1.2 SG fluid
    - As a reference – A typical passenger vehicle tyres exert approx. 200kPa on ground
  - Ground/Site Preparation
    - Assessment would be on a site-specific basis
    - Recommended final site preparation of a bedding material of loose sand or fine aggregate to ensure uniform loading under frame members
    - A depth of not less than 100mm would ensure final levelling could be performed during assembly of the panels
    - Alternatively, a ground level tolerance of +/- 16mm should be used to ensure uniform loading under frame members
    - To reduce the likelihood of erosion, it is recommended that a spoon drain or similar be provided around the structure, particularly on the high side or where there is the potential for significant runoff to impact the structure. This would also be site specific.

FlexiPond Structural Integrity

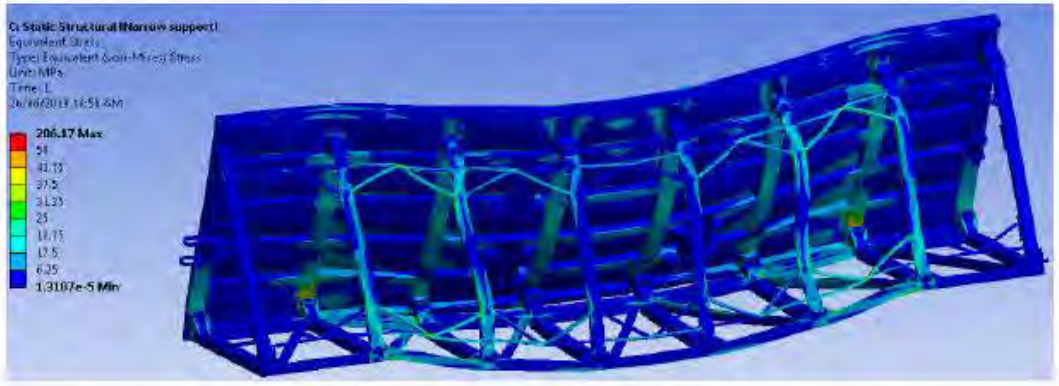
FlexiPond is designed and Engineered to AS3990 Mechanical Equipment – Steelwork

Finite Element Analysis (FEA) software used to simulate various load conditions and confirm engineering calculations





Deformed Scale = 300 times



Deformed Scale = 340 times

Structural FEA of Frame Connecting Pin

- Supported in the middle of the pin
- Loaded on either end of the pin

Structural FEA simulating erosion under 70% of Structure

- Supported on either side by the ground and horizontal cables
- Fluid hydrostatic pressure acting on the panel surface

Appendix D Chemical Risk Assessment

Prepared for
Origin Energy B2 Pty Ltd
ABN: 42 105 431 525

AECOM

Beetaloo Exploration and Appraisal Program - Hydraulic Fracturing Chemical Risk Assessment

07-Jul-2022

aecom.com

Delivering a better world

Beetaloo Exploration and Appraisal Program - Hydraulic Fracturing Chemical Risk Assessment

Client: Origin Energy B2 Pty Ltd

ABN: 42 105 431 525

Prepared by

AECOM Australia Pty Ltd
Level 3, 9 Cavenagh Street, Darwin NT 0800, GPO Box 3175, Darwin NT 0801, Australia
T +61 8 8942 6200 F +61 8 8942 6299 www.aecom.com
ABN 20 093 846 925

07-Jul-2022

Job No.: 60480548

AECOM in Australia and New Zealand is certified to ISO9001, ISO14001 and ISO45001.

Quality Information

Document	Beetaloo Exploration and Appraisal Program - Hydraulic Fracturing Chemical Risk Assessment
Ref	60480548
Date	07-Jul-2022
Prepared by	Cindy Cheung, Tiffany Teo
Reviewed by	Mark Chapman

Revision History

Rev	Revision Date	Details	Authorised	
			Name/Position	Signature
A	6 August 2019	Draft	Hayden Seear Project Manager	
0	16-Dec-2019	Final	Hayden Seear Project Manager	
1	18-Mar-2020	Addition of Perfomatrol chemical to drilling fluid	Hayden Seear Project Manager	
2	23-Feb-2022	Addition of chemicals to drilling fluid	Alana Court Project Manager	
3	07-Jul-2022	Minor update to CRA	Alana Court Project Manager	

Table of Contents

1.0 Background

Chemical risk assessments for the hydraulic fracturing fluid systems were undertaken to assess the potential human health effects of the chemicals proposed to be used in the Beetaloo Exploration and Appraisal Program.

The following fluid systems were assessed:

- Hydraulic fracture stimulation fluids;
- Hydraulic fracture chemical tracers; and
- Drilling fluids.

The risk assessment aligns with the *Northern Territory Government, Department of Environment, Parks and Water Security, Environment Management Plan Content Guideline, 2021* (herein referred to as DEPWS 2021).

The methods used for this updated chemical risk assessment also follow the guidance provided by the *Department of the Environment and Energy, Exposure Draft - Chemical Risk Assessment Guidance Manual: for chemicals associated with coal seam gas extraction, 2017* (DoEE, 2017) and the methodology adopted for the chemical risk assessment is in general accordance with the following:

- National Industrial Chemicals Notification and Assessment Scheme (NICNAS), National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia, 2017 (herein referred to as NICNAS 2017)
- enHealth. Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards, 2012
- National Environment Protection (Assessment of Site Contamination) Measure 1999 (ASC NEPM); Schedule B4, Site-specific health risk assessment methodology, 2013

The chemical risk assessment comprised the following tasks:

- Hazard assessment. An evaluation of the environmental hazard of the chemical additives in the hydraulic fracturing fluid systems, based on their environmental persistence, bioaccumulation and aquatic toxicity properties. Also included was an evaluation of human health effects (i.e. genotoxicity, carcinogenicity, reproductive toxicity, oral toxicity, inhalation toxicity, dermal toxicity, chronic repeated dose toxicity).
- Exposure assessment. The exposure assessment comprised of an evaluation of surface and sub-surface exposure pathways assessment and mass balance calculation to identify the amount of each chemical additive of the hydraulic fracturing fluid system.
- Screening and validation processes via Tier 1 and Tier 2 assessments. Determination of chemicals known to be of low concern, and identification of chemicals for further risk assessment.
    - Tier 1: using published information about each chemical proposed to be used in the hydraulic fracturing fluid systems.
    - Tier 2: A quantitative evaluation of the risks using toxicity values and quantitative estimates of chemical intake to provide an estimate of potential human health risk associated with the hydraulic fracturing activities, based on the identification of complete exposure pathways and hazard identification.

2.0 Hydraulic Fracture Chemical Risk Assessment Tier 1 Screen

2.1.1 Outcome of Tier 1 Screen – Stimulation Fluid Recipes

Three stimulation fluid recipes (SW, Hybrid and HVFR) will be used for the Beetaloo Exploration and Appraisal Program.

Comparison of the chemicals with the assessment criteria as presented in DoEE (2017) indicated that 10 chemicals were not considered to require a Tier 2 assessment. The chemicals have been assessed under the National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia using the adapted IMAP screening process (NICNAS 2017) and were identified to be of low concern because of low hazard. **Table 1** presents a summary of the chemicals identified to be of low concern to human health for the hydraulic fracture stimulation fluid recipes.

Table 1 Chemicals identified to be of low human health concern (Tier 1) – Stimulation Fluid Recipes

CAS	Chemical	Reasoning
9003-04-7	Sodium polyacrylate	NICNAS (2017) low concern chemical
25987-30-8	Acrylamide acrylate copolymer	NICNAS (2017) low concern chemical
25987-30-8	Acrylamide, sodium acrylate polymer	NICNAS (2017) low concern chemical
107-21-1	Ethylene glycol	NICNAS (2017) low concern chemical
67-48-1	Choline chloride	NICNAS (2017) low concern chemical
77-92-9	Citric acid	NICNAS (2017) low concern chemical
7681-82-5	Sodium iodide	NICNAS (2017) low concern chemical
9000-30-0	Guar gum	NICNAS (2017) low concern chemical
7757-82-6	Sodium sulfate	NICNAS (2017) low concern chemical
126-96-5	Sodium diacetate	NICNAS (2017) low concern chemical

Based on the Tier 1 screening, most chemicals (24 from SW, 30 from Hybrid and 25 from HVFR) were identified to require a Tier 2 assessment. It is to be noted that none of these chemicals were identified to be persistent and bioaccumulative.

The Tier 1 screening is provided in **Appendix A** to **Appendix C**, and the chemical toxicological profiles are provided in **Appendix D** to **Appendix H**.

2.1.2 Outcome of Tier 1 Screen – Drilling Fluids

Comparison of the chemicals with the assessment criteria indicated that 30 chemicals were not considered to require a Tier 2 assessment. 22 chemicals have been assessed by NICNAS (2017) and were identified to be of low concern. In following the IMAP screening process, a further 8 chemicals (which were not assessed by NICNAS 2017) were identified to be of low concern to human health.

Table 2 presents a summary of the chemicals identified to be of low concern to human health for the drilling fluid recipe.

Table 2 Chemicals identified to be of low human health concern (Tier 1) – Drilling Fluids

CAS	Chemical	Reasoning
Not Applicable	Proprietary Chemical	NICNAS (2017) low concern chemical
77-92-9	Citric acid	NICNAS (2017) low concern chemical
9004-32-4	Poly Anionic Cellulose	NICNAS (2017) low concern chemical
7447-40-7	Potassium Chloride	NICNAS (2017) low concern chemical
144-55-8	Sodium Bicarbonate	NICNAS (2017) low concern chemical

CAS	Chemical	Reasoning
7647-14-5	Sodium Chloride	NICNAS (2017) low concern chemical
6381-77-7	Sodium erythorbate	NICNAS (2017) low concern chemical
11138-66-2	Xanthan Gum	NICNAS (2017) low concern chemical
1317-65-3	Calcium Carbonate	NICNAS (2017) low concern chemical
1310-73-2	Sodium hydroxide	Acute toxicity only. No evidence of systemic toxicity. Due to the unavailability of a NOAEL, quantification of risks from repeated exposure is not possible. However, due to dissociation into ions which are subject to homeostatic controls in the human body, systemic effects from repeated exposures to sodium hydroxide are not expected (NICNAS 2017).
1310-58-3	Potassium Hydroxide	Acute toxicity only. No evidence of systemic toxicity. Similar results were reported for sodium hydroxide (NICNAS 2017).
9005-25-8	Starch	NICNAS polymer of low concern (PLC)
12199-37-0	Smectite	No chronic data available. Read across to bentonite which is listed as a NICNAS (2017) low concern chemical.
38193-60-1	Polyacrylamide	NICNAS PLC
1332-58-7	Plagioclase Feldspar/Kaolinite	Listed in US Food and Drug Administration (FDA) Generally Recognized as Safe (GRAS) list and Inert Ingredients Eligible for US Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) 25(b) pesticide products.
Proprietary	Performatrol*	A low weight and stable polymer that is highly biodegradable with low environmental toxicity.
13462-86-7	Barite	NICNAS (2017) low concern chemical
9003-05-8	Partially hydrolysed polyacrylamide	NICNAS (2017) low concern chemical
9004-32-4	Polyanionic cellulose, low viscosity	NICNAS (2017) low concern chemical
7727-43-7	Barium sulphate	NICNAS (2017) low concern chemical
7439-92-1	Lead	Maximum concentration below Australian Drinking Water Guidelines (NHMRC, 2018) and Australian and New Zealand Guidelines for Fresh and Marine Water Quality (ANZG, 2018).
7782-42-5	Graphite	NICNAS (2017) low concern chemical
14807-96-6	Talc	NICNAS (2017) low concern chemical
8042-47-5	Mineral oil	NICNAS (2017) low concern chemical
7440-50-8	Copper	NICNAS (2017) low concern chemical
7440-66-6	Zinc	NICNAS (2017) low concern chemical
1305-78-8	Calcium oxide	NICNAS (2017) low concern chemical
7429-90-5	Aluminium not powder, dust or fume	NICNAS (2017) low concern chemical
1317-38-0	Copper (II) Oxide	NICNAS (2017) low concern chemical
64-02-8	Tetrasodium ethylenediaminetetraacetate	NICNAS (2017) low concern chemical

*CAS number not provided to AECOM, information obtained via chemical manufacturer's SDS

Based on the Tier 1 screening 26 drilling fluid chemicals were identified to require a Tier 2 assessment. It is to be noted that none of these chemicals were identified to be persistent and bioaccumulative.

Two of the chemicals are proprietary. For one of the chemicals, the CAS number and name have been redacted from the public submission to protect the intellectual property of chemical manufacturer. Although the proprietary details of the chemical have been redacted in this report, AECOM had access to the chemical name and CAS number and the assessment of risk from the redacted chemical is presented in this report. For the other proprietary chemical (Performatrol), the CAS number was not provided by the chemical manufacturer, however the information in its SDS was utilised to inform this assessment.

The Tier 1 screening is provided in **Appendix D**, and the chemical toxicological profiles are provided in **Appendix G**.

2.1.3 Outcome of Tier 1 Screen – Chemical Tracers

The following chemical tracers may be used for the Beetaloo Exploration and Appraisal Program – CFT, GFT and WFT. The proprietary chemical CAS numbers and names have been redacted from the public submission to protect the intellectual property of chemical manufacturers. Although the proprietary details of the chemicals have been redacted in this report, AECOM had access to the chemical names and CAS numbers (with the exception of Performatrol) and the assessment of risk from the redacted chemicals is presented in this report.
Comparison of the chemicals with the assessment criteria indicated that all chemicals were considered to require a Tier 2 assessment. However, none of these chemicals were identified to be persistent and bioaccumulative.

The Tier 1 screening is provided in **Appendix E**, and the chemical toxicological profiles are provided in **Appendix H**.

3.0 Hydraulic Fracture Chemical Risk Assessment Tier 2 Screen

3.1.1 Tier 2 Screen Methodology

The Tier 2 assessment evaluated the toxicity of the individual chemicals and characterised the cumulative risks of the total fluid mixtures to Workers. The methodology incorporated an assessment of potential exposures to the Workers, with the following identified as the only potentially complete exposure pathways:

- Incidental ingestion and dermal contact of flowback fluid by Workers during the hydraulic stimulation period for a maximum duration of 1 month; and
- Inhalation of mist from the evaporation units at the flowback tank by Workers for a maximum duration of 1 year.

These scenarios are also deemed protective of the following due to the less frequent and short duration of these exposures occurring:

- Worker exposure during a spill (i.e. a coupling breaks on a tank and releases product onto the worker) or leak scenarios

Based on the risk mitigation measures identified in the NT Government *Scientific Inquiry into Hydraulic Fracturing in the Northern Territory*, the *Code of Practice for Onshore Petroleum Activities* in the Northern Territory and mitigation measures outlined by Origin in its EMPs, no potentially complete exposure pathways were identified for hydraulic fracturing chemicals to impact groundwater that is used for beneficial use in the project area. The specific controls implemented by Origin focussed on the protection of aquifers follow industry standard practice and include:

- the physical vertical separation distances of 1,400 m between the aquifer and target formation to prevent any migration of stimulation fluid to aquifer units;
- the horizontal separation distance between the exploration well and the closest groundwater extraction bores of at least 1 km, as per the Code;
- use of double lined wastewater tanks with leak detection;

- implementation of spill management plan;
- use of enclosed tanks and freeboard requirements; and
- mandatory secondary containment requirements.

Potential exposures to hydraulic fracturing chemicals at the project area were therefore assessed to be limited to the above ground storage and handling of flowback water. Management of flowback water involves temporary storage in above ground fluid holding tanks for evaporation.

3.1.2 Chemicals of Potential Concern

Exposure point concentrations (EPC) were developed for each of the hydraulic fracturing fluid systems using theoretical calculations, where it was conservatively assumed that 100% of the mass of the chemicals injected into the well will be present in the flowback water.

A summary of the chemicals that require further assessment are presented in **Table 3** to **Table 7**.

Table 3 Chemicals requiring further assessment (Tier 2) – Stimulation Fluid SW Recipe (24 chemicals)

CAS	Chemical Name
7647-01-0	Hydrochloric acid
68937-66-6	Alcohols, C6-12, ethoxylated propoxylated
69227-22-1	Alcohols, C10-16, ethoxylated propoxylated
7647-14-5	Sodium Chloride
64-19-7	Acetic acid
81741-28-8	Tributyl tetradecyl phosphonium chloride
25322-68-3	Polyethylene glycol
7631-90-5	Sodium bisulfite
104-55-2	Cinnamaldehyde
111-46-6	Diethylene glycol
67-56-1	Methanol
61788-90-7	Amine oxides, cocoalkyldimethyl
1310-73-2	Sodium hydroxide
100-52-7	Benzaldehyde
64-17-5	Ethanol
64742-47-8	Hydrotreated light petroleum distillate
61791-00-2	Fatty acids, tall-oil, ethoxylated
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)
71-36-3	Butyl alcohol
68131-39-5	Alcohols, C12-15, ethoxylated
68551-12-2	Alcohols, C12-16, ethoxylated
107-13-1	Acrylonitrile
111-42-2	Diethanolamine
111-30-8	Glutaraldehyde

Table 4 Chemicals requiring further assessment (Tier 2) – Stimulation Fluid Hybrid Recipe (30 chemicals)

CAS	Chemical Name
7647-01-0	Hydrochloric acid
68937-66-6	Alcohols, C6-12, ethoxylated propoxylated
1319-33-1	Ulexite
102-71-6	Triethanol amine
7647-14-5	Sodium Chloride
1310-73-2	Sodium hydroxide
69227-22-1	Alcohols, C10-16, ethoxylated propoxylated
64-19-7	Acetic acid
111-42-2	Diethanolamine
81741-28-8	Tributyl tetradecyl phosphonium chloride
7631-90-5	Sodium bisulfite
7758-19-2	Chlorous acid, sodium salt
12008-41-2	Disodium octaborate tetrahydrate
104-55-2	Cinnamaldehyde
25322-68-3	Polyethylene glycol
111-46-6	Diethylene glycol
14808-60-7	Crystalline silica, quartz
67-56-1	Methanol
7775-27-1	Sodium persulfate
61788-90-7	Amine oxides, cocoalkyldimethyl
100-52-7	Benzaldehyde
64-17-5	Ethanol
64742-47-8	Hydrotreated light petroleum distillate
61791-00-2	Fatty acids, tall-oil, ethoxylated
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)
71-36-3	Butyl alcohol
68131-39-5	Alcohols, C12-15, ethoxylated
68551-12-2	Alcohols, C12-16, ethoxylated
107-13-1	Acrylonitrile
111-30-8	Glutaraldehyde

Table 5 Chemicals requiring further assessment (Tier 2) – Stimulation Fluid HVFR Recipe (25 chemicals)

CAS	Chemical Name
64-19-7	Acetic acid
69227-22-1	Alcohols, C10-16, ethoxylated propoxylated
68131-39-5	Alcohols, C12-15, ethoxylated
68551-12-2	Alcohols, C12-16, ethoxylated
68937-66-6	Alcohols, C6-12, ethoxylated propoxylated
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)
61788-90-7	Amine oxides, cocoalkyldimethyl
100-52-7	Benzaldehyde
71-36-3	Butyl alcohol
104-55-2	Cinnamaldehyde
111-42-2	Diethanolamine
111-46-6	Diethylene glycol
64-17-5	Ethanol
68439-54-3	Ethoxylated branched C13 alcohol
61791-00-2	Fatty acids, tall-oil, ethoxylated
7647-01-0	Hydrochloric acid
64742-47-8	Hydrotreated light petroleum distillate
67-56-1	Methanol
25322-68-3	Polyethylene glycol
1338-43-8	Sobitan, mono-9-octadecenoate, (Z)
7631-90-5	Sodium bisulfite
1310-73-2	Sodium hydroxide
9005-65-6	Sorbitan monooleate polyoxyethylene derivative
81741-28-8	Tributyl tetradecyl phosphonium chloride
10486-00-7	Sodium perborate tetrahydrate

Table 6 Chemicals requiring further assessment (Tier 2) – Drilling Fluids (26 chemicals)

CAS	Chemical Name
78330-21-9	Alcohol, C11-14, ethoxylated
64742-47-8	Distillates, hydrotreated light
68909-77-3	Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivatives residues
111-30-8	Glutaraldehyde
107-22-2	Glyoxal <1%
67-56-1	Methanol
5064-31-3	Nitrilotriacetic acid, trisodium salt monohydrate
14808-60-7	Quartz/Cristobite

CAS	Chemical Name
497-19-8	Sodium Carbonate
533-74-4	Tetrahydro-3,5-dimethyl-1,3,5-thiadiazine-2-thione
50-01-1	Guanidine, hydrochloride (1:1)
4719-04-4	Triazine based biocide C572,2',2"-(hexahydro-1,3, 5-triazine-1,3,5-triyl) triethano
10192-30-0	Ammonium hydrogensulfite
68909-77-3	Filming amine Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivs. Residues
848301-67-7	Distillates (Fischer-Tropsch), C8-26 - Branched and Linear
68990-47-6	Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine
34590-94-8	(2-methoxymethylethoxy)propanol
1120-36-1	1-tetradecene
68155-20-4	Amides, tall oil fatty N,N-bis (hydroxyethyl)
68910-93-0	Fatty acids, tall-oil, reaction products with polyethylenepolyamines
68585-36-4	Phosphoric ester of ethoxylated fatty alcohol
629-73-2	Hexadec-1-ene
64742-52-5	Distillates (petroleum), hydrotreated heavy naphthenic
64742-53-6	Distillates (petroleum), hydrotreated light naphthenic < 3% DMSO
64741-44-2	Distillates (petroleum), straight-run middle
8052-42-4	Bitumen
68457-79-4	Phosphorodithioic acid, mixed o,o-bis(iso-bu and pentyl) esters, zinc salts
4719-04-4	Triazine based biocide C572,2',2"-(hexahydro-1,3, 5-triazine-1,3,5-triyl) triethano
10192-30-0	Ammonium hydrogensulfite
68909-77-3	Filming amine Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivs. Residues
848301-67-7	Distillates (Fischer-Tropsch), C8-26 - Branched and Linear
68990-47-6	Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine
34590-94-8	(2-methoxymethylethoxy)propanol
1120-36-1	1-tetradecene
68155-20-4	Amides, tall oil fatty N,N-bis (hydroxyethyl)
68910-93-0	Fatty acids, tall-oil, reaction products with polyethylenepolyamines
68585-36-4	Phosphoric ester of ethoxylated fatty alcohol
629-73-2	Hexadec-1-ene
64742-52-5	Distillates (petroleum), hydrotreated heavy naphthenic
64742-53-6	Distillates (petroleum), hydrotreated light naphthenic < 3% DMSO
64741-44-2	Distillates (petroleum), straight-run middle
8052-42-4	Bitumen
68457-79-4	Phosphorodithioic acid, mixed o,o-bis(iso-bu and pentyl) esters, zinc salts

Table 7 Chemicals requiring further assessment (Tier 2) – Chemical Tracers (4 chemicals)

CAS	Chemical Name
Proprietary	CFT (one chemical selected to represent a group of 20 similar chemicals)
Proprietary	GFT (one chemical selected to represent a group of 15 similar chemicals)
Proprietary	WFT
Proprietary	WFT

3.1.3 Outcome of Tier 2 Screen

For the assessment of the overall potential for adverse human health effects posed by simultaneous exposure to multiple chemicals, the estimated daily intake of the chemicals by inhalation and direct (ingestion and dermal) contact were compared to acceptable risk-based intakes to calculate an individual hazard quotient (HQ) and then summed for all constituents into a hazard index (HI).

Consistent with Australian risk assessment methodologies, if the HI is less than or equal to 1, then no adverse health effects are likely associated with exposures. However, if the total HI is greater than 1, adverse health effects may be possible and therefore the assumptions inherent in the risk characterisation process warrant further evaluation

3.1.3.1 Stimulation Fluids

A summary of the calculated risks for the workers that are relevant to the assessment of potential exposure to COPCs in stimulation fluids on-site, based on the available data is presented in **Table 8**.

Table 8 Risk associated with potential exposure to Workers – Stimulation Fluids

Receptor and Pathway	Threshold Hazard Index
	100% Mass Return
Worker - Exposure to Stimulation Fluid SW Recipe	
Ingestion of chemicals via incidental contact with flowback water	0.01
Dermal exposure to chemicals via incidental contact with flowback water	0.20
Inhalation of mist from the evaporation units containing flowback water	0.05
Total Risk	**0.3**
Worker - Exposure to Stimulation Fluid Hybrid Recipe	
Ingestion of chemicals via incidental contact with flowback water	0.03
Dermal exposure to chemicals via incidental contact with flowback water	0.08
Inhalation of mist from the evaporation units containing flowback water	0.74
Total Risk	**0.9**
Worker - Exposure to Stimulation Fluid HVFR Recipe	
Ingestion of chemicals via incidental contact with flowback water	0.01
Dermal exposure to chemicals via incidental contact with flowback water	0.22
Inhalation of mist from the evaporation units containing flowback water	0.05
Total Risk	**0.3**

The following can be noted from the table above:

- The calculated risks associated with potential exposure to COPC identified in flowback water, where either SW, Hybrid or HVFR stimulation fluid recipes are used and assuming 100% mass recovery, are below the target 1, hence, risks are considered to be low and acceptable.

3.1.3.2 Drilling Fluid

A summary of the calculated risks for the workers that are relevant to the assessment of potential exposure to COPCs in the drilling fluid on-site, based on the available data is presented in **Table 9**.

Table 9 Risk associated with potential exposure to Workers – Drilling Fluid

Receptor and Pathway	Threshold Hazard Index
	100% Mass Return
Worker	
Ingestion of chemicals via incidental contact with flowback water	0.004
Dermal exposure to chemicals via incidental contact with flowback water	0.007
Inhalation of mist from the evaporation units containing flowback water	0.2
Total Risk	**0.3**

The following can be noted from the table above:

- The calculated risks associated with potential exposure to COPC identified in flowback water, where drilling fluid recipe is used and assuming 100% mass recovery are below the target 1, hence, risks are considered to be low and acceptable.

3.1.3.3 Chemical Tracers

A summary of the calculated risks for the workers that are relevant to the assessment of potential exposure to COPCs in the Chemical Tracers on-site, based on the available data is presented in **Table 10**.

Table 10 Risk associated with potential exposure to Workers – Chemical Tracers

Receptor and Pathway	Threshold Hazard Index
	100% Mass Return
Worker – Exposure to Chemical Tracer CFT Recipe	
Ingestion of chemicals via incidental contact with flowback water	0.0000032
Dermal exposure to chemicals via incidental contact with flowback water	0.000010
Inhalation of mist from the evaporation units containing flowback water	0.000018
Total Risk	**0.00003**
Worker – Exposure to Chemical Tracer GFT Recipe	
Ingestion of chemicals via incidental contact with flowback water	0.0000047
Dermal exposure to chemicals via incidental contact with flowback water	0.0010
Inhalation of mist from the evaporation units containing flowback water	0.000026
Total Risk	**0.001**
Worker – Exposure to Chemical Tracer WFT Recipe	
Ingestion of chemicals via incidental contact with flowback water	0.30
Dermal exposure to chemicals via incidental contact with flowback water	0.012

Receptor and Pathway	Threshold Hazard Index
	100% Mass Return
Inhalation of mist from the evaporation units containing flowback water	-
Total Risk	**0.3**

The following can be noted from the table above:

- The calculated risks associated with potential exposure to COPC identified in flowback water, where either CFT, GFT or WFT chemical tracer recipes are used and assuming 100% mass recovery, are below the target 1, hence, risks are considered to be low and acceptable.

3.1.3.4 Combination of Hydraulic Fracturing Fluid Systems

A summary of the calculated risks for the workers that are relevant to the assessment of potential exposure to COPCs from combinations of hydraulic fracturing fluid systems on-site, based on the available data is presented in **Table 11**.

Table 11 Risk associated with potential exposure to Workers – Combination of Hydraulic Fracturing Fluid Systems

Receptor	Threshold Hazard Index
	100% Mass Return
Worker	
Exposure to SW + Drilling Fluid + Chemical Tracer CFT Recipes	0.5
Exposure to Hybrid + Drilling Fluid + Chemical Tracer CFT Recipes	1
Exposure to HVFR+ Drilling Fluid + Chemical Tracer CFT Recipes	0.5
Exposure to SW + Drilling Fluid + Chemical Tracer GFT Recipes	0.5
Exposure to Hybrid + Drilling Fluid + Chemical Tracer GFT Recipes	1
Exposure to HVFR+ Drilling Fluid + Chemical Tracer GFT Recipes	0.5
Exposure to SW + Drilling Fluid + Chemical Tracer WFT Recipes	0.8
Exposure to Hybrid + Drilling Fluid + Chemical Tracer WFT Recipes	1
Exposure to HVFR+ Drilling Fluid + Chemical Tracer WFT Recipes	0.8

The following can be noted from the table above:

- On the basis of the risk evaluation, no unacceptable risk to Workers was identified in all of the possible recipe combinations of stimulation fluids, drilling fluids and chemical tracers. It is noted that conservative risk scenarios assessed included regular exposure to the flowback water during the hydraulic stimulation and evaporation phases, with exposures to high theoretical concentrations of COPC in the flowback water. This may result in overestimation of the risk.

It is to be noted that this assessment does not replace the requirement for appropriate occupational health and safety procedures and management plans. Crystalline silica is scheduled by Safe Work Australia as a chemical for which health monitoring may be required.

The Tier 2 assessment is provided in **Appendix A to Appendix E**, the chemical toxicological profiles are provided in **Appendix F** to **Appendix H.**

4.0 Chemical Transport, Storage and Handling

Origin aligns its transport, storage, and handling of hazardous chemicals with WHS Regulations, and the prescribed chemical legislation including all obligations and duties for storage and handling of hazardous chemicals and eliminating risks to workers from potential exposure and the potential requirements for health monitoring.

The following prescribed chemical legislation, as defined by the Petroleum (Environment) Regulations 2016, will be followed as it relates to the transport, storage, and handling of HFS chemicals:

- *Medicines, Poisons and Therapeutic Goods Act 2012 and Medicines, Poisons and Therapeutic Goods Regulations 2014*
- *Dangerous Goods Act 1998*
- *Water Act 1992*
- *Waste Management and Pollution Control Act 1998*
- *Work Health and Safety (National Uniform Legislation) Act 2011*
- *Radiation Protection Act 2004.*

5.0 References

AECOM (2019). Beetaloo 2019 Campaign – Chemical Risk Assessments, April 2019.

ANZG (2018). Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Australian and New Zealand Governments and Australian state and territory governments, Canberra ACT, Australia. Available at www.waterquality.gov.au/anz-guidelines

DoEE (2017). Department of the Environment and Energy, Exposure Draft - Chemical Risk Assessment Guidance Manual: for chemicals associated with coal seam gas extraction, 2017

enHealth (2012). Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards, 2012

ASC NEPM (2013). National Environment Protection (Assessment of Site Contamination) Measure 1999; Schedule B4, Site-specific health risk assessment methodology, 2013

NICNAS (2017). National Industrial Chemicals Notification and Assessment Scheme, National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia, 2017

NHMRC (2018). Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management

DEPWS (2021). Northern Territory Government, Department of Environment, Parks and Water Security, Environment Management Plan Content Guideline, 2021

Origin (2019). Beetaloo Basin Drilling Stimulation and Well Testing Program, Environmental Management Plan, 2019

Scientific Inquiry into Hydraulic Fracturing in the Northern Territory, Draft Final Report, December 2017.

Appendix A

Chemical Risk Assessment Hydraulic Fracture Stimulation Fluid – Hybrid

Chemical Name	CAS Number	Density (kg/L)	Volume of Chemical (L)	Volume Fraction (%v/v)	Chemical Mass in Fluid (kg)	Mass Fraction (% w/w)	Concentration in Injected Fluid (mg/L)	Parent Compound Purpose	Ecotoxicity[1]	Toxicity[2]	Biodegradation[1,3]	Bioaccummulative[1]	Tier 1 Screening Assessment	Tier 2 Assessment Worker Ingestion Risk	Tier 2 Assessment Worker Dermal Risk	Tier 2 Assessment Worker Aerosol Inhalation Risk	Hazard Quotient	Outcome of Tier 2 Worker Risk Assessment[1]
Proprietary	Proprietary	1.1	24,720	0.0950%	27,192	0.0973%	1,096	Clay Stabiliser	96-hour fish LC50 value is >100 mg/L 48-hour in vertebrate EC50 is 349 mg/L 72-hour EC50 to Pseudokirchneriella subcapitata is >1,000 mg/L 21-day Daphnia NOEC value is 30.2 mg/L	Based on Chronic: Low	Choline chloride is readily biodegradable and thus it does not meet the screening criteria for persistence.	Not Bioaccumulative (based on a measured log Kow of -3.77 and a calculated BCF of 0.59)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Guar gum	9000-30-0	0.7	23,649	0.0909%	16,555	0.0592%	667	Gelling agent	lowest measured ecotoxicity endpoint for fish was reported to be 218 mg/L.	Based on Acute: Low	Guar gum is a naturally occurring polysaccharide which would be expected to readily biodegrade. Thus, it is not expected to meet the screening criteria for persistence	Not Bioaccumulative based on the molecular weight of guar gum (ranges from 200,000 to 300,000 daltons), and it is also water soluble.	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Hydrochloric acid	7647-01-0	1.152	10,206	0.0392%	11,757	0.0421%	474	Acid	Algae = 0.492 mg/L Daphnia = 0.492 mg/L Fish = 4.92 mg/L Daphnia (chronic) = 62 mg/L	Based on Chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	NA. Acute toxity only	NA. Acute toxity only	NA. Acute toxity only	NA. Acute toxity only	NA. Acute toxity only
Alcohols, C6-12, ethoxylated propoxylated	68937-66-6	0.94	5,253	0.0202%	4,938	0.0177%	199	Surfactant	LC50 (96h) 0.59 mg/L (Pleuronectes platessa) EC50 (48h) 0.14 mg/L (Daphnia magna) EC50 (96h) 0.7 mg/L (Pseudokirchneriella subapitata) NOEC 4.4 mg/L (Pimephales promelas, juvenile)	Based on Chronic: Moderate	Expected to be readily biodegradable based on similar substances	Not Bio accumulative (Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8)	Tier 2	1.40E-03	7.78E-05	7.79E-03	9.26E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Ethylene glycol	107-21-1	1.11	3,723	0.0143%	4,132	0.0148%	166	Crosslinker	LC50 for fish = 22800 mg/L LC50 for Daphnia =7800 mg/L NOEC for Algae =100 mg/L	Based on Acute: Low	Readily biodegradable	No based on the measured log Kow of -1.36 and a measured BCF of 10	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Ulexite	1319-33-1	1.49	3,476	0.0134%	5,175	0.0185%	209	Crosslinker	Chronic endpoints for Boric acid were available for Daphnia (6 mg/L) and Fish (2.1 mg/L).	Based on Chronic: Moderate	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	7.63E-03	3.21E-03	4.25E-02	5.33E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Triethanol amine	102-71-6	1.1245	3,309	0.0127%	3,721	0.0133%	150	Crosslinker	Fish: 96h-LC50 of 11,800 mg/ Daphnia: 24h-EC50 of 1,390 mg/l Daphnia: 21 d NOEC of 16 mg/l Algae:96 h EC50 of 910 mg/l	Based on Chronic: Low	Inherently biodegradable	Not Bio accumulative (Based on an estimated log Kow value of -1.0, and BCF value of <3.9)	Tier 2	4.21E-04	9.55E-06	2.35E-03	2.78E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sodium Chloride	7647-14-5	2.165	2,859	0.0110%	6,189	0.0221%	249	Stabiliser	EC50 = 400 to 30000 mg/L EC50 Fish = 1290 mg/L NOEC = 314 mg/L (Daphnia)	Based on Chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	Low chronic toxicity, insufficient data to establish toxicity value	Low chronic toxicity, insufficient data to establish toxicity value	Low chronic toxicity, insufficient data to establish toxicity value	Low chronic toxicity, insufficient data to establish toxicity value	Low chronic toxicity, insufficient data to establish toxicity value
Sodium polyacrylate	9003-04-7	1.32	2,370	0.0091%	3,128	0.0112%	126		96 hr LC50 for fish is >1000 mg/L NOEC from a chronic early life stage test for the fathead minnow is 56 mg/L 48 hr LC50 for Dapnia magna is >1000 mg/L NOEC for a 21day chronic reproductive test on Daphnia magna is 5.6 mg/L EC10 for Scenedesmus is 180 mg/L	Based on Chronic: Moderate to low	Sodium polyacrylate has limited biodegradation potential and thus meets the screening criteria for persistence.	Bioaccumulation of sodium polyacrylate is unlikely due to the high molecular weight of the polymer.	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Sodium hydroxide	1310-73-2	1.515	2,059	0.0079%	3,119	0.0112%	126	pH buffer	Measured acute endpoints were available for fish (196 mg/L). Measured chronic endpoint were available for Daphnia (240 mg/L)	Based on Chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	NA. Acute toxicity only (irritant and corrosive), not systemically available in body	NA. Acute toxicity only (irritant and corrosive), not systemically available in body	NA. Acute toxicity only (irritant and corrosive), not systemically available in body	NA. Acute toxicity only (irritant and corrosive), not systemically available in body	NA. Acute toxicity only (irritant and corrosive), not systemically available in body
Alcohols, C10-16, ethoxylated propoxylated	69227-22-1	0.94	1,876	0.0072%	1,763	0.0063%	71	Friction Reducer, Surfactant	LC50 (96h) 0.59 mg/L (Pleuronectes platessa EC50 (48h) 0.14 mg/L (Daphnia magna) ErC50 (48h) 0.7 mg/L (Skeletonema costatum) ErC50 (16.9h) > 10 g/L (Pseudomonas putida)	Based on Acute: Very high	Expected to be readily biodegradable based on similar substances	Not Bio accumulative (Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8)	Tier 2	4.99E-04	6.59E-02	2.78E-03	6.92E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Acetic acid	64-19-7	1.05	1,558	0.0060%	1,636	0.0059%	66	Acid	Acute endpoints: Fish = 75 mg/L, Daphnia EC50 = 32 mg/L Chronic endpoints: Daphnia = 150 mg/L	Based on Chronic: Low	Readily biodegradable	Not Bio accumulative (Based on log Kow = -0.136)	Tier 2	1.93E-05	4.93E-06	1.07E-04	1.32E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Diethanolamine	111-42-2	1.1	1,459	0.0056%	1,605	0.0057%	65	Breaker Activator	Fish 96-h LC50 = 1370 mg/l Invertebrates 48-h EC50 = 55 mg/l Pseudokirchneriella subcapitata 96-h ErC50 = 2.2 mg/l Microorganisms 16-h TTC = 16 mg/l Daphnia magna, the NOEC (21 days) was 0.78 mg/l	Based on Chronic: High	Readily biodegradable	Not Bioaccumulative. Based on a measured log Kow of -2.18 and a calculated BCF of 3.16	Tier 2	1.62E-02	3.37E-04	9.04E-02	1.07E-01	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Tributyl tetradecyl phosphonium chloride	81741-28-8	0.95	736	0.0028%	700	0.0025%	28	Biocide	LC50: (96 hour) 0.46 mg/L (Oncorhynchus mykiss) LC50: (96 hour) 0.06 mg/l (Lepomis macrochirus) LC50: (96 hour) 0.58 mg/l (fish) TLM96: 1.6 mg/l (Crangon crangon) TLM48: 0.025 mg/l (Daphnia magna Modelled acute endpoint: Daphnia is 16.788 mg/L Fish is 1059.2530 mg/L	Based on Acute: Very high	Not available, however it has been observed to biodegrade in sediment.	Not bioaccumulative (Based on an estimated log Kow value of 6.26)	Tier 2	NA. Acute toxity only	NA. Acute toxity only	NA. Acute toxity only	NA. Acute toxity only	NA. Acute toxity only
Acrylamide acrylate copolymer	9003-06-9	0.75	730	0.0028%	548	0.0020%	22	Scale Inhibitor	96 hour LC50 for fish = 1 400 mg/L 48 hour EC50 for Daphnia magna = 1 200 mg/L 21 day EC50 for Daphnia magna = 680 mg/L 21 day NOEC for algae = 380 mg/L	Based on Chronic: Low	Polymers are not readily biodegradable, hence they meet the screening criteria for persistence.	Polymers are expected to have very high molecular weights and poor water solubility. They are not expected to be bioavailable.	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Sodium bisulfite	7631-90-5	2.44	483	0.0019%	1,179	0.0042%	47	Scale Inhibitor	72h-EC50 = 36.8 mg sodium sulfite/L (alga) NOEC of >8.41 mg sodium sulfite/L (Daphnia)	Based on Chronic: Moderate	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	1.59E-05	3.04E-11	8.85E-05	1.04E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Chlorous acid, sodium salt	7758-19-2	2.47	458	0.0018%	1,131	0.0040%	46	Breaker	LC50 values above 100 mg/l (fish) LC50 48-hour = 0.063 mg/l (daphnia) ECr50 value at 72 h as 1.2 mg/l (algae)	Based on Acute: Very High	No. Not expected to be persistent due to its instability.	No. Based on an estimated log Kow value of 3	Tier 2	4.10E-03	1.56E-08	2.29E-02	2.70E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Disodium octaborate tetrahydrate	12008-41-2	1.874	336	0.0013%	630	0.0023%	25	Crosslinker	Algae: EC10 (3 d) 96.5 mg/L (Pseudokirchneriella subcapitata) Fish: LC50 (96 h) 314.6 mg/L (Pimephales promelas), NOEC (34 d) 25.2 mg/L (Danio rerio) Invertebrates: NOEC (21 d) 42.5 mg/L (Daphnia magna) Microorganism: EC50 (3 h) > 39371 mg/L (activated sludge)	Based on Chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	9.29E-04	3.90E-04	5.17E-03	6.49E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Cinnamaldehyde	104-55-2	1.048	332	0.0013%	348	0.0012%	14	Corrosion Inhibitor	Danio rerio (Zebrafish) 96 h LC50 = 3.1 mg/L; Daphnia magna (Water flea) 48 h EC50 = 3.86 mg/L; Pseudokirchneriella subcapitata (Green algae) 72 h EC50 = 4.07 mg/L. 72 h NOEC value = 2.0 mg/L Pseudokirchneriella subcapitata (Green algae)	Based on Chronic: Moderate	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	2.46E-05	5.89E-05	1.37E-04	2.21E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Polyethylene glycol	25322-68-3	1.21	328	0.0013%	397	0.0014%	16	Scale Inhibitor	LC50 = 100 mg/L (fish) LC50 = 1000 mg/L (invertebrates) EC 50 = 15.91 mg/L (algae)	Based on Acute: Moderate	Expected to be readily biodegradable	No based on BCF of 3.2	Tier 2	7.03E-06	6.92E-09	3.92E-05	4.62E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Diethylene glycol	111-46-6	1.12	303	0.0012%	339	0.0012%	14	Corrosion Inhibitor	LC 50 = >100 mg/L (fish, invertebrates, algae)	Based on Acute: Low	Readily biodegradable	No based on the estimated BCF of 3	Tier 2	1.60E-04	3.07E-06	8.91E-04	1.05E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Crystalline silica, quartz	14808-60-7	2.6	235	0.0009%	611	0.0022%	25	Crosslinker	no acute toxicity to fish, Daphnia, or algae, though some physical effects were observed with loading rates of greater than or equal to 10 g/L (OECD 2004). Any harmful effects to aquatic ecosystems are therefore not ecotoxicological in nature. No chronic toxicity data were identified.	Based on Acute: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	NA. Not toxic via oral exposure as not absorbed via GI tract	NA. Not toxic via dermal exposure.	5.62E-01	5.62E-01	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).

Chemical Name	CAS Number	Density (kg/L)	Volume of Chemical (L)	Volume Fraction (%v/v)	Chemical Mass in Fluid (kg)	Mass Fraction (% w/w)	Concentration in Injected Fluid (mg/L)	Parent Compound Purpose	Ecotoxicity[1]	Toxicity[2]	Biodegradation[1,3]	Bioaccummulative[1]	Tier 1 Screening Assessment	Tier 2 Assessment Worker Ingestion Risk	Tier 2 Assessment Worker Dermal Risk	Tier 2 Assessment Worker Aerosol Inhalation Risk	Hazard Quotient	Outcome of Tier 2 Worker Risk Assessment[1]
Methanol	67-56-1	0.791	125	0.0005%	99	0.0004%	4	Corrosion Inhibitor, Surfactant	LC50s ranged from 15,400 to 29,400 mg/L (fish) 24-hour and 48-hour EC50s were > 10,000 mg/L (Daphnia) 28 days NOEC was 446.7 mg/L (fish) 21 days NOEC was 208 mg/L (invertebrates)	Based on Chronic: Low	Readily biodegradable	No based on the Log Kow of -0.74	Tier 2	3.76E-04	5.52E-05	2.10E-03	2.53E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sodium persulfate	7775-27-1	1.68	116	0.0004%	194	0.0007%	8	Breaker	LC50 fish = 163 to 771 mg/L. EC50 invertebrates = 133 and 519 mg/L EC50 algae = 116 mg/L	Based on Acute: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	4.10E-05	1.33E-08	2.29E-04	2.70E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Amine oxides, cocoalkyldimethyl	61788-90-7	0.716	103	0.0004%	74	0.0003%	3	Corrosion Inhibitor	LC50/EC50/ErC50 values: 0.60-32 mg/L for fish 0.50-10.8 mg/L for Daphnia magna 0.010-5.30 mg/L for algae NOEC/ EC20: 0.010-1.72 mg/L for algae 0.28 mg/L for Daphnia 0.31 mg/L for fish	Based on Chronic: Very High	Readily biodegradable	No based on the calculated Log Kow of <2.7 and BCF <87	Tier 2	1.30E-04	6.18E-03	7.27E-04	7.04E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Citric acid	77-92-9	1.542	69	0.0003%	106	0.0004%	4	Corrosion Inhibitor	LC50/EC50 > 100 mg/L (fish, daphnia, algae) 8 day NOEC = 425 mg/L (algae)	Based on Chronic: Low	Readily biodegradable	No based on low log Kow	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Benzaldehyde	100-52-7	1.0415	47	0.0002%	48	0.0002%	2	Corrosion Inhibitor	Acute LC50 for freshwater fish is 1.07 mg/L, freshwater invertebrates is 16.2 mg/L and EC10 for freshwater algae is 20 mg/L. Chronic NOEC for freshwater fish is 0.12 mg/L.	Based on Chronic: High	Expected to be readily biodegradable	No based on Log Pow of 1.4	Tier 2	2.29E-05	4.03E-05	1.27E-04	1.91E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Ethanol	64-17-5	0.7864	45	0.0002%	35	0.0001%	1	Surfactant	LC50/EC50 > 1000 mg/L (fish, daphnia, algae) NOEC for invertebrates is 9.6 mg/L (10 day reproduction), plants it is 280 mg/L (7 day study)	Based on Chronic: High	Readily biodegradable	No based on calculated logBCF=0.5	Tier 2	2.07E-07	5.11E-08	1.15E-06	1.41E-06	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Hydrotreated light petroleum distillate	64742-47-8	0.8	43	0.0002%	35	0.0001%	1	Friction Reducer, Surfactant	96 hr LL50 was 2 to 5 mg/L (fish) 48 hr EL50 was 1.4 mg/L (daphnia) 21 d NOEL = 0.48 mg/L (daphnia)	Based on Chronic: High	Readily biodegradable	Yes based on calculated log BCF values for constituents that range from 2.78 to 4.06, and calculated BCF values of 598 to 11,430 L/kg wet-weight	Tier 2	4.90E-07	4.41E-04	2.73E-06	4.45E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Fatty acids, tall-oil, ethoxylated	61791-00-2	1.054	23	0.0001%	24	0.0001%	1	Surfactant	96h-LL50 > 100 mg/L (fish) 48h-EL50 = 12.41 mg/L (invertebrates) 72h-EL50 = 39.7 mg/L (algae) 72h-EL10 = 7.08 mg/L (algae)	Based on Acute: High	Readily biodegradable (read across)	No based on low BCF values of < 100 L/kg ww	Tier 2	3.37E-07	3.27E-06	1.88E-06	5.48E-06	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Amides, tall-oil fatty, N,N-bis(hydroxyethyl)	68155-20-4	0.9	22	0.0001%	20	0.0001%	1	Surfactant	LC50 (96h) 6.7 mg/L (Danio rerio) (similar substance) LC50 (21d) = 0.1 mg/L (Daphnia magna) LC50 (48h) = 2.15 mg/L EC50 (72h) 2.2 mg/L (Scendesmus subspicatus) (similar substance)	Based on Chronic: High	Readily biodegradable (read across)	No Log Kow 3	Tier 2	5.67E-06	1.86E-04	3.16E-05	2.23E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Butyl alcohol	71-36-3	0.81	22	0.0001%	18	0.0001%	1	Surfactant	Fish, LC50 (96h) 1376 mg/l Invertebrates, EC50 (48h) 1328 mg/L) Algae, EC50 (96h) 225 mg/L EC10 (17h) Pseudomonas putida = 2476 mg/L Chronic NOECrepro (21d) = 4.1 mg/L for Daphnia magna	Based on Chronic: Moderate	Readily biodegradable	No based on low log Kow values of 1	Tier 2	1.98E-06	2.11E-06	1.10E-05	1.51E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Alcohols, C12-15, ethoxylated	68131-39-5	0.867	20	0.0001%	18	0.0001%	1	Friction Reducer, Surfactant	96 h LC50 Oncorhynchus mykiss was 5 - 7 mg/L Lepomis macrochirus, NOEC (30 days) was 0.11 – 0.33 mg/L. Daphnia magna, EC50 (48 h) was 2.5 mg/L. Daphnia magna, NOEC (21 days) was 0.77 – 1.75 mg/L. Green algae, EC50 (96 h) was 1.4 mg/L. EC50 (3 h) for microorganisms was 140 mg/L.	Based on Chronic: High	Readily biodegradable	No. Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8, it is not expected to be bioaccumulative.	Tier 2	4.97E-06	3.39E-06	2.77E-05	3.61E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Alcohols, C12-16, ethoxylated	68551-12-2	0.97	20	0.00008%	19	0.00007%	1	Corrosion Inhibitor, Surfactant	96 h LC50 Oncorhynchus mykiss was 5 - 7 mg/L Lepomis macrochirus, NOEC (30 days) was 0.11 – 0.33 mg/L. Daphnia magna, EC50 (48 h) was 2.5 mg/L. Daphnia magna, NOEC (21 days) was 0.77 – 1.75 mg/L. Green algae, EC50 (96 h) was 1.4 mg/L. EC50 (3 h) for microorganisms was 140 mg/L.	Based on Chronic: High	Readily biodegradable	No. Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8, it is not expected to be bioaccumulative.	Tier 2	5.42E-06	2.24E-03	3.02E-05	2.27E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sodium iodide	7681-82-5	3.67	5	0.00002%	19	0.00007%	1	Corrosion Inhibitor	96 hour LC50 for fish is > 860 mg/l 7 days NOEC for fish is 100 mg/L 48hrs-EC50 for Daphnia magna is 1.27 mg/L NOEC for algae is 66 mg/L	Based on Chronic: Low	N.A.(Inorganic)	N.A.(Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Acrylonitrile	107-13-1	0.806	2	0.00001%	2	0.00001%	0.1	Surfactant	96h LC50 for freshwater fish = 10 - 20 mg/l 96h LC50 for saltwater fish 8.6 mg/l 48h EC50 for Daphnia = 7.6 mg/l 30d NOEC for fish of 0.17 mg/l	Based on Chronic: High	Biodegradable	No based on the low log Pow (0.00-0.30)	Tier 2	1.11E-04	5.95E-05	6.21E-04	7.92E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sodium Sulfate	7757-82-6	2.68	0	0.0000004%	0	0.00000%	0.01	Scale Inhibitor	acute studies all show a toxicity of sodium sulfate higher than 100 mg/l	Based on Acute: Low	N.A.(Inorganic)	N.A.(Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Glutaraldehyde	111-30-8	1.05	0	0.0000001%	0	0.00000%	0.001	Biocide	96 h acute Bluegill sunfish LC50 = 11.2 mg/L 48 h acute Oyster larvae LC550 = 2.1 mg/L 96 h acute Green crabs LC50 = 465 mg/L 96 h acute Grass shrimp LC50 = 41 mg/L 48 acute Daphnia magna LC50 = 0.35 mg/L 48 acute Daphnia magna LC50 = 16.3 mg/L 21 d reproductn Daphnia magna LOEC = 4.3 mg/L, NOEC = 2.1 mg/L 96 h algal growth inhibition Selenastrum capricornutum ILm = 3.9 mg/L (median inhibitory limit) 96 h algal growth inhibition Scenedesmus subspicatus EC50 = 1.0 mg/L Bacterial inhibition Sewage microbes IC50 = 25-34 mg/L	Based on Chronic: Moderate	Readily biodegradable	No based on the Log Pow of -0.01	Tier 2	7.47E-05	1.12E-05	4.16E-04	5.02E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
																Total Risk	**0.85**	The calculated risks associated with potential exposure to COPC identified in flowback water, where the HYBRID Recipe is used and assuming 100% mass recovery is below the target of 1, respectively. Hence, chronic health risks are considered to be low and acceptable.

Notes
Tier 1 (NICNAS) - Chemical identified as of low concern for human health, as published in the National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia (NICNAS 2017).
1 - Please refer to the individual toxicity profiles for further detail.
2 - Toxicity assessed using Commonwealth of Australia 2013 Ecotoxicity Assessment Guidelines (presented as Table 4 in the Northern Territory Government Draft Guideline for the Preparation of an Environment Management Plan under the Petroleum Regulations (2019))
3- Biodegradation assessed as per Northern Territory Government Draft Guideline for the Preparation of an Environment Management Plan under the Petroleum Regulations (2019) and Australian Government Department of Health National Industrial Chemicals Notification and Assessment Scheme (NICNAS)
BCF - Bioconcentration Factor
NA - Not Applicable
MOE - Margin of Exposure
NICNAS 2017 - National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia
DOE 2017 - Draft Risk Assessment Guidance Manual: For Chemicals Associated with Coal Seam Gas Extraction, Australian Government, Department of Energy

Toxicity and Dermal Absorption Parameters

C = calculated from chronic value, Ch = chronic value adopted

CAS#	Chemical	Oral/Dermal Exposures				Inhalation Exposures								
		Threshold Chronic TDI or RfD (mg/kg/day)		Dermal Permeability (cm/hr)	Reference	Inhalation Unit Risk $(ug/m^3)^{-1}$	Non-Threshold Slope Factor $(mg/kg/day)^{-1}$		Threshold Chronic TC or RfC (mg/m^3)		NOAEC or LOAEC (mg/m^3)	NOAEL or LOAEL (mg/kg bw/d)	Reference	UF	Reference
	COPC in Hydraulic Fracturing Fluid Injected into Well														
1319-33-1	Boronatrocalcite/Ulexite[A]	0.096	D	9.14E-04	EPI (as boric acid)				0.336	converted from RFD		9.6	NICNAS (2017)	100	NICNAS (2017)
68937-66-6	Alcohols, C6-12, ethoxylated propoxylated[B]	0.5	D	1.21E-04	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
69227-22-1	Alcohols, C10-16, ethoxylated propoxylated[B]	0.5	D	2.87E-01	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
64-19-7	Acetic acid	12	D	5.56E-04	EPI				42	converted from RFD		1200	NICNAS (2017)	100	NICNAS (2017)
25322-68-3	Polyethylene glycol	8	D	2.14E-06	EPI				28	converted from RFD		8000	REACH	1000	D
7631-90-5	Sodium bisulfite[C]	10.5	D	4.16E-09	EPI				36.75	converted from RFD		1050	NICNAS (2017)	100	NICNAS (2017)
104-55-2	Cinnamaldehyde	2	D	5.20E-03	EPI				7	converted from RFD		200	NTP (2004); REACH	100	D
67-56-1	Methanol	0.037	D	3.19E-04	EPI				0.13	converted from RFD		3.7	NICNAS (2017)	100	NICNAS (2017)
61788-90-7	Amine oxides, cocoalkyldimethyl	0.08	D	1.03E-01	EPI				0.28	converted from RFD		80	OECD (2001)	1000	D
100-52-7	Benzaldehyde	0.3	D	3.83E-03	EPI				1.05	converted from RFD		300	OECD (2002); REACH; NICNAS	1000	D
64-17-5	Ethanol	24	D	5.38E-04	EPI				84	converted from RFD		2400	NICNAS (2017)	100	NICNAS (2017)
64742-47-8	Hydrotreated light petroleum distillate	10	D	1.96E+00	EPI				35	converted from RFD		1000	NICNAS (2017)	100	NICNAS (2017)
61791-00-2	Fatty acids, tall-oil, ethoxylated	10	D	2.11E-02	EPI				35	converted from RFD		1000	REACH	100	D
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)	0.5	D	7.14E-02	EPI				1.75	converted from RFD		50	USEPA (2010)	100	D
71-36-3	Butyl alcohol	1.25	D	2.31E-03	EPI				4.375	converted from RFD		125	OECD (2001)/NICNAS	100	D
68131-39-5	Alcohols, C12-15, ethoxylated[B]	0.5	D	1.48E-03	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
68551-12-2	Alcohols, C12-16, ethoxylated[B]	0.5	D	8.97E-01	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
107-13-1	Acrylonitrile	0.0025	D	1.16E-03	EPI				0.00875	converted from RFD		0.25	OECD (2005); NICNAS	100	D
111-42-2	Diethanolamine	0.014	D	4.51E-05	EPI				0.049	converted from RFD		14	REACH; OECD (2002); NICNAS	1000	D
111-30-8	Glutaraldehyde	0.04	D	3.25E-04	EPI				0.14	converted from RFD		4	NICNAS (2017)	100	NICNAS (2017)
102-71-6	Triethanol amine	1.25	D	4.93E-05	EPI				4.375	converted from RFD		125	NICNAS (2017)	100	NICNAS (2017)
7758-19-2	Chlorous acid, sodium salt	0.039	D	8.27E-09	EPI				0.1365	converted from RFD		3.9	NICNAS (2017)	100	NICNAS (2017)
12008-41-2	Disodium octaborate tetrahydrate[A]	0.096	D	9.14E-04	EPI (as boric acid)				0.336	converted from RFD		9.6	NICNAS (2017)	100	NICNAS (2017)
7775-27-1	Sodium persulfate	0.67	D	7.05E-07	EPI				2.345	converted from RFD		67	NICNAS (2017)	100	NICNAS (2017)
68439-54-3	Ethoxylated branched C13 alcohol	0.5	D	1.06E-03	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
1338-43-8	Sobitan, mono-9-octadecenoate, (Z)	25	JECFA	5.02E-02	EPI				87.5	converted from RFD		-	JECFA(1973); US FDA; FSANZ (2018)	-	-
9005-65-6	Sorbitan monooleate polyoxyethylene derivative	10	EFSA	3.54E-09	EPI				35	converted from RFD		-	EFSA (2017)	-	-
111-46-6	2,2''-oxydiethanol (diethylene glycol)	0.3	D	4.17E-05	EPI				1.05	converted from RFD		300	Health Council of the Netherlands (2007); NICNAS	1000	D
7631-90-5	Sodium bisulfate[C]	10.5	D	9.29E-09	EPI				36.75	converted from RFD		1050	NICNAS (2017)	100	NICNAS (2017)
14808-60-7	Crystalline silica, quartz	Not toxic via oral/dermal exposure							0.003	USEPA (2019)		-	-	-	-
10486-00-7	Sodium perborate tetrahydrate	0.05	D	1.81E-06	EPI				0.175	converted from RFD		50	REACH	1000	D

Notes:

A - Read across data from Boric Acid

B - Read across data from Alcohol ethoxylates C6-C12

C - Read across data from Sodium Sulfite

References:

D - Derived (refer to individual Toxicity Profiles)

EFSA - European Food Safety Authority

EPI - USEPA Estimation Programs Interface (EPI) Suite

FSANZ - Food Standards Australia New Zealand

J- JECFA - Joint FAO/WHO Expert Committee on Food Additives

NICNAS - National Industrial Chemicals Notification and Assessment Scheme. Assessed at https://www.nicnas.gov.au/

NICNAS (2017) - Department of the Environment and Energy 2017 , National assessment of chemicals associatedwith coal seam gas extraction in Australia, prepared by the National Industrial ChemicalsNotification and Assessment Scheme

NTP - US Department of Health and Serices National Toxicology Program

OECD - Organisation for Economic Co-operation and Development Screening Information Data Set (SIDS)

REACH - ECHA REACH European Chemicals Agency Database: http://apps.echa.europa.eu

Exposure to Chemicals via Incidental Ingestion of Flowback fluid - Hybrid Recipe

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Flowback Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Ingestion Rate (IRw)	L/day or L/hr	0.005	Assume Incidental ingestion of 5 ml (1 tsp) of water per day during fraccing.
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**4.2E-09** **3.5E-06**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Intake Factor (ref: USEPA 1989)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

	Chemical	Toxicity Data				Concentration	Daily Intake		Calculated Risk	
		Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% Chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)	in Water	NonThreshold	Threshold	NonThreshold Risk	Chronic Hazard Quotient
		(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
68937-66-6	Alcohols, C6-12, ethoxylated propoxylatedB		5.0E-01		5.0E-01	198.94	8.3E-07	7.0E-04	--	1.4E-03
1319-33-1	BoronatrocalciteUlexiteA		9.6E-02		9.6E-02	208.50	8.7E-07	7.3E-04	--	7.6E-03
102-71-6	Triethanol amine		1.3E+00		1.3E+00	149.92	6.3E-07	5.3E-04	--	4.2E-04
69227-22-1	Alcohols, C10-16, ethoxylated propoxylatedB		5.0E-01		5.0E-01	71.05	3.0E-07	2.5E-04	--	5.0E-04
64-19-7	Acetic acid		1.2E+01		1.2E+01	65.91	2.8E-07	2.3E-04	--	1.9E-05
111-42-2	Diethanolamine		1.4E-02		1.4E-02	64.65	2.7E-07	2.3E-04	--	1.6E-02
7631-90-5	Sodium bisulfiteC		1.1E+01		1.1E+01	47.49	2.0E-07	1.7E-04	--	1.6E-05
7758-19-2	Chlorous acid, sodium salt		3.9E-02		3.9E-02	45.57	1.9E-07	1.6E-04	--	4.1E-03
12008-41-2	Disodium octaborate tetrahydrateA		9.6E-02		9.6E-02	25.38	1.1E-07	8.9E-05	--	9.3E-04
104-55-2	Cinnamaldehyde		2.0E+00		2.0E+00	14.02	5.9E-08	4.9E-05	--	2.5E-05
25322-68-3	Polyethylene glycol		8.0E+00		8.0E+00	16.01	6.7E-08	5.6E-05	--	7.0E-06
111-46-6	2,2''-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01	13.65	5.7E-08	4.8E-05	--	1.6E-04
67-56-1	Methanol		3.7E-02		3.7E-02	3.98	1.7E-08	1.4E-05	--	3.8E-04
7775-27-1	Sodium persulfate		6.7E-01		6.7E-01	7.82	3.3E-08	2.7E-05	--	4.1E-05
61788-90-7	Amine oxides, cocoalkyldimethyl		8.0E-02		8.0E-02	2.97	1.2E-08	1.0E-05	--	1.3E-04
100-52-7	Benzaldehyde		3.0E-01		3.0E-01	1.95	8.2E-09	6.9E-06	--	2.3E-05
64-17-5	Ethanol		2.4E+01		2.4E+01	1.41	5.9E-09	5.0E-06	--	2.1E-07
64742-47-8	Hydrotreated light petroleum distillate		1.0E+01		1.0E+01	1.39	5.8E-09	4.9E-06	--	4.9E-07
61791-00-2	Fatty acids, tall-oil, ethoxylated		1.0E+01		1.0E+01	0.96	4.0E-09	3.4E-06	--	3.4E-07
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)		5.0E-01		5.0E-01	0.81	3.4E-09	2.8E-06	--	5.7E-06
71-36-3	Butyl alcohol		1.3E+00		1.3E+00	0.71	3.0E-09	2.5E-06	--	2.0E-06
68131-39-5	Alcohols, C12-15, ethoxylatedB		5.0E-01		5.0E-01	0.71	3.0E-09	2.5E-06	--	5.0E-06
68551-12-2	Alcohols, C12-16, ethoxylatedB		5.0E-01		5.0E-01	0.77	3.2E-09	2.7E-06	--	5.4E-06
107-13-1	Acrylonitrile		2.5E-03		2.5E-03	0.08	3.3E-10	2.8E-07	--	1.1E-04
111-30-8	Glutaraldehyde		4.0E-02		4.0E-02	0.85	3.6E-09	3.0E-06	--	7.5E-05
								Total Risk (mixture)		**3.21E-02**

Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

Dermal Exposure to Chemicals via Contact with Flow Back Water - Hybrid Recipe

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Flow Back Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Surface Area (SAw)	cm^2	2300	Hands and forearms exposed (enHealth 2012) Occupational HSE would require long pants and closed shoes on Australian work sites; forearms conservatively included
Exposure Time (ET)	hr/day	1	Assume contact with flow back water for 1 hours per day
Conversion Factor (CF)	L/cm^3	1.E-03	Conversion of units
Intake Factor = $\frac{SAw*ET*CF*EF*ED}{BW*AT}$	L-hr/(cm-kg-day)	**1.9E-06** **1.6E-03**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor (ref: USEPA 1989, 2004)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

	Chemical	Toxicity Data					Concentration	Daily Intake		Calculated Risk	
		Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)	Dermal Permeability	in Water	NonThreshold	Threshold	NonThreshold Risk	Chronic Hazard Quotient
		$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
68937-66-6	Alcohols, C6-12, ethoxylated propoxylatedB		5.0E-01		5.0E-01	1.2E-4	198.94	4.6E-08	3.9E-05	--	7.8E-05
1319-33-1	Boronatrocalcite/UlexiteA		9.6E-02		9.6E-02	9.1E-4	208.50	3.7E-07	3.1E-04	--	3.2E-03
102-71-6	Triethanol amine		1.3E+00		1.3E+00	4.9E-5	149.92	1.4E-08	1.2E-05	--	9.6E-06
69227-22-1	Alcohols, C10-16, ethoxylated propoxylatedB		5.0E-01		5.0E-01	2.9E-1	71.05	3.9E-05	3.3E-02	--	6.6E-02
64-19-7	Acetic acid		1.2E+01		1.2E+01	5.6E-4	65.91	7.0E-08	5.9E-05	--	4.9E-06
111-42-2	Diethanolamine		1.4E-02		1.4E-02	4.5E-5	64.65	5.6E-09	4.7E-06	--	3.4E-04
7631-90-5	Sodium bisulfiteC		1.1E+01		1.1E+01	4.2E-9	47.49	3.8E-13	3.2E-10	--	3.0E-11
7758-19-2	Chlorous acid, sodium salt		3.9E-02		3.9E-02	8.3E-9	45.57	7.2E-13	6.1E-10	--	1.6E-08
12008-41-2	Disodium octaborate tetrahydrateA		9.6E-02		9.6E-02	9.1E-4	25.38	4.5E-08	3.7E-05	--	3.9E-04
104-55-2	Cinnamaldehyde		2.0E+00		2.0E+00	5.2E-3	14.02	1.4E-07	1.2E-04	--	5.9E-05
25322-68-3	Polyethylene glycol		8.0E+00		8.0E+00	2.1E-6	16.01	6.6E-11	5.5E-08	--	6.9E-09
111-46-6	2,2"-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01	4.2E-5	13.65	1.1E-09	9.2E-07	--	3.1E-06
67-56-1	Methanol		3.7E-02		3.7E-02	3.2E-4	3.98	2.4E-09	2.0E-06	--	5.5E-05
7775-27-1	Sodium persulfate		6.7E-01		6.7E-01	7.1E-7	7.82	1.1E-11	8.9E-09	--	1.3E-08
61788-90-7	Amine oxides, cocoalkyldimethyl		8.0E-02		8.0E-02	1.0E-1	2.97	5.9E-07	4.9E-04	--	6.2E-03
100-52-7	Benzaldehyde		3.0E-01		3.0E-01	3.8E-3	1.95	1.4E-08	1.2E-05	--	4.0E-05
64-17-5	Ethanol		2.4E+01		2.4E+01	5.4E-4	1.41	1.5E-09	1.2E-06	--	5.1E-08
64742-47-8	Hydrotreated light petroleum distillate		1.0E+01		1.0E+01	2.0E+0	1.39	5.3E-06	4.4E-03	--	4.4E-04
61791-00-2	Fatty acids, tall-oil, ethoxylated		1.0E+01		1.0E+01	2.1E-2	0.96	3.9E-08	3.3E-05	--	3.3E-06
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)		5.0E-01		5.0E-01	7.1E-2	0.81	1.1E-07	9.3E-05	--	1.9E-04
71-36-3	Butyl alcohol		1.3E+00		1.3E+00	2.3E-3	0.71	3.1E-09	2.6E-06	--	2.1E-06
68131-39-5	Alcohols, C12-15, ethoxylatedB		5.0E-01		5.0E-01	1.5E-3	0.71	2.0E-09	1.7E-06	--	3.4E-06
68551-12-2	Alcohols, C12-16, ethoxylatedB		5.0E-01		5.0E-01	9.0E-1	0.77	1.3E-06	1.1E-03	--	2.2E-03
107-13-1	Acrylonitrile		2.5E-03		2.5E-03	1.2E-3	0.08	1.8E-10	1.5E-07	--	5.9E-05
111-30-8	Glutaraldehyde		4.0E-02		4.0E-02	3.3E-4	0.85	5.3E-10	4.5E-07	--	1.1E-05
									Total Risk (mixture)		**7.92E-02**

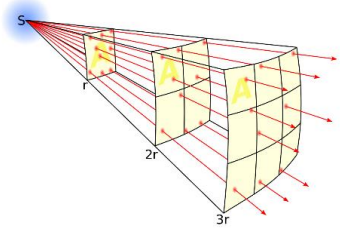
Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

Aerosol Exposure - Hybrid Recipe

The concentration of COPC in aerosol spray was estimated by calculating the concentration for driftable droplets using a mixed box model in which steady state
An emission factor for driftable aerosol was estimated using the algorithm presented below.

Emission Factor for Driftable Aerosol Algorithm

$$Emission\ Factor_{driftable\ aerosol}\left(\frac{L}{m^3}\right) = \frac{Box\ Centre^2(m) \times \left(\frac{Spray\ Vol\ \left(\frac{L}{hr}\right) \times Aerosol_{driftable}(\%)}{BoxVR\ \left(\frac{m^3}{hr}\right)}\right)}{BoxDistance^2\ (m)}$$



Aerosol Exposure Modelling Notes:

1) The inhalation of chemicals in mist/aerosol resultant from irrigation activities is dependent upon the concentration in water, the amount of water used per unit time, how close a person stands to the spray generation, how long they are in a position of exposure and the extent of spray drift (determined by the size of the water droplets and speed/direction of the wind). These equations are applicable for non-volatile contaminants that are inhaled.
2) These equations calculate the concentration for driftable droplets using a simple well mixed box model in which steady state air concentrations are calculated. The 'Inverse square law' is then applied to approximate the air concentration at a distance from the virtual air box. This law assumes the further away a receptor is from the spray source, the density of the droplets will decrease. The density of the spray droplets is inversely proportional to the square of the distance from the source.

Parameter	Units	Value	Description
Spray box length	m	3	Assume a 'spray box' of 3 m long.
Spray box width	m	3	Assume a 'spray box' of 3 m wide.
Box Centre	m	1.5	Distance to centre of box is 1.5 m.
$Box_{Distance}$	m	2	Distance the irrigation worker is from the 'spray box'. Assumed a distance of 2 m.
$Aerosol_{driftable}$	unitless	0.2	Proportion of aerosol spray that drifts outside the 'spray box' and available for exposure. Assumed 0.2, based on a droplet size of 400 – 500 µm that falls approximately 0.3 m in less than 10 seconds, with a lateral drift of approximately 3.5 m in a 5 km/hr wind (i.e. a light breeze) (Grisso et al. 2013).
Spray Volume	L/hr	1800.0	1800 L/min, irrigation value adopted from NZ MtE (2011) Appendix 5A.
Wind speed	m/hr	9000	Based on windspeed of 2.5 m/sec
BoxVR	m^3/hr	81000.0	Ventilation rate of spray in the 'spray box'. Assumed to be 81,000 m3/hr based on a wind speed of 9000 m/hr, and a 'spray box' dimension of 3 x 3 m.

CAS	Chemical	Concentration in Water mg/L	Generation rate of chemical in volume mg/hr	Driftable Aerosol Emission Factor L/m^3
68937-66-6	Alcohols, C6-12, ethoxylated propoxyla	198.94	71619.76796	2.500000E-03
1319-33-1	Boronatrocalcite/UlexiteA	208.50	75061.62126	2.500000E-03
102-71-6	Triethanol amine	149.92	53969.54143	2.500000E-03
69227-22-1	Alcohols, C10-16, ethoxylated propoxy	71.05	25578.48856	2.500000E-03
64-19-7	Acetic acid	65.91	23729.05222	2.500000E-03
111-42-2	Diethanolamine	64.65	23274.23026	2.500000E-03
7631-90-5	Sodium bisulfateC	47.49	17096.46645	2.500000E-03
7758-19-2	Chlorous acid, sodium salt	45.57	16404.17744	2.500000E-03
12008-41-2	Disodium octaborate tetrahydrateA	25.38	9138.176627	2.500000E-03
104-55-2	Cinnamaldehyde	14.02	5046.11094	2.500000E-03
25322-68-3	Polyethylene glycol	16.01	5761.962715	2.500000E-03
111-46-6	2,2"-oxydiethanol (diethylene glycol)	13.65	4915.303675	2.500000E-03
67-56-1	Methanol	3.98	1431.585551	2.500000E-03
7775-27-1	Sodium persulfate	7.82	2816.350509	2.500000E-03
61788-90-7	Amine oxides, cocoalkyldimethyl	2.97	1069.713505	2.500000E-03
100-52-7	Benzaldehyde	1.95	703.1990079	2.500000E-03
64-17-5	Ethanol	1.41	508.3231621	2.500000E-03
64742-47-8	Hydrotreated light petroleum distillate	1.39	501.7922025	2.500000E-03
61791-00-2	Fatty acids, tall-oil, ethoxylated	0.96	345.0387202	2.500000E-03
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxye	0.81	290.4332727	2.500000E-03
71-36-3	Butyl alcohol	0.71	254.0323025	2.500000E-03
68131-39-5	Alcohols, C12-15, ethoxylatedB	0.71	254.9143591	2.500000E-03
68551-12-2	Alcohols, C12-16, ethoxylatedB	0.77	278.0084294	2.500000E-03
107-13-1	Acrylonitrile	0.08	28.55172359	2.500000E-03
111-30-8	Glutaraldehyde	0.85	306.4351619	2.500000E-03
14808-60-7	Crystalline silica, quartz	24.60	8857.767115	2.500000E-03

Exposure to Chemicals via Inhalation of Mist from the Evaporation Units - Hybrid Recipe

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Inhalation of Mist by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays
Exposure Duration (ED)	years	1	Maximum duration that the flowback tank will be on-site
Exposure Time (ET)	hr/day	1	Professional judgement for irrigation exposure. Assume worker to be near tank for 1 hours every working day.
Driftable aerosol emission factor (EMF)	L/m3	2.50E-03	Calculated
Aerosol Inhalation Bioavailability (AAF)	unitless	1.0	Assume 100% bioavailability
Averaging Time - Threshold (AT)	years	1.0	USEPA 1989 and CSMS 1996
$ITF_{inh,w,shwr} = \frac{EmF \times AAF \times ET_{iw} \times EF \times ED}{365 \frac{days}{year} \times 24 \frac{hours}{day} \times AT}$			

Daily Intake = Concentration in Water x Intake Factor (ref: USEPA 1989)
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

CAS	Chemical	Groundwater Concentration	Aerosol Inhalation Bioavailability	Driftable Aerosol Emission Factor	RfC (Background Corrected)	Threshold Intake and Risk Calculations: Adult Exposure Factor (threshold)	Adult Exposure Adjusted Air Concentration (threshold)	Hazard Index (Adult)
		mg/L	(unitless)	(L/m^3)	(mg/m^3)	(L/m^3)	(mg/m^3)	(unitless)
68937-66-6	Alcohols, C6-12, ethoxylated propoxylatedB	1.99E+02	1.00	2.50E-03	1.75E+00	6.85E-05	1.36E-02	7.79E-03
1319-33-1	Boronatrocalcite/UlexiteA	2.09E+02	1.00	2.50E-03	3.36E-01	6.85E-05	1.43E-02	4.25E-02
102-71-6	Triethanol amine	1.50E+02	1.00	2.50E-03	4.38E+00	6.85E-05	1.03E-02	2.35E-03
69227-22-1	Alcohols, C10-16, ethoxylated propoxylatedB	7.11E+01	1.00	2.50E-03	1.75E+00	6.85E-05	4.87E-03	2.78E-03
64-19-7	Acetic acid	6.59E+01	1.00	2.50E-03	4.20E+01	6.85E-05	4.51E-03	1.07E-04
111-42-2	Diethanolamine	6.47E+01	1.00	2.50E-03	4.90E-02	6.85E-05	4.43E-03	9.04E-02
7631-90-5	Sodium bisulfateC	4.75E+01	1.00	2.50E-03	3.68E+01	6.85E-05	3.25E-03	8.85E-05
7758-19-2	Chlorous acid, sodium salt	4.56E+01	1.00	2.50E-03	1.37E-01	6.85E-05	3.12E-03	2.29E-02
12008-41-2	Disodium octaborate tetrahydrateA	2.54E+01	1.00	2.50E-03	3.36E-01	6.85E-05	1.74E-03	5.17E-03
104-55-2	Cinnamaldehyde	1.40E+01	1.00	2.50E-03	7.00E+00	6.85E-05	9.60E-04	1.37E-04
25322-68-3	Polyethylene glycol	1.60E+01	1.00	2.50E-03	2.80E+01	6.85E-05	1.10E-03	3.92E-05
111-46-6	2,2''-oxydiethanol (diethylene glycol)	1.37E+01	1.00	2.50E-03	1.05E+00	6.85E-05	9.35E-04	8.91E-04
67-56-1	Methanol	3.98E+00	1.00	2.50E-03	1.30E-01	6.85E-05	2.72E-04	2.10E-03
7775-27-1	Sodium persulfate	7.82E+00	1.00	2.50E-03	2.35E+00	6.85E-05	5.36E-04	2.29E-04
61788-90-7	Amine oxides, cocoalkyldimethyl	2.97E+00	1.00	2.50E-03	2.80E-01	6.85E-05	2.04E-04	7.27E-04
100-52-7	Benzaldehyde	1.95E+00	1.00	2.50E-03	1.05E+00	6.85E-05	1.34E-04	1.27E-04
64-17-5	Ethanol	1.41E+00	1.00	2.50E-03	8.40E+01	6.85E-05	9.67E-05	1.15E-06
64742-47-8	Hydrotreated light petroleum distillate	1.39E+00	1.00	2.50E-03	3.50E+01	6.85E-05	9.55E-05	2.73E-06
61791-00-2	Fatty acids, tall-oil, ethoxylated	9.58E-01	1.00	2.50E-03	3.50E+01	6.85E-05	6.56E-05	1.88E-06
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)	8.07E-01	1.00	2.50E-03	1.75E+00	6.85E-05	5.53E-05	3.16E-05
71-36-3	Butyl alcohol	7.06E-01	1.00	2.50E-03	4.38E+00	6.85E-05	4.83E-05	1.10E-05
68131-39-5	Alcohols, C12-15, ethoxylatedB	7.08E-01	1.00	2.50E-03	1.75E+00	6.85E-05	4.85E-05	2.77E-05
68551-12-2	Alcohols, C12-16, ethoxylatedB	7.72E-01	1.00	2.50E-03	1.75E+00	6.85E-05	5.29E-05	3.02E-05
107-13-1	Acrylonitrile	7.93E-02	1.00	2.50E-03	8.75E-03	6.85E-05	5.43E-06	6.21E-04
111-30-8	Glutaraldehyde	8.51E-01	1.00	2.50E-03	1.40E-01	6.85E-05	5.83E-05	4.16E-04
14808-60-7	Crystalline silica, quartz	2.46E+01	1.00	2.50E-03	3.00E-03	6.85E-05	1.69E-03	5.62E-01
							Total Threshold Risk (mixture)	**0.7**

Summary of Risk to Workers - Hybrid Recipe
Exposure fo Target Chemicals - Theoretical Data

Receptor/Exposure Pathway	Calculated HI 100% Mass Return
Use of Stimulation Fluid in Hydraulic Fracturing	
HYBRID Recipe	
Workers	
Ingestion of Chemicals via Incidental Contact with Flowback Water	0.03
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water	0.08
Inhalation of mist from the evaporation units	0.74
Total Risk	**0.85**

Appendix B

Chemical Risk Assessment Hydraulic Fracture Stimulation Fluid – HVFR

Chemical Name	CAS Number	Density (kg/L)	Volume of Chemical (L)	Volume Fraction (%v/v)	Chemical Mass in Fluid (kg)	Mass Fraction (% w/w)	Concentration in Injected Fluid (mg/L)	Parent Compound Purpose	Ecotoxicity[1]	Toxicity[2]	Biodegradation[1,3]	Bioaccummulative[1]	Tier 1 Screening Assessment	Tier 2 Assessment Worker Ingestion Risk	Tier 2 Assessment Worker Dermal Risk	Tier 2 Assessment Worker Aerosol Inhalation Risk	Hazard Quotient	Outcome of Tier 2 Worker Risk Assessment
Acetic acid	64-19-7	1.05	1050.64	0.0032%	1,103	0.0032%	35	Acid	Acute endpoints: Fish = 75 mg/L, Daphnia EC50 = 32 mg/L Chronic endpoints: Daphnia = 150 mg/L	Based on Chronic: Low	Readily biodegradable	Not Bio accumulative (Based on log Kow = -0.136)	Tier 2	1.03E-05	2.63E-06	5.72E-05	7.01E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Acrylamide acrylate copolymer	9003-06-9	0.75	1991.54	0.0061%	1,494	0.0043%	47	Scale Inhibitor	96 hour LC50 for fish = 1 400 mg/L 48 hour EC50 for Daphnia magna = 1 200 mg/L 21 day EC50 for Daphnia magna = 680 mg/L 21 day NOEC for algae = 380 mg/L	Based on Chronic: Low	Polymers are not readily biodegradable, hence they meet the screening criteria for persistence.	Polymers are expected to have very high molecular weights and poor water solubility. They are not expected to be bioavailable.	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Acrylamide, sodium acrylate polymer	25987-30-8	0.75	19778.02	0.0603%	14,834	0.0424%	472	Corrosion Inhibitor	LC50 (96h) 0.59 mg/L (Pleuronectes platessa) EC50 (48h) 0.14 mg/L (Daphnia magna) EC50 (96h) 0.7 mg/L (Pseudokirchneriella subapitata) NOEC 4.4 mg/L (Pimephales promelas, juvenile)	96 hour LC50 for fish = 1 400 mg/L 48 hour EC50 for Daphnia magna = 1 200 mg/L 21 day EC50 for Daphnia magna = 680 mg/L 21 day NOEC for algae = 380 mg/L	Based on Chronic: Low	Polymers are not readily biodegradable, hence they meet the screening criteria for persistence.	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Alcohols, C10-16, ethoxylated propoxylated	69227-22-1	0.94	1950.67	0.0059%	1,834	0.0052%	58	Friction Reducer, Surfactant	LC50 (96h) 0.59 mg/L (Pleuronectes platessa EC50 (48h) 0.14 mg/L (Daphnia magna) ErC50 (48h) 0.7 mg/L (Skeletonema costatum) ErC50 (16.9h) > 10 g/L (Pseudomonas putida)	Based on Acute: Very high	Expected to be readily biodegradable based on similar substances	Not Bio accumulative (Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8)	Tier 2	4.10E-04	5.41E-02	2.28E-03	5.68E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Alcohols, C12-15, ethoxylated	68131-39-5	0.867	1679.39	0.0051%	1,456	0.0042%	46	Friction Reducer, Surfactant	96 h LC50 Oncorhynchus mykiss was 5 - 7 mg/L Lepomis macrochirus, NOEC (30 days) was 0.11 – 0.33 mg/L. Daphnia magna, EC50 (48 h) was 2.5 mg/L. Daphnia magna, NOEC (21 days) was 0.77 – 1.75 mg/L. Green algae, EC50 (96 h) was 1.4 mg/L. EC50 (3 h) for microorganisms was 140 mg/L.	Based on Chronic: High	Readily biodegradable	No. Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8, it is not expected to be bioaccumulative.	Tier 2	3.25E-04	2.21E-04	1.81E-03	2.36E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Alcohols, C12-16, ethoxylated	68551-12-2	0.97	1.25	0.0000%	1	0.0000%	0	Corrosion Inhibitor, Surfactant	96 h LC50 Oncorhynchus mykiss was 5 - 7 mg/L Lepomis macrochirus, NOEC (30 days) was 0.11 – 0.33 mg/L. Daphnia magna, EC50 (48 h) was 2.5 mg/L. Daphnia magna, NOEC (21 days) was 0.77 – 1.75 mg/L. Green algae, EC50 (96 h) was 1.4 mg/L. EC50 (3 h) for microorganisms was 140 mg/L.	Based on Chronic: High	Readily biodegradable	No. Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8, it is not expected to be bioaccumulative.	Tier 2	2.70E-07	1.11E-04	1.51E-06	1.13E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Alcohols, C6-12, ethoxylated propoxylated	68937-66-6	0.94	5461.88	0.0166%	5,134	0.0147%	163	Surfactant	LC50 (96h) 0.59 mg/L (Pleuronectes platessa) EC50 (48h) 0.14 mg/L (Daphnia magna) EC50 (96h) 0.7 mg/L (Pseudokirchneriella subapitata) NOEC 4.4 mg/L (Pimephales promelas, juvenile)	Based on Chronic: Moderate	Expected to be readily biodegradable based on similar substances	Not Bio accumulative (Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8)	Tier 2	1.15E-03	6.38E-05	6.39E-03	7.60E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Amides, tall-oil fatty, N,N-bis(hydroxyethyl)	68155-20-4	0.9	1843.23	0.0056%	1,659	0.0047%	53	Surfactant	LC50 (96h) 6.7 mg/L (Danio rerio) (similar substance) LC50 (21d) = 0.1 mg/L (Daphnia magna) LC50 (48h) = 2.15 mg/L EC50 (72h) 2.2 mg/L (Scendesmus subspicatus) (similar substance)	Based on Chronic: High	Readily biodegradable (read across)	No based on Log Kow of 3	Tier 2	3.71E-04	1.22E-02	2.06E-03	1.46E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Amine oxides, cocoalkyldimethyl	61788-90-7	0.716	6.50	0.0000%	5	0.0000%	0	Corrosion Inhibitor	LC50/EC50/ErC50 values: 0.60-32 mg/L for fish 0.50-10.8 mg/L for Daphnia magna 0.010-5.30 mg/L for algae NOEC/ EC20: 0.010-1.72 mg/L for algae 0.28 mg/L for Daphnia 0.31 mg/L for fish	Based on Chronic: Very High	Readily biodegradable	No based on the calculated Log Kow of <2.7 and BCF <87	Tier 2	6.50E-06	3.08E-04	3.62E-05	3.51E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Benzaldehyde	100-52-7	1.0415	2.94	0.0000%	3	0.0000%	0	Corrosion Inhibitor	Acute LC50 for freshwater fish is 1.07 mg/L, freshwater invertebrates is 16.2 mg/L and EC10 for freshwater algae is 20 mg/L. Chronic NOEC for freshwater fish is 0.12 mg/L.	Based on Chronic: High	Expected to be readily biodegradable	No based on Log Pow of 1.4	Tier 2	1.14E-06	2.01E-06	6.35E-06	9.49E-06	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Butyl alcohol	71-36-3	0.81	1791.35	0.0055%	1,451	0.0041%	46	Surfactant	Fish, LC50 (96h) 1376 mg/l Invertebrates, EC50 (48h) 1328 mg/L) Algae, EC50 (96h) 225 mg/L EC10 (17h) Pseudomonas putida = 2476 mg/L Chronic NOECrepro (21d) = 4.1 mg/L for Daphnia magna	Based on Chronic: Moderate	Readily biodegradable	No based on low log Kow values of 1	Tier 2	1.30E-04	1.38E-04	7.22E-04	9.90E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Choline Chloride	67-48-1	1.1	31430.04	0.0958%	34,573	0.0988%	1099	Clay Stabiliser	96-hour fish LC50 value is >100 mg/L 48-hour in vertebrate EC50 is 349 mg/L 72-hour EC50 to Pseudokirchneriella subcapitata is >1,000 mg/L 21-day Daphnia NOEC value is 30.2 mg/L	Based on Chronic: Low	Choline chloride is readily biodegradable and thus it does not meet the screening criteria for persistence.	Not Bioaccumulative (based on a measured log Kow of -3.77 and a calculated BCF of 0.59)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Cinnamaldehyde	104-55-2	1.048	20.95	0.0001%	22	0.0001%	1	Corrosion Inhibitor	Danio rerio (Zebrafish) 96 h LC50 = 3.1 mg/L; Daphnia magna (Water flea) 48 h EC50 = 3.86 mg/L; Pseudokirchneriella subcapitata (Green algae) 72 h EC50 = 4.07 mg/L. 72 h NOEC value = 2.0 mg/L Pseudokirchneriella subcapitata (Green algae)	Based on Chronic: Moderate	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	1.23E-06	2.93E-06	6.83E-06	1.10E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Citric acid	77-92-9	1.542	144.39	0.0004%	223	0.0006%	7	Corrosion Inhibitor	LC50/EC50 > 100 mg/L (fish, daphnia, algae) 8 day NOEC = 425 mg/L (algae)	Based on Chronic: Low	Readily biodegradable	No based on low log Kow	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Diethanolamine	111-42-2	1.1	133.12	0.0004%	146	0.0004%	5	Breaker Activiator	Fish 96-h LC50 = 1370 mg/l Invertebrates 48-h EC50 = 55 mg/l Pseudokirchneriella subcapitata 96-h ErC50 = 2.2 mg/l Microorganisms 16-h TTC = 16 mg/l Daphnia magna, the NOEC (21 days) was 0.78 mg/l	Based on Chronic: High	Readily biodegradable	No. Based on a measured log Kow of -2.18 and a calculated BCF of 3.16	Tier 2	1.17E-03	2.42E-05	6.51E-03	7.70E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).

Chemical Name	CAS Number	Density (kg/L)	Volume of Chemical (L)	Volume Fraction (%v/v)	Chemical Mass in Fluid (kg)	Mass Fraction (% w/w)	Concentration in Injected Fluid (mg/L)	Parent Compound Purpose	Ecotoxicity[1]	Toxicity[2]	Biodegradation[1,3]	Bioaccummulative[1]	Tier 1 Screening Assessment	Tier 2 Assessment Worker Ingestion Risk	Tier 2 Assessment Worker Dermal Risk	Tier 2 Assessment Worker Aerosol Inhalation Risk	Hazard Quotient	Outcome of Tier 2 Worker Risk Assessment
Diethylene glycol	111-46-6	1.12	19.09	0.0001%	21	0.0001%	1	Corrosion Inhibitor	LC 50 = >100 mg/L (fish, invertebrates, algae)	Based on Acute: Low	Readily biodegradable	No based on the estimated BCF of 3	Tier 2	7.96E-06	1.53E-07	4.44E-05	5.25E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Ethanol	64-17-5	0.7864	3692.09	0.0113%	2,903	0.0083%	92	Surfactant	LC50/EC50 > 1000 mg/L (fish, daphnia, algae) NOEC for invertebrates is 9.6 mg/L (10 day reproduction), plants it is 280 mg/L (7 day study)	Based on Chronic: High	Readily biodegradable	No based on calculated logBCF=0.5	Tier 2	1.35E-05	3.34E-06	7.53E-05	9.21E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Ethoxylated branched C13 alcohol	68439-54-3	0.8	1019.49	0.0031%	816	0.0023%	26		96 h LC50 Oncorhynchus mykiss was 5 - 7 mg/L Lepomis macrochirus, NOEC (30 days) was 0.11 – 0.33 mg/L. Daphnia magna, EC50 (48 h) was 2.5 mg/L. Daphnia magna, NOEC (21 days) was 0.77 – 1.75 mg/L. Green algae, EC50 (96 h) was 1.4 mg/L. EC50 (3 h) for microorganisms was 140 mg/L.	Based on Chronic: High	Readily biodegradable	No.	Tier 2	1.82E-04	8.88E-05	1.02E-03	1.29E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Ethylene glycol	107-21-1	1.11	2040.97	0.0062%	2,265	0.0065%	72	Crosslinker	LC50 for fish = 22800 mg/L LC50 for Daphnia =7800 mg/L NOEC for Algae =100 mg/L	Based on Acute: Low	Readily biodegradable	No based on the measured log Kow of -1.36 and a measured BCF of 10	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Fatty acids, tall-oil, ethoxylated	61791-00-2	1.054	1869.83	0.0057%	1,971	0.0056%	63	Surfactant	96h-LL50 > 100 mg/L (fish) 48h-EL50 = 12.41 mg/L (invertebrates) 72h-EL50 = 39.7 mg/L (algae) 72h-EL10 = 7.08 mg/L (algae)	Based on Acute: High	Readily biodegradable (read across)	No based on low BCF values of < 100 L/kg ww	Tier 2	2.20E-05	2.14E-04	1.23E-04	3.58E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Hydrochloric acid	7647-01-0	1.152	4292.88	0.0131%	4,945	0.0141%	157	Acid	Algae = 0.492 mg/L Daphnia = 0.492 mg/L Fish = 4.92 mg/L Daphnia (chronic) = 62 mg/L	Based on Chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	Acute Toxicity Only	Acute Toxicity Only	Acute Toxicity Only	Acute Toxicity Only	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Hydrotreated light petroleum distillate	64742-47-8	0.8	18843.51	0.0574%	15,075	0.0431%	479	Friction Reducer, Surfactant	96 hr LL50 was 2 to 5 mg/L (fish) 48 hr EL50 was 1.4 mg/L (daphnia) 21 d NOEL = 0.48 mg/L (daphnia)	Based on Chronic: High	Readily biodegradable	Yes based on calculated log BCF values for constituents that range from 2.78 to 4.06, and calculated BCF values of 598 to 11,430 L/kg wet-weight	Tier 2	1.68E-04	1.52E-01	9.38E-04	1.53E-01	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Methanol	67-56-1	0.791	191.40	0.0006%	151	0.0004%	5	Corrosion Inhibitor, Surfactant	LC50s ranged from 15,400 to 29,400 mg/L (fish) 24-hour and 48-hour EC50s were > 10,000 mg/L (Daphnia) 28 days NOEC was 446.7 mg/L (fish) 21 days NOEC was 208 mg/L (invertebrates)	Based on Chronic: Low	Readily biodegradable	No based on the Log Kow of -0.74	Tier 2	4.55E-04	6.68E-05	2.54E-03	3.06E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Polyethylene glycol	25322-68-3	1.21	341.37	0.0010%	413	0.0012%	13	Scale Inhibitor	LC50 = 100 mg/L (fish) LC50 = 1000 mg/L (invertebrates) EC 50 = 15.91 mg/L (algae)	Based on Acute: Moderate	Expected to be readily biodegradable	No based on BCF of 3.2	Tier 2	5.77E-06	5.68E-09	3.21E-05	3.79E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sobitan, mono-9-octadecenoate, (Z)	1338-43-8	1.06	1002.94	0.0031%	1,063	0.0030%	34	Surfactant	96 h LC50 for fish = 75 mg/L	Based on Acute: Low	Readily biodegradable	No. Based on a calculated BCF of 2.832 and a BAF of 36.4	Tier 2	4.75E-06	1.10E-04	2.65E-05	1.41E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sodium bisulfite	7631-90-5	2.44	614.20	0.0019%	1,499	0.0043%	48	Scale Inhibitor	72h-EC50 = 36.8 mg sodium sulfite/L (alga) NOEC of >8.41 mg sodium sulfite/L (Daphnia)	Based on Chronic: Moderate	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	1.59E-05	3.05E-11	8.88E-05	1.05E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sodium diacetate	126-96-5	1.01	941.81	0.0029%	951	0.0027%	30	pH buffer	96 h LC 50 for fish = 184.7 mg/L 48h EC 50 for daphnia > 141 mg/L 72 h EC50 for algae = 164 mg/L	Based on Acute: Low	Readily biodegradable	No. Based on a log Kow of -3.72 and a calculated BCF of 3.16	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Sodium hydroxide	1310-73-2	1.515	1213.57	0.0037%	1,839	0.0053%	58	pH buffer	Measured acute endpoints for fish (196 mg/L). Measured chronic endpoint for Daphnia (240 mg/L)	Based on Chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	Acute toxicity only (irritant and corrosive), not systemically available in body	Acute toxicity only (irritant and corrosive), not systemically available in body	Acute toxicity only (irritant and corrosive), not systemically available in body	Acute toxicity only (irritant and corrosive), not systemically available in body	Acute toxicity only (irritant and corrosive), not systemically available in body
Sodium iodide	7681-82-5	3.67	0.33	0.0000%	1	0.0000%	0	Corrosion Inhibitor	96 hour LC50 for fish is > 860 mg/l 7 days NOEC for fish is 100 mg/L 48hrs-EC50 for Daphnia magna is 1.27 mg/L NOEC for algae is 66 mg/L	Based on Chronic: Low	N.A.(Inorganic)	N.A.(Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Sodium polyacrylate	9003-04-7	1.32	3013.30	0.0092%	3,978	0.0114%	126	Gelling Agent	96 hr LC50 for fish is >1000 mg/L NOEC from a chronic early life stage test for the fathead minnow is 56 mg/L 48 hr LC50 for Dapnia magna is >1000 mg/L NOEC for a 21day chronic reproductive test on Daphnia magna is 5.6 mg/L EC10 for Scenedesmus is 180 mg/L	Based on Chronic: Moderate to low	Sodium polyacrylate has limited biodegradation potential and thus meets the screening criteria for persistence.	Bioaccumulation of sodium polyacrylate is unlikely due to the high molecular weight of the polymer.	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Sorbitan monooleate polyoxyethylene derivative	9005-65-6	1.06	915.65	0.0028%	971	0.0028%	31	Surfactant	EC50 in algae was reported to be 100 mg/L	Based on Acute: Low	Not readily biodegradable	No. Based on a log Kow of -2.03 and a calculated BCF of 3.16	Tier 2	1.08E-05	1.77E-11	6.04E-05	7.12E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Tributyl tetradecyl phosphonium chloride	81741-28-8	0.95	936.32	0.0029%	890	0.0025%	28	Biocide	LC50: (96 hour) 0.46 mg/L (Oncorhynchus mykiss) LC50: (96 hour) 0.06 mg/l (Lepomis macrochirus) LC50: (96 hour) 0.58 mg/l (fish) TLM96: 1.6 mg/l (Crangon crangon) TLM48: 0.025 mg/l (Daphnia magna Modelled acute endpoint: Daphnia is 16.788 mg/L Fish is 1059.2530 mg/L	Based on Acute: Very high	Not available, however it has been observed to biodegrade in sediment.	Not bioaccumulative (Based on an estimated log Kow value of 6.26)	Tier 2	Acute toxicity only	Acute toxicity only	Acute toxicity only	Acute toxicity only	Acute toxicity only
Sodium perborate tetrahydrate	10486-00-7	0.65	3060.09	0.0093%	1,989	0.0057%	63	TBD	96hr LC50 for fish is estimated to be 2610 mg/L 48 hr LC50 for daphnids is estimated to be 1241 mg/L 96 hr EC50 for algae is estimated to be 444 mg/L	Based on Acute: Low	Readily biodegradable (read across)	Not bioaccumulative	Tier 2	4.44E-03	3.70E-06	2.48E-02	2.92E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
																Total Risk	**0.28**	The calculated risks associated with potential exposure to COPC identified in flowback water, where the HVFR Recipe is used and assuming 100% mass recovery is below the target of 1, respectively. Hence, chronic health risks are considered to be low and acceptable.

Notes
Tier 1 (NICNAS) - Chemical identified as of low concern for human health, as published in the National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia (NICNAS 2017).
1 - Please refer to the individual toxicity profiles for further detail.
2 - Toxicity assessed using Commonwealth of Australia 2013 Ecotoxicity Assessment Guidelines (presented as Table 4 in the Northern Territory Government Draft Guideline for the Preparation of an Environment Management Plan under the Petroleum Regulations (2019))
3- Biodegradation assessed as per Northern Territory Government Draft Guideline for the Preparation of an Environment Management Plan under the Petroleum Regulations (2019) and Australian Government Department of Health National Industrial Chemicals Notification and Assessment Scheme (NICNAS)
BCF - Bioconcentration Factor
NA - Not Applicable
TBD - To be determined
NICNAS 2017 - National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia
DOE 2017 - Draft Risk Assessment Guidance Manual: For Chemicals Associated with Coal Seam Gas Extraction, Australian Government, Department of Energy

Toxicity and Dermal Absorption Parameters

C = calculated from chronic value, Ch = chronic value adopted

CAS#	Chemical	Oral/Dermal Exposures				Inhalation Exposures									
		Threshold Chronic TDI or RfD (mg/kg/day)		Dermal Permeability (cm/hr)	Reference	Inhalation Unit Risk $(ug/m^3)^{-1}$	Non-Threshold Slope Factor $(mg/kg/day)^{-1}$		Threshold Chronic TC or RfC (mg/m^3)		NOAEC or LOAEC (mg/m^3)	NOAEL or LOAEL (mg/kg bw/d)	Reference	UF	Reference
	COPC in Hydraulic Fracturing Fluid Injected into Well														
1319-33-1	Boronatrocalcite/Ulexite[A]	0.096	D	9.14E-04	EPI (as boric acid)				0.336	converted from RFD		9.6	NICNAS (2017)	100	NICNAS (2017)
68937-66-6	Alcohols, C6-12, ethoxylated propoxylated[B]	0.5	D	1.21E-04	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
69227-22-1	Alcohols, C10-16, ethoxylated propoxylated[B]	0.5	D	2.87E-01	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
64-19-7	Acetic acid	12	D	5.56E-04	EPI				42	converted from RFD		1200	NICNAS (2017)	100	NICNAS (2017)
25322-68-3	Polyethylene glycol	8	D	2.14E-06	EPI				28	converted from RFD		8000	REACH	1000	D
7631-90-5	Sodium bisulfite[C]	10.5	D	4.16E-09	EPI				36.75	converted from RFD		1050	NICNAS (2017)	100	NICNAS (2017)
104-55-2	Cinnamaldehyde	2	D	5.20E-03	EPI				7	converted from RFD		200	NTP (2004); REACH	100	D
67-56-1	Methanol	0.037	D	3.19E-04	EPI				0.13	converted from RFD		3.7	NICNAS (2017)	100	NICNAS (2017)
61788-90-7	Amine oxides, cocoalkyldimethyl	0.08	D	1.03E-01	EPI				0.28	converted from RFD		80	OECD (2001)	1000	D
100-52-7	Benzaldehyde	0.3	D	3.83E-03	EPI				1.05	converted from RFD		300	OECD (2002); REACH; NICNAS	1000	D
64-17-5	Ethanol	24	D	5.38E-04	EPI				84	converted from RFD		2400	NICNAS (2017)	100	NICNAS (2017)
64742-47-8	Hydrotreated light petroleum distillate	10	D	1.96E+00	EPI				35	converted from RFD		1000	NICNAS (2017)	100	NICNAS (2017)
61791-00-2	Fatty acids, tall-oil, ethoxylated	10	D	2.11E-02	EPI				35	converted from RFD		1000	REACH	100	D
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)	0.5	D	7.14E-02	EPI				1.75	converted from RFD		50	USEPA (2010)	100	D
71-36-3	Butyl alcohol	1.25	D	2.31E-03	EPI				4.375	converted from RFD		125	OECD (2001)/NICNAS	100	D
68131-39-5	Alcohols, C12-15, ethoxylated[B]	0.5	D	1.48E-03	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
68551-12-2	Alcohols, C12-16, ethoxylated[B]	0.5	D	8.97E-01	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
107-13-1	Acrylonitrile	0.0025	D	1.16E-03	EPI				0.00875	converted from RFD		0.25	OECD (2005); NICNAS	100	D
111-42-2	Diethanolamine	0.014	D	4.51E-05	EPI				0.049	converted from RFD		14	REACH; OECD (2002); NICNAS	1000	D
111-30-8	Glutaraldehyde	0.04	D	3.25E-04	EPI				0.14	converted from RFD		4	NICNAS (2017)	100	NICNAS (2017)
102-71-6	Triethanol amine	1.25	D	4.93E-05	EPI				4.375	converted from RFD		125	NICNAS (2017)	100	NICNAS (2017)
7758-19-2	Chlorous acid, sodium salt	0.039	D	8.27E-09	EPI				0.1365	converted from RFD		3.9	NICNAS (2017)	100	NICNAS (2017)
12008-41-2	Disodium octaborate tetrahydrate[A]	0.096	D	9.14E-04	EPI (as boric acid)				0.336	converted from RFD		9.6	NICNAS (2017)	100	NICNAS (2017)
7775-27-1	Sodium persulfate	0.67	D	7.05E-07	EPI				2.345	converted from RFD		67	NICNAS (2017)	100	NICNAS (2017)
68439-54-3	Ethoxylated branched C13 alcohol	0.5	D	1.06E-03	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
1338-43-8	Sobitan, mono-9-octadecenoate, (Z)	25	JECFA	5.02E-02	EPI				87.5	converted from RFD		-	JECFA(1973); US FDA; FSANZ (2018)	-	-
9005-65-6	Sorbitan monooleate polyoxyethylene derivative	10	EFSA	3.54E-09	EPI				35	converted from RFD		-	EFSA (2017)	-	-
111-46-6	2,2''-oxydiethanol (diethylene glycol)	0.3	D	4.17E-05	EPI				1.05	converted from RFD		300	Health Council of the Netherlands (2007); NICNAS	1000	D
7631-90-5	Sodium bisulfate[C]	10.5	D	9.29E-09	EPI				36.75	converted from RFD		1050	NICNAS (2017)	100	NICNAS (2017)
14808-60-7	Crystalline silica, quartz	Not toxic via oral/dermal exposure							0.003	USEPA (2019)		-	-	-	-
10486-00-7	Sodium perborate tetrahydrate	0.05	D	1.81E-06	EPI				0.175	converted from RFD		50	REACH	1000	D

Notes:

A - Read across data from Boric Acid

B - Read across data from Alcohol ethoxylates C6-C12

C - Read across data from Sodium Sulfite

References:

D - Derived (refer to individual Toxicity Profiles)

EFSA - European Food Safety Authority

EPI - USEPA Estimation Programs Interface (EPI) Suite

FSANZ - Food Standards Australia New Zealand

J- JECFA - Joint FAO/WHO Expert Committee on Food Additives

NICNAS - National Industrial Chemicals Notification and Assessment Scheme. Assessed at https://www.nicnas.gov.au/

NICNAS (2017) - Department of the Environment and Energy 2017 , National assessment of chemicals associatedwith coal seam gas extraction in Australia, prepared by the National Industrial ChemicalsNotification and Assessment Scheme

NTP - US Department of Health and Serices National Toxicology Program

OECD - Organisation for Economic Co-operation and Development Screening Information Data Set (SIDS)

REACH - ECHA REACH European Chemicals Agency Database: http://apps.echa.europa.eu

Exposure to Chemicals via Incidental Ingestion of Flowback fluid - HVFR Recipe

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Flowback Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Ingestion Rate (IRw)	L/day or L/hr	0.005	Assume Incidental ingestion of 5 ml (1 tsp) of water per day during fraccing.
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**4.2E-09** **3.5E-06**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Intake Factor (ref: USEPA 1989)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

CAS	Chemical	Toxicity Data				Concentration	Daily Intake		Calculated Risk	
		Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% Chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)	in Water	NonThreshold	Threshold	NonThreshold Risk	Chronic Hazard Quotient
		$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
64-19-7	Acetic acid		1.2E+01		1.2E+01	35.08	1.5E-07	1.2E-04	--	1.0E-05
69227-22-1	Alcohols, C10-16, ethoxylated propoxylatedB		5.0E-01		5.0E-01	58.31	2.4E-07	2.0E-04	--	4.1E-04
68131-39-5	Alcohols, C12-15, ethoxylatedB		5.0E-01		5.0E-01	46.30	1.9E-07	1.6E-04	--	3.3E-04
68551-12-2	Alcohols, C12-16, ethoxylatedB		5.0E-01		5.0E-01	0.04	1.6E-10	1.4E-07	--	2.7E-07
68937-66-6	Alcohols, C6-12, ethoxylated propoxylatedB		5.0E-01		5.0E-01	163.27	6.8E-07	5.7E-04	--	1.1E-03
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)		5.0E-01		5.0E-01	52.75	2.2E-07	1.9E-04	--	3.7E-04
61788-90-7	Amine oxides, cocoalkyldimethyl		8.0E-02		8.0E-02	0.15	6.2E-10	5.2E-07	--	6.5E-06
100-52-7	Benzaldehyde		3.0E-01		3.0E-01	0.10	4.1E-10	3.4E-07	--	1.1E-06
71-36-3	Butyl alcohol		1.3E+00		1.3E+00	46.14	1.9E-07	1.6E-04	--	1.3E-04
104-55-2	Cinnamaldehyde		2.0E+00		2.0E+00	0.70	2.9E-09	2.5E-06	--	1.2E-06
111-42-2	Diethanolamine		1.4E-02		1.4E-02	4.66	1.9E-08	1.6E-05	--	1.2E-03
111-46-6	2,2"-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01	0.68	2.8E-09	2.4E-06	--	8.0E-06
64-17-5	Ethanol		2.4E+01		2.4E+01	92.33	3.9E-07	3.2E-04	--	1.4E-05
61791-00-2	Fatty acids, tall-oil, ethoxylated		1.0E+01		1.0E+01	62.67	2.6E-07	2.2E-04	--	2.2E-05
64742-47-8	Hydrotreated light petroleum distillate		1.0E+01		1.0E+01	479.38	2.0E-06	1.7E-03	--	1.7E-04
67-56-1	Methanol		3.7E-02		3.7E-02	4.81	2.0E-08	1.7E-05	--	4.6E-04
25322-68-3	Polyethylene glycol		8.0E+00		8.0E+00	13.14	5.5E-08	4.6E-05	--	5.8E-06
1338-43-8	Sobitan, mono-9-octadecenoate, (Z)		2.5E+01		2.5E+01	33.81	1.4E-07	1.2E-04	--	4.7E-06
9005-65-6	Sorbitan monooleate polyoxyethylene derivative		1.0E+01		1.0E+01	30.86	1.3E-07	1.1E-04	--	1.1E-05
10486-00-7	Sodium perborate tetrahydrate		5.0E-02		5.0E-02	63.25	2.6E-07	2.2E-04	--	4.4E-03
68439-54-3	Ethoxylated branched C13 alcohol		5.0E-01		5.0E-01	25.94	1.1E-07	9.1E-05	--	1.8E-04
7631-90-5	Sodium bisulfiteC		1.1E+01		1.1E+01	47.66	2.0E-07	1.7E-04	--	1.6E-05
								Total Risk (mixture)		**8.90E-03**

Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

Dermal Exposure to Chemicals via Contact with Flow Back Water - HVFR Recipe

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Flow Back Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Surface Area (SAw)	cm^2	2300	Hands and forearms exposed (enHealth 2012) Occupational HSE would require long pants and closed shoes on Australian work sites; forearms conservatively included
Exposure Time (ET)	hr/day	1	Assume contact with flow back water for 1 hours per day
Conversion Factor (CF)	L/cm^3	1.E-03	Conversion of units
Intake Factor = SAw*ET*CF*EF*ED / BW*AT	L-hr/(cm-kg-day)	**1.9E-06** **1.6E-03**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor (ref: USEPA 1989, 2004)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

CAS	Chemical	Toxicity Data					Concentration	Daily Intake		Calculated Risk	
		Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)	Dermal Permeability	in Water	NonThreshold	Threshold	NonThreshold Risk	Chronic Hazard Quotient
		$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
64-19-7	Acetic acid		1.2E+01		1.2E+01	5.6E-4	35.08	3.8E-08	3.2E-05	--	2.6E-06
69227-22-1	Alcohols, C10-16, ethoxylated propoxylatedB		5.0E-01		5.0E-01	2.9E-1	58.31	3.2E-05	2.7E-02	--	5.4E-02
68131-39-5	Alcohols, C12-15, ethoxylatedB		5.0E-01		5.0E-01	1.5E-3	46.30	1.3E-07	1.1E-04	--	2.2E-04
68551-12-2	Alcohols, C12-16, ethoxylatedB		5.0E-01		5.0E-01	9.0E-1	0.04	6.6E-08	5.6E-05	--	1.1E-04
68937-66-6	Alcohols, C6-12, ethoxylated propoxylatedB		5.0E-01		5.0E-01	1.2E-4	163.27	3.8E-08	3.2E-05	--	6.4E-05
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)		5.0E-01		5.0E-01	7.1E-2	52.75	7.2E-06	6.1E-03	--	1.2E-02
61788-90-7	Amine oxides, cocoalkyldimethyl		8.0E-02		8.0E-02	1.0E-1	0.15	2.9E-08	2.5E-05	--	3.1E-04
100-52-7	Benzaldehyde		3.0E-01		3.0E-01	3.8E-3	0.10	7.2E-10	6.0E-07	--	2.0E-06
71-36-3	Butyl alcohol		1.3E+00		1.3E+00	2.3E-3	46.14	2.1E-07	1.7E-04	--	1.4E-04
104-55-2	Cinnamaldehyde		2.0E+00		2.0E+00	5.2E-3	0.70	7.0E-09	5.9E-06	--	2.9E-06
111-42-2	Diethanolamine		1.4E-02		1.4E-02	4.5E-5	4.66	4.0E-10	3.4E-07	--	2.4E-05
111-46-6	2,2"-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01	4.2E-5	0.68	5.5E-11	4.6E-08	--	1.5E-07
64-17-5	Ethanol		2.4E+01		2.4E+01	5.4E-4	92.33	9.6E-08	8.0E-05	--	3.3E-06
61791-00-2	Fatty acids, tall-oil, ethoxylated		1.0E+01		1.0E+01	2.1E-2	62.67	2.5E-06	2.1E-03	--	2.1E-04
64742-47-8	Hydrotreated light petroleum distillate		1.0E+01		1.0E+01	2.0E+0	479.38	1.8E-03	1.5E+00	--	1.5E-01
67-56-1	Methanol		3.7E-02		3.7E-02	3.2E-4	4.81	3.0E-09	2.5E-06	--	6.7E-05
25322-68-3	Polyethylene glycol		8.0E+00		8.0E+00	2.1E-6	13.14	5.4E-11	4.5E-08	--	5.7E-09
1338-43-8	Sobitan, mono-9-octadecenoate, (Z)		2.5E+01		2.5E+01	5.0E-2	33.81	3.3E-06	2.7E-03	--	1.1E-04
9005-65-6	Sorbitan monooleate polyoxyethylene derivative		1.0E+01		1.0E+01	3.5E-9	30.86	2.1E-13	1.8E-10	--	1.8E-11
10486-00-7	Sodium perborate tetrahydrate		5.0E-02		5.0E-02	1.8E-6	63.25	2.2E-10	1.8E-07	--	3.7E-06
68439-54-3	Ethoxylated branched C13 alcohol		5.0E-01		5.0E-01	1.1E-3	25.94	5.3E-08	4.4E-05	--	8.9E-05
7631-90-5	Sodium bisulfiteC		1.1E+01		1.1E+01	4.2E-9	47.66	3.8E-13	3.2E-10	--	3.1E-11
									Total Risk (mixture)		**2.19E-01**

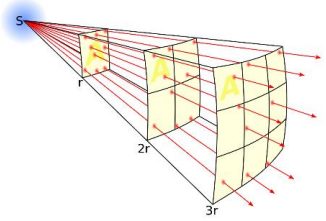
Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

Aerosol Exposure - HVFR Recipe

The concentration of COPC in aerosol spray was estimated by calculating the concentration for driftable droplets using a mixed box model in which steady state concentrations
An emission factor for driftable aerosol was estimated using the algorithm presented below.

Emission Factor for Driftable Aerosol Algorithm

$$Emission\ Factor_{driftable\ aerosol}\left(\frac{L}{m^3}\right) = \frac{Box\ Centre^2(m) \times \left(\frac{Spray\ Vol\ \left(\frac{L}{hr}\right) \times Aerosol_{driftable}(\%)}{BoxVR\ \left(\frac{m^3}{hr}\right)}\right)}{BoxDistance^2\ (m)}$$



Aerosol Exposure Modelling Notes:

1) The inhalation of chemicals in mist/aerosol resultant from irrigation activities is dependent upon the concentration in water, the amount of water used per unit time, how close a person stands to the spray generation, how long they are in a position of exposure and the extent of spray drift (determined by the size of the water droplets and speed/direction of the wind). These equations are applicable for non-volatile contaminants that are inhaled.

2) These equations calculate the concentration for driftable droplets using a simple well mixed box model in which steady state air concentrations are calculated. The 'Inverse square law' is then applied to approximate the air concentration at a distance from the virtual air box. This law assumes the further away a receptor is from the spray source, the density of the droplets will decrease. The density of the spray droplets is inversely proportional to the square of the distance from the source.

Parameter	Units	Value	Description
Spray box length	m	3	Assume a 'spray box' of 3 m long.
Spray box width	m	3	Assume a 'spray box' of 3 m wide.
Box Centre	m	1.5	Distance to centre of box is 1.5 m.
$Box_{Distance}$	m	2	Distance the irrigation worker is from the 'spray box'. Assumed a distance of 2 m.
$Aerosol_{driftable}$	unitless	0.2	Proportion of aerosol spray that drifts outside the 'spray box' and available for exposure. Assumed 0.2, based on a droplet size of 400 – 500 µm that falls approximately 0.3 m in less than 10 seconds, with a lateral drift of approximately 3.5 m in a 5 km/hr wind (i.e. a light breeze) (Grisso et al. 2013).
Spray Volume	L/hr	1800.0	1800 L/min, irrigation value adopted from NZ MtE (2011) Appendix 5A.
Wind speed	m/hr	9000	Based on windspeed of 2.5 m/sec
BoxVR	m^3/hr	81000.0	Ventilation rate of spray in the 'spray box'. Assumed to be 81,000 m3/hr based on a wind speed of 9000 m/hr, and a 'spray box' dimension of 3 x 3 m.

CAS	Chemical	Concentration in Water mg/L	Generation rate of chemical in volume mg/hr	Driftable Aerosol Emission Factor L/m^3
64-19-7	Acetic acid	35.08	12629.04069	2.500000E-03
69227-22-1	Alcohols, C10-16, ethoxylated propoxylated	58.31	20991.25298	2.500000E-03
68131-39-5	Alcohols, C12-15, ethoxylatedB	46.30	16668.49372	2.500000E-03
68551-12-2	Alcohols, C12-16, ethoxylatedB	0.04	13.84454389	2.500000E-03
68937-66-6	Alcohols, C6-12, ethoxylated propoxylatedB	163.27	58775.50834	2.500000E-03
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)	52.75	18991.02585	2.500000E-03
61788-90-7	Amine oxides, cocoalkyldimethyl	0.15	53.27067097	2.500000E-03
100-52-7	Benzaldehyde	0.10	35.01861274	2.500000E-03
71-36-3	Butyl alcohol	46.14	16610.81728	2.500000E-03
104-55-2	Cinnamaldehyde	0.70	251.2913171	2.500000E-03
111-42-2	Diethanolamine	4.66	1676.321009	2.500000E-03
111-46-6	2,2"-oxydiethanol (diethylene glycol)	0.68	244.7772451	2.500000E-03
64-17-5	Ethanol	92.33	33238.54124	2.500000E-03
61791-00-2	Fatty acids, tall-oil, ethoxylated	62.67	22561.59975	2.500000E-03
64742-47-8	Hydrotreated light petroleum distillate	479.38	172575.2226	2.500000E-03
67-56-1	Methanol	4.81	1733.209245	2.500000E-03
25322-68-3	Polyethylene glycol	13.14	4728.6147	2.500000E-03
1338-43-8	Sobitan, mono-9-octadecenoate, (Z)	33.81	12170.50102	2.500000E-03
9005-65-6	Sorbitan monooleate polyoxyethylene deriv	30.86	11111.21667	2.500000E-03
10486-00-7	Sodium perborate tetrahydrate	63.25	22770.58133	2.500000E-03
68439-54-3	Ethoxylated branched C13 alcohol	25.94	9336.793624	2.500000E-03
7631-90-5	Sodium bisulfiteC	47.66	17156.33574	2.500000E-03

Exposure to Chemicals via Inhalation of Mist from the Evaporation Units - HVFR Recipe

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Inhalation of Mist by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays
Exposure Duration (ED)	years	1	Maximum duration that the flowback tank will be on-site
Exposure Time (ET)	hr/day	1	Professional judgement for irrigation exposure. Assume worker to be near tank for 1 hours every working day.
Driftable aerosol emission factor (EMF)	L/m3	2.50E-03	Calculated
Aerosol Inhalation Bioavailability (AAF)	unitless	1.0	Assume 100% bioavailability
Averaging Time - Threshold (AT)	years	1.000	USEPA 1989 and CSMS 1996
$ITF_{inh,w,shwr} = \frac{EmF \times AAF \times ET_{iw} \times EF \times ED}{365\ \frac{days}{year} \times 24\ \frac{hours}{day} \times AT}$			

Daily Intake = Concentration in Water x Intake Factor (ref: USEPA 1989)
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

CAS	Chemical	Groundwater Concentration	Aerosol Inhalation Bioavailability	Driftable Aerosol Emission Factor	RfC (Background Corrected)	Threshold Intake and Risk Calculations: Adult Exposure Factor (threshold)	Adult Exposure Adjusted Air Concentration (threshold)	Hazard Index (Adult)
		mg/L	(unitless)	(L/m^3)	(mg/m^3)	(L/m^3)	(mg/m^3)	(unitless)
64-19-7	Acetic acid	3.51E+01	1.00	2.50E-03	4.20E+01	6.85E-05	2.40E-03	5.72E-05
69227-22-1	Alcohols, C10-16, ethoxylated propoxylatedB	5.83E+01	1.00	2.50E-03	1.75E+00	6.85E-05	3.99E-03	2.28E-03
68131-39-5	Alcohols, C12-15, ethoxylatedB	4.63E+01	1.00	2.50E-03	1.75E+00	6.85E-05	3.17E-03	1.81E-03
68551-12-2	Alcohols, C12-16, ethoxylatedB	3.85E-02	1.00	2.50E-03	1.75E+00	6.85E-05	2.63E-06	1.51E-06
68937-66-6	Alcohols, C6-12, ethoxylated propoxylatedB	1.63E+02	1.00	2.50E-03	1.75E+00	6.85E-05	1.12E-02	6.39E-03
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)	5.28E+01	1.00	2.50E-03	1.75E+00	6.85E-05	3.61E-03	2.06E-03
61788-90-7	Amine oxides, cocoalkyldimethyl	1.48E-01	1.00	2.50E-03	2.80E-01	6.85E-05	1.01E-05	3.62E-05
100-52-7	Benzaldehyde	9.73E-02	1.00	2.50E-03	1.05E+00	6.85E-05	6.66E-06	6.35E-06
71-36-3	Butyl alcohol	4.61E+01	1.00	2.50E-03	4.38E+00	6.85E-05	3.16E-03	7.22E-04
104-55-2	Cinnamaldehyde	6.98E-01	1.00	2.50E-03	7.00E+00	6.85E-05	4.78E-05	6.83E-06
111-42-2	Diethanolamine	4.66E+00	1.00	2.50E-03	4.90E-02	6.85E-05	3.19E-04	6.51E-03
111-46-6	2,2"-oxydiethanol (diethylene glycol)	6.80E-01	1.00	2.50E-03	1.05E+00	6.85E-05	4.66E-05	4.44E-05
64-17-5	Ethanol	9.23E+01	1.00	2.50E-03	8.40E+01	6.85E-05	6.32E-03	7.53E-05
61791-00-2	Fatty acids, tall-oil, ethoxylated	6.27E+01	1.00	2.50E-03	3.50E+01	6.85E-05	4.29E-03	1.23E-04
64742-47-8	Hydrotreated light petroleum distillate	4.79E+02	1.00	2.50E-03	3.50E+01	6.85E-05	3.28E-02	9.38E-04
67-56-1	Methanol	4.81E+00	1.00	2.50E-03	1.30E-01	6.85E-05	3.30E-04	2.54E-03
25322-68-3	Polyethylene glycol	1.31E+01	1.00	2.50E-03	2.80E+01	6.85E-05	9.00E-04	3.21E-05
1338-43-8	Sobitan, mono-9-octadecenoate, (Z)	3.38E+01	1.00	2.50E-03	8.75E+01	6.85E-05	2.32E-03	2.65E-05
9005-65-6	Sorbitan monooleate polyoxyethylene derivative	3.09E+01	1.00	2.50E-03	3.50E+01	6.85E-05	2.11E-03	6.04E-05
10486-00-7	Sodium perborate tetrahydrate	6.33E+01	1.00	2.50E-03	1.75E-01	6.85E-05	4.33E-03	2.48E-02
68439-54-3	Ethoxylated branched C13 alcohol	2.59E+01	1.00	2.50E-03	1.75E+00	6.85E-05	1.78E-03	1.02E-03
7631-90-5	Sodium bisulfiteC	4.77E+01	1.00	2.50E-03	3.68E+01	6.85E-05	3.26E-03	8.88E-05
							Total Risk (mixture)	**0.050**

Summary of Risk to Workers - HVFR Recipe
Exposure fo Target Chemicals - Theoretical Data

Receptor/Exposure Pathway	Calculated HI 100% Mass Return
Use of Stimulation Fluid in Hydraulic Fracturing	
HVFR Recipe	
Workers	
Ingestion of Chemicals via Incidental Contact with Flowback Water	0.0089
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water	0.22
Inhalation of mist from the evaporation units	0.050
Total Risk	**0.28**

Appendix C

Chemical Risk Assessment Hydraulic Fracture Stimulation Fluid – SW

Chemical Name	CAS Number	Density (kg/L)	Volume of Chemical (L)	Volume Fraction (%v/v)	Chemical Mass in Fluid (kg)	Mass Fraction (% w/w)	Concentration in Injected Fluid (mg/L)	Parent Compound Purpose	Ecotoxicity[1]	Toxicity[2]	Biodegradation[1,3]	Bioaccummulative[1]	Tier 1 Screening Assessment	Tier 2 Assessment Worker Ingestion Risk	Tier 2 Assessment Worker Dermal Risk	Tier 2 Assessment Worker Aerosol Inhalation Risk	Hazard Quotient	Outcome of Tier 2 Worker Risk Assessment[1]
Choline Chloride	67-48-1	1.1	24720	0.0848%	27192	0.0869%	977	Clay Stabiliser	96-hour fish LC50 value is >100 mg/L 48-hour in vertebrate EC50 is 349 mg/L 72-hour EC50 to Pseudokirchneriella subcapitata is >1,000 mg/L 21-day Daphnia NOEC value is 30.2 mg/L	Based on Chronic: Low	Choline chloride is readily biodegradable and thus it does not meet the screening criteria for persistence.	Not Bioaccumulative (based on a measured log Kow of -3.77 and a calculated BCF of 0.59)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Hydrochloric acid	7647-01-0	1.152	23649	0.0811%	27244	0.0871%	979	Acid	Algae (acute) = 0.492 mg/L Daphnia (acute) = 0.492 mg/L Fish (acute) = 4.92 mg/L Daphnia (chronic) = 62 mg/L	Based on Chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	NA. Acute toxity only	NA. Acute toxity only	NA. Acute toxity only	NA. Acute toxity only	NA. Acute toxity only
Alcohols, C6-12, ethoxylated propoxylated	68937-66-6	0.94	10,206	0.0350%	9,593	0.0307%	345	Surfactant	LC50 (96h) 0.59 mg/L (Pleuronectes platessa) EC50 (48h) 0.14 mg/L (Daphnia magna) EC50 (96h) 0.7 mg/L (Pseudokirchneriella subapitata) NOEC 4.4 mg/L (Pimephales promelas, juvenile)	Based on Chronic: Moderate	Expected to be readily biodegradable based on similar substances	Not Bio accumulative (Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8)	Tier 2	2.42E-03	1.35E-04	1.35E-02	1.60E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Alcohols, C10-16, ethoxylated propoxylated	69227-22-1	0.94	5,253	0.0180%	4,938	0.0158%	177	Friction Reducer, Surfactant	LC50 (96h) 0.59 mg/L (Pleuronectes platessa EC50 (48h) 0.14 mg/L (Daphnia magna) ErC50 (48h) 0.7 mg/L (Skeletonema costatum) ErC50 (16.9h) > 10 g/L (Pseudomonas putida)	Based on Acute: Very high	Expected to be readily biodegradable based on similar substances	Not Bio accumulative (Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8)	Tier 2	1.25E-03	1.64E-01	6.94E-03	1.73E-01	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sodium polyacrylate	9003-04-7	1.32	3723	0.0128%	4914	0.0157%	177		96 hr LC50 for fish is >1000 mg/L NOEC from a chronic early life stage test for the fathead minnow is 56 mg/L 48 hr LC50 for Dapnia magna is >1000 mg/L NOEC for a 21day chronic reproductive test on Daphnia magna is 5.6 mg/L EC10 for Scenedesmus is 180 mg/L	Based on Chronic: Moderate to low	Sodium polyacrylate has limited biodegradation potential and thus meets the screening criteria for persistence.	Bioaccumulation of sodium polyacrylate is unlikely due to the high molecular weight of the polymer.	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Sodium Chloride	7647-14-5	2.165	3476	0.0119%	7525	0.0241%	270	Stabiliser	EC50 = 400 to 30000 mg/L EC50 Fish = 1290 mg/L NOEC = 314 mg/L (Daphnia)	Based on Chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	Low chronic toxicity, insufficient data to establish toxicity value	Low chronic toxicity, insufficient data to establish toxicity value	Low chronic toxicity, insufficient data to establish toxicity value	Low chronic toxicity, insufficient data to establish toxicity value	Low chronic toxicity, insufficient data to establish toxicity value
Acrylamide acrylate copolymer	25987-30-8	0.75	3309	0.0113%	2482	0.0079%	89	Scale Inhibitor	96 hour LC50 for fish = 1 400 mg/L 48 hour EC50 for Daphnia magna = 1 200 mg/L 21 day EC50 for Daphnia magna = 680 mg/L 21 day NOEC for algae = 380 mg/L	Based on Chronic: Low	Polymers are not readily biodegradable, hence they meet the screening criteria for persistence.	Polymers are expected to have very high molecular weights and poor water solubility. They are not expected to be bioavailable.	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Acetic acid	64-19-7	1.05	2859	0.0098%	3002	0.0096%	108	Acid	Acute endpoints: Fish = 75 mg/L, Daphnia EC50 = 32 mg/L Chronic endpoints: Daphnia = 150 mg/L	Based on Chronic: Low	Readily biodegradable	Not Bio accumulative (Based on log Kow = -0.136)	Tier 2	3.16E-05	8.07E-06	1.76E-04	2.15E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Tributyl tetradecyl phosphonium chloride	81741-28-8	0.95	2370	0.0081%	2251	0.0072%	81	Biocide	LC50: (96 hour) 0.46 mg/L (Oncorhynchus mykiss) LC50: (96 hour) 0.06 mg/l (Lepomis macrochirus) LC50: (96 hour) 0.58 mg/l (fish) TLM96: 1.6 mg/l (Crangon crangon) TLM48: 0.025 mg/l (Daphnia magna Modelled acute endpoint: Daphnia is 16.788 mg/L Fish is 1059.2530 mg/L	Based on Acute: Very high	Not available, however it has been observed to biodegrade in sediment.	Not bioaccumulative (Based on an estimated log Kow value of 6.26)	Tier 2	NA. Acute toxity only	NA. Acute toxity only	NA. Acute toxity only	NA. Acute toxity only	NA. Acute toxity only
Polyethylene glycol	25322-68-3	1.21	2059	0.0071%	2491	0.0080%	89	Scale Inhibitor	LC50 = 100 mg/L (fish) LC50 = 1000 mg/L (invertebrates) EC 50 = 15.91 mg/L (algae)	Based on Acute: Moderate	Expected to be readily biodegradable	No based on BCF of 3.2	Tier 2	3.93E-05	3.87E-08	2.19E-04	2.58E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sodium bisulfite	7631-90-5	1.348	1876	0.0064%	2529	0.0081%	91	Scale Inhibitor	72h-EC50 = 36.8 mg sodium sulfite/L (alga) NOEC of >8.41 mg sodium sulfite/L (Daphnia)	Based on Chronic: Moderate	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	3.04E-05	5.81E-11	1.69E-04	2.00E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Ethylene glycol	107-21-1	1.11	1558	0.0053%	1729	0.0055%	62	Crosslinker	LC50 for fish = 22800 mg/L LC50 for Daphnia =7800 mg/L NOEC for Algae =100 mg/L	Based on Acute: Low	Readily biodegradable	No based on the measured log Kow of -1.36 and a measured BCF of 10	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Cinnamaldehyde	104-55-2	1.048	1459	0.0050%	1529	0.0049%	55	Corrosion Inhibitor	Danio rerio (Zebrafish) 96 h LC50 = 3.1 mg/L; Daphnia magna (Water flea) 48 h EC50 = 3.86 mg/L; Pseudokirchneriella subcapitata (Green algae) 72 h EC50 = 4.07 mg/L. 72 h NOEC value = 2.0 mg/L Pseudokirchneriella subcapitata (Green algae)	Based on Chronic: Moderate	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	9.64E-05	2.31E-04	5.37E-04	8.64E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Diethylene glycol	111-46-6	1.12	736	0.0025%	825	0.0026%	30	Corrosion Inhibitor	LC 50 = >100 mg/L (fish, invertebrates, algae)	Based on Acute: Low	Readily biodegradable	No based on the estimated BCF of 3	Tier 2	3.47E-04	6.65E-06	1.93E-03	2.29E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Methanol	67-56-1	0.791	730	0.0025%	578	0.0018%	21	Corrosion Inhibitor, Surfactant	LC50s ranged from 15,400 to 29,400 mg/L (fish) 24-hour and 48-hour EC50s were > 10,000 mg/L (Daphnia) 28 days NOEC was 446.7 mg/L (fish) 21 days NOEC was 208 mg/L (invertebrates)	Based on Chronic: Low	Readily biodegradable	No based on the Log Kow of -0.74	Tier 2	1.96E-03	2.88E-04	1.09E-02	1.32E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Amine oxides, cocoalkyldimethyl	61788-90-7	0.716	483	0.0017%	346	0.0011%	12	Corrosion Inhibitor	LC50/EC50/ErC50 values: 0.60-32 mg/L for fish 0.50-10.8 mg/L for Daphnia magna 0.010-5.30 mg/L for algae NOEC/ EC20: 0.010-1.72 mg/L for algae 0.28 mg/L for Daphnia 0.31 mg/L for fish	Based on Chronic: Very High	Readily biodegradable	No based on the calculated Log Kow of <2.7 and BCF <87	Tier 2	5.45E-04	2.58E-02	3.04E-03	2.94E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sodium hydroxide	1310-73-2	1.515	458	0.0016%	694	0.0022%	25	pH buffer	Measured acute endpoints for fish (196 mg/L). Measured chronic endpoint for Daphnia (240 mg/L)	Based on Chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	NA. Acute toxicity only (irritant and corrosive), not systemically available in body	NA. Acute toxicity only (irritant and corrosive), not systemically available in body	NA. Acute toxicity only (irritant and corrosive), not systemically available in body	NA. Acute toxicity only (irritant and corrosive), not systemically available in body	NA. Acute toxicity only (irritant and corrosive), not systemically available in body
Citric acid	77-92-9	1.542	336	0.0012%	518	0.0017%	19	Corrosion Inhibitor	LC50/EC50 > 100 mg/L (fish, daphnia, algae) 8 day NOEC = 425 mg/L (algae)	Based on Chronic: Low	Readily biodegradable	No based on low log Kow	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Benzaldehyde	100-52-7	1.0415	332	0.0011%	346	0.0011%	12	Corrosion Inhibitor	Acute LC50 for freshwater fish is 1.07 mg/L, freshwater invertebrates is 16.2 mg/L and EC10 for freshwater algae is 20 mg/L. Chronic NOEC for freshwater fish is 0.12 mg/L.	Based on Chronic: High	Expected to be readily biodegradable	No based on Log Pow of 1.4	Tier 2	1.45E-04	2.56E-04	8.10E-04	1.21E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Ethanol	64-17-5	0.7864	328	0.0011%	258	0.0008%	9	Surfactant	LC50/EC50 > 1000 mg/L (fish, daphnia, algae) NOEC for invertebrates is 9.6 mg/L (10 day reproduction), plants it is 280 mg/L (7 day study)	Based on Chronic: High	Readily biodegradable	No based on calculated logBCF=0.5	Tier 2	1.36E-06	3.36E-07	7.56E-06	9.25E-06	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Hydrotreated light petroleum distillate	64742-47-8	0.8	303	0.0010%	242	0.0008%	9	Friction Reducer, Surfactant	96 hr LL50 was 2 to 5 mg/L (fish) 48 hr EL50 was 1.4 mg/L (daphnia) 21 d NOEL = 0.48 mg/L (daphnia)	Based on Chronic: High	Readily biodegradable	Yes based on calculated log BCF values for constituents that range from 2.78 to 4.06, and calculated BCF values of 598 to 11,430 L/kg wet-weight	Tier 2	3.05E-06	2.75E-03	1.70E-05	2.77E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Fatty acids, tall-oil, ethoxylated	61791-00-2	1.054	235	0.0008%	248	0.0008%	9	Surfactant	96h-LL50 > 100 mg/L (fish) 48h-EL50 = 12.41 mg/L (invertebrates) 72h-EL50 = 39.7 mg/L (algae) 72h-EL10 = 7.08 mg/L (algae)	Based on Acute: High	Readily biodegradable (read across)	No based on low BCF values of < 100 L/kg ww	Tier 2	3.12E-06	3.03E-05	1.74E-05	5.08E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Amides, tall-oil fatty, N,N-bis(hydroxyethyl)	68155-20-4	0.9	125	0.0004%	112	0.0004%	4	Surfactant	LC50 (96h) 6.7 mg/L (Danio rerio) (similar substance) LC50 (21d) = 0.1 mg/L (Daphnia magna) LC50 (48h) = 2.15 mg/L EC50 (72h) 2.2 mg/L (Scendesmus subspicatus) (similar substance)	Based on Chronic: High	Readily biodegradable (read across)	No Log Kow 3	Tier 2	2.83E-05	9.31E-04	1.58E-04	1.12E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).

Chemical Name	CAS Number	Density (kg/L)	Volume of Chemical (L)	Volume Fraction (%v/v)	Chemical Mass in Fluid (kg)	Mass Fraction (% w/w)	Concentration in Injected Fluid (mg/L)	Parent Compound Purpose	Ecotoxicity[1]	Toxicity[2]	Biodegradation[1,3]	Bioaccummulative[1]	Tier 1 Screening Assessment	Tier 2 Assessment Worker Ingestion Risk	Tier 2 Assessment Worker Dermal Risk	Tier 2 Assessment Worker Aerosol Inhalation Risk	Hazard Quotient	Outcome of Tier 2 Worker Risk Assessment[1]
Butyl alcohol	71-36-3	0.81	116	0.0004%	94	0.0003%	3	Surfactant	Fish, LC50 (96h) 1376 mg/l Invertebrates, EC50 (48h) 1328 mg/L) Algae, EC50 (96h) 225 mg/L EC10 (17h) Pseudomonas putida = 2476 mg/L Chronic NOECrepro (21d) = 4.1 mg/L for Daphnia magna	Based on Chronic: Moderate	Readily biodegradable	No based on low log Kow values of 1	Tier 2	9.45E-06	1.00E-05	5.26E-05	7.21E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Alcohols, C12-15, ethoxylated	68131-39-5	0.867	103	0.0004%	89	0.0003%	3	Friction Reducer, Surfactant	96 h LC50 Oncorhynchus mykiss was 5 - 7 mg/L Lepomis macrochirus, NOEC (30 days) was 0.11 – 0.33 mg/L. Daphnia magna, EC50 (48 h) was 2.5 mg/L. Daphnia magna, NOEC (21 days) was 0.77 – 1.75 mg/L. Green algae, EC50 (96 h) was 1.4 mg/L. EC50 (3 h) for microorganisms was 140 mg/L.	Based on Chronic: High	Readily biodegradable	No. Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8, it is not expected to be bioaccumulative.	Tier 2	2.25E-05	1.53E-05	1.26E-04	1.63E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Alcohols, C12-16, ethoxylated	68551-12-2	0.97	69	0.0002%	67	0.0002%	2	Corrosion Inhibitor, Surfactant	96 h LC50 Oncorhynchus mykiss was 5 - 7 mg/L Lepomis macrochirus, NOEC (30 days) was 0.11 – 0.33 mg/L. Daphnia magna, EC50 (48 h) was 2.5 mg/L. Daphnia magna, NOEC (21 days) was 0.77 – 1.75 mg/L. Green algae, EC50 (96 h) was 1.4 mg/L. EC50 (3 h) for microorganisms was 140 mg/L.	Based on Chronic: High	Readily biodegradable	No. Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8, it is not expected to be bioaccumulative.	Tier 2	1.68E-05	6.93E-03	9.36E-05	7.04E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sodium iodide	7681-82-5	3.67	47	0.0002%	171	0.0005%	6	Corrosion Inhibitor	96 hour LC50 for fish is > 860 mg/l 7 days NOEC for fish is 100 mg/L 48hrs-EC50 for Daphnia magna is 1.27 mg/L NOEC for algae is 66 mg/L	Based on Chronic: Low	N.A.(Inorganic)	N.A.(Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Acrylonitrile	107-13-1	0.806	45	0.0002%	36	0.0001%	1	Surfactant	96h LC50 for freshwater fish = 10 - 20 mg/l 96h LC50 for saltwater fish 8.6 mg/l 48h EC50 for Daphnia = 7.6 mg/l 30d NOEC for fish of 0.17 mg/l	Based on Chronic: High	Biodegradable	No based on the low log Pow (0.00-0.30)	Tier 2	1.81E-03	9.67E-04	1.01E-02	1.29E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Diethanolamine	111-42-2	1.1	43	0.0001%	48	0.0002%	2	Breaker Activiator	Fish 96-h LC50 = 1370 mg/l Invertebrates 48-h EC50 = 55 mg/l Pseudokirchneriella subcapitata 96-h ErC50 = 2.2 mg/l Microorganisms 16-h TTC = 16 mg/l Daphnia magna, the NOEC (21 days) was 0.78 mg/l	Based on Chronic: High	Readily biodegradable	No. Based on a measured log Kow of -2.18 and a calculated BCF of 3.16	Tier 2	4.29E-04	8.89E-06	2.39E-03	2.83E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Glutaraldehyde	111-30-8	1.05	23	0.0001%	24	0.0001%	1	Biocide	96 h acute Bluegill sunfish LC50 = 11.2 mg/L 48 h acute Oyster larvae LC550 = 2.1 mg/L 96 h acute Green crabs LC50 = 465 mg/L 96 h acute Grass shrimp LC50 = 41 mg/L 48 acute Daphnia magna LC50 = 0.35 mg/L 48 acute Daphnia magna LC50 = 16.3 mg/L 21 d reproducfn Daphnia magna LOEC = 4.3 mg/L, NOEC = 2.1 mg/L 96 h algal growth inhibition Selenastrum capricornutum ILm = 3.9 mg/L (median inhibitory limit) 96 h algal growth inhibition Scenedesmus subspicatus EC50 = 1.0 mg/L Bacterial inhibition Sewage microbes IC50 = 25-34 mg/L	Based on Chronic: Moderate	Readily biodegradable	No based on the Log Pow of -0.01	Tier 2	7.47E-05	1.12E-05	4.16E-04	5.02E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
																Total Risk	**0.26**	The calculated risks associated with potential exposure to COPC identified in flowback water, where the SW Recipe is used and assuming 100% mass recovery is below the target of 1, respectively. Hence, chronic health risks are considered to be low and acceptable.

Notes
Tier 1 (NICNAS) - Chemical identified as of low concern for human health, as published in the National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia (NICNAS 2017).
1 - Please refer to the individual toxicity profiles for further detail.
2 - Toxicity assessed using Commonwealth of Australia 2013 Ecotoxicity Assessment Guidelines (presented as Table 4 in the Northern Territory Government Draft Guideline for the Preparation of an Environment Management Plan under the Petroleum Regulations (2019)
3- Biodegradation assessed as per Northern Territory Government Draft Guideline for the Preparation of an Environment Management Plan under the Petroleum Regulations (2019) and Australian Government Department of Health National Industrial Chemicals Notification and Assessment Scheme (NICNAS)
BCF - Bioconcentration Factor
NA - Not Applicable
NICNAS 2017 - National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia
DOE 2017 - Draft Risk Assessment Guidance Manual: For Chemicals Associated with Coal Seam Gas Extraction, Australian Government, Department of Energy

Toxicity and Dermal Absorption Parameters

C = calculated from chronic value, Ch = chronic value adopted

CAS#	Chemical	Oral/Dermal Exposures				Inhalation Exposures									
		Threshold Chronic TDI or RfD (mg/kg/day)		Dermal Permeability (cm/hr)	Reference	Inhalation Unit Risk $(ug/m^3)^{-1}$	Non-Threshold Slope Factor $(mg/kg/day)^{-1}$		Threshold Chronic TC or RfC (mg/m^3)		NOAEC or LOAEC (mg/m^3)	NOAEL or LOAEL (mg/kg bw/d)	Reference	UF	Reference
	COPC in Hydraulic Fracturing Fluid Injected into Well														
1319-33-1	Boronatrocalcite/Ulexite[A]	0.096	D	9.14E-04	EPI (as boric acid)				0.336	converted from RFD		9.6	NICNAS (2017)	100	NICNAS (2017)
68937-66-6	Alcohols, C6-12, ethoxylated propoxylated[B]	0.5	D	1.21E-04	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
69227-22-1	Alcohols, C10-16, ethoxylated propoxylated[B]	0.5	D	2.87E-01	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
64-19-7	Acetic acid	12	D	5.56E-04	EPI				42	converted from RFD		1200	NICNAS (2017)	100	NICNAS (2017)
25322-68-3	Polyethylene glycol	8	D	2.14E-06	EPI				28	converted from RFD		8000	REACH	1000	D
7631-90-5	Sodium bisulfite[C]	10.5	D	4.16E-09	EPI				36.75	converted from RFD		1050	NICNAS (2017)	100	NICNAS (2017)
104-55-2	Cinnamaldehyde	2	D	5.20E-03	EPI				7	converted from RFD		200	NTP (2004); REACH	100	D
67-56-1	Methanol	0.037	D	3.19E-04	EPI				0.13	converted from RFD		3.7	NICNAS (2017)	100	NICNAS (2017)
61788-90-7	Amine oxides, cocoalkyldimethyl	0.08	D	1.03E-01	EPI				0.28	converted from RFD		80	OECD (2001)	1000	D
100-52-7	Benzaldehyde	0.3	D	3.83E-03	EPI				1.05	converted from RFD		300	OECD (2002); REACH; NICNAS	1000	D
64-17-5	Ethanol	24	D	5.38E-04	EPI				84	converted from RFD		2400	NICNAS (2017)	100	NICNAS (2017)
64742-47-8	Hydrotreated light petroleum distillate	10	D	1.96E+00	EPI				35	converted from RFD		1000	NICNAS (2017)	100	NICNAS (2017)
61791-00-2	Fatty acids, tall-oil, ethoxylated	10	D	2.11E-02	EPI				35	converted from RFD		1000	REACH	100	D
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)	0.5	D	7.14E-02	EPI				1.75	converted from RFD		50	USEPA (2010)	100	D
71-36-3	Butyl alcohol	1.25	D	2.31E-03	EPI				4.375	converted from RFD		125	OECD (2001)/NICNAS	100	D
68131-39-5	Alcohols, C12-15, ethoxylated[B]	0.5	D	1.48E-03	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
68551-12-2	Alcohols, C12-16, ethoxylated[B]	0.5	D	8.97E-01	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
107-13-1	Acrylonitrile	0.0025	D	1.16E-03	EPI				0.00875	converted from RFD		0.25	OECD (2005); NICNAS	100	D
111-42-2	Diethanolamine	0.014	D	4.51E-05	EPI				0.049	converted from RFD		14	REACH; OECD (2002); NICNAS	1000	D
111-30-8	Glutaraldehyde	0.04	D	3.25E-04	EPI				0.14	converted from RFD		4	NICNAS (2017)	100	NICNAS (2017)
102-71-6	Triethanol amine	1.25	D	4.93E-05	EPI				4.375	converted from RFD		125	NICNAS (2017)	100	NICNAS (2017)
7758-19-2	Chlorous acid, sodium salt	0.039	D	8.27E-09	EPI				0.1365	converted from RFD		3.9	NICNAS (2017)	100	NICNAS (2017)
12008-41-2	Disodium octaborate tetrahydrate[A]	0.096	D	9.14E-04	EPI (as boric acid)				0.336	converted from RFD		9.6	NICNAS (2017)	100	NICNAS (2017)
7775-27-1	Sodium persulfate	0.67	D	7.05E-07	EPI				2.345	converted from RFD		67	NICNAS (2017)	100	NICNAS (2017)
68439-54-3	Ethoxylated branched C13 alcohol	0.5	D	1.06E-03	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
1338-43-8	Sobitan, mono-9-octadecenoate, (Z)	25	JECFA	5.02E-02	EPI				87.5	converted from RFD		-	JECFA(1973); US FDA; FSANZ (2018)	-	-
9005-65-6	Sorbitan monooleate polyoxyethylene derivative	10	EFSA	3.54E-09	EPI				35	converted from RFD		-	EFSA (2017)	-	-
111-46-6	2,2''-oxydiethanol (diethylene glycol)	0.3	D	4.17E-05	EPI				1.05	converted from RFD		300	Health Council of the Netherlands (2007); NICNAS	1000	D
7631-90-5	Sodium bisulfate[C]	10.5	D	9.29E-09	EPI				36.75	converted from RFD		1050	NICNAS (2017)	100	NICNAS (2017)
14808-60-7	Crystalline silica, quartz	Not toxic via oral/dermal exposure							0.003	USEPA (2019)		-	-	-	-
10486-00-7	Sodium perborate tetrahydrate	0.05	D	1.81E-06	EPI				0.175	converted from RFD		50	REACH	1000	D

Notes:

A - Read across data from Boric Acid

B - Read across data from Alcohol ethoxylates C6-C12

C - Read across data from Sodium Sulfite

References:

D - Derived (refer to individual Toxicity Profiles)

EFSA - European Food Safety Authority

EPI - USEPA Estimation Programs Interface (EPI) Suite

FSANZ - Food Standards Australia New Zealand

J- JECFA - Joint FAO/WHO Expert Committee on Food Additives

NICNAS - National Industrial Chemicals Notification and Assessment Scheme. Assessed at https://www.nicnas.gov.au/

NICNAS (2017) - Department of the Environment and Energy 2017 , National assessment of chemicals associatedwith coal seam gas extraction in Australia, prepared by the National Industrial ChemicalsNotification and Assessment Scheme

NTP - US Department of Health and Serices National Toxicology Program

OECD - Organisation for Economic Co-operation and Development Screening Information Data Set (SIDS)

REACH - ECHA REACH European Chemicals Agency Database: http://apps.echa.europa.eu

Exposure to Chemicals via Incidental Ingestion of Flowback fluid - SW Recipe

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Flowback Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Ingestion Rate (IRw)	L/day or L/hr	0.005	Assume Incidental ingestion of 5 ml (1 tsp) of water per day during fraccing.
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**4.2E-09**	**NonThreshold**
		3.5E-06	**Threshold**

Daily Intake from Water = Concentration in Water x Intake Factor (ref: USEPA 1989)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

CAS	Chemical	Toxicity Data				Concentration	Daily Intake		Calculated Risk	
		Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% Chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)	in Water	NonThreshold	Threshold	NonThreshold Risk	Chronic Hazard Quotient
		$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
68937-66-6	Alcohols, C6-12, ethoxylated propoxylatedB		5.0E-01		5.0E-01	344.58	1.4E-06	1.2E-03	--	2.4E-03
69227-22-1	Alcohols, C10-16, ethoxylated propoxylatedB		5.0E-01		5.0E-01	177.36	7.4E-07	6.2E-04	--	1.2E-03
64-19-7	Acetic acid		1.2E+01		1.2E+01	107.82	4.5E-07	3.8E-04	--	3.2E-05
25322-68-3	Polyethylene glycol		8.0E+00		8.0E+00	89.47	3.7E-07	3.1E-04	--	3.9E-05
7631-90-5	Sodium bisulfiteC		1.1E+01		1.1E+01	90.84	3.8E-07	3.2E-04	--	3.0E-05
104-55-2	Cinnamaldehyde		2.0E+00		2.0E+00	54.91	2.3E-07	1.9E-04	--	9.6E-05
111-46-6	2,2"-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01	29.63	1.2E-07	1.0E-04	--	3.5E-04
67-56-1	Methanol		3.7E-02		3.7E-02	20.75	8.7E-08	7.3E-05	--	2.0E-03
61788-90-7	Amine oxides, cocoalkyldimethyl		8.0E-02		8.0E-02	12.42	5.2E-08	4.4E-05	--	5.5E-04
100-52-7	Benzaldehyde		3.0E-01		3.0E-01	12.42	5.2E-08	4.4E-05	--	1.5E-04
64-17-5	Ethanol		2.4E+01		2.4E+01	9.27	3.9E-08	3.3E-05	--	1.4E-06
64742-47-8	Hydrotreated light petroleum distillate		1.0E+01		1.0E+01	8.69	3.6E-08	3.1E-05	--	3.1E-06
61791-00-2	Fatty acids, tall-oil, ethoxylated		1.0E+01		1.0E+01	8.89	3.7E-08	3.1E-05	--	3.1E-06
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)		5.0E-01		5.0E-01	4.03	1.7E-08	1.4E-05	--	2.8E-05
71-36-3	Butyl alcohol		1.3E+00		1.3E+00	3.36	1.4E-08	1.2E-05	--	9.4E-06
68131-39-5	Alcohols, C12-15, ethoxylatedB		5.0E-01		5.0E-01	3.21	1.3E-08	1.1E-05	--	2.3E-05
68551-12-2	Alcohols, C12-16, ethoxylatedB		5.0E-01		5.0E-01	2.39	1.0E-08	8.4E-06	--	1.7E-05
107-13-1	Acrylonitrile		2.5E-03		2.5E-03	1.29	5.4E-09	4.5E-06	--	1.8E-03
111-42-2	Diethanolamine		1.4E-02		1.4E-02	1.71	7.1E-09	6.0E-06	--	4.3E-04
111-30-8	Glutaraldehyde		4.0E-02		4.0E-02	0.85	3.6E-09	3.0E-06	--	7.5E-05
								Total Risk (mixture)		**9.19E-03**

Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

Dermal Exposure to Chemicals via Contact with Flow Back Water - SW Recipe

Chronic Exposures

General Data/ Equations		Units	Exposure Calculations (RME) Dermal Contact with Flow Back Water by Workers	
Exposure Parameters				
Exposure Frequency (EF)		days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)		years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)		kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)		days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)		days	30.42	USEPA 1989 and CSMS 1996
Surface Area (SAw)		cm^2	2300	Hands and forearms exposed (enHealth 2012) Occupational HSE would require long pants and closed shoes on Australian work sites; forearms conservatively included
Exposure Time (ET)		hr/day	1	Assume contact with flow back water for 1 hours per day
Conversion Factor (CF)		L/cm^3	1.E-03	Conversion of units
Intake Factor =	**SAw*ET*CF*EF*ED / BW*AT**	L-hr/(cm-kg-day)	**1.9E-06** **1.6E-03**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor (ref: USEPA 1989, 2004)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

Chemical		Toxicity Data				Concentration	Daily Intake		Calculated Risk	
		Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)	Dermal Permeability	in Water	NonThreshold	Threshold	NonThreshold Risk	Chronic Hazard Quotient
		$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
68937-66-6	Alcohols, C6-12, ethoxylated propoxylatedB		5.0E-01		5.0E-01	1.2E-4	344.58	8.0E-08	6.7E-05	--	1.3E-04
69227-22-1	Alcohols, C10-16, ethoxylated propoxylatedB		5.0E-01		5.0E-01	2.9E-1	177.36	9.8E-05	8.2E-02	--	1.6E-01
64-19-7	Acetic acid		1.2E+01		1.2E+01	5.6E-4	107.82	1.2E-07	9.7E-05	--	8.1E-06
25322-68-3	Polyethylene glycol		8.0E+00		8.0E+00	2.1E-6	89.47	3.7E-10	3.1E-07	--	3.9E-08
7631-90-5	Sodium bisulfiteC		1.1E+01		1.1E+01	4.2E-9	90.84	7.3E-13	6.1E-10	--	5.8E-11
104-55-2	Cinnamaldehyde		2.0E+00		2.0E+00	5.2E-3	54.91	5.5E-07	4.6E-04	--	2.3E-04
111-46-6	2,2"-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01	4.2E-5	29.63	2.4E-09	2.0E-06	--	6.7E-06
67-56-1	Methanol		3.7E-02		3.7E-02	3.2E-4	20.75	1.3E-08	1.1E-05	--	2.9E-04
61788-90-7	Amine oxides, cocoalkyldimethyl		8.0E-02		8.0E-02	1.0E-1	12.42	2.5E-06	2.1E-03	--	2.6E-02
100-52-7	Benzaldehyde		3.0E-01		3.0E-01	3.8E-3	12.42	9.1E-08	7.7E-05	--	2.6E-04
64-17-5	Ethanol		2.4E+01		2.4E+01	5.4E-4	9.27	9.6E-09	8.1E-06	--	3.4E-07
64742-47-8	Hydrotreated light petroleum distillate		1.0E+01		1.0E+01	2.0E+0	8.69	3.3E-05	2.8E-02	--	2.8E-03
61791-00-2	Fatty acids, tall-oil, ethoxylated		1.0E+01		1.0E+01	2.1E-2	8.89	3.6E-07	3.0E-04	--	3.0E-05
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)		5.0E-01		5.0E-01	7.1E-2	4.03	5.5E-07	4.7E-04	--	9.3E-04
71-36-3	Butyl alcohol		1.3E+00		1.3E+00	2.3E-3	3.36	1.5E-08	1.3E-05	--	1.0E-05
68131-39-5	Alcohols, C12-15, ethoxylatedB		5.0E-01		5.0E-01	1.5E-3	3.21	9.1E-09	7.7E-06	--	1.5E-05
68551-12-2	Alcohols, C12-16, ethoxylatedB		5.0E-01		5.0E-01	9.0E-1	2.39	4.1E-06	3.5E-03	--	6.9E-03
107-13-1	Acrylonitrile		2.5E-03		2.5E-03	1.2E-3	1.29	2.9E-09	2.4E-06	--	9.7E-04
111-42-2	Diethanolamine		1.4E-02		1.4E-02	4.5E-5	1.71	1.5E-10	1.2E-07	--	8.9E-06
111-30-8	Glutaraldehyde		4.0E-02		4.0E-02	3.3E-4	0.85	5.3E-10	4.5E-07	--	1.1E-05
									Total Risk (mixture)		**2.0E-1**

Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

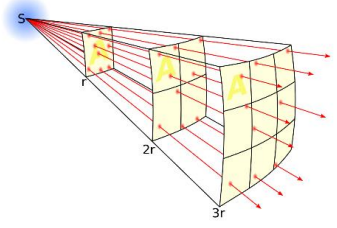
Aerosol Exposure - SW Recipe

The concentration of COPC in aerosol spray was estimated by calculating the concentration for driftable droplets using a mixed box model in which steady state concentrations were calculated. The 'inverse square law' was then applied to approximate the air concentration at a distance from the virtual air box. This law assumes that the density of the spray droplets is inversely proportional to the square of the distance from the source. That is, the further away a receptor is from the spray source, the density of the droplets (and therefore the concentration) will decrease.

An emission factor for driftable aerosol was estimated using the algorithm presented below.

Emission Factor for Driftable Aerosol Algorithm

$$Emission\ Factor_{driftable\ aerosol}\left(\frac{L}{m^3}\right) = \frac{Box\ Centre^2(m) \times \left(\frac{Spray\ Vol\ \left(\frac{L}{hr}\right) \times Aerosol_{driftable}(\%)}{BoxVR\ \left(\frac{m^3}{hr}\right)}\right)}{BoxDistance^2\ (m)}$$



Aerosol Exposure Modelling Notes:

1) The inhalation of chemicals in mist/aerosol resultant from irrigation activities is dependent upon the concentration in water, the amount of water used per unit time, how close a person stands to the spray generation, how long they are in a position of exposure and the extent of spray drift (determined by the size of the water droplets and speed/direction of the wind). These equations are applicable for non-volatile contaminants that are inhaled.
2) These equations calculate the concentration for driftable droplets using a simple well mixed box model in which steady state air concentrations are calculated. The 'Inverse square law' is then applied to approximate the air concentration at a distance from the virtual air box. This law assumes the further away a receptor is from the spray source, the density of the droplets will decrease. The density of the spray droplets is inversely proportional to the square of the distance from the source.

Parameter	Units	Value	Description
Spray box length	m	3	Assume a 'spray box' of 3 m long.
Spray box width	m	3	Assume a 'spray box' of 3 m wide.
Box Centre	m	1.5	Distance to centre of box is 1.5 m.
$Box_{Distance}$	m	2	Distance the irrigation worker is from the 'spray box'. Assumed a distance of 2 m.
$Aerosol_{driftable}$	unitless	0.2	Proportion of aerosol spray that drifts outside the 'spray box' and available for exposure. Assumed 0.2, based on a droplet size of 400 – 500 µm that falls approximately 0.3 m in less than 10 seconds, with a lateral drift of approximately 3.5 m in a 5 km/hr wind (i.e. a light breeze) (Grisso et al. 2013).
Spray Volume	L/hr	1800.0	1800 L/min, irrigation value adopted from NZ MtE (2011) Appendix 5A.
Wind speed	m/hr	9000	Based on windspeed of 2.5 m/sec
BoxVR	m^3/hr	81000.0	Ventilation rate of spray in the 'spray box'. Assumed to be 81,000 m3/hr based on a wind speed of 9000 m/hr, and a 'spray box' dimension of 3 x 3 m.

CAS	Chemical	Concentration in Water	Generation rate of chemical in volume	Driftable Aerosol Emission Factor
		mg/L	mg/hr	L/m^3
68937-66-6	Alcohols, C6-12, ethoxylated propoxyla	344.58	124049.7383	2.500000E-03
69227-22-1	Alcohols, C10-16, ethoxylated propoxyl	177.36	63849.13002	2.500000E-03
64-19-7	Acetic acid	107.82	38815.27443	2.500000E-03
25322-68-3	Polyethylene glycol	89.47	32209.40603	2.500000E-03
7631-90-5	Sodium bisulfiteC	90.84	32700.84622	2.500000E-03
104-55-2	Cinnamaldehyde	54.91	19768.14867	2.500000E-03
111-46-6	2,2"-oxydiethanol (diethylene glycol)	29.63	10665.31447	2.500000E-03
67-56-1	Methanol	20.75	7469.078692	2.500000E-03
61788-90-7	Amine oxides, cocoalkyldimethyl	12.42	4472.513177	2.500000E-03
100-52-7	Benzaldehyde	12.42	4470.713715	2.500000E-03
64-17-5	Ethanol	9.27	3338.494405	2.500000E-03
64742-47-8	Hydrotreated light petroleum distillate	8.69	3130.000401	2.500000E-03
61791-00-2	Fatty acids, tall-oil, ethoxylated	8.89	3201.205801	2.500000E-03
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyetl	4.03	1452.129696	2.500000E-03
71-36-3	Butyl alcohol	3.36	1210.554971	2.500000E-03
68131-39-5	Alcohols, C12-15, ethoxylatedB	3.21	1154.770361	2.500000E-03
68551-12-2	Alcohols, C12-16, ethoxylatedB	2.39	861.0898092	2.500000E-03
107-13-1	Acrylonitrile	1.29	464.4655534	2.500000E-03
111-42-2	Diethanolamine	1.71	615.1041839	2.500000E-03
111-30-8	Glutaraldehyde	0.85	306.4351619	2.500000E-03

Exposure to Chemicals via Inhalation of Mist from the Evaporation Units - SW Recipe

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Inhalation of Mist by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays
Exposure Duration (ED)	years	1	Maximum duration that the flowback tank will be on-site
Exposure Time (ET)	hr/day	1	Professional judgement for irrigation exposure. Assume worker to be near tank for 1 hours every working day.
Driftable aerosol emission factor (EMF)	L/m3	2.50E-03	Calculated
Aerosol Inhalation Bioavailability (AAF)	unitless	1.0	Assume 100% bioavailability
Averaging Time - Threshold (AT)	years	1.0	USEPA 1989 and CSMS 1996
$ITF_{inh,w,shwr} = \frac{EmF \times AAF \times ET_{iw} \times EF \times ED}{365\ \frac{days}{year} \times 24\ \frac{hours}{day} \times AT}$			

Daily Intake = Concentration in Water x Intake Factor (ref: USEPA 1989)
Hazard Quotients = (Daily Intake from Aerosol for Threshold Effects/ADI)

CAS	Chemical	Concentration in Water	Aerosol Inhalation Bioavailability	Driftable Aerosol Emission Factor	RfC (Background Corrected)	Threshold Intake and Risk Calculations: Adult Exposure Factor (threshold)	Adult Exposure Adjusted Air Concentration (threshold)	Hazard Quotient (Adult)
		mg/L	(unitless)	(L/m^3)	(mg/m^3)	(L/m^3)	(mg/m^3)	(unitless)
68937-66-6	Alcohols, C6-12, ethoxylated propoxylatedB	3.45E+02	1.00	2.50E-03	1.75E+00	6.85E-05	2.36E-02	1.35E-02
69227-22-1	Alcohols, C10-16, ethoxylated propoxylatedB	1.77E+02	1.00	2.50E-03	1.75E+00	6.85E-05	1.21E-02	6.94E-03
64-19-7	Acetic acid	1.08E+02	1.00	2.50E-03	4.20E+01	6.85E-05	7.38E-03	1.76E-04
25322-68-3	Polyethylene glycol	8.95E+01	1.00	2.50E-03	2.80E+01	6.85E-05	6.13E-03	2.19E-04
7631-90-5	Sodium bisulfiteC	9.08E+01	1.00	2.50E-03	3.68E+01	6.85E-05	6.22E-03	1.69E-04
104-55-2	Cinnamaldehyde	5.49E+01	1.00	2.50E-03	7.00E+00	6.85E-05	3.76E-03	5.37E-04
111-46-6	2,2''-oxydiethanol (diethylene glycol)	2.96E+01	1.00	2.50E-03	1.05E+00	6.85E-05	2.03E-03	1.93E-03
67-56-1	Methanol	2.07E+01	1.00	2.50E-03	1.30E-01	6.85E-05	1.42E-03	1.09E-02
61788-90-7	Amine oxides, cocoalkyldimethyl	1.24E+01	1.00	2.50E-03	2.80E-01	6.85E-05	8.51E-04	3.04E-03
100-52-7	Benzaldehyde	1.24E+01	1.00	2.50E-03	1.05E+00	6.85E-05	8.51E-04	8.10E-04
64-17-5	Ethanol	9.27E+00	1.00	2.50E-03	8.40E+01	6.85E-05	6.35E-04	7.56E-06
64742-47-8	Hydrotreated light petroleum distillate	8.69E+00	1.00	2.50E-03	3.50E+01	6.85E-05	5.96E-04	1.70E-05
61791-00-2	Fatty acids, tall-oil, ethoxylated	8.89E+00	1.00	2.50E-03	3.50E+01	6.85E-05	6.09E-04	1.74E-05
68155-20-4	Amides, tall-oil fatty, N,N-bis(hydroxyethyl)	4.03E+00	1.00	2.50E-03	1.75E+00	6.85E-05	2.76E-04	1.58E-04
71-36-3	Butyl alcohol	3.36E+00	1.00	2.50E-03	4.38E+00	6.85E-05	2.30E-04	5.26E-05
68131-39-5	Alcohols, C12-15, ethoxylatedB	3.21E+00	1.00	2.50E-03	1.75E+00	6.85E-05	2.20E-04	1.26E-04
68551-12-2	Alcohols, C12-16, ethoxylatedB	2.39E+00	1.00	2.50E-03	1.75E+00	6.85E-05	1.64E-04	9.36E-05
107-13-1	Acrylonitrile	1.29E+00	1.00	2.50E-03	8.75E-03	6.85E-05	8.84E-05	1.01E-02
111-42-2	Diethanolamine	1.71E+00	1.00	2.50E-03	4.90E-02	6.85E-05	1.17E-04	2.39E-03
111-30-8	Glutaraldehyde	8.51E-01	1.00	2.50E-03	1.40E-01	6.85E-05	5.83E-05	4.16E-04
							Total Risk (mixture)	**0.05**

Summary of Risk to Workers - SW Recipe
Exposure fo Target Chemicals - Theoretical Data

Receptor/Exposure Pathway	Calculated HI 100% Mass Return
Use of Stimulation Fluid in Hydraulic Fracturing	
SW Recipe	
Workers	
Ingestion of Chemicals via Incidental Contact with Flowback Water	0.01
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water	0.20
Inhalation of mist from the evaporation units	0.05
Total Risk	**0.26**

Appendix D

Chemical Risk Assessment – Drilling Fluid

Chemical Name	CAS Number	Concentration in Injected Fluid (mg/L)	Ecotoxicity[1]	Toxicity[2]	Biodegradation[1,3]	Bioaccummulative[1]	Tier 1 Screening Assessment	Tier 2 Assessment Worker Ingestion Risk	Tier 2 Assessment Worker Dermal Risk	Tier 2 Assessment Worker Aerosol Inhalation Risk	Hazard Quotient	Outcome of Tier 2 Worker Risk Assessment[1]
Alcohol, C11-14, ethoxylated	78330-21-9	1.5	96 h LC50, Oncorhynchus mykiss = 5 - 7 mg/L 30 d Lepomis macrochirus, NOEC = 0.11 – 0.33 mg/L. 48 h EC50 Daphnia magna = 2.5 mg/L. 21 d NOEC Daphnia magna = 0.77 – 1.75 mg/L. 96 h EC50 (green algae) = 1.4 mg/L EC50 (3 h) for microorganisms = 140 mg/L	Based on chronic: High	Readily biodegradable	Not bioaccumulative	Tier 2	1.1E-05	6.16E-03	2.9E-07	6.2E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Proprietary	Proprietary	Proprietary	Proprietary	Proprietary	Proprietary	Proprietary	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Performatrol	Proprietary	Proprietary	Proprietary	Proprietary	Proprietary	Proprietary	Tier 1	NA	NA	NA	NA	NA
Citric Acid, monohydrate	77-92-9	1	96 h LC50 fish = 440 to 1,516 mg/L 24 h EC50 value for invertebrates is 85 mg/L 7 d toxic limit concentration values for algae = 300 to 640 mg/L 8 d freshwater static test for the algae Scenedesmus quadricauda, NOEC = 425 mg/L	Based on chronic: Low	Expected to be readily biodegradable	Not bioaccumulative	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Distillates, hydrotreated light	64742-47-8	1.5	Lowest acute endpoint for Daphnia = 0.018 mg/L (modelled)	Based on acute: Very high	Readily biodegradable	Yes based on calculated log BCF values for constituents that range from 2.78 to 4.06, and calculated BCF values of 598 to 11,430 L/kg wet-weight	Tier 2	5.3E-07	4.75E-04	2.9E-06	4.8E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Glutaraldehyde	111-30-8	0.3	96 h acute Bluegill sunfish LC50 = 11.2 mg/L 48 h acute Oyster larvae LC550 = 2.1 mg/L 96 h acute Green crabs LC50 = 465 mg/L 96 h acute Grass shrimp LC50 = 41 mg/L 48 acute Daphnia magna LC50 = 0.35 mg/L 48 acute Daphnia magna LC50 = 16.3 mg/L 21 d reproduct'n Daphnia magna LOEC = 4.3 mg/L, NOEC = 2.1 mg/L 96 h algal growth inhibition Selenastrum capricornutum ILm = 3.9 mg/L (median inhibitory limit) 96 h algal growth inhibition Scenedesmus subspicatus EC50 = 1.0 mg/L Bacterial inhibition Sewage microbes IC50 = 25-34 mg/L	Based on chronic: Moderate	Readily biodegradable	No based on the Log Pow of -0.01	Tier 2	2.6E-05	3.94E-06	1.5E-04	1.8E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Glyoxal <1%	107-22-2	2.2	96 h-LC50 fish = 215 mg/L Invertebrates EC50 > 100 mg/L NOEC fish = 119 mg/L (a.i.)	Based on chronic: Low	Expected to be readily biodegradable	Not bioaccumulative	Tier 2	5.8E-05	1.57E-06	3.2E-04	3.8E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Methanol	67-56-1	0.3	Acute LC50s = 15,400 to 29,400 mg/L Invertebrates, chronic NOEC = 32,000 mg/L.	Based on acute: Low	Readily biodegradable	No based on the Log Kow of -0.74	Tier 2	2.8E-05	4.16E-06	1.6E-04	1.9E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Nitrilotriacetic acid, trisodium salt monohydrate	5064-31-3	1	Fish 96 h LC50 = 98 - 487 mg/L Fish NOEC = 54 mg/L Invertebrates NOEC = 9.3 mg/L	Based on chronic: Moderate	Readily biodegradable	No based on the Log Pow of -13.2	Tier 2	3.5E-04	1.83E-12	2.0E-03	2.3E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Plagioclase Feldspar/Kaolinite	1332-58-7	10	Daphnia pulex (water flea) 24- and 48-h LC50 >1.1 g/L P. trilineatum 12-h LC50 = 170 mg/L O. fasciatus 12-h LC50 = 710 mg/L	Based on acute: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 1	NA	NA	NA	NA	NA
Poly Anionic Cellulose	9004-32-4	1.5	96 h LC50 for Brachydanio rerio is >2,500 mg/L 48 h LC50 for Daphnia magna is >5,000 mg/L 96 h EC50 for Selenastrum capricornutum is 500 mg/L	Based on acute: Low	Not readily biodegradable	Not bioaccumulative	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Potassium Chloride	7447-40-7	18	96 h LC50 in Pimephales promelas = 880 mg/L 48 h LC50 Lepomis macrochirus, Oncorhyncusmykiss and Ictalurus punctatus = 720 - 2010 mg/L	Based on chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Potassium Hydroxide	1310-58-3	0.3	96-hour fish LC50 value = 80 mg/L 48-hr invertebrate EC50 value = 40 mg/L 120-hr algae EC50 value = 1337 mg/L	Based on acute: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 1	NA	NA	NA	NA	NA. Acute toxicity only (irritant and corrosive), not systemically available in body
Quartz/Cristobite	14808-60-7	10	acute data >10 g/L	Based on acute: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	NA. Not toxic via oral exposure as not absorbed via GI tract	NA. Not toxic via dermal exposure.	2.3E-01	2.3E-01	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Smectite	12199-37-0	10	96 hr Oncorhynchus mykiss (Rainbow Trout) LC50 = 19000 mg/L	Based on acute: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 1	NA	NA	NA	NA	NA
Sodium Bicarbonate	144-55-8	0.5	21 d Daphnia NOEC = 576 mg/L	Based on chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Sodium Carbonate	497-19-8	0.29	96-hour LC50 Bluegill sunfish (Lepomis macrochirus) = 300 mg/L 96-hour LC50 to mosquitofish (Gambusia affinis) = 740 mg/L 48-hour EC50 to the invertebrate Ceriodaphnia cf. dubia = 200 to 227 mg/L	Based on acute: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	1.1E-05	1.49E-10	5.9E-05	6.9E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sodium Chloride	7647-14-5	17.61	acute endpoint for Fish = 1290 mg/L NOEC for Daphnia = 314 mg/L	Based on chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Sodium erythorbate	6381-77-7	0.2	96 h LC50 Fish > 100 mg/L 48 h EC50 Daphnia magna = 84 - 100 mg/L 72 h NOEC alga = 20 mg/L	Based on acute: Low	Not readily biodegradable	Not bioaccumulative	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Sodium hydroxide	1310-73-2	0.3	Measured acute endpoints for fish = 196 mg/L Measured chronic endpoint for Daphnia = 240 mg/L	Based on acute: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 1	NA	NA	NA	NA	NA. Acute toxicity only (irritant and corrosive), not systemically available in body
Starch	9005-25-8	4	Crassostrea virginica 96 h = 1000 mg/L Orthopristis chrysoptera 96 h = 5000 mg/L Bairdiella chrysoura 96 h = 5000 mg/L	Based on acute: Low	Expected to be readily biodegradable	Not bioaccumulative	Tier 1	NA	NA	NA	NA	NA
Tetrahydro-3,5-dimethyl-1,3,5-thiadiazine-2-thione	533-74-4	4	Daphnia magna (Water flea), 48 h, static, EC50 = 0.3 mg/L Salmo gairdneri (Rainbow trout), 96 h, static, LC50 = 0.16 mg/L Ankistrodesmus bribaianus (Green alga), 72 h, static, EC50 = 1.08 mg/L Colinus virginianus (Bobwhite quail), 21 d, LD50 = 415 mg/kg bw Colinus virginianus (Bobwhite quail), 25 weeks, NOEL = 100 mg/kg food	Based on acute: Very high	Expected to be readily biodegradable	Not bioaccumulative	Tier 2	2.8E-03	6.53E-04	1.6E-02	1.9E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Xanthan Gum	11138-66-2	1.5	Acute Fish (measured) = 420 mg/L	Based on acute: Low	Expected to be readily biodegradable	Not bioaccumulative	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Guanidine, hydrochloride (1:1)	50-01-1	7	96-h-LC50 for fish = 690 mg/L NOEC for fish = 181 mg/L EC50 for Daphnia = 70.2 mg/L NOEC for Daphnia = 2.9 mg/L EC50 for algae = 33.5 mg/L NOEC for algae = 6.3 mg/L	Based on chronic: Moderate	Expected to be readily biodegradable	Not bioaccumulative	Tier 2	2.5E-04	4.37E-09	1.4E-06	2.5E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Polyacrylamide	38193-60-1	1.5	LC50 = 357 mg/L (fish) LC50 = 212 mg/L (invertebrates) EC 50 = >1000 mg/L (algae)	Based on acute: Low	Not readily biodegradable	Not bioaccumulative	Tier 1	NA	NA	NA	NA	NA
Calcium Carbonate	1317-65-3	15	96h EC50 for fish >100mg/L 48 h EC50 for Daphnia >100 mg/L 72 h ERC50 for algae >14 mg/L	Based on acute: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Barite	13462-86-7	0.12	Long-term toxicity data are available for three trophic levels: 33 days NOEC: 1.26 mg/L (Fish) 21 days NOEC: 2.9 mg/L (Invertebrates) 72 hrs NOEC: 1.15 mg/L (Algae)	Based on chronic: Moderate	N.A.(Inorganic)	N.A. (Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Triazine based biocide C572,2',2''-(hexahydro-1,3, 5-triazine-1,3,5-triyl) triethanol	4719-04-4	0.00101	LC50 for fish 240.04 mg/L LC50 for invertebrates 60.67 mg/L EC50 for freshwater algae: 6.6 mg/L	Based on acute: High	Expected to be readily biodegradable.	Not bioaccumulative	Tier 2	5.5E-08	1.86E-12	3.1E-07	3.6E-07	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Ammonium hydrogensulfite	10192-30-0	0.00071	Algae NOEC/EC10 = 28 mg SO32-/L Invertebrates NOEC/EC10 = ≥8.41 mg SO32-/L Fish NOEC/EC10 = 50 mg SO32-/L	Based on chronic: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 2	2.2E-08	6.36E-13	1.2E-07	1.5E-07	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Sulphur Dioxide (Impurity)	7446-09-5	0.00071	Sulfur dioxide is not present as a substance. It is formed during decomposition. Sulphur dioxide is a gaseous substance and does not remain present in the aquatic environment.	NA	NA	NA	NA	NA	NA	NA	NA	NA
Partially hydrolysed polyacrylamide	9003-05-8	0.00117	Fathead minnow LC50: 810 mg/L Rainbow trout LC50: > 100 mg/L Bluegill sunfish LC50: >300 mg/L Daphnia magna LC50: 470 mg/L	Based on acute: Low	Not readily biodegradable	Not bioaccumulative	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Polyanionic cellulose, low viscosity	9004-32-4	0.00338	Brachydanio rerio 96-hour LC50 >2,500 mg/L Daphnia magna 48-hour EC50 >5,000 mg/L Daphnia magna 48-hour EC50 87.26 mg/L Selenastrum capricornutum 96-hour EC50 500 mg/L	Based on acute: Low	Not readily biodegradable	Not bioaccumulative	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Barium sulphate	7727-43-7	0.08743	Long-term toxicity data are available for three trophic levels: 33 days NOEC: 1.26 mg/L (Fish) 21 days NOEC: 2.9 mg/L (Invertebrates) 72 hrs NOEC: 1.15 mg/L (Algae)	Based on chronic: Moderate	N.A.(Inorganic)	N.A. (Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Filming amine Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivs. Residues	68909-77-3	0.005	LC50 (96 h) for fish: 681.2 mg/L EC50 for daphnids: > 122 mg/L ErC50(72h) for algae: 45 mg/L	Based on acute: Moderate	Not readily biodegradable	Not bioaccumulative	Tier 2	1.8E-08	1.11E-11	9.8E-08	1.2E-07	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Distillates (Fischer-Tropsch), C8-26 - Branched and Linear	848301-67-7	0.0000001	Short-term toxicity: NOEC (48 h): 1000 mg/L (fish) LC50 (7 day): >100000 mg/L (fish) EL50 (72 h): >1000 mg/L (invertebrates) EL50 (48 h): 1000 mg/L (crustaceans) EL50 (72 h): 1000 mg/L (algae) Long-term toxicity: NOEL (33 day): >100 mg/L (fish) NOEL (21 day): <100 mg/L (invertebrates)	Based on acute: Low	Expected to be readily biodegradable	No. Based on log BCF of 3.17 or BCF of 1479.	Tier 2	1.8E-12	1.10E-09	9.8E-12	1.1E-09	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine	68990-47-6	0.007	Short term toxicity: LC50 (4 days): 100 mg/L (fish) NOEC (4 days): 100 mg/L (fish) LOEC (4 days): 100 mg/L (fish) IC50 (48 h): 100 mg/L (invertebrates)	Based on acute: Low	Not readily biodegradable	Yes. Based on the estimated Log Kow of 11 (Log Kow > 4.2).	Tier 2	2.5E-08	1.14E-08	1.4E-07	1.7E-07	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
(2-methoxymethylethoxy)propanol	34590-94-8	0.007	Short term toxicity data: EC50s/LC50s >1000 mg/l in daphnia (48 hr), fish (96 hr) and algae (7 days). Long term toxicity data: NOEC: 0.5 mg/L (daphnia)	Based on chronic: High	Expected to be readily biodegradable	Not bioaccumulative. Based on the Log Kow of 0.004 at 25 °C (Log Kow < 4.2).	Tier 2	2.5E-08	1.54E-09	1.4E-07	1.6E-07	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).

Chemical Name	CAS Number	Concentration in Injected Fluid (mg/L)	Ecotoxicity[1]	Toxicity[2]	Biodegradation[1,3]	Bioaccumulative[1]	Tier 1 Screening Assessment	Tier 2 Assessment Worker Ingestion Risk	Tier 2 Assessment Worker Dermal Risk	Tier 2 Assessment Worker Aerosol Inhalation Risk	Hazard Quotient	Outcome of Tier 2 Worker Risk Assessment[1]
1-tetradecene	1120-36-1	0.0000001	Short term toxicity: LC50 (4 days): 3.4 µg/L (fish) EC50 (48 h): 2.8 µg/L (invertebrates) EC50 (4 days): 4.5 µg/L (algae)	Based on chronic: Low	Expected to be readily biodegradable.	Yes. Based on the estimated Log Kow of 7.3 (Log Kow > 4.2)	Tier 2	3.5E-12	1.02E-08	2.0E-11	1.0E-08	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Amides, tall oil fatty N,N-bis (hydroxyethyl)	68155-20-4	0.0000001	Based on read across: Daphnia: EC50 (24-hour): 3.3 mg active matter/l Daphnia: 48-hour LC50 = 2.15 and 2.64 mg/l 21 d NOEC = 0.08 mg/L	Based on chronic: Very high	Expected to be readily biodegradable.	Not bioaccumulative. Based on BAF = 108 and log Kow of 3 (estimated)	Tier 2	4.7E-13	1.54E-11	2.6E-12	1.8E-11	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Fatty acids, tall-oil, reaction products with polyethylenepolyamines	68910-93-0	0.0000001	Short term toxicity data: 96h-LL50 > 100 mg/L (fish) 48h-EL50 = 12.41 mg/L (invertebrates) 72h-EL50 = 39.7 mg/L (algae)	Based on acute: Moderate	Expected to be readily biodegradable.	Not bioaccumulative	Tier 2	3.5E-13	1.63E-13	2.0E-12	2.5E-12	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Phosphoric ester of ethoxylated fatty alcohol	68585-36-4	0.0000001	Short term toxicity data: 96h-LL50 > 100 mg/L (fish) 48h-EL50 = 12.41 mg/L (invertebrates) 72h-EL50 = 39.7 mg/L (algae)	Based on acute: Moderate	Expected to be readily biodegradable.	Not bioaccumulative	Tier 2	3.5E-13	1.63E-13	2.0E-12	2.5E-12	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Hexadec-1-ene	629-73-2	0.0000001	Short term toxicity 96-hr LC50 > solubility Actual concentration negligible. Fish 96-hr LL0 = 1000 mg/L (nominal)	Based on chronic: Very high	Expected to be readily biodegradable.	Not bioaccumulative	Tier 2	3.5E-12	3.18E-08	2.0E-11	3.2E-08	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Distillates (petroleum), hydrotreated heavy naphthenic	64742-52-5	0.0000001	Short term toxicity data: LL50 was > 100 mg/L (fish) EL50 was >10,000 mg/L (invertebrates) Long term toxicity data: 21 day NOEL: 10 mg/L (invertebrates)	Based on chronic: Low	Not readily biodegradable	Yes. Calculated BCF for constituents of this substance range between 0.4 and 71100 L/kg	Tier 2	4.4E-13	5.09E-08	2.4E-12	5.1E-08	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Lead	7439-92-1	0.0000001	Short-term toxicity data: LC50 (96 h) 40.8 µg/L (Fish) LC50 (48 h) 26 µg/L (Invertebrates) EC50 (72 h) 20.5 µg/L (algae)	Based on chronic: Very high	N.A.(Inorganic)	N.A. (Inorganic)	Tier 1 (below ADWG and ANZECC)	NA	NA	NA	NA	NA
Graphite	7782-42-5	0.0000001	The short-term toxicity: LC50 > 100 mg/L for the LC50 and NOEC > 100 mg/L (fish) EC50 > 100 mg/L for the EC50 and NOEC > 100 mg/L for the NOEC (daphnids)	Based on acute: Low	N.A.(Inorganic)	N.A. (Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Talc	14807-96-6	0.0000001	No data	Based on low bioavailability: Low	Not readily biodegradable	Not bioaccumulative	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Mineral oil	8042-47-5	0.0000001	Rainbow trout 96 hr LL50 (48 h) 100 mg/L	Based on acute: Low	N.A.(UVCB)	No. Not readily biodegradable based on read across study.	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Copper	7440-50-8	0.0000001	Fish: 2.6 µg/L (Ptylocheilus oregonensis, from 7-day LC50) 131 µg/L (Pimephales promelas, 7-day LC50) Crustaceans: 1.7 µg/L (D. pulex and G. pulex, NOEC, reproduction & mortality) 12.1 µg/L (Hyalella azteca, from 10 to 14-day LC50).	Based on chronic: Very high	N.A. (Inorganic)	N.A. (Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Zinc	7440-66-6	0.0000001	Fish: 24 µg/L (Oncorhynchus tshawytscha; from LC50) Amphibians: Ambystoma opacum, 180 µg/L (from LOEC) Crustaceans: 5.5 µg/L (C. dubia; from LC50)	Based on acute: Very high	N.A. (Inorganic)	N.A. (Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Calcium oxide	1305-78-8	0.0000001	Oncorhynchus mykiss 96-hour LC50: 50.6 mg/L Daphnia magna 48-hour EC50: 49.1 mg/L Pseudokirchneriella subcapitata 72 hour EC10: 79.22 mg/L Crangon septemspinosa 14-day: EC10 of 32 mg/L	Based on acute: Moderate	N.A. (Inorganic)	N.A. (Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Distillates (petroleum), hydrotreated light naphthenic < 3% DMS	64742-53-6	0.0000001	Short term toxicity data: LL50 was > 100 mg/L (fish) EL50 was >10,000 mg/L (invertebrates) Long term toxicity data: 21 day NOEL: 10 mg/L (invertebrates)	Based on chronic: Low	Not readily biodegradable	Yes. Calculated BCF for constituents of this substance range between 0.4 and 71100 L/kg	Tier 2	4.4E-13	3.96E-10	2.4E-12	4.0E-10	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Aluminum not powder, dust or fume	7429-90-5	0.0000001	8-day LC50 0.17 mg/L (fish) 8-day LC50 of 2.28 mg/L (amphibian)	Based on chronic: High	N.A. (Inorganic)	N.A. (Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Distillates (petroleum), straight-run middle	64741-44-2	0.0000001	96h LL50 21 mg/L (fish) NOEL: 0.068 mg/L (fish) 48h EL50 68 mg/L (daphnia) 21 d NOEL: 0.167 mg/L (daphnia) 72 h ErL50: 22 mg/L (algae)	Based on chronic: High	Expected to be readily biodegradable.	Yes. Log Kow values in the range 3.9 to greater than 6.	Tier 2	1.2E-11	7.32E-09	6.5E-11	7.4E-09	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Bitumen	8052-42-4	0.0000001	Short term toxicity: LL50 (4 days): 1 g/L (fish) LL50 (48 h): 1 g/L (invertebrates) EL50 (72 h): 1 g/L (algae) Long term toxicity: LL50 (28 days): 1 g/L (fish)	Based on chronic: Low	Expected to be readily biodegradable.	N.A. (UVCB)	Tier 2	1.8E-12	0.00E+00	9.8E-12	1.2E-11	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Copper (II) Oxide	1317-38-0	0.0000001	Fish: 2.6 µg/L (Ptylocheilus oregonensis, from 7-day LC50) 131 µg/L (Pimephales promelas, 7-day LC50) Crustaceans: 1.7 µg/L (D. pulex and G. pulex, NOEC, reproduction & mortality) 12.1 µg/L (Hyalella azteca, from 10 to 14-day LC50).	Based on chronic: Very high	N.A. (Inorganic)	N.A. (Inorganic)	Tier 1 (NICNAS)	NA	NA	NA	NA	NA
Phosphorodithioic acid, mixed o,o-bis(iso-bu and pentyl) esters,	68457-79-4	0.0000001	Short term toxicity: LC50 (4 days): 46 mg/L (fish) LL50 (4 days): 4.5 mg/L (fish) EL50 (48 h): 23 mg/L (invertebrates) EL50 (72 h): 21 mg/L (algae) Long term toxicity: NOEC (21 days): 0.4 mg/L (invertebrates)	Based on chronic: High	Not readily biodegradable	Not bioaccumulative. Based on the measured log Kow value of less than 3.	Tier 2	2.2E-12	3.10E-09	1.2E-11	3.1E-09	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
Tetrasodium ethylenediaminetetraacetate	64-02-8	0.0000001	Danio rerio: 35 d-NOEC > 26.8 mg/L Daphnia magna: 21d-NOEC = 22 mg/L; Scenedesmus subspicatus: 72h-EC10 = > 100 mg/L. For Na2EDTA, Daphnia magna; 21d-NOEC = 25 mg/L.	Based on chronic: Low	Not readily biodegradable	Not bioaccumulative	Tier 1 (NICNAS)	NA	NA	NA	NA	NA

Notes
1 - Please refer to the individual toxicity profiles for further detail.
2 - Toxicity assessed using NT (2021)
3 - Biodegradation assessed as per NT (2021) and DoEE (2017)
BCF - Bioconcentration Factor
NA - Not Applicable
MOE - Margin of Exposure
NICNAS 2017 - National Assessment of Chemicals Associated with Coal Seam Gas Extraction in Australia
DoEE 2017 - Draft Risk Assessment Guidance Manual: For Chemicals Associated with Coal Seam Gas Extraction, Australian Government, Department of Energy
NT 2021 - Northern Territory Government, Department of Environment, Parks and Water Security, Environment Management Plan Content Guideline, 2021

Toxicity and Dermal Absorption Parameters

C = calculated from chronic value, Ch = chronic value adopted

CAS#	Chemical	Oral/Dermal Exposures				Inhalation Exposures								
		Threshold Chronic TDI or RfD (mg/kg/day)		Dermal Permeability (cm/hr)	Reference	Inhalation Unit Risk $(ug/m^3)^{-1}$	Non-Threshold Slope Factor $(mg/kg/day)^{-1}$		Threshold Chronic TC or RfC (mg/m^3)		NOAEC or LOAEC (mg/m^3)	NOAEL or LOAEL (mg/kg bw/d)	Reference	UF	Reference
	COPC in Hydraulic Fracturing Fluid Injected into Well														
67-56-1	Methanol	0.037	D	3.19E-04	EPI				0.13	converted from RFD		3.7	NICNAS (2017)	100	NICNAS (2017)
64742-47-8	Hydrotreated light petroleum distillate	10	D	1.96E+00	EPI				35	converted from RFD		1000	NICNAS (2017)	100	NICNAS (2017)
111-30-8	Glutaraldehyde	0.04	D	3.25E-04	EPI				0.14	converted from RFD		4	NICNAS (2017)	100	NICNAS (2017)
14808-60-7	Crystalline silica, quartz	Not toxic via oral/dermal exposure							0.003	USEPA (2019)		-			
78330-21-9	Alcohol, C11-14, ethoxylated[B]	0.5	D	1.27E+00	EPI				1.75	converted from RFD		50	NICNAS (2017)	100	NICNAS (2017)
68909-77-3	Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivatives residues	1	D	1.38E-06	EPI				3.5	converted from RFD		1000	REACH	1000	D
107-22-2	Glyoxal <1% (Ethanedial)	0.133	D	5.88E-05	EPI				0.4655	converted from RFD		13.3	NICNAS (2017)	100	NICNAS (2017)
5064-31-3	Nitrilotriacetic acid, trisodium salt monohydrate	0.01	D	1.13E-11	EPI				0.035	converted from RFD		10	ADWG (2018)	1000	ADWG (2018)
497-19-8	Sodium Carbonate	0.0967	D	3.08E-08	EPI				0.338	converted from RFD		9.67	NICNAS (2017)	100	NICNAS (2017)
533-74-4	Tetrahydro-3,5-dimethyl-1,3,5-thiadiazine-2-thione	0.005	D	5.05E-04	EPI				0.018	converted from RFD		0.5	NRA (1997)	100	NRA (1997)
50-01-1	Guanidine, hydrochloride (1:1)	0.1	D	3.86E-08	EPI				0.350	converted from RFD		100	REACH	1000	D
34590-94-8	(2-methoxymethylethoxy)propanol	1	D	1.36E-04	EPI				3.500	converted from RFD		1000	REACH	1000	D
68155-20-4	Amides, tall oil fatty N,N-bis (hydroxyethyl)	0.75	D	7.14E-02	EPI				2.625	converted from RFD		750	REACH	1000	D
64741-44-2	Distillates (petroleum), straight-run middle	0.03	D	1.36E+00	EPI				0.105	converted from RFD		30	REACH	1000	D
68457-79-4	Phosphorodithioic acid, mixed o,o-bis(iso-bu and pentyl) esters, zinc salts	0.16	D	3.07E+00	EPI				0.560	converted from RFD		160	REACH	1000	D
4719-04-4	Triazine based biocide C572,2',2"-(hexahydro-1,3, 5-triazine-1,3,5-triyl) triethano	0.064	D	7.29E-08	EPI				0.224	converted from RFD		64	REACH	1000	D
10192-30-0	Ammonium hydrogensulfite	0.113	D	6.26E-08	EPI				0.396	converted from RFD		113	REACH	1000	D
848301-67-7	Distillates (Fischer-Tropsch), C8-26 - Branched and Linear	0.2	D	1.36E+00	EPI				0.700	converted from RFD		200	REACH	1000	D
68909-77-3	Filming amine Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivs. Residues	1	D	1.38E-06	EPI				3.500	converted from RFD		1000	REACH	1000	D
68990-47-6	Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine	1	D	1.01E-03	EPI				3.500	converted from RFD		1000	REACH	1000	D
1120-36-1	1-tetradecene	0.1	D	6.29E+00	EPI				0.350	converted from RFD		100	REACH	1000	D
68910-93-0	Fatty acids, tall-oil, reaction products with polyethylenepolyamines	1	D	1.01E-03	EPI				3.500	converted from RFD		1000	REACH	1000	D
68585-36-4	Phosphoric ester of ethoxylated fatty alcohol	1	D	1.01E-03	EPI				3.500	converted from RFD		1000	REACH	1000	D
629-73-2	Hexadec-1-ene	0.1	D	1.97E+01	EPI				0.350	converted from RFD		100	REACH	1000	D
64742-52-5	Distillates (petroleum), hydrotreated heavy naphthenic	0.8	D	2.52E+02	EPI				2.800	converted from RFD		800	USEPA (2011)	1000	D
64742-53-6	Distillates (petroleum), hydrotreated light naphthenic < 3% DMSO	0.8	D	1.96E+00	EPI				2.800	converted from RFD		800	USEPA (2011)	1000	D
8052-42-4	Bitumen	0.2	D	1.00E-03	EPI				0.700	converted from RFD		200	REACH	1000	D

Notes:

A - Read across data from Boric Acid

B - Read across data from Alcohol ethoxylates C6-C12

C - Read across data from Sodium Sulfite

References:

D - Derived (refer to individual Toxicity Profiles)

EFSA - European Food Safety Authority

EPI - USEPA Estimation Programs Interface (EPI) Suite

J- JECFA - Joint FAO/WHO Expert Committee on Food Additives

NICNAS - National Industrial Chemicals Notification and Assessment Scheme. Assessed at https://www.nicnas.gov.au/

NICNAS (2017) - Department of the Environment and Energy 2017 , National assessment of chemicals associatedwith coal seam gas extraction in Australia, prepared by the National Industrial ChemicalsNotification and Assessment Scheme

NTP - US Department of Health and Serices National Toxicology Program

OECD - Organisation for Economic Co-operation and Development Screening Information Data Set (SIDS)

REACH - ECHA REACH European Chemicals Agency Database: http://apps.echa.europa.eu

Exposure to Chemicals via Incidental Ingestion of Flowback fluid - Drilling Fluids

Chronic Exposures

General Data/ Equations		Units	Exposure Calculations (RME) Ingestion of Flowback Water by Workers	
	Exposure Parameters			
	Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
	Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Use of Drilling	Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
	Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
	Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
	Ingestion Rate (IRw)	L/day or L/hr	0.005	Assume Incidental ingestion of 5 ml (1 tsp) of water per day during fraccing.
	Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
	Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**4.2E-09**	**NonThreshold**
			3.5E-06	**Threshold**

Daily Intake from Water = Concentration in Water x Intake Factor (ref: USEPA 1989)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

	Chemical	Toxicity Data: Non-Threshold Slope Factor	Toxicity Data: Chronic Threshold TDI	Toxicity Data: Background Intake (% Chronic TDI)	Toxicity Data: Chronic TDI Allowable for Assessment (TDI-Background)	Concentration in Water	Daily Intake: NonThreshold	Daily Intake: Threshold	Calculated Risk: NonThreshold Risk	Calculated Risk: Chronic Hazard Quotient
		(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
67-56-1	Methanol		3.7E-02		3.7E-02	0.30	1.3E-09	1.1E-06	--	2.8E-05
64742-47-8	Hydrotreated light petroleum distillate		1.0E+01		1.0E+01	1.50	6.3E-09	5.3E-06	--	5.3E-07
111-30-8	Glutaraldehyde		4.0E-02		4.0E-02	0.30	1.3E-09	1.1E-06	--	2.6E-05
78330-21-9	Alcohol, C11-14, ethoxylatedB		5.0E-01		5.0E-01	1.50	6.3E-09	5.3E-06	--	1.1E-05
107-22-2	Glyoxal <1% (Ethanedial)		1.3E-01		1.3E-01	2.20	9.2E-09	7.7E-06	--	5.8E-05
5064-31-3	Nitrilotriacetic acid, trisodium salt monohydrate		1.0E-02		1.0E-02	1.00	4.2E-09	3.5E-06	--	3.5E-04
497-19-8	Sodium Carbonate		9.7E-02		9.7E-02	0.29	1.2E-09	1.0E-06	--	1.1E-05
533-74-4	Tetrahydro-3,5-dimethyl-1,3,5-thiadiazine-2-thione		5.0E-03		5.0E-03	4.00	1.7E-08	1.4E-05	--	2.8E-03
50-01-1	Guanidine, hydrochloride (1:1)		1.0E-01		1.0E-01	7.00	2.9E-08	2.5E-05	--	2.5E-04
34590-94-8	(2-methoxymethylethoxy)propanol		1.0E+00		1.0E+00	0.007	2.9E-11	2.5E-08	--	2.5E-08
68155-20-4	Amides, tall oil fatty N,N-bis (hydroxyethyl)		7.5E-01		7.5E-01	0.0000001	4.2E-16	3.5E-13	--	4.7E-13
64741-44-2	Distillates (petroleum), straight-run middle		3.0E-02		3.0E-02	0.0000001	4.2E-16	3.5E-13	--	1.2E-11
68457-79-4	Phosphorodithioic acid, mixed o,o-bis(iso-bu and pentyl) esters, zinc salts		1.6E-01		1.6E-01	0.0000001	4.2E-16	3.5E-13	--	2.2E-12
4719-04-4	Triazine based biocide C572,2',2"-(hexahydro-1,3, 5-triazine-1,3,5-triyl) triethano		6.4E-02		6.4E-02	0.00101	4.2E-12	3.5E-09	--	5.5E-08
10192-30-0	Ammonium hydrogensulfite		1.1E-01		1.1E-01	0.00071	3.0E-12	2.5E-09	--	2.2E-08
848301-67-7	Distillates (Fischer-Tropsch), C8-26 - Branched and Linear		2.0E-01		2.0E-01	0.0000001	4.2E-16	3.5E-13	--	1.8E-12
68909-77-3	Filming amine Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivs. Residues		1.0E+00		1.0E+00	0.005	2.1E-11	1.8E-08	--	1.8E-08
68990-47-6	Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine		1.0E+00		1.0E+00	0.007	2.9E-11	2.5E-08	--	2.5E-08
1120-36-1	1-tetradecene		1.0E-01		1.0E-01	0.0000001	4.2E-16	3.5E-13	--	3.5E-12
68910-93-0	Fatty acids, tall-oil, reaction products with polyethylenepolyamines		1.0E+00		1.0E+00	0.0000001	4.2E-16	3.5E-13	--	3.5E-13
68585-36-4	Phosphoric ester of ethoxylated fatty alcohol		1.0E+00		1.0E+00	0.0000001	4.2E-16	3.5E-13	--	3.5E-13
629-73-2	Hexadec-1-ene		1.0E-01		1.0E-01	0.0000001	4.2E-16	3.5E-13	--	3.5E-12
64742-52-5	Distillates (petroleum), hydrotreated heavy naphthenic		8.0E-01		8.0E-01	0.0000001	4.2E-16	3.5E-13	--	4.4E-13
64742-53-6	Distillates (petroleum), hydrotreated light naphthenic < 3% DMSO		8.0E-01		8.0E-01	0.0000001	4.2E-16	3.5E-13	--	4.4E-13
8052-42-4	Bitumen		2.0E-01		2.0E-01	0.0000001	4.2E-16	3.5E-13	--	1.8E-12
								Total Risk (mixture)	--	**3.5E-03**

Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

Dermal Exposure to Chemicals via Contact with Flow Back Water - Drilling Fluids

Chronic Exposures

	General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Flow Back Water by Workers	
	Exposure Parameters			
	Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
	Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Use of Drilling	Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
	Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
	Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
	Surface Area (SAw)	cm^2	2300	Hands and forearms exposed (enHealth 2012) Occupational HSE would require long pants and closed shoes on Australian work sites; forearms conservatively included
	Exposure Time (ET)	hr/day	1	Assume contact with flow back water for 1 hours per day
	Conversion Factor (CF)	L/cm^3	1.E-03	Conversion of units
	Intake Factor = SAw*ET*CF*EF*ED / BW*AT	L-hr/(cm-kg-day)	**1.9E-06** **1.6E-03**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor (ref: USEPA 1989, 2004)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

	Chemical	Toxicity Data					Concentration	Daily Intake		Calculated Risk	
		Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)	Dermal Permeability	in Water	NonThreshold	Threshold	NonThreshold Risk	Chronic Hazard Quotient
		$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
67-56-1	Methanol		3.7E-02		3.7E-02	3.2E-4	0.30	1.8E-10	1.5E-07	--	4.2E-06
64742-47-8	Hydrotreated light petroleum distillate		1.0E+01		1.0E+01	2.0E+0	1.50	5.7E-06	4.8E-03	--	4.8E-04
111-30-8	Glutaraldehyde		4.0E-02		4.0E-02	3.3E-4	0.30	1.9E-10	1.6E-07	--	3.9E-06
78330-21-9	Alcohol, C11-14, ethoxylatedB		5.0E-01		5.0E-01	1.3E+0	1.50	3.7E-06	3.1E-03	--	6.2E-03
107-22-2	Glyoxal <1% (Ethanedial)		1.3E-01		1.3E-01	5.9E-5	2.20	2.5E-10	2.1E-07	--	1.6E-06
5064-31-3	Nitrilotriacetic acid, trisodium salt monohydrate		1.0E-02		1.0E-02	1.1E-11	1.00	2.2E-17	1.8E-14	--	1.8E-12
497-19-8	Sodium Carbonate		9.7E-02		9.7E-02	3.1E-8	0.29	1.7E-14	1.4E-11	--	1.5E-10
533-74-4	Tetrahydro-3,5-dimethyl-1,3,5-thiadiazine-2-thione		5.0E-03		5.0E-03	5.1E-4	4.00	3.9E-09	3.3E-06	--	6.5E-04
50-01-1	Guanidine, hydrochloride (1:1)		1.0E-01		1.0E-01	3.9E-8	7.00	5.2E-13	4.4E-10	--	4.4E-09
34590-94-8	(2-methoxymethylethoxy)propanol		1.0E+00		1.0E+00	1.4E-4	0.007	1.8E-12	1.5E-09	--	1.5E-09
68155-20-4	Amides, tall oil fatty N,N-bis (hydroxyethyl)		7.5E-01		7.5E-01	7.1E-2	0.0000001	1.4E-14	1.2E-11	--	1.5E-11
64741-44-2	Distillates (petroleum), straight-run middle		3.0E-02		3.0E-02	1.4E+0	0.0000001	2.6E-13	2.2E-10	--	7.3E-09
68457-79-4	Phosphorodithioic acid, mixed o,o-bis(iso-bu and pentyl) esters, zinc salts		1.6E-01		1.6E-01	3.1E+0	0.0000001	5.9E-13	5.0E-10	--	3.1E-09
4719-04-4	Triazine based biocide C572,2',2"-(hexahydro-1,3, 5-triazine-1,3,5-triyl) triethano		6.4E-02		6.4E-02	7.3E-8	0.00101	1.4E-16	1.2E-13	--	1.9E-12
10192-30-0	Ammonium hydrogensulfite		1.1E-01		1.1E-01	6.3E-8	0.00071	8.5E-17	7.2E-14	--	6.4E-13
848301-67-7	Distillates (Fischer-Tropsch), C8-26 - Branched and Linear		2.0E-01		2.0E-01	1.4E+0	0.0000001	2.6E-13	2.2E-10	--	1.1E-09
68909-77-3	Filming amine Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivs. Residues		1.0E+00		1.0E+00	1.4E-6	0.005	1.3E-14	1.1E-11	--	1.1E-11
68990-47-6	Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine		1.0E+00		1.0E+00	1.0E-3	0.007	1.4E-11	1.1E-08	--	1.1E-08
1120-36-1	1-tetradecene		1.0E-01		1.0E-01	6.3E+0	0.0000001	1.2E-12	1.0E-09	--	1.0E-08
68910-93-0	Fatty acids, tall-oil, reaction products with polyethylenepolyamines		1.0E+00		1.0E+00	1.0E-3	0.0000001	1.9E-16	1.6E-13	--	1.6E-13
68585-36-4	Phosphoric ester of ethoxylated fatty alcohol		1.0E+00		1.0E+00	1.0E-3	0.0000001	1.9E-16	1.6E-13	--	1.6E-13
629-73-2	Hexadec-1-ene		1.0E-01		1.0E-01	2.0E+1	0.0000001	3.8E-12	3.2E-09	--	3.2E-08
64742-52-5	Distillates (petroleum), hydrotreated heavy naphthenic		8.0E-01		8.0E-01	2.5E+2	0.0000001	4.8E-11	4.1E-08	--	5.1E-08
64742-53-6	Distillates (petroleum), hydrotreated light naphthenic < 3% DMSO		8.0E-01		8.0E-01	2.0E+0	0.0000001	3.8E-13	3.2E-10	--	4.0E-10
8052-42-4	Bitumen										
									Total Risk (mixture)		**7.3E-03**

Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

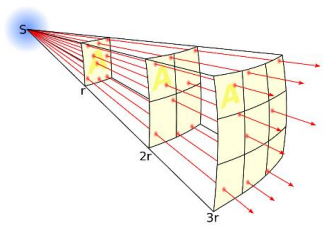
Aerosol Exposure - Drilling Fluids

The concentration of COPC in aerosol spray was estimated by calculating the concentration for driftable droplets using a mixed box model in which steady state concentrations were
An emission factor for driftable aerosol was estimated using the algorithm presented below.

Emission Factor for Driftable Aerosol Algorithm

Use of Drilling Fluid

$$Emission\ Factor_{driftable\ aerosol}\left(\frac{L}{m^3}\right) = \frac{Box\ Centre^2(m) \times \left(\frac{Spray\ Vol\ \left(\frac{L}{hr}\right) \times Aerosol_{driftable}(\%)}{BoxVR\ \left(\frac{m^3}{hr}\right)}\right)}{BoxDistance^2\ (m)}$$



Aerosol Exposure Modelling Notes:

1) The inhalation of chemicals in mist/aerosol resultant from irrigation activities is dependent upon the concentration in water, the amount of water used per unit time, how close a person stands to the spray generation, how long they are in a position of exposure and the extent of spray drift (determined by the size of the water droplets and speed/direction of the wind). These equations are applicable for non-volatile contaminants that are inhaled.

2) These equations calculate the concentration for driftable droplets using a simple well mixed box model in which steady state air concentrations are calculated. The 'Inverse square law' is then applied to approximate the air concentration at a distance from the virtual air box. This law assumes the further away a receptor is from the spray source, the density of the droplets will decrease. The density of the spray droplets is inversely proportional to the square of the distance from the source.

Parameter	Units	Value	Description
Spray box length	m	3	Assume a 'spray box' of 3 m long.
Spray box width	m	3	Assume a 'spray box' of 3 m wide.
Box Centre	m	1.5	Distance to centre of box is 1.5 m.
$Box_{Distance}$	m	2	Distance the irrigation worker is from the 'spray box'. Assumed a distance of 2 m.
$Aerosol_{driftable}$	unitless	0.2	Proportion of aerosol spray that drifts outside the 'spray box' and available for exposure. Assumed 0.2, based on a droplet size of 400 – 500 µm that falls approximately 0.3 m in less than 10 seconds, with a lateral drift of approximately 3.5 m in a 5 km/hr wind (i.e. a light breeze) (Grisso et al. 2013).
Spray Volume	L/hr	1800.0	1800 L/min, irrigation value adopted from NZ MtE (2011) Appendix 5A.
Wind speed	m/hr	9000	Based on windspeed of 2.5 m/sec
BoxVR	m^3/hr	81000.0	Ventilation rate of spray in the 'spray box'. Assumed to be 81,000 m3/hr based on a wind speed of 9000 m/hr, and a 'spray box' dimension of 3 x 3 m.

CAS	Chemical	Concentration in Water	Generation rate of chemical in volume	Driftable Aerosol Emission Factor
		mg/L	mg/hr	L/m^3
67-56-1	Methanol	0.30	108	2.5E-03
64742-47-8	Hydrotreated light petroleum distillate	1.50	540	2.5E-03
111-30-8	Glutaraldehyde	0.30	108	2.5E-03
78330-21-9	Alcohol, C11-14, ethoxylatedB	0.01	2.6	2.5E-03
107-22-2	Glyoxal <1% (Ethanedial)	2.20	792	2.5E-03
5064-31-3	Nitrilotriacetic acid, trisodium salt monohydrate	1.00	360	2.5E-03
497-19-8	Sodium Carbonate	0.29	104.4	2.5E-03
533-74-4	Tetrahydro-3,5-dimethyl-1,3,5-thiadiazine-2-thione	4.00	1440	2.5E-03
50-01-1	Guanidine, hydrochloride (1:1)	7.00	2520	2.5E-03
14808-60-7	Crystalline silica, quartz	10.00	3600	2.5E-03
34590-94-8	(2-methoxymethylethoxy)propanol	0.007	2.52	2.5E-03
68155-20-4	Amides, tall oil fatty N,N-bis (hydroxyethyl)	0.0000001	0.000036	2.5E-03
64741-44-2	Distillates (petroleum), straight-run middle	0.0000001	0.000036	2.5E-03
68457-79-4	Phosphorodithioic acid, mixed o,o-bis(iso-bu and pentyl) esters, zinc salts	0.0000001	0.000036	2.5E-03
4719-04-4	Triazine based biocide C572,2',2"-(hexahydro-1,3, 5-triazine-1,3,5-triyl) triethano	0.00101	0.3636	2.5E-03
10192-30-0	Ammonium hydrogensulfite	0.00071	0.2556	2.5E-03
848301-67-7	Distillates (Fischer-Tropsch), C8-26 - Branched and Linear	0.0000001	0.000036	2.5E-03
68909-77-3	Filming amine Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivs. Residues	0.0050000	1.8	2.5E-03
68990-47-6	Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine	0.0070000	2.52	2.5E-03
1120-36-1	1-tetradecene	0.0000001	0.000036	2.5E-03
68910-93-0	Fatty acids, tall-oil, reaction products with polyethylenepolyamines	0.0000001	0.000036	2.5E-03
68585-36-4	Phosphoric ester of ethoxylated fatty alcohol	0.0000001	0.000036	2.5E-03
629-73-2	Hexadec-1-ene	0.0000001	0.000036	2.5E-03
64742-52-5	Distillates (petroleum), hydrotreated heavy naphthenic	0.0000001	0.000036	2.5E-03
64742-53-6	Distillates (petroleum), hydrotreated light naphthenic < 3% DMSO	0.0000001	0.000036	2.5E-03
8052-42-4	Bitumen	0.0000001	0.000036	2.5E-03

Exposure to Chemicals via Inhalation of Mist from the Evaporation Units - Drilling Fluids

Chronic Exposures

Use of Drilli

General Data/ Equations	Units	Exposure Calculations (RME) Inhalation of Mist by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays
Exposure Duration (ED)	years	1	Maximum duration that the flowback tank will be on-site
Exposure Time (ET)	hr/day	1	Professional judgement for irrigation exposure. Assume worker to be near tank for 1 hours every working day.
Driftable aerosol emission factor (EMF)	L/m3	2.50E-03	Calculated
Aerosol Inhalation Bioavailability (AAF)	unitless	1.0	Assume 100% bioavailability
Averaging Time - Threshold (AT)	years	1.0	USEPA 1989 and CSMS 1996
$ITF_{inh,w,shwr} = \frac{EmF \times AAF \times ET_{iw} \times EF \times ED}{365\ \frac{days}{year} \times 24\ \frac{hours}{day} \times AT}$			

Daily Intake = Concentration in Water x Intake Factor (ref: USEPA 1989)
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

CAS	Chemical	Groundwater Concentration	Aerosol Inhalation Bioavailability	Driftable Aerosol Emission Factor	RfC (Background Corrected)	Threshold Intake and Risk Calculations: Adult Exposure Factor (threshold)	Adult Exposure Adjusted Air Concentration (threshold)	Hazard Index (Adult)
		mg/L	(unitless)	(L/m^3)	(mg/m^3)	(L/m^3)	(mg/m^3)	(unitless)
67-56-1	Methanol	0.3	1.00	2.50E-03	1.30E-01	6.85E-05	2.05E-05	1.58E-04
64742-47-8	Hydrotreated light petroleum distillate	1.5	1.00	2.50E-03	3.50E+01	6.85E-05	1.03E-04	2.94E-06
111-30-8	Glutaraldehyde	0.3	1.00	2.50E-03	1.40E-01	6.85E-05	2.05E-05	1.47E-04
78330-21-9	Alcohol, C11-14, ethoxylatedB	0.0	1.00	2.50E-03	1.75E+00	6.85E-05	5.00E-07	2.85E-07
107-22-2	Glyoxal <1% (Ethanedial)	2.2	1.00	2.50E-03	4.66E-01	6.85E-05	1.51E-04	3.24E-04
5064-31-3	Nitrilotriacetic acid, trisodium salt monohydrate	1	1.00	2.50E-03	3.50E-02	6.85E-05	6.85E-05	1.96E-03
497-19-8	Sodium Carbonate	0.29	1.00	2.50E-03	3.38E-01	6.85E-05	1.99E-05	5.87E-05
533-74-4	Tetrahydro-3,5-dimethyl-1,3,5-thiadiazine-2-thione	4	1.00	2.50E-03	1.75E-02	6.85E-05	2.74E-04	1.57E-02
50-01-1	Guanidine, hydrochloride (1:1)	0	1.00	2.50E-03	3.50E-01	6.85E-05	5.00E-07	1.43E-06
14808-60-7	Crystalline silica, quartz	10	1.00	2.50E-03	3.00E-03	6.85E-05	6.85E-04	2.28E-01
34590-94-8	(2-methoxymethylethoxy)propanol	0.007	1.00	2.50E-03	3.50E+00	6.85E-05	4.79E-07	1.37E-07
68155-20-4	Amides, tall oil fatty N,N-bis (hydroxyethyl)	0.0000001	1.00	2.50E-03	2.63E+00	6.85E-05	6.85E-12	2.61E-12
64741-44-2	Distillates (petroleum), straight-run middle	0.0000001	1.00	2.50E-03	1.05E-01	6.85E-05	6.85E-12	6.52E-11
68457-79-4	Phosphorodithioic acid, mixed o,o-bis(iso-bu and pentyl) esters, zinc salts	0.0000001	1.00	2.50E-03	5.60E-01	6.85E-05	6.85E-12	1.22E-11
4719-04-4	Triazine based biocide C572,2',2"-(hexahydro-1,3, 5-triazine-1,3,5-triyl) triethano	0.00101	1.00	2.50E-03	2.24E-01	6.85E-05	6.92E-08	3.09E-07
10192-30-0	Ammonium hydrogensulfite	0.00071	1.00	2.50E-03	3.96E-01	6.85E-05	4.86E-08	1.23E-07
848301-67-7	Distillates (Fischer-Tropsch), C8-26 - Branched and Linear	0.0000001	1.00	2.50E-03	7.00E-01	6.85E-05	6.85E-12	9.78E-12
68909-77-3	Filming amine Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivs. Residues	0.005	1.00	2.50E-03	3.50E+00	6.85E-05	3.42E-07	9.78E-08
68990-47-6	Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine	0.007	1.00	2.50E-03	3.50E+00	6.85E-05	4.79E-07	1.37E-07
1120-36-1	1-tetradecene	0.0000001	1.00	2.50E-03	3.50E-01	6.85E-05	6.85E-12	1.96E-11
68910-93-0	Fatty acids, tall-oil, reaction products with polyethylenepolyamines	0.0000001	1.00	2.50E-03	3.50E+00	6.85E-05	6.85E-12	1.96E-12
68585-36-4	Phosphoric ester of ethoxylated fatty alcohol	0.0000001	1.00	2.50E-03	3.50E+00	6.85E-05	6.85E-12	1.96E-12
629-73-2	Hexadec-1-ene	0.0000001	1.00	2.50E-03	3.50E-01	6.85E-05	6.85E-12	1.96E-11
64742-52-5	Distillates (petroleum), hydrotreated heavy naphthenic	0.0000001	1.00	2.50E-03	2.80E+00	6.85E-05	6.85E-12	2.45E-12
64742-53-6	Distillates (petroleum), hydrotreated light naphthenic < 3% DMSO	0.0000001	1.00	2.50E-03	2.80E+00	6.85E-05	6.85E-12	2.45E-12
8052-42-4	Bitumen	0.0000001	1.00	2.50E-03	7.00E-01	6.85E-05	6.85E-12	9.78E-12
							Total Threshold Risk (mixture)	**0.25**

Summary of Risk to Workers - Drilling Fluids
Exposure fo Target Chemicals - Theoretical Data

Receptor/Exposure Pathway	Calculated HI 100% Mass Return
Use of Drilling Fluid	
Workers	
Ingestion of Chemicals via Incidental Contact with Flowback Water	0.004
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water	0.007
Inhalation of mist from the evaporation units	0.2
Total Risk	**0.3**

Appendix E

Chemical Risk Assessment - Tracers

Human Health Screening Assessment
Chemical Tracers

Tracer Name	Concentration in Injected Fluid (mg/L)	Ecotoxicity	Toxicity	Persistence	Bioaccummulative	Tier 1 Screening Assessment	Tier 2 Assessment Worker Ingestion Risk	Tier 2 Assessment Worker Dermal Risk	Tier 2 Assessment Worker Aerosol Inhalation Risk	Hazard Quotient	Outcome of Tier 2 Worker Risk Assessment[1]
CFT (20 chemicals)	0.75	Algae EC50 = 33.1 mg/L Fish LC50 = 44.6 mg/L Daphnia EC50 > 100 mg/L Algae EC10 = 3.4 mg/L Fish NOEC 28 d = 120 mg/L Daphnia NOEC 21 d = 25 mg/L	Based on chronic: Low	Expected to be readily biodegradable	No based on calculated log Kow of 1.87	Tier 2	3.19E-06	1.01E-05	1.78E-05	3.11E-05	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
GFT (15 chemicals)	1.35	Fish 96h LC50 > 100 mg/L Invertebrates 48h EC50 > 0.1 mg/L Microorganism 3h EC50 > 100 mg/L Fish 96h NOEC = 1000 mg/L	Based on chronic: Low	Not readily biodegradable	Yes based on calculated log Kow of > 4.5	Tier 2	4.74E-06	1.04E-03	2.64E-05	1.08E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
WFT (1 chemical)	200,000	LC50 fish (96 h) > 120 mg/L EC50 daphnia (48h) > 125 mg/L EC50 plants (48h) > 125 mg/L	Based on acute: Low	Not readily biodegradable	No based on log Kow of -10.7	Tier 2	2.34E-01	1.23E-02	NA. Not volatile	2.46E-01	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).
WFT (1 chemical)	200,000	Fish 96 h LC50 = 87 mg/L Daphnia 48 h EC50 = 182 mg/L Algae ErC50 > 100 mg/L	Based on acute: Low	Expected to be readily biodegradable	No based on log Kow of 0.07	Tier 2	7.02E-02	6.66E-12	NA. Not volatile	7.02E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to individual toxicity profile and risk calculations for further detail).

Toxicity and Dermal Absorption Parameters

C = calculated from chronic value, Ch = chronic value adopted

CAS#	Chemical	Oral/Dermal Exposures				Inhalation Exposures									
		Threshold Chronic TDI or RfD (mg/kg/day)		Dermal Permeability (cm/hr)	Reference	Inhalation Unit Risk $(ug/m^3)^{-1}$	Non-Threshold Slope Factor $(mg/kg/day)^{-1}$		Threshold Chronic TC or RfC (mg/m^3)		NOAEC or LOAEC (mg/m^3)	NOAEL or LOAEL (mg/kg bw/d)	Reference	UF	Reference
	COPC in Hydraulic Fracturing Fluid Injected into Well														
	CFT	0.825	D	6.88E-03	EPI				2.8875	converted from RFD		825.0	OECD (2004)	1000	D
	GFT	1	D	4.79E-01	EPI				3.5	converted from RFD		1000	REACH	1000	D
	WFT	3	EFSA	1.14E-04	EPI				-	Not volatile		-	-	-	-
	WFT	10	JECFA	2.06E-13	EPI				-	Not volatile		-	-	-	-

Notes:

A - Read across data from Boric Acid

B - Read across data from Alcohol ethoxylates C6-C12

C - Read across data from Sodium Sulfite

References:

D - Derived (refer to individual Toxicity Profiles)

EFSA - European Food Safety Authority

EPI - USEPA Estimation Programs Interface (EPI) Suite

FSANZ - Food Standards Australia New Zealand

J- JECFA - Joint FAO/WHO Expert Committee on Food Additives

NICNAS - National Industrial Chemicals Notification and Assessment Scheme. Assessed at https://www.nicnas.gov.au/

NICNAS (2017) - Department of the Environment and Energy 2017 , National assessment of chemicals associatedwith coal seam gas extraction in Australia, prepared by the National Industrial ChemicalsNotification and Assessment Scheme

NTP - US Department of Health and Serices National Toxicology Program

OECD - Organisation for Economic Co-operation and Development Screening Information Data Set (SIDS)

REACH - ECHA REACH European Chemicals Agency Database: http://apps.echa.europa.eu

Exposure to Chemicals via Incidental Ingestion of Flowback fluid - CFT

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Flowback Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Ingestion Rate (IRw)	L/day or L/hr	0.005	Assume Incidental ingestion of 5 ml (1 tsp) of water per day during fraccing.
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**4.2E-09** **3.5E-06**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Intake Factor (ref: USEPA 1989)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

Chemical	Toxicity Data: Non-Threshold Slope Factor	Toxicity Data: Chronic Threshold TDI	Toxicity Data: Background Intake (% Chronic TDI)	Toxicity Data: Chronic TDI Allowable for Assessment (TDI-Background)	Concentration in Water	Daily Intake: NonThreshold	Daily Intake: Threshold	Calculated Risk: NonThreshold Risk	Calculated Risk: Chronic Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
CFT		8.3E-01		8.3E-01	0.75	3.1E-09	2.6E-06	--	3.2E-06
							Total Risk (mixture)	**--**	**3.19E-06**

Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

Dermal Exposure to Chemicals via Contact with Flow Back Water - CFT

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Flow Back Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Surface Area (SAw)	cm^2	2300	Hands and forearms exposed (enHealth 2012) Occupational HSE would require long pants and closed shoes on Australian work sites; forearms conservatively included
Exposure Time (ET)	hr/day	1	Assume contact with flow back water for 1 hours per day
Conversion Factor (CF)	L/cm^3	1.E-03	Conversion of units
Intake Factor = SAw*ET*CF*EF*ED / BW*AT	L-hr/(cm-kg-day)	**1.9E-06** **1.6E-03**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor (ref: USEPA 1989, 2004)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

Chemical	Toxicity Data				Dermal Permeability	Concentration in Water	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	NonThreshold Risk	Chronic Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
CFT		8.3E-01		8.3E-01	6.9E-3	0.75	9.9E-09	8.3E-06	--	1.0E-05
								Total Risk (mixture)		**1.01E-05**

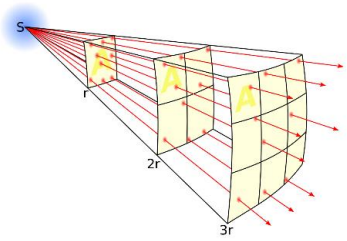
Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

Aerosol Exposure - CFT

The concentration of COPC in aerosol spray was estimated by calculating the concentration for driftable droplets using a mixed box model in which steady state
An emission factor for driftable aerosol was estimated using the algorithm presented below.

Emission Factor for Driftable Aerosol Algorithm

$$Emission\ Factor_{driftable\ aerosol}\ \left(\frac{L}{m^3}\right) = \frac{Box\ Centre^2(m) \times \left(\frac{Spray\ Vol\ \left(\frac{L}{hr}\right) \times Aerosol_{driftable}\ (\%)}{BoxVR\ \left(\frac{m^3}{hr}\right)}\right)}{BoxDistance^2\ (m)}$$



Aerosol Exposure Modelling Notes:

1) The inhalation of chemicals in mist/aerosol resultant from irrigation activities is dependent upon the concentration in water, the amount of water used per unit time, how close a person stands to the spray generation, how long they are in a position of exposure and the extent of spray drift (determined by the size of the water droplets and speed/direction of the wind). These equations are applicable for non-volatile contaminants that are inhaled.

2) These equations calculate the concentration for driftable droplets using a simple well mixed box model in which steady state air concentrations are calculated. The 'Inverse square law' is then applied to approximate the air concentration at a distance from the virtual air box. This law assumes the further away a receptor is from the spray source, the density of the droplets will decrease. The density of the spray droplets is inversely proportional to the square of the distance from the source.

Parameter	Units	Value	Description
Spray box length	m	3	Assume a 'spray box' of 3 m long.
Spray box width	m	3	Assume a 'spray box' of 3 m wide.
Box Centre	m	1.5	Distance to centre of box is 1.5 m.
$Box_{Distance}$	m	2	Distance the irrigation worker is from the 'spray box'. Assumed a distance of 2 m.
$Aerosol_{driftable}$	unitless	0.2	Proportion of aerosol spray that drifts outside the 'spray box' and available for exposure. Assumed 0.2, based on a droplet size of 400 – 500 µm that falls approximately 0.3 m in less than 10 seconds, with a lateral drift of approximately 3.5 m in a 5 km/hr wind (i.e. a light breeze) (Grisso et al. 2013).
Spray Volume	L/hr	1800.0	1800 L/min, irrigation value adopted from NZ MtE (2011) Appendix 5A.
Wind speed	m/hr	9000	Based on windspeed of 2.5 m/sec
BoxVR	m^3/hr	81000.0	Ventilation rate of spray in the 'spray box'. Assumed to be 81,000 m3/hr based on a wind speed of 9000 m/hr, and a 'spray box' dimension of 3 x 3 m.

CAS	Chemical	Concentration in Water	Generation rate of chemical in volume	Driftable Aerosol Emission Factor
		mg/L	mg/hr	L/m^3
	CFT	0.75	270	2.500000E-03

Exposure to Chemicals via Inhalation of Mist from the Evaporation Units - CFT

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Inhalation of Mist by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays
Exposure Duration (ED)	years	1	Maximum duration that the flowback tank will be on-site
Exposure Time (ET)	hr/day	1	Professional judgement for irrigation exposure. Assume worker to be near tank for 1 hours every working day.
Driftable aerosol emission factor (EMF)	L/m3	2.50E-03	Calculated
Aerosol Inhalation Bioavailability (AAF)	unitless	1.0	Assume 100% bioavailability
Averaging Time - Threshold (AT)	years	1.0	USEPA 1989 and CSMS 1996
$ITF_{inh,w,shwr} = \frac{EmF \times AAF \times ET_{iw} \times EF \times ED}{365\ \frac{days}{year} \times 24\ \frac{hours}{day} \times AT}$			

Daily Intake = Concentration in Water x Intake Factor (ref: USEPA 1989)
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

						Threshold Intake and Risk Calculations		
CAS	Chemical	Groundwater Concentration	Aerosol Inhalation Bioavailability	Driftable Aerosol Emission Factor	RfC (Background Corrected)	Adult Exposure Factor (threshold)	Adult Exposure Adjusted Air Concentration (threshold)	Hazard Index (Adult)
		mg/L	(unitless)	(L/m^3)	(mg/m^3)	(L/m^3)	(mg/m^3)	(unitless)
	CFT	0.8	1.00	2.50E-03	2.89E+00	6.85E-05	5.14E-05	1.78E-05
							Total Threshold Risk (mixture)	**1.78E-05**

Exposure to Chemicals via Incidental Ingestion of Flowback fluid - GFT

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Flowback Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Ingestion Rate (IRw)	L/day or L/hr	0.005	Assume Incidental ingestion of 5 ml (1 tsp) of water per day during fraccing.
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**4.2E-09** **3.5E-06**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Intake Factor (ref: USEPA 1989)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

Chemical	Toxicity Data: Non-Threshold Slope Factor	Toxicity Data: Chronic Threshold TDI	Toxicity Data: Background Intake (% Chronic TDI)	Toxicity Data: Chronic TDI Allowable for Assessment (TDI-Background)	Concentration in Water	Daily Intake: NonThreshold	Daily Intake: Threshold	Calculated Risk: NonThreshold Risk	Calculated Risk: Chronic Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
GFT		1.0E+00		1.0E+00	1.35	5.6E-09	4.7E-06	--	4.7E-06
							Total Risk (mixture)	--	**4.74E-06**

Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

Dermal Exposure to Chemicals via Contact with Flow Back Water - GFT

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Flow Back Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Surface Area (SAw)	cm^2	2300	Hands and forearms exposed (enHealth 2012) Occupational HSE would require long pants and closed shoes on Australian work sites; forearms conservatively included
Exposure Time (ET)	hr/day	1	Assume contact with flow back water for 1 hours per day
Conversion Factor (CF)	L/cm^3	1.E-03	Conversion of units
Intake Factor = SAw*ET*CF*EF*ED / BW*AT	L-hr/(cm-kg-day)	**1.9E-06** **1.6E-03**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor (ref: USEPA 1989, 2004)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

Chemical	Toxicity Data				Dermal Permeability	Concentration in Water	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	NonThreshold Risk	Chronic Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
GFT		1.0E+00		1.0E+00	4.8E-1	1.35	1.2E-06	1.0E-03	--	1.04E-03
								Total Risk (mixture)		**1.0E-03**

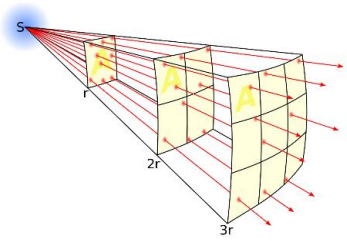
Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

Aerosol Exposure - GFT

The concentration of COPC in aerosol spray was estimated by calculating the concentration for driftable droplets using a mixed box model in which steady state
An emission factor for driftable aerosol was estimated using the algorithm presented below.

Emission Factor for Driftable Aerosol Algorithm

$$Emission\ Factor_{driftable\ aerosol}\ \left(\frac{L}{m^3}\right) = \frac{Box\ Centre^2(m) \times \left(\frac{Spray\ Vol\ \left(\frac{L}{hr}\right) \times Aerosol_{driftable}\ (\%)}{BoxVR\ \left(\frac{m^3}{hr}\right)}\right)}{BoxDistance^2\ (m)}$$



Aerosol Exposure Modelling Notes:

1) The inhalation of chemicals in mist/aerosol resultant from irrigation activities is dependent upon the concentration in water, the amount of water used per unit time, how close a person stands to the spray generation, how long they are in a position of exposure and the extent of spray drift (determined by the size of the water droplets and speed/direction of the wind). These equations are applicable for non-volatile contaminants that are inhaled.

2) These equations calculate the concentration for driftable droplets using a simple well mixed box model in which steady state air concentrations are calculated. The 'Inverse square law' is then applied to approximate the air concentration at a distance from the virtual air box. This law assumes the further away a receptor is from the spray source, the density of the droplets will decrease. The density of the spray droplets is inversely proportional to the square of the distance from the source.

Parameter	Units	Value	Description
Spray box length	m	3	Assume a 'spray box' of 3 m long.
Spray box width	m	3	Assume a 'spray box' of 3 m wide.
Box Centre	m	1.5	Distance to centre of box is 1.5 m.
$Box_{Distance}$	m	2	Distance the irrigation worker is from the 'spray box'. Assumed a distance of 2 m.
$Aerosol_{driftable}$	unitless	0.2	Proportion of aerosol spray that drifts outside the 'spray box' and available for exposure. Assumed 0.2, based on a droplet size of 400 – 500 µm that falls approximately 0.3 m in less than 10 seconds, with a lateral drift of approximately 3.5 m in a 5 km/hr wind (i.e. a light breeze) (Grisso et al. 2013).
Spray Volume	L/hr	1800.0	1800 L/min, irrigation value adopted from NZ MtE (2011) Appendix 5A.
Wind speed	m/hr	9000	Based on windspeed of 2.5 m/sec
BoxVR	m^3/hr	81000.0	Ventilation rate of spray in the 'spray box'. Assumed to be 81,000 m3/hr based on a wind speed of 9000 m/hr, and a 'spray box' dimension of 3 x 3 m.

CAS	Chemical	Concentration in Water mg/L	Generation rate of chemical in volume mg/hr	Driftable Aerosol Emission Factor L/m^3
	GFT	1.35	486	2.500000E-03

Exposure to Chemicals via Inhalation of Mist from the Evaporation Units - GFT

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Inhalation of Mist by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays
Exposure Duration (ED)	years	1	Maximum duration that the flowback tank will be on-site
Exposure Time (ET)	hr/day	1	Professional judgement for irrigation exposure. Assume worker to be near tank for 1 hours every working day.
Driftable aerosol emission factor (EMF)	L/m3	2.50E-03	Calculated
Aerosol Inhalation Bioavailability (AAF)	unitless	1.0	Assume 100% bioavailability
Averaging Time - Threshold (AT)	years	1.0	USEPA 1989 and CSMS 1996
$ITF_{inh,w,shwr} = \frac{EmF \times AAF \times ET_{iw} \times EF \times ED}{365\ \frac{days}{year} \times 24\ \frac{hours}{day} \times AT}$			

Daily Intake = Concentration in Water x Intake Factor (ref: USEPA 1989)
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

						Threshold Intake and Risk Calculations		
CAS	Chemical	Groundwater Concentration	Aerosol Inhalation Bioavailability	Driftable Aerosol Emission Factor	RfC (Background Corrected)	Adult Exposure Factor (threshold)	Adult Exposure Adjusted Air Concentration (threshold)	Hazard Index (Adult)
		mg/L	(unitless)	(L/m^3)	(mg/m^3)	(L/m^3)	(mg/m^3)	(unitless)
64742-47-8	GFT	1.4	1.00	2.50E-03	3.50E+00	6.85E-05	9.25E-05	2.64E-05
							Total Threshold Risk (mixture)	**2.64E-05**

Exposure to Chemicals via Incidental Ingestion of Flowback fluid - WFT

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Flowback Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Ingestion Rate (IRw)	L/day or L/hr	0.005	Assume Incidental ingestion of 5 ml (1 tsp) of water per day during fraccing.
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**4.2E-09**	**NonThreshold**
		3.5E-06	**Threshold**

Daily Intake from Water = Concentration in Water x Intake Factor (ref: USEPA 1989)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

Chemical	Toxicity Data: Non-Threshold Slope Factor	Toxicity Data: Chronic Threshold TDI	Toxicity Data: Background Intake (% Chronic TDI)	Toxicity Data: Chronic TDI Allowable for Assessment (TDI-Background)	Concentration in Water	Daily Intake: NonThreshold	Daily Intake: Threshold	Calculated Risk: NonThreshold Risk	Calculated Risk: Chronic Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
WFT		3.0E+00		3.0E+00	200000	8.4E-04	7.0E-01	--	2.3E-01
WFT		1.0E+01		1.0E+01	200000	8.4E-04	7.0E-01	--	7.0E-02
							Total Risk (mixture)	**--**	**3.04E-01**

Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

Dermal Exposure to Chemicals via Contact with Flow Back Water - WFT

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Flow Back Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Surface Area (SAw)	cm^2	2300	Hands and forearms exposed (enHealth 2012) Occupational HSE would require long pants and closed shoes on Australian work sites; forearms conservatively included
Exposure Time (ET)	hr/day	1	Assume contact with flow back water for 1 hours per day
Conversion Factor (CF)	L/cm^3	1.E-03	Conversion of units
Intake Factor = $\frac{SAw*ET*CF*EF*ED}{BW*AT}$	L-hr/(cm-kg-day)	**1.9E-06** **1.6E-03**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor (ref: USEPA 1989, 2004)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

Chemical	Toxicity Data				Concentration	Daily Intake		Calculated Risk	
	Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)	Dermal Permeability	in Water	NonThreshold	Threshold	NonThreshold Risk	Chronic Hazard Quotient
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
WFT		3.0E+00		3.0E+00	1.1E-4	200000.00	4.4E-05	3.7E-02	--	1.2E-02
WFT		1.0E+01		1.0E+01	2.1E-13	200000.00	7.9E-14	6.7E-11	--	6.7E-12
								Total Risk (mixture)		**1.23E-02**

Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

Summary of Risk to Workers - Chemical Tracers
Exposure fo Target Chemicals - Theoretical Data

Receptor/Exposure Pathway	Calculated HI 100% Mass Return
Use of Chemical Tracers in Hydraulic Fracturing	
CFT Recipe	
Workers	
Ingestion of Chemicals via Incidental Contact with Flowback Water	0.0000032
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water	0.000010
Inhalation of mist from the evaporation units	0.000018
Total Risk	**0.00003**
GFT Recipe	
Workers	
Ingestion of Chemicals via Incidental Contact with Flowback Water	0.0000047
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water	0.0010
Inhalation of mist from the evaporation units	0.000026
Total Risk	**0.001**
WFT Recipe	
Workers	
Ingestion of Chemicals via Incidental Contact with Flowback Water	0.30
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water	0.012
Inhalation of mist from the evaporation units	-
Total Risk	**0.3**

Appendix F

Toxicological Profiles for SW, HYBRID and HVFR Recipes

AECOM Imagine it. Delivered.

Toxicity Summary - 2-hydroxy-N,N,N-trimethylethanaminium (Choline Chloride)

Chemical and Physical Properties[1,2,3,4]	
CAS number	67-48-1
Molecular formula	$C_5H_{14}NOCl$
Molecular weight	139.63 g/mole
Solubility in water	Very soluble in water and alcohol
Melting point	247°C
Boiling point	Decomposition upon heating
Vapour pressure	6.57 x 10^{-8} Pa at 25°C
Henrys law constant	2.06*10E-11 Pa*m³/mole at 25°C
Explosive potential	Not explosive
Flammability potential	Combustible. Gives off irritating or toxic fumes (or gases) in a fire.
Colour/Form	white crystalline solid
Overview	Choline chloride is a quaternary amine salt, it dissociates in water into the corresponding positively charged quaternary hydroxyl alkylammonium ion and the negatively charged chloride ion. Choline chloride has neither explosive nor oxidizing properties due to its molecular structure Choline is a dietary component and found in foods as free choline and as esterified forms such as phosphocholine, glycerophosphocholine, sphingomyeline, and phosphatidylcholine. It functions as a precursor for acetylcholine, phospholipids, and the methyl donor betaine and is important for the structural integrity of cell membranes, methyl metabolism, cholinergic neurotransmission, transmembrane signalling, and lipid and cholesterol transport and metabolism. Evidence from animal studies and from human exposure indicates that choline chloride has low toxicity, is not mutagenic and has no developmental toxicity. This is not unexpected in view of its presence in the diet and its production in metabolic processes in the body; it fulfils key roles in nerve transmission, cell membrane integrity, and lipid metabolism. Only limited animal data are available on effects on fertility, but the normal exposure of humans to appreciable amounts of choline chloride both from the diet and formed from normal metabolic processes, would argue against it having any significant adverse effects on fertility. This is supported by the fact that it has been widely used as an animal feed additive for decades with no apparent adverse effects being noted on fertility. A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that this chemical was identified as low concern to human health.
Environmental Fate[1,3,4]	
Soil/Water/Air	Distribution modelling using Mackay Level I indicates water (100 %) to be the main target compartment. The amount in the other compartments is with < 0.0001 % negligible. Choline chloride is readily biodegradable according to OECD-criteria (MITI-I Test; BOD measurements) reaching 93 % degradation within 14 days. Due to the chemical structure hydrolysis can be excluded. In the atmosphere choline chloride will be rapidly degraded according to a half-life time (t½) of about 6.9 hours for hydroxyl-radicals based on a 12 hours day. Due to the measured and calculated logKow of –3.77 and –5.16 both at 25°C, respectively, and a calculated logKoc of 0.37 a bio- or geoaccumulation is not to be expected.

Human Health Toxicity Summary [1,3,4,5]	
Chronic Repeated Dose Toxicity	A 72-week feeding study was conducted to investigate the impact of choline chloride on the liver tumour promoting activity of phenobarbital and DDT in diethylnitroamineinitiated Fischer 344 rats (Shivapurkar *et al.*, 1986). Animals received approximately 500 mg/kg-day choline chloride. Following the end of the exposure period, the animals were kept on the same untreated diet as the control group until study termination at week 103. Histopathology was limited to the liver and organs that developed gross abnormalities. There were no significant differences between treated and control animals on survival rates, body weights, and relative liver weights. Neither was there any increased number of neoplastic liver nodules, hepatocellular carcinomas, lung tumours, leukaemia nor other tumours between treated and control animals. The NOAEL for choline chloride in this study is 500 mg/kg/day In humans, oral administration of 10,000 mg/day choline chloride in a pilot study treating a small number of patients with Alzheimer's disease, resulted in a slight hypotensive effect (Boyd *et al.*, 1977). This dose was regarded as a LOAEL by the Standing Committee on the Scientific Evaluation of Dietary Reference Intake (2000).
Carcinogenicity	No studies were located.
Mutagenicity/ Genotoxicity	Choline chloride was not mutagenic to bacteria in reverse mutation assays (Haworth *et al.*, 1984; JETOC, 1997; Litton Bionetics, 1977). A small, but statistically significant, and dose-related increase in sister chromatid exchanges (SCEs) in Chinese Hamster Ovary (CHO) cells was reported at 50 and 500 µg/ml choline chloride in the absence of S9 only (Bloom *et al.*, 1982). No higher concentrations were examined. These results could not be confirmed in another study using CHO cells at concentrations of choline chloride up to 5,000 µg/ml. (Galloway *et al.*, 1985). In a gene conversion assay with *Saccharomyces cerevisiae* strain D4, choline chloride was negative in the presence and absence of metabolic activation (Litton Bionetics, 1977). No *in vivo* genotoxicity studies were available.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Pregnant female mice were given in their feed 1,250 to 20,000 mg/kg choline chloride during gestational days 1 to 18 (BASF AG, 1966). Maternal body weight gain was reduced in all treated groups except for the 1,250 mg/kg group. Determination of maternal weight gain of dams with embryonic/foetal absorptions showed that there was no All foetuses were resorbed in the 20,000 mg/kg group. Embryonic/foetal lethality of 35% and 69% were seen in the 4,160 and 10,800 mg/kg groups, respectively. No resorptions occurred in the 1,250 mg/kg group. Developmental toxicity was seen in all but the 1,250 mg/kg group. No statistically significant increases in malformations were observed in any dose group. The NOAELs for maternal and developmental toxicity is 1,250 mg/kg/day.
Acute Toxicity	The oral LD50 in rats was reported to be between 3,150 and 5,000 mg/kg (BASF AG, 1963a, 1969).
Irritation	Application of a 70% aqueous solution to the skin of rabbits for 20 hours under occlusive conditions resulted in only minor skin irritation (BASF AG, 1963b). Slight eye irritation was seen in the eyes of rabbits after instillation of a 70% aqueous solution of choline chloride; no effects were seen one day after exposure (BASF AG, 1963c).
Sensitisation	No data are available in animals. In a Human Repeated Insult Patch Test, there was no evidence of dermal sensitization in two hundred subjects given 0.5% (w/v) aqueous solution of choline chloride during the induction phase and 0.2% (w/v) aqueous solution during the challenge phase (Colgate-Palmolive, 2003).
Health Effects Summary	This chemical has been identified by NICNAS to be of low concern to human health.

Key Study/Critical Effect for Screening Criteria	The Standing Committee on the Scientific Evaluation of Dietary Reference Intakes selected hypotension as the critical effect from the study by Boyd *et al.* (1977) when deriving a Tolerable Upper Intake Level. Boyd *et al.* (1977) reported a LOAEL of 10,000 mg/day choline chloride (7,500 mg/day choline). An uncertainty factor of 2 was chosen because of the limited data regarding hypotension and the inter-individual variation in response to cholinergic effects. Thus, the value for the Tolerable Upper Intake Value for repeated exposure of adults to choline is 3,500 mg/day choline. The oral RfD for choline chloride is derived by using the LOAEL of 10,000 mg/day from the Boyd *et al.* (1977) study, which is divided by an uncertainty factor of 2, to obtain a value of 5,000 mg/day or 71 mg/kg/day for a 70 kg person. Oral RfD = 71 mg/kg/day Drinking water guideline value = 248 ppm
Ecological Toxicity [4]	
Aquatic Toxicity	The 96-hour fish LC50 value is >100 mg/L (nominal and measured) in *Oryzias latipes* (MOE Japan, 1999a), and the 48-hour in vertebrate EC50 is 349 mg/L (nominal and measured) in *Daphnia magna* (MOE Japan, 1999b). The 72-hour EC50 to *Pseudokirchneriella subcapitata* is >1,000 mg/L (nominal and measured) based on growth rate; the 72-hour NOEC is 32 mg/L (MOE Japan, 1999c). In a 21-day *Daphnia magna* reproduction test, the nominal and measured NOEC was reported to be 30.2 mg/L (MOE Japan, 1999d).
Determination of PNEC aquatic	PNECaquatic: Experimental results are available for three trophic levels. Acute E(L)C50 values are available for fish (>100 mg/L), invertebrates (349 mg/L), and algae (>1,000 mg/L). Results from chronic studies are available for invertebrates (21-day NOEC = 30.2 mg/L) and algae (72-hour NOEC = 32 mg/L). On the basis that the data consists of chronic studies on two trophic levels, an assessment factor of 10 has been applied to the lowest reported NOEC of 30 mg/L for Daphnia. The PNECaquatic is 3.02 mg/L.
Current Regulatory Controls	
Australian Hazard Classification	No data available
Australian Occupational Exposure Standards	No data available
International Occupational Exposure Standards	No data available
Australian Food Standards	No data available
Australian Drinking Water Guidelines	No data available
Aquatic Toxicity Guidelines	No data available
PBT Assessment [3]	
P/vP Criteria fulfilled?	Choline chloride is readily biodegradable and thus it does not meet the screening criteria for persistence.
B/vB criteria fulfilled?	Based on a measured log Kow of -3.77 and a calculated BCF of 0.59, choline chloride does not meet the screening criteria for bioaccumulation.
T criteria fulfilled?	The chronic toxicity data on choline chloride show NOECs of >0.01 mg/L. Thus, choline chloride does not meet the screening criteria for toxicity.
Overall conclusion	Not a PBT substance (based on screening data).
Revised	December 2018

References

1. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2017, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
2. IPCS Inchem, Choline Chloride, CAS# 67-48-1
3. National Industrial Chemicals Notification and Assessment Scheme (NICNAS), IMAP Environment Tier I summary all tranches, 10 Mar 2017.
4. OECD (2004). SIDS Initial Assessment Report for Choline chloride (CAS No. 67-48-1)
5. UNEP Publications.Standing Committee on the Scientific Evaluation of Dietary Reference Intake. Institute of Medicine (2000). Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline. National Academy Press, Washington D.C.
6. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2018: https://www.nicnas.gov.au

AECOM Imagine it. Delivered.

Toxicity Summary - Acetic acid

Chemical and Physical Properties[1,6]	
CAS number	64-19-7
Molecular formula	$C_2H_4O_2$
Product name	Acetic Acid 60%
Molecular weight	60 g/mol
Solubility in water	1000 g/L at 25°C
pH	1.38
Melting point	16.6 °C
Boiling point	117.9 °C
Vapour pressure	1.5 kPa at 20°C
Henrys law constant	0.0101 Pa m^3/mol
Explosive potential	Above 39°C explosive vapour mixtures may be formed. Risk of fire and explosion on contact with strong oxidants.
Flammability potential	Flammable. Flashpoint = 39°C
Colour/Form	Clear colourless liquid with a pungent vinegar smell
Overview	Acetic acid is naturally occurring as the acid in apple cider vinegar and other fruit derived products. Acetic acid is recognised by Food Standards Australia New Zealand (FSANZ) and the US Food and Drug Administration (FDA) as safe as a food additives (e.g. flavouring agent, preservative).
Environmental Fate[1]	
Soil/Water/Air	When released into the environment, acetic acid is not expected to adsorb onto suspended solids or sediments. Acetic acid dissociates in aqueous media to H+ and the acetate anion ($CH_3CO_2^-$). The compound is expected to be present in the dissociated form, where volatilisation is not an important process. Based on the range of expected Koc values, acetic acide is expected to have a very high to moderate mobility in soil. In air acetic acid will exist soley in the vapour phase where it is degraded with photochemically produced hydroxyl radicals with a half-life for this reaction in air of approximately 22 days. Acetic acid is readily biodegradable, and biodegrades rapidly under aerobic and anaerobic conditions. Based on an estimated bioconcentration factor of 3.2, the potential for bioaccumulation is low.

Human Health Toxicity Summary [1,2,5,6]	
Chronic Repeated Dose Toxicity	In a six-month repeat dose oral toxicity study (Lamb and Evard 1919), pigs were initially fed acetic acid at 155 mg/kg bw/day with the dose was raised every 10 to 30 days until a final dose of 380 to 450 mg/kg bw/day was reached after 60 days. There was no mortality and no effects on body weight or acid-base balance noted in this study (REACH 2013). A NOAEL was not established in this study. Repeated intra-gastric administration of the chemical at 3% concentration in animals (unspecified) for six months resulted in chronic inflammation of the oesophageal mucosa (HSDB 2013). Similarly, intra-gastric administration to rats of 3 mL of a 10% solution for 90 days produced a drop in haemoglobin concentration and erythrocyte count (HSDB 2013). In another similar study, pigs were fed daily diets containing the chemical at 0, 240, 720, 960 and 1200 mg/kg bw/day for successive 30-day periods for a total of 150 days (HSDB 2013).There were no significant differences in growth rate, weight gain, early morning urinary ammonia and terminal blood pH between controls and test groups. A NOEL or NOAEL was not indicated by the authors. Based on the available information and taking a conservative approach, the NOAEL in the study is considered to be 1200 mg/kg bw/day, the highest tested dose with no adverse effects. This NOAEL will be used for human health risk assessment. In the only available dermal repeat dose toxicity study (Slaga et al. 1975), acetic acid was applied dermally to mice at doses of 1 to 40 mg/animal, one to three times/week for 32 weeks. Single dermal applications of acetic acid at doses of up to 40 mg/animal did not induce mortality. However, more than one application per week of 10 mg acetic acid or more caused excessive mortality. 33% of mice died when 10 mg acetic acid/animal was applied dermally three times/week and approximately 50% of mice died when 20 mg was applied twice a week. No biochemical or histopathological effects were reported. A LOAEL of 10 mg/animal was suggested by the authors, however it was expressed in terms of ‘mg/animal’ rather than ‘mg/kg bw/day’ and it therefore cannot be adopted. Dermal NOAEL or LOAEL for acetic acid are not available. Repeated oral, inhalation and dermal exposure of humans to pure acetic acid has been reported to have effects on the gastrointestinal tract and to cause digestive disorders including heartburn and constipation, chronic inflammation of the respiratory tract, pharyngitis, catarrhal bronchitis, darkening of skin, skin dermatitis and erosion of the exposed front teeth enamel. In addition, skin on the palms of hands can become dry, cracked and hyperkeratotic. These observed effects were not associated with any systemic findings, suggesting the effects observed could be due to its corrosive action (EC 2012; HSDB 2013).
Carcinogenicity	In a carcinogenicity study (Slaga et al. 1975), acetic acid was tested as the promoter for tumour development in mice. Acetic acid was applied dermally to mice initiated with a carcinogenic agent, dimethylbenz(a)anthracene (DMBA) at doses of 1 to 40 mg/animal, one to three times/week for 32 weeks. Control animals received acetic acid dermally once per week. No further details were provided about the exposure duration. Single dermal application of acetic acid at doses of up to 40 mg/animal did not induce mortality. However, more than one application per week of 10 to 40 mg acetic acid caused excessive mortality. Thirty three per cent of mice died when 10 mg acetic acid/animal was applied dermally three times/week and approximately 50% of mice died when 20 mg was applied twice a week. No biochemical or histopathological effects were reported. Acetic acid did not produce any carcinogenic effects in mice (REACH 2013). In another study, oral administration of the chemical as a 3% solution in rats, three times/week for eight months did not induce tumours in the oesophagus and fore-stomach, although epithelial hyperplasia was observed. When dosed in combination with the known carcinogen, N-nitrososarcosine ethyl ester (positive control), there was an increase in oesophageal/stomach tumour formation (REACH 2013). Based on the limited available data, acetic acid is not likely to be a carcinogen.

Mutagenicity/ Genotoxicity	Acetic acid was not mutagenic in bacterial reverse mutation assays using Salmonella typhimurium strains TA100, TA1535, TA97 and TA98 with and without metabolic activation (Ishidate et al. 1984). Acetic acid was negative in the chromosome aberration assay using Chinese hamster lung fibroblasts at concentrations of up to 1 mg/mL with or without metabolic activation. In one study using Chinese hamster ovary KI cells, acetic acid induced chromosomal aberrations at the initial pH of 6.0 or below (about 10 to 14 mM of acid) both with and without S9 mix (REACH 2013). However, when the culture medium was neutralised to pH 7.2 with sodium hydroxide, no clastogenic activity was observed. Moreover, pH lower than 6.0 (pH 5.7 or below) were also found to be cytotoxic. Chromosomal aberrations induced at these high concentrations were therefore considered to be artefacts due to acidification of the culture medium. Acetic acid was concluded not to be clastogenic when tested in cultured Chinese hamster K1 cells (REACH 2013; HSDB 2013). It was concluded that acetic acid is not mutagenic.
Reproductive Toxicity	No data available
Developmental Toxicity/Teratogenicity	In two developmental toxicity studies conducted according to the EU Method B.31 (prenatal developmental toxicity study), acetic acid was administered by gavage to pregnant female Wistar rats and CD-1 mice at 16, 74.3, 345, and 1600 mg/kg bw/day during gestation days 6 to 15 (10 consecutive doses) (REACH 2013). In a similar study, the chemical was administered by gavage to female Dutch rabbits at 16, 74.3, 345, and 1600 mg/kg bw/day during gestation days 6 to 18 (13 consecutive doses) (REACH 2013). There were no clearly discernible effects on implantation, maternal survival or foetal survival in any species at any of the doses. The number of abnormalities seen in either soft or skeletal tissues of the test groups did not differ significantly from those occurring spontaneously in the controls. No NOAEL could be established for maternal toxicity or foetal developmental effects. Based on the available data, the chemical does not show developmental toxicity.
Acute Toxicity	Acetic acid was of low acute toxicity in animal tests following oral exposure. The median lethal dose (LD50) observed in two rat studies is greater than 2000 mg/kg bw (REACH2013). In one study, groups of unfasted rats were fed 2239, 2512, 2859, 3100, 3500, 4000, 4467 mg/kg bw sodium acetate and observed for six days (REACH 2013). The acute oral median lethal dose (LD50) of the sodium salt of acetic acid was found to be 3310 mg/kg bw for rats. Acetic acid was of moderate acute toxicity in rabbits following dermal exposure. The LD50 in rabbits was 1060 mg/kg bw (HSDB 2013). Details regarding the concentration of the administered test substance were not provided. The moderate acute dermal toxicity is believed to be due to its local corrosive effects rather than any systemic toxicity. Acetic acid was of low acute toxicity in animal tests following inhalation exposure. In an acute inhalation study, mice were exposed to various concentrations of acetic acid (experimental details and concentration range not provided) (HSDB 2013). Clinical signs of respiratory irritation were evident at concentrations of 2.46 mg/L and higher. Animals exposed to concentrations higher than 11.07 mg/L died within 27 hours of exposure. Surviving mice recovered quickly and showed no abnormalities three days after exposure. The median lethal concentration (LC50) was determined by the Weil's method and was estimated to be 13.8 mg/L in the mouse. Severe health effects have been reported in humans following accidental exposure to acetic acid by different routes, mainly due to the local corrosive effects of the chemical leading to systemic effects (HSDB 2013).

Irritation	Pure acetic acid is corrosive to skin. In animal studies, severe skin burns were reported in guinea pigs following application to intact or abraded skin of patches of 80% solution of the chemical, moderate to severe burns at 50 to 80% solution, mild injury at 50% solution, and no effect at 10% solution (HSDB 2013). In a study with rabbits, the chemical was considered to be slightly irritating at concentrations of 3.3% and 10% (REACH 2013). In another study with rabbits, a concentration of 2.5% of the chemical was not irritating while concentrations of 10 to 25% caused moderate to severe erythema, slight to severe oedema, skin lesions over the application site and eschar formation (REACH 2013). A 10% solution was therefore considered a skin irritant. As part of a study to select the optimum testing conditions for predicting hazard to the human eye, 3%and 10% aqueous acetic acid were tested in rabbit eyes (REACH 2013). Materials were applied directly to the central corneal surface. Irritation was followed for up to 21 days and scored according to the Draize scale. The 3% acetic acid gave moderate irritation and 10% acetic acid was severely irritating or corrosive. In other studies, instillation of 0.5 mL of a 1% acetic acid solution in the eyes of rabbits caused a severe burn (Smyth et al. 1951). Solutions of 5% induced injury in eyes of rabbits which healed by 14 days, while a 10% solution resulted in severe permanent damage (Henschler 1973). Based on the results of the studies pure acetic acid is considered to be corrosive to eyes. In an acute inhalation study in mice, clinical signs of respiratory irritation were evident at concentrations of 2.46 mg/L and higher (see Section A28.5.2.3). Acetic acid vapours were reported to cause damage to nose, throat and lungs in humans (SCOEL 2012). Acetic acid is considered to be a respiratory tract irritant. Chemical burns and eye and nasal irritation have been reported in humans following exposure
Sensitisation	No experimental data were available, however the US National Institute of Occupational Safety and Health (NIOSH) Pocket Guide to Chemical Hazards mentions skin sensitisation as one of the symptoms of acetic acid exposure (NIOSH 2010). A 1994 report (Kivity et al. 1994) describes a late asthmatic response to inhaled glacial acetic acid by an asthma patient. Based on reports of patients with bronchial asthma reacting to acetic acid challenge, it is believed that acetic acid may cause allergic reactions in humans (HSDB 2013). Some researchers consider acetic acid capable of causing a syndrome known as ‘reactive airways dysfunction’, which resembles bronchial asthma. Symptoms include dyspnoea, wheezing, and cough.
Health Effects Summary	Acetic acid has low acute oral and inhalation toxicity but moderate dermal toxicity. LD50 for oral, dermal and inhalation routes were >3100 mg/kg bw, 1060 mg/kg bw and 13.8 mg/L, respectively in laboratory animals. It is corrosive to skin, eyes and respiratory tract. Acetic acid has low repeat dose toxicity by oral and dermal routes. Information on toxicity by the inhalation route is not available. It is not genotoxic or carcinogenic and does not have any developmental effects in animals. Information on effects on fertility is not available. The critical health effect of acetic acid for risk characterisation is its corrosivity.
Key Study/Critical Effect for Screening Criteria	A NOEL or NOAEL was not established in any of the repeat dose studies. Based on the available information and taking a conservative approach, the highest tested dose with no adverse effects in the repeat dose oral study (1200 mg/kg bw/day) was taken as the NOAEL for human health risk assessment.
Ecological Toxicity [2]	
Aquatic Toxicity	Acute endpoints: Fish = 75 mg/L (measured), Daphnia EC50 = 32 mg/L (Dept. Env. (2013a) in LMC, 2012 Chronic endpoints: Daphnia = 150 mg/L (measured)
Determination of PNEC aquatic	PNECaquatic: On the basis of the chronic results for Daphnia, an assessment factor of 10 has been applied to the lowest reported effect concentration of 150 mg/L. The PNECaquatic is determined to be 15 mg/L.

Current Regulatory Controls	
Australian Hazard Classification	Acetic acid is classified as hazardous, with the following risk phrase for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia 2013): C; R35 (Corrosive, causes severe burns). Mixtures containing the chemical are classified as hazardous with the following risk phrases based on the concentration (Conc) of the chemical in the mixtures: Conc >=90%: C; R35 (Corrosive, causes severe burns) ≥25% Conc <90%: C; R34 (Corrosive, causes burns) ≥10% Conc <25%: Xi; R36/38 (Irritant, Irritating to eyes and skin).
Australian Occupational Exposure Standards	The chemical has an exposure standard of 25 mg/m³ (10 ppm) Time Weighted Average (TWA) and 37 mg/m³ (15 ppm) Short-Term Exposure Limit (STEL) (Safe Work Australia 2013).
International Occupational Exposure Standards	The following exposure standards are identified in Galleria Chemica (2013). Occupational Exposure limit (TWA): 10 to 25 mg/m^3 [China, Canada, Denmark, Germany, Ireland, South Africa, Spain, Sweden, Switzerland, and the US]. An exposure limit (STEL): 15 to 50 mg/m^3 [China, Canada, France, Ireland, Singapore, South Africa, Spain, Sweden, Switzerland, and the US].
Australian Food Standards	Acetic acid is allotted the following International Numbering System of food additives number: INS 260 (Food Standards Australia New Zealand 2013).
Australian Drinking Water Guidelines	No data found
Aquatic Toxicity Guidelines	No data found
PBT Assessment	
P/vP Criteria fulfilled?	No. The acetate ion of acetic acid is readily biodegradable and thus it does not meet the screening criteria for persistence.
B/vB criteria fulfilled?	The log Kow for acetic acid is reported to be -0.136. Acetate is also found in the body and is metabolized as part of the body's biochemical pathways. Thus, acetic acid (specifically, the acetate ion) does not meet the screening criteria for bioaccumulation.
T criteria fulfilled?	No. The NOECs from the chronic aquatic toxicity data on acetic acid are >1 mg/L, hence does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier II Assessment for Acetic acid, Retrieved 2019: https://www.nicnas.gov.au
2. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2015, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
3. ECHA REACH, Acetic Acid, Retrieved 2015: http://apps.echa.europa.eu
4. JECFA http://apps.who.int/ipsc/database/evaluations/chemical.aspx?chemID=4785
5. U.S. EPA HPVIS database, http://www.epa.gov/chemrtk/hpvis/index.html
6. OECD, Acetic Acid, Retrieved 2015: http://www.echemportal.org
7. IPCS Acetic Acid, Retrieved 2015: http://www.inchem.org

AECOM Imagine it. Delivered.

Toxicity Summary - Acrylamide polymers: Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer and Polymer of 2-acrylamido-2- ethylpropanesulfonic acid sodium salt and methyl acrylate

Chemical and Physical Properties [2, 3, 4]	
CAS number	38193-60-1, 136793-29-8, 9003-06-9, 25987-30-8
Molecular formula	38193-60-1: $(C_7H_{13}NO_4S.C_3H_5NO.Na)_x$ 136793-29-8: $C_{11}H_{18}NNaO_6S$
Molecular weight	Likely >1000 MW
Solubility in water	No data available.
Melting point	No data available.
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	No data available.
Overview	No studies are available for the Acrylamide polymers. Information for 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt will be referenced in the following sections. 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salts are generally incorporated into polymers. As such, the fate of the monomer is tied to the polymer and no hydrolysis, movement, biodegradation or bioaccumulation of the polymer is expected. A Tier 1 Human Health Assessment for Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer has been conducted by NICNAS which concluded that this chemical was identified as low concern to human health.
Environmental Fate [2]	
Soil/Water/Air	The polymers are not expected to be readily biodegradable. Biodegradation is limited due to the very high molecular weights and the low water solubilities of the polymers. Due to their high molecular weight, the polymers are not expected to bioaccumulate.
Human Health Toxicity Summary [2]	
Chronic Repeated Dose Toxicity	A repeat dose 28 day oral toxicity study carried out in rats with a 50% aqueous solution of 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt indicated no treatment related toxic effects.
Carcinogenicity	No information available.
Mutagenicity/ Genotoxicity	2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt did not induce a mutagenic response in bacteria and no clastogenicity was observed when a 50% solution of the notified chemical was tested in albino mice cells in vitro.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No data available.

Acute Toxicity	Aqueous solutions containing 50% of 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt exhibited low acute oral and dermal toxicity in rats (LD50 > 5 000 mg/kg, and 2 000 mg/kg respectively).
Irritation	2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt was non-irritant to rabbit skin and a slight eye irritant in rabbits.
Sensitisation	A 50% aqueous solution of 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt showed minimal sensitisation potential when tested in guinea pigs.
Health Effects Summary	This chemical has been identified by NICNAS to be of low concern to human health.
Key Study/Critical Effect for Screening Criteria	No data available
Ecological Toxicity [2]	
Aquatic Toxicity	Limited information is available. The polymers are expected to be a low concern for toxicity to aquatic organisms. Due to their poor solubility and high molecular weights, they are not expected to be bioavailable. The polymers do not contain any reactive functional groups. Ecotoxicity data for 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt indicate that it is practically non-toxic to fish, water fleas and algae.
Determination of PNEC aquatic	No PNEC values were calculated.
Current Regulatory Controls [5]	
Australian Hazard Classification	No data available
Australian Occupational Exposure Standards	No data available
International Occupational Exposure Standards	No data available
Australian Food Standards	No data available
Australian Drinking Water Guidelines	Based on health considerations, the concentration of acrylamide in drinking water should not exceed 0.0002 mg/L.
Aquatic Toxicity Guidelines	No data available
PBT Assessment [1, 2]	
P/vP Criteria fulfilled?	The polymers are not readily biodegradable, hence they meet the screening criteria for persistence.
B/vB criteria fulfilled?	The polymers are expected to have very high molecular weights and poor water solubility. They are not expected to be bioavailable, hence the polymers do not meet the criteria for bioaccumulation.
T criteria fulfilled?	There are no aquatic toxicity studies on the polymers. They are expected to have low aquatic toxicity because of their very high molecular weights and poor water solubility. As such, the polymers do not meet the criteria for toxicity.
Overall conclusion	Not PBT substances
Revised	December 2018

References

1. Categorization Results from the Canadian Domestic Substance List, CAS# 38193-60-1

2. National Industry Chemicals Notification and Assessment Scheme. 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt, July 1997.
3. U.S National Library of Medicine, Toxicology Data Network, ChemIDplus, CAS#38193-60-1. Accessed 2017 at https://chem.nlm.nih.gov/chemidplus/rn/38193-60-1
4. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2018: https://www.nicnas.gov.au
5. National Health and Medical Research Council, Natural Resources Management Ministerial Council, national Water Quality Management Strategy, Australian Drinking Water Guidelines 6, 2011, Version 3.3 Updated November 2016.

AECOM Imagine it. Delivered.

Toxicity Summary - Acrylonitrile

Chemical and Physical Properties [1,2,3,4]	
CAS number	107-13-1
Molecular formula	C3H3N
Molecular weight	53.06
Solubility in water	73 g/L at 20 °C
Melting point	– 88.55 °C
Boiling point	77.3 °C
Vapour pressure	12.4 kPa at 20 °C
Henrys law constant	9.0 Pa ·m^3/mole at 20 °C
Explosive potential	Sax (1989) presents that acetonitrile forms explosive mixtures with air. The lower explosive limit is 3.05% in volume and the upper explosive limit 17% in volume.
Flammability potential	Acetonitrile is highly flammable, with a lower flammability limit of 4.4% in volume and an upper flammability limit of 16% in volume.
Colour/Form	Volatile, colourless liquid with a sweet ether-like odour
Overview	Acrylonitrile was first prepared in 1893 but had no significant technical or commercial applications until the late 1930s when a synthetic rubber based on a co-polymer of butadiene and acrylonitrile was introduced in Germany (Langvardt, 1984). In USA, projects relating to nitrile rubber received special support during World War II because of their strategic importance and acrylonitrile became established as a monomer of commercial importance. Demand for acrylonitrile began to soar following the introduction of acrylic fibres in 1950. Today, acrylonitrile is an industrial intermediate used predominantly in the production of polymeric materials, with acrylic fibres accounting for 60% and plastics for 25% of world consumption (SRI, 1995). Other uses include the production of adiponitrile and acrylamide monomers and the co-polymerisation with other monomers to produce polymer emulsions, elastomers and nitrile rubber. From the early 1940s to the mid-1960s, acrylonitrile was mainly manufactured by the dehydration of ethylene cyanohydrin produced from ethylene oxide and aqueous hydrocyanic acid. Nowadays, all acrylonitrile is produced by direct catalytic conversion of propene, oxygen (as air) and ammonia (SRI, 1995). Processes based on propane or ethylene have been developed and may become commercially viable in the future where propane or ethylene feedstock is readily available. In 1995, global acrylonitrile capacity amounted to 4.5 million metric tonnes (t) (SRI, 1995).
Environmental Fate[1]	
Soil/Water/Air	Acrylonitrile is readily to fairly degradable in water, soil and in the troposphere. Its toxicity to aquatic vertebrates and invertebrates, algae and aquatic plants is slight to moderate. Bioaccumulation is expected to be slight to negligible. As there are no readily hydrolysable groups on the acrylonitrile molecule, hydrolysis is not expected to be an environmentally significant process. The vapour pressure of acrylonitrile puts it in the category of highly volatile chemicals (Mensink et al., 1995). However, the water solubility is also high. The Henry's Law constant can provide an indication of the volatility characteristics of compounds (Lyman et al., 1982). The characteristics of acrylonitrile indicate that although the volatilisation from aquatic systems is not rapid, it may be a significant removal process in the environment. Therefore, the high vapour pressure is mediated by the high water solubility. The volatilisation half-life of acrylonitrile in a typical pond, river and lake has been estimated at 6, 1.2 and 4.8 days respectively (Howard, 1989). The US EPA has previously suggested that although acrylonitrile is quite volatile, large spillages of the substance could lead to groundwater contamination (DoE, 1993).
Human Health Toxicity Summary [1,2,3]	

Chronic Repeated Dose Toxicity	Repeated-dose toxicity studies involving inhalation, ingestion or subcutaneous or intraperitoneal injection of acrylonitrile for 1-12 months in rats, mice, guinea pigs, rabbits, cats, dogs and monkeys showed a narrow range between lethal and no observed adverse effect levels. The most consistently observed effects were decreased body weight gain, irritation of the respiratory tract, kidney damage and reversible ataxia or paralysis. Retching and vomiting, adrenal hyperplasia, increased liver weight, hyperplasia of the gastric mucosa and biochemical effects such as small reductions in haemoglobin, haematocrit and erythrocyte counts and small increases in alkaline phosphatase were observed in some studies.
Carcinogenicity	The carcinogenic potential of acrylonitrile has been investigated in three strains of rats exposed to 5-80 ppm in air (2 studies), 1-500 ppm in drinking water (5 studies), or 0.1-10 mg/kg by gavage (2 studies). Exposure-related tumours were found in all studies. The most common forms were astrocytomas of the CNS and carcinomas of the zymbal gland, both of which rarely occur spontaneously in experimental animals. Tumours of the mammary gland, tongue, small intestine and forestomach (oral exposure only) were less consistent across studies. A 2-year bioassay in mice, where metabolism via CNEO plays a greater role than in rats, is currently underway within the US National Toxicology Program. Acrylonitrile has also been evaluated by the International Agency for Research on Cancer (IARC). In 1979 and 1987, IARC concluded that there was limited evidence of carcinogenicity of acrylonitrile in humans and sufficient evidence of carcinogenicity in animals and therefore assigned the chemical to group 2A: agents that are probably carcinogenic to humans (IARC, 1979, 1987). In February 1998, all published literature on acrylonitrile was re-evaluated by an IARC working group comprising 30 experts from 12 countries. The group concluded that although additional studies confirmed that acrylonitrile is a potent multi-site carcinogen in rats, the combined epidemiological evidence did not support a credible association between acrylonitrile exposure and cancer. As such, IARC determined that there was inadequate evidence in humans but sufficient evidence in experimental animals for the carcinogenicity of acrylonitrile and re-classified the chemical in group 2B: agents that are possibly carcinogenic to humans (IARC, 1999).
Mutagenicity/ Genotoxicity	The genetic toxicity of acrylonitrile has been investigated in numerous in vitro and in vivo test systems. In vitro, it was weakly positive in several bacterial, fungal and mammalian mutagenicity assays and mammalian and fungal cytogenetic tests, particularly in the presence of metabolic activation. Where CNEO was tested in parallel assays, it was mutagenic in the absence of metabolic activation. In vivo, acrylonitrile tested negative in several dominant lethal, micronucleus and chromosome aberration assays. Studies in Drosophila using various genetic markers gave positive results. In vitro and in vivo assays for DNA binding and unscheduled DNA synthesis yielded negative results in tests using the most reliable techniques. On balance, it appears that acrylonitrile has little affinity for DNA, whereas the metabolite CNEO is a direct-acting mutagen in vitro. It is conceivable that the lack of genotoxicity of acrylonitrile in several in vivo tests is due to limited formation and/or rapid degradation of CNEO in intact mammals.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	In a 3-generation rat study, up to 35 mg/kg/day had no effect on fertility. In sub-acute studies in rats and mice, there was evidence of defective spermatogenesis at oral doses approaching acutely toxic levels, whereas several long-term studies found no abnormalities in male reproductive organs. In developmental toxicity studies in rats, hamsters, and rat embryos exposed in vitro, acrylonitrile showed some potential to cause foetal toxicity, but developmental effects in vivo occurred only at exposure levels associated with marked maternal toxicity.
Acute Toxicity	Acrylonitrile is acutely toxic by all routes of administration. In the rat, the LD50 is 72-186 mg/kg from oral and 148-282 mg/kg from skin exposure, and the 4 h LC50 from inhalation is 138-558 ppm (0.47-1.2 mg/L). The acute toxicity is roughly similar in other species, including mice, guinea pigs, rabbits, cats and dogs. Irrespective of route or test species, a lethal dose causes central nervous system (CNS) excitation followed by paralysis and respiratory arrest. The target organs are the gastrointestinal tract (bleeding), adrenals (haemorrhagic necrosis), brain (oedema) and lungs (oedema).
Irritation	Acrylonitrile is irritating to the skin and eyes. Repeated airborne exposure induces inflammatory and hyperplastic changes in the nasal mucosa, indicating a potential for irritation of the respiratory system.

Sensitisation	A guinea pig maximisation test for skin sensitisation was strongly positive. There are no data on respiratory sensitisation.
Health Effects Summary	Acrylonitrile is acutely toxic to humans by inhalation, in contact with skin and if swallowed. It is also a severe eye irritant and may cause sensitization by skin contact. Repeat dose toxicity studies in animals have shown treatment related changes in the gastrointestinal tract, central nervous system and adrenal gland. There are occasional reports of liver and kidney damage. It is a rodent carcinogen, tumours being observed in the brain, Zymbal gland, gastrointestinal tract and mammary gland. Detailed, recent epidemiological studies do not however provide evidence of human carcinogenicity. Acrylonitrile is an in vitro mutagen, indicating that the mechanism of carcinogenicity may be genotoxic. This is not however supported by the results of in vivo mutagenicity studies. It is concluded that there is a need for active management of the identified risk and further consideration of the risk management measures currently being applied in relation to workers, consumers and the population exposed via the environment.
Key Study/Critical Effect for Screening Criteria	In animals repeated exposure to acrylonitrile results in damage to the gastrointestinal tract, central nervous system and adrenal gland. There are occasional reports of liver and kidney damage. The respiratory tract is also affected following inhalation exposure, based on histopathological changes in the nasal turbinates of rats in the Quast et al.,(1980) two year study. A LO(A)EL of 20 ppm was established in the study, treatment-related nasal changes being evident at this exposure level, and this was used as a starting point in the risk assessment in relation to inhalation exposure. A No Adverse Effect Level (NAEL) of 4 ppm for the inhalation route was been derived from the LO(A)EL of 20 ppm, by application of a safety factor of 5. In relation to oral administration of acrylonitrile, the N(A)OEL is estimated to be 3 ppm (0.25 mg/kg/day) in drinking water, based on the information from the Biodynamics study (1980) study in rats which showed systemic toxicity, probably attributable to metabolic release of cyanide.
Ecological Toxicity [6]	
Aquatic Toxicity	The data set for acrylonitrile includes a wide range of information on short and long term toxicity in fish, Daphnia and other aquatic invertebrates. Acrylonitrile is moderately toxic to fish, with 96-hour LC50 for fresh water fish generally lying in the range of 10 - 20 mg/l (nominal). A recent short term study in the saltwater species Cyprinodon variegatus, carried out in full compliance with current protocols, reported a 96-hour LC50 of 8.6 mg/l. The lowest 48 hour EC50 for Daphnia was 7.6 mg/l. The fish early life stage toxicity test in Pimephales promelas, using flow-through conditions, provided a LOEC/NOEC of 0.34 mg/l, while a 30 day flow through test in mature fish of the same species provided a long-term LC50 of 2.6 mg/l. If the value of 0.34 mg/l is taken as a LOEC, a NOEC may be derived by application of safety factor of 2, giving a NOEC of 0.17 mg/l.
Determination of PNEC aquatic	Applying an assessment factor of 10 to the NOEC (0.17 mg/l) derived from the fish early life stage toxicity test gives a PNEC of 17 µg/l.
Current Regulatory Controls [1,7]	
Australian Hazard Classification	The chemical is classified as hazardous, with the following risk phrases for human health in the Hazardous Chemical Information System (HCIS) (Safe Work Australia): H225 (Highly flammable liquid and vapour) H350 (May cause cancer) H331 (Toxic if inhaled) H311 (Toxic in contact with skin) H301 (Toxic if swallowed) H335 (May cause respiratory irritation) H315 (Causes skin irritation) H318 (Causes serious eye damage) H317 (May cause an allergic skin reaction) H411 (Toxic to aquatic life with long-lasting effects)
Australian Occupational Exposure Standards	The current national occupational exposure standard for acrylonitrile in Australia is 2 ppm (4.3 mg/m3) expressed as an 8 h TWA airborne concentration, Carcinogen Category 2, with a ‘skin’ notation (NOHSC, 1995a).

International Occupational Exposure Standards	The following exposure standards are identified: 8h TWA: Austria 2 ppm (4.5 mg/m^3) Belgium 2 ppm (4.3 mg/m^3) Denmark 2 ppm (4.0 mg/m^3) Finland 2 ppm (4.3 mg/m^3) France 2 ppm (4.0 mg/m^3) Germany 3 ppm (7.0 mg/m^3) Hungary 0.23 ppm (0.5 mg/m^3) India 2 ppm (4.3 mg/m^3) Ireland 2 ppm (4.5 mg/m^3) Japan 2 ppm (4.3 mg/m^3) Netherlands 4 ppm (9 mg/m^3) Philippines 20 ppm (43 mg/m^3) Poland 5 ppm (10 mg/m^3) Russia 0.23 ppm (0.5 mg/m^3) Spain 2 ppm (4.5 mg/m^3) Sweden 2 ppm (4.5 mg/m^3) Turkey 20 ppm (43 mg/m^3) United Kingdom 2 ppm (4 mg/m^3) USA (NIOSH) 1 ppm (2.2 mg/m^3) USA (OSHA) 2 ppm (4.3 mg/m^3) Short-term exposure limits (STEL): Finland 4 ppm (9 mg/m^3) France 15 ppm (32.5 mg/m^3) Netherlands 10 ppm (22 mg/m^3) Sweden 6 ppm (14 mg/m^3) USA (NIOSH) 10 ppm (22 mg/m^3) USA (OSHA) 10 ppm (22 mg/m^3)
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	A freshwater low reliability trigger value of 160 µg/L was calculated for acetonitrile using an AF of 1000. In the absence of marine data, this was adopted as a marine low reliability trigger value.
PBT Assessment	
P/vP Criteria fulfilled?	No. Acrylonitrile is readily to fairly degradable in water, soil and in the troposphere
B/vB criteria fulfilled?	No. The low log Pow (0.00-0.30) measures for acrylonitrile suggest bioaccumulation will not occur.
T criteria fulfilled?	Yes. Chronic toxicity data <1 mg/L in fish, thus acrylonitrile meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. NICNAS (1998) Priority Existing Chemical 10, Acrylonitrile: Retrieved 2019: https://www.nicnas.gov.au
2. OECD (1998) European Union Risk Assessment Report, Acetonitrile, Retrieved 2019: http://www.echemportal.org
3. ECHA REACH, Acrylonitrile, Retrieved 2019: https://echa.europa.eu
4. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2019, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
5. ANZECC & ARMCANZ (2000), Australian and New Zealand Guidelines for Fresh and Marine Water Quality
6. OECD (2005) SIDS Initial Assessment Profile on Acrylonitrile

7. Hazardous Chemical Information System (HCIS), Safe Work Australia. Retrieved 2019: http://hcis.safeworkaustralia.gov.au/

AECOM Imagine it. Delivered.

Toxicity Summary - Alcohols, C10-16, ethoxylated propoxylated

Chemical and Physical Properties[1]	
CAS number	69227-22-1
Molecular formula	No data available.
Molecular weight	No data available.
Solubility in water	Soluble in water
Melting point	-3 °C
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	Yellow liquid, mild odour
Overview	Principle Route of Exposure: Eye or skin contact, inhalation Causes severe eye irritation which may damage tissue. Causes skin irritation. Harmful if swallowed.
Environmental Fate[1]	
Soil/Water/Air	This substance is expected to be readily biodegradable (84% @ 28d) (similar substances). Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8, it is not expected to be bioaccumulative. Mobility in soil: KOC = >4
Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	No data available to indicate product or components present at greater than 0.1% are chronic health hazards.
Carcinogenicity	Did not show carcinogenic effects in animal experiments (similar substances)
Mutagenicity/ Genotoxicity	In vitro tests did not show mutagenic effects. In vivo tests did not show mutagenic effects. (similar substances)
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Animal testing did not show any effects on fertility.
Acute Toxicity	LD50 Oral: 600 mg/kg (Rat) (similar substance) LD50 Dermal: > 5200 mg/kg (Rabbit) (similar substance) LD50 Inhalation: > 0.22 mg/L (saturated concentration) (Rat) (similar substance)
Irritation	May cause mild respiratory irritation. Causes severe eye irritation which may damage tissue. Causes skin irritation.
Sensitisation	Did not cause sensitization on laboratory animals (guinea pig) (similar substances)
Health Effects Summary	Causes severe eye irritation which may damage tissue. Causes skin irritation. Harmful if swallowed.
Key Study/Critical Effect for Screening Criteria	
Ecological Toxicity [1]	

Aquatic Toxicity	Toxicity to fish: LC50 (96h) 0.59 mg/L (Pleuronectes platessa) (similar substance) LC50 (96h) 1.6 mg/L (Pimephales promelas) (similar substace) LC50 (96h) 3 mg/L (Brachydanio rerio) (similar substance) Toxicity to invertebrates: EC50 (48h) 0.14 mg/L (Daphnia magna) (similar substance) EC50 (48h) 2 mg/L (Daphnia magna) (similar substance) Toxicity to algae: EC50 (72h) 0.75 mg/L (Pseudokirchnerella subcapitata) (similar substance) ErC50 (48h) 0.7 mg/L (Skeletonema costatum) EC10 9.79 mg/L (Selenastrum capricornutum) (similar substance) ErC50 1.1 mg/L (Scenedesmus subspicatus) (similar substance) Toxicity to microorganisms: ErC50 (16.9h) > 10 g/L (Pseudomonas putida) (similar substance)
Determination of PNEC aquatic	On the basis that the data consists of short term results from three trophic levels, an assessment factor of 1000 has been applied to the lowest reported effect concentration of 0.14 mg/L for Daphnia. The PNEC aquatic is 0.14 µg/L.
Current Regulatory Controls [1]	
Australian Hazard Classification	H302 - Harmful if swallowed H315 - Causes skin irritation H318 - Causes serious eye damage
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Expected to be readily biodegradable based on similar substances.
B/vB criteria fulfilled?	No. Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8, it is not expected to be bioaccumulative.
T criteria fulfilled?	No. The acute aquatic toxicity of this chemical is >0.01 mg/L. Thus, it is not expected to meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. Redacted

AECOM Imagine it. Delivered.

Toxicity Summary - Alcohols, C6-12, ethoxylated propoxylated

Chemical and Physical Properties[1]	
CAS number	68937-66-6
Molecular formula	No data available.
Molecular weight	No data available.
Solubility in water	Soluble in water
Melting point	-3 °C
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	Yellow liquid, mild odour
Overview	Principle Route of Exposure: Eye or skin contact, inhalation Causes severe eye irritation which may damage tissue. Causes skin irritation. Harmful if swallowed.
Environmental Fate[1]	
Soil/Water/Air	This substance is expected to be readily biodegradable (60% @ 28d) (similar substances). Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8, it is not expected to be bioaccumulative. Mobility in soil: KOC = >4
Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	No data available to indicate product or components present at greater than 0.1% are chronic health hazards.
Carcinogenicity	Did not show carcinogenic effects in animal experiments (similar substances)
Mutagenicity/ Genotoxicity	In vitro tests did not show mutagenic effects. In vivo tests did not show mutagenic effects. (similar substances)
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Animal testing did not show any effects on fertility.
Acute Toxicity	LD50 Oral: 600 mg/kg (Rat) (similar substance) LD50 Dermal: > 5200 mg/kg (Rabbit) (similar substance) LD50 Inhalation: > 0.22 mg/L (saturated concentration) (Rat) (similar substance)
Irritation	May cause mild respiratory irritation. Causes severe eye irritation which may damage tissue. Causes skin irritation.
Sensitisation	Did not cause sensitization on laboratory animals (guinea pig) (similar substances)
Health Effects Summary	Causes severe eye irritation which may damage tissue. Causes skin irritation. Harmful if swallowed.
Key Study/Critical Effect for Screening Criteria	
Ecological Toxicity [1]	

Aquatic Toxicity	Toxicity to fish: LC50 (96h) 0.59 mg/L (Pleuronectes platessa) (similar substance) LC50 (96) 1.4 mg/L (Pimephales promelas) (similar substance) NOEC 4.4 mg/L (Pimephales promelas, juvenile) Toxicity to invertebrates: EC50 (48h) 0.14 mg/L (Daphnia magna) (similar substance) EC50 (48h) 0.39 mg/L (Cerodaphnia dubia) (similar substance) Toxicity to algae: EC50 (72h) 0.75 mg/L (Pseudokirchnerella subcapitata) (similar substance) EC50 (96h) 0.7 mg/L (Pseudokirchneriella subapitata) (similar substance) CD10 8 mg/L (Pseudokirchneriella subapitata) EC10 2 mg/L (Brachionus calyciflorus) Toxicity to microorganisms: EC50 (48h) 0.39 mg/L (Cerodaphnia dubia) (similar substance)
Determination of PNEC aquatic	On the basis that the data consists of short term results from three trophic levels, an assessment factor of 1000 has been applied to the lowest reported effect concentration of 0.14 mg/L for Daphnia. The PNEC aquatic is 0.14 µg/L.
Current Regulatory Controls [1]	
Australian Hazard Classification	H302 - Harmful if swallowed H315 - Causes skin irritation H318 - Causes serious eye damage
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Expected to be readily biodegradable based on similar substances.
B/vB criteria fulfilled?	No. Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8, it is not expected to be bioaccumulative.
T criteria fulfilled?	Not applicable. Chronic toxicity data >1 mg/L in invertebrates, thus sodium hydroxide does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. Redacted

Toxicity Summary - Ethoxylated of aliphatic alcohols (>C6)

Chemical and Physical Properties [1,2,3]	
CAS number	112-59-4, 3055-93-4, 3055-94-5, 3055-95-6, 3055-97-8, 4536-30-5, 5274-68-0, 25190-05-0, 9002-92-0, 9004-95-9, 9004-98-2, 9005-00-9, 9043-30-5, 31726-34-8, 24938-91-8, 26183-52-8, 26468-86-0, 27252-75-1, 27306-79-2, 31943-12-1, 32128-65-7, 37281-47-3, 37702-39-9, 39587-22-9, 52292-17-8, 61723-78-2, 68439-45-2, 68439-46-3, 68439-49-6, 68439-50-9, 68439-54-3, 61791-13-7, 61791-28-4, 61827-42-7, 64425-86-1, 66455-14-9, 66455-15-0, 69227-20-9, 67254-71-1, 68002-97-1, 68131-39-5, 68131-40-8, 68155-01-1, 68213-23-0, 68526-94-3, 68551-12-2, 97953-22-5, 68920-66-1, 68991-48-0, 78330-21-9
Molecular formula	Unspecified
Molecular weight	Unspecified
Solubility in water	0.1876 - 13.18 mg/L at 25 °C (C12-14 ethoxylated, 1-2.5 EO) (CAS 68131-39-5) 1.69 - 246.7 mg/L at 25 °C (C9-11, ethoxylated (EO < 2.5) (CAS 68439-46-3)
Melting point	7.2 °C at 101.3 kPa (CAS 68131-39-5) -20 °C at 101.3 kPa (CAS 68439-46-3)
Boiling point	271.11 - 516.11 °C (CAS 68131-39-5) 260 °C (CAS 68439-46-3)
Vapour pressure	< 1 Pa at 25 °C (CAS 68131-39-5) 0.004 - 117 Pa at 20 °C (CAS 68439-46-3)
Henrys law constant	No data available.
Explosive potential	Non explosives
Flammability potential	Non flammable
Colour/Form	Organic liquid, colourless to light yellow
Overview	The chemicals in this group are structurally related alcohol ethoxylates (AEs), ethoxylated ethers of aliphatic alcohols, where the alky chain length is six carbons or higher. Ethoxylates of shorter chain alcohols (C<6) do not show the same degree of surfactancy compared to the chemicals in this group. Commercially available AEs generally consist of a mixture of various AE homologues of varying carbon chain lengths and degree of ethoxylation. The chemicals contain a hydrophobic alkyl chain attached via an ether linkage to a hydrophilic ethylene oxide (EO) chain that gives them their characteristic surfactant properties. The hydrophobic alkyl and the hydrophilic EO chains can vary in length depending on method of production and source of the precursor chemicals (HERA, 2009). Although most of chemicals of this group are polymers according to the definition in the Industrial Chemicals (Notification and Assessment) Act (1989), the individually named members do not necessarily meet the polymer of low concern (PLC) criteria as the number-average molecular weight (NAMW) >1000 Da. Lower molecular weight forms of these chemicals (MW <500) are expected to be used in commercial, domestic and cosmetic products. The chemicals are used extensively as non-ionic surfactants in a wide range of cosmetic and domestic products. The chemicals in this group are expected to have similar physicochemical and toxicological properties, which depend on the alkyl chain length and the number of EO units.
Environmental Fate [2,3]	
Soil/Water/Air	Alcohol ethoxylates are readily biodegradable under aerobic conditions and also anaerobically biodegradable (HERA, 2009). The main mechanism of primary biodegradation for the linear and essentially linear AE is the central cleavage of the molecule, leading to the formation of long chain alcohol and polyethylene glycol (HERA, 2009; Marcomini et al., 2000a; Marcomini et al., 2000b). Long chain alcohols themselves are readily biodegradable up to C18 (SIDS, 2006). Abiotic degradation in water, soil, sediment and air is not expected to occur because of the chemical structures of AE homologues. Neither hydrolysis under normal

	environmental conditions (pH range from 4 to 9) nor photolysis in the atmosphere, in water, or when absorbed to soil and sediment surfaces, is to be considered (HERA, 2009). Experimentally determined BCF-values given for pure homologues and summarized in the publication of Tolls et al. (2000) are used as read-across data for the endpoint bioaccumulation in water. It can be stated that bioaccumulation of alcohol ethoxylates is regarded to be negligible as the surfactants will be rapidly metabolised. For more detail see endpoint summary for bioaccumulation. Concerning transport and distribution of the alcohol ethoxylate mixtures a high adsorption of the substances is determined by using QSAR-models. Adsorption onto surfaces is an intrinsic property of alcohol ethoxylates and thus a high Koc-value is expected.
Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	The chemicals in this group are not expected to cause serious damage to health fr In several 90-day oral feeding studies in rats (similar to OECD TG 407), the NOAEL was established between 50 and 700 mg/kg bw/day (calculated from dietary levels) for group members (CAS Nos. 68439-50-9 and 68131-39-5, ranging from C12–15 with EO7). Effects observed at higher concentrations included reduction in mean body weights, and increases in relative liver and kidney weights. These changes were considered to be adaptive and related to the poor palatability of the test chemicals. No treatment related histopathological changes were reported (SCCS, 2007; HERA 2009; CIR, 2012). Similar effects were seen in longer-term studies. Alcohols, C12-13, ethoxylated (CAS No. 66455-14-9; EO6.5) and alcohols, C14-15, ethoxylated (CAS No. 68951-67-7, EO7, not listed on AICS) were given to rats in one- and two-year chronic feeding studies at levels between 0.1 and 1 %. The NOAEL was established between 50 and 192 mg/kg bw/day (calculated from dietary level). Effects observed at higher levels included reduction in mean body weights, and increase in relative liver and kidney weights. These changes were considered to be adaptive and may be due to poor palatability of the test chemicals. No treatment related lesions were observed (SCCS, 2007; HERA, 2009; CIR, 2012).om repeated oral and dermal exposure. In a 90-day study (OECD TG 411), Fischer rats were exposed to the chemical (C9–11 with 6 EO units, CAS No. 68439-46-3) at 1, 10 or 25 % concentration, 3 days/week. The application site was shaved but not covered. There were no significant treatment related effects at any concentration. Dry and flaky skin was observed in the 10 and 25 % dose groups. Increased relative kidney weights were observed in the 25 % dose groups. However, no histological lesions were observed. The NOAEL was established at 10 %, equivalent to 80 mg/kg bw/day (HERA, 2009).
Carcinogenicity	Based on the data available, the chemicals in this group are not considered to be carcinogenic. Two chemicals, alcohols, C12-13, ethoxylated (CAS No. 66455-14-9; EO6.5) and alcohols, C14-15, ethoxylated (CAS No. 68951-67-7, EO7, not listed on AICS) were administered at up to 1 % in the diet to rats for one and two years, respectively. No treatment related histopathological effects or increased tumour incidences were observed in either study (HERA, 2009; CIR, 2012). The chemicals are synthesised through processes which may result in 1,4-dioxane as an impurity. This impurity is classified as a Carcinogen—Category 3 (R40—Limited evidence of a carcinogenic effect). However, it is reported that cosmetic industry uses additional purification steps to remove the 1,4-dioxane residual in PEG before blending into cosmetic formulations (CIR, 2012).

Mutagenicity/ Genotoxicity	Based on the data available, the chemicals in this group are not considered to be genotoxic. The group members (CAS Nos. 68439-50-9, 68131-39-5 and 64425-86-1) and several analogue chemicals (ranging from C12–18 and EO3–21) produced negative results in several in vitro and in vivo tests for gene mutation and clastogenicity. Negative results were reported in bacterial reverse mutation tests for mutagenicity against Salmonella typhimurium (strains TA98, TA100, TA102, TA104, TA1535, TA1537 and TA1538) and Escherichia coli (strains WP2 and WP2uvrA pKM101), with or without metabolic activation. Negative results were also reported in chromosomal aberration tests in Chinese hamster V79, Chinese hamster ovary, mouse lymphoma and rat liver cell lines (SCCP, 2007; HERA, 2009; CIR, 2012). These chemicals did not induce chromosomal damage in Chinese hamster or Tunstall Wistar rat bone marrow cells after acute oral doses ranged between 250 and 3400 mg/kg bw (HERA, 2009).
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Based on the data available, the chemicals of this group are not considered to cause reproductive or developmental toxicity. In a two-generation reproductive and developmental toxicity study, the chemical (C14-15EO7) was administered in the diet of Charles River CD rats (n=25/sex/group, at doses of 0, 25, 50 or 250 mg/kg bw/day). The NOAEL for reproductive toxicity was established as 250 mg/kg bw/day (or 0.5 % of the diet). No treatment related effects were reported with respect to fertility, gestation, or viability indices or other histopathological parameters. The NOAEL for developmental toxicity was established as 50 mg/kg bw/day based on reduced pup body weights in the second generation at 250 mg/kg bw/day (HERA 2009; CIR, 2012). In a two-generation reproductive and developmental toxicity study, the chemical (C9-11EO6) was applied dermally to Fischer 344 rats (n=30/sex/group, at doses of 0,10, 100 or 250 mg/kg bw/day, 3 times a week except mating periods). No treatment related effects were reported with respect to mating, fertility, gestation, or viability indices and mean gestational length in both generations. No effects on testicular weights or sperm counts were observed in the male rats. The NOAEL for reproductive toxicity was established as 250 mg/kg bw/day. The NOAEL for developmental toxicity was >250 mg/kg bw/day, based on no effects seen in growth and development in the offspring up to the highest dose tested (HERA 2009; CIR, 2012). In a two generation study, the chemical (C12EO6) was administered in the diet of female rabbits at doses of 0, 50, 100 or 200 mg/kg bw/day from gestation days 2 to 16. Ataxia and a slight decrease in body weight were observed at 100 and 200 mg/kg bw/day, indicating maternal toxicity. Nine rabbits in the control group and 31 in the treatment groups died during the study (details not available). There were no treatment related effects on implantations, number of live foetuses and spontaneous abortions. The NOAEL for maternal toxicity was reported as >50 mg/kg bw/day (HERA, 2009). Although certain short chain monoethylene glycol ethers such as 2-ethoxyethanol (CAS No. 110-80-5) are known reproductive toxicants, the ability of the glycol ethers to cause testicular toxicity decreases with increasing chain length, with effects not observed with chain lengths greater than C2 (OECD, 2004).

Acute Toxicity	Based on the available animal (rats, mice and guinea pigs) studies, the chemicals in this group are expected to have low to moderate acute oral toxicity (REACHa-h; OECD, 2005; HERA, 2009; CIR, 2012). The LD50 in rats ranged from 600 mg/kg bw to greater than 20 g/kg bw. Observed sublethal effects for the chemical with the highest toxicity (C15–16 and EO10) included diarrhoea, pilo-erection, ataxia, abnormal posture, difficult laboured breathing, salivation, lacrimation, bloody noses and lethargy. Data from HERA assessment studies show that the chemicals with ethoxylate chains (EO) between 5 and 15 units were more toxic by the oral route than those with less than 4 or greater than 21 units. No relationship between the alcohol chain length and toxicity was observed (HERA, 2009). The chemicals of this group exhibit low acute dermal and inhalation toxicity. The chemicals (C9 to C15 with 3–13 EO units) were of low acute toxicity in rats and rabbits following dermal exposure. The LD50 ranged from 2000 to 5000 mg/kg bw. Sub-clinical effects included wet appearance of the fur, little or no urine, laboured breathing, lethargy, diarrhoea, ataxia, muscle tremours and decreased activity. There was no relationship between the alcohol chain length or number of ethoxylate groups and toxicity. Very high dermal doses of the chemicals (>16000 mg/kg bw) applied dermally for 24 hours in rabbits led to severe skin irritation, ataxia and lung lesions (HERA, 2009; CIR, 2012). In a guideline study (Test Guideline (TG) 403), a single static inhalation exposure to substantially saturated vapour (equivalent to 131.58 ppm - calculated) of C6EO1-2.5 (CAS No. 112-59-4), resulted in no mortality or other signs of inhalation toxicity in Sprague- Dawley (SD) rats (REACHa).
Irritation	The chemicals in this group are reported to be moderate to severe skin irritants in animal studies. The degree of irritation was reported to be dependent on the type of patch (occluded vs semi-occluded), exposure time (ranging from 4 hours up to 4 weeks) and the concentration used. Undiluted chemicals were moderately to severely irritating, 1–10 % was mildly irritating and 0.1 % and 0.5 % were non-irritating. There was also a general trend between the severity of irritation and the degree of ethoxylation. Chemicals with three and less ethoxylate units appeared to be more irritating than chemicals with higher degree of ethoxylation. No trend in irritation potential with respect to the length of carbon chain could be established. Available data indicates that undiluted AEs can produce varying degrees of eye irritation ranging from moderate to severe irritancy. The severity of irritation was found to be concentration dependent, with up to 1 % minimally irritating and concentrations in the range of 1 to 10 % slightly to moderately irritating. In most cases, following exposure, the eyes of the treated animals recovered a few days after exposure. Further tests showed that rinsing the eye 30 seconds after application with tap water may reduce the severity of the effects. No clear relationship could be established between the number of EO units or carbon chain length and eye irritation potency.
Sensitisation	Based on available data, the chemicals in this group are not skin sensitisers.
Health Effects Summary	The chemicals in this group are synthesised from linear alcohols (primary or secondary) or branched alcohols. The commercial AEs may also contain un-reacted alcohol as reaction by-products at about 5 % but with variations between different commercial products (HERA, 2009). Available data on linear and branched chain alcohols show that they have low acute and systemic toxicity and exhibit similar patterns of absorption, metabolism, and excretion to alcohol ethoxylates. They are also shown to have no skin sensitisation potential. A potential for skin and eye irritation exists with alcohols >11 carbon chain length (OECD, 2006; OECD, 2006a).
Key Study/Critical Effect for Screening Criteria	The critical human health effects for risk characterisation are acute oral toxicity and skin and eye irritation. The irritant effects are similar to those produced by other surfactants, and the severity of irritation appears to increase directly with concentration and generally decrease with an increasing number of ethoxylate units.

Ecological Toxicity [2,3]	
Aquatic Toxicity	The 96 h LC50 value for Alcohols, C9-11, ethoxylated with Oncorhynchus mykiss was 5 - 7 mg/L based on nominal concentrations. In the long term toxicity test to Lepomis macrochirus, the NOEC (30 days) was 0.11 – 0.33 mg/L. In the short–term toxicity test to Daphnia magna, the EC50 (48 h) was 2.5 mg/L. In the long term toxicity test to Daphnia magna, the NOEC (21 days) was 0.77 – 1.75 mg/L. In the short–term toxicity test to Pseudokirchneriella subcapitata (green algae), the EC50 (96 h) was 1.4 mg/L. The EC50 (3 h) for microorganisms was 140 mg/L. In a study conducted with two different fish species (bluegill sunfish and fathead minnow) the effects of C14 -15 alcohol ethoxylates (7EO) were determined (Dorn et al., 1995, Shell). In two experiments fish were exposed for 10 d in a laboratory assay and for 30 d in an outdoor stream mesocosm. Effect parameters determined were survival and growth of juvenile bluegills and survival and reproduction of fathead minnows. In the laboratory experiment the NOEC for survival and swimming performance of bluegills and for survival of fathead minnows was 0.16 mg/L. In the stream mesocosm the NOEC for bluegill survival and growth was >0.33 mg/L and for fathead minnow survival 0.28 mg/L. There was an indication of decreased egg laying by fathead minnow in the streams at concentrations of 0.33 mg/L or greater. On the basis of the reported results a worst-case NOEC of 0.16 mg/L is assumed. One publication is available for an alcohol ethoxylate mixture with a chain length of C12 - C13 and approximately 6.5 ethoxy groups (Gillespie et al. 1999). The 21 days flow-through chronic experiment on daphnids is conducted according to the guidelines USEPA-TSCA (U.S. EPA, 1992) and ASTM (1988) and is well documented in the paper. Nevertheless the degree of ethoxylation of the tested mixture described in the paper (6.5 EO) is higher than the degree of ethoxylation described for CAS 68131-39-5 (2.5 EO). The NOEC of 0.77 mg/L for reproduction can be used for read-across.
Determination of PNEC aquatic	A PNECaquatic of 11 µg/L was calculated using the lowest chronic endpoint of NOEC of 0.11 mg/L for Daphnia magna. An assessment factor of 10 was used.
Current Regulatory Controls [1]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No specific exposure standards are available.
International Occupational Exposure Standards	No specific exposure standards are available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	Trigger values for freshwater (95% species) (ANZECC 2000): Alcohol ethoxyolated sulfate (AES) = 650 μgL^{-1} Alcohol ethoxylated surfactants (AE) = 140 μgL^{-1}
PBT Assessment	
P/vP Criteria fulfilled?	No. These chemicals were found to be readily biodegradable. Thus, it does not meet the screening criteria for persistence.
B/vB criteria fulfilled?	No. Bioaccumulation in organisms is expected to be negligible, due to biotransformation and excretion of alcohol ethoxylates.
T criteria fulfilled?	No. The NOECs from the chronic aquatic toxicity data are >0.01 mg/L, hence does not meet the screening criteria for toxicity.

Overall conclusion	Not PBT
Revised	January 2019

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier II Assessment for Ethoxylates of aliphatic alcohols (>C6):, Retrieved 2019: https://www.nicnas.gov.au
2. ECHA REACH, Alcohols, C9-11 ethoxylated, < 2.5 EO, Retrieved 2017: https://echa.europa.eu/information-on-chemicals/registered-substances
3. ECHA REACH, Alcohols, C12-15 ethoxylated, Retrieved 2017: https://echa.europa.eu/information-on-chemicals/registered-substances

AECOM Imagine it. Delivered.

Toxicity Summary - Alcohols, C6-12, ethoxylated propoxylated

Chemical and Physical Properties[1]	
CAS number	68937-66-6
Molecular formula	No data available.
Molecular weight	No data available.
Solubility in water	Soluble in water
Melting point	-3 °C
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	Yellow liquid, mild odour
Overview	Principle Route of Exposure: Eye or skin contact, inhalation Causes severe eye irritation which may damage tissue. Causes skin irritation. Harmful if swallowed.
Environmental Fate[1]	
Soil/Water/Air	This substance is expected to be readily biodegradable (60% @ 28d) (similar substances). Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8, it is not expected to be bioaccumulative. Mobility in soil: KOC = >4
Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	No data available to indicate product or components present at greater than 0.1% are chronic health hazards.
Carcinogenicity	Did not show carcinogenic effects in animal experiments (similar substances)
Mutagenicity/ Genotoxicity	In vitro tests did not show mutagenic effects. In vivo tests did not show mutagenic effects. (similar substances)
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Animal testing did not show any effects on fertility.
Acute Toxicity	LD50 Oral: 600 mg/kg (Rat) (similar substance) LD50 Dermal: > 5200 mg/kg (Rabbit) (similar substance) LD50 Inhalation: > 0.22 mg/L (saturated concentration) (Rat) (similar substance)
Irritation	May cause mild respiratory irritation. Causes severe eye irritation which may damage tissue. Causes skin irritation.
Sensitisation	Did not cause sensitization on laboratory animals (guinea pig) (similar substances)
Health Effects Summary	Causes severe eye irritation which may damage tissue. Causes skin irritation. Harmful if swallowed.
Key Study/Critical Effect for Screening Criteria	
Ecological Toxicity [1]	

Aquatic Toxicity	Toxicity to fish: LC50 (96h) 0.59 mg/L (Pleuronectes platessa) (similar substance) LC50 (96) 1.4 mg/L (Pimephales promelas) (similar substance) NOEC 4.4 mg/L (Pimephales promelas, juvenile) Toxicity to invertebrates: EC50 (48h) 0.14 mg/L (Daphnia magna) (similar substance) EC50 (48h) 0.39 mg/L (Cerodaphnia dubia) (similar substance) Toxicity to algae: EC50 (72h) 0.75 mg/L (Pseudokirchnerella subcapitata) (similar substance) EC50 (96h) 0.7 mg/L (Pseudokirchneriella subapitata) (similar substance) CD10 8 mg/L (Pseudokirchneriella subapitata) EC10 2 mg/L (Brachionus calyciflorus) Toxicity to microorganisms: EC50 (48h) 0.39 mg/L (Cerodaphnia dubia) (similar substance)
Determination of PNEC aquatic	On the basis that the data consists of short term results from three trophic levels, an assessment factor of 1000 has been applied to the lowest reported effect concentration of 0.14 mg/L for Daphnia. The PNEC aquatic is 0.14 µg/L.
Current Regulatory Controls [1]	
Australian Hazard Classification	H302 - Harmful if swallowed H315 - Causes skin irritation H318 - Causes serious eye damage
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Expected to be readily biodegradable based on similar substances.
B/vB criteria fulfilled?	No. Based on an estimated log Kow value of 4.3 – 5.36, and BCF value of 1.1 – 1.8, it is not expected to be bioaccumulative.
T criteria fulfilled?	Not applicable. Chronic toxicity data >1 mg/L in invertebrates, thus sodium hydroxide does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. Redacted

AECOM Imagine it. Delivered.

Toxicity Summary - Amides, tall-oil fatty, N,N-bis(hydroxyethyl)

Chemical and Physical Properties [1,2]	
CAS number	68155-20-4
Molecular formula	UVCB
Molecular weight	370 (typical C18 monounsaturated)
Solubility in water	Dispersible
Melting point	<25 °C (liquid)
Boiling point	>300 °C (estimated)
Vapour pressure	$<1.0\times10^{-10}$ (estimated)
Henrys law constant	$<1.0\times10^{-10}$ (estimated)
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	Liquid
Overview	Non-confidential information in the IUR indicated that the industrial processing and uses of the chemical include other basic organic chemical manufacturing as surface active agents and intermediates; pesticide and other agricultural chemical manufacturing as surface active agents; soap and cleaning compound manufacturing as surface active agents; support activities for mining as surface active agents; and petrochemical manufacturing as surface active agents. Non-confidential commercial and consumer uses of this chemical include lubricants, greases and fuel additives.
Environmental Fate [1,2]	
Soil/Water/Air	The members of the fatty nitrogen derived amides category are long-chain alkyl substituted amides used in commercial product mixtures. The category consists of three subcategories: Subcategory I, fatty acid amides; Subcategory II, fatty alkanolamides; and Subcategory III, fatty acid reaction products with amines. For the purpose of this discussion only, a one-member Subcategory, Subcategory IV, which contains CASRN, 61790-63-4, has been considered as part of Subcategory II. The components of Subcategory I are solids possessing low vapor pressure and low water solubility. The substances in Subcategory II contain solids and liquids with negligible to low vapor pressure and tend to be dispersible in water. The substances in Subcategory III also contain solids and liquids possessing negligible to low vapor pressure that tend to be dispersible in water. The fatty acid amides (Subcategory I) and the fatty acid reaction products with amines (Subcategory III) are expected to possess low mobility in soil. The fatty alkanolamides (Subcategory II) are expected to possess moderate to high mobility in soil. Volatilization is low to moderate for the fatty acid amides and low for the fatty alkanolamides and the fatty acid reaction products with amines. The rate of hydrolysis is considered negligible for all category members. The rate of atmospheric photooxidation is considered moderate to rapid for members of each subcategory; however, this is not expected to be an important environmental fate process since these substances are not expected to exist in the vapor phase in the atmosphere. The overall weight of evidence suggests that the members of the fatty nitrogen derived amides category should possess low persistence (P1) and low bioaccumulation potential (B1) with the exception of two members of subcategory III. Fatty acids, tall-oil, reaction products with tetraethylenepentamine and fatty acids, tall-oil, reaction products with polyethylenepolyamines are expected to possess low persistence (P1), but moderate bioaccumulation potential (B2).
Human Health Toxicity Summary [1,2, 3]	

Chronic Repeated Dose Toxicity	Based on read-across from CAS 120-40-1, an oral repeated dose toxicity study reported NOEL = 0.1% which corresponds to 50 mg/kg/day. No rats died as a result of being treated with the test substance. Two males treated with diet containing 1.0% test substance were euthanized on Days 23 and 58 because of weight loss and respiratory distress. Extensive lung abscess formation was seen at autopsy and bronchopneumonia was confirmed histologically. Growth was inhibited significantly in males and females at and above the 0.5% dietary concentration. Food intake was reduced at all dietary levels except 0.1%, and was attributed to an effect of the test substance on palatability of the diet. The rats in the palatability study showed exclusive preference to the control feed than the treated feed, virtually no test diet was consumed at any dietary levels incorporated. Hematological examination revealed statistically significant reductions in hemoglobin levels and red cell counts in females at the 2.0 and 1.0% dietary concentration and in hemoglobin levels in males at the 2.0% level. Examination of the femoral bone marrow smears showed not deviation from normality. Serum chemistry revealed significantly high serum levels of glutamic-oxaloacetic transaminase in females at the 0.5% level and higher, but only at the 0.5% level in males. Urinalysis was comparable across all groups for males and females. Gross examinations were unremarkable. Statistically significant increases in relative kidney weight in all test groups except at 0.1% in females and at 2.0 and 1.0% in males; and increases in relative liver weight in females at 2.0 and 1.0% were seen. These were attributed to the decreases in body weight. Types and incidence of pathological lesions seen histologically were comparable in control and test groups. Gonads were examined histologically, thus this study meets SIDS requirements for a reproductive screen.
Carcinogenicity	Not regarded as carcinogenic.
Mutagenicity/ Genotoxicity	Based on read-across from CAS 120-40-1, the test substance did not induce reverse mutations in the tested strains of Salmonella typhimurium in the presence or absence of S-9 activation.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Based on read-across from CAS 68603-42-9, the results from a developmental toxicity study showed that repeated oral administration of COMPERLAN KD to pregnant rats on day 6 through 15 of gestation, caused no symptoms of cumulative toxicity up to a dose level of 1000 mg/kg/day. With the exception of salivation and propulsion of the head during the dose administration, there were no treatment-related effects. Also, COMPERLAN KD does not reveal any embryotoxic or teratogenic potential at dose levels up to 1000 mg/kg/day (author of the report).
Acute Toxicity	Acute oral and dermal toxicities of CASRN 68140-00-1 in rat and rabbit, respectively, are low. Based on read-across from CAS 68140-00-1, an oral acute toxicity test on rats reported LD50 > 5 g/kg. All animals survived the 8-day observation period and no adverse effects were observed. With respect to the determined LD50 value, it is assumed that the LD50 value for female rats also exceeds the limit dose of > 2000 mg/kg body weight. In a dermal acute toxicity test on rabbits, LD50 > 2 g/kg was reported. All animals survived. All animals appeared normal through day 14. Two females that had abraded skin lost weight (0.01 and 0.25 kg) over the 14-day post-exposure period. All remaining rabbits gained weight through day 14. Swiss-Webster mice (4 males/dose) were administered “Alkanolamide #1”, identified in the robust summary as CASRN 68144-20-4, via whole body exposure for 3 hours. Doses were 86- 219 mg/m3 (0.086 – 0.219 mg/L). Animals were observed for several days. No mortality was observed. LC50 > 0.219 mg/L
Irritation	The test article produced sensory irritation later in the exposure at low concentrations. Pulmonary irritation also occurred later in these exposures.
Sensitisation	Did not cause sensitization on laboratory animals (similar substances)

Health Effects Summary	Acute oral and dermal toxicities of CASRN 68140-00-1 in rat and rabbit, respectively, are low. CASRNs 142-78-9 and 68140-00-1 were negative for gene mutations in bacteria in vitro. No data are available for the repeated-dose/reproductive/developmental toxicity and genetic toxicity (chromosomal aberrations) endpoints. The repeated-dose/reproductive/developmental toxicity and genetic toxicity (chromosomal aberrations) endpoints are identified as data gaps
Key Study/Critical Effect for Screening Criteria	
Ecological Toxicity [1, 3]	
Aquatic Toxicity	Based on read-across for CAS No: 68603-42-9 Daphnia: EC50 (24-hour): 3.3 mg active matter/l Daphnia: 48-hour LC50 = 2.15 and 2.64 mg/l Based on read-across for CAS No: 112-84-5 The experiment measured the survival and reproduction of Daphnia magna over a 21-day exposure to the test and control substances. Daphnids were cultured in the laboratory using Elendt M7 medium and a daily feeding regiment of green algal cells (Chlorella vulgaris). Four experimental groups: control (Elendt M7 medium), solvent control (0.1 ml methanol/l), 33 µg/l, and 100 µg/l (nominal concentrations) were used in a static-renewal exposure system. All test solutions were prepared with Elendt M7 medium. Replicate test vessels consisted of 4 oz glass bottles containing 100 ml of test solution. There were 10 replicates per experimental group. On the day of test initiation, neonate daphnids were removed from cultures and placed in a crystallizing dish containing Elendt M7 medium. One daphnid was placed in each replicate test vessel, and each vessel was randomly placed in the testing area. Light intensity was not measured, but ambient laboratory lighting was provided with a photoperiod of 16 hours light/8 hours dark. Each day, test solutions were renewed, and the daphnids were fed 1.7 x 10(5) cells/ml of Chlorella vulgaris. Adult survival and reproduction was assessed each day and neonates were removed daily. The pH, dissolved oxygen (DO) and total hardness (as mg/l CaCO(3)) were measured on test days 0, 1, every Tuesday and Friday and on day 21. Means and ranges for temperature, water pH, DO and total hardness were 19.7 °C (14.5 - 25.0 °C), 7.6 (7.2 - 8.1), 8.2 mg/l (4.5 - 9.3 mg/l) and 245 mg/l (234 - 256 mg/l) as CaCO(3), respectively. Concentrations of the test substance in exposure solutions were measured on test days 0, 1, 5, 9, 12, 16 and 19 in both the old and the new solutions. Effect concentrations were based on mean measured concentrations. 21 d NOEC = 0.08 mg/L
Determination of PNEC aquatic	Applying an assessment factor of 1000 to the NOEC (0.08 mg/l) gives a PNEC of 0.08 µg/l.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Expected to be readily biodegradable based on similar substances.

B/vB criteria fulfilled?	No. Based on BAF = 108 and log Kow of 3 (estimated)
T criteria fulfilled?	No. Acute toxicity data was >1 mg/L.
Overall conclusion	Not PBT
Revised	January 2019

References

1. OECD, Amides, tall-oil fatty, N,N-bis(hydroxyethyl), Retrieved 2019: http://www.echemportal.org
2. USEPA Hazard Characterization Document, Fatty Nitrogen Derived (FND) Amides Category, September 2010
3. Redacted

AECOM Imagine it. Delivered.

Toxicity Summary - Amine oxides, cocoalkyldimethyl

Chemical and Physical Properties	
CAS number	61788-90-7
Molecular formula	CH3.(CH2)R.N(CH3)2:O, where R is 9-17
Molecular weight	237 (70% C12: 30% C14) (molecular weight will vary depending on structure)
Solubility in water	409.5 g/L
Melting point	Average: 130.5
Boiling point	Decomposes before boiling
Vapour pressure	Predicted vapour pressure values are < 4.6E-7 hPa
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	No data available.
Overview	Surfactants known as amine oxides (AO) contain even numbered linear alkyl chains ranging from C8 to C20. Also known as fatty alkyl dimethyl AOs, they are usually produced by reacting alkyl dimethyl amines with hydrogen peroxide in water. The AOs are produced, transported and used in water solutions, typically at a 25-35% activity level. The AOs are produced and used either as single chain length substances (e.g., C12) or as a mixture of different chain lengths (e.g., C12 to C18). All of the substances in this category are surfactants, consisting of a polar “head” (the amine oxide) and a relatively inert, hydrophobic “tail” (the long alkyl substituent). AOs are used in cleaning and personal care products as foam stabilizers, thickeners, emollients, emulsifying and conditioning agents. Primary uses are in liquid hard surface cleaners, laundry and dishwashing detergents, shampoos and hair conditioning products.
Environmental Fate[1]	
Soil/Water/Air	AOs are highly water soluble (C10-16 AO = 409.5g/L). AO is fully biodegradable under both aerobic and anaerobic conditions and is effectively removed during wastewater sewer transport (“pipe loss” >90%) and in biological wastewater treatment (~98%). It has low potential for bioaccumulation (BCF <87 L/kg). These characteristics help to minimize the potential for environmental exposure, and for indirect human exposures via drinking water and/or fish consumption.
Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	In four repeated-dose studies with rats and mice exposed to AO via oral and dermal routes (all with CAS No 70592-80-2), three dermal studies were designed to assess the effect of repeated exposure on skin at maximum doses of 1.5 mg AO/kg-bw/day. Higher doses were tested in a 90-day dietary study with rabbits. No treatment-related clinical chemistry, hematology and histopathological changes were observed. In these studies, LOAELs ranged from 87 to 150 mg AO/kg bw/day with the highest oral NOAEL below the lowest LOAEL as 80 mg AO/kg bw/day. Signs of toxicity observed in the oral study included suppressed mean body weight gain, lenticular opacities and diarrhoea; in the dermal studies, local dermal irritation was evident.
Carcinogenicity	The carcinogenic potential of amine oxides has been thoroughly investigated in three carcinogenicity studies in rats or mice by dermal, dietary, or drinking water routes. In all cases the substances demonstrated no evidence of a carcinogenic response.

Mutagenicity/ Genotoxicity	In five in vitro bacterial (Salmonella) mutagenicity studies, AO shows no evidence of mutagenicity either with or without S9 metabolic activation at concentrations up to 250 ug/plate (higher concentrations caused cytotoxicity). Three in vivo studies investigated clastogenic effects on a close structural analog of the category, 1-(methyldodecyl)dimethylamine-N-oxide including: a mouse micronucleus, a Chinese hamster micronucleus and a Chinese hamster cytogenetics study. These studies were all negative showing no increase in micronuclei or chromosome aberrations. An in vivo mouse dominant lethal assay showed no evidence of heritable effects. Two AOs (CAS No 1643-20-5 and CAS No 3332-27-2) were negative in an in vitro cell transformation assay tested at concentrations up to 20 ug/ml.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No evidence of reproductive toxicity or fertility effects was observed in a study in which rats were given dietary doses of AO in the diet over two generations (CAS No 1643-20-5). No macroscopic or histopathological changes were attributable to treatment with the test substance. The maternal NOAEL from this reproductive study was >40 mg AO/kg bw/day, which was the highest dose tested. At all treatment levels, the rate of bodyweight gain for the F1 and F2 offspring was reduced during the lactation period, however, this reduction was not greater than 10%. This effect appeared to be dose-related, but was not statistically significant until after weaning in the mid and high dose levels. This was not considered an adverse effect since the body weight change only reached statistical significance when the rat pups were getting the majority of their calories from solid food (Developmental NOAEL >40 mg/kg bw/day). In three developmental toxicity studies via gavage in rats and rabbits (with CAS No 1643-20-5 & 70592-80-2), effects such as decreased fetal weight or delayed ossification, were most often observed only at maternally toxic doses and were associated with the irritation effects of AO on the gastrointestinal tract. No decreases in litter size, no changes in litter parameters, no malformations or significant differences in skeletal defects were observed at oral doses up to 25 mg/kg bw/day in rats (based on decreased fetal weight at 100 mg/kg bw/day) and >160 mg/kg bw/day in rabbits (the highest dose tested).
Acute Toxicity	In rat oral acute toxicity limit tests, no deaths occurred at single doses of 600 mg C10-16 AO/kg bw or less (for CAS No 70592-80-2). In multi-dose studies, acute oral LD50 values for rats ranged from 846 mg AO/kg bw to 3873 mg AO/kg bw (both values for CAS No 61788-90-7), with several other AO's having rat oral LD50's falling within this range. In single dose acute dermal toxicity limit tests, no deaths occurred at a dose of 520 mg AO/kg bw (CAS No 70592-80-2). This dose was equivalent to 2 mL/kg of a 30% formulation. There were no deaths observed in a rat acute inhalation study to aerosol droplets of a consumer product providing a dose of 0.016 mg AO/L.
Irritation	In a series of studies on rabbits, AO's of varying chain length showed consistent results and all 1) were not irritating to the skin or eyes at low concentrations (1%), 2) were moderately irritating at 5%, and 3) more severely irritating when tested as produced (e.g., ~30% aqueous solutions). In studies that included rinsing, eye irritation effects diminished with rinsing after 30 seconds of exposure and were slight with rinsing after 4 seconds of exposure. In Draize rabbit eye irritation tests using ~30% AO solutions, rabbits experienced severe to moderate irritation. (The maximum concentration of AO is 10% active in consumer products.) Accidental eye exposure in manufacturing employee incidents and consumer incidents established that eye irritation effects of exposure during manufacturing and use of products containing AO and other surfactants are moderate, transient and reversible
Sensitisation	There is no indication of skin sensitization for the AO category based on the available animal and human data.
Health Effects Summary	The chemicals in this category present properties indicating a hazard for human health (skin and eye irritation). However, these hazards do not warrant further work as they are related to reversible, transient and non-lasting effects. Nevertheless, these hazards should be noted by chemical safety professionals and users.
Key Study/Critical Effect for Screening Criteria	Skin and eye irritation.

Ecological Toxicity [1,2,3]	
Aquatic Toxicity	Extensive aquatic toxicity data are available for commercially representative amine oxides (C10 to C18) that are single chain length as well as mixtures. Based on hazard data, freshwater green algae are considered the most sensitive species, for acute and chronic endpoints. Acute toxicity is affected by chain length for fish and invertebrates. Chain length affects hydrophobicity, wherein longer chain-lengths increase the rate of uptake and decrease depuration. All but four supporting AO's have been tested for acute toxicity in fish, daphnia, and algae. The range of acute LC50/EC50/ErC50 values based on a review of the aquatic toxicity data on AO were 0.60-32 mg/L for fish, 0.50-10.8 mg/L for Daphnia magna and 0.010-5.30 mg/L for algae. Chronic toxicity data were normalized to a chain length of 12.9 carbon atoms, as this average chain length represents the largest volume product for North America (CAS No 70952-80-2). Chronic toxicity (NOEC, EC20) for an amine oxide of average chain length of C12.9 ranged as follows for the different trophic levels: 0.010-1.72 mg/L for algae, 0.28 mg/L for Daphnia (flow through) and 0.31 mg/L for fish (flow through). These are based on geometric mean values, and a dataset of 21 chronic toxicity studies. Based on a chronic periphyton microcosm bioassay that included 110 taxa of algae (most sensitive species), a NOEC value of 0.050 mg/L was derived when normalized for a C12.9 amine oxide.
Determination of PNEC aquatic	Chronic toxicity values are reported for fish, aquatic invertebrates, and freshwater algae. Since there is valid chronic toxicity data for three trophic levels, an assessment factor of 10 is used (in accordance with EU guidance). Based on the NOEC for freshwater algae (the most sensitive species), the aquatic PNEC is 0.01 µg/L.
Current Regulatory Controls [4]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. AOs are highly removed by conventional sewage treatment systems and biodegrade rapidly and completely under aerobic and anaerobic conditions.
B/vB criteria fulfilled?	No. BCFWIN predictions using the calculated logKow value of < 2.7 as input parameters (derived for C10-16 AO), calculated bioconcentration factor < 87 for C12-14 AO (The Procter & Gamble Company, 2002C). Thus the potential for bioaccumulation of AOs in aquatic organisms is considered to be low.
T criteria fulfilled?	Yes. Chronic toxicity data < 1 mg/L fish, aquatic invertebrate and/or algae, thus AO does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. OECD (2001) SIDS Initial Assessment Profile for Amine Oxides (AO)

AECOM Imagine it. Delivered.

Toxicity Summary - Benzaldehyde

Chemical and Physical Properties[1,2,3]	
CAS number	100-52-7
Molecular formula	C7H6O
Molecular weight	106.12
Solubility in water	6.55 g/L at 25°C
Melting point	-26°C
Boiling point	179.2°C
Vapour pressure	0.130 kPa (0.97 mmHg) at 20°C
Henrys law constant	2.85 Pa.m³.mol-1 @ 25 °C
Explosive potential	Non explosive
Flammability potential	Non flammable
Colour/Form	Colourless or yellow liquid with an almond-like odour.
Overview	Benzaldehyde is a colourless liquid that becomes yellowish with age. It smells a little like almond and has a burning, aromatic taste. Benzaldehyde is very soluble in water. Benzaldehyde occurs naturally in plants. It can be formed in the atmosphere from the reaction of some chemicals with sunlight. It has been detected in air associated with volcanoes. Benzaldehyde is an important commercial chemical that is used to make other chemicals. It is also used as a preservative in cosmetics, personal care products, food and select car detailing products. It is used as a solvent for oils, flavouring, and in synthetic perfumes. It may be a tobacco additive. It was formerly used as an insecticide.
Environmental Fate [2,3]	
Soil/Water/Air	The test substance is readily biodegradable. The test substance was shown to degrade under influence of light with a DT50 of 9.4 hours. In addition under anaerobic conditions complete biodegradation is expected. As the logKow is 1.4, the potential for bioaccumulation and sorption of the test substance is considered to be low. The Henry Constant was calculated to be 2.85 Pa m^3/mol. A calculation with Simple Treat shows that the test substance will degrade in the Sewage Treatment Plant for > 88% with at maximum about 12% to end up in the water compartment.

Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	Based on the data available, the chemical is not considered to cause serious damage to health from repeated oral and inhalation exposure. In a repeated dose oral toxicity study, Fischer rats (male/female, 10/sex/dose) were administered the chemical by oral gavage at doses of 0, 50, 100, 200, 400 or 800 mg/kg bw/day, five days a week, for 13 weeks. Mortalities and histopathological changes including lesions in the brain (degeneration and necrosis of the cerebellum and necrosis in the hippocampus), renal tubular necrosis, hyperplasia and/or hyperkeratosis of the forestomach, and degeneration of the liver were observed in both sexes at the highest tested dose level. Depressed body weights (26 % lower than controls) were also observed for male rats at this dose. A no observed adverse effect level (NOAEL) of 400 mg/kg bw/day was established (NTP, 1990; OECD, 2002; CIR, 2006; REACH). A similar repeated dose oral toxicity study on B6C3F1 mice (male/female, 10/sex/dose) was also conducted. The mice were administered the chemical by oral gavage at doses of 0, 75, 150, 300, 600 or 1200 mg/kg bw/day, five days a week, for 13 weeks. Within the first week of dosing, 9/10 males and 1/10 females died at the highest tested dose. Mild to moderate renal tubular degeneration in all males was observed in the high dose group and 1/10 males in the 600 mg/kg/day group. Depressed body weights (9 % lower than controls) were also observed for the males at 600 mg/kg bw/day. The NOAEL was determined to be 300 mg/kg bw/day for male mice and 600 mg/kg bw/day for female mice (NTP, 1990; OECD, 2002; CIR, 2006; REACH). In another repeated dose oral toxicity study, similar to OECD TG 408, groups of Osborne–Mendel rats (male/female, five/sex/dose) were fed a powdered diet containing the chemical at concentrations of 1000 ppm for 28 weeks, or 10000 ppm (approximately 500 mg/kg bw/day) daily for 16 weeks. No effects on body weight or haematological parameters and no macroscopic/microscopic changes in selected organs were noted at 10000 ppm (CIR, 2006; REACH). In a repeated dose inhalation toxicity study conducted similarly to OECD TG 412, groups of Sprague Dawley (SD) rats (male/female, 14/sex/dose) were exposed (whole body) to the vapours of the chemical at 0, 500, 750 and 1000 ppm, six hours a day for 14 days. Significant reduction in body weight was observed for all males but only at 1000 ppm for females. Mortalities occurred in the two higher dose groups. All groups exhibited clinical toxicity symptoms including reduced motor activity, hypothermia, respiratory problems and nasal and ocular irritation. With increased concentrations, the severity of nasal and ocular irritation increased. At the two highest doses, the rats displayed aggressive behaviour and central nervous system symptoms (tremors, piloerection, diuresis, seizures and sensitivity to noise). The most prominent histopathological observation was goblet cell metaplasia in the respiratory epithelial lining of the nasal septum, which was found in males at doses 500 and 1000 ppm, but not in females. A no observed adverse effect concentration (NOAEC) could not be determined due to the clinical observations (indicative of neurotoxicity), hypothermia, and goblet cell metaplasia which were seen at concentrations of 500 ppm and above. The lowest observed adverse effect concentration (LOAEC) was reported to be 500 ppm in this study (CIR, 2006; HSDB; REACH). In another repeated dose inhalation toxicity study with limited documentation (non-guideline), rats were exposed to the chemical at 186 ppm (803 mg/m^3), four hours a day, five days a week for two weeks. Respiratory irritation was observed during exposure. No other effects were reported (EC, 2000; OECD 2002).

Carcinogenicity	Although the chemical has been reported to have 'some evidence of carcinogenic activity' in B6C3F1 mice, there was 'no evidence of carcinogenic activity' in Fischer 344 rats receiving 200 or 400 mg/kg bw/day (NTP, 1990). It was further concluded that the increased incidences of pancreatic acinar cell neoplasms in male rats and squamous cell papillomas of the forestomach in mice were probably due to the high concentrations of corn oil (mild irritant and mitogen) used as a vehicle in these studies (US EPA, 2001). The chemical is also considered not to have mutagenic or genotoxic potential (see Genotoxicity). Therefore, the chemical is not considered to have carcinogenic potential. In a combined chronic toxicity–carcinogenicity study (OECD TG 451), groups of eight-week-old Fischer 344 rats (male/female, 50/sex/dose) were administered (gavage) the chemical in corn oil at doses of 200 or 400 mg/kg bw, five days a week for two years. At the highest dose, mortality in male rats was significantly higher than the controls. No dose-related effects on body weight and clinical signs were observed. As squamous cell papillomas of the forestomach were seen in only two female rats in the high dose group and there was a lack of supporting hyperplasia, these were not considered to be due to the administration of the chemical. Significant increases in the incidences of pancreatic acinar cell hyperplasia and tumours were observed in male rats only at the high dose. Unpublished National Toxicology Program (NTP) studies indicated that pancreatic acinar cell tumours found in rats gavaged with corn oil were not autunomous as these tumours failed to transplant. Therefore, based on the facts that these tumours failed to transplant, were present in variable numbers in control animals, and increased only at the high dose, it was concluded that pancreatic acinar cell hyperplasia and tumours were not considered as evidence of carcinogenic activity for the chemical (NTP, 1990; EC, 2000; HSDB; REACH). It was further concluded that the increased incidence of tumours specific to male rats in this study was probably due to the use of corn oil as a vehicle in this study (US EPA, 2001). In the same carcinogenicity study, groups of eight-week-old B6C3F1 mice (male and female, 50/sex/dose) were administered (gavage) the chemical in corn oil at doses of 200 or 400 mg/kg bw (in males), 300 or 600 mg/kg bw (in females), five days a week for two years. Although no significant differences in mean body weights and survival were observed between any groups of mice, effects were noted in the forestomach of mice. The incidences of uncommonly occurring squamous cell papillomas of the forestomach in both exposure groups were significantly greater as compared to the controls (male: vehicle control, 1/50; low dose, 2/50; high dose, 5/50; female: 0/50; 5/50; 6/50). The increased incidences of papillomas were accompanied by significantly increased incidences of focal hyperplasia in the forestomach in both sexes of the 400 mg/kg bw group and in females of the 200 mg/kg bw group, compared with vehicle controls. The NTP considered that the increase in papillomas was due to a concurrent increase in hyperplasia following treatment with the chemical and concluded that there was 'some evidence of carcinogenicity' in mice. It was also concluded male and female mice might have been able to tolerate higher doses (NTP, 1990; REACH).

Mutagenicity/ Genotoxicity	Overall, the data indicate that the chemical has no mutagenic or genotoxic potential. Although there is no mutagenic activity in bacterial systems, the chemical does have weak clastogenic effects in some mammalian cell assays. There are also no in vivo data available. The chemical gave negative results in several in vitro bacterial reverse mutation assays with Salmonella typhimurium at concentrations up to 3333 mg/plate. Induction of chromosomal aberrations was also not observed in Chinese hamster ovary (CHO) cells, treated with the chemical up to 500 mg/mL in the absence of S9 or with up to 1600 ug/mL with S9 (NTP, 1990; REACH). In an in vitro chromosomal aberration assay (OECD TG 473) in the Chinese hamster cell line B241, a significant percentage (13 %; 21/162) of the cells displayed abnormalities following exposure to a concentration of 5.3 nM of the chemical for 24 hours (CIR, 2006). Cytogenetic tests with CHO cells reported an increased number of sister chromatid exchanges at doses of 50 mg/ml and 160 mg/ml in the absence of S9 or at 1600 mg/mL with S9 (NTP, 1990; HSDB; REACH). The chemical gave positive results in a mouse lymphoma forward mutation assay (OECD TG 476) with mouse lymphoma L5178Y cells. The concentrations of the chemical tested in this assay were 0, 50, 100, 200, 400, and 800 mg/mL. Although significant increases in mutant fractions were observed at a dose of 400 mg/mL, the positive response was noted to be close to the cytotoxic dose of 640 mg/ml (HSDB; REACH). Negative results were obtained with the chemicals in an in vivo sex-linked recessive lethal test with Drosophila melanogaster (NTP, 1990; OECD, 2002; HSDB; REACH).
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Although limited data are available, the available information indicates that the chemical does not show specific reproductive or developmental toxicity. Benzyl derivatives, including benzaldehyde, have been reported to produce no evidence of reproductive and developmental toxicity during various studies. It was also stated that as benzyl derivatives generally follow similar metabolic pathways, studies conducted on benzyl derivatives provide adequate evidence for benzaldehyde (US EPA, 2001). As part of reviewing the reproductive toxicity and teratogenicity of benzaldehyde and related compounds (benzyl acetate, benzyl alcohol, and benzoic acid and its salts), the Joint Food and Agriculture Organization of the United Nations (FAO) and the World Health Organization (WHO) Expert Committee on Food Additives concluded that 'delayed development and reduced foetal and postnatal pup body weights were observed in developmental toxicity studies in rats, mice, hamsters and rabbits, but only at doses that were toxic to the mother' (CIR, 2006). In a poorly-documented one-generation reproductive toxicity study (non-guideline), male and female rats were administered the chemical by oral gavage at doses of 0 or 5 mg/kg bw/day in oil, once every second day for 32 weeks. Dosing commenced at 75 days before breeding with untreated males; two pregnancies per rat were studied, one at 75 days and one at 180 days. The number of gestating females, number of live-born offspring, pup weights at birth and on postnatal days 7 and 21, and pup viability were recorded. The incidences of pregnancy were reported to be lower for treated females compared with controls. All other parameters were reported to be similar between the treatment and control groups. It was concluded that the treatment did not cause a significant change in any of the reproductive parameters measured. (US EPA, 2001; OECD, 2002; CIR, 2006; REACH).

Acute Toxicity	In an acute oral toxicity study conducted similar to the Organisation for Economic Co-operation and Development (OECD) Test Guideline (TG) 401, groups of male Wistar rats were administered (by gavage) the chemical at doses of 0.8, 1.0, 1.1, 1.2, 1.3, 1.5, and 1.8 mL/kg bw and observed twice daily for 14 days. The acute median lethal dose (LD50) was reported to be 1.43 mL/kg bw (1430 mg/kg bw), with a mortality rate of 100 % (10/10) at the highest tested dose. Observed sub-lethal effects included sedation, staggering, weight loss and a rough coat (REACH). In another acute oral toxicity study with limited data, male and female rats were administered the chemical at doses of 1100–1540 mg/kg bw. An LD50 of 1300 mg/kg bw was established (OECD, 2002; REACH). Although limited information is available, the chemical is likely to have low acute dermal toxicity in animal tests following dermal exposure. In an acute dermal toxicity study in rabbits with limited available data, an LD50 of >1250 mg/kg bw was reported (OECD, 2002; HSDB; REACH). Although limited data are available, the available information indicates that the chemical has moderate acute toxicity in animal tests following inhalation exposure and is recommended for classification. In an acute inhalation toxicity study conducted according to OECD TG 436, Wistar rats (male/female) were exposed (nose only) to the vapours of the chemical at 1 and 5 mg/L for four hours and observed up to 14 days. Clinical effects were observed in most animals following exposure at 5 mg/L including lethargy, flat/hunched postures, ventrolateral recumbency, respiratory difficulties and piloerection. Four animals out of six (one male and three females) died following exposure at 5 mg/L. A median lethal concentration (LC50) of <5mg/L was established, based on mortalities at the highest tested dose (REACH). An increased incidence of respiratory symptoms was noted among workers exposed to vapour of the chemical at atmospheric concentrations of >5 mg/m^3 (OECD, 2002).
Irritation	Although limited data are available, the available information indicates that the chemical is not likely to be a skin irritant. In two skin irritation studies (non-guideline) with limited data, the undiluted chemical (500 mg) was applied to the intact or abraded skin of New Zealand White rabbits for 24 hours with observation up to seven days. Although the exact details were not provided, slight skin irritation was observed (EC, 2000). Although limited data are available, the chemical had been reported to be an eye irritant in animal studies. The available information is not sufficient to support a classification. In an eye irritation study (non-guideline), one drop of the undiluted chemical was applied to the conjunctival sac of a rabbit. Observations were made at one, 24 and 48 hours following application. Immediate irritation effects were noted at one hour and within 24 hours, the anterior portion of the cornea was damaged. The cornea was cleared within 48 hours and only erythema of the conjunctiva and nictitating membrane was noted at this stage. Although the rabbit died on the sixth day, the death was not related to the application of the chemical (CIR, 2006; REACH). In another eye irritation study (non-guideline) with limited data, the chemical (100 µL, concentration not stated) was instilled into the eyes of two rabbits and observed for seven days. The chemical was observed to be slightly irritating to the eyes (REACH).

Sensitisation	Although the chemical has produced skin sensitisation reactions in some tests, based on the weight of evidence, the chemical is not likely to be a skin sensitiser. It is also noted that the chemical is rapidly metabolised to benzoic acid in the skin. Clinical reports of allergy to the chemical are rare and benzoic acid has also been reported not to produce sensitisation in clinical trials in humans (CIR, 2006). In a Magnusson-Kligman skin sensitisation test conducted by the US EPA, guinea pigs (10/group) were initially exposed to the chemical intradermally by a 0.1 mL injection of 3 % chemical in paraffin oil followed by topical application to a patch of skin (occluded for 48 hours) of 15 % chemical in petrolatum. The skin was later challenged by a topical application (occluded for 24 hours) of 7 % chemical in petrolatum on a patch of skin. As the chemical failed to induce erythema in either group, the chemical was concluded not to be a skin sensitiser (CIR, 2006). In a skin sensitisation study that compared four testing methods of 32 fragrance materials on Himalayan guinea pigs, the chemical tested positive for allergenicity in the Draize test (DT), the maximisation test (MT) and Freund's complete adjuvant (FCA) test. The guinea pigs were injected intradermally with the chemical at doses of 0.05 mL (0.1 % solution), 0.1 mL (5 % solution) and 0.05 mL (undiluted) for DT, MT and FCA, respectively (EC, 2000; CIR, 2006; REACH). The chemical was reported to be non-sensitising in the open epicutaneous test (OET) for the same study as reported above. The guinea pigs were exposed to the chemical (undiluted, 0.03 , 0.1, 0.3, 1, 3, 10, or 30 %) at a dose of 0.1 mL on an 8 cm2 area of shaved skin on the flank. Applications were repeated once a day for 21 days and the sites were scored for signs of irritation 24 hours following each treatment. The acute minimum irritating concentration was 10 % and after 21 exposures was 3 %. The animals were challenged with 3 % (minimum irritating concentration for day 21) or an unspecified lower concentration on a 2 cm2 area of shaved skin at two weeks post-exposure. The sites were scored at 24, 48 and 72 hours. No sensitisation effects were observed (CIR, 2006; REACH). In a guinea pig skin maximisation test (OECD TG 406), animals were injected intradermally with 2.7 % of the chemical and followed by three epidermal challenges with 2.1, 2.1 and 0.64 % of the chemical. It was noted that only one intradermal induction was performed and no additional topical induction. Also, there were three challenge reactions instead of one. The time between induction and challenge applications was also not stated. No sensitisation effects were observed (REACH).
Health Effects Summary	The critical health effects for risk characterisation include systemic acute effects (acute toxicity from oral and inhalation exposure).
Key Study/Critical Effect for Screening Criteria	The chemical has been reported to possibly cause respiratory failure, depression of the CNS and convulsions at high concentrations (HSDB). A young woman died after ingesting 50–60 ml (700–2000 mg/kg) of the chemical. At autopsy, yellowish-white pulp with a strong odour of bitter almond was found in the stomach. The time between consumption and death was not specified. In another case, a man had to be revived from near death following ingestion of 40 ml of a derivative of the chemical (o-hydroxybenzaldehyde). Based on these two studies, a lethal oral dose of 600–900 mg/kg bw was calculated for the chemical in the absence of prompt treatment (NTP, 1990; EC, 2000; CIR, 2006). In a case study, workers exposed to vapour of the chemical at atmospheric concentrations of >5 mg/m^3 reported an increased incidence of respiratory symptoms (OECD, 2002). In an inhalation toxicity study, human volunteers were exposed to 4.5 ppm (19.5 mg/m^3) of the chemical for one minute. Irritation of the eyes and upper respiratory tract were observed. In an occupational study, workers exposed to the chemical vapour at atmospheric concentrations of >5 mg/m^3 reported symptoms of slight eye irritation and considerable skin irritation (OECD, 2002).

Ecological Toxicity [2,3]	
Aquatic Toxicity	Acute LC50 for freshwater fish is 1.07 mg/L, freshwater invertebrates is 16.2 mg/L and EC10 for freshwater algae is 20 mg/L. Chronic NOEC for freshwater fish is 0.12 mg/L. The overall acute dataset on aquatic organisms yields a lowest LC50 value for fish of 1.07 mg/L and a NOEC of 0.12 mg/L. However, the substance is readily biodegradable and has a low potential for bioaccumulation. Based on the second ATP to CLP the test substance was classified as Chronic category 3 for aquatic toxicity.
Determination of PNEC aquatic	Ecotoxicological data indicate that benzaldehyde is acutely toxic to fish, harmful to daphnia and very slightly toxic to algae. Using an uncertainty factor of 100 on the lowest LC50 to fish a PNEC (Predicted No Effect Concentration) of 10.7 ug/L is calculated, for aquatic organisms.
Current Regulatory Controls [1]	
Australian Hazard Classification	The chemical is classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia): Harmful if swallowed, Xn; R22 (Acute toxicity)
Australian Occupational Exposure Standards	No specific exposure standards are available.
International Occupational Exposure Standards	The following exposure standards are identified (Galleria Chemica). The chemical has an exposure standard of 5 mg/m^3 time weighted average (TWA) in Bulgaria, Hungary, Latvia and Russia; 10 mg/m^3 in Poland; and 2 ppm in the USA. Short-term exposure limits (STEL) of 4 ppm in the USA and Canada; 10 mg/m^3 in Hungary; and 40 mg/m^3 in Poland have been reported.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Expected to be readily biodegradable.
B/vB criteria fulfilled?	No. As the Log Pow is 1.4 (Log Pow < 4.5), it is not expected to be bioaccumulative.
T criteria fulfilled?	No. The chronic aquatic toxicity of the chemical is > 0.01 mg/L. Hence the substance does not fulfil the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier II Assessment for Benzaldehyde: Retrieved 2019: https://www.nicnas.gov.au
2. ECHA REACH, Benzaldehyde, Retrieved 2019: https://echa.europa.eu/
3. OECD (2002) SIDS Initial Assessment Profile for Benzaldehyde

AECOM Imagine it. Delivered.

Toxicity Summary - Butyl alcohol

Chemical and Physical Properties[1,2,3]	
CAS number	71-36-3
Molecular formula	C4H10O
Molecular weight	74.12
Solubility in water	77 g/l at 20 °C
Melting point	-89.9 °C
Boiling point	117.6 °C
Vapour pressure	0.56 kPa at 20 °C
Henrys law constant	0.054 Pa m³/mol
Explosive potential	Non-explosive
Flammability potential	Flammable
Colour/Form	Colourless liquid with a mildly alcoholic odour.
Overview	n-Butyl alcohol is used as a solvent in surface coatings. These can include varnishes, resins, waxes and gums. It is also used in the manufacture of other butyl compounds. n-Butyl alcohol is a product of fermentation. It has also been detected in the volatiles of foods such as cheese, muskmelon and cooked rice. People that work in industries where products containing n-butyl alcohol are used will have the highest exposure. These could include varnishing of automobiles, painting shops and fabric coating. Exposure will happen by eating foods containing n-butyl alcohol and breathing in fumes from cooking certain foods. n-Butyl alcohol can be found in surface water and air. It is often found in indoor air of new construction. It breaks down in air by reaction with radicals. It is expected to evaporate from soil and water surfaces. n-Butyl alcohol that remains in soil or water will be broken down by microorganisms. It is not expected to build up in aquatic organisms.
Environmental Fate[1]	
Soil/Water/Air	Based on level III fugacity modelling, BA will partition 83.5% in air, 5.9% in soil, 10.6% in water, <0.1% in suspended solids, and <0.1% in biota and in sediment. BA degrades in air by reaction with hydroxyl radicals, having a half-life in air of 1.2 to 2.3 days. The volatilization half-life for BA in water is estimated to be 2.4 hours for streams, 3.9 hours for rivers and 126 days for lakes.
Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	A no observed adverse effect level (NOAEL) of 125 mg/kg bw/day and a lowest observed adverse effect level (LOAEL) of 500 mg/kg bw/day in male and female CD rats was reported based on results from a repeat dose oral study using the chemical (OECD 2001). Groups of male and female rats (30/sex/group) were administered the chemical via gavage at 0, 30, 125 or 500 mg/kg/day for 13 weeks. It was reported that ataxia (impaired muscle coordination) and hypoactivity were observed at the highest dose during the final six weeks of the study. No treatment related effects were reported in the 30 and 125 mg/kg/ bw/day dose groups (OECD 2001). In a non-guideline study, the chemical was applied to the skin of rabbits under occlusive conditions over a period of 21 days. Local effects were reported such as drying of the skin, cracking, wrinkling and exfoliation of the epidermis. However, no systemic toxicity was reported (REACH). In another non-guideline repeat dose dermal study on rabbits, 42 to 55 mL/kg of the chemical applied to the skin of rabbits over four consecutive days resulted in 100 % mortality. However, the same study reported that 30 applications of 20 mL/kg of the chemical over six weeks did not produce any deaths (OECD 2001).

Carcinogenicity	OECD (2001) reported that based on the number of negative mutagenicity and clastogenicity findings, the chemical is not expected to be a carcinogen. A weight of evidence study reported that the chemical is not expected to have carcinogenic potential as it does not contain structural components to support carcinogenicity (REACH, HSDB).
Mutagenicity/ Genotoxicity	The chemical is not expected to be genotoxic. The chemical tested negative in a number of tests for genotoxicity. These included several in vitro tests (OECD Guideline 473: mammalian chromosome aberration test on Chinese hamster lung fibroblasts V79; OECD Guideline 471: bacterial reverse mutation assay on S. typhimurium TA 98, TA 100, TA 98, TA 1535 and TA 1537; OECD Guideline 476: mammalian cell gene mutation test on Chinese hamster lung fibroblasts V79) and in vivo tests (OECD Guideline 474: mouse micronucleus) (OECD 2001, REACH).
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	The chemical is not expected to be toxic to reproduction (OECD 2001). In a non-guideline study, male and female Sprague Dawley (SD) rats were exposed to the chemical via inhalation at 0, 3000 or 6000 ppm for seven hours/day. Female rats were exposed to the chemical throughout gestation, while males were exposed to the chemical for six weeks prior to mating. No harmful effects on fertility or pregnancy rate were reported at any of the dose levels. In another non-guideline study, no testicular toxicity (effect on testes weight or histopathology) was reported in SD male rats that were administered the chemical via oral intubation at 533 mg/kg bw/day over six days (OECD 2001). Any developmental effects were only reported to be observed secondary to maternal toxicity, so the chemical is not expected to be a developmental toxin. OECD (2001) reported that the chemical showed mild foetotoxicity and developmental variations in offspring only at or near the maternally toxic and, in some cases, lethal dose of 8000 ppm. Offspring of female SD rats exposed via inhalation to 0, 3500, 6000 or 8000 ppm of the chemical on gestations days 1 to 19, reported a reduction of foetal weights at 6000 and 8000 ppm and a slight increase in skeletal malformations at 8000 ppm but not at the lower dosage levels. At a maternally toxic dose of 8000 ppm, decreased weight gain, food consumption and dam deaths were reported. The NOAEL for offspring and dams was 3500 ppm as there was a slight decrease in foetal weight at the 6000 ppm dose level. In another 20 day study in male and female SD rats exposed to 0, 3000 or 6000 ppm of the chemical via inhalation, a small number of behavioural and neurochemical variations in offspring at 6000 ppm were reported. No maternal toxicity was reported throughout gestation for females or for six weeks prior to mating for males as a result of maternal or paternal exposure. However, the effects observed in offspring were not regarded as biologically significant by the authors due to inconsistences between dose-response patterns.
Acute Toxicity	The chemical is reported to be slightly acutely toxic via the oral route of exposure. Oral median lethal doses (LD50s) in rats were reported between 790 and 4360 mg/kg bw (OECD 2001). The chemical is reported to have low toxicity via the dermal route of exposure. The lowest LD50 in rabbits was reported to be 3402 mg/kg bw (OECD 2001). The chemical is reported to be of low acute toxicity via the inhalation route of exposure. The median lethal concentration (LC50) in rats was reported to be greater than 5000 ppm (OECD 2001).

Irritation	Based on an inhalation study in mice, it was reported that 1268 ppm (3909 mg/ m³) of the chemical was predicted to be intolerable in humans, 127 ppm (390.9 mg/ m³) would be uncomfortable in humans and 13 ppm (40 mg/ m³) was expected to have no effect on humans (OECD 2001). Moderate irritation was reported in a 24 hour patch test (non-guideline study) where 405 or 500 mg of the chemical was applied to the skin of the rabbits. It was reported that these effects may be due to the chemical's defatting (chemical dissolving of dermal lipids from the skin) and drying characteristics (OECD 2001). Another non-guideline study reported the chemical was a skin irritant in several Vienna white rabbits exposed to 0.5 mL of the chemical for five minutes, one hour or two hours under occlusive conditions. The animals were observed for eight days. The authors concluded that exposure for two hours under occlusive conditions resulted in higher Draize scores and observed superficial necrosis (death of tissue). However, there was no full thickness destruction of the skin (REACH). The chemical was reported to be a severe eye irritant when tested according to OECD Test Guideline (TG) 405 using 0.1 mL of the chemical applied to three New Zealand white rabbits. Severe occular lesions were present at the end of the seven day observation period, indicating severe eye damage and irreversible effects on the eye (REACH). The chemical was reported to be a severe eye irritant in rabbits in non-guideline studies where 1.62 or 20 mg of the chemical was applied into rabbit eyes over a 24 or 72 hour period (OECD 2001). An additional non-guideline study reported severe corneal irritation when 0.005 mL of the chemical was applied into rabbit eyes.
Sensitisation	Based on available repeat dose dermal studies, the chemical is not expected to be a skin sensitiser. OECD (2001) reported that human studies and experience show that the chemical is not likely to be a skin sensitiser.
Health Effects Summary	The critical health effects for risk characterisation include local effects (serious damage to the eyes and respiratory irritation). The chemical also possesses hazardous properties such as skin irritation, harm if ingested and chemical vapours causing drowsiness and dizziness.
Key Study/Critical Effect for Screening Criteria	n-Butyl alcohol was only slightly toxic to experimental animals following acute oral, dermal, or inhalation exposure. The acute oral LD50 values for female rats ranged from 790 to 4360 mg/kg.
Ecological Toxicity [3]	
Aquatic Toxicity	Results on acute aquatic toxicity are available for fish (Pimephales promelas, LC50 (96h) 1376 mg/l), invertebrates (Daphnia magna, EC50 (48h) 1328 mg/L), and algae (Selenastrum capricornutum, EC50 (96h) 225 mg/L). EC10 (17h) as determined for Pseudomonas putida was 2476 mg/L. Furthermore, based on the chronic NOECrepro (21d) of 4.1 mg/L for Daphnia magna butan-1-ol is very likely not harmful to aquatic organisms. Thus, no adverse effects were observed.
Determination of PNEC aquatic	A PNECaqua = 0.082 mg/L can be calculated based on the lowest chronic toxicity value (21 day NOEC = 4.1 mg/L) for aquatic invertebrates (Daphnia) with the assessment factor of 50.
Current Regulatory Controls[4]	
Australian Hazard Classification	The chemical is classified as hazardous with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) Safe Work Australia: Xn; R22 (Harmful if swallowed) Xi; R37/38-41 (Irritating to respiratory system and skin. Risk of serious damage to eyes) R67 (Vapours may cause drowsiness and dizziness)
Australian Occupational Exposure Standards	The chemical has an exposure standard of 152 mg/m³ (50 ppm) Peak limitation Time Weighted Average (Ceiling TWA).

International Occupational Exposure Standards	The following exposure standards were identified (Galleria Chemica): Ceiling TWA: 150- 152 mg/m³ (50 ppm). India, Indonesia, Japan (OEL), Malaysia and USA [National Institute for Occupational Safety and Health (NIOSH)]. Ceiling TWA: 90 mg/m³ (30 ppm). Canada (British Colombia), Estonia, Russia and Sweden. TWA: 150- 154 mg/m³ (50 ppm). Canada (Yukon), Chile, Denmark, Egypt, Iceland, Poland and Switzerland. TWA: 300- 310 mg/m³ (100 ppm). Germany, Greece, Taiwan and USA [Occupational Safety and Health Administration (OSHA)]. TWA: 45- 75 mg/m³ (15-25 ppm). Canada (Alberta, British Colombia, Saskatchewan), Estonia, Hungary, Ireland, Japan [Workplace Exposure Standards (WES) and Working Environment Evaluation Standards (WEES)], Norway and Sweden.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. n-Butanol is considered readily biodegradable.
B/vB criteria fulfilled?	No. Due to the low log Pow (1.0), accumulation in organisms is not to be expected.
T criteria fulfilled?	Not applicable. Chronic toxicity data >1 mg/L in invertebrates, thus butyl alcohol does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. NICNAS (2017) Human Health Tier II Assessment for 1-Butanol: Retrieved 2019: https://www.nicnas.gov.au
2. OECD (2005) SIDS Initial Assessment Profile on 1-Butanol
3. ECHA REACH, 1-Butanol, Retrieved 2019: https://echa.europa.eu/

Toxicity Summary - Chlorous acid, sodium salt

Chemical and Physical Properties [1,2,3]	
CAS number	7758-19-2
Molecular formula	ClHO2.Na
Molecular weight	90.4
Solubility in water	571 g/L at 20 °C
Melting point	234 °C
Boiling point	Decomposes > 170 °C. Poor purity of test substance, accurate value cannot be obtained.
Vapour pressure	1.1 x 10^{-7} Pa at 25°C
Henrys law constant	No data available.
Explosive potential	At normal temperature and pressure, the natural form of chlorine dioxide is unstable, highly reactive (an oxidizing agent) and explosive. It is explosive when its concentration in air exceeds 10% v/v when it is easily detonated by sunlight, heat, contact with mercury or carbon monoxide (O'Neil et al. 2001).
Flammability potential	Non-flammable
Colour/Form	White crystals or crystalline powder, odourless
Overview	The commercial production of sodium chlorite is carried out in two steps: firstly, sodium chlorate is reacted with an acid to generate chlorine dioxide (gas) and secondly, chlorine dioxide is reacted with caustic soda, catalysed by hydrogen peroxide, to form sodium chlorite. The industrial product formed is a solution of 34.5%; the commercial grade is obtained by dilution with water. Chlorine dioxide may also be produced from sodium chlorate. The total amount of sodium chlorite (as 100%) sold on average in the EU Member States (15) for the years 1998-2000 was 11 800 tonnes per year. This includes use as preservatives for liquid cooling and processing systems; food and feed area disinfectants; food or feedstocks; molluscicides; and slimicides and other non-defined biocidal use. The estimated annual total consumption of sodium chlorite in Japan is 4000 tonnes.
Environmental Fate [2]	
Soil/Water/Air	Irradiation of sodium chlorite solutions indicated a photodegradation half-life of about 30 minutes with a steady increase in pH (pH 8 to 12.6) and major products identified as hydroxide, chlorine dioxide and chloride with chlorate and hypochlorite as minor products and trace amounts of chlorine. The radiation dose (9000 j/m^2) needed to produce a 50% reduction in chlorite concentration suggests that the doses (200-250 j/m^2) used for drinking water disinfection would not result in a significant reduction in chlorite concentrations (Cosson and Ernst, 1994; Leitner et al., 1992). It is not considered technically appropriate to perform a ready biodegradation test on sodium chlorite. As ready biodegradation studies measure oxygen consumption or carbon dioxide production, none of these techniques can be used to analyse mineralization of this compound. However, sodium chlorite is expected to be rapidly reduced to sodium chloride in the environment, especially in anaerobic conditions. Due to its extremely low lipophilicity and high instability in water, sodium chlorite and hence chlorine dioxide are not expected to bioaccumulate in fish.

Human Health Toxicity Summary [1,2]	
Chronic Repeated Dose Toxicity	In a study used by the World Health Organization (WHO) to establish a drinking water guideline for chlorite in 1993, rats were administered sodium chlorite at doses of 0, 10, 50, 100, 250 and 500 mg/L (equivalent to 0, 1, 5, 10, 25 and 50 mg/kg bw/day) via drinking water for 30, 60 or 90 days (Heffernan et al. 1979). After 30 days, haematological parameters were depressed indicating slight anaemia at 10 and 25 mg/kg bw/day. These were correcting at 60 days and returned to near normal levels by 90 days. Decreases in erythrocyte glutathione levels were observed at 5 mg/kg bw/day and above, but given the magnitude of variations normally seen in mammals, the toxicological significance of these changes was uncertain. The No Observed Adverse Effect Level (NOAEL) established from this study was 5 mg/kg bw/day. In a 14-day range finding study conducted to OECD TG 407, rats were administered sodium chlorite daily by gavage at doses of 0, 25, 50, 100 or 200 mg/kg bw day (CMA 1992a; Harrington et al. 1995a). At 200 mg/kg bw/day, 3 of 10 animals died. At 100 mg/kg bw/day, changes in haematological parameters were seen and body weight gains were reduced. At 50 mg/kg bw/day, body weights in males were reduced and at both 25 and 50 mg/kg bw/day haematocrits were slightly reduced. A follow-up 90-day study was performed in which rats were administered sodium chlorite daily by gavage at doses of 0, 10, 25 or 80 mg/kg bw day (CMA 1992b; Harrington et al. 1995a). At 80 mg/kg bw/day, four of 30 animals died and surviving animals displayed hypoactivity, piloerection and hunched posture. At 25 mg/kg bw/day, one of 30 animals died. Increased salivation was observed at both doses. Treatment-related haematological changes consisting of reduced erythrocyte counts, reduced associated erythrocyte parameters and morphological changes in erythrocytes were observed at 80 mg/kg bw/day. These were accompanied by increases in absolute and relative spleen weights, histopathological abnormalities in the spleen and evidence of irritation of the gastric mucosa. At 25 mg/kg bw/day, minor clinical signs and occasional histopathological abnormalities in the stomach mucosa were seen. There were no haematological changes considered treatment related at this dose. A NOAEL was established at 10 mg/kg bw/day. Data on repeat dose toxicity were also available from a two-generation reproductive toxicity study in rats conducted to OECD TG 416 (Chlorine Dioxide Panel of the Chemical Manufacturers Association 1996; Gill et al. 2000). This study was used by the WHO to revise an earlier drinking water quality guideline for chlorite and chlorate (WHO 2005). A NOAEL of 35 ppm (approximately 3.9 mg/kg bw/day) was derived based on decreased liver weights in two generations. Repeated dose toxicity studies have also been performed in mice. Mice were treated for 30 days with doses equivalent to 0, 0.19, 1.9 and 19 mg/kg bw/day sodium chlorite in drinking water (Moore and Calabrese 1980). Slight changes in haematological parameters suggestive of effects on erythrocyte cell membranes were seen at 19 mg/kg bw/day. A NOAEL of 1.9 mg/kg bw day was established. Similarly, in more limited studies, mice were administered sodium chlorite in drinking water at doses up to approximately 17 mg/kg bw/day for 30, 90 or 180 days. No effects on water consumption, body weight gain, kidney weights or kidney histology were seen (Connor et al. 1985). Also, no dose-related immunomodulatory effects were seen in a study of immunotoxicity in mice receiving sodium chlorite in drinking water at levels up to 30 mg/L for 28 days (Karrow et al. 2001). In conclusion, several rodent studies of 30 to 90 days' duration have reported haemotoxicity from repeated doses of sodium chlorite. A guideline 90-day repeated dose toxicity study in rats reported reduced erythrocyte counts, reduced associated erythrocyte parameters and morphological changes in erythrocytes at 80 mg/kg bw/day. At lower doses, minor clinical signs and occasional histopathological abnormalities in the stomach mucosa were seen. A NOAEL for repeated dose oral toxicity was established from this 90-day study at 10 mg/kg bw/day.

Carcinogenicity	A limited number of carcinogenicity studies indicated that sodium chlorite is not carcinogenic in laboratory animals. In an oral carcinogenicity study conducted similarly to OECD TG 451, groups of 50 male and 50 female rats were exposed to sodium chlorite in drinking water at concentrations of 0, 300 or 600 mg/L (estimated to be 0, 18 or 32 mg/kg bw/day for males and 0, 28 or 41 mg/kg bw/day for females) for 85 weeks. The original study envisaged an exposure period of 104 weeks, but was stopped at 85 weeks due to infections in all groups. At this time there were no significant changes in organ weights and haematological or clinical chemistry findings between groups. Tumours developed in the testis, uterus, pituitary gland, thyroid gland (males) and adrenal gland (males) of both treated and control rats. However, the incidences of tumours and non-neoplastic lesions in the three groups were not significantly different. There were no findings suggestive of a carcinogenic effect of sodium chlorite (Shimoyama et al., 1985). In another oral carcinogenicity study conducted similarly to OECD TG 451, groups of 50 male and 50 female B6C3F1 mice were exposed to sodium chlorite in drinking water at concentrations of 0, 250 or 500 mg/L (estimated to be 0, 36 and 71 mg/kg bw/day) for 85 weeks (Yokose et al., 1987). After 85 weeks, surviving animals were euthanised and histopathological examinations were performed. Although tumours developed in a variety of organs in all animals including controls, the only significant change was an increase in lung adenomas in highest dose males: 5/43 (12 %) in this group, compared with 0/35 (0 %) in the control group. Based on an absence of dose-related increases in the incidence of lung adenomas and the lack of increased incidence of lung adenocarcinomas, the authors concluded that sodium chlorite had no carcinogenic potential.
Mutagenicity/ Genotoxicity	Sodium chlorite is not mutagenic or genotoxic. In vitro genotoxicity test results for sodium chlorite are not available. In the three in vivo tests that looked at chromosomal damage or sperm head abnormality, sodium chlorite gave negative results for genotoxicity (Meier et al., 1985). In vitro tests using chlorine dioxide have been reported in the literature. Chlorite (and chlorate) ions are produced following dissolution of chlorine dioxide in aqueous media. Therefore, in vitro test results for chlorine dioxide are regarded as relevant to sodium chlorite. Two of the three in vitro tests, the mouse lymphoma forward mutation assay and in vitro transformation of BALB/3T3 cells, were negative for chlorine dioxide, whereas the chromosome aberration frequencies test in Chinese hamster ovary cells was positive (Scopas, 1986a, Scopas, 1986b and Scopas, 1986c). Across all available studies, data suggest that sodium chlorite (and chlorine dioxide) has low genotoxic potential.

Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Based on a series of studies of fertility and sperm parameters in rats, sodium chlorite is not considered to be toxic to the reproductive system. Studies in rats and rabbits did not show any effect of sodium chlorite on development. In a rabbit study conducted according to US EPA guidelines, sodium chlorite was administered via drinking water to groups of 16 pregnant New Zealand White rabbits at concentrations of 0, 200, 600 or 1200 mg/L during gestation days (GD) 7–19 (Harrington et al., 1995b). At 600 and 1200 mg/L, dose-related reductions in water consumption (due to palatability problems), food consumption and body weight gain were observed. No treatment-related abnormalities were observed at maternal necropsy. Overall, data indicate that sodium chlorite does not cause developmental toxicity at doses below those associated with maternal toxicity. In a two-generation reproduction study in rats conducted according to OECD TG 416 (Gill et al. 2000), groups of 30 male and 30 female Sprague-Dawley rats were administered sodium chlorite via drinking water at doses of 0, 35, 70 or 300 ppm (approximately 0, 4, 7.6 or 28.2 mg/kg bw/day for males and 0, 3.9, 8 and 38.7 mg/kg bw/day for females) (Chlorine Dioxide Panel of the Chemical Manufacturers Association 1996; Gill et al. 2000). Dosing was conducted in the parental F0 generation commencing 10 weeks prior to mating, until weaning of the F2 generation. Males were exposed through mating and then sacrificed. Females were exposed through mating, pregnancy and lactation and were sacrificed following weaning of litters. F1 pups were continued on the same treatment regime as the parents. At 14 weeks they were mated to produce the F2 generation. Reductions in food and water consumption and body weight gain were observed for all generations, attributed to unpalatability of the formulated drinking water. At 35 and 70 ppm, minor reductions in several haematological parameters were observed in F1 female pups. These appeared within the range of historical control data and were not regarded as toxicologically significant. At 70 ppm, a reduction in liver weight was also observed in F0 females and F1 males and females. A slight decrease in the maximum response to auditory startle stimulus was also observed in F2 pups. At 300 ppm, reductions in haematological parameters were seen in F1 male and female pups and adults. Reduced liver weights were seen in F0 adult males, F1 adult males and females and F1 pups. Reduced thymus and spleen weights were also seen in both generations. A slight decrease in absolute brain weight was seen in F1 male pups at post-natal day (PND) 11 but not at PND 25. In F2 pups at this dose, there was a slightly lowered incidence of normal righting reflexes and a slight decrease in the maximum response to auditory startle stimulus. Reduced pup body weight at birth and during lactation in F1 and F2 generations were also observed. Delays in preputial separation and vaginal openings were reported for F1 pups. Despite systemic toxicity, the authors reported no treatment-related changes to oestrous cyclicity, sperm motility, sperm morphology, or mating, fertility or gestational indices. Also, there were no treatment-related changes in number of pups born, sex ratios, live birth index or pup survival indices. There were no treatment-related changes in serum T3 or T4 in F1 pups or F1 adults. On the basis of historical data, delays in preputial separation and vaginal openings reported for F1 pups were attributed to reduced body weight rather than a direct treatment-related effect. Similarly, slight decreases in brain weight in male pups were consistent with decreased body weight. The toxicological significance of decreases in auditory startle stimulus response at 70 and 300 ppm was unclear. The magnitude of responses was small compared to known neuroactive chemicals, dose response to the stimulus was weak, there was a lack of corroborative evidence from neuropathology or other test of motor function or arousal, and the decreases in response were not replicated upon later examination of the same animals at PND 60 (Gill et al. 2000). A NOAEL of 35 ppm (approximately 3.9 mg/kg bw/day) with a LOAEL at 70 ppm (approximately 7.6 mg/kg bw/day) were derived based on decreased liver weights.

Acute Toxicity	Sodium chlorite has moderate acute oral toxicity. An acute oral toxicity study in rats, similar to OECD Test Guideline TG 401, derived a lethal median dose (LD50) of 284 mg/kg bw for sodium chlorite. At doses of 250 mg/kg bw and above, the main clinical signs were prostration and cyanosis (Atochem, 1984). Sodium chlorite has high acute dermal toxicity. In a dermal toxicity study in rabbits, conducted according to US EPA test guidelines, various doses of an aqueous slurry (80 %) of sodium chlorite were administered under semi-occlusive dressings to over 10 % of the body surface area for 24 hours. Animals were observed for clinical signs immediately after dosing, at one and four hours and then once daily for 14 days following exposure. Slight depression and dose-related dermal irritation consisting of skin thickening, epidermal scaling, necrosis and sloughing were noted in all animals. The study reported a dermal LD50 of 134 mg/kg bw (Degussa Corporation, 1984).
Irritation	Sodium chlorite is a severe skin irritant. Necrosis was observed in rabbits in the skin irritation studies. In one skin irritation study conducted according to US EPA test guidelines, 0.5 g sodium chlorite powder (80 % pure) was applied to three male and three female New Zealand White rabbits under occlusive conditions for four hours. Dermal responses were assessed at 30–60 minutes on day one, and once daily for 21 days after application. Irritation consisted of erythema (grades 1–3) in all sites at 30–60 minutes and 24 hours after dosing, persisting through day seven at two sites. Oedema (grade one) was observed at one site at 30–60 minutes and at two sites at 48 hours. Other dermal effects included blanching, thickening, necrosis, sloughing, and blackened areas (REACH, 2014). In another study in rabbits, edema cutis and subcutis were observed immediately after patch removal followed by formation of eschar within 24–48h. Dose and other details of the test were not provided (REACH, 2014) A 34.5 % solution of sodium chlorite, applied to rabbit skin for four hours under semi-occlusive conditions, did not elicit any irritation effects. Only one of three animals displayed slight erythema and dryness of the skin (Elf Atochem SA, 1994). In the only eye irritation study available and conducted according to US EPA test guidelines, sodium chlorite was found to be a severe eye irritant. A 31.5 % sodium chlorite solution was applied to the eyes of rabbits. Six of the nine rabbits showed corneal opacity that did not reverse by rinsing the eyes 30 seconds after instillation. All animals showed iris damage and exhibited moderate to severe redness and chemosis which was also not abolished by rinsing. Superficial corneal vascularisation and transient cases of haemorrhaging and adhesion of conjunctivae to cornea were also seen (Atochem, 1985).
Sensitisation	Sodium chlorite is not considered to be a skin sensitiser. A guinea pig maximisation test conducted according to OECD TG 406 reported no clinical signs and no cutaneous reactions upon a challenge application of 1 % sodium chlorite in normal saline. Sodium chlorite was concluded not to be a skin sensitiser (CEFIC sodium chlorite sector group, 2002).
Health Effects Summary	The critical health effects for risk characterisation include acute effects from oral and dermal exposure, and severe skin and eye irritation and repeated dose toxicity from oral exposure.
Key Study/Critical Effect for Screening Criteria	A guideline two-generation reproductive toxicity study in rats also reported haemotoxicity, as well as hepatotoxicity and slight neurobehavioural changes at doses below those associated with no effects in repeated dose studies. The study reported no effects on fertility or development. Accordingly, a NOAEL for hepatotoxicity was established from this 2- generation study at 3.9 mg/kg bw/day. The LOAEL was approximately 7.6 mg/kg bw/day. This NOAEL is used for this human health risk assessment.
Ecological Toxicity [2]	

Aquatic Toxicity	Sodium chlorite, in general, shows low acute toxicity to fish with LC50 values above 100 mg/l for zebrafish, sheepshead minnow and rainbow trout and slightly lower for bluegill sunfish. Due to extremely low lipophilicity and high instability in water, sodium chlorite is not expected to bioaccumulate in fish. Sodium chlorite is more toxic to invertebrates with high toxicity to Daphnia magna (sodium chlorite, LC50 48-hour = 0.063 mg/l) and the crustacean, Mysidopsis bahia (sodium chlorite LC50 96-hour = 0.65 mg/l). However, the mollusc, Crassostrea virginica was much less sensitive (sodium chlorite 96 hours NOEC was 70.6 mg/l and the EC50 (shell growth) was 129 mg/l). The green algae were more sensitive to sodium chlorite than fish or oyster and toxicity increased with time (ECr50 value at 72 hours was recorded as 1.2 mg/l).
Determination of PNEC aquatic	Using an uncertainty factor of 100 on the lowest LC50 to Daphnia a PNEC (Predicted No Effect Concentration) of 0.63 ug/L is calculated, for aquatic organisms.
Current Regulatory Controls[1]	
Australian Hazard Classification	The chemical is not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).
Australian Occupational Exposure Standards	There is no specific exposure standard for sodium chlorite. However, the permissible exposure limits for dusts apply: · Time Weighted Average (TWA): 10 mg/m^3 measured as inspirable dust.
International Occupational Exposure Standards	There are no specific exposure standards for sodium chlorite. However, the following exposure standards for particulates are identified (Galleria Chemica 2013). TWA: · 10 mg/m^3 [Canada, Ireland, Spain] · 5 mg/m^3 [US] · 1 mg/m^3 [Latvia].
Australian Food Standards	Sodium chlorite has the following listings in the Australia New Zealand Food Standards Code – Standard 1.3.3 Processing Aids (Food Standards Australia and New Zealand 2013): · As a permitted bleaching agent, washing and peeling agent (maximum level 1 mg/kg available chlorine) · As a permitted processing aid with miscellaneous functions (anti-microbial agent for meat, fish, fruit and vegetables; maximum level is the limit of determination for chlorite, chlorate, chlorous acid and chlorine dioxide).
Australian Drinking Water Guidelines	The National Health and Medical Research Council (NHMRC) Australian Drinking Water Guidelines lists chlorite under microbial, chemical and physical characteristics as a by-product of chlorine dioxide disinfection. The guideline value for chlorite based on health considerations is 0.8 mg/L (NHMRC 2011).
Aquatic Toxicity Guidelines	No data available.
PBT Assessment [3]	
P/vP Criteria fulfilled?	No. Not expected to be persistent due to its instability.
B/vB criteria fulfilled?	No. There is no concern for potential bioaccumulation from chlorine chlorite.
T criteria fulfilled?	Yes. Acutely toxic to aquatic invertebrates.
Overall conclusion	Not PBT
Revised	January 2019

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier II Assessment for Chlorous acid, sodium salt: Retrieved 2019: https://www.nicnas.gov.au

2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS, 2017). National assessment of chemicals associated with coal seam gas extraction in Australia. Human health hazards of chemicals associated with coal seam gas extraction in Australia.
3. OECD (2009) SIDS Initial Assessment Profile on Sodium chlorite and chlorine dioxide
4. ECHA REACH, Sodium chlorite, Retrieved 2019: https://echa.europa.eu/

AECOM Imagine it. Delivered.

Toxicity Summary - Cinnamaldehyde

Chemical and Physical Properties [1,2,3,4]	
CAS number	104-55-2
Molecular formula	C9H8O
Molecular weight	132.16
Solubility in water	2.11 g/L at 22 °C
Melting point	-18 °C
Boiling point	250°C
Vapour pressure	3.85 Pa at 25 °C
Henrys law constant	0.162 Pa.m³.mol-1 at 25 °C
Explosive potential	Non-explosive
Flammability potential	Non-flammable
Colour/Form	Yellowish oily liquid with strong odour of cinnamon
Overview	Cinnamaldehyde is a plant natural product that is present in some essential oils extracted from plants. For large scale applications such as in the flavouring and fragrance industries, this chemical is synthesised.
Environmental Fate [1,3]	
Soil/Water/Air	Cinnamaldehyde is expected to remain in soil, or partition to water and sediment, when released as a result of industrial uses. It is not expected to be persistent in the environment and is expected to undergo rapid and ultimate biodegradation in water. Cinnamaldehyde is not expected to bioaccumulate in aquatic organisms. No evidence has been identified to indicate that Cinnamaldehyde biomagnify through the aquatic food chain. The atmospheric oxidation half-life of cinnamaldehyde was estimated using the level III multimedia model. It was estimated that the substance is not persistent in air medium as the half-life period of cinnamaldehyde in air is only 0.31 days. This indicates that cinnamaldehyde is rapidly phototransformed in air. The Hydrolysis rate constant of Cinnamaldehyde is estimated to be 3.36 x 10-17 cm3/molecule-sec. at half-life of 3.411 days indicating that the substance is slowly hydrolysable.
Human Health Toxicity Summary [2,4]	
Chronic Repeated Dose Toxicity	Cinnamaldehyde is 'generally regarded as safe' for use as a flavour ingredient by the US Food and Drug Administration (US FDA, 2015), reflecting the low level of concern regarding its potential for long-term toxicity via the oral route. Considering the no observed adverse effect levels (NOAELs) of 68–200 mg/kg bw/day, based on 17-week to 2-year rat studies (read across), and no toxicologically significant treatment-related effects reported in various studies, repeated oral exposure to the chemical is not considered to cause serious damage to health. Based on the limited data available, the chemical is not considered to cause serious damage to health by repeated dermal exposure.
Carcinogenicity	Based on the limited data available for cinnamaldehyde and trans-cinnamaldehyde (CAS No. 14371-10-9), the chemical is not expected to have carcinogenic potential. In a two-year carcinogenicity study, groups of F344/N rats and B6C3F1 mice (50 animals/sex/dose) were fed microencapsulated trans-cinnamaldehyde (CAS No. 14371-10-9) by daily gavage at doses of 0, 1000, 2100 or 4100 ppm (equivalent to 0, 50, 100 or 200 mg/kg bw/day). Increased incidences of preputial and prostate gland adenomas and mononuclear cell leukaemia were considered to be within the historical range in controls, or likely to represent biological variations unrelated to exposure to the chemical. No other treatment-related neoplasms or non-neoplastic lesions were reported in either species (Adams et al., 2004; NTP, 2004; REACH; US HPVIS, 2009).

Mutagenicity/ Genotoxicity	The chemical cinnamaldehyde contains an a,b-unsaturated aldehyde group, a common structural alert for genotoxicity due to the ability of the chemical to form DNA adducts. However, based on the available data, the chemical is not considered to be genotoxic. The chemical cinnamaldehyde and the isomer trans-cinnamaldehyde (CAS No. 14371-10-9) were negative for point mutations in almost all strains of Salmonella typhimurium in the Ames test. A positive result was found only with TA100 strain, and in only two out of eleven tests. Evidence of genotoxic activity was also observed in isolated mammalian cells. However, these results were weakly positive and observed at cytotoxic concentrations. A sex-linked recessive lethal test in Drosophila melanogaster demonstrated that systemically-available chemical (administered via injection) could enter germ cells and induce mutations; however, oral dosing did not produce the same effect. Importantly, the reported activity in in vitro and insect studies did not translate into significant genotoxic activity in mammalian systems in vivo.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	The chemical is not expected to have the potential for reproductive or developmental toxicity. Any developmental effects were only observed secondary to maternal toxicity. In a two-generation study in rats (strains not reported), cinnamaldehyde (absolute dose 2 mg—route not specified) was dosed every two days for 223 and 210 days and did not have any effects on body weight gain, reproductive ability, development or viability of offspring (NTP, 2004). Cinnamaldehyde in olive oil was administered to female SD rats via oral gavage at doses of 0, 5, 25 or 250 mg/kg bw/day on gestation days (GD) 7–17. Treatment-related, increased incidence of defective cranial ossification in all dose groups was observed. Renal abnormalities including dilated pelvis and reduced papilla and dilated ureters were observed at low and mid doses, but not at high dose. Offspring at ≥25 mg/kg bw/day had significantly increased instances of reduced ossification of the tympanic bulla. An increase in the incidence of abnormal sternebrae was also reported in the 25 mg/kg bw/day group. However, these effects were not found to be dose-related and may be attributed to a decrease in maternal weight gain that was noted in the mid- and high-dose groups. A LOAEL of 5 mg/kg bw/day for developmental toxicity was reported based on the reduced cranial ossification and kidney variations. A LOAEL of 25 mg/kg bw/day was reported for maternal toxicity based on the reduced weight gain observed in the dams (Adams et al., 2004; NTP, 2004; US HPVIS, 2009; HSDB; REACH). No signs of toxicity were reported in the dams or in the offspring of CD-1 mice after exposure to 1200 mg/kg bw/day during GD 6–13 (cinnamaldehyde) or GD 7–14 (trans-cinnamaldehyde) (NTP, 2004; US HPVIS, 2009; REACH).
Acute Toxicity	Cinnamaldehyde has low acute oral toxicity based on animal studies. The median lethal dose (LD50) in rats is >2000 mg/kg bw. Cinnamaldehyde has moderate acute dermal toxicity based on animal studies, warranting hazard classification. The dermal LD50 in rabbits was in the range of 620–1260 mg/kg bw (Bickers et al., 2005; Cocchiara et al., 2005; FFHBVC, 2005; and US HPVIS, 2009). Albino rabbits (2 animals/dose) were administered a single dose of cinnamaldehyde (0, 0.25, 0.50, 1.0, 2.0 or 4.0 mL/kg bw—equivalent to 0, 263, 525, 1050, 2100 or 4200 mg/kg bw) by application to intact and abraded skin. All animals in the 1.0 mL/kg and higher dose groups died after treatment. The LD50 was reported to be 620 mg/kg bw (Cocchiara et al., 2005; FFHPVC, 2005; US HPVIS, 2009; REACH).
Irritation	Respiratory irritation was assessed in CF-1 female mice by recording their respiratory rate following exposure to nebulised cinnamaldehyde for 1 minute, either through nose-only breathing or via a tracheal cannula. Marked respiratory depression with nose-only inhalation was observed. The ED25 (dose providing a 25 % reduction in respiratory rate) was calculated to be 241 µg/L. No significant effects were observed when inhalation was through the tracheal cannula (Cocchiara et al., 2005). Cinnamaldehyde produced severe irritation in rabbits when applied undiluted, mild irritation in mice and guinea pigs at concentrations of 3–5 %, and was non-irritating to rabbits at 1 % (Bickers et al., 2005). The US EPA considers cinnamaldehyde a strong skin irritant in guinea pigs (no study details provided) (US HPVIS, 2009). Several international agencies have concluded that cinnamaldehyde is an eye irritant (US HPVIS, 2009; REACH), and a number of notifications to the Classification and Labelling Inventory by industry in the European Union have indicated the chemical as irritating to the eyes (ECHA C&L).

Sensitisation	The chemical was considered to be a moderate to strong skin sensitiser based on the positive results in several local lymph node assays (LLNA). The EC3 value (concentration required to provoke a 3-fold increase in lymph node cell proliferative activity compared with controls) was reported to be as low as 0.2 % (SCCS, 2012).
Health Effects Summary	Cinnamaldehyde is a well-recognised and frequently reported consumer contact allergen (SCCNFP, 1999; RIVM, 2009; SCCS, 2012; IFRA, 2013). It is one of eight components of the diagnostic test, the fragrance mix, used by dermatologists to determine if a patient has allergies to common chemicals used in fragrances. It is an established contact allergen in humans according to the Scientific Committee on Consumer Safety (2012), and accounts for 5–36 % of the reactions to the fragrance mix (SCCNFP, 1999). A number of human repeat insult patch tests (HRIPTs) have been undertaken to determine the skin sensitisation potential of cinnamaldehyde in healthy volunteers, as well as groups of subjects suspected of skin allergies to fragrances (SCCNFP, 1999; NTP, 2004; Cocchiara et al., 2005). Although fewer cases of sensitisation were found when the concentration of the chemical was less than 1 %, positive allergic responses have been reported in cases where the administered concentration of cinnamaldehyde was as low as 0.2 % (Cocchiara et al., 2005). Skin irritation effects were generally predominant at concentrations above 3 % cinnamaldehyde, and often impeded the interpretation of results from the patch testing (SCCNFP, 1999; NTP, 2004). Many cases of skin sensitisation have occurred following occupational and consumer exposure to the chemical. Workers in spice manufacturing plants, hairdressing salons and bakeries have reported cases of contact dermatitis that were traced back to cinnamaldehyde. In addition, exposure of consumers to toothpaste, cosmetics and perfumes containing the chemical as a fragrance ingredient have resulted in a number of case studies identifying cinnamaldehyde as an agent responsible for the allergic reactions (see SCCNFP, 1999; NTP, 2004; Cocchiara et al., 2005 for review).
Key Study/Critical Effect for Screening Criteria	The critical health effect for risk characterisation is skin sensitisation. Other observed health effects include systemic acute effects (acute toxicity from dermal exposure) and local effects (eye/skin/respiratory irritation).
Ecological Toxicity [1]	
Aquatic Toxicity	The following data are measured acute toxicity values for cinnamaldehyde: Danio rerio (Zebrafish) EC Directive 92/69/EEC C.1 Acute Toxicity for Fish: 96 h LC50 = 3.1 mg/L; Daphnia magna (Water flea) OECD TG 202: 48 h EC50 = 3.86 mg/L; Pseudokirchneriella subcapitata (Green algae) OECD TG 201: 72 h EC50 = 4.07 mg/L. In the chronic toxicity study, the 72 h NOEC value of 2.0 mg/L was reported for Pseudokirchneriella subcapitata (Green algae) OECD TG 201.
Determination of PNEC aquatic	A PNECaqua = 0.2 mg/L can be calculated based on the chronic toxicity value (72 h NOEC = 2 mg/L) for green algae with the assessment factor of 10.
Current Regulatory Controls[4]	
Australian Hazard Classification	The chemical is not listed in the Hazardous Substances Information System (HSIS) (Safe Work Australia).
Australian Occupational Exposure Standards	No specific exposure standards are available for the chemical.
International Occupational Exposure Standards	The US Temporary Emergency Exposure Limits (TEELs) for cinnamaldehyde are 14, 150 and 670 mg/m^3 (Galleria Chemica).
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.

PBT Assessment	
P/vP Criteria fulfilled?	Not applicable (inorganic salt, ionic species ubiquitous in environment)
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to these inorganic ions; sodium and hydroxide ions are ubiquitous and are present in most water, soil and sediment.
T criteria fulfilled?	Not applicable. Chronic toxicity data >1 mg/L in invertebrates, thus sodium hydroxide does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. NICNAS (2017a) Environment Tier II Assessment for Cinnamic Aldehydes
2. NICNAS (2017b) Human Health Tier II assessment for 2-Propenal, 3-phenyl-
3. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2015, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
4. ECHA REACH, Cinnamaldehyde, Retrieved 2017: https://echa.europa.eu/information-on-chemicals/registered-substances

AECOM Imagine it. Delivered.

Toxicity Summary - Citric acid

Chemical and Physical Properties[2,3,5]	
CAS number	77-92-9
Molecular formula	C6-H8-O7
Product name	--
Molecular weight	192.124
Solubility in water	1000000 mg/L
pH	2 to 2.2
Melting point	Decomposition > 175 C
Boiling point	152 to159 C
Vapour pressure	White powder or granules
Henrys law constant	1.7 $x10^{-8}$ mm Hg at 25 deg C
Explosive potential	4.39 x 10^{-09} $Pa.m^3/mol$
Flammability potential	Dust explosion possible if powder or granular form, mixed with air
Colour/Form	Melts and decomposes in fire, a non-hazardous reaction.
Overview	Citric acid is a water soluble organic solid. It is a natural substance that appears as an intermediate in the basic physiological citric acid or Krebs cycle in every eukaryote cell. Citric acid has been produced for many years in high volumes. It has wide dispersive use, being added to processed food and beverages, used in pharmaceutical preparations and in household cleaners as well as in special technical applications. Citric acid is recognised by Food Standards Australia New Zealand (FSANZ) and the WHO JECFA as safe as a multipurpose food additive. No upper limit of concentrations has been established in food products. This chemical has been identified by NICNAS to be of low concern to human health based on an initial screening approach and thus required no further assessment.
Environmental Fate[2,5]	
Soil/Water/Air	Citric acid is highly mobile in the environment and is extremely soluble in water. The pKa of citric acid is 2.79, indicating that this compound will exist almost entirely in the anion form in the environment. The compound does not sorb to soil or particles in the water column and is readily and rapidly degraded in surface waters and in soil. (OECD, hsdb)

Human Health Toxicity Summary [1,2,4,5]	
Chronic Repeated Dose Toxicity	A 2-year chronic oral study in rats being given 5% or 3% citric acid in feed (approx. 2 resp. 1.2 g/kg/d) found slightly decreased growth in the higher dosage group but no tissue abnormalities in the major organs. From the lower dosage a NOAEL of 1200 mg/kg/d results. Similarly, NOAELs of 1500 mg/kg/d (rabbit) and of 1400 mg/kg/d (dog) have been determined. In general, citric acid is a strong chelating agent, the dietary uptake of which may interfere with biological availability, absorption and excretion of metals. Further, loss of superficial enamel and erosion of teeth as well as local irritation result from frequent ingestion of citric acid in beverages including natural fruit juices; citric acid fumes were reported to apparently affect the teeth of exposed workers. The average daily intake of citric acid from natural sources in the diet and food additives was estimated at about 40 mg/kg for women, 130 mg/kg for infants and 400 mg/kg for individuals on slimming diets; maximum daily intake is reported to reach levels of 500 mg/kg. No formal ADI (acceptable daily intake) level has been specified for citric acid and its common salts by the Joint FAO/WHO Expert Committee on Food Additives nor by the EC Scientific Committee for Food.
Carcinogenicity	Citric acid has not been classified by the IARC.
Mutagenicity/ Genotoxicity	In several in vitro and in vivo tests citric acid was not mutagenic. The substance was not mutagenic either in bacterial tests with Salmonella typhimurium (Ames test, 2 studies) and Escherichia coli, with and without metabolic activation.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	In a two-generation 90 days study with male and female rats fed 1.2 % citric acid no adverse effect on reproductive parameters nor any teratogenicity of dietary citric acid was seen. There were no indications of teratogenic or other adverse effects in three shorter term reproductive studies in rats with dietary dosage of either 5% citric acid (approx. 2.5 g/kg/d) previous, during and after mating (NOEL = 2500 mg/kg/d), or 295 mg/kg/d (route unspecified) during days 6–15 of pregnancy
Acute Toxicity	Citric acid has a low acute toxicity by oral application in both rat (LD50 = 3,000–12,000 mg/kg, 3 different values) and mouse (LD50 = 5,400 mg/kg). General effects comprised physiological disturbances (acidosis and calcium deficiency), while “high” doses caused nervous system effects as well as severe damage to the stomach mucosa.
Irritation	Local effects of citric acid to the skin (rabbit) are reported as slightly irritating in two studies and as not irritating in a third study using a 30% aqueous solution. In an acute eye irritation/corrosion test in rabbits according to OECD 405 citric acid was highly irritating.
Sensitisation	The sensitising potential is low.
Key Study/Critical Effect for Screening Criteria	A 2-year chronic oral study in rats being given 5% or 3% citric acid in feed resulted in a NOAEL of 1200 mg/kg/d. Uncertainty factors: 10 (interspecies variability) and 10 (intraspecies variability). Drinking water guideline = 4.7 ppm
Ecological Toxicity [1,5]	
Aquatic Toxicity	The 96-hour LC50 values for citric acid to fish are from 440 to 1,516 mg/L. The acute toxicity 24 hour EC50 value for invertebrates is 85 mg/L. The 7 day toxic limit concentration (TLC) values for algae range from 300 to 640 mg/L. In an 8 day freshwater static test for the algae Scenedesmus quadricauda, the NOEC is 425 mg/L. In freshwater, citric acid appears to be of low toxicity to aquatic acute test standard organisms, fish, daphnia and algae, with consistent LC50/EC50 values of several hundred milligrams per litre.

Determination of PNEC aquatic	$PNEC_{aquatic}$: Experimental results are available for three trophic levels. Acute $E(L)C_{50}$ values are available for fish (440 mg/L), Daphnia (85 mg/L). A TLC value of 300 mg/L was obtained for algae from which no dependable EC50 can be derived. Even though a NOEC was obtained from the algae study, there were no chronic studies conducted on fish or Daphnia. 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 effect concentration of 85 mg/L for Daphnia Magna. The $PNEC_{aquatic}$ was calculated to be 0.085 mg/L.
Current Regulatory Controls	
Australian Hazard Classification	
Australian Occupational Exposure Standards	
International Occupational Exposure Standards	
Australian Food Standards	
Australian Drinking Water Guidelines	No data found
Aquatic Toxicity Guidelines	No data found
Australian Hazard Classification	
PBT Assessment[1]	
P/vP Criteria fulfilled?	Citric acid is expected to be readily biodegradable and does not persist in the environment
B/vB criteria fulfilled?	Based on the low Log Kow and widespread natural occurrence, citric acid is not expected to have potential for bioaccumulation.
T criteria fulfilled?	Long term data not available (acute data >0.1 mg/L); potentially not toxic.
Overall conclusion	Not a PBT substance (based on screening data).

References

1. ECHA REACH, Citric Acid, Retrieved 2015: http://apps.echa.europa.eu
2. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2015, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
3. IPCS Citric Acid, Retrieved 2015: http://www.inchem.org
4. JECFA http://apps.who.int/ipsc/database/evaluations/chemical.aspx?chemID=4785
5. OECD, Citric Acid, Retrieved 2015: http://www.echemportal.org

AECOM Imagine it. Delivered.

Toxicity Summary - Crystalline silica-cristobalite, crystalline silica-quartz

Chemical and Physical Properties[1,3]	
CAS number	Crystalline Silica (Cristobalite) : 14464-46-1 Crystalline Silica (Quartz): 14808-60-7 Diatomacous Earth (Calcined silica): 91053-39-3
Molecular formula	Crystalline Silica (Cristobalite): SiO_2 Crystalline Silica (Quartz): SiO_2 Diatomacous Earth (Calcined silica): SiO_2
Molecular weight	60.09 g/mol
Solubility in water	Insoluble/negligible
pH	-
Melting point	1713◦C (Cristobalite) 1610◦C (Quartz)
Boiling point	2230 ◦C
Vapour pressure	NA
Henrys law constant	NA
Explosive potential	Not explosive
Flammability potential	Not flammable
Colour/Form	Transparent crystals
Overview	Silica is an off-white granule that occurs naturally in various crystalline and amorphous or other non-crystalline forms. Crystalline silica is characterized by silicon dioxide (SiO2) 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. Uncalcined diatomaceous earth typically contains around 1%crystalline silica. When diatomaceous earth is subjected to pressure or is processed ("calcined") at temperatures above 1000°C some of the amorphous silica is converted to crystalline silica in the form of cristobalite. Calcined diatomaceous earth can contain anywhere from 1% to 75% cristobalite.
Environmental Fate [1,2]	
Soil/Water/Air	Crystalline Silica consists of diatomaceous earth, a naturally occurring material. Its primary component, silica, is found in common materials like quartz, sand and agate. The materials are ubiquitous and unlikely to react chemically with any other substance in the environment.

Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	A number of animal studies have found that cristobalite is more toxic to the lung than quartz, and more tumorigenic (e.g., King et al. 1953; Wagner et al. 1980). However, several other authors concluded that this is not the case (Bolsaitis and Wallace 1996; Guthrie and Heaney 1995). OSHA (2013) has examined evidence on the comparative toxicity of the silica polymorphs (quartz, cristobalite, and tridymite) and found no difference in toxicity effects between cristobalite and quartz. Furthermore, no difference in toxicity between cristobalite and quartz has been observed in epidemiologic studies (NIOSH 2002). There is no information on the repeat dose oral, inhalation or dermal effect of calcined silica. However, since calcined diatomaceous earth contains varying amounts of crystalline silica in the form of cristobalite, and may also contain small amounts of quartz and tridymite, it is expected that any long-term health hazards associated with diatomaceous earth would mainly be due to the effects of crystalline silica. In humans, the most prevalent effect identified from long term exposure in occupational settings is silicosis, a diffused nodular pulmonary fibrosis (US EPA 1996).
Carcinogenicity	IARC (2012) concluded that there is sufficient evidence in humans for the carcinogenicity of inhaled crystalline silica in the form of quartz or cristobalite from occupational sources. There is sufficient evidence in experimental animals for the carcinogenicity of quartz and cristobalite. The IARC has also concluded that inhaled crystalline silica in the form of cristobalite or quartz from occupational sources is carcinogenic to humans (Group 1) (IARC 2012).
Mutagenicity/ Genotoxicity	Conflicting results have been reported in genotoxicity studies with crystalline quartz or cristobalite, and a direct genotoxic effect for crystalline silica has not been confirmed or ruled out. Studies on genotoxicity of calcined diatomaceous silica are not available.
Reproductive Toxicity Developmental Toxicity/Teratogenicity	No data available.
Acute Toxicity	No data available.
Irritation	No data available. Most acute toxicity studies for quartz or cristobalite were conducted using intratracheal instillation. 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 general, exposure to high concentrations of dust may cause coughing and mild, temporary irritation (CCOHS 2001).
Sensitisation	No data available. However, based on the structure and physico-chemical properties, the three forms of crystalline silica or the calcined diatomaceous silica are not expected to cause skin sensitisation.
Health Effects Summary	The substances are not skin or eye irritants but acute inhalation of dust may cause discomfort and stress as well as signs of local irritation to nasal, bronchiolar and ocular mucous membranes. Based on the evaluation of the epidemiological data it is concluded that inhalation exposure to crystalline silica results in lung cancer. This conclusion is also supported by animal studies in which inhalation and intratracheal exposure to crystalline silica resulted in lung tumours. The most common types of lung tumour observed in rats were lung adenocarcinomas.
Key Study/Critical Effect for Screening Criteria	Not applicable.

Ecological Toxicity [1,2,3]	
Aquatic Toxicity	Aquatic toxicity studies performed at saturation concentrations of synthetic amorphous silica showed no acute toxicity to fish, Daphnia, or algae, though some physical effects were observed with loading rates of greater than or equal to 10 g/L (OECD 2004). Any harmful effects to aquatic ecosystems are therefore not ecotoxicological in nature. No chronic toxicity data were identified.
Determination of PNEC aquatic	Not applicable.
Current Regulatory Controls [3]	
Australian Hazard Classification	Quartz and cristobalite are listed in the Hazardous Substances Information System (HSIS) (Safe Work Australia 2014a) as hazardous substances. Calcined silica is not listed in the HSIS.
Australian Occupational Exposure Standards	Time Weighted Average (TWA) occupational exposure standard of 0.1 mg/m³ for quartz and cristobalite are recommended in Australia (Safework Australia 2013). A Short-Term Exposure Limit (STEL) is not recommended for any of the compounds.
International Occupational Exposure Standards	TWA for quartz, cristobalite: Canada: 0.025 mg/m³ France: 0.05 mg/m^3 Japan: 0.03 mg/m³ Sweden: 0.05 mg/m^3 US (ACGIH): 0.025 mg/m^3 US (NIOSH): 0.05 mg/m^3 US (OSHA): 0.1 mg/m^3 US: 0.3, 0.9, 1.5, 500 mg/m^3 Temporary Emergency Exposure Limits (TEEL) (Diatomaceous silica, calcined)
Australian Food Standards	No data found.
Australian Drinking Water Guidelines	The Australian Drinking Water Guidelines state: 'To minimise an undesirable scale build up on surfaces, silica (SiO¬2) within drinking water should not exceed 80 mg/L' (National Health and Medical Research Council (NHMRC) 2001).
Aquatic Toxicity Guidelines	No data found.
PBT Assessment [3]	
P/vP Criteria fulfilled?	No. Not applicable, inorganic substance, ubiquitous in environment.
B/vB criteria fulfilled?	No. Not applicable, inorganic substance, ubiquitous in environment.
T criteria fulfilled?	No. Long term data not available (acute data >0.1 mg/L).
Overall conclusion	It is not currently possible to categorise the environmental hazards of metals and other inorganic chemicals according to standard persistence, bioaccumulation and toxicity (PBT) hazard criteria. These criteria were developed for organic chemicals and do not take into account the unique properties of inorganic substances and their behaviour in the environment (UNECE 2007; US EPA 2007). Further assessment of the environmental risks from the use of this chemical is not required as identified by DoEE
Revised	April 2018

References

1. HSDB. Hazardous Substances Data Bank. Retrieved 2015, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
2. OECD-SIDS Initial Targeted Assessment Profile on Quartz and Cristobalite, SIAM 32, 19-21 April 2011.
3. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme

AECOM Imagine it. Delivered.

Toxicity Summary - Diethanolamine

Chemical and Physical Properties [1,2,4]	
CAS number	111-42-2
Molecular formula	C4H11NO2
Molecular weight	105.14
Solubility in water	1,000 g/L @ 20 °C
Melting point	27 °C at 101.3 kPa
Boiling point	269.9 °C at 101.325 kPa
Vapour pressure	0.0028 hPa (25 °C)
Henrys law constant	3.97 x 10-6 Pa*m^3/mol
Explosive potential	Non explosive
Flammability potential	Non flammable
Colour/Form	Colourless crystals or a white syrupy liquid with a mild ammonical odour.
Overview	2,2'-Iminodiethanol (diethanolamine, DEA) belongs to the ethanolamines group that includes monoethanolamine (MEA), diethanolamine (DEA) and triethanolamine (TEA). Large-scale production of DEA is carried out by the reaction of ethylene oxide and excess ammonia, followed by fractionation of the three ethanolamines (mono-, di- and triethanolamine). Ethanolamines are used widely as intermediates in the production of anionic and non-ionic surfactants, which have become commercially important as detergents, textile and leather chemicals, and emulsifiers. Their uses range from drilling and cutting oils to medicinal soaps and high-quality toiletries. DEA is an important additive of corrosion inhibitors, particularly in coolants for automobile engines. DEA is also employed as an additive in lubricants and in cement/concrete production. Large amounts of DEA are used as such in closed systems for absorptive gas purification to remove weakly acidic components. In the production of detergents, cleaners, fabric softeners and metalworking fluids DEA is used for acid neutralization and to prevent soil deposition. DEA is also used as an intermediate in the production of morpholine, photographic chemicals and polyurethanes. In addition, DEA is used as a building block for agrochemicals.
Environmental Fate[4]	
Soil/Water/Air	The colourless solid DEA is completely miscible with water at ambient temperature and has a negligible vapour pressure of 0.0028 hPa (25 °C). The measured log KOW of -2.18 (25 °C) and the calculated BCF of 3.16 indicate a low potential for bioaccumulation. The Henry's law constant of 3.97 x 10-6 Pa*m^3/mol (uncharged) is considered as an indication for low volatility. The calculated Koc of uncharged DEA is 1 (corrected log Koc = 0). Thus, the potential for adsorption to soil, sediment, and suspended solid may be low. However, binding of the substance to the matrix of soils (and sediments) with high capacities for cation exchange (e.g. clay) cannot be excluded for the charged molecule. The measured pKa value of 8.92 (23 °C) indicates that at environmentally relevant conditions of pH 6 – 8, the molecule will predominantly occur in the charged (cationic) form. At pH values > 9, DEA will predominantly be present as the uncharged species. According to Mackay Level I modelling, uncharged DEA will distribute almost completely into water (99.99 %). DEA is readily biodegradable according to OECD criteria. Potential for anaerobic degradation of DEA was also observed. In the atmosphere, it will be photodegraded by reactions with OH radicals (calculated half-life of the uncharged molecule for a 12-hour day and 1.5E06 OH/cm^3: 2.4 hours = 0.1 day; for a 24-h day and 0.5E06 OH/cm^3: 4.2 hours = 0.2 days). At environmental pH conditions hydrolysis is not expected to be a relevant degradation process due to the absence of hydrolysable groups
Human Health Toxicity Summary [1,2]	

Chronic Repeated Dose Toxicity	In a 90 day oral gavage study conducted similarly to OECD TG 408 in F344 rats, lowest observed adverse effect levels (LOAEL) of 320 and 160 ppm (equivalent to 25 and 14 mg/kg bw/day, respectively) was reported in male and female rats, respectively. These were the lowest doses tested. Mortality was observed in males (2/10 animals) at the highest dose (5000 ppm) before the completion of the study (REACH; OECD, 2008). Signs of toxicity were observed across all dose groups (160 - 2500 ppm), and included tremors, extreme weight loss, abnormal posture and a dose dependent increase in microcytic anaemia. Dose related (≥ 320 ppm in males and ≥ 160 ppm in females) changes in kidney weights were associated with an increase in nephropathy and renal cell necrosis. Dose related (≥ 320 ppm in males and ≥ 630 ppm in females) increase in liver weight was associated with a moderate increase in serum bile acid concentration (REACH; OECD, 2008). Based on treatment-related effects reported with a LOAEL of 32 and 80 mg/kg bw/day in rat and mouse studies, respectively, the chemical is considered to cause serious damage to health from repeated oral exposure. In a 90 day dermal application study conducted similarly to OECD TG 411 in F344 rats, a LOAEL of 32 mg/kg bw/day was reported in male and female rats. Mortality occurred in one male and two female rats administered the highest dose of 500 mg/kg bw/day (REACH; OECD, 2008). Ulceration, inflammation, hyperkeratosis, and acanthosis occurred at all administered doses (32 - 500 mg/kg bw/day). Other signs of toxicity included reductions in body weight gain, anaemia, renal function changes and liver weight increases. Demyelination in the brain, nephropathy and renal tubular necrosis were also observed (REACH; OECD, 2008). In a similar study conducted similarly to OECD TG 411 in B6C3F1 mice, a LOAEL of 80 mg/kg bw/day was reported in male and female mice. Effects on the skin were noted at all doses (80 - 1250 mg/kg bw/day) and consisted of acanthosis at the lower doses and a dose-dependent increase in ulcerations, inflammation and hyperkeratosis at higher dose levels (630 and 1250 mg/kg bw/day in males and females, respectively) (REACH; OECD, 2008). Further signs of toxicity included dose dependent increases in liver and kidney weights. The increase in liver weight was associated with hepatocellular changes consisting of enlarged hepatocytes and, at the higher dose levels, the presence of multinucleated, giant hepatocytes. Liver damage (hepatocellular necrosis) was observed in male mice only (REACH; OECD, 2008). Based on the available data no adverse systemic toxicity was evident. Local effects were observed at a lowest observed adverse effect concentration (LOAEC) of 0.15 mg/L in one study. The available data do not warrant a hazard classification for repeated dose inhalation toxicity. However, a classification for respiratory irritation is warranted. In a 90 day inhalation study conducted according to OECD TG 413 in Wistar rats, a LOAEC of 0.15 mg/L was reported in male and female rats. Local inflammation (focal squamous metaplasia and hyperplasia) was evident in the larynx (0.15 mg/L) and trachea (0.4 mg/L) in a concentration dependent manner (REACH, SIDS, 2008). Marginal increases in liver weight and serum alkaline phosphatase levels occurred at the mid - high doses (0.15 and 0.4 mg/L, respectively), although, no histopathological changes were noted. In females, erosions of the glandular stomach occurred in a dose dependent manner (0.15 mg/L and 0.4 mg/L) (REACH; OECD, 2008). A further study conducted according to OECD TG 413 in male and female Wistar rats using lower doses (0.0015, 0.003 or 0.008 mg/L) showed similar local irritation effects (focal squamous metaplasia) after 90 days of exposure. After 90 days of exposure to the chemical, a group of 10 animals were given three months of recovery. At the end of the recovery period, no treatment related systemic effects were observed, indicating reversibility in the laryngeal epithelium up to the highest dose administered (0.008 mg/L) (REACH, OECD, 2008).

Carcinogenicity	Limited data are available on the carcinogenicity of DEA. A two-year carcinogenicity study was conducted by the United States National Toxicology Program (NTP, 1999). Based on the pattern of occupational and consumer exposure, dermal administration was considered the most appropriate route for the carcinogenicity study in rats and mice. Groups of 50 male F344/N rats were administered dermal doses of 0, 16, 32, or 64 mg/kg bw DEA in ethanol solutions, 5 days per week for 103 weeks. Female rats were administered 0, 8, 16, or 32 mg/kg bw, and male and female B6C3F1 mice were administered 0, 40, 80, or 160 mg/kg bw DEA dermally, 5 days per week for 103 weeks. Mean body weights of treated rats were generally lower than those of the control rats. The only clinical finding attributed to DEA administration was irritation of the skin at the site of application. This effect was dose-related. Exudate, consisting of focal accumulations of serum and cellular debris on the epidermal surface, occurred at significantly increased incidences in 64 mg/kg bw males and in all dosed female groups. In rats, the main histopathological effects were noted in kidneys of female rats with nephropathy, renal tubular epithelial cell necrosis and/or mineralisation, which increased in incidence and/or severity in a dose-dependent manner. The incidence of nephropathy in dosed female groups was significantly greater than that in the vehicle controls; but no such effects were seen in male rats. There was no neoplastic response in the skin or any organ associated with DEA exposure during the two-year study. The incidence of basophilic foci was significantly decreased in all dosed groups of males and females. The incidence of fibroadenoma in mammary glands in female rats occurred with a negative trend, being lower in all dosed groups compared to the historical control range. In mice, mean body weights of treated groups were depressed, more so in female mice than in male mice. The liver was clearly the most affected organ, and female mice were more sensitive than males. Exposure to diethanolamine for two years produced a marked neoplastic response in the liver characterised by significant increases in the incidences and multiplicity of hepatocellular adenomas (males: 31/50, 42/50, 49/50, 45/50 and females: 32/50, 50/50, 48/50, 48/50) and hepatocellular carcinoma (males: 12/50, 17/50, 33/50, 34/50 and females: 5/50, 19/50, 38/50, 42/50) at 0, 40, 80 and 160 mg/kg bw/day, respectively. The microscopic appearance of these liver neoplasms was typical of those usually observed spontaneously in B6C3F1 mice. There was a morphologic continuum from adenoma to carcinoma, with less differentiation and typical trabecular formations in the carcinomas. Increased mortality was noted in female mice and this, along with reduced body weights, was considered to be a consequence of the presence of liver neoplasms. The incidence of hepatoblastomas, uncommon phenotypic variants of hepatocellular carcinoma, was significantly increased in male mice, but not in females. In addition, the incidence of syncytial alteration, a non-neoplastic lesion characterised by the presence of hepatocytes containing multiple (three or more) nuclei, was increased in all groups of dosed mice; this lesion was not present in the controls. Centrilobular cytoplasmic alteration was increased in treated males but was not present in females. There were no neoplasms of the skin in mice. Effects in the kidneys included increased organ weights and increased incidence of tubular epithelial cell necrosis. The incidences of renal tubule adenoma and renal tubule adenoma or carcinoma (combined) occurred with a positive trend in male mice, but renal tubule carcinoma did not follow the same pattern. Detailed evaluation of the renal neoplasms indicated a treatment- and dose-related increase in the incidences of renal tubule adenoma (1/50, 4/50, 6/50 and 6/50) and adenoma or carcinoma (combined) (3/50, 5/50, 6/50 and 8/50 at 0, 40, 80 and 160 mg/kg, respectively). Diethanolamine is eliminated in urine as the parent compound. The data on the mode of action are insufficient to conclude that diethanolamine-induced tumours in mice are relevant for humans and, therefore, based on the available information, diethanolamine is not classified for carcinogenicity.

Mutagenicity/ Genotoxicity	The chemical tested negative in several in vitro (Ames test with and without metabolic activation, reverse mutation assay, cytogenic assay and the mouse lymphoma assay) and in vivo (micronucleus assay and the alkaline elution assay) tests for gene mutation and clastogenicity (NICNAS; OECD, 2008).
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No reproductive toxicity studies are available for diethanolamine. Repeated dose studies were conducted in F344/N rats and B6C3F1 mice of both sexes for 13 weeks (10/sex/species/dose) to characterise the effects of oral and dermal exposure (NTP, 1992). No reproductive toxicity in male or female rats was reported following dermal administration of the chemical for 13 weeks. There were no morphological effects on male or female reproductive organs or in sperm parameters (NTP, 1992). It is likely that testicular degeneration in a 90-day drinking water study is a direct toxic effect of diethanolamine. However, no effect on the reproductive organs of the female rats was noted. The NOAEL for reproductive effects in males is 630 ppm (48 mg/kg bw/day). In an inhalation study, conducted according to OECD TG 413, male and female Wistar rats were exposed to the chemical via inhalation (0.015, 0.15 or 0.4 mg/L), five times a week for 90 days. Reproductive effects in males were reported at the highest concentration (0.4 mg/L) and these included testicular atrophy and slight atrophy of the prostate. No changes were observed in female rats (OECD, 2008). The effects of diethanolamine on the male reproductive system are indicative of a potential to impair reproductive capability. However, more detailed reproductive toxicity studies are needed to confirm the potential effects on fertility observed in male rats. The current information is insufficient to classify diethanolamine for reproductive toxicity. Developmental effects were tested following exposure of dams to diethanolamine by oral, dermal and inhalation routes. In almost all the rodent studies, developmental effects were seen only at higher doses, at which maternal effects were also noted. In a dermal study in rabbits, the overall incidence of malformation was similar to the incidence seen in control animals. The current data therefore do not allow for a clear delineation of reproductive and developmental toxicity of diethanolamine in experimental animals. Classification of diethanolamine for reproductive and developmental toxicity is, therefore, not recommended at this stage.

Acute Toxicity	The reported oral median lethal dose (LD50) values in rats ranged from 780 - 3540 mg/kg bw (OECD, 2008). In one study male Sprague Dawley (SD) rats administered a single oral dose of aqueous DEA (100 – 6400 mg/kg bw) resulting in 90 % mortality at the highest dose. Doses greater than 100 mg/kg bw resulted in an increase in liver weight. An increase in the relative kidney weight was observed at doses greater than 1600 mg/kg bw. Clinical chemistry changes were reported for the liver at doses greater than 200 mg/kg bw and for the kidney at greater than 400 mg/kg bw (OECD, 2008). The chemical was of low acute toxicity in animal tests following dermal exposure. The median lethal dose (LD50) in rabbits is greater than 12000 mg/kg bw (IUCLID, 2000). The chemical was of low acute toxicity in animal tests following inhalation exposure. The median lethal concentration (LC50) in rats is 6.4 mg/L. The available data do not warrant hazard classification. Acute inhalation exposure to the chemical for 1.5 – 4 hours at concentrations between 30 – 1476 ppm (0.13 - 6.4 mg/L) caused mortality in 5/8 rats after 105 minutes of exposure to 6.4 mg/L. Exposure to 3.35 mg/L (768 ppm) for up to 4 hours resulted in no mortality. It was reported that the exposure was to vapours or aerosols (most likely at the higher concentration). Observed sub-lethal effects included lethargy, increased breathing, increased blood pressure, congestion in the lung and discolouration in the kidney and thymus (REACH; OECD 2008).
Irritation	The chemical on unabraded rabbit skin produced skin irritation after 1 - 15 minutes and marked irritation after 20 hours. Over 72 hours, erythema increased and oedema decreased (REACH). After 20 hours of exposure the mean Draize scores for erythema and oedema formation were 2 and 1.33, respectively. While the Draize scores for erythema and oedema returned to normal after 8 days, severe desquamation of the skin persisted. The chemical is also reported to cause ulceration, inflammation and hyperkeratosis following repeated exposure. In an eye irritation study in Vienna White rabbits, 0.05 mL of the chemical was instilled into the rabbit's eyes and observed for eight days. The chemical caused signs of severe irritation consisting of superficial corrosion, corneal opacity, conjunctival bleeding, conjunctivitis and oedema (OECD, 2008; REACH). Extensive corrosion was evident at the end of the observation period. In a further study, 0.1 g of the chemical was applied into the conjunctival sac of New Zealand White rabbits. This resulted in strong irritation of the cornea, iris and conjunctiva, which did not completely resolve over seven days of observation (OECD, 2008).
Sensitisation	The chemical was not found to induce dermal sensitisation in the Guinea pig maximization test conducted according to OECD Test Guideline (TG) 406 (OECD, 2008).
Health Effects Summary	The critical health effects for risk characterisation include systemic acute effects (acute toxicity by the oral route of exposure) and local effects (skin, eye and respiratory irritation). The chemical may also cause harmful effects following repeated exposure through oral and dermal routes.
Key Study/Critical Effect for Screening Criteria	The lowest observed adverse effect levels (LOAEL) of 320 and 160 ppm (equivalent to 25 and 14 mg/kg bw/day, respectively) were reported in male and female rats, respectively, based on kidney and liver weights in the drinking water study (US NTP, 1992). In mice, the LOAEL was 630 ppm (104 mg/kg bw/day for males and 142 mg/kg bw/day for females) based on liver weight changes. It is reported that the fatal oral dose of the chemical is 20g in humans (HSDB).
Ecological Toxicity [3,4]	

Aquatic Toxicity	The lowest reliable acute toxicity values for aquatic species were as follows: Pimephales promelas (fish) 96-h LC50 = 1370 mg/l (nominal) Daphnia magna (invertebrates) 48-h EC50 = 55 mg/l (nominal) Pseudokirchneriella subcapitata 96-h ErC50 = 2.2 mg/l (nominal) Pseudomonas sp. (microorganisms) 16-h TTC = 16 mg/l (nominal) In a chronic toxicity test on reproduction of the water flea Daphnia magna, the NOEC (21 days) was 0.78 mg/l (nominal, based on analytical verification).
Determination of PNEC aquatic	Using an uncertainty factor of 50 on the lowest NOEC to Daphnia a PNEC (Predicted No Effect Concentration) of 0.02 mg/L is calculated, for aquatic organisms.
Current Regulatory Controls [1]	
Australian Hazard Classification	The chemical is classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia): Xn; R22 (Acute toxicity) Xi; R38/41 (Irritation) Xn; R48/22 (Repeated dose toxicity)
Australian Occupational Exposure Standards	The chemical has an exposure standard of 13 mg/m³ (3 ppm) time weighted average (TWA).
International Occupational Exposure Standards	The following exposure standards are identified (Galleria Chemica): An exposure limit (TWA) of 2 - 15 mg/m³ (0.46 – 3 ppm) in different countries such as USA (Alaska, Hawaii), Canada (Yukon), Norway and Switzerland.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment [4]	
P/vP Criteria fulfilled?	No. DEA is readily biodegradable according to OECD criteria.
B/vB criteria fulfilled?	No. Based on a measured log Kow of -2.18 and a calculated BCF of 3.16, this chemical does not meet the screening criteria for bioaccumulation.
T criteria fulfilled?	No. The chronic aquatic toxicity of the chemical is > 0.01 mg/L. Hence the substance does not fulfil the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). Human Health Tier II Assessment for Ethanol, 2,2'-iminobis-, Retrieved 2019: https://www.nicnas.gov.au
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). Human Health Tier III Assessment for Ethanol, 2,2'-iminobis-, Retrieved 2019: https://www.nicnas.gov.au
3. ECHA REACH, 2,2'-iminodiethanol, Retrieved 2019: https://echa.europa.eu/
4. OECD (2002) SIDS Initial Assessment Profile for 2,2'-iminodiethanol (diethanolamine, DEA)

AECOM Imagine it. Delivered.

Toxicity Summary - 2,2''-oxydiethanol (Diethylene glycol)

Chemical and Physical Properties[1,2,3,4]	
CAS number	111-46-6
Molecular formula	$C_4H_{10}O_3$
Molecular weight	106.1 g/mol
Solubility in water	Miscible
Melting point	-10°C
Boiling point	245°C
Vapour pressure	It has a low vapour pressure (<0.01 kPa at 25°C).
Henrys law constant	$2.0X10^{-9}$ atm-cu m/mol at 25 °C
Explosive potential	Not explosive
Flammability potential	Combustible
Colour/Form	Odourless, colourless, viscous and hygroscopic liquid with a sharply sweetish taste
Overview	Diethylene glycol (DEG) is produced via a non-catalytic reaction between ethylene oxide and water at high pressure temperature. The resulting crude ethylene glycols (EG) are dried. The water-free glycol mixture is subsequently fractionated by vacuum distillation into mono, di and triethylene glycol. Biodegradation of polyethylene glycols results in chain shortening with concomitant formation of ethylene glycol and diethylene glycol in nature DEG is a widely used chemical in industrial and household applications. It is also used in cosmetics for topical use. DEG is not an approved food additive in Australia. However, DEG is allowable in food in Australia as an impurity in polyethylene glycol (PEG) used as a processing aid or miscellaneous food additive. PEG used for this purpose must contain no more than 0.25% w/w DEG.
Environmental Fate[1,4]	
Soil/Water/Air	EGs emitted to the atmosphere readily undergo hydroxyl radical induced photodegradation, with half-lives ranging from about 2 to 15 hours. Particulate-phase EGs may be physically removed from the atmosphere by wet deposition (SRC, 2003). EGs have limited volatility, decreasing with increasing molecular weight. Level III fugacity modelling and Henry's Law constants ranging from 1.31×10^{-7} to 7.62×10^{-15} atm-m^3/mole indicate that volatilization from water to the atmosphere is limited. EGs are inherently to readily biodegraded in water. Since these substances are resistant to water hydrolysis, abiotic degradative processes in water are not major elimination pathways. Fugacity modelling indicates that EGs have a high affinity for soil as well as water. Low soil/sediment coefficients (Koc = 1 to 10) suggest that these substances are highly mobile in soil, have limited tendency to adsorb onto suspended solids and sediment, and are therefore subject to biodegradative elimination in either soil or water. Overall, the data suggest that EGs do not persist in the environment and that they have limited potential for bioaccumulation.

Human Health Toxicity Summary [1,2,3,4,5]	
Chronic Repeated Dose Toxicity	Two well-conducted studies were identified from which effect levels from long-term oral DEG administration could be derived (OECD, 2004; Health Council of the Netherlands 2007). In these two studies by Gaunt et al. (1976*) using DEG doses in food of 0%-4% (0.3-3.7 g/kg bw/d) for 98 days and 0%-2% (0.05-1.5 g/kg bw/d) for 225 days in Wistar rats (10-15/sex/dose), kidney effects were reported consisting of oxalate crystalluria, increased urine volumes and histopathological evidence of hydropic degeneration and tubular necrosis. For the crystalluria and increased urine volumes, there were inconsistent findings between male and female rats and questionable dose-response relationships. For example, the number of male rats with urinary oxalate crystals was not increased at the highest male dose of 1.2 g/kg bw/d in the 225 day study. In addition, the observed increase in urinary volumes was possibly caused by the osmotic diuretic effect of DEG and the oxalate crystalluria could not be explained in view of oxalic acid being a minor metabolite of DEG in rats. Therefore, the significance of elevated production of oxalate was regarded as unclear (Health Council of the Netherlands, 2007) and was viewed as a biomarker and not an indication of toxicity (OECD, 2004). OECD (2004) identified a LOAEL for kidney effects of 230 mg/kg bw/d from the 225 day study based on increases in urine volume. The NOAEL was 100 mg/kg bw/d. Health Council of the Netherlands (2007) regarded a NOAEL based on renal histopathological findings as more relevant than a NOAEL based on increased urine volumes. From the 98 day study, a LOAEL based on renal hydropic degeneration was established at 1.6 g/kg bw/day with the NOAEL at 300 mg/kg bw/d (Health Council of the Netherlands, 2007).
Carcinogenicity	The International Agency for Research on Cancer (IARC) has not evaluated DEG as a carcinogen. Urinary bladder calculus and tumour responses were recorded in some long-term oral studies in the rat. Bladder tumours were found associated with the formation of oxalate containing bladder stones in a 2-year feeding study by Fitzhugh and Nelson (1946*). On the other hand, Weil et al. (1965*, 1967*) found that DEG did not induce bladder tumours in rats unless a foreign body or lesion was present, such as an oxalate- containing bladder stone or a surgery-induced bladder lesion. These authors concluded that the bladder tumours seen were due to mechanical irritation by oxalate-containing bladder stones rather than the carcinogenic response to DEG. In more recent studies such as Ito et al. (1988*), Masui (1988*) and Hiasa et al. (1990* and 1991*), DEG did not demonstrate any evidence of carcinogenic effects after oral administration. Several studies in mice also showed that DEG is not carcinogenic after dermal application. No information was found in the literature concerning the occurrence of bladder stones in humans after ingestion of DEG. Overall, although some human carcinogenicity information are available, data are insufficient (e.g. lack of a quantitative estimate of DEG exposure and sound methodology) to evaluate the carcinogenic potential of DEG.
Mutagenicity/ Genotoxicity	DEG was shown to be negative in the majority of gene mutation and chromosome aberration studies in vitro. Some indications of chromosomal damage were seen in vivo only at high doses. Taken together, DEG is considered non-genotoxic.

Reproductive Toxicity / Developmental Toxicity/Teratogenicity	In oral studies, adverse effects on fertility were seen in mice and foetal abnormalities occurred in rats and mice. Inhalation and injection studies in rabbits and hamsters also revealed foetal abnormalities and other adverse effects on the foetus. However, reduced fertility was observed only at high doses of DEG, up to 6.1 g/kg bw/d in mice with maternal toxicity. With regard to developmental toxicity, a significant decrease in mean foetal body weight in mice was seen at 10 g/kg bw/d in the presence of maternal toxicity. In addition, at an oral dose of 6.1 g/kg bw/d in a 2-generation study in mice, craniofacial malformations, including exencephaly and cleft palate, and related mortality were observed in the presence of maternal toxicity. In rats, a decreased foetal body weight with increased skeletal variations was seen at 4.5 g/kg bw/d in the presence of maternal toxicity. Foetal malformations were not observed at dose levels up to 8.9 g/kg bw/d. From these studies, the NOAEL for fertility and developmental effects is established at 3.1 g/kg bw/d with a LOAEL of 6.1 g/kg bw/d based on reductions in litters/pair, live pups/litter and live pup weight
Acute Toxicity	In animals, the acute oral, dermal and inhalational toxicity of DEG are low. Oral toxicity is similar for both rats and mice with LD50 values ranging 13-30 g/kg bw across both species. A single study of dermal toxicity in rabbits derived an LD50 value of 12.5 or 13.3 g/kg bw . Acute inhalational toxicity has also been tested in rats and mice. The 4-hour LC50 in rats was 4600 mg/m^3. In humans, mortality and morbidity are high in cases of inadvertent DEG ingestion, with most deaths occurring within the first 2 weeks post exposure. Neurological impairments observed after exposure include encephalopathy, demyelinating neuropathy, optic neuritis, facial paralysis, cerebral oedema and haemorrhages. Acute anuric renal failure with metabolic acidosis and concomitant severe neurological abnormalities progressing to coma and finally death were also noted during severe intoxications after uptake of DEG in patients with burns. A median lethal oral dose of 1.49 g/kg bw DEG (range 0.25-4.9 g/kg bw) was estimated from large-scale intoxication of Haitian children with a paracetamol syrup contaminated with DEG. However, large overlaps in ranges of lethal and non-lethal doses have been observed for adults and children. Accidents in humans following acute DEG exposure have been recorded. A large number of mass poisonings in humans involving substitution of DEG for more expensive, non-toxic, glycols in medicinal preparations have been documented over the past 70 years. Typical features of acute toxicity include neurological impairment, metabolic acidosis and acute renal failure. Early mortality and morbidity are high, with most deaths occurring within the first two weeks following DEG exposure. Humans appear to be 10 times more susceptible to acute oral toxic effects of DEG compared with experimental animals, with median lethal dose of 1490 mg/kg bw in humans compared with > 15000 mg/kg bw in rats (NICNAS, 2009).
Irritation	Overall, available data indicate that DEG causes no or only minimal skin and eye irritation in laboratory animals. Respiratory depression was reported in mice although the characteristics were reported as not typical of a pure airway irritant (OECD, 2004). No other information on respiratory irritation was available. Similar to experimental animals, DEG causes no or only minimal skin irritation in humans. Data for eye irritation in humans were not available.
Sensitisation	DEG does not cause skin sensitisation in guinea pigs. In humans, there is a single case study reporting skin sensitisation 2-4 weeks after a man had started smoking a brand of cigarettes containing DEG. However, overall, available data indicate that DEG is not a skin sensitiser in humans.
Health Effects Summary	The critical health effects for risk characterisation include systemic acute effects (acute toxicity from oral exposure).

Key Study/Critical Effect for Screening Criteria	The effects of diethylene glycol on the liver and kidneys after prolonged oral exposure are considered as the critical effects. Key study is the oral exposure study in rats carried out by Gaunt *et al.* (1976). the NOAEL for hydropic degeneration is 300 mg/kg bw/day (0.4% diethylene glycol in food) in the male rats (Health Council of the Netherlands, 2007). Uncertainty factors: 10 (interspecicies variability); 10 (intraspecies variability); 10 (sub-chronic to chronic) Oral RfD = 300/1000 = 0.3 mg/kg/day Drinking water guidance value = 1.17 mg/L
Ecological Toxicity [1,4]	
Aquatic Toxicity	Fish acute toxicity (measured as LC50 in mg/L) for DEG ranges from >1000 mg/L to 77900 mg/L. The lowest acute toxicity (LC50) to invertebrates (Daphnia) value was >100 mg/L (48hr LC50) . Algal toxicity has been tested for DEG with an EC50 of >1000 mg/L. Chronic toxicity to fish was also tested which resulted in a 7 day LC50 of 61,000 mg/L and chronic toxicity data on pentaEG are available for algae (NOEC – 100 mg/L)
Determination of PNEC aquatic	On the basis that 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 for algae (100 mg/L). The PNEC aquatic is 2.0 mg/L.
Current Regulatory Controls [6]	
Australian Hazard Classification	The chemical is classified as hazardous with the following risk phrase for human health in HSIS (Safe Work Australia): Xn; R22 (Harmful if swallowed)
Australian Occupational Exposure Standards	TWA (time weighted average) = 100 mg/m^3 (Safe Work Australia).
International Occupational Exposure Standards	TWA = 101 mg/m^3 [UK] (HSE, 2013).
Australian Food Standards	No data available
Australian Drinking Water Guidelines	No data available
Aquatic Toxicity Guidelines	No data available
PBT Assessment [1,4]	
P/vP Criteria fulfilled?	DEG is readily biodegradable and as such not persistent in the environment.
B/vB criteria fulfilled?	An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low.
T criteria fulfilled?	The acute aquatic toxicity of DEG is > 0.01 mg/L. Hence the substance does not fulfill the screening criteria for toxic (T).
Overall conclusion	Not a PBT substance (based on screening data).
Revised	December 2018

References

1. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2017, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS), 2009. Diethylene glycol (DEG)—hazard assessment
3. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). Human Health Tier II assessment for Ethanol, 2,2'-oxybis-, CAS Number: 111-46-6, Retrieved 2018: https://www.nicnas.gov.au

4. OECD (2004) SIDS Initial Assessment Profile for Ethylene Glycols Category, CAS Number 107-21-1, 111-46-6, 112-27-6, 112-60-7, 4792-15-8
5. Health Council of the Netherlands. 2007. Diethylene glycol; Health based recommended occupational exposure limit. The Hague; Health Council of the Netherlands; publication no 2007/03OSH. Accessed 2017 at https://www.gezondheidsraad.nl/sites/default/files/200703osh_diethylene_glycol.pdf
6. Safe Work Australia 2011. Workplace Exposure Standards for Airborne Contaminants.

AECOM Imagine it. Delivered.

Toxicity Summary - Boric acid/sodium tetraborate / boronatrocalcite / boron sodium oxide

Chemical and Physical Properties[1,3,5,8]	
CAS number	Boric Acid: 10043-35-3 Sodium Tetraborate: 1330-43-4 Boronatrocalcite: 1319-33-1 Boron sodium oxide: 12008-41-2
Molecular formula	Boric acid: H_3BO_3 Sodium Tetraborate: $Na_2B_4O_7$ Boronatrocalcite: $CaNaH_{12}(BO_3)5.2H_2O$ Boron sodium oxide: $B_8Na_2O_{13}$
Molecular weight	Boric acid: 61.833 g/mol Sodium Tetraborate: 201.220 g/mol Boronatrocalcite: 405.23 g/mol Boron sodium oxide: 340.47
Solubility in water	Boric acid: 49.20 g/l @ 20± 0.5 °C Sodium Tetraborate: 3.1% at 25 °C Boronatrocalcite: no data found Boron sodium oxide: 223.65 g/L @ 20 °C
pH	Boric acid: 6.1 in a 0.1% (wt) solution Sodium Tetraborate: 9.3 at 20 °C (3% solution) Boronatrocalcite: no data found Boron sodium oxide: no data found
Melting point	Boric Acid: 170.9 °C Sodium Tetraborate: 743 °C Boronatrocalcite: no data found Boron sodium oxide: 813 °C
Boiling point	Boric Acid: 300 C Sodium Tetraborate: 1,575 °C (decomposes) Boronatrocalcite: no data found Boron sodium oxide: no data found
Vapour pressure	Boric acid: 9.9×10^{-6} Pa @ 25 °C Sodium Tetraborate: Negligible at 20 °C Boronatrocalcite: no data found Boron sodium oxide: no data found
Henrys law constant	No data found
Explosive potential	Not explosive
Flammability potential	Not flammable
Colour/Form	Boric Acid: Colourless, transparent crystals or white granules or powder. Sodium Tetraborate: Colourless, monoclinic crystalline salt; also occurs as a white powder. Boronatrocalcite: Silky white rounded crystalline masses or parallel fibres. Boron sodium oxide: Solid white powder. Odourless.

Overview	Limited toxicity data is available for sodium tetraborate (Borax anhydrous) and boronatrocalcite (Ulexite) as such; this toxicity profile includes data on boron and boric acid. Boric acid and borate salts exist naturally in rocks, soil, plants and water as forms of the naturally occurring element boron. Anhydrous Borax is a free flowing mixture of clear, glass-like particles and white granules formed by the crushing of relatively large masses of fused materials. Borax is a salt of boric acid. Borax occurs naturally in evaporite deposits produced by the repeated evaporation of seasonal lakes and has many applications in chemistry, mining and pharmaceuticals. Ulexite is a sodium-calcium-hydroborate and, like other borates, is a structurally complex mineral. It is composed of hydrogen (3.98 %), sodium (5.67 %), calcium (9.89 %), boron (13.34 %), and oxygen (67.12 %) There is a lack of data available in the literature to directly assess the toxicity of the chemical. The major component of the chemical is a borate ion, which is likely to be associated with human health hazards of the chemical. The other constituents are considered to be of low concern to human health (NICNAS, 2013). As the chemical will readily break down in the stomach pH to boric acid (H_3BO_3) following ingestion, the toxicokinetics and toxicity of the chemical will be driven predominantly by borate ions. Boron is a naturally occurring element that is found in the form of borates in the oceans, sedimentary rocks, coal, shale, and some soils. Boron is widely distributed in nature, with concentrations of about 10 mg/kg in the earth's crust (range 5 mg/kg in basalts to 100 mg/kg in shales) and about 4.5 mg/L in the ocean. Borates are used in glass, ceramics, detergents, wood treatment and insulation fiberglass industries. Boric acid and other borates are also used in a range of consumer products including cosmetic and personal care products and also in detergents. Moreover, borates are essential for all plants – their use as fertilizers increases crop yields (including grapes, potatoes, sugar beets, alfalfa and olives) and quality. Boron occurs in foods as borate and boric acid. Boron has not been established to be an essential nutrient for humans and no specific biochemical function for boron has been identified in higher animals or man. There is some evidence that, in humans, boron intake within the usual dietary range may influence the metabolism and utilisation of other nutrients, particularly calcium, and may have a beneficial effect on bone calcification and maintenance.
Environmental Fate[2,4]	
Soil/Water/Air	All of the chemical in this group will transform into boric acid in the aquatic environment. This simple mononuclear boron compound is highly water soluble and is the predominant form of dissolved boron in surface waters. It is a mobile species in the environment and is to be found in all major environmental compartments.

Human Health Toxicity Summary [2,3,4,8,9]	
Chronic Repeated Dose Toxicity	The haematological system and the testes have been identified as the major targets after oral repeat dose exposure to Boric acid. Studies after repeated dermal or inhalation exposure to boric acid are not available. A NOAEL for effects on testes and the blood system of 17.5 mg boron/kg bw/day can be derived (with a LOAEL of 58.5 mg boron/kg bw/day) from two 2-year studies in rats on boric acid. Results obtained with boric acid can be supported by findings obtained from other borates thus indicating that the boron ion is the toxicologically relevant species
Carcinogenicity	Boric acid is not listed as an IARC carcinogen. In long term feeding studies on boric acid and disodium tetraborate decahydrate in both rats and dogs, no carcinogenic effects were observed.
Mutagenicity/ Genotoxicity	Boric acid is not mutagenic either in vitro or in vivo.
Reproductive Toxicity Developmental Toxicity/Teratogenicity	Results from animal experiments demonstrate that boric acid adversely effects fertility and development. Feeding studies in different animal species (rats, mice and dogs) have consistently demonstrated that the male reproductive system is the principle target in experimental animals, although effects on the female reproductive system have also been reported. Testicular damage ranging from mildly inhibited spermiation to complete atrophy has been demonstrated following oral administration of boric acid. Effects on fertility were observed at lower dose levels compared to dose levels, where signs of general toxicity appeared. Based on data from the two-year feeding studies with boric acid and borax in rats, 17.5 mg boron /kg bw/day (equivalent to 100 mg boric acid/kg bw/day)_was derived as a NOAEL for male and female fertility. Developmental effects have been observed in three species, rats, mice and rabbits. The most sensitive species appears to be rats, in which the effects observed at non maternally toxic doses include a reduction in foetal body weight and minor skeletal variations which, with the exception of short rib XIII, had reversed by 21 days post-natal. The NOAEL for developmental effects is 9.6 mg boron/kg bw/day (55 mg boric acid/kg/day).
Acute Toxicity	Boric acid is of low acute toxicity. LD50 oral rat > 3765 mg/kg bw (659 mg boron/kg/bw); LD50 dermal rabbits > 2000 mg/kg bw/day; 4 hour LC50 inhalation rat ≥ 2.03 mg/L.
Irritation	In rabbits, boric acid caused no/mild skin irritation, induced reversible conjunctival redness and chemosis with minor effects on the iris. In rats and mice, boric acid acts as a sensory irritant. The substance may irritate the eyes, nasal mucous membranes, skin and the respiratory tract, and may cause effects on the gastrointestinal tract, liver and kidneys.
Sensitisation	No borate tested has displayed skin sensitisation in Bheuler studies. No evidence of skin sensitisation has been seen in humans exposed occupationally to sodium borates, or in a human patch test with a 3% aqueous boric acid solution.
Health Effects Summary	Borates are of low acute toxicity and low skin irritation potential. It may cause sensory irritant effects on animals and humans with acute exposure. Borates were shown not to be skin sensitisers, genotoxic or carcinogenic. Repeated exposures to boron as boric acid induced effects on fertility (testes), development and the blood system.
Key Study/Critical Effect for Screening Criteria	The critical lowest No Observed Adverse Effect (NOAEL) level for the purposes of risk assessment is 9.6 mg boron/kg bw/day. This NOAEL was the equivalent of 55 mg boric acid/kg bw/day; 38 mg disodium octaborate anhydrate/kg bw/day and 85 mg borax/kg bw/day), from feeding (dietary intake) studies based on developmental effects. Uncertainty factors: 10 (interspecies variability); 10 (intraspecies variability); 10 (subacute to chronic). Drinking water guideline for boron: 3.5 ppm

Ecological Toxicity [3,9]	
Aquatic Toxicity	The most sensitive tests report that acute effects on fish are in the range of 10-20 mg-B/L although the quality of these studies was rated low. The lowest daphnid acute value is 133 mg-B/L. Algal and microbial inhibition studies suggest less toxicity: Selenastrum growth was not affected at 93 mg-B/L and activated sludge respiration showed minimal effects at 683 mg/L boric acid (119 mg-B/L). Chronic endpoints for Boric acid were available for Daphnia (6 mg/L) and Fish (2.1 mg/L).
Determination of PNEC aquatic	Canadian Water Quality Guidelines for the Protection of Aquatic Life: Long–term Exposure to Boron is 1.5 mg/L (2009). An assessment factor of 100 has been applied to the lowest reported chronic effect concentration of 2.1 mg/L for Fish. The PNECaquatic is 0.021 mg/L.
Current Regulatory Controls [9]	
Australian Hazard Classification	Boric acid and borax are classified as hazardous for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia 2013) with the following risk phrases: · Toxic to reproduction (Repr.) Cat. 2; R60 (May impair fertility) · Repr. Cat. 2; R61 (May cause harm to the unborn child) Mixtures containing boric acid and borax are classified as hazardous with the following risk phrases based on the concentration (conc) of the chemicals in the mixtures. · Boric acid: Conc ≥5.5%: Toxic (T); R60; R61 · Borax: Conc ≥8.5%: T; R60; R61.
Australian Occupational Exposure Standards	There are no specific exposure standards for boric acid or disodium octaborate anhydrate. However, the permissible exposure limits (as the time weighted average (TWA)) for dusts apply (10 mg/m^3 measured as inspirable dust) (Safe Work Australia 2013b). The exposure standard for borax is 5 mg/m^3 TWA (Safe Work Australia 2013a).
International Occupational Exposure Standards	The following exposure standards were identified (Galleria Chemica 2013): · Boric acid − Canada 2 mg/m^3 TWA, 6 mg/m^3 Short-term exposure limit (STEL) (borate compounds) − Germany 10 mg/m^3 TWA; 1 mg/m^3 STEL − Spain 10 mg/m^3 TWA (insoluble particles) − US 2 mg/m^3 TWA; 6 mg/m^3 STEL (borate compounds), 5 mg/m^3 TWA (particulates, respirable fraction) · Disodium octaborate anhydrate − Canada 10 mg/m^3 TWA, (insoluble particles) − Spain 10 mg/m^3 TWA (particulates, inhalable fraction) − US 5 mg/m^3 TWA (particulates, respirable fraction) · Borax − Canada 1 to 5 mg/m^3 TWA, 6 mg/m^3 STEL (inorganic borate compounds) − Denmark 1 to 2 mg/m^3 TWA − Germany 0.5 mg/m^3 TWA − Spain 5 mg/m^3 TWA − Sweden and UK 2 mg/m^3 TWA − US 2 mg/m^3 TWA (inorganic borate compounds); 5 to 10 mg/m^3 TWA.
Australian Food Standards	No data found.
Australian Drinking Water Guidelines	No aesthetic or health-related guidance values exist specifically for boric acid, disodium octaborate anhydrate or borax. However, the guidelines note that boron in the environment is likely to be predominantly in the form of boric acid and that based on health considerations, the concentration of boron in drinking water should not exceed 4 mg/L (NHMRC 2011).
Aquatic Toxicity Guidelines	For boron: 90 µg/L (ANZECC 2000 99% Freshwater)
PBT Assessment [9]	
P/vP Criteria fulfilled?	For the purposes of this PBT assessment, the persistent criteria is not considered applicable to this inorganic substance.

B/vB criteria fulfilled?	For the purposes of this PBT assessment, the bioaccumulation criteria is not considered applicable to this inorganic substance.
T criteria fulfilled?	No. The chronic toxicity data is >1 mg/L.
Overall conclusion	Not PBT
Revised	April 2018

References

1. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2015, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
2. ECHA REACH, Acetic Acid, Retrieved 2015: http://apps.echa.europa.eu
3. Human and Environmental Risk Assessment (HERA) on Ingredients of Household Cleaning Products: Boric Acid, 10043-35-3, 2005. http://www.heraproject.com
4. EFSA, Opinion of the Scientific Panel on Dietetic Products, Nutrition and Allergies on a request from the Commission related to the Tolerable Upper Intake Level of Boron (Sodium Borate and Boric Acid), 2004
5. Draft European Union Risk Assessment Report. Disodium tetraborate, Anhydrous Boric Acid, Boric Acid, Crude natural (1) Risk Assessment. 2007
6. IPCS Sodium Tetraborate, Retrieved 2016: http://www.inchem.org
7. EFSA, European Food Safety Authority, Scientific Opinioni on the re-evaluation of boric acid (E284) and sodium tetraborate (borax) (E285) as food additives. 2013
8. NICNAS, National Industrial Chemicals Notification and Assessment Scheme, Human Health Tier II Assesssment for Ulexite, CAS Number: 1319-33-1, 2015
9. National Industrial Chemicals Notification and Assessment Scheme (NICNAS, 2017). National assessment of chemicals associated with coal seam gas extraction in Australia. Human health hazards of chemicals associated with coal seam gas extraction in Australia.
10. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme

AECOM Imagine it. Delivered.

Toxicity Summary - Ethanol

Chemical and Physical Properties[1,2,3]	
CAS number	64-17-5
Molecular formula	C2H6O
Molecular weight	46.07
Solubility in water	1 x 10_3 g/L at 25 °C
Melting point	114.14 °C
Boiling point	78.3 °C
Vapour pressure	57.3 hPa at 20°C
Henrys law constant	0.000252
Explosive potential	Non explosive
Flammability potential	Highly flammable (100%)
Colour/Form	Clear, colourless liquid with a characteristic pleasant odour and burning taste.
Overview	Ethanol, also known as grain alcohol, is a clear, colourless liquid. It has an alcohol odour a burning taste. Ethanol mixes easily with water. Ethanol is present in emissions from plants, fires, volcanoes, animal wastes, insects and natural fermentation of sugars. Ethanol is an important commercial chemical used in alcoholic beverages, which may contain up to 50% ethanol. It is also used as a solvent in cleaners and as a fuel additive. Ethanol is used in the production of other chemicals, pharmaceuticals, perfumes, and cosmetics. It is also used as a fungicide and to regulate plant growth. It is an ingredient in many consumer products, such as cleaners, sprays, inks, mouthwash, perfume and aftershave, and human and veterinary medicines. Ethanol is a food additive.
Environmental Fate[3]	
Soil/Water/Air	Ethanol is stable to hydrolysis but is readily biodegradable (74% after 5 days) and is not likely to bioaccumulate (calculated logBCF=0.5). Ethanol is not persistent in the environment. Fugacity-based modelling shows that ethanol released into the environment will become distributed mainly into air and water. Relative distributions between compartments based on an emission pattern of 1000:100:10 were 57 % in air, 34 % in water, and 9 % in soil. These predictions are supported by the limited data available on prevailing concentrations, which shows that ethanol has been detected in outdoor air and in river water. The total tropospheric half-life of ethanol is estimated to be 10-36 hours, with degradation due to hydroxyl, NOx and SOx radical-mediated photooxidation. As a volatile organic compound in the atmosphere, ethanol is a potential contributor to tropospheric ozone formation under certain conditions, however its photochemical ozone creation potential is considered to be moderate to low (40-45 relative to ethylene as 100).
Human Health Toxicity Summary [1]	

Chronic Repeated Dose Toxicity	Many repeated dose studies of chemical have been conducted in many species, predominantly with the aim of assessing adverse effects associated with the consumption of alcoholic beverages. Consequently, these are mostly conducted through oral exposure and with doses well in excess of those that might be encountered in occupational exposure or consumer products (OECD, 2005), or unintentional public exposures from environmental contamination. Considering the lowest observed adverse effect level (LOAEL) available from a 90-day rat study (3600 mg/kg bw/day), and based on the treatment-related effects reported in various repeated dose toxicity studies, the chemical is not considered to cause serious damage to health from repeated oral exposure, except from exposure to high doses. In a well-conducted repeated dose toxicity study, the chemical was administered (in a liquid diet) to Sprague Dawley (SD) rats at a 1, 2, 3, 4, 5, and 10 % concentration for 90 days. Water consumption in the 10 % group was reduced relative to controls. There were no adverse clinical signs or mortality during the study. Serum liver enzymes were unaffected by treatment and kidney findings were reported to be minimal. A LOAEL was established at 3 % (approximately 3600 mg/kg bw/day), based on dose-related hepatic yellowing, centrilobular steatosis, increased frequency and severity of Mallory bodies (hyaline), and acidophilic degeneration and necrosis. The no observed adverse effect level (NOAEL) was 2 % (approximately 2400 mg/kg bw/day) (OECD, 2005; REACH). In another repeated dose toxicity study conducted in accordance with national test guidelines of USA (EPA OPPTS 870.3100), the chemical was administered in drinking water to Fischer 344 (F344) rats and B6C3F1 mice at a single dose of 5 % concentration for 90 days. Even though male rats showed minor changes in thymus weights, and some slight but inconsistent changes in haematology and clinical chemistry, these effects were not considered adverse. Based on water consumption data, this single dose study established a 5 % nominal NOAEL for male rats (approximately 3250 mg/kg bw/day). Although minor changes in clinical chemistry were also seen in female rats, some female rats (4/10) also exhibited liver nodules (diaphragmatic nodules) and small increases in liver weights. As no NOAEL could be established for female rats, a LOAEL of 4400 mg/kg bw/day was established. For male mice, a LOAEL at 9700 mg/kg bw/day was established, based on increased organ weights (liver, heart, kidney and lung) and decreased sperm counts in the cauda epididymis. Although female mice showed small changes in the length of dioestrus and pro-oestrus, the overall cycle length was unchanged. As biological significance of these changes was unclear, a NOAEL for female mice was established at 5 % (9400 mg/kg bw/day) (OECD, 2005; REACH). As properly conducted studies in animals are not available, there are no valid data on the effects of repeated inhalation exposure to the chemical. However, limited information is presented below to indicate that the chemical is likely to be of low toxicity following repeated inhalation exposure. In a repeated dose toxicity study, SD male rats (10/dose) were exposed to the chemical through inhalation (whole body exposure) continuously at 20 mg/L for three, six, nine, and 26 days. Although initial exposure to the chemical produced a number of transient effects (lethargy, ataxia and intoxication, mild hepatic vacuolisation and changes to clinical chemistry parameters), animals adapted and appeared normal at the end of the study. Induction of metabolic tolerance to the chemical was also indicated as it was noted that the levels of the chemical in the blood of animals exposed for 26 days were much lower than those exposed for shorter periods (REACH). In another repeated dose toxicity study, the chemical was administered through inhalation at 0 or 6300 ppm (1 ppm = 1.92 mg/m³) to SD rats (10/sex/dose) for six hours/day, five days/week, for four weeks (total of 20 days exposure). Additional groups of animals (five/sex/dose) were also included in the study to determine reversibility of effects for a further four weeks following cessation of treatment. There were no treatment-related clinical signs of toxicity and there were also no gross pathological or histological changes reported of the major organs. Body weights, liver enzyme levels, haematology, and clinical chemistry parameters were otherwise normal (REACH).

Carcinogenicity	The International Agency for Research on Cancer (IARC) has concluded that there is sufficient evidence in humans and experimental animals to establish carcinogenicity of alcohol consumption and ethanol, respectively. It was also concluded that there is sufficient evidence in experimental animals to establish carcinogenicity of acetaldehyde (major metabolite of ethanol). Consequently, IARC has classified that 'alcohol consumption is carcinogenic to humans (Group 1)' and that 'ethanol in alcoholic beverages is carcinogenic to humans (Group 1)'. This conclusion was supported by an analysis of the expanded human dataset that carcinogenic effects appeared independent of the type of alcoholic beverage (IARC, 2010; IARC, 2012). As the use of the chemical in alcoholic beverages is not considered in this report, the above assessment of carcinogenicity of alcohol beverages may not be relevant to occupational exposure to the chemical or from using the chemical in consumer products (OECD, 2005). Furthermore, studies in animals conducted mostly through oral exposure at very high doses, exceeding the 'maximum tolerated dose', may be of little relevance when assessing risks associated with occupational exposure or using consumer products containing the chemical (OECD, 2005). Thus, classification is not considered appropriate.
Mutagenicity/ Genotoxicity	Overall, the data indicate that the chemical has no mutagenic or genotoxic potential (OECD, 2005; REACH). The results from numerous bacterial mutation assays of the chemical have generally been negative. A very weak positive effect of the chemical was found in an Escherichia coli DNA repair test but not in Ames tests with Salmonella typhimurium conducted by the same authors. In separate studies, there have been positive results reported in Ames tests, but only at concentrations of the chemical significantly greater than those specified in test guidelines. The chemical is therefore not considered mutagenic in bacteria.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	The chemical does not show specific reproductive or developmental toxicity. Any reproductive and developmental effects were only observed secondary to maternal toxicity. As results of inhalation studies showed no developmental toxicity from chemical exposures even at maternally toxic doses, it can be concluded that deliberate oral consumption of alcoholic beverages is required for any reproductive or developmental toxicity (OECD, 2005).
Acute Toxicity	The chemical has low acute toxicity by oral exposure in animal tests. The median lethal dose (LD50) in rats is >2000 mg/kg bw. Observed sub-lethal effects included central nervous system depression, e.g. inebriation, disturbances of gait, dose-related decreases in responses to painful stimuli, respiratory depression, and coma. Deaths were reported due to cardiorespiratory failure (OECD, 2005; HSDB; REACH). Few studies are available on the dermal toxicity of the chemical. A poorly documented rabbit study reported death in one of four animals following a dose of 20000 mg/kg bw. Although limited data are available, the apparent low dermal toxicity from this study is regarded as consistent with low uptake of ethanol through intact skin. The median lethal dose (LD50) in rats is greater than 2000 mg/kg bw. Observed sub-lethal effects were not reported for the study (OECD, 2005; REACH). The chemical has low acute toxicity by inhalation exposure in animal tests. The lowest reported median lethal concentration (LC50) is 124.7 mg/L/four hours in rats. Observed sub-lethal effects included attempts to escape, reddish-watery eyes, nasal secretions, closing of eyelids, snout wiping, intermittent respiration, loss of pain reflex, abdominal position, and apathy (OECD, 2005; REACH).

Irritation	The chemical is not regarded as irritating to skin. In a skin irritation study conducted in accordance with OECD Test Guideline (TG) 404, the chemical was applied to six New Zealand White rabbits for four hours using exposure chambers. The mean score for erythema was one at 24 hours and remained zero at all other time points (48, 72 hours); the mean score for oedema remained zero at all time points (24, 48, 72 hours). The chemical was concluded not to be irritating to the skin of rabbits. Another skin irritation study in rabbits, where the chemical was applied under occlusion for 24 hours, also showed only very slight skin irritation (OECD, 2005; REACH). The chemical produced irritant effects in several eye irritation studies in rabbits. In an eye irritation study conducted in accordance with US Federal guideline (Fed. Reg. Vol. 38, No. 187, 1973), the chemical (0.1 mL) was applied on the conjuctival sac of one eye of each of three New Zealand White rabbits. Irritation responses were observed at 24, 48 and 72 hours and eight days following application. Mean Draize scores following grading at 24, 48 and 72 hours for three rabbits were 1 for corneal opacity, 0.22 for iritis, 2.45 for conjunctivitis, and 1.89 for chemosis. Mean Draize scores following grading at day eight were 0.67 for corneal opacity, 1.67 for conjunctivitis, and 1.33 for chemosis. While iris lesions were fully reversible by day eight, other eye lesions were not fully reversible at this time. Given the observation period did not extend to 21 days, it is difficult to conclude any findings on the reversibility of the irritation. The average response of 2/3 animals was sufficiently severe in terms of conjunctival effects (>2.5) and chemosis (³2) observed, that classification as an eye irritant is warranted (REACH). In another eye irritation study (OECD TG 405), the chemical (0.1 mL) was applied to the eyes of three rabbits (strain not specified) and observed up to 14 days. Mean Draize scores at 24, 48 and 72 hours were 2.11 for conjunctivitis, 1.33 for chemosis, 0.44 for iritis, and 1.11 for corneal opacity. Although all symptoms subsided by day 14, conjunctivitis was still present at day seven. As positive responses for corneal opacity (mean score >1 for 2/3 animals) and conjunctival redness (mean score >2 for 2/3 animals) were noted in the study, the chemical is considered to be an eye irritant (category 2A) (OECD, 2005; REACH). In an eye irritation study (OECD TG 405), the chemical (0.1 mL) was applied into the lower conjunctival sac of one eye of six New Zealand White rabbits and observed up to 72 hours. Reported average Draize scores at 24, 48 and 72 hours were 2.39 for redness of the conjunctivae, 1.2 for chemosis, 0.28 for iritis, and 1.2 for corneal opacity. As conjuctival redness persisted for 24 hours with a mean score of >2 and corneal opacity was noted with a mean score >1, the chemical is considered to be an eye irritant (category 2A) (OECD, 2005; REACH). In an eye irritation study conducted in accordance with US Federal guideline (Fed. Reg. 28 (119), 5582, 1963), the chemical (0.1 mL) was applied on the lower lid of one eye of six New Zealand White rabbits. The eyes were examined at 24, 48, and 72 hours and at day seven following administration of the chemical. Mean Draize scores following grading at 24, 48 and 72 hours were 1.72 for conjunctivitis, 1.78 for chemosis, 0.83 for iritis, and 1.28 for corneal opacity. While iris lesions were fully reversible at day seven, other eye lesions were not. Mean Draize scores following grading at day seven were 0.83 for conjunctivitis, 0.83 for chemosis, and 1.17 for corneal opacity. As corneal opacity was noted with a mean score >1, the chemical is considered an eye irritant (category 2A). In addition, whilst mean scores for conjunctival redness and chemosis were <2, scores ³2 were noted in four out of six animals (OECD, 2005; REACH).

Sensitisation	The available data indicate that the chemical does not induce skin sensitisation in animals. The chemical, at 75 % concentration, was used as a solvent in a Magnusson and Kligman guinea pig maximisation test of a polyalkalene glycol. Skin reactions were not observed at challenge with the polyalkalene glycol in 75 % ethanol in either the test or negative control animals (OECD, 2005). In a mouse ear swelling test, no increase in ear thickness was observed following a challenge application of the chemical at 95 % (OECD, 2005; REACH). In a mouse local lymph node assay (LLNA) (OECD TG429) the chemical, or diethyl phthalate, were used as vehicles to examine the skin sensitisation potential of four test fragrance materials. The concentration of the chemical in this study varied from 0–100 %. The level of induced T-lymphocyte proliferation was low for the chemical compared with that for fragrance materials known to be mild to moderate skin sensitisers, and comparable with the other negative control vehicle (diethyl phthalate). On the basis of a lack of sensitising potential up to a concentration of 100 %, the test concluded that the chemical is an appropriate vehicle for use in a local lymph node assay (REACH).
Health Effects Summary	While exposure to the chemical through consuming alcoholic beverages is associated with an increased risk of carcinogenicity and reproductive and developmental toxicity, these risks increase in a dose-dependent manner and are not considered relevant at doses relating to occupational exposure and using consumer products containing the substance such as mouthwash. Therefore the critical health effect for risk characterisation from industrial use of the chemical is a local effect: eye irritation.
Key Study/Critical Effect for Screening Criteria	Overall, the most sensitive endpoint for ethanol is repeat dose toxicity. The oral NOAEL was 2,400 mg/kg bw/day. This NOAEL is used in this human health risk assessment.
Ecological Toxicity [2,3]	
Aquatic Toxicity	The aquatic toxicity data in fish, invertebrates, and algae indicate a low order of acute toxicity with LC50/EC50 values greater than 1000 mg/L. The most sensitive species were algae Chlorella vulgaris with a 96hr EC50 of 1000 mg/L and the invertebrate Artemia Salina with a 24hr LC50 of 1833 mg/L. Valid chronic toxicity data are available for two trophic levels. NICNAS (2017) reported a measured chronic endpoint of 7800 mg/L for Daphnia.
Determination of PNEC aquatic	A PNECaqua = 780 mg/L can be calculated based on the chronic toxicity value (NOEC = 7800 mg/L) for aquatic invertebrates (Daphnia) with the assessment factor of 10.
Current Regulatory Controls [1,4]	
Australian Hazard Classification	The chemical is not classified for health hazards on the Hazardous Substances Information System (HSIS) (Safe Work Australia).
Australian Occupational Exposure Standards	The chemical has an exposure standard of 1880 mg/m³ (1000 ppm) time weighted average (TWA).
International Occupational Exposure Standards	The following exposure standards are identified (Galleria Chemica): An exposure limit (TWA) of 960–1920 mg/m³ (500-1000 ppm) in countries such as Canada, Denmark, Germany, Sweden, South Africa, Switzerland, United Kingdom, and the United States of America. An exposure limit (STEL) of 1900–1920 mg/m³ (1000 ppm) in countries such as Canada, Sweden, and Switzerland.

Australian Food Standards	Ethanol has the following listings in the Australia New Zealand Food Standards Code (Food Standards Australia and New Zealand 2013): · as a permitted food additive subject to GMP (ethanol) (Standard 1.3.1 Food additives) · as a generally permitted processing aid (ethyl alcohol) (Standard 1.3.3 Processing aids) · as a permitted component of wine (alcohol) (Standard 2.7.3 Fruit wine and vegetable wine) · as subject to a composition limit in brewed soft drinks (no more than 1.15% alcohol/volume) (Standard 2.6.2 Non-alcoholic beverages and brewed soft drinks) · As subject to a composition limit in: − wine and sparkling wine (no less than 45mL ethanol/L and not to contain added ethanol) − fortified wine (no less than 150 mL ethanol/L and no more than 220 mL ethanol/L) − brandy (must contain no less than 250 mL/L of the spirit distilled at a strength of no more than 830 mL ethanol/L at 20°C (Standard 4.5.1 Wine production requirements).
Australian Drinking Water Guidelines	No aesthetic or health-related guidance values were identified for this chemical in the Australian Drinking Water Guidelines (NHMRC 2011).
Aquatic Toxicity Guidelines	1400 µg/L (95% protection level) (ANZECC & ARMCANZ, 2000)
PBT Assessment [2]	
P/vP Criteria fulfilled?	No. Ethanol is readily biodegradable (74% after 5 days).
B/vB criteria fulfilled?	No. Ethanol is not likely to bioaccumulate (calculated logBCF=0.5).
T criteria fulfilled?	No. Chronic aquatic toxicity (NOEC) >1mg/l, thus ethanol does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier II Assessment for Ethanol: Retrieved 2019: https://www.nicnas.gov.au
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS, 2017). National assessment of chemicals associated with coal seam gas extraction in Australia. Human health hazards of chemicals associated with coal seam gas extraction in Australia.
3. ECHA REACH, Ethanol, Retrieved 2019: https://echa.europa.eu/
4. OECD (2005) SIDS Initial Assessment Profile for Ethanol
5. ANZECC & ARMCANZ (2000), Australian and New Zealand Guidelines for Fresh and Marine Water Quality

AECOM Imagine it. Delivered.

Toxicity Summary - Ethylene glycol

Chemical and Physical Properties [1,2]	
CAS number	107-21-1
Molecular formula	C2H6O2
Molecular weight	62.07 g/mol
Solubility in water	Miscible with water.
pH	No data found
Melting point	-12.69 ºC
Boiling point	197.3 ºC
Vapour pressure	0.092 mm/Hg at 25C
Henrys law constant	Low. 6.00X10-8 atm-cu m/mol at 25 deg C
Explosive potential	Not explosive
Flammability potential	Lower flammable limit of 3.2% by volume; Flashpoint of 232 deg F (111 deg C). Not combustible.
Colour/Form	Colourless odourless liquid
Overview	Ethylene glycol is a clear, colourless, syrupy liquid with a sweet taste but no odour. It has low volatility. It is miscible with water and some other solvents, slightly soluble in ether, but practically insoluble in benzene, chlorinated hydrocarbons, petroleum ethers, and oils. As a small molecular weight alcohol, ethylene glycol readily passes through biological membranes and will be effectively absorbed from the gastrointestinal tract and via inhalation exposure. It is rapidly distributed in body water. The chemical has numerous domestic and commercial uses, and is found in cleaning products, cosmetics, hydraulic brake fluids, anti-freeze agents and corrosion inhibitors. Ethylene glycol has been assessed by NICNAS to be of low environmental concern when used in coal seam gas extraction.
Environmental Fate [1,3,5]	
Soil/Water/Air	Ethylene glycol released to the atmosphere will be degraded by reaction with hydroxyl radicals; the half-life for the compound in this reaction has been estimated at between 0.3 and 3.5 days. No hydrolysis of ethylene glycol is expected in surface waters. The compound has little or no capacity to bind to particulates and will be mobile in soil. The low octanol/water partition coefficient and measured bioconcentration factors indicate low capacity for bioaccumulation Ethylene glycol is readily biodegradable in standard tests using sewage sludge. Rapid degradation has been reported in surface waters (less in salt water than in fresh water), groundwater, and soil.

Human Health Toxicity Summary [1,2,3,4,6,7]	
Chronic Repeated Dose Toxicity	Considering the lowest observed adverse effect levels (LOAELs) available from 13–104 week studies (300–3000 mg/kgbw/d) (ATSDR, 2010), and based on the treatment-related effects reported in various repeated dose toxicity studies, the chemical is not considered to cause serious damage to health from repeated oral exposure. However, there is evidence of cumulative effects, as the nephropathy observed at high doses in acute toxicity studies also occurs after repeated exposure at lower doses. The National Toxicology Program (NTP) conducted a 13 week and a two year study in B6C3F1 mice. In the 13 week study, 10 male and 10 female mice were administered 0, 3200, 6300, 12500, 25000 or 50000 ppm ethylene glycol incorporated into feed. There were no reported deaths and no chemical-related clinical findings were reported. Histopathology showed chemical-related kidney and liver lesions, which were significantly elevated in the 25000 and 50000 ppm male mice. These lesions included nephropathy and centrilobular hepatocellular hyaline degeneration (NTP, 1993). The two year study used 60 male mice dosed with the chemical at 0, 6250, 12500 or 25000 ppm and 60 females dosed at 0, 12500, 25000 or 50000 ppm in feed. The doses in ppm were reported as being equivalent to: males - 0, 1500, 3000 or 6000 mg/kg bw/d and females - 0, 3000, 6000 or 12000 mg/kg bw/d. There were no significant differences in survival although male mice in the high dose (6000 mg/kg bw/d) group had to be housed separately after week 54 due to excessive fighting. Survival of mice was not affected by ethylene glycol administration at all doses. As with the 13 week study, mice did not show any adverse clinical signs. Histopathology showed hepatocellular degeneration in the mid and high dose male and high dose female mice. Pulmonary arterial hyperplasia occurred at a higher incidence in female mice than male mice exposed to the chemical. Some male mice in the high dose group had oxalate-like crystals and/or calculi in the renal system (NTP, 1993). Mice appear to be less sensitive than rats to ethylene glycol. A two-year study conducted in Fischer-344 (F344) rats found that administration of the chemical (40, 200 or 1000 mg/kg bw/d) resulted in excessive mortality in male rats in the high dose group after nine months. All male rats in the high dose group (1000 mg/kg bw/d) were reported dead by 15 months of the study. Survival was significantly reduced in male rats in the 1000 mg/kg bw/d group only. (Cruzan et al., 2004; DePass et al., 1986). Pathology investigation of the male rats concluded that extensive kidney damage was the reason for increased mortality in the 1000 mg/kg bw/d group. The NOAEL for male rats was reported as 200 mg/kg bw/d in this study (DePass et al., 1986). A further study indicates that the Wistar rat strain is more sensitive than the F344 strain. In a 16-week study, 10 male rats of each strain were exposed to the chemical (0, 50, 150, 500 or 1000 mg/kg bw/d) by incorporation in a normal diet. Mortality was reported in two Wistar rats at the highest dose and significant weight loss was observed in Wistar rats administered 500 and 1000 mg/kg bw/d, respectively. Both strains of rats treated with ≥ 500 mg/kg bw/d had increased calcium oxalate crystals in the kidney tubules as well as crystal associated nephropathy; this was reported as being more severe in the Wistar rat strain (Cruzan et al., 2004). Further repeated dose studies conducted in rodents have reported no observed adverse effect levels (NOAELs) in the range of 150–2000 mg/kg bw/d depending on species and strain studied. Overall, repeated oral exposure to ethylene glycol is consistently associated with adverse effects on the kidney such as crystal nephropathy in rodents (ATSDR, 2010).

	In a study conducted according to OECD TG 410, five male Beagle dogs per group were dermally exposed (60 % of the total body surface area) to 0.5, 2.0 or 8 mL/kg bw/d Glysantin G 105 (automotive coolant which contains ≥ 92.5 % ethylene glycol and ≥1.4 % p-tert.-butyl benzoate (PTBBA)) daily for four weeks. Mortality (4/5 animals) was reported at the highest dose (8 mL/kg). Prior to death, animals showed signs of toxicity including staggering gait, vomiting, diarrhoea and reduced food intake. Clinical analysis showed increased creatinine and urea levels and increased incidence of calcium oxalate crystals. Pathology investigation reported oxalate nephrosis, testicular atrophy and uraemic gastroenteritis. Similar pathology findings were reported at the mid dose (2 mL/kg), but only in one animal. No mortality or any further clinical or pathological adverse effects were reported at the mid and lower doses. Further studies conducted comparing pure ethylene glycol to Glysantin G105 showed that the testicular atrophy was associated with the presence of PTBBA in Glysantin G105 and not ethylene glycol (REACH). PTBBA has known testicular toxicity (NICNAS). Mortality was reported in 1/15 rats, 3/15 guinea pigs, 1/3 rabbits, 0/3 dogs and 0/3 monkeys after exposure to 12 mg/m3 of ethylene glycol aerosol for 90 days. Apart from mortality, no specific signs of clinical toxicity were reported. In a further study, no mortality or toxicity was observed in the same range of animal species exposed to either 10 or 57 mg/m3 ethylene glycol. The authors noted that as the exposure was whole body, further oral intake from grooming may have occurred, and therefore a reliable LOAEL could not be established (ATSDR, 2010).
Carcinogenicity	Based on the available data, ethylene glycol is not considered to be a carcinogen. Histopathological investigations showed no evidence of carcinogenicity in studies conducted in various rodent species. No tumours were reported in SD rats administered up to 3000 mg/kg bw/day in the diet for two years, F344 rats administered 1000 mg/kg bw/day in the diet for one year, B6C3F1 mice administered up to 12000 mg/kg bw/day in the diet for two years and CD-1 mice administered up to 1000 mg/kg bw/day in the diet for two years (NTP, 2004; WHO, 2002). A limited number of epidemiological studies have reported that exposure to the chemical does not increase the risk of cancer. Ethylene glycol exposure (inhalation) in 1666 chemical plant employees was not found to increase the odds ratio (OR) for any type of cancer (ATSDR, 2010).
Mutagenicity/ Genotoxicity	Based on the weight of evidence from the available in vitro and in vivo genotoxicity studies the chemical is not considered to be genotoxic. An Ames assay conducted according to OECD TG 471 reported that the chemical did not induce bacterial mutations in Salmonella typhimurium strains TA 1535, TA 1537, TA 98, TA 100 and Escherichia coli WP2 at a concentration up to 5000 □g/plate with or without metabolic activation (REACH). Further in vitro genotoxicity tests conducted with bacterial and mammalian cell lines were all negative for gene mutations and DNA strand breaks respectively (ATSDR, 2010). An in vivo study in mice reported no chromosomal aberrations in Swiss mice exposed to 638 mg/kg bw/day for two days (WHO, 2002). Negative results were found for dominant lethal mutations in F344 rats after administration of up to 1000 mg/kg bw/d ethylene glycol in a 155-day multi-generational study.

Reproductive Toxicity Developmental Toxicity/Teratogenicity	The available data from rat studies suggest that developmental effects were only observed secondary to maternal toxicity, so the chemical does not show specific developmental toxicity. The chemical is not toxic to reproduction. Having reviewed the available data the Centre for the Evaluation of Risks to Human Reproduction (CERHR) expert panel concluded that there are sufficient data to conclude that the chemical is not toxic to reproduction in rats orally exposed to 1000 mg/kg bw/day in diet (NTP, 2004). A study in mice gave negative results at doses up to 2826 mg/kg bw/day via drinking water. The expert panel also concluded that exposure of CD-1 mice to the chemical by the dermal route for 6 hours/d on gestation days (GD) 6-15 resulted in no evidence of developmental toxicity up to a dose of 3549 mg/kg bw/d. Developmental toxicity was also not observed in rabbits exposed orally via gavage on GD 6-19 to doses as high as 2000 mg/kg bw/d. Severe maternal toxicity was observed at the high dose with maternal deaths as well as oxalate crystals in the kidney. Data suggested that oral exposure to high doses of the chemical (≥500 mg/kg bw/d in CD-1 mice and ≥1000 mg/kg bw/d in SD rats) on GD 6-15 causes developmental effects in mice and rats such as axial skeletal malformations, external malformations, reduced body weights and increased post-implantation loss (NTP, 2004). The CERHR expert panel concluded that developmental toxicity may not be attributed directly to the chemical but from the accumulation of glycolic acid, which is a metabolic breakdown product of ethylene glycol. The developmental effects are seen at doses that exceed saturation of glycolic acid metabolism. Observations from rat studies suggest that oral doses resulting in developmental toxicity (1000 mg/kg bw/d) are greater than those associated with maternal and renal toxicity at 500 mg/kg bw/d.
Acute Toxicity	Ethylene glycol has low acute toxicity via oral, inhalation, or dermal exposure. LD50s for the oral administration of ethylene glycol in rats range from 4000 to 10 020 mg/kg body weight, while reported values in guinea-pigs and mice are 6610 mg/kg body weight and 5500–8350 mg/kg body weight, respectively. The minimum lethal oral dose in rats is 3.8 g/kg body weight (Clark et al., 1979). Oral LD50s of 5500 and 1650 mg ethylene glycol/kg body weight have also been reported in dogs and cats, respectively. A dermal LD50 of 10 600 mg/kg body weight has been reported for rabbits. In rats and mice, the lethal concentration following 2-h inhalation exposure has been reported to be >200 mg/m3.
Irritation	The available data show that the chemical is a mild skin irritant in animals. Mild dermal irritation was reported in rabbits and guinea pigs. No dermal effects were reported in female CD-1 mice exposed to 3549 mg/kg bw/day ethylene glycol under occlusive conditions for 6 hours/day on gestation days 6-15 (NTP, 2004; WHO, 2002). The available data indicate that the chemical is a mild eye irritant in animals. In a study conducted in six New Zealand White rabbits, 0.05 mL of the chemical (4 or 40 %) applied to one eye (while the other eye served as a control) at 10 minute intervals for a total of 35 applications in a six hour period was reported to cause chemosis, swelling and conjunctival redness. All eyes exposed to the chemical were reported to be normal on day seven of observation and no evidence of systemic toxicity was reported (REACH).
Sensitisation	The chemical was not found to induce dermal sensitisation when tested according to OECD Test Guideline (TG) 406 (REACH).
Health Effects Summary	Ethylene glycol demonstrates acute oral toxicity, is a mild skin and eye irritant and a respiratory irritant in humans. The chemical is not a skin sensitiser. Consistent adverse effects associated with repeated exposure to ethylene glycol in animals are the kidney effects, characterised by calcium oxalate crystal deposition and consequent renal lesions.

Key Study/Critical Effect for Screening Criteria	The key study chosen for the determination of a drinking water guidance value is the one-year rat feeding study by Wilson et al. (2005). No adverse chronic renal effects from ethylene glycol dosing were seen in animals exposed below 150 mg/kg/day. The oral RfD for ethylene glycol is thus based on the NOAEL of 150 mg/kg/day. Uncertainty factors: 10 (interspecies variability); 10 (intraspecies variability) Oral RfD = 150/100 = 1.5 mg/kg/dayDrinking water guideline value = 0.59 ppm
Ecological Toxicity [3,8]	
Aquatic Toxicity	The aquatic toxicity of the 'ethylene glycol and higher glycols' (mono-, di-, tri-, tetra- and pentaethylene glycol) is evaluated as a category. Fish acute toxicity (measured as LC50 in mg/L) has been tested for all category members and ranges from 22800 for EG to greater than 50000 for pentaEG. Toxicity to Daphnia (measured as LC50 in mg/L) is greater than 20,000 for all category members except tetraEG (LC50=7800 mg/L) indicating low toxicity, but the toxicity was not as uniform as in fish. Toxicity evaluations in another invertebrate, brine shrimp (Artemia salina) were imprecise, but appear to be more consistent than the measured Daphnia toxicity values (no toxicity observed at the highest tested dose, 20g/l for EG, 10 g/l for DEG, TEG and tetraEG). Algal toxicity has been tested for EG, DEG, TEG, and PentaEG, and no toxicity was found at concentrations less than or equal to 100 mg/L. As a worst case assumption the limit test concentration of 100 mg/L was used as NOEC value for the PNEC derivation.
Determination of PNEC aquatic	PNECaquatic: An assessment factor of 10 has been applied to the lowest reported effect concentration of 100 mg/L. The PNECaquatic is determined to be 10 mg/L.
Current Regulatory Controls [7]	
Australian Hazard Classification	Xn (Harmful); R22 (Harmful if swallowed) (Safe Work Australia 2013) Acute Toxicity: Harmful if swallowed – Cat 4 (H302) (NICNAS)
Australian Occupational Exposure Standards	Ethylene glycol has an exposure standard of 10 mg/m^3 time weighted average (TWA). A further exposure standard for ethylene glycol (vapour) is 52 mg/m^3 (20 ppm) TWA and a short-term exposure limit (STEL) of 104 mg/m^3 (40 ppm) (Safe Work Australia 2013)
International Occupational Exposure Standards	TWA: 50 mg/m3 (20 ppm) [Belgium, Hungary, UK, Finland] 26 mg/m³ (10 ppm) [Denmark, Iceland, Sweden] 25 to 50 mg/m³ (63 to 125 ppm) [Mexico, Norway] 5 mg/m³ [Russia] STEL: 20 to 40 mg/m3 (50 to 104 ppm) [Belgium, Hungary, UK, Finland, Peru, Sweden] 10 mg/m³ [Russia]
Australian Food Standards	No data found.
Australian Drinking Water Guidelines	No data found
Aquatic Toxicity Guidelines	No data found
PBT Assessment [1,3,5]	
P/vP Criteria fulfilled?	Ethylene glycol is readily biodegradable both aerobically and anaerobically and as such not persistent in the environment.
B/vB criteria fulfilled?	Based on the measured log Kow of -1.36 and a measured BCF of 10, Ethylene glycol is not bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of Ethylene glycol is > 0.01 mg/L. Hence the substance does not fulfill the screening criteria for toxic (T)
Overall conclusion	Not a PBT substance (based on screening data).
Revised	April 2018

References

1. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2016, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS), 2014. Inventory Multi-Tiered Assessment and Prioritisation (IMAP), Human Health Tier II Assessment for 1,2 – Ethanediol, CAS Number 107-21-1.
3. OECD (2004). Screening Information Dataset (SIDS) Initial Assessment Profile for Ethylene Glycols Category (CAS No.107-21-1, 111-46-6, 112-27-6, 112-60-7, 4792-15-8)
4. US Environmental Protection Agency, Integrated Risk Information System (IRIS), Chemical Assessment Summary, Ethylene Glycol, CASRN 107-21-1
5. World Health Organisation (2000), Concise International Chemical Assessment Document (CICAD) 22, Ethylene Glycol: Environmental Aspects
6. World Health Organisation (2002), Concise International Chemical Assessment Document (CICAD) 45, Ethylene Glycol: Human Health Aspects
7. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme
8. ECHA REACH, Ethane-1,2-diol, Retrieved 2019: https://echa.europa.eu/

AECOM Imagine it. Delivered.

Toxicity Summary - Fatty acids, tall-oil, ethoxylated

Chemical and Physical Properties[1]	
CAS number	61791-00-2
Molecular formula	C(18-50)H(34-98)O(3-8)
Molecular weight	UVCB
Solubility in water	No data available.
Melting point	-85 °C at 101.3 kPa
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	Non-explosive (100%)
Flammability potential	Not classified
Colour/Form	Liquid
Overview	This substance is used by consumers, in articles, by professional workers (widespread uses), in formulation or re-packing, at industrial sites and in manufacturing.
Environmental Fate[1]	
Soil/Water/Air	One study investigating the adsorption/desorption behaviour of Fatty acids, tall-oil, ethoxylated (CAS 61791-00-2) is available. The study was performed according to GLP and OECD guideline 121 (BASF 2017). 6 different peaks were observed with log Koc values ranging from < 1.8 to > 5.63. The two main components (> 85%) show log Koc values > 4. Thus, adsorption of Fatty acids, tall-oil, ethoxylated to solid soil is expected. The test with the source substance was conducted according to OECD Guideline 301B, under GLP conditions (BASF 2005). Domestic, non-adapted activated sludge was exposed to the test substance for 28 days at 22°C, and biodegradation was measured by CO2 consumption. After 28 days, the test substance 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. The test substance consists of components with log Kow values in the range of 5 to > 10 (KOWWIN v1.68) indicating a potential for bioaccumulation. But due to rapid environmental biodegradation, metabolisation via enzymatic hydrolysis (monoesters and diesters) as well as sterical hindrance of crossing biological mebranes (high molecular weight of diesters) a relevant uptake and bioaccumulation in aquatic organisms is not expected. This is supported by low BCF values of < 100 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, the test substance is not considered to be bioaccumulative.
Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	Under the conditions of this Combined Repeated Dose Toxicity Study with the Reproduction/Developmental Toxicity Screening Test, the oral administration by gavage of test substance to Wistar rats revealed no adverse signs of toxicity in male and female animals at a dose level of 1000 mg/kg bw/d. Thus, the no observed adverse effect level (NOAEL) for general systemic toxicity was 1000 mg/kg bw/d for male and female Wistar rats.
Carcinogenicity	No data available.

Mutagenicity/ Genotoxicity	The test substance is not mutagenic in bacteria, as determined in an OECD 471 study. The test substance is not chromosome damaging, as determined in an OECD 487 study. The test substance is not mutagenic in mammalian cells, as determined in an OECD 476 study.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Under the conditions of this Combined Repeated Dose Toxicity Study with the Reproduction/Developmental Toxicity Screening Test, the oral administration by gavage of the test substance to Wistar rats revealed no adverse signs of toxicity in male and female animals at a dose level of 1000 mg/kg bw/d. Thus, the no observed adverse effect level (NOAEL) for general systemic toxicity was 1000 mg/kg bw/d for male and female Wistar rats. The NOAEL for reproductive performance and fertility was set to 1000 mg/kg bw/d for male and female Wistar rats.
Acute Toxicity	In an acute oral toxicity study performed similar to OECD guideline 401 (BASF 1971), three groups of rats consisting of 10 animals/sex/dose were treated by single gavage application with an aqueous solution of the test substance (10000, 8000, 6400 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 occurred at 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 6400 mg/kg bw. Based on the results obtained under the test conditions of this study, the acute oral LD50 was determined to be > 10000 mg/kg bw. To evaluate the potential acute inhalation toxicity of the test substance an Inhalation Risk Test conducted according to a BASF internal testing method (BASF 1971). The test demonstrates the toxicity of an atmosphere saturated with vapours of the volatile components of a test substance at the temperature chosen for vapour generation (20 °C). Rats were exposed sequentially to the vapours, generated by bubbling 200 l/h air through a substance column of about 5 cm above a fritted glass disc in a glass cylinder. The animals were exposed for 8 hour. The exposure concentration was estimated to be 0.28 mg/L based on evaporated substance. In addition to mortality, clinical signs were recorded and necropsy on surviving animals performed. No mortality occurred and no clinical sign were noted during exposure and observation period. In one animal exposed for 8 hours hydrometra was observed after necropsy. Since no mortality occurred at the concentrations tested an LC50 estimation cannot be made. In another Inhalation Risk Test of similar design, Rats (12 animals) were exposed sequentially to the vapours, generated by bubbling 200 l/h air through a substance column of about 5 cm above a fritted glass disc in a glass cylinder. This time vapours were generated at 20 °C as well as 50 °C. The exposure concentrations were 0.04 mg/L and 0.34 mg/L. Rats were exposed for 8 hour. As in the previous study, no mortality occurred after exposure up to 8 hours. Clinical sings observed in the animals exposed to the vapour generated at 20°C included mild escape attempts when exposure began and at the end of the exposure period slight eye irritation was observed. The next day, the animals were without symptoms. In the animals exposed to the vapour generated at 50 °C escape attempts were noted in the first 60 minutes of exposure. Exposure to the saturated atmosphere caused slight eye irritation. At the end of the exposure period, all clinical signs were resolved. Since no mortality occurred at the concentrations tested an LC50 estimation cannot be made. Based on the inhalation studies, no conclusion on LC50 can be drawn, because the tested concentrations are too low in relation to the classification criteria.

Irritation	The test substance was not irritant or corrosive to the skin in a GLP-compliant OECD 431 and 439 study. The test substance was not irritant to the eyes in a GLP-compliant OECD 492 study. Based on the available information, classification for skin and eye irritation is not warranted, in accordance with EU Classification, Labelling and Packaging of Substances and Mixtures (CLP) Regulation No. (EC) 1272/2008.
Sensitisation	The test substance did not show an indication of skin sensitising potential in an OECD 429 (LLNA) study. However, an earlier Buehler test (OECD 406) did indicate skin sensitising potential of the substance.
Health Effects Summary	Possible sensitiser.
Key Study/Critical Effect for Screening Criteria	
Ecological Toxicity [1]	
Aquatic Toxicity	Short-term toxicity tests with the target substance for all trophic levels (fish, daphnia, algae) are available. The test substance did not indicate to be harmful to freshwater fish (96h-LL50 > 100 mg/L), but showed to be toxic to aquatic invertebrates (48h-EL50 = 12.41 mg/L) and harmful to algae (72h-EL50 = 39.7 mg/L). Hence, aquatic invertebrates were most susceptible to the test substance and this effect value was used for the PNEC derivation. Long-term toxicity data with the source substance are only available for algae. The algal test revealed the substance to be of low toxicity to algae (72h-EL10 = 7.08 mg/L). In addition, data are available for toxicity to microorganisms. A test on respiration inhibition with activated sludge resulted in an 3h-EC10 of > 10000 mg/L indicating that detrimental effects in STPs are not to be expected.
Determination of PNEC aquatic	A PNECaqua = 0.012 mg/L can be calculated based on the lowest acute toxicity value (EL50 = 12.41 mg/L) for aquatic invertebrates (Daphnia) with the assessment factor of 1000.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1]	
P/vP Criteria fulfilled?	No. Based on the results from the read-across substance, Fatty acids, tall oil, ethoxylated (EO > 1 < 2.5) (CAS 61791-00-2) is considered to be readily biodegradable.
B/vB criteria fulfilled?	No. The test substance consists of components with log Kow values in the range of xx to > 10 (KOWWIN v1.68) indicating a potential for bioaccumulation. But due to rapid environmental biodegradation, metabolisation via enzymatic hydrolysis (monoesters and diesters) as well as sterical 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 BCF values of < 100 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, the test substance is not considered to be B or vB.
T criteria fulfilled?	No. Available short-term and long-term toxicity tests with aquatic organisms resulted in effect values > 1 mg/L. Thus, this substance does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. ECHA REACH, Fatty acids, tall-oil, ethoxylated, Retrieved 2019: https://echa.europa.eu/

AECOM Imagine it. Delivered.

Toxicity Summary - Glutaraldehyde

Chemical and Physical Properties [1,2,3]	
CAS number	111-30-8
Molecular formula	C5H8O2
Molecular weight	100.11
Solubility in water	Soluble in all proportions in water and ethanol; soluble in benzene and ether.
Melting point	-14°C
Boiling point	188°C
Vapour pressure	2.03 x 10^{-3} kPa at 25 °C (50% solution)
Henrys law constant	0.011 Pa m³/mol @ 25 °C
Explosive potential	Non explosive
Flammability potential	Non flammable
Colour/Form	Colourless oily liquid. In the vapour state, glutaraldehyde has a pungent odour, with an odour threshold of 0.04 ppm.
Overview	Glutaraldehyde is manufactured in Germany by BASF and in the USA by Union Carbide Corporation. It is usually sold commercially as a 45% or 50% aqueous solution. Glutaraldehyde has a wide variety of uses throughout the world with its use spread over a number of different industries. It is used primarily as a biocide but it also has wide use as a fixative, and some use as a therapeutic agent. The principal health effects of glutaraldehyde are irritation of the skin, eye and respiratory tract, skin sensitisation and occupational asthma. Exposure data indicated that, in some situations, particularly the health care industry (disinfection), x-ray film processing and the animal health industry (spray use), health concerns may arise where available control measures such as ventilation have not been implemented to minimise exposure. Due to low and intermittent exposure, the public health risk from the industrial use of glutaraldehyde is minimal. For the use of glutaraldehyde in cosmetics, a safety margin of >400 for extensive use indicated low concern.
Environmental Fate[1]	
Soil/Water/Air	Glutaraldehyde is a hydrophilic substance that will be mainly associated with the aquatic compartment, with minor amounts partitioning to the atmosphere, following release to the environment. Hydrolysis is slow, but glutaraldehyde, like other aldehydes, undergoes aerial oxidation in solution. It biodegrades rapidly in aerobic and anaerobic aquatic environments at subcidal concentrations (below 10 mg/L) and will not bioaccumulate. Tropospheric degradation is also rapid.
Human Health Toxicity Summary [1,2,3]	

Chronic Repeated Dose Toxicity	A two-year chronic study was conducted in male and female Fischer 344 rats (NICNAS 1994). Groups of 100 male and 100 female rats were administered 0, 50, 250, or 1000 ppm w/v glutaraldehyde in drinking water (4, 17 and 64 mg/kg bw/day for the males and 6, 25 and 86 mg/kg/day for the females). The mortality rate over the treatment period was 25 to 30% for males and 19 to 23% for females with no dose-related increase. The major cause of death in all rats (control and dose groups) was large granular cell lymphatic leukaemia (LGLL). Small dose-related decreases in absolute body weight and body weight gain occurred at 250 and 1000 ppm in males and at 1000 ppm in females. Dose-related decrease in urine volumes and associated increase in osmolality were observed in higher dose animals. At necropsy at 52, 78 and 104 weeks, the only statistically significant changes in organ weights were for the kidney. Relative kidney weights were increased for males and females at 52 and 78 weeks. A significant dose-related increase in kidney weight relative to final body weight occurred for males and females in the 250 and 1000 ppm groups, including an increase in absolute kidney weight for the female rats. Changes in final body weights and the weights of other organs were minor and / or sporadic and were unlikely to be related to glutaraldehyde exposure. The total leucocyte count was significantly increased at week 104 in males at 250 and 1000 ppm, and in females at 250 ppm only. The variation in counts was large, possibly due to the large monocyte count at 250 and 1000 ppm. Changes in clinical chemistry parameters included decreases in the activities of some enzymes at 250 and 1000 ppm and occasional decreases in total protein, globulin and phosphorous; these were probably due to reduced food consumption and body weight. Gross pathology showed evidence of gastric inflammation, particularly in rats sacrificed at the end of the study, with irritation observed as ulceration, a multifocal colour change and thickening of the mucosa (dose groups not specified). Histologic examination of the tissues revealed squamous epithelial hyperplasia and keratinised cysts and oedema. Based on the observations, a NOAEL of 4 mg/kg bw/day for males and 6 mg/kg bw/day for females was established in this study. For the purpose of human health risk assessment, the lowest NOAEL (4 mg/kg bw/day) established in the two-year chronic study in rats will be used.
Carcinogenicity	In a two-year chronic/carcinogenicity study by Van Miller et al. (2002), groups of 100 male and 100 female Fischer 344 rats were treated with 0, 50, 250, or 1000 ppm w/v glutaraldehyde in drinking water. The mean glutaraldehyde consumption for each of the three groups was 4, 17 and 64 mg/kg bw/day for the males and 6, 25 and 86 mg/kg bw/day for the females. The mortality rate during the study period was 25 to 30% for males and 19 to 23% for females and was not dose-related. Gross pathology showed evidence of gastric inflammation. The main finding of the study was an increased incidence of large granular lymphocytic leukaemia (LGLL) in the spleen and liver of male and female rats in all groups, including the control group. Treated females showed a significantly increased incidence of LGLL and analysis for dose-response trend for the severity of LLGL revealed an increased severity in females at the higher dosages (53% in spleen and 54% in liver versus respectively 20% and 23% in untreated females) while no such observation were made for the males. No other significant oncogenic effects were observed during the study. Occurrence of LGLL was seen in all groups including controls; the incidence of LGLL in the 1000 ppm group was high compared to controls but no clear dose-response relationship was evident, and LGLL mainly affected treated females whereas the incidence in treated males was within the control range (REACH 2013). Historical control data for untreated Fischer 344 rats in NTP studies also indicates that the ranges for this tumour are 10 to 72% in males and 6 to 31% in females (REACH 2013). The control data in the Van Miller et al. study fitted in with the historical control data reported from NTP studies. The variability in control data for LGLL and the wide variation reported in the literature makes a definitive conclusion difficult. Base on this study, glutaraldehyde was considered not to be carcinogenic.

Mutagenicity/ Genotoxicity	Glutaraldehyde has been extensively tested for genetic activity in vitro and in vivo, however there is disagreement in the literature regarding glutaraldehyde's genetic activity (Zeiger et al. 2005). While all in vivo genotoxicity tests with glutaraldehyde gave negative results, mixed results were reported for in vitro mutagenicity tests. Early in vitro tests were negative (Watts 1984), but some recent bacterial assays and tests in mammalian cells indicated that glutaraldehyde could be mutagenic in vitro. A series of reverse mutation assays was carried out with various Salmonella typhimurium strains, with and without metabolic activation (REACH 2013). All assays with TA 100, 1535, 1537 and 98 were negative. Some assays with TA 102 and 104 gave positive results. Tests with Escherichia coli also yielded both positive as well as negative results. Glutaraldehyde induced sister chromatid exchanges in CHO cells with and without S9 metabolic activation in one laboratory, but was negative without S9 and only weakly positive with S9 in the second laboratory (NICNAS 1994). The difference in the results was attributed to slight differences between the data evaluation systems used in the two laboratories. Glutaraldehyde was not mutagenic in any of the in vivo assays such as peripheral blood micronucleus test, rat bone marrow chromosomal aberration assay and the Drosophila melanogaster sex-linked recessive lethal test (NICNAS 1994; REACH 2013). Chromosome aberrations in bone marrow cells were reported in only one out of eight studies using rats and mice, micronuclei were not induced in bone marrow cells of mice, and dominant lethal mutations were not induced in mice. Glutaraldehyde did not induce cell transformation in Syrian hamster embryo cells in vitro (Zeiger et al. 2005). In vivo, inhalation of glutaraldehyde induced cell proliferation in nasal tissue in rats and mice, but did not induce DNA damage at these sites. Based on these observations, it is concluded that glutaraldehyde is not a genotoxin.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Studies on the incidence of miscarriage in pregnant women have shown no difference between those exposed to glutaraldehyde and those not exposed to the chemical. Studies in female rats and mice have resulted in embryotoxicity/foetotoxicity for glutaraldehyde, but only at doses which are maternally toxic. A number of studies have found no evidence of teratogenicity.

Acute Toxicity	Several acute oral toxicity studies with glutaraldehyde have been reported in rats and other species. In one reliable study, administration of 0.2, 0.3, 0.5, 1.0, 1.7 mL/kg bw glutaraldehyde (corresponding to 226, 339, 565, 1130 and 1921 mg/kg bw, respectively) to male/female Wistar rats by gavage gave a median lethal dose (LD50) of 226 mg/kg bw (REACH 2013). Necropsy of animals that died during the observation period revealed congestion of the lungs and the abdominal viscera. In another study in Sprague-Dawley rats, the oral LD50 was 316 mg/kg bw for males and 285 mg/kg bw for females, when 10 mL of 2.15, 3.16, 4.64, 14.7% glutaraldehyde (corresponding to 215, 316, 464 and 1470 mg/kg bw) was administered by oral gavage (REACH 2013). In a separate study using different strengths of glutaraldehyde, Ballantyne (1986) showed that the oral LD50 for glutaraldehyde in rats varied with the concentration of the glutaraldehyde used. By using different concentrations of glutaraldehyde solutions (1% to 50%) and varying the administration volume to maintain a constant dose, oral LD50 in the range 66 to 733 mg/kg bw were obtained. These studies indicate that glutaraldehyde has high acute oral toxicity. Of the 18 acute dermal toxicity studies reported in REACH (2013) dossiers, results from 14 studies indicated LD50 higher than 2000 mg/kg bw. In four other studies, LD50 ranged between 250 and 1432 mg/kg bw. These studies however did not follow international guidelines and have low reliability. Based on these studies, glutaraldehyde is considered to have low acute dermal toxicity. In a well-defined study, 10 male and 10 female Sprague-Dawley rats per dose group were exposed to glutaraldehyde as liquid aerosol at 0.22, 0.31 and 0.63 mg/L for 4 hours (REACH 2013). Exposure was followed by an observation period of 14 days. During the exposure period slight nasal discharge, snout wiping, flank respiration and irregular to intermittent respiration were reported in rats. During the post-exposure period, bloody nasal discharge, red crusts surrounding the nose, whooping or gasping respiration with rasping sounds and a tremulous gait were observed. These symptoms disappeared in the surviving animals within 5 to 9 days post-exposure. Mortalities were noted in all treated groups. The determination of the LC50 values was based on the Probit Analysis. An LC50 of 0.48 mg/L was calculated for both male and female rats. In another acute inhalation study conducted in a similar manner to the above study, Sprague-Dawley rats, 10 rats per sex per dose group, were exposed to 0.1, 0.18, 0.28, 0.39 and 0.44 mg/L glutaraldehyde as liquid aerosol for 4 hours (REACH 2013). During and after exposure, mortality and clinical signs of toxicity were recorded at regular time intervals. The LC50 in this study was established as 0.28 mg/L for females and 0.39 mg/L for males. Based on the above studies, glutaraldehyde is considered to have high acute inhalation toxicity.
Irritation	Glutaraldehyde is corrosive to the skin and eyes of rabbits at high concentrations, with signs of skin irritation evident at 2%, and eye irritation at 0.2%. Exposure to glutaraldehyde vapours in acute inhalational studies resulted in nasal irritation and respiratory difficulties. Joint irritation was seen in rabbits after intra-articular administration.
Sensitisation	The skin sensitisation effect of glutaraldehyde was demonstrated in tests with guinea pigs.
Health Effects Summary	Glutaraldehyde has high acute oral and inhalation toxicity and low to moderate acute dermal toxicity. Based on human and animal data, it is corrosive, the vapours are irritating to the respiratory tract, and it has skin and respiratory sensitisation potential. Glutaraldehyde has high repeat dose oral and inhalation toxicity, with an oral No-Observed-Adverse-Effect Level (NOAEL) of 4 mg/kg bw/day based on changes in liver and kidney weights and clinical chemistry parameters. Glutaraldehyde is not genotoxic or carcinogenic. It did not have any adverse effects on the reproductive system of adult rats or on the development of foetuses. The critical adverse health effects of glutaraldehyde are corrosivity, skin and respiratory tract sensitisation and acute and repeat dose oral and inhalation toxicity.
Key Study/Critical Effect for Screening Criteria	From the hazard characterisation, the critical (most sensitive) adverse health effects for repeated exposures to the chemical are changes in clinical chemistry parameters and relative organ (liver and kidney) weights. Glutaraldehyde has high repeat dose oral toxicity with an oral NOAEL of 4 mg/kg bw/day. This NOAEL is used in this human health risk assessment.
Ecological Toxicity [1,2,3,4]	

Aquatic Toxicity	96 h acute Bluegill sunfish LC50 = 11.2 mg/L 48 h acute Oyster larvae LC550 = 2.1 mg/L 96 h acute Green crabs LC50 = 465 mg/L 96 h acute Grass shrimp LC50 = 41 mg/L 48 acute Daphnia magna LC50 = 0.35 mg/L 48 acute Daphnia magna LC50 = 16.3 mg/L 21 d reproduct'n Daphnia magna LOEC = 4.3 mg/L, NOEC = 2.1 mg/L 96 h algal growth inhibition Selenastrum capricornutum ILm = 3.9 mg/L (median inhibitory limit) 96 h algal growth inhibition Scenedesmus subspicatus EC50 = 1.0 mg/L Bacterial inhibition Sewage microbes IC50 = 25-34 mg/L In summary, the test results indicate that glutaraldehyde is slightly to moderately toxic to aquatic fauna and moderately to highly toxic to algae. In some instances, glutaraldehyde appeared to be rapidly lost from test waters in the laboratory. Such behaviour in aquatic toxicity tests generally means that their results will underestimate the inherent toxicity of a substance. However, the toxicity that will prevail under environmental conditions is likely to be lower than that recorded in the laboratory in view of the rapid degradation that would be expected to occur in natural surface waters.
Determination of PNEC aquatic	As a wide selection of species is available, applying a safety factor of 10 to the NOEC (2.1 mg/L) derived from Daphnia seems most appropriate, giving a PNEC of 2100/10 = 0.21 mg/L for faunal species
Current Regulatory Controls [1,2,4]	
Australian Hazard Classification	Glutaraldehyde is classified as hazardous in the Hazardous Substances Information System (HSIS) with the following risk phrase (Safe Work Australia 2013): · T (Toxic); R23/25 (Toxic by inhalation and if swallowed) · C (Corrosive ; R34 (causes burns) · R42/43 (May cause sensitisation by inhalation and skin contact). Mixtures containing the chemical are classified as hazardous with the following risk phrases based on the concentration (Conc) of the chemical in the mixtures. The risk phrases for this chemical are: · Conc ≥50%: T; R23/25; R34; R42/43 (Toxic; toxic by inhalation and if swallowed; causes burns; may cause sensitisation by inhalation and skin contact) · ≥25% Conc <50%: T; R23; R22; R34; R42/43 (Toxic; toxic by inhalation, harmful if swallowed, causes burns; may cause sensitisation by inhalation and skin contact) · ≥10% Conc <25%: C; R20/22; R34; 42/43 (Corrosive; harmful by inhalation and if swallowed; causes burns; may cause sensitisation by inhalation and skin contact) · ≥2% Conc <10%: Xn; R20/22; R37/38; R41; R42/43 (Harmful; harmful by inhalation and if swallowed; irritating to respiratory system and skin; risk of serious eye damage; may cause sensitisation by inhalation and skin contact) · ≥1% Conc <2%: Xn; R36/37/38 R42/43 (Harmful; Irritating to eyes, respiratory system and skin; may cause sensitisation by inhalation and skin contact) · ≥0.5% Conc <1%: Xi; R36/37/38; R43 (Irritating; irritating to eyes, respiratory system and skin; may cause sensitisation by skin contact)
Australian Occupational Exposure Standards	The chemical has an exposure standard of 0.41 mg/m³, 0.1 ppm; Time Weighted Average (TWA).
International Occupational Exposure Standards	The following exposure standards are identified in Galleria Chemica (2013): · Occupational Exposure limit (TWA) of 0.2 mg/m3 [Canada, China, Denmark, Japan, Korea, UK] · 0.4 mg/m3 TWA [Sweden] · 0.8 mg/m3 TWA [US (NIOSH), Greece]
Australian Food Standards	No Australian food standards relating to the chemical have been identified (Food Standards Australia New Zealand 2013).
Australian Drinking Water Guidelines	No aesthetic or health-related guidance values were identified for this chemical in the Australian Drinking Water Guidelines. (National Health and Medical Research Council (NHMRC) 2011).

Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Readily biodegradable and as such not persistent in the environment.
B/vB criteria fulfilled?	No. As the Log Pow is -0.01 (Log Pow < 4.5), it is not expected to be bioaccumulative.
T criteria fulfilled?	No. Chronic toxicity data >1 mg/L in invertebrates, thus glutaraldehyde does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. NICNAS (1994) Priority Existing Chemical 3, Glutaraldehyde: Retrieved 2019: https://www.nicnas.gov.au
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS, 2017). National assessment of chemicals associated with coal seam gas extraction in Australia. Human health hazards of chemicals associated with coal seam gas extraction in Australia.
3. OECD (1995) SIDS Initial Assessment Profile on Glutaraldehyde
4. ECHA REACH, Glutaral, Retrieved 2019: https://echa.europa.eu/
5. Hazardous Chemical Information System (HCIS), Safe Work Australia. Retrieved 2019: http://hcis.safeworkaustralia.gov.au/
6. National Occupational Health and Safety Commission, Approved Criteria for Classifying Hazardous Substances [NOHSC:0006(1993)], AGPS, Canberra, 1993.

AECOM Imagine it. Delivered.

Toxicity Summary - Guar gum

Chemical and Physical Properties[1,2,7]	
CAS number	9000-30-0
Molecular formula	NA.
Product name	
Molecular weight	220,000 g/mol
Solubility in water	Completely soluble in water
pH	No data were found.
Melting point	No data were found.
Boiling point	No data were found.
Vapour pressure	solid
Henrys law constant	NA
Explosive potential	NA
Flammability potential	NA
Colour/Form	NA
Overview	Guar gum is a yellowish-white free-flowing powder. It is completely soluble in water and practically insoluble in oils, greases, hydrocarbons, ketones and esters. Water solutions are tasteless, odourless and a pale, translucent grey colour and neutral. The powder has 5 to 8 times the thickening power of starch. Water solution may be converted to a gel by adding a small amount of borox and are stable to heat. Guar gum is extensively used, eg typically used as a protective colloid, stabilizer, thickening and film forming agent for cheese, salad dressing, milk products including ice cream and soups; disintegration agent in tablet formulations; in pharmaceutical jelly formulations; in suspension, emulsions, lotions, creams and toothpastes; in bulk laxatives and appetite depressants; in mining industry as a flocculent, for hydraulic fracturing aid in oil well recovery and as a filtering ages; gelling and waterproofing agent in explosive and in water treatment as a coagulant. Guar gum is approved for use as a food additive by the U.S. Food and Drug Administration and is on the list of substances "generally recognized as safe" (CFR 1974). This chemical has been identified by NICNAS to be of low concern to human health based on an initial screening approach and thus required no further assessment.
Environmental Fate[1]	
Soil/Water/Air	No information was found. Guar gum, being a polysaccharide composed of galactomannan, would be expected to be readily biodegradable

Human Health Toxicity Summary [1,2,3,5,6,7,8,9]	
Chronic Repeated Dose Toxicity	F344 rats and B6C3F1 mice were given diets containing 0, 6,300, 12,500, 25,000, 50,000 or 100,000 ppm guar gum for 13 weeks (NTP, 1982). Mean body weights were decreased in male rats (100,000 ppm group) and in female mice (50,000 and 100,000 ppm). 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. F344 rats and B6C3F1 mice were given diets containing 0, 25,000 ppm or 50,000 ppm guar gum for 103 weeks (NTP, 1982). 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.
Carcinogenicity	F344 rats and B6C3F1 mice were given diets containing 0, 25,000 ppm or 50,000 ppm guar gum for 103 weeks (NTP, 1982). There were increased incidences of adenomas of the pituitary in male rats and pheochromocytomas of the adrenal 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 are combined, the statistical differences disappear. 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 highdose group. It was concluded that under conditions of this bioassay, guar gum was not carcinogenic for F344 rats or B6C3F1 mice.
Mutagenicity/ Genotoxicity	Guar gum induced no consistent responses in dominant lethal gene tests to suggest that it was mutagenic to the rat. Guar gum was not mutagenic to Salmonella typhimurium TA 1530 or G-46 when tested without metabolic activation; however, it was mutagenic to Saccharomyces cerevisiae D- 3 (Green, 1977). Guar gum also was reported to cause chromosomal aberrations in human embryonic lung cells WI-38 (Green, 1977). No in vivo genotoxicity studies have been conducted on guar gum.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	The developmental effects of guar gum were evaluated in groups of 20 rabbits by daily dermal administration of the test substance for 6 hours/day at dose levels of 0, 2, 10 and 50 mg/kg/day on days 6 through 18 of gestation. The number of early resorptions was significantly increased and the number of viable foetuses was correspondingly decreased at 50 mg/kg/day ($p<0.05$). The NOEL was 2 mg/kg/day. The frequency of foetal malformations and variations in the treated groups was comparable to that of the control group at all dose levels. Female rabbits were given daily (6 hours/day) dermal administration of 0, 2, 10 and 50 mg/kg guar gum during gestational days 6 through 18 (IRDC, 1988). Mortalities included 2 deaths at 50 mg/kg and 1 death at 10 mg/kg. A single animal was killed in extremis. A dose-related increase in dermal irritation (including erythema, edema, and desquamation) was observed in animals receiving 10 and 50 mg/kg. The number of early resorptions was significantly increased and the number of viable fetuses was correspondingly decreased at 50 mg/kg/day ($p<0.05$). The frequency of fetal malformations and variations in the treated groups was comparable to that of the control group at all dose levels. The NOEL for this study is 2 mg/kg/day.
Acute Toxicity	Guar gum has been blamed for causing esophageal obstruction. A death has the use of one guar gum tablet product, which apparently swelled in the esophagus, resulting in complications that caused the fatality. Mildly toxic by ingestion. The oral LD50 is 8,100 mg/kg for mice and 9,400 mg/kg for rats.
Irritation	No data were found.
Sensitisation	Occupational asthma has been reported in subjects of guar gum. A respiratory sensitizer There are reports of respiratory sensitization in workers exposed occupationally to guar gum dusts (Maio, 1986).

Key Study/Critical Effect for Screening Criteria	The key studies for the determination of a drinking water guidance value is the NTP two year chronic bioassays. The LOAELs are based on decreased mean body weights in female mice and rats fed 50,000 ppm guar gum in diet for 103 weeks. The NOAELs for these studies are 25,000 ppm guar gum. Rat: NOAEL (mg/kg/day) = 25,000 ppm * 0.05 = 1,250 mg/kg/day Mouse: NOAEL (mg/kg/day) = 25,000 ppm * 0.13 = 3,250 mg/kg/day Where 0.05 and 0.13 are the fraction of body weight that rats and mice, respectively, consume per day as food (U.S. EPA). The lowest NOAEL of 1,250 mg/kg/day for the rat will be used to derive a drinking water guidance value. Uncertainty factors: 10 (interspecies variability); 10 (intraspecies variability) Oral RfD = 1,250/100 = 12.5 mg/kg/day Drinking water guideline = 49 ppm
Ecological Toxicity [1,7]	
Aquatic Toxicity	The lowest measured ecotoxicity endpoint for fish was reported to be 218 mg/L.
Determination of PNEC aquatic	PNECaquatic: On the basis that the data consists of only one short-term result from one trophic level, an assessment factor of 1,000 has been applied to the reported effect concentration of 218 mg/L for Fish. The PNECaquatic is 0.218 mg/L.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
Australian Hazard Classification	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No biodegradation information was found on guar gum. However, guar gum is a naturally occurring polysaccharide which would be expected to readily biodegrade. Thus, it is not expected to meet the screening criteria for persistence
B/vB criteria fulfilled?	The molecular weight of guar gum ranges from 200,000 to 300,000 daltons, and it is also water soluble. Thus, guar gum is not expected to meet the criteria for bioaccumulation
T criteria fulfilled?	The acute aquatic toxicity of guar gum is >0.1 mg/L. Thus, guar gum is not expected to meet the screening criteria for toxicity
Overall conclusion	Not a PBT substance.

References

1. Hazardous Substances and New Organisms (HSNO) 2013, Chemical Classification and Information Database (CCID). Guar Gum. New Zealand Environmental Protection Authority, New Zealand Government.
2. Glicksman M. (1969). Gum technology in the Food Industry, pp. 590, Academic Press, New York; cited in Yoon, S.-J., Chu, D.-C., and Juneja, L.R. (2008) Chemical and physical properties, safety and application of partially hydrolyzed guar gum as dietary fiber. J. Clin. Biochem. Nutr. 42: 1-7.
3. Green, S. (1977). Present and future uses of mutagenicity tests for assessment of the safety of food additives. J. Environ. Pathol. Toxicol. 1: 49-54.

4. International Research and Development Corp (1988). Teratology Study of Guar Gum in Rabbits. TSCATS database, EPA Doc. No. 88-920004924, Fiche No. OTS0542101; cited in NZ HSNO CCID. http://www.epa.govt.nz/search-databases/Pages/cciddetails. aspx?SubstanceID=1930

5. Maio, J.L., Cartier, A., L'Archevêque, J., Ghezzo, H., Soucy, F., Somers, J., and Dolovich, J. (1990). Prevalence of occupational asthma and immunologic sensitization to guar gum among employees at a carpet-manufacturing plant. J. Allergy Clin. Immunol. 86: 562-569.

6. NTP (1982). NTP Technical Report on the Carcinogenesis Bioassay of Guar Gum (CAS No. 9000-30-0) in F344 Rats and B6C3F1 Mice (Feed Study), National Toxicology Program, Research Triangle Park, NC

7. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme

AECOM Imagine it. Delivered.

Toxicity Summary - Hydrochloric acid

Chemical and Physical Properties [1,2]	
CAS number	7647-01-0
Molecular formula	HCl
Molecular weight	36.46 g/mol
Solubility in water	Soluble
Melting point	-114.22 °C
Boiling point	-85.05°C
Vapour pressure	35,424 mm Hg at 25 deg C
Henrys law constant	2.04 x106 mol/L atm
Explosive potential	Reacts with most metals producing explosive hydrogen gas
Flammability potential	Not combustible
Colour/Form	liquid
Overview	CAS Registry number. Since the gas becomes the acid in aqueous systems and volatilization of the gas can occur from aqueous systems, it is often difficult to determine which is being considered in a specific item in the literature. If released to water, hydrogen chloride dissociates readily in water to chloride and hydronium ions, decreasing the pH of the water. The solution in water is a strong acid, it reacts violently with bases and is corrosive. Reacts violently with oxidants forming toxic gas (chlorine). Attacks many metals in the presence of water forming flammable/explosive gas (hydrogen). Hydrochloric acid is one of the most widely used industrial chemicals. Uses include pickling and cleaning metals, food process, and cleaning of industrial equipment.
Environmental Fate [3,4]	
Soil/Water/Air	Hydrochloric acid is readily dissociated in water into hydrated protons and chloride ions. The increase in the concentration of hydrochloric acid in water decreases the pH in the aquatic ecosystem. Generally, the buffer capacity to maintain the pH in the aquatic ecosystem is important and the equilibrium between CO2, HCO3 - and CO3 2- in the aquatic ecosystem is mainly responsible for the buffer capacity of receiving water.

Human Health Toxicity Summary [1,2,3,8]	
Chronic Repeated Dose Toxicity	Frequent contact with aqueous solutions of hydrochloric acid may lead to dermatitis. For repeated dose toxicity, local irritation effects were observed in the groups of 10 ppm and above in a 90-day inhalation study. Rats were fed diets containing 280 to 1,250 mmol/kg hydrochloric acid (10.2 to 45.6 mg/kg) for 7-12 weeks. There was increased water intake in all treated groups. All animals fed diet containing 937 mmol/kg and above for 9 weeks, and half of the animals fed diet containing 900 mmol/kg for 12 weeks died. Also at doses >937 mmol/kg, there was decreased body weight, food consumption, blood pH, femur length, rate of ash in bone (Upton and L'Estrange, 1977). In another study with rats, hydrochloric acid was administered via drinking water at pH 2-3 (study duration not provided). Decreased protein levels in urine and decreased urine volumes were observed in the treatment groups (Clausing and Gottschalk, 1989).
Carcinogenicity	HCl is not classifiable as a human carcinogen. No evidence of treatment related carcinogenicity was observed either in other animal studies performed by inhalation, oral or dermal administration. In three industry-based human case studies conducted in the U.S, no association between hydrogen chloride exposure and cancers of the lung, brain, or kidney was observed. In one U.S study of steel-pickling workers an excess risk for cancer of the lung was identified in workers exposed primarily to hydrochloric acid. Under IARC definitions, HCl is not classifiable as to its carcinogenicity to humans (Group 3).
Mutagenicity/ Genotoxicity	In single studies, HCl induced mutation and chromosomal aberrations in mammalian cells and induced chromosomal aberrations in insects and in plants. It did not induce mutation in bacteria. For genetic toxicity, a negative result has been shown in the Ames test. A positive result, which is considered to be an artefact due to the low pH, has been obtained in a chromosome aberration test using Hamster ovary cells. The effects of low pH in in vitro studies are not a problem in vivo as the proton level is regulated systemically. Hydrochloric acid is not considered to be genotoxic.
Reproductive Toxicity Developmental Toxicity/Teratogenicity	No reliable studies have been reported regarding toxicity to reproduction and development in animals after oral, dermal or inhalation exposure to hydrogen chloride/hydrochloric acid. As protons and chloride ions are normal constituents in the body fluid of animal species, low concentrations of hydrogen chloride gas/mist or solution do not seem to cause adverse effects to animals. The cells of gastric glands secrete hydrochloric acid into the cavity of the stomach. No reliable conclusion could be drawn on the potential reproductive toxicity of hydrogen chloride/hydrochloric acid.

Acute Toxicity	Rapid evaporation of the liquid may cause frostbite. The substance is corrosive to the eyes, the skin and the respiratory tract and can cause serious skin burns and blurred/reduced vision or blindness. Inhalation of high concentrations of the gas may cause pneumonitis and lung oedema, resulting in reactive airways dysfunction syndrome. The effects may be delayed. Exposure to hydrochloric acid can produce burns on the skin and mucous membranes, with severity related to the concentration of the solution. Subsequent ulceration may occur, followed by keloid and retractile scarring. Dental decay, including yellowing, softening and breaking of teeth, and related digestive diseases have been recorded after exposures to hydrochloric acid. Mortality has been observed following ingestion of hydrochloric acid. Female rats orally administered 3.3% hydrochloric acid yielded an acute oral median lethal dose (LD50) in a range from 238 to 277 mg/kg bw (Hoechst 1966). No details of the study were available. In another study in rats, administration of a solution of undisclosed concentration induced stomach ulceration, inflammation of the intestine, discolouration of the liver and hyperaemia of the lung (Monsanto 1976). An LD50 of 700 mg/kg bw was reported. An acute dermal LD50 was established as >5010 mg/kg bw in rabbits however the dose levels administered were not reported (Monsanto 1976). Acute median lethal concentration (LC50) values of 8.3 mg/L and 3.2 mg/L were observed in rats and mice respectively after a 30 minute inhalation exposure to aerosolised hydrochloric acid (Darmer et al. 1974).
Irritation	In a skin irritation test in rabbits performed according to OECD TG 404, 37% hydrochloric acid (0.5 mL) was applied by both semi-occlusion and occlusion (Potokar 1985). The chemical was found to be corrosive under both conditions after one hour exposure. Concentrations >17% also caused corrosion in rabbits. Concentrations >3.3% caused skin irritation to rabbits after application for 5 days. Hydrochloric acid caused mild to severe eye irritation in animal studies. There were no data available for respiratory irritation however; inhalation of hydrochloric acid vapours is expected to cause irritation. In humans, the chemical was determined to be ‘irritating to skin’ (York et al. 1996).
Sensitisation	May cause dermatitis with frequent contact of aqueous solutions of hydrochloric acid.
Health Effects Summary	Hydrochloric acid has demonstrated acute oral toxicity, corrosive effects to the skin and eye, and irritant effects to the respiratory system. Hydrochloric acid is not a skin sensitiser based on the available studies. Only limited information on the repeated oral toxicity of hydrochloric acid is available. However, as the component ions are normal constituents of the human body (particularly the stomach), only localised effects are expected. No systemic effects from repeated exposures are expected. The chemical is not genotoxic. No evidence of treatment-related carcinogenicity was observed in animal studies performed by inhalation or dermal administration. In humans, no association between hydrogen chloride exposure and tumour incidence was observed. No reliable studies were identified regarding specific toxicity to reproduction and development in animals after exposure to hydrochloric acid/hydrogen chloride. Because protons and chloride ions are normal constituents in the body fluids, low concentrations of hydrochloric acid/hydrogen chloride would not be expected to cause adverse reproductive effects to animals. This conclusion is supported by the 90-day inhalation study of hydrogen chloride where no effects on the gonads of rodents were observed.
Key Study/Critical Effect for Screening Criteria	The Australian drinking water guideline value for pH may apply to hydrochloric acid.

Ecological Toxicity [1,3,4,8]	
Aquatic Toxicity	The measured acute endpoint for: Algae = 0.492 mg/L Daphnia = 0.492 mg/L Fish = 4.92 mg/L The measured chronic endpoint for Daphnia is 62 mg/L
Determination of PNEC aquatic	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 Chronic endpoint of 62 mg/L for Daphnia. The PNECaquatic is 6.2 mg/L.
Current Regulatory Controls [8]	
Australian Hazard Classification	C (Corrosive); R34 (Causes burns) Xi (Irritant); R37 (Irritating to respiratory system).
Australian Occupational Exposure Standards	There are no specific exposure standards for hydrochloric acid. However, the permissible exposure limits for hydrogen chloride gas apply (Safe Work Australia 2013): Time Weighted Average (TWA) of 7.5 mg/m^3 (5 ppm).
International Occupational Exposure Standards	The following exposure standards were identified for hydrogen chloride (Galleria Chemical 2013). TWA: 7 to 8 mg/m³ (5 ppm) [Austria, Belgium, Denmark, EU, Hungary, Japan, Korea, Mexico, The Netherlands, New Zealand, Norway, Sweden, Turkey] 2 to 5 mg/m³ (1-2 ppm) [Germany, Poland, Switzerland, UK]. Short Term Exposure Limit (STEL): 15 mg/m^3 (10 ppm) [Austria, Belgium, EU, Hungary]
Australian Food Standards	Hydrochloric acid is an additive permitted in accordance with Good Manufacturing Practice (GMP) in processed foods specified in Schedule 1 of the Australia New Zealand Food Standards Code – Standard 1.3.1 – Food Additives (Food Standards Australia New Zealand 2013).
Australian Drinking Water Guidelines	Hydrochloric acid is listed as an endorsed drinking water treatment chemical in the Australian Drinking Water Guidelines (National Health and Medical Research Council (NHMRC) 2011).
Aquatic Toxicity Guidelines	No data found
PBT Assessment	
P/vP Criteria fulfilled?	Hydrochloric acid is an organic 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 most water, soil and sediment. Thus, the persistent criteria is not considered applicable to this inorganic salt.
B/vB criteria fulfilled?	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.
T criteria fulfilled?	No chronic toxicity data exist on hydrochloric acid; however, the acute EC(L)50s are >0.1 mg/L in fish, invertebrates and algae. Thus, hydrochloric acid does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	April 2018

References

1. U.S. National Library of Medicine, Toxicology Data Network HSDB (Hazardous Substances Data Bank) http://toxnet.nlm.nih.gov/
2. OECD SIDS. (1992), *UNEP Publications 5; Hydrochloric Acid (IARC Summary & Evaluation, Volume 54*). Obtained from IPCS INCHEM http://www.inchem.org/documents/iarc/vol54/03-hydrochloric-acid.html
3. IARC (International Agency for Research on Cancer). (2011), *Agents Classified by the IARC Monographs, Volumes 1 -102.*

4. IARC (International Agency for Research on Cancer). (1992), *Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Man* (Multi-volume work).
5. OECD (2002). IUCLID Data Set for Hydrogen chloride (CAS No. 7647-01-0), UNEP Publications.
6. OECD (2002). Screening Information Dataset (SIDS) Initial Assessment Report for Hydrogen chloride (CAS No. 7647-01-0), UNEP Publications.
7. Safe Work Australia Workplace Exposure Standards for Airborne Contaminants, 2013.
8. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme

AECOM Imagine it. Delivered.

Toxicity Summary - Distillates, Hydrotreated Light

Chemical and Physical Properties [1,2,3,4]	
CAS number	64742-47-8
Molecular formula	C48H94
Molecular weight	Not applicable - unknown or variable composition, complex reaction products or biological materials (UVCB)
Solubility in water	0.009 to 6.45 mg/L (at 25°C)
Melting point	-49 °C
Boiling point	146 to 299 °C
Vapour pressure	1 to 3.7 kPa at 37.8 °C
Henrys law constant	No data found.
Explosive potential	Above 66°C explosive vapour/air mixtures may be formed
Flammability potential	Combustible
Colour/Form	Liquid at room temperature
Overview	Distillates, hydrotreated light (also called deodorised kerosene) is a petroleum substance. The C_9-C_{14} Aliphatic [< 2% Aromatic] Hydrocarbon Solvents Category is comprised of complex aliphatic hydrocarbon solvents that contain >98% aliphatic constituents with carbon numbers in the range of C9-C14 and less than 2% aromatic constituents. The chemical is used as a component of a drilling fluid formulation for coal seam gas extraction.
Environmental Fate[1]	
Soil/Water/Air	Members of the C_9-C_{14} Aliphatic [≤2% aromatics] Hydrocarbon Solvents Category have the potential to volatilize from surface waters, based on Henry's Law constants (HLC) representing volatility for category members that range from 4.76×10^4 to 1.67×10^6 Pa-m^3/mole (at 25°C). In the air, category members have the potential to rapidly degrade through indirect photolytic processes mediated primarily by hydroxyl radicals (•OH) with calculated degradation half-lives ranging from 0.42 to 1.10 days or 10.8 to 26.4 hours based on a 12-hr day and an •OH concentration of 1.5×10^6 •OH/cm^3. These chemicals are unlikely to degrade by hydrolysis as they lack a functional group that is hydrolytically reactive.

Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	In a 90-day study conducted in accordance with OECD TG 408, Sprague-Dawley rats were administered deodorized kerosene by gavage at doses of 0, 100, 500 or 1000 mg/kg bw/day (REACH 2013). Microscopic changes, such as incidence of a2µ-globulin, were seen in male kidneys. These effects are not considered relevant to humans. No other treatment-related effects were observed. No Lowest Observed Adverse Effect Level (LOAEL) or No Observed Adverse Effect Level (NOAEL) could be established in this study. Repeated dermal exposures to members of the kerosene/jet fuel category showed minimal systemic effects (API 2010). Animal data on repeat dermal toxicity of kerosene (petroleum) are summarised from REACH (2013) and presented in Table A29.2. The LOAELs and NOAELs are indicated for each study. Prolonged skin exposure to kerosene (petroleum) in rats and rabbits were consistently associated with local irritation. In rabbits only, systemic effects included changes in bodyweight and organ weights. It is expected that deodorized kerosene would have similar effects in the animals. In a 13-week study, rats (strain not specified) were exposed to deodorized kerosene vapour at concentrations of 0, 0.02, 0.048 or 0.10 mg/L for six hours/day, five days/week. No treatment-related effects were reported (REACH 2013).
Carcinogenicity	A study for deodorized kerosene is available in the REACH Dossier (REACH 2013) but was not reported in enough detail to be able to determine the carcinogenicity of the substance. In a study conducted similarly to OECD TG 451, B6C3F1 mice were applied 0, 250 or 500 mg/kg bw/day kerosene (petroleum) in the interscapular region (type of wrapping not specified) for 103 weeks (REACH 2013). At the end of the study, less than 10% decrease in bodyweight gain was observed at the top dose in both sexes. Mortality in females was significantly higher at the two doses compared to controls. Increased incidence and severity of chronic dermatitis was seen in all treatment groups. At the top dose, increased incidence of the following non-neoplastic lesions was reported: amyloid in the liver, kidney, adrenal cortex (males only), spleen; granulocytic hyperplasia in the bone marrow; and hyperplasia of the axillary lymph nodes (females only). The only indication of neoplastic lesions was an increased incidence of malignant lymphomas observed in treated female animals but the values were within the range of historical controls. Under the conditions of the test, kerosene (petroleum) was not carcinogenic. The LOAEL for systemic effects is 250 mg/kg bw/day. The International Agency for Research on Cancer (IARC) concluded that there is inadequate evidence for the carcinogenicity of kerosene (petroleum) in experimental animals and humans, placing the chemical in Group 3 (Not classifiable as to its carcinogenicity to humans) (IARC 1989). Deodorized kerosene is not carcinogenic, based on reading across the information available for kerosene (petroleum).
Mutagenicity/ Genotoxicity	In vitro tests reported deodorized kerosene as negative both with and without metabolic activation in Ames tests conducted in accordance with OECD TG 471 (REACH 2013; OECD 2011) and in chromosomal aberration tests conducted in accordance with OECD TG 473 (OECD 2011, 2012). In an in vivo study, deodorized kerosene was negative in a dominant lethal assay, conducted in accordance with OECD TG 478, in male Swiss mice and Long Evans rats administered 10% deodorized kerosene intraperitoneally (REACH 2013). These studies demonstrate that deodorized kerosene is not genotoxic.

Reproductive Toxicity / Developmental Toxicity/Teratogenicity	C9-C14 aliphatic (≤2% aromatic) hydrocarbon solvents and C14-C20 aliphatic (≤2% aromatic) hydrocarbon solvents are not toxic to fertility (OECD 2011, 2012). Members of the kerosene/jet fuel category are not toxic to fertility (API 2010). Sprague-Dawley rats were administered undiluted kerosene (petroleum) by gavage at doses of 0, 750, 1500 or 3000 mg/kg bw/day in males treated for 70-90 days and 0, 325, 750 or 1500 mg/kg bw/day in females treated for 21 weeks. At 750 and 1500 mg/kg bw/day, increased absolute liver weight was observed in females but with no corresponding changes in clinical chemistry or histopathology. In females only, other effects included perianal dermatitis at 1500 mg/kg bw/day and stomach hyperplasia at 750 and 1500 mg/kg bw/day. These parameters were not measured in males. In males, the study indicated dose dependent decrease in male bodyweight that was linked to nephropathy specific to male rats. Data for this effect were not provided in the study description. There were no treatment related effects on fertility in both sexes (REACH 2013). The NOAEL for systemic effects in females only was 325 mg/kg bw/day. No NOAEL can be established for fertility effects. C9-C14 aliphatic (≤2% aromatic) hydrocarbon solvents and C14-C20 aliphatic (≤2% aromatic) hydrocarbon solvents are not developmental toxicants (OECD 2011, 2012). Members of the kerosene/jet fuel category are not developmental toxicants (API 2010). In a study conducted in accordance with OECD TG 414, Sprague-Dawley rats were administered kerosene (petroleum) by gavage on gestation days (GD) 6 to 15 at doses of 0, 500, 1000, 1500 or 2000 mg/kg bw/day (REACH 2013). Bodyweight gain was decreased at 1500 and 2000 mg/kg bw/day. Foetal weight was decreased at 1500 and 2000 mg/kg bw/day which may be attributed to decreased maternal bodyweight gain. No malformations were reported. The maternal NOAEL is 1000 mg/kg bw/day. In another study, Sprague-Dawley rats were exposed (whole body) to kerosene (petroleum) in air at concentrations of 0, 106 or 364 ppm on GD 6-15. There were no treatment-related effects observed in the dams and offspring (REACH 2013). Deodorized kerosene is not considered a developmental toxicant, based on reading across data available for kerosene (petroleum).
Acute Toxicity	The chemicals have low acute toxicity based on results from animal tests following oral exposure. The median lethal dose (LD50) in rats is >2000 mg/kg bw (OECD, 2011; US EPA, 2011; OECD, 2012a; OECD, 2012b; OECD, 2012c). The chemicals have low acute toxicity based on results from animal tests following dermal exposure. The LD50 in rats and rabbits is >2000 mg/kg bw (OECD, 2011; US EPA, 2011; OECD, 2012a; OECD, 2012b; OECD, 2012c). The chemicals have low acute toxicity based on results from animal tests following inhalation exposure.
Irritation	Semi-occlusive applications of commercial grade deodorized kerosene produced slight irritation in New Zealand White and SPF rabbits in dermal irritation studies conducted in accordance with OECD TG 404. The studies reported the range of erythema and oedema scores to be 0.3-0.9 and 0.2-1.0, respectively, based on Draize scoring at 24, 48 and 72 hours. Deodorized kerosene is slightly irritating to rabbit skin. Several studies conducted similarly to OECD TG 405 showed minimal effects to the eye with the reported range of conjunctival redness score to be 0-0.2 from instillation of undiluted deodorized kerosene in the eyes of New Zealand White and SPF rabbits (OECD 2011). Deodorized kerosene is slightly irritating to rabbit eye.
Sensitisation	The C9-C14 aliphatic (≤2% aromatics) Category members do not cause skin sensitization.

Health Effects Summary	Deodorised kerosene is an aspiration hazard since it has low viscosity and is composed of aliphatic and aromatic hydrocarbons up to 10%. Deodorised kerosene has low acute oral, dermal and inhalation toxicity, and is slightly irritating to the skin and eyes. The substance is not a skin sensitiser, based on reading across data available for kerosene (petroleum). No treatment-related effects were reported in repeated oral and inhalation exposures to deodorised kerosene. Prolonged dermal exposure to kerosene (petroleum) reported local irritation in rats and rabbits, and changes in bodyweight and organ weights in rabbits. It is expected that these effects would be similar for deodorised kerosene. Based on the absence of adverse effects observed in repeat dose toxicity studies, for the purposes of quantifying the health risk to the general worker and public, the highest dose tested in the study conducted in rats (1 000 mg/kg bw/day) is used in this risk assessment. The substance is not genotoxic. It is neither a carcinogen nor a reproductive toxicant, based on reading across data available for kerosene (petroleum).
Key Study/Critical Effect for Screening Criteria	The most appropriate No-Observed-Adverse-Effect Level (NOAEL) for risk assessment is 1 000 mg/kg bw/day based on maternal toxicity (decreased bodyweight gain) at the Lowest- Observed-Adverse-Effect Level (LOAEL) of 1 500 mg/kg bw/day from a developmental toxicity study on kerosene (petroleum).
Ecological Toxicity [2]	
Aquatic Toxicity	Lowest acute endpoint for Daphnia = 0.018 mg/L (modelled)
Determination of PNEC aquatic	Based on the lowest acute endpoint for Daphnia (0.018 mg/L), an assessment factor of 100 has been applied, resulting in a PNECaquatic of 1.80E-04 mg/L.
Current Regulatory Controls [2]	
Australian Hazard Classification	All of the chemicals are classified as hazardous, with the following risk phrase for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia): Xn; R65 (acute toxicity) Mixtures containing the substance are classified as hazardous with the following risk phrase based on the concentration (Conc) of the substance in the mixtures: Conc ≥10%: Xn; R65 (May cause lung damage if swallowed)
Australian Occupational Exposure Standards	No specific exposure standards are available.
International Occupational Exposure Standards	No specific exposure standards are available for this chemical.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	Oils and greases (including petrochemicals) for freshwater production: <300[6] µg/L (ANZECC 2000)
PBT Assessment	
P/vP Criteria fulfilled?	No. This chemical is expected to be biodegradable. The ready biodegradability of SHELLSOL NF a solvent naphtha (petroleum), heavy aromatics (consists predominantly of C9 aromatics 25%m/m; C10 aromatics 65%, and indanes 10%) was studied in mineral nutrient medium inoculated with activated sludge (mixed liquor suspended solids 100-101 mg/L, pH 6.9) and incubated for 28 days at 20°C. SHELLSOL NF is readily biodegrade after 28 days but not within the 10 day window.
B/vB criteria fulfilled?	Category members have a potential to bioaccumulate, based on calculated log BCF values for constituents that range from 2.78 to 4.06, and calculated BCF values of 598 to 11,430 L/kg wet-weight, based on the Arnot and Gobas model, that take into account biotransformation of the chemicals in fish tissue. This chemical also has a log Kow of 6.025.

T criteria fulfilled?	Yes. The lowest acute endpoint is <1 mg/L.
Overall conclusion	Not PBT
Revised	January 2019

Human Health Risk Assessment

Occupational Exposure

Table 2 presents the calculated internal doses for adult workers associated with drilling chemical exposure/hydraulic fracturing chemical exposure.

Table 2 Calculated Internal Doses for Adult Workers

Occupational Activity	E_{derm} (mg/kg bw/day)	E_{inh} (mg/kg bw/day)	E_{total} (mg/kg bw/day)
Transport and storage	Negligible*	Negligible*	Negligible*
Mixing/blending drilling of hydraulic fracturing chemicals	0.06	0.750	**0.810**
Injection of drilling chemicals	Negligible*	Negligible*	Negligible*
Cleaning and maintenance (hydraulic fracturing)	0.012	0.150	**0.162**
Combined exposure Mixing/blending and cleaning and maintenance			**0.972**
Transport and storage of drilling muds	Negligible*	Negligible*	Negligible*

Ederm - Internal dose from dermal exposure; Einh – Internal dose from inhalation exposure; Etotal – Total internal dose from all routes.

* In the absence of accidents/incidents, repeated occupational exposures during transport and storage, or during injection of mixed/blended chemicals, are negligible. Similarly, repeated occupational exposures to the chemical via the transport and storage of drilling muds are negligible (NICNAS 2017).

Human Health Risk Characterisation

Uncertainty Factors

Using the Margin of Exposure (MOE) approach, conservative default uncertainty factors for intra- and inter-species variability are assumed to be 10 each. A MOE of less than 100 is considered a concern (NICNAS 2017).

Acute Health Risks

Acute exposure to the chemical is unlikely to result in adverse health effects. In addition, given the low concentration in the drilling fluids, exposure to the chemical via these fluids is of low concern for workers.

Chronic long-term health risks

The critical (most sensitive) adverse health effect is maternal toxicity (decreased bodyweight gain). The NOAEL established for this effect is 1000 mg/kg bw/day from a reproductive toxicity study. There are no adverse effects observed from repeated exposures to the chemical at any dose tested, up to 1000 mg/kg bw/day. This highest no-effect dose is applicable for a general worker. Margins of Exposure (MOE) for adverse health effects from repeated occupational exposures are calculated by comparing the NOAEL with exposures estimated for different occupational activities and combined activities. **Table 3** presents Margin of Exposure calculated for Adult Workers associated with drilling

chemical exposure/hydraulic fracturing chemical exposure. Risk characterisation calculations are presented in **Attachment A**.

Table3 Margins of exposure calculated for adult workers

Adult worker exposure scenario	E_{total} (mg/kg bw/day)	NOAEL (mg/kg bw/day)	Critical effect	MOE (NOAEL / E_{total})	Chemical is of concern? (MOE < 100)
Occupational Activity					
Mixing/blending drilling of hydraulic fracturing chemicals	0.810	1000	Maternal toxicity in rats	1235	No
Cleaning and maintenance (hydraulic fracturing)	0.162			6173	
Combined exposure Mixing/blending and cleaning and maintenance	0.972			1029	

Based on uncertainty factors derived for this risk characterisation, the MOEs indicate that the chemical is of low concern for workers from repeated exposures during certain operations.

References

1. OECD (2012) SIDS Initial Assessment Profile on C_9-C_{14} Aliphatic [≤2% aromatic] Hydrocarbon Solvents Category. Available at: http://webnet.oecd.org/HPV/UI/SIDS_Details.aspx?id=476560b6-e2b7-4466-9c52-0b278c8b71a7
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS, 2017). National assessment of chemicals associated with coal seam gas extraction in Australia. Human health hazards of chemicals associated with coal seam gas extraction in Australia.
3. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier II Assessment for Kerosene, Retrieved 2019: https://www.nicnas.gov.au
4. ECHA REACH, Distillates (petroleum), hydrotreated light, Retrieved 2017: https://echa.europa.eu/information-on-chemicals/registered-substances
5. ICSC Distillates (petroleum), hydrotreated light, Retrieved 2017: http://www.inchem.org
6. ANZECC (2000) Australian and New Zealand Guidelines for Fresh and Marine Water Quality for protection for aquatic ecosystems

AECOM Imagine it. Delivered.

Toxicity Summary - Methanol

Chemical and Physical Properties[1,3,4]	
CAS number	67-56-1
Molecular formula	CH4O
Molecular weight	32.04
Solubility in water	1,000 g/L at 20 °C
Melting point	-98 °C
Boiling point	65 °C
Vapour pressure	16.927 kPa at 25 °C
Henrys law constant	0.461 Pa m³/mol
Explosive potential	Vapour/air mixtures are explosive
Flammability potential	Highly flammable
Colour/Form	Clear colourless liquid
Overview	Methanol occurs naturally in humans, animals and plants. The general population is exposed to methanol mainly through consumption of food and beverages and through use of consumer products such as paints, sealers and adhesives that contain methanol as a solvent.
Environmental Fate [1,3]	
Soil/Water/Air	Air is the main target compartment, based on a fugacity model calculation (Mackay Level III) with about 73 % of environmental methanol distributing to air and 16 % to water. Methanol is degraded in the atmosphere by photochemical, hydroxyl-radical dependent reactions. The estimated elimination half-life is calculated to be about 17-18 days with a rate constant of 0.93 x 10-2 cm3/molecule-sec. Methanol is completely miscible in water and has a low octanol/water partition coefficient. These properties are indicative of high mobility in soil.
Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	Considering the no observed adverse effect level (NOAEL) available from a 90-day rat study (500 mg/kg bw/day), the chemical is not considered to cause serious damage to health by repeated oral exposure. In a 20-day inhalation study in monkeys, 3.9 mg/L (3000 mL/m3) was identified as the LOAEL (continuous exposure) where neurotoxic lesions appeared to progress in monkeys (according to NEDO 1987). This exposure concentration correlated with methanol blood levels 80 mg/L and formate levels 30 mg/L. There was no evidence of adverse effects in rats exposed to methanol up to 6.6 mg/L, six hours/day for 28 days, except local nasal irritation and increased relative spleen weights, which were observed only at the middle dose and not considered treatment-related (Andrews et al. 1987). A NOAEL could not be established in this study. In the chronic exposure studies in rats and mice, slight treatment-related decreases in body and organ weights were reported at the highest dose. These are however not considered as 'adverse' effects. In monkeys, slight degeneration of the inside nucleus of the thalamus was observed at 0.13 and 1.3 mg/L after seven months or more (NEDO 1987). One monkey at 0.13 mg/L and two at 1.3 mg/L showed slight but clear changes in peroneal nerves indicating damage to peripheral nerves. Some signs of fibrosis at 1.3 mg/L, which were considered borderline. There were mild but significant effects on heart and kidney at 0.13 and 1.3 mg/L. Histologically, a significant increase of Sudan positive granules was noted in the 1.3 mg group without pathological manifestations (e.g. fibrosis). Although the authors considered the lowest dose (0.013 mg/L) as the LOAEL, it was observed that effects at this dose were very mild and reversible and therefore not considered to be adverse effects. Based on these observations, a NOAEL of 0.013 mg/L was established in this study.

Carcinogenicity	The chemical is not likely to be a carcinogen. In a chronic inhalation study, Fisher rats and B6C3F1 mice were exposed to 0.013, 0.13, and 1.3 mg/L methanol for 24 and 18 months, respectively (NEDO 1987). No differences in survival were noted in the treatment groups compared with the control group. There was no evidence of an increase in liver tumours in rats or in the spontaneous liver tumour rate in mice. In the rats, some tumours such as papillary lung adenomas (males only), adrenal phaeochromocytomas (females only) and metastatic (transition) tumours appeared at a somewhat higher incidence in high-dose group rats after week 79 and 104 without clear dose-response relationship. However these tumour incidences were not statistically significantly different from those in the control group. In the mice, there were no appreciable differences from the control in either numbers of animals with tumours or in degree of malignancy observed. Proliferative effects on the astroglia cells were observed in monkeys continuously exposed to 0.013, 0.13 and 1.3 mg/L methanol by the inhalation route (NEDO 1987). These effects however were of a transient nature and disappeared after a six-month recovery period. There were no signs of histological degeneration.
Mutagenicity/ Genotoxicity	Methanol has been examined in numerous in vitro and in vivo test systems, including bacterial, mammalian and fungal test systems. Most in vitro studies did not demonstrate mutagenic activity. A small number of studies gave ambiguous results. All other studies produced negative results consistently. The majority of in vivo assays were negative for mutagenicity and clastogenicity (OECD 2004). Methanol was therefore concluded to be not mutagenic.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No impairment of fertility or reproductive performance was reported in male and female rats exposed to the chemical, except at very high doses. Male mice had morphological anomalies in spermatozoa after repeated oral dosing at 1000 mg/kg bw/day (blood level > 500 to 1000 mg/L in mice) (OECD 2004). Rodent studies indicate that methanol has developmental toxicity effects. The rodent data on developmental toxicity are relevant for humans despite the known differences in methanol metabolism between the two species. However, rodents are considered adequate models for humans only at levels where formate does not accumulate (NTP 2003). Blood methanol levels associated with serious developmental effects in rodents were in the range associated with formate accumulation (1000 to 2000 mg methanol per litre of blood), which is likely to result in metabolic acidosis, and visual and clinical effects in humans (NTP 2003; OECD 2004). The limited data available in humans do not show an association between reproductive and developmental toxicity and methanol (NTP 2003). Following a review of the developmental toxicity studies, the NTP concluded that there is evidence to suggest that females with low folate levels may be more susceptible to the adverse developmental effects of methanol, but more information was necessary to clarify this issue (NTP 2003). Based on the data available, the chemical is not considered to have reproductive or developmental toxicity in humans.

Acute Toxicity	In rats, mice, rabbits and dogs, the LD50 values after single oral administration range from about 5600 to 14 400 mg/kg bw (EHC 1997). Adverse effects noted in these animals were ataxia, narcosis and coma after high methanol doses. The animals did not exhibit acidosis and ophthalmologic changes typically seen in humans at high lethal and sub-lethal doses In rhesus monkeys, no deaths were reported at doses of 1000 to 2000 mg/kg bw, while animals receiving 3000 to 8000 mg/kg bw died within two days (OECD 2004). Treated animals showed acidosis, and some exhibited semi-coma and ophthalmologic changes. Human data, however, indicate acute oral toxicity at comparatively lower doses of 300 to 1000 mg/kg bw (EHC 1997). The reported median lethal doses (LD50) for experimental animals are 7300 mg/kg bw (mouse), 5628 mg/kg bw (rat), 14 200 mg/kg bw (rabbit) and 7000 mg/kg bw (monkey). The lowest lethal dose (LDLo) for humans ranges from 143 to 428 mg/kg bw (ChemIDplus 2012). There are limited available dermal toxicity studies in animals. In one dermal exposure study all the rats survived after application of 35 000 mg/kg bw methanol to the skin under occlusive conditions, while deaths were reported at 45 000 mg/kg bw (Eulner and Gedicke 1955). In rabbits, a dermal LD50 of 17 000 mg/kg bw was reported although no details of the study were provided (Carnegie-Mellon 1981). Limited data in monkeys indicate that the chemical is toxic via the dermal route (McCord 1931). Humans have been found to be more susceptible to methanol as compared to monkeys. Therefore, acute dermal toxicity with methanol is expected in humans (OECD 2004). The lowest reported dermal LD50 is 17 000 mg/kg bw, which was recorded in rabbits. Median lethal concentrations (LC50) of 87.5 and 128.2 mg/L were reported in rats following six and four hour inhalation exposures to methanol, respectively (BASF 1980a, 1980b). Clinical signs of toxicity were secretions from eyes and nose, laboured breathing, staggering, apathy and narcosis. A similar LC50 value (79 mg/L) was reported for mice following 2.25 hours exposure (Von Burg 1994). In cats, LC50 values after six-hour exposures ranged from 26 to 48 mg/L. A shorter duration of 4.5 hours led to an LC50 of 85.4 mg/L (Von Burg 1994). Studies in Rhesus monkeys indicated lethal concentrations (percent mortality not reported) at 13 mg/L after 18 hour exposure and 52 mg/L after one to four hour exposure (OECD 2004).
Irritation	The chemical is not a skin irritant. The chemical is a slight eye irritant in rabbits. High concentration of methanol vapours may cause irritation of the respiratory tract. In a short-term exposure study (details not available), exposure of rats to an atmosphere saturated with methanol vapours produced severe irritation of mucous membranes and milky corneal opacity (BASF 1975). All animals died after eight hours (BASF 1975).
Sensitisation	The chemical is not a skin sensitiser.
Health Effects Summary	Methanol has low acute oral, dermal and inhalation toxicity in experimental animals but moderate to high acute oral and dermal toxicity in humans. A Lowest Lethal Dose (LDLo) of 143 - 428 mg/kg bw (humans) has been reported. It is not a skin or eye irritant but is expected to be a moderate respiratory irritant, based on its effect on the mucous membrane in rats exposed to methanol vapours and on the effects observed in repeat dose inhalation studies. Tests with guinea pigs indicated that methanol is not a skin sensitiser. The critical effects to human health are acute toxicity from inhalation, skin contact and swallowing, and possible irreversible effects from acute oral exposure. No deaths were reported in Rhesus monkeys dosed at 2 000 mg/kg bw, but treated animals showed acidosis, and some exhibited semi-coma and ophthalmic changes. Human data, however, indicate acute oral toxicity and ophthalmic changes at comparatively lower doses of 300 - 1 000 mg/kg bw. Information on repeated dose toxicity by the dermal route is not available. Methanol was not genotoxic or carcinogenic. Reproductive and developmental toxicity studies did not show any significant effects of relevance to humans.

Key Study/Critical Effect for Screening Criteria	A No-Observed-Adverse-Effect-Concentration (NOAEC) of 0.013 mg/L (13 mg/m3) is used for this risk assessment. This NOAEC is derived from a chronic inhalation study in monkeys, in which degenerative effects in the brain and slight damage to the optic and peripheral nerves were noted at 0.13 mg/L and above. Changes in peroneal nerves were also noted in higher dosed animals, indicating damage to peripheral nerves. An oral No Observed Adverse Effect Level (NOAEL) of 500 mg/kg bw/day was also established in rats in a 90-day oral study based on increased liver enzymes (enzymes not specified) and decreased absolute brain weights at the highest dose. This value is not used in this risk assessment because acute oral data indicate that humans are more sensitive to methanol toxicity than rodents.
Ecological Toxicity [2,3]	
Aquatic Toxicity	In several 96-hour studies in fish in which methanol concentrations were measured during the tests, LC50s ranged from 15,400 to 29,400 mg/L. In the chronic toxicity study to invertebrates, the NOEC was 32,000 mg/L.
Determination of PNEC aquatic	A PNECaqua = 3.20E+03 mg/L can be calculated based on the lowest chronic toxicity value for aquatic invertebrates (Daphnia) with the assessment factor of 10.
Current Regulatory Controls[4]	
Australian Hazard Classification	The chemical is classified as hazardous with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia): T; R23/24/25 (acute toxicity) T; R39/23/24/25 (irreversible effects from acute exposure) Mixtures containing the chemical are classified as hazardous based on the concentration (Conc) of the chemical in the mixtures. The risk phrases for this chemical are: Conc ≥20%: T; R23/24/25; (Toxic: Toxic by inhalation, in contact with skin and if swallowed); R39/23/24/25; (Toxic: danger of very serious irreversible effects through inhalation, in contact with skin and if swallowed) 10% ≤Conc <20%: T; R20/21/22; (Toxic: Harmful by inhalation, in contact with skin and if swallowed); R39/23/24/25; (Toxic: danger of very serious irreversible effects through inhalation, in contact with skin and if swallowed) 3% ≤Conc <10%: Xn; R20/21/22; (Harmful: Harmful by inhalation, in contact with skin and if swallowed); R68/20/21/22; (Harmful: possible risk of irreversible effects through inhalation, in contact with skin and if swallowed).
Australian Occupational Exposure Standards	The chemical has an exposure standard of 262 mg/m³ (200 ppm) Time Weighted Average (TWA) and 328 mg/m³ (250 ppm) Short-Term Exposure Limits (STEL) (Safe Work Australia).

International Occupational Exposure Standards	The following were identified (Galleria Chemica): 250-270 mg/m³ (200 ppm) TWA in USA, Canada, Denmark, United Kingdom, Germany, France, Estonia, Greece, Hungary, South Africa, Spain, Singapore, Taiwan, Sweden, Malta, Malaysia, Latvia, Japan, Indonesia, India, Iceland, Egypt, Ireland, Mexico, Philippines and Switzerland; 250-350 mg/m³ (250-328 ppm) STEL in USA, Canada, United Kingdom, Greece, South Africa, Singapore, Sweden, India, Egypt and Mexico; 50 mg/m³ TWA in Bulgaria; 100 mg/m³ TWA and 300 mg/m³ STEL in Poland; 133 mg/m³ TWA in Netherlands; 25 mg/m³ TWA and 50 mg/m³ STEL in China; 1300 mg/m³ (1000 ppm) STEL in France; and 1040 mg/m³ STEL in Hungary and Switzerland.
Australian Food Standards	No Australian food standards were identified (FSANZ 2013)
Australian Drinking Water Guidelines	No aesthetic or health-related guidance values were identified for methanol in the Australian Drinking Water Guidelines (National Health and Medical Research Council (NHMRC) 2011).
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Methanol is expected to be readily biodegradable.
B/vB criteria fulfilled?	No. The Log Kow for methanol is -0.82 to -0.64. Thus, methanol does not meet the screening criteria for bioaccumulation.
T criteria fulfilled?	No. The EC50s from the acute aquatic toxicity data on methanol are >1 mg/L, hence does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. NICNAS (2017) Human Health Tier II Assessment for Methanol
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS, 2017). National assessment of chemicals associated with coal seam gas extraction in Australia. Human health hazards of chemicals associated with coal seam gas extraction in Australia.
3. OECD (2008) SIDS Initial Assessment Profile on Methanol
4. ECHA REACH, Methanol, Retrieved 2017: https://echa.europa.eu/information-on-chemicals/registered-substances
5. IPCS Acetic Acid, Retrieved 2015: http://www.inchem.org

AECOM Imagine it. Delivered.

Toxicity Summary - Polyethylene glycol

Chemical and Physical Properties	
CAS number	25322-68-3
Molecular formula	(C2H4O)nH2O
Molecular weight	UVCB
Solubility in water	40 g/L @ 30 °C
Melting point	-10 °C at 101.3 kPa
Boiling point	870 °C at 101.3 kPa
Vapour pressure	0 Pa @ 25 °C
Henrys law constant	
Explosive potential	Non explosive
Flammability potential	Non flammable
Colour/Form	Odourless, viscous transparent organic liquid
Overview	Polyethylene glycols, also known as PEGs, are clear, colourless, thick liquids to waxy solids, depending on the molecular weight. The molecular weight of PEGs ranges from 200 to over 6000. Some may have a faint odour and bitter taste. PEGs mix easily with water. PEGs are important commercial chemicals. They are used to make other chemicals, paper coatings, solvents, plasticizers and used in many household products, cosmetics and pharmaceuticals. One formulation, PEG 3500, is used as a laxative. PEGs are also used as food and animal feed additives.
Environmental Fate[1]	
Soil/Water/Air	Koc value of PEG was estimated as 10 L/kg by means of MCI method. This indicates that PEG will have a negligible tendency of sorption to soil and sediment and therefore have rapid migration potential to groundwater. The estimated half-life of the substance indicates that the substance is rapidly hydrolysable.

Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	The substance PEG exhibits repeated dose toxicity by oral, dermal and inhalation route. A study was designed to investigate the subacute repeated dose toxicity effects of Polyethylene Glycols (PEG 400) in Wistar rats (male/female) by oral route, in an overall study period of 90 days. Dose group (5 animals per group) was fed a solution ofPEG400 equivalent to 0, 2000, 4000, 8000, 16000 or 24000 mg/kg/day in the diet. The control group received no polyethylene glycol. During the study period, body weight as a ratio to the amount of nutrient consumed, body weight, liver weight, kidney weight, micro pathology of liver and kidneys were examined. No effects upon male and female rats were observed when PEG 400 was present in the diet at a level up to 8000 mg/kg/day (8%concentration) for 90 days study period. But at 16000 mg/kg/day it showed effects on organ weight (liver and kidney heavier than that of control rats); and a decrease in weight gain was observed. Thus, from overall conclusion of the study the NOAEL (no observed adverse effect level) for repeated dose oral toxicity was considered to be 8000 mg/kg/day. And the LOAEL (low observed adverse effect level) for subacute repeated dose toxicity was considered to be 16000 mg/kg/day. Rats were exposed to airborne concentrations of 100 mg/m^3 and 1000 mg/m^3 of PEG-200 for periods up to 13 weeks. Toxicological, physiological, hematological, blood chemical, and pathological effects were evaluated during the course of the exposures. No significant lesions observed in this study occurred exclusively in exposed animals and the severity of lesions which were found was not dose-related. It is our impression that there were no PEG 200 induced lesions in rat tissue at the dosage level and exposure/post exposure periods evaluated in this study. Organ:body weight ratios in rats at all concentrations and for the 6- and 13-week exposure periods and the 30-day post exposure period showed no pattern of significance that could be related to PEG 200. The mice organ:body weights for the 6-week·exposure period are unavailable. No pattern of significance could be related to PEG 200 exposure for the 13-week or the 30-day post exposure periods. There were no consistently significant changes in rat blood chemistry at the end of the 6- or 13-week exposures or the 30-day post exposure period. It appears that PEG-200 produced no positive effects in the rodents at the Inn and 1000 mg/m3 PEG 200 concentrations over the 13 weeks of exposure used in this study. Thus it is concluded that the NOAEC value of PEG-200 in rats was observed at dose level of 1000 mg/m^3. The NOAEL value of PEG in rabbit was observed at dose level of 760 mg/kg bw/day. The supporting study indicates the TDLo (TDLo - Lowest published toxic dose) of PEG was observed at a dose concentration of 30 mL/kg (30000 mg/kg) in a 30 days study period where the dosage of PEG was intermittently given to rodent-rabbit by the dermal route(full study is not available). Considering the above results it is concluded that PEG is non-toxic by dermal route.
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	PEG was found to be non-genotoxic.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	The one generation reproductive toxicity NOAEL (no observed adverse effect level) of PEG in rat was observed at a dose concentration of 1698.09 mg/kg bw/day. On the basis of this NOAEL value it indicates that PEG does not exhibit toxic effects to rat below the above mention dose.
Acute Toxicity	Acute toxicity of PEG to mouse by the oral route indicates that the substance does not exhibits acute toxicity by the oral route. Similarly the acute values of inhalation also indicate that the substance does not exhibits acute toxicity by the inhalative route. Thus, it can be inferred that the target substance is non-toxic to any of the oral, dermal and inhalation route of exposure.
Irritation	The available studies indicate that the substance PEG is not classified as a skin and eye irritant according to CLP regulation within the dose levels mentioned in the study.
Sensitisation	In the human repeat insult patch test 216 subjects were enrolled and 200 subsequently completed the study. PEG 200 caused some degree of sensitization response in 1 of the 200 subjects. This subject was a 61 year old white woman.

Health Effects Summary	PEG is non acute toxic to oral, dermal and inhalation route, shows no irritation effect to skin and eye, is not genotoxic and is not developmental and reproductive toxic.
Key Study/Critical Effect for Screening Criteria	Oral: In chronic repeated dose toxicity study by polyethylene glycols (PEG) 400 showed no effect upon male and female dogs when present in the diet at a level of 500 mg/kg/day (2% concentration) for one year. Thus NOAELs (no observed adverse effect level) for repeated dose oral toxicity was considered to be 500 mg/kg/day. Inhalation: The NOAEC value of PEG-200 in rats was observed at dose level of 1000 mg/m^3. Dermal: The NOAEL value of PEG in rabbit was observed at dose level of 760 mg/kg bw/day.
Ecological Toxicity [1]	
Aquatic Toxicity	The toxicity values of fish, invertebrates and algae are LC50 = 100 mg/L, LC50 = 1000 mg/L and EC 50 = 15.91 mg/L, respectively.
Determination of PNEC aquatic	Acute LC50 values are reported for fish, aquatic invertebrates, and freshwater algae. Since there is valid acute toxicity data for three trophic levels, an assessment factor of 1000 is used (in accordance with EU guidance). Based on the EC50 for freshwater algae (the most sensitive species in short term tests), the aquatic PNEC is 15.91 µg/L.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment [1]	
P/vP Criteria fulfilled?	No. PEG is non persistent in nature and so is considered to have rapid biodegradation in the environment.
B/vB criteria fulfilled?	No. The calculated BCF of PEG is 3.2 dimensionless and below the threshold of 2000.
T criteria fulfilled?	No. Acute toxicity data >1 mg/L in fish, invertebrates and algae, thus PEG does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. ECHA REACH European Chemicals Agency Database: http://apps.echa.europa.eu

Toxicity Summary - Sodium bisulfite

Chemical and Physical Properties [1]	
CAS number	7631-90-5
Molecular formula	H2O3S.Na
Molecular weight	104.06
Solubility in water	724 g/L @ 20 °C
Melting point	No data available.
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	No data available.
Overview	Sulfites in aqueous solutions involve complex equilibria among the different species of sulfur oxidation state IV. The composition of their mixture in solutions depends on the pH and temperature. Sulfur dioxide may be produced from sulfites at low pH. At a pH closer to 7, the concentration ratio of bisulfite ($HSO3^-$) to sulfur dioxide (SO2) is very high (Gunnison and Jacobsen, 1987). Sulfites occur naturally in some foods and beverages as a result of fermentation (e.g. in beer and wine). A small percentage of the population (up to 1 %) is sensitive to sulfites (FDA, cited in Grotheer et al., 2005), as sulfur dioxide may be generated from sulfites in the stomach at low pH (Simon, 1986). The sensitivity to sulfur dioxide can cause a wide range of reactions in humans ranging from mild to severe dermatological, pulmonary, gastrointestinal, or cardiovascular symptoms (Grotheer et al., 2005).
Environmental Fate[1]	
Soil/Water/Air	The substance has a very low vapour pressure, and also does not sublime. Therefore, the substance will not be present as a gas and no radical reactions can be expected. According to its chemical properties, hydrolysis is not expected/probable. Photodegradation in water is not relevant because it dissociates rapidly into ions and decomposes in water, and it not susceptible to visible light. The substance is an inorganic compound which does not undergo biodegradation. The substance readiliy dissociates in aqueous solution, as with soil moisture. Bioaccumulation is not to be expected. a low log Kow underlines this statement. Due to the ionic salt-character and other physico-chemical properties (negligible vapour pressure, very high water solubility and decomposition in water), the Henry constant is near to zero. Because of its ionic nature, sodium hydrogensulfite as well as its dissociation products are not volatile from aqueous solutions. Relevant adsorption onto soils, sediments or suspended matter is not expected.

Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	Based on the data available for sodium metabisulfite, Sulfites are not considered to cause serious damage to health by repeated oral and inhalation exposure. In an 8-week study, SD rats (normal and sulfite oxidase enzyme—which oxidises sulfite to sulfate—deficient) were exposed to sodium metabisulfite (CAS No. 7681-57-4) or a mixture containing sodium metabisulfite and acetaldehyde hydroxysulfonate, in drinking water at doses of 0, 7, 70 or 175 mg/kg bw/day (as SO2). A no observed effect level (NOEL) for sodium metabisulfite was established as 70 mg/kg bw/day (as SO2) for all treated rats (normal and enzyme deficient), based on severe gastric lesions, significant body weight reduction and increased urine excretion with sulfites observed at the highest dose. The NOEL for the mixture was 7 mg/kg bw/day (as SO2) for enzyme-deficient rats, based on severe gastric and hepatic lesions at higher doses. At necropsy, lung oedema was observed in sodium metabisulfite treated, enzyme-deficient rats (Hui et al., 1989 cited in CIR, 2003). Groups of six rats (Sprague Dawley) were exposed to sodium sulfite (CAS No: 7757-83-7) aerosols with a particle size of approximately 1 µm at concentrations of 0.1, 1, 5 or 15 mg/m3 for three days. Mild pulmonary oedema at 5 mg/m3 and irritation of the tracheal epithelium at 15 mg/m^3 were observed (CIR, 2003). In a repeated dose study, eight dogs (beagle) were exposed to 1 mg/m3 of sodium metabisulfite (CAS No: 7681-57-4) aerosols with a mass median aerodynamic diameter (MMAD) of 0.63 µm for 290 days. Severe epithelial changes were observed with hyperplastic foci in the respiratory region of the nasal cavity. An increase in the nonciliated cell numbers in the membranous portion of the trachea of the animals was also observed. No other effects were reported (CIR, 2003).
Carcinogenicity	Based on a 104-week repeated dose toxicity study in rats, with up to 2 % sodium bisulfite in the diet, sodium bisulfite is not considered carcinogenic to rats (OECD, 2001).
Mutagenicity/ Genotoxicity	Based on the data available, Sulfites are not considered to be genotoxic. A mixture of sodium bisulfite (CAS No. 7631-90-5) and sodium sulfite (1:3) was tested at concentrations of 0.05–1 mmol/L in human peripheral lymphocytes. Positive results were obtained for chromosomal aberrations: micronucleus formation, and sister chromatid exchange (WHO, 1999). In an in vitro unscheduled DNA synthesis test with rat hepatocytes (OECD TG 486), and in an in vivo micronucleus test (OECD TG 474), sodium bisulfite (CAS No. 7631-90-5) did not show any evidence of mutagenicity (SCCNFP, 2003). Sodium bisulfite gave both positive and negative results in the mutagenicity testing. The positive results in Salmonella typhimurium strains containing his-G46 and his-D6610 mutations, and in some E.coli strains were suggested to be due to the presence of sulfurous acid under acidic conditions. At a neutral pH and lower concentrations, sodium bisulfite was not mutagenic to these strains. However, sodium bisulfite alone gave negative results in all in vivo studies with mammalian systems (rats and mice) (CIR, 2003).
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Based on the data available, Sulfites are not considered to cause reproductive or developmental toxicity. Pregnant rats (Wistar) were exposed by gavage to sodium bisulfite (CAS No. 7631-90-5) at 0, 1, 5, 24, or 110 mg/kg bw/day on days 6–15 of gestation. The NOAEL for maternal toxicity or embryo foetotoxicity was 110 mg/kg bw/day. A NOAEL of 123 mg/kg bw/day was established in a study with pregnant rabbits (Dutch belted) exposed to sodium metabisulfite (CAS No. 7681-57-4) at 0, 1.23, 5.71, 26.5 or 123 mg/kg bw/day on days 6–18 of gestation. In both these studies, there were no treatment related effects reported on nidation (nesting behaviour), maternal or foetal survival. The number of abnormalities in soft or skeletal tissues of the treated groups were similar to controls (OECD, 2001).

Acute Toxicity	Sodium bisulfite has an oral LD50 of 2000 mg/kg bw in rats (ChemIDplus). Based on the limited data available, sulfites are considered to be of low acute dermal toxicity. The LD50 for sodium metabisulfite in rats is >2000 mg/kg bw. Sulfites exhibit low acute toxicity in animal tests (US EPA, 2007). Based on the limited data available, no conclusion can be made on the acute inhalation toxicity of the chemicals in this group. A group of guinea pigs was exposed (whole body) for one hour to 0.204, 0.395 or 1.152 mg/m^3 of sodium sulfite (CAS No. 7757-83-7) aerosols with a mass median aerodynamic diameter (MMAD) of 0.36 µm. The chemical caused dose-related changes in the lung capacity parameters (bronchoconstriction) with a lowest observed adverse effect concentration (LOAEC) of 0.204 mg/m^3 (Chen et al., 1987 cited in CIR, 2003). Sodium bisulfite are classified as hazardous with the risk phrase 'Contact with acid liberates toxic gas' (Xi; R31) in the Hazardous Substances Information System (HSIS) (Safe Work Australia).
Irritation	No data are available on respiratory tract irritation from a single exposure. A 3-day repeated dose study indicated irritation of the tracheal epithelium in rats from exposure to sodium sulfite (CAS No. 7757-83-7) aerosols at 15 mg/m^3 (CIR, 2003). In acute dermal irritation studies (OECD TG 404) with sodium sulfite, sodium bisulfite and potassium sulfite, no skin irritation was observed in albino rabbits (SCCNFP, 2003). In acute eye irritation studies (OECD TG 405) with sodium sulfite and sodium bisulfite in rabbits, slight to severe effects in the cornea and the iris in most of the exposed animals persisted during the observation periods (eight and 15 days, respectively). Slight to moderate conjunctival effects (erythema and oedema) were also observed up to the end of the observation periods. Due to the persistency of eye effects, especially of increased corneal opacity, both chemicals were considered as severe eye irritants (SCCNFP, 2003).
Sensitisation	Based on the available data, Sulfites are not likely to be skin sensitisers.
Health Effects Summary	Severe eye irritation effects; acute oral toxicity; and the possibility of liberating toxic gas when the chemical is in contact with acids. Sensitivity to sulfites that causes allergic reactions in a small percentage of the population should also be considered.
Key Study/Critical Effect for Screening Criteria	The main critical effects to human health are severe eye irritation and acute oral toxicity. The chemicals in this group will liberate toxic gas when in contact with acid and therefore may cause effects in individuals with a high acid content in the stomach. A small percentage of the population (up to 1 %) are sensitive to sulfites (FDA, cited in Grotheer et al., 2005). Those who have asthma are most at risk to sulfite sensitivity and other forms of sulfite reactions. This sensitivity can cause a wide range of allergic reactions ranging from mild to severe.
Ecological Toxicity [2]	
Aquatic Toxicity	Acute and chronic toxicity data were available for the three main aquatic trophic levels that are considered for classification purposes. Classification is based on the lowest acute and chronic value, referred to as the acute and chronic toxicity reference value (TRV). The lowest acute effect concentration was observed for the alga S. subspicatus (72h-EC50), and was 36.8 mg sodium sulfite/L. Translating this value to HNaSO3 results in an acute TRV of 47.9 mg/L for this substance. For sulfite/disulfite compounds, the lowest chronic value was a NOEC of >8.41 mg sodium sulfite/L for the invertebrate D. magna. Translating this value to HNaSO3 results in a chronic TRV of 10.9 mg/L for this substance, i.e., > 1 mg/L.

Determination of PNEC aquatic	The lowest value for chronic toxicity was and unbounded NOEC of 8.41 mg sodium sulfite/L. Applying the AF of 10 results in a PNECaquatic of 0.84 mg sodium sulfite/L.Translating this value to HNaSO3 gives a PNECaquatic of 1.09 mg test substance/L. As the lowest NOEC-value is an unbounded value (i.e., no effect was noted at the highest test concentration), this value can be considered as a worst-case estimate. Further refinement of the NOEC-value for daphnids could increase the PNECaquatic up to a maximum value of 2.8 mg sodium sulfite/L (i.e., an assessment factor of 10 on the algal 72h-EC10 value), which is equivalent to 3.64 mg test substance/L.
Current Regulatory Controls [1]	
Australian Hazard Classification	Sodium bisulfite is classified as hazardous with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia): Sodium bisulfite (CAS No. 7631-90-5): Xn; R22 (acute toxicity) Xi; R31 (contact with acid liberates toxic gas)
Australian Occupational Exposure Standards	Sodium bisulfite has an exposure standard of 5 mg/m^3 time weighted average (TWA). The exposure standard for sulfur dioxide of 5.2 mg/m^3 (2 ppm) (TWA) is also relevant to uses of these chemicals that may generate sulfur dioxide.
International Occupational Exposure Standards	An exposure limit (OEL, TWA, STEL, PEL or STV) of 5–10 mg/m^3 in different countries such as USA, United Kingdom, Canada, Ireland, Spain, Norway and Switzerland.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment [2]	
P/vP Criteria fulfilled?	Not applicable (inorganic substance)
B/vB criteria fulfilled?	Not applicable (inorganic substance)
T criteria fulfilled?	Not applicable (inorganic substance)
Overall conclusion	Not PBT
Revised	January 2019

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier II Assessment for Ethoxylates of aliphatic alcohols (>C6): Retrieved 2019: https://www.nicnas.gov.au
2. ECHA REACH, Sodium hydrogensulfite, Retrieved 2019: https://echa.europa.eu/

AECOM Imagine it. Delivered.

Toxicity Summary - Sodium chloride

Chemical and Physical Properties [1,4]	
CAS number	7647-14-5
Molecular formula	NaCl
Molecular weight	58.44 g/mol
Solubility in water	3.57 x 10 5 g/m3 at 25oC
pH	In aqueous solution is neutral
Melting point	1 mm Hg at 865oC
Boiling point	1670 ^{o}C
Vapour pressure	No data found
Henrys law constant	No data found
Explosive potential	Not explosive
Flammability potential	Not flammable
Colour/Form	light brown liquid or colourless crystals
Overview	Sodium, together with potassium is an essential mineral for the regulation of body fluid balance. Sodium is the most abundant cation in the extracellular fluid and sodium salts account for more than 90% of the osmotically active solute in the plasma and interstitial fluid. Consequently, sodium load is the major determinant of extracellular volume. Chloride is also important in maintaining the fluid balance and is an essential component of the gastric and intestinal secretions Sodium chloride occurs naturally as rock salt which comprises 95% to 99% NaCl. It is also widely used in food products. The NHMRC has established dietary guidelines for the intake of sodium per day (adults should consume less than 2300 mg sodium per day). This chemical has been identified by NICNAS to be of low concern to human health based on an initial screening approach and thus required no further assessment.
Environmental Fate [2,3]	
Soil/Water/Air	Due to its high solubility, sodium chloride is highly mobile in the environment. Once dissociated, chloride ions will migrate readily, however sodium ions will sorb to clay-rich materials limiting mobility. If released into the environment, sodium chloride is not likely to sorb to solid particles in the water column, is readily dissociated to form chloride and sodium ions, is not bioaccumulative in aquatic species or the food chain.

Human Health Toxicity Summary [2,3]	
Chronic Repeated Dose Toxicity	High sodium chloride intakes increase calcium excretion and may increase the risk of kidney stone formation. There is evidence for a causal relationship between the consumption of sodium (mainly from common salt) and both blood pressure and the age-related rise in blood pressure. Data suggest that30% of a normotensive population may be salt sensitive. Sodium chloride has been demonstrated to be a gastric tumour promoter in experimental animals and high sodium chloride intakes have been associated with incidence of stomach cancer in human populations with traditional diets of highly concentrated, salted foods.
Carcinogenicity	Not listed with IARC.
Mutagenicity/ Genotoxicity	No data available.
Reproductive Toxicity Developmental Toxicity/Teratogenicity	No data available.
Acute Toxicity	No data available.
Irritation	Although rare, acute toxicity may be caused by ingestion of 500 – 1000 mg sodium chloride/kg body weight. Symptoms include vomiting, ulceration of the gastrointestinal tract, muscle weakness and renal damage, leading to dehydration, metabolic acidosis and severe peripheral and central neural effects.
Sensitisation	No data available.
Health Effects Summary	Sodium is an essential mineral for the regulation of body fluid balance. This chemical has been identified by NICNAS to be of low concern to human health.
Key Study/Critical Effect for Screening Criteria	The Australian drinking water guideline value for sodium and chloride may apply.
Ecological Toxicity [2,3,4]	
Aquatic Toxicity	A large number of studies are available in relation to the aquatic toxicity of sodium chloride with the USEPA ECOTOX database identifying 1712 records. The evaluation of ecological effects of sodium chloride has been evaluated in detail for the assessment of the use of rock salt in the US on roadways during the winter months. The following has been summarised from the US review: The presence of sodium chloride may result in the increased mobilisation of other contaminants (metals, nutrients etc) and a shift in the acid buffering capacity may compromise aquatic ecosystems. Most sensitive species are birds where a safe concentration of 1000 mg/L sodium chloride can be established. Salt tolerance of aquatic species varies significantly with EC50 concentrations ranging from 400 to 30000 mg/L. The measured acute endpoint for Fish was reported at 1290 mg/L. The measured NOEC for Daphnia is 314 mg/L.
Determination of PNEC aquatic	PNECaquatic: On the basis of the chronic results for Daphnia, an assessment factor of 100 has been applied to the lowest reported effect concentration of 314 mg/L. The PNECaquatic is determined to be 3.14 mg/L.
Current Regulatory Controls	
Australian Hazard Classification	No data available
Australian Occupational Exposure Standards	No data available
International Occupational Exposure Standards	No data available
Australian Food Standards	No data available

Australian Drinking Water Guidelines	No data available
Aquatic Toxicity Guidelines	No data available
PBT Assessment [4]	
P/vP Criteria fulfilled?	Sodium chloride is an organic salt that dissociates completely to sodium and chloride ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions; both sodium and chloride ions are also ubiquitous and are present in most water, soil and sediment. The persistent criteria is not considered applicable to this inorganic salt.
B/vB criteria fulfilled?	Sodium and chloride ions are essential to all living organisms and their intracellular and extracellular concentrations are actively regulated. Thus, sodium chloride is not expected to bioaccumulate.
T criteria fulfilled?	The measured chronic toxicity data for sodium chloride was 314 mg/L for Daphnia Thus, sodium chloride does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	April 2018

References

1. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2015, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
2. UK 2003. Expert Group on Vitamins and Minerals, Risk Assessment - Sodium Chloride
3. US, 2007. Hazard Identification for Human and Ecological Effects of Sodium Chloride Rock Salt. Prepared by the New Hampshire Department of Environmental Services
4. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme

AECOM Imagine it. Delivered.

Toxicity Summary - Sodium hydroxide

Chemical and Physical Properties	
CAS number	1310-73-2
Molecular formula	Na-O-H
Product name	40 g/mol
Molecular weight	1.11E+06 mg/L at 20C
Solubility in water	13
Melting point	318 °C
Boiling point	1388 °C
Vapour pressure	Negligible at 25 deg C
Henrys law constant	No data found.
Explosive potential	No
Flammability potential	No
Colour/Form	Anhydrous (pure) NaOH is a solid – *refer melting point above*. However it is a hygoscopic, ionic solid, and will absorb water from air and is highly soluble
Incompatibility	Avoid contact of solid NaOH with water due to strong exothermic reaction, leather, wood, acids, organic halogen compounds or organic nitro compounds. Carbon monoxide gas can form upon contact with reducing sugars, food and beverage products in enclosed spaces. NAoH is neither explosive, flammable, nor oxidising.
Overview	Vegetable oil refining, regenerating iron exchange resins, organic fusions, peeling of fruits and vegetables in the food industry, etching and electroplating.
Environmental Fate[1]	
Soil/Water/Air	Sodium hydroxide is highly soluble, not volatile and unlikely to materially adsorb to soil and is therefore predominately found in the aquatic environment if released to the environment. NaOH will readily dissociate to be present in the environment as sodium and hydroxyl ions, both being ubiquitous in the environment. NaOH is a strong alkali, so it's dissolution in water may locally raise the pH of the affected environment. The dissolution reaction is also strongly exothermic.

Human Health Toxicity Summary [1,2,,3]	
Chronic Repeated Dose Toxicity	No animal data are available on repeated dose toxicity studies by oral or dermal routes for sodium hydroxide. In a repeat dose inhalation study, twenty seven white rats died within a month, mostly from bronchopneumonia, after being exposed twice weekly to an aerosol of unknown airborne concentration of sodium hydroxide, generated from an aqueous 40% sodium hydroxide solution (NIOSH 1975). When exposed to an aerosol generated from a 20% sodium hydroxide solution, the bronchi were dilated, the epithelial cover was thin and frequently desquamated, and the septa were dilated and cracked. A light round cell infiltration of the sub-mucus membrane tissue was also observed. Few changes occurred in a group of rats exposed to aerosols from 10% sodium hydroxide, but rats exposed to an aerosol of 5% sodium hydroxide had dilation of the bronchi and a slight degeneration of the mucus membrane and thickened strata of lymphadenoid tissue surrounding the bronchi. A NOAEL could not be established in this study. Workers exposed to 0.24 to 1.86 mg/m^3 sodium hydroxide for 2 to 15 minutes reported throat irritation and watery eyes (NIOSH 1975). Based on the observations of the irritant effects on workers exposed to 1 to 40 mg/m^3 sodium hydroxide, it was concluded that 2 mg/m^3 represented a concentration that is 'noticeably but not extensively irritant' (NIOSH 1975). Obstructive airway disease has been reported following chronic occupational exposure to sodium hydroxide mist (IPCS 1996). The patient developed cough, dyspnoea and tachypnoea after a 20-year exposure to sodium hydroxide.
Carcinogenicity	IARC Category 3 - not classifiable as to human carcinogenicity
Mutagenicity/ Genotoxicity	In vitro and vivo genetic toxicity testing reported no evidence of mutagenic activity.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No valid studies were identified regarding reproduction toxicity after oral, dermal or inhalation exposure to NaOH. Sodium hydroxide is not expected to be systemically available to the body under normal handling and use conditions.
Acute Toxicity	Exposure to the solid or concentrated liquid can cause severe burns to the eyes, skin and gastrointestinal tract which may cause death. An oral LD50 of a 1-10% solution of NaOH in rabbits was 325 mg/kg bw (as 100% NaOH). An oral LD50 of 140 to 340 mg/kg in rats has also been reported (National Research Council 2011), however details of the study are not available. In an acute dermal study, mice were treated dermally with 50% sodium hydroxide, and the treated area was irrigated with water at various intervals (OECD 2002). The mortality of mice was 20, 40, 80 and 71% when they were irrigated at 30 minutes, one hour, two hours or not at all after the application. All animals developed rapidly progressive burns. No mortality or burns were observed when the treated area was irrigated immediately after the application. A 5% aqueous solution of sodium hydroxide produced severe necrosis when applied to the skin of rabbits for four hours (Clayton and Clayton 1993). A dermal LD50 of 1350 mg/kg has been reported in rabbits (National Research Council 2011), however details of the study are not available. Caustic dusts are irritating to the upper respiratory system. Prolonged exposure to high concentrations may cause discomfort and ulceration of nasal passages. Cases of fatality due to ingestion of liquid sodium hydroxide have been reported in humans.
Irritation	Sodium hydroxide is a corrosive irritant to skin, eyes and mucous membranes. A NaOH solution of 8% can be considered corrosive based on animal data. Human data indicate that concentrations of 0.5 to 4% were irritating.
Sensitisation	Sodium hydroxide has no skin sensitisation potential.

Health Effects Summary	An oral LD50 of 325 mg/kg in rats and a dermal LD50 of 1350 mg/kg in rabbits were reported for sodium hydroxide. Lethality has been reported in animals at oral doses of 240 mg/kg bw. Inhalational LC50 is not available. Sodium hydroxide is corrosive to skin, eyes and gastrointestinal and respiratory tracts. Based on human data, concentrations of 0.5 to 4.0% are irritating to the skin, while a concentration of 8.0% is corrosive. Sodium hydroxide is not a skin sensitiser. No animal data were available on repeated dose toxicity by oral or dermal routes for sodium hydroxide. In the single reported repeat dose inhalation study, a NOAEL could not be established. Both in vitro and in vivo genetic toxicity tests indicated no evidence of a mutagenic activity. Information is not available on reproductive and developmental toxicity and carcinogenicity of sodium hydroxide. Due to dissociation into ions which are subject to homeostatic controls in the human body, systemic effects from repeated exposures to sodium hydroxide are not expected. The critical health effect of sodium hydroxide is its corrosive effect.
Key Study/Critical Effect for Screening Criteria	No oral TRV apply. Acute toxicity only (irritant and corrosive), not systemically available in body. The Australian drinking water guideline value for pH may apply to sodium hydroxide.
Ecological Toxicity [1,2,3]	
Aquatic Toxicity	Measured acute endpoints were available for fish (196 mg/L). Measured chronic endpoint were available for Daphnia (240 mg/L)
Determination of PNEC aquatic	An assessment factor of 10 has been applied to the lowest reported NOEC of 240 mg/L for Daphnia. The PNECaquatic is 24 mg/L.
Current Regulatory Controls[4]	
Australian Hazard Classification	C: R35 (Corrosive, causes severe burns)
Australian Occupational Exposure Standards	Sodium hydroxide has an exposure standard of 2 mg/m³, Time Weighted Average (Safe Work Australia 2013).
International Occupational Exposure Standards	Occupational Exposure Limit (OEL) or limit values in working environment of 2 mg/m³ [Argentina, Belgium, Bulgaria, Canada, China, India, Japan and the US (NIOSH 1975)]. Occupational exposure standard: 2 mg/m³ [Korea] Occupational exposure limit values: 0.5 mg/m³ [Latvia] Short Term Exposure Limit (STEL): 2 mg/m³ [UK] US Department of Energy Temporary Emergency Exposure Limits (TEELs) = 0.5 mg/m³ (TEEL-0 and TEEL-1), 5 mg/m³ (TEEL-2) and 50 mg/m³ (TEEL-3).
Australian Food Standards	Processing aids - Generally permitted - permitted for use as acidity regulator (FSANZ 2013). Sodium hydroxide is allotted an International Numbering System (INS) of food additives number: INS 524 (Food Standards Australia New Zealand 2013).
Australian Drinking Water Guidelines	No data found. However, since sodium hydroxide readily dissociates in water into sodium and hydroxyl ions, the Australian Drinking Water Guidelines for sodium state that, based on aesthetic considerations (taste), the concentration of sodium in drinking water should not exceed 180 mg/L (National Health and Medical Research Council (NHMRC) 2011). No health-based guideline value is proposed for sodium.
Aquatic Toxicity Guidelines	No data found.
Occupational Exposure Limits	Peak limitation – 2 mg/m^3
PBT Assessment	
P/vP Criteria fulfilled?	Not applicable (inorganic salt, ionic species ubiquitous in environment)
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to these inorganic ions; sodium and hydroxide ions are ubiquitous and are present in most water, soil and sediment.

T criteria fulfilled?	Not applicable. Chronic toxicity data >1 mg/L in invertebrates, thus sodium hydroxide does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT

References

1. OECD SIDS Sodium Hydroxide, UNEP Publications, March 2002
2. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved March 2015, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
3. European Commission (EC) Joint Research Centre (JRC) Institute for Health and Consumer Protection - European Chemical Substances Information System (ESIS), Sodium Hydroxide, Summary Risk Assessment Report, 2008
4. Safe Work Australia, Hazardous Substances System, sodium hydroxide

AECOM Imagine it. Delivered.

Toxicity Summary - Sodium iodide

Chemical and Physical Properties [1,2,3]	
CAS number	7681-82-5
Molecular formula	INa
Molecular weight	149.92
Solubility in water	165 – 1,800 g/L @ 25 °C
Melting point	651 - 659 °C at 101.3 kPa
Boiling point	1,304 °C at 101.3 kPa
Vapour pressure	-1.301 @ 25 °C
Henrys law constant	0.015 Pa.m³.mol-1 @ 25 °C
Explosive potential	Non explosive
Flammability potential	Non flammable
Colour/Form	Solid, colourless cubic crystals, odourless
Overview	Iodides are used by the thyroid gland in hormone production. Iodides have been utilized to treat iodine disorders, hyperthyroidism, bacterial, fungal or protozoal infections and also were traditionally as expectorants because of their stimulatory effects on bronchial secretions. A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health.
Environmental Fate [2]	
Soil/Water/Air	Sodium iodide is very stable under ordinary conditions of use and storage. The phototransformation in air is irrelevant to sodium iodide, because few sodium iodide can be distributed in air for the low vapour pressure and high water solubility. Hydrolysis is not a concern to such inorganic substance which can be completely ionized in water phase. sodium iodide will completely dissociate in water giving sodium ion and iodide anion. The sodium iodide is readily absorbed by organisms as Na+ and I-, which are both small (an)ions and well known to not likely to be bioaccumulative. Based on the intrinsic prosperities of sodium iodide, the substance can be expected to have a low potential for adsorption (completely ionized to small ions in water phase). The sodium ion and iodide anion are uniformly distributed in water phase. In the air, these two basic (an)ions is negligible, due to high water solubility and low vapour pressure. To sediment and soil phases, these two (an)ions are mostly distributed in the pore water.
Human Health Toxicity Summary [1]	

Chronic Repeated Dose Toxicity	The most likely route for human exposure is via digestion, so the dermal and inhalation route are irrelevant in the repeated toxicity assessment. Boyages et al. (1989) compared thyroid status in groups of children 7–15 years of age who resided in two areas of China where drinking-water iodide concentrations were either 462.5 µg/l (n = 120) or 54 µg/l (n =51). Urinary iodine concentrations were 1236 µg/g creatinine in the high-iodine group and 428 µg/g creatinine in the low-iodine group. Although the subjects were all euthyroid, with normal values for serum thyroid hormones and TSH concentrations, TSH concentrations were significantly higher ($P < 0.05$) in the high-iodine group. The high-iodine group had a 65% prevalence of goitre and a 15% prevalence of Grade 2 goitre compared with 15% for goitre and 0% for Grade 2 goitre in the low-iodine group. To transform the measured urinary iodine levels into estimates of iodine intakes, steady state baseline dietary intakes of iodide were assumed to be equivalent to the reported 24 h urinary iodine excretion rates. Assuming a body weight of 40 kg and lean body mass of 85% of body weight, the urinary iodine/creatinine ratios reported by Boyages et al. (1989) can be converted to approximate equivalent intake rates of 1150 µg/day (0.029 mg/kg body weight per day) and 400 µg/day (0.01 mg/kg body weight per day) for the high- and low-iodine groups, respectively. Thus, the NOAEL for this study is considered to be 0.01 mg/kg body weight per day. From the Boyages et al. (1989) study, supported by the studies of Gardner et al. (1988), Paul et al. (1988), and others, a TDI of 0.01 mg/kg body weight, based upon reversible subclinical hypothyroidism, can be established by dividing the NOAEL of 0.01 mg/kg body weight per day by an uncertainty factor of 1.
Carcinogenicity	A chronic toxicity and carcinogenicity study, in which male and female F344/DuCrj rats were administrated iodide (KI) in the drinking water at concentrations of 0, 10, 100 or 1000 ppm for 104 weeks was conducted. In the test, neither focal hyperplasias, adenomas nor carcinomas derived from the follicular epithelium were increased, despite the fact that iodide was administered for 2 yr. It was therefore concluded that long-term treatment of iodide per se does not result in thyroid tumour induction in rats. In contrast, SCCs were observed in the submandibular gland in the 1000 ppm groups of both sexes, along with focal acinar atrophy and/or ductular proliferation, frequently accompanied by squamous metaplasia. Based on the fact that the cell proliferation of these proliferating ductules was higher in cases with metaplasia, and the evidence of a morphological continuum from meta-plasias to squamous cell carcinomas, a histogenetic relationship is suspected, which was also described in previous investigation (Takegawa et al., 1998). Based on these findings, it suggests that excess iodide has a thyroid tumour-promoting effect, but iodide per se does not induce thyroid tumours in rats. In the salivary gland, iodide was suggested to have carcinogenic potential via an epigenetic mechanism, only active at a high dose (1000 ppm in drinking water). The default value of volume of drinking water for rat is well accepted of 10 ml/100g bw·day, and the average body weight for rat is 250g. Based on these the LOAEL for salivary glands for carcinogenicity is proposed to be 100 mg/kg bw·day of iodide by drinking water

Mutagenicity/ Genotoxicity	The mutagenic potential for iodide (in potassium iodide) was studied using the L5178Y mouse (TK+/-) lymphoma assay (Kessler et al., 1980), The established mutagens ethylmethanesulphonate (EMS) and dimethylnitrosamine (DMN)were highly active in this assay, whereas iodide (KI) was inactive. Using the BALB/c 3T3 transformation assay well assessed the transformational capacities of these same agents and the positive mutagen N-ethyl-N-nitro-N-nitrosoguanidine (MNNG). All concentrations of the iodide tested were inactive in this assay it can be concluded that KI did not possess any biologically significant mutagenic cell transforming ability. Another study (J.M. Poul, and P. Sanders, 2004) on genotoxic effects of potassium iodide was conducted in vitro using the alkaline comet assay at concentration of 0.625, 1.25, 2.5, 5 and 10 mM. Additionally in the test cell viability was also measured using the Trypan blue exclusion method and expressed as proportion of total cells. The test results showed that potassium iodide did not induced DNA damage or cytotoxicity in the alkaline comet assay for doses up to 10 mM. In the same study, the chromosome damage effects of potassium iodide were evaluated in vitro using cytokinesis-block micronucleus test at concentration of 0.625, 1.25, 2.5, 5 and 10 mM. Additionally in the test cytotoxicity was also measured by the binucleated (BN) cell ratio between treated and control slides. The test results showed that potassium iodide did not induce chromosome damage or cytotoxicity in the alkaline comet assay for doses up to 10 mM. In an in vivo chromosome aberration test on embryonic hepatocytes, Stable iodine of 10 mg/kg is administered to the rats 7 days after fertilization. Then the embryonic liver was homogenated and the cells in metaphase were stained and checked under metaphase. The chromosome aberration cells were counted respectively for the concentration group and control group. The chromosome aberration rate in the concentration group was compared with that in the control group. The result showed there was no significant difference between iodide dosed group with the control group. Therefore, it can be concluded that the iodide has neither genetic toxicity nor cytotoxicity to mammalian cells.

Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Iodide (KI) was fed to male and female rats before and during breeding, to females only during gestation and lactation, and to their offspring after weaning (day 21 after birth) through to day 90, at levels of 0, 0.025, 0.05 or 0.1% (w/w) of the diet. There was no evidence suggesting that potassium iodide was embryotoxic. Litter size was significantly reduced, but birth weights and external morphology among those born alive were not significantly altered. No change in thyroid weight was observed indicating that these doses were not overtly thyrotoxic. Thyroid hormones were not assessed, however, and it is possible that thyroid function could have been altered in these animals. Nevertheless, the data are consistent with a picture of impaired thyroid function. Several tests of post-weaning behaviour showed effects at the lowest dose, 0.025 % potassium iodide. M-maze errors were increased at this dose and rotorod performance decreased. However, because these effects were not found at the higher doses it appears unlikely that they were related to potassium iodide. At present, these effects can only described as 'false positives'. The only effect on post-weaning behaviour that appeared to be consistently related to potassium iodide exposure was the reduction in nocturnal running-wheel activity found among the tested females. It may be that female cyclicity makes them more sensitive to the influence of chronic moderate iodide exposure than males and this could explain the contrast with the results of an acute test of activity and exploration, the open-field test, on which no consistent iodide-related effects were found. According to REACH guidance “R 10.8 of Guidance on information requirements and chemical safety assessment Chapter R.10: Characterisation of dose [concentration]-response for environment” The NOAEL can be calculated with the equation R 10-7:NOAEL(mg/kg bw day) = NOEC (mg/kg food)/CONV Where NOEC (mg/kg food) is 0.1, and CONV for Rattus norvegicus (> 6 weeks) is 20, and 10 for Rattus norvegicus (≤6 weeks). Therefore under this study the NOAEL for rats is 50 mg/kg bw day (developmental). In another study, twenty-five thyroiditis-prone BB/W rats were prenatally and postnatally exposed to iodine in drinking-water at dosages equivalent to 0, 0.059, or 59 mg/kg body weight per day for about 12 weeks. An increase in the number of lysosomes and lipid droplets was observed in the treated animals, especially in the higher exposure group. However, the test organism is not healty, as well as not enough information in the study, the effects cannot be considered to be dose related. Additionally, old studies were conducted with rabbits hamsters, rats and swine (Arrington LR, et al., 1965) to determine the effects of excess iodine intake. Females were bred to normal males, potassium or sodium iodide was added to the diet during the latter portion of gestation and the females were permitted to litter normally. Observations were made for length of gestation, parturition time, lactation and survival of young. 250 to 1000 ppm iodide fed for 2 to 5 days caused increasing mortality of new born rabbits. Hamsters were not affected by 2500 ppm iodine except for slightly re duced feed intake and decreased weaning weight of the young. Gestation time for rats and hamsters was not affected by iodine. Female rats and rabbits re-bred after removal from dietary iodine produced and nursed litters normally. Swine were not affected by dietary levels of iodine which were toxic to rabbits and rats. In conclusion, the iodide is not reproductive, embryonic toxicity, but the developmental toxicity was showed under concentration of 0.1% in diet, corresponding NOAEL as 50 mg/kg bw day (developmental).

Acute Toxicity	The most relevant study on vertebrates by oral route is a company study (A. Hausner, G. Weise, and A. Hofmann, 1980). In the test the effects of iodide were studied in male and female Wistar rats. 10 male and 10 female in each dose and control groups were administrated with potassium iodide for 14 days at dose of 0 (control), 2000, 2500, 2800 3200, 3600, and 4000 mg/kg body weight mg/kg bw respectively. The key value of LD50 was calculated by Probit-analysis (Fink und Hund 1965). It shows the 24 hour and 7-14 days of LD50 to rats (male/female) was respectively 3118 and 2779 mg/kg bw under test conditions. Therefore the key value which is used in the hazard classification and chemical safety assessment is 3118 mg/kg bw.
Irritation	Iodine has been used for dermal application in human as disinfectant (as Iodine and Povidine Iodine) for long time. The mechanism of disinfecting is oxidizing bactericide by iodine; meanwhile the iodine is reduced to iodide. It means after application of iodine on skin, the iodide is left on skin. In addition, based on information from assessment report of WHO, in a human assay, five patients were applied with potassium iodide in concentrations ranging from 5% to 20% in petrolatum, the reactions were negative. With such evidence, it can be concluded that iodide has no effect to the human skin.
Sensitisation	No adverse effect observed (not sensitising) for skin and respiratory sensitisation.
Health Effects Summary	This chemical has been identified by NICNAS to be of low concern to human health
Key Study/Critical Effect for Screening Criteria	TDI of iodide is 0.01 mg/kg body weight.
Ecological Toxicity [2]	
Aquatic Toxicity	The 96 hours acute toxicity test to Rainbow Trout (Laverock, M.J., M. Stephenson, and C.R. MacDonald, 1995) was conducted according to Protocol to determine the acute lethality of liquid effluents to fish, which was established by Ontario Ministry of the Environment. The results showed that the 96 hour LC50 is over 860 mg/l. The acute toxicity to daphnia of iodide was determined (INERIS Parc Technologique ALATA, 2012) according to OECD test guideline 202 following GLP procedure to give a result of 48hrs-EC50 as 1.27 mg/L (95%CL, 1.19 -1.38 mg/L). There is another data on daphnia acute toxiciy (Laboratoire d'Ecotoxicologie Parc technologique ALATA, 1996) of KI according to method of “French standard”, which was similar to OECD test guideline 202, which is 48 hrs- EC50 as 7.5 mg/l. As the study for NaI gives lower tolerance value for daphnia and the test itself is more reliable (Klimisch score 1), the 48 hrs- EC50 of 1.27 mg/l is taken as the key value. One study of acute toxicity of iodide to algae was published in well-known journal “water research” (Bringmann, G., and R. Kuhn, 1980). It was not a standard test and without declaration of GLP compliance, and in the test the 7 days cell multiplication inhibition test was applied to the model organism, Scenedesmus quadricauda (green algae) for iodide, but fulfilled basically scientific principles. The results showed the toxicity threshold (≥3% inhibition of the biomass of green algae) of iodide to green algae is 2370 mg/l.
Determination of PNEC aquatic	PNECaquatic: On the basis of the acute results for Daphnia, an assessment factor of 100 has been applied to the lowest reported effect concentration of 1.27 mg/L. The PNECaquatic is determined to be 1.27 µg/L.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.

International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment [2]	
P/vP Criteria fulfilled?	Not applicable (inorganic salt, ionic species ubiquitous in environment).
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to these inorganic ions; sodium and iodide ions are ubiquitous and are present in most water, soil and sediment.
T criteria fulfilled?	Not applicable. Acute toxicity data >0.01 mg/L in invertebrates, thus sodium iodide does not meet the screening criteria for toxicity.
Overall conclusion	Not applicable.
Revised	January 2019

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.nicnas.gov.au
2. ECHA REACH, Sodium iodide, Retrieved 2019: https://echa.europa.eu/
3. HSDB (n.d.). Hazardous Substances Data Bank. Retrieved 2015, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

AECOM Imagine it. Delivered.

Toxicity Summary - Sodium Persulfate

Chemical and Physical Properties[1,2,3,4]	
CAS number	7775-27-1
Molecular formula	Na2O8S2
Molecular weight	238
Solubility in water	730 g/l at 25 °C
Melting point	Decomposes at > 180°C
Boiling point	No data available
Vapour pressure	0 Pa at 25 °C (negligible)
Henrys law constant	No data available
Explosive potential	Non-explosive
Flammability potential	Non-flammable
Colour/Form	White crystals or powder
Overview	The persulfates category includes molecules with similar chemical structure and similar physical-chemical properties. Substances of the persulfate category are inorganic salts sharing the persulfate anion moiety. The inorganic substances differ only by the cationic portion of the salt, which is not expected to influence the hazardous properties of the molecule. The anionic part is identical and is expected to display the same environmental, ecotoxicological and toxicological behaviour based on the available data.
Environmental Fate[1,3]	
Soil/Water/Air	Substances of the persulfate category are not stable in the environment. Persulfates are not expected to adsorb to soil due to their dissociation properties, instability (hydrolysis) and high water solubility. They should behave as free ions or decompose into sulfate ions. In soils, upon decomposition, the cation could form more stable sulfate or bisulfate salts. Persulfates are not expected to bioaccumulate in the soil or in aqueous solution. They will decompose into inorganic sulfate or bisulfate.
Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	The persulfates have low repeat dose toxicity. Twenty-eight-day repeated dose oral (dietary) toxicity studies were conducted in rats with 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 potasium salt). Tests were performed in male rats only. The no observed adverse effect levels (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). 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).
Carcinogenicity	Based on the limited data available, there is no evidence of carcinogenicity of any of the persulfate salt. 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).

Mutagenicity/ Genotoxicity	Based on the limited available data, sodium persulfate was not mutagenic. An in vitro unscheduled DNA synthesis test was also negative for sodium persulfate (FMC, 1990d). The ammonium salt was not clastogenic in Chinese hamster fibroblasts in the absence of metabolic activation in a chromosome aberration test (Ishidate et al., 1988). 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). 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).
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Based on the limited data available for ammonium persulfate, the sodium persulfate is not toxic to reproduction or development. 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).
Acute Toxicity	Persulfate salts are considered to have moderate acute toxicity by the oral route. The acute oral median lethal dose (LD50) values for soidum persulfate (in rats) was reported as 895-930 mg/kg bw (Degussa AG, 1979). Clinical signs were ocular and oral discharge, irregular breathing and loss of muscle control. Persulfate salts have low acute dermal toxicity. The acute dermal LD50 was greater than 10,000 mg/kg bw (rabbits) for sodium persulfates (FMC, 1979c). Ocular and nasal discharge and slight irritation were reported in animals dermally exposed to high levels of persulfates (FMC, 1979b). Persulfates have low acute inhalation toxicity. Acute inhalation studies with sodium persulfates performed according to OECD guidelines in rats, indicated median lethal concentration (LC50) values of greater than the maximum attainable concentrations, 5.1 mg/L. 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).

Irritation	The chemicals are classified as hazardous with the risk phrase 'Irritating to Respiratory system' (Xi; R37) in the HSIS (Safe Work Australia). Groups of male ND4 Swiss Webster mice were exposed, head-only, to sodium persulfate dust for 30 minutes at concentrations of 0.26 to 3.22 mg/L. Mortality was observed in all except the lowest exposure group during the 7-day post-exposure period with clinical signs that included ocular and nasal discharge and decreased respiratory rate. Abnormal gait and whole body tremors were observed in animals exposed to the highest concentration of dust. The concentration of dust which produced a 50 % decrease in respiratory rate (RD50) was 2.25 mg/L, indicating that sodium persulfate was a respiratory system irritant (FMC, 1994). Sodium persulfates were not found to be skin irritants in animal studies. However human observations support the existing classification as skin irritants. Three brief study reports submitted by industry on sodium persulfate showed at most a slight skin irritant potential in rabbits (FMC, 1979d; FMC, 1980). The chemicals are classified as hazardous with the risk phrase 'Irritating to eyes' (Xi; R36) in the HSIS (Safe Work Australia). 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).
Sensitisation	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). 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).
Health Effects Summary	Although the persulfate salts are harmful by the oral route, potential for acute toxicity was generally not demonstrated via the dermal or inhalation routes. The persulfate salts were irritating to eyes and respiratory system but not skin irritants in animal studies, while studies in humans indicate that persulfates can cause skin irritation. The persulfates are capable of inducing skin and respiratory sensitisation in animals and these are also the major chronic effects observed in humans. Mouse LLNA results for ammonium and sodium persulfate suggest that persulfates are moderate to strong sensitisers. Overall, the main critical effects to human health are skin and respiratory sensitisation and irritation.
Key Study/Critical Effect for Screening Criteria	
Ecological Toxicity [2]	
Aquatic Toxicity	The LC50 values for acute toxicity to fish ranged between 163 to 771 mg/L for sodium persulfate. The acute toxicity EC50 values for invertebrates were between 133 and 519 mg/L for sodium persulfate. In algae, the EC50 for sodium persulfate 116 mg/L.
Determination of PNEC aquatic	A PNECaquatic of 116 µg/L was calculated using the lowest endpoint of EC50 of 116 mg/L for algae. An assessment factor of 1000 was used.
Current Regulatory Controls	

Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Biodegradation is not applicable to substances of the Persulfate Category, as the substances are inorganic. Upon contact with water or water vapour substances of the persulfate category hydrolyse into cation and persulfate anion. The persulfate anion, independent of the cation, undergoes further decomposition in normal water or acid conditions, readily oxidizing water to oxygen, producing sulphate and hydrogen ions. All final persulfate degradation products are ubiquitous to the environment.
B/vB criteria fulfilled?	No. Persulfates are very soluble in water and are not expected to bioaccumulate in soil or aqueous solutions.
T criteria fulfilled?	Based on measured acute toxicity endpoints of greater than 1 mg/L, sodium persulfate does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	January 2019

References

1. NICNAS (2017) Human Health Tier II Assessment for Persulfates
2. OECD (2005) SIDS Initial Assessment Profile on Persulfates
3. ECHA REACH, Disodium peroxodisulphate, Retrieved 2017: https://echa.europa.eu/information-on-chemicals/registered-substances
4. ICSC Sodium Persulfates, Retrieved 2017: http://www.inchem.org

AECOM Imagine it. Delivered.

Toxicity Summary - Sodium sulphate

Chemical and Physical Properties[1,3,4,5]	
CAS number	7757-82-6
Molecular formula	Na2SO4
Product name	142.04 g/mol
Molecular weight	161 g/l at 20 °C
Solubility in water	No data found.
Melting point	884 °C
Boiling point	Decomposition occurs above 884°C.
Vapour pressure	Solid
Henrys law constant	Expected to be extremely low
Explosive potential	No data found.
Flammability potential	No data found.
Colour/Form	Not combustible. Gives off irritating or toxic fumes/gases in a fire.
Overview	Sodium sulfate is widely distributed in nature; it occurs as mineral salts (e.g. thenardite, mirabilite), it is present in almost all fresh and salt waters and sulfate as such is normally present in almost all natural foodstuffs. Both sodium and sulfate ions are among the most common ions found in all living organisms. In mammals, sulfate is an normal metabolite of sulfur-containing amino-acids, it is normally incorporated in a variety of body compounds and it plays an important role in detoxification/ excretion processes due to sulfoconjugation Sodium sulfate has been produced for many years in high volumes for use in detergents, glass and paper manufacture and a variety of smaller industrial uses National Industrial Chemicals Notification and Assessment Scheme (NICNAS) has performed an IMAP environment Tier 1 summary which concluded that sodium sulphate is an inorganic substance comprising ions of low ecotoxicological concern. This chemical is not expected to pose an unreasonable risk to the environment provided that ANZECC water quality guidelines for physical and chemical stressors are not exceeded.
Environmental Fate[1,4,5]	
Soil/Water/Air	Sodium sulphate is a solid inorganic salt well soluble in water. In water solutions it is fully dissociated to sodium and sulfate ions. In anaerobic environments sulfate is biologically reduced to (hydrogen) sulphide by sulfate reducing bacteria, or incorporated into living organisms as source of sulphur, and thereby included in the sulphur cycle. The BCF of sodium sulfate is very low (0.5) and therefore bioconcentration is not expected. Sodium and sulfate ions are essential to all living organisms and their intracellular and extracellular concentrations are actively regulated. However some plants (e.g. corn and *Kochia Scoparia*), are capable of accumulating sulfate to concentrations that are potentially toxic to ruminants.
Human Health Toxicity Summary [1,2,4,5]	
Chronic Repeated Dose Toxicity	Valid oral repeated dose toxicity studies with 21, 28 and 35 day studies in hens and pigs are available. Toxicity was confined to changes in bodyweight, water and feed intake and diarrhoea. These changes occurred only at very high doses of sodium sulfate. In ruminants, high concentrations of sulfate in food may result in the formation of toxic amounts of sulfites by bacterial reduction the rumen, leading to poly-encephalomalacia. The available data do not allow the derivation of a NOAEL. Based on available consumer data, a daily dose of around 25 mg/kg/day is well tolerated by humans

Carcinogenicity	There is no valid oral carcinogenicity study. Limited data from experimental studies support the notion that a substance that is abundantly present in and essential to the body is unlikely to be carcinogenic.
Mutagenicity/ Genotoxicity	Sodium sulfate has been shown to be without effect in the Ames test using various strains of *S. typhimurium* (TA1535, TA1537, TA100, TA98) both with and without S9 activation in a GLP standardised test Based on the natural intra- and extracellular occurrence of the substance it can be concluded that sodium sulfate is highly unlikely to be mutagenic
Reproductive Toxicity	Limited data of poor validity did not provide an indication of toxicity to reproduction.
Developmental Toxicity/Teratogenicity	No data were found.
Acute Toxicity	The acute toxicity (LD50) of sodium sulfate has not been reliably established but is probably far in excess of 5000 mg/kg. In an inhalation study with an aerosol, no adverse effects were found at 10 mg/m3. Also human data indicate a very low acute toxicity of sodium sulfate. Human clinical experience indicates that very high oral doses of sodium sulfate, 300 mg/kg bw up to 20 grams for an adult, are well tolerated, except from (intentionally) causing severe diarrhoea. WHO/FAO did not set an ADI for sodium sulfate. There is no data on acute dermal toxicity, but this is probably of no concern because of total ionisation in solution.
Irritation	Sodium sulfate is not irritating to the skin and slightly irritating to the eyes. Respiratory irritation has never been reported.
Sensitisation	Sodium sulphate is not a skin or respiratory sensitiser
Key Study/Critical Effect for Screening Criteria	The Australian Drinking Water Guidelines for sodium and sulphate may apply to sodium sulphate.
Ecological Toxicity [3,4,5]	
Aquatic Toxicity	Algae were shown to be the most sensitive to sodium sulfate; EC50 120h = 1,900 mg/l. For invertebrates *(Daphnia magna)* the EC50 48h = 4,580 mg/l and fish appeared to be the least sensitive with a LC50 96h = 7,960 mg/l for *Pimephales promelas*. No data were found for long term toxicity. The acute studies all show a toxicity of sodium sulfate higher than 100 mg/l, no bioaccumulation is expected
Determination of PNEC aquatic	An assessment factor of 1000 has been applied to the lowest reported effect concentration of 1900 mg/L for Daphnia. The PNEC aquatic is 1.9 mg/L.
Current Regulatory Controls	
Australian Hazard Classification	The chemical is not listed in the Hazardous Substance Information System (HSIS) (Safe Work Australia 2013).
Australian Occupational Exposure Standards	No data found
International Occupational Exposure Standards	No data found
Australian Food Standards	No data found
Australian Drinking Water Guidelines	The Australian Drinking Water Guideline for sulphate is 250 mg/L (aesthetic) and sodium is 180 mg/L (aesthetic).
Aquatic Toxicity Guidelines	No data found
PBT Assessment	
P/vP Criteria fulfilled?	Sodium sulphate is an inorganic salt that dissociates completely to sodium and sulphate ions in aqueous solutions. The persistent criterion is not considered applicable to this inorganic salt.
B/vB criteria fulfilled?	The BCF of sodium sulfate is very low (0.5) and therefore bioconcentration is not

	expected.
T criteria fulfilled?	The acute aquatic toxicity of sodium sulfate is > 0.01 mg/L. Hence the substance does not fulfill the screening criteria for toxic (T)
Overall conclusion	Not a PBT substance (based on screening data).

References

1. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2017, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
2. National Health and Medical Research Council, Natural Resources Management Ministerial Council, national Water Quality Management Strategy, Australian Drinking Water Guidelines 6, 2011, Version 3.3 Updated November 2016.
3. National Industrial Chemicals Notification and Assessment Scheme (NICNAS), IMAP Environment Tier I Summary all tranches, 2016.
4. OECD (2005a) Screening Information Dataset (SIDS) Initial Assessment Report for Sodium Sulfate, CAS Number 7757-82-6, UNEP Publications
5. OECD (2005b) SIDS Initial Assessment Profile for Sodium Sulfate, CAS Number 7757-82-6, UNEP Publications

AECOM Imagine it. Delivered.

Toxicity Summary - Tributyl tetradecyl (TTPC)

Chemical and Physical Properties	
CAS number	81741-28-8
Molecular formula	C26-H56P.Cl
Product name	BE9
Molecular weight	435.15 g/mol
Solubility in water	miscible
Melting point	45 °C
Boiling point	439 °C (estimated)
Vapour pressure	Solid
Henrys law constant	1.04 x 10-8 kPa at 25 °C (estimated)
Explosive potential	No data found
Flammability potential	No data found
Colour/Form	No data found
Overview	Limited toxicity information was located for this alkyl phosphonium salt.
Environmental Fate[1]	
Soil/Water/Air	No data found
Human Health Toxicity Summary [1,2]	
Chronic Repeated Dose Toxicity	No data were found.
Carcinogenicity	No data were found.
Mutagenicity/ Genotoxicity	No data were available for TTPC. A brief report for TBPB noted that the chemical tested negative in an Ames bacterial mutagenicity assay, a Chinese hamster ovary (CHO) chromosome aberration test and a cell transformation test using Hamster Embryo Cells (HEC) although further details were not provided (Dunn et al. 1982). Therefore, TBPB is not mutagenic under the conditions tested and, on the basis of this limited evidence; it is assumed that TTPC is not genotoxic.
Reproductive Toxicity	No data were found.
Developmental Toxicity/Teratogenicity	No data were found.
Acute Toxicity	An inhalation study (EPA Office of Prevention, Pesticides and Toxic Substances (OPPTS) 870.1300) in rats exposed nose-only to TTPC (particle size 1.7 to 2.1 µm) reported hypoactivity, gasping, irregular respiration, red nasal discharge, ano-genital staining and abdominal distension at 0.05 mg/L (US EPA 2012b). Six of the 10 animals died within three days of a four-hour exposure. Gross necropsy revealed red coloured lungs, distension of stomach and / or intestines and / or mottled liver. The single exposure acute inhalation LC50 for this study was identified as <0.05 mg/L. This study shows that TTPC is highly toxic by the inhalation route in rats. No oral or dermal information was available for TTPC. However, based on analogue data available for THPB, TBPC and TBPB from animal studies, acute toxicity of TTPC by oral and dermal route is likely to be moderate

Irritation	No information was available for TTPC but data were available for the analogues THPB and TBPC.for skin irritation. Overall, the effects observed with the analogues THPB and TBPC, albeit after a 24-hour exposure period compared with the four-hour exposure specified by the equivalent OECD TG, demonstrate the likely corrosive potential of TTPC to the skin. No information was available for TTPC but data were available for the analogues THPB, TBPC and TBPB for eye irritation. The effects observed in all tests with the analogues THPB, TBPC and TBPB demonstrate the likely corrosive potential of TTPC to the eyes. In an inhalation study with TTPC in rats, a red nasal discharge and facial staining was noted (US EPA 2012b). While the information in the study is limited based on the analogues being corrosive to the skin it is likely that the chemicals are also irritant to the respiratory mucosa. TTPC is therefore likely to be a respiratory irritant.
Sensitisation	No data were available for TTPC. TBPC at 0.1% concentration in normal saline solution was determined as not sensitising to the skin following dermal applications (undisclosed induction and one challenge treatment) in guinea pigs (US EPA 1978). TBPC is not a skin sensitiser in guinea pigs and therefore a sensitisation potential for TTPC is not expected. No data were available for respiratory sensitisation.
Health Effects Summary	TTPC demonstrates high acute toxicity by the inhalation route. Based on read across data available from THPB, TBPC and TBPB, the chemical has moderate acute toxicity by oral and dermal routes and is corrosive to the skin and eye and is a respiratory irritant. Data available for TBPC and TBPB indicate that the chemical is not a skin sensitiser or genotoxic, respectively. No repeat dose, carcinogenicity or reproductive toxicity data were available for the chemical or suitable analogues. Chronic exposure may be considered as inappropriate given the nature of TTPC and analogues as direct acting corrosives mediating severe adverse effects at the site of contact. In conclusion, the critical health effect of TTPC is its acute inhalation toxicity.
Key Study/Critical Effect for Screening Criteria	No data are available for determining the critical effect and the LOAEL/NOAEL for an oral reference dose.
Ecological Toxicity [1,2]	
Aquatic Toxicity	The modelled acute endpoint for Daphnia is 16.788 mg/L and Fish is 1059.2530 mg/L.
Determination of PNEC aquatic	PNECaquatic: On the basis that the modelled data consists of short-term results from two trophic levels, an assessment factor of 1,000 has been applied to the lowest reported effect concentration of 16.788 mg/L for Daphnia. The PNECaquatic is calculated to be 0.0168 mg/L.
Current Regulatory Controls	
Australian Hazard Classification	The chemical is not listed in the Hazardous Substance Information System (HSIS) (Safe Work Australia 2013).
Australian Occupational Exposure Standards	No data found
International Occupational Exposure Standards	No data found
Australian Food Standards	No data found

Australian Drinking Water Guidelines	No data found
Aquatic Toxicity Guidelines	No data found
PBT Assessment	
P/vP Criteria fulfilled?	No information is available on biodegradation.
B/vB criteria fulfilled?	Not Bioaccumulative (Based on an estimated log Kow value of 6.26)
T criteria fulfilled?	No chronic toxicity data are available for TTPC. The lowest modelled acute endpoint of TTPC is 16.788 mg/L in invertebrates. Since this value is >0.1 mg/L, TTPC does not meet the screening criteria for toxicity.
Overall conclusion	Inconclusive.

References

1. Material Safety Data Sheet for Bellacide 350, BWA Water Additives, SDS No. 10794
2. National Information System of the Regional Integrated Pest Management (IPM) Centers, U.S. Department of Agriculture and National Institutes of Food and Agriculture (www.ipmcenters.org).

Toxicity Summary - 2,2`,2"- Nitrilotriethanol

Chemical and Physical Properties [1,2, 3,6]	
CAS number	102-71-6
Molecular formula	C6H15NO3
Molecular weight	149.19 g/mol
Solubility in water	Miscible with water.
pH	10.5
Melting point	17-21.6 °C
Boiling point	153 °C at 0.1007 kPa 192.87 °C at 0.7996 kPa 236.69 °C at 5.01 kPa 320 °C at 101 kPa
Vapour pressure	$3.59x10^{-6}$ mm Hg at 25 °C
Henrys law constant	$7.05x10^{-13}$ atm-cu m/mole at 25 °C
Explosive potential	No data found.
Flammability potential	Combustible, when exposed to heat or flame. Gives off irritating or toxic fumes (or gases) in a fire.
Colour/Form	Pale yellow to colourless viscous liquid with a slight ammonia odour.
Overview	Triethanolamine is a member of the ethanolamines family that combines the properties of amines and alcohols. Triethanolamine is typically supplied as a pale colourless to yellow liquid with an ammonia-like odor. Triethanolamine is primarily used in detergents, personal-care products, and textile finishing. Triethanolamine may also be used as in other applications including adhesives, agricultural products, concrete additives, gas treating processes, rubber, surfactants, photographic chemicals, and urethane foams. Contact with triethanolamine may cause slight to severe eye irritation. Brief contact is essentially nonirritating to the skin, but repeated exposure may cause irritation and burns. Skin contact may cause an allergic skin reaction. At room temperature, exposure to vapour is minimal due to low volatility; single exposure is not likely to be hazardous. This product has very low toxicity if swallowed. Harmful effects are not anticipated from swallowing small amounts, but swallowing larger amounts may cause injury. This product has been toxic to the fetus in laboratory animals at doses toxic to the mother. Findings from a study by the National Toxicology Program suggest an increased incidence of liver tumors in mice, but their relevant to humans is not clear. Triethanolamine is water soluable and biodegradable according to the OECD 301A test for biodegradation. It is not expected to bioaccumulate or persist in the environment. Triethanolimine is practically non-toxic to aquatic organisms on an acute basis. However large releases may increase the pH of aquatic systems to levels that may be toxic to aquatic organisms.

Environmental Fate [1,3,4,6]	
Soil/Water/Air	If released to soil, triethanolamine is expected to have very high mobility based upon an estimated Koc of 7. However, the pKa of triethanolamine is 7.8, indicating that this compound will primarily exist in cation form; and cations generally adsorb to organic carbon and clay more strongly than their neutral counterparts. Volatilization from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry's Law constant of 7.1X10-13 atm-cu m/mole. If released into water, triethanolamine is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Triethanolamine biodegraded in a biochemical oxygen demand (BOD) test at an initial concn 50 ppm. After 10 days, the ThOD (theoretical oxygen demand) was 70% using acclimated water as seed and sewage as inoculum. Volatilization from water surfaces is not expected to be an important fate process based upon this compound's estimated Henry's Law constant. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyze under environmental conditions

Human Health Toxicity Summary [1,2,3,4,5,6]	
Chronic Repeated Dose Toxicity	Fischer 344 rats and B6C3F1 mice were administered 0, 500, 1000, 2000, 4000 or 8000 mg/100 mL triethanolamine in drinking water (NTP 1990). Water consumption was reduced at the top two doses. No other details were provided. In a 91-day study conducted in accordance with OECD TG 408, Cox CD rats were administered 88.5% triethanolamine in the diet at doses of 0, 250, 500 or 1000 mg/kg bw/day (REACH 2013). There were no significant dose-dependent changes in bodyweight, organ weight, histopathology, pathology and haematology. No Lowest Observed Adverse Effect Level (LOAEL) or No Observed Adverse Effect Level (NOAEL) can be established for this study. In a 90-day study, rats (strain not specified) were administered doses of 5 to 2610 mg/kg bw/day triethanolamine in the diet (Smyth et al. 1951). The study reported microscopic lesions and mortality at doses of 730 mg/kg bw/day and above. The authors indicated the NOAEL as 80 mg/kg bw/day. No other details were provided. In 60- and 120-day studies in rats (strain not specified) given 200 to 1800 mg/kg bw/day triethanolamine, effects observed included liver changes at all treatment doses after 60 and 120 days administration, kidney changes at 400 mg/kg bw/day after 60 and 120 days administration, and kidney damage at >800 mg/kg bw/day after 60 and 120 days administration (Kindsvatter 1940). The specific changes in the liver and kidney were not described. No other details were provided. The LOAEL for this study was 200 mg/kg bw/day. Repeated dermal dose toxicity with triethanolamine application was consistently associated with inflammation at the treatment site. Systemic effects included changes in bodyweight and organ to bodyweight ratios. The critical study for determining the effects of repeated dermal exposures to the chemical is the 90-day study cited in REACH (2013) conducted similarly to OECD TG 411. The NOAELs for this study are 125 mg/kg bw/day for males and 250 mg/kg bw/day for females. In an inhalation study, Fischer 344 rats were exposed to 0, 125, 250, 500, 1000 or 2000 mg/m3 triethanolamine for 16 days (NTP 1985b). The effects observed included decreased bodyweight at 2000 mg/m3 for both sexes, increased liver weight in males at 2000 mg/m3, increased kidney weight in males at concentrations ≥500 mg/m3, and increased kidney weight in females at concentrations ≥250 mg/m3. Minimal to slight acute inflammation of the larynx was reportedbut the doses for which this effect was seen were not specified. The LOAECs are 500 mg/m3 in males and 250 mg/m3 in females. The NOAECs are 250 and 125 mg/m3 in males and females, respectively. Wistar rats were exposed through the head and nose to 0, 0.02, 0.1 or 0.5 mg/L aerosolised triethanolamine in a 28-day study conducted in accordance with OECD TG 412 (Gamer et al., 2008). There were no treatment-related effects seen on bodyweight, haematology, clinical chemistry and neurobehavioural parameters. Local effects, such as minimal to moderate focal inflammation in the submucosa of the larynx region, were reported at all treatment concentrations. The LOAEC and NOAEC for systemic effects cannot be established. The LOAEC for local effects is 0.02 mg/L. B6C3F1 mice exposed to 0, 125, 250, 500, 1000 or 2000 mg/m3 triethanolamine for 14 days showed minimal acute inflammation of the laryngeal submucosa (NTP 1985a). The doses for which this effect was seen were not specified.
Carcinogenicity	The International Agency for Research on Cancer (IARC) has classified the chemical as ‘not classifiable as to its carcinogenicity to humans’ (Group 3), based on inadequate evidence for carcinogenicity in humans and experimental animals (IARC, 2000). There was no evidence of carcinogenicity by oral (up to 1000 mg/kg/day for 104 weeks, and up to 3334 mg/kg/day for 82 weeks amongst rats and mice respectively) or dermal routes (dose unknown) in studies of 14-18 months duration using rats and mice. No inhalation data were available.

Mutagenicity/ Genotoxicity	Triethanolamine was not genotoxic in a number of in vitro studies (bacterial reverse mutation, mammalian cell cytogenetics, and unscheduled DNA synthesis). On the basis of the negative results observed in a range of in vitro studies, in vivo genotoxicity is not anticipated.
Reproductive Toxicity Developmental Toxicity/Teratogenicity	Triethanolamine is not considered to be toxic to fertility and not considered to be a developmental toxicant. There were no effects observed in the reproductive organs of the animals treated with the chemical from repeated oral, dermal and inhalation toxicity studies. In a reproduction/developmental toxicity screening test conducted in accordance with OECD TG 421, Wistar rats were administered 0, 100, 300 or 1000 mg/kg bw/day triethanolamine by gavage (REACH 2013). The animals were treated during pre-mating (two weeks for both sexes), mating (maximum of two weeks for both sexes), post-mating (one week in males), and the entire gestation period and four days of lactation in females. There were no parental systemic effects reported in all of the treated animals. Most of the animals treated at the top dose showed transient salivation, which could be attributed to the unpalatability of the chemical or local irritation of the upper digestive tract. There were no effects on fertility observed in any of the treated animals. The parental LOAEL and NOAEL for local effects are 1000 and 300 mg/kg bw/day, respectively. The developmental LOAEL and NOAEL are 1000 and 300 mg/kg bw/day, respectively. The LOAEL and NOAEL for fertility cannot be established. A dye formulation containing 0.15, 1.5 or 2% triethanolamine was applied to the shaved skin of CD-1 rats (Burnett et al. 1976). The application occurred seven times during the gestation period. There were no systemic or local effects observed. No developmental effects were reported.
Acute Toxicity	The chemical was of low acute toxicity in animal tests following oral exposure. The median lethal dose (LD50) in experimental rats studies ranged from is 4190–11300 mg/kg bw triethanolamine. Two studies in mice (strain not specified), two studies in rabbits (strain not specified), and three studies in guinea pigs (strain not specified) reported acute oral LD50s of 5400 to 7800, 2200 to 5200, and 2200 to 8000 mg/kg bw, respectively.Observed sub-lethal effects included agitation, elevated respiration and reduced grooming (NIWL, 2003; CIR, 2011). The chemical was of low acute toxicity in animal tests following dermal exposure. The median lethal dose (LD50) in rabbits is greater than 2000 mg/kg bw. Observed sublethal effects included mild erythema 24 hours after exposure, resolving after 6 –10 days (REACH; CIR, 2011). Due to the low vapour pressure of the chemical, the highest attainable vapour concentration is 1.8 mg/m³. In a study conducted in rats (strain not specified) exposed to the chemical (1.8 mg/m³), no deaths were reported. One out of 12 rats exposed showed signs of chronic bronchitis (REACH).
Irritation	Based on the available data, the chemical is considered a respiratory and eye irritant. In two studies conducted similarly to OECD TG 405 the average Draize scores for corneal opacity, redness of the conjunctivae and chemosis were 1, 2 and 1.75 respectively (REACH). In one study, the corneal opacity in one animal had not fully resolved by day eight of the observation period. However, based on the results seen in the other animals, it is expected that the corneal opacity would fully resolve had the observation period continued for 21 days. The chemical was not irritating to skin in studies that were performed in accordance with OECD Test Guideline (TG) 404 (REACH). In one study, three Vienna White Rabbits were dermally exposed to the chemical (85 % concentration of triethanolamine and 15 % diethanolamine) through a occlusive patch for four hours. Neither oedema nor erythema was observed throughout the observation period (REACH). In animal studies with repeated exposures, the chemical was applied to rabbit ears over 10 open applications, with 10 unoccluded applications to abdominal intact skin, or with three semi-occluded 24-hour applications to abraded skin. These exposures resulted in slight to moderate irritation (CIR, 2013). In a two-year repeated dose dermal study, the chemical caused lesions consisting of acanthosis (thickened skin), ulceration and chronic active inflammation at the application site. In the repeated dose inhalation studies, minimal to slight acute inflammation of the larynx was observed in rats and mice (NTP 1985a, 1985b). In a more recent 28-day inhalation study, minimal to moderate focal inflammation in the submucosa of the larynx was observed in rats (Gamer et al. 2008).

Sensitisation	Triethanolamine is not a skin sensitizer in animals. The negative results observed for the chemical in several guinea pig maximisation tests and one local lymph node assay support a conclusion that the chemical is not a skin sensitiser (REACH; CIR, 2013).
Health Effects Summary	Triethanolamine has low acute oral and dermal toxicity but may cause eye and respiratory irritation. Triethanolamine was non-irritating to the skin in rabbit studies, whilst studies in humans indicate that the chemical can cause skin irritation. The chemical is not a skin sensitiser. The chemical is neither genotoxic, carcinogenic nor a reproductive toxicant.
Key Study/Critical Effect for Screening Criteria	The most appropriate NOAELs for risk assessment, determined from the 90-day repeat dermal dose toxicity study cited in REACH (2013) are 125 (males) and 250 (females) mg/kg bw/day based on systemic effects. Uncertainty factors: 10 (interspecies variability); 10 (intraspecies variability); 10 (subchronic to chronic) Oral RfD = 125/1000 = 0.125 mg/kg/day Drinking water guideline value = 0.49 ppm

Ecological Toxicity [1,3, 4,6]	
Aquatic Toxicity	Triethanolamine is of low acute toxicity to fish and aquatic invertebrates. The most sensitive fish species tested was the fathead minnow Pimephales promelas for which a 96h-LC50 of 11,800 mg/l was determined. Triethanolamine was slightly more toxic to Daphnia, which had a 24h-EC50 of 1,390 mg/l. In a 21 day reproduction test with Daphnia magna, a NOEC of 16 mg/l and an EC50 of 2,038 mg/l were determined in a semi-static test (concentrations measured twice during the test). Triethanolamine appears to be more toxic to algal species. Toxicity tests have been carried out at both constant pH and allowing the pH to increase with increasing triethanolamine concentration. In two cases triethanolamine appears to be more toxic when the test is carried out allowing the pH to increase. In one case, using the green algae Scenedesmus quadricauda, the 7-8 day toxicity threshold (defined as the concentration which just caused an effect of cell multiplication of around 3% compared with controls - can be considered as a NOEC) for triethanolamine was found to be much lower at constant pH (toxicity threshold = 1.8 mg/l) than when the pH was allowed to vary (toxicity threshold = 715 mg/l). The EC50 was reported as 910 mg/l for Scenedesmus subspicatus (algae) for 96 hour exposure under test conditions where the test media was neutralised.
Determination of PNEC aquatic	PNECaquatic: 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 of1.8 mg/L for Scenedesmus quadricauda mg/L for invertebrates. The PNECaquatic is 0.18 mg/L.
Current Regulatory Controls [2]	
Australian Hazard Classification	Triethanolamine is listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia 2013) with a recommended Exposure Standard.
Australian Occupational Exposure Standards	Time Weighted Average (TWA) of 5 mg/m^3 (Safe Work Australia 2013).
International Occupational Exposure Standards	TWA: 5 mg/m^3 [Belgium, Finland, Iceland, New Zealand, Peru] 0.5 mg/m^3 [Denmark].
Australian Food Standards	Triethanolamine is listed as a permitted processing aid in bleaching agents, washing and peeling agents, water used as an ingredient in other foods, and miscellaneous functions under the conditions of Good Manufacturing Practice (GMP) (Food Standards Australia New Zealand 2013).
Australian Drinking Water Guidelines	No data found
Aquatic Toxicity Guidelines	No data found
PBT Assessment [1,3,4,6]	
P/vP Criteria fulfilled?	There are conflicting findings from standard ready biodegradability tests regarding the rate of biodegradation of triethanolamine. Some studies indicate relative rapid biodegradation, whereas some closed bottle studies indicate slow biodegradation under the test conditions (OECD 1995). However, the chemical is inherently biodegradable. The results of a test using OECD test guideline 302B showed that 89% of the chemical is degraded after 14 days (OECD 1995). Thus, Triethanolamine is categorised as Persistent.
B/vB criteria fulfilled?	Based on the measured log Kow of -1.0 and a measured BCF of <3.9 L/kg in fish, triethanolamine has low bioaccummulation potential and is considered not bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of triethanolamine is > 0.01 mg/L. Hence the substance does not fulfill the screening criteria for toxic (T)
Overall conclusion	Not a PBT substance (based on screening data). Further assessment of the environmental risks from the use of this chemical is not required as identified by DoEE.
Revised	April 2018

References

1. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2016, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS), 2014. Inventory Multi-Tiered Assessment and Prioitisation (IMAP), Human Health Tier II Assessment for Ethanol, 2,2',2''- nitrilotris-, CAS Number 102-71-6.
3. OECD (1995) SIDS Initial Assessment Report for Triethanolamine, CAS Number 102-71-6
4. DOW Product Safety Assessment Triethanolamine, 2014
5. International Agency for Research on Cancer (IARC) – Summaries & Evaluations, Triethanolamine, 2000
6. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme

AECOM Imagine it. Delivered.

Toxicity Summary - Sodium perborate tetrahydrate

Chemical and Physical Properties	
CAS number	10486-00-7
Molecular formula	NaBO3. 4H2O / NaBO2. H2O2. 3H2O
Molecular weight	153.9
Solubility in water	g/100ml at 20°C: 2.3
Melting point	ca. 60-65.5°C
Boiling point	Decomposes.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	No data available.
Overview	Perborates are salts of perboric acid. Sodium perborate degrades into sodium metaborate and water with the generation of hydrogen peroxide and oxygen. Sodium metaborate is the salt of a strong base (sodium hydroxide) and a weak acid (boric acid). Undissociated boric acid is the main species present in the blood of mammals following exposure to these chemicals. Sodium perborate tetrahydrate (CAS No. 10486-00-7) have reported domestic use as bleaching agents.
Environmental Fate[1]	
Soil/Water/Air	Perborates have high water solubility. Perborates dissociate to borates and hydrogen peroxide in aqueous environments. The most important borate species, which will be formed in aqueous solutions, is boric acid (H3BO3). At relevant environmental pH values of ≤ 7 no significant adsorption of boron compounds in soil and the aquatic compartments are to be expected (EPA, 1975; Koehnlein, 1972).
Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	In a repeated dose toxicity study, sodium perborate tetrahydrate (CAS No. 10486-00-7) was administered (by gavage) to rats at 1000 mg/kg bw/day (70 mg boron/kg bw/day) for 28 days. Acanthosis and hyperkeratosis in the forestomach, and hyperplasia of the fundic mucosa were noted. At the end of the study, the red blood cell count, haemoglobin, haematocrit and number of lymphocytes were significantly decreased; the number of platelets was significantly increased. The spleen size and splenic parenchyma were reduced. Although a significant (18 %) decrease in absolute testicular weights was recorded, the authors attributed this to a generalised weight reduction of 15 %; histological examination of the testes revealed no adverse effects. The lowest observed adverse effect level (LOAEL) was 1000 mg/kg bw/day (70 mg boron/kg bw/day), based on effects on the stomach, spleen and the haematopoietic system. It was concluded that the no observed adverse effect level (NOAEL) for males or females was below 1000 mg/kg bw/day (EU RAR, 2007; SCCS, 2010; REACH). In a repeated dose toxicity study, sodium perborate tetrahydrate (CAS No. 10486-00-7) was applied at 200 mg/kg bw/day (as a 10 % aqueous solution) to the abraded skin of New Zealand White rabbits for three weeks. After exposure, the skin was near normal (signs of mild irritation in some cases) and there were no adverse microscopic findings in different organs. A NOAEL of 200 mg/kg bw/day was established, being the highest tested dose (EU RAR, 2007; SCCS, 2010; REACH). In another repeated dose toxicity study, sodium perborate tetrahydrate (CAS No. 10486-00-7) was applied at 50 mg/kg bw (as a 2.5 % aqueous solution) to the

	intact skin of New Zealand White rabbits (three/sex), five days/week for 13 weeks. The treatment caused no skin irritation and there were no adverse effects on blood parameters or on the gross histopathology of selected organs. An NOAEL of 50 mg/kg bw/day was established, being the highest tested dose (EU RAR, 2007; SCCS, 2010; REACH).
Carcinogenicity	Not likely to have any carcinogenic potential.
Mutagenicity/ Genotoxicity	Not considered to have mutagenic or genotoxic potential.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	In a repeated dose toxicity study, sodium perborate tetrahydrate (CAS No. 10486-00-7) was administered (by gavage) to rats at 1000 mg/kg bw/day (70 mg boron/kg bw/day) for 28 days. The authors recorded a significant (18 %) decrease in absolute testicular weights but this was attributed to a generalised weight reduction of 15 %. A histological examination of the testes revealed no adverse effects. It has also been argued that more sensitive methods of histopathology than used in this study (fixed with formalin) could have revealed more subtle effects. Therefore, using reduced testes weights as early signs of testicular toxicity cannot be dismissed in view of the known testicular toxicity of the borates. It was concluded that the NOAEL for males or females was below 1000 mg/kg bw/day (EU RAR, 2007; SCCS, 2010; REACH). In a developmental toxicity study, sodium perborate tetrahydrate (CAS No. 10486-00-7) was administered (by gavage) to 25 pregnant Crl:Cd (SD) rats on gestation days (GD) 6–15 at doses of 0, 100, 300 and 1000 mg/kg bw/day. The NOAEL for maternal toxicity was established as 100 mg/kg bw/day (7 mg boron/kg bw/day), based on significant reductions in body weight gain at the two highest doses. It is also noted that even though reduced maternal weight gain might partly be due to an increased number of resumptions and reduced foetal weights, other toxicological studies have supported the view that doses above 100 mg/kg bw/day administered via gavage are toxic to the dams. A dose-related effect was found on the ossification and bone system. While various incomplete ossifications and wavy ribs occurred at 300 mg/kg bw/day, malformations (fused ribs) were observed at 1000 mg/kg bw/day. The NOAEL for developmental toxicity was established as 100 mg/kg bw/day (7 mg boron/kg bw/day) (EU RAR, 2007; SCCS, 2010; REACH).
Acute Toxicity	The reported oral LD50 for sodium perborate tetrahydrate is 2567 mg/kg bw (CAS No. 10486-00-7). The chemical is likely to have low acute toxicity following dermal exposure. It is also noted that the dermal absorption through intact skin is very low. The available data (median lethal concentration—LC50, inhalation) for sodium perborate tetrahydrate is 1.65 mg/L. Reported signs of toxicity included gasping, red nasal discharge, and compound-covered faeces (EU RAR, 2007; SCCS, 2010; REACH).
Irritation	The chemicals in the group are classified as hazardous, with hazard category Specific Target Organ Toxicity (Single Exposure) – Category 3 and hazard statement 'May cause respiratory irritation' (H335) in the HCIS (Safe Work Australia). Although slight skin irritant effects were reported in animal studies, the effects were not sufficient to warrant a hazard classification for the chemicals in this group. The sodium perborates are classified as hazardous with hazard category 'Eye Damage – Category 1' and the hazard statement 'Causes serious eye damage' (H318) in the HCIS (Safe Work Australia). In an eye irritation study conducted according to Federal Hazardous Substances Act Regulations 191.12 (1964-09) of the USA, 0.1 mL of sodium perborate tetrahydrate (CAS No. 10486-00-7) was placed once into the right eyes of six albino rabbits. The chemical was judged to be corrosive as severe corneal damage, severe iritis and severe conjunctivitis were observed in all animals (EU RAR, 2007; SCCS, 2010; REACH).
Sensitisation	Not likely to be skin and respiratory sensitisers.
Health Effects Summary	The critical health effects for risk characterisation include systemic long-term effects (reproductive toxicity, developmental toxicity), systemic acute effects

	(acute toxicity from oral/inhalation exposure) and local effects (respiratory and eye irritation).
Key Study/Critical Effect for Screening Criteria	The lowest NOAEL of 50 mg/kg bw/day from the repeated dose dermal study will be used for risk assessment.
Ecological Toxicity[3]	
Aquatic Toxicity	The following aquatic toxicity endpoints are based on modelled estimates of sodium perborate (CAS 7632-04-4) from ECOSAR: The 96hr LC50 for fish is estimated to be 2610 mg/L The 48 hr LC50 for daphnids is estimated to be 1241 mg/L The 14 day LC50 for earthworms is estimated to be 164.5 mg/L The 96 hr EC50 for algae is estimated to be 444 mg/L
Determination of PNEC aquatic	In a recent publication Dyer (2001) used a probabilistic approach to derive a PNEC0.05 (Predicted No Effect Concentration for 95% of the species) from chronic studies that were available for boron for all trophic levels. Mean toxicity levels per taxa were determined and then converted to a cumulative probability term and curve-fit assuming a log-logistic distribution. The PNEC 0.05 derived from this analysis was 3.45 mg B/l when all species data with uniform chronic toxicity endpoints (NOEC, LC10) were considered.
Current Regulatory Controls[4]	
Australian Hazard Classification	Reproductive toxicity – category 1B Acute toxicity – category 4 Specific target organ toxicity (single exposure) – category 3 Eye damage – category 1
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Expected to be biodegradable based on Ecosar prediction using sodium perborate.
B/vB criteria fulfilled?	No. Estimated log Kow for sodium perborate: 0.08 (Log Kow < 4.5)
T criteria fulfilled?	No. Acute toxicity values > 1 mg/L.
Overall conclusion	Not PBT
Revised	October 2019

References

1. IPCS INCHEM, Sodium perborate tetrahydrate. Retrieved 2019: http://www.inchem.org/documents/icsc/icsc/eics1046.htm
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier II Assessment for Perborates: Retrieved 2019: https://www.nicnas.gov.au

3. HERA Risk Assessment of Sodium Perborate mono- and tetrahydrate. Retrieved 2019: https://www.heraproject.com/files/7-F-04-HERA%20sodium%20perborate%20full%20web%20wd.pdf
4. Hazardous Chemical Information System (HCIS), http://hcis.safeworkaustralia.gov.au/

Appendix G

Toxicological Profiles for Drilling Fluids

AECOM Imagine it. Delivered.

Toxicity Summary - Sodium Erythorbate

Chemical and Physical Properties[1,2]	
CAS number	6381-77-7
Molecular formula	C6H7NaO6
Molecular weight	199.13
Solubility in water	Soluble; 146 g/L at 20 °C and pH 6
Melting point	160 °C at 101.3 kPa
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	Non-flammable (100%)
Colour/Form	White, free-flowing crystals
Overview	Sodium erythorbate is a synthetic antioxidant used in food and cosmetic formulations. Foliar application of sodium erythorbate sprays and dusts are used to control young tree decline in citrus trees and to reduce ozone damage to Thompson seedless grapes. It is also used in hydraulic fracturing mixtures to prevent precipitation of metal oxides (iron control). This chemical has been identified by NICNAS to be of low concern to human health based on an initial screening approach and thus required no further assessment.
Environmental Fate[1]	
Soil/Water/Air	The chemical is not expected to be readily biodegradable. The chemical achieved 56% degradation in 28 days according to test guidelines OECD 301E. However, the degradation after 28 d was not yet finished as a plateau is not yet visible in the degradation curve; thus, a further degradation of the product seems to be possible.
Human Health Toxicity Summary[1]	
Chronic Repeated Dose Toxicity	Male 6-week-old F344 rats were given doses of 5% Sodium Erythorbate in feed for 168 days. Parameters of urinary excretion were investigated and the urinary bladder epithelium was examined using light and scanning electron microscopy at weeks 8, 16, and 24. The urine of rats fed Sodium Erythorbate had increased pH, elevated content of crystals and sodium, and decreased osmolality; however, no morphological alterations such as hyperplasia were detected in the mucosa. The urine values and urinary bladder mucosa were similar to controls at doses below 5 g/kg/day.

Carcinogenicity	F344/DuCrj rats of both sexes (6-week-old) were given 1.25% or 2.5% Sodium Erythorbate in drinking water for 104 weeks and untreated water for 8 additional weeks. Rats of the control group were given untreated water only. Each group consisted of 52 male and 50 female rats. Cumulative consumption of Sodium Erythorbate by male rats was 217 g/rat (1.25%) and 430 g/rat (2.5%). Consumption by females was 206 g/rat (1.25%) and 583 g/rat (2.5%). Body weight of rats given 2.5% Sodium Erythorbate was reduced by 8.5% for males and 15.5% for females at weeks 88 and 85, respectively, compared to controls. Body weight gain was normal in rats of the low dose group. All male treated and control rats (except two of the high-dose group) had testicular interstitial cell tumours. Various tumours occurred in 80% of control males, 69% of males given the low dose, and 78% of males given the high dose. A 6-18% incidence of leukaemia, pheochromocytoma, mammary fibroadenoma, and mesothelioma was observed. Of the females of the control, 1.25%, and 2.5% dose groups, 94%, 88%, and 78% had tumours, respectively. Twenty to 43% of females (all groups) had leukaemia, mammary fibroadenoma, endometrial stromal polyp and/or pituitary adenoma. Females given 2.5% Sodium Erythorbate had significantly fewer tumours than control females. The pattern of occurrence of the various types of tumours was similar among the groups. Sodium Erythorbate did not enhance the development of rare spontaneous tumours or transform benign tumours (e.g., solid adenoma of the thyroid) to carcinomas. The investigators concluded that Sodium Erythorbate was not carcinogenic in F344 rats.
Mutagenicity/ Genotoxicity	Sodium Erythorbate (99.8% pure; 5.0 mg/plate) was non-mutagenic in S. typhimurium strains TA92, TA94, TA98, TA100, TA1535, and TA1537 with and without S9 activation. Sodium Erythorbate (0.25 mg/mL plate) was also negative in the chromosomal aberration assay using Chinese hamster fibroblasts; Sodium Erythorbate did not induce the formation of polyploid cells after 48 hours, and caused 1 % chromosomal breaks after 24 hours.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Sodium erythorbate did not cause maternal or fetal toxicity when administered to female rats and mice during gestation by oral intubation at dosages up to 1,030 mg/kg/day. Developmental toxicity did not occur after pregnant rats were given up to 5% sodium erythorbate in feed during a 13-week teratogenesis study. It produced negative results in the Ames test, the host-mediated assay using S. typhimurium, chromosomal aberration tests using Chinese hamster ovary fibroblasts, the dominant lethal test using rats, and the B. subtilis rec assay.
Acute Toxicity	Sodium erythorbate powder was applied to the intact and abraded skin of six rabbits as a single 2 g/kg dose. A substantial amount of residual compound was observed 24 hours after dosing. No erythema, edema, 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.
Irritation	Sodium erythorbate powder did not cause signs of dermal irritation when applied to the intact and abraded skin of rabbits. Instillation of sodium erythorbate powder to the conjunctival sac of rabbits caused slight and transient reddening of the conjunctiva that cleared within 24 hours.
Sensitisation	In a dermal sensitization study (according to OECD 429) 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). In this study, Sodium erythorbate was not considered a potential skin sensitizer.
Health Effects Summary	Sodium erythorbate did not show signs of toxicity, carcinogenicity, mutagenicity, irritation and sensitisation in the studies reported. This chemical has been identified by NICNAS to be of low concern to human health.
Key Study/Critical Effect for Screening Criteria	The Australian drinking water guideline value for sodium may apply.

Ecological Toxicity [1,2]	
Aquatic Toxicity	The acute toxicity of the sodium erythorbate to the freshwater fish rainbow trout (Oncorhynchus myldss) has been investigated and gave a 96-Hour LC50 of greater than 100 mg/L (semi-static). The acute toxicity of sodium erythorbate to Daphnia magna gave an EC50 (48 h) of 84 - 100 mg/L. The effect of the test item on the growth of Pseudokirchneriella subcapitata has been investigated over a 72-Hour period. The EC50 (72 h) was 160 mg/L while the NOEC (72 h) was 20 mg/L.
Determination of PNEC aquatic	A PNECaquatic of 84 µg/L was calculated using the lowest endpoint of EC50 of 84 mg/L for Daphnia magna. An assessment factor of 1000 was used.
Current Regulatory Controls[4]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	Could potentially be persistent as it is not readily biodegradable.
B/vB criteria fulfilled?	No. The Log Pow is -3.29 (Log Pow < 4.5) which does not meet the screening criteria for bioaccumulation.
T criteria fulfilled?	No. Based on measured acute toxicity endpoints of greater than 1 mg/L Sodium erythorbate does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	April 2019

References

1. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2015, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
2. ECHA REACH, 2,3-didehydro-3-O-sodio-D-erythro-hexono-1,4-lactone, Retrieved 2019: https://echa.europa.eu/
3. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme

AECOM Imagine it. Delivered.

Toxicity Summary - Starch

Chemical and Physical Properties[1,2,4,6]	
CAS number	9005-25-8
Molecular formula	(C6H10O5)n
Molecular weight	UVCB
Solubility in water	In cold water, starch absorbs water reversibly and swells slightly. In hot water, irreversible swelling occurs, producing gelatisation.
Melting point	No data available.
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	Combustible
Flammability potential	No data available.
Colour/Form	White powder, tasteless and has no smell
Overview	Starch is a high –polymeric carbohydrate material primarily composed of amylopectin and amylose. It is usually derived from cereal grains such as corn, wheat and sorghum and from roots and tubers such as potatoes and tapioca. It includes starch which has been pregelatinized by heating in the presence of water. This chemical has been identified by NICNAS to be of low concern to human health and thus required no further assessment.
Environmental Fate[7]	
Soil/Water/Air	Based on information from NICNAS (2006): In a ready biodegradation test, the notified polymer (Potato Starch Modified) showed an 86.87% degradation during a Modified Sturm Test (OECD Test Guideline 301B) indicating that it was readily biodegradable. The test was verified using a sodium benzoate standard which showed 93.77% degradation at the end of the study. In addition a toxicity control consisting of a mixture of the test substance and sodium benzoate showed 83.49% degradation at the end of the study period, indicating that the test material did not inhibit the microbial activity. The notified polymer does potentially contain cationic and anionic functional groups, however based on the typical dissociation constants for the functionalities and their ratio within the polymer it is expected to have a net anionic charge throughout most of the environmental pH range, becoming slightly cationic only at the low end of the range. In landfill and the sewer, the notified chemical is expected to be relatively readily degraded by biotic and abiotic pathways to ultimately yield water and oxides of carbon and nitrogen and salts of chlorine and sodium. Any incineration of the notified polymer would result in its destruction and the formation of carbon dioxide and water and ash containing salts of chlorine and sodium. The notified polymer has a high molecular weight not expected to bioaccumulate.

Human Health Toxicity Summary[2,3]	
Chronic Repeated Dose Toxicity	A long-term study was carried out on the effects of inoculating 1.5 g of starch powder into the peritoneal cavity of rats. After an initial considerable inflammatory reaction, the intense vascular reaction subsided, leaving firm adhesions that were still present in animals sacrificed at 18 months (Ell90). Feeding of unmodified cornstarch and potato starch to groups of rats at dietary levels up to 30% (equivalent to 27.4-33.6 g/kg bw/d) in a 2-year test and 10% (food intake not indicated) in a 3-generation test did not result in distinct toxicologically significant effects (Gro74). Rats fed a cooked diet containing 62% unmodified maize starch (equivalent to 51.1 g/kg bw/d*) for 2 years also did not show significant toxicological effects, including reproductive effects over 3 generations (Tru79). Slight growth retardation was seen in rats exposed for 4 weeks to raw potato starch at a dietary level of 40% (equivalent to 46.0-52.8 g/kg bw/d) (Fer73).
Carcinogenicity	Not classifiable as a human carcinogen (A4)
Mutagenicity/ Genotoxicity	There were no indications for significant toxicity, carcinogenicity or reproduction toxicity of starch in rats fed 27.4-52.8 g/kg bw/day.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	There were no indications for significant toxicity, carcinogenicity or reproduction toxicity of starch in rats fed 27.4-52.8 g/kg bw/day.
Acute Toxicity	Toxicity of starch given orally to albino rats were studied. Experiments were performed upon CBL albino rats weighing 140 ± 15 (mean ± S.D.). Starch was given daily for 14 consecutive days in total daily oral doses of 36, 72, 120 and 168 g/kg and in animals which did not succumb to gastric rupture in doses gradually increasing to 204, 240 and 288 g/kg. Gastric rupture appeared in 50 – 75% of rats given starch in amounts of 168 g/kg and over. Of the survivors of gastric rupture, 5% died of pneumonia and 20% from bowel obstruction during the 14 days of starch administration. Doses of one tenth body weight and above produced some inhibition of growth, doses of one fifth body weight and above increased the susceptibility to pneumonia and bowel obstruction owing to the inability of the animal to evacuate the starch calculi. It was noted that doses of this order could not readily be taken by humans and smaller doses had insignificant toxicity. Acute respiratory effects after exposure to dust from the refining process of potato starch have been described (personal sampling: 3.9-56.0 mg/m^3, total dust). The responsible agent could not be identified although the authors suspected endotoxin to be the causative agent (Hol94). Millers and bakers occupationally exposed to grain and flour dusts (personal sampling: 1.1-14.3 mg/m^3, total dust) showed significantly higher incidences of coughing and chronic bronchitis compared to a non-exposed reference group (Mas95, Mas96). A dose-response relationship was observed between dust exposure levels and chronic respiratory symptoms (Mas95). Although flour is a complex product that is mainly made up of starch (70%) and gluten (12%), it may also contain mite dust and endotoxins. The causative role of starch in the observed respiratory symptoms is therefore not clear. The intraperitoneal LD50 of starch in mice is 6600 mg/kg (ACG99).
Irritation	Skin contact with a total dose of 300 µg of starch, intermittently applied over a 3-day period, resulted in a mild erythema and slight oedema of the skin in humans (ACG99).
Sensitisation	No data available.
Health Effects Summary	This chemical has been identified by NICNAS to be of low concern to human health.
Key Study/Critical Effect for Screening Criteria	The intraperitoneal LD50 of starch in mice is 6600 mg/kg (ACG99).

Ecological Toxicity[7]	
Aquatic Toxicity	Based on QSAR modelling: Crassostrea virginica 96 h = 1000 mg/L Orthopristis chrysoptera 96 h = 5000 mg/L Bairdiella chrysoura 96 h = 5000 mg/L
Determination of PNEC aquatic	Based on the lack of ecotoxicity data, PNECaquatic was not determined.
Current Regulatory Controls[2,4]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	TWA = 10 mg/m^3
International Occupational Exposure Standards	TLV: 10 mg/m^3, as TWA The current administrative occupational exposure limit (MAC) for starch in the Netherlands is 10 mg/m^3, 8-hour TWA, equal to the occupational exposure limit for nuisance dust.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. This substance is expected to be readily biodegradable.
B/vB criteria fulfilled?	No. This substance is not expected to be bioaccumulative.
T criteria fulfilled?	Based on QSAR modelling, this substance is not expected to meet the toxicity criteria.
Overall conclusion	Not PBT
Revised	April 2019

References

1. ECHA REACH, Starch, Retrieved 2019: https://echa.europa.eu/
2. IPCS Starch, Retrieved 2019: http://www.inchem.org
3. Boyd, E. M., & Liu, S. J. (1968). Toxicity of starch administered by mouth. Canadian Medical Association journal, 98(10), 492–499.
4. Health Council of the Netherlands: Committee on Updating of Occupational Exposure Limits. Starch; Health-based Reassessment of Administrative Occupational Exposure Limits. The Hague: Health Council of the Netherlands, 2002; 2000/15OSH/038.
5. Safe Work Australia, Hazardous Substances System, Starch, Retrieved 2019: http://hcis.safeworkaustralia.gov.au/
6. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.nicnas.gov.au
7. Daugherty,F.M.J Sewage Ind. Wastes 23(10):1282-1286. Effects of Some Chemicals Used in Oil Well Drilling on Marine Animals
8. NICNAS (2006) Potato Starch Modified, Full Public Report, File No PLC/639

AECOM Imagine it. Delivered.

Toxicity Summary - Tetrahydro-3,5-dimethyl-1,3,5-thiadiazine-2-thione

Chemical and Physical Properties[1,2,3,5]	
CAS number	533-74-4
Molecular formula	C5H10N2S2
Molecular weight	162.28
Solubility in water	3.5 g/l at 20 °C at pH 5, pH 7and pH 9
Melting point	103.2 – 105.2 °C
Boiling point	No data available.
Vapour pressure	5.8 x 10-6 Pa at 20 °C (extrapolated)
Henrys law constant	2.66X10-10 atm-cu m/mole
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	Off-white to yellowish solid of sulphurous odour
Overview	Dazomet (Tetrahydro-3,5-dimethyl-1,3,5-thiadiazine-2-thione) is a soil fumigant effective for the control of nematodes, insects, germinating weeds and soil fungi. Dazomet is strongly phytotoxic, acting by virtue of the chemical release of methylisothiocyanate (MITC).
Environmental Fate[1]	
Soil/Water/Air	Dazomet's production may result in its release to the environment through various waste streams; its use as a soil sterilant, nematicide, fungicide, slimicide in pulp and paper manufacture, and as a preservative in adhesives and glues will result in its direct release to the environment. If released to air, a vapour pressure of 2.80X10-6 mm Hg at 20 deg C indicates dazomet will exist in both the vapour and particulate phases in the atmosphere. Vapour-phase dazomet will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 1.4 hours. Particulate-phase dazomet will be removed from the atmosphere by wet or dry deposition; hydrolysis of this compound during rain events or in clouds may occur. It has been suggested that dazomet may also undergo direct photolytic degradation and this process may contribute to atmospheric removal. If released to soil, dazomet is expected to have high mobility based upon an estimated Koc of 52; however it is expected to hydrolyse before extensive leaching occurs. Volatilization from moist soil surfaces is not expected to be an important fate process based upon a Henry's Law constant of 2.66X10-10 atm-cu m/mole. When dazomet is applied to soil, either to the surface or incorporated, it quickly hydrolyzes in the presence of moisture. The major degradate is methyl isothiocyanate, but formaldehyde, monomethylamine, hydrogen sulfide and (in acid soils) carbon disulfide, are also formed. The half-life of dazomet in soil has been reported as less than 1 day (pH >5). The rate of disappearance was found to be the same in both unamended and sterilized soils and in different soil types, indicating that chemical hydrolysis and not biodegradation is the primary removal process. Dazomet is not expected to volatilize from dry soil surfaces based upon its vapour pressure. If released into water, dazomet is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilization from water surfaces is not expected to be an important fate process based upon this compound's Henry's Law constant. In water, dazomet is expected to undergo hydrolysis rapidly, forming methyl isothiocyanate and formaldehyde. Half-lives of 3.6, 2.4, 2.8, and 4.0 hours have been reported at pH values of 4.4, 5.7, 7.0, and 8.0, respectively. In salt water (0.15 M), a half-life of 6.1 hours was reported. An estimated BCF of 2.4 suggests the potential for bioconcentration in aquatic organisms is low.

Human Health Toxicity Summary[1]	
Chronic Repeated Dose Toxicity	In a 78 week study, mice were given dazomet in the diet at 0, 20, 80 and 320 ppm. Compound intakes were estimated as follows: males - 0, 4, 16 and 68 mg/kg/d; females - 0, 6, 22 and 93 mg/kg/d. Survival was not affected and there were no noteworthy clinical signs, or bodyweight or food consumption changes. There was a significant elevation of liver weight at the high dose and an increased number of mid-dose and high dose animals with liver discolouration, liver masses and centrilobular lipid deposition. At the high dose, females showed a slightly increased incidence of hepatocellular adenomas (3, 0, 1 and 7 females, out of 50, in the control, low dose, mid dose and high dose groups, respectively) and a significantly increased incidence of basophilic foci. Increased splenic haemosiderin deposition and extramedullary haematopoiesis were noted at the mid dose (males) and high dose. Three/60 females from each dose group had malignant lymphoma at one or more sites; because of the low incidence, lack of a dose-response, and lack of any effect in males, it was not considered to be directly compound-related. The NOEL was 20 ppm (about 4 mg/kg/d in males, 6 mg/kg/d in females).
Carcinogenicity	Rat studies showed no clear evidence of any carcinogenic effect of dazomet. In mice, there was a slight increase in hepatocellular adenomas (not carcinomas) following 78 weeks of treatment at the high dose (320 ppm). There was also an increase in malignant lymphoma in females, but because of the low incidence, the lack of effect in males and the lack of any dose-response, it was not considered to be directly compound-related. The lack of a carcinogenic effect of dazomet is consistent with the data for MITC.
Mutagenicity/ Genotoxicity	An acceptable package of mutagenicity tests has been conducted covering all three end points. The results are the genotoxicity tests are not clear cut. While the majority of tests gave negative results, there were sufficient positive results to indicate some genotoxic potential of dazomet. In summary, there were positive results in one gene mutation assay (HGPRT locus in Chinese hamster ovary cells), equivocal results in another gene mutation assay (TK locus in mouse lymphoma L5178Y cells), and positive results in two chromosome aberration assays (both in vitro assays in mouse lymphoma L5178Y cells), in one in vitro assay for of unscheduled DNA synthesis in primary rat hepatocytes and in one in vitro assay of sister chromatid exchange. In all cases, the positive findings were relatively weak. There were no positive in vivo studies and there was a trend for results to only be positive (or to be stronger) in the absence of metabolic activation than in its presence. This suggests that unchanged dazomet has greater genotoxic potential than the metabolites of dazomet. The unscheduled DNA synthesis assay was the only assay which gave results suggesting that the metabolites of dazomet may have some genotoxic potential, even if only weak.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Dazomet was fed to rats at 0, 5, 30 and 180 ppm for at least 70 days prior to mating, throughout mating and lactation, during production of F_1a and F_1b litters. Selected F_1a pups were maintained on compound-containing diets post-weaning to produce F_2 litters. Hepatotoxicity was observed in both generations, mainly at the high dose, but to some extent at the mid dose. Liver weights were increased and there was an increased severity of liver fatty change. Some serum enzyme and serum protein changes also indicated effects on the liver. There was no impairment of mating or reproductive performance and no adverse effect on reproductive organs or pup development. The NOEL with respect to reproductive function in rats was 180 ppm (about 18 mg/kg/d), while that for systemic toxicity was 5 ppm (about 0.5 mg/kg/d). An oral (gavage) developmental study was conducted in rats at dazomet doses of 0, 3, 10 and 30 mg/kg/d. Food intake and body weight and also uterine weights were reduced at the high dose and to a lesser extent at the mid dose. There was a higher incidence of runts at 10 mg/kg and above, however, without a clear dose-response relationship. There was no evidence of teratogenic effects. The NOEL for maternal and foetal effects was 3 mg/kg/d.
Acute Toxicity	Dazomet is of moderate acute oral toxicity. The oral LD50 values for dazomet from two different studies in rats were about 600 - 900 mg/kg for males and 400 - 550 mg/kg for females. The LD50 of dazomet, given subcutaneously to mice, was 248 mg/kg. The LD50 of dazomet, given subcutaneously to rats, was 470 and 550 mg/kg in males and females, respectively. The dermal LD50 of dazomet in rats was greater than 2000 mg/kg. Symptoms associated with acute dazomet toxicity were shaking, salivation, tonic convulsions, trembling, dyspnoea and lassitude.

Irritation	In two studies, the introduction of 39 or 50 mg dazomet into the eye of rabbits caused slight irritation (moderate conjunctival erythema and slight oedema). Results of two acute dermal irritation studies employing 50% aqueous preparations of dazomet in rabbits were reported. No irritation was observed in the study employing a 4 h exposure period. After a 20 h exposure period, moderate erythema and oedema were observed. Application of the EUP, Basamid Granular (2 g coated on a cottonwool carrier), to the rabbit ear for 20 h caused slight inflammation.
Sensitisation	Skin sensitisation was not observed in two studies following the application of dazomet or Basamid Granular to the guinea pig. No justification was given for the doses / concentrations used in one of these studies and positive control compounds were not tested in these studies.
Health Effects Summary	Dazomet has moderate to low acute oral, dermal and inhalational toxicity. It appears that the toxicity of dazomet is somewhat greater by the oral route than by the dermal and inhalational routes. Dazomet is only a slight dermal and ocular irritant.
Key Study/Critical Effect for Screening Criteria	An ADI of 0.005 mg/kg/d is calculated based on a NOEL of 0.5 mg/kg (established in a 1-year dietary dog study and a 2-year dietary rat reproductive study) and a safety factor of 100.
Ecological Toxicity [1,2,3]	
Aquatic Toxicity	Daphnia magna (Water flea), 48 h, static, EC50 = 0.3 mg/L Salmo gairdneri (Rainbow trout), 96 h, static, LC50 = 0.16 mg/L Ankistrodesmus bribaianus (Green alga), 72 h, static, EC50 = 1.08 mg/L Colinus virginianus (Bobwhite quail), 21 d, LD50 = 415 mg/kg bw Colinus virginianus (Bobwhite quail), 25 weeks, NOEL = 100 mg/kg food
Determination of PNEC aquatic	An assessment factor of 10 has been applied to the lowest reported LC50 of 0.16 mg/L for Rainbow trout. The PNECaquatic is 0.016 mg/L.
Current Regulatory Controls[4]	
Australian Hazard Classification	Acute toxicity – category 4 Eye irritation – category 2 Hazardous to the aquatic environment (acute) – category 1 Hazardous to the aquatic environment (chronic) – category 1
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1,3,5]	
P/vP Criteria fulfilled?	The half-life of dazomet in soil has been reported as less than 1 day (half-life in soil < 6 months). Thus, it is not expected to be persistent.
B/vB criteria fulfilled?	As the Log Pow is 0.63 at 20 °C (Log Pow < 4.5) and estimated BCF is 2.4, it is not expected to be bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of this chemical is >0.01 mg/L. Thus, it is not expected to meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	April 2019

References

1. National Registration Authority for Agricultural and Veterinary Chemicals, NRA Special Review of Metham Sodium, Dazomet and Methylisothiocyanate (MITC) Volume II, June 1997
2. ECHA REACH, 1-Butanol, Retrieved 2019: https://echa.europa.eu/
3. Food and Agriculture Organization of The United Nations (2001), FAO Specifications and Evaluations for Plant Protection Prducts, Dazoment
4. Safe Work Australia, Hazardous Substances System, Dazomet, Retrieved 2019: http://hcis.safeworkaustralia.gov.au/
5. HSDB (n.d.). Hazardous Substances Data Bank. Retrieved 2019, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

Toxicity Summary - Trisodium Nitrilotriacetate

Chemical and Physical Properties[1,2,3]	
CAS number	5064-31-3
Molecular formula	C6H9NO6.3Na
Molecular weight	257.0
Solubility in water	640 g/l at 20 °C
Melting point	410 °C with decomposition above 200 °C
Boiling point	No data available
Vapour pressure	No data available
Henrys law constant	No data available
Explosive potential	Non-explosive (100%)
Flammability potential	Non-flammable (100%)
Colour/Form	colourless crystalline powder
Overview	The chemicals in this group are known as nitrilotriacetic acid (NTA) and its trisodium and tripotassium salts, trisodium nitrilotriacetate (trisodium NTA) and tripotassium nitrilotriacetate (tripotassium NTA). The trisodium salt also occurs as its monohydrate form (trisodium nitrilotriacetate monohydrate; CAS No. 18662-53-8). The chemical NTA is an aminocarboxylic acid with three functional carboxylate groups. The chemical forms water-soluble complexes with multivalent metal ions. The chemical NTA and trisodium NTA dissociate to form a common moiety, nitrilotriacetate ion. Thus the systemic toxicity of these chemicals is similar (Health Canada, 2010; SCCS 2010). Tripotassium NTA is considered to be functionally similar to trisodium NTA. The chemicals, NTA and trisodium NTA are used to soften water and to remove traces of heavy metals. These chemicals are commonly used as chelating and sequestering agents, and as builders in detergent and cleaning formulations for domestic and commercial use (EU RAR, 2008; SCCS, 2010).
Environmental Fate[1]	
Soil/Water/Air	Trisodium NTA was tested for ready biodegradability according to OECD 301 E (BASF, 1983b,c), OECD 301 F (in addition to a combined CO2/DOC test, see Strotmann et al., 1995), and Sturm Test (BASF, 1983d), and in a die away test (Takahashi et al, 1997) as well as for inherent biodegradability according to OECD 302 B (BASF, 1983a). These tests resulted in 75 -100 % degradation after 7 to 28 days with lag phases ranging between 1 and 16 days. According to results from ready biodegradation tests, trisodium NTA can be regarded as readily biodegradable. In accordance with column 2 of REACH Annex IX, trisodium NTA has a log octanol-water partition coefficient of -13.2 at pH 7, is highly water-soluble, and is unlikely, due to its polar nature, to be taken up by fish gills or across other biological membranes. Due to the ionic structure of the substance a relevant adsorption of trisodium NTA onto the organic fraction of soils, sediments or suspended solids is not expected. However, interaction with the mineral phase may be possible. This assumption is in line with available study results (Dunlap et al., 1971; Bolton et al., 1993) which demonstrate that trisodium NTA is neither strongly sorbed by loam, clay-loam and sandy soils or marine surface sediments (Kp sediment-water = 1.6 l/kg).

Human Health Toxicity Summary[1]	
Chronic Repeated Dose Toxicity	The available data suggest that the chemicals have harmful effects following repeated oral dosing, based on results from animal tests. However, the effects were not sufficient to warrant hazard classification. In a 4-week study, Charles River and Fischer 344 (F344/N) (five or ten animals/group) rats were fed either 0 % or 1.5 % NTA in the diet. Effects observed included reduced growth, increased relative kidney weight, urinary calcium, haematuria and hydronephrosis. A lowest observed adverse effect level (LOAEL) of 1.5 % NTA (equivalent to 750 mg/kg bw/day) was reported (EU RAR, 2008; Health Canada, 2010). In a 10-week study in male Sprague Dawley (SD) rats, trisodium NTA was administered to the rats in drinking water at 0 %, 0.01 %, 0.1 % or 1 % (equivalent to 0, 10, 100 or 1000 mg/kg bw/day). Increased kidney weights were observed in the rats treated at 0.1 % (100 mg/kg bw/day) and marked vacuolisation of the renal tubules was observed at 1 % trisodium NTA (1000 mg/kg bw/day dose) group. A LOAEL of 100 mg/kg bw/day (0.1 % trisodium NTA) was reported (EU RAR, 2008; Health Canada, 2010; SCCS, 2010). Trisodium NTA was administered to male SD rats by gavage at 0, 0.73 or 7.3 mmol/day (equivalent to 0, 187 or 1876 mg/kg bw/day) for 30 days. Cytoplasmic vacuolisation, focal haemorrhage, necrosis, erosion and hyperplasia of the epithelium of the proximal convoluted tubules were observed in all treated animals. An oral LOAEL of 0.73 mmol/day (187 mg/kg bw/day) was reported (EU RAR, 2008; Health Canada, 2010; SCCS, 2010; REACHb). In a 90-day study in rats (strain not reported), NTA was administered to male rats at 0, 100, 1000 or 5000 mg/L in drinking water. All treated animals showed reduced serum potassium levels (EU RAR, 2008; Health Canada, 2010). In two different studies (28-days and 91-days), New Zealand White (NZW) rabbits (six/group) were treated with either 0 or 2.5 % trisodium NTA on intact or abraded skin. No treatment-related effects were observed with or without abrasion (EU RAR, 2008; Health Canada, 2010; SCCS, 2010; REACHa & b). In a 4-week repeated dose inhalation toxicity study, NTA was administered in SD rats, trueblood albino guinea pigs and cynomolgus monkeys at 0, 10, 213 or 343 mg/m^3 concentrations for 6 hours/day by whole body exposure. No respiratory irritation or discomfort was observed at the highest tested concentration. The only treatment-related effects included diarrhoea in monkeys and dyspnoea in rats and guinea pigs. The no observed adverse effect concentration (NOAEC) of 213 mg/m^3 and the lowest observed adverse effect concentration (LOAEC) of 343 mg/m^3 were reported (EU RAR, 2008; Health Canada, 2010; REACHa & b). In another study, male albino rats were treated with NTA at 0, 2, 20, 200 or 2000 mg/m^3 concentrations for 6 hours/day for four consecutive days by inhalation exposure. All animals in the 2000 mg/m^3 showed signs of nasal, respiratory and eye irritation, which were fully reversed on day 14 (EU RAR, 2008; Health Canada, 2010).

Carcinogenicity	Trisodium NTA is classified as hazardous with hazard catergory 'Carcinogenicity – Category 2' and hazard statement 'Suspected of causing cancer' (H351) in the HCIS (Safe Work Australia). The available data support the classification for trisodium NTA. Additionally, the classification for carcinogenicity is considered appropriate for NTA. The International Agency for Research on Cancer (IARC) has classified NTA and its salts as 'Possibly carcinogenic to humans' (Group 2B), based on inadequate evidence for carcinogenicity in humans, but sufficient evidence for carcinogenicity in animal tests (IARC, 1990; IARC, 1995). In two-year carcinogenicity studies in Charles River (CD) rats and B6C3F1 mice, oral administration of Na3NTA induced benign and malignant tumours of the urinary system in both male and female rats at 80–100 mg/kg bw/day and haematopoietic tumours in male mice at 500–600 mg/kg bw. Trisodium NTA was reported to induce renal tubular adenomas and adenocarcinomas in male rats when administered orally (IARC, 1990; IARC, 1995; EU RAR, 2008; Health Canada, 2010; SCCS, 2010; REACHa & b).
Mutagenicity/ Genotoxicity	Based on the weight of evidence from the available in vitro and in vivo genotoxicity studies, the chemicals are not considered to be genotoxic. Several in vitro and in vivo micronucleus tests for gene mutation and clastogenicity were negative, although several positive results were reported (IARC, 1990; IARC, 1995; EU RAR, 2008; Health Canada, 2010; SCCS, 2010; REACHa & b).
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Based on the available information, the chemicals do not cause specific reproductive or developmental toxicity. In different two-generation reproductive and developmental toxicity studies, oral administration of up to 0.5 % trisodium NTA (equivalent to 450 mg/kg bw/day) in the diet of Charles River rats, up to 250 mg/kg bw/day trisodium NTA by gavage in pregnant NZW rabbits, and up to 0.2 % NTA (equivalent to 570 mg/kg bw/day) in drinking water in Naval Medical Research Institute (NMRI) mice, caused no significant maternal, embryonic or foetal effects. No effect on neonatal development was seen in any of the above studies (NTP, 1977; IARC, 1995; EU RAR, 2008; Health Canada, 2010; SCCS, 2010; HSDB; REACHa & b). In a developmental study, female NZW rabbits (groups of 20) were treated by gavage with trisodium NTA in drinking water at 0, 2.5, 25, 100 or 250 mg/kg bw/day during gestation days 7–16. All animals were sacrificed on day 28 of gestation. No treatment-related effects were observed (EU RAR, 2008; Health Canada, 2010; SCCS, 2010; REACHa & b). A study was conducted in pregnant NMRI albino mice (10 animals/group) treated with 0 or 0.2 % trisodium NTA (equivalent to 0 or 570 mg/kg bw/day) in drinking water on 6–18 days of gestation. No significant differences in maternal weight gains and no developmental effects were observed (EU RAR, 2008; Health Canada, 2010; SCCS, 2010; REACHa & b).

Acute Toxicity	Trisodium NTA is classified as hazardous with hazard category 'Acute Toxicity – category 4' and hazard statement 'Harmful if swallowed' (H302) in the HCIS (Safe Work Australia). The available data (median lethal dose—LD50 of 1470 mg/kg bw in female rats and 750 mg/kg bw in monkeys) support this classification. Reported signs of toxicity include ataxia, tremors, hypopnoea, hypothermia, hypoactivity, prostration, staggering, twitching, opisthotonus, tonic convulsion, apathy, salivation and dyspnoea. Available data for NTA indicate an LD50 >6400 mg/kg in rats. The chemicals have low acute toxicity based on results from an animal test in rabbits following dermal exposure. In an acute dermal toxicity study, a 25 % aqueous solution of trisodium NTA monohydrate was applied occlusively to intact skin of rabbits (one animal/sex/dose) at 1000, 1580, 2510, 3980, 6310 or 10000 mg/kg bw. Mild muscle weakness and reduction in activity and appetite were seen in the higher dose groups. No local symptoms or muscular uncoordination were reported. An LD50 of >10,000 mg/kg bw was reported (EU RAR, 2008; REACHa & b). The chemicals have low acute toxicity based on results from animal tests following inhalation exposure. A median lethal concentration (LC50) in rats of >5.0 mg/L was reported for NTA (EU RAR, 2008; Health Canada, 2010; SCCS, 2010).
Irritation	Trisodium NTA is slightly irritating to the animal skin. The effects were not sufficient to warrant a hazard classification. Trisodium NTA is classified as hazardous with hazard category 'Eye Irritation – category 2A' and hazard statement 'Causes serious eye irritation' (H319) in HCIS (Safe Work Australia). The available data support this classification. In an eye irritation study in rabbits, trisodium NTA was found to be irritating. Conjunctivitis and marked corneal effects were observed at 24, 48 and 72 hours after application (ECHA, 2006). Effects were not reversible within the 7-day period. In a study, albino rabbits had considerable discomfort immediately after application of 100 mg of trisodium NTA monohydrate. Effects observed one hour after application included copious discharge, oedema with partial eversion of the lids, moderate redness and congestion with obscure iris. Discharge and oedema reduced on washing the eyes with saline solution after 24 hours. Complete reversal oedema occurred but mild redness and slight corneal dullness were observed on days 5 to 7 (EU RAR, 2008; Health Canada, 2010; SCCS, 2010; REACHb). In another study conducted according to OECD Test Guideline (TG) 405, trisodium NTA (0.1 mL of 38 % solution) applied to the conjunctival sac of three albino rabbits caused slight eye irritation. The average scores for conjunctival redness and chemosis after 24 hours were 2.0 and 0.7, respectively. The conjunctival redness score was 0.1 after 48 hours and no chemosis was present. The conjunctival redness was reversible within 8 days after application. No effects on the cornea and iris were reported (EU RAR, 2008; Health Canada, 2010; SCCS, 2010; REACHb).
Sensitisation	Based on the available data, the chemicals are not considered to be skin sensitisers.
Health Effects Summary	The critical health effects for risk characterisation include systemic long-term effects (carcinogenicity) for all three chemicals, and systemic acute effects (acute toxicity from oral exposure) and local effects (eye irritation) for trisodium NTA and tripotassium NTA only.
Key Study/Critical Effect for Screening Criteria	The Australian Drinking Water Guideline for NTA is 0.2 mg/L.

Ecological Toxicity[4]	
Aquatic Toxicity	Tests on acute toxicity to fish resulted in 96-hour LC50 values in the range of 98 – 487 mg/l. In a generation-cycle test over 224 days on Pimephales promelas (Arthur et al., 1974), there were no observable differences in survival, spawning activity, and egg hatchability at the highest tested concentration of 54 mg/l trisodium NTA (the active test substance was Ca- or Mg-NTA). Based in this study, the NOEC for fish is determined to 54 mg/L. All tests on acute toxicity to invertebrates showed effects only when the trisodium NTA concentration exceeded the stoichiometric metal levels of the medium. It is expected that effects are caused by the uncomplexed agent. This is supported by the increased effect values in hard water. In long-term tests, the most sensitive organism was the amphipod Gammarus pseudo limnaeus. In a generation-cycle test over 21 weeks exposure, the lowest tested concentration without significant effects was 9.3 mg/l trisodium NTA. Based in this study, the NOEC for invertebrates is determined to 9.3 mg/l. At this concentration, NTA is mainly complexed with Ca and Mg.
Determination of PNEC aquatic	Reliable long-term data was available for a fish, invertebrate and algae. The lowest NOEC of 9.3 mg/L was a result for testing with Gammarus pseudolimnaeus (Arthur et al. 1974). An assessment factor of 10 was used for a resulting PNEC for intermittent releases of 0.93 mg/L.
Current Regulatory Controls[1]	
Australian Hazard Classification	Trisodium NTA is classified as hazardous, with the following risk phrases for human health in the Hazardous Chemicals Information System (HCIS) (Safe Work Australia): Acute toxicity – category 4; H302 (Harmful if swallowed) Eye irritation – category 2; H319 (Causes serious eye irritation) Carcinogenicity – category 2; H351 (Suspected of causing cancer).
Australian Occupational Exposure Standards	The chemical NTA has an Australian drinking water guideline value of 0.2 mg/L (NHMRC, 2011; FSANZ).
International Occupational Exposure Standards	The following exposure standards are identified (Galleria Chemica; Protective Action Criteria (PAC)): Temporary Emergency exposure limits (TEELs) defined by the US Department of Energy (DOE): TEEL-1= 3.7 - 9.2 mg/m^3; TEEL-2= 40 - 100 mg/m^3; TEEL-3= 220 - 110 mg/m^3.
Australian Food Standards	The chemical NTA has an Australian drinking water guideline value of 0.2 mg/L (NHMRC, 2011; FSANZ).
Australian Drinking Water Guidelines	The chemical NTA has an Australian drinking water guideline value of 0.2 mg/L (NHMRC, 2011; FSANZ).
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	NTA is readily biodegradable and as such not persistent in the environment.
B/vB criteria fulfilled?	Trisodium NTA has a log octanol-water partition coefficient of -13.2 at pH 7, is highly water-soluble. Thus, it is not expected to be bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of NTA is > 0.01 mg/L. Hence the substance does not fulfil the screening criteria for toxic (T)

Overall conclusion	Not PBT
Revised	March 2019

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier II Assessment for Nitrilotriacetic acid and salts: Retrieved 2019: https://www.nicnas.gov.au
2. ECHA REACH, Trisodium nitrilotriacetate, Retrieved 2019: https://echa.europa.eu/
3. HSDB (n.d.). Hazardous Substances Data Bank. Retrieved 2019, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

AECOM Imagine it. Delivered.

Toxicity Summary - Xanthan Gum

Chemical and Physical Properties[1,3]	
CAS number	11138-66-2
Molecular formula	Unspecified
Molecular weight	high-molecular weight (of the order of 1000 kDa)
Solubility in water	Water-soluble
Melting point	No data available.
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	No data available.
Overview	Xanthan gum is a high molecular weight anionic polysaccharide secreted by the bacteria *Xanthomonas compestris*. It is used as a stabilizer and thickener for foods, pharmaceuticals, and cosmetics, for rheology control in water-based systems, and in oil and gas drilling. Xanthan gum is used for controlling the viscosity of drilling muds (DoE 2014). This chemical has been identified by NICNAS to be of low concern to human health based on an initial screening approach and thus required no further assessment.
Environmental Fate[1]	
Soil/Water/Air	Xanthan gum is expected to exhibit similar behaviour to that of guar gum because the two compounds are chemically similar. Thus, it is expected to adsorb strongly to soil and sediment and there is limited potential for it to reach surface waters via dissolved runoff and / or to leach into ground water. Volatilisation from soils and water is not considered to be a likely transport process in the environment (US EPA 2005). Xanthan gum is expected to readily undergo microbial biodegradation in the environment (on the bases that it is polysaccharide and expected to be readily biodegradable), and the potential to bioaccumulate in organisms is considered to be low.

Human Health Toxicity Summary[2]	
Chronic Repeated Dose Toxicity	Groups of 30 male and 30 female Charles River CD strain rats were fed diets for 104 weeks supplying O, 0.25, 0.5, or 1.0 g/kg b.w./day xanthan gum. No abnormalities which could be attributed to ingestion of these experimental diets were found with regard to survival, body-weight gain, food consumption, behaviour, or appearance. Ophthalmic and haematologic examination yielded normal results. Analysis of blood for glucose, SGOT, and prothrombin time showed no abnormalities in test groups. Organ weights were within normal limits and no lesions attributable to xanthan gum were found on gross and histopathological examination (Woodard et al., 1973). Xanthan gum was administered in the diet at levels supplying 0, 0.25, 0.37, or 1.0 g/kg b.w./day to groups of 4 male and 4 female beagle dogs for 107 weeks. No effects attributable to administration of the gum were seen in the treated animals with regard to survival, food intake, body-weight gain, electrocardiograms, blood pressure, heart rate, body temperature, or ophthalmic and neurological examinations. Haemoglobin, total and differential white cell counts, coagulation and prothrombin times, thrombocyte counts, serum alkaline phosphatase, blood urea nitrogen, blood glucose, SGOT, and SPGT were the same in control and treated animals. Urine pH, glucose concentrations, and sediment contents were comparable between test and control groups, but there was a dose-related increase in urine SG and a more frequent appearance of urinary albumin in dogs consuming 1.0 g/kg b.w./day of gum than in the other groups. Stool consistency was normal at the 0.37 g/kg level, but stools were loose at the top-dose level. The weight of the faeces showed a dose-related increase, as would be expected from feeding a non-absorbed hydrophilic gum at high-dose levels. The increased urinary SG is consistent with physiological adjustment for the extra water excreted in the faeces. Examination of the appearance and weights of organs and histopathological examinations failed to detect any adverse effects of treatment with xanthan gum at any dose level (Woodward et al.,1973).
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	No data available.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	A three-generation reproduction study was carried out using groups of 10 male and 20 female rats in the first generation and 20 male and 20 female rats in subsequent generations. Dosage levels of 0, 0.25, and 0.5 g/kg/day were administered in the diet. Criteria evaluated were survival, body weight, general appearance, behaviour, the number of litters produced, number of live births and still births, physical condition of the young, weight at birth and weaning, and survival of the young. Females that had fewer than two litters were examined to determine whether there was foetal resorption. Malformations in offspring were recorded and gross and micropathological examinations were made on the offspring of the second and third generations. No adverse effects attributable to xanthan gum were found in this study (Woodard et al., 1973).
Acute Toxicity	A study was carried out on an unspecified number of rats fed diets containing 7.5 or 10% xanthan gum for 99-110 days. No adverse effects were observed in extensive investigatins on these animals (Booth et al., 1963). In a 91-day feeding study, a reduced rate of weight gain was found in groups of rats receiving 7.5 or 15% xanthan gum in the diet. Diets containing 3 or 6% gum did not reduce weight gain. No significant alterations in haemoglobin, red or white cell counts, or organ weights were observed in these rats. Histological examination of tissues from rats at the 15% level showed no pathological effects. At the highest-dose level the animals produced abnormally large faecal pellets, but diarrhoea did not occur. A paired-feeding test was used to compare the growth of rats ingesting a diet containing 7.5% xanthan gum and comparable rats restricted to the same intake of control diet. No differences in weight gain were found at the end of 18 days, indicating the absence of a growth-inhibiting factor (Booth et al.,1963). Groups of 3 male and 3 female beagle dogs were fed diets supplying 0, 0.25, or 0.5 g/kg b.w./day xanthan gum for 12 weeks. Animals in the high-dose group had softer stools than normal, but no diarrhoea. Growth was slightly retarded in the males and the serum cholesterol level was lowered in both sexes of the high-dose group. No other adverse effects were seen. The no-adverse-effect-level in this test was considered to be 0.25 g/kg b.w./day (USDA, 1964).

Irritation	Daily application of a 1% solution for 15 days to rat skin produced no signs of irritation. Daily application of a 1% solution for five days to rabbit conjunctiva produced no signs of irritation.
Sensitisation	Intradermal challenge tests in guinea-pigs did not produce evidence of sensitization (Hendrickson & Booth, sine data).
Health Effects Summary	A mild skin and eye irritant
Key Study/Critical Effect for Screening Criteria	The Joint FAO/WHO Expert Committee on Food Additives allocated an Acceptable Daily Intake (ADI) of "not specified".
Ecological Toxicity [1,2,3]	
Aquatic Toxicity	Acute Fish (measured) = 420 mg/L
Determination of PNEC aquatic	Based on acute fish toxicity of 420 mg/L, an assessment factor of 1000 was used for a resulting PNEC of 0.42 mg/L.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No biodegradation information was found on xanthan gum. However, xantham gum is a naturally occurring polysaccharide which would be expected to readily biodegrade. Thus, it is not expected to meet the screening criteria for persistence
B/vB criteria fulfilled?	Xantham gum is not expected to meet the criteria for bioaccumulation.
T criteria fulfilled?	Not applicable. Acute toxicity data >1 mg/L in fish, thus xanthan gum does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	March 2019

References

1. Department of the Environment and Energy 2017, National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme
2. IPCS INCHEM, Xanthan Gum, Retrieved 2019: http://www.inchem.org/
3. Food and Agriculture Organization of the United Nations (FAO) 2016, 82nd JECFA - Chemical and Technical Assessment (CTA),XANTHAN GUM

AECOM Imagine it. Delivered.

Toxicity Summary - Acrylamide polymers: Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer and Polymer of 2-acrylamido-2- ethylpropanesulfonic acid sodium salt and methyl acrylate

Chemical and Physical Properties [2, 3, 4]	
CAS number	38193-60-1 and 136793-29-8
Molecular formula	38193-60-1: $(C_7H_{13}NO_4S.C_3H_5NO.Na)_x$ 136793-29-8: $C_{11}H_{18}NNaO_6S$
Molecular weight	Likely >1000 MW
Solubility in water	No data available.
Melting point	No data available.
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	No data available.
Overview	No studies are available for the Acrylamide polymers. Information for 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt will be referenced in the following sections. 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salts are generally incorporated into polymers. As such, the fate of the monomer is tied to the polymer and no hydrolysis, movement, biodegradation or bioaccumulation of the polymer is expected. A Tier 1 Human Health Assessment for Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer has been conducted by NICNAS which concluded that this chemical was identified as low concern to human health.
Environmental Fate [2]	
Soil/Water/Air	The polymers are not expected to be readily biodegradable. Biodegradation is limited due to the very high molecular weights and the low water solubilities of the polymers. Due to their high molecular weight, the polymers are not expected to bioaccumulate.
Human Health Toxicity Summary [2]	
Chronic Repeated Dose Toxicity	A repeat dose 28 day oral toxicity study carried out in rats with a 50% aqueous solution of 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt indicated no treatment related toxic effects.
Carcinogenicity	No information available.
Mutagenicity/ Genotoxicity	2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt did not induce a mutagenic response in bacteria and no clastogenicity was observed when a 50% solution of the notified chemical was tested in albino mice cells in vitro.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No data available.

Acute Toxicity	Aqueous solutions containing 50% of 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt exhibited low acute oral and dermal toxicity in rats (LD50 > 5 000 mg/kg, and 2 000 mg/kg respectively).
Irritation	2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt was non-irritant to rabbit skin and a slight eye irritant in rabbits.
Sensitisation	A 50% aqueous solution of 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt showed minimal sensitisation potential when tested in guinea pigs.
Health Effects Summary	This chemical has been identified by NICNAS to be of low concern to human health.
Key Study/Critical Effect for Screening Criteria	No data available
Ecological Toxicity [2]	
Aquatic Toxicity	Limited information is available. The polymers are expected to be a low concern for toxicity to aquatic organisms. Due to their poor solubility and high molecular weights, they are not expected to be bioavailable. The polymers do not contain any reactive functional groups. Ecotoxicity data for 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt indicate that it is practically non-toxic to fish, water fleas and algae.
Determination of PNEC aquatic	No PNEC values were calculated.
Current Regulatory Controls[5]	
Australian Hazard Classification	No data available
Australian Occupational Exposure Standards	No data available
International Occupational Exposure Standards	No data available
Australian Food Standards	No data available
Australian Drinking Water Guidelines	Based on health considerations, the concentration of acrylamide in drinking water should not exceed 0.0002 mg/L.
Aquatic Toxicity Guidelines	No data available
PBT Assessment [1, 2]	
P/vP Criteria fulfilled?	The polymers are not readily biodegradable, hence they meet the screening criteria for persistence.
B/vB criteria fulfilled?	The polymers are expected to have very high molecular weights and poor water solubility. They are not expected to be bioavailable, hence the polymers do not meet the criteria for bioaccumulation.
T criteria fulfilled?	There are no aquatic toxicity studies on the polymers. They are expected to have low aquatic toxicity because of their very high molecular weights and poor water solubility. As such, the polymers do not meet the criteria for toxicity.
Overall conclusion	Not PBT substances
Revised	December 2018

References

1. Categorization Results from the Canadian Domestic Substance List, CAS# 38193-60-1

2. National Industry Chemicals Notification and Assessment Scheme. 2-Acrylamido-2-methylpropanesulfonic acid, ammonium salt, July 1997.
3. U.S National Library of Medicine, Toxicology Data Network, ChemIDplus, CAS#38193-60-1. Accessed 2017 at https://chem.nlm.nih.gov/chemidplus/rn/38193-60-1
4. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2018: https://www.nicnas.gov.au
5. National Health and Medical Research Council, Natural Resources Management Ministerial Council, national Water Quality Management Strategy, Australian Drinking Water Guidelines 6, 2011, Version 3.3 Updated November 2016.

AECOM Imagine it. Delivered.

Toxicity Summary - Potassium chloride

Chemical and Physical Properties [1,2,3,8,9,10]	
CAS number	7447-40-7
Molecular formula	KCl
Molecular weight	74.55 g/mol
Solubility in water	34.20 at 20 ◦C
pH	7
Melting point	771.00 ◦C
Boiling point	1500 ◦C
Vapour pressure	No data found
Henrys law constant	No data found
Explosive potential	Not explosive
Flammability potential	Not flammable
Colour/Form	White crystals or crystalline powder
Overview	Potassium is an essential element in the body. It is the main intracellular cation with 98% of total body potassium located within the cells. It is mainly used in fertilisers, medicine, lethal injections, scientific applications, feedstock, food processing and as a sodium substitute in table salt. Potassium chloride is an essential element with homeostatic physiological processes regulating levels in the body. In cases of increased exposure to high levels of potassium significant health effects in people with kidney disease or other conditions, such as heart disease may result. Potassium chloride as an inorganic salt is not subjected to further degradation processes in the environment once it dissociates into its respective ions. In water, potassium chloride is highly water soluble, and readily undergoes dissociation. In soil, transport and leaching of potassium and chloride ions is affected by the clay minerals (type and content), pH, and organic matter. This chemical has been identified by NICNAS to be of low concern to human health based on an initial screening approach and thus required no further assessment.
Environmental Fate [1,3,8,9]	
Soil/Water/Air	KCl is a solid inorganic salt that is highly soluble in water (342 g/L at 20º C). Potassium chloride fully dissociates in aqueous solutions to K+ and Cl- ions. Cl, either as an inorganic salt or as K+ and Cl- ions, is ubiquitous in the environment. There is no potential for bioaccumulation or bioconcentration. Potassium and chloride ions are essential to all living organisms and their intracellular and extracellular concentrations are actively regulated.

Human Health Toxicity Summary [1,3,8,9]	
Chronic Repeated Dose Toxicity	Fourteen female rats were given KCl in their drinking water (approximately 5,250 mg/kg/day) for 105 days. Ten rats were sacrificed after 105 days of exposure for examination of the heart, kidneys and the adrenals; four rats (recovery group) were kept for an additional month. KCl exposure resulted in decreased heart weight, increased kidney weight, and enlargement of part of the adrenals. All changes were reversible within one month of exposure (Bacchus, 1951).F344/Slc male rats were given 0, 110, 450 or 1,820 mg/kg/day KCl in feed for two years. At the end of the study, survival rates were 48%, 64%, 58% and 84% in the controls, 110, 45 and 1,820 mg/kg/day groups. Nephritis was reported to be predominant in all groups, including the controls. The only treatment-related effect observed was gastritis(inflammation of the stomach lining). The incidence of gastritis and ulcers were 6%, 18%, 18% and 30% in the controls, 110, 450 and 1,820 mg/kg/day groups (Imai *et al.*, 1968). Male and female Wistar rats were fed diets containing 0 or 3% KCl over a total period of 30 months: Examination after 13 weeks (10 rats/sex/group), after 18 months (15 rats/sex/group) and after 30 months (50 rats/sex /group). Due to the reduction of feed intake the mean test substance intake and mean body weight decreased in time. After 30 months of treatment, there was hypertrophy of the zona glomerulosa in the adrenals (24/50 treated rats versus 4/50 in controls); and cystitis in the urinary bladder (males: 3/59; females 3/50) and single epithelial hyperplasia of the bladder (males 3/50; females 2/50) (Lina *et al.*, 1994; Lina and Kuijpers, 2004)**.**
Carcinogenicity	Potassium chloride has not been evaluated and is not listed by the IARC as a carcinogen. In a long-term study, male rats (50 per group) were fed potassium chloride in the diet at levels of 110, 450 or 1820 mg/kg bw/day for 2 years. No carcinogenic effects were observed in male rats.
Mutagenicity/ Genotoxicity	No gene mutation ns were reported in bacterial tests, with and without metabolic activation. However, high concentrations of potassium chloride showed positive results in a range of genotoxic screening assays using mammalian cells in culture. The action of potassium chloride in culture seems to be an indirect effect therefore further *in vivo* studies were not considered necessary.
Reproductive Toxicity Developmental Toxicity/Teratogenicity	A developmental study revealed no foetotoxic or teratogenic effects of potassium chloridel in doses up to 235 mg/kg/day (mice) and 310 mg/kg/day (rats). No fertility study has been located. Further human and ecological assessment was not recommended by the OECD SIDS.
Acute Toxicity	Potassium chloride is an essential element with homeostatic physiological processes regulating levels in the body. In cases of increased exposure to high levels of potassium significant health effects in people with kidney disease or other conditions, such as heart disease may result. Adverse health effects due to consumption of potassium from drinking water are unlikely to occur in healthy individuals. Acute effects are rare in humans although under particular circumstances severe effects may occur. Lethal effects were observed in a 2 month old baby fed 15,000 mg potassium chloride for 2 days and in another case report where an adult woman had ingested slow released potassium chloride tablets (35, 000 mg). The most common form of ingestion is through drinking water. It is not considered necessary to establish a health-based guideline value for potassium in drinking water due to its lack of toxicity.
Irritation	Slight skin and eye irritant. A threshold concentration for skin irritancy of 60% was seen when potassium chloride in aqueous solution was in contact with skin of human volunteers. The threshold concentration when applied to broken skin was 5%.
Sensitisation	No data found.
Health Effects Summary	This chemical has been identified by NICNAS to be of low concern to human health and it is listed by the US Food and Drug Administration (FDA) as a Generally Recognised as Safe (GRAS) substance.

Key Study/Critical Effect for Screening Criteria	In a two-year rat feeding study, there was an increased incidence of gastritis and ulcers at dose levels of >110 mg/kg/day (Imai *et al.*, 1968). There was no NOAEL. Thus, the LOAEL for this study is 110 mg/kgday. Since the gastritis and ulcers are the result of a localized irritation effect of the test substance (site of contact) in the gastrointestinal tract, an uncertainty factor for interspecies variability is deemed unnecessary. For systemic effects, the NOAEL for the two-year rat feeding study is considered to be 1,820 mg/kg/day, the highest dose tested. Uncertainty factors: 10 (intraspecies variability); 10 (interspecies variability); 1 (intraspecies variability) Oral Reference Dose = 1,820/100 = 18.2 mg/kg/day Drinking water guideline: 71 ppm
Ecological Toxicity [1,3,8,9,10]	
Aquatic Toxicity	In a guideline study, the 96-hour LC50 in *Pimephales promelas* was reported to be 880 mg/L (Mount *et al.*, 1997). The 48-hour LC50 values from two studies on *Lepomis macrochirus* (Patrick *et al.*, 1968; Trama, 1954), and one study each on *Oncorhyncusmykiss* and *Ictalurus punctatus* (Waller *et al.*, 1993) ranged from 720 to 2,010 mg/L. In a guideline study, the 48-hour EC50s in *Daphnia magna* and *Ceriodaphnia dubia* were 660 and 630 mg/L, respectively (Mount *et al.*, 1997; ECHA REACH database). The 48-hour EC50 in *Daphnia magna* in another study was also reported to be 177 mg/L (Biesinger and Christensen, 1972).The toxicity of KCl has been investigated in one algae species (*Nitzschia linearis*), showing 120 hour-EC50 (growth rate) of 1,337 mg/L (Patrick *et al.*, 1968). The 72-hour EC50 to *Scenedesmus subspicatus* is >100 mg/L (growth rate), with a NOEC of >100 mg/L (ECHA REACH database). In a fish early-life-stage test with the fathead minnow (*Pimephales promelas*), the 7-day NOEC is 500 mg/L (ECHA REACH database). A long term (21-day) study has been performed on *Daphnia magna* where effects on reproduction were investigated for several metals. A 16% impairment of reproduction (LOEC) was observed at a concentration of 53 mg/L of K +, equal to KCl concentration of 101 mg/L (Biesinger and Christensen, 1972). The measured NOEC for Daphnia is 373 mg/L
Determination of PNEC aquatic	PNECaquatic: On the basis of the chronic results for Daphnia, an assessment factor of 100 has been applied to the lowest reported effect concentration of 373 mg/L. The PNECaquatic is determined to be 3.73 mg/L.
Current Regulatory Controls	
Australian Hazard Classification	No data available
Australian Occupational Exposure Standards	No data available
International Occupational Exposure Standards	No data available
Australian Food Standards	No data available
Australian Drinking Water Guidelines	No data available
Aquatic Toxicity Guidelines	No data available
PBT Assessment [1,8,9,10]	
P/vP Criteria fulfilled?	Potassium chloride is an organic salt that dissociates completely to potassium and chloride ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions; both potassium and chloride ions are also ubiquitous and are present in most water, soil and sediment. For the purposes of this PBT assessment, the persistent criteria is not considered applicable to this inorganic salt.
B/vB criteria fulfilled?	Potassium and chloride ions are essential to all living organisms and their intracellular and extracellular concentrations are actively regulated. Thus, potassium chloride is not expected to bioaccumulate.

T criteria fulfilled?	The measured chronic toxicity data for potassium chloride was 373 mg/L for Daphnia. Thus, potassium chloride does not meet the screening criteria for toxicity
Overall conclusion	Not PBT
Revised	April 2018

References

1. WHO (2009). Potassium in drinking-water. Background document for development of Guidelines for Drinking-water Quality. World Health Organization WHO/HSE/WSH/09.01/7.
2. HSDB Hazardous Substance Databank (HSDB) Potassium Chloride. Toxnet http://toxnet.nlm.nih.gov U.S. National Library of Medicine.
3. IARC, 2009: IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. International Agency for Research on Cancer. World Health Organisation.
4. Material Safety Data Sheet Potassium chloride. ScienceLabs.com Inc. http://www.sciencelab.com/msds.php?msdsId=9927402
5. WHO Poisons Information Monograph for Potassium Chloride. Electronic record accessed from www.inchem.org World Health Organization.
6. UNEP Potassium Chloride Screening Information Dataset (SIDS) Initial Assessment Report for 13th SIAM (Bern, 6-9 November 2001. United Nations Environment Programme (UNEP) http://www.inchem.org/documents/sids/sids/KCHLORIDE.pdf
7. ECHA REACH database: http://apps.echa.europa.eu/registered/registered-sub.aspx
8. IUCLID Data Set for Potassium chloride (CAS No. 7447-40-7), UNEP Publications.
9. OECD (2001b). OECD-Screening Information Assessment Report (SIAR) for Potassium chloride (CAS No. 7447-40-7), UNEP Publications.
10. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme

AECOM Imagine it. Delivered.

Toxicity Summary - 2-Propenoic acid, polymer with sodium phosphinate and 2-Propenoic acid, sodium salt, polymer with 2-propenamide

Chemical and Physical Properties[1,2,3]	
CAS number	129898-01-7 25085-02-3
Molecular formula	(C3H4O2.H3O2P.Na)x.xNa (C3H5NO.C3H4O2.Na)x
Molecular weight	Likely >1000 MW
Solubility in water	No data available.
Melting point	No data available.
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	No data available.
Overview	No studies are available. The polymer is not expected to be readily biodegradable. Biodegradation is limited due to the very high molecular weight and the low water solubility of the polymer. Due to its high molecular weight, the polymer is not expected to bioaccumulate. This chemical has been identified by NICNAS to be of low concern to human health and thus required no further assessment.
Environmental Fate[2]	
Soil/Water/Air	The polymer is not expected to be readily biodegradable. Biodegradation is limited due to the very high molecular weight and the low water solubility of the polymer. Due to its high molecular weight, the polymer is not expected to bioaccumulate.
Human Health Toxicity Summary	
Chronic Repeated Dose Toxicity	No data available.
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	No data available.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No data available.
Acute Toxicity	No data available.
Irritation	No data available.
Sensitisation	No data available.
Health Effects Summary	This chemical has been identified by NICNAS to be of low concern to human health.

Key Study/Critical Effect for Screening Criteria	No data available.
Ecological Toxicity[2]	
Aquatic Toxicity	Limited information is available. The polymer is expected to be a low concern for toxicity to aquatic organisms. Due to its poor solubility and high molecular weight, it is not expected to be bioavailable. It does not contain any reactive functional groups.
Determination of PNEC aquatic	No PNEC values were calculated.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	The polymer is not readily biodegradable, hence it meets the screening criteria for persistence.
B/vB criteria fulfilled?	The polymer is expected to have a very high molecular weight and poor water solubility. It is not expected to be bioavailable, hence this polymer does not meet the criteria for bioaccumulation.
T criteria fulfilled?	There are no aquatic toxicity studies on the polymer. It is expected to have low concern for aquatic toxicity because of its very high molecular weight and poor water solubility. As such, the polymer does not meet the critera for toxicity.
Overall conclusion	Not PBT
Revised	April 2019

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.nicnas.gov.au
2. Categorization Results from the Canadian Domestic Substance List, 2-Propenoic acid, polymer with sodium phosphinate
3. U.S National Library of Medicine, Toxicology Data Network, ChemIDplus, CAS#38193-60-1. Accessed 2017 at https://chem.nlm.nih.gov/chemidplus/rn/38193-60-1

AECOM Imagine it. Delivered.

Toxicity Summary - Calcium Carbonate

Chemical and Physical Properties[1,2]	
CAS number	1317-65-3
Molecular formula	Not applicable
Molecular weight	Not applicable
Solubility in water	No data available
Melting point	Approximately 900°C (Oates 1998).
Boiling point	No data available
Vapour pressure	No data available
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	No data available
Colour/Form	Solid
Overview	Limestone is the name given to a type of rock mostly composed of calcium carbonate. It also contains minor impurities of iron, magnesium, quartz, clay, pyrite, phosphate, and organic matter (Pohl 2011). It is used widely in agriculture to increase calcium concentrations and the pH of soils (Upjohn et al. 2005). Limestone is used industrially on a very large scale as an ingredient in concrete production and in metallurgy (Oates 1998; Pohl 2011). In the Australian coal seam gas industry, it is used as a bridging agent in drilling fluid formulations. A Tier 1 Human Health Assessment for these chemicals has been conducted by NICNAS which concluded that these chemicals were identified as low concern to human health by application of expert validated rules.
Environmental Fate[2]	
Soil/Water/Air	Limestone dissolves slowly in water, releasing calcium and carbonate ions as well as other trace elements, such as iron and magnesium (Deer et al. 1992; Clair and Hindar 2005; Pohl 2011). These trace elements are naturally ubiquitous in the environment and are subject to natural biogeochemical processes. Calcium oxide reacts immediately upon exposure to water, forming calcium hydroxide, which itself reacts with carbon dioxide to form calcium carbonate. The final reaction products of both limestone and calcium oxide in the environment are therefore essentially the same, although calcium oxide typically has lower concentrations of magnesium and other inorganic chemicals than limestone and produces a higher initial concentration of hydroxide ions (Upjohn et al. 2005). Calcium and carbonate ions occur naturally in all environmental compartments, and are important nutrients for various organisms. Calcium is mobile in soil (ANZECC and ARMCANZ 2000) and, if released to the environment, should be expected to experience significant partitioning to the water compartment. However, calcium ions may also form insoluble precipitates with anions present in the environment, such as carbonate ions, and settle out of the aqueous phase. Carbonate is an important component of the global carbon cycle (Wetzel 2001).
Human Health Toxicity Summary[3]	
Chronic Repeated Dose Toxicity	No systemic toxicological findings could be detected in rats after repeated administration of uncoated nano calcium carbonate by the oral route for a period of 90 days. The results of this study are read across to bulk calcium carbonate. Several potential adverse effects have been reported following calcium supplementation e.g. effects on kidney function, milk-alkali syndrome, kidney stones and interactions with minerals. However, these effects are more prevalent in those people suffering from renal insufficiency and following the ingestion of high doses of calcium.

	No systemic toxicity was observed in a 90-day inhalation study where rats were exposed to uncoated calcium carbonate at a maximum concentration of 0.399 mg/L, stated as the NOEC. The local effects observed at this level were limited to increased lung weights accompanied by increases in BAL-derived inflammation and cytotoxicity biomarkers, which were reversible in males but were not fully reversible in females within a 4-week recovery period. Hence the NOAEC for local effects was established as 0.212 mg/L and the LOAEC at 0.399 mg/L. The results of this study are read across to bulk calcium carbonate.
Carcinogenicity	Uncoated nano calcium carbonate is not expected to pose a risk of carcinogenicity.
Mutagenicity/ Genotoxicity	Uncoated nano calcium carbonate was negative in the following assays: In vitro gene mutation study in bacteria (OECD TG 471) using Salmonella typhimurium strains TA 98, TA 100, TA 1535 and TA 1537 and Escherichia coli WP2 uvrA with and without metabolic activation (S9). In vitro chromosome aberration study in mammalian cells (OECD TG 473) using human lymphocytes in the presence and absence of metabolic activation. In vitro gene mutation study in mammalian cells (OECD TG 476) using mouse lymphoma L5178Y cells in the presence and absence of metabolic activation. The results of these studies are read across to bulk calcium carbonate.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Under the conditions of the OECD TG 422 study, uncoated nano calcium carbonate administered to male and female rats up to a dose level of 1000 mg/kg bw/day for a period of up to 48 days is not toxic to reproduction and has no effect on fertility or development. No treatment-related effects were observed for reproduction; hence, a NOEL for reproductive toxicity was considered to be 1000 mg/kg bw/day. The results of this study are read across to bulk calcium carbonate. The prenatal developmental toxicity study also demonstrated that calcium carbonate was neither foetotoxic nor teratogenic at the concentrations used. Since no adverse effects were noted at the highest dose level tested (1.25% Ca in diet), the NOAEL for teratogenic and maternal toxic effects in rats is in excess of 1.25% Ca, equivalent to approximately 1963 - 2188 mg/kg bw/day of calcium carbonate.
Acute Toxicity	Bulk calcium carbonate is not considered to be acutely harmful by the oral, dermal or inhalation routes.
Irritation	Bulk calcium carbonate is not considered to be irritating to the skin or eyes.
Sensitisation	Based on the results of an OECD TG 429 study performed using nano calcium carbonate and read across to bulk calcium carbonate, where the Stimulation Index was < 3, bulk calcium carbonate is considered to be a non-sensitiser
Health Effects Summary	This chemical has been identified by NICNAS to be of low concern to human health.
Key Study/Critical Effect for Screening Criteria	In the repeated dose toxicity study, NOAEC for local effects was established as 0.212 mg/L and the LOAEC at 0.399 mg/L.
Ecological Toxicity[2]	
Aquatic Toxicity	Calcium carbonate has low toxicity to aquatic and terrestrial organisms. Ecotoxicological endpoint values for aquatic organisms generally greatly exceed 100 mg/L (LMC 2014), indicating very low toxicity.
Determination of PNEC aquatic	Experimental results are available for three trophic levels. Acute LC50 values are available for fish, invertebrates, and algae. On the basis that the data consists of short term results from three trophic levels, an assessment factor of 1000 has been applied to the lowest reported effect concentration of 310 mg/L for invertebrates. The PNEC aquatic is 0.3 mg/L.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.

International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	Not applicable (inorganic chemical, ionic species ubiquitous in environment)
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to these inorganic ions.
T criteria fulfilled?	Not applicable. Expected to have low toxicity to aquatic organisms.
Overall conclusion	Not PBT
Revised	October 2019

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.nicnas.gov.au
2. Department of the Environment and Energy 2017, National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme
3. ECHA REACH, Calcium carbonate, Retrieved 2019: https://echa.europa.eu/

AECOM Imagine it. Delivered.

Toxicity Summary - Cellulose, carboxymethyl ether, sodium salt

Chemical and Physical Properties[1,2]	
CAS number	9004-32-4
Molecular formula	No data available.
Molecular weight	No data available.
Solubility in water	No data available.
Melting point	No data available.
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	White odourless hygroscopic granules or powder.
Overview	Sodium carboxycellulose is the sodium salt of carboxymethylcellulose. Carboxymethyl cellulose is a cellulose derivative with carboxymethyl groups (-CH2COOH) bound to some of the hydroxyl groups of the glucopyranose monomers that make up the cellulose backbone. Sodium carboxycellulase is a listed as GRAS (Generally Regarded as Safe) by the U.S. Food and Drug Administration (FDA GRAS database). It is an approved food additive in the EU (EC, 1995) and may be added to all foodstuffs following quantum satis principle, except in products for the dietary management of metabolic disorders, where the limit of use is 10 g/L or kg (EC, 1999). Sodium carboxycellulase is also listed as an Inert Ingredient Eligible for US Federal Insecticide, Fungicide, and Rodenticide Act (FIFRA) 25(b) pesticide products and US EPA List 4A. The Joint FAO/WHO Expert Committee on Food Additives has determined an Acceptable Daily Intake (ADI) for sodium carboxymethyl cellulose of “Not Specified” (no upper limit) (JECFA, 1989). A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health.
Environmental Fate[1]	
Soil/Water/Air	Carboxymethyl cellulose (DS 0.7) showed 25% biodegradation after 28 days in a OECD 301A test. Thus, sodium carboxymethyl cellulose is not readily biodegradable. Other studies have also shown partial degradation of carboxymethyl cellulose in ready and inherent biodegradability tests.
Human Health Toxicity Summary	
Chronic Repeated Dose Toxicity	No data available.
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	No data available.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No data available.

Acute Toxicity	No data available.
Irritation	No data available.
Sensitisation	No data available.
Health Effects Summary	No data available.
Key Study/Critical Effect for Screening Criteria	No data are available for determining the critical effect and the LOAEL/NOAEL for an oral reference dose.
Ecological Toxicity [1]	
Aquatic Toxicity	Carboxymethyl cellulose has been tested in several acute aquatic toxicity tests. The 96-hour LC50 for Brachydanio rerio is >2,500 mg/L; the 48-hour LC50 for Daphnia magna is >5,000 mg/L; and the 96-hour EC50 for Selenastrum capricornutum is 500 mg/L.
Determination of PNEC aquatic	PNECaquatic: Experimental results are available for three trophic levels. Acute E(L)C50 values are available for fish (>2,500 mg/L), Daphnia (>5,000 mg/L), and algae (>500 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 effect concentration of 500 mg/L for algae. The PNECaquatic is 0.5 mg/L.
Current Regulatory Controls[4]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	Sodium carboxymethyl cellulose is a water-soluble semisynthetic polymer and is not readily biodegradable. Thus, it meets the screening criteria for persistence.
B/vB criteria fulfilled?	Sodium carboxymethyl cellulose is a water-soluble semisynthetic polymer and is expected to have a molecular weight of >1,000 which limits its bioavailability to aquatic organisms. Thus, it is not expected to bioaccumulate.
T criteria fulfilled?	The acute EC(L)50 of sodium carboxymethylcellulose is >0.1 mg/L in fish, invertebrates and algae. Thus, it does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	April 2019

References

1. Van Ginkel, C.G., and Gayton, S. (1996). The biodegradability and nontoxicity of carboxymethyl cellulose (DS 0.7) and intermediates. Environ. Toxicol. Chem. 15: 270-274
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.nicnas.gov.au
3. EC (1995). European Parliament and Council Directive No 95/2/EC of 20 February 1995 on food additives other than colours and sweeteners, OJ L 61, 18.3.1995, p. 1-63.

4. EC (1999). Food additives permitted in dietary foods for infants and young children for special medical purposes as defined in Directive 1999/21/EC (Commission Directive 1999/21/EC of 25 March 1999 on dietary foods for special medical purposes, (OJ L 91, 7.4.1999, p. 29).
5. FDA GRAS Database: http://www.fda.gov/Food/IngredientsPackagingLabeling/GRAS/SCOGS/ucm260737.htm
6. JECFA (1989). http://apps.who.int/ipsc/database/evaluations/chemical.aspx?chemID=3773

AECOM Imagine it. Delivered.

Toxicity Summary - Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivatives residues

Chemical and Physical Properties[1]	
CAS number	68909-77-3
Molecular formula	C36H78N6O14
Molecular weight	UVCB
Solubility in water	100 g/L at 20 °C
Melting point	-20 °C at 101.3 kPa
Boiling point	223 °C at 101.3 kPa
Vapour pressure	0.55 - 20 Pa at 20 - 25 °C
Henrys law constant	No data available
Explosive potential	Non-explosive (100%)
Flammability potential	Not classified (50%), Non-flammable (50%)
Colour/Form	Liquid
Overview	The residuum from the reaction of diethylene glycol and ammonia. It consists predominantly of morpholine-based derivatives such as [(aminoethoxy)ethyl]morpholine, [(hydroxyethoxy)ethyl]morpholine, 3-morpholinone, and 4,4'-(oxydi-2,1-ethanediyl)bis[morpholine].
Environmental Fate[1]	
Soil/Water/Air	The substance is hydrolytically stable at pH 4, 7 and 9 at 25°C. Adsorption to solid soil phase is not to be expected. Based on the assessment of the components, it can be concluded that the mixture will not evaporate into the atmosphere. The substance is not readily biodegradable. Based on the low log Kow, the substance will have a low potential for bioaccumulation. Over time, the mixture will preferentially distribute into the compartment water.
Human Health Toxicity Summary[1]	
Chronic Repeated Dose Toxicity	No adverse effects were observed in male and female rats in a 28 days and a 90 days repeated dose toxicity test conducted according to OECD 407 and OECD 408 respectively (Calvert Laboratories, Inc., 2011 and Envigo Research Limited, 2018) in which animals were exposed orally (gavage) to 0, 100, 500 or 1000 mg/kg bw/d (28 days study) and to 0, 10, 100 or 1000 mg/kg bw/d (90 days study). In both studies, an NOAEL of 1000 mg/kg bw was determined.
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	In an in vivo micronucleus test on mouse bone marrow erythrocytes (BioReliance, 2010), performed according to OECD guideline 474, the animals were exposed orally (via gavage) with 500, 1000, 2000 mg/kg. This did not induce a statistically positive increase in micronuclei in the hemopoietic cells of the mouse bone marrow at the time intervals evaluated under the experimental condition of this assay. No toxicity was observed. Vehicle and positive controls were valid.

Reproductive Toxicity / Developmental Toxicity/Teratogenicity	A Reproductive / Development Toxicity Screening Testing in Rats was performed according to OECD guideline 421 (Calvert Laboratories, Inc., 2011, Klimisch 1). In this GLP-compliant study, male and female Sprague-Dawley rats (10 animals/sex/dose) were exposed to 1000 mg/kg bw/day. Control animals received concurrent vehicle (water). The rats were exposed on a daily basis, starting two weeks prior to cohabitation until the day before the scheduled euthanasia (minimum of 4 weeks) for males and starting for a minimum of 15 days prior to cohabitation, during cohabitation and from presumed gestation days 0 through day 19 of gestation for females. One female animal was found dead on lactation day 2. All other females survived until scheduled sacrifice. No treatment-related effects were observed in clinical signs, mortality, body weight (gain), food consumption, gross pathology, organ weights or histopathology in the parental animals. There was no treatment-related effect on reproductive performance or in reproductive parameters like for instance number of gravid animals, corpora lutea per dam, total implantations, litter viability or foetal sex ratios. The incidence of neonates born alive/found dead, stillborn or missing between lactation days 0 -4 was comparable among study groups. No treatment-related malformations were observed for neonates. The body weight of neonates was statistically significantly increased with an unknown biological significance for this finding. A NOAEL of 1000 mg/kg bw/day was established.
Acute Toxicity	The oral LD50 in male and female Sprague-Dawley rats was determined to be 5000 mg/kg bw in a reliable, key study conducted similarly to OECD guideline 420 (Calvert Laboratories, Inc., 2010). Calvert Laboratories Inc (2010) determined acute dermal toxicity in 10 male / female rats following a GLP-compliant study performed equivalent to OECD guideline 402 (key study, Klimisch 1). Only one dose was tested (2000 mg/kg bw). No mortality was observed. Shortly after exposure, local erythema and oedema effects were observed in both males and females. The effects seemed to be reversible in males and 1 female. In the 4 other females, necrosis and slouching appeared.
Irritation	The skin irritation potential of the test substance is investigated in a key, reliable study performed according to OECD guideline 404 (Allen, 1994; Klimisch 2) in 3 rabbits. Semi-occlusive patches were removed from shaved test sites after exposure periods of 3 min, 1h, and 4h to 0.5mL of the test substance. The Draize scoring system was used to evaluate the results. No erythema/eschar formation and no oedema formation is observed after 3 min and 1 hour exposure. After 4 hours of exposure, very slight erythema in all 3 animals and very slight oedema in 2 animals was observed, but this was fully reversible within 24 - 48 hrs. Calvert Laboratories, Inc. (2010) investigated the eye irritation potential of the test substance in a key, reliable study performed according to EU method B.5 (Klimisch 1) in 3 rabbits. The treated eyes (with 0.1mL test substance) will remain unrinsed for at least 24 hours after instillation. The 24-, 48- and 72-hours scores will be added separately for each animal and each total divided by 3 to yield the individual mean scores for each animal. Based on the data and according to the criterial of the CLP regulation, the test substance is classified as irritant to the eyes (category 2).
Sensitisation	Based on a guinea pig maximization study, performed according to the OECD guideline 406 (Allen, 1996; Klimisch 2), the test substance is considered not sensitising to the skin.
Health Effects Summary	This chemical may cause skin and eye irritation.
Key Study/Critical Effect for Screening Criteria	The critical lowest No Observed Adverse Effect (NOAEL) level for the purposes of risk assessment is 1000 mg/kg bw/day from the 90 day repeated oral toxicity study.
Ecological Toxicity [1]	

Aquatic Toxicity	In a static test following the procedures of the German national standard DIN 38412 using Leuciscus idus as test species a LC50 (96 h) of 681.2 mg/L (nominal) was determined [BASF AG, 1988; Study No. 10F0118/885140]. In conclusion, the substance is with high probability not acutely harmful to fish. The EC50 of the test item on daphnids was found to be greater than 122 mg/L (measured value) in a GLP guideline study according to OECD 202 [BASF SE, 2010; Study No. 50E0396/09E012]. Therefore, the test substance is with high probability acutely not harmful to aquatic invertebrates. A study was performed to assess the effect of the test item on the growth of the green alga Pseudokirchneriella subcapitata. The method followed that described in the OECD Guidelines for Testing of Chemicals (2006) No 201, "Freshwater Alga and Cyanobacteria, Growth Inhibition Test" referenced as Method C.3 of Commission Regulation (EC) No 440/2008. The effect of the test item on the growth of Pseudokirchneriella subcapitata has been investigated over a 72-Hour period. the ErC50(72h) of the test item is 45 mg/L for Pseudokirchneriella subcapitata. The toxicity to microorganisms was determined in a GLP short-term respiration test according to OECD guideline 209 using activated sludge from a municipal sewage treatment plant. After 180 minutes no inhibition effect on the respiration rate at the highest test concentration (1000 mg/L) was observed [BASF 2010; Study No. 08G0396/09G004]. Therefore, it can be concluded that inhibition of the degradation activity of activated sludge is not anticipated when introduced in appropriately low concentrations.
Determination of PNEC aquatic	The available short-term aquatic tests covering the three trophic levels (fish, daphnids, algae) showed the lowest L(E)C50 to be 45 mg/L in algae. An assessment factor of 1000 was used for a resulting PNEC for of 0.045 mg/L.
Current Regulatory Controls[4]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1]	
P/vP Criteria fulfilled?	Not readily biodegradable. Thus, it is expected to meet the screening criteria for persistence.
B/vB criteria fulfilled?	As the Log Pow is 0.565 at 20 °C (Log Pow < 4.5), it is not expected to be bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of this chemical is >0.1 mg/L. Thus, it is not expected to meet the screening criteria for toxicity
Overall conclusion	Not PBT
Revised	March 2019

References

1. ECHA REACH, Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivs. residues, Retrieved 2019: https://echa.europa.eu/

AECOM Imagine it. Delivered.

Toxicity Summary - Glyoxal (Ethanedial)

Chemical and Physical Properties[1,2,3]	
CAS number	107-22-2
Molecular formula	C2H2O2
Molecular weight	58.04
Solubility in water	600 g/L at 25 °C
Melting point	15 °C
Boiling point	50.4 °C
Vapour pressure	29.33 kPa at 20 °C
Henrys law constant	No data available.
Explosive potential	Non explosive
Flammability potential	Not classified
Colour/Form	Light yellow liquid with a mild odour at ambient temperatures; yellow crystals at 15 °C.
Overview	Glyoxal is generally available as an aqueous solution, typically containing 30-50% glyoxal in which hydrated oligomers are present. This chemical is used as a chemical intermediate in the production of pharmaceuticals and dyestuffs, as a cross-linking agent in the production of polymers, as a biocide, and as a disinfecting agent. Due to microbial activity as well as non-enzymatic autoxidation of oil or browning reactions of saccharides, glyoxal is frequently detected in fermented food and beverages. It is found in beer, wine and tea.
Environmental Fate[1]	
Soil/Water/Air	Glyoxal's production and use as a crosslinking agent in permanent-press fabrics, textiles, organic synthesis, glues, and biocides may result in its release to the environment through various waste streams. Glyoxal is also released to the environment from the combustion of wood, automobile exhaust, and the atmospheric degradation of aromatic and olefinic hydrocarbons. It may also be produced as a disinfection byproduct during the treatment of drinking water. Glyoxal is also endogenously produced by a variety of enzyme-independent pathways. If released to air, an extrapolated vapor pressure of 255 mm Hg at 25 deg C indicates glyoxal will exist solely in the vapor-phase. Vapor-phase glyoxal is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 34 hours. Glyoxal also undergoes direct photolysis, with an estimated atmospheric lifetime of 5 hours. If released to soil, glyoxal is expected to have very high mobility based upon an estimated Koc of 1. Volatilization from moist soil surfaces is not expected to be an important fate process based upon a Henry's Law constant of 3.33X10-9 atm-cu m/mole. The potential for volatilization of glyoxal from dry soil surfaces may exist based upon the extrapolated vapor pressure of this compound. Screening studies using sewage seed have indicated that glyoxal is readily biodegradable. If released into water, glyoxal is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Volatilization from water surfaces is not expected to be an important fate process based upon this compound's Henry's Law constant. Photolysis in sunlit surface waters is expected to be an important fate process because glyoxal absorbs light greater than 290 nm and undergoes direct photolysis in the atmosphere. Glyoxal is not expected to undergo hydrolysis in the environment due to the lack of hydrolyzable functional groups. An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low.

Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	From an oral 28 day repeat dose toxicity test conducted in accordance with OECD TG 407 a NOAEL was established at 40 mg/kg bw/day (active substance), based on dose-related changes in body weight gain at higher doses. A single inhalation toxicity study in rats revealed no systemic toxicity even at the highest dose of 0.4 mg/m^3.
Carcinogenicity	Results from several carcinogenicity studies, tumour initiation/promotion studies and in vitro cell transformation assays show that ethanedial is not carcinogenic.
Mutagenicity/ Genotoxicity	Ethanedial was shown to be mutagenic in both bacterial and mammalian cells in vitro. Unscheduled DNA synthesis was reported in one study in mice in vivo, but only within the pyloric sphincter and liver and not in more remote organs.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Available data on ethanedial and an analogue of ethanedial present in aqueous solutions suggest no effects on fertility or developmental toxicity in the absence of material toxicity.
Acute Toxicity	Ethanedial is moderately toxic via the oral and inhalation routes. In a guideline study in rats, an oral LD50 for a 40% ethanedial aqueous solution was reported at 3300 mg/kg bw. This corresponds to 1320 mg/kg bw/day for the active ingredient. An LC50 for inhalation toxicity was established at 2.44 g/L (active ingredient). Ethanedial is therefore considered to be of low dermal toxicity.
Irritation	Animal studies indicate that ethanedial is a skin and eye irritant
Sensitisation	Based on both animal and human studies, ethanedial is also considered a skin sensitiser.
Health Effects Summary	Ethanedial is moderately toxic via the oral and inhalation routes. In a guideline study in rats, an acute oral median lethal dose (LD50) for a 40% ethanedial aqueous solution was reported at 3 300 mg/kg bw. This corresponds to 1 320 mg/kg bw day for the active ingredient. A median lethal concentration (LC50) for inhalation toxicity was established at 2.44 g/L (active ingredient). Ethanedial is of low dermal toxicity. Animal studies indicate that ethanedial is a skin and eye irritant. From both animal and human studies, ethanedial is also a skin sensitiser.
Key Study/Critical Effect for Screening Criteria	A single repeat dose inhalation toxicity study in rats revealed no systemic toxicity even at the highest dose of 10 mg/m^3. From an oral 28-day repeat dose toxicity test conducted in accordance with OECD TG 407, a No-Observed-Adverse-Effect Level (NOAEL) was established at 40 mg/kg bw/day (active substance), based on dose related changes in body weight gain at higher doses. An adjustment factor of three is applied for inadequate duration of this study, as the no-effect dose was derived from a 28 day study. Consequently, for the purposes of quantifying the health risk of the chemical, an adjusted NOAEL of 13.3 mg/kg bw/day is used in this risk assessment.
Ecological Toxicity [1,2,3]	
Aquatic Toxicity	215 mg/L 96 h-LC50 fish. The result of the key study on freshwater invertebrates (BASF, 1988) indicates no acute toxicity of glyoxal (40% in aqueous solution) to Daphnia magna. The EC50 value is above 100 mg/L even when it is considered that no analytical monitoring was performed since glyoxal was shown to be stable at least for this 48-h period. In a GLP guideline study following OECD 210, the chronic treatment of early-life-stages of fish with the test item (Glyoxal 40%) under flow-through conditions resulted in no substance-related effects. Referring to the nominal concentrations of the active substance glyoxal, the NOEC was 119 mg a.i./L (BASF, 2009).
Determination of PNEC aquatic	An assessment factor of 100 has been applied to the reported LC50 of 215 mg/L for fish. The PNECaquatic is 2.15 mg/L.

Current Regulatory Controls[4]	
Australian Hazard Classification	Ethanedial is classified as hazardous for human health in the Hazardous Substances Information System (HSIS) with the following risk phrases (Safe Work Australia 2013): • Muta. Cat. 3 (Mutagenic Substances, Category 3) • R68 (Possible risk of irreversible effects) • Xn; R20 (Harmful by inhalation) • Xi; R36/38 (Irritating to eyes and skin) • R43 (May cause sensitisation by skin contact)
Australian Occupational Exposure Standards	No specific exposure standards were available.
International Occupational Exposure Standards	The following exposure standards are identified (Galleria Chemica 2013). Time Weighted Average (TWA): • 0.1 mg/m³ [Belgium, Columbia, Canada (Alberta, British Columbia, Saskatchewan), • Italy, Nicaragua, Portugal, Spain, United States of America] • 0.5 mg/m³ (0.2 ppm) [Denmark]. • Short Term Exposure Limit (STEL): • 0.3 mg/m³ [Canada (Saskatchewan)].
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No aesthetic or health-related guidance values were identified for this chemical in the Australian Drinking Water Guidelines (National Health and Medical Research Council (NHMRC) 2011).
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1,2]	
P/vP Criteria fulfilled?	Expected to be readily biodegradable and as such not persistent in the environment.
B/vB criteria fulfilled?	As the Log Pow is 0.85 (Log Pow < 4.5), it is not expected to be bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of this chemical is >0.01 mg/L. Thus, it is not expected to meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	April 2019

References

1. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme
2. ECHA REACH, Glyoxal, Retrieved 2019: https://echa.europa.eu/
3. HSDB (n.d.). Hazardous Substances Data Bank. Retrieved 2019, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

AECOM Imagine it. Delivered.

Toxicity Summary - Guanidine, hydrochloride (1:1)

Chemical and Physical Properties[2]	
CAS number	50-01-1
Molecular formula	CH5N3.ClH
Molecular weight	95.53 g/mol
Solubility in water	2,150 g/L at 20 °C
Melting point	188 °C
Boiling point	No data available.
Vapour pressure	For the pure solid guanidinium chloride the vapour pressure is expected to be much lower than 0.000005 Pa.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	Solid, powder, odourless
Overview	This substance is used in the following products: laboratory chemicals, extraction agents and pharmaceuticals. This substance has an industrial use resulting in manufacture of another substance (use of intermediates).
Environmental Fate[2]	
Soil/Water/Air	The guanidine ion is expected to have such a long hydrolysis half-life at environmentally relevant pH that the measurement is not feasable. Due to the low vapour pressure the substance under investigation will not be present in the gas phase in the atmosphere in appreciable amounts and therefore the elimination path photodegradation in air will be only of minor importance. Guanidine chloride is inherently biodegradable. Guanidine chloride is highly water soluble. For the inorganic solid a negligible vapour pressure is expected. According to the measured log Kow < -1.7, a low potential for adsorption is expected (non-ionic adsorption).
Human Health Toxicity Summary[2]	
Chronic Repeated Dose Toxicity	A No Observed Adverse Effect Level (NOAEL) of 100 mg/kg body weight/day for repeated dose toxicity was established from an oral sub chronic toxicity study on Wistar rats according to OECD guideline 408 with Guanidine hydrochloride.
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	There is no evidence for genotoxic properties from gene mutation assays in bacteria and mammalian cells, as well as chromosome aberration in mammalian cells.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	A NOAEL of 350 mg/kg body weight/day for developmental toxicity was established from a developmental toxicity study according to OECD guideline 414 with Guanidine hydrochloride.
Acute Toxicity	Acute toxicity data on Guanidine hydrochloride are available for the oral, inhalation and dermal route. The data available from three studies for the oral route all indicate LD50 values for Guanidine hydrochloride in the range between 773.6 and 1120 mg/kg bw. The LC50 from an inhalation study for female rats is 3.181 mg/L air (LC50 for male rats = 7.655 mg/L air). The dermal LD50 is > 2000 mg/kg bw.
Irritation	Based on the available data Guanidine hydrochloride is irritating to the skin and irritating to the eye.

Sensitisation	Not sensitising
Health Effects Summary	After oral exposure signs of systemic toxicity including death were observed in acute toxicity studies, thus absorption of guanidine hydrochloride has occurred. As a consequence, it is likely that the substance will also be absorbed if inhaled. This assumption is supported by data from an acute inhalation toxicity study, were systemic effects and death were observed. The substance is irritating to the skin and eye. The substance is not skin sensitising and there is no evidence of genotoxic toxicity.
Key Study/Critical Effect for Screening Criteria	NOAEL (rat) of 100 mg/kg bw/day from sub-chronic oral toxicity study.
Ecological Toxicity[2]	
Aquatic Toxicity	Short-term toxicity to aquatic organisms: Fish: LC50 (96 h) = 690 mg/L a.i. for Pimephales promelas (test with read-across substance Guanidine nitrate). Invertebrates: EC50 (48h) = 70.2 mg/L for Daphnia magna (test with read-across substance Guanidine nitrate, similar to OECD 202). Algae and cyanobacteria: ErC50 (72 h) = 33.5 mg/L for Pseudokirchneriella subcapitata (test with read-across substance Guanidine nitrate Long-term toxicity to aquatic organisms: Fish: NOEC = 181 mg/L for Fathead minnow (test with read-across substance Guanidine nitrate, similar to OECD 210). Invertebrates: NOEC = 2.9 mg/L for Daphnia magna (test with read-across substance Guanidine nitrate, similar to OECD 211).
Determination of PNEC aquatic	PNEC not calculated. Acute and chronic results for species for all three tropic levels are above 1 mg/L.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Guanidine chloride is inherently biodegradable.
B/vB criteria fulfilled?	No. Log Kow is -1.7 @ 20 °C and BCF is 3.2 L/kg ww
T criteria fulfilled?	No. Acute and chronic toxicity data >1 mg/L for all three tropic levels.
Overall conclusion	Not PBT
Revised	October 2019

References

1. ECHA REACH, Guanidinium chloride, Retrieved 2019: https://echa.europa.eu/

AECOM Imagine it. Delivered.

Toxicity Summary - Kaolin

Chemical and Physical Properties[1,2,4,5]	
CAS number	1332-58-7
Molecular formula	H2Al2Si2O8 H2O
Molecular weight	258 (approx)
Solubility in water	Insoluble
Melting point	No data available.
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	Not combustible
Colour/Form	White, greyish-white, or slightly coloured
Overview	Kaolin is a mixture of different minerals. Its main component is kaolinite and it frequently contains quartz, mica, feldspar, illite and montmorlilonite. Kaolinite composition is tiny sheets of triclinic crystals with pseudohexagonal morphology. It is formed by rock weathering. Kaolin is used in paper production, in paints, rubber, plastic, ceramic, chemical, pharmaceutical and cosmetic industries. It has a high fusion point and is the most refractory of all clays. Kaolin is listed in FIFRA 25(b) and US EPA List 4A. It is also listed as GRAS (Generally Regarded as Safe) by the U.S. Food and Drug Administration (FDA GRAS database). A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health.
Environmental Fate[4]	
Soil/Water/Air	Kaolin is a natural component of the soil and occurs widely in ambient air. It has a density of 2.1–2.6 g/cm^3. The cation exchange capacity of kaolinite is considerably less than that of montmorillonite, in the order of 2–10 meq/100 g, depending on the particle size, but the rate of the exchange reaction is rapid, almost instantaneous (Grim, 1968). Kaolinite adsorbs small molecular substances such as lecithin, quinoline, paraquat, and diquat, but also proteins, polyacrylonitrile, bacteria, and viruses (McLaren et al., 1958; Mortensen, 1961; Weber et al., 1965; Steel & Anderson, 1972; Wallace et al., 1975; Adamis & Timár, 1980; Schiffenbauer & Stotzky, 1982; Lipson & Stotzky, 1983). The adsorbed material can be easily removed from the particles because adsorption is limited to the surface of the particles (planes, edges), unlike the case with montmorillonite, where the adsorbed molecules are also bound between the layers (Weber et al., 1965). Upon heating, kaolinite starts to lose water at approximately 400 °C, and the dehydration approaches completeness at approximately 525 °C (Grim, 1968). The dehydration depends on the particle size and crystallinity.
Human Health Toxicity Summary [1,4]	
Chronic Repeated Dose Toxicity	Long-term exposure to kaolin may lead to a relatively benign pneumoconiosis, known as kaolinosis. Deterioration of lung function has been observed only in cases with prominent radiological alterations. Based on data from China clay workers in the United Kingdom, it can be very roughly estimated that kaolin is at least an order of magnitude less potent than quartz.
Carcinogenicity	A4; Not classifiable as a human carcinogen

Mutagenicity/ Genotoxicity	Recently, manufactured nano/microparticles such as fullerenes (C60), carbon black (CB) and ceramic fiber are being widely used because of their desirable properties in industrial, medical and cosmetic fields. However, there are few data on these particles in mammalian mutagenesis and carcinogenesis. To examine genotoxic effects by C60, CB and kaolin, an in vitro micronuclei (MN) test was conducted with human lung cancer cell line, A549 cells. In addition, DNA damage and mutations were analyzed by in vivo assay systems using male C57BL/6J or gpt delta transgenic mice which were intratracheally instilled with single or multiple doses of 0.2 mg per animal of particles. In in vitro genotoxic analysis, increased MN frequencies were observed in A549 cells treated with C60, CB and kaolin in a dose-dependent manner. These three nano/microparticles also induced DNA damage in the lungs of C57BL/6J mice measured by comet assay. Moreover, single or multiple instillations of C60 and kaolin, increased either or both of gpt and Spi- mutant frequencies in the lungs of gpt delta transgenic mice. Mutation spectra analysis showed transversions were predominant, and more than 60% of the base substitutions occurred at G:C base pairs in the gpt genes. The G:C to C:G transversion was commonly increased by these particle instillations. Manufactured nano/microparticles, CB, C60 and kaolin, were shown to be genotoxic in in vitro and in vivo assay systems.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No data available.
Acute Toxicity	Occupationally inhaled kaolin produced chronic pulmonary fibrosis. In an acute oral study in which 120 rats were fed doses of Kaolin ranging from 100 to 210 g/kg. Fourteen rats were controls. Kaolin was inert and nonstatic except for the danger of bowel obstruction resulting in perforation. The clinical signs were listlessness, anorexia, oliguria, hypothermia, and dyspnea. These were a pathological reaction from over distension of the alimentary canal by an inert solid. The number of fatalities and the incidence and advance of bowel obstruction along the small intestine were dose related. The dose that killed 50% of the rats by bowel obstruction was 149 g/kg.
Irritation	Causes moderate eye irritation. May cause irritation of the respiratory system
Sensitisation	No data available.
Health Effects Summary	Kaolin is toxic to a variety of mammalian cells in vitro, and it produces transient inflammation in the lungs of experimental animals after intratracheal instillation.
Key Study/Critical Effect for Screening Criteria	No data available.

Ecological Toxicity [4]	
Aquatic Toxicity	The 24- and 48-h LC50 values for kaolinite toxicity to the water flea (Daphnia pulex) were >1.1 g/litre (Lee, 1976). Georgia kaolin caused <10% mortality of sea urchin (Strongylocentrosus purpuratus), Japanese clam (Tapes japonica), hermit crab (Pagurus hirsutiusculus), isopod (Sphaeroma pentodon), mud snail (Nassarius obsoletus), blue mussel (Mytilus edulis), and tunicates (Molgula manhattensis and Styela montereyensis) within 5–12 days. The 200-h LC10 values for coast mussel (Mytilus californianus), black-spotted bay shrimp (Crangon nigromaculata), migrant prawn (Palaemon macrodactylus), dungeness crab (Cancer magister), and the polychaete Neanthes succinea were 26, 16, 24, 10, and 9 g/litre, respectively. The 100-h LC10 values for the tunicate Ascidia ceratodes, amphipod Anisogammarus confervicolus, and shiner perch (Cymatogaster aggregata) were 7, 38, and 1 g/litre, respectively (McFarland & Peddicord, 1980). No effect on the hatching success or egg development rate of four marine fish species — red seabream (Pagrus major), black porgy (Acanthopagrus schlegeli), striped knifefish (Oplegnathus fasciatus), and threeline grunt (Parapristipoma trilineatum) — was observed at kaolinite concentrations up to 10 g/litre for 24 h. Larvae were more sensitive to kaolinite: the 12-h LC50 values were 170 and 710 mg/litre for P. trilineatum and O. fasciatus, respectively; mortality was also observed for P. major at concentrations of 1000 mg/litre and above (Isono et al., 1998).
Determination of PNEC aquatic	Kaolin has low toxicity to aquatic species, a large number of which have been tested. As such, $PNEC_{aquatic}$ has not been determined.
Current Regulatory Controls [2,3]	
Australian Hazard Classification	No hazard classification according to GHS criteria
Australian Occupational Exposure Standards	TWA: 10 mg/m^3
International Occupational Exposure Standards	TLV: (respirable fraction): 2 mg/m^3, as TWA
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	Not applicable (inorganic salt, ionic species ubiquitous in environment)
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to these inorganic ions.
T criteria fulfilled?	Not applicable. Acute toxicity data >1 mg/L in water flea, thus Kaolin does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	April 2019

References

1. HSDB (n.d.). Hazardous Substances Data Bank. Retrieved 2019, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
2. IPCS Kaolin, Retrieved 2019: http://www.inchem.org
3. Safe Work Australia, Hazardous Substances System, Retrieved 2019: http://hcis.safeworkaustralia.gov.au/

4. IPCS INCHEM; Environmental Health Criteria (EHC) Monographs. Bentonite, kaolin, and selected clay minerals (EHC 231). Available from, as of June 25, 2007: http://www.inchem.org/pages/ehc.html
5. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.nicnas.gov.au

AECOM Imagine it. Delivered.

Toxicity Summary - Potassium Hydroxide

Chemical and Physical Properties[1,2,3]	
CAS number	1310-58-3
Molecular formula	KOH
Molecular weight	56.11
Solubility in water	1100 g/l at 25°C
Melting point	406°C
Boiling point	1327°C
Vapour pressure	1.3 hPa at 719°C
Henrys law constant	No data available.
Explosive potential	The solution in water is a strong base. It reacts violently with acid and is corrosive to metals such as aluminium, tin, lead and zinc. This produces a combustible / explosive gas. Reacts with ammonium salts. This produces ammonia. This generates fire hazard. Contact with moisture and water may generate heat.
Flammability potential	Not combustible. Contact with moisture or water may generate sufficient heat to ignite combustible materials.
Colour/Form	White or slightly yellow odourless lumps, rods, pellets.
Overview	Potassium hydroxide is a strong alkaline substance that dissociates completely in water to K+ and OH- ions. KOH is commercialised as a solid or as solutions with varying concentrations. It has many industrial uses; less than 2% is for wide dispersive use. It is used in paint and varnish removers, drain cleaners, degreasing agents and dairy pipeline cleaners.
Environmental Fate[4]	
Soil/Water/Air	The high water solubility and low vapour pressure indicate that KOH will be found predominantly in the aquatic environment. KOH is present in the environment as potassium and hydroxyl ions, which implies that it will not adsorb on particulate matter or surfaces and will not accumulate in living tissues.
Human Health Toxicity Summary[1,3,4]	
Chronic Repeated Dose Toxicity	No studies were identified regarding the repeated dose toxicity of KOH in animals
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	There is no evidence for a mutagenic activity. K+ and OH- are not expected to be systemically available in the body over the normal limits, under non-irritating conditions. A genotoxic effect is also not very likely because both the K+ and OH- ions are naturally present in the human body.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Studies to the reproduction of KOH are not available. Based on the results of corresponding potassium salts like KCl and K2CO3, effects in non-irritating doses/concentrations to reproduction or development are not expected for KOH. The calculated NOAEL for the potassium ion is approximately 164 mg/kg bw.

Acute Toxicity	Potassium hydroxide has moderate acute toxicity based on results from three animal studies in rats following oral exposure. The median lethal dose (LD50) in rats is reported as 273–1230 mg/kg bw. The concentrations used in these tests were not reported. Observed sub-lethal effects included hyperexcitability, followed by apathy and weakness. Haemorrhaging of the stomach and intestine, and adhesions of abdominal organs (stomach, pancreas, spleen, liver and small intestine) were seen following administration of both lethal and sub-lethal doses (OECD, 2002). In contrast, the LD50 value in rats of potassium chloride, 3000 mg/kg bw, is much higher than that of potassium hydroxide, indicating low toxicity of the potassium ion (OECD, 2002).
Irritation	Solid KOH is corrosive. Depending on the concentration, solutions of KOH are non-irritating, irritating or corrosive and they cause direct local effects on the skin, eyes and gastrointestinal tract. Systemic effects are not to be expected. Solutions with concentrations higher than 2% are corrosive, while concentrations of about 0.5 to about 2.0 % are irritating.
Sensitisation	Based on the reported negative results in a guinea pig study and human experience, potassium hydroxide is not considered to be a skin sensitiser (OECD, 2002). Potassium hydroxide has been used extensively for many decades by industry and by consumers. However, skin sensitisation has never been described as secondary to skin irritation or burns. As discussed previously, both the potassium and the hydroxide constituents are ions that are naturally present in the body. For this reason, it is very unlikely that skin sensitisation would result from exposure to the chemical (OECD, 2002)
Health Effects Summary	Potassium hydroxide is corrosive to the skin, eyes, and gastrointestinal and respiratory tracts. Based on human data, concentrations of 0.5–2.0 % are irritating to the skin, while a concentration greater than 2.0 % is corrosive (OECD, 2002). The constituent ions of potassium hydroxide are naturally present in the body. Chronic systemic health effects such as repeated dose toxicity (apart from alkalosis), carcinogenicity and reproductive toxicity are not expected following exposures at non-irritating concentrations. There are limited available data on systemic health effects of potassium hydroxide in vivo (REACH). The very limited data on potassium chloride (OECD, 2002) concludes that there is no evidence of systemic toxicity of the endogenous potassium ion. In addition, similar results were reported for sodium hydroxide (NICNAS). Potassium salts are generally considered by NICNAS to be of low concern to human health (NICNAS, 2012).
Key Study/Critical Effect for Screening Criteria	No oral TRV apply. Acute toxicity only (irritant and corrosive). Systemic effects are not to be expected. The Australian drinking water guideline value for pH may apply to potassium hydroxide.
Ecological Toxicity [4]	
Aquatic Toxicity	The hazard of KOH for the environment is caused by the hydroxyl ion (pH effect). For this reason the effect of KOH on the organisms depends on the buffer capacity of the aquatic or terrestrial ecosystem. Also the variation in acute toxicity for aquatic organisms can be explained for a significant extent by the variation in buffer capacity of the test medium. The LC50 value of acute fish toxicity was in the order of 80 mg/l. It was 880 mg/l for KCl and ranged between 125-189 mg/l for NaOH. The LC50 values of acute invertebrate toxicity for KCl was 660 mg/l (Daphnia magna) and 630 mg/l (Ceriodaphnia dubia), and for NaOH 40 mg/l (Ceriodaphnia dubia). The EC50 algae value (Nitscheria linearis) was 1337 mg/l for KCl.
Determination of PNEC aquatic	It is not considered useful to calculate a PNEC for potassium hydroxide because factors such as the buffer capacity, the natural pH, and the fluctuation of the pH are very specific for a certain ecosystem. Based on the information above, a PNECaquatic was not derived for potassium hydroxide.
Current Regulatory Controls[1]	

Australian Hazard Classification	The chemical is classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia): Xn; R22 (acute toxicity) C; R35 (corrosivity)
Australian Occupational Exposure Standards	TWA: 2 mg/m^3 (peak limitation), Safe Work Australia
International Occupational Exposure Standards	The following exposure standards are identified (Galleria Chemica): An exposure limit of 0.5–2 mg/m^3 time weigh ted average (TWA) in different countries such as Bulgaria, Chile, Denmark, Poland and Sweden and 1–2 mg/m^3 short-term exposure limit (STEL) in countries such as the United Kingdom, Spain, South Africa and Poland.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	Not applicable (ionic species ubiquitous in environment)
B/vB criteria fulfilled?	Not applicable (ionic species ubiquitous in environment)
T criteria fulfilled?	No chronic toxicity data exist on potassium hydroxide; however, the acute EC(L)50s for KCl are >0.1 mg/L in fish, invertebrates and algae. Thus, potassium hydroxide does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	April 2019

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier II Assessment for Potassium hydroxide: Retrieved 2019: https://www.nicnas.gov.au
2. HSDB (n.d.). Hazardous Substances Data Bank. Retrieved 2015, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
3. IPCS Potassium Hydroxide, Retrieved 2015: http://www.inchem.org
4. OECD (2008). Screening Information Dataset (SIDS) Initial Assessment Report for Potassium Hydroxide (CAS No. 1310-58-3)
5. Safe Work Australia Workplace Exposure Standards for Airborne Contaminants, 2013.
6. ECHA REACH, Potassium Hydroxide, Retrieved 2015: http://echa.europa.eu

AECOM Imagine it. Delivered.

Toxicity Summary - Smectite

Chemical and Physical Properties[1,2,3]	
CAS number	12199-37-0
Molecular formula	No data available.
Molecular weight	No data available.
Solubility in water	No data available.
Melting point	No data available.
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	Off-white to tan fine flakes or powder
Overview	Smectites commonly result from the weathering of basic rocks. Smectite formation is favoured by level to gently sloping terranes that are poorly drained, mildly alkaline (such as in marine environments), and have the high Si and Mg potentials (Borchardt, 1977). Other factors that favour the formation of smectites include the availability of Ca and the paucity of K (Deer and others, 1975). Poor drainage is necessary because otherwise water can leach away ions (e.g. Mg) freed in the alteration reactions. Smetites are used in the industry as fillers, carriers, absorbents and a component in drilling fluids (Grim, 1962). A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health.
Environmental Fate[4*]	
Soil/Water/Air	Limited data is available for smectite, read across data has been obtained from bentonite. Bentonite is a rock formed of highly colloidal and plastic clays composed mainly of montmorillonite, a clay mineral of the smectite group, and is produced by in situ devitrification of volcanic ash. Bentonite's production and use in domestic products, cat litter, construction materials, ceramics, pharmaceuticals, beer and wine production and cosmetics may result in its release to the environment through various waste streams. Its use in drilling muds, in agricultural practice as a carrier and an animal feed binder will result in its direct release to the environment. Bentonite is a colloidal native hydrated aluminum silicate (clay) found in midwest of USA and in Canada. Occupational exposure to bentonite may occur through inhalation of dust and dermal contact with this compound at workplaces where bentonite is produced or used. Use data indicate that the general population may be exposed to bentonite via ingestion of and dermal contact with consumer products containing bentonite.
Human Health Toxicity Summary[4*]	
Chronic Repeated Dose Toxicity	Mice maintained on diets containing bentonite displayed slightly reduced growth rates. Mice treated with higher doses showed minimal growth and fatty livers and fibrosis of the liver and benign hepatomas. Bentonite increased the susceptibility of mice to pulmonary infection.
Carcinogenicity	No adequate studies are available on the carcinogenicity of bentonite.

Mutagenicity/ Genotoxicity	The genotoxic potential of bentonite particles (diameter < 10 um) with an a-quartz content of up to 6% and different chemical modifications (alkaline, acidic, organic) was investigated. Human lung fibroblasts (IMR90) were incubated for 36 hr, 48 hr, or 72 hr with bentonite particles in concentrations ranging from 1 to 15 ug/sq cm. Genotoxicity was assessed using the micronucleus (MN) assay and kinetochore analysis. The generation of reactive oxygen species (ROS) caused by bentonite particles via Fenton-like mechanisms was measured acellularly using electron spin resonance (ESR) technique and intracellularly by applying an iron chelator. The results show that bentonite-induced genotoxic effects in human lung fibroblasts are weak. The formation of micronuclei was only slightly increased after exposure of IMR90 cells to an acidic sample of bentonite dust with a quartz content of 4-5% for 36 hr (15 ug/sq cm), 48 hr (5 ug/sq cm), and 72 hr (1 ug/sq cm), to an alkaline sample with a quartz content of 5% for 48 hr and 72 hr (15 ug/sq cm), and to an acidic bentonite sample with 1% quartz for 72 hr (1 ug/sq cm). Native (untreated) and organic activated bentonite particles did not show genotoxic effects in most of the experiments. Also, bentonite particles with a quartz content < 1% were negative in the micronucleus assay. Generation of ROS measured by ESR was dependent on the content of transition metals in the sample but not on the quartz content or the chemical modification. Reduction of MN after addition of the iron chelator 2,2'-dipyridyl showed that ROS formation also occurs intracellularly. It was concluded that the genotoxic potential of bentonite particles is generally low but can be altered by the content of quartz and available transition metals.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Limited studies did not demonstrate developmental toxicity in rats after oral exposure to bentonite.
Acute Toxicity	Single intratracheal injection into rodents of bentonite and montmorillonite with low quartz content caused dose and particle side dependent effects, as well as transient local inflammation, which included oedema and increased lung weight. Single intratracheal exposures of rats to bentonite caused storage foci in the lungs. After intratracheal exposure of rats to this material with high quartz content, fibrosis is noted.
Irritation	The powder may contain large amounts of free silica which can produce pneumoconiosis with chronic inhalation.
Sensitisation	No data available.
Health Effects Summary	The substance can be absorbed into the body by inhalation. The substance is mildly irritating to the eyes and skin. The substance may have effects on the lungs. This may result in fibrosis.
Key Study/Critical Effect for Screening Criteria	No study available.
Ecological Toxicity [4*]	
Aquatic Toxicity	The 96-h LC50 for rainbow trout (Oncorhynchus mykiss) of Wyoming bentonite, used as a viscosifier in drilling fluids, was 19 g/litre (Sprague & Logan, 1979).
Determination of PNEC aquatic	PNEC has not been calculated.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.

Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[4]	
P/vP Criteria fulfilled?	No data available for Smectite. Information on bentonite reported that Biodegradation of bentonite appears to be minimal.
B/vB criteria fulfilled?	No, bioaccumulation appear minimal for montmorillonite compounds
T criteria fulfilled?	No, read across data from bentonite reported 96h LC50 for fish was > 1 mg/L. Thus, it is not expected to meet the toxicity criteria.
Overall conclusion	Not PBT
Revised	April 2019

* No data available for Smectite. Toxicity data for Bentonite is presented as a surrogate.

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.nicnas.gov.au
2. HSDB Hazardous Substances Data Bank. U.S. National Library of Medicine, Retrieved 2019: http://toxnet.nlm.nih.gov/
3. USGS Coastal and Marine Geology Program, Smectite Group. Retrieved 2019: https://pubs.usgs.gov/of/2001/of01-041/htmldocs/clays/smc.htm
4. IPCS Bentonite, Kaolin and Selected Clay Minerals, Retrieved 2015: http://www.inchem.org

AECOM Imagine it. Delivered.

Toxicity Summary - Sodium bicarbonate

Chemical and Physical Properties [1,2,4,5,6]	
CAS number	144-55-8
Molecular formula	NaHCO3
Molecular weight	84.01
Solubility in water	96 g/L (at 20 °C)
Melting point	Decomposes when heated over 50 °C
Boiling point	Decomposes
Vapour pressure	Negligible, ionizable inorganic compound
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	No data available
Colour/Form	white, odourless, crystalline powder
Overview	Sodium bicarbonate is classified by the U.S. Food and Drug Administration (FDA) as a 'Generally Recognised as Safe' (GRAS) ingredient in food with no other limitation than current good manufacturing practice (FDA, 1978; FDA, 1983). In the EU it is approved as a food additive (EU, 2000) and a feed ingredient (EU, 1998).In Australia it is recognised by Food Standards Australia New Zealand (FSANZ) as a food additive. Sodium bicarbonate is used as animal feed additive, human food additive and it is used in pharmaceuticals. It is also used for the production of other chemicals and used in cosmetics and detergents and other household cleaning products. A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health.
Environmental Fate[3]	
Soil/Water/Air	The high water solubility and low vapour pressure indicate that sodium bicarbonate will be found predominantly in the aquatic environment. Sodium bicarbonate is present in the environment as sodium and bicarbonate ions, which implies that it will not adsorb on particulate matter or surfaces and will not accumulate in living tissues.
Human Health Toxicity Summary [2,3]	
Chronic Repeated Dose Toxicity	There are no directly relevant studies on repeated dose exposure, however, knowledge of prior use and available literature does not indicate any adverse effects of long-term use of exposure via any route. In humans there is a long history of sodium bicarbonate used as an antacid in doses up to 4 g without adverse effects of long-term use, although it is recommended not to use high doses of pure sodium bicarbonate instead of antacids. In addition, sodium bicarbonate is an important extracellular buffer in vertebrates and is therefore readily regulated in the body.
Carcinogenicity	As with other sodium salts, high doses of sodium bicarbonate promote carcinoma formation in rat urinary bladder after pre-exposure to initiator or BBN. However, when rats were only exposed to sodium bicarbonate no carcinogenic effect on the urinary bladder was found. Based on the available information there are no indications that sodium bicarbonate has carcinogenic effects.
Mutagenicity/ Genotoxicity	*In vitro* bacterial and mammalian cell tests showed no evidence of genotoxic activity.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Sodium bicarbonate did not induce developmental effects when administered orally at the following doses: 580 mg/kg bw (mice), 340 mg/kg bw (rats) and 330 mg/kg bw (rabbits). Furthermore the substance will usually not reach the foetus when the exposure to sodium bicarbonate is sufficiently low, as it does not become systemically available.

Acute Toxicity	Acute oral ingestion by the patients may result in a ruptured stomach due to excessive gas development. Acute or chronic excessive oral ingestion may cause metabolic alkalosis, cyanosis and hypernatraemia. These conditions are usually reversible, and will not cause adverse effects.
Irritation	Sodium bicarbonate is a minimal or mild ocular and skin irritant
Sensitisation	No data available
Health Effects Summary	This chemical has been identified by NICNAS to be of low concern to human health.
Key Study/Critical Effect for Screening Criteria	The Australian drinking water screening value for sodium (180 ppm, aethestic) and pH may apply to sodium bicarbonate.
Ecological Toxicity [3]	
Aquatic Toxicity	In a 96-hr acute flow-through test with rainbow trout (Oncorhynchus mykiss) a NOEC of 2,300 mg/l and a LC50 of 7,700 mg/l were determined. The test was conducted under GLP conditions and according to FIFRA Guideline Reference number 72-1. In a 96-hr acute flow-through test with bluegill sunfish (Lepomis macrochirus) a NOEC of 5,200 mg/l and a LC50 of 7,100 mg/l were determined. The test was conducted under GLP conditions and according to FIFRA Guideline Reference number 72-1. In a 48-hr acute flow-through test with Daphnia magna a NOEC of 3,100 mg/l and a LC50 of 4,100 mg/l were determine. The test was conducted under GLP conditions and according to FIFRA Guideline Reference number 72-2. A (chronic) reproduction test with Daphnia magna was carried out. Test solutions were prepared to contain the appropriate concentrations of salts to yield a total hardness of170 mg/l $CaCO_3$. At the tested concentration $NaHCO_3$ of 576 mg/l the survival was 100% and the cumulative number of offspring per female did not significantly differ from the control. This demonstrates that the 21-day Daphnia magna NOEC is higher than 576 mg/l. Standard toxicity tests with algae or aquatic plants have not been found, but test medium for acute algae tests contain 50 mg/l sodium bicarbonate. Glass slides were exposed to a portion of a small stream with an addition of sodium bicarbonate to a concentration of 45 mg/l for a period of 63 days. An increasing algal standing crop compared to the controls was found. Except for a small increase of Cyanophycea species, no shift in species was determined.
Determination of PNEC aquatic	It is not considered useful to calculate a PNEC for sodium bicarbonate because factors such as the buffer capacity, the natural pH, and the fluctuation of the pH are very specific for a certain ecosystem. Based on the information above, a PNECaquatic was not derived for sodium bicarbonate.
Current Regulatory Controls[4]	
Australian Hazard Classification	No data available
Australian Occupational Exposure Standards	No data available
International Occupational Exposure Standards	No data available
Australian Food Standards	No data available
Australian Drinking Water Guidelines	No data available
Aquatic Toxicity Guidelines	No data available
PBT Assessment	
P/vP Criteria fulfilled?	Sodium bicarbonate is an inorganic salt that is present in the environment as sodium and bicarbonate ions. Biodegradation is not applicable to these inorganic ions. Thus, the persistent criterion is not considered applicable to this inorganic salt.
B/vB criteria fulfilled?	Sodium and bicarbonate ions are essential to all living organisms and its

	extracellular concentrations are actively regulated. Thus, sodium bicarbonate is not expected to bioaccumulate.
T criteria fulfilled?	The 21 d chronic NOEC is 576 mg/L for Daphnia. Thus, sodium bicarbonate does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	March 2019

References

1. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2015, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
2. IPCS Sodium Bicarbonate, Retrieved 2015: http://www.inchem.org
3. OECD (2008). Screening Information Dataset (SIDS) Initial Assessment Report for Sodium Bicarbonate (CAS No. 144-55-8).
4. FSANZ 2014, Food Standards Australia New Zealand Food Additives – Alphabetical list.
5. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.nicnas.gov.au
6. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme

AECOM Imagine it. Delivered.

Toxicity Summary - Sodium carbonate

Chemical and Physical Properties[1,2,3,4,6]	
CAS number	497-19-8
Molecular formula	Na_2CO_3
Molecular weight	105.99 g/mol
Solubility in water	215 g/l at 20 °C
Melting point	851 °C
Boiling point	Decomposition
Vapour pressure	No data found
Henrys law constant	No data found
Explosive potential	It reacts violently with acids and reacts with magnesium, phosphorous pentoxide causing explosion hazard
Flammability potential	Reacts with fluorine causing fire hazard
Colour/Form	White powder
Overview	Sodium carbonate has been reviewed in the OECD-SIDS program (OECD, 2002a,b).Sodium carbonate is a strong alkaline compound with a pH of 11.6 for a 0.1M aqueous solution. The pKa of carbonate (CO3 2-) is 10.33, which means that at a pH of 10.33 both carbonate and bicarbonate are present in equal amounts. In water, sodium carbonate dissociates into sodium ion (Na+) and carbonate (CO3 2-). The carbonate ions will react with water, resulting in the formation of bicarbonate and hydroxide, until equilibrium is established. Sodium carbonate is used in many countries (e.g. U.S. and EU) as a food additive. It is regarded as a 'Generally Recognised as Safe' (GRAS) substance in food with no limitation other than current good manufacturing practice. Sodium carbon is extensively used across a range of industries and processes such as in the manufacturing of sodium salts, glass, soap/detergents and aluminium
Environmental Fate[1,2,3,4]	
Soil/Water/Air	The high water solubility and low vapor pressure indicate that sodium carbonate will be found predominantly in the aquatic environment. In water, sodium carbonate dissociates into sodium (Na+) and carbonate (CO3 2-) and both ions will not adsorb on particulate matter or surfaces and will not accumulate in living tissues.
Human Health Toxicity Summary[1]	
Chronic Repeated Dose Toxicity	No chronic oral and dermal data are available. Due to the biological importance of the products formed by the stomach acid (biocarbonate and carbon dioxide), systemic toxicity is not expected. In rats, histopathological changes of the respiratory tract and the lungs were seen following repeated inhalation exposure to sodium carbonate (70 mg/m^3 aqueous sodium cabonate at pH 11.6 for 3.5 months) and potassium carbonate (0.4 mg/L potassium carbonate at pH 9.9 for 21days). These effects were considered local responses to the high alkalinity of this group of chemicals (OECD, 2002; REACHa; REACHb).
Carcinogenicity	No data are available. Based on the available data from carcinogenicity studies with related substances (sodium bicarbonate and potassium bicarbonate), the chemicals in this group are not considered carcinogenic (OECD, 2002; REACHa; REACHb). Carbonate ions are neutralised under physiological conditions to form bicarbonate ions and/or carbon dioxide, which are major products of all human metabolic activities; therefore, systemic toxicity is not expected.

Mutagenicity/ Genotoxicity	Based on the available data, this chemical is not considered to be genotoxic (OECD, 2002; REACHa; REACHb). Carbonate ions are neutralised under physiological conditions to form bicarbonate ions and/or carbon dioxide, which are major products of all human metabolic activities; therefore, systemic toxicity is not expected.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Based on the limited information available, this chemical does not show specific reproductive or developmental toxicity (OECD, 2002; REACHa; REACHb). Carbonate ions are neutralised under physiological conditions to form bicarbonate ions and/or carbon dioxide, which are major products of all human metabolic activities; therefore, systemic toxicity is not expected.
Acute Toxicity	In animal tests, this chemical was of low acute toxicity following oral exposure. The median lethal dose (LD50) was >2000 mg/kg bw in rats (OECD, 2002; REACHa; REACHb).The majority of the animals that died following acute oral exposure to sodium carbonate at concentrations up to 2600 mg/kg/bw showed oral or nasal discharge, lesions in the liver, mottled lungs, mottled or pale kidneys and a red or partly gas-filled gastro-intestinal tract. In animal tests, this chemical was of low acute toxicity following dermal exposure. The median lethal dose (LD50) was >2000 mg/kg bw in rats (OECD, 2002; REACHa; REACHb). No systemic effects were observed following dermal exposure to sodium carbonate. Local severe skin irritation (severe erythema and oedema) was seen at the application site (OECD, 2002; REACHa; REACHb). In animal tests, this chemical was of low acute toxicity following inhalation exposure. The median lethal dose (LC50) was >2000 mg/m^3 in rats (OECD, 2002; REACH, a & b). Signs of respiratory impairment including dyspnoea, wheezing, excessive salivation and a distended abdomen were observed immediately after inhalation exposure to sodium carbonate of up to 2300 mg/m^3. Excessive salivation, repeated swallowing and a lack of appetite were observed 2–5 hours after exposure. Animals that died had lesions in the anterior trachea, posterior pharynx and larynx, along with an accumulation of mucus, vesiculation and mucosal oedema (REACHa).
Irritation	Sodium carbonate is classified as hazardous with the risk phrase 'Irritating to eyes' (Xi; R36) in HSIS (Safe Work Australia). However, in several eye irritation studies in rabbits, sodium carbonate was found to be severely irritating to the eyes, with effects including conjunctivitis, marked corneal opacity and iritis, which persisted for seven days (REACHa; REACHb). The available data support an amendment to the current HSIS eye irritation classification for sodium carbonate.
Sensitisation	Based on the limited data available, sodium carbonate is not considered to be skin sensitisers (OECD, 2002; REACHa; REACHb). No structural flags for sensitisation are present.
Health Effects Summary	The critical health effects for risk characterisation include serious eye damage and respiratory irritation because of the high basicity of the chemicals in this group. Skin irritation and corrosion of eyes and mucous membranes are also of concern where long-term exposure to the solid or concentrated solutions may occur. These effects are particularly relevant to domestic use of the chemicals. Sodium carbonate was not genotoxic or carcinogenic. Reproductive toxicity studies are not available; however, no effects on reproductive organs were noted when rats were exposed to sodium carbonate aerosol for over three months. Developmental studies with rats did not show any toxicity.
Key Study/Critical Effect for Screening Criteria	A No Observed Adverse Effect Level (NOAEL) was not available. Based on the absence of adverse effects observed in a repeat dose inhalation toxicity study, for the purposes of quantifying potential health risk, the highest dose tested in the inhalation exposure study in rats of 70 mg/m^3 (equivalent to 9.67 mg/kg bw/day) is used in the human health risk assessment.
Ecological Toxicity [1,2,3,4]	
Aquatic Toxicity	The acute 96-hour LC50 to three sizes of Bluegill sunfish (*Lepomis macrochirus*) exposed to sodium carbonate is 300 mg/L for all sizes. The acute 96-hour LC50 to mosquitofish (*Gambusia affinis*) is 740 mg/L. The acute 48-hour EC50 value to the invertebrate *Ceriodaphnia* cf. *dubia* is from 200 to 227 mg/L.

Determination of PNEC aquatic	PNECaquatic: Experimental results are available for two trophic levels. Acute E(L)C50 values are available for fish (300 mg/L) and *Ceriodaphnia* (200 mg/L). On the basis that the data consists of short-term results from two trophic levels, an assessment factor of 1,000 has been applied to the lowest reported effect concentration of 200 mg/L for Daphnia. The PNECaquatic is 0.2 mg/L.
Current Regulatory Controls[1]	
Australian Hazard Classification	Sodium carbonate is classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia): 'Xi; R36 (Irritating to eyes)'.
Australian Occupational Exposure Standards	Sodium carbonate has an exposure standard of 7.5 mg/m^3 (5 ppm) time weighted average (TWA) and 15 mg/m^3 (10 ppm) short-term exposure limit (STEL) (Safework Australia).
International Occupational Exposure Standards	Occupational exposure standard limits for sodium and potassium carbonate recommended by other countries are provided below (Galleria Chemica, 2013): US Dept of Energy (DOE) Temporary Emergency Exposure Limits (TEELs): Sodium carbonate: TEEL-0 = 10 mg/m^3 , TEEL-1 = 30 mg/m^3 , TEEL-2 = 50 mg/m^3, TEEL-3 = 500 mg/m^3 No other country has an occupational exposure limit specifically for sodium and potassium carbonate, although many countries have assigned a generic TWA exposure limits of 10 mg/m^3 (inhalable dust), and 3 mg/m^3 (respirable dust) for particles not otherwise classified (PNOC).
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[4,6]	
P/vP Criteria fulfilled?	Not applicable, inorganic substance, ubiquitous in environment.
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to these inorganic ions.
T criteria fulfilled?	No chronic toxicity data exist; however, the acute EC(L)50s are >0.1 mg/L. Thus, does not meet the screening criteria for toxicity
Overall conclusion	Not PBT
Revised	March 2019

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier II Assessment for Alkaline Salts-Carbonates: Retrieved 2019: https://www.nicnas.gov.au
2. HSDB Hazardous Substances Data Bank. U.S. National Library of Medicine, < http://toxnet.nlm.nih.gov/>,
3. OECD (2011) SIDS Initial Assessment Report for SIAM 15 (OECD SIDS). *Sodium carbonate: CAS Nº:497-19-8*. United Nations Environment Programme (UNEP) Publications. From http://www.chem.unep.ch/irptc/sids/OECDSIDS/Naco.pdf,
4. ICPS (2004). *Sodium carbonate (anhydrous): Summary*. October 2004. International Programme on Chemical Safety and the Commission of the European Communities (IPCS and CEC). From http://www.inchem.org/documents/icsc/icsc/eics1135.htm
5. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme
6. ECHA REACH, Sodium carbonate, Retrieved 2019: https://echa.europa.eu/

AECOM Imagine it. Delivered.

Toxicity Summary - PERFORMATROL®

Chemical and Physical Properties[1,2]	
CAS number	Not provided
Molecular formula	No data available.
Molecular weight	No data available.
Solubility in water	Water soluble
Melting point	No data available.
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	Clear, colourless, odourless, viscous liquid
Overview	PERFORMATROL® shale stabilizer is a low weight polymer that stabilizes reactive clays and shale by inhibiting the uptake of water and thereby mitigating their swelling or dispersion tendencies. PERMORMATROL shale stabilizer can also flocculate any dispersed clays or colloidal particles and aid their removal by solids control equipment. PERFOMATROL shale stabilizer is effective in freshwater or monovalent brines, is shear thinning, provides lubricity, has a low environmental toxicity, is highly biodegradable and is non-hazardous to rig personnel. PERFORMATROL shale stabilizer is stable to 250°F (121°C) but may achieve higher temperature stability with the use of oxygen scavengers.
Environmental Fate	
Soil/Water/Air	No data available.
Human Health Toxicity Summary[1,2]	
Chronic Repeated Dose Toxicity	No data available to indicate product or components present at greater than 0.1% are chronic health hazards.
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	No data available.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No data available.
Acute Toxicity	No data available.
Irritation	Non-irritating to rabbit's eye.
Sensitisation	No data available.
Health Effects Summary	No data available.
Key Study/Critical Effect for Screening Criteria	No data available.

Ecological Toxicity	
Aquatic Toxicity	The polymers are expected to be a low concern for toxicity to aquatic organisms. Due to their poor solubility and high molecular weights, they are not expected to be bioavailable.
Determination of PNEC aquatic	No PNEC values were calculated.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Expected to be highly biodegradable.
B/vB criteria fulfilled?	Polymers are expected to have very high molecular weights and poor water solubility. They are not expected to be bioavailable, hence the polymers do not meet the criteria for bioaccumulation.
T criteria fulfilled?	There are no aquatic toxicity studies on this polymer. Polymers are expected to have low aquatic toxicity because of their very high molecular weights and poor water solubility. As such, the polymers do not meet the criteria for toxicity.
Overall conclusion	Not PBT
Revised	February 2020

References

1. PERFORMATROL®, Product Data Sheet, Haliburton, Dated: 8/31/2010
2. PERFORMATROL®, Safety Data Sheet, Haliburton, Revision date: 30 September 2015, Revision number: 24

Toxicity Summary - Hexadec-1-ene

Chemical and Physical Properties[1,2,3]	
CAS number	629-73-2
Molecular formula	C16H32
Molecular weight	224.42
Solubility in water	0.00144 at 25°C
Melting point	4.1
Boiling point	284.9 at 1013 hPa
Vapour pressure	0.00352 hPa at 25°C
Henrys law constant	0.541 – 16.9 atm-m^3/mole
Explosive potential	No data available
Flammability potential	No data available
Colour/Form	Hexadec-1-ene are liquids at room temperature.
Overview	Hexadec-1-ene also known as 1-hexadecene are mono-olefins. It is an alkene in the C6-C18 range. These products are produced commercially in closed systems and are used primarily as intermediates in the production of other chemicals. No non-intermediate applications have been identified. Any occupational exposures that do occur are most likely by the inhalation and dermal routes.
Environmental Fate[1]	
Soil/Water/Air	Members of this category do not contain any hydrolysable functional groups, so will not undergo hydrolysis. Category members with carbon numbers from C6 to C24 have been shown to be readily biodegradable in biodegradation screening tests. The estimated half-life of 1-hexene in air is 10.2 hours. The soil adsorption coefficients (Koc) range from 149 for C6 to 230,800 for C18, indicating increasing partitioning to soil/sediment with increasing carbon number. It is expected that C16-C18 olefins would partition primarily to soil. Volatilization from water is predicted to occur rapidly (hours to days).
Human Health Toxicity Summary[1,2,3]	
Chronic Repeated Dose Toxicity	Repeated-dose studies, using the inhalation (C6 alpha), dermal (C12-C16), or oral (C6 alpha and internal linear/branched; C8 and C14 alpha; and C16, C18 and C20-C24 internal linear/branched) routes of exposure, have shown comparable levels of low toxicity in rats. In females, alterations in body and organ weights, changes in certain clinical chemistry/hematology values, and liver effects were noted (NOELs of ≥ 100 mg/kg oral or ≥ 3.44 mg/L (1000 ppm) inhalation). In males, alterations in organ weights, changes in certain clinical chemistry/hematology values, liver effects, and male rat-specific kidney damage that is likely associated with the alpha 2- globulin protein were noted (LOELs ≥ 100 mg/kg oral only). The male rat kidney damage was seen in oral studies with C6, C8 and C14 linear alpha olefins and C6 internal branched olefins, but was not seen in studies with C16/C18 or C20 - C24 internal linear/branched olefins. The noted liver effects were seen in oral studies with C14 alpha olefins (minimal-to-mild hepatocyte cytoplasmic vacuolation with increased liver weight in males and females) and with C20-C24 internal olefins (minimal centrilobular hepatocyte hypertrophy with increased liver weight in females only). No effects were present in the study with C20-C24 internal olefins following a 4-week recovery period, indicating reversibility of the observed effects. These liver effects seen only with the larger molecules may be indirect effects of an intensified liver burden, rather than a direct toxic effect of the olefin. Based on evidence from neurotoxicity screens included in repeated dose studies with C6 and C14 alpha olefins and with C6, C16/C18 and C20-C24 internal linear/branched olefins, the category members are not neurotoxic.

Carcinogenicity	No carcinogenicity tests have been conducted on C6 – C18 alpha or internal olefins; however, there are no structural alerts indicating a potential for carcinogenicity in humans.
Mutagenicity/ Genotoxicity	Based on the weight of evidence from studies with alpha and internal olefins, category members are not genotoxic.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Based on evidence from reproductive/developmental toxicity screens in rats with C6 and C14 alpha olefins and C6 and C18 linear/branched internal olefins, along with the findings of no biologically significant effects on male or female reproductive organs in repeated dose toxicity studies, the category members are not expected to cause reproductive or developmental toxicity.
Acute Toxicity	Olefins (alkenes) ranging in carbon number from C6 to C24, alpha (linear) and internal (linear and branched) demonstrate low acute toxicity by the oral, inhalation and dermal routes of exposure: Rat oral LD50 >5 g/kg; rat 4-hr inhalation LC50 range = 110 mg/L (32,000 ppm) to 6.4 mg/L (693 ppm) for C6 to C16; and rat/rabbit dermal LD50 > highest doses tested (1.43 - 10 g/kg).
Irritation	These materials are not eye irritants. Prolonged exposure of the skin for many hours may cause skin irritation.
Sensitisation	These materials are not skin sensitizers.
Health Effects Summary	Olefins (alkenes) ranging in carbon number from C6 to C24, alpha (linear) and internal (linear and branched) demonstrate low acute and chronic toxicity by the oral, inhalation and dermal routes of exposure.
Key Study/Critical Effect for Screening Criteria	The repeated dose toxicity in rats was considered the most sensitive endpoint with a NOEL of 100 mg/kg.
Ecological Toxicity [1,2,3]	
Aquatic Toxicity	Short term toxicity 96-hr LC50 > solubility Actual concentration negligible. Fish 96-hr LL0 = 1000 mg/L (nominal) Long term toxicity: NOEC (21 days) 19.4 µg/L (invertebrates)
Determination of PNEC aquatic	An assessment factor of 1000 is applied to the lowest NOEC of 19.4 µg/L (invertebrates). A PNECaqua of 0.0019 µg/L was derived.
Current Regulatory Controls[4]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1,2]	
P/vP Criteria fulfilled?	No. Readily biodegradable. The C6-C18 olefins have been shown to degrade to an extent of approximately 8 to 81% in standard 28-day biodegradation tests.
B/vB criteria fulfilled?	No. Based on calculated bioconcentration factors, hexadec-1-ene are not expected to bioaccumulate (BCF = 71).

T criteria fulfilled?	No. Chronic toxicity data >0.01 mg/L in fish, thus the substance does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	December 2021

References

1. ECHA REACH, Hexadec-1-ene, Retrieved 2021: https://echa.europa.eu/
2. OECD (2005) SIDS Initial Assessment Profile on Higher Olefins
3. NCBI, 2021. National Center for Biotechnology Information. Available at: https://pubchem.ncbi.nlm.nih.gov/. Retrieved December 2021.
4. ANZG, 2018. Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Australian and New Zealand Governments and Australian state and territory governments, Canberra ACT, Australia. Available at www.waterquality.gov.au/anz-guidelines.
5. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
6. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
7. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.

Toxicity Summary - Lead

Chemical and Physical Properties[1,2,3,4]	
CAS number	7439-92-1
Molecular formula	Pb
Molecular weight	207.2
Solubility in water	Insoluble
Melting point	326 °C at 101.3 kPa
Boiling point	600 °C at 101.3 kPa
Vapour pressure	0
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	No data available
Colour/Form	Blueish-white metal with bright lustre, very soft, highly malleable
Overview	Lead is a naturally occurring element found in the Earth's crust at an average concentration of approximately 15 to 20 mg/kg. Lead is used principally in the production of batteries, metal alloys, X-ray shielding materials, ammunition, chemical resistant linings and pigments. It has also been used historically as an additive in petrol and also in many paints. Lead is a poor conductor of electricity and is very resistant to corrosion. Lead is rarely found in its metallic form in nature and commonly occurs as a mineral with sulphur or oxygen.
Environmental Fate[1]	
Soil/Water/Air	The atmosphere is the main environmental transport media for lead that is deposited onto surface water and soils. Upon release to the atmosphere, lead particles are dispersed and ultimately removed from the atmosphere by wet or dry deposition. Lead deposition is typically greatest closer to lead emission sources. An important factor in determining the atmospheric transport of lead is particle size distribution. Large particles settle out of the atmosphere more rapidly and are deposited relatively close to emission sources and smaller particles may be transported much farther distances. After deposition, particles may be resuspended and redeposited. The cycling of lead in aquatic environments is governed by chemical, biological, and mechanical processes. The exchange between sediment and surface water will be affected by pH, ionic strength, formation of organic complexes with Pb ions, and oxidation-reduction potential of the environment.
Human Health Toxicity Summary[4]	
Chronic Repeated Dose Toxicity	Oral: A lowest observed adverse effect level (LOAEL) of 200 ppm (corresponding to PbB levels of 40–60 mg/dL) was derived for lead acetate from a repeated dose toxicity study in Sprague Dawley (SD) rats following the guidelines set out in a US EPA chronic feeding study. Lead acetate was administered in drinking water (which was freely accessible [ad libitum]) to male rats (18 animals/dose group) at 0, 200, 500 or 1000 ppm per day for four, eight or 12 weeks. Decreased body weight and increased kidney weight as a percentage of body weight were reported at all dose ranges at four weeks of exposure. Dermal: In a report available on repeated dose toxicity during dermal exposure, rats were exposed to lead acetate, lead oleate, lead arsenate or tetraethyl lead for 24 hours. The test groups had lead compounds applied either directly to the skin or to skin that had been mechanically injured. Dermal absorption of lead was shown to occur in both test groups. However, comparatively greater absorption of lead was reported in the groups where the skin had been mechanically injured.

	Inhalation: Aerosolised lead nitrate was administered to mice (Swiss Webster) by inhalation at 2.5 mg/m³ per day for 14 or 28 days. It was determined, considering the total retention of the inhaled lead, that each mouse received a dose of 80 µg/day of lead. A statistically significant reduction in the relative size of the spleen and thymus in both test groups was reported when compared with the control group. Increased lung weight was noted in both test groups and an increase in lead concentration was reported in the liver, lung and kidney; although the 28-day group was noted to show a greater concentration than the 14-day group. There were no apparent differences in body weight and food consumption noted for either test group.
Carcinogenicity	A review conducted by the International Agency for Research on Cancer (IARC), indicated that there was sufficient evidence in experimental animals and limited evidence in humans for the carcinogenicity of inorganic lead compounds. The review resulted in the classification of inorganic lead compounds as probably carcinogenic to humans (Group 2A).
Mutagenicity/ Genotoxicity	Lead compounds are considered genotoxic to mammalian cells.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	In a reproductive and developmental toxicity screening test in SD rats, lead acetate was administered in drinking water to nine females at 0.6 % weight per volume (w/v) (equivalent to 502 mg/kg bw/day) on gestation days 5–21. A stillbirth rate of 19 % was recorded in the test group compared with a 2 % rate noted in the control group. The dams and offspring in the test group had PbB levels >200 µg/dL. In a subsequent reproductive and developmental toxicity screening test in SD rats, lead acetate was administered in drinking water to 10 females at 0.05 % w/v, eight females at 0.15 % w/v and nine females at 0.45% w/v, on gestation days 5–21. Stillbirth rates of 3(±3), 10(±6) and 28(±8) % were recorded for increasing dose groups respectively compared with a 4(±3) % rate noted in the control group. At birth, the male pups had PbB levels of 40(±1), 83(±8) and 120(±120) µg/dL for increasing dose groups respectively, while the female pups had PbB levels of 42(±7), 67(±16) and 197(±82) µg/dL. A developmental LOAEL of 0.05 % (equivalent to 42 mg/kg bw/day) was reported for this study. Recent studies have investigated the effect of lead exposure in occupational groups and in general populations living near industrial plants. Although the evidence reported is predominantly qualitative and dose-effect relationships have largely not been established, it has been suggested that moderately high PbB levels in humans could result in spontaneous abortion, pre-term delivery, alterations in sperm and decreased male fertility. Data pertaining to low level exposure to lead contributing to developmental toxicity in infants and young children were recently reviewed. Consensus exists between the reports, which suggest that PbB levels in humans >10 µg/dL can affect paediatric intellectual development. In addition, data regarding the effects on children of higher levels of lead exposure were reviewed. Although neurobehavioral deficits were reported in children with PbB levels <10 µg/dL, there is uncertainty regarding the reported effects of estimates. Even so, the US Centres for Disease Control and Prevention (CDC) has a reference level of 5 µg/dL, for which any levels above it is recommended that public health action be initiated.
Acute Toxicity	Lead oxides are generally demonstrated to be of low acute toxicity in animal tests following oral exposure. The rate oral medial lethal doses (LD50s) for lead oxides are generally reported to be > 2000 mg/kg bw for male and female rats. No clinical signs were reported. Several lead compounds, including lead oxides, were reported to exhibit low acute toxicity in animal tests. Dermal median lethal dose (LD50) values in rats are reported to be >2000 mg/kg bw. The rat median lethal concentrations (LC50s) for lead oxide (PbO) is reported to be > 5.05 mg/L for male and female rats. No abnormal signs were observed. Lead metal is expected to have lower bioavailability.
Irritation	Lead compounds are not considered to irritate the skin, eyes or cause serious eye damage.
Sensitisation	Non-sensitisers

Health Effects Summary	The critical health effects for risk characterisation include systemic long-term effects (reproductive and developmental toxicity, carcinogenicity and mutagenicity). The chemical may also cause harmful effects following repeated exposure and harmful systemic effects following a single exposure.
Key Study/Critical Effect for Screening Criteria	The lowest blood lead levels studied were ≤5 µg/dL which has been associated with serious adverse effects.
Ecological Toxicity[1,5]	
Aquatic Toxicity	Short-term toxicity data: LC50 (96 h) 40.8 µg/L (Fish) LC50 (48 h) 26 µg/L (Invertebrates) EC50 (72 h) 20.5 µg/L (algae) Long-term toxicity data: NOEC (53 days) 13.3 µg/L (Fish) NOEC (42 days) 5.9 µg/L (Invertebrates) EC10 (72 h) 6.1 µg/L (algae)
Determination of PNEC aquatic	The PNEC freshwater is 2.4 µg Pb/L.
Current Regulatory Controls[4,5,6,7,8,9]	
Australian Hazard Classification	Lead metal (CAS No. 7439-92-1 as lead, inorganic dusts & fumes (as Pb)) is listed in the Hazardous Substances Information System (HSIS), but no classification is specified. For classification purposes, the chemical is considered to be covered by the generic 'lead and lead compounds' classification as hazardous with the following risk phrases for human health in HSIS: Xn; R20/R22 (Harmful by inhalation and if swallowed) Xn; R33 (Danger of cumulative effects) Repr. Cat. 1; R61 (Reproductive toxicity—may cause harm to the unborn child) Repr. Cat. 3; R62 (Reproductive toxicity—possible risk of impaired fertility)
Australian Occupational Exposure Standards	Time weighted average (TWA): 0.15 mg/m^3 for lead compounds (as lead). Short-term exposure limits (STEL): No specific exposure standards are available
International Occupational Exposure Standards	For lead compounds in general, the following exposure limits were identified: TWA = 0.05 mg/m^3 [Bulgaria, Canada, China, Italy, Malaysia, USA] TWA = 0.10 mg/m^3 [Austria, New Zealand, Republic of South Africa, Sweden] TWA = 0.15 mg/m^3 [Argentina, Egypt, EU (Directive 98/24/EC), Malta, Singapore] TWA = 0.20 mg/m^3 [Thailand] STEL: 0.10 mg/m^3 [Austria] STEL: 0.15 mg/m^3 [Canada] STEL: 0.45 mg/m^3 [Argentina, Egypt]
Australian Food Standards	The tolerable limit for lead is 25 µg/kg bw/week.
Australian Drinking Water Guidelines	Based on health considerations, the concentration of lead in drinking water should not exceed 0.01 mg/L.
Aquatic Toxicity Guidelines	A high reliability freshwater trigger value for lead of 3.4 µg/L was calculated using the statistical distribution method at 95% protection. A marine high reliability trigger value for lead of 4.4 µg/L was calculated using the statistical distribution method with 95% protection.
PBT Assessment[1]	
P/vP Criteria fulfilled?	Not applicable (lead as a metal do not degrade and traditional persistence measures used for organic substances do not equally apply to metals).
B/vB criteria fulfilled?	Not applicable. Due to their natural occurrence, biota will naturally accumulate metals at least to some degree without deleterious effect and non-essential metals such as lead are homeostatically regulated to some extent.
T criteria fulfilled?	Yes. Lead fulfils the toxicity criteria based on the most sensitive NOEC, HC5-50 and PNEC values, which are below 10µg/L.

Overall conclusion	It is not currently possible to categorise the environmental hazards of metals and other inorganic chemicals according to standard persistence, bioaccumulation and toxicity (PBT) hazard criteria. These criteria were developed for organic chemicals and do not take into account the unique properties of inorganic substances and their behaviour in the environment.
Revised	December 2021

References

1. ECHA REACH, Lead, Retrieved 2021: https://echa.europa.eu/
2. USEPA, 2021. Regional Risk Levels. November 2021. https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables. Retrieved December 2021.
3. ATSDR, 2020. Toxicological Profile for Lead. Agency for Toxic Substances and Disease Registry. August 2020.
4. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier II Assessment for Lead: Retrieved 2021: https://www.industrialchemicals.gov.au/.
5. ANZG, 2018. Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Australian and New Zealand Governments and Australian state and territory governments, Canberra ACT, Australia. Available at www.waterquality.gov.au/anz-guidelines.
6. Food Standards Australia New Zealand (FSANZ) 20th Australian Total Diet Survey. Retrieved 2021: https://www.foodstandards.gov.au/publications/pages/20thaustraliantotaldietsurveyjanuary2003/20thaustraliantotaldietsurveyfullreport/partb20thatds/partbmetals.aspx.
7. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.7 Updated January 2022. National Water Quality Management.
8. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
9. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.

Toxicity Summary - Phosphorodithioic acid, mixed O,O-bis(isobutyl and pentyl) esters, zinc salts

Chemical and Physical Properties[1,2,3]	
CAS number	68457-79-4
Molecular formula	C16H36O4P2S4Zn
Molecular weight	548.1
Solubility in water	1.658 g/L at 22°C and pH 5
Melting point	-21°C
Boiling point	Decomposes before boiling
Vapour pressure	0.003 - 0.107 Pa at 25 - 70°C
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	No data available
Colour/Form	Viscous, amber-coloured liquid capable of producing an odour characteristic of sulphur-containing compounds
Overview	The uses and applications for this substance include: Antioxidant; lubricating oil additive for corrosion and wear resistance; accelerator for rubber. A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health.
Environmental Fate[1]	
Soil/Water/Air	The test substance is hydrolytically stable at pH 4, 7 and 9 as defined by the OECD 111 criterion of a < 10% change in the concentration of the parent compound. The substance has a low octanol water partition coefficient. It is not readily biodegradable under test conditions. Based on the weight of evidence from read across to structurally similar ZDDP substances with BCF data in fish (from Japanese MITI data, US EPA database, CAESAR database), measured Log Kow data, and QSAR predictions, this substance is expected to have low bioaccumulation potential.
Human Health Toxicity Summary[1,2,3]	
Chronic Repeated Dose Toxicity	The oral repeat dose toxicity was evaluated with rats at doses as high as 160 mg/kg/day for up to 52 consecutive days in accordance with OECD 422. Substance-related toxicity was limited to moribundity, adverse clinical signs, and epithelial hyperplasia, hyperkeratosis, and inflammation of the stomach. The NOAEL for systemic toxicity was 160 mg/kg/day. The NOEL for portal of entry irritation and related secondary effects parental toxicity was 40 mg/kg/day.
Carcinogenicity	Not expected to be carcinogenic.
Mutagenicity/ Genotoxicity	No non-threshold mode of action is associated with this substance, in particular, the test substance has no genotoxic potential. The weight of evidence suggests that the test substance is not expected to present a significant risk for mutagenicity or carcinogenicity in humans,
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	The reproductive toxicity of this substance was evaluated with rats at doses as high as 160 mg/kg/day for up to 52 consecutive days in accordance with OECD 422. The NOAEL and NOEL for reproductive fertility and neonatal toxicity was determined to be 160 mg/kg/day.
Acute Toxicity	This substance does not show any evidence of toxicity via the oral route of exposure in animals when tested in accordance with OECD Guideline 401. The rat oral LD50 is 3,600 mg/kg in male rats. Sublethal effects of lethargy, diarrhea, piloerection, chromodacryorrhea, chromorhinorrhea and ptosis were observed. Necropsy observations included lung and gastrointestinal abnormalities, but no

	specific organ toxicity is significant; all animals showed expected bodyweight gain during the course of study. This substance does not show adverse toxicity effects via the dermal route of exposure in animals when tested in accordance with OECD Guideline 402. The rat dermal LD50 is greater than 20,000 mg/kg in rabbits. No mortality occurred. Toxic signs observed included lethargy, diarrhea, ataxia, ptosis, alopecia, emaciation, and yellow nasal discharge. No specific organ toxicity is evident.
Irritation	The substance is a skin and eye irritant.
Sensitisation	Not a skin sensitizer.
Health Effects Summary	The substance causes skin and eye irritation. Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.
Key Study/Critical Effect for Screening Criteria	The oral repeated dose toxicity in rats was considered the most sensitive endpoint with a NOEL of 160 mg/kg bw/day.
Ecological Toxicity [1,2,3]	
Aquatic Toxicity	Short term toxicity: LC50 (4 days): 46 mg/L (fish) LL50 (4 days): 4.5 mg/L (fish) EL50 (48 h): 23 mg/L (invertebrates) EL50 (72 h): 21 mg/L (algae) Long term toxicity: NOEC (21 days): 0.4 mg/L (invertebrates)
Determination of PNEC aquatic	Data from short-term tests with three trophic levels and one long-term test on invertebrates are available. An assessment factor of 100 is applied to the lowest NOEC of 0.4 mg/L (invertebrates). A PNECaqua of 0.004 mg/L was derived.
Current Regulatory Controls[4,5,6]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1]	
P/vP Criteria fulfilled?	Yes. Not readily biodegradable.
B/vB criteria fulfilled?	No. Based on the measured log Kow value of less than 3, this substance is not bioaccumulative.
T criteria fulfilled?	Not applicable. Chronic toxicity data >1 mg/L in invertebrates, thus sodium hydroxide does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	December 2021

References

1. ECHA REACH, Phosphorodithioic acid, mixed O,O-bis(iso-Bu and pentyl) esters, zinc salts, Retrieved 2021: https://echa.europa.eu/
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2021: https://www.industrialchemicals.gov.au/.
3. NCBI, 2021. National Center for Biotechnology Information. Available at: https://pubchem.ncbi.nlm.nih.gov/. Retrieved December 2021
4. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
5. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.
6. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.

Toxicity Summary - Sulphur dioxide

Chemical and Physical Properties[1,2,3]	
CAS number	7446-09-5
Molecular formula	SO_2
Molecular weight	64.064
Solubility in water	114 g/L at 20 °C
Melting point	-75.5 - -74.5 °C
Boiling point	-10.05 - -10 °C at 101.3 - 101.325 kPa
Vapour pressure	327.1 kPa at 20 °C
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	No data available
Colour/Form	Colourless gas with a characteristic, irritating, pungent odour
Overview	Sulphur dioxide is a colourless gas with a pungent odour. It is a liquid when under pressure. Sulphur dioxide dissolves in water very easily. It cannot catch fire. Sulphur dioxide in the air results primarily from activities associated with the burning of fossil fuels (coal, oil) such as at power plants or from copper smelting. In nature, sulphur dioxide can be released to the air, for example, from volcanic eruptions.
Environmental Fate[1,3]	
Soil/Water/Air	Once released into the environment, sulphur dioxide moves to the air. In the air, sulphur dioxide can be converted to sulfuric acid, sulphur trioxide, and sulphates. Sulphur dioxide dissolves in water. Once dissolved in water, sulphur dioxide can form sulphurous acid. Soil can absorb sulphur dioxide, with uptake being dependent on the pH and moisture content of the soil.
Human Health Toxicity Summary [1,2,3,4]	
Chronic Repeated Dose Toxicity	Based on the available data, repeated inhalation exposure to sulphur dioxide is associated with local effects. The airway response to the chemical indicates a defence mechanism to local irritation, such as mild to moderate pathological changes in tracheal and lung tissues, that may lead to persistent defects with prolonged exposure. In a non-guideline study, three groups of male Sprague-Dawley (SD) rats (70/group) were treated with 0, 10, or 30 ppm (0, 28.2, or 84.6 mg/m^3) sulphur dioxide for 21 weeks (six hours/day, five days/week) by whole body exposure. Mild to moderate pathological changes in tracheal and lung tissues were detected at the 10 and 30 ppm groups, with no significant recovery detected in the respiratory tract during the four-week post-exposure period. In another non-guideline study, male SD rats were exposed to 1 ppm (2.8 mg/m^3) sulphur dioxide for either four or eight months (five hours/day, five days/week) by whole body exposure. Temporary bronchiolar epithelial hyperplasia was observed at four months only. Respiratory function was impaired at four months (not examined at eight months). No other details of the study were provided. No adverse systemic effects were reported in multiple non-guideline chronic or subchronic studies in dogs, rats, guinea pigs and cynomolgous monkeys treated daily for various durations and a range of concentrations of the chemical.
Carcinogenicity	Based on the available data, the chemical is not considered to be carcinogenic. In a non-guideline study, male SD rats were exposed to 10 or 30 ppm (28.2 or 84.6 mg/m^3) sulphur dioxide for 21 weeks (six hours/day, five days/week) and followed for up to two years. The rats exposed to the chemical had normal survival and showed increases in tumour occurrence over their lifetimes. Lack of carcinogenic potential was supported by another nonguideline study, where no increases in lung tumours were seen in rats (sex and strain not specified) exposed chronically to 10

	ppm sulphur dioxide for 534 days (five hours/day, five days/week) and observed for further 260 days. In a non-guideline study, male and female mice (strain not specified) treated with daily short-term exposures (five minutes/day, five days/week) to a high concentration of 500 ppm (1410 mg/m^3) sulphur dioxide over their lifetime (300 days or more) had increased incidence and larger primary lung tumours at an earlier age when compared to untreated controls.
Mutagenicity/ Genotoxicity	Based on the available data, the chemical is potentially mutagenic.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Based on the available data, the chemical is not considered to be a reproductive toxicant. Some evidence exists for the chemical to potentially cause developmental toxicity.
Acute Toxicity	In a non-guideline study, male CD-1 rats (8/dose) were exposed to sulphur dioxide gas concentrations of 224, 593, 965, 1168, or 1319 ppm (632, 1670, 2720, 3295, or 3720 mg/m^3) for four hours and observed for 14 days following exposure. The median lethal concentration (LC50) was between 965–1168 ppm/4 hours. No deaths occurred at the 593 ppm concentration, while the 1319 ppm concentration was lethal to 100% of rats. Clinical signs included respiratory difficulties followed by exhaustion and death. In another non-guideline study, male Syrian hamsters were exposed to sulphur dioxide gas at concentrations of 40, 200, or 400 ppm (113, 564, or 1130 mg/m^3) for 4–6 hours. All hamsters died due to development of respiratory distress following exposure to 400 ppm of the chemical. No deaths occurred at 40 and 200 ppm. Ciliary loss in the trachea was observed at 40 and 200 ppm. The calculated LC50 values of sulphur dioxide for male Swiss mice were 9,600 ppm (27,080 mg/m^3)/ 5 min, 4,800 ppm (13,540 mg/m^3)/ 10-min, 3,800 ppm (10,720 mg/m^3)/ 15-min, and 3,400 ppm (9,590 mg/m^3)/ 30-min. Clinical signs and cause of deaths were not reported.
Irritation	Sulphurous acid, which is formed when sulphur dioxide comes in contact with moist surfaces, is the primary cause of irritation and corrosivity of the chemical
Sensitisation	Available data suggest potential respiratory sensitisation potential for the chemical. In a non-guideline study, male Dunkin-Hartley or female Dunkin-Hartley Pirbright-White guinea pigs were exposed to 0.1–16 ppm (0.28–45.1 mg/m^3) sulphur dioxide for five to eight hours a day for five consecutive days, and additionally exposed to ovalbumin aerosol on days 3, 4 and 5 for 45 minutes/day, followed by provocation on day 13 by 1 % ovalbumin aerosol. Exposure to the chemical at the low concentration of 0.1 ppm significantly enhanced the development of ovalbumin-induced asthmatic reactions (increases in airway resistance and infiltration of inflammatory cells and epithelial damage in bronchial and lung tissue) in guinea pigs. Exposure to sulphur dioxide alone had no effect. In another non-guideline study, male Hartley guinea pigs (12/group) were exposed to sulphur dioxide. The initial phase consisted of intraperitoneal (i.p.) injection of 10 mg Candida albicans in physiological saline vehicle. Two weeks later, the guinea pigs were exposed to 5 ppm of the chemical 30 times (four hours/day, five days/week). Two weeks after exposure to the chemical, the animals were exposed to C. albicans for 30 minutes. Exposure of guinea pigs to the chemical increased sensitivity to C. albicans and resulted in significantly increased numbers of animals with prolonged expiration and/or inspiration and in a decrease of respiratory rate and even mortality in 25% of sulphur dioxide exposed animals.
Health Effects Summary	The critical health effects for risk characterisation include local effects (corrosive effects on the eyes, skin and respiratory tract).
Key Study/Critical Effect for Screening Criteria	An minimal risk level (MRL) of 0.01 ppm has been derived for acute-duration exposure (14 days or less) to sulphur dioxide. This MRL is derived from the study by Sheppard et al. (1981) in which exercising mild asthmatics were exposed to ≥0.1 ppm sulphur dioxide for 10 minutes. The two most sensitive subjects developed slight bronchoconstriction after inhaling 0.1 ppm sulphur dioxide (ATSDR).
Ecological Toxicity [1,2,3]	
Aquatic Toxicity	Sulphur dioxide, however, is a gaseous substance and does not remain present in the aquatic environment under this form: Sulphur dioxide will react with water (or water vapour) to form sulphurous acid. Consequently, an E(L)C50, EC10 or NOEC

	expressed as mg SO2/L cannot be determined (i.e., no acute or chronic reference values can be generated). Secondly, as SO2 is not present in the aquatic compartment for a relevant time period, this substance will not cross biological membranes, or will not interact with it in another way.
Determination of PNEC aquatic	Not determined
Current Regulatory Controls[1,5]	
Australian Hazard Classification	The chemical is classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HSIS) (Safe Work Australia): Acute toxicity – category 3 Skin corrosion – category 1B Gases under pressure
Australian Occupational Exposure Standards	The following exposure standards are identified (Galleria Chemica): Time-weighted average (TWA) of 5.2 mg/m^3 (2 ppm) Short-term exposure limits (STEL) 13 mg/m^3 (5ppm)
International Occupational Exposure Standards	The following exposure standards are identified (Galleria Chemica): An exposure limit (occupational exposure limit (OEL) or TWA) of 1 – 5.3 mg/m^3 and STEL of 5-13 mg/m^3 in most countries. The STEL established by American Conference of Governmental Industrial Hygienists (ACGIH) is 0.25 ppm (0.7 mg/m^3). The chemical is included in US NIOSH Substances Immediately Dangerous to Life or Health (IDLH) List at a level of 100 ppm (282 mg/m^3). US Department of Energy (DOE) has Temporary Emergency Exposure Limits (TEELs) for Protective Action Criteria (PAC): PAC-1 at 0.2, PAC-2 at 0.75 and PAC-3 at 30 ppm (84.6 mg/m^3).
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	Not applicable (inorganic substance, ionic species ubiquitous in environment)
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to this inorganic substance.
T criteria fulfilled?	Not applicable.
Overall conclusion	It is not currently possible to categorise the environmental hazards of inorganic chemicals according to standard persistence, bioaccumulation and toxicity (PBT) hazard criteria. These criteria were developed for organic chemicals and do not take into account the unique properties of inorganic substances and their behaviour in the environment.
Revised	December 2021

References

1. ECHA REACH, Sulfur dioxide, Retrieved 2021: https://echa.europa.eu/
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Sulfur dioxide: Human health tier II assessment: Retrieved 2021: https://www.industrialchemicals.gov.au/.
3. ATSDR, 1998. Toxicological Profile for Sulfur Dioxide. Agency for Toxic Substances and Disease Registry. December 1998.
4. Sheppard D, Saisho A, Nadel JA, et al. 198 1. Exercise increases sulfur dioxide-induced bronchoconstriction in asthmatic subjects. Am Rev Respir Dis 123:486-491.
5. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.

Toxicity Summary - 1,3,5-Triazine-1,3,5(2H,4H,6H)-triethanol

Chemical and Physical Properties[1,2,3]	
CAS number	4719-04-4
Molecular formula	C9H21N3O3
Molecular weight	219.28
Solubility in water	Miscible at 20°C and at pH 5, 7, and 9
Melting point	-79 °C
Boiling point	110.1°C at 101.325 kPa
Vapour pressure	0 Pa at 25 °C
Henrys law constant	0 Pa m^3/mol at 25 °C
Explosive potential	Non-explosive
Flammability potential	Not classified
Colour/Form	Viscous yellow liquid
Overview	The substance in is generally used as a biocide to control bacterial growth.
Environmental Fate[1]	
Soil/Water/Air	After evaporation or exposure to the air, the substance will be rapidly degraded by photochemical processes. Based upon a calculated log Koc adsorption to soil phase is not expected. From the water surface the substance will not evaporate into the atmosphere. The substance will preferentially distribute into the compartment water.
Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	In a subchronic oral toxicity study in Wistar rats with administration of the test substance in drinking water for 3 months, the NOAEL was determined to be 64 mg/kg/day based on reduced water consumption at this dose level but without any corroborating changes in-life or pathologically (BASF SE, 2002). In a repeated dose oral toxicity 90-day study conducted according to the OECD TG 442, the chemical was administered to Wistar CrlGlxBrlHan rats (10/sex/dose) at dietary concentrations of 200 ppm (14 mg/kg bw/day in males; 21 mg/kg bw/day in females), 1000 ppm (64 mg/kg bw/day in males; 91 mg/kg bw/day in females), and 5000 ppm (285 mg/kg bw/day in males; 339 mg/kg bw/day in females). The animals were observed for signs of toxicity or mortality up to twice a day for 3 months. At the end of the study, neither mortality nor clinical symptoms of toxicity were observed, and the appearance and behaviour of the animals showed no treatment related changes. Repeat dose exposure to the chemical via dermal route is not considered to be hazardous. In a subchronic dermal toxicity 90-day study, male and female Charles River rats (10 animals per sex per dose) were treated with the chemical under semi-occlusive conditions for 6 hours/day, 5 days/week for 90 days. Doses were 0, 5, 50 or 250 mg/kg bw/day. The application site was not washed between doses. No mortality occurred during the test. There were no treatment related clinical signs. Yellow staining at the site of application in the 50 and 250 mg/kg bw/day groups was seen. In a repeated dose inhalation toxicity study (OECD Guideline 412) Wistar rats (10 animals per sex per dose) were exposed (nose only) to the aerosol chemical at 3, 10, 30 and 100 mg/m^3. The highest concentration was decreased to 50 mg/m^3 after the first exposure day for females and the second exposure day for males due to clinical signs indicative of a severe irritant response. The animals were exposed for 6 hrs/day for 5 consecutive days per week for 4 weeks. The target concentrations were maintained throughout the exposure period. Severe clinical signs of toxicity (gasping, intermittent respiration, respiration sound, red encrusted nose, hypothermia, poor general state and yellow discoloured fur), significantly reduced body weight change in males and premature death of 5 of the 10 males were observed in the highest dose group (initially 100 mg/m^3, then lowered to 50 mg/m^3). In the 30 mg/m^3 and 10 mg/m^3 groups, intermittent respiration, rales, red

	encrusted nose, squamous metaplasia occurred in all treated groups. The presence of erosion/ulceration of the larynx, squamous metaplasia of the nasal cavity, squamous metaplasia of the carina epithelium, necrosis of the u-shaped cartilage of the larynx, epithelial hyperplasia of the larynx and degeneration of the bronchial epithelium for both sexes were noted. In the lowest dose group (3 mg/m^3): multifocal squamous metaplasia of the larynx in all animals; necrosis of the u-shaped cartilage of the larynx in 1/10 males; degeneration of the bronchial epithelium in 3/10 males and 7/10 females and squamous metaplasia of the carina epithelium in 4/10 males and 3/10 females were noted). In conclusion, exposure of male and female Wistar rats to the aerosol of the chemical caused concentration-related local irritation of the respiratory tract. Systemic toxicity was not observed in clinical chemistry, haematology or in histological examinations up to 30 mg/m^3. The reduced body weight gain and premature death were considered to be associated with the severe local irritation. Based on histopathology findings in larynx, trachea and lung, a no observed adverse effect concentration (NOAEC) could not be established for the local irritation effect under the current study conditions. For systemic effects the NOAEC is 30 mg/m^3.
Carcinogenicity	Carcinogenicity studies for the chemical are not available. In a poorly documented dermal study with only limited number of animals (NMRI mice), limited scope of parameters examined and with short study duration, the chemical did not result in any carcinogenic effects. Many methodological details of the study are lacking. The test substance was applied to a shaved area of the upper part of the back. Applications, 0.15%, 1.5 % and 15% of the chemical (purity not specified) were made three times a week, over 31 consecutive weeks. All mice survived to the end of the study. Slight dysplasia was reported in two high-dose animals. Hyperplasia occurred in one mid-dose and seven high-dose mice. Three of the high-dose animals had degenerative changes (amyloid deposition) in the kidney, but not the spleen or liver. The test substance did not induce papillomas. No information is provided on clinical observations in the treated animals.
Mutagenicity/ Genotoxicity	Genotoxicity potential of the chemical was tested in several in vitro and in vivo genotoxicity tests. Based on the weight of evidence from the available data, the chemical is not considered to be genotoxic.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Studies for reproductive toxicity are not available. In a prenatal developmental toxicity study in rats, artificially inseminated female Sprague-Dawley rats (24/group) were administered the aqueous chemical (78.5% 1,3,5-tris(2-hydroxyethyl)hexahydro-1,3,5-triazine) by gavage at doses of 0, 250, 500, and 750 mg/kg/day in deionised water on gestation days 6 through 15. All animals survived the duration of the study. High dose females exhibited post-dosing salivation. Rales, laboured breathing, wheezing, and tachypnea were observed occasionally in the mid and high dose groups toward the end of the dosing period. No other clinical signs were reported. Maternal body weight gain and food consumption were significantly lower in the high dose females during the dosing period than the controls. Stomach lesions characterised by ulceration and/or scarring of the mucosa were observed in 14 of 20 high dose females. No gross abnormalities were reported in the other dosage groups. No differences were seen between the control and treated dams with respect to pregnancy rates, number of corpora lutea, implantation sites, number of live foetuses, or early and late resorptions. There were no abortions and no premature deliveries. At these doses, developmental toxicity as measured by foetal pup weight, external, or visceral, abnormalities was not seen. There were increased incidences of vestigial 14th ribs and retarded ossification of the vertebral thoracic centra which appeared to be dose-related. The effects were not statistically significant, and the incidence of these abnormalities is highly variable in rats, they are not considered treatment related. The maternal no observed adverse effect level (NOAEL) is 500 mg/kg bw/day, based on decreased body weight gain, ulcerations and/or scarring of the stomach mucosa at the higher dose. The NOAEL for developmental toxicity is 750 mg/kg bw/day.
Acute Toxicity	In the only available oral acute toxicity study (OECD Guideline 401) groups of 10 fasted Wistar rats (5 per sex) were given a single oral dose of the test substance at dose levels of 500, 1000 or 2000 mg/kg bw. Four males and all females in the 2000 mg/kg bw dose group and two males and four females in the 1000 mg/kg bw

	dose group died within two days after administration. Necroscopy findings of the animals that died included agonal congestion, erythema, erosion in the glandular stomach and discolouration of the mucosa of the forestomach and the glandular stomach. Observed sub-lethal effects included general depressed activity, staggering, paresis and diarrhoea. The median lethal dose (LD50) was calculated as 763 mg/kg bw in rats. The chemical has low acute toxicity based on results from an animal test following dermal exposure. The LD50 in rats in this study was >4000 mg/kg bw. The chemical has high acute toxicity following inhalation exposure based on results from animal tests. The median lethal concentration (LC50) in rats is 0.371 mg/L.
Irritation	The chemical did not cause irritation to the skin in rabbits exposed dermally to 0.5 mL of the unchanged substance for four hours via a test patch moistened with the substance. Slight irritation was observed in rabbits administered 0.1 mL of the chemical in the conjunctival sac of the right eye but was reversible within 8 days. No eye lesions remained in any of the test animals at the end of the three-week observation period
Sensitisation	The substance was considered to be a skin sensitiser in studies with guinea pigs. Case studies on humans have indicated that the chemical is a skin sensitising agent.
Health Effects Summary	The critical health effects for risk characterisation include acute toxicity effects from oral and inhalation exposure and skin sensitisation.
Key Study/Critical Effect for Screening Criteria	The subchronic oral repeated dose toxicity in rats was considered the most sensitive endpoint with a NOAEL of 64 mg/kg bw/day.
Ecological Toxicity[1]	
Aquatic Toxicity	Fish: LC50 (4 days) 16.07 - 240.04 mg/L LC100 (4 days) 58.9 mg/L Invertebrates: EC50 (48 h) 11.9 mg/L LC50 (48 h) 60.67 mg/L EC100 (48 h) 17.5 mg/L Algae: EC50 for freshwater algae: 6.6 mg/L EC50 for marine water algae: 21 mg/L EC10 or NOEC for freshwater algae: 3.4 mg/L EC10 or NOEC for marine water algae: 10 mg/L
Determination of PNEC aquatic	Data from short-term tests with three trophic levels are available. An assessment factor of 1000 is applied to the lowest EC50 of 6.6 mg/L (algae). A PNECaqua of 7 µg/L was derived.
Current Regulatory Controls[2,4,5,6]	
Australian Hazard Classification	The chemical is classified as hazardous, with the following risk phrases for human health in the Hazardous Substances Information System (HCIS): Skin sensitisation – category 1 Specific target organ toxicity (repeated exposure) – category 1 Acute toxicity (inhalation) - category 3 Acute toxicity (ingestion) - category 4
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	The following exposure standards are identified (Galleria Chemica). US DOE Temporary Emergency Exposure Limits (TEELs) TEEL 1: 2.3 mg/m^3; TEEL 2: 25 mg/m^3 and TEEL 3: 150 mg/m^3.

Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1]	
P/vP Criteria fulfilled?	No. Expected to be readily biodegradable.
B/vB criteria fulfilled?	No. Based on Log Kow = -2.3 - -1.3 at 24 °C and pH 5 – 9 (Log Kow < 4.2)
T criteria fulfilled?	No. Acute toxicity data >1 mg/L in fish, invertebrates and algae, thus the substance does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	December 2021

References

1. ECHA REACH, 2,2',2''-(hexahydro-1,3,5-triazine-1,3,5-triyl)triethanol, Retrieved 2021: https://echa.europa.eu/
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, 1,3,5-Triazine-1,3,5(2H,4H,6H)-triethanol: Human health tier II assessment: Retrieved 2021: https://www.industrialchemicals.gov.au/.
3. NCBI, 2021. National Center for Biotechnology Information. Available at: https://pubchem.ncbi.nlm.nih.gov/. Retrieved December 2021
4. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
5. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.
6. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.

Toxicity Summary - Zinc

Chemical and Physical Properties[1,2,3,4]	
CAS number	7440-66-6
Molecular formula	Zn
Molecular weight	65.38
Solubility in water	Insoluble
Melting point	409°C
Boiling point	No data
Vapour pressure	1 at 487°C
Henrys law constant	Not applicable
Explosive potential	No data
Flammability potential	Not flammable
Colour/Form	Bluish-white, shiny metal
Overview	Zinc is a naturally occurring element found in the earth's surface rocks. Because of its reactivity, zinc metal is not found as the free element in nature. Powdered zinc is explosive and may burst into flames if stored in damp places. Zinc is found in the air, soil, and water and is present in all foods. Metallic zinc is used in industry to coat steel and iron as well as other metals to prevent rust and corrosion. Metallic zinc is also mixed with other metals to form alloys such as brass and bronze. Metallic zinc is also used to make dry cell batteries. A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health.
Environmental Fate[3]	
Soil/Water/Air	Zinc partitions to the air, water, and soil. Zinc occurs in the environment mainly in the +2 oxidation state (ATSDR, 2005). Adsorption is the dominant fate of zinc, resulting in enrichment of zinc in suspended and bed sediments. Zinc can occur in both suspended and dissolved forms in surface water. In the aquatic environment, zinc partitions to sediments or suspended solids in surface waters through sorption onto hydrous iron and manganese oxides, clay minerals, and organic material. The transport of zinc in the aquatic environment is controlled by anion species. In natural waters, complexing agents, such as humic acid, can bind zinc. The stability of zinc complexes depends on the pH of the water and the nature of the complex. Zinc sorbs strongly onto soil particulates. The mobility of zinc in soil depends on the solubility of the speciated forms of the element and on soil properties such as cation exchange capacity, pH, redox potential, and chemical species present in soil.
Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	Following longer-term exposure to lower doses (~0.5–2 mg zinc/kg/day) of zinc compounds, the observed symptoms generally result from a decreased absorption of copper from the diet, leading to early symptoms of copper deficiency. The most noticeable manifestation of the decreased copper levels is anaemia, manifesting as decreased erythrocyte number or decreased hematocrit. High-dose zinc administration has also resulted in reductions in leukocyte number and function. Some studies have also found decreases in high-density lipoprotein (HDL) levels in humans exposed to increased levels of zinc; however, not all studies have confirmed this observation. Long-term consumption of excess zinc may also result in decreased iron stores, although the mechanism behind this effect is not presently clear.
Carcinogenicity	Available studies of zinc-induced carcinogenic effects in humans and animals following both oral or inhalation exposure have not adequately demonstrated an increase in cancer incidence following long term exposure to zinc compounds.

Mutagenicity/ Genotoxicity	Genotoxicity studies conducted in a variety of test systems have failed to provide evidence for mutagenicity of zinc. However, there are indications of weak clastogenic effects following zinc exposure.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Available studies have not presented evidence of reproductive or developmental effects in humans or animals following inhalation of zinc compounds. Effects on reproductive or developmental end points have been noted in oral-exposure animal studies, but generally only at very high doses (>200 mg/kg/day).
Acute Toxicity	The effects of inhalation exposure to zinc and zinc compounds vary somewhat with the chemical form of the zinc compound, but the majority of the effects seen will occur within the respiratory tract. Following inhalation of zinc oxide, and to a lesser extent zinc metal and many other zinc compounds, the most commonly reported effect is the development of “metal fume fever” which is characterized by chest pain, cough, dyspnoea, reduced lung volumes, nausea, chills, malaise, and leucocytosis. Symptoms generally appear a few hours after exposure and are reversible 1–4 days following cessation of exposure.
Irritation	Not irritating.
Sensitisation	Not sensitising.
Health Effects Summary	Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.
Key Study/Critical Effect for Screening Criteria	The chronic reference dose (RfD) was based on the average LOAEL of 0.91 mg/kg/day for blood effects observed in four principal studies on male and female adults.
Ecological Toxicity[1,5]	
Aquatic Toxicity	Fish: 24 µg/L (Oncorhynchus tshawytscha; from LC50) to 1316 µg/L (Ptylocheilus oregonensis; from LC50). Amphibians: Ambystoma opacum, 180 µg/L (from LOEC). Crustaceans: 5.5 µg/L (C. dubia; from LC50) to 25.3 µg/L (C. dubia). Molluscs: 54 µg/L (Dreissena polymorpha) to 11,200 µg/L (Velesunio ambigua), a NOEC of 487 µg/L was measured for Physa gyrina. Annelid: one species, Limnodrilus hoffmeisteri, 560 µg/L (from LC50).
Determination of PNEC aquatic	The PNECaquatic (freshwater) is determined to be 20.6 µg/L.
Current Regulatory Controls[5,6,7,8]	
Australian Hazard Classification	H260 (In contact with water releases flammable gases which may ignite spontaneously) H250 (Catches fire spontaneously if exposed to air) H410 (Very toxic to aquatic life with long-lasting effects)
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	An exposure limit for zinc and its inorganic compounds (inhalable fraction) (TWA) of 2 mg/m^3 and (respirable fraction) (TWA) of 0.1 mg/m^3 in Germany.
Australian Food Standards	Tolerable limit = 45 mg/person/day
Australian Drinking Water Guidelines	Based on aesthetic considerations (taste), the concentration of zinc in drinking water should be less than 3 mg/L. No health-based guideline value is proposed for zinc.
Aquatic Toxicity Guidelines	A freshwater and marine high reliability trigger value of 8 µg/L was calculated for zinc.
PBT Assessment	
P/vP Criteria fulfilled?	Not applicable (zinc is an essential element and is ubiquitous in environment).

B/vB criteria fulfilled?	No. As an essential element, zinc is commonly regulated by the organism and do not bioaccumulate or biomagnify.
T criteria fulfilled?	Not applicable. Zinc is an essential nutrient for all living organisms.
Overall conclusion	It is not currently possible to categorise the environmental hazards of metals and other inorganic chemicals according to standard persistence, bioaccumulation and toxicity (PBT) hazard criteria. These criteria were developed for organic chemicals and do not take into account the unique properties of inorganic substances and their behaviour in the environment.
Revised	December 2021

References

1. ECHA REACH, Zinc, Retrieved 2021: https://echa.europa.eu/
2. USEPA, 2021. Regional Risk Levels. November 2021. https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables. Retrieved December 2021.
3. ATSDR, 2005. Toxicological Profile for Zinc. Agency for Toxic Substances and Disease Registry. August 2005.
4. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.industrialchemicals.gov.au/.
5. ANZG, 2018. Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Australian and New Zealand Governments and Australian state and territory governments, Canberra ACT, Australia. Available at www.waterquality.gov.au/anz-guidelines.
6. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
7. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
8. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.
9. Food Standards Australia New Zealand (FSANZ) 19th ATDS Supplementary Information – Part 1. Retrieved 2021: https://www.foodstandards.gov.au/publications/documents/tables%201-8.pdf.

Toxicity Summary - Distillates (Fischer-Tropsch), C8-26-branched and linear

Chemical and Physical Properties[1,2]	
CAS number	848301-67-7
Molecular formula	Unknown or variable composition, complex reaction products or biological materials (UVCB)
Molecular weight	UVCB
Solubility in water	1 mg/L at 20°C and pH 5.1 - 5.3
Melting point	-20°C
Boiling point	218 - 357 °C at 101.1 kPa
Vapour pressure	0.54 Pa at 25°C
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	No data available
Colour/Form	Colourless, liquid, mild-paraffinic odour
Overview	Gas-to-liquid (GTL) products are synthetic hydrocarbons produced from natural gas using a Fischer–Tropsch process. This process yields a synthetic crude oil that consists of saturated hydrocarbons, primarily linear alkanes, with increasing amounts of branched (methyl-groups) alkanes as the chains get longer. In addition, small amounts of cycloalkanes (branched cyclopentanes and cyclohexanes) may be formed as the polymerisation reaction prolongs. This synthetic crude can subsequently be refined to a range of products very similar to petroleum refining. However, in contrast to their petroleum-derived analogues, GTL products are essentially free of unsaturated or aromatic constituents and also no sulphur-, oxygen-, or nitrogen-containing constituents are present.
Environmental Fate[1]	
Soil/Water/Air	The substance is expected to be readily biodegradable. It has a Log Koc of > 5.63 and is expected to be immobile in soils. If released into water, the substance is expected to adsorb to suspended solids and sediment based upon the Log Koc.
Human Health Toxicity Summary [1,2]	
Chronic Repeated Dose Toxicity	NOAEL (rat, oral): 200 mg/kg bw/day
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	The substance was found to be non-mutagenic.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No data available.
Acute Toxicity	The acute oral median lethal dose (LD50) of the test material in the female Sprague-Dawley CD strain rat was estimated to be greater than 5000 mg/kg bodyweight.
Irritation	Not irritating based on read across data.
Sensitisation	Not sensitising based on read across data.
Health Effects Summary	The critical health effect for risk characterisation is chronic repeated dose toxicity from oral exposure.
Key Study/Critical Effect for Screening Criteria	The repeated dose toxicity in rats via oral exposure was considered the most sensitive endpoint with a NOAEL of 200 mg/kg bw/day.

Ecological Toxicity [1,8]	
Aquatic Toxicity	Short-term toxicity: NOEC (48 h): 1000 mg/L (fish) LC50 (7 day): >100000 mg/L (fish) EL50 (72 h): >1000 mg/L (invertebrates) EL50 (48 h): 1000 mg/L (crustaceans) EL50 (72 h): 1000 mg/L (algae) Long-term toxicity: NOEL (33 day): >100 mg/L (fish) NOEL (21 day): <100 mg/L (invertebrates)
Determination of PNEC aquatic	Based on the lowest chronic endpoint for aquatic toxicity (100 mg/L), an assessment factor of 100 has been applied, resulting in a PNECaquatic of 1 mg/L.
Current Regulatory Controls[2,3,4,5,6]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	Oils and greases (including petrochemicals) for freshwater production: <300[3] µg/L (ANZECC, 2000)
PBT Assessment[1]	
P/vP Criteria fulfilled?	No. Readily biodegradable.
B/vB criteria fulfilled?	No. Based on log BCF of 3.17 or BCF of 1479.
T criteria fulfilled?	No. Acute toxicity data >1 mg/L in fish and invertebrates, thus the substance does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	February 2022

References

1. ECHA REACH, Distillates (Fischer-Tropsch), C8-26-branched and linear, Retrieved 2021: https://echa.europa.eu/
2. Peter J. Boogaard, Juan-Carlos Carrillo, Linda G. Roberts & Graham F. Whale (2017) Toxicological and ecotoxicological properties of gas-to-liquid (GTL) products. 1. Mammalian toxicology, Critical Reviews in Toxicology, 47:2, 121-144, DOI: 10.1080/10408444.2016.1214676.
3. ANZECC (2000) Australian and New Zealand Guidelines for Fresh and Marine Water Quality for protection for aquatic ecosystems.
4. Food Standards Australia New Zealand (FSANZ) 20th Australian Total Diet Survey. Retrieved 2021: https://www.foodstandards.gov.au/publications/pages/20thaustraliantotaldietsurveyjanuary2003/20thaustraliantotaldietsurveyfullreport/partb20thatds/partbmetals.aspx.
5. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
6. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.

7. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.
8. Shell GTL Saraline 185V Synthetic Base Fluid for High Performance Drilling. Retrieved 2022: https://www.shell.com/business-customers/chemicals/our-products/shell-gtl-drilling-fluids/_jcr_content/par/textimage_1477979319.stream/1580450325747/4322827486b4e1ef86e8858f64ce9b8100f1c2da/saraline-brochure.pdf.

Toxicity Summary - Fatty acids, tall-oil, reaction products with polyethylenepolyamines

Chemical and Physical Properties[1]	
CAS number	68910-93-0
Molecular formula	Unknown or variable composition, complex reaction products or biological materials (UVCB)
Molecular weight	UVCB
Solubility in water	No data available.
Melting point	-85 °C at 101.3 kPa
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	Non-explosive (100%)
Flammability potential	Not classified
Colour/Form	Liquid
Overview	This substance is used by consumers, in articles, by professional workers (widespread uses), in formulation or re-packing, at industrial sites and in manufacturing.
Environmental Fate[1]	
Soil/Water/Air	One study investigating the adsorption/desorption behaviour of Fatty acids, tall-oil, ethoxylated (CAS 61791-00-2) is available. The study was performed according to GLP and OECD guideline 121 (BASF 2017). 6 different peaks were observed with log Koc values ranging from < 1.8 to > 5.63. The two main components (> 85%) show log Koc values > 4. Thus, adsorption of Fatty acids, tall-oil, ethoxylated to solid soil is expected. The test with the source substance was conducted according to OECD Guideline 301B, under GLP conditions (BASF 2005). Domestic, non-adapted activated sludge was exposed to the test substance for 28 days at 22°C, and biodegradation was measured by CO2 consumption. After 28 days, the test substance 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. The test substance consists of components with log Kow values in the range of 5 to > 10 (KOWWIN v1.68) indicating a potential for bioaccumulation. But due to rapid environmental biodegradation, metabolisation via enzymatic hydrolysis (monoesters and diesters) as well as sterical hindrance of crossing biological mebranes (high molecular weight of diesters) a relevant uptake and bioaccumulation in aquatic organisms is not expected. This is supported by low BCF values of < 100 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, the test substance is not considered to be bioaccumulative.
Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	Under the conditions of this Combined Repeated Dose Toxicity Study with the Reproduction/Developmental Toxicity Screening Test, the oral administration by gavage of test substance to Wistar rats revealed no adverse signs of toxicity in male and female animals at a dose level of 1000 mg/kg bw/d. Thus, the no observed adverse effect level (NOAEL) for general systemic toxicity was 1000 mg/kg bw/d for male and female Wistar rats.
Carcinogenicity	No data available.

Mutagenicity/ Genotoxicity	The test substance is not mutagenic in bacteria, as determined in an OECD 471 study. The test substance is not chromosome damaging, as determined in an OECD 487 study. The test substance is not mutagenic in mammalian cells, as determined in an OECD 476 study.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Under the conditions of this Combined Repeated Dose Toxicity Study with the Reproduction/Developmental Toxicity Screening Test, the oral administration by gavage of the test substance to Wistar rats revealed no adverse signs of toxicity in male and female animals at a dose level of 1000 mg/kg bw/d. Thus, the no observed adverse effect level (NOAEL) for general systemic toxicity was 1000 mg/kg bw/d for male and female Wistar rats. The NOAEL for reproductive performance and fertility was set to 1000 mg/kg bw/d for male and female Wistar rats.
Acute Toxicity	In an acute oral toxicity study performed similar to OECD guideline 401 (BASF 1971), three groups of rats consisting of 10 animals/sex/dose were treated by single gavage application with an aqueous solution of the test substance (10000, 8000, 6400 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 occurred at 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 6400 mg/kg bw. Based on the results obtained under the test conditions of this study, the acute oral LD50 was determined to be > 10000 mg/kg bw. To evaluate the potential acute inhalation toxicity of the test substance an Inhalation Risk Test conducted according to a BASF internal testing method (BASF 1971). The test demonstrates the toxicity of an atmosphere saturated with vapours of the volatile components of a test substance at the temperature chosen for vapour generation (20 °C). Rats were exposed sequentially to the vapours, generated by bubbling 200 l/h air through a substance column of about 5 cm above a fritted glass disc in a glass cylinder. The animals were exposed for 8 hour. The exposure concentration was estimated to be 0.28 mg/L based on evaporated substance. In addition to mortality, clinical signs were recorded and necropsy on surviving animals performed. No mortality occurred and no clinical sign were noted during exposure and observation period. In one animal exposed for 8 hours hydrometra was observed after necropsy. Since no mortality occurred at the concentrations tested an LC50 estimation cannot be made. In another Inhalation Risk Test of similar design, Rats (12 animals) were exposed sequentially to the vapours, generated by bubbling 200 l/h air through a substance column of about 5 cm above a fritted glass disc in a glass cylinder. This time vapours were generated at 20 °C as well as 50 °C. The exposure concentrations were 0.04 mg/L and 0.34 mg/L. Rats were exposed for 8 hour. As in the previous study, no mortality occurred after exposure up to 8 hours. Clinical sings observed in the animals exposed to the vapour generated at 20°C included mild escape attempts when exposure began and at the end of the exposure period slight eye irritation was observed. The next day, the animals were without symptoms. In the animals exposed to the vapour generated at 50 °C escape attempts were noted in the first 60 minutes of exposure. Exposure to the saturated atmosphere caused slight eye irritation. At the end of the exposure period, all clinical signs were resolved. Since no mortality occurred at the concentrations tested an LC50 estimation cannot be made. Based on the inhalation studies, no conclusion on LC50 can be drawn, because the tested concentrations are too low in relation to the classification criteria.
Irritation	The test substance was not irritant or corrosive to the skin in a GLP-compliant OECD 431 and 439 study. The test substance was not irritant to the eyes in a GLP-compliant OECD 492 study.

	Based on the available information, classification for skin and eye irritation is not warranted, in accordance with EU Classification, Labelling and Packaging of Substances and Mixtures (CLP) Regulation No. (EC) 1272/2008.
Sensitisation	The test substance did not show an indication of skin sensitising potential in an OECD 429 (LLNA) study. However, an earlier Buehler test (OECD 406) did indicate skin sensitising potential of the substance.
Health Effects Summary	Possible sensitiser.
Key Study/Critical Effect for Screening Criteria	The NOAEL for general systemic toxicity of 1000 mg/kg bw/d was selected as the key study.
Ecological Toxicity [1]	
Aquatic Toxicity	Short-term toxicity tests with the target substance for all trophic levels (fish, daphnia, algae) are available. The test substance did not indicate to be harmful to freshwater fish (96h-LL50 > 100 mg/L), but showed to be toxic to aquatic invertebrates (48h-EL50 = 12.41 mg/L) and harmful to algae (72h-EL50 = 39.7 mg/L). Hence, aquatic invertebrates were most susceptible to the test substance and this effect value was used for the PNEC derivation. Long-term toxicity data with the source substance are only available for algae. The algal test revealed the substance to be of low toxicity to algae (72h-EL10 = 7.08 mg/L). In addition, data are available for toxicity to microorganisms. A test on respiration inhibition with activated sludge resulted in an 3h-EC10 of > 10000 mg/L indicating that detrimental effects in STPs are not to be expected.
Determination of PNEC aquatic	A PNECaqua = 0.012 mg/L can be calculated based on the lowest acute toxicity value (EL50 = 12.41 mg/L) for aquatic invertebrates (Daphnia) with the assessment factor of 1000.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1]	
P/vP Criteria fulfilled?	No. Based on the results from the read-across substance, Fatty acids, tall oil, ethoxylated (EO > 1 < 2.5) (CAS 61791-00-2) is considered to be readily biodegradable.
B/vB criteria fulfilled?	No. The test substance consists of components with log Kow values of > 10 (KOWWIN v1.68) indicating a potential for bioaccumulation. But due to rapid environmental biodegradation, metabolisation via enzymatic hydrolysis (monoesters and diesters) as well as sterical 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 BCF values of < 100 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, the test substance is not considered to be B or vB.
T criteria fulfilled?	No. Available short-term and long-term toxicity tests with aquatic organisms resulted in effect values > 1 mg/L. Thus, this substance does not meet the screening criteria for toxicity.

Overall conclusion	Not PBT
Revised	January 2022

References

1. ECHA REACH, Fatty acids, tall-oil, ethoxylated, Retrieved 2019: https://echa.europa.eu/

Toxicity Summary - Mineral Oil

Chemical and Physical Properties[1,2,3]	
CAS number	8042-47-5
Molecular formula	UVCB
Molecular weight	UVCB
Solubility in water	Insoluble
Melting point	-60 - 0 °C at 101.3 - 101.325 kPa
Boiling point	218 - 800 °C at 101.3 kPa
Vapour pressure	10 Pa at 20 °C
Henrys law constant	No data available
Explosive potential	Non-explosive
Flammability potential	Non-flammable
Colour/Form	Liquid, odourless
Overview	A highly refined petroleum mineral oil consisting of a complex combination of hydrocarbons obtained from the intensive treatment of a petroleum fraction with sulphuric acid and oleum, or by hydrogenation, or by a combination of hydrogenation and acid treatment. Additional washing and treating steps may be included in the processing operation. It consists of saturated hydrocarbons having carbon numbers predominantly in the range of C15 through C50. A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health.
Environmental Fate[3]	
Soil/Water/Air	The environmental fate assessment of these chemicals indicates they have low to very low vapor pressures, very low solubility in water, high octanol-water partition coefficients, and high sorption to organic matter. Thus, these chemicals will exhibit very poor migration, due to their high sorption and low solubility in water, as well as low potential for volatility. Fugacity modelling suggests they would remain partitioned to the terrestrial phase, remaining sorbed to soil or the foliar surfaces to which they are applied.
Human Health Toxicity Summary [2,3]	
Chronic Repeated Dose Toxicity	The effects of long-term exposure include possible dermatitis with repeated or prolonged contact with skin
Carcinogenicity	Evidence of carcinogenicity is inadequate in humans and inadequate or limited in experimental animals.
Mutagenicity/ Genotoxicity	The mutagenicity of various test materials were all characterized as being non-mutagenic, in general, but with problems due to the presence of suspended oil droplets, due to the poor water solubility of the test materials.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	It was concluded from dermal dosing studies, that mineral oil had no effects (on mortality, clinical signs of toxicity, body weight, food consumption, absolute organ weights, microscopic changes in reproductive organs of parental animals, number of corpora lutea, implantation sites, live pups per litter, no gross anomalies, and body weights of pups or weight gains of pups). In a 4-week inhalation study, there were no treatment related effects on sperm morphology. In a one-generation reproduction study, both males and females were dosed by gavage, and there were no adverse effects (no clinical findings, growth weights and food consumption was normal, no effects on fertility and mating indices in either males or females, and at necropsy, organ weights and histopathology were considered normal by the study authors). Two other studies were reported with white mineral oil, both via single daily gavage doses. In one study, both sexes were dosed, and some effects were observed, which the study authors concluded were within the “spectrum of malformations [which] occurs spontaneously in Sprague-Dawley rat.” In the

	companion study in which only pregnant females were dosed, foetal effects were noted, but "the study authors considered these malformations to be minor and within the normal ranges for the strain of rat" (SpragueDawley). In general, these studies were performed at very high dosages, from about 900 mg/kg-bw/day (1 mL/kg-bw/day) to about 4500 mg/kg-bw/day (5 mL/kg-bw/day).
Acute Toxicity	A short-term exposure duration dermal NOAEL of 2000 mg/kg/day was observed in a 28-day repeat-dose study, in which no adverse effects were observed at the highest test concentration (2000 mg/kg/day). A short-term exposure duration inhalation LOAEL of 146.64 mg/kg/day was observed in a 28-day inhalation study. Adverse effects were reported at the lowest exposure dosage, 0.5 mg/L, based on the following observations: (1) multiple lung effects, (2) increased white blood cell counts in males, (3) increased absolute liver weight, (4) accessory spleens and/or abnormally coloured spleens, and (5) additional microscopic findings. An intermediate-term exposure duration inhalation NOAEL of 26.1 mg/kg/day was observed in a 90-day inhalation study, in which effects were observed at 0.9 mg/L, but there were no adverse effects observed at 0.1 mg/L
Irritation	Slight eye irritation in rats and rabbits.
Sensitisation	Not a dermal sensitizer.
Health Effects Summary	Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.
Key Study/Critical Effect for Screening Criteria	The intermediate-term inhalation NOAEL of 26.1 mg/kg/day, derived from a 90-day inhalation study, based on effects observed at 0.9 mg/L, with no adverse effects observed at 0.1 mg/L was considered the most sensitive endpoint.
Ecological Toxicity [1]	
Aquatic Toxicity	Rainbow trout 96 hr LL50 (48 h) 100 mg/L
Determination of PNEC aquatic	This substance has a low acute toxicity concern to aquatic organisms and thus required no further assessment.
Current Regulatory Controls[4]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	MAK: (respirable fraction): 5 mg/m^3; peak limitation category: II(4); pregnancy risk group: C
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment [1]	
P/vP Criteria fulfilled?	No. Not readily biodegradable based on read across study.
B/vB criteria fulfilled?	Not applicable. This substance is a UVCB.
T criteria fulfilled?	No. The acute LL50 value in fish is >1 mg/L. Thus, it does not meet the criteria for toxicity.
Overall conclusion	Not PBT
Revised	February 2022

References

1. ECHA REACH, White mineral oil (petroleum), Retrieved 2022: https://echa.europa.eu/.
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2022: https://www.industrialchemicals.gov.au/
3. USEPA 2007. Revised Reregistration Eligibility Decision for Aliphatic Solvents, 29 November 2007. US Environmental Protection Agency Office of Pesticide Programs.
4. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved February 2022: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
5. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved February 2022: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.

Toxicity Summary - Partially hydrolysed polyacrylamide

Chemical and Physical Properties[1,2,3,4]	
CAS number	9003-05-8
Molecular formula	(C3H5NO)x
Molecular weight	1,000,000 to > 50,000,000 g/mol for polyacrylamide copolymers used as flocculants
Solubility in water	Water soluble
Melting point	No data available.
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	No data available.
Overview	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. Anionic polyacrylamide polymer can then be formed from the hydrolysis of the acrylamide homopolymer either simultaneously during the polymerisation process or as a subsequent step. Anionic polyacrylamide polymer can also be formed from the copolymerisation of acrylamide and acrylic acid. A Tier 1 Human Health and Environmental Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health and the environment and thus required no further assessment.
Environmental Fate [3]	
Soil/Water/Air	No studies on the environmental fate of anionic polyacrylamide are available. As a high-molecular weight, water-soluble polymer, it is not expected to biodegrade or bioaccumulate. The environmental fate of anionic polyacrylamide will be determined primarily by adsorption. 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.
Human Health Toxicity Summary [1,2,4]	
Chronic Repeated Dose Toxicity	No data available.
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	No data available.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No data available.
Acute Toxicity	Mouse LD50 (oral): 12950 mg/kg Rabbit LD50 (oral): 11250 mg/kg Rat LD50 (oral): >1000 mg/kg
Irritation	No data available.
Sensitisation	No data available.
Health Effects Summary	Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.

Key Study/Critical Effect for Screening Criteria	The oral acute toxicity in rats was considered the most sensitive endpoint with a LD50 of 1000 mg/kg.
Ecological Toxicity [3]	
Aquatic Toxicity	Fathead minnow LC50: 810 mg/L Rainbow trout LC50: > 100 mg/L Bluegill sunfish LC50: >300 mg/L Daphnia magna LC50: 470 mg/L
Determination of PNEC aquatic	Anionic polyacrylamide has a low acute toxicity concern to aquatic organisms and thus required no further assessment.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment [3]	
P/vP Criteria fulfilled?	Yes. Anionic 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.
B/vB criteria fulfilled?	No. Pharmacokinetic studies showed that anionic polyacrylamide was not bioavailable to rats when ingested; this is most likely due to its large size (high molecular weight) and presumed resistance to break down in the gastrointestinal tract. Anionic polyacrylamide is thus not expected to be bioavailable to aquatic or terrestrial organisms. It is not expected to meet the criteria for bioaccumulation.
T criteria fulfilled?	No. The acute LC50 values in fish and invertebrates are >1 mg/L. Thus, it does not meet the criteria for toxicity.
Overall conclusion	Not PBT
Revised	February 2022

References

1. Department of the Environment and Energy 2017, National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2022: https://www.industrialchemicals.gov.au/.
3. EHS Support, Anionic Polyacrylamide. Available at: https://www.santos.com/wp-content/uploads/2021/04/Anionic-Polyacrylamide-March-2021.pdf. Retrieved February 2022.
4. ChemIDplus, Polyacrylamide, Retrieved February 2022: https://chem.nlm.nih.gov/chemidplus/rn/9003-05-8.
5. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved February 2022: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
6. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved February 2022: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.

Toxicity Summary - Phosphoric ester of ethoxylated fatty alcohol

Chemical and Physical Properties[1]	
CAS number	68585-36-4
Molecular formula	Unknown or variable composition, complex reaction products or biological materials (UVCB)
Molecular weight	UVCB
Solubility in water	No data available.
Melting point	-85 °C at 101.3 kPa
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	Non-explosive (100%)
Flammability potential	Not classified
Colour/Form	Liquid
Overview	This substance is used by consumers, in articles, by professional workers (widespread uses), in formulation or re-packing, at industrial sites and in manufacturing.
Environmental Fate[1]	
Soil/Water/Air	One study investigating the adsorption/desorption behaviour of Fatty acids, tall-oil, ethoxylated (CAS 61791-00-2) is available. The study was performed according to GLP and OECD guideline 121 (BASF 2017). 6 different peaks were observed with log Koc values ranging from < 1.8 to > 5.63. The two main components (> 85%) show log Koc values > 4. Thus, adsorption of Fatty acids, tall-oil, ethoxylated to solid soil is expected. The test with the source substance was conducted according to OECD Guideline 301B, under GLP conditions (BASF 2005). Domestic, non-adapted activated sludge was exposed to the test substance for 28 days at 22°C, and biodegradation was measured by CO2 consumption. After 28 days, the test substance 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. The test substance consists of components with log Kow values in the range of 5 to > 10 (KOWWIN v1.68) indicating a potential for bioaccumulation. But due to rapid environmental biodegradation, metabolisation via enzymatic hydrolysis (monoesters and diesters) as well as sterical hindrance of crossing biological mebranes (high molecular weight of diesters) a relevant uptake and bioaccumulation in aquatic organisms is not expected. This is supported by low BCF values of < 100 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, the test substance is not considered to be bioaccumulative.
Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	Under the conditions of this Combined Repeated Dose Toxicity Study with the Reproduction/Developmental Toxicity Screening Test, the oral administration by gavage of test substance to Wistar rats revealed no adverse signs of toxicity in male and female animals at a dose level of 1000 mg/kg bw/d. Thus, the no observed adverse effect level (NOAEL) for general systemic toxicity was 1000 mg/kg bw/d for male and female Wistar rats.
Carcinogenicity	No data available.

Mutagenicity/ Genotoxicity	The test substance is not mutagenic in bacteria, as determined in an OECD 471 study. The test substance is not chromosome damaging, as determined in an OECD 487 study. The test substance is not mutagenic in mammalian cells, as determined in an OECD 476 study.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Under the conditions of this Combined Repeated Dose Toxicity Study with the Reproduction/Developmental Toxicity Screening Test, the oral administration by gavage of the test substance to Wistar rats revealed no adverse signs of toxicity in male and female animals at a dose level of 1000 mg/kg bw/d. Thus, the no observed adverse effect level (NOAEL) for general systemic toxicity was 1000 mg/kg bw/d for male and female Wistar rats. The NOAEL for reproductive performance and fertility was set to 1000 mg/kg bw/d for male and female Wistar rats.
Acute Toxicity	In an acute oral toxicity study performed similar to OECD guideline 401 (BASF 1971), three groups of rats consisting of 10 animals/sex/dose were treated by single gavage application with an aqueous solution of the test substance (10000, 8000, 6400 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 occurred at 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 6400 mg/kg bw. Based on the results obtained under the test conditions of this study, the acute oral LD50 was determined to be > 10000 mg/kg bw. To evaluate the potential acute inhalation toxicity of the test substance an Inhalation Risk Test conducted according to a BASF internal testing method (BASF 1971). The test demonstrates the toxicity of an atmosphere saturated with vapours of the volatile components of a test substance at the temperature chosen for vapour generation (20 °C). Rats were exposed sequentially to the vapours, generated by bubbling 200 l/h air through a substance column of about 5 cm above a fritted glass disc in a glass cylinder. The animals were exposed for 8 hour. The exposure concentration was estimated to be 0.28 mg/L based on evaporated substance. In addition to mortality, clinical signs were recorded and necropsy on surviving animals performed. No mortality occurred and no clinical sign were noted during exposure and observation period. In one animal exposed for 8 hours hydrometra was observed after necropsy. Since no mortality occurred at the concentrations tested an LC50 estimation cannot be made. In another Inhalation Risk Test of similar design, Rats (12 animals) were exposed sequentially to the vapours, generated by bubbling 200 l/h air through a substance column of about 5 cm above a fritted glass disc in a glass cylinder. This time vapours were generated at 20 °C as well as 50 °C. The exposure concentrations were 0.04 mg/L and 0.34 mg/L. Rats were exposed for 8 hour. As in the previous study, no mortality occurred after exposure up to 8 hours. Clinical sings observed in the animals exposed to the vapour generated at 20°C included mild escape attempts when exposure began and at the end of the exposure period slight eye irritation was observed. The next day, the animals were without symptoms. In the animals exposed to the vapour generated at 50 °C escape attempts were noted in the first 60 minutes of exposure. Exposure to the saturated atmosphere caused slight eye irritation. At the end of the exposure period, all clinical signs were resolved. Since no mortality occurred at the concentrations tested an LC50 estimation cannot be made. Based on the inhalation studies, no conclusion on LC50 can be drawn, because the tested concentrations are too low in relation to the classification criteria.
Irritation	The test substance was not irritant or corrosive to the skin in a GLP-compliant OECD 431 and 439 study. The test substance was not irritant to the eyes in a GLP-compliant OECD 492 study.

	Based on the available information, classification for skin and eye irritation is not warranted, in accordance with EU Classification, Labelling and Packaging of Substances and Mixtures (CLP) Regulation No. (EC) 1272/2008.
Sensitisation	The test substance did not show an indication of skin sensitising potential in an OECD 429 (LLNA) study. However, an earlier Buehler test (OECD 406) did indicate skin sensitising potential of the substance.
Health Effects Summary	Possible sensitiser.
Key Study/Critical Effect for Screening Criteria	The NOAEL for general systemic toxicity of 1000 mg/kg bw/d was selected as the key study.
Ecological Toxicity [1]	
Aquatic Toxicity	Short-term toxicity tests with the target substance for all trophic levels (fish, daphnia, algae) are available. The test substance did not indicate to be harmful to freshwater fish (96h-LL50 > 100 mg/L), but showed to be toxic to aquatic invertebrates (48h-EL50 = 12.41 mg/L) and harmful to algae (72h-EL50 = 39.7 mg/L). Hence, aquatic invertebrates were most susceptible to the test substance and this effect value was used for the PNEC derivation. Long-term toxicity data with the source substance are only available for algae. The algal test revealed the substance to be of low toxicity to algae (72h-EL10 = 7.08 mg/L). In addition, data are available for toxicity to microorganisms. A test on respiration inhibition with activated sludge resulted in an 3h-EC10 of > 10000 mg/L indicating that detrimental effects in STPs are not to be expected.
Determination of PNEC aquatic	A PNECaqua = 0.012 mg/L can be calculated based on the lowest acute toxicity value (EL50 = 12.41 mg/L) for aquatic invertebrates (Daphnia) with the assessment factor of 1000.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1]	
P/vP Criteria fulfilled?	No. Based on the results from the read-across substance, Fatty acids, tall oil, ethoxylated (EO > 1 < 2.5) (CAS 61791-00-2) is considered to be readily biodegradable.
B/vB criteria fulfilled?	No. The test substance consists of components with log Kow values of > 10 (KOWWIN v1.68) indicating a potential for bioaccumulation. But due to rapid environmental biodegradation, metabolisation via enzymatic hydrolysis (monoesters and diesters) as well as sterical 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 BCF values of < 100 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, the test substance is not considered to be B or vB.
T criteria fulfilled?	No. Available short-term and long-term toxicity tests with aquatic organisms resulted in effect values > 1 mg/L. Thus, this substance does not meet the screening criteria for toxicity.

Overall conclusion	Not PBT
Revised	January 2022

References

1. ECHA REACH, Fatty acids, tall-oil, ethoxylated, Retrieved 2019: https://echa.europa.eu/

Toxicity Summary - Polyanionic cellulose, low viscosity

Chemical and Physical Properties[1,2,3,4]	
CAS number	9004-32-4
Molecular formula	C8H15NaO8
Molecular weight	262.19
Solubility in water	The sodium salt disperses and its solubility in water depends upon the degree of substitution.
Melting point	300°C
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	White or slightly yellowish, almost odourless and tasteless hydroscopic powder, consisting of very fine particles, fine granules or fine fibres.
Overview	Sodium carboxymethyl cellulose (CMC) is used in drilling muds, detergents, resin emulsion paints, adhesives, printing inks, and textile sizes. It is also used as a protective colloid, a stabilizer for foods, and a pharmaceutical additive. A Tier 1 Human Health and Environmental Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health and the environment and thus required no further assessment.
Environmental Fate [4]	
Soil/Water/Air	Sodium carboxymethylcellulose is biodegradable, but is not considered to be readily biodegradable. It is not expected to bioaccumulate. All of the polymers in this group are expected to be water soluble. If discharged into natural waters, sodium carboxymethylcellulose is expected to be present as a polyanion as a result of the ionisation of the carboxymethyl substituents. Comparatively complex partitioning behaviour in aquatic systems may occur based on the well-established interactions between colloids and carboxymethylcellulose, which is a key part of the function of this polymer in laundry detergents. No experimental partition coefficient data are available for sodium carboxymethylcellulose. Based on its high water solubility, the substance is likely to be mobile in the environment.
Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	Ten rats received 300 to 500 mg of CMC daily for two months without any adverse effect. Another group of 10 rats received a diet containing 20% of CMC for 63 days. Slight growth retardation and a laxative effect were observed. Organ weights and both gross and microscopic pathological examination revealed no abnormalities. Oral rat TDLo: 227 g/kg/13W (continuous)
Carcinogenicity	Carboxymethyl cellulose sodium salt is a “suspected carcinogen”.
Mutagenicity/ Genotoxicity	Carboxymethylcellulose has been used often as the vehicle control in a number of genotoxicity studies as the control agent or vehicle and as such would not be expected to show activity in these types of studies.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	In several studies, carboxymethylcellulose and its sodium salt have been used as the vehicle in developmental, embryotoxic and teratogenic studies on rats, mice or rabbits and as such would not be expected to have any adverse effect.
Acute Toxicity	Rats, guinea pigs and rabbits showed no symptoms after administration by stomach tube of 3000 mg/kg in three divided doses. Rat LD50 (oral): 270000 mg/kg/bw Guinea pig LD50 (oral): 160000 mg/kg/bw

	A 4-hr inhalation LC50 value of 5.8 g/m^3 has been reported for the sodium salt in rats.
Irritation	No data available.
Sensitisation	Suspected skin sensitiser
Health Effects Summary	Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.
Key Study/Critical Effect for Screening Criteria	The oral rat chronic toxicity TDLo: 227 g/kg/13W (continuous) was considered the most sensitive endpoint.
Ecological Toxicity [4]	
Aquatic Toxicity	Brachydanio rerio 96-hour LC50 >2,500 mg/L Daphnia magna 48-hour EC50 >5,000 mg/L Daphnia magna 48-hour EC50 87.26 mg/L Selenastrum capricornutum 96-hour EC50 500 mg/L
Determination of PNEC aquatic	This compound has a low acute toxicity concern to aquatic organisms and thus required no further assessment.
Current Regulatory Controls [5,6]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment [4]	
P/vP Criteria fulfilled?	No. Sodium carboxymethylcellulose is a water-soluble semisynthetic polymer that is not readily biodegradable. Therefore, it meets the screening criteria for persistence.
B/vB criteria fulfilled?	No. Not expected to bioaccumulate.
T criteria fulfilled?	No. The acute EC50 of sodium carboxymethylcellulose is >1 mg/L in fish, invertebrates and algae. Therefore, it does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	February 2022

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2022: https://www.industrialchemicals.gov.au/.
2. NCBI, 2022. National Center for Biotechnology Information. Available at: https://pubchem.ncbi.nlm.nih.gov/. Retrieved February 2022.
3. Toxicological profile for sodium carboxy methyl cellulose, Retrieved February 2022: https://tobacco-information.hpa.gov.tw/common/Download.ashx?t=CLI8001&f=54368658_336/54368658_336_A0191.pdf
4. EHS Support, Sodium Carboxymethylcellulose. Available at: https://www.santos.com/wp-content/uploads/2021/04/Sodium-Carboxymethylcellulose-March-2021.pdf. Retrieved February 2022.

5. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved February 2022: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
6. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved February 2022: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.

Toxicity Summary - Disodium, Trisodium, Tetrasodium EDTA

Chemical and Physical Properties [1,2,3]	
CAS number	139-33-3 – Disodium EDTA 150-38-9 – Trisodium EDTA 64-02-8 – Tetrasodium EDTA
Molecular formula	Na2EDTA – Disodium EDTA NA3EDTA – Trisodium EDTA NA4EDTA – Tetrasodium EDTA
Molecular weight	336.21 g/mol - Disodium EDTA 380.17 g/mol – Tetrasodium EDTA
Solubility in water	$1.0X10^{+6}$ mg/L (miscible) at 25 ^{o}C - Disodium EDTA
Melting point	242 ^{o}C - Disodium EDTA >300 ^{o}C – Tetrasodium EDTA
Boiling point	252 ^{o}C (decomposes) - Disodium EDTA
Vapour pressure	Negligible
Henrys law constant	Negligible
Explosive potential	No data found
Flammability potential	No data found
Colour/Form	Solid granular materials
Overview	Disodium, trisodium and tetrasodium EDTA are members of the Amino Carboxylic Acid-Based Chelants Category. EDTA is a metal-complexing agent and may act to mobilise some heavy metals in the environment. EDTA is used widely in industry and agriculture. It is used in laundry detergents, water softening, electroplating, textile and paper production, as a food additive, and in cosmetics. Most of these uses will result in the release of EDTA to the aquatic environment. It is also used as a drug in chelation therapy, particularly in cases involving lead poisoning. EDTA is poorly absorbed in the gut and does not form any significant metabolites. It does not accumulate in the body. Long-term feeding studies with rats and dogs reported no interference to mineral metabolism. Results from other studies have been affected by the formation of zinc complexes in the gastrointestinal tract,which prevents the zinc from being absorbed. As metal-organic salts, or inner salts, all category members decompose before melting upon sufficient heating (generally at temperatures > 200 °C). Therefore true melting points are not applicable. Chelants that are metal salts do not exist as discrete neutral molecules, and therefore cannot volatilize, exert appreciable vapour pressure, or boil. Therefore, vapour pressure and boiling point data are not applicable for such chelants and are not determined. Henry's law constants are also expected to be negligible. Chelants that exist as neutral molecules (not metal salts) can exert vapour pressure, but in this case the vapour pressure is exceedingly low. All category members are highly soluble to miscible in water (generally > 10,000 mg/L) and insoluble in organic solvents, therefore also possessing negative partition coefficients (log Kows). The ability of chelants to remove and add ions to solution is the mechanism whereby these chemicals produce toxicity. Environmental fate and ecological and mammalian toxicity profiles are consistent within the category. A Tier 1 Human Health Assessment for these chemicals has been conducted by NICNAS which concluded that these chemicals were identified as low concern to human health.

Environmental Fate [1,2,3]	
Soil/Water/Air	EDTAs have demonstrated high stability to hydrolysis, and most are commercially available primarily or solely in aqueous solution. EDTAs emitted to waterways will remain dissolved in this environmental compartment. If emitted to soil or sediment, they will exhibit high water solubility and soil mobility. This behaviour is based on the presence of multiple carboxylate anion groups in the molecular structure, and is supported by the demonstrated high water solubility and negligible vapor pressure of EDTAs. Results of recent studies indicate that EDTA, calcium EDTA and Na2EDTA can biodegrade under certain conditions.
Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	In a 13-week repeated-dose toxicity study, rats (both sexes) fed Na2EDTA (0, 1, 5, 10%) showed mortality at the highest dose. In addition, there was decreased food consumption (emaciation at 10%) and diarrhea at doses of 5% (approximately 4206 mg/kg bw/day) and above. The NOAEL was 1% (approximately 692 mg/kg bw/day). Range finding studies with higher dose levels revealed diarrhea, emaciation, loss of body weight and sometimes parakeratosis in esophagus and forestomach as well as decreased hemoglobin and hematocrit levels. In a 2- year bioassay in rats and mice (both sexes) with Na3EDTA (0, 3750 or 7500 ppm) a NOAEL of 7500 ppm (approximately 500 mg/kg bw/day in rats and 938 mg/kg bw/day in mice; highest dose tested) was determined.
Carcinogenicity	An oral two-year study with Na3EDTA trihydrate in mice and rats indicated no evidence of carcinogenicity. The amino carboxylic acid-based chelants category members are not expected to be carcinogens.
Mutagenicity/ Genotoxicity	Available data indicate disodium and trisodium EDTA do not induce gene mutations or chromosomal aberrations in vitro or in vivo.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Chronic studies with Na3EDTA that included histological examination of gonadal tissues for evidence of adverse effects also showed no adverse effects on reproductive organs. The weight of evidence from a two-generation reproductive toxicity study in rats shows that dietary ingestion of 1% Na2EDTA (approx. 920 mg/kg bw/day) had no effect on reproduction; however, no litters were produced at 5% (approx. 4600 mg/kg bw/day); the NOAEL for reproductive toxicity was 920 mg/kg bw/day. Developmental toxicity data are available for EDTA, CaNa2EDTA, Na2EDTA, Na3EDTA, and Na5DTPA. Data from multigenerational and prenatal developmental toxicity studies suggest that developmental effects are observed in the presence of maternal toxicity and are related to plasma zinc concentrations. Studies on developmental toxicity showed a specific fetotoxic and teratogenic potential of EDTA, Na2EDTA and CaNa2EDTA; a LOAEL of 1000 mg/kg bw/day was determined. Increased proportions/litter and significantly lower fetal body weights are indicative for an impaired fetal development. The pattern of malformations comprised cleft palate, severe brain deformities, eye defects, micro- or agnathia, syndactyly, clubbed legs and tail anomalies. These effects were exhibited in studies using maternally toxic dose levels. The mechanism resulting in developmental effects is found to occur via zinc depletion resulting in zinc deficit. These effects are independent of whether the acid or sodium or calcium salts are applied.
Acute Toxicity	Limited acute inhalation toxicity data with atmospheres enriched in the dusts of certain of the chelants were generally without effect in rats. However, inhalation of respirable dust aerosols of Na2EDTA in male rats exposed to 30, 300 or 1103 mg/m^3 6 hours/day for up to 5 days produced adverse effects at all concentration levels. Mortality was observed at 1103 mg/m^3 following a single 6-h exposure. These effects were fully reversed in surviving animals after a 14-day recovery. Acute dermal toxicity studies in rats, oral LD50 values for Na2EDTA, Na3EDTA were > 2000 mg/kg bw
Irritation	The aminocarboxylic acid-based chelants are not irritating to moderately irritating to the skin, and slightly to moderately irritating to the eyes in rabbits. The irritancy potential is related to the pH of the individual salt. Thus, more acidic members of the category such as disodium EDTA have inherently greater irritancy potential.

Sensitisation	The aminocarboxylic acid-based chelants are not skin sensitisers based on studies in mice and guinea pigs.
Health Effects Summary	These chemicals have been identified by NICNAS to be of low concern to human health.
Key Study/Critical Effect for Screening Criteria	The Australian Drinking Water Guideline (0.25 mg/L, health) may be used. for EDTA
Ecological Toxicity [1,2,3]	
Aquatic Toxicity	According to the results from different ecotoxicological studies, EDTA mainly influences the pathway of metal ions. For EDTA long-term studies with fish, daphnids and algae are available. The following results were found: *Danio rerio*: 35 d-NOEC > 26.8 mg/L (CaNa2EDTA); *Daphnia magna*: 21d-NOEC = 22 mg/L; *Scenedesmus subspicatus*: 72h-EC10 = > 100 mg/L. For Na2EDTA, *Daphnia magna*: 21d-NOEC = 25 mg/L.
Determination of PNEC aquatic	The effects assessment of EDTA is based on long-term tests, which are available for fish,daphnids and algae. The most sensitive endpoint could be found for *Daphnia magna* with a NOEC of 22 mg/l H4EDTA. An assessment factor of 10 has been used leading to a PNECaqua of 2.2 mg/l.
Current Regulatory Controls [4]	
Australian Hazard Classification	No data available
Australian Occupational Exposure Standards	No data available
International Occupational Exposure Standards	No data available
Australian Food Standards	No data available
Australian Drinking Water Guidelines	The Australian Drinking Water Guideline for EDTA is 0.25 mg/L.
Aquatic Toxicity Guidelines	No data available
PBT Assessment [1,2,3]	
P/vP Criteria fulfilled?	EDTAs are not readily biodegradable and as such are persistent in the environment.
B/vB criteria fulfilled?	EDTAs have a low potential for bioaccumulation.
T criteria fulfilled?	The acute aquatic toxicity of EDTAs are > 0.01 mg/L. Hence the substances do not fulfill the screening criteria for toxic (T)
Overall conclusion	Not a PBT substance (based on screening data).
Revised	December 2018

References

1. European Commission Joint Research Centre 2004, European Union Risk Assessment Report, Tetrasodium Ethylenediaminetetracetate (Na4EDTA), CAS no. 64-02-8.
2. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2017, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB
3. OECD (2012) SIDS Initial Assessment Profile for Amino Carboxylic Acid-Based Chelants Category
4. National Health and Medical Research Council, Natural Resources Management Ministerial Council, national Water Quality Management Strategy, Australian Drinking Water Guidelines 6, 2011, Version 3.3 Updated November 2016.
5. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2018: https://www.nicnas.gov.au

Toxicity Summary - Talc

Chemical and Physical Properties [1,4]	
CAS number	14807-96-6
Molecular formula	H2-O3-Si 3/4Mg or Mg3Si4O10(OH)2
Molecular weight	78.10 (estimate)
Solubility in water	Insoluble in water, cold acids or in alkalis
pH	9.0 to 9.5
Melting point	800-900°C (disintegration; WHO 2005)
Boiling point	549.7°C (estimate)
Vapour pressure	NA
Henrys law constant	NA
Explosive potential	NA
Flammability potential	Not flammable
Colour/Form	white to gray-white, fine crystalline powder.
Overview	Talc finely powdered hydrous magnesium silicate mineral sometimes found in association with asbestos. After being mined, it is processed to remove impurities and powdered. Talc is a useful commercial product due to its fragrance retention, luster, purity, softness, and whiteness as well as its chemical inertness and oil and grease adsorption. Talc is a mineral composed of hydrated magnesium silicate. Talc refers to both mineral talc and industrial mineral products that are marketed under the name talc and contain proportions of mineral talc that range from about 35% to almost 100%. Industrial talc generally refers to products that contain abundant minerals other than talc; cosmetic talc now normally contains >98% talc but the content may have been lower in the past. Pharmaceutical talc contains >99% talc. Talcum powder is cosmetic-grade talc. This chemical has been identified by NICNAS to be of low concern to human health based on an initial screening approach and thus required no further assessment. Further assessment of the environmental risks from the use of this chemical is also not required.
Environmental Fate [2,3]	
Soil/Water/Air	As a mineral, talc does not biodegrade

Human Health Toxicity Summary [1,2]	
Chronic Repeated Dose Toxicity Carcinogenicity	Talc-based body powder, when used perineally, is classified by IARC as group 2B as possibly carcinogenic to humans. However, talc for general use not containing asbestos or asbestiform fibres is classified as group 3 as not classifiable to its carcinogenicity to humans. Talc containing asbestiform fibres is classified by IARC as group 1 for carcinogenic to humans. Talc alone failed to induce respiratory tumors, granulomas or mesothelial proliferation in a hamster study but produced tumours of the larynx, trachea and lungs when tested in association with benzo(a)pyrene. In a rat study of aerosol talc there was some evidence of carcinogenic activity of talc in male F344/N rats and clear evidence of carcinogenic activity in female F344/N rats. No evidence of carcinogenicity was evident in intraperitoneal or inhalation studies in hamsters. Male and female Wistar rats were given in their diet 0 or 50 mg/kg of commercial talc [characteristics unspecified] for the life of the animals (average survival was 702 and 649 days, respectively). There was no significant difference in the talc-fed animals compared with control animals (Gibel *et al.*, 1976). In humans and experimental animals, the effects of talc are dependent on the route of exposure, and the dose and properties of the talc. Talc pneumoconiosis was somewhat more prevalent and severe among miners exposed to talc containing asbestiform minerals and/or asbestos than among those exposed to talc without such contaminants. However, the role of quartz and asbestos in the observed pneumoconiosis could not be ruled out. Among drug users, intravenous injection of talc present as a filler in the drugs resulted in microembolization in a variety of organs and alterations in pulmonary function. In animal studies, talc has been shown to cause granulomas and mild inflammation when inhaled. Observations of the effects that occurred in the lungs of rats exposed by inhalation to talc suggested that the operative mechanisms may be similar to those identified for carbon black, and talc is known to cause the release of cytokines, chemokines and growth factors from pleural mesothelial cells. IARC: There is *inadequate evidence* in humans for the carcinogenicity of inhaled talc not containing asbestos or asbestiform fibres. There is *limited evidence* in experimental animals for the carcinogenicity of talc not containing asbestos or asbestiform fibres. Inhaled talc not containing asbestos or asbestiform fibres is *not classifiable as to its carcinogenicity (Group 3)*.
Mutagenicity/ Genotoxicity	Talc was not mutagenic in host-mediated assays in mice. It did not produce chromosomal aberrations or dominant lethal mutations in rats. The IARC (1987) review of talc included unpublished results from a 1974 study conducted by Litton Bionetics that showed no mutagenic activity for talc *in vitro* or *in vivo.* Talc did not induce mutations in *Salmonella typhimurium* strains TA1530 or HisG46, or in the yeast, *Saccharomyces cerevisiae***.** No chromosomal aberrations were observed in human fibroblasts treated with talc *in vitro. In vivo* tests conducted in rats gave negative results for induction of chromosomal aberrations in bone marrow cells and dominant lethal mutations in germinal cells
Reproductive Toxicity Developmental Toxicity/Teratogenicity	No teratological effects were observed in hamsters, rats, mice, or rabbits after oral administration of 900-1600 mg/kg. No teratologic effects were observed in hamsters, rats, mice, or rabbits after oral administration of talc. The doses used were 1,600 mg/kg for rats and mice on days *6* through 15 of gestation; 1,200 mg/kg for hamsters on day 6 through 10 of gestation; and 900 mg/kg for rabbits on days 6 through 18 of gestation
Acute Toxicity	Acute inhalation exposure to talc causes symptoms such as cough, dyspnea, sneezing, vomiting, and cyanosis. Other inhalation exposure symptoms include diffuse pleural thickening and fibrous adhesions of pleural surfaces. Respiratory distress syndrome has been reported in children after massive accidental inhalation of talcum powder. Animal (rat, dog, rabbit) studies showed internal accumulation of talc after short- and long-term inhalation exposure as well as numerous lung afflictions such as fibrosis and inflammation.
Irritation	In monkey eyes, talc in the anterior chamber has induced persistent glaucoma. Talc can induce severe granulomatous reactions when introduced into wounds. It has induced granulomas in and about the human eye when as a dusting powder for surgeons' gloves.

Sensitisation	Talc particles are smaller than 1 um and these particles are respirable and produce an intense inflammatory response characterized by cough, rhinitis, dyspnea, and vomiting.
Health Effects Summary	This chemical has been identified by NICNAS to be of low concern to human health, and it is listed by the US Food and Drug Administration (FDA) as a Generally Recognised as Safe (GRAS) substance.
Key Study/Critical Effect for Screening Criteria	There are no adequate studies for which to derive am oral reference dose. Talc is poorly absorbed from the gastrointestinal tract, if at all, and the limited data available by the oral route indicate that talc is essentially non-toxic by the oral route of exposure
Ecological Toxicity [2,3,4]	
Aquatic Toxicity	No data were found. Talc is expected to have low toxicity to the environmental based on its ubiquity in the environment, its low bioavailability, and its widespread use in consumer products (Zazenski et al. 1995).
Determination of PNEC aquatic	PNEC values for talc cannot be calculated.
Current Regulatory Controls	
Australian Hazard Classification	No data available
Australian Occupational Exposure Standards	TWA: 2.5 mg/m^3
International Occupational Exposure Standards	NIOSH: TWA 2 mg/m^3
Australian Food Standards	No data available
Australian Drinking Water Guidelines	No data available
Aquatic Toxicity Guidelines	No data available
PBT Assessment [4]	
P/vP Criteria fulfilled?	Talc does not biodegrade in the environment. It is a naturally-occurring mineral and is persistent in the environment. However, for the purposes of this PBT assessment, it does not meet the criteria for persistence.
B/vB criteria fulfilled?	Talc is not expected to be bioavailable to aquatic organisms; thus, it is does not meet the criteria for bioaccumulation
T criteria fulfilled?	Talc is not expected to be bioavailable to aquatic organisms; thus, it is does not meet the criteria for toxicity.
Overall conclusion	It is not currently possible to categorise the environmental hazards of metals and other inorganic chemicals according to standard persistence, bioaccumulation and toxicity (PBT) hazard criteria. These criteria were developed for organic chemicals and do not take into account the unique properties of inorganic substances and their behaviour in the environment (UNECE 2007; US EPA 2007).
Revised	April 2018

References

1. HSDB (n.d.). *Hazardous Substances Data Bank*. Retrieved 2015, from Toxnet, Toxicology Data Network, National Library of Medicine: http://toxnet.nlm.nih.gov/cgi-bin/sis/htmlgen?HSDB

2. IARC (2010) Carbon Black, Titanium Oxide and Talc. Volume 93. International Agency for Research on Cancer Monographs on the Evaluation of Carcinogenic Risks to Humans. Available at http://monographs.iarc.fr/ENG/Monographs/vol93/mono93.pdf.

3. Pfizer (2006) Material Safety Data Sheet for Gemfibrozil Tablets, 90mg. Available at http://www.pfizer.com/files/products/material_safety_data/CI-719.pdf.

4. Department of the Environment and Energy 2017 , National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme

Toxicity Summary - Amides, tall-oil fatty, N,N-bis(hydroxyethyl)

Chemical and Physical Properties [1,2]	
CAS number	68155-20-4
Molecular formula	UVCB
Molecular weight	370 (typical C18 monounsaturated)
Solubility in water	Dispersible
Melting point	<25 °C (liquid)
Boiling point	>300 °C (estimated)
Vapour pressure	$<1.0\times10^{-10}$ (estimated)
Henrys law constant	$<1.0\times10^{-10}$ (estimated)
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	Liquid
Overview	Non-confidential information in the IUR indicated that the industrial processing and uses of the chemical include other basic organic chemical manufacturing as surface active agents and intermediates; pesticide and other agricultural chemical manufacturing as surface active agents; soap and cleaning compound manufacturing as surface active agents; support activities for mining as surface active agents; and petrochemical manufacturing as surface active agents. Non-confidential commercial and consumer uses of this chemical include lubricants, greases and fuel additives.
Environmental Fate [1,2]	
Soil/Water/Air	The members of the fatty nitrogen derived amides category are long-chain alkyl substituted amides used in commercial product mixtures. The category consists of three subcategories: Subcategory I, fatty acid amides; Subcategory II, fatty alkanolamides; and Subcategory III, fatty acid reaction products with amines. The components of Subcategory I are solids possessing low vapor pressure and low water solubility. The substances in Subcategory II contain solids and liquids with negligible to low vapor pressure and tend to be dispersible in water. The substances in Subcategory III also contain solids and liquids possessing negligible to low vapor pressure that tend to be dispersible in water. The fatty acid amides (Subcategory I) and the fatty acid reaction products with amines (Subcategory III) are expected to possess low mobility in soil. The fatty alkanolamides (Subcategory II) are expected to possess moderate to high mobility in soil. Volatilization is low to moderate for the fatty acid amides and low for the fatty alkanolamides and the fatty acid reaction products with amines. The rate of hydrolysis is considered negligible for all category members. The rate of atmospheric photooxidation is considered moderate to rapid for members of each subcategory; however, this is not expected to be an important environmental fate process since these substances are not expected to exist in the vapor phase in the atmosphere. The overall weight of evidence suggests that the members of the fatty nitrogen derived amides category should possess low persistence (P1) and low bioaccumulation potential (B1) with the exception of two members of subcategory III. Fatty acids, tall-oil, reaction products with tetraethylenepentamine and fatty acids, tall-oil, reaction products with polyethylenepolyamines are expected to possess low persistence (P1), but moderate bioaccumulation potential (B2). As there is limited toxicological data on amides, tall oils fatty, N,N-bis(hydroxyethyl), read across information has been obtained from oleamide DEA (CAS No. 93-83-4) because amides, tall oils fatty, N,N-bis(hydroxyethyl) is

	predominantly diethanolamides of unsaturated C18 fatty acids similar to the composition of oleamide DEA.
Human Health Toxicity Summary [1,2, 3,4]	
Chronic Repeated Dose Toxicity	Based on read-across from CAS 93-83-4, an oral sub-acute repeated dose toxicity study reported NOAEL = 750 mg/kg/day. Groups of 10 male and 10 female Wistar rats were orally gavaged with the substance diluted in olive oil, 5 d/week for 28 d at doses of 0, 70, 250, 750 (Days 1-14) and 1500 (Days 15-28) mg/kg bw/d. Clinical signs, bodyweight, haematology, clinical chemistry, urinalysis, gross and microscopic pathology were recorded. Additional groups of 5 male and 5 female rats were kept for a 4 month recovery period. No treatment-related adverse effects were observed at any of the doses. Changes in the forestomach at some doses including controls were attributed to the use of olive oil and found to be reversible after end of exposure. Under the study conditions, the 28 d NOAEL to rats was considered to be >750 mg/kg bw/day (Potokar, 1983).
Carcinogenicity	Not regarded as carcinogenic.
Mutagenicity/ Genotoxicity	Based on read-across from CAS 93-83-4, the test substance was negative in short-term in vitro and in vivo genotoxicity tests.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Based on read-across from CAS 68603-42-9, the results from a developmental toxicity study showed that repeated oral administration of COMPERLAN KD to pregnant rats on day 6 through 15 of gestation, caused no symptoms of cumulative toxicity up to a dose level of 1000 mg/kg/day. With the exception of salivation and propulsion of the head during the dose administration, there were no treatment-related effects. Also, COMPERLAN KD does not reveal any embryotoxic or teratogenic potential at dose levels up to 1000 mg/kg/day (author of the report).
Acute Toxicity	Acute oral and dermal toxicities of CAS 68140-00-1 in rat and rabbit, respectively, are low. Further, CAS 93-83-4 is not considered acutely toxic via oral route of exposure with a LD50 of 10,000 mg/kg in rats. Based on read-across from CAS 68140-00-1, an oral acute toxicity test on rats reported LD50 > 5 g/kg. All animals survived the 8-day observation period and no adverse effects were observed. With respect to the determined LD50 value, it is assumed that the LD50 value for female rats also exceeds the limit dose of > 2000 mg/kg body weight. In a dermal acute toxicity test on rabbits, LD50 > 2 g/kg was reported. All animals survived. All animals appeared normal through day 14. Two females that had abraded skin lost weight (0.01 and 0.25 kg) over the 14-day post-exposure period. All remaining rabbits gained weight through day 14. Swiss-Webster mice (4 males/dose) were administered “Alkanolamide #1”, identified in the robust summary as CASRN 68144-20-4, via whole body exposure for 3 hours. Doses were 86- 219 mg/m3 (0.086 – 0.219 mg/L). Animals were observed for several days. No mortality was observed. LC50 > 0.219 mg/L
Irritation	CAS 93-83-4 is considered irritating to skin and eyes.
Sensitisation	The test substance did not cause sensitisation on laboratory animals.
Health Effects Summary	Acute oral and dermal toxicities of CAS 93-83-4 are low. It is considered a skin and eye irritant but does not cause skin sensitisation. It is considered not toxic via repeated oral doses and not genotoxic or carcinogenic. It has no reported adverse reproductive or developmental effects.
Key Study/Critical Effect for Screening Criteria	The oral repeated dose toxicity in rats was considered the most sensitive endpoint with a NOAEL of 750 mg/kg bw/day.
Ecological Toxicity [1, 3]	
Aquatic Toxicity	Based on read-across for CAS No: 68603-42-9 Daphnia: EC50 (24-hour): 3.3 mg active matter/l Daphnia: 48-hour LC50 = 2.15 and 2.64 mg/l Based on read-across for CAS No: 112-84-5 The experiment measured the survival and reproduction of Daphnia magna over a 21-day exposure to the test and control substances. Daphnids were cultured in

	the laboratory using Elendt M7 medium and a daily feeding regiment of green algal cells (Chlorella vulgaris). Four experimental groups: control (Elendt M7 medium), solvent control (0.1 ml methanol/l), 33 µg/l, and 100 µg/l (nominal concentrations) were used in a static-renewal exposure system. All test solutions were prepared with Elendt M7 medium. Replicate test vessels consisted of 4 oz glass bottles containing 100 ml of test solution. There were 10 replicates per experimental group. On the day of test initiation, neonate daphnids were removed from cultures and placed in a crystallizing dish containing Elendt M7 medium. One daphnid was placed in each replicate test vessel, and each vessel was randomly placed in the testing area. Light intensity was not measured, but ambient laboratory lighting was provided with a photoperiod of 16 hours light/8 hours dark. Each day, test solutions were renewed, and the daphnids were fed 1.7 x 10(5) cells/ml of Chlorella vulgaris. Adult survival and reproduction was assessed each day and neonates were removed daily. The pH, dissolved oxygen (DO) and total hardness (as mg/l CaCO(3)) were measured on test days 0, 1, every Tuesday and Friday and on day 21. Means and ranges for temperature, water pH, DO and total hardness were 19.7 °C (14.5 - 25.0 °C), 7.6 (7.2 - 8.1), 8.2 mg/l (4.5 - 9.3 mg/l) and 245 mg/l (234 - 256 mg/l) as CaCO(3), respectively. Concentrations of the test substance in exposure solutions were measured on test days 0, 1, 5, 9, 12, 16 and 19 in both the old and the new solutions. Effect concentrations were based on mean measured concentrations. 21 d NOEC = 0.08 mg/L
Determination of PNEC aquatic	Applying an assessment factor of 1000 to the NOEC (0.08 mg/l) gives a PNEC of 0.08 µg/l.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Expected to be readily biodegradable based on similar substances.
B/vB criteria fulfilled?	No. Based on BAF = 108 and log Kow of 3 (estimated)
T criteria fulfilled?	No. Acute toxicity data was >1 mg/L.
Overall conclusion	Not PBT
Revised	January 2019

References

1. OECD, Amides, tall-oil fatty, N,N-bis(hydroxyethyl), Retrieved 2019: http://www.echemportal.org
2. USEPA Hazard Characterization Document, Fatty Nitrogen Derived (FND) Amides Category, September 2010
3. Halliburton Safety data sheet Date / Revised: 31.08.2018 Version: 3 Product: DCA-32014
4. ECHA REACH, Amides, C18-unsatd., N,N-bis(hydroxyethyl), Retrieved 2022: https://echa.europa.eu/

Toxicity Summary - Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivatives residues

Chemical and Physical Properties[1]	
CAS number	68909-77-3
Molecular formula	C36H78N6O14
Molecular weight	UVCB
Solubility in water	100 g/L at 20 °C
Melting point	-20 °C at 101.3 kPa
Boiling point	223 °C at 101.3 kPa
Vapour pressure	0.55 - 20 Pa at 20 - 25 °C
Henrys law constant	No data available
Explosive potential	Non-explosive (100%)
Flammability potential	Not classified (50%), Non-flammable (50%)
Colour/Form	Liquid
Overview	The residuum from the reaction of diethylene glycol and ammonia. It consists predominantly of morpholine-based derivatives such as [(aminoethoxy)ethyl]morpholine, [(hydroxyethoxy)ethyl]morpholine, 3-morpholinone, and 4,4'-(oxydi-2,1-ethanediyl)bis[morpholine].
Environmental Fate[1]	
Soil/Water/Air	The substance is hydrolytically stable at pH 4, 7 and 9 at 25°C. Adsorption to solid soil phase is not to be expected. Based on the assessment of the components, it can be concluded that the mixture will not evaporate into the atmosphere. The substance is not readily biodegradable. Based on the low log Kow, the substance will have a low potential for bioaccumulation. Over time, the mixture will preferentially distribute into the compartment water.
Human Health Toxicity Summary[1]	
Chronic Repeated Dose Toxicity	No adverse effects were observed in male and female rats in a 28 days and a 90 days repeated dose toxicity test conducted according to OECD 407 and OECD 408 respectively (Calvert Laboratories, Inc., 2011 and Envigo Research Limited, 2018) in which animals were exposed orally (gavage) to 0, 100, 500 or 1000 mg/kg bw/d (28 days study) and to 0, 10, 100 or 1000 mg/kg bw/d (90 days study). In both studies, an NOAEL of 1000 mg/kg bw was determined.
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	In an in vivo micronucleus test on mouse bone marrow erythrocytes (BioReliance, 2010), performed according to OECD guideline 474, the animals were exposed orally (via gavage) with 500, 1000, 2000 mg/kg. This did not induce a statistically positive increase in micronuclei in the hemopoietic cells of the mouse bone marrow at the time intervals evaluated under the experimental condition of this assay. No toxicity was observed. Vehicle and positive controls were valid.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	A Reproductive / Development Toxicity Screening Testing in Rats was performed according to OECD guideline 421 (Calvert Laboratories, Inc., 2011, Klimisch 1). In this GLP-compliant study, male and female Sprague-Dawley rats (10 animals/sex/dose) were exposed to 1000 mg/kg bw/day. Control animals received concurrent vehicle (water). The rats were exposed on a daily basis, starting two weeks prior to cohabitation until the day before the scheduled euthanasia (minimum of 4 weeks) for males and starting for a minimum of 15 days prior to cohabitation, during cohabitation and from presumed gestation days 0 through day 19 of gestation for females. One female animal was found dead on lactation day 2. All other females survived until scheduled sacrifice. No treatment-related effects were observed in clinical signs, mortality, body weight (gain), food consumption, gross pathology, organ weights or histopathology in the parental

	animals. There was no treatment-related effect on reproductive performance or in reproductive parameters like for instance number of gravid animals, corpora lutea per dam, total implantations, litter viability or foetal sex ratios. The incidence of neonates born alive/found dead, stillborn or missing between lactation days 0 -4 was comparable among study groups. No treatment-related malformations were observed for neonates. The body weight of neonates was statistically significantly increased with an unknown biological significance for this finding. A NOAEL of 1000 mg/kg bw/day was established.
Acute Toxicity	The oral LD50 in male and female Sprague-Dawley rats was determined to be 5000 mg/kg bw in a reliable, key study conducted similarly to OECD guideline 420 (Calvert Laboratories, Inc., 2010). Calvert Laboratories Inc (2010) determined acute dermal toxicity in 10 male / female rats following a GLP-compliant study performed equivalent to OECD guideline 402 (key study, Klimisch 1). Only one dose was tested (2000 mg/kg bw). No mortality was observed. Shortly after exposure, local erythema and oedema effects were observed in both males and females. The effects seemed to be reversible in males and 1 female. In the 4 other females, necrosis and slouching appeared.
Irritation	The skin irritation potential of the test substance is investigated in a key, reliable study performed according to OECD guideline 404 (Allen, 1994; Klimisch 2) in 3 rabbits. Semi-occlusive patches were removed from shaved test sites after exposure periods of 3 min, 1h, and 4h to 0.5mL of the test substance. The Draize scoring system was used to evaluate the results. No erythema/eschar formation and no oedema formation is observed after 3 min and 1 hour exposure. After 4 hours of exposure, very slight erythema in all 3 animals and very slight oedema in 2 animals was observed, but this was fully reversible within 24 - 48 hrs. Calvert Laboratories, Inc. (2010) investigated the eye irritation potential of the test substance in a key, reliable study performed according to EU method B.5 (Klimisch 1) in 3 rabbits. The treated eyes (with 0.1mL test substance) will remain unrinsed for at least 24 hours after instillation. The 24-, 48- and 72-hours scores will be added separately for each animal and each total divided by 3 to yield the individual mean scores for each animal. Based on the data and according to the criterial of the CLP regulation, the test substance is classified as irritant to the eyes (category 2).
Sensitisation	Based on a guinea pig maximization study, performed according to the OECD guideline 406 (Allen, 1996; Klimisch 2), the test substance is considered not sensitising to the skin.
Health Effects Summary	This chemical may cause skin and eye irritation.
Key Study/Critical Effect for Screening Criteria	The critical lowest No Observed Adverse Effect (NOAEL) level for the purposes of risk assessment is 1000 mg/kg bw/day from the 90 day repeated oral toxicity study.
Ecological Toxicity [1]	
Aquatic Toxicity	In a static test following the procedures of the German national standard DIN 38412 using Leuciscus idus as test species a LC50 (96 h) of 681.2 mg/L (nominal) was determined [BASF AG, 1988; Study No. 10F0118/885140]. In conclusion, the substance is with high probability not acutely harmful to fish. The EC50 of the test item on daphnids was found to be greater than 122 mg/L (measured value) in a GLP guideline study according to OECD 202 [BASF SE, 2010; Study No. 50E0396/09E012]. Therefore, the test substance is with high probability acutely not harmful to aquatic invertebrates. A study was performed to assess the effect of the test item on the growth of the green alga Pseudokirchneriella subcapitata. The method followed that described in the OECD Guidelines for Testing of Chemicals (2006) No 201, "Freshwater Alga and Cyanobacteria, Growth Inhibition Test" referenced as Method C.3 of Commission Regulation (EC) No 440/2008. The effect of the test item on the growth of Pseudokirchneriella subcapitata has been investigated over a 72-Hour

	period. the ErC50(72h) of the test item is 45 mg/L for Pseudokirchneriella subcapitata. The toxicity to microorganisms was determined in a GLP short-term respiration test according to OECD guideline 209 using activated sludge from a municipal sewage treatment plant. After 180 minutes no inhibition effect on the respiration rate at the highest test concentration (1000 mg/L) was observed [BASF 2010; Study No. 08G0396/09G004]. Therefore, it can be concluded that inhibition of the degradation activity of activated sludge is not anticipated when introduced in appropriately low concentrations.
Determination of PNEC aquatic	The available short-term aquatic tests covering the three trophic levels (fish, daphnids, algae) showed the lowest L(E)C50 to be 45 mg/L in algae. An assessment factor of 1000 was used for a resulting PNEC for of 0.045 mg/L.
Current Regulatory Controls[4]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1]	
P/vP Criteria fulfilled?	Not readily biodegradable. Thus, it is expected to meet the screening criteria for persistence.
B/vB criteria fulfilled?	As the Log Pow is 0.565 at 20 °C (Log Pow < 4.5), it is not expected to be bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of this chemical is >0.1 mg/L. Thus, it is not expected to meet the screening criteria for toxicity
Overall conclusion	Not PBT
Revised	March 2019

References

1. ECHA REACH, Ethanol, 2,2'-oxybis-, reaction products with ammonia, morpholine derivs. residues, Retrieved 2019: https://echa.europa.eu/

Toxicity Summary - (2-Methoxymethylethoxy)propanol

Chemical and Physical Properties[1,2,3]	
CAS number	34590-94-8
Molecular formula	C7H16O3
Molecular weight	148.20
Solubility in water	1 g/L at 25 °C and pH 7
Melting point	-83 °C at 101.325 kPa
Boiling point	190 °C at 101.325 kPa
Vapour pressure	37.1 Pa at 20 °C
Henrys law constant	No data available
Explosive potential	Non-explosive
Flammability potential	Non-flammable
Colour/Form	Colourless organic liquid with a mild odour
Overview	(2-Methoxymethylethoxy) propanol is used as hydraulic fluid and as a high boiling solvent. A Tier 1 Human Health and Environmental Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health and the environment.
Environmental Fate[1]	
Soil/Water/Air	The substance has a low Kow and a high water solubility, therefore has a low potential for adsorption to soil or sediments, and a low potential for bioaccumulation in biota. If released to air, The substance will rapidly react in the atmosphere with hydroxyl radicals. If released directly to water, the substance will remain in the water compartment and ultimately biodegrade, as the substance meets the criteria for “ready biodegradation reaching the 10 day window
Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	The 28-day oral gavage study in rats is of high quality and considered to be reliable without restrictions. The only effects observed during this study were salivation and increased liver weights at the highest dose level. The liver weight increase observed at the highest dose level was only slight and no histopathologic changes, except for hypertrophy, accompanied this effect. There were no changes in clinical chemistry (ALP, ASP) indicating a liver damage. The same effect was observed with other structurally related molecules, e.g. propylene glycol methyl ether has been shown to cause liver weight increases via a phenobarbital-like enzyme induction mode of action and it is highly likely that dipropylene glycol methyl ether liver weight increases occur via the same mode of action. As this is an adaptive effect typical for many glycol ethers, it is not considered as adverse. Based on the results of this study a no observed adverse effect level (NOAEL) of 1000 mg/kg bw/day and a no observed effect level (NOEL) of 200 mg/kg/day can be established in rats under the conditions of this study. The two studies via the dermal route are both reliable with restrictions as they were not conducted under GLP, but are equivalent to OECD guidelines. No adverse effects were observed up to 1000 mg/kg bw/day in a 28 -day study in rats. In a 90-day study in rabbits dipropylene glycol methyl ether produced some narcosis at 10 ml/kg bw/day and 5 ml/kg bw/day. No narcosis was observed at lower dose levels (1.0 and 3.0 ml/kg bw/day). Mortality was high at the 10.0 ml/kg dose level, some mortality was observed at 5.0 ml/kg bw/day and no mortality was observed at the 1.0 and 3.0 ml/kg bw/day dose levels. No haematological changes occurred at any dosage level. No significant organ weight changes occurred at any dosage level. Observations for gross pathology revealed only gastric distension and occasional gastric irritation in those animals dying at the 10 ml/kg dosage level. Histopathological analysis done on the liver, lung, spleen, adrenal, heart, testes and stomach of those animals receiving the 5.0 and 10.0 ml/kg bw/day dose levels

	revealed no changes. The kidneys of those animals on the 10.0 ml/kg bw/day level showed some granular and some hydropic changes, at the 5.0 ml/kg same kidney abnormalities were observed but they were of no greater intensity than those observed in some of the controls. The effect of severe (repeated and prolonged) exposure to the skin was slight, being similar to that caused by distilled water under similar conditions. Based on the results of this study a NOAEL of 3.0 ml/kg bw/day (2850 mg/kg/day) was established for dermal exposure to dipropylene glycol methyl ether. No significant adverse effects were observed in rats, rabbits, guinea pigs and monkeys after repeated inhalation exposure to dipropylene glycol methyl ether at any of the test concentrations. The 90 -day inhalation studies in rats and rabbits were selected as key studies as these studies are reliable without restrictions. The highest concentration tested in these studies were 200 ppm which was identified as the NOAEC. Based on the molecular weight of 148, this converts to 1232 mg/m^3 at 20 deg Celcius and 1 atm.
Carcinogenicity	No specific studies for the substance are available. Two inhalation studies with propylene glycol methyl ether in rats and mice are available for read-across to dipropylene glycol methyl ether. Both studies are reliable without restrictions as they were conducted under GLP and according to OECD guideline 453. No carcinogenic effect as evidenced by any increase in tumour incidence occurred from exposure to propylene glycol methyl ether
Mutagenicity/ Genotoxicity	The substance was not mutagenic in bacteria (Salmonella typhimuriumTA 1535, TA 1537, TA 1538, TA 98, and TA 100) and in yeast, and no cytogenetic effect were observed in mammalian cells. The data available indicates that the substance is not genotoxic.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No treatment related adverse effects - no maternal toxicity, no embryo-/fetotoxicity and no teratogenicity - were observed in rats or rabbits at the highest attainable concentration of dipropylene glycol methyl ether. The studies in both species are of good quality and reliable without restrictions. The no observed adverse effect level for dipropylene glycol methyl ether is 300 ppm in both species.
Acute Toxicity	Oral - All acute toxicity studies via the oral route reported LD50 values greater than 5000 mg/kg for dipropylene glycol methyl ether. The key study identified for acute oral toxicity is the BASF (1979) study in rats with a reported LD50 of greater than 5000 mg/kg body weight. Inhalation - Via the inhalation route no mortality was observed at the highest attainable concentration (i.e. LC0 values > ca. 552.6 ppm, 3404.47 mg/m^3) in three independent studies. The key study identified is the BASF (1979) study in rats with a LC0 greater than 275 ppm (duration 7 hours) which would be equivalent to approximately 1.69422 mg/L (based on conversion equation at 20 degree celsius and 1 atmosphere). Using Haber's law for converting this 7-hour exposure to a 4 -hour exposure, the equivalent LC0 value is greater than 2.04 mg/L or 2040 mg/m^3. Dermal - For the dermal route, two studies reported no mortality up to the highest dose tested (20 ml/kg bw) in rats and rabbits. One study in rabbits reported a dermal LD50 of 10 ml/kg bw (9510 mg/kg bw). The lowest LD50 will be taken into account for the risk assessment. The other study reported LD50 greater than 19020 mg/kg body weight in rats.
Irritation	Several non-GLP studies in rabbits equivalent or similar to OECD guidelines 404 and 405 are available for the substance. These studies are supported by a human volunteer study for eye irritation and a 90-day dermal study in rabbits. No irritation was observed in rabbits and humans
Sensitisation	No sensitization reaction was observed with the substance in the study with human volunteers.
Health Effects Summary	Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.
Key Study/Critical Effect for Screening Criteria	The oral repeated dose toxicity in rats was considered the most sensitive endpoint with a NOAEL of 1000 mg/kg bw/day.
Ecological Toxicity[1,2,3]	
Aquatic Toxicity	Acute toxicity studies have been conducted in fish, daphnia and algae. In summary, for the aquatic compartment dipropylene glycol methyl ether shows

	EC50s/LC50s that exceed 1000 mg/l in daphnia (48 hr), fish (96 hr) and algae (7 days). The NOEC for reproduction of Daphnia magna corresponds to the highest concentration tested of 0.5 mg/L in the long-term test, which was set very low considering the low acute toxicity of the substance on Daphnia magna. The low chronic toxicity is highlighted in a freshwater algae test with a NOEC at 1000 mg/L. An activated sludge respiration inhibition test showed an EC50 of 4168 mg/L for micro-organisms.
Determination of PNEC aquatic	Data from short-term tests with three trophic levels and one long-term test on invertebrates are available. An assessment factor of 100 is applied to the lowest NOEC of 0.5 mg/L (daphnia). A PNECaqua of 0.005 mg/L was derived.
Current Regulatory Controls[4,5,6]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	A TWA of 50 ppm (308 mg/m^3) is recommended to protect for eye, nose and throat irritation in exposed workers
International Occupational Exposure Standards	TLV: 100 ppm as TWA; 150 ppm as STEL; (skin). MAK: 310 mg/m^3, 50 ppm; peak limitation category: I(1); pregnancy risk group: D. EU-OEL: 308 mg/m^3, 50 ppm as TWA; (skin)
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	No. Expected to be readily biodegradable.
B/vB criteria fulfilled?	No. Based on the Log Kow of 0.004 at 25 °C (Log Kow < 4.2).
T criteria fulfilled?	No. Chronic toxicity data >0.01 mg/L in invertebrates, thus the substance does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	December 2021

References

1. ECHA REACH, 2,2',2''-(hexahydro-1,3,5-triazine-1,3,5-triyl)triethanol, Retrieved 2021: https://echa.europa.eu/
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.industrialchemicals.gov.au/.
3. NCBI, 2021. National Center for Biotechnology Information. Available at: https://pubchem.ncbi.nlm.nih.gov/. Retrieved December 2021
4. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
5. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.
6. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.

Toxicity Summary - 1-Tetradecene

Chemical and Physical Properties[1,2]	
CAS number	1120-36-1
Molecular formula	C14H28
Molecular weight	196.37
Solubility in water	4.0 x 10^{-4} mg/L at 25°C (estimated)
Melting point	-12°C
Boiling point	233.0 °C
Vapour pressure	1.5 x 10^{-2} mm Hg at 25°C
Henrys law constant	8.48 atm-cu m/mole at 25°C (estimated)
Explosive potential	No data available.
Flammability potential	Non-flammable
Colour/Form	Watery liquid; colourless; mild pleasant odour.
Overview	1-Tetradecene is an anthropogenic compound which is used as a specialty solvent. It may be released to the environment as a fugitive emission during its production and use, and as a result of the burning of plastics.
Environmental Fate[1,2]	
Soil/Water/Air	If released to soil, 1-tetradecene will be essentially immobile. It may rapidly volatilize from moist soil to the atmosphere although its expected strong adsorption to soil may attenuate the rate of this process. 1-Tetradecene will not volatilize from dry soil to the atmosphere. Pure culture studies indicate that 1-tetradecene has the potential to biodegrade in soil and water under aerobic conditions. If released to water, 1-tetradecene will bioconcentrate in fish and aquatic organisms and strongly adsorb to sediment and suspended organic matter. It may rapidly volatilize from water to the atmosphere. The estimated half-life for volatilization from a model river is 4.1 hrs. Its expected strong adsorption to sediment and suspended organic matter may attenuate the rate of this process. The estimated half-life for volatilization from a model pond, which takes into account adsorptive processes, is 7.3 months. If released to the atmosphere, 1-tetradecene may undergo removal by gas-phase reaction with atmospheric oxidants. Estimated half-lives for the reaction with photochemically produced hydroxyl radicals and ozone are 9.3 hrs and 23 hrs.
Human Health Toxicity Summary[1,2]	
Chronic Repeated Dose Toxicity	Guideline repeat dose toxicity studies in rats have been conducted for fourteen members of the higher olefin category, covering C6 to C20-24. The majority of these investigations (27 studies) have used oral (gavage) exposure, with three sub-acute (28-day), nine screening (OECD 421/422), and seven sub-chronic (90-day) studies available for this route. Two sub-acute dermal, two sub-acute inhalation and one sub-chronic inhalation tests, are also available; eight short-term repeat dose range-finding studies are also available. For the oral studies, systemic toxicity findings were typically limited to body weight, liver changes, and effects on clinical chemistry parameters as well as organ weights. Some of the effects observed were adaptive rather than adverse. While most of the studies revealed no systemic toxicity at doses up to 1000 mg/kg bw/day, a conservative NOAEL for this category was determined to be 100 mg/kg bw/day, based on minor effects observed with some category members. The inhalation toxicity NOAEC was determined to be 3,000 ppm (10,326 mg/m^3).
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	There was no evidence of mutagenicity or genotoxicity in any of the studies.

Reproductive Toxicity / Developmental Toxicity/Teratogenicity	The weight of evidence from oral reproductive and developmental toxicity studies, accompanied with data from oral and inhalation sub-chronic toxicity studies in rats indicate that category members have little or no potential to be considered reproductive/developmental toxicants.
Acute Toxicity	Not acutely hazardous after ingestion, inhalation or skin contact, based on read across animal test data. The acute oral LD50 for hex-1-ene (Neodene 6) alpha olefin in male and female rats was reported as >5600 mg/kg. To assess acute oral toxicity of alkenes, C20-24, groups of 5 fasted female Sprague-Dawley CD strain rats were given a single oral dose (2000 mg/kg bw) of ENORDET O241 and observed for 14 days (Sanders, 2008). There were no treatment related clinical signs, necropsy findings or changes in body weight. The oral LD50 was determined to be greater than 2000 mg/kg in this single sex study.
Irritation	Not irritating to skin and eyes.
Sensitisation	There was no evidence of dermal sensitization in any of the studies.
Health Effects Summary	The substance is expected to have low acute toxicity and is not an irritant.
Key Study/Critical Effect for Screening Criteria	A conservative NOAEL for this category was determined to be 100 mg/kg bw/day, based on minor effects observed with some category members.
Ecological Toxicity[1,2]	
Aquatic Toxicity	Short term toxicity: LC50 (4 days): 3.4 µg/L (fish) EC50 (48 h): 2.8 µg/L (invertebrates) EC50 (4 days): 4.5 µg/L (algae) Long term toxicity: NOEC (21 days): 19.4 µg/L (invertebrates)
Determination of PNEC aquatic	Based on the lowest chronic endpoint for aquatic toxicity (19.4 µg/L), an assessment factor of 100 has been applied, resulting in a PNECaquatic of 0.194 µg/L.
Current Regulatory Controls[3,4,5]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1,4]	
P/vP Criteria fulfilled?	No. Expected to be readily biodegradable.
B/vB criteria fulfilled?	Yes. Bioaccumulation of this substance may occur in aquatic organisms based on the estimated Log Kow of 7.3 (Log Kow > 4.2)
T criteria fulfilled?	No. Chronic toxicity data >0.01 mg/L in invertebrates, thus the substance does not meet the screening criteria for toxicity.

Overall conclusion	Not PBT
Revised	December 2021

References

1. ECHA REACH, Tetradec-1-ene, Retrieved 2021: https://echa.europa.eu/
2. NCBI, 2021. National Center for Biotechnology Information. Available at: https://pubchem.ncbi.nlm.nih.gov/. Retrieved December 2021
3. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
4. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.
5. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.

Toxicity Summary - Aluminium

Chemical and Physical Properties[1,2,3,4,5]	
CAS number	7429-90-5
Molecular formula	Al
Molecular weight	26.982
Solubility in water	Insoluble
Melting point	660.32°C
Boiling point	2,327°C
Vapour pressure	0
Henrys law constant	No data available
Explosive potential	No data
Flammability potential	Finely divided aluminium dust is easily ignited
Colour/Form	Silver white, malleable, ductile metal, cubic crystal, odourless
Overview	Aluminium is the most abundant metal in the earth's crust and it is widely distributed. Aluminium is a very reactive element and is never found as the free metal in nature. It is found combined with other elements, most commonly with oxygen, silicon, and fluorine. These chemical compounds are commonly found in soil, minerals (e.g., sapphires, rubies, turquoise), rocks (especially igneous rocks), and clays. Aluminium as the metal is obtained from aluminium-containing minerals, primarily bauxite. A Tier 1 Human Health and Environmental Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health and the environment.
Environmental Fate[1]	
Soil/Water/Air	Aluminium is the most abundant metal in the earth's crust, but is never found in its elemental state in nature. In compounds, aluminium occurs in its only oxidation state (+3). The transport and partitioning of aluminium in the environment is determined by its chemical properties, as well as the characteristics of the environmental matrix that affect its solubility. At a pH >5.5, naturally occurring aluminium compounds exist predominantly in an undissolved form such as gibbsite, Al(OH)3, or as aluminosilicates except in the presence of high amounts of dissolved organic material or fulvic acid, which binds with aluminium and can cause increased dissolved aluminium concentrations in streams and lakes. As an element, aluminium cannot be degraded in the environment, but may undergo various precipitation or ligand exchange reactions. The solubility of aluminium in the environment will depend on the ligands present and the pH.
Human Health Toxicity Summary [1,2,3,4,5]	
Chronic Repeated Dose Toxicity	Aluminium has been implicated in causing neurological and hematopoietic effects in individuals with impaired renal function. Respiratory and neurological effects have been observed in workers exposed to finely ground aluminium and aluminium welding fumes. Impaired lung function has been observed in workers employed in various aluminium industries including potrooms, foundry, and welders. Other studies have provided some suggestive evidence that aluminium exposure can result in occupational asthma or pulmonary fibrosis. A common limitation of most of these occupational exposure studies is co-exposure to other compounds, such as silica, which can also damage the respiratory tract. Subtle neurological effects have been observed in workers exposed to aluminium dust in the form of McIntyre powder, aluminium dust and fumes in potrooms, and aluminium fumes during welding. Studies examining the systemic toxicity of aluminum following chronic oral exposure have identified two potential targets of toxicity: the nervous system and the hematopoietic system.

Carcinogenicity	The current weight of evidence does not support an association between inhalation exposure to aluminium metal/aluminium oxide and cancers in the respiratory organs. The weight of evidence also does not support a systemic carcinogenic effect from exposure to aluminium metal and aluminium oxide.
Mutagenicity/ Genotoxicity	Several in vitro studies have found significant increases in the occurrence of micronuclei formation and chromosome aberrations in human lymphocytes; no human in vivo studies were identified. One study examined the in vivo genotoxicity of aluminium and found clastogenic changes in mice receiving an intraperitoneal injection of aluminium chloride. In vitro studies in mammalian and bacterial systems have not found mutagenic alterations.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No studies were located regarding reproductive effects of various forms of aluminium following inhalation, oral, or dermal exposure in humans. No histological alterations were observed in the reproductive tissues of rats or guinea pigs exposed to airborne aluminium chlorhydrate. A number of oral-exposure studies examining reproductive end points in several animal species were identified. In general, the results of these studies suggest that aluminium is not associated with alterations in fertility, mating success, or number of implantations, implantation losses, or litter size.
Acute Toxicity	Aluminium metal (dust/powder) is not to be classified for acute oral, inhalation and dermal toxicity. Oral LD50 (rat) > 2000 mg/kg bw Inhalation LC50 (rat) > 888 mg/m^3 Inhalation NOAEC (rat) = 10 mg/m^3
Irritation	Not irritating to eye and skin.
Sensitisation	Not sensitising
Health Effects Summary	Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.
Key Study/Critical Effect for Screening Criteria	The toxicological effects of Al in rodents suggests that neurotoxicological and developmental (including neurodevelopmental) endpoints are among the most sensitive indicators of Al toxicity. The LOAEL of 100 mg Al/kg-day for minimal neurotoxicity in the offspring of mice is selected as the basis for the chronic reference dose (RfD). Application of an uncertainty factor (UF) of 100 (3 for use of a minimal LOAEL, 10 for interspecies extrapolation and 3 for intra-human variability where the critical effects have been observed in a sensitive sub-group) results in a provisional RfD of 1 mg Al/kg-day.
Ecological Toxicity[6]	
Aquatic Toxicity	8-day LC50 0.17 mg/L (fish) 8-day LC50 of 2.28 mg/L (amphibian)
Determination of PNEC aquatic	PNEC freshwater: 74.9 µg/L
Current Regulatory Controls[6,7,8,9]	
Australian Hazard Classification	Aluminium powder (pyrophoric): H261 (In contact with water releases flammable gas) H250 (Catches fire spontaneously if exposed to air) Aluminium powder (stabilised): H261 (In contact with water releases flammable gas) H228 (Flammable solid)
Australian Occupational Exposure Standards	Time Weighted Average (TWA): Aluminium (metal dust) = 10 mg/m^3 Aluminium (welding fumes) (as Al) = 5 mg/m^3 Aluminium, alkyls (NOC) (as Al) = 2 mg/m^3 Aluminium, pyro powders (as Al) = 5 mg/m^3 Aluminium, soluble salts (as Al) = 2 mg/m^3

International Occupational Exposure Standards	TLV: 1 mg/m^3, as TWA; A4 (not classifiable as a human carcinogen). MAK: (inhalable fraction): 4 mg/m3; (respirable fraction): 1.5 mg/m3; pregnancy risk group: D
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	A freshwater moderate reliability trigger value of 55 µg/L was derived for aluminium at pH >6.5 using the statistical distribution method (Burr distribution as modified by CSIRO, ANZECC & ARMCANZ 2000 Section 8.3.3.3) with 95% protection and an ACR of 8.2. A freshwater low reliability trigger value of 0.8 µg/L was derived for aluminium at pH <6.5 using an assessment factor (AF) of 20 (essential element) on the low pH trout LC50 figure. The low reliability figures should only be used as indicative interim working levels. There were limited marine data and procedures for calculating an Environmental Concern Level (ECL) (ANZECC & ARMCANZ 2000 Section 8.3.4.5) were used to calculate a low reliability marine trigger value of 0.5 µg/L derived for aluminium using an AF of 200. This figure should only be used as an indicative interim working level but could be revisited as more data become available. The factor of 200 was used because the ECL factor of 1000 was considered excessive for such a commonly found element.
PBT Assessment[1]	
P/vP Criteria fulfilled?	Not applicable (aluminium as a metal do not degrade and traditional persistence measures used for organic substances do not equally apply to metals).
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to inorganic compounds; aluminium ions are ubiquitous and are present in most water, soil and sediment.
T criteria fulfilled?	No. LC50 >0.1 mg/L in fish (The lowest measured chronic figure was an 8-day LC50 of 0.17 mg/L for fish).
Overall conclusion	It is not currently possible to categorise the environmental hazards of metals and other inorganic chemicals according to standard persistence, bioaccumulation and toxicity (PBT) hazard criteria. These criteria were developed for organic chemicals and do not take into account the unique properties of inorganic substances and their behaviour in the environment.
Revised	December 2021

References

1. ECHA REACH, Aluminium, Retrieved 2021: https://echa.europa.eu/
2. USEPA, 2021. Regional Risk Levels. November 2021. https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables. Retrieved December 2021.
3. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.industrialchemicals.gov.au/.
4. ATSDR, 2008. Toxicological Profile for Aluminium. Agency for Toxic Substances and Disease Registry. September 2008.
5. USEPA, 2006. Provisional Peer-Reviewed Toxicity Values for Aluminium. U.S. Environmental Protection Agency, Washington, DC, EPA/690/R-06/001F.
6. ANZG, 2018. Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Australian and New Zealand Governments and Australian state and territory governments, Canberra ACT, Australia. Available at www.waterquality.gov.au/anz-guidelines.
7. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
8. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
9. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.

Toxicity Summary - Ammonium hydrogensulfite

Chemical and Physical Properties[1,2,3]	
CAS number	10192-30-0
Molecular formula	H3N.H2O3S
Molecular weight	99.11
Solubility in water	718 - 6 200 g/L at 0 - 60 °C
Melting point	No data available
Boiling point	No data available
Vapour pressure	No data available
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	No data available
Colour/Form	Colourless to yellow crystals
Overview	Ammonium hydrogensulfite are soluble in water. It is non-combustible. It is corrosive to aluminium. It is a strong irritant to skin and mucous membranes. It is toxic by skin absorption.
Environmental Fate[1]	
Soil/Water/Air	The substance has a very low vapour pressure, and also does not sublime. Therefore, the substance will not be present as a gas and no radical reactions can be expected. According to its chemical properties, hydrolysis is not expected/probable. Photodegradation in water is not relevant because it dissociates rapidly into ions and decomposes in water, and it not susceptible to visible light. The substance is an inorganic compound which does not undergo biodegradation. The substance readiliy dissociates in aqueous solution, as with soil moisture. Bioaccumulation is not to be expected. a low log Kow underlines this statement. Due to the ionic salt-character and other physico-chemical properties (negligible vapour pressure, very high water solubility and decomposition in water), the Henry constant is near to zero. Because of its ionic nature, ammonium hydrogensulfite as well as its dissociation products are not volatile from aqueous solutions. Relevant adsorption onto soils, sediments or suspended matter is not expected.
Human Health Toxicity Summary[1,2,3]	
Chronic Repeated Dose Toxicity	Male and female rats received 0, 0.125, 0.25, 0.5, 1.0 or 2.0% $Na_2S_2O_5$ in a thiamine-containing diet (50 ppm) for 104 weeks. 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 (or 0.215% accounting for the loss of metabisulfite). The corrected dose level corresponded to a dose of 108 mg/kg bw/d $Na_2S_2O_5$ or an equivalent dose of 113 mg/kg bw/day ammonium hydrogensulfite. Because there was no evidence of systemic toxicity following chronic treatment, the NOAEL for systemic effects can be expected above the highest dose of 2% sodium metabisulfite corresponding to 955 mg/kg bw/d of $Na_2S_2O_5$ or 996 mg/kg bw/d ammonium hydrogensulfite.
Carcinogenicity	Not considered to be carcinogenic.
Mutagenicity/ Genotoxicity	Not considered to be genotoxic
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Not considered to cause reproductive or developmental toxicity

Acute Toxicity	Based on the described read-across methodology information from sodium sulfite (CAS 7757-83-7), sodium metabisulfite (CAS 7681 -57 -4) and potassium metabisulfite (CAS 16731 -55 -8) were used to determine acute toxicity values (oral, dermal and inhalatiion) for ammonium hydrogensulfite. In total, four reliable animal studies on acute oral exposure for sulfite substances are available, conducted equivalent or similar to OECD guideline 401. One study (Grundler, 1981) indicates a LD50 value of >2610 mg/kg/bw (male and female rats) for the test item sodium sulfite (CAS 7757 -83 -7). One study performed with potassium metabisulfite (CAS 16731 -55 -8) as test item indicated a LD50 >2000 mg/kg/bw (no clinical symptoms were observed in the concentration rang 200 - 2000 mg/kg bw). Two animal study reports on acute oral exposure to sodium metabisulfite (CAS 7681 -57 -4) are available (Hofmann & Jung, 1987 and Zeller&Hofmann, 1974), conducted according to or equivalent/similar to OECD guideline 401. The study of Hofmann & Jung indicated a LD50 >1540 mg/kg/bw. whereas the study performed by Zeller & Hofmann indicated a LD50 value of >3200 mg/kg bw. One study on acute dermal toxicity, performed according to OECD 402 for the test item sodium sulfite (CAS 7757 -83 -7) is available. LD50 value was determined to be greater than 2000 mg/kg/bw (limit test).No systemic clinical observations were observed during clinical examination. No local effects were observed. One study equivalent or similar to OECD 403 for sodium sulfite (CAS 7757 -83 -7) has been performed which indicated a LC50 >5.5 mg/l (limit test). During exposure nothing abnormal was detected. After exposure: substance-contaminated heads, and unstable, staggering gait. After one day nothing abnormal was detected.
Irritation	Not irritating
Sensitisation	Not likely to be skin sensitisers
Health Effects Summary	The main critical effects to human health are severe eye irritation and acute oral toxicity. This chemical will liberate toxic gas when in contact with acid and therefore may cause effects in individuals with a high acid content in the stomach.
Key Study/Critical Effect for Screening Criteria	The chronic repeated dose study in rats with a NOAEL of 113 mg/kg bw/day ammonium hydrogensulfite was used in the risk assessment.
Ecological Toxicity[1]	
Aquatic Toxicity	Algae NOEC/EC10 = 28 mg SO_3^{2-}/L Invertebrates NOEC/EC10 = ≥8.41 mg SO_3^{2-}/L Fish NOEC/EC10 = 50 mg SO_3^{2-}/L
Determination of PNEC aquatic	The lowest value for chronic toxicity was the NOEC for invertebrates of 8.41 mg SO_3^{2-}/L. Applying the AF of 10 results in a PNECaquatic of 0.84 mg SO_3^{2-}/L. Translating this value to H3N.H2O3S gives a PNECaquatic of 1.04 mg test substance/L.
Current Regulatory Controls[2,4,5,6,7]	
Australian Hazard Classification	Acute toxicity – category 4 Eye damage – category 1
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	The following exposure standards are identified for sulfites: An exposure limit (OEL, TWA, STEL, PEL or STV) of 5 – 10 mg/m^3 in different countries such as USA, United Kingdom, Canada, Ireland, Spain, Norway and Switzerland.
Australian Food Standards	The ADI value of 0-0.7 mg/kg bw/day for sulphites was used by FSANZ for the dietary risk assessment.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	Inorganic substance comprising ions of low ecotoxicological concern. This chemical is not expected to pose an unreasonable risk to the environment provided

	that ANZECC water quality guidelines for physical and chemical stressors are not exceeded.
PBT Assessment[1]	
P/vP Criteria fulfilled?	Not applicable (inorganic substance, ionic species ubiquitous in environment)
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to these inorganic substances.
T criteria fulfilled?	No. Inorganic substance comprising ions of low ecotoxicological concern.
Overall conclusion	It is not currently possible to categorise the environmental hazards of inorganic chemicals according to standard persistence, bioaccumulation and toxicity (PBT) hazard criteria. These criteria were developed for organic chemicals and do not take into account the unique properties of inorganic substances and their behaviour in the environment.
Revised	December 2021

References

1. ECHA REACH, Ammonium hydrogensulfite, Retrieved 2021: https://echa.europa.eu/
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Sulfites: Human health tier II assessment: Retrieved 2021: https://www.industrialchemicals.gov.au/.
3. NCBI, 2021. National Center for Biotechnology Information. Available at: https://pubchem.ncbi.nlm.nih.gov/. Retrieved December 2021
4. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
5. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.
6. Food Standards Australia New Zealand (FSANZ). Retrieved December 2021: https://www.foodstandards.gov.au/consumer/additives/sulphite/Pages/default.aspx
7. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.

Toxicity Summary - Barite

Chemical and Physical Properties[1,2,3]	
CAS number	13462-86-7
Molecular formula	Ba(SO4)
Molecular weight	233.39
Solubility in water	3.1 mg/L at 20°C
Melting point	1580°C
Boiling point	1600°C at 760 mm Hg (Decomposes)
Vapour pressure	No data available
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	No data available
Colour/Form	Odourless white powder
Overview	Barium sulphate is an inorganic compound. It is partially soluble in water, dissociating into barium and sulphate ions; both are ubiquitous in the environment. The ions will not adsorb on particulate matter or surfaces and will not accumulate in living tissues A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health.
Environmental Fate[1]	
Soil/Water/Air	The dissolution of barium substances in the environment and corresponding dissolved Ba levels are controlled by the solubility of barite (BaSO4) and witherite (BaCO3), two naturally occurring barium minerals and the concentration of dissolved Ba cations in freshwater is rather low. However, in the dissolved state, the divalent barium cation, is the predominant form in soil, sediments and water. The solubility of barium compounds increases as solution pH decreases. Barium in soils is not expected to be very mobile because of the formation of water-insoluble salts (sulphate and carbonate) and its inability to form soluble complexes with humic and fulvic materials. Transport, fate, and toxicity of barium in the environment are largely controlled by the solubility of barium minerals. The barium cation is the moiety of toxicological concern, and thus the hazard assessment is based on Ba2+.
Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	The NOAEL for barium toxicity in this study is based on depressed body weight gains, elevated phosphorus levels, neurobehavioral effects and chemically related lesions in the kidney and lymphoid tissue at the highest dose level of 4000 pm. Individual effects observed at 2000 ppm barium chloride in drinking water (corresponding to the final barium dose of 61.1 and 80.9 mg Ba/kg bw/day to male and female rats respectively) were regarded as not treatment-related, and this dose level therefore represents the NOAEL. No systemic toxicity was shown to result from long term inhalation exposure in humans to high concentrations of barium sulphate. Particle overload is observed for insoluble particles such as barium sulphate, whereby the rat is the most sensitive species studied, and species-specific differences are demonstrated in various mechanistic animal studies. It has been demonstrated with reasonable certainty that lung overload conditions are not relevant for human health.
Carcinogenicity	There was no evidence of carcinogenic activity (showing no chemical related increase of malignant or benign neoplasms) of barium chloride in both sexes of rats and mice that received 500, 1250, and 2500 ppm. Thus, the concentration of 2500 ppm represents a NOAEL (corresponding to barium doses of 60 and 75 mg/kg bw/d to male and female rats, respectively, and 160 and 200 mg/kg bw/d to male and female mice, respectively).

Mutagenicity/ Genotoxicity	Not likely to be mutagenic or genotoxic.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Only a screening test is available. However, based on this study there are no indications of a substantial impairment of fertility in rats up to the highest dose tested. Thus, the NOAEL was 4000 ppm (to average doses of 201.5 and 179.5 mg Ba/kg bw/d to male and and female rats, respectively). NOAELs on developmental toxicity for rats of 4000 ppm were derived. However, this NOAEL is of limited value to evaluate the potential for barium to induce developmental effects because there was no exposure of the females during gestation.
Acute Toxicity	The toxicity of barium sulphate and barium chloride is based on the Ba2+cation and therefore on the solubility of the test substance. Barium chloride is well water soluble (ca. 375 g/L) at pH ca. 6.5 (pH of artificial sweat solution), whereas barium sulphate is low soluble (3.1 mg/L at pH 9). Due to the fact that Barium chloride seems to be no toxic via the dermal route it can be concluded that barium sulphate will result in a dermal LD50 of >>2000 mg/kg bw and should therefore not classified as acute toxic to the dermal route.
Irritation	Not irritating to skin or eyes.
Sensitisation	Barium sulphate is expected to be not sensitizing to skin.
Health Effects Summary	Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.
Key Study/Critical Effect for Screening Criteria	The repeated dose toxicity via oral application was considered the key study. Rats were dosed at 61.1 and 80.9 mg Ba2+ /kg bw/day to male and female rats via feed for 90 days. The values refer to 104 and 138 mg/kg bw/day. The NOAEL for barium toxicity in this study is based on depressed body weight gains, elevated phosphorus levels, neurobehavioral effects and chemically related lesions in the kidney and lymphoid tissue at the highest dose level. Individual effects observed were regarded as non-treatment related.
Ecological Toxicity[1]	
Aquatic Toxicity	Short-term toxicity EC/LC50 values of barium sulphate available for 3 trophic levels: 96 hrs LC50: >3.5 mg/L (Fish) 48 hrs LC50: 14.5 mg/L (Invertebrates) 72 hrs EC50: 1.15 mg/L (Algae) Long-term toxicity data are available for three trophic levels: 33 days NOEC: 1.26 mg/L (Fish) 21 days NOEC: 2.9 mg/L (Invertebrates) 72 hrs NOEC: 1.15 mg/L (Algae)
Determination of PNEC aquatic	Data from short and long-term tests with three trophic levels are available. An assessment factor of 10 is applied to the lowest EC50 of 1.15 mg/L (algae). A PNECaqua of 115 µg/L was derived.
Current Regulatory Controls[5,6,7,8]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.

PBT Assessment[1]	
P/vP Criteria fulfilled?	Not applicable (inorganic salt, ionic species ubiquitous in environment)
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to these inorganic ions; barium and sulphate ions are ubiquitous and are present in most water, soil and sediment.
T criteria fulfilled?	Not applicable. Chronic toxicity data >1 mg/L, thus barium sulphate does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	December 2021

References

1. ECHA REACH, Barite sulfate, Retrieved 2021: https://echa.europa.eu/
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.industrialchemicals.gov.au/.
3. NCBI, 2021. National Center for Biotechnology Information. Available at: https://pubchem.ncbi.nlm.nih.gov/. Retrieved December 2021.
4. EHS Support, Barium Sulfate. Available at: https://www.santos.com/wp-content/uploads/2021/04/Barium-sulfate-March-2021.pdf. Retrieved December 2021.
5. ANZG, 2018. Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Australian and New Zealand Governments and Australian state and territory governments, Canberra ACT, Australia. Available at www.waterquality.gov.au/anz-guidelines.
6. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
7. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
8. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.

Toxicity Summary - Barium Sulphate

Chemical and Physical Properties[1,2,3]	
CAS number	13462-86-7, 7727-43-7
Molecular formula	Ba(SO4)
Molecular weight	233.39
Solubility in water	3.1 mg/L at 20°C
Melting point	1580°C
Boiling point	1600°C at 760 mm Hg (Decomposes)
Vapour pressure	No data available
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	No data available
Colour/Form	Odourless white powder
Overview	Barium sulphate is an inorganic compound. It is partially soluble in water, dissociating into barium and sulphate ions; both are ubiquitous in the environment. The ions will not adsorb on particulate matter or surfaces and will not accumulate in living tissues A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health.
Environmental Fate[1]	
Soil/Water/Air	The dissolution of barium substances in the environment and corresponding dissolved Ba levels are controlled by the solubility of barite (BaSO4) and witherite (BaCO3), two naturally occurring barium minerals and the concentration of dissolved Ba cations in freshwater is rather low. However, in the dissolved state, the divalent barium cation, is the predominant form in soil, sediments and water. The solubility of barium compounds increases as solution pH decreases. Barium in soils is not expected to be very mobile because of the formation of water-insoluble salts (sulphate and carbonate) and its inability to form soluble complexes with humic and fulvic materials. Transport, fate, and toxicity of barium in the environment are largely controlled by the solubility of barium minerals. The barium cation is the moiety of toxicological concern, and thus the hazard assessment is based on Ba2+.
Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	The NOAEL for barium toxicity in this study is based on depressed body weight gains, elevated phosphorus levels, neurobehavioral effects and chemically related lesions in the kidney and lymphoid tissue at the highest dose level of 4000 pm. Individual effects observed at 2000 ppm barium chloride in drinking water (corresponding to the final barium dose of 61.1 and 80.9 mg Ba/kg bw/day to male and female rats respectively) were regarded as not treatment-related, and this dose level therefore represents the NOAEL. No systemic toxicity was shown to result from long term inhalation exposure in humans to high concentrations of barium sulphate. Particle overload is observed for insoluble particles such as barium sulphate, whereby the rat is the most sensitive species studied, and species-specific differences are demonstrated in various mechanistic animal studies. It has been demonstrated with reasonable certainty that lung overload conditions are not relevant for human health.
Carcinogenicity	There was no evidence of carcinogenic activity (showing no chemical related increase of malignant or benign neoplasms) of barium chloride in both sexes of rats and mice that received 500, 1250, and 2500 ppm. Thus, the concentration of 2500 ppm represents a NOAEL (corresponding to barium doses of 60 and 75 mg/kg bw/d to male and female rats, respectively, and 160 and 200 mg/kg bw/d to male and female mice, respectively).

Mutagenicity/ Genotoxicity	Not likely to be mutagenic or genotoxic.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Only a screening test is available. However, based on this study there are no indications of a substantial impairment of fertility in rats up to the highest dose tested. Thus, the NOAEL was 4000 ppm (to average doses of 201.5 and 179.5 mg Ba/kg bw/d to male and and female rats, respectively). NOAELs on developmental toxicity for rats of 4000 ppm were derived. However, this NOAEL is of limited value to evaluate the potential for barium to induce developmental effects because there was no exposure of the females during gestation.
Acute Toxicity	The toxicity of barium sulphate and barium chloride is based on the Ba2+cation and therefore on the solubility of the test substance. Barium chloride is well water soluble (ca. 375 g/L) at pH ca. 6.5 (pH of artificial sweat solution), whereas barium sulphate is low soluble (3.1 mg/L at pH 9). Due to the fact that Barium chloride seems to be no toxic via the dermal route it can be concluded that barium sulphate will result in a dermal LD50 of >>2000 mg/kg bw and should therefore not classified as acute toxic to the dermal route.
Irritation	Not irritating to skin or eyes.
Sensitisation	Barium sulphate is expected to be not sensitizing to skin.
Health Effects Summary	Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.
Key Study/Critical Effect for Screening Criteria	The repeated dose toxicity via oral application was considered the key study. Rats were dosed at 61.1 and 80.9 mg Ba2+ /kg bw/day to male and female rats via feed for 90 days. The values refer to 104 and 138 mg/kg bw/day. The NOAEL for barium toxicity in this study is based on depressed body weight gains, elevated phosphorus levels, neurobehavioral effects and chemically related lesions in the kidney and lymphoid tissue at the highest dose level. Individual effects observed were regarded as non-treatment related.
Ecological Toxicity[1]	
Aquatic Toxicity	Short-term toxicity EC/LC50 values of barium sulphate available for 3 trophic levels: 96 hrs LC50: >3.5 mg/L (Fish) 48 hrs LC50: 14.5 mg/L (Invertebrates) 72 hrs EC50: 1.15 mg/L (Algae) Long-term toxicity data are available for three trophic levels: 33 days NOEC: 1.26 mg/L (Fish) 21 days NOEC: 2.9 mg/L (Invertebrates) 72 hrs NOEC: 1.15 mg/L (Algae)
Determination of PNEC aquatic	Data from short and long-term tests with three trophic levels are available. An assessment factor of 10 is applied to the lowest EC50 of 1.15 mg/L (algae). A PNECaqua of 115 µg/L was derived.
Current Regulatory Controls[5,6,7,8]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.

PBT Assessment[1]	
P/vP Criteria fulfilled?	Not applicable (inorganic salt, ionic species ubiquitous in environment)
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to these inorganic ions; barium and sulphate ions are ubiquitous and are present in most water, soil and sediment.
T criteria fulfilled?	Not applicable. Chronic toxicity data >1 mg/L, thus barium sulphate does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	December 2021

References

1. ECHA REACH, Barite sulfate, Retrieved 2021: https://echa.europa.eu/
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.industrialchemicals.gov.au/.
3. NCBI, 2021. National Center for Biotechnology Information. Available at: https://pubchem.ncbi.nlm.nih.gov/. Retrieved December 2021.
4. EHS Support, Barium Sulfate. Available at: https://www.santos.com/wp-content/uploads/2021/04/Barium-sulfate-March-2021.pdf. Retrieved December 2021.
5. ANZG, 2018. Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Australian and New Zealand Governments and Australian state and territory governments, Canberra ACT, Australia. Available at www.waterquality.gov.au/anz-guidelines.
6. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
7. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
8. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.

Toxicity Summary - Bitumen

Chemical and Physical Properties[1,2]	
CAS number	8052-42-4
Molecular formula	Unknown or variable composition, complex reaction products or biological materials (UVCB)
Molecular weight	UVCB
Solubility in water	No data available
Melting point	30 - 128°C at 101.3 - 101.325 kPa
Boiling point	320 - 500°C at 101.325 kPa
Vapour pressure	1 hPa @ 20 °C
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	No data available
Colour/Form	Black or dark brown solid or semi-solid at 20°C and 101.3 kPa
Overview	A very complex combination of high molecular weight organic compounds containing a relatively high proportion of hydrocarbons having carbon numbers predominantly greater than C25 with high carbon-to-hydrogen ratios. It also contains small amounts of various metals such as nickel, iron, or vanadium. It is obtained as the non-volatile residue from distillation of crude oil or by separation as the raffinate from a residual oil in a deasphalting or decarbonization process. Bitumen is also commonly known as asphalt.
Environmental Fate[1]	
Soil/Water/Air	Hydrocarbons are degraded (under aerobic conditions) via mono-oxygenases or di-oxygenases and are subsequently carboxylated and ultimately hydroxylated. In further assessing the type of metabolites formed, it has been demonstrated that for all the major classes of hydrocarbons, the major metabolites are in most cases less toxic, and always less bioaccumulative than the parent molecule.
Human Health Toxicity Summary[1,2]	
Chronic Repeated Dose Toxicity	Based on the data available, the chemicals in this group are not considered to cause serious damage to health from repeated dermal exposure. In a GLP-compliant study conducted similarly to OECD TG 410, residues, petroleum, vacuum (CAS No. 64741-56-6) was administered at dosages of 200, 1000, or 2000 mg/kg bw three times a week for four weeks. Clinical observations included slight oedema, flaking skin, wheezing and decreased food-intake (qualitative observation), resulting in reduced body weight gain in all dose groups when compared to controls. There were statistically significant reduced body weight gains in males in the high-dose group. There were no significant changes in clinical chemistry, haematology parameters or reproductive organs reported. A no observed adverse effect level (NOAEL) for local effects of 200 mg/kg bw/day was reported based on dermal irritation. A NOAEL for systemic effects of 1000 mg/kg bw/day was reported based on decreased body weight (which was considered to be secondary to the reduced food intake). Based on the data available, the chemicals in this group are not considered to cause serious damage to health from repeated inhalation exposure. The fume condensate from oxidized asphalt (CAS No. 64742-93-4) was tested in rats in a combined repeated dose and reproductive and developmental screening test conducted in accordance with OECD TG 474. Wistar rats were exposed (nose only) to concentrations of approximately 30, 100 or 300 mg/m^3 for 28 days. A no observed adverse effect concentration (NOAEC) was established as 100 mg/m^3 based on slight histopathological changes observed in the lungs observed at the highest dose.

	Asphalt fume condensate collected over a paving asphalt tank was tested in a repeated dose inhalation study conducted in accordance with OECD TG 413. Wistar rats were exposed (nose-only) to concentrations of approximately 5, 28 or 149 mg/m^3 for 90 days. The NOAEC was established as 28 mg/m^3 based on reduced body weights and histopathological changes in the nasal and paranasal cavities observed at the highest dose.
Carcinogenicity	Based on the available data, the chemicals in this group as whole materials are not considered carcinogenic, although dilution in organic solvents may produce some carcinogenic effects following prolonged dermal exposure. Exposure to asphalt emissions during certain occupations has been linked to increased risks of carcinogenicity.
Mutagenicity/ Genotoxicity	Based on the weight of evidence, the chemicals in this group (as whole materials) are not considered to be mutagenic. Asphalt fume condensates are mutagenic, with the level of mutagenic activity related to the temperature at which they are generated and levels of PACs.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	There are no reproductive or developmental toxicity studies available on asphalt or asphalt fumes. Based on the limited data available, and the low concentration of PACs generated in asphalt fumes, a classification for reproductive or developmental effects is not warranted. In a GLP-compliant two-generation reproduction toxicity study conducted in accordance with OECD TG 416, rats were exposed (oral gavage) to the analogue chemical, distillates (Fischer-Tropsch), heavy, C18-50- branched, cyclic and linear (CAS No. 848301-69-9) at dosages of 0, 50, 250 or 1000 mg/kg bw/day. The analogue chemical is mainly comprised of saturated oil components, which may be found in asphalts. There were histopathological lesions in the lungs (chronic interstitial/alveolar inflammation) of the F0- and F1-generations. There were corresponding macroscopic findings and/or increased lung weights, and effects in the kidneys (renal tubular hyaline droplets likely associated with alpha-2μ-globulin) of the F1 males only. The study authors stated that the lung lesions were most likely secondary to aspiration of the chemical and, therefore, not relevant for human risk assessment. The renal effects are specific to male rats. These are induced by hydrocarbons and have no relevance for humans. An equivocal, non-adverse slight decrease in F2 pup brain weights was reported. A NOAEL of 1000 mg/kg bw/day was determined for reproductive and systemic toxicity, based on no adverse effects on the male and female reproductive systems, non-reproductive tissues, and other parameters (such as body weight, feed consumption, and clinical observations).
Acute Toxicity	Oral: Based on the data available, the chemicals in this group have low acute toxicity based on results from animal tests following oral exposure to residues, petroleum, vacuum (CAS No. 64741-56-6). The median lethal dose (LD50) in rats is >5000 mg/kg bw. Observed sub-lethal effects included hypoactivity and diarrhoea. Dermal: Based on the data available, the chemicals in this group have low acute toxicity based on results from animal tests following dermal exposure to residues, petroleum, vacuum (CAS No. 64741-56-6). The LD50 value in rats is >2000 mg/kg bw. Inhalation: Based on the data available, the chemicals in this group have low acute toxicity following inhalation exposure. No mortality or significant signs of toxicity were noted in rats exposed to fumes generated from condensates collected from the headspace of a bitumen storage unit. Mean exposures were estimated to be 182 mg/m^3 for four hours. No mortality or toxic effects have been reported in several other studies in which rats were repeatedly exposed up to 300 mg/m^3.
Irritation	Based on the available data, the chemicals in this group may slightly irritate skin in animal studies, particularly following repeated exposure. Based on the available data, the chemicals in this group may be, at most, slightly irritating to the eye in animal studies.

	Exposure to asphalt vapours was reported to cause only minor, transient conjunctivitis in the eyes of rabbits.
Sensitisation	The negative results observed for residues, petroleum, vacuum (CAS No. 64741-56-6), in several skin sensitisation animal studies conducted in accordance with OECD TG 406 (Buehler test), support a conclusion that the chemicals in this group are not skin sensitisers.
Health Effects Summary	The critical health effects for risk characterisation relate to the use of the chemicals at elevated temperatures. Fumes from asphalts have been associated with carcinogenicity and mutagenicity in humans and animals. There is considered to be an increased risk for fumes containing higher levels of PACs. The levels of PACs are affected by the temperature of fume generation. Exposure to asphalt fumes could also cause irritant effects (skin, eye, nasal and throat) and respiratory effects. Severe burns to the skin have been reported in workers from hot asphalt (usually used at temperatures from 150 to 190°C).
Key Study/Critical Effect for Screening Criteria	The repeated dose toxicity in rats via dermal application was considered the most sensitive endpoint with a NOAEL of 200 mg/kg bw/day.
Ecological Toxicity[1]	
Aquatic Toxicity	Short term toxicity: LL50 (4 days): 1 g/L (fish) LL50 (48 h): 1 g/L (invertebrates) EL50 (72 h): 1 g/L (algae) Long term toxicity: LL50 (28 days): 1 g/L (fish)
Determination of PNEC aquatic	Based on the lowest endpoint for aquatic toxicity (1 g/L), an assessment factor of 100 has been applied, resulting in a PNECaquatic of 0.01 g/L.
Current Regulatory Controls[2,3,4]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	Asphalt as bitumen fumes, has an occupational exposure limit (OEL) of 5 mg/m^3 time weighted average (TWA).
International Occupational Exposure Standards	The following exposure standards are identified (Galleria Chemica) for asphalt: An OEL of 0.5–10 mg/m^3 TWA and 1.5–10 mg/m^3 short-term exposure limit (STEL) in different countries such as Canada, Chile, China, Germany, Indonesia, Ireland, Malaysia, Mexico, Norway, Singapore, South Africa, Spain, Switzerland, United Kingdom, and USA. The American Conference of Governmental Industrial Hygienists (ACGIH) recommends a threshold limited value (TLV) of 0.5 mg/m^3 (TWA) as benzene-soluble (or equivalent method) inhalable aerosol. 'This value is intended to minimize the potential for mucous membrane and ocular irritation'.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1]	
P/vP Criteria fulfilled?	No. Based on QSAR modelling, the substance is expected to be readily biodegradable.
B/vB criteria fulfilled?	Not applicable (substance is a UVCB). Calculated BCF for constituents of this substance range between 0.4 and 13300 L/kg.
T criteria fulfilled?	No. Chronic toxicity data >1 g/L in invertebrates, thus the substance does not meet the screening criteria for toxicity.

Overall conclusion	Not PBT. These chemicals are a UVCB. They do not contain PBT constituents included in the Substances of Very High Concern (SVHC) candidate list at concentrations above 0.1%.
Revised	December 2021

References

1. ECHA REACH, Asphalt, Retrieved 2021: https://echa.europa.eu/
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Asphalt: Human health tier II assessment: Retrieved 2021: https://www.industrialchemicals.gov.au/.
3. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
4. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.

Toxicity Summary - Calcium oxide

Chemical and Physical Properties[1,2,3,4]	
CAS number	1305-78-8
Molecular formula	CaO
Molecular weight	56.08
Solubility in water	1.19 g/L at 20 °C
Melting point	2572°C
Boiling point	2850°C
Vapour pressure	Negligible at 25 °C
Henrys law constant	No data available
Explosive potential	Non-explosive
Flammability potential	Non-flammable
Colour/Form	Greyish yellow, odourless, hygroscopic solid
Overview	Calcium oxide (CaO), is an inorganic compound commonly known as quicklime or burnt lime, is a widely used chemical compound. The chemical is used as a component of a hydraulic fracturing fluid formulation for coal seam gas extraction. A Tier 1 Human Health and Environmental Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health and the environment.
Environmental Fate[5]	
Soil/Water/Air	Calcium oxide reacts immediately upon exposure to water, forming calcium hydroxide, which itself reacts with carbon dioxide to form calcium carbonate. The final reaction products of both limestone and calcium oxide in the environment are therefore essentially the same, although calcium oxide typically has lower concentrations of magnesium and other inorganic chemicals than limestone and produces a higher initial concentration of hydroxide ions. Calcium and carbonate ions occur naturally in all environmental compartments and are important nutrients for various organisms. Calcium is mobile in soil and, if released to the environment, should be expected to experience significant partitioning to the water compartment. However, calcium ions may also form insoluble precipitates with anions present in the environment, such as carbonate ions, and settle out of the aqueous phase.
Human Health Toxicity Summary[2]	
Chronic Repeated Dose Toxicity	Several repeat dose studies using analogues of calcium oxide (calcium hydroxide, calcium carbonate, calcium gluconate) investigating the effect of calcium ions on various metabolic functions in experimental animals are available in the literature. However, all these studies were considered inappropriate for derivation of a No Observed Adverse Effect Level (NOAEL) by the study authors, as they did not follow any international guidelines (ECHA REACH).
Carcinogenicity	No data available. Using a read across study, calcium oxide is considered not likely to be carcinogenic.
Mutagenicity/ Genotoxicity	Calcium oxide is not mutagenic.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	In two developmental toxicity studies conducted according to methods equivalent or similar to the OECD TG 414 (Prenatal Developmental Toxicity Study), calcium oxide was administered by gavage to pregnant female Wistar rats up to 680 mg/kg bw/day and CD-1 mice up to 440 mg/kg bw/day during gestation days 6 to 15 (10 consecutive doses). There were no clear discernible effects on implantation, maternal survival or foetal survival in any species at any of the doses. The number of abnormalities seen in either soft or skeletal tissues of the test groups did not

	differ significantly from those occurring spontaneously in the controls. No NOAEL could be established for maternal toxicity or foetal developmental effects. Based on the available data, calcium oxide is not considered to be a developmental toxicant.
Acute Toxicity	A study on acute oral toxicity of calcium oxide in female rats was conducted by a scientifically accepted method. Different doses of calcium oxide suspended in polyethylene glycol (0.2 g/mL) were administered to rats by gavage. No deaths were observed at 2000 mg/kg bw, indicating that the oral median lethal dose (LD50) for rats is >2000 mg/kg bw. No adverse effects were observed following treatment. No macroscopic findings were observed at necropsy. Calcium oxide has low oral acute toxicity with an oral LD50 of >2000 mg/kg bw. Acute dermal toxicity studies with calcium oxide are not available. An acute dermal toxicity study was conducted in rabbits using moistened calcium hydroxide (Ca(OH)2). As calcium oxide (CaO) is converted to Ca(OH)2 in the presence of moisture, the test results for Ca(OH)2 are also applicable for CaO. No animal deaths were observed at 2500 mg/kg bw Ca(OH)2, indicating that the dermal LD50 for male/female rabbits is >2500 mg/kg bw. No adverse effects were observed following the treatment. Based on the results with Ca(OH)2, calcium oxide is considered to have low acute dermal toxicity.
Irritation	Results from two skin irritation studies with calcium hydroxide (hydrated calcium oxide) indicated that calcium hydroxide causes skin irritation. The US Occupational Health Guideline for calcium oxide states 'calcium oxide causes irritation of the eyes, nose, throat and skin. Severe burns may result from contact with this chemical'. Calcium oxide is also considered to be a severe eye irritant.
Sensitisation	No data available.
Health Effects Summary	Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.
Key Study/Critical Effect for Screening Criteria	Calcium oxide has low acute oral and dermal toxicity, is a skin and respiratory irritant and a severe eye irritant. Calcium oxide is not genotoxic or carcinogenic and does not have any developmental effects in animals. Given the constituent ions of calcium oxide which are subject to tight homeostatic control in the body, repeated exposure to calcium oxide is regarded to have no significant systemic effects. In an epidemiological study, no significant adverse effects were observed in lime-kiln workers exposed to 1.2 mg/m^3 lime dust. This atmospheric concentration was taken as an overall NOAEC for calcium oxide. This NOAEC will be carried forward for human health risk assessment. The critical health effects of calcium oxide are skin and respiratory irritation and severe eye irritation.
Ecological Toxicity [2,5]	
Aquatic Toxicity	Oncorhynchus mykiss 96-hour LC50: 50.6 mg/L Daphnia magna 48-hour EC50: 49.1 mg/L Pseudokirchneriella subcapitata 72 hour EC10: 79.22 mg/L A 42-day Oncorhynchus mykiss test showed that enhanced Ca2+ diets (60 mg Ca2+) had no effects on survival. Mean fish weights remained constant across all treatments. A 14-day Crangon septemspinosa test showed an EC10 of 32 mg/L.
Determination of PNEC aquatic	A Tier 1 assessment of the environmental risks from the use of substances in the Limestone and its derivatives group is not required.
Current Regulatory Controls[2]	
Australian Hazard Classification	Calcium oxide is listed as hazardous in the Hazardous Substances Information System (HSIS). No risk phrases have been assigned to this chemical.
Australian Occupational Exposure Standards	The chemical has an exposure standard of 2 mg/m^3, Time Weighted Average (TWA)

International Occupational Exposure Standards	The following exposure standards are identified in Galleria Chemica (2013): Occupational Exposure limit (TWA) of 2 mg/m^3 [Canada, Denmark, Korea, UK, US (NIOSH)] Permissible Exposure Limits (PEL) of 5 mg/m^3 [US (OSHA 1978)].
Australian Food Standards	Calcium oxide is allotted the following International Numbering System of food additives number: INS 529 (Food Standards Australia New Zealand 2013).
Australian Drinking Water Guidelines	Calcium oxide is used in water treatment to correct pH and adjust alkalinity, for coagulation optimisation, corrosion control and water softening. Typical calcium oxide concentrations used in drinking water treatment depend on the quality of the water to be treated and the purpose of treatment (e.g. water softening, pH adjustment or alkalinity increase) and can vary from 5 to 500 mg/L.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	Not applicable (inorganic salt, ionic species ubiquitous in environment).
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to these inorganic ions; sodium and hydroxide ions are ubiquitous and are present in most water, soil and sediment.
T criteria fulfilled?	No. Chronic and acute toxicity data >1 mg/L, calcium oxide does not meet the screening criteria for toxicity.
Overall conclusion	It is not currently possible to categorise the environmental hazards of metals and other inorganic chemicals according to standard persistence, bioaccumulation and toxicity (PBT) hazard criteria. These criteria were developed for organic chemicals and do not take into account the unique properties of inorganic substances and their behaviour in the environment.
Revised	December 2021

References

1. ECHA REACH, Calcium oxide, Retrieved 2021: https://echa.europa.eu/
2. Department of the Environment and Energy 2017, National assessment of chemicals associated with coal seam gas extraction in Australia, prepared by the National Industrial Chemicals Notification and Assessment Scheme
3. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2021: https://www.industrialchemicals.gov.au/.
4. NCBI, 2021. National Center for Biotechnology Information. Available at: https://pubchem.ncbi.nlm.nih.gov/. Retrieved December 2021.
5. EHS Support, Calcium oxide, calcium hydroxide. Available at: https://www.santos.com/wp-content/uploads/2021/05/Calcium-oxide-and-calcium-hydroxide-March-2021.pdf. Retrieved December 2021.
6. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
7. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.

Toxicity Summary - Copper (II) oxide

Chemical and Physical Properties[1,2,3,4]	
CAS number	1317-38-0
Molecular formula	CuO
Molecular weight	79.55
Solubility in water	Insoluble
Melting point	1,326 °C
Boiling point	No data available.
Vapour pressure	No data available.
Henrys law constant	No data available.
Explosive potential	No data available.
Flammability potential	No data available.
Colour/Form	Black to brownish-black amorphous or crystalline powder or granules
Overview	CuO is an inorganic compound. It is a product of copper mining and is used for the production of other copper-containing products. A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health.
Environmental Fate[1]	
Soil/Water/Air	Among the copper species released/transformed, Cu (II) is thus the most environmental relevant species. It is further recognised that Cu (II) ions - commonly named free cupric ions- are the most active copper species and that total Cu or Cu(II) concentrations are usually not directly related to ecological effects since exposure of biota may be limited by processes that render Cu unavailable for uptake. Assessing the species of Cu (II) therefore has ecotoxicological relevance. After being released into the environment, the Cu(II) ions typically bind to inorganic and organic ligands contained within water, soil, and sediments. In water Cu(II) binds to dissolved organic matter (e. g. humic or fulvic acids). The Cu(II) ion forms stable complexes with -NH2, -SH, and, to a lesser extent, -OH groups in these organic acids. Cu(II) will also bind with varying affinities to inorganic and organic components in sediments and soils. For example, Cu(II) binds strongly to hydrous manganese and iron oxides in clay and to humic acids, but much less strongly to aluminosilicates in sand. In all environmental compartments (water, sediment, soil), the binding affinities of Cu(II) with inorganic and organic matter is dependent on pH, the oxidation-reduction potential in the local environment, and the presence of competing metal ions and inorganic anions.
Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	The chronic toxicity of CuO is based on studies on copper sulphate. The pivotal repeat dose study was a 90-day study by the oral route with copper sulphate pentahydrate. In rats and mice, ingestion of copper sulphate pentahydrate produced forestomach lesions that could be due to the irritant effects of the compound. The no-observed-adverse-effect level (NOAEL) for this effect was 16.7 mg Cu/kg bw/day in rats and 97 and 126 mg Cu/kg bw/day in male and female mice respectively. In rats, inflammation of the liver was observed. The NOAEL for liver and kidney damage were 16.7 mg Cu/kg bw/day in rats.
Carcinogenicity	The carcinogenicity of copper has not been adequately studied. An increase in cancer risk has been found among copper smelters; however, the increased risk has been attributed to concomitant exposure to arsenic. Increased lung and stomach cancer risks have also been found in copper miners. However, a high occurrence of smoking and exposure to radioactivity, silica, iron, and arsenic obscure the association of copper exposure with carcinogenesis. Animal studies have not found increased cancer risks in orally exposed rats or mice.

Mutagenicity/ Genotoxicity	The available genotoxicity studies support the indication that copper compounds have no carcinogenic potential. The studies include Ames assays in Salmonella typhimurium on copper II sulphate pentahydrate; a micronucleus study on copper II sulphate pentahydrate and an unscheduled DNA synthesis ex vivo study in rat liver on copper II sulphate. The Ames tests indicated that copper sulphate had no mutagenic activity. No evidence of an increase in the incidence of micronuclei was detected in the mouse micronucleus study when mice were orally administered two doses of 447 mg/kg copper sulphate, 24 h apart. There was also no evidence of unscheduled DNA synthesis in the rat liver. These studies are consistent and show a lack of in vitro mutagenic activity or in vivo clastogenic potential associated with soluble copper compounds. The results of these studies do not highlight a concern regarding the genotoxic potential of copper compounds.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	The two-generation study in the rat indicate that that under the conditions of this study, the NOAEL for reproductive toxicity was 1500 ppm, the highest concentration tested. The NOAEL for P1 and F1 rats and F1 and F2 offspring during lactation was 1000 ppm, based on reduced spleen weight in P1 adult females, and F1 and F2 male and female weanlings at 1500 ppm however the transient reduced spleen weights are not considered a reproductive endpoint as it did not affect growth or fertility.
Acute Toxicity	In a study to assess the acute oral toxicity of copper oxide following a single oral administration by gavage, there were no mortalities or signs of systemic toxicity among any of the animals treated with copper oxide at the test concentration of 2000 mg/kg bw. An LD50 of >2500 mg/kg bw can be estimated using the flow chart in annex 2d of OECD Guidelines for the Testing of Chemicals No. 423 “Acute Oral Toxicity – Acute Toxic Class Method. The acute median lethal dose (LD50) of copper oxide in the Sprague-Dawley CD (Crl: CD (SD) IGS BR) strain of rats study to assess the acute dermal toxicity of copper oxide was found to be >2000 mg/kg bw.
Irritation	Not irritating to the skin and eyes.
Sensitisation	Not sensitising.
Health Effects Summary	Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.
Key Study/Critical Effect for Screening Criteria	The NOAEL of 16.7 mg Cu/kg bw/day for liver and kidney damage in rats is used in the risk characterisation.
Ecological Toxicity [1,3]	
Aquatic Toxicity	Based on copper ecotoxicity data: Fish: 2.6 µg/L (Ptylocheilus oregonensis, from 7-day LC50) 131 µg/L (Pimephales promelas, 7-day LC50) Crustaceans: 1.7 µg/L (D. pulex and G. pulex, NOEC, reproduction & mortality) 12.1 µg/L (Hyalella azteca, from 10 to 14-day LC50). Insects: 2.2 µg/L (Tanytarsus dissimilis, from 10-day LC50) 11 µg/L (Chironomus tentans, 10 to 20-day LC50). Molluscs: 1.64 µg/L (Flumicola virens, from 14-day LC50) 56.2 (Corbicula manilensis, from 7 to 42-day LC50).
Determination of PNEC aquatic	The PNECaquatic for freshwater is determined to be 7.8 µg/L based on the conclusion that the copper concentrations in food items and daily dietary copper dose in fish are unlikely to cause negative effects at this threshold.
Current Regulatory Controls[4]	
Australian Hazard Classification	H410 (Very toxic to aquatic life with long-lasting effects)

Australian Occupational Exposure Standards	No data availanle
International Occupational Exposure Standards	TWA = 1 mg/m^3 (dust & mists) TWA = 0.2 mg/m^3 (fume)
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	Based on health considerations, the concentration of copper in drinking water should not exceed 2 mg/L. Based on aesthetic considerations, the concentration of copper in drinking water should not exceed 1 mg/L.
Aquatic Toxicity Guidelines	A freshwater high reliability trigger value for copper of 1.4 µg/L was derived using the statistical distribution method with 95% protection. A marine high reliability trigger value for copper of 1.3 µg/L was derived using the statistical distribution method with 95% protection.
PBT Assessment	
P/vP Criteria fulfilled?	Not applicable (ionic species ubiquitous in environment).
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to these inorganic ions; copper ions are ubiquitous and are present in most water, soil and sediment.
T criteria fulfilled?	Not applicable. Copper is an essential nutrient for all living organisms.
Overall conclusion	It is not currently possible to categorise the environmental hazards of metals and other inorganic chemicals according to standard persistence, bioaccumulation and toxicity (PBT) hazard criteria. These criteria were developed for organic chemicals and do not take into account the unique properties of inorganic substances and their behaviour in the environment.
Revised	December 2021

References

1. ECHA REACH, Copper (II) oxide, Retrieved 2021: https://echa.europa.eu/
2. NCBI, 2021. National Center for Biotechnology Information. Available at: https://pubchem.ncbi.nlm.nih.gov/. Retrieved December 2021.
3. ANZG, 2018. Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Australian and New Zealand Governments and Australian state and territory governments, Canberra ACT, Australia. Available at www.waterquality.gov.au/anz-guidelines.
4. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.industrialchemicals.gov.au/.
5. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
6. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
7. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.

Toxicity Summary - Copper

Chemical and Physical Properties[1,2,3,4]	
CAS number	7440-50-8
Molecular formula	Cu
Molecular weight	63.546
Solubility in water	Insoluble
Melting point	1,057 – 1,059 °C
Boiling point	No data
Vapour pressure	1 (1,628 °C)
Henrys law constant	No data
Explosive potential	No data
Flammability potential	No data
Colour/Form	Reddish, solid
Overview	Copper is a reddish metal that occurs naturally in rock, soil, water, sediment, and at low levels in air. Copper's unique chemical and physical properties include high thermal conductivity, high electrical conductivity, malleability, low corrosion, alloying ability, and pleasing appearance. Properties of metallic copper such as electrical conductivity and fabricability vary markedly with purity. A Tier 1 Human Health Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health.
Environmental Fate[3]	
Soil/Water/Air	Copper is released to the atmosphere in the form of particulate matter or adsorbed to particulate matter. Atmospheric copper is removed by gravitational settling, dry deposition, and wet deposition (rain and snow). Much of the copper discharged into waterways is in particulate matter and settles out. In the water column and in sediments, copper adsorbs to organic matter, hydrous iron and manganese oxides, and clay. Copper binds primarily to organic matter in estuarine sediment unless the sediment is low in organic matter content. Most copper deposited on soil from the atmosphere, agricultural use, and solid waste and sludge disposal will be adsorbed with greater concentrations of copper measured in the upper 5 – 10 centimetres of soil in comparison to lower soil depths, except in sandy soils where the lability of bound copper is greater. Copper's movement in soil is determined by a host of physical and chemical interactions of copper with the soil components. In general, copper will adsorb to organic matter, carbonate minerals, clay minerals, or hydrous iron and manganese oxides. Sandy soils with low pH have the greatest potential for leaching. Copper binds strongly to soils with high organic content.
Human Health Toxicity Summary[3,4]	
Chronic Repeated Dose Toxicity	Liver damage (necrosis, fibrosis, abnormal biomarkers of liver damage) have been reported in individuals ingesting lethal doses of copper sulphate. There is some evidence from animal studies to suggest that exposure to airborne copper or high levels of copper in drinking water can damage the immune system. Impaired cell-mediated and humoral-mediated immune function have been observed in mice. Studies in rats, mice, and mink suggest that exposure to high levels of copper in the diet can result in decreased embryo and foetal growth.
Carcinogenicity	The carcinogenicity of copper has not been adequately studied. An increase in cancer risk has been found among copper smelters; however, the increased risk has been attributed to concomitant exposure to arsenic. Increased lung and

	stomach cancer risks have also been found in copper miners. However, a high occurrence of smoking and exposure to radioactivity, silica, iron, and arsenic obscure the association of copper exposure with carcinogenesis. Animal studies have not found increased cancer risks in orally exposed rats or mice. The IARC has classified the pesticide, copper 8-hydroxyquinoline, in Group 3, unclassifiable as to carcinogenicity in humans and EPA has classified copper in Group D, not classifiable as to human carcinogenicity.
Mutagenicity/ Genotoxicity	No data on the genotoxicity of copper in humans were located. The available genotoxicity data suggest that copper is a clastogenic agent. Several studies have also shown that exposure to copper can result in DNA damage.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No studies were located regarding developmental effects in humans and animals following inhalation exposure to copper.
Acute Toxicity	One of the most commonly reported adverse health effect of copper is gastrointestinal distress. Nausea, vomiting, and/or abdominal pain have been reported, usually occurring shortly after drinking a copper sulphate solution, beverages that were stored in a copper or untinned brass container, or first draw water (water that sat in the pipe overnight).
Irritation	Copper is a respiratory tract irritant and causes coughing, sneezing, runny nose, pulmonary fibrosis, and increased vascularity of the nasal mucosa.
Sensitisation	Not sensitising.
Health Effects Summary	Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.
Key Study/Critical Effect for Screening Criteria	The chronic oral reference dose (RfD) of 4 x 10^{-2} mg/kg/day is based drinking water standard of 1.3 mg/L, assuming a water consumption rate of 2 L/day and a body weight of 70 kg.
Ecological Toxicity[1,5]	
Aquatic Toxicity	Based on copper ecotoxicity data: Fish: 2.6 µg/L (Ptylocheilus oregonensis, from 7-day LC50) 131 µg/L (Pimephales promelas, 7-day LC50) Crustaceans: 1.7 µg/L (D. pulex and G. pulex, NOEC, reproduction & mortality) 12.1 µg/L (Hyalella azteca, from 10 to 14-day LC50). Insects: 2.2 µg/L (Tanytarsus dissimilis, from 10-day LC50) 11 µg/L (Chironomus tentans, 10 to 20-day LC50). Molluscs: 1.64 µg/L (Flumicola virens, from 14-day LC50) 56.2 (Corbicula manilensis, from 7 to 42-day LC50).
Determination of PNEC aquatic	The PNECaquatic for freshwater is determined to be 7.8 µg/L based on the conclusion that the copper concentrations in food items and daily dietary copper dose in fish are unlikely to cause negative effects at this threshold.
Current Regulatory Controls[5,6,7,8,9]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	TWA = 1 mg/m^3 (dust & mists) TWA = 0.2 mg/m^3 (fume)
International Occupational Exposure Standards	TWA = 1 mg/m^3 (dust & mists) TWA = 0.2 mg/m^3 (fume)

Australian Food Standards	Tolerable limit = 0.2 mg/kg bw/day
Australian Drinking Water Guidelines	Based on health considerations, the concentration of copper in drinking water should not exceed 2 mg/L. Based on aesthetic considerations, the concentration of copper in drinking water should not exceed 1 mg/L.
Aquatic Toxicity Guidelines	A freshwater high reliability trigger value for copper of 1.4 µg/L was derived using the statistical distribution method with 95% protection. A marine high reliability trigger value for copper of 1.3 µg/L was derived using the statistical distribution method with 95% protection.
PBT Assessment	
P/vP Criteria fulfilled?	Not applicable (copper is an essential element and is ubiquitous in environment).
B/vB criteria fulfilled?	No. As an essential element, copper is commonly regulated by the organism and do not bioaccumulate or biomagnify.
T criteria fulfilled?	Not applicable. Copper is an essential nutrient for all living organisms.
Overall conclusion	It is not currently possible to categorise the environmental hazards of metals and other inorganic chemicals according to standard persistence, bioaccumulation and toxicity (PBT) hazard criteria. These criteria were developed for organic chemicals and do not take into account the unique properties of inorganic substances and their behaviour in the environment.
Revised	December 2021

References

1. ECHA REACH, Copper, Retrieved 2021: https://echa.europa.eu/
2. USEPA, 2021. Regional Risk Levels. November 2021. https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables. Retrieved December 2021.
3. ATSDR, 2004. Toxicological Profile for Copper. Agency for Toxic Substances and Disease Registry. September 2004.
4. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2019: https://www.industrialchemicals.gov.au/.
5. ANZG, 2018. Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Australian and New Zealand Governments and Australian state and territory governments, Canberra ACT, Australia. Available at www.waterquality.gov.au/anz-guidelines.
6. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
7. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
8. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.
9. Food Standards Australia New Zealand (FSANZ) 20th Australian Total Diet Survey. Retrieved 2021: https://www.foodstandards.gov.au/publications/pages/20thaustraliantotaldietsurveyjanuary2003/20thaustraliantotaldietsurveyfullreport/partb20thatds/partbmetals.aspx.

Toxicity Summary - Distillates (petroleum), hydrotreated light naphthenic

Chemical and Physical Properties[1,2]	
CAS number	64742-53-6
Molecular formula	Unknown or variable composition, complex reaction products or biological materials (UVCB)
Molecular weight	UVCB
Solubility in water	No data available
Melting point	0°C at 101.325 kPa
Boiling point	207 - 750°C at 101.325 kPa
Vapour pressure	10 Pa at 20 °C
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	Non-flammable
Colour/Form	Liquid, petroleum product
Overview	These chemicals are refined distillate base oils derived from crude oil. It undergoes a series of extractive or transforming processes that improve the base stocks' performance characteristics and remove or reduce undesirable components such as polyaromatic compounds (PACs). The chemicals are complex mixtures of straight and branched-chain paraffinic, naphthenic and aromatic hydrocarbons having carbon numbers in the C15–C50 range. The chemical composition of these chemicals depends on both the original crude oil and on the refining process. The toxicity profile of these chemicals is dictated by the levels of PACs. Mineral oils containing <3 % w/w dimethylsulfoxide (DMSO) extractables (as measured by the IP346 assay) are considered highly or severely refined. Only white oils are considered highly refined by definition.
Environmental Fate[1]	
Soil/Water/Air	Hydrocarbons are degraded (under aerobic conditions) via mono-oxygenases or di-oxygenases and are subsequently carboxylated and ultimately hydroxylated. In further assessing the type of metabolites formed, it has been demonstrated that for all the major classes of hydrocarbons, the major metabolites are in most cases less toxic, and always less bioaccumulative than the parent molecule.
Human Health Toxicity Summary[1,2,3]	
Chronic Repeated Dose Toxicity	In separate 4-week single-dose studies, New Zealand White rabbits were dermally exposed to 1000 mg/kg bw/day of the chemicals identified by CAS Nos. 64741-88-4, 64742-56-9 and 64742-65-0. There were no significant signs of systemic toxicity. Signs of irritation were observed both macroscopically (erythema and oedema) and microscopically (inflammation, hyperplasia and hyperkeratosis). In a 13-week study, Sprague Dawley (SD) rats were dermally exposed to 800 or 2000 mg/kg bw/day, for five days a week. The no observed adverse effect level (NOAEL) for systemic effects was reported as 800 mg/kg bw/day based on increased liver weights and histopathological changes observed in high-dose females. Signs of irritation were observed at both dose levels, both macroscopically (erythema and oedema) and microscopically (inflammation, hyperplasia and hyperkeratosis). As application sites were not covered, exposure may have been less than intended. Increased liver weights were also observed in a 3-week dermal study with CAS No. 64742-70-7. In this study, SD rats were dermally exposed to 1720 mg/kg bw/day (five days/week). Effects were observed in both males and females. As application sites were not covered, exposure may have been less than intended.

Carcinogenicity	These chemicals are classified as hazardous, Category 2 carcinogens with the risk phrase 'May cause cancer'. Skin tumours have been observed in mice following exposure to several unrefined or mildly refined base oils. In a dermal carcinogenicity study in mice, the chemical with CAS No. 64742-53-6 increased the incidence of skin tumours. Petroleum substances containing <3 % w/w DMSO extractables (as measured by the IP346 assay), or which are negative in the modified Ames test (mutagenicity index <1) are not carcinogenic to skin.
Mutagenicity/ Genotoxicity	The genetic toxicity of the chemicals is expected to be related to the level of refinement and associated removal of PACs. The results for the chemical CAS No. 64742-53-6 were positive in an in vitro mouse lymphoma mutation assay but the results for CAS No. 64742-52-5 were negative in a similar study.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No data are available for the chemicals. Certain petroleum streams have been shown to be developmentally toxic from dermal exposure. Effects include increased incidence of resorptions and decrease in foetal body weight. The developmental toxicity of the chemicals is expected to be correlated with the level of refinement of the chemicals.
Acute Toxicity	These chemicals are considered to be of low acute toxicity following oral and dermal exposure. The chemical identified by CAS Nos. 64742-53-6, has a reported median lethal dose (LD50) in rabbits of greater than 2000 mg/kg bw. Signs of skin irritation (slight to severe for erythema and oedema and desquamation) were observed in some studies. Based on data available, the chemicals in this group are expected to have low to moderate toxicity following inhalation exposure. Based on the reported median lethal concentration (LC50) value (2.18 mg/L/4-h) for an insufficiently refined oil, classification is considered appropriate for the chemicals in this group. However, this classification need not apply for highly or severely refined oils, containing <3 % w/w DMSO extractables (as measured by the IP346 assay). In an acute inhalation toxicity study, rats were exposed to an aerosol of the chemical identified by CAS No. 64742-53-6 at concentrations of 1, 1.5, 2.5, 3.5 and 5 mg/L for four hours. All the animals in the two high dose groups, and three males and three females in the 2.5 mg/L dose group died. The LC50 was 2.18 mg/L/4-h. Observed sublethal effects included decreased activity, rapid breathing and congestion, and inflammation of the lungs. The test chemical was reported to have a DMSO extractable content of >3 % as measured by the IP346 assay. Two further acute inhalation studies were available for the chemical identified by CAS No. 64742-53-6. In a single dose study in rats, 80 % mortality was observed at 5.7 mg/L/4-h. The DMSO extractable content was not reported. In another rat study, the LC50 was reported to be 10.5 mg/L/4-h for males and 9.5 mg/L/4-h for females. The cause of death appeared to be associated with lung congestion and/or oedema. The test chemical was reported to be highly or severely refined oils containing <3 % w/w DMSO extractables. Acute inhalation data were available for several highly or severely refined oils containing <3 % w/w DMSO extractables. In general, no mortalities were observed.
Irritation	Animal skin irritation data were available for several highly or severely refined oils containing <3 % w/w DMSO extractables. In general, slight skin irritation effects were considered to be not sufficient for classification. These minimally irritant effects are consistent with irritant responses observed in humans. Very slight irritant effects were observed in four separate repeated insult patch tests in human volunteers with highly or severely refined oils, containing <3 % w/w DMSO extractables. CAS No. 64742-53-6 produced erythema and oedema (mean scores greater than two over 72 hours for both endpoints) following application to intact rabbit skin. Effects were fully reversible within 14 days. The test chemical was reported to have a DMSO extractable content (as measured by the IP346 assay) of >3 %. CAS No. 64742-53-6 also reported to be a slight eye irritant in animal studies. Based on the data available, the chemicals are considered to be, at most, slightly irritating to eyes.
Sensitisation	The limited data available do not indicate a potential for skin sensitisation. CAS No. 64742-53-6 was negative in Buehler-type studies in guinea pigs.

Health Effects Summary	The critical health effects for the chemicals in this group are dependent upon the level of refinement. The critical health effects for less refined base oils (DMSO extractable content > 3 % in the IP346 assay) include carcinogenicity and developmental toxicity, acute effects (acute toxicity by the inhalation route of exposure) and local effects (skin irritation). Exposure to mists may also cause respiratory symptoms such as cough and phlegm.
Key Study/Critical Effect for Screening Criteria	The repeated dose toxicity via dermal application was considered the most sensitive endpoint with the NOAEL for systemic effects reported at 800 mg/kg bw/day.
Ecological Toxicity[1]	
Aquatic Toxicity	Short-term toxicity to fish: In a key static 96-hour short-term fathead minnow (Pimephales promelas) limit test (OECD 203; KS=1), 10 animals/loading were exposed to the WAF of Basestock Solvent Neutral 600 (MRD-94 -981) at a nominal concentration of 100 mg/L. The LL50 was > 100 mg/L and the NOEL was ≥100 mg/L. Long-term toxicity to fish: For other lubricant base oils, read across has been applied for the long-term toxicity in fish endpoint, using the results of long-term toxicity testing on invertebrates (Daphnia magna). Toxic effects of hydrocarbons are primarily caused by narcosis and occur in a narrow range of molar concentrations across aquatic taxa; hence, read across between species is justified. Results of computer modelling to estimate aquatic chronic toxicity of other lubricant base oils in a 28-day freshwater fish study show no chronic toxicity to freshwater fish at or below its maximum attainable water solubility. This supports the applied interspecies read across. Short-term toxicity to aquatic invertebrates: In a key static 48-hour short-term Daphnia magna toxicity test (OECD 202; KS = 2), 10 animals/loading were exposed to the WAF of an other lubricant base oil, MVI(N) 40 base oil (CAS # 64742-53-6 or 64741-97-5), at nominal concentrations of 0, 10, 100, 1000, and 10,000 mg/L. The EL50 was >10,000 mg/L based on mobility and the NOEL was ≥ 1000 mg/L. Long-term toxicity to aquatic invertebrates: In a key semi-static 21-day long-term Daphnia magna toxicity test (OECD 211; KS = 2), 10 animals/loading were exposed to the WAF of other lubricant base oil LVIN 38 (CAS #64742-53-6) at nominal concentrations of 1, 10, 100 and 1000 mg/L. The NOEL was 10 mg/L based on reproduction. The loss of all daphnids in the 100 mg/L WAF was attributed to a non-treatment related effect, the cause of which was unknown. Further testing would be required to clarify the consequences of exposure to a 100 mg/L WAF of the base oil. Toxicity to aquatic algae: In a key static 72-hour algal (Pseudokirchneriella subcapitata) limit test (OECD 201; KS = 2), the freshwater alga was exposed to the WAF of another lubricant base oil (N100DW; CAS # 72623-87-1), at a nominal concentration of 100 mg/L. The NOEL was ≥ 100 mg/L based upon average specific growth rate and cell yield. Toxicity to microorganisms: In a key static 4-day Photobacterium phosphoreum luminescence inhibition study (KS=2) using other lubricant base oils as control substances, no significant luminescence inhibition was observed for Spindle oil (BP 320-400 ºC) and Neutral oil Ro NIII (BP 400-450 ºC), as well as for the n-paraffin dodecane. The actual NOEL for Spindle oil was > 1.93 mg/L and the actual NOEL for Neutral oil Ro NIII was > 2.17 mg/L.
Determination of PNEC aquatic	Based on the lowest chronic endpoint for Daphnia (10 mg/L), an assessment factor of 100 has been applied, resulting in a PNECaquatic of 0.1 mg/L.
Current Regulatory Controls[2,3,4,5,6]	
Australian Hazard Classification	Acute toxicity – category 4 Carcinogenicity – category 1B

	Skin irritation – category 2 Reproductive toxicity – category 2
Australian Occupational Exposure Standards	No specific exposure standards are available for specific chemicals. The exposure standard for oil mist, refined mineral is 5 mg/m^3 time weighted average (TWA). The applicability of this exposure standard for the chemicals will depend on the level of refinement.
International Occupational Exposure Standards	A number of countries have exposure standards for oil mist mineral including an exposure limit of 0.2–5 mg/m^3 (TWA) in different countries such as the United Arab Emirates, Japan, Austria, United States of America (USA) and Canada; and a short-term exposure limit (STEL) of 3–10 mg/m^3 in countries such as Sweden, Egypt, the USA, Canada, Singapore and Poland. The American Conference of Governmental Industrial Hygienists (ACGIH) recommends a threshold limit value (TLV) of 5 mg/m^3 (TWA) for pure, highly and severely refined mineral oils. The ACGIH does not recommend application of this TLV to poorly- and mildly-refined mineral oils. No TLV is assigned based on insufficient data, although an A2 suspected human carcinogen classification is assigned.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	Oils and greases (including petrochemicals) for freshwater production: <300^3 µg/L (ANZECC, 2000)
PBT Assessment[1]	
P/vP Criteria fulfilled?	Yes. In a supporting biodegradability study, Solvent Neutral 600 Base Oil (MRD-94 -981), was determined to be inherently biodegradable but not readily biodegradable with a mean degradation of 31.13% by day 28. In an additional supporting biodegradability study, an other lubricant base oil (GOHC 1468) was determined not to be readily biodegradable when it attained 2 to 4 % degradation within 28 days.
B/vB criteria fulfilled?	Calculated BCF for constituents of this substance range between 0.4 and 71100 L/kg.
T criteria fulfilled?	No. Chronic and acute toxicity data >1 mg/L, these chemicals do not meet the screening criteria for toxicity.
Overall conclusion	Not PBT. These chemicals are a UVCB. They do not contain PBT constituents included in the Substances of Very High Concern (SVHC) candidate list at concentrations above 0.1%.
Revised	December 2021

References

1. ECHA REACH, Distillates (petroleum), hydrotreated heavy naphthenic, Retrieved 2021: https://echa.europa.eu/
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Selected refined base oils: Human health tier II assessment: Retrieved 2021: https://www.industrialchemicals.gov.au/.
3. ANZECC (2000) Australian and New Zealand Guidelines for Fresh and Marine Water Quality for protection for aquatic ecosystems.
4. Food Standards Australia New Zealand (FSANZ) 20th Australian Total Diet Survey. Retrieved 2021: https://www.foodstandards.gov.au/publications/pages/20thaustraliantotaldietsurveyjanuary2003/20thaustraliantotaldietsurveyfullreport/partb20thatds/partbmetals.aspx.
5. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
6. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.

Toxicity Summary - Distillates (petroleum), straight-run middle

Chemical and Physical Properties[1,2]	
CAS number	64741-44-2
Molecular formula	Unknown or variable composition, complex reaction products or biological materials (UVCB)
Molecular weight	UVCB
Solubility in water	No data available
Melting point	-21 - 6°C at 101.325 kPa
Boiling point	150 - 399°C at 101.3 kPa
Vapour pressure	4 hPa at 40°C
Henrys law constant	No data available
Explosive potential	Non-explosive
Flammability potential	Non-flammable
Colour/Form	Liquid, petroleum product
Overview	Whilst other compositional characteristics could influence toxicity, the toxicity profile of this chemical is expected to be dictated by the levels of polycyclic aromatic compounds (PACs), particularly those composed of 3, 4, 5, 6 and/or 7 fused aromatic rings. Due to the hydrotreating process, the chemicals in this group are expected to contain low levels of these PACs.
Environmental Fate[1]	
Soil/Water/Air	Hydrocarbons are degraded (under aerobic conditions) via mono-oxygenases or di-oxygenases and are subsequently carboxylated and ultimately hydroxylated. In further assessing the type of metabolites formed, it has been demonstrated that for all the major classes of hydrocarbons, the major metabolites are in most cases less toxic, and always less bioaccumulative than the parent molecule.
Human Health Toxicity Summary[1,2]	
Chronic Repeated Dose Toxicity	A key 'read across' 90-day dermal study in rats was identified in which vacuum tower overheads was applied to the shaved skin of rats, 5 days a weeks for 90-days. The NOAEL was 30 mg/kg/day, based on findings in liver, thymus and blood. A 28 day repeated dose toxicity studies in rabbits was identified for dermal exposure, plus a supporting 28 day dermal study in rats. There was one key read-across 90-day repeated dose toxicity study (OECD 413) for inhalation. For the read-across 90-day inhalation study, a NOAEC of 0.88 mg/L for local effects on the lung (increased relative wet weight in the absence of histopathological change) was established in rats expose to aerosol. A NOAEC of greater than or equal to 1.71 mg/L is established for systemic effects, based on no significant findings at this level. For the 28-day dermal study, a LOAEL of 200 mg/kg/day was established based on local irritation. No NOEL was determined for local irritation. The NOAEL for systemic effects in rabbits following repeated dermal exposure was greater than or equal to 2000 mg/kg/day.
Carcinogenicity	Distillates (petroleum), straight-run middle has been reported to produce squamous cell carcinomas and fibrosarcomas (20–25 % incidence) in long-term dermal carcinogenicity studies in mice when applied undiluted. However, data from other straight run gas oils that have been applied in diluted form indicate that the tumorigenic activity of straight-run middle distillates, with low levels of PACs, is likely to be a consequence of a non-genotoxic process associated with frequent cell damage and repair. In these studies, when the irritant effects were reduced, there were no significant increases in tumours relative to controls.

Mutagenicity/ Genotoxicity	In the key in vitro modified bacteria Ames study (similar to OECD 471), there was no evidence of mutagenic activity. This result was supported by other studies with straight run gas oils and related materials, the majority of which were negative. A key in vivo chromosome aberration assay (OECD 475) was identified, in which straight run middle distillate was not found to be mutagenic in male rat bone marrow cells. An additional chromosome aberration assay also showed negative results for mutagenicity (OECD 475).
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Based on the expected negligible amounts of PACs with 3–7 rings, the chemicals are not expected to show specific reproductive or developmental toxicity.
Acute Toxicity	The substance is considered to have low acute toxicity following oral and dermal exposure and moderate acute toxicity following inhalation exposure. The reported median lethal dose (LD50) for oral exposure in rats for distillates (petroleum), straight-run middle is >5000 mg/kg bw. Reported signs of toxicity included hypoactivity, diarrhoea and hair loss. In general, gas oils produced from secondary processing are considered to have low acute toxicity following oral exposure. The reported LD50 for dermal exposure in rats for distillates (petroleum), straight-run middle is >2000 mg/kg bw. Whilst no systemic effects were reported slight to moderate dermal irritation was observed. In an acute inhalation study conducted similarly to the Organisation for Economic Co-operation and Development Test Guideline (OECD TG) 403 with distillates (petroleum), straight-run middle, the median lethal concentration (LC50) was determined to be 1.78 mg/L. Reported signs of toxicity included reduced body weight gain, gross necropsy findings and acute histopathological changes in the lung.
Irritation	In general, gas oils are considered to be slightly to moderately irritating to the skin. In a skin irritation study in New Zealand White rabbits, distillates (petroleum), straight-run middle was applied to intact and abraded clipped skin on the back and flank of six rabbits, under occlusion for 24 hours. For intact skin, the mean erythema and oedema scores were 1.80 and 1.58, respectively. Effects were reversible within 14 days. Given that the chemical was tested under occlusive patch conditions and for longer periods of time than specified in the OECD TG 404 conditions, irritant responses might be more pronounced than would be expected in a standard study. Distillates (petroleum), straight-run middle were reported to be non-irritating to the eyes (unrinsed and rinsed) when tested equivalently or similarly to OECD TG 405. The mean conjunctival, iridial and corneal scores at 24-, 48- and 72-hours post-exposure were 0.
Sensitisation	Gas oils produced by secondary processing and distillates (petroleum), straight-run middle were not skin sensitisers in the guinea pig Buehler test.
Health Effects Summary	The critical health effect for risk characterisation is acute toxicity from inhalation exposure. The chemicals also have the potential to cause chemical pneumonitis if aspirated. Due to the hydrotreating process, the chemicals in this group are expected to contain low levels of PACs composed of 3–7 fused aromatic rings and, as such, are not considered to be genotoxic carcinogens. The chemicals are considered unlikely to cause skin tumours in the absence of prolonged skin irritation.
Key Study/Critical Effect for Screening Criteria	The 90-day repeated dose toxicity in rats via dermal application was considered the most sensitive endpoint with a NOAEL of 30 mg/kg bw/day.
Ecological Toxicity[1]	
Aquatic Toxicity	The 96h LL50 for freshwater fish is 21 mg/L. The estimated freshwater fish NOEL (No Observed Effect Level) value is 0.068 mg/L based on mortality. The 48 h EL50 for Daphnia was 68 mg/L. The estimated freshwater invertebrate NOEL (No Observed Effect Level) value is 0.167 mg/L based on immobility and numbers of live young produced per adult by Day 21.

	The 72 h ErL50 for algae was 22 mg/L.
Determination of PNEC aquatic	Based on the lowest endpoint for aquatic toxicity (0.167 mg/L), an assessment factor of 100 has been applied, resulting in a PNECaquatic of 0.001 mg/L.
Current Regulatory Controls[4,5]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	Oils and greases (including petrochemicals) for freshwater production: <300[3] µg/L (ANZECC, 2000)
PBT Assessment[1]	
P/vP Criteria fulfilled?	No. Degradation was achieved at varying levels in the available tests. Two tests indicate that the substance is readily biodegradable (ignoring the 10-day window). As the 10-day window is not relevant to UVCB substances, therefore the substance is considered readily biodegradable
B/vB criteria fulfilled?	Gas oils components have log Kow values in the range 3.9 to greater than 6.
T criteria fulfilled?	No. Aquatic toxicity data >1 mg/L, thus the substance does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT. These chemicals are a UVCB. They do not contain PBT constituents included in the Substances of Very High Concern (SVHC) candidate list at concentrations above 0.1%.
Revised	December 2021

References

1. ECHA REACH, Distillates (petroleum), straight-run middle, Retrieved 2021: https://echa.europa.eu/
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Hydrocracked gas oils: Human health tier II assessment: Retrieved 2021: https://www.industrialchemicals.gov.au/.
3. ANZECC (2000) Australian and New Zealand Guidelines for Fresh and Marine Water Quality for protection for aquatic ecosystems.
4. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
5. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.

Toxicity Summary - Distillates, petroleum, hydrotreated heavy naphthenic

Chemical and Physical Properties[1,2]	
CAS number	64742-52-5
Molecular formula	Unknown or variable composition, complex reaction products or biological materials (UVCB)
Molecular weight	UVCB
Solubility in water	No data available
Melting point	0°C at 101.325 kPa
Boiling point	207 - 750°C at 101.325 kPa
Vapour pressure	10 Pa at 20 °C
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	Non-flammable
Colour/Form	Liquid, petroleum product
Overview	These chemicals are refined distillate base oils derived from crude oil. It undergoes a series of extractive or transforming processes that improve the base stocks' performance characteristics and remove or reduce undesirable components such as polyaromatic compounds (PACs). The chemicals are complex mixtures of straight and branched-chain paraffinic, naphthenic and aromatic hydrocarbons having carbon numbers in the C15–C50 range. The chemical composition of these chemicals depends on both the original crude oil and on the refining process. The toxicity profile of these chemicals is dictated by the levels of PACs. Mineral oils containing <3 % w/w dimethylsulfoxide (DMSO) extractables (as measured by the IP346 assay) are considered highly or severely refined. Only white oils are considered highly refined by definition.
Environmental Fate[1]	
Soil/Water/Air	Hydrocarbons are degraded (under aerobic conditions) via mono-oxygenases or di-oxygenases and are subsequently carboxylated and ultimately hydroxylated. In further assessing the type of metabolites formed, it has been demonstrated that for all the major classes of hydrocarbons, the major metabolites are in most cases less toxic, and always less bioaccumulative than the parent molecule.
Human Health Toxicity Summary[1,2,3]	
Chronic Repeated Dose Toxicity	In separate 4-week single-dose studies, New Zealand White rabbits were dermally exposed to 1000 mg/kg bw/day of the chemicals identified by CAS Nos. 64741-88-4, 64742-56-9 and 64742-65-0. There were no significant signs of systemic toxicity. Signs of irritation were observed both macroscopically (erythema and oedema) and microscopically (inflammation, hyperplasia and hyperkeratosis). In a 13-week study, Sprague Dawley (SD) rats were dermally exposed to 800 or 2000 mg/kg bw/day, for five days a week. The no observed adverse effect level (NOAEL) for systemic effects was reported as 800 mg/kg bw/day based on increased liver weights and histopathological changes observed in high-dose females. Signs of irritation were observed at both dose levels, both macroscopically (erythema and oedema) and microscopically (inflammation, hyperplasia and hyperkeratosis). As application sites were not covered, exposure may have been less than intended. Increased liver weights were also observed in a 3-week dermal study with CAS No. 64742-70-7. In this study, SD rats were dermally exposed to 1720 mg/kg bw/day (five days/week). Effects were observed in both males and females. As application sites were not covered, exposure may have been less than intended.

Carcinogenicity	These chemicals are classified as hazardous, Category 2 carcinogens with the risk phrase 'May cause cancer'. Skin tumours have been observed in mice following exposure to several unrefined or mildly refined base oils. In a dermal carcinogenicity study in mice, the chemical with CAS No. 64742-53-6 increased the incidence of skin tumours. Petroleum substances containing <3 % w/w DMSO extractables (as measured by the IP346 assay), or which are negative in the modified Ames test (mutagenicity index <1) are not carcinogenic to skin.
Mutagenicity/ Genotoxicity	The genetic toxicity of the chemicals is expected to be related to the level of refinement and associated removal of PACs. The results for the chemical CAS No. 64742-53-6 were positive in an in vitro mouse lymphoma mutation assay but the results for CAS No. 64742-52-5 were negative in a similar study.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No data are available for the chemicals. Certain petroleum streams have been shown to be developmentally toxic from dermal exposure. Effects include increased incidence of resorptions and decrease in foetal body weight. The developmental toxicity of the chemicals is expected to be correlated with the level of refinement of the chemicals.
Acute Toxicity	These chemicals are considered to be of low acute toxicity following oral and dermal exposure. The chemical identified by CAS Nos. 64742-53-6, has a reported median lethal dose (LD50) in rabbits of greater than 2000 mg/kg bw. Signs of skin irritation (slight to severe for erythema and oedema and desquamation) were observed in some studies. Based on data available, the chemicals in this group are expected to have low to moderate toxicity following inhalation exposure. Based on the reported median lethal concentration (LC50) value (2.18 mg/L/4-h) for an insufficiently refined oil, classification is considered appropriate for the chemicals in this group. However, this classification need not apply for highly or severely refined oils, containing <3 % w/w DMSO extractables (as measured by the IP346 assay). In an acute inhalation toxicity study, rats were exposed to an aerosol of the chemical identified by CAS No. 64742-53-6 at concentrations of 1, 1.5, 2.5, 3.5 and 5 mg/L for four hours. All the animals in the two high dose groups, and three males and three females in the 2.5 mg/L dose group died. The LC50 was 2.18 mg/L/4-h. Observed sublethal effects included decreased activity, rapid breathing and congestion, and inflammation of the lungs. The test chemical was reported to have a DMSO extractable content of >3 % as measured by the IP346 assay. Two further acute inhalation studies were available for the chemical identified by CAS No. 64742-53-6. In a single dose study in rats, 80 % mortality was observed at 5.7 mg/L/4-h. The DMSO extractable content was not reported. In another rat study, the LC50 was reported to be 10.5 mg/L/4-h for males and 9.5 mg/L/4-h for females. The cause of death appeared to be associated with lung congestion and/or oedema. The test chemical was reported to be highly or severely refined oils containing <3 % w/w DMSO extractables. Acute inhalation data were available for several highly or severely refined oils containing <3 % w/w DMSO extractables. In general, no mortalities were observed.
Irritation	Animal skin irritation data were available for several highly or severely refined oils containing <3 % w/w DMSO extractables. In general, slight skin irritation effects were considered to be not sufficient for classification. These minimally irritant effects are consistent with irritant responses observed in humans. Very slight irritant effects were observed in four separate repeated insult patch tests in human volunteers with highly or severely refined oils, containing <3 % w/w DMSO extractables. CAS No. 64742-53-6 produced erythema and oedema (mean scores greater than two over 72 hours for both endpoints) following application to intact rabbit skin. Effects were fully reversible within 14 days. The test chemical was reported to have a DMSO extractable content (as measured by the IP346 assay) of >3 %. CAS No. 64742-53-6 also reported to be a slight eye irritant in animal studies. Based on the data available, the chemicals are considered to be, at most, slightly irritating to eyes.
Sensitisation	The limited data available do not indicate a potential for skin sensitisation. CAS No. 64742-53-6 was negative in Buehler-type studies in guinea pigs.

Health Effects Summary	The critical health effects for the chemicals in this group are dependent upon the level of refinement. The critical health effects for less refined base oils (DMSO extractable content > 3 % in the IP346 assay) include carcinogenicity and developmental toxicity, acute effects (acute toxicity by the inhalation route of exposure) and local effects (skin irritation). Exposure to mists may also cause respiratory symptoms such as cough and phlegm.
Key Study/Critical Effect for Screening Criteria	The repeated dose toxicity via dermal application was considered the most sensitive endpoint with the NOAEL for systemic effects reported at 800 mg/kg bw/day.
Ecological Toxicity[1]	
Aquatic Toxicity	Short-term toxicity to fish: In a key static 96-hour short-term fathead minnow (Pimephales promelas) limit test (OECD 203; KS=1), 10 animals/loading were exposed to the WAF of Basestock Solvent Neutral 600 (MRD-94 -981) at a nominal concentration of 100 mg/L. The LL50 was > 100 mg/L and the NOEL was ≥100 mg/L. Long-term toxicity to fish: For other lubricant base oils, read across has been applied for the long-term toxicity in fish endpoint, using the results of long-term toxicity testing on invertebrates (Daphnia magna). Toxic effects of hydrocarbons are primarily caused by narcosis and occur in a narrow range of molar concentrations across aquatic taxa; hence, read across between species is justified. Results of computer modelling to estimate aquatic chronic toxicity of other lubricant base oils in a 28-day freshwater fish study show no chronic toxicity to freshwater fish at or below its maximum attainable water solubility. This supports the applied interspecies read across. Short-term toxicity to aquatic invertebrates: In a key static 48-hour short-term Daphnia magna toxicity test (OECD 202; KS = 2), 10 animals/loading were exposed to the WAF of an other lubricant base oil, MVI(N) 40 base oil (CAS # 64742-53-6 or 64741-97-5), at nominal concentrations of 0, 10, 100, 1000, and 10,000 mg/L. The EL50 was >10,000 mg/L based on mobility and the NOEL was ≥ 1000 mg/L. Long-term toxicity to aquatic invertebrates: In a key semi-static 21-day long-term Daphnia magna toxicity test (OECD 211; KS = 2), 10 animals/loading were exposed to the WAF of other lubricant base oil LVIN 38 (CAS #64742-53-6) at nominal concentrations of 1, 10, 100 and 1000 mg/L. The NOEL was 10 mg/L based on reproduction. The loss of all daphnids in the 100 mg/L WAF was attributed to a non-treatment related effect, the cause of which was unknown. Further testing would be required to clarify the consequences of exposure to a 100 mg/L WAF of the base oil. Toxicity to aquatic algae: In a key static 72-hour algal (Pseudokirchneriella subcapitata) limit test (OECD 201; KS = 2), the freshwater alga was exposed to the WAF of another lubricant base oil (N100DW; CAS # 72623-87-1), at a nominal concentration of 100 mg/L. The NOEL was ≥ 100 mg/L based upon average specific growth rate and cell yield. Toxicity to microorganisms: In a key static 4-day Photobacterium phosphoreum luminescence inhibition study (KS=2) using other lubricant base oils as control substances, no significant luminescence inhibition was observed for Spindle oil (BP 320-400 ºC) and Neutral oil Ro NIII (BP 400-450 ºC), as well as for the n-paraffin dodecane. The actual NOEL for Spindle oil was > 1.93 mg/L and the actual NOEL for Neutral oil Ro NIII was > 2.17 mg/L.
Determination of PNEC aquatic	Based on the lowest chronic endpoint for Daphnia (10 mg/L), an assessment factor of 100 has been applied, resulting in a PNECaquatic of 0.1 mg/L.
Current Regulatory Controls[2,3,4,5,6]	
Australian Hazard Classification	Acute toxicity – category 4 Carcinogenicity – category 1B

	Skin irritation – category 2 Reproductive toxicity – category 2
Australian Occupational Exposure Standards	No specific exposure standards are available for specific chemicals. The exposure standard for oil mist, refined mineral is 5 mg/m^3 time weighted average (TWA). The applicability of this exposure standard for the chemicals will depend on the level of refinement.
International Occupational Exposure Standards	A number of countries have exposure standards for oil mist mineral including an exposure limit of 0.2–5 mg/m^3 (TWA) in different countries such as the United Arab Emirates, Japan, Austria, United States of America (USA) and Canada; and a short-term exposure limit (STEL) of 3–10 mg/m^3 in countries such as Sweden, Egypt, the USA, Canada, Singapore and Poland. The American Conference of Governmental Industrial Hygienists (ACGIH) recommends a threshold limit value (TLV) of 5 mg/m^3 (TWA) for pure, highly and severely refined mineral oils. The ACGIH does not recommend application of this TLV to poorly- and mildly-refined mineral oils. No TLV is assigned based on insufficient data, although an A2 suspected human carcinogen classification is assigned.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	Oils and greases (including petrochemicals) for freshwater production: <300[3] µg/L (ANZECC, 2000)
PBT Assessment[1]	
P/vP Criteria fulfilled?	Yes. In a supporting biodegradability study, Solvent Neutral 600 Base Oil (MRD-94 -981), was determined to be inherently biodegradable but not readily biodegradable with a mean degradation of 31.13% by day 28. In an additional supporting biodegradability study, an other lubricant base oil (GOHC 1468) was determined not to be readily biodegradable when it attained 2 to 4 % degradation within 28 days.
B/vB criteria fulfilled?	Calculated BCF for constituents of this substance range between 0.4 and 71100 L/kg.
T criteria fulfilled?	No. Chronic and acute toxicity data >1 mg/L, these chemicals do not meet the screening criteria for toxicity.
Overall conclusion	Not PBT. These chemicals are a UVCB. They do not contain PBT constituents included in the Substances of Very High Concern (SVHC) candidate list at concentrations above 0.1%.
Revised	December 2021

References

1. ECHA REACH, Distillates (petroleum), hydrotreated heavy naphthenic, Retrieved 2021: https://echa.europa.eu/
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Selected refined base oils: Human health tier II assessment: Retrieved 2021: https://www.industrialchemicals.gov.au/.
3. ANZECC (2000) Australian and New Zealand Guidelines for Fresh and Marine Water Quality for protection for aquatic ecosystems.
4. Food Standards Australia New Zealand (FSANZ) 20th Australian Total Diet Survey. Retrieved 2021: https://www.foodstandards.gov.au/publications/pages/20thaustraliantotaldietsurveyjanuary2003/20thaustraliantotaldietsurveyfullreport/partb20thatds/partbmetals.aspx.
5. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
6. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.

Toxicity Summary - Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine

Chemical and Physical Properties[1,2]	
CAS number	68990-47-6
Molecular formula	Unknown or variable composition, complex reaction products or biological materials (UVCB)
Molecular weight	UVCB
Solubility in water	2.17 mg/L
Melting point	No data available
Boiling point	No data available
Vapour pressure	No data available
Henrys law constant	No data available
Explosive potential	Non-explosive
Flammability potential	No data available
Colour/Form	Solid with a dark colour at room temperature
Overview	This substance is used by consumers, in articles, by professional workers (widespread uses), in formulation or re-packing, at industrial sites and in manufacturing.
Environmental Fate[1]	
Soil/Water/Air	The substance is not expected to be readily biodegradable. On the basis of the very low water solubility and its chemical nature, the substance is expected to have a high ability to absorb to soil. Due to its complex composition, methods for the experimental measurement of octanol -water partition coefficient (Kow) of Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine are technically not applicable. On the basis of the high solubility in octanol (> 30 mg/L) compared to the solubility in water (2.17 ppm), and the chemical nature, Kow value for the substance is expected to be high. Estimated Log Kow value for the smallest molecule arising from the chemical synthesis is 11.
Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	Test item-related histopathological changes were restricted to the lung. Multifocal subacute bronchopneumonia, characterized by peribronchial foci of prominent fibrosis, with re-epithelialization, infiltration with mononuclear cells, histiocytes and occasional multinucleated cells, was observed in a small proportion of treated males and females of all dose groups, without dose relationship. In addition, a mild amount of intrahistiocytic black material was seen in the lung of each one male treated at 300 or 1000 mg/kg/day. As a conclusion, based on the pathological evaluation, a No-Observed-Effect-Level (NOEL) could not be determined in this study.
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	The test item Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine is considered to be non-clastogenic.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Based on the data generated from this combined repeated dose toxicity and reproduction/ developmental toxicity screening test with Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine, no effects were reported on reproductive/ developmental toxicity parameters measured in this study. There

	were also no effects reported on general toxicity parameters except for the reported macroscopic/microscopic lung changes. Due to the lack of clear dose-response relationship (solely restricted to histopathological lung changes) observed in this study, the suitable NOAEL (No observed adverse effect level) general toxicity could not be determined. However, for reproductive/ developmental toxicity, the NOAEL could be set at 1000 mg/kg bw.
Acute Toxicity	The test substance was assessed for its acute oral toxicity potential when administered to albino rats. The acute oral LD50, as indicated by the data, is greater than 2020 mg/kg in males and females.
Irritation	Not irritating to skin and eye.
Sensitisation	Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine caused reactions identified as sensitisation at the tested concentration.
Health Effects Summary	The substance is expected to have low acute toxicity and is not an irritant. The substance may cause skin sensitisation.
Key Study/Critical Effect for Screening Criteria	The reproductive/ developmental toxicity in rats was considered the most sensitive endpoint with a NOAEL of 1000 mg/kg bw/day.
Ecological Toxicity[1,2,3]	
Aquatic Toxicity	Short term toxicity: LC50 (4 days): 100 mg/L (fish) NOEC (4 days): 100 mg/L (fish) LOEC (4 days): 100 mg/L (fish) IC50 (48 h): 100 mg/L (invertebrates) NOEC (48 h): 100 mg/L (invertebrates) LOEC (48 h): 100 mg/L (invertebrates) EC50 (72 h): 100 mg/L (algae)
Determination of PNEC aquatic	Data from short-term tests with three trophic levels are available. An assessment factor of 1000 is applied to the lowest NOEC of 100 mg/L. A PNECaqua of 0.1 mg/L was derived.
Current Regulatory Controls[4]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1]	
P/vP Criteria fulfilled?	Yes. Not inherently biodegradable.
B/vB criteria fulfilled?	Yes. Bioaccumulation of this substance may occur in aquatic organisms based on the estimated Log Kow of 11 (Log Kow > 4.2).
T criteria fulfilled?	No. Acute toxicity data >1 mg/L in fish, invertebrates and algae, thus the substance does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT

Revised	December 2021

References

1. ECHA REACH, Fatty acids, tall-oil, reaction products with diethylenetriamine, maleic anhydride, tetraethylenepentamine and triethylenetetramine, Retrieved 2021: https://echa.europa.eu/.
2. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
3. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
4. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.

Toxicity Summary - Graphite

Chemical and Physical Properties[1,2]	
CAS number	7782-42-5
Molecular formula	C
Molecular weight	12.011
Solubility in water	Insoluble
Melting point	600°C at 101.3 kPa
Boiling point	No data available
Vapour pressure	No data available
Henrys law constant	No data available
Explosive potential	No data available
Flammability potential	No data available
Colour/Form	Odourless black solid powder
Overview	Graphite is a naturally-occurring form of crystalline carbon. It is a native element mineral found in metamorphic and igneous rocks. It is extremely soft, cleaves with very light pressure, and has a very low specific gravity. In contrast, it is extremely resistant to heat and nearly inert in contact with almost any other material. A Tier 1 Human Health and Environmental Assessment for this chemical has been conducted by NICNAS which concluded that it was low concern to human health and the environment.
Environmental Fate[1,2]	
Soil/Water/Air	Graphite is a crystal modification of the chemical element carbon, an inorganic substance with negligible water solubility. Therefore, neither hydrolysis, biodegradation, nor adsorption is of relevance for the fate of the molecule. Transport and distribution is of no relevance by the negligible solubility of the substance and as element "C" in its overall availability in different organic and inorganic forms in the environment.
Human Health Toxicity Summary[1,7]	
Chronic Repeated Dose Toxicity	Oral: - One study according to OECD 422 (subacute) was conducted - Concentrations tested were up to the limit dose specified in OECD 422 = 1000 mg/kg bw/day (nominal) - No effects due to Graphite exposure were found, neither on systemic toxicity nor on reproductive/developmental toxicity Inhalation: - Two studies according to OECD 412 (subacute) were conducted - Synthetic Graphite (SG; w/o Quartz) and Expanded Graphite (EG; with Quartz) were compared separately - Testing of SG resulted in a NOAEL of 12 mg/m^3, whereas testing of EG resulted in a NOAEL of 8 mg/m^3 - Both qualities showed effects that were to be expected for a poorly soluble dust with low toxicity, with partly recovery after 28 days - Exposure was generally well tolerated - Despite the respiratory system no other organs were affected at all - No sign of systemic toxicity was observed
Carcinogenicity	No data available.
Mutagenicity/ Genotoxicity	No evidence for any genotoxic potential of Graphite.

Reproductive Toxicity / Developmental Toxicity/Teratogenicity	OECD 422 (combined repeated dose toxicity study with the reproductive/developmental toxicity screening test) - Oral administration via food (incl. analytical verification) - Graphite was tested up to the limit dose given in OECD 422 (nominal 1000 mg/kg bw/day) - Result: No signs of systemic toxicity were observed, no signs of any effects on development, reproduction, or fertility - NOAEL based on nominal food intake = 1000 mg/kg bw/day
Acute Toxicity	Oral (OECD 423, conducted as limit test): - None of the animals showed any clinical signs of reaction to the treatment. - LD50 > 2000 mg/kg bw Inhalation (OECD 403, conducted as limit test): - Upon cessation of exposure via inhalation none of the rats exposed to Graphite showed any signs of toxicity. - Only usual signs of discomfort after exposure to particles were observed. Grooming activity started immediately after the end of exposure. - LC50 > 2000 mg/m^3
Irritation	Not irritating to skin and eyes.
Sensitisation	Not sensitising
Health Effects Summary	A harmful concentration of airborne particles can be reached quickly when dispersed, especially if powdered. Repeated or prolonged inhalation of dusts may cause effects on the lungs. This may result in graphite pneumoconiosis. Poses no unreasonable risk to human health based on Tier I assessment under the NICNAS IMAP assessment framework.
Key Study/Critical Effect for Screening Criteria	Nominal doses up to 1000 mg/kg bw/day were well tolerated and did not show any sign for systemic toxicity. Since the study was conducted as a combined repeated dose toxicity study with the reproductive/developmental toxicity screening test, several NOAELs were obtained, all representing the nominal dose of 1000 mg/kg bw/day. However, the actual substance intake varied from about 813 mg/kg bw/day up to 1159 mg/kg bw/day. The derived no effect levels were calculated using the NOAEL of 813 mg/kg bw/day.
Ecological Toxicity[1]	
Aquatic Toxicity	The short-term fish toxicity was determined to be > 100 mg/L for the LC50 and > 100 mg/L for the NOEC. The short-term toxicity for aquatic invertebrates (daphnids) was determined to be > 100 mg/L for the EC50 and > 100 mg/L for the NOEC. Based in the result obtained by a valid GLP-OECD 201 study in algae with graphite as test item, no toxic effects were found up to the highest tested concentration of 100 mg/L.
Determination of PNEC aquatic	A Tier 1 assessment of the environmental risks of graphite is not required.
Current Regulatory Controls[4,5,6,7]	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	Time Weighted Average (TWA): 3 mg/m^3
International Occupational Exposure Standards	Threshold limit value, TLV: (respirable fraction): 2 mg/m^3, as TWA. Maximum workplace concentration, MAK: (inhalable fraction): 4 mg/m^3. MAK: (respirable fraction): 0.3 mg/m^3; peak limitation category: II(8); pregnancy risk group: C; carcinogen category: 4
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.

Aquatic Toxicity Guidelines	No data available.
PBT Assessment[1]	
P/vP Criteria fulfilled?	Not applicable (inorganic mineral, ionic species ubiquitous in environment)
B/vB criteria fulfilled?	Not applicable. Bioaccumulation is not applicable to these inorganic, insoluble minerals.
T criteria fulfilled?	No. Acute data >1 mg/L. Thus, it is not expected to meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	December 2021

References

1. ECHA REACH, Graphite, Retrieved 2021: https://echa.europa.eu/
2. EHS Support, Graphite. Available at: https://www.santos.com/wp-content/uploads/2021/04/Graphite-March-2021.pdf. Retrieved December 2021.
3. National Industrial Chemicals Notification and Assessment Scheme (NICNAS). IMAP, Human Health Tier 1 Assessment. Retrieved 2021: https://www.industrialchemicals.gov.au/.
4. ANZG, 2018. Australian and New Zealand Guidelines for Fresh and Marine Water Quality. Australian and New Zealand Governments and Australian state and territory governments, Canberra ACT, Australia. Available at www.waterquality.gov.au/anz-guidelines.
5. NHMRC, 2018. Australian Drinking Water Guidelines 6, 2011, Version 3.5 Updated August 2018. National Water Quality Management.
6. HCIS, Hazardous Chemical Information System, Safe Work Australia, Retrieved December 2021: http://hcis.safeworkaustralia.gov.au/HazardousChemical.
7. ILO, International Labour Organisation, International Chemical Safety Cards (ICSCs), Retrieved December 2021: http://www.ilo.org/dyn/icsc/showcard.listcards3?p_lang=en.

Appendix H

Toxicological Profiles for Chemical Tracers

AECOM Imagine it. Delivered.

Toxicity Summary - Water Flow Assurance Tracer (WFT)

Chemical and Physical Properties[1,2,3,4]	
CAS number	One chemical (proprietary)
Molecular formula	Proprietary
Molecular weight	534.36
Solubility in water	167.05 g/L at 20 °C and pH 7
Melting point	347.1 °C
Boiling point	909.54 °C at 101.325 kPa
Vapour pressure	7.43 X 10-22 mm Hg at 25°C (calculated)
Henrys law constant	10-15 atm-m^3/mol (estimated)
Explosive potential	Non-explosive (100%)
Flammability potential	Non-flammable (100%)
Colour/Form	Bright, odourless, orange-yellow powder
Overview	This chemical is used as a food, drug, and cosmetic colorant. It is used to colour confectionary, bakery goods, animal feeds, aqueous drug solutions, toothpastes, bath salts, hair rinses, and printing inks for use in and on foods, drugs, and cosmetics and on food, drug, and cosmetic packaging materials. This chemical is an azo dye. Azo compounds are formed from arenediazonium ions reacting with highly reactive aromatic compounds, in what is called a diazo coupling reaction. Azo compounds are generally deeply coloured because the azo linkage brings the two aromatic rings into conjugation (Solomon, 1996).
Environmental Fate[2]	
Soil/Water/Air	This chemical's production as a dye for wool, silks and as a colorant in food, drugs and cosmetics may result in its release to the environment through various waste streams. If released to air, this chemical will exist solely in the particulate phase in the atmosphere since it is a salt and will be non-volatile. Particulate-phase this chemical will be removed from the atmosphere by wet or dry deposition. This chemical may be susceptible to direct photolysis by sunlight; after exposure to sunlight, This chemical in distilled water exhibited a first order rate constant of $2.31X10^{-3}$ per day, corresponding to a half-life of 300 days. If released to soil, this chemical is expected to be mobile since this compound is expected to exist almost entirely 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. Volatilization from moist soil surfaces is not expected to be an important fate process because the compound exists as an anion and anions do not volatilize. If released into water, this chemical is not expected to adsorb to suspended solids and sediment based upon this compound's ionic nature in the environment. This chemical passed through pilot scale treatment activated sludge processes relatively unchanged, indicating that biodegradation is not expected to be an important environmental fate process. This chemical will exist almost entirely in the anion form at pH values of 5 to 9 and therefore volatilization from water surfaces is not expected to be an important fate process. Measured BCF values of <0.29 and <3.0 in carp suggests bioconcentration in aquatic organisms is low. Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyse under environmental conditions.

Human Health Toxicity Summary [1,2,3,4]	
Chronic Repeated Dose Toxicity	Two separate but concurrent studies in rats given 0%, 0.1%, 1% or 2% in the diet or 0% or 5% in the diet for between 113 and 125 weeks showed decreases in body weight in females at 1% in the diet and in males (12.2% decrease) and females (16.9% decrease) at 5% in the diet, but there were no effects at 2% in the diet. The FAO/WHO Expert Committee on Food Additives concluded that 2% in the diet, equal to 984 mg/kg bw per day, was the NOAEL for this study. During a 2-year study in Fischer 344 rats given This chemical in the drinking water at a concentration of 0%, 1% or 2%, statistically significant increases in mesothelioma in the abdominal cavity in males and endometrial stromal polyps in females in the 1% concentration groups were reported. The incidences of these tumours were not dose dependent, and the authors noted that the incidences were within the historical control range for these tumours in this rat strain.
Carcinogenicity	A 104-week carcinogenicity study in mice given 0%, 0.5%, 1.5% or 5% This chemical in the diet showed no effects other than reductions in body weight at various time points in both sexes at 5% in the diet and slight, but statistically significant, increases in feed consumption in males at 5% in the diet. Although the authors considered the NOAEL to be the highest dose tested, the FAO/WHO Expert Committee on Food Additives concluded that 1.5% in the diet, equal to 2173 mg/kg bw per day, was the NOAEL for this study, on the basis of a body weight reduction concurrent with an increase in feed consumption at the higher dose in males.
Mutagenicity/ Genotoxicity	The FAO/WHO Expert Committee on Food Additives concluded that the overall weight of evidence indicates that this chemical is not genotoxic.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	Reproductive and developmental parameters were assessed in the rat chronic toxicity studies that included an in utero exposure phase. No significant effects on reproduction or body weights of the offspring were observed. The FAO/WHO Expert Committee on Food Additives concluded that 5% in the diet, equal to 2641 mg/kg bw per day, the highest dose tested, was the NOAEL for reproductive end-points in this study. No reproductive effects were observed in two developmental neurotoxicity studies. Also, no effects on reproductive parameters were observed in several other developmental neurotoxicity studies in rats using a mixture of colours, including This chemical, as the test substance. Two developmental toxicity studies were available in rats, one with dietary administration and one with drinking-water administration of This chemical during gestation days 0–19; these showed no adverse effects at doses up to 1000 mg/kg bw per day.
Acute Toxicity	In reports submitted to the World Health Organization, the acute oral LD50 in mice was reported to be 12,750 mg/kg bw [National Institute of Hygienic Sciences of Japan, 1964]. In rats, the LD50 by intraperitoneal injection was reported to be 2,000 mg/kg bw and the LD50 by intravenous injection was reported to be 1,000 mg/kg bw [Deutsche Forschungsgemeinschaft, 1957].
Irritation	No irritating effects were observed both for skin and for eye.
Sensitisation	The results of the available tests about the evaluation of dermal effects on human showed no sensitizing effects.
Health Effects Summary	A number of case reports have been published showing intolerance or hypersensitivity reactions to This chemical. Although some of these reactions have been shown to be quite severe, their prevalence appears to be very low (0.12% in the general population).
Key Study/Critical Effect for Screening Criteria	An average daily intake (ADI) of 0-10 mg/kg bw per day was assigned by JECFA in 2016.

Ecological Toxicity [1]	
Aquatic Toxicity	Acute short-term administration on fish: LC50 fish (96 h) > 120 mg/L Acute short-term administration on invertebrates: Both of the acute toxicity to Daphnia magna studies does not show any toxic effects. EC50(48h) > 125 mg/L Acute short-term administration on aquatic plants: Both of the acute toxicity to aquatic plants studies does not show any toxic effects. EC50(48h) > 125 mg/L
Determination of PNEC aquatic	On the basis of the three acute toxicity data points, an assessment factor of 1000 has been applied to the lowest reported effect concentration of 120 mg/L. The PNECaquatic is determined to be 0.12 mg/L.
Current Regulatory Controls[3,4]	
Australian Hazard Classification	This chemical is a permitted food colour in both Australia and New Zealand.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	This chemical is a certified colour additive approved by the FDA in the United States to colour food, drugs and cosmetics.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	Not readily biodegradable. Thus, it is expected to meet the screening criteria for persistence.
B/vB criteria fulfilled?	As the estimated Log Pow is -10.7 (Log Pow < 4.5), it is not expected to be bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of this chemical is >0.01 mg/L. Thus, it is not expected to meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	April 2019

References

Redacted

AECOM Imagine it. Delivered.

Toxicity Summary - Water Flow Assurance Tracer (WFT)

Chemical and Physical Properties[3,4,8,9]	
CAS number	One chemical (proprietary)
Molecular formula	Proprietary
Product name	--
Molecular weight	194.19
Solubility in water	$2.16x10^{4}$ mg/L at 25 deg C
pH	6.9
Melting point	236.2 deg C
Boiling point	178 deg C
Vapour pressure	Odourless white crystals or crystalline powder
Henrys law constant	$9.0x10^{-7}$ mm Hg at 25 deg C
Explosive potential	$1.1X10^{-11}$ atm-cu m/mole at 25 deg C
Flammability potential	Combustible. Gives off irritating of toxic fumes in a fire.
Colour/Form	No data found
Overview	This WFT is a naturally occurring substance in various plant species. The use in food is the predominant way of human exposure and of exposure of the environment. It is generally recognised as safe (GRAS) as a food additive by the US FDA.
Environmental Fate[4,8,9]	
Soil/Water/Air	If released to air, a vapor pressure of 9.0X10-7 mm Hg at 25 deg C indicates this chemical will exist in both the vapor and particulate phases in the atmosphere. In vapor-phase the chemical will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 20 hours. The Henry's law constant of 0.00000363 Pa m^3/mol indicates that the substance is non-volatile from water surfaces. If released to soil, this chemical is expected to have low to no mobility based upon Koc values of 741 and 7762 determined in silt and sandy loam soils. An approximated Koc of 71 suggests high mobility in sand which contains no clay and very low organic carbon content. Volatilization from moist soil surfaces is not expected to be an important fate process based upon an estimated Henry's Law constant of 1.1X10-11 atm-cu m/mole. Various biodegradation studies have found this chemical to be readily biodegradable. If released into water, this chemical is expected to adsorb to suspended solids and sediment based upon the Koc. Volatilization from water surfaces is not expected to be an important fate process based upon this compound's estimated Henry's Law constant. An estimated BCF of 3 (log Kow of -0.07) suggests the potential for bioconcentration in aquatic organisms is low. The hydrolysis half-life of this chemical in water is reported to be >1 year. Degradation in natural water can occur through photodegradation and biodegradation.

Human Health Toxicity Summary [1,2,3,5,6,7,8,9]	
Chronic Repeated Dose Toxicity	This chemical was tested for carcinogenicity in five studies in rats by oral administration. In two of these studies, no significant difference in the incidence of tumours at any site was found. The other three studies were found to be inadequate for evaluation. Studies on oral and intraperitoneal administration of this chemical to mice were found to be inadequate for evaluation. In one study, decaffeinated coffee to which this chemical was added was tested by oral administration to rats; overall, no increase in tumours at any site was observed as compared to appropriate controls. Administration of this chemical in combination with known carcinogens resulted in decreased incidences of lung tumours in mice treated with urethane, of mammary tumours in rats treated with diethylstilboestrol and of skin tumours in mice treated with either ultra-violet light or cigarette-smoke condensate. This chemical did not influence the incidence of bladder tumours induced in rats by N-nitroso-N-butyl(4-hydroxybutyl)amine in three experiments or of pancreatic tumours induced in rats by 4-hydroxyaminoquinoline-1-oxide in another study. Nawrot et al. (2003) concluded in their review of the effects of this chemical on human health that "for the healthy adult population, moderate daily this chemical intake at a dose level up to 400 mg/day (equivalent to 6 mg/kg body weight/day in a 65-kg person) is not associated with adverse effects such as general toxicity, cardiovascular effects, effects on bone status and calcium balance (with consumption of adequate calcium), changes in adult behaviour, increased incidence of cancer and effects on male fertility." It was indicated that habitual daily use of this chemical at greater than 500-600 mg/day (8.3 - 10 mg/kg) could be considered a health risk. For women, this chemical intake greater than 400 mg/day (6.7 mg/kg) "may increase the risk of detrusor instability (unstable bladder) development in women". The EFSA's panel on dietetic products, nutrition and allergies concluded that single doses of caffeine up to 200 mg (3 mg/kg/bw) from all sources do not raise safety concerns for the general healthy adult population. Intakes up to 400 mg per day (5.7 mg/kg bw) consumed throughout the day do not raise safety concerns for healthy adults in the general population, except pregnant women. A safety level of 3 mg/kg bw per day is also proposed for habitual caffeine consumption by children and adolescents.
Carcinogenicity	IARC evaluates that this chemical is not classifiable as to its carcinogenicity to humans (group 3).
Mutagenicity/ Genotoxicity	The potential for this chemical to induce genotoxicity has been evaluated in both in vitro an in vivo studies, with in vitro studies indicating both genotoxic and non-genotoxic results; in vivo studies have shown that, overall, this chemical is not genotoxic .
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	This chemical has been shown to cause adverse reproductive and developmental effects in mice, rats, rabbits and monkeys. Testicular atrophy was observed at high dose levels in rats. Reproductive studies in mice showed no effect on pregnancy but there was a decrease in litter size at birth. Teratogenic effects were usually associated with high, single, daily doses that were also associated with other signs of maternal toxicity. High daily levels given as divided doses were less toxic to the conceptus that when given as a single dose. Reduced fetal body weight was observed in rats. A reversible delay in ossification of the sternum was observed in rats at a relative low dose given by gavage. With administration in drinking-water, similar effects were seen, but at higher doses. One epidemiological study revealed no effect of this chemical on the sex ratio of their children. In lymphocytes of normal, this chemical-exposed people, chromosomal aberrations were not observed. An increased frequency of micronucleated blood cells was observed in otherwise healthy splenectomized people exposed to this chemical. Urine of this chemical-exposed persons was not mutagenic to Salmonella typhimurium.

Acute Toxicity	After oral application the LD50 for rats (10 animals/group/sex) was found to be 261-383 mg/kg bw; as clinical symptoms of toxicity, dyspnoea and staggering were seen after oral intake. In further reports the oral LD50 for rats was reported to be 200-400 mg/kg bw and for mice 185 mg/kg bw. The inhalation of the substance by rats as an aerosol for a period of 4 h resulted in an LC50-value of ca. 4.94 mg/l. Irregular and accelerated respiration were noted in this study. The LD50 for dermal application was >2000 mg/kg bw; no clinical symptoms of toxicity were observed. In animals studies this chemical showed moderate toxicity after oral uptake and inhalation and a low acute toxicity after dermal treatment .
Irritation	The undiluted substance was not irritating to the eyes of rabbits. Mean irritation indices were 0.9 (corneal opacity), 0 (iritis), 1.6 (conjunctival erythema) and 0.6 (conjunctival edema). The strongest signs of irritation were observed in 3/3 animals within the first 24h. By day 8 only one animal showed slight corneal opacity and conjunctival redness. The substance in a 50% aqueous dilution was not irritating to the skin of rabbits (Irritation index was 0) (OECD guideline 404 and 405). This chemical is not irritating to skin and eyes.
Sensitisation	No data available.
Key Study/Critical Effect for Screening Criteria	The American College of Obstetricians and Gynaecologists (2010) concluded that moderate chemical consumption (<200 mg/day) does not appear to be a major contributing factor in miscarriage or preterm birth. The EFSA's panel on dietetic products, nutrition and allergies concluded that single doses of caffeine up to 200 mg (3 mg/kg/bw) from all sources do not raise safety concerns for the general healthy adult population Thus, the acceptable daily intake of this chemical will be set at 200 mg/person/day for the derivation of a drinking water guidance value. Assuming that humans consume 2 litres of water a day, the drinking water guidance value for this chemical is determined to be 100 mg/L.
Ecological Toxicity [8,9]	
Aquatic Toxicity	Acute toxicity guideline studies have been conducted in fish, invertebrates and algae (OECD, 2002a,b; ECHA REACH database). A 96-hour LC50 in Leuciscus idus was reported to be 87 mg/L; the 48-hour EC50 in Daphnia magna was reported to be 182 mg/L. and the ErC50 in Scenedesmus subspicatus was reported to be >100 mg/L. .
Determination of PNEC aquatic	Based on the lowest acute toxicity value of 87 mg/L in fish and an assessment factor of 1,000, a PNECaquatic is determined to be 0.087 mg/L
Current Regulatory Controls	
Australian Hazard Classification	No data found
Australian Occupational Exposure Standards	No data found
International Occupational Exposure Standards	No data found
Australian Food Standards	No data found
Australian Drinking Water Guidelines	No data found
Aquatic Toxicity Guidelines	No data found
Australian Hazard Classification	No data found
Australian Occupational Exposure Standards	No data found

PBT Assessment	
P/vP Criteria fulfilled?	This chemical is expected to be readily biodegradable and thus would not be expected to meet the screening criteria for persistence.
B/vB criteria fulfilled?	This chemical is water-soluble and bioaccumulation is not expected according to the log Kow (0.07). Thus, this chemical is not likely to meet the screening criteria for bioaccumulation.
T criteria fulfilled?	Long term data not available (acute data >0.1 mg/L); potentially not toxic.
Overall conclusion	Not a PBT substance (based on screening data).

References

Redacted

AECOM Imagine it. Delivered.

Toxicity Summary - Water SoluableTracers (CFTs) - Benzoic acid used as analogue data

Chemical and Physical Properties[1]	
CAS number	20 chemicals (proprietary)
Molecular formula	Proprietary
Molecular weight	140 – 260 (approximate)
Solubility in water	3.5 g/L at 25 °C
Melting point	122.4 °C
Boiling point	249.2 °C
Vapour pressure	0.11 Pa at 20 °C
Henrys law constant	No data available.
Explosive potential	Non-flammable
Flammability potential	Non explosive
Colour/Form	A white crystalline powder with a pleasant odour.
Overview	CFTs are organic compounds. Benzoic acid has been used as analogue data.
Environmental Fate[1,2,3]	
Soil/Water/Air	If released to air, a vapor pressure of 7.0X10-4 mm Hg at 25 deg C indicates benzoic acid will exist solely as a vapor in the atmosphere. Vapor-phase benzoic acid will be degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 9 days. Benzoic acid absorbs light at wavelengths >290 nm and, therefore, may be susceptible to direct photolysis by sunlight. If released to soil, benzoic acid is expected to have very high mobility based upon an estimated Koc of 15 (log Kow of 1.87). The pKa of benzoic acid is 4.20, indicating that this compound will exist almost entirely in the anion form in the environment and anions generally do not adsorb more strongly to soils containing organic carbon and clay than their neutral counterparts. Volatilization from moist soil is not expected because the compound exists as an anion and anions do not volatilize. Benzoic acid is not expected to volatilize from dry soil surfaces based upon its vapor pressure. If released into water, benzoic acid is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. Biodegradation half-lives of 0.85 and 3.6 days using inoculum from a polluted river and a reservoir, respectively, suggest that biodegradation may be an important fate process in water. Measured BCF values of <10, 14, and 21 were reported for Golden ide (Leuciscus idus melanotus)(1), trout(2), and mosquito fish (Gambusia affinis)(3), respectively. This BCF range suggests the potential for bioconcentration in aquatic organisms is low.

Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	Based on the weight of evidence the chemical is not considered to cause serious damage to health by repeated oral exposure (no observed adverse effect level (NOAEL) of 825 mg/kg bw/d). Effects observed at > 1000 mg/kg bw/d included increased mortality, reduced weight gain, and liver and kidney effects (OECD, 2004). Available animal data suggest that the chemical is not likely to cause serious damage to health via repeated dermal exposure. No treatment-related effects in rabbits at doses of up to 2500 mg/kg bw/d applied 5 d/wk for 3 weeks (OECD, 2004). Available animal data suggest that the chemical is not likely to cause serious damage to health via repeated inhalation exposure. The only available rat study for this chemical reported 2/20 mortalities at 1.2 mg/L 6 h/d (5 d/wk over 4 wk). Local reddish discharge around the nostrils and inflammatory cell infiltrates and interstitial fibrosis of the lung secondary to local irritant effects were also observed at ³ 0.25 mg/L. On the basis of systemic effects, the NOAEC is considered to be > 0.25 mg/L 6 h/d (ECHA, 2011).
Carcinogenicity	Based on the available data, the chemical is not considered carcinogenic. The chemical was not carcinogenic (NOAEL 500 mg/kg bw/d) in a lifetime 3-generation study in rats when given with the diet at doses up to 500 mg/kg bw/d. No increase in the lifetime tumour incidence, clinical abnormalities or histopathological changes were observed (OECD, 2004). A lifelong study using male/female Swiss Albino mice given the chemical (2 %) continuously in drinking water showed no carcinogenic effect (such as effect on survival or incidence of tumours) (CICAD, 2000).
Mutagenicity/ Genotoxicity	Based on the weight of the evidence of the in vitro and in vivo genotoxicity data, the chemical is not considered mutagenic or clastogenic. In vitro data using the reverse mutation assays with various strains of Salmonella typhimurium (with and without metabolic activation) and sister chromatid exchange assays (except one equivocal result) were negative. Weak genotoxic effects or equivocal results were observed in most of the chromosome aberration assays in three mammalian cell lines and two of the recombination assays in Bacillus subtilis (no further information available, only summary given) (REACH). No genotoxicity was observed in the in vivo cytogenetic, micronucleus, or other assays at either somatic or germ cell level (OECD, 2004).
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No evidence of reproductive or developmental toxicity was observed for the chemical.

Acute Toxicity	The chemical is of low acute toxicity in animal tests following oral exposure. The median lethal dose (LD50) in rats and mice is greater than 2000 mg/kg bw/d. LD50 in rats ranged from 1700-3040 mg/kg bw/d and in mouse ranged from 1940-2370 mg/kg bw/d. However, the studies that reported the lower LD50s were all pre-guideline studies and no further information was available to critically assess the data. LD50s reported in two reliable studies were 2250 mg/kg bw/d (mice) and 2565 mg/kg bw/d (rats) (OECD, 2004). The chemical is of low acute toxicity in animal tests following oral exposure. The median lethal dose (LD50) in rats and mice is greater than 2000 mg/kg bw/d. LD50 in rats ranged from 1700-3040 mg/kg bw/d and in mouse ranged from 1940-2370 mg/kg bw/d. However, the studies that reported the lower LD50s were all pre-guideline studies and no further information was available to critically assess the data. LD50s reported in two reliable studies were 2250 mg/kg bw/d (mice) and 2565 mg/kg bw/d (rats) (OECD, 2004). The chemical exhibits low acute toxicity in animal tests as evidenced by reported dermal LD50 (median lethal concentration) in rats of greater than 2000 mg/kg bw (OECD, 2004). The chemical exhibits low acute toxicity in animal tests following inhalation exposure. No mortalities or toxic effects were observed in rats and mice with the reported median lethal concentration (LC50) > 12.2 mg/L/4-h (ECHA, 2011; OECD, 2004).
Irritation	Inhalation toxicity of the chemical was evaluated in one rat study (0, 0.025, 0.25 and 1.2 mg/L, 6 h/d 5 d/wk over 4 weeks) using fine benzoic acid dust (see Repeat dose toxicity - Inhalation). A reddish discharge around the nostrils was seen in the mid and high dose groups. An increased incidence and intensity of interstitial inflammatory cell infiltrate and interstitial fibrosis (indicating upper respiratory tract irritation) was noted at all doses. Observed histopathological changes were most likely due to a persistent irritating effect of the test substance on the lung. No changes in gross pathology were noted (REACH). The chemical was irritating (erythema and swelling of the ear lobe) in the guinea pig ear swelling test at ³ 1%, particularly when dissolved in ethanol, although it was not found irritating in the rabbit (OECD, 2004). The chemical was highly irritating in rabbit eyes, causing irreversible corneal opacity and chemosis in 2/3 animals, and increasing conjunctival redness severity with white/grey discoloration after 2-day observation. A Draize score of 35 was given based on the effects (REACH). In another rabbit study an irritation score of 65.0/110 was noted. No further details were available from this study (OECD, 2004).
Sensitisation	The negative results seen for the chemical from several skin sensitisation animal studies including guinea pig maximisation test (GPMT), Buehler test and local lymph node assay (LLNA) support a conclusion that the chemical is not a skin sensitiser (REACH). The chemical did not induce sensitisation in healthy volunteers although some allergic reactions were noted in 34/537 patients with suspected contact dermatitis (at 2 %) (SCCP, 2005) and 9/121 patients with dermatoses and 10/57 patients with chronic urticaria (at 5 %) (ECHA, 2011).
Health Effects Summary	The critical health effects associated with the chemical (but not the salts) are skin, eye and respiratory tract irritation. However, no systemic effects were seen with benzoic acid. The salts are expected to exist almost entirely as the benzoate ion under normal physiological conditions and will not have the local irritant properties that arise from the acidity of benzoic acid. Therefore, it is unlikely that any systemic effects will be observed with the salts of benzoic acid.
Key Study/Critical Effect for Screening Criteria	The critical lowest No Observed Adverse Effect (NOAEL) level for the purposes of risk assessment is 825 mg/kg bw/day from the repeated chronic oral toxicity study.

Ecological Toxicity [2]	
Aquatic Toxicity	Studies on three trophic levels are available with the lowest EC50 found in algae (33.1 mg/L). In this study the concentrations decreased significantly over the exposure period of 72 hours. The LC50 for fish is 44.6 mg/L and for daphnia an EC50 of > 100 mg/L was derived. The EC10 from the algae study is 3.4 mg/L, which is much lower than the NOEC for fish (120 mg/L in a 28 day study) and daphnia (25 mg/L in 21 day reproduction test).
Determination of PNEC aquatic	Long-term data was available for a fish, invertebrate and algae. An assessment factor of 10 was used on the lowest NOEC of 3.4 mg/L for algae for a resulting PNEC of 0.34 mg/L.
Current Regulatory Controls[1]	
Australian Hazard Classification	The chemical is not listed on the Hazardous Substances Information System (HSIS) (Safe Work Australia).
Australian Occupational Exposure Standards	No specific exposure standards are available.
International Occupational Exposure Standards	The following exposure standards are identified (Galleria Chemica): An exposure limit (TWA) of 5–10 mg/m^3 in different countries such as USA (California, Tennessee), Canada and England.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	Benzoic acid is readily biodegradable and as such not persistent in the environment.
B/vB criteria fulfilled?	Based on the measured BCF values of <10 to 21 and a log Kow of 1.87 benzoic acid is not expected to be bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of this chemical is >0.01 mg/L. Thus, it is not expected to meet the screening criteria for toxicity.
Overall conclusion	Not PBT
Revised	April 2019

References

Redacted

AECOM Imagine it. Delivered.

Toxicity Summary - Gas Phase Frac Tracers (GFTs)

Chemical and Physical Properties[1,2,3]	
CAS number	15 chemicals (proprietary).
Molecular formula	Proprietary
Molecular weight	~300 – 500
Solubility in water	Insoluble
Melting point	~-37 °C
Boiling point	~76 °C
Vapour pressure	666 @ 25 °C
Henrys law constant	No data available
Explosive potential	Non explosive
Flammability potential	Non-flammable
Colour/Form	Colourless, odourless liquid
Overview	GFTs tracers are compounds that consist of a carbon and fluorine atoms joined by covalent bonds. GFTs are very stable because of the strength of the carbon–fluorine bond. GFTs are chemically inactive, nontoxic, and non-flammable compounds that are found in the atmosphere at very low levels. They are chemical inert, have no biological effects and are very safe. GFTs present no known danger to humans if inhaled or ingested. There are no regulatory restrictions on the use or emission of GFTs. Information for Perfluorocarbons (PFCs) used as analogue data.
Environmental Fate[1]	
Soil/Water/Air	GFTs as a class are extremely stable. They are not susceptible to hydrolysis, and not affected by light (including UV).
Human Health Toxicity Summary [1,2,3]	
Chronic Repeated Dose Toxicity	Two-week repeat dose preliminary inhalation toxicity (rat at a target concentration of 10,000 ppm (10%), no treatment-related effects were noted for clinical signs, body weight, food consumption, water consumption, macroscopic pathology or organ weights. 90 day inhalation study in rats: no treatment-related effects were observed in this study in which rats were exposed to 5,000 ppm, 15,000 ppm, and 50,000 ppm of the test material for 6 hours per day, 5 days per week for a total of 13 weeks. These results indicate that the toxicity of the test material following repeated inhalation exposure is very low and suggest that the gas can be treated as a simple asphyxiant. In a short term repeated Dose 28 Day oral toxicity study in rodents conducted in accordance to the OECD Guideline 407, the test subjects showed no toxic effect at a dosage of 1000 mg/kg/day over 28 days. The NOEL was determined to be 1000 mg/kg/day.
Carcinogenicity	Chromosomal aberration test in cultured mammalian cells: non-clastogenic
Mutagenicity/ Genotoxicity	Bacterial mutation assay salmonella typhimurium (strains ta 1535, ta 1537, ta 1538, ta 98 and ta 100): negative.
Reproductive Toxicity / Developmental Toxicity/Teratogenicity	No data available.

Acute Toxicity	Inhalation 4-hour LC50 : > 800,000 ppm in rats Effects observed in animals by inhalation include decreased growth rate, pulmonary changes, irregular respiration, increased urine volume and creatinine, reversible pathological changes in the kidneys, and increased urinary fluoride concentration. One study showed no arrhythmogenic effects in dogs at a concentration of 20 %, while another study did show some arrhythmogenic effects in both guinea pigs and dogs. Long-term inhalation exposures resulted in an initial decrease in growth rate, but no other adverse changes were noted. No animal test reports are available to define carcinogenic, developmental, or reproductive hazards. The compound does not produce genetic damage in bacterial cell cultures but has not been tested in animals. Acute inhalation toxicity study (rat): the 4-hour LC50 is above 110,000 ppm. These results suggest that on an acute inhalation basis the test material can be considered as a simple asphyxiant.
Irritation	Non-irritating
Sensitisation	Not sensitising
Health Effects Summary	The chemicals have been used in various medical applications, both in trials and in routine use, in human subjects, for some forty years, indicating these materials have zero toxicity to humans.
Key Study/Critical Effect for Screening Criteria	The NOEL level for the purposes of risk assessment is 1000 mg/kg bw/day from the repeated short term oral toxicity study.
Ecological Toxicity [1]	
Aquatic Toxicity	Fish 96h LC50 > 100 mg/L Invertebrates 48h EC50 > 0.1 mg/L Microorganism 3h EC50 > 100 mg/L Pimephales promelas (fathead minnow) 96 h NOEC = 1000 mg/L
Determination of PNEC aquatic	$PNEC_{aquatic}$ has not been calculated. The substance exhibits no toxicity.
Current Regulatory Controls	
Australian Hazard Classification	No data available.
Australian Occupational Exposure Standards	No data available.
International Occupational Exposure Standards	No data available.
Australian Food Standards	No data available.
Australian Drinking Water Guidelines	No data available.
Aquatic Toxicity Guidelines	No data available.
PBT Assessment	
P/vP Criteria fulfilled?	Yes, GFTs are not biodegradable. However, they are volatile and are quickly removed from the environment.
B/vB criteria fulfilled?	The estimated Log Kow is generally > 4.5 (EPI Suite), thus it is expected to be bioaccumulative.
T criteria fulfilled?	No. Fish 96 h NOEC = 1000 mg/L. Thus, it is not expected to meet the screening criteria for toxicity.
Overall conclusion	Not PBT

Revised	April 2019

References

Redacted

Appendix D.2 Beetaloo Recycled Flowback Water Risk Assessment Update

Recycled Flowback Data

Chemical Name	CAS Number	Maximum Concentration in Flowback Fluid (mg/L)	Tier 1 Screening Assessment	Discussion	Tier 2 Assessment Worker Ingestion Risk	Tier 2 Assessment Worker Dermal Risk	Tier 2 Assessment Worker Aerosol Inhalation Risk	Hazard Quotient	Outcome of Tier 2 Worker Risk Assessment
Benzene	71-43-2	0.007	Tier 2	Maximum concentration above NHMRC 2011 Health Guideline	Threshold Risk = 6.15E-06 Non Threshold Risk = 1.0E-12	Threshold Risk = 1.4E-06 Non Threshold Risk = 2.4E-13	Threshold Risk = 1.6E-05 Non Threshold Risk = 4.1E-14	Threshold Risk =2.4E-05 Non Threshold Risk = 1.3E-12	Based on the calculated risks the chemical is of low concern for workers (refer to risk calculations for further detail).
Ethylbenzene	100-41-4	0.01	Tier 1	Maximum concentration below NHMRC 2011 Health Guideline	NA	NA	NA	NA	NA
Toluene	108-88-3	0.048	Tier 1	Maximum concentration below NHMRC 2011 Health Guideline	NA	NA	NA	NA	NA
Xylene Total	1330-20-7	0.23	Tier 1	Maximum concentration below NHMRC 2011 Health Guideline	NA	NA	NA	NA	NA
Alkalinity (Bicarbonate) as CaCO3	471-34-1	716	Tier 1	NA. No human health guideline available*	NA	NA	NA	NA	NA
Alkalinity (Total) as CaCO3	471-34-1	716	Tier 1	NA. No human health guideline available*	NA	NA	NA	NA	NA
Ammonia (filtered)	007664-41-7	34	Tier 1	Maximim concentration below the WHO Drinking Water Guideline	NA	NA	NA	NA	NA
Anions Total	-	724	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Bicarbonate	-	873.52	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Bromide (filtered)	7726-95-6	260	Tier 1	NA. No human health guideline available*	NA	NA	NA	NA	NA
Calcium (filtered)	7440-70-2	1740	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Carbonate	-	0.6	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Cations Total	-	718	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Chloride	16887-00-6	25400	Tier 1	NA. No human health guideline available*	NA	NA	NA	NA	NA
Electrical Conductivity (Lab)	-	59600	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Fluoride	16984-48-8	1.2	Tier 1	Maximum concentration below NHMRC 2011 Health Guideline	NA	NA	NA	NA	NA
Kjeldahl Nitrogen Total	-	65.6	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Magnesium (filtered)	7439-95-4	370	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Methane	74-82-8	8.37	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Nitrite + Nitrate (as N)	014797-55-8	0.26	Tier 1	Maximim concentration below the WHO Drinking Water Guideline	NA	NA	NA	NA	NA
Nitrogen (Total)	7727-37-9	65.6	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
pH (Lab)	-	6.74	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Phosphorus	7723-14-0	1.07	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Potassium (filtered)	7440-09-7	83	Tier 1	NA. No human health guideline available*	NA	NA	NA	NA	NA
Silicon as Si	7440-21-3	16	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Silicon as SiO2	7631-86-9	33	Tier 1	NA. No human health guideline available	NA	NA	NA	NA	NA
Sodium (filtered)	7440-23-5	13900	Tier 1	NA. No human health guideline available*	NA	NA	NA	NA	NA
Sulphate as SO4 (filtered)	14808-79-8	42	Tier 1	NA. No human health guideline available*	NA	NA	NA	NA	NA
Total Dissolved Solids (filtered)	-	49200	Tier 1	NA. No human health guideline available*	NA	NA	NA	NA	NA
Total Dissolved Solids (Calculated)	-	37900	Tier 1	NA. No human health guideline available*	NA	NA	NA	NA	NA
Total Hardness as CaCO3 (filtered)	-	5560	Tier 1	NA. No human health guideline available*	NA	NA	NA	NA	NA
Aluminium	7429-90-5	0.3	Tier 1	Maximum concentration below the USEPA RSL 2022 Guideline	NA	NA	NA	NA	NA
Arsenic	007440-38-2	0.084	Tier 2	Maximum concentration above NHMRC 2011 Health Guideline	1.48E-04	6.79E-05	5.75E-03	5.97E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
Barium	7440-39-3	110	Tier 2	Maximum concentration above NHMRC 2011 Health Guideline	1.93E-03	8.89E-04	1.08E-02	1.36E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
Boron	7440-42-8	54.5	Tier 2	Maximum concentration above NHMRC 2011 Health Guideline	9.57E-04	8.19E-04	5.33E-03	7.11E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
Chromium (III+VI) (filtered)		0.048	Tier 1	Maximum concentration below the USEPA RSL 2022 Guideline	NA	NA	NA	NA	NA
Cobalt	7440-48-4	0.024	Tier 2	Maximum concentration above USEPA RSL 2022 Guidelline	6.02E-05	1.11E-05	1.64E-02	1.65E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
Iron	7439-89-6	97	Tier 2	Maximum concentration above USEPA RSL 2022 Guidelline	4.87E-04	2.24E-04	2.71E-03	3.42E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
Manganese	7439-96-5	3.09	Tier 2	Maximum concentration above NHMRC 2011 Health Guideline	6.78E-05	3.12E-05	3.78E-04	4.77E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
Mercury	007439-97-6	0.026	Tier 2	Maximum concentration above NHMRC 2011 Health Guideline	1.52E-04	2.12E-05	8.90E-03	9.08E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
Nickel	7440-02-0	0.04	Tier 2	Maximum concentration above NHMRC 2011 Health Guideline	1.17E-05	1.08E-06	1.37E-01	1.37E-01	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
Strontium	7440-24-6	170	Tier 2	Maximum concentration above USEPA RSL 2022 Guidelline	9.95E-04	3.28E-04	5.54E-03	6.87E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
Zinc		0.13	Tier 1	Maximum concentration below the USEPA RSL 2022 Guideline	NA	NA	NA	NA	NA
2-methylnaphthalene	91-57-6	0.046	Tier 2	Maximum concentration above USEPA RSL 2022 Guidelline	4.04E-06	1.70E-04	2.25E-05	1.97E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
3-&4-methylphenol		0.0113	Tier 1	Maximum concentration below the USEPA RSL 2022 Guideline	NA	NA	NA	NA	NA
Naphthalene	91-20-3	0.043	Tier 2	Maximum concentration above USEPA RSL 2022 Guidelline	7.55E-06	1.62E-04	2.95E-04	4.64E-04	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
Phenol		0.004	Tier 1	Maximum concentration below the USEPA RSL 2022 Guideline	NA	NA	NA	NA	NA
TPH C6 - C9 Fraction[A]	-	0.31	Tier 1	Maximim concentration below the WHO Drinking Water Guideline	NA	NA	NA	NA	NA
TPH C10 - C14 Fraction[A]	-	0.93	Tier 2	Maximum concentration above WHO Drinking Water Guidelline	7.08E-05	2.26E-03	1.91E-04	2.52E-03	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
TPH C15 - C28 Fraction[A]	-	3.07	Tier 2	Maximum concentration above WHO Drinking Water Guidelline	3.23E-04	4.82E-02	1.80E-03	5.03E-02	Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
TPH C29 - C36 Fraction[A]	-	1.72	Tier 2	Maximum concentration above WHO Drinking Water Guidelline					Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
TPH C34 - C40 Fraction[A]	-	0.65	Tier 2	Maximum concentration above WHO Drinking Water Guidelline					Based on the calculated HQ the chemical is of low concern for workers (refer to risk calculations for further detail).
					Total Risk		**Non-Threshold**	**1.3E-12**	The calculated risks associated with potential exposure to COPC measured in recycled flowback water is below the Non-Threshold target of 1E-06 and Threshold target of 1, respectively. Hence, chronic health risks are considered to be low and acceptable.
							Threshold	**2.5E-01**	

Notes
NA - Not Applicable
* - Listed as naturally occuring chemical for which drinking water guideline values have not been established (WHO 2017)
A - No fractionation into aromatic and aliphatic components was undertaken for the identified TPH impacts. The approach adopted for the fractionation of TPH into aromatic and aliphatic components is summarised below:
• For TPH C6-C9 it is assumed that the total BTEX concentration reported is representative of the aromatic portion of TPH, and that the remaining TPH (i.e., total TPH C6-C9 minus total BTEX) is representative of the aliphatic portion of the TPH fraction. If the total BTEX concentration is less than the total TPH C6-C9 concentration, the TPH C6-C9 aliphatic portion has been calculated as the remaining concentration of TPH C6-C9;
•The higher TPH fractions are split conservatively, assuming a 50/50 ratio for aromatic and aliphatic components;
•TPH 15+ is the sum of the C15 - C40 concentrations

NHMRC 2011 Australian Drinking Water Guidelines 6. Volume 1. National Water Quality Management Strategy. National Health and Medical Research Council. Updated September 2022.
WHO Drinking Water Guidelines 2017 - World Health Organisation Drinking Water Guidelines and rolling revisions
USEPA RSLs 2022 - USEPA Regional Screening Levels

Toxicity and Dermal Absorption Parameters

C = calculated from chronic value, Ch = chronic value adopted

CAS#	Chemical	Oral/Dermal Exposures						Inhalation Exposures			
		Non-Threshold Slope Factor $(mg/kg/day)^{-1}$		Threshold Chronic TDI or RfD (mg/kg/day)		Dermal Permeability (cm/hr)	Reference	Inhalation Unit Risk $(ug/m^3)^{-1}$		Threshold Chronic TC or RfC (mg/m^3)	
	Chemicals of Potential Concern										
71-43-2	Benzene	3.50E-02	NHMRC (2011)	0.0040	USEPA IRIS	5.00E-04	USEPA (1995) as per NEPC (2013)	6.00E-06	WHO (2010)	3.00E-02	USEPA IRIS
007440-38-2	Arsenic			0.0020	NEPC (2013)	1.00E-03	RAIS			1.00E-03	RIVM (2001)
7440-39-3	Barium			0.2000	ATSDR (2007)	1.00E-03	RAIS			0.7	converted from RFD
7440-42-8	Boron			0.2000	USEPA RSL (2022)	1.86E-03	EPI			0.7	converted from RFD
7440-48-4	Cobalt			0.0014	RIVM (2001)	4.00E-04	RAIS			1.00E-04	WHO (2006)
7439-89-6	Iron			0.7000	PPRTV (USEPA RSL (2022)	1.00E-03	RAIS			2.45	converted from RFD
7439-96-5	Manganese			0.1600	ATSDR (2008)	1.00E-03	RAIS			0.56	converted from RFD
007439-97-6	Mercury			0.0006	WHO (2017)	3.03E-04	RAIS			2.00E-04	WHO (2003)
7440-02-0	Nickel			0.0120	WHO (2017)	2.00E-04	RAIS			2.00E-05	EA (2009)
7440-24-6	Strontium			0.6000	USEPA RSL (2022)	7.17E-04	EPI			2.1	converted from RFD
91-57-6	2-methylnaphthalene			0.0400	ATSDR (2005)	9.17E-02	RAIS			0.14	converted from RFD
91-20-3	Naphthalene			0.0200	IRIS	4.66E-02	RAIS			1.00E-02	WHO (2010)
-	TPH C10 - C14 Fraction Aromatic[E]			0.0300	TPHCWG	6.94E-02	TPHCWG			0.2	TPHCWG
-	TPH C10 - C14 Fraction Aliphatic[E]			0.1000	TPHCWG	6.94E-02	TPHCWG			1	TPHCWG
-	TPH C15+ Fraction Aromatic[E]			0.0300	TPHCWG	3.24E-01	TPHCWG			0.105	converted from RFD
-	TPH C15+ Fraction Aliphatic[E]			2.0000	TPHCWG	3.24E-01	TPHCWG			7	converted from RFD

Notes:

E - No fractionation into aromatic and aliphatic components was undertaken for the identified TPH impacts. The approach adopted for the fractionation of TPH into aromatic and aliphatic components is summarised below:

- For TPH C6-C9 it is assumed that the total BTEX concentration reported is representative of the aromatic portion of TPH, and that the remaining TPH (i.e., total TPH C6-C9 minus total BTEX) is representative of the aliphatic portion of the TPH fraction. If the total BTEX concentration is less than the total TPH C6-C9 concentration, the TPH C6-C9 aliphatic portion has been calculated as the remaining concentration of TPH C6-C9;
- The higher TPH fractions are split conservatively, assuming a 50/50 ratio for aromatic and aliphatic components;
- TPH 15+ is the sum of the C15 - C40 concentrations

References:

IRIS - Integrated Risk Information System (USEPA)

RAIS - US Department of Energy Office of Environmental Management, Risk Assessment Information System

ATSDR - Agency for Toxic Substance and Disease Registry toxicity profiles for individual compounds.

PPRTV - Provisional Peer Reviewed Toxicity Values (USEPA, Office of Superfund Remediation and Technology Innovation (OSRTI))

NHMRC (2011) Australian Drinking Water Guidelines 6. Volume 1. National Water Quality Management Strategy. National Health and Medical Research Council. Updated September 2022.

USEPA (2022) Regional Screening Levels. Updated May 2022. https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables

NEPC (2013) National Environmental Protection (Assessment of Site Contamination) Measure 1999 as ammended May 2013. National Environmental Protection Council, May 2013.

TPHCWG - TPH Criteria Working Group. Development of fraction specific reference doses (RfDs) and reference concentrations (RfCs) for total petroleum hydrocarbons (TPH), 1997

WHO (2010) Guidelines for Indoor Air Quality

WHO (2017) - World Health Organisation Drinking Water Guidelines and rolling revisions

Exposure to Chemicals via Incidental Ingestion of Flowback fluid - Recycled

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Ingestion of Flowback Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Ingestion Rate (IRw)	L/day or L/hr	0.005	Assume Incidental ingestion of 5 ml (1 tsp) of water per day during fraccing.
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**4.2E-09** **3.5E-06**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Intake Factor (ref: USEPA 1989)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

CAS	Chemical	Toxicity Data				Concentration	Daily Intake		Calculated Risk	
		Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% Chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)	in Water	NonThreshold	Threshold	NonThreshold Risk	Chronic Hazard Quotient
		$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
71-43-2	Benzene	3.5E-02	4.0E-03		4.0E-03	0.01	2.9E-11	2.5E-08	1.0E-12	6.1E-06
007440-38-2	Arsenic		2.0E-03		2.0E-03	0.08	3.5E-10	3.0E-07	--	1.5E-04
7440-39-3	Barium		2.0E-01		2.0E-01	110.00	4.6E-07	3.9E-04	--	1.9E-03
7440-42-8	Boron		2.0E-01		2.0E-01	54.50	2.3E-07	1.9E-04	--	9.6E-04
7440-48-4	Cobalt		1.4E-03		1.4E-03	0.02	1.0E-10	8.4E-08	--	6.0E-05
7439-89-6	Iron		7.0E-01		7.0E-01	97.00	4.1E-07	3.4E-04	--	4.9E-04
7439-96-5	Manganese		1.6E-01		1.6E-01	3.09	1.3E-08	1.1E-05	--	6.8E-05
007439-97-6	Mercury		6.0E-04		6.0E-04	0.03	1.1E-10	9.1E-08	--	1.5E-04
7440-02-0	Nickel		1.2E-02		1.2E-02	0.04	1.7E-10	1.4E-07	--	1.2E-05
7440-24-6	Strontium		6.0E-01		6.0E-01	170.00	7.1E-07	6.0E-04	--	1.0E-03
91-57-6	2-methylnaphthalene		4.0E-02		4.0E-02	0.05	1.9E-10	1.6E-07	--	4.0E-06
91-20-3	Naphthalene		2.0E-02		2.0E-02	0.04	1.8E-10	1.5E-07	--	7.6E-06
-	TPH C10 - C14 Fraction Aromatic[A]		0.0300		3.0E-02	0.47	1.9E-09	1.6E-06	--	5.4E-05
-	TPH C10 - C14 Fraction Aliphatic[A]		0.1000		1.0E-01	0.47	1.9E-09	1.6E-06	--	1.6E-05
-	TPH C15+ Fraction Aromatic[A]		0.0300		3.0E-02	2.72	1.1E-08	9.6E-06	--	3.2E-04
-	TPH C15+ Fraction Aliphatic[A]		2.0000		2.0E+00	2.72	1.1E-08	9.6E-06	--	4.8E-06
								Total Risk (mixture)	**1.0E-12**	**5.22E-03**

Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

A - No fractionation into aromatic and aliphatic components was undertaken for the identified TPH impacts. The approach adopted for the fractionation of TPH into aromatic and aliphatic components is summarised below:
- For TPH C6-C9 it is typically assumed that the total BTEX concentration reported is representative of the aromatic portion of TPH, and that the remaining TPH (i.e., total TPH C6-C9 minus total BTEX) is representative of the aliphatic portion of the TPH fraction. If the total BTEX concentration is less than the total TPH C6-C9 concentration, the TPH C6-C9 aliphatic portion has been calculated as the remaining concentration of TPH C6-C9;
- The higher TPH fractions are split conservatively, assuming a 50/50 ratio for aromatic and aliphatic components;
- TPH 15+ is the sum of the C15 - C40 concentrations

Dermal Exposure to Chemicals via Contact with Flow Back Water - Recycled

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Dermal Contact with Flow Back Water by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	20	Assume work 5 days per week for 1 month during the fraccing period
Exposure Duration (ED)	years	0.083	Maximum duration of the frac. Works will be complete in one month.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	30.42	USEPA 1989 and CSMS 1996
Surface Area (SAw)	cm^2	2300	Hands and forearms exposed (enHealth 2012) Occupational HSE would require long pants and closed shoes on Australian work sites; forearms conservatively included
Exposure Time (ET)	hr/day	1	Assume contact with flow back water for 1 hours per day
Conversion Factor (CF)	L/cm^3	1.E-03	Conversion of units
Intake Factor = $\frac{SAw*ET*CF*EF*ED}{BW*AT}$	L-hr/(cm-kg-day)	**1.9E-06** **1.6E-03**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor (ref: USEPA 1989, 2004)
NonThreshold Risk = Daily Intake from Water for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

CAS	Chemical	Toxicity Data				Dermal Permeability	Concentration in Water	Daily Intake		Calculated Risk	
		Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	NonThreshold Risk	Chronic Hazard Quotient
		$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
71-43-2	Benzene	3.5E-02	4.0E-03		4.0E-03	5.0E-4	0.01	6.7E-12	5.7E-09	2.4E-13	1.4E-06
007440-38-2	Arsenic		2.0E-03		2.0E-03	1.0E-3	0.08	1.6E-10	1.4E-07	--	6.8E-05
7440-39-3	Barium		2.0E-01		2.0E-01	1.0E-3	110.00	2.1E-07	1.8E-04	--	8.9E-04
7440-42-8	Boron		2.0E-01		2.0E-01	1.9E-3	54.50	1.9E-07	1.6E-04	--	8.2E-04
7440-48-4	Cobalt		1.4E-03		1.4E-03	4.0E-4	0.02	1.8E-11	1.6E-08	--	1.1E-05
7439-89-6	Iron		7.0E-01		7.0E-01	1.0E-3	97.00	1.9E-07	1.6E-04	--	2.2E-04
7439-96-5	Manganese		1.6E-01		1.6E-01	1.0E-3	3.09	5.9E-09	5.0E-06	--	3.1E-05
007439-97-6	Mercury		6.0E-04		6.0E-04	3.0E-4	0.03	1.5E-11	1.3E-08	--	2.1E-05
7440-02-0	Nickel		1.2E-02		1.2E-02	2.0E-4	0.04	1.5E-11	1.3E-08	--	1.1E-06
7440-24-6	Strontium		6.0E-01		6.0E-01	7.2E-4	170.00	2.3E-07	2.0E-04	--	3.3E-04
91-57-6	2-methylnaphthalene		4.0E-02		4.0E-02	9.2E-2	0.05	8.1E-09	6.8E-06	--	1.7E-04
91-20-3	Naphthalene		2.0E-02		2.0E-02	4.7E-2	0.04	3.9E-09	3.2E-06	--	1.6E-04
-	TPH C10 - C14 Fraction Aromatic[A]		3.0E-02		3.0E-02	6.9E-2	0.47	6.2E-08	5.2E-05	--	1.7E-03
-	TPH C10 - C14 Fraction Aliphatic[A]		1.0E-01		1.0E-01	6.9E-2	0.47	6.2E-08	5.2E-05	--	5.2E-04
-	TPH C15+ Fraction Aromatic[A]		3.0E-02		3.0E-02	3.2E-1	2.72	1.7E-06	1.4E-03	--	4.7E-02
-	TPH C15+ Fraction Aliphatic[A]		2.0E+00		2.0E+00	3.2E-1	2.72	1.7E-06	1.4E-03	--	7.1E-04
									Total Risk (mixture)	**2.36E-13**	**5.32E-02**

Note:
This scenario is deemed protective of the following scenarios due to the less frequent and short duration of exposures:
- Worker exposure during a spill (i.e.a couple breaks on a tank and releases product onto the worker) or leak scenarios

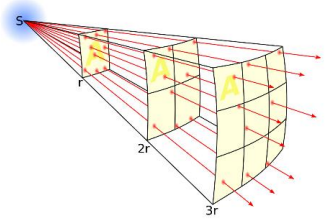
A - No fractionation into aromatic and aliphatic components was undertaken for the identified TPH impacts. The approach adopted for the fractionation of TPH into aromatic and aliphatic components is summarised below:
• For TPH C6-C9 it is typically assumed that the total BTEX concentration reported is representative of the aromatic portion of TPH, and that the remaining TPH (i.e., total TPH C6-C9 minus total BTEX) is representative of the aliphatic portion of the TPH fraction. If the total BTEX concentration is less than the total TPH C6-C9 concentration, the TPH C6-C9 aliphatic portion has been calculated as the remaining concentration of TPH C6-C9;
•The higher TPH fractions are split conservatively, assuming a 50/50 ratio for aromatic and aliphatic components;
•TPH 15+ is the sum of the C15 - C40 concentrations

Aerosol Exposure - Recycled Flowback

The concentration of COPC in aerosol spray was estimated by calculating the concentration for driftable droplets using a mixed box model in which steady state concentrations An emission factor for driftable aerosol was estimated using the algorithm presented below.

Emission Factor for Driftable Aerosol Algorithm

$$Emission\ Factor_{driftable\ aerosol}\left(\frac{L}{m^3}\right) = \frac{Box\ Centre^2(m) \times \left(\frac{Spray\ Vol\ \left(\frac{L}{hr}\right) \times Aerosol_{driftable}(\%)}{BoxVR\ \left(\frac{m^3}{hr}\right)}\right)}{BoxDistance^2\ (m)}$$



Aerosol Exposure Modelling Notes:

1) The inhalation of chemicals in mist/aerosol resultant from irrigation activities is dependent upon the concentration in water, the amount of water used per unit time, how close a person stands to the spray generation, how long they are in a position of exposure and the extent of spray drift (determined by the size of the water droplets and speed/direction of the wind). These equations are applicable for non-volatile contaminants that are inhaled.

2) These equations calculate the concentration for driftable droplets using a simple well mixed box model in which steady state air concentrations are calculated. The 'Inverse square law' is then applied to approximate the air concentration at a distance from the virtual air box. This law assumes the further away a receptor is from the spray source, the density of the droplets will decrease. The density of the spray droplets is inversely proportional to the square of the distance from the source.

Parameter	Units	Value	Description
Spray box length	m	3	Assume a 'spray box' of 3 m long.
Spray box width	m	3	Assume a 'spray box' of 3 m wide.
Box Centre	m	1.5	Distance to centre of box is 1.5 m.
$Box_{Distance}$	m	2	Distance the irrigation worker is from the 'spray box'. Assumed a distance of 2 m.
$Aerosol_{driftable}$	unitless	0.2	Proportion of aerosol spray that drifts outside the 'spray box' and available for exposure. Assumed 0.2, based on a droplet size of 400 – 500 µm that falls approximately 0.3 m in less than 10 seconds, with a lateral drift of approximately 3.5 m in a 5 km/hr wind (i.e. a light breeze) (Grisso et al. 2013).
Spray Volume	L/hr	1800.0	1800 L/min, irrigation value adopted from NZ MtE (2011) Appendix 5A.
Wind speed	m/hr	9000	Based on windspeed of 2.5 m/sec
BoxVR	m^3/hr	81000.0	Ventilation rate of spray in the 'spray box'. Assumed to be 81,000 m3/hr based on a wind speed of 9000 m/hr, and a 'spray box' dimension of 3 x 3 m.

CAS	Chemical	Concentration in Water mg/L	Generation rate of chemical in volume mg/hr	Driftable Aerosol Emission Factor L/m^3
71-43-2	Benzene	0.01	2.52	2.500000E-03
007440-38-2	Arsenic	0.08	30.24	2.500000E-03
7440-39-3	Barium	110.00	39600	2.500000E-03
7440-42-8	Boron	54.50	19620	2.500000E-03
7440-48-4	Cobalt	0.02	8.64	2.500000E-03
7439-89-6	Iron	97.00	34920	2.500000E-03
7439-96-5	Manganese	3.09	1112.4	2.500000E-03
007439-97-6	Mercury	0.03	9.36	2.500000E-03
7440-02-0	Nickel	0.04	14.4	2.500000E-03
7440-24-6	Strontium	170.00	61200	2.500000E-03
91-57-6	2-methylnaphthalene	0.05	16.56	2.500000E-03
91-20-3	Naphthalene	0.04	15.48	2.500000E-03
-	TPH C10 - C14 Fraction Aromatic[A]	0.47	167.4	2.500000E-03
-	TPH C10 - C14 Fraction Aliphatic[A]	0.47	167.4	2.500000E-03
-	TPH C15+ Fraction Aromatic[A]	2.72	979.2	2.500000E-03
-	TPH C15+ Fraction Aliphatic[A]	2.72	979.2	2.500000E-03

A - No fractionation into aromatic and aliphatic components was undertaken for the identified TPH impacts. The approach adopted for the fractionation of TPH into aromatic and aliphatic components is summarised below:

- For TPH C6-C9 it is typically assumed that the total BTEX concentration reported is representative of the aromatic portion of TPH, and that the remaining TPH (i.e., total TPH C6-C9 minus total BTEX) is representative of the aliphatic portion of the TPH fraction. If the total BTEX concentration is less than the total TPH C6-C9 concentration, the TPH C6-C9 aliphatic portion has been calculated as the remaining concentration of TPH C6-C9;
- The higher TPH fractions are split conservatively, assuming a 50/50 ratio for aromatic and aliphatic components;
- TPH 15+ is the sum of the C15 - C40 concentrations

Exposure to Chemicals via Inhalation of Mist from the Evaporation Units - Recycled Flowback

Chronic Exposures

General Data/ Equations	Units	Exposure Calculations (RME) Inhalation of Mist by Workers	
Exposure Parameters			
Exposure Frequency (EF)	days/year	240	Exposure for 5 days per week minus 4 weeks holidays
Exposure Duration (ED)	years	1	Maximum duration that the flowback tank will be on-site
Exposure Time (ET)	hr/day	1	Professional judgement for irrigation exposure. Assume worker to be near tank for 1 hours every working day.
Driftable aerosol emission factor (EMF)	L/m3	2.50E-03	Calculated
Aerosol Inhalation Bioavailability (AAF)	unitless	1.0	Assume 100% bioavailability
Averaging Time - Threshold (AT)	years	1.000	USEPA 1989 and CSMS 1996
$ITF_{inh,w,shwr} = \frac{EmF \times AAF \times ET_{iw} \times EF \times ED}{365\ \frac{days}{year} \times 24\ \frac{hours}{day} \times AT}$			

Daily Intake = Concentration in Water x Intake Factor (ref: USEPA 1989)
Hazard Quotients = (Daily Intake from Water for Threshold Effects/ADI)

CAS	Chemical	Groundwater Concentration	Aerosol Inhalation Bioavailability	Driftable Aerosol Emission Factor	RfC (Background Corrected)	Threshold Intake and Risk Calculations: Adult Exposure Factor (threshold)	Adult Exposure Adjusted Air Concentration (threshold)	Hazard Index (Adult)	Non-Threshold Intake and Risk Calculations: Inhalation Unit Risk	Adult Exposure Factor (non-threshold)	Lifetime Exposure Factor (non-threshold)	Lifetime Exposure Adjusted Air Concentration (non-threshold)	Lifetime Excess Cancer Risk
		mg/L	(unitless)	(L/m^3)	(mg/m^3)	(L/m^3)	(mg/m^3)	(unitless)	$(mg/m^3)^{-1}$	(L/m^3)	(L/m^3)	(mg/m^3)	(unitless)
71-43-2	Benzene	0.01	1.00	2.50E-03	3.00E-02	6.85E-05	4.79E-07	1.60E-05	6.00E-06	9.78E-07	9.78E-07	6.85E-09	4.11E-14
007440-38-2	Arsenic	0.08	1.00	2.50E-03	1.00E-03	6.85E-05	5.75E-06	5.75E-03	-	-	-	-	-
7440-39-3	Barium	110.00	1.00	2.50E-03	7.00E-01	6.85E-05	7.53E-03	1.08E-02	-	-	-	-	-
7440-42-8	Boron	54.50	1.00	2.50E-03	7.00E-01	6.85E-05	3.73E-03	5.33E-03	-	-	-	-	-
7440-48-4	Cobalt	0.02	1.00	2.50E-03	1.00E-04	6.85E-05	1.64E-06	1.64E-02	-	-	-	-	-
7439-89-6	Iron	97.00	1.00	2.50E-03	2.45E+00	6.85E-05	6.64E-03	2.71E-03	-	-	-	-	-
7439-96-5	Manganese	3.09	1.00	2.50E-03	5.60E-01	6.85E-05	2.12E-04	3.78E-04	-	-	-	-	-
007439-97-6	Mercury	0.03	1.00	2.50E-03	2.00E-04	6.85E-05	1.78E-06	8.90E-03	-	-	-	-	-
7440-02-0	Nickel	0.04	1.00	2.50E-03	2.00E-05	6.85E-05	2.74E-06	1.37E-01	-	-	-	-	-
7440-24-6	Strontium	170.00	1.00	2.50E-03	2.10E+00	6.85E-05	1.16E-02	5.54E-03	-	-	-	-	-
91-57-6	2-methylnaphthalene	0.05	1.00	2.50E-03	1.40E-01	6.85E-05	3.15E-06	2.25E-05	-	-	-	-	-
91-20-3	Naphthalene	0.04	1.00	2.50E-03	1.00E-02	6.85E-05	2.95E-06	2.95E-04	-	-	-	-	-
-	TPH C10 - C14 Fraction Aromatic[A]	0.47	1.00	2.50E-03	2.00E-01	6.85E-05	3.18E-05	1.59E-04	-	-	-	-	-
-	TPH C10 - C14 Fraction Aliphatic[A]	0.47	1.00	2.50E-03	1.00E+00	6.85E-05	3.18E-05	3.18E-05	-	-	-	-	-
-	TPH C15+ Fraction Aromatic[A]	2.72	1.00	2.50E-03	1.05E-01	6.85E-05	1.86E-04	1.77E-03	-	-	-	-	-
-	TPH C15+ Fraction Aliphatic[A]	2.72	1.00	2.50E-03	7.00E+00	6.85E-05	1.86E-04	2.66E-05	-	-	-	-	-
							Total Risk (mixture)	**0.195**					**4.11E-14**

A - No fractionation into aromatic and aliphatic components was undertaken for the identified TPH impacts. The approach adopted for the fractionation of TPH into aromatic and aliphatic components is summarised below:
- For TPH C6-C9 it is typically assumed that the total BTEX concentration reported is representative of the aromatic portion of TPH, and that the remaining TPH (i.e., total TPH C6-C9 minus total BTEX) is representative of the aliphatic portion of the TPH fraction. If the total BTEX concentration is less than the total TPH C6-C9 concentration, the TPH C6-C9 aliphatic portion has been calculated as the remaining concentration of TPH C6-C9;
- The higher TPH fractions are split conservatively, assuming a 50/50 ratio for aromatic and aliphatic components;
- TPH 15+ is the sum of the C15 - C40 concentrations

Summary of Risk to Workers - Recycled Flowback Exposure fo Target Chemicals

Receptor/Exposure Pathway	Calculated Non-Threshold Risk 100% Mass Return	Calculated HI 100% Mass Return
Use of Stimulation Fluid in Hydraulic Fracturing		
HVFR Recipe		
Workers		
Ingestion of Chemicals via Incidental Contact with Flowback Water	1.02E-12	0.0052
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water	2.36E-13	0.05
Inhalation of mist from the evaporation units	4.11E-14	0.195
Total Risk	**1.30E-12**	**0.25**

RECYCLED FLOWBACK DATA

Client Name: Origin
Project Name: Beetaloo Chemical Risk Assessment

			BTEX							Inorganics																		
		Anionic Surfactants as MBAS	Benzene	Ethylbenzene	Toluene	Xylene (m & p)	Xylene (o)	Xylene Total	Sum of BTEX	Alkalinity (Bicarbonate) as CaCO3	Alkalinity (Total) as CaCO3	Ammonia (filtered)	Anions Total	Bicarbonate	Bromide (filtered)	Calcium (filtered)	Carbonate	Cations Total	Chloride	Electrical Conductivity (Lab)	Fluoride	Kjeldahl Nitrogen Total	Magnesium (filtered)	Methane	Nitrite + Nitrate (as N)	Nitrogen (Total)	pH (Lab)	Phosphorus
		mg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	mg/L	mg/L	mg/L	meq/L	mg/L	mg/L	mg/L	mg/L	meq/L	mg/L	µS/cm	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	pH_Units	mg/L
EQL		0.1	1	1	1	1	1	1	1	1	1	0.01	0.01		0.05	0.2		0.01	1	1	0.1	0.05	0.1	0.001	0.01	0.1	0.01	0.01
NHMRC (2011) Australian Drinking Water Guidelines			**1**	**300**	**800**			**600**													**1.5**							
WHO (2017) Drinking Water Guidelines (mg/L)												**35** (taste only)													**50**			
USEPA (2022) Regional Screening Levels																												
Field ID	**Date**																											
AMUNGEE NW-1H	**15/11/2016**																											
BET_PW001_Fe_15.3%	**11/11/2016**	0.2	3	<2	2	<2	<2	<2	5	364	364		639	444.08		1,320	0.6	599	22,400	54,400	1.1	55.1	271	4.76	<0.01	55.1	6.5	<0.05
BET_PW001_Fe_15.8%	**17/11/2016**	0.1	3	<2	2	<2	<2	<2	5	364	364		684	444.08		1,400	0.6	612	24,000	54,800	1.1	50.1	282	5.22	0.04	50.1	6.4	<0.05
BET_PW001_Fe_16.0%	**20/11/2016**	0.2	3	<2	2	<2	<2	<2	5	390	390		685	475.8		1,410	0.6	617	24,000	54,900	1.1	54.8	275	6.48	<0.01	54.8	6.44	0.3
BET-PW001	**8/09/2021**		3	3	14	60	14	75	95	140	140	34		170.8	260	1,500			21,000	59,600		37	370		<0.1		6	<0.02
BET-PW001_Fe14.1%	**30/10/2016**	0.2	3	<2	2	<2	<2	<2	5	498	498		622	607.56		1,270	0.6	640	21,700	57,300	1.2	57.3	277	3.99	0.02	57.3	6.47	0.16
BET-PW001_Fe14.5%	**2/11/2016**	<0.1	4	<2	3	<2	<2	<2	7	465	465		633	567.3		1,330	0.6	666	22,100	57,000	1.2	55.5	284	4.29	0.01	55.5	6.43	0.12
BET-PW001_Fe14.8%	**5/11/2016**	0.2	3	<2	2	<2	<2	<2	5	441	441		638	538.02		1,380	0.6	688	22,300	57,300	1.2	56.3	306	5.41	0.02	56.3	6.43	0.1
BET-PW001_Fe15.1%	**8/11/2016**	0.2	3	<2	2	<2	<2	<2	5	342	342		644	417.24		1,380	0.6	688	22,600	58,300	1.2	55.2	305	5.27	0.01	55.2	6.39	0.06
BET-PW001_Fe_9	**29/09/2016**	<0.1	4	<2	3	2	<2	2	9	474	474		408	578.28		853	0.6	464	14,100	40,600	1.2	52.2	147	1.2	0.04	52.2	6.54	0.41
BET-PW001_Fe_9.4	**5/10/2016**	<0.1	7	<2	6	2	<2	2	15	716	716		398	873.52		774	0.6	446	13,600	39,000	1.2	51.6	133	6.29	0.26	51.9	6.74	1.07
BET-PW001_Fe_10.6	**7/10/2016**	<0.1	3	<2	3	<2	<2	<2	6	540	540		443	658.8		980	0.6	503	15,300	44,100	1.1	50.6	165	5.39	0.17	50.8	6.63	0.47
BET-PW001_Fe_11.5%	**15/10/2016**	<0.1	3	<2	2	2	<2	2	7	506	506		524	617.32		1,220	0.6	610	18,200	49,000	1.1	45.1	253	5.46	0.03	45.1	6.47	0.22
BET-PW001_Fe_12.5%	**19/10/2016**	<0.1	4	<2	3	<2	<2	<2	7	472	472		540	575.84		1,360	0.6	627	18,800	51,100	1.1	48	269	5.5	0.12	48.1	6.45	0.12
BET-PW001_Fe_12.15%	**17/10/2016**	0.1	4	<2	3	<2	<2	<2	7	474	474		526	578.28		1,230	0.6	593	18,300	50,500	1.1	65.6	252	7.09	0.02	65.6	6.5	0.16
BET-PW001_Fe_13%	**22/10/2016**	<0.1	4	<2	3	<2	<2	<2	7	566	566		556	690.52		1,200	0.6	555	19,300	52,600	1.1	61.3	233	6.5	<0.01	61.3	6.51	<0.1
BET-PW001_Fe_13.5%	**25/10/2016**	<0.1	3	<2	2	<2	<2	<2	5	556	556		575	678.32		1,210	0.6	551	20,000	53,500	1.1	59.4	235	6.49	<0.01	59.4	6.55	0.15
BET-PW001_Fe_16.2	**23/12/2016**	0.1	3	<2	2	<2	<2	<2	5	377	377		628	459.94		1,600	0.6	674	22,000	51,700	1	58	272	7.35	<0.01	58	6.56	<0.05
BET-PW001_Fe_16.5%	**28/12/2016**	<0.1	2	<2	<2	<2	<2	<2	2	367	367		611	447.74		1,740	0.6	718	21,400	52,800	1.1	61.5	295	7.75	<0.01	61.5	6.5	0.1
BET-PW001_FE_16.4	**26/12/2016**	0.2	2	<2	<2	<2	<2	<2	2	371	371		684	452.62		1,650	0.6	686	24,000	52,300	1.1	60	283	8.37	<0.01	60	6.5	<0.05
BET-PW001_FE_16.6%	**30/12/2016**	0.2	2	<2	<2	<2	<2	<2	2	384	384		724	468.48		1,740	0.6	713	25,400	52,300	1.1	62.1	295	7.72	<0.01	62.1	6.5	<0.05
BET-PW001 2209 Sep	**22/09/2021**		5	10	48	180	42	230	290	460	460	27		561.2	190	1,100			15,000	37,400		32	270		<0.1		6.3	<0.02
	Maximum Concentration	0.2	7	10	48	180	42	230	290	716	716	34	724	873.52	260	1740	0.6	718	25400	59600	1.2	65.6	370	8.37	0.26	65.6	6.74	1.07

Notes:

NHMRC (2011) Australian Drinking Water Guidelines 6. Volume 1. National Water Quality Management Strategy. National Health and Medical Research Council, updated September 2022.

WHO (2017) World Health Organisation Drinking Water Guidelines and rolling revisions

USEPA (2022) Regional Screening Levels. Updated May 2022. https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables

No fractionation into aromatic and aliphatic components was undertaken for the identified TPH impacts. The approach adopted for the fractionation of TPH into aromatic and aliphatic components is summarised below:

- For TPH C6-C9 it is assumed that the total BTEX concentration reported is representative of the aromatic portion of TPH, and that the remaining TPH (i.e., total TPH C6-C9 minus total BTEX) is representative of the aliphatic portion of the TPH fraction. If the total BTEX concentration is less than the total TPH C6-C9 concentration, the TPH C6-C9 aliphatic portion has been calculated as the remaining concentration of TPH C6-C9;
- The higher TPH fractions are split conservatively, assuming a 50/50 ratio for aromatic and aliphatic components;
- TPH 15+ is the sum of the C15 - C40 concentrations

RECYCLED FLOWBACK DATA

Client Name: Origin
Project Name: Beetaloo Chemical Risk Assessment

										Metals													PAH's and Phenolics			
		Potassium (filtered)	Silicon as Si	Silicon as SiO2	Sodium (filtered)	Sulphate as SO4 (filtered)	Total Dissolved Solids (filtered)	Total Dissolved Solids (Calculated)	Total Hardness as CaCO3 (filtered)	Aluminium	Arsenic (filtered)	Barium (filtered)	Boron (filtered)	Chromium (III+VI) (filtered)	Cobalt	Iron	Manganese	Manganese (filtered)	Mercury	Nickel (filtered)	Strontium	Zinc	Zinc (filtered)	2-methylnaphthalene	3-&4-methylphenol	Naphthalene	Phenol
		mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	mg/L	µg/L	µg/L	µg/L	µg/L
EQL		0.01	0.02	0.05	0.5	1	10	1	1	0.001	0.0005	0.001	0.001	0.0005	0.0002	0.001	0.0005	0.0005	0.0001	0.0005	0.001	0.001	0.001	1	1	1	1
NHMRC (2011) Australian Drinking Water Guidelines											**0.01**	**2**	**4**				**0.5**	**0.5**	**0.001**	**0.02**							
WHO (2017) Drinking Water Guidelines (mg/L)																											
USEPA (2022) Regional Screening Levels										**20**				**22**	**0.006**	**14**					**12**	**6**	**6**	**1.1**	**370** (as 4 methylphenol)	**0.12**	**5800**
Field ID	**Date**																										
AMUNGEE NW-1H	**15/11/2016**																										
BET_PW001_Fe_15.3%	**11/11/2016**	70			11,700	<10	44,200	35,400	4,410		<0.01	60.7	34.6	0.01				2.26		0.018			<0.05		5.2	<1	2.8
BET_PW001_Fe_15.8%	**17/11/2016**	70			11,900	<10	46,600	35,600	4,660		<0.01	65.7	33.1	0.015				2.42		<0.01			<0.05		5.6	<1	2.1
BET_PW001_Fe_16.0%	**20/11/2016**	69			12,000	<10	49,200	35,700	4,650		<0.01	66.5	34.8	0.013				2.53		0.028			<0.05		10.3	<1	3.5
BET-PW001	**8/09/2021**	53	16	33	8,800	2	35,000			0.3	<0.025	110	12	<0.025	0.024	79	1.9	1.7	0.026	0.03	170	0.13	0.11	46	<1	43	2
BET-PW001_Fe14.1%	**30/10/2016**	76			12,700	<10	45,500	37,200	4,310		<0.01	68.8	45.4	0.031				2.74		0.018			<0.05		6.8	<1	2.8
BET-PW001_Fe14.5%	**2/11/2016**	80			13,200	<10	45,300	37,000	4,490		0.011	74.8	44	0.032				2.9		0.014			<0.05		4.4	<1	2
BET-PW001_Fe14.8%	**5/11/2016**	83			13,600	<10	45,600	37,200	4,700		<0.01	77.8	43.9	0.033				3.09		<0.01			<0.05		4.3	<1	2.1
BET-PW001_Fe15.1%	**8/11/2016**	83			13,600	<10	44,300	37,900	4,700		<0.01	68.5	45.4	0.031				2.4		0.012			<0.05		4.7	<1	2.6
BET-PW001_Fe_9	**29/09/2016**	55			9,370	20	33,600	26,400	2,740		0.084	35.6	50.9	0.035				1.82		0.04			<0.05		<2	<1	1.5
BET-PW001_Fe_9.4	**5/10/2016**	58			9,080	17	30,400	25,400	2,480		0.011	30.5	54.5	0.048				1.95		0.012			<0.05		<2	<1	<1
BET-PW001_Fe_10.6	**7/10/2016**	60			10,100	42	32,300	28,700	3,130		<0.01	42	49.4	0.034				1.8		0.01			<0.05		<2	<1	<1
BET-PW001_Fe_11.5%	**15/10/2016**	72			12,100	<10	38,800	31,800	4,090		<0.010	51.9	45.9	0.042				2.38		<0.010			<0.050		2.6	<1.0	2.3
BET-PW001_Fe_12.5%	**19/10/2016**	74			12,300	38	39,000	33,200	4,500		<0.010	59.1	43.5	0.033				2.43		<0.010			<0.050		2.9	<1.0	2.2
BET-PW001_Fe_12.15%	**17/10/2016**	70			11,700	26	37,400	32,800	4,110		<0.010	53.9	44.5	0.03				2.24		<0.010			<0.050		2.1	<1.0	2.2
BET-PW001_Fe_13%	**22/10/2016**	64			10,900	<1	37,700	34,200	3,960		<0.01	63.5	40	0.032				2.34		0.014			<0.05		2.7	<1	1.6
BET-PW001_Fe_13.5%	**25/10/2016**	65			10,800	<1	31,800	34,800	3,990		<0.01	68.4	41.1	0.03				2.44		0.018			<0.05		7	<1	1.7
BET-PW001_Fe_16.2	**23/12/2016**	68			13,100	<10	42,000	33,600	5,120		<0.010	66.2	35.1	0.025				2.31		0.021			<0.050		2.5	<1.0	2.8
BET-PW001_Fe_16.5%	**28/12/2016**	70			13,900	<10	44,800	34,300	5,560		<0.010	77.8	31	0.031				2.64		<0.010			<0.050		5.8	<1.0	3.1
BET-PW001_FE_16.4	**26/12/2016**	67			13,300	<10	44,200	34,000	5,280		<0.010	71.6	34.2	0.028				2.56		0.017			<0.050		3.3	<1.0	2.5
BET-PW001_FE_16.6%	**30/12/2016**	70			13,800	<10	44,500	34,000	5,560		<0.010	80.1	34	0.029				2.75		<0.010			<0.050		11.3	<1.0	4
BET-PW001 2209 Sep	**22/09/2021**	39	7.3	16	6,200	1	29,000			<0.05	<0.005	90	8.2	<0.005	0.0032	97	1.9	1.8	0.0025	<0.005	150	0.097	0.07	42	<1	38	2
	Maximum Concentration	83	16	33	13900	42	49200	37900	5560	0.3	0.084	110	54.5	0.048	0.024	97	1.9	3.09	0.026	0.04	170	0.13	0.11	46	11.3	43	4

Notes:

NHMRC (2011) Australian Drinking Water Guidelines 6. Volume 1. National Water Quality Management Strategy. National Health and Medical Research Council, updated September 2022.

WHO (2017) World Health Organisation Drinking Water Guidelines and rolling revisions

USEPA (2022) Regional Screening Levels. Updated May 2022. https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables

No fractionation into aromatic and aliphatic components was undertaken for the identified TPH impacts. The approach adopted for the fractionation of TPH into aromatic and aliphatic components is summarised below:

- For TPH C6-C9 it is assumed that the total BTEX concentration reported is representative of the aromatic portion of TPH, and that the remaining TPH (i.e., total TPH C6-C9 minus total BTEX) is representative of the aliphatic portion of the TPH fraction. If the total BTEX concentration is less than the total TPH C6-C9 concentration, the TPH C6-C9 aliphatic portion has been calculated as the remaining concentration of TPH C6-C9;
- The higher TPH fractions are split conservatively, assuming a 50/50 ratio for aromatic and aliphatic components;
- TPH 15+ is the sum of the C15 - C40 concentrations

RECYCLED FLOWBACK DATA

Client Name: Origin
Project Name: Beetaloo Chemical Risk Assessment

		Radionuclides		TPH											
		Gross alpha activity_	Gross beta activity (excluding activity of K-40)	C6 - C9 Fraction	C6 - C10 Fraction	C6 - C10 Fraction minus BTEX (F1)	C10 - C14 Fraction	C10 - C16 Fraction	C10 - C16 Fraction minus Naphthalene (F2)	C15 - C28 Fraction	C16 - C34 Fraction	C29 - C36 Fraction	C34 - C40 Fraction	C10 - C36 Fraction (Sum)	C10 - C40 Fraction (Sum)
		Bq/L	Bq/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L	µg/L
EQL		0	0	10	10	10	10	10	10	50	50	50	50	50	50
NHMRC (2011) Australian Drinking Water Guidelines		**0.5**	**0.5**												
WHO (2017) Drinking Water Guidelines (mg/L)				**15000 aliphatic**			**100 aromatic 300 aliphatic**			**90 aromatic 300 alipatic**		**90 aromatic 300 alipatic**	**90 aromatic 300 alipatic**		
USEPA (2022) Regional Screening Levels															
Field ID	**Date**														
AMUNGEE NW-1H	**15/11/2016**	9.22	5.22												
BET_PW001_Fe_15.3%	**11/11/2016**	11	5.32	50	40	40	<50	<100	<100	1,080	1,020	<50	<100	1,080	1,020
BET_PW001_Fe_15.8%	**17/11/2016**	10.2	5.08	100	90	80	60	<100	<100	410	410	<50	<100	470	410
BET_PW001_Fe_16.0%	**20/11/2016**	9.3	4.8	110	90	80	80	<100	<100	200	220	<50	<100	280	220
BET-PW001	**8/09/2021**	12	8.8	220	260	170	380	420	400	320	160	<50	<50	700	580
BET-PW001_Fe14.1%	**30/10/2016**	3.06	17.2	80	80	80	70	<100	<100	610	620	<50	<100	680	620
BET-PW001_Fe14.5%	**2/11/2016**	2.86	17.8	130	130	120	120	100	100	130	<100	<50	<100	250	100
BET-PW001_Fe14.8%	**5/11/2016**	5.13	18.3	60	50	40	<50	<100	<100	530	490	<50	<100	530	490
BET-PW001_Fe15.1%	**8/11/2016**	5.08	15.9	60	60	60	130	160	160	1,180	1,160	<50	<100	1,310	1,320
BET-PW001_Fe_9	**29/09/2016**	<0.62	<1.25	50	60	50	110	120	120	430	490	120	<100	660	610
BET-PW001_Fe_9.4	**5/10/2016**			100	100	80	90	130	130	3,070	4,160	1,720	650	4,880	4,940
BET-PW001_Fe_10.6	**7/10/2016**	2.43	5.99	50	50	40	180	190	190	240	260	60	<100	480	450
BET-PW001_Fe_11.5%	**15/10/2016**	8.82	15.4	60	60	50	110	<100	<100	470	600	200	<100	780	600
BET-PW001_Fe_12.5%	**19/10/2016**	8.38	8.31	80	80	70	240	120	120	100	110	<50	<100	340	230
BET-PW001_Fe_12.15%	**17/10/2016**	6.31	7.55	80	80	70	160	<100	<100	<100	110	<50	<100	160	110
BET-PW001_Fe_13%	**22/10/2016**	8.57	9.76	90	90	80	270	240	240	170	210	<50	<100	440	450
BET-PW001_Fe_13.5%	**25/10/2016**	12.4	12.7	80	80	80	190	140	140	180	280	150	130	520	550
BET-PW001_Fe_16.2	**23/12/2016**			110	110	100	<50	<100	<100	490	570	120	<100	610	570
BET-PW001_Fe_16.5%	**28/12/2016**			200	200	200	<50	<100	<100	450	470	70	<100	520	470
BET-PW001_FE_16.4	**26/12/2016**			130	130	130	<50	<100	<100	470	440	<50	<100	470	440
BET-PW001_FE_16.6%	**30/12/2016**			70	70	70	<50	<100	<100	610	640	90	<100	700	640
BET-PW001 2209 Sep	**22/09/2021**	7	5.2	310	390	100	930	630	550	<50	<50	<50	<50	930	630
	Maximum Concentration	12.4	18.3	310	390	200	930	630	550	3070	4160	1720	650	4880	4940

Notes:

NHMRC (2011) Australian Drinking Water Guidelines 6. Volume 1. National Water Quality Management Strategy. National Health and Medical Research Council, updated September 2022.

WHO (2017) World Health Organisation Drinking Water Guidelines and rolling revisions

USEPA (2022) Regional Screening Levels. Updated May 2022. https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables

No fractionation into aromatic and aliphatic components was undertaken for the identified TPH impacts. The approach adopted for the fractionation of TPH into aromatic and aliphatic components is summarised below:

- For TPH C6-C9 it is assumed that the total BTEX concentration reported is representative of the aromatic portion of TPH, and that the remaining TPH (i.e., total TPH C6-C9 minus total BTEX) is representative of the aliphatic portion of the TPH fraction. If the total BTEX concentration is less than the total TPH C6-C9 concentration, the TPH C6-C9 aliphatic portion has been calculated as the remaining concentration of TPH C6-C9;
- The higher TPH fractions are split conservatively, assuming a 50/50 ratio for aromatic and aliphatic components;
- TPH 15+ is the sum of the C15 - C40 concentrations

Appendix E Spill Management Plan

THE BEETALOO EXPLORATION PROJECT

Spill Management Plan

Review record

Rev	Date	Reason for issue	Authors	Consolidator	Approver
2.0	29/06/2022	Minor update to include delineation EMP	LP		MK
2.1	6/10/2022	Edits to Table 2 and Appendix A	LP		MK

Review due: 18/05/2023

Spill Management Plan

NT-2050-15-027

Table of contents

List of Figures

List of Tables

Review due: 18/05/2023

Appendices

Appendix A: Chemical volumes per well and storage areas

1. Purpose

This Spill Management Plan (SMP) has been prepared to support Origin's Beetaloo exploration program. The SMP is a mandatory requirement prepared in accordance with the Code of Practice for Petroleum Activities in the Northern Territory (the Code of Practice). This SMP is designed to provide the strategy for the management of spills across Origin's Beetaloo exploration activities.

The Environmental Management Plans (EMPs) covered by this plan are:

- NT-2050-15-MP-025 Origin Energy Beetaloo Kyalla 117 N2 Drilling, Stimulation and Well Testing EMP
- NT-2050-15-MP-032 Origin Energy Beetaloo Velkerri 76 S2 Drilling, Stimulation and Well Testing EMP
- NT-2050-15-MP-038 Origin Beetaloo Sub-Basin Kyalla 117 N2 Multiwell Drilling, Stimulation and Well Testing EMP
- CDN/ID NT-2050-35-PH-0018 Origin Beetaloo Sub-Basin Amungee NW-1H EMP
- NT-2050-15-MP-039 Beetaloo W-1 EMP
- NT-2050-MP-040 Kalala S1 EMP
- NT-2050-15-MP-041 Beetaloo Sub-Basin Multi-well Drilling, Stimulation and Well Testing EMP
- NT-2050-15-MP-0088 Amungee NW Delineation Program EMP

This plan will reference the relevant sections within each of the various EMPs to avoid duplication. This plan should be read in conjunction with the chemical risk assessment and operation risk assessment appended to each EMP, in accordance with section 3.4 of the Code.

2. Key legislation

Key legislation and documents consulted in the development of this plan are provided below (a full list of applicable legislation is provided in the corresponding management plans):

- **Code of Practice: Onshore Petroleum Activities in the Northern Territory:** Mandatory code of practice legislating the management of chemicals and wastewater onsite, including the use of secondary containment, lined tanks and spill management plan,
- ***Transport of Dangerous Goods by Road and Rail (National Uniform Legislation) Act 2010:*** Covers the transportation of goods by road in the NT. This also covers licences for vehicles and drivers carrying dangerous goods.
- ***Workplace Health and Safety (National Uniform Legislation) Act 2011:*** Covers the storage and handling of chemicals on site.
- ***Waste Management and Pollution Control Act 1998:*** Covers the requirements for the transportation and disposal of waste within the NT. This includes the requirements for contractors, vehicles and facilities managing listed wastes to be licenced.

3. Chemicals and wastewater description

The chemicals and wastewater typically stored onsite includes:

- Chemicals used for drilling
- Waste drilling fluids

- Chemicals used for stimulation
- Flowback wastewater
- Completions and well suspension fluids
- General use chemicals such as condensate and oil, diesel and fuels, general equipment maintenance chemicals (hydraulic oils, degreasers etc.)

The full list of chemicals and wastewater stored onsite, including their volume and location are provided in Appendix A. For chemicals and maximum volumes for other EMPs refer to Table 1. Where available, links are provided to the relevant sections and appendices.

The assessment of chemicals, including evaluation of the environmental hazard of the chemical additives in the hydraulic fracturing fluid systems, based on their environmental persistence, bioaccumulation and aquatic toxicity properties; evaluation of human health effects; and exposure assessment is provided in the relevant hydraulic fracturing chemical risk assessment.

Table 1: Types of chemicals and wastewater relevant to each EMP

EMP	Drilling chemicals and waste fluids	Stimulation chemicals	Flowback wastewater	Completion and well suspension fluids	General use
NT-2050-15-MP-025 Kyalla 117 N2 EMP: • Chemical RA Appendix C • Risk assessment Appendix J	x	x	x	x	x
NT-2050-15-MP-032 Velkerri 76 S2 EMP: • Chemical RA Appendix C • Risk assessment Appendix N	x	x	x	x	x
NT-2050-15-MP-038 Kyalla 117 N2 Multiwell EMP (ORI6): • Chemical RA Appendix C • Risk Assessment Appendix K	x	x	x	x	x
CDN/ID NT-2050-35-PH-0018 Amungee NW-1H (ORI7): • Section 2.1.1, Table 2, Table 3	N/A	N/A	x	x	x
NT-2050-15-MP-039 Beetaloo W-1 EMP (ORI8): • Section 3.9, Table 8	N/A	N/A	N/A	Incidental volumes may be generated	x
NT-2050-MP-040 Kalala S1 EMP (ORI9): • Section 3.8, Table 8	N/A	N/A	N/A	Incidental volumes may be generated	x
NT-2050-15-MP-041 Beetaloo Sub-basin Multi-well EMP (ORI10): • Chemical RA Appendix E	x	x	x	x	x

EMP	Drilling chemicals and waste fluids	Stimulation chemicals	Flowback wastewater	Completion and well suspension fluids	General use
• Risk assessment Appendix M					
NT-2050-15-MP-0088 Amungee NW Delineation Program EMP: • Section 3.13.2 and Section 3.15 • Chemical RA Appendix D • Risk assessment Appendix L	x	x	x	x	x

4. Spill failure scenarios

Potential spill scenarios associated with exploration activities are summarised in Table 2. These scenarios include:

- Spills from chemical and wastewater handling and storage activities onsite
- Spills from chemical and wastewater during transportation (offsite)
- Tank, drilling sump and containment vessel overflows and structural failures

The loss of containment due to the failure of well barriers is covered under the Well Operations Management Plan (WOMP).

Table 2: Spill scenario summary table

Spill scenario	Activity duration	Mechanisms	Location	Quality	Quantity	Key management controls	Monitoring	Receptors	Effectiveness of controls
Spills from chemical and wastewater handling and storage activities onsite	• Drilling–45 days • Stimulation–15-30 days • Well testing 30– 180 days	Container rupture Spill during chemical storage, handling and mixing Contaminants in water and soil pass through the food chain and bioaccumulate	• Chemical storage area •Drilling rig • Stimulation spread • Drilling sumps • Flowback storage tanks • Well testing equipment	Potentially hazardous fluids such as: •Saline and synthetic based mud (SBM) drilling fluids •Saline flowback • Chemicals listed in EMP **NB:** All added chemicals have been assessed and verified to not be toxic and persistent and bio-accumulative (see EMP Appendix D).	<1,000L <1,000L <200L	• Designated storage areas with appropriate segregation of incompatible chemicals • Secondary containment to be deployed under high-risk spill/leak storage and handling areas • Spill kits available • Routine inspection of chemical stores • Sites are manned during operations, with continuous leak detection and level monitoring at all other times • Wastewater management plan	Routine inspection of chemical stores, sumps and tanks during operations Tank leak detection	Retained on-site	High – use of secondary containment reduces the probability of a spill. High – controls managing the storage of chemicals and wastes are mature with secondary containment measures limiting potential pathways to receptors. The scientific certainty around the effectiveness of secondary containment in preventing groundwater contamination is high and mature.
Loss of containment during transfer onsite (leakage from pipes, hoses, fittings etc)	• Drilling–45 days • Stimulation– 15-30 days • Well testing 30– 180 days	Coupling, valve, hosing and equipment failure Contaminants in water and soil pass through the food chain and bioaccumulate	Chemical mixing and transfer areas on the drill rig, mixing hoppers and wastewater storages	Potentially hazardous fluids such as: •Saline and SBM drilling fluids and wastewater. • Chemicals listed in EMP Appendix D. **NB:** All added chemicals have been assessed and verified to not be toxic and persistent and bio-accumulative (see EMP Appendix D).	<5,000L	• Secondary containment to be deployed under high-risk spill/leak storage and handling areas • Spill kits available • Routine inspection of chemical stores • Sites are manned during operations, with continuous leak detection and level monitoring at all other times • Wastewater management plan	Routine inspection of all chemical handling areas, including wastewater transfer points and chemical mixing areas	Retained on-site	High – use of secondary containment reduces the probability of a spill High – controls managing the storage of various fluids are in accordance with the requirements of the Code, which limit potential pathways to receptors. The scientific certainty around the effectiveness of secondary containment and transfer in preventing groundwater contamination is high and mature.
Spills from chemical and wastewater during transportation (off-site)	• Drilling chemical transfer—1–5 days of bulk chemical transfer generally pre-drilling • Stimulation chemical transfer 2–3 truckloads	Transport spill Traffic accident (total or partial release) Contaminants in water and soil pass through	Off-site along highway	Potentially hazardous fluids such as: •Combustible fluids (e.g. diesel)	<1,000L for transport spill <50,000L	• All transport companies to be appropriately licenced to transport chemicals and waste *(Dangerous goods and Waste Management and Pollution Control Act)*	Performance of contractors to be monitored as a part of transportation contractors	• Chemical transport between Darwin/South Australia and Queensland/	High – use of secondary containment reduces the probability of a spill High – controls managing the storage of various fluids following code measures

Spill scenario	Activity duration	Mechanisms	Location	Quality	Quantity	Key management controls	Monitoring	Receptors	Effectiveness of controls
	of chemicals per week for ~6 weeks • Wastewater disposal over 3 weeks—up to ~22 truck movements total over the duration	the food chain and bioaccumulate		•Various chemicals as listed in EMP Appendix D. • Saline wastewater **NB:** All added chemicals have been assessed and verified to not be toxic and persistent and bio-accumulative (see EMP Appendix D).	for total loss of B- triple carrying flowback	including the requirement to detect and respond to spills • Wastewater management plan		and Daly Waters • Wastewater transportation between Daly Waters and Queensland Via Tennant Creek	limiting potential pathways to receptors •The scientific certainty around the transportation of chemicals and wastes is high and mature, and well understood across Australia, limiting exposure to personnel, the public and surrounding receptors.
Tank, drilling sump and containment vessel overflows and structural failures	• Duration of all activities plus ongoing wastewater storage which may be extended beyond 12-months to allow for ongoing evaporation of fluids	Overfilling of a sump and flowback tank Structural failure of embankment or tank wall Contaminants in water and soil pass through the food chain and bioaccumulate	Sumps and tanks on lease	Potentially hazardous fluids such as: •Saline wastewater with TDS >50,000 mg/L **NB:** All added chemicals have been assessed and verified to not be toxic and persistent and bio-accumulative (see EMP Appendix D).	>10,000L	• Lease pads bunded during the storage of flowback • Enclosed tanks used during wet seasons operations •Open tanks with 1:1000ARI freeboard • Tanks constructed to Australian Standards Routine tank and sump inspections	Routine tank and sump level and structural integrity (visual) inspections	Retained on lease pad within bund	High – controls managing the storage of various fluids are in accordance with the requirements of the Code, which limit potential pathways to receptors. The scientific certainty around the effectiveness of conservative freeboard as a mitigation is high and mature.

5. Potential receptors

The location of Origin's Beetaloo exploration activities is remote. A description of the environment, including environmental and cultural sensitivities, with the potential to be impacted by a spill is provided in each of the EMPs. Figure 1 illustrates the separation distance from sensitive receptors such as:

- Watercourses
- Communities
- Homesteads
- Heritage places
- Vegetation communities
- Protected areas

Maps regarding sacred sites and restricted work areas are also applicable and will be provided to work crews to ensure awareness of these features.

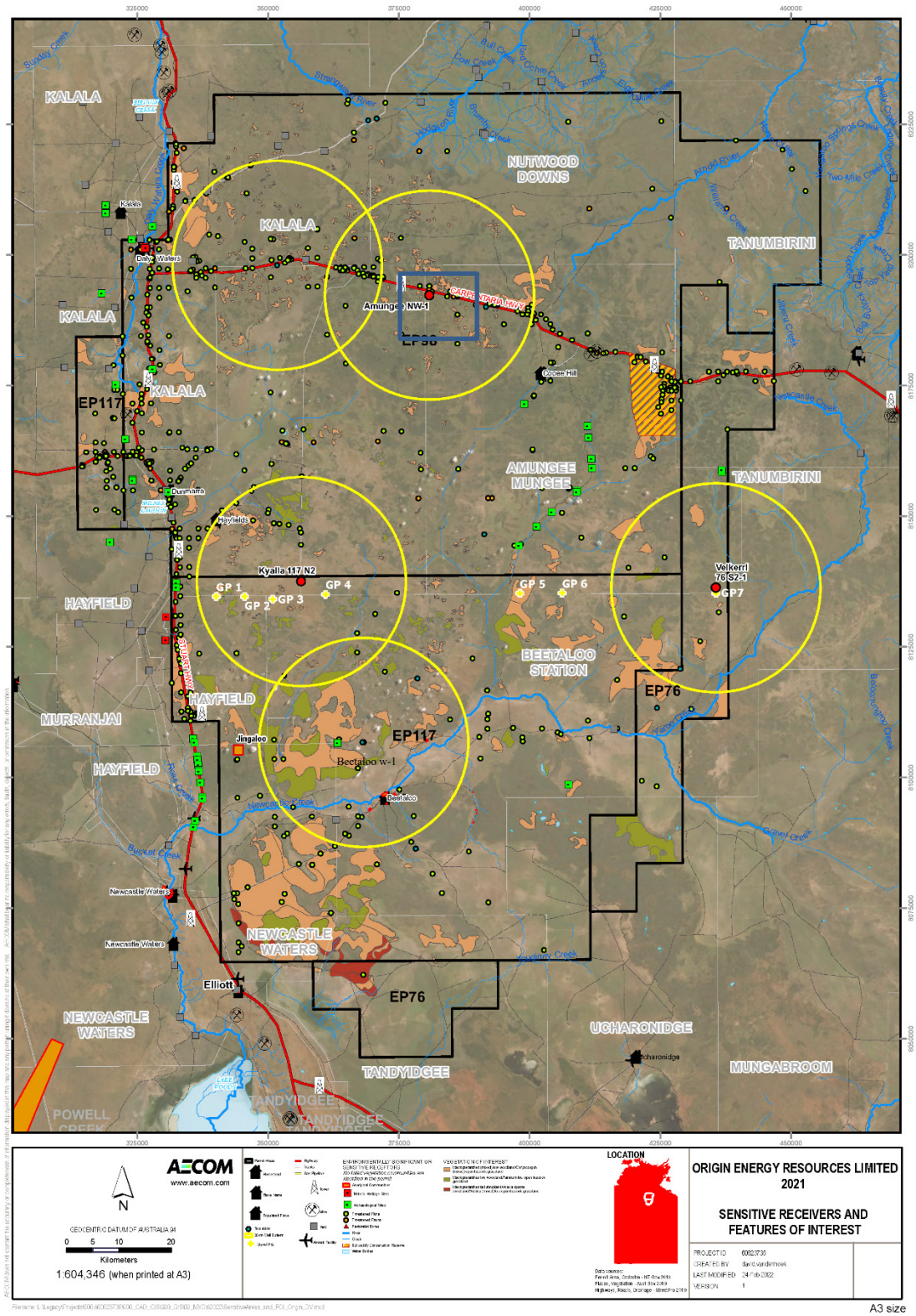


Figure 1: Location of activities and potential receptors

Review due: 18/05/2023

6. Risk assessment

The risk of spills associated with all drilling, stimulation and well testing activities is covered under the EMPs.

7. Control measures

Controls measures to manage spills associated with exploration activities are provided in the EMPs and summarised in Table 2. The key management controls include:

- Contractors are required to develop spill management procedures to comply with the requirements of this plan
- All flowback, completion fluids, chemicals, oil and fuel storage will be equipped with secondary containment (or dual liners), as per the codes of practice
- Drilling will be lined, with enough freeboard to manage a 1:1000ARI wet season (~1300mm)
- Flare pits will be designed to manage a 1:1000ARI 24-hour storm event (377mm)
- Tanks will be designed, installed and operated as per the manufacturer's specifications and COP
- Where flowback is being stored on a lease pad, the wastewater tanks shall be earthen bunded to prevent release to surrounding areas in the case of a catastrophic failure.
- The earthen bund shall be designed to hold 110% of the volume of the largest wastewater tank onsite
- The earthen bund shall be constructed to withstand a failure event, with the bund appropriately compacted and stabilised
- Well sites are designed and constructed to prevent spills of hazardous chemicals; this includes
  - compacting the lease pad surface to 100kpa prevent infiltration
  - provision of bunded (lined) chemical segregation areas
- Monitoring to detect spills will be undertaken in accordance with Section 9
- Procedures will be developed by contractors designed to detect, remediate and report any spills. This includes:
  - Chemical handling procedures
  - Chemical storage and handling inspection procedures
  - Spill prevention, detection and response procedures
- The transport of hydraulic fracturing chemicals and wastewater during the wet season will be avoided, unless a site-specific risk assessment indicates the risk is equal to or below a moderate
- Effective spill clean-up material readily available at each work site and on all mobile service trucks or vehicles, where hydrocarbons and chemicals are stored and/or used
- Inspection reports and maintenance records of secondary containment shall be kept and available for review upon request
- Spill response mock-up drills to be completed as a part of routine emergency response.

8. Spill response and management

The following section provides an overview of the response to spills during drilling, stimulation and well testing activities. Where the spill is the result of an emergency situation that is still active, the Beetaloo Exploration Emergency Response Plan (NT-2050-15-MP-024) will take precedence over this plan.

8.1 Rapid spill assessment

When a spill occurs, the on-site Supervisor will carry out a rapid assessment to determine the potential hazards and the type and location of emergency assistance required. This assessment shall include the following:

- Determine the physical (volume and state) and location of the spill
- Determine the appropriate spill category and type of response as per section 12.1.
- Assess the hazard of the material spilled, including any potential hazards associated with chemical mixing (such as oxidising and reducing agents)
- Determine the safety hazard to immediate response personnel and whether additional resources (such as emergency services or specialised equipment or advice) are required to manage the spill safely
- Determine spill movement, factors affecting the movement (i.e. impending weather, topography, drainage lines, etc) and spill response priorities, as per Table 3.

Table 3: Spill response priorities

Spill priority	Response considerations
People and communities	• Evacuate and muster (if deemed necessary) • Account for all people and determine missing persons • Stop unauthorised access • Provide a technical resource to the Emergency Services (if required) • Protect community and pastoralists
Environment and sacred sites	• For emergencies that are safeto manage, onsite personnel will respond with available resources to limit the extent of the impact to the environment or a protected site • For larger incidents, or where it is unsafe for onsite personnel to respond, trained people will be mobilised to control and contain the emergency to minimise the impact to the environment or protected site
Regulators	• Notify Regulators as per incident reporting requirements
Assets	• Monitor automatic shutdown of the equipment or part thereof, or initiate manual shutdowns where it is safe to do so • Mobilise emergency services to intervene
Reputation	• Notify neighbours (if required)

8.2 Spill containment and clean up procedures

Generic spill containment clean-up procedures must be developed and implemented by each drilling, stimulation and well testing contractor aligning with the requirements of this plan. These procedures shall be adapted (where appropriate) to consider the site and chemical specific hazards associated with each spill event.

The procedures shall consider the following generic spill containment and response procedure:

- Move all people out of harm's way
- Alert others nearby
- Assess the situation—determine what substances are involved, the potential receptors (people and the environment) and if additional support is required. The substance must be known prior to taking any action (refer to SDS)
- If applicable; remove any possible risk escalating factors (e.g. ignition hazards in case of flammable/combustible spills); approach from up-wind to reduce fume risks, isolate the spill source (close containment valve, similar). Ensure appropriate controls requirements are met, e.g. PPE, first aid support, etc., prior to conducting spill clean up
- If it is safe to do so; stop the source of the leak (if possible) and contain the spill using onsite equipment to
- Prevent from leaving site or entering a waterway or sensitive feature
- Recover free liquid and contaminated material as soon as practicable (i.e. immediately) to mitigate infiltration. Material recovery should consider the benefit of recovery versus the additional impact that recovery of all contaminated material could cause as per the National Environment Protection (Assessment of Site Contamination) Measure
- Prevent people, livestock and wildlife access to hazardous material through fencing or other barriers
- Store contaminated material in a manner to minimise the risk of additional contamination
- For Level 2 spills and higher, the Project Manager shall be notified as soon as it is safe to do so, but within 2-hours
- Project Manager to ensure appropriate external incident reporting requirements are actioned in accordance with the impact of the spill
- For Level 2 spills and higher, Origin Project Manager to seek expertise as to whether additional testing and remediation is required upon completion of the initial containment and clean up. This consideration will be undertaken in in accordance with the National Environment Protection (Assessment of Site Contamination) Measure
- Upon rectification of a reportable spill, an incident investigation shall be completed as per the Petroleum (Environment) Regulations. This shall include the root cause of the incident, actions taken to mitigate the impact and ongoing monitoring and maintenance required to ensure the site is stable and non-polluting.

8.3 Contaminated material disposal

Contaminated material disposal will be undertaken in consideration of the following:

- During a spill clean-up, the storage of contaminated material must be undertaken in a manner that minimises additional contamination
- Offsite disposal must be undertaken in accordance with the NT *Waste Management and Pollution Control Act 1998*

- All listed waste transportation shall be undertaken by licenced contractors, be tracked and disposed of at licenced waste management facilities.

9. Monitoring and inspections

The monitoring and inspection programs to identify spills is summarised in Table 4.

Table 4: Spill monitoring and inspections

Monitoring Program	Frequency	Methodology	Purpose	Minimum volume of leak
Tank and sump level monitoring (when wastewater is stored on-site)	During operations: Daily All other times: • Weekly during the dry season • Daily during the wet season	Instrument Or Level dip/ visual assessment	Prevent the overtopping of tanks	10's of litres
Tank leak detection (when wastewater is stored on-site)	Continuous	Instrument	Detect the migration of fluid through primary containment	10's of litres
Chemical storage areas (when chemical stored On-site)	During operations: Daily All other times: Weekly	Visual (a camera may be used where sites are unmanned)	Detection of leaks	Litres
Tank structural integrity (when wastewater is stored onsite)	Weekly	Visual inspection	Detect potential structural weakness	N/A

10. Roles and responsibilities

The critical roles and responsibilities set out in Table 5 are for the main members of the Spill Response Group. This team represents the core group of resources that will lead a spill response with the support of the broader Origin Energy team.

Table 5: Roles and responsibilities

Position	Role and responsibility
Project Manager	Ultimately accountable for the implementation of the spill management plan (SMP). Role, or delegate, will liaise with Origin Environment Specialists to determine remediation requirements and external reporting obligations.
On-site Supervisor	Responsible for the initial response to a spill. This role will be delegated to the well site representative or nominated contractor in charge of a work program. Role will undertake the initial spill assessment, engage emergency services (if required) and co-ordinate immediate spill clean-up operations associations to minimise the potential impacts to people, places and the environment.
Environment/HSE Lead	Report Spill to Regulatory Authorities. Provide expertise on clean up requirements and ongoing monitoring and management requirements. Interface with government and regulatory bodies for communication and consents.
Emergency Response Lead	Provide specialist technical advice (Emergency Response) to support spill management activities.

11. Waste transportation and disposal

All contractors engaged to perform drilling, stimulation and well testing will be required to comply with this plan. A bridging SPMP will be developed by each contractor summarising the activities to be undertaken to comply with this plan and the CoP.

12. Spill reporting

12.1 Spill rating

Table 6 provides a summary of the spill classification based upon the volume and location of spill. The hazards of the potential spill to people and the environment should be assessed independently, to ensure incident specific hazards are considered in the spill response. This table provides guidance as to the likely spill scenarios that may trigger the different incident reporting requirements.

When classifying spills and determining the reporting requirements, Ministerial conditions and environmental performance objectives and criteria should also be considered when determining the whether the event is a recordable or reportable event.

The spill tiers include:

- **Level 1:** Spills that can be contained within the well site and can be cleaned up by the operator without involvement of external organisations. Most Tier 1 spills are likely to be less than 2,500L and would include diesel spills during fuel transfer, oil spillage during

routine maintenance or small wastewater spills during well testing. Clean up time is generally less than 1-day. These spills will most likely be classified as recordable incidents as per Section 12.

- **Level 2:** Spills that have not been completely contained within the site boundary and/or may require additional resources to clean up. Clean up time is generally less than a week. Level 2 spills are typically reportable incidents as defined in section 12 and may also require notification under the *Waste Management and Pollution Control Act.*
- **Level 3:** Severe spills that cannot be contained by the operator and requires substantial additional resources to manage the spill. Clean up time is generally greater than a week. Level 3 spills are reportable incidents.

Table 6: Spill tier levels

		Spill (L)		
		20-200L	200-2,500 L	>2,500 L
Receiving environment	Bund or contained impervious area	**Not reportable***	**Level 1**	**Level 1**
	Onsite *(lease pad, camp pad, hardstand, road or work area)* compacted or sealed surface**	**Not reportable***	**Level 1**	**Level 2**
	Offsite permeable surfaces- areas adjacent to lease pads, camp pads, roads where spills have moved beyond the approved activity area. **	**Level 1**	**Level 2**	**Level 3**
	Sensitive environmental or cultural feature (such as a waterway, drainage lines, wetland, high valued habitat and sacred site) or where the spill has, or has the potential to, cause material or serious environmental harm **	**Level 2**	**Level 2**	**Level 3**

Notes: * *Non-reportable spills must be recorded in Origin's OCIS (and made available for review by Contractor), with monthly reviews. For certain substances, such as flowback, there may be site specific requirements outlined int eh approval notice. The approvals notice should be reviewed. ** spills of Dangerous goods or wastes offsite may need to be reported under NT Dangerous Goods Act or Waste Management and Pollution control Act 1998.*

12.2 Incident reporting

Incidents may require reporting under the *Petroleum (Environment) Regulations and Waste Management Pollution Control Act.*

12.2.1 *Petroleum (Environment) Act* incident reporting

12.2.1.1 Reportable environmental incident reporting

The Petroleum (Environment) Regulations define a reportable incident as an incident arising from a regulated activity that has caused, or has the potential to cause, material environmental harm or serious environmental harm as defined under the *Petroleum Act.*

An interest holder must notify (this may be oral or in writing) DEPWS of a reportable incident as soon as practicable but no later than two-hours after the first occurrence of the incident or after the time the interest holder becomes aware of the incident.

DEPWS can be notified through the DEPWS Onshore gas non-compliance hotline on 1800 413 567.

Any verbal report to DEPWS must be followed up by a written report from the Project Manager within three days in accordance with the *Petroleum (Environment) Regulations.*

12.2.1.2 Recordable incidents

The *Petroleum (Environment) Regulations* define a recordable incident as an incident arising from a regulated activity that:

I. Has resulted in an environmental impact or environmental risk not specified in the current plan for the activity; or
II. Has resulted in a contravention of an environmental performance standard specified in the current plan for the activity; or
III. Is inconsistent with an environmental outcome specified in the current plan for the activity; and
IV. Is not a reportable incident.

These types of spills are typically a Level 1 type spill as defined in Table 7.

An interest holder must notify (this may be oral or in writing) DEPWS of a recordable incident as soon as practicable but no later than 15-days after the reporting period (agreed period or each 90-day period after the day on which the EMP is approved).

12.2.2 *Waste Management and Pollution Control Act 1998* incident reporting

In accordance with the Waste Management and Pollution Control Act, where contaminants or waste is not confined within the land on which the petroleum activities are undertaken (i.e. the approved disturbance areas where the petroleum activity is occurring), Origin will notify the EPA of any incident causing or threatening to cause pollution as soon as practicable, but no less than 24 hours after becoming aware of the incident.

A notifiable incident is defined as an incident that causes, or is threatening or may threaten to cause, pollution resulting in material environmental harm or serious environmental harm.

A notification must include:

a) the incident causing or threatening to cause pollution;
b) the place where the incident occurred;
c) the date and time of the incident;
d) how the pollution has occurred, is occurring or may occur;
e) the attempts made to prevent, reduce, control, rectify or clean up the pollution or resultant environmental harm caused or threatening to be caused by the incident; and
f) the identity of the person notifying.

The notification shall be made to the NT EPA Pollution Hotline 1800 064 567.

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

NOTE: In accordance with the Code, a chemical risk assessment has been completed on all listed chemicals, which have been verified to not be toxic and persistent and bioaccumulative.

Material name	Typical volume	Maximum volume	Unit	Storage area	Hazardous (Y/N)
Acetic acid – 60%	3,000	9,000	L	Stimulation chemical storage area	No
BE-9 Biocide	17,000	51,000	L	Stimulation chemical storage area	Yes
Caustic Soda Liquid	15,000	45,000	L	Stimulation chemical storage area	No
DCA-11001 Breaker activator	5,000	15,000	L	Stimulation chemical storage area	Yes
DCA-13002 Breaker	300	900	kg	Stimulation chemical storage area	Yes
DCA-13003 Breaker	10,000	30,000	L	Stimulation chemical storage area	Yes
DCA-16001 Clay Stabiliser	42,000	126,000	L	Stimulation chemical storage area	No
DCA-17001 Corrosion inhibiter	1,000	3,000	L	Stimulation chemical storage area	Yes
DCA-19001 Crosslinker	600	1,800	kg	Stimulation chemical storage area	Yes
DCA-19002 Crosslinker	10,000	30,000	L	Stimulation chemical storage area	Yes
DCA-23001 Friction reducer	5,000	15,000	kg	Stimulation chemical storage area	No
DCA-23003 Friction reducer	18,000	54,000	L	Stimulation chemical storage area	No
DCA-25005 Gelling agent	35,000	105,000	kg	Stimulation chemical storage area	No
DCA-30001 Scale inhibitor	15,000	45,000	L	Stimulation chemical storage area	No
DCA-32002 Surfactant	15,000	45,000	L	Stimulation chemical storage area	Yes
DCA-32014 Surfactant	200	600	L	Stimulation chemical storage area	Yes
FE-2 Buffer	200	600	kg	Stimulation chemical storage area	No
Hydrochloric acid – 32%	50,000	150,000	L	Stimulation chemical storage area	Yes
100 mesh sand	91,000	273,000	kg	Stimulation chemical storage area	No
4070 sand	1,650,000	4,950,000	kg	Stimulation chemical storage area	No
30/50 sand	610,000	1,830,000	kg	Stimulation chemical storage area	No

Material name	Typical volume	Maximum volume	Unit	Storage area	Hazardous (Y/N)
Sodium chloride	15,000	45,000	kg	Completion chemical storage area	No
ALDACIDE G	500	1,500	L	Completion chemical storage area	Yes
OXYGON	100	300	kg	Completion chemical storage area	No
BARACOR 100	2,000	6,000	L	Completion chemical storage area	Yes
CON-DET	50	150	kg	Drilling chemical storage area	No
SAPP	50	150	kg	Drilling chemical storage area	No
Bentonite	3,000	9,000	kg	Drilling chemical storage area	No
Caustic soda	1,400	4,200	kg	Drilling chemical storage area	No
EZ MUD DP or EZ MUD Liquid	2,000	6,000	kg	Drilling chemical storage area	No
ALDACIDE G	336	1008	kg	Drilling chemical storage area	Yes
STOPPIT	1,000	3,000	kg	Drilling chemical storage area	No
Soda ash	350	1050	kg	Drilling chemical storage area	Yes
BARACOR 100	250	750	kg	Drilling chemical storage area	Yes
Sodium chloride (flossy salt)	96,000	288,000	kg	Drilling chemical storage area	No
Barite	500	1,500	kg	Drilling chemical storage area	No
BARACARB	500	1,500	kg	Drilling chemical storage area	Yes
Citric acid	500	1,500	kg	Drilling chemical storage area	Yes
BARADEFOAM HP	500	1,500	kg	Drilling chemical storage area	No
Sodium Bicarbonate	500	1,500	kg	Drilling chemical storage area	No
PERFORMATROL	500	1,500	kg	Drilling chemical storage area	Yes
SOURSCAV	500	1,500	kg	Drilling chemical storage area	No
DRIL-N-SLIDE	500	1,500	kg	Drilling chemical storage area	No
STEELSEAL	500	1,500	kg	Drilling chemical storage area	Yes
BARAZAN D or BARAZAN D Plus	4,150	12,450	kg	Drilling chemical storage area	No
PAC L	2,300	6,900	kg	Drilling chemical storage area	Yes
Potassium chloride	22,500	67,500	kg	Drilling chemical storage area	No
GEM CP/GP	500	1,500	kg	Drilling chemical storage area	No
QUIK-FREE	500	1,500	kg	Drilling chemical storage area	No
BAROFIBRE, BAROFIBRE Superfine and	500	1,500	kg	Drilling chemical storage area	No

Material name	Typical volume	Maximum volume	Unit	Storage area	Hazardous (Y/N)
BAROFIBRE COARSE					
BaraBlend-657	500	1,500	kg	Drilling chemical storage area	Yes
N-DRIL HT Plus	500	1,500	kg	Drilling chemical storage area	Yes
DEXTRID LTE			kg	Drilling chemical storage area	No
BARABUF	500	1,500	kg	Drilling chemical storage area	No
BORE-HIB	500	1,500	kg	Drilling chemical storage area	No
BDF 933 or BaraLube W-933			kg	Drilling chemical storage area	Yes
BAROLIFT	500	1,500	kg	Drilling chemical storage area	No
OXYGON	500	1,500	kg	Drilling chemical storage area	No
ENVIRO-THIN	500	1,500	kg	Drilling chemical storage area	No
Lime	500	1,500	kg	Drilling chemical storage area	Yes
BDF 677	4,770	14,310	kg	Drilling chemical storage area	No
BDF 988	3,390	10,170	kg	Drilling chemical storage area	No
Waste Drilling Fluids	2,500	7,500	m^3	Drill mud sump	Yes
Completion Fluids	1.4	4.2	ML	Drilling sump/onsite tank	No
Condensate	160	480	KL	Condensate storage area	Yes
Diesel	250	750	KL	Diesel storage tanks	Yes
Hydraulic Oil	1,000	3,000	L	Workshop	Yes
Engine Oil	1,000	3,000	L	Workshop	Yes
Degreasers	100	300	L	Workshop	Yes
Flowback	3.2	9.5 (per site)	ML	Flowback tanks	Yes

Appendix F Wastewater Management Plan

THE BEETALOO EXPLORATION PROJECT

Wastewater Management Plan

Review record

Rev	Date	Reason for issue	Author	Reviewer	Approver
1.5	15/05/2021	Minor update to address DEPWS comments			MK
1.6	01/07/2021	Minor updates to included Beetaloo W-1 activities			MK
1.7	25/08/2021	Minor update to address DEPWS comments	MK		MK
1.8	21/11/2021	Minor update to include multiwell EMP	TK		MK
1.9	26/06/2022	Minor update to include delineation EMP	LP		MK
1.10	01/09/2022	Removal of reinjection from section 5.2 and Table 2	LP		MK
1.11	14/10/2022	Minor edits to include recycling / re-use of flowback water	LP		MK

Review due: 18/05/2023

Beetaloo Exploration WWMP

NT-2050-15-MP-028

Table of contents

List of tables

List of appendices

Review due: 18/05/2023

Beetaloo Exploration WWMP

NT-2050-15-MP-028

1. Introduction

This Wastewater Management Plan (WWMP) has been prepared to support Origin's Beetaloo exploration program. The WWMP is a mandatory requirement prepared in accordance with the Code of Practice for Petroleum Activities in the Northern Territory (the CoP).

This plan is designed to provide the strategy for how wastewater will be managed across Origin's Beetaloo exploration activities.

The current Environment Management Plans (EMPs) covered by this plan are:

- NT-2050-15-MP-025 Origin Energy Beetaloo Kyalla 117 N2 Drilling, Stimulation and Well Testing EMP
- NT-2050-15-MP-032 Origin Energy Beetaloo Velkerri 76 S2 Drilling, Stimulation and Well Testing EMP
- NT-2050-15-MP-038 Origin Beetaloo Sub-Basin Kyalla 117 N2 Multiwell Drilling, Stimulation and Well Testing EMP
- CDN/ID NT-2050-35-PH-0018 Origin Beetaloo Sub-Basin Amungee NW-1H EMP
- NT-2050-MP-039- Beetaloo W-1 EMP
- NT-2050-MP-040 Kalala S1 EMP
- NT-2050-MP-041 Beetaloo Sub-Basin Multiwell Drilling, Stimulation and Well Testing EMP
- NT-2050-15-MP-0088 Amungee NW Delineation Program EMP

This plan will reference the related sections within each of the various EMPs to avoid duplication.

2. Description of Activity

Wastewater, as defined in the CoP, includes the following:

- Drilling fluid, drill cuttings and cement returns
- Flowback fluid, generated during the well testing phase
- Completion fluids, kill fluids and well suspension fluids.

Wastewater is produced through the following activities:

- **Drilling**: waste drilling fluids are generated from drilling activities. Drilling fluids primary objective is to provide primary well barrier during well construction (unless underbalance drilling is preferred drilling technique) where bottom hole hydrostatic pressure exerted by drilling fluids is used to overbalance formation pore pressure. Drilling fluids are also used to cool the bit and assist in transporting formation cuttings to surface (rock such as shale, mudstone, siltstone etc.). Excess cement when cementing a casing string and waste drill fluids and cuttings are stored in a lined mud sump, tested and either disposed of on-site or disposed of off-site at a licensed waste facility.
- **Stimulation 'flow back' water:** After the completion of hydraulic fracture stimulation, the exploration well is "flowed back" to remove all recoverable injected fluid from the formation. Flowback wastewater is stored in on-site tanks, recycled / reused, evaporated and then disposed of off-site at a licenced facility.
- **Well production test:** During production testing the well flows gas and water to the surface. The water coming to surface is defined as 'production water' and is separated from the gas

stream and is stored in the on-site tanks, evaporated and then disposed of at a licenced facility. The production water is the same quality as the flow back water from stimulation.

- **Completion activities:** Completion fluids, such as kill fluids or well suspension fluids, are used to supress the formation pressure within the reservoir. The use of these fluids is a form of well control and may need to be removed from the well and disposed of where well interventions are required (i.e. the well may be suspended with fluid post drilling, with the fluid removed prior to completion and stimulation activities).

The wastewater generating activities within the scope of each EMP covered by this plan is presented in Table 1.

Table 1: Wastewater generating activities per Beetaloo Exploration EMP

EMP	Drilling	Stimulation	Well production test	Completions
NT-2050-15-MP-025 Kyalla 117 N2 Section 3.10	x	x	x	x
NT-2050-15-MP-032 Velkerri 76 S2 Section 3.11	x	x	x	x
NT-2050-15-MP-038 Kyalla 117 N2 Multi-well (ORI6) Section 3.9	x	x	x	x
CDN/ID NT-2050-35-PH-0018 Amungee NW-1H (ORI7) Section 7.1	N/A	N/A	x	x
NT-2050-15-MP-039 Beetaloo W-1 EMP (ORI8) Section 7 and Section 10	N/A	N/A	N/A	Not anticipated- with incidental volumes possible
NT-2050-MP-040 Kalala S1 EMP (ORI9) Section 3.13	N/A	N/A	N/A	Not anticipated- with incidental volumes possible

EMP	Drilling	Stimulation	Well production test	Completions
NT-2050-15-MP-041 Beetaloo Sub-basin Multi-well EMP (ORI10) Section 3.13	x	x	x	x
NT-2050-15-MP-0088 Amungee NW Delineation Program EMP Section 3.15	x	x	x	x

3. Waste management framework

Wastewater will be managed with the objective of achieving optimal environmental outcomes and in accordance with the following hierarchy principals:

1. **Avoid:** eliminate the generation of waste through design modification
2. **Reduce:** reduce unnecessary resource use or substitute a less resource intensive product or service
3. **Re-use:** re-use a waste without further processing
4. **Recycle:** recover resources from a waste
5. **Treatment:** treat the waste to reduce the hazard of the waste prior to disposal
6. **Disposal:** disposal of waste if there is no viable alternative.

It is recognised that the options for avoiding, reducing or re-using wastewater generated during exploration are limited. This is largely restricted to:

- Maximising the re-use and recycling of drilling fluids during operations
- Minimising the use of suspension fluids by minimising re-entry activities (i.e. multiple entries into a well requiring fluid to be unloaded)
- Minimise the off-site transportation of flowback through maximisation of recycling / re-use, and evaporation within the designated treatment tanks.

The amount of cuttings produced during the drilling activity is dictated by the regional stratigraphy (target zone depth) and lateral length of the horizontal well, whereas the volume of the flowback is a function of stimulation design and number of stages completed during stimulation. There is however an ability to minimise the volume of waste disposed of off-site, through careful flowback recycling / re-use, waste management and treatment.

4. Wastewater risk assessment

The risks associated with wastewater are covered in the risk assessments within each of the EMPs.

Detailed assessments of the site-specific risk associated with the disposal of drilling fluids and muds as per condition C.4.1.2 of the CoP, will be undertaken upon completion of drilling activities.

Geogenic chemical composition of flowback water was sampled as a part of the Amungee NW-1H, Kyalla 117 N2-1H and Shenandoah 1 a hydraulic fracture activity. These data indicate the risk associated with flowback are largely to do with salts—specifically chlorides. The presence of other compounds, such as hydrocarbons and heavy metals are also likely.

The hazards associated with flowback management have been addressed by the CoP and within specific EMPs. Specific controls covered by the CoP and EMP's preventing environmental harm include:

- Well operations management plan designed to ensure the risk of the well to surrounding aquifers is mitigated; including the requirement for multiple, verified well barriers containing steel and cement
- Use of enclosed tanks
- Use of double lined tanks with leak detection
- Secondary containment requirements for all pumps and high-risk spill locations
- Prohibition of wastewater discharges and reinjection
- Groundwater monitoring bores
- Spill management plan
- Freeboard requirements to accommodate a 1:1000 ARI total wet season.

5. Wastewater management overview

A summary of how each wastewater stream is managed to optimise the environmental outcomes is provided in Table 2. An individual description of each wastewater stream is provided in the following section.

5.1 Drilling fluid and cuttings

Approximately 750 m^3 of solid drilling muds and cuttings and 1-2ML of drilling fluids will be generated from the drilling of each exploration well. Except for synthetic based muds (SBM) which are recovered and reused, water-based drilling fluids and wastes are saline, polymer/bentonite-based material which are stored in lined sumps on-site. The primary contaminants associated with drilling fluids and wastes are likely to be from chlorides.

Drilling fluids and muds will be managed in accordance with the following:

- All drilling fluids, water-based drilling muds and drilling cuttings stored in engineered lined Coletanche (or similar) sumps
- Synthetic based muds will be separated from the drill cuttings, reused and ultimately removed from site for further reuse. Drill cuttings will be stored in the engineered line Coletanche sumps
- Sumps will be designed with a 1:1000 ARI freeboard calculated in accordance with the methodology outlined in Appendix A
- The Maximum Water Level (1.3M wet season and 0.3M dry season freeboard) will be clearly marked on the side of the sump

- All lease pads will be fenced to prevent livestock and fauna ingress into open sumps
- Supernatant liquids will be transferred to lined wastewater storage tanks from the sump upon completion of activity to allow the muds and cuttings contained in lined sump to dry out with liquids evaporated in lined wastewater storage tanks
- Drilling cuttings and muds may be removed from the sump between wells, or as required, to maintain the safe operating level of the sump. Drilling waste material will be stored in pit/sump (in compliance with the CoP) with an impermeable liner, with any free water removed to the sump or wastewater tanks. During the wet season, dried drilling muds and cuttings will be covered to prevent rain ingress into the stored area
- Drilling muds and fluids may be moved between sites to manage sump volume and disposal requirements.
- Any residual liquids will be transported to a licenced interstate disposal facility (Westrex, Jackson, Queensland) with the appropriate interstate waste transport consignment authority as per the *National Environmental Protection (Movement of Controlled Waste between States and Territories) Measure 1998* (NEPM) as implemented under the NT *Waste Management and Pollution Control Act 1998* and Queensland *Environmental Protection Act 1994*
- Leachability testing of drill cuttings and muds will be undertaken in accordance with Table 10 of the CoP
- A disposal option assessment will be completed by a suitably qualified person (as outlined in section C.4.1.2 of the CoP), with on-site disposal to land only undertaken where environmental harm will not result from the disposal activities.

5.2 Produced water and flowback management

All produced water and flowback fluids will be stored in accordance with the CoP.

The volume of flowback generated from the activity is dependent on the number of stages of stimulation, with approximately 20% to 80% fluid recovery expected (based on US ranges). For Origin Stage 2 activities, it is anticipated that approximately 3-12ML of flowback from stimulation activities will be generated from each exploration well, with a final off-site disposal figure of approximately 0.5–2ML per E&A well (post treatment). Flowback and production water will be highly saline, with a summary of the anticipated quality provided in Appendix B —Table 6 and Table 7. Further details on the wastewater generated and stored on-site is found in the water balance section of each EMP.

Management controls for flowback implemented during the program include:

- Recycling of flowback in make-up fluid
- No disposal of flowback wastewater to surface water
- Wastewater stored in above ground tanks
- Tanks to be double lined with built in leak detection
- All wastewater to be stored in enclosed tanks
- The site will have enough enclosed storage to deal with the total volume of wastewater stored at any time
- Appropriate venting of enclosed tanks to prevent the build-up of explosive gasses

- Tank design, construction and operation will consider environmental factors, such as wind loading, temperature bushfires and structural integrity
- All working evaporation tanks will have a minimum freeboard to allow for a 1 in 1000-year average recurrence intensity wet or dry season (depending on which season operations are undertaken in) as calculated in Appendix C
- Off-site wastewater disposal will be minimised through the treatment of wastewater through evaporation. Evaporation tanks will be used to treat wastewater all times, except during periods of significant rainfall
- Mechanical evaporators will be used in each tank to increase evaporation to reduce the volume of flowback. Evaporators will be positioned in a manner to avoid off-site drift and have automated wind direction and speed cut-offs
- Wastewater may be transferred between approved sites to maximise the efficient use of tank capacity.
- The freeboard requirements will be clearly marked on each of the tanks as the Maximum Water Level (MWL)
- During the wet season, wastewater will be stored in enclosed tanks, with some additional treatment capacity available via the evaporation tank
- During the dry season, evaporation tank capacity will be increased to facilitate wastewater treatments
- Wastewater on location must be able to be transferred into enclosed tanks within 72-hours of becoming aware of a significant rainfall event. This transfer must be completed at least 8-hours prior to the predicted commencement of the significant rainfall event. The determination of a significant rainfall event is provided in section 7.1
- Pumping infrastructure must be available to transfer wastewater into enclosed storage within 24-hours (noting wastewater must be transferred 8-hours prior to the onset of the rainfall event). Sufficient pumping redundancy must be available to accommodate pump failures
- Storage tanks that are connected will be designed to prevent uncontrolled release from multiple tanks
- Tanks are to be designed and constructed to the relevant Australian Standard (including AS1554.1 and AS3990) with a quality assurance and quality control (QA/QC) plan and installation procedures implemented by the contractor
- Tanks will be designed to prevent the ingress of stock and fauna, with each exploration site fenced to prevent stock and public access
- Monitoring of pond levels will be undertaken daily, with management response criteria implemented to prevent tank overtopping. This includes shutting in operations where freeboard requirements cannot be met. Monitoring may be in person or via remote methods.
- Residual flowback liquids after recycling / re-use and evaporation will be evaporated and transported to a licenced interstate disposal facility (such as Westrex, Jackson, Queensland) with the appropriate interstate waste transport consignment authority as per the NEPM as implemented under the NT *Waste Management and Pollution Control Act 1998* and Queensland *Environmental Protection Act 1994.*

- When the tanks are decommissioned the associated residual solids, brines and liners are removed and disposed of at an appropriately licensed waste disposal facility by a licenced contractor as per NT *Waste Management and Pollution Control Act.*
- Daily inspections of all wastewater storages will be implemented during operations (active well testing), with continuous level logging and leak detection implemented when sites are unmanned.
- Flowback may be transferred between operating sites to centralise wastewater storage.

5.3 Drilling and completion fluids (suspension and kill fluids)

Drilling and completion fluids (suspension and kill fluids) may be used to maintain bit lubrication and circulation and for well control/suppress formation pressure. Drilling and completion fluids are likely to have an elevated salinity, with sodium and potassium-based salts being the main compounds.

It is anticipated that up to 0.5–1ML of drilling and completion fluids could be produced per well, with fluids stored in the drill sump or tanks (depending on whether tanks have been installed on-site at the stage). The fluids will be evaporated, and any residual transported off-site for final disposal at a licenced facility.

Table 2: Wastewater management summary and implementation plan

Wastewater	Quantity produced	Properties	Hazards	Implementation plan					Final management	Final disposal volume[1]	Alternative management options considered
				Storage	Handling	Operational controls	Routine inspections	Monitoring			
Flowback / production water	3 – 12 ML per well, depending on stimulation volume	Composition influenced by chemical composition of shale formation. Geogenic sourced contaminants include: Salinity (Electrical Conductivity 50,000us/cm-250,000us/cm) with elevated, sodium, chloride, boron, barium and hydrocarbons as per Appendix B	High salinity wastewater representing a hazard to groundwater, surface water and soils from chloride dominated salts if released into the environment	• Stored on-site in double lined above ground enclosed tanks and double lined working evaporation Tanks with leak detection • All tanks have been sized with regards to the 1:1000 average recurrence interval rainfall event as per Appendix A • Maximum water levels (MWL) to be clearly marked on each tank	• Transferred to storage facilities from on-site separators or directly from the well as required under B.4.13.2 (k) of the CoP • Secondary containment used under all pumps and connections	• Recycling of flowback in make-up fluid • Storage volumes of ponds to be monitored daily through visual inspections or telemetry during wastewater storage • Wastewater stored in enclosed tanks during wet season, with some evaporation tank surplus capacity • Evaporation tank capacity increased during dry season to facilitate treatment • Wastewater (equivalent of wet season freeboard) to be transferred into enclosed storage when a significant rainfall event is predicted as per section 7 • Evaporators to be strategically located on or within the boundaries of the pond with drift prevention controls (automated wind	• Storage facilities and handling areas inspected daily during operations via electronic or manual means • Visual inspections of tanks completed weekly	As per section 6	Evaporated on-site using fractionating evaporators to reduce final volumes. Potentially onsite treatment using brine crystallisation to create solid salt. Then trucked off-site to a licenced waste disposal facility (where locally available or Westrex in QLD) in accordance with *NT Waste Management and Pollution Control Act* waste consignment authority approval	Up to 2ML/well	• Due to the saline nature of the material, limited re-use or recycling options exist during exploration • Treatment using Reverse Osmosis or other mechanical filtration has been considered; salinity and scaling constraint posed by wastewater restricted the use of conventional water treatment • Request for proposal (RFP) has been released to identify additional technologies for a potential future trial

[1] Note these values are indicative and the final values are outlined in the respective EMP.

Wastewater	Quantity produced	Properties	Hazards	Implementation plan					Final management	Final disposal volume[1]	Alternative management options considered
				Storage	Handling	Operational controls	Routine inspections	Monitoring			
						direction and speed cut offs)					
Drilling muds, cuttings and fluids	750m^3/well	Saline (KCL and NaCl) polymer/bentonite based drilling fluids with formation cuttings Synthetic based muds (SBM) separated from formation cuttings and reused (no onsite disposal of SBM required)	KCL and NaCL may represent a hazard in residual drilling muds and cuttings if not segregated prior to disposal. Formation cuttings may contain low level of hydrocarbons, which are likely to be degraded quickly in the open sump.	• Stored on-site in lined drilling sumps with sufficient freeboard to accommodate a 1:1000 average recurrence interval rainfall event as per Appendix A • Maximum water levels (MWL) to be clearly marked on each tank and sump	• Transferred directly from rig via the shakers into the sump • Fluid stored in lined tanks as per CoP	• Storage volumes of sumps to be monitored daily • Material to be dried out after completion of activity, with supernatant fluids evaporated in a separate tank (CoP compliant storage) • Material to be tested prior to determining final disposal requirements	• Sump level to be monitored daily during operations via electronic or manual means • Sump liner and embankments to be inspected weekly during operations	As per section 6	• Supernatant fluids will be segregated from muds upon completion of activity and evaporated (in a CoP compliant tank) • Fluids to be transported to a licenced waste disposal facility (where available locally or Westrex in QLD) in accordance with *NT Waste Management and Pollution Control Act* and related interstate waste consignment authority approval • Final disposal solution of muds and cuttings to be determine through on-site characterisation and risk assessment by third party • For on-site disposal, muds and cuttings to be mixed, buried and covered on-site • For off-site disposal, material will be transported to a licenced waste disposal facility (where available locally or Westrex in QLD) in accordance with *NT Waste Management and Pollution Control Act* and related interstate	750m^3/well	There are no other viable options currently available in addition to what has currently been considered (off-site and on-site disposal)

Wastewater	Quantity produced	Properties	Hazards	Implementation plan					Final management	Final disposal volume[1]	Alternative management options considered
				Storage	Handling	Operational controls	Routine inspections	Monitoring			
									waste consignment authority approval		
Drilling, completion, suspension and kill fluids	0.5-1ML/well (or incidental volumes during maintenance)	KCL or NaCl based fluids with a TDS >50,000us/cm	High salinity wastewater representing a hazard to groundwater, surface water and soils from chloride dominated salts if released into the environment	• Stored on-site in the sump and transferred (as required) to the double lined above ground enclosed tanks and evaporation tanks with leak detection • All tanks have been sized with regards to the 1:1000 average recurrence interval rainfall event as per Appendix A • Maximum water levels (MWL) to be clearly marked on each tank and sump	• Transferred to flowback storage facilities directly from well • Secondary containment used under all pumps and connections	• Storage volumes of ponds to be monitored daily during operations • Evaporators to be strategically located on or within the boundaries of the pond with drift prevention controls (automated wind direction and speed cut offs) • All wastewater to be transferred into enclosed storage when a significant rainfall event is predicted as per section 7.1	• Storage facilities and handling areas inspected daily during operations • Visual inspections of tanks completed weekly	As per section 6	Stored in flowback tanks. Evaporated on-site using fractionating evaporators to reduce final volumes. Potentially onsite treatment using brine crystallisation to create solid salt. Then trucked off-site (if required) to a licenced waste disposal facility (Westrex in QLD) in accordance with *NT Waste Management and Pollution Control Act* waste consignment authority approval. Currently, it is assumed all drilling wastewater will be evaporated with limited water removed from site.	0–0.5ML	• Due to the saline nature of the material, limited re-use or recycling options exist during exploration • Treatment using reverse osmosis or other mechanical filtration has been considered; salinity and scaling constrain the use of conventional water treatment • Request for proposal (RFP) has been released to identify additional technologies for a potential future trial

6. Wastewater monitoring program

A wastewater sampling program will be implemented to characterise the quality of the wastewater during flowback activities. The monitoring program is summarised in Table 3 below.

Table 3: Minimum monitoring requirements

Monitoring Program	Location	Monitoring Requirements	Frequency
Significant rainfall event detection	Each site	Daily review of 8-day total rain forecast as per section 7.1	Daily during wastewater storage
Flowback characterisation	Post separator— prior to entering storage tanks	Electrical conductivity, pH, temperature and volume of flowback	Continuous (at least one sample every 24 hours)
		Testing samples of flowback for analytes listed in Appendix C	Weekly until the EC level stabilises (<10% change over 2 weeks) and then monthly until practical completion of flowback activities
Stimulation fluid— pre-injection	Post blender— prior to injection	Testing sample of stimulation fluid for analytes listed in Appendix C	1 sample pre-injection for each stimulation fluid utilised
Flowback storage tanks	Each storage tank	Testing samples of flowback for analytes listed in Appendix C	6-monthly
	Each storage tank	Level— estimated evaporation rates	Daily - through either visual inspections or telemetered meter.
Drilling material	Determined by suitably qualified person	Testing samples of drilling cuttings for analytes listed in Table 10 of the Codes of Practice, Naturally Occurring Radiation Material (NORMs) and volume	Prior to disposal
Fauna interactions	Wastewater tanks and surrounding lease area	1. Ad hoc bird and fauna observations and photos to be taken around wastewater tanks 2. Wastewater tank inspection for bird carcasses	1. Continuous 2. Daily 3. Weekly 4. During final decommissioning

Monitoring Program	Location	Monitoring Requirements	Frequency
		3. Inspections around area adjacent to lease (within 50 m of boundary) 4. Carcasses present during tank emptying	

6.1 Sampling methodology

- Water samples will be collected in accordance with the methodology outlined in Table 4
- All samples will be collected by appropriately qualified personnel, with all meters calibrated in accordance with the manufacturer's instructions
- Samples will be collected in laboratory supplied sampling containers and placed in chilled eskies and transported under chain of custody (COC) procedures
- Analysis will be performed by laboratories with National Association of Testing Authorities (NATA) accredited analysis methodology
- Each sample will have a unique identifier that would be cross referenced to the monitoring location and time of sampling. Due to the remote location, samples will be couriered to the laboratory to minimise sample holding time violations.
- In accordance with of C.5.1 (d) in the Code of Practice, where there are no NATA accredited laboratories for a specific analyte or substance, then duplicate samples must be sent to at least two separate laboratories for independent testing or evaluation.

Table 4: Monitoring program methodologies

Program	Sampling methodology
Drilling sump characterisation	• National Environment Protection (Assessment of Site Contamination) Measure • AS4482.1-2005 guide to the investigation and sampling of sites with potentially contaminated soil
Flowback and drilling fluid monitoring	• Australian and New Zealand Guidelines for Fresh and Marine Water Quality 2000 (ANZECC Guidelines) • AS/NZ5667.1: 1998. Water Quality Sampling Part 1: Guidance on the design of sampling programs, sampling techniques and the preservation and handling of samples

7. Wastewater storage management response criteria

To minimise the risk of overtopping a tank or sump, the criteria outlined in Table 5 will be implemented when hydraulic fracturing wastewater is stored on-site.

Table 5: Wastewater storage management response criteria

Monitoring program	Criteria Description	Criteria	Criteria Response
Significant rainfall event	Significant rainfall event predicted	The 4-day total rainfall exceeds 300mm within the 8-day forecast	All flowback fluid must be transferred to enclosed storage at least 8-hours prior to the predicted commencement of the significant rainfall event
Wastewater tank level monitoring	Enclosed storage level exceedance	The total volume of hydraulic fracturing wastewater stored on-site exceeds the available closed/covered tank storage capacity	Flowback activities to cease, unless authorised by DEPWS to continue operations. Origin to provide written notification to DEPWS within 48-hours of exceedance, along with the proposed plan to return to compliance.
Drilling sump level monitoring	Drilling sump storage level exceedance	The total volume of drilling wastewater exceeds the freeboard capacity of the drilling sump, with no additional storage available within other on-site tanks	Drilling wastewater disposal activities to cease, unless authorised by DEPWS to continue operations. Origin to provide written notification to DEPWS within 48-hours of exceedance, along with the proposed plan to return to compliance.

7.1 Significant rainfall events

The 8-day Bureau of Meteorology 4-day total rain forecast[2] shall be reviewed daily to identify periods of significant rainfall. Significant rainfall is defined in this WWMP as an event where greater than 300mm of total rainfall is predicted over a 4-day period. This type of rainfall level is consistent with that from a significant rainfall event, such as a monsoonal trough, tropical low or cyclone.

Commencement time to transfer the flowback fluid will be selected to ensure that it is completed at least 8-hours prior to the predicted commencement of the significant rainfall event.

8. Waste transportation and disposal

All wastewater transport providers will be licenced under the NT *Waste Management and Pollution Control Act 1998.*

All wastewater will be transported interstate to a licenced waste storage and treatment facility. Westrex, at Jackson, Queensland is the current default option for wastewater disposal, with other interstate disposal locations available. The transportation of wastewater between states/territories, will require an Interstate waste transport consignment authority as per the NEPM as implemented under the NT *Waste Management and Pollution Control Act 1998* and relevant accepting state/territory (such as the Queensland *Environmental Protection Act 1994*).

All wastewater storage and treatment facilities will be licenced as per the relevant accepting state/territory (such as the Queensland *Environmental Protection Act 1994*).

9. Waste tracking and reporting

The movement of wastewater will be tracked in accordance with the following:

i. Volumes of wastewater produced from the well

ii. Volumes of wastewater transferred into each tank

iii. Estimates for evaporation rates from each tank updated weekly

iv. Volumes of wastewater reused

vi. Volumes of water removed from site (whether by vehicle or pipeline).

- Wastewater tracking will be documented and available upon request
- Off-site wastewater tracking must be in accordance with tracking requirements of listed wastes as per the *Waste Management and Pollution Control Act*, NEPM and (where relevant) the *Radiation Protection Act*
- Wastewater tracking documentation must be reported to the Minister at least annually in the annual environment performance report for the relevant EMP.

The following measurement criteria have been developed to demonstrate the risks associated with wastewater storage are reduced as low as reasonably practicable:

- Zero wastewater tank overtopping events
- No off-site releases of wastewater
- No reportable spills of wastewater

[2] Refer http://www.bom.gov.au/jsp/watl/rainfall/pme.jsp

10. Incident reporting

The reporting of incidents shall comply with the Petroleum (Environment) Regulations 2016 (the Regulations) and the *Waste Management and Pollution Control Act 1998.*

10.1 Reportable environmental incident reporting

The Regulations define a reportable incident as an incident arising from a regulated activity that has caused, or has the potential to cause, material environmental harm or serious environmental harm as defined under the *Petroleum Act 1984*.

An interest holder must notify (this may be oral or in writing) DEPWS of a reportable incident as soon as practicable but no later than two-hours after the first occurrence of the incident or after the time the interest holder becomes aware of the incident.

DEPWS can be notified through the DEPWS Onshore gas non-compliance hotline on 1800 413 567.

Any verbal report to DEPWS must be followed up by a written report from the Project Manager within three days in accordance with the Regulations.

10.2 Recordable incidents

The Regulations define a recordable incident as an incident arising from a regulated activity that:

I. Has resulted in an environmental impact or environmental risk not specified in the current plan for the activity; or

II. Has resulted in a contravention of an environmental performance standard specified in the current plan for the activity; or

III. Is inconsistent with an environmental outcome specified in the current plan for the activity; and

IV. Is not a reportable incident.

An interest holder must notify (this may be oral or in writing) DEPWS of a recordable incident as soon as practicable but no later than 15-days after the reporting period (agreed period or each 90-day period after the day on which the EMP is approved).

10.3 *Waste Management and Pollution Control Act 1998* incident reporting

In accordance with the Waste Management Pollution Control Act, where contaminants or waste is not confined within the land on which the petroleum activities are undertaken (i.e. the approved disturbance areas where the petroleum activity is occurring), Origin will notify the regulator of incidents causing or threatening to cause pollution as soon as practicable, but no later than 24-hours after becoming aware of the incident.

A notifiable incident is defined as an incident that causes, or is threatening or may threaten to cause, pollution resulting in material environmental harm or serious environmental harm.

A notification must include:

(a) the incident causing or threatening to cause pollution
(b) the place where the incident occurred
(c) the date and time of the incident
(d) how the pollution has occurred, is occurring or may occur
(e) the attempts made to prevent, reduce, control, rectify or clean up the pollution or resultant environmental harm caused or threatening to be caused by the incident

(f) the identity of the person notifying.

The notification shall be made to the NT EPA Pollution Hotline 1800 064 567.

11. Emergency response

An Emergency Response Plan (NT-2050-15-MP019) has been developed covering the proposed activities within the EMP. The ERP provides a broad framework for managing potential emergency incidents to minimise the potential risk to human safety and the environment. The ERP should be referenced for any emergency response activities.

Spills must be reported to the Minister in accordance with the requirements of spill management plan (NT-2050-15-MP-027) and reportable and recordable incidents of the Regulations.

Appendix A 1:1000 ARI Calculation

Monthly rainfall totals were analysed from the Scientific Information for Land Owners (SILO) data for to interpolate rainfall data from 1900 to the present day. Consistent with industry accepted methodology associated with practices (such as dam risk assessments which calculate the wet season based on your geographical location) a 3-month period was determined applicable.

The highest 3-month rainfall period during the wet and dry seasons was predicted for every year from 1900 till 2018. These values were then used to fit a Log Pearson III distribution to the data to allow us to extrapolate to the 1000-year, 3-month duration wet season (1) and 3-month dry season (figure 2). This method is consistent with the *Australian Rainfall & Runoff* methodologies. The median 1 in 1000-year 3-month wet season is 1,289mm and 3-month dry season is 300mm. These figure does not include any evaporation and are therefore considered extremely conservative.

Based on the assessment, a 1,300mm wet season and 300mm dry season freeboard will be applied to all open sumps and tanks.

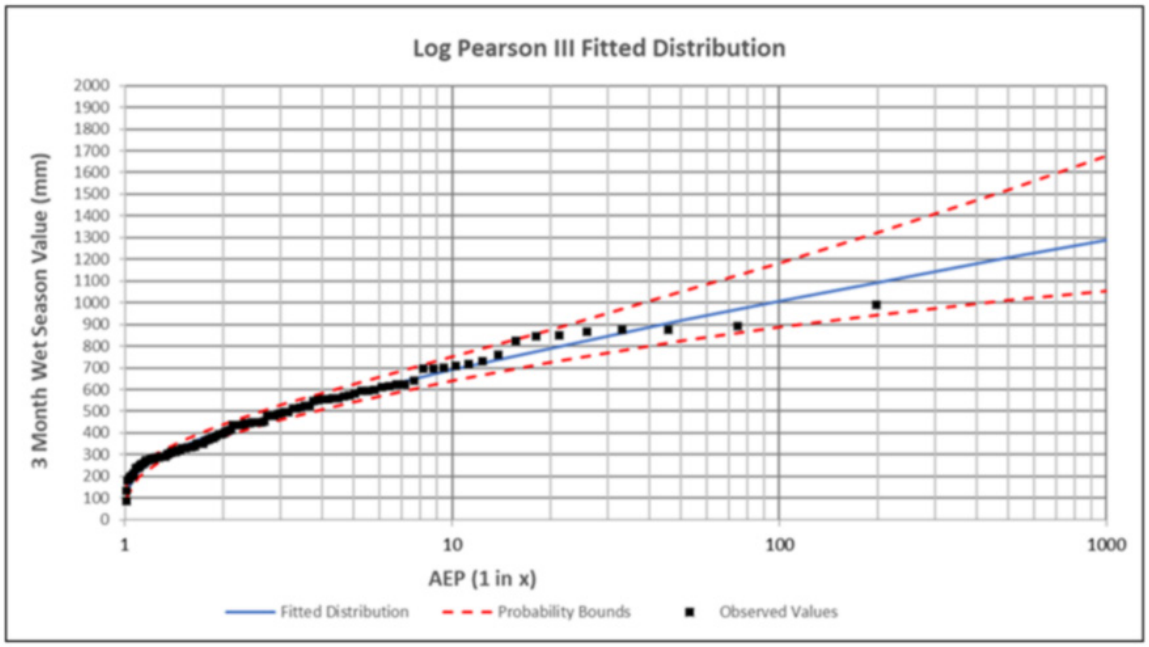


Figure 1: Log Pearson determination of 1:1000 Wet Season ARI

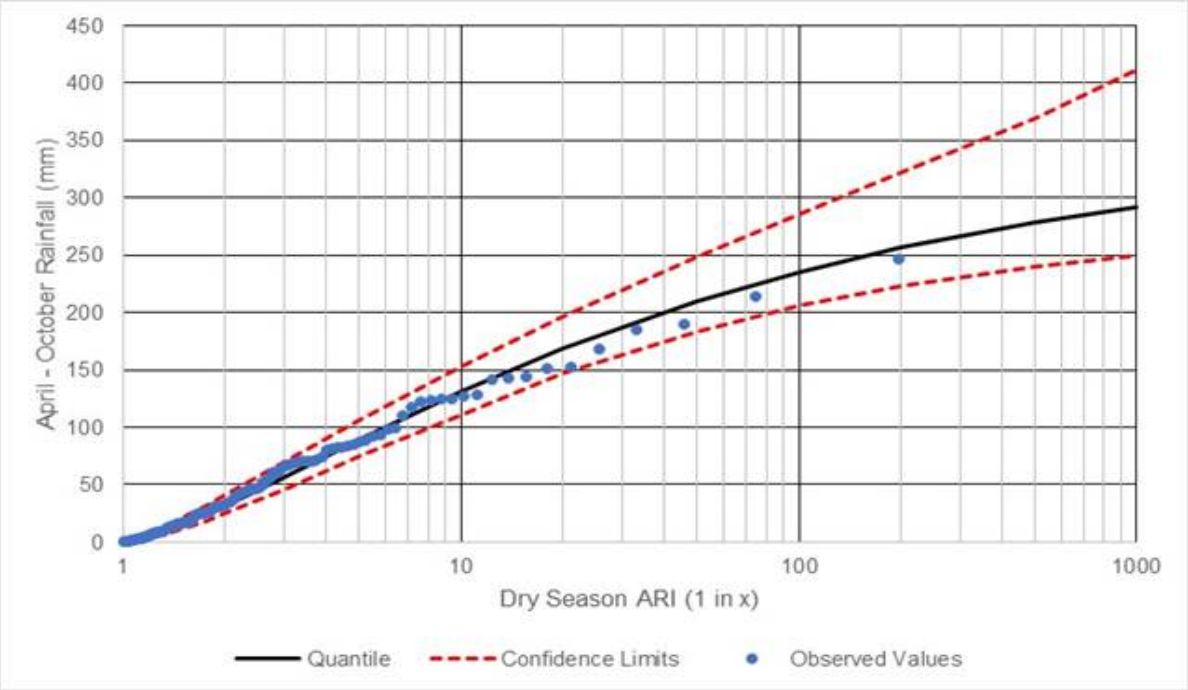


Figure 2: Log Pearson determination of 1:1000 Dry Season ARI

Appendix B Flowback characteristic summary

Table 6: Anticipated flowback quality of the Velkerri formation based on Amungee NW-1H flowback results

Parameter	Flow back levels
BTEX compounds	BTEX levels are anticipated to be low ranging between 2 and 15 µg/L.
Total nitrogen (as N)	Maximum value of 62.1 mg/L observed within flowback.
Salinity (TDS)	Saline with total dissolved solids level exceeding 49,000 mg/L.
pH	Slightly acidic with a median value of 6.74.
Major ions	Predominantly Na and Cl dominated. Bicarbonate present at levels consistent with stimulation fluid.
Dissolved metals	Barium and boron are the main metal elements anticipated to be present at elevated levels. Maximum levels of 80.1 mg/L for barium and 54.5 mg/L for boron were recorded during the Amungee NW 1H flowback. Lower level of other metals such ash Arsenic and Manganese were observed, with maximum concentration of 0.084 mg/L and 3.09 mg/L, respectively.
Polycyclic Aromatic Hydrocarbons	Expected to be below detection level.
Petroleum Hydrocarbons	All fractions of TPH are anticipated to be elevated.
Phenolic Compounds	Low level of phenolic compounds expected, with only Phenol (max 4 µg/L) and 3-&4- methylphenol (max 11.3 ug/L).
Radionuclides	Maximum Gross Alpha Activity and Gross Beta Activity of 12.4 Bq/L and 18.3 Bq/L were recorded in the flowback of offset wells. The primary component being radium-226.

Table 7: Flowback quality based on Kyalla 117 N2-1 flowback results

Parameter	Flow back levels
BTEX compounds	Total BTEX levels in the flowback ranged between 63 and 190 µg/L.
Total nitrogen (as N)	Maximum value of 180mg/L observed within flowback.
Salinity (TDS)	Saline with total dissolved solids level from 120,000–290,000 mg/L.
pH	Slightly acidic with a median value of 6.54.

Parameter	Flow back levels
Major ions	Flowback predominantly Na and Cl dominated, with elevated levels of calcium and magnesium.
Dissolved metals	All detected dissolved metal concentrations within the flowback were low, except for barium (1029 mg/L), gallium (290 mg/L) and strontium (279 mg/L).
Polycyclic Aromatic Hydrocarbons	All values in the flowback below laboratory Limit of Reporting (LOR).
Petroleum Hydrocarbons	All fractions of TPH are anticipated to be elevated, with Total Petroleum Hydrocarbon levels likely to range from 25 mg/L–150 mg/L.
Phenolic Compounds	Low levels of phenolic compounds detected in flowback with phenol and phenol compounds <3 µg/L.
Radionuclides	Maximum Gross Alpha Activity and Gross Beta Activity of 36.2 Bq/L and 97Bq/L encountered in the flowback, the anticipated source is likely to be radium-226.

Appendix C Wastewater monitoring analyte list

Parameter	Reporting units	Limit of reporting	Method
Physical Parameters			
Electrical Conductivity (EC)	us/cm	1	Field
Total Dissolved Solids (TDS)	mg/L	10	APHA 2540C
Total Suspended Solids (TSS)	mg/L	5	APHA 2540C
pH		0.1	Field
Sodium Adsorption Ratio	ratio	0.01	APHA 4500 Ca, Mg, Ca, NA
Temperature	°C	0.1	Field
Nutrients			
Nitrate	mg/L	0.01	APHA VC13
Nitrite	mg/L	0.01	APHA 4500 NO2
Total Nitrogen	mg/L	0.1	APHA 4500 NORG
Total Kjeldahl Nitrogen	mg/L	0.1	APHA NORG/TKN
Ammonia	mg/L	0.01	APHA NH4
Reactive Phosphorous	mg/L	0.01	APHA 4500P
Total Phosphorous	mg/L	0.01	APHA 4500P
Anions			
Sulfate	mg/L	1	APHA 4500-SO4-C
Chloride	mg/L	1	APHA 4500-Cl-C
Carbonate	mg/L	1	APHA 2320 B
Bicarbonate (as $CaCO_3$ equivalent)	mg/L	1	APHA 2310 B
Bicarbonate Alkalinity (as $CaCO_3$ equivalent)	mg/L	1	APHA 2320 B
Hydroxide Alkalinity (as $CaCO_3$ equivalent)	mg/L	0.01	APHA 2320 B
Total Alkalinity (as $CaCO_3$ equivalent)	mg/L	0.01	APHA 2320 B
Fluoride	mg/L	0.1	APHA 4500 F-C
Bromide	mg/L	0.01	APHA 4110B
Major Cations			
Sodium	mg/L	1	APHA 4500 Na
Magnesium	mg/L	1	APHA 4500 Mg
Potassium	mg/L	1	APHA 4500 K
Calcium	mg/L	1	APHA 4500 Ca

Parameter	Reporting units	Limit of reporting	Method
Metals and Metalloids (total and dissolved)			
Aluminium	mg/L	0.001	USEPA 6010 ICP/AES
Antimony	mg/L	0.001	USEPA 6010 ICP/AES
Arsenic	mg/L	0.001	USEPA 6010 ICP/AES
Barium	mg/L	0.001	USEPA 6010 ICP/AES
Beryllium	mg/L	0.001	USEPA 6010 ICP/AES
Boron	mg/L	0.001	USEPA 6010 ICP/AES
Bromide	mg/L	0.001	USEPA 6010 ICP/AES
Cadmium	mg/L	0.001	USEPA 6010 ICP/AES
Chromium	mg/L	0.001	USEPA 6010 ICP/AES
Copper	mg/L	0.001	USEPA 6010 ICP/AES
Iron	mg/L	0.001	USEPA 6010 ICP/AES
Lead	mg/L	0.001	USEPA 6010 ICP/AES
Manganese	mg/L	0.001	USEPA 6010 ICP/AES
Mercury	mg/L	0.001	USEPA 6010 ICP/AES
Molybdenum	mg/L	0.001	USEPA 6010 ICP/AES
Nickel	mg/L	0.001	USEPA 6010 ICP/AES
Selenium	mg/L	0.001	USEPA 6010 ICP/AES
Silica	mg/L	0.1	USEPA 6010 ICP/AES
Silver	mg/L	0.001	USEPA 6010 ICP/AES
Strontium	mg/L	0.001	USEPA 6010 ICP/AES
Thorium	mg/L	0.001	USEPA 6010 ICP/AES
Tin	mg/L	0.001	USEPA 6010 ICP/AES
Uranium	mg/L	0.001	USEPA 6010 ICP/AES
Vanadium	mg/L	0.001	USEPA 6010 ICP/AES
Zinc	mg/L	0.001	USEPA 6010 ICP/AES
Naturally Occurring Radioactive Material (NORM)			
alpha radiation	Bq/L	0.05	ASTM D7283-06
beta radiation	Bq/L	0.05	ASTM D7283-06
BTEX			
Benzene	µg/L	1	USEPA 5030/8260 HS or P&T/GC/MS
Toluene	µg/L	2	USEPA 5030/8260 HS or P&T/GC/MS
Ethylbenzene	µg/L	2	USEPA 5030/8260 HS or P&T/GC/MS
M and P Xylene	µg/L	2	USEPA 5030/8260 HS or P&T/GC/MS

Parameter	Reporting units	Limit of reporting	Method
O Xylene	µg/L	2	USEPA 5030/8260 HS or P&T/GC/MS
Total Xylene	µg/L	2	USEPA 5030/8260 HS or P&T/GC/MS
Hydrocarbons			
TRH C6 - C10	µg/L	20	USEPA 5030/8260 HS or P&T/GC/MS
TRH C6 - C10 less BTEX	µg/L	20	USEPA 5030/8260 HS or P&T/GC/MS
TRH >C10 - C16	µg/L	100	USEPA 5030/8260 HS or P&T/GC/MS
TRH >C10 - C16 less Napthalene	µg/L	100	USEPA 5030/8260 HS or P&T/GC/MS
TRH >C16 - C34	µg/L	100	USEPA 5030/8260 HS or P&T/GC/MS
TRH >C34 - C40	µg/L	100	USEPA 5030/8260 HS or P&T/GC/MS
Total TRH C6 - C40	µg/L	100	USEPA 5030/8260 HS or P&T/GC/MS
Polycyclic Aromatic Hydrocarbons			
3-Methylcholanthrene	mg/L	0.001	USEPA 3510/8270 GC/MS
7, 12-Dimethylbenz(a)anthracene	mg/L	0.001	USEPA 3510/8270 GC/MS
Acenaphthene	mg/L	0.001	USEPA 3510/8270 GC/MS
Acenaphthylene	mg/L	0.001	USEPA 3510/8270 GC/MS
Anthracene	mg/L	0.001	USEPA 3510/8270 GC/MS
Benzo (a) pyrene	mg/L	0.0005	USEPA 3510/8270 GC/MS
Benzo (b) fluoranthene	mg/L	0.001	USEPA 3510/8270 GC/MS
Benzo (ghi) perylene	mg/L	0.001	USEPA 3510/8270 GC/MS
Benzo (k) fluoranthene	mg/L	0.001	USEPA 3510/8270 GC/MS
Benzo (a) anthracene	mg/L	0.001	USEPA 3510/8270 GC/MS
Chrysene	mg/L	0.001	USEPA 3510/8270 GC/MS
Dibenz (ah) anthracene	mg/L	0.001	USEPA 3510/8270 GC/MS
Fluoranthene	mg/L	0.001	USEPA 3510/8270 GC/MS
Fluorene	mg/L	0.001	USEPA 3510/8270 GC/MS
Indeno (1,2,3-cd) pyrene	mg/L	0.001	USEPA 3510/8270 GC/MS
Napthalene	mg/L	0.001	USEPA 3510/8270 GC/MS
Phenanthrene	mg/L	0.001	USEPA 3510/8270 GC/MS
Pyrene	mg/L	0.001	USEPA 3510/8270 GC/MS

Parameter	Reporting units	Limit of reporting	Method
Carcinogenic PAHs (benzo[a}pyrene equivalents	mg/L	0.001	USEPA 3510/8270 GC/MS
Total PAH	mg/L	0.0005	USEPA 3510/8270 GC/MS
Volatile Organic Compounds			
2,3,4,6-Tetrachlorophenol	µg/L	1	USEPA 3510/8270 GC/MS
2,4,5-Trichlorophenol	µg/L	1	USEPA 3510/8270 GC/MS
2,4,6-Trichlorophenol	µg/L	1	USEPA 3510/8270 GC/MS
2,4-Dichlorophenol	µg/L	1	USEPA 3510/8270 GC/MS
2,4-Dimethylphenol	µg/L	1	USEPA 3510/8270 GC/MS
2,4-Dinitrophenol	µg/L	1	USEPA 3510/8270 GC/MS
2,6-Dichlorophenol	µg/L	1	USEPA 3510/8270 GC/MS
2-Chlorophenol	µg/L	1	USEPA 3510/8270 GC/MS
2-Methyl-4,6-dinitrophenol	µg/L	1	USEPA 3510/8270 GC/MS
2-Nitrophenol	µg/L	1	USEPA 3510/8270 GC/MS
4-Chloro-3-methylphenol	µg/L	1	USEPA 3510/8270 GC/MS
4-Nitrophenol	µg/L	1	USEPA 3510/8270 GC/MS
Dinoseb	µg/L	1	USEPA 3510/8270 GC/MS
Formaldehyde	µg/L	1	USEPA 3510/8270 GC/MS
Hexachlorophene	µg/L	1	USEPA 3510/8270 GC/MS
m- and p-Cresol	µg/L	1	USEPA 3510/8270 GC/MS
Pentachlorophenol	µg/L	1	USEPA 3510/8270 GC/MS
Phenol	µg/L	1	USEPA 3510/8270 GC/MS
Organic Carbon			
Dissolved Organic Carbon	mg/L	1	APHA 5310 B
Total Organic Carbon	mg/L	1	APHA 5310 B

Appendix G Erosion and Sediment Control Plan

BEETALOO BASIN EXPLORATION PROJECT

Erosion and Sediment Control Plan

EP76, EP98 and EP117

This document outlines the basic principles for Contractors to develop site specific erosion and sediment control plans for Beetaloo Basin Exploration Program.

Review record

Rev	Date	Reason for issue	Reviewer/s	Consolidator	Approver
0	29/03/2019	Issued for use	A.Court	M.Kernke	M.Hanson
1	28/06/2019	Revised based on comments received by DEPWS	A.Court/J.Jentz	M.Kernke	M.Hanson
2	16/07/2019	Updated Primary ESCP	A.Court/J.Jentz	M.Kernke	M.Hanson
3	19/11/2021	Update overarching ESCP	P.Szamosi/J.Jentz	A.Court	M.Kernke
4	23/02/2022	Update overarching ESCP	P.Szamosi/J.Jentz	A.Court	M.Kernke
5	08/07/2022	Update for Amungee delineation area	A.Court	A.Court	M.Kernke
6	13/09/2022	Update overarching ESCP	J.Jentz	A.Court	L. Pugh

Review due: 13/09/2025

Erosion and Sediment Control Plan

NT-2050-15-MP-019.

Table of contents

Table of figures

Review due: 13/09/2025

List of tables

List of appendices

1. Introduction

This Erosion and Sediment Control Plan (ESCP) has been developed to ensure best practice erosion and sediment controls are implemented during Origin's Exploration activities within permit EP76, EP98 and EP117 to prevent erosion and offsite impacts such as sedimentation of waterways.

This ESCP has been developed to provide direction for Origin and contractors to implement erosion and sediment control (ESC) during construction of the lease pads and associated infrastructure, worker camps and access tracks, seismic lines as well as during ongoing maintenance and monitoring once sites are established.

The design of the exploration well pads, seismic lines and access tracks will comply with Northern Territory and local government statutory laws and regulations and are to be designed to meet all relevant and applicable codes and standards. This ESCP has been developed in accordance with the following guidelines:

- *Code of Practice for Petroleum Activities in the Northern Territory* (Department of Environment and Natural Resources (DENR), 2019)
- *Best Practice Erosion and Sediment Control* (International Erosion Control Association (IECA), 2008)
- *Land Clearing Guidelines* (Department of Environment, Parks and Water Security (DEPWS), 2021)
- *Erosion and Sediment Control Guidelines for Rural Development Environment Fact Sheet* (DLRM, 2018).

The location of the proposed exploration activities are shown on **Figure 1**.

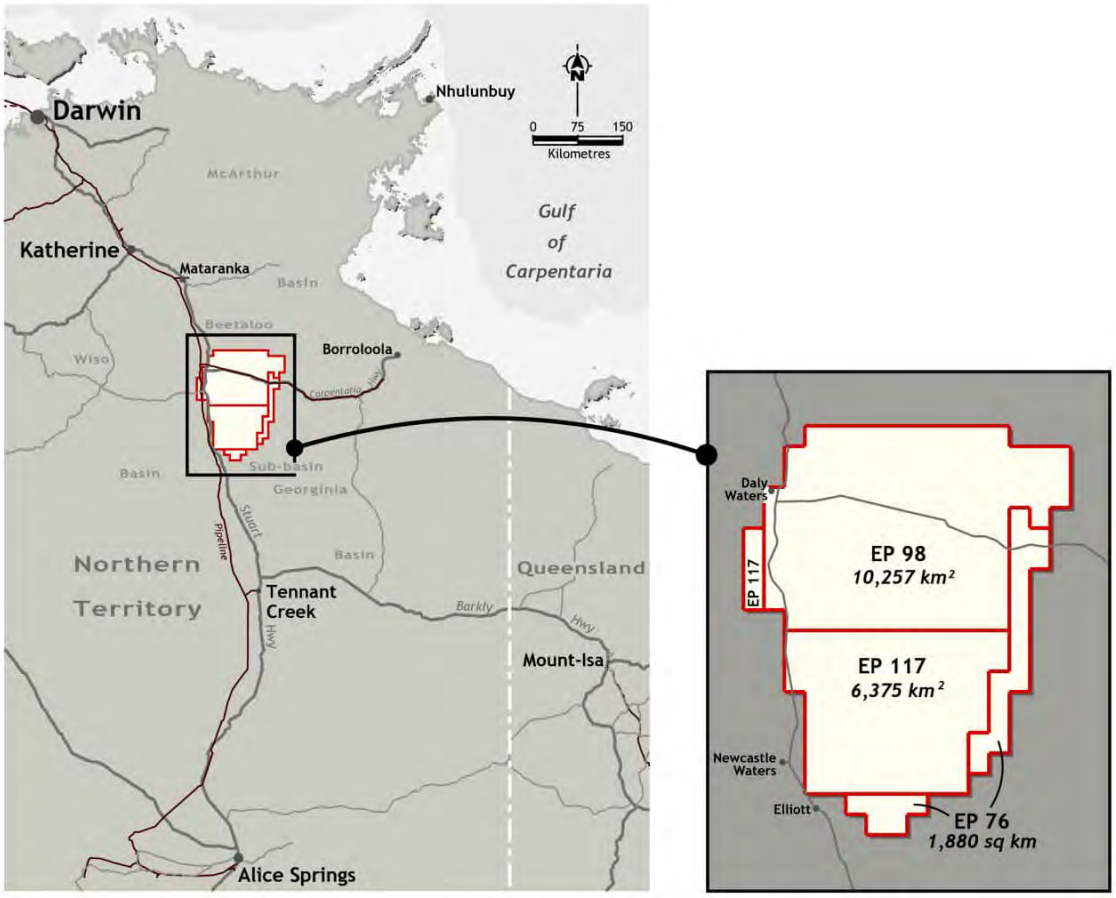


Figure 1 Location of Origin Permit Area

2. Project Context

This plan covers all civil, seismic acquisition, well drilling, stimulating, rehabilitation and routine maintenance/monitoring activities undertaken by Origin and their contractors within permit EP76, EP98 and EP117 as detailed in Table 1 and shown in Figure 2 and Figure 3.

Table 1 Coordinates of centroid 2D Seismic and exploration lease areas

2D Seismic Line Reference	Seismic line coordinates			
	Start of line		End of line	
	Lat	Long	Lat	Long
001-SR	-16.32434	133.82875	-16.39386	133.89996
002-SR	-16.32112	133.85894	-16.35325	133.89186
003-SR	-16.34104	133.87802	-16.39438	133.93218
004-SR	-16.36162	133.93763	-16.41430	133.99165
005-SR	-16.34667	133.95114	-16.39806	134.00384
006-SR	-16.37223	133.86042	-16.37795	134.00306
007-SR	-16.34267	133.88364	-16.34584	133.88032
008-SR	-16.34459	133.88562	-16.34777	133.88229
009-SR	-16.34652	133.88759	-16.34970	133.88427
010-SR	-16.34845	133.88957	-16.35163	133.88624
Lease Area, Access Track and Gravel Pits	**Lease Pads**			
	Zone	**Easting**	**Northing**	
Velkerri 98 E1	53	415515	8180683	
Amungee NW	53	415515	8180683	
Amungee NW-2	53	381039	8192324	
Amungee NW-3	53	389841.4	8190093	
Amungee NW-4	53	376611	8193100	
Amungee NW-5	53	390313.6	8187337	
Kyalla 98 W1	53	364955	8177458	
Kalala S1	53	351740	8198030	
Velkerri 76 S1	53	424362	8113273	
Velkerri 76 N1	53	440940	8107032	
Velkerri 76 S2	53	435488	8136321	
Kyalla 117 N2	53	356175	8137500	
Velkerri 117 E1	53	428861	8120782	
Beetaloo W (Kyalla 117 W1)	53	368312	8106695	

*Grey shading are new lease pads for 2022/2023 | * Universal Transverse Mercator (UTM) geographic coordinate system is GDA 94*

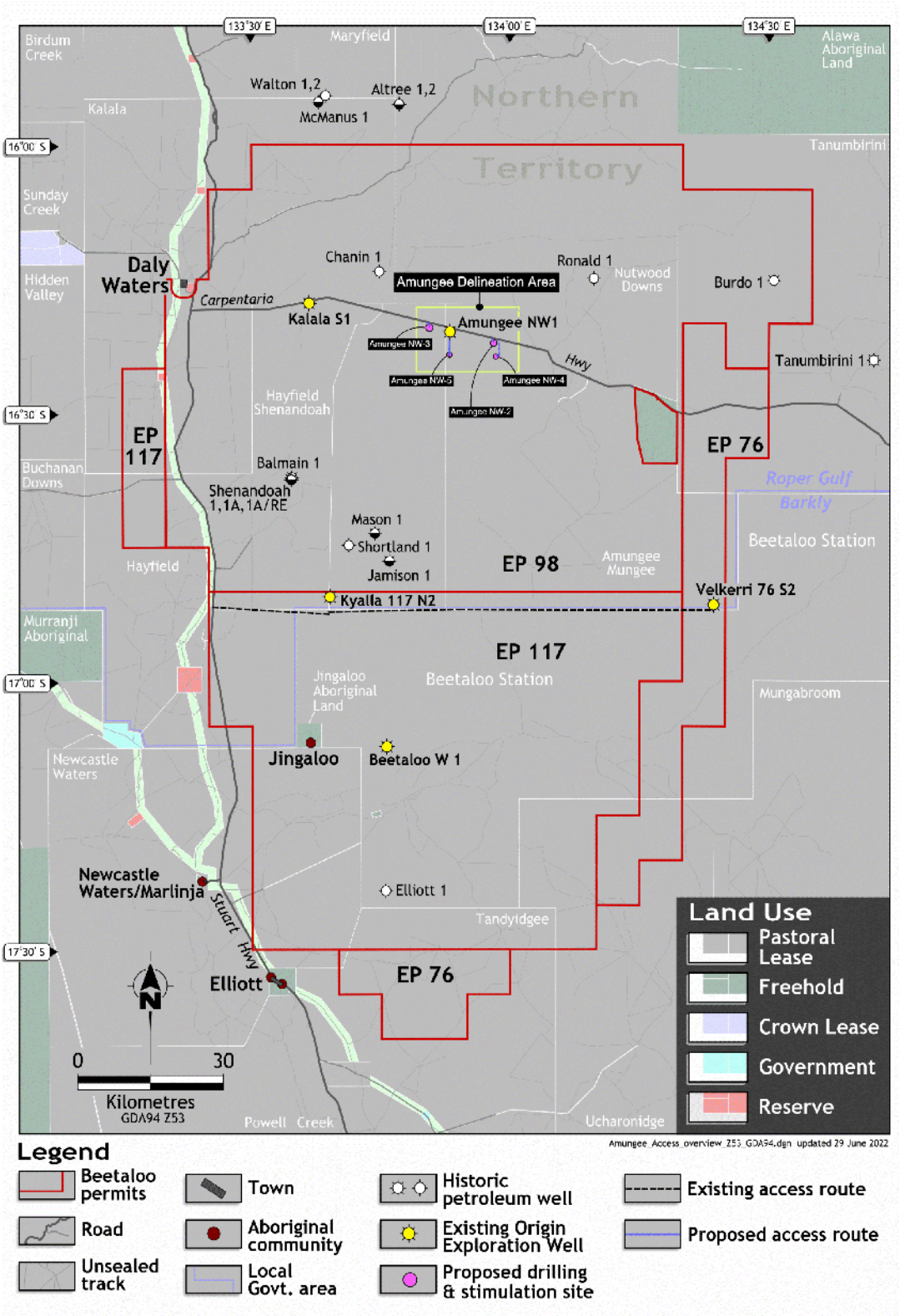


Figure 2 Location of Origin Exploration Lease Areas

Review due: 13/09/2025

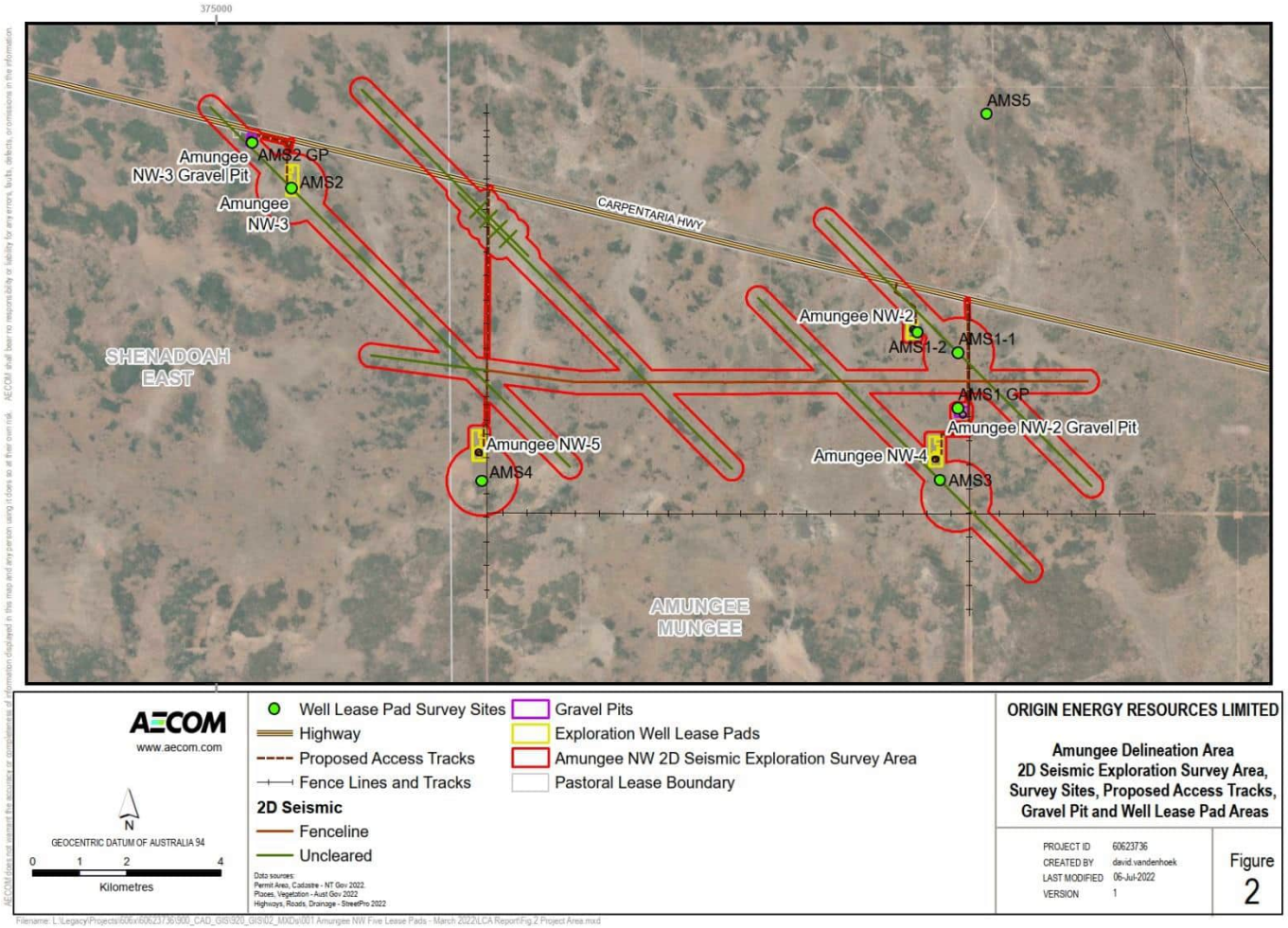


Figure 3 Location of Origin Amungee Seismic Survey Area

Review due: 13/09/2025

The primary activities subject to this ESCP are:

- Construction and or ongoing maintenance of exploration well pads, camp pads, stockpile areas, helipad and wet weather storage area.
- Minor intersection upgrade works at the intersection with the Stuart Highway and Carpentaria Highway in accordance with approved Road Agency approval (2018-0186-D2) and Permit to Work within NT Government Road Reserve.
- Seismic line preparation, data collection and rehabilitation.
- Construction and ongoing maintenance of access tracks.
- Gravel extraction, as required, for construction and maintenance of drill pads and sections of the access tracks.
- All other activities ancillary to the seismic survey and drilling, stimulation and well testing of an exploration well.

2.1 Legislation

The activities outlined within the EMP, which this management sub-plan is a component of, aim to comply with relevant guidelines associated with exploration activities, such as *International Erosion Control Association (IECA) Best Practice for Erosion and Sediment Control (2008)*, *IECA Appendix P: Land Based Pipeline Construction December 2015* (Addendum to IECA 2008), the *Australian Pipeline Industry Association Code of Environmental Practice for Onshore Pipelines 2017* and the *Code of Practice for Onshore Petroleum Activities in the Northern Territory 2019*.

2.1.1 *Code of Practice for Onshore Petroleum Activities in the Northern Territory 2019*

The *Code of Practice for Petroleum Activities in the Northern Territory* is a mandatory code of practice for the petroleum industry to ensure that petroleum activities in the Northern Territory are managed according to minimum acceptable standards to ensure that risks to the environment can be managed to a level that is as low as reasonably practical (ALARP) and acceptable.

Under these regulations Origin is required to submit an EMP prior to any petroleum exploration or production activity. The EMP for a petroleum activity must include a primary Erosion and Sediment Control Plan (ESCP) outlining all activities. This should be developed by a suitably qualified person in accordance with relevant guidelines including specific environmental outcomes and environmental performance standards to be included in the Implementation Strategy in the EMP. The ESCP must include:

- A risk assessment in relation to the potential impact to the environment from erosion and sedimentation associated with the proposed activities. Including an assessment of site-specific conditions and the nature and timing of works with the Land Clearing Guidelines as published on the Department of Environment, Parks and Water Security (DEPWS) website and any amendments.
- Where the Primary ESCP requires it, a further ESCP must be developed by a suitably qualified person in relation to the relevant matters identified in the Primary ESCP and implemented by the interest holder.
- Road and pipeline designs must:
    - minimise erosion of exposed road surfaces and drains
    - ensure that roads and pipeline surface water flow paths minimise erosion of all exposed surfaces and drains
    - Comply with legislative requirements.
- The requirements of the Land Clearing Guidelines as published on the DEPWS website and amended from time to time must be complied with in relation to protection of natural waterways as a result of land disturbance and ensure the following:
    - appropriate buffers are implemented around natural waterways

- disturbance in the wet season is minimised
- the number of crossing points is minimised
- crossings are established as close as practicable to right angles to the waterway
- material changes in the shape of the waterway are avoided
- material changes in the volume, speed or direction of flow or likely flow of water in the waterway are avoided
- alteration to the stability of the bed or banks of the waterway (including by removal of vegetation) is avoided
- erosion risk, sedimentation and pollution of waterways is minimised through the appropriate design and implementation of best practice erosion and sediment control measures.

3. Aim and Objective

The ESCP aims to:

- Address key soil and water management issues, including legislative and client requirements.
- Determine the "Type" of ESC to be implemented during construction, post construction and until exploration activities are completed.
- Where practical identify, eliminate and reduce hazards and associated risks inherent in specific work activities, which if untreated could lead to a diminished product or create the potential for an accident, dangerous occurrence or environmental incident.

The objective of this ESCP is to manage Origin's activities within the Permit Area in a manner that minimises the impacts upon soil, vegetation and surface water which may result from soil disturbance activities including seismic line preparation, land clearing associated with well pad establishment.

This ESCP may be amended as required, in response to the monitoring and maintenance programs described herein to avoid significant and/or sustained deterioration in downstream water quality. Standard drawings are provided as a guide, with the Construction Supervisor and Origin Engineers making final determination on site.

Strategies shall be developed, implemented and reviewed on a regular basis, so that risks are identified, measured and recorded throughout the course of the project.

Due to potential chance for activities to lead up to the wet season, wet weather contingencies have been identified in this plan and the overarching EMP (BOM, 2012). It is anticipated that due to the known ground conditions across the region, ground conditions following rainfall events can make access impossible. The primary mitigation will be to monitor weather forecasts daily during the program and where rainfall is likely to result in an event that has potential to limit access, the subcontractor will stabilise the current work areas and go into standby mode until such time can assess the track conditions to recommence activities.

Further strategies will be developed, implemented and reviewed on a regular basis so that risks are identified, measured and recorded throughout the course of exploration activities. Any substantial changes to the ESCP will be subject to review and approval by the Department of Environment, Parks and Water Security (DEPWS) Land Management Team.

3.1 Compliance with IECA Guideline

The ESCP has been prepared by suitably qualified and experienced personnel that understand the intent and minimum standards of IECA. The team that prepared the plan consist of the following:

- Alana Court – BEnvSci, PGDipEnvMgt. Principal Environmental Scientist with over 18 years' experience and completed the IECA erosion and sediment control training (2013). Over 20 years' experience providing advice to managing environmental requirements in the Beetaloo Basin including erosion and sediment control.
- James Jentz – BEng, RPEQ, CPEng. Civil Engineer with over 30 years' experience in the design and documentation of civil engineering projects. James has signed off all civil drawings under his qualification.

4. Civil Construction Schedule

The Exploration schedule for Origin's activities for the 2022/2025 exploration period will primarily occur from April each year extending into October while rainfall risk rating is considered very low (0 to 30 mm).

Implementation of the ESCP will commence as soon as access is granted and continued throughout the exploration activities until such time that the site is stabilised.

In the event that exploration activities continue through to the wet season, Origin will implement the wet weather contingency planning.

This revision will occur during October for approval by DEPWS Land Management Team and will be implemented between 1 November to the 31 March, based on the rainfall conditions in that permit area.

5. Permit Area Erosion Susceptibility

Erosion susceptibility varies throughout the Origin permit area, dependent upon the soil types, slope and extent of ground disturbance. Apart from the erosive impact of climatic conditions, soil erosion is influenced mainly by the inherent properties of the soils and the processes which occurred during the formation of the landscapes.

Erosion will occur in the permit area if the land is used beyond its capacity, as is seen if land is overstocked or vehicle movements not controlled, for example. The locations of the exploration lease areas for 2022/2025 program have been examined in the field to determine the risk of erosion occurring from Origin activities.

Factors considered include the following:

- Season (R Factor) – the timing of the project works will occur mostly within the dry season of the NT, which has low amounts of rainfall and is considered a low-risk factor. Risk levels of rainfall data of Daly Waters and Newcastle waters can be seen in Table 2 and Table 3 which present the erosion risk rating based on average monthly rainfall using the rating system provided in the IECA (2008) Table 4.4.2 for Daly Waters (northern sites) and Newcastle Waters (southern sites).

Table 2 Erosion Risk Rating based on average monthly rainfall at Daly Waters

-Item	Jan	Feb	Mar	Apr	May	June	Jul	Aug	Sep	Oct	Nov	Dec
Rainfall (mm)	165.4	165.4	120.1	23.6	5.0	5.6	1.5	1.7	4.9	22.5	59.4	110
Erosion Risk*	H	H	H	VL	VL	VL	VL	VL	VL	VL	M	H

Table 3 Erosion Risk Rating based on average monthly rainfall at Newcastle Waters

Item	Jan	Feb	Mar	Apr	May	June	Jul	Aug	Sep	Oct	Nov	Dec
Rainfall (mm)	125.5	130.9	93.7	24.6	9.3	5.3	3.4	1.0	5.4	20.9	35.7	77.3
Erosion Risk*	H	H	M	VL	VL	VL	VL	VL	VL	VL	L	M

* E = Extreme (>225 mm); H = High (100+ to 225 mm); M = Moderate (45+ to 100 mm); L = Low (30+ to 45 mm); VL = Very Low (0 to 30 mm)

- Soil type (K Factor) – soils with higher clay content are prone to generation of bulldust and are easily eroded by wind and water. Gravelly soils tend to be more robust to disturbance on the scale expected for Origin exploration activities. The primary soil type encountered across the permit can generally be described as silty SAND, SM with some gravel. These soils are considered to have a low to medium erodibility potential when the soils are disturbed.
- Slope length – the slope of the exploration area is one of the characteristics that will help to determine the risk of erosion during rainfall events, with steeply inclined areas a higher risk than small undulations in the landform. The Origin exploration areas subject to this ESCP are generally flat with a slope of <1%. There are some slight undulations that occur throughout the area, generally being less than 2% gradient, however some areas are known to be in excess of 2%. Treatments are defined for sections less than 2% and greater than 2% in this plan. The relevant treatment will be considered on a case by case basis.
- Aspect – the position of the seismic lines, access tracks and pads in relation to the direction of the contour should be considered and creation of tracks and the lease pads across (as opposed to parallel with) the contour should be avoided.
- Groundcover – clearing will be conducted to construct access tracks, establish gravel pits and earthworks relating to construction of the exploration well pad and associated camps, as well as line preparation for seismic exploration. The method that will be used for seismic line preparation will consist of dozer and grader, ensuring that topsoil and root stock is retained.

The clearing method used will consist of a dozer to initially clear vegetation and then dozer or grader to strip topsoil, ensuring that rootstock is retained in the stockpiled topsoil. Expected machinery includes grader, 4W loader, tip truck, water truck, water tanks, excavators and compactors.

- Drainage line crossings – potential for minor drainage lines to occur across the exploration area. Generally these can be trafficable with minimal modification of the creek bed required.

5.1 Erosion Hazard Assessment for EP76, EP98 and EP117

5.1.1 Erosion Hazard Assessment for EP76, EP98 and EP117 – Lease Pads, Access Tracks

An Erosion Hazard Assessment for all sites subject to this ESCP has been completed to inform the specific issues and actions that will be required for conducting activities within the permit areas. Table 4 presents the results of the assessment for exploration well lease pads. The IECA (2008) Explanatory Notes for the assessment are presented Appendix A.

Table 4 Erosion Hazard Assessment for EP76, EP98 and EP117

Condition (as described by IECA, 2008)	Points	Erosion Hazard Score					Trigger value
		Amungee Delineation Area	Kalala S1	Kyalla 117 N2	Velkerri 76 S2	Beetaloo W	
AVERAGE SLOPE OF DISTURBANCE AREA [1]							
• not more than 3% [3% ≙ 33H:1V]	0	**0**	**0**	**0**	**0**	**0**	4
• more than 3% but not more than 5% [5% = 20H:1V]	1	Comment - Topographical survey of lease areas indicated (low relief) with a slope <1% (refer Appendix B)					
• more than 5% but not more than 10% [10% = 10H:1V]	2						
• more than 10% but not more than 15% [15% ≙ 6.7H:1V]	4						
• more than 15%	6						
SOIL CLASSIFICATION GROUP (AS1726) [2]							
• GW, GP, GM, GC	0	**2**	**2**	**2**	**2**	**2**	-
• SW, SP, OL, OH	1	Comment – Geotechnical testing indicated SM - Silty sands, poorly graded sand-silt mixtures (refer Appendix C).					
• SM, SC, MH, CH	2						
• ML, CL, or if imported fill is used, or if soils are untested	3						
EMERSON (DISPERSION) CLASS NUMBER [3]							
• Class 4, 6, 7, or 8	0	**0**	**0**	**0**	**0**	**0**	6
• Class 5	2	Comment – Class 4 – Sand Material, therefore Emerson test not applicable.					
• Class 3, (default value if soils are untested)	4						
• Class 1 or 2	6						
DURATION OF SOIL DISTURBANCE [4]							
• not more than 1 month	0	**2**	**2**	**0**	**0**	**0**	6
• more than 1 month but not more than 4 months	2	Comment – Clearing and earthworks are expected to be between 1 and 4 months.					
• more than 4 months but not more than 6 months	4						
• more than 6 months	6						

Condition (as described by IECA, 2008)	Points	Erosion Hazard Score					Trigger value
		Amungee Delineation Area	Kalala S1	Kyalla 117 N2	Velkerri 76 S2	Beetaloo W	
AREA OF DISTURBANCE [5]							
• not more than 1000 m2	0	**6**	**6**	**6**	**6**	**6**	6
• more than 1000 m2 but not more than 5000 m2	1	Comment – All exploration lease areas are greater then 4 ha but less then 10 ha of disturbance.					
• more than 5000 m2 but not more than 1 ha	2						
• more than 1 ha but not more than 4 ha	4						
• more than 4 ha	6						
WATERWAY DISTURBANCE [6]							
• No disturbance to a watercourse, open drain or channel	0	**0**	**0**	**0**	**0**	**0**	2
• Involves disturbance to a constructed open drain or channel	1	Comment – Not in close proximity to natural water courses (refer Appendix D).					
• Involves disturbance to a natural watercourse	2						
REHABILITATION METHOD [7] Percentage of area (relative to total disturbance) revegetated by seeding without light mulching (i.e. worst-case revegetation method).							
• not more than 1%	1	**1**	**1**	**1**	**1**	**1**	-
• more than 1% but not more than 5%	2	Comment – Top soil replaced along batters to commence assisted natural regeneration.					
• more than 5% but not more than 10%	3						
• more than 10%	4						
RECEIVING WATERS [8]							
• Saline waters only	0	**2**	**2**	**2**	**2**	**2**	-
• Freshwater body (e.g. creek or freshwater lake or river)	2	Comment – not located within the major flow pathway (refer to flood assessment in the Amungee Delineation Area Land Condition Assessment).					
SUBSOIL EXPOSURE [9]							
• No subsoil exposure except of service trenches	0	**0**	**0**	**0**	**0**	**0**	-
• Subsoils are likely to be exposed	2						
EXTERNAL CATCHMENTS [10]							
• No external catchment	0	**1**	**1**	**1**	**1**	**1**	-
• External catchment diverted around the soil disturbance	1	Comment – refer to Civil Design Drawings (Appendix E to Appendix M)					
• External catchment not diverted around the soil disturbance	2						
ROAD CONSTRUCTION [11]							
• No road construction	0	**2**	**2**	**2**	**2**	**2**	-

Condition (as described by IECA, 2008)	Points	Erosion Hazard Score					Trigger value
		Amungee Delineation Area	Kalala S1	Kyalla 117 N2	Velkerri 76 S2	Beetaloo W	
• Involves road construction works	2						
pH OF SOILS TO BE REVEGETATED [12]							
• more than pH 5.5 but less than pH 8	0	**0**	**0**	**0**	**0**	**0**	-
• other pH values, or if soils are untested	1	Comment – Soil pH 5.5 to 8.0					
Total Score [13]		**16**	**16**	**16**	**16**	**16**	
For guidance purposes only: [13] A primary ESCP must be submitted to the local government for approval during the planning phase for any development that obtains a total point score of 17 or greater or when any trigger value is scored or exceeded							

5.1.2 Erosion Hazard Assessment for EP98 – 2D Seismic Survey

Table 5 presents the results of the assessment for the 2D seismic program.

Table 5 Erosion Hazard Assessment for EP76, EP98 and EP117

Condition (as described by IECA, 2008)	Points	Score EP98 Seismic Survey	Trigger value
AVERAGE SLOPE OF DISTURBANCE AREA [1]			
• not more than 3% [3% = 33H:1V]	0	**1**	4
• more than 3% but not more than 5% [5% = 20H:1V]	1	**Comment** - Topographical data of lease areas indicated (low relief) with a slope <1-2%. Isolated areas increase to 3% to 5%. Value of 1 adopted as worse as scenario.	
• more than 5% but not more than 10% [10% = 10H:1V]	2		
• more than 10% but not more than 15% [15% = 6.7H:1V]	4		
• more than 15%	6		
SOIL CLASSIFICATION GROUP (AS1726) [2]			
• GW, GP, GM, GC	0	**2**	-
• SW, SP, OL, OH	1	**Comment** – Initial soil testing during the baseline survey indicated SM - Silty sands, poorly graded sand-silt mixtures (refer EMP).	
• SM, SC, MH, CH	2		
• ML, CL, or if imported fill is used, or if soils are untested	3		
EMERSON (DISPERSION) CLASS NUMBER [3]			
• Class 4, 6, 7, or 8	0	**6**	6
• Class 5	2	**Comment** – Class 2 – Specific testing for Emerson Class not conducted. Therefore, default value used.	
• Class 3, (default value if soils are untested)	4		
• Class 1 or 2	6		
DURATION OF SOIL DISTURBANCE [4]			
• not more than 1 month	0	**2**	6
• more than 1 month but not more than 4 months	2	**Comment** – Line preparation to rehabilitation will be less than 1-month duration, however worst-case allowance used.	
• more than 4 months but not more than 6 months	4		
• more than 6 months	6		

Condition (as described by IECA, 2008)	Points	Score EP98 Seismic Survey	Trigger value
AREA OF DISTURBANCE [5]			
• not more than 1000 m^2	0	**1**	6
• more than 1,000 m^2 but not more than 5,000 m^2	1	**Comment –** Due to the tread lightly approach of the line preparation using existing tracks and minimising tree and shrub clearing and the re-instatement of topsoil and vegetation as soon as possible after acquisition, results in no more than 5,000 m^2 assessed at any one time.	
• more than 5,000 m^2 but not more than 1 ha	2		
• more than 1 ha but not more than 4 ha	4		
• more than 4 ha	6		
WATERWAY DISTURBANCE [6]			
• No disturbance to a watercourse, open drain or channel	0	**2**	2
• Involves disturbance to a constructed open drain or channel	1	**Comment –** Activities require crossing of some minor drainage lines. Not considered to be major works and will be re-instated as completion of acquisition	
• Involves disturbance to a natural watercourse	2		
REHABILITATION METHOD [7] Percentage of area (relative to total disturbance) revegetated by seeding without light mulching (i.e. worst-case revegetation method).			
• not more than 1%	1	**1**	-
• more than 1% but not more than 5%	2	**Comment –** Topsoil and vegetated material to be replaced over disturbance within 2 weeks post activity for natural regeneration.	
• more than 5% but not more than 10%	3		
• more than 10%	4		
RECEIVING WATERS [8]			
• Saline waters only	0	**2**	-
• Freshwater body (e.g. creek or freshwater lake or river)	2	**Comment –** Minor drainage lines, with no flowing water at time of acquisition.	
SUBSOIL EXPOSURE [9]			
• No subsoil exposure except of service trenches	0	**0**	-
• Subsoils are likely to be exposed	2		
EXTERNAL CATCHMENTS [10]			
• No external catchment	0	**0**	-
• External catchment diverted around the soil disturbance	1	**Comment –** Not considered applicable based on the activities being completed are temporary seismic lines.	
• External catchment not diverted around the soil disturbance	2		
ROAD CONSTRUCTION [11]			
• No road construction	0	**0**	-
• Involves road construction works	2	**Comment –** only temporary seismic lines required. No construction of new tracks is necessary. Existing pastoral tracks to be treated post activity.	
pH OF SOILS TO BE REVEGETATED [12]			
• more than pH 5.5 but less than pH 8	0	**0**	-
• other pH values, or if soils are untested	1	**Comment –** Majority soils recorded within Soil pH range 5.5-8 across exploration area. Some areas recorded outside range but considered minimal risk to seismic program.	

Condition (as described by IECA, 2008)	Points	Score	Trigger value
		EP98	
		Seismic Survey	
Total Score [13]		17	
For guidance purposes only: [13] A primary ESCP must be submitted to the local government for approval during the planning phase for any development that obtains a total point score of 17 or greater or when any trigger value is scored or exceeded.			

The Erosion Hazard Assessment for the Origin permit area for the 2022/2025 program all report equal or just below the point score of 17. Based on the trigger value being met the ESCP is required.

5.2 Soil Loss Estimate

IECA (2008) soil loss estimation has been used to determine the type of controls the project should adopt to limit soil loss during construction when soils are exposed to rainfall. Long term average soil loss resulting from sheet and rill flow can be predicted using the Revised Universal Soil Loss Equation (RUSLE).

Soil loss calculated using RUSLE for the project area was calculated as follows:

A = R . K . LS . C . P

Where A = annual soil loss due to erosion [tonnes/hectare/year (t/ha/yr)]

R = rainfall erosivity factor based on = **6297**)

K = soil erodibility factor of **0.055** for silty sand)

LS = topographic factor derived from slope length and slope gradient (**0.24**)

C = cover and management factor (**1**)

P = erosion control practice factor (**1.3**)

It is noted that the **annual R-factor of 6297** for the Katherine Region has been adopted as per comment received by DEPWS Land Management team. Since preparation of the initial ESCP, additional geotechnical information has been obtained which provides a larger sample size of the proposed permit areas. The geotechnical sampling completed on the sites has shown that the top 0.3 m of the site is “Silty Sand”. As such, the K-factor has been revised to 0.055 for “Silty Loam” from Table E4 of the IECA Guidelines.

Revision of the LS-factor on more detailed design drawings shows a total slope length of approx. 200 m at a gradient of 0.00120 m/m (0.12%), indicative of the gradients across both sites. A LS factor of 0.24 was adopted, indicating a 200 m slope at 0.01 m/m (1%).

Based on the reviewed RUSLE soil loss methodology, **the Annual Soil Loss estimate using these values is 108 t/ha/yr**. Type 3 sediment controls are adequate with the revision to the RUSLE equation. In addition, Type 2 controls have been allowed for in design including settlement pond on the drill pads and rock filter dams at the Stuart Highway Intersection.

All the proposed activities for the exploration program are planned during the dry season (July to October) when the erosion risk rating for rainfall is very low (refer to Table 2 and Table 3. Where activities occur outside, Origin’s Wet Weather Contingency Plan will be implemented.

5.3 Erosion Risk and Determination of ESC

Erosion risk ratings for the Project area have been determined based on the average monthly erosivity (R-factor of 6297), average monthly rainfall depth (mm) (refer Table 2 and Table 3 above) and soil loss (estimated at 108t/ha/yr). As indicated in Table 6, the Project has an erosion risk rating of “very low” to “extreme”.

Table 6 Erosion Risk Rating (adapted from IECA, 2008, Tables 4.4.1, 4.4.2 and 4.4.3)

Erosion Risk Rating	Average Monthly Erosivity (R-Factor)	Average Monthly Rainfall Depth (mm)	Soil Loss (t/ha/yr)
Very Low	0 to 60	0 to 30*	0 to 150

Erosion Risk Rating	Average Monthly Erosivity (R-Factor)	Average Monthly Rainfall Depth (mm)	Soil Loss (t/ha/yr)
Low	60+ to 100	30+ to 45	150+ to 225
Moderate	100+ to 285	45+ to 100	225+ to 500
High	285+ to 1,500	100+ to 225	500+ to 1,500
Extreme	>1,500*	>225	>1,500

** It is noted that the monthly erosivity factor would only be triggered during rainfall events. The construction period is proposed to occur from July to October and based on assessment of the average monthly rainfall for the region (refer* Table 2 and Table 3*), the erosion risk rating is considered very low (0 to 30mm during this time). It is anticipated that at completion of construction the site would be stabilised for normal operation.*

Table 7, provides an indication of the "Type" of erosion and sediment controls that should be deployed during construction depending on annual soil loss. Based on the proposed construction schedule during the dry season, the Project is determined to trigger the use of Type 3 erosion and sediment controls, with some Type 2 controls allowed for in design including settlement pond on the drill pads and rock filter dams at the Stuart Highway Intersection.

Table 7 Sediment Control Standard (adapted from IECA, 2008, Table 4.5.1)

Catchment Area (m^2)	Soil Loss Rate Limit (t/ha/yr)		
	Type 1	Type 2	Type 3
250	N/A	N/A	All Cases
1000	N/A	N/A	All Cases
2500	N/A	>75	75
>2500	>150	150	75

Table 8 provides a range of erosion and sediment controls that can be deployed on the Project for each 'Erosion and Sediment Control Type'.

Table 8 Classifications of Sediment Controls

Type 1	Type 2	Type 3
Sheet Flow		
Buffer Zone capable of infiltrating 100% of stormwater runoff or processed water Infiltration basin or sand filter bed capable of infiltration of 100% of flow	Buffer Zone capable of infiltrating 100% of stormwater runoff **Compost/Mulch Berm**	**Buffer Zone capable of infiltrating 100% of stormwater runoff** Filter Fence Modular Sediment Trap Sediment Fence
Concentrated Flow		
Sediment basin (sized in accordance with design standard)	Sediment Basin (smaller than the design standard) Filter Tube Dam Rock Filter Dam Sediment Trench Sediment Weir	Coarse Sediment Trap Modular Sediment Trap U-shaped Sediment Trap
Dewatering Sediment Control		
Type F/D Sediment Basin Stilling Pond	Filter Bag or Filter Tube Filter Pond	Compost Berm Filter Fence

Type 1	Type 2	Type 3
	Filter Tube Dam Portable Sediment Tank Settling Pond Sump Pit	Grass Filter Bed Hydrocyclone Portable Sediment Tank Sediment Fence
In-stream sediment control		
Pump sediment laden water to an off-stream Type F/D Sediment Basin or high filtration system	Filter Tube Barrier Modular Sediment Barrier Rock Filter Dam Sediment Weir	Modular Sediment Barrier Sediment Filter Cage

The ESCPs are provided in **Appendix E to Appendix M**.

Standard drawings that may be applicable for the Project, including controls for access tracks and stream crossings are provided in **Appendix N**. The final design of the ESC controls will be dependent on decisions made in the field by the Supervising Engineer and site conditions. Any significant changes to those identified in this ESCP will be reported through to DEPWS Land Management Team for review and approval. Origin and its civil contractors will be responsible for notifying of any changes.

Standard drawings for erosion and sediment controls are available at:
http://www.austieca.com.au/publications/book-6-standard-drawings.

5.3.1 Modifying the ESC Measures

It is possible that some ESC measures will require modification as the project is constructed and in response to the performance of ESC measures or changes in project circumstances. The modifications may be considered minor, moderate or significant. Moderate and minor changes will occur, and it is expected that significant modifications will be the exception. If significant erosion events occur, significant changes to the measures used will be required and should be approved by a CPESC or suitably qualified consulting engineer.

To accommodate the range of circumstances likely to occur, a change management decision matrix is presented in Table 9. Where changes are required these will be risked assessed through a change management process and kept in a change management register.

Table 9 Change management decision matrix

Authority required	Minor	Moderate		Significant
	Maintenance of all measures	Removal or relocation of minor temporary controls	Permanent measure relocation	Permanent measure removal/revisions to ESCP
Origin Onsite Company Rep	✓	×	×	×
Site Supervisor	-	✓	×	×
CPESC	-	-	✓	✓
Consulting Engineer	-	-	✓	✓

✓ Authorised to undertake.
× Not Authorised to undertake.
- Denotes that authority level is not required

Examples of different types of sediment controls can be seen in Table 7. Examples of minor temporary controls would fall under Type 3 Sediment Controls while Type 2 Sediment Controls provide examples of permanent measures.

It is noted that minor and permanent are not indications of how long the sediment controls are in place. At completion of the activities, the disturbed areas to be restored and/or rehabilitated to pre-disturbed conditions consistent with the surrounding land use.

If ESC measures are observed to be ineffective (*e.g.* obvious sediment deposition has occurred, or is occurring in a waterway), the source of the sediment must be identified, and corrective ESC measures implemented.

6. Erosion and Sediment Controls

6.1.1 Well Exploration Areas

Based on the erosion susceptibility of the exploration area, the ESCP measures to be adopted for the 2022/2025 program are summarised in Table 10 below. These ESCP measures have been considered during the design of the exploration well pads and associated infrastructures and will be implemented by the Origin Contractors during the construction and maintenance activities.

Table 10 Measures to be implemented for Erosion and Sediment Control

Activity	Management Controls
Land Clearing	- Undertake selective clearing (only clearing areas that are necessary for construction and ESC activities), using lighter machinery such as graders or smaller bulldozers, taking care not to overwork the site. Overworking the site can lead to the loss of topsoil, compaction, formation of windrows and wheel rutting. - Minimise tree clearing activities only during the dry season (April to October) to allow the ground surface to stablise before the onset of the wet season (November to March). - Retain vegetation buffers surrounding streams and creeks, as outlined in the *NTG Land Clearing Guidelines 2010*. - Undertake clearing for each stage in small units over time, keeping the disturbed areas small and time of exposure short, in conjunction with progressive re-vegetation (assisted natural regeneration using available topsoil). - Take all reasonable and practicable measures to minimise the removal of, or disturbance to, trees, shrubs and ground covers (organic or inorganic) that are to be retained. - If bulk tree clearing is required, it must occur in a manner that minimises disturbance to existing ground cover (organic or inorganic). - Bulk tree clearing and grubbing of the site must be immediately followed by specified temporary stabilisation measures (e.g. gravel, soil berm) prior to commencement of each stage of construction works. - Land clearing should not occur unless preceded by the installation of appropriate drainage and sediment control measures. The exception would be any land clearing necessary to allow installation of these control measures. Prior to land clearing, establish tree protection zones around vegetation to be retained e.g. identify with high-visibility tape, or light fencing. - All land clearing must be in accordance with the Federal, Territory and local government vegetation clearing requirements and IECA Table 4.4.7 Best practice land clearing and rehabilitation requirements. - All reasonable and practicable steps to be taken to apply best practice Erosion control measures following earthworks and site stabilised prior to anticipated rainfall. Disturbed areas will be stabilised with a minimum 60% cover within 30 days of completion if rainfall is reasonably possible.

Activity	Management Controls
Access Tracks	- Where possible, use existing roads and tracks to access the lease areas, and where new tracks are required, they should be located along the most direct and practicable route to the lease area (noting Velkerri 76 S1 access track has been diverted around the sensitive Bullwaddy/Lancewood vegetation type). - Trucks entering and exiting the site will be constrained in such a manner to prevent dropping or tracking material on the Highway in accordance with the Road Agency Approval (ref 2018-0186-D2). - Monitor Stuart Highway during construction and operation. Where tracked material on the road pavement becomes a potential safety issue, Origin and its contractors will sweep and clean material off the road. If Stuart Highway Turn-in results in dust, dirt creating hazard to road users, additional ESC will be considered including installation of shaker grid or rock pad. - Minimise track width and surface disturbance (e.g. topsoil, seed and root stock) as far as practicable to allow safe passage of required equipment. Disturbed areas will be stabilised with a minimum 60% cover 30 days of completion if rainfall possible. - Where gravelling is warranted (Stuart Highway and Carpentaria Turn-in), the formation process can remove undesirable material and/or box the imported material where it is required. Track formation will be required for the following reasons: • Drainage control, especially in areas where erosion or sediment influences are evident, any vegetation, topography, wheel rutting or compaction is likely to intercept, concentrate and channel water. • Where the topography of the track location or the drainage characteristics of the soil are likely to hinder access for a protracted time period following rain (e.g. 1 to 2 weeks). • Where natural side-slope poses a safety hazard to potential users of the track (e.g. Contractors, Land Owners). - Place scrub and vegetation cleared from the route adjacent to the route where practical to facilitate its return to the disturbed area. Where this occurs, spread the material out rather than form windrows. Allow disturbed areas to be stabilised and natural regeneration of the native grasses to occur. - Construct access tracks with table drains that are free draining. - Avoid road crowning to allow water to naturally cross the road. - Form tracks to allow off-road drainage. Where track intercepts the direction of overland flow and re-directs this flow to a non-natural drainage line, install erosion control works to minimise potential erosion. - The design and position of erosion control measures to be determined in the field by experienced operator and site engineer, based on the site characteristics of the access track location. - Where construction of table drains are deemed necessary, they should have a broad flat base at least 1 m wide and should not be graded to produce a 'V' shape. To minimise erosion, the slope should be no greater than 0.5% on erodible soils or 1% on stable soils. - Where encounter dispersive / erosive soils they should be stabilised with gypsum or other stabiliser, as determined by laboratory analysis of soils.

Activity	Management Controls
	- Where cut-out drains are required, they should be spaced based on the slope of the area i.e. 0.5% slope, allow for cut-out draining every 170-180 m or 1 % slope, allow for cut-out drainage every 120-130 m etc. (refer to NT Road Drainage Fact Sheet). It is noted that the recommended distance between turn-out drains is a guide and may not apply to all locations along the access track. - Monitor road conditions to ensure deterioration does not occur. Assist in the maintenance and repair work on roads and tracks used. - Following completion of activities and within 2 years after the surrender of a lease, the land surrounding or affected by the installation of access tracks shall be restored in accordance with the site-specific rehabilitation plan and final determination of asset (i.e. if transferring asset ownership to landholder).
Pad construction	- Pad construction to be in accordance with the typical ESCP (refer **Appendix E**). The topsoil berm dimension to be in accordance with the IECA Figure 1 Standard Drawing MB-01 presented in **Appendix N**. • Use topsoil berms to divert upstream runoff from undisturbed areas ('clean' water) around and away from disturbed areas, and back to the environment. • Use topsoil berms to contain / manage runoff from disturbed construction areas ('dirty' water) and prevent release to environment without treatment. • Treat runoff from construction areas through suitable sediment controls (e.g. sediment traps). • Configure berms so that upstream runoff does not mix with construction area runoff prior to treatment of construction area runoff. - Where topsoil stripping is required, the stripping depth would be in accordance with Technical Instruction (NT-2050-15-TI-0001) and amelioration rates agreed with the Construction Supervisor, Origin engineers and by a suitably qualified ESC practitioner. It is noted that the expected nominal depth of topsoil across the lease pads at both locations range from <100 mm to 150 mm. Final strip depth will be confirmed in the field. Any changes to the adopted ESCs will be reflected in the ESCP and to satisfaction of DEPWS. - Stockpiled felled trees nearby for future use in rehabilitation. - Inspect on a regular basis in accordance with Section 5 Maintenance. - Damage or maintenance is undertaken by an appropriately qualified person i.e. contractor / Origin. - Following completion of activities and within 2 years after the surrender of a lease, the land surrounding or affected by the exploration wells shall be restored in accordance with the site-specific rehabilitation plan and final determination of asset (i.e. if transferring asset ownership to landholder).
Stream and Creek Crossings	Where a crossing is required to be upgraded, a bed level crossing as detailed in **Appendix L**, will be installed in accordance with the following: - Crossings will be aligned perpendicular to the water flow. - Crossing will be constructed from clean rocks (minimal fine material) that are an equivalent or larger size than the natural bed material at the crossing. - The surface is to be left rough and not to be over compacted (e.g. track-rolled finish or rougher).

Activity	Management Controls
	- The lowest point of the bed level crossing will be installed at the level of the lowest point of the natural stream bed (preconstruction), within the footprint of the proposed crossing. - There must be a height difference of at least 100 mm up to ≤ 300mm from the lowest point of the crossing to the edges of the low flow section of the crossing. Where scour protection is required: - Scour protection must abut the surface edge of the crossing at the same level (this is to ensure that there is no drop in elevation at the join). - If the crossing is set below bed level then the surface of the scour protection must also be below bed level. - The stream bed must abut the scour protection at the same level (this is to ensure that there is no drop in elevation at the join). - The scour protection is installed at a gradient no steeper than 1 in 20 or the natural channel gradient, whichever is steeper. - Scour protection must incorporate a low flow channel. Use clean rocks (minimal fine material), at least 100 mm diameter. - Ensure the rock armouring is not over compacted but left at the same level and uneven (track-rolled finish or rougher). - Use clean rocks (minimal fine material), at least 100 mm diameter. - The retention of vegetation buffers, as outlined in the NTG Land Clearing Guidelines 2019, as they relate to stream order has been considered for the siting of proposed access tracks and pads. - Site specific progressive ECP's should be approved by DEPWS prior to any disturbance. - Should activities pushout to the wet season, the ESCP to be reviewed and updated for Wet Season conditions. The revision to be reviewed and approved by DEPWS during October to allow implementation of the plan prior to the onset of the wet season. Wet season ESCP to be implemented between 1 November to 31 March.
Soil and Stockpile Management	- Stockpile existing topsoil, where available, so that it can be reused on the site for ESC and future rehabilitation at completion of project. - Stockpiles of erodible material that has the potential to cause environmental harm if displaced, must be: (i) Appropriately protected from wind, rain, concentrated surface flow and excessive up-slope stormwater surface flows. (ii) Located at least 2m from any hazardous area or retained vegetation. (iii) Located up-slope of an appropriate sediment control system. (iv) Provided with an appropriate protective cover (synthetic or vegetative) if the materials are likely to be stockpiled for more than 28 days. (v) Provided with an appropriate protective cover (synthetic or vegetative) if the materials are likely to be stockpiled for more than 10 days during those months that have an erosion risk rating higher than medium. - A suitable flow diversion system must be established immediately up-slope of a stockpile of erodible material that has the potential to cause environmental harm if displaced, if the up-slope catchment area draining to the stockpile exceeds 1,500m^2.

Activity	Management Controls
	- Avoid creating windrows. Do not create windrows across creeks, use rollers when putting in tracks in preference to dozers, or walk the dozer with the blade raised off the ground.
Site Management	- All disturbed areas identified as very low, low, medium or high erosion risk must be suitably stabilised prior to anticipated rainfall, from the day that soil disturbances on the area have been finalised- IECA Table 4.4.7. - Tracks to be regularly inspected for early signs of compaction, erosion and soil degradation (generation of bulldust). Ongoing maintenance and repair work should be implemented as required on tracks. - No off-lease or off-road driving. - The construction schedule must aim to minimise the duration that any and all areas of soil are exposed to the erosive effects of wind, rain and surface water flow. - Land-disturbing activities must: (i) allow stormwater to pass through the site in a controlled manner and at non-erosive flow velocities. (ii) minimise soil erosion resulting from rain, water flow and/or wind. (iii) minimise adverse effects of sediment runoff, including safety issues. (iv) prevent, or at least minimise, environmental harm resulting from work-related soil erosion and sediment runoff. (v) ensure that the value and use of land/properties adjacent to the site (including access roads) are not diminished as a result of the adopted ESC measures. - Additional and/or alternative ESC measures must be implemented in the event that unacceptable off-site sedimentation is occurring as a result of the work activities. - Sediment deposited off the site as a direct result of an on-site activity, must be collected and the area appropriately rehabilitated as soon as reasonable and practicable, and in a manner that gives appropriate consideration to the safety and environmental risks associated with the sediment deposition.
Drainage Control	- Where reasonable and practicable, stormwater runoff entering the site, must be diverted around or through the area in a manner that minimises soil erosion and the contamination of water for all discharges. - All reasonable and practicable measures must be implemented to control flow velocities a manner that prevents soil erosion along drainage paths and at the entrance and exit of all drains and drainage pipes during storms up to the relevant design storm discharge. - Where reasonable and practicable, all waters discharged during construction must discharge onto stable land, in a non-erosive manner.
Erosion Control	- If synthetic reinforced erosion control mats or blankets are required, they must not be placed in, or adjacent to, riparian zones and watercourses if such materials are likely to cause environmental harm to wildlife or wildlife habitats.

Activity	Management Controls
	- A minimum 60% ground cover must be achieved on all non-completed earthworks exposed to accelerated soil erosion. If further construction activities or soil disturbances are likely to be suspended for more than 30 days during months when the expected rainfall erosivity is less than 60; • minimum 70% cover within 30 days if between 60 and 100; • minimum 70% cover within 20 days if between 100 and 285; • minimum 80% cover within 10 days if between 285 and 1,500; and • minimum 95% cover within 5 days if greater than 1,500.
Sediment Control	- Optimum benefit must be made of every opportunity to trap sediment within the work site, and as close as practicable to its source. - Sediment pond to be installed and operated to both collect and retain sediment (refer to Drawing NT-2050-15-MP-0021 and NT-2050-15-MP-022 in Appendix E). Design details of the sediment pond is provided in NT-2050-20-DD-0023. - All reasonable and practicable measures must be taken to prevent, or at least minimise, the release of sediment from the site. - Sediment control devices must be de-silted and made fully operational as soon as reasonable and practicable after a sediment-producing event, if the device's sediment retention capacity falls below 75% of its design retention capacity. - Materials removed from sediment control devices must be disposed of in a manner that does not cause ongoing soil erosion or environmental harm.
Wet weather contingency	- 7-day forecast from the Bureau of Meteorology (BOM) to be monitored and the civil and water bore construction activities planned around the forecasts. - Where forecasts indicate rainfall is likely to result in an event that has potential to limit access to the work area, the civil and water bore contractor will stabilise the current work areas and go into standby mode until such time they can assess the track condition after an event to recommence activities. - Emergency response – a post-rainfall/flood damage reconnaissance and assessment will be undertaken as soon as the area becomes accessible. Any damage observed would be repaired as soon as practicable after the event and ensure the controls and measures are in place prior to the next rainfall event.
Site Rehabilitation	- Following completion of works, disturbed areas are to be restored and/or rehabilitated. - Gravel pits to have topsoil returned and re-profiled. - All compacted areas will be ripped and scarified to promote regeneration of vegetation, this may require assistance through spread of native seed stock. - All disturbed areas will be allowed to naturally regenerate or be revegetated on completion of use. - Compacted areas will be contour ripped to 0.5m depth where practicable. - At completion of activities, establish vegetation similar to adjacent vegetation, unless agreement with landowner for alternative use.

Activity	Management Controls
	- All disturbed areas identified as very low, low, medium or high erosion risk must be suitably stabilised prior to anticipated rainfall, from the day that soil disturbances on the area have been finalised- IECA Table 4.4.7. - Stabilise disturbed areas quickly to reduce the potential for erosion. Methods of stabilisation will be site specific and based, in part, on laboratory analysis of soils for erosive and dispersive characteristics. - Previously removed vegetation and topsoil will be uniformly re-spread over disturbed area to assist with rehabilitation process through agencies of increased infiltration and return of seed-bearing topsoil. If required, additional native seed mix from the area could be respread to speed up rehabilitation process - Windrows of debris that cannot be removed should be aligned down the contour or in a manner appropriate to avoid channelling and concentrating runoff. All other windrows are to be removed as soon as practicable. - The type of ground cover applied to completed earthworks is compatible with the anticipated long-term land use, environmental risk, and site rehabilitation measures.

6.1.2 2D Seismic Activities

Based on the erosion susceptibility of the exploration area, the ESCP measures to be adopted for the 2D Seismic exploration program are summarised in Table 11 below. These ESCP measures have been considered during the design of the seismic program and will be implemented by the Origin Contractors during the construction and maintenance activities.

Table 11 Measures to be implemented for Erosion and Sediment Control

Activity	Management Controls
Vegetation clearing	- Undertake selective clearing (only clearing areas that are necessary for surveying lines and only where an alternative route is unavoidable), using lighter machinery such as graders or smaller bulldozers, taking care not to overwork tracks. Overworking the site can lead to the loss of topsoil, compaction, formation of windrows and wheel rutting. Refer to the first dot point in the Seismic Line Preparation and access track and camp establishment/maintenance section below. - Ground surface to be stabilised before the onset of the wet season (November to March). - Undertake clearing for each stage in small units over time, keeping the disturbed areas small and exposure time short, in conjunction with progressive re-vegetation (assisted natural regeneration using available topsoil and removed vegetation). - Take all reasonable and practicable measures to minimise the removal of, or disturbance to, trees, shrubs and ground covers (organic or inorganic) that are to be retained.

<table>
<tr><th>Activity</th><th>Management Controls</th></tr>
<tr><td></td><td>- All vegetation clearing must be in accordance with the Federal, Territory and local government vegetation clearing requirements and IECA Table 4.4.7 Best practice land clearing and rehabilitation requirements detailed Appendix O.
- Best practice erosion control measures will be implemented in accordance with the ESCP following earthworks and site stabilised prior to anticipated rainfall. Disturbed areas will be stabilised in accordance with the Rehabilitation Plan.</td></tr>
<tr><td>Creek and Drainage Line Crossings</td><td>- Minimise disturbance in the riparian buffers in accordance with the stream order of the encountered drainage line in accordance with the buffers provided below:
<table>
<tr><th>Riparian class</th><th>Stream order</th><th>Minimum buffer width</th><th>Measured from</th></tr>
<tr><td>Drainage depression</td><td>Not applicable</td><td>25</td><td>The outer edge of the drainage depression, which is the extent of the associated poorly drained soils and associated vegetation</td></tr>
<tr><td>Intermittent streams</td><td>First</td><td>25</td><td>The outer edge of the riparian vegetation or levee (whichever is greater). If braided channels are present, the edge of the outer most stream channel</td></tr>
<tr><td>Intermittent streams</td><td>Second</td><td>50</td><td>As above</td></tr>
<tr><td>Creeks</td><td>Third and fourth</td><td>100</td><td>As above</td></tr>
<tr><td>Rivers</td><td>Fifth or higher</td><td>250</td><td>As above</td></tr>
</table>
- No additional material will be used for the seismic acquisition to cross over the creek crossing. Existing crossings will not be altered.
- The activities shall be completed in a manner that does not cause a:
• material change to the shape of a waterway,
• material change to the volume, speed or direction of flow or likely flow of water in or into a waterway, or
• alteration to the stability of the bed or banks of a waterway, including by removal of vegetation.
- Ongoing monitoring of creek and drainage crossing condition prior to, during and at completion of rehabilitation.
- Reinstate the original topography of the creek or drainage bed following seismic acquisition.</td></tr>
<tr><td>Seismic Line Preparation</td><td>- The method for line preparation described in the EMP is to use existing pastoral station tracks wherever practicable, or minimise the complete removal of the vegetation, with vehicles to traverse over or around the vegetation instead, leaving as much intact as possible. Assessment of the survey area indicates that in the order of 80 to 90% of the undisturbed areas will be traversed as a blade up exercise.</td></tr>
</table>

Activity	Management Controls
	- Minimising vegetation and soil disturbance is the default position for the seismic program. Wherever possible vegetation and soil shall not be disturbed when establishing survey lines (i.e. blade up). If disturbance is required, establishment of survey lines which will form a runoff channel is to be avoided. - Seismic vehicles that enter and exit the site will be constrained in such a manner to prevent dropping or tracking material on the Highway in accordance with the Road Agency Approval. - Place scrub and vegetation cleared from the route adjacent to the route where practical to facilitate its return to the disturbed area. Where this occurs, spread the material out rather than form windrows. Allow disturbed areas to be stabilised and natural regeneration of the native grasses to occur.
Site management	- All plant and equipment brought to site is to be certified a "free" of weeds, soil pathogens and pests. - All disturbed areas identified as very low, low, medium or high erosion risk must be suitably stabilised prior to anticipated rainfall, from the day that soil disturbances on the area have been finalised - IECA Table 4.4.7 in Appendix O. - Land-disturbing activities must: • allow stormwater to pass through the site in a controlled manner and at non-erosive flow velocities. Where this cannot be achieved, reference should be made to installing controls as detailed in the following section. • minimise soil erosion resulting from rain, water flow and/or wind. • minimise adverse effects of sediment runoff, including safety issues. • prevent, or at least minimise, environmental harm resulting from work-related soil erosion and sediment runoff. • ensure that the value and use of land/properties adjacent to the site (including access roads) are not diminished as a result of the adopted ESC measures. - Additional and/or alternative ESC measures must be implemented in the event that unacceptable off-site sedimentation is occurring as a result of the work activities. - Sediment deposited off the site as a direct result of an on-site activity, must be collected and the area appropriately rehabilitated as soon as reasonable and practicable, and in a manner that gives appropriate consideration to the safety and environmental risks associated with the sediment deposition.

Activity	Management Controls
Wet weather contingency	- 7-day forecast from the Bureau of Meteorology (BOM) to be monitored and the seismic exploration activities planned around the forecasts. - Where forecasts indicate rainfall is likely to result in an event that has potential to limit access to the work area, the seismic contractor will stabilise the current work areas and go into standby mode until such time they can assess the track condition after an event to recommence activities. - Emergency response - a post-rainfall/flood damage reconnaissance and assessment will be undertaken as soon as area becomes accessible. Any damage observed would be repaired as soon as practicable after the event.
Site rehabilitation	- Within 2 weeks of the activities being completed, disturbed areas are to be restored and/or rehabilitated. Reference should be made to Origin's Amungee delineation area Rehabilitation Plan 2022/25. - All compacted areas will be ripped and scarified to promote regeneration of vegetation. - All disturbed areas will be allowed to naturally regenerate or be revegetated on completion of use. - At completion of activities, establish vegetation to the standard of that registered in the pre-assessment, or better. - All disturbed areas identified as very low, low, medium or high erosion risk must be suitably stabilised prior to anticipated rainfall, from the day that soil disturbances on the area have been finalized as per the requirements of IECA Table 4.4.7 (Appendix O). - Stabilise disturbed areas quickly to reduce the potential for erosion. - Previously removed vegetation and topsoil will be uniformly re-spread over disturbed area to assist with rehabilitation process through agencies of increased infiltration and return of seed-bearing topsoil. If required, additional native seed mix from the area could be respread to speed up rehabilitation process. This will be confirmed during rehabilitation monitoring activities. - Windrows to be removed as soon as practicable. - The type of ground cover applied to completed earthworks is compatible with the anticipated long-term land use, environmental risk, and site rehabilitation measures. - At completion, the disturbed areas are to be restored and/or rehabilitated to original pre-disturbed condition consistent with surrounding landuse.

6.2 ESC Treatment Options for Specific Situations

Appendix N to Appendix P contain typical erosion and sediment control measures that are to be applied throughout the project when required. Treatments are identified for specific situations and should be applied appropriately. Five different seismic line treatments are identified below.

- Blade up areas where only wheel tracks will develop – no treatments required.
- Surface bladed by grader to smooth out ground surface to allow vehicle movements. No tree removal. Topsoil will be bladed off by grader and windrowed for later respreading at completion of data recording, to preserve the soil structure. Whoa boys or roll over banks to be provided as per details in Appendix P.
    - At the conclusion of activities, or as part of progressive rehabilitation, or the anticipated onset of a significant rainfall event which will require the site to be abandoned, topsoil would be respread and ripped into the soil surface.
    - Works on grade (>2%)– Surface bladed by grader to smooth out ground surface to allow vehicle movements. No tree removal. Topsoil will be bladed off by grader and windrowed for later respreading at completion of data recording, to preserve the soil structure. Whoa boys or roll over banks to be provided as per details in Appendix P.
    - At the conclusion of activities, or as part of progressive rehabilitation, or the anticipated onset of a significant rainfall event which will require the site to be abandoned, topsoil would be respread and ripped into the soil surface.
- Wooded communities e.g. Lancewood/Bullwaddy – For the majority of the program wherever practical, activities should be planned to avoid impacts to Lancewood and Bullwaddy vegetation communities. Where this is not possible, the vegetation community would require measures as follows:
    - A survey line of 5 m maximum should be cleared by the dozer removing the trees. Felled trees should be pushed to the side to enable vehicle access through the site.
    - Following clearing the topsoil bladed off by grader and windrowed for later respreading with the vegetated material at completion of data recording.
    - The line preparation will require blading to a sufficient depth, no greater than 150 mm, to enable the safe access of the vehicles. The purpose of the blading is to reduce the risk of tyre puncture from the Lancewood which is known to snap off at ground level leaving a spike protruding.
    - Whoa boys or roll over banks to be provided as per detail in Appendix P.
    - At the conclusion of activities, or as part of progressive rehabilitation, or the anticipated onset of a significant rainfall event which will require the site to be abandoned, topsoil would be respread at a thickness of 150 mm and ripped into the soil surface.
    - Felled vegetation will be evenly spread over the top soiled area to provide additional protection against erosion.
- Seasonally inundated areas - Similar to the wooded communities described above, high clay content soils (vertosols) are also found in seasonally inundated areas and in the southern survey area. Unlike the wooded areas these clays continue at depth, making the scraping back of topsoil less effective in keeping bulldust down and preserving soil structure. The recommendation in these locations is that line preparation would consist primarily of the vehicles traversing directly of the annual grasses, flattening or slashing for data acquisition i.e. blade up.

7. Monitoring

7.1 Construction

Monitoring for soil erosion and related issues is best undertaken at critical stages, such as:

- At the time of the baseline land condition assessment.
- During siting of access tracks and exploration areas, this is when there is the greatest opportunity to avoid erosion problems.
- After completion of a specific phase of activity, all disturbed areas will be monitored before and after the wet season.
- When accessing the site after the wet season, all disturbed areas should be inspected for signs of erosion. If significant impacts are identified remediation works may need to be conducted prior to continued vehicular access.
- In the unlikely event that water is required to be released from the sediment pond, the stored water will be visually assessed (no sheen, or turbidity) and physical parameters (pH, EC) taken to ensure release water will not impact on any downgradient sensitive receiving environments (refer Section 7.3). It is noted that lease areas do not have any sensitive receiving water bodies located in close proximity to the sites.

When accessing the site after the wet season, all disturbed areas should be inspected for signs of erosion. If significant impacts are identified remediation works may need to be conducted.

7.2 Operations

Visual inspections will be undertaken throughout the 2D seismic survey activities to assess the impact risk level of the regulated activities being undertaken and the likelihood of erosion occurring. A review of mitigation measures that are implemented throughout the project phase will be conducted regularly to assess the efficacy and that the standard is maintained.

All other areas to be inspected before and after the wet season to identify the occurrence of erosion and sedimentation. Where erosion is observed, maintenance activities shall be undertaken. Ongoing Monitoring and maintenance shall occur throughout the life of the infrastructure until the land is handed back.

7.3 ESC Trigger Action Response Plan

The following Trigger Action Response Plan (TARP) is to be implemented during construction:

- **Monitoring Requirements:**
  - Daily visual inspection of access track, lease pads and campsite conditions for duration of civil construction activities.
  - Routine visual inspections of the creek and drainage line access track crossings and the wastewater containment system at the camp weekly or following a rainfall event (i.e. greater than 20 mm in 24 hours).
- **Action:**
  - On establishment of each exploration lease pad, undertake jar testing work to determine anticipated settling rate of sediments on site. This will inform flocculent dosing requirements as required.
  - Repair of ESC devices immediately when found not to comply.
  - Where monitoring has indicated weather condition have impacted the integrity of the erosion and sediment controls, operators must adopt one of the treatment plans from Section 6.0 to mitigate the impacts of rainfall and ensure that the ESC devices are reinstated as soon as physically practicable after the event.

- Inspection of all ESC devices across the worksite and physical water quality testing (physical parameters only) at the lease pad sediment basin should be conducted prior to discharge of water offsite. Water quality discharge indicators include:
  - No visible oil, grease or other hydrocarbons
  - pH: Between 6.0-8.0
  - EC: 250 uS/cm.

  The adopted discharge criteria are based on ANZECC 2000 Table 3.3.4 and Table 3.3.5 default trigger values for pH and conductivity (EC, salinity) indicative of slightly disturbed ecosystems in tropical Australia, as well as consideration of the distance and type of nearby sensitive surface water receptors as ephemeral drainage lines and creeks.

- **Response:**
  - If water quality conditions meet discharge indicators, beneficial reuse of water may be considered for construction activities.
  - External NATA accredited laboratory testing of soil/sediment or surface water would only be required for the following triggers:
    - Work area has a known existing contaminating event in the preceding 3 months that could influence stormwater discharge quality (refer to Origin's Spill Management Plan appended to the EMP).
    - The visual inspection and physical water quality testing indicated potential contamination.
    - Where there is a sensitive receiving water body within 200 m of the discharge point.

7.4 Rehabilitation

Well lease pad, access tracks, gravel pits and camp

Where rehabilitation of a site is required, rehabilitation monitoring will be undertaken annually to assess the rehabilitation success and determine whether additional remedial works are required. Success criteria are defined in the relevant EMP and include:

- Safe for humans and wildlife
- Non-polluting
- Stable, with appropriate vegetation cover
- Land condition suitable for existing pastoral land use.

Seismic Line Acquisition

Rehabilitation will be undertaken along all newly cleared survey lines concurrently with the completion of the survey process. Reference should be made to Origin's Rehabilitation Plan prepared in support of the Amungee delineation area EMP. Rehabilitation of all areas must be undertaken in accordance with the methodologies described in the Rehabilitation Plan and treatments in Appendix P of this document.

Rehabilitation monitoring will be undertaken before and after the initial wet season and then annually for 5 years to assess the rehabilitation success and determine whether additional remedial works are required. Success criteria are defined in the relevant EMP and include:

- safe for humans and wildlife
- non-polluting
- stable, with appropriate vegetation cover
- waterways are not materially changed.

- land condition suitable for existing pastoral land use.

7.5 Incident Reporting

The Constructor must follow incident reporting requirements covered in the Origin Incident Management Directive.

Sediment release and turbidity increase incidents can require some assessment to determine if they are reportable, as controls are only designed to cope with certain rain events (refer to IECA, 2008).

The Constructor must:

- Report sediment release and turbidity increase incidents.
- Include justification in each case of why the incident is, or is not, reportable to the regulator based on:
  - The state of the controls prior to the rainfall
  - The design standard applied (IECA, 2008)
  - The actual rainfall received, based on the nearest data source available
  - Whether the design storm event was exceeded or not; and
  - Whether environmental harm was caused or not.

7.6 Records

Records shall be retained demonstrating areas have been inspected. Photographic records will be maintained over the duration of the activities for documenting soil disturbance.

All environmentally relevant incidents are to be recorded in a field log that must remain accessible to all relevant regulatory authorities.

Minimum records to be retained for each site include:

Location of disturbance	Area of disturbance	Date	Close out

7.7 ESCP Revisions

Where major changes are required to the proposed controls in the ESCP through Origin's change management processes, DEPWS would be advised and a revised ESCP provided. Should any civils be required during the wet season, the wet weather contingency plan outlined in Table 10 will be implement.

7.8 Maintenance

All temporary erosion and sediment control measures, including drainage control measures, must be fully operational and maintained in proper working order at all times during the project.

When undertaking construction work, erosion and sediment control measures must be inspected:

- at least daily (when work is occurring on-site during the wet season)
- within 24 hours of expected rainfall
- within 18 hours of a rainfall event of sufficient intensity and duration to cause runoff on-site or greater than 20mm in 24 hours.

Once operational, inspections of the site will continue daily while onsite, and before and after the wet season. Where erosion is observed, maintenance activities shall be undertaken.

Sediment removed from sediment traps and places of sediment deposition must be disposed of in a lawful manner that does not cause ongoing soil erosion or environmental harm.

Prior to the completion of activities on the ground, the construction areas will be stabilised to the satisfaction of the Construction Supervisor.

References

Catchment and Creeks Pty Ltd. 2012. *Erosion & Sediment Control – A Field Guide for Construction Site Managers V5.* Catchment and Creeks. Brisbane. QLD.

Department of Environment and Natural Resource, 2019, *Land Clearing Guidelines*, Northern Territory Government (dated February 2019).

Department of Natural Resources, Environment, The Arts and Sport (NRETAS) 2010. *Land Clearing Guidelines*. Northern Territory Government.

Department of Agriculture, Fisheries and Forestry. 2013. *Code for Self-Assessable Development Minor Waterway Barrier Works Part 4: Bed Level Crossings Code Number WWBW01 April 2013.* State of Queensland, Qld.

IECA. 2008. *Best Practice Erosion and Sediment Control – for building and construction sites*. Picton, NSW: International Erosion Control Association (Australasia).

Origin Energy Resources Limited. 2018. *Draft Beetaloo Basin Groundwater Monitoring Bore Installation Program Environmental Management Plan*.

Scientific Inquiry into Hydraulic Fracturing in the Northern Territory. 2018. *Scientific Inquiry into Hydraulic Fracturing in the Northern Territory – Final Report.*

Appendix A Erosion Hazard Assessment Explanatory Notes

reference: IECA, 2008, *Best Practice Erosion and Sediment Control Hazard Assessment Form)*

Requirements: Specific issues or actions required by the proponent.

Warnings: Issues that should be considered by the proponent.

Comments: General information relating to the topic.

[1] **REQUIREMENTS:**

For sites with an average slope of proposed land disturbance greater than 10%, a preliminary ESCP must be submitted to the regulatory authority for approval during planning negotiations.

Proponents must demonstrate that adequate erosion and sediment control measures can be implemented on-site to effectively protect downstream environmental values.

If site or financial constraints suggest that it is not reasonable or practicable for the prescribed water quality objectives to be achieved for the proposal, then the proponent must demonstrate that alternative designs or construction techniques (e.g. pole homes, suspended slab) cannot reasonably be implemented on the site.

WARNINGS:

Steep sites usually require more stringent drainage and erosion controls than flatter grade sites.

COMMENTS:

The steeper the land, the greater the need for adequate drainage controls to prevent soil and mulch from being washed from the site.

[2] **REQUIREMENTS:**

If the actual soil K-factor is known from soil testing, then the Score shall be determined from Table 1.

If a preliminary ESCP is required during planning negotiations, then it must be demonstrated that adequate space is available for the construction and operation of any major sediment traps, including the provision for any sediment basins and their associated embankments and spillways. It must also be demonstrated that all reasonable and practicable measures can be taken to divert the maximum quantity of sediment-laden runoff (up to the specified design storm) to these sediment traps throughout the construction phase and until the contributing catchment is adequately stabilised against erosion.

WARNINGS:-

The higher the point score, the greater the need to protect the soil from raindrop impact and thus the greater the need for effective erosion control measures. A point score of 2 or greater will require a greater emphasis to be placed on revegetation techniques that do not expose the soil to direct rainfall contact during vegetation establishment, e.g. turfing and *Hydromulching*.

COMMENTS:

Table 2 provides an *indication* of soil conditions likely to be associated with a particular Soil group based on a statistical analysis of soil testing across NSW. This table provides only an initial estimate of the likely soil conditions.

The left-hand-side of the table provides an indication of the type of sediment basin that will be required (Type C, F or D). The right-hand-side of the table provides an indication of the likely erodibility of the soil based on the Revised Universal Soil Loss Equation (RUSLE) K-factor.

Table 3 provides some general comments on the erosion potential of the various soil groups.

Table 1 – Score if soil K-factor is known

	RUSLE soil erodibility K-factor			
	K < 0.02	0.02<K<0.04	0.04<K<0.06	K > 0.06
Score	0	1	2	3

Table 2 – Statistical analysis of NSW soil data [1]

Unified Soil Class System	Likely sediment basin classification (%)			Probable soil erodibility K-factor (%) [2]			
	Dry	Wet		Low	Moderate	High	Very High
	Type C	Type F	Type D	K < 0.02	0.02<K<0.04	0.04<K<0.06	K > 0.06
GM	30	**58**	12	12	**51**	26	12
GC	**42**	33	25	13	**71**	17	0
SW	40	**48**	12	**49**	39	12	0
SP	**53**	32	15	**76**	18	5	1
SM	21	**67**	12	26	**48**	25	1
SC	26	**50**	24	16	**64**	18	2
ML	5	**63**	32	4	35	**45**	16
CL	9	**51**	39	12	**56**	19	13
OL	2	**80**	18	34	**61**	5	1
MH	12	41	**48**	15	19	**41**	25
CH	5	44	**51**	39	**43**	11	7

Notes: [1] Analysis of soil data presented in Landcom (2004).

[2] Soil erodibility based on Revised Universal Soil Loss Equation (RUSLE) K-factor.

Unified Soil Classification System (USCS)

GW Well graded gravels, gravel-sand mixtures, little or no fines

GP Poorly graded gravels, gravel-sand mixture, little or no fines

GM Silty gravels, poorly graded gravel-sand-silt mixtures

GC Clayey gravels, poorly graded gravel-sand-clay mixtures

SW Well graded sands, gravelly sands, little or no fines

SP Poorly graded sands, gravelly sands, little or no fines

SM Silty sands, poorly graded sand-silt mixtures

SC Clayey sands, poorly graded sand-clay mixtures

ML Inorganic silts & very fine sands, rock flour, silty or clayey fine sands with slight plasticity

CL Inorganic clays, low–medium plasticity, gravelly clays, sandy clays, silty clays, lean clays

OL Organic silts and organic silt-clays of low plasticity

MH Inorganic silts, micaceous or diatomaceous fine sandy or silty soils, elastic silts

CH Inorganic clays of high plasticity, fat clays

OH Organic clays of medium to high plasticity

Table 3 – Typical properties of various soil groups [1]

Soil Groups	Typical properties [2]
GW, GP	• Low erodibility potential.
GM, GC	• Low to medium erodibility potential. • May create turbid runoff if disturbed as a result of the release of silt and clay particles.
SW, SP	• Low to medium erodibility potential.
SM, SC	• Medium erodibility potential. • May create turbid runoff if disturbed as a result of the release of silt and clay particles.
MH, CH	• Highly variable (low to high) erodibility potential. • Will generally create turbid runoff if disturbed.
ML, CL	• High erodibility potential. • Tendency to be dispersive. • May create some turbidity in runoff if disturbed.

Note: [1] After Soil Services & NSW DLWC (1998).

[2] Any soil can represent a high erosion risk if the binding clays or silts are unstable.

Table 4 provides **general** guidelines on the suitability of various soil groups to various engineering applications.

Table 4 – Engineering suitability based on Unified Soil Classification [1]

Unified Soil Class	USC Group	Embankments		Fill	Slope stability	Untreated roads
		Water retaining	Non-water retaining			
Well graded gravels	GW	Unsuitable	Excellent	Excellent	Excellent	Average
Poorly graded gravel	GP	Unsuitable	Average	Excellent	Average	Unsuitable
Silty gravels	GM	Unsuitable	Average	Good	Average	Average
Clayey gravels	GC	Suitable	Average	Good	Average	Excellent
Well graded sands	SW	Unsuitable	Excellent	Excellent	Excellent	Average
Poorly graded sands	SP	Unsuitable	Average	Good	Average	Unsuitable
Silty sands	SM	Suitable [2]	Average	Average	Average	Poor
Clayey sands	SC	Suitable	Average	Average	Average	Good
Inorganic silts	ML	Unsuitable	Poor	Average	Poor	Unsuitable
Inorganic clays	CL	Suitable [2]	Good	Average	Good	Poor

Organic silts	OL	Unsuitable	Unsuitable	Poor	Unsuitable	Unsuitable
Inorganic silts	MH	Unsuitable	Poor	Poor	Poor	Unsuitable
Inorganic clays	CH	Suitable [2]	Average	Unsuitable	Average	Unsuitable
Organic clays	OH	Unsuitable	Unsuitable	Unsuitable	Unsuitable	Unsuitable
Highly organic soils	Pt	Unsuitable	Unsuitable	Unsuitable	Unsuitable	Unsuitable

Notes: [1] Modified from Hazelton & Murphy (1992)

[2] Suitable only after modifications to soil such as compaction and/or erosion protection

[3] If the soils have not been tested for Emerson Class, then adopt a score of 4.

REQUIREMENTS:

Works proposed on sites containing Emerson Class 1 or 2 soils have a very high pollution potential and must submit a conceptual ESCP to the regulatory authority for review and/or approval (as required by the authority) during planning negotiations.

WARNINGS:

Class 3 and 5 soils disturbed by cut and fill operations or construction traffic are highly likely to discolour stormwater (i.e. cause turbid runoff). Chemical stabilisation will likely be required if these soils are placed immediately adjacent to a retaining wall. Any disturbed Class 1, 2, 3 and 5 soils that are to be revegetated must be covered with a non-dispersive topsoil as soon as possible (unless otherwise agreed by the regulatory authority).

Class 1 and 2 soils are highly likely to discolour (pollute) stormwater if exposed to rainfall or flowing water. Treatment of these soils with gypsum (or other suitable substance) will most likely be required. These soils should not be placed directly behind a retaining wall unless it has been adequately treated (stabilised) or covered with a non-dispersible soil.

[4] The duration of disturbance refers to the total duration of soil exposure to rainfall up until a time when there is at least 70% coverage of all areas of soil.

REQUIREMENTS:

All land developments with an expected soil disturbance period greater than 6 months must submit a conceptual ESCP to the regulatory authority for review and/or approval (as required by the authority) during planning negotiations.

COMMENTS:

Construction periods greater than 3 months will generally experience at least some significant storm events, independent of the time of year that the construction (soil disturbance) occurs.

[5] **REQUIREMENTS:**

Development proposals with an expected soil disturbance in excess of 1ha must submit a conceptual ESCP to the regulatory authority for review and/or approval (as required by the regulatory authority) during planning negotiations.

The area of disturbance refers to the total area of soil exposed to rainfall or dust-producing winds either as a result of:

(a) the removal of ground cover vegetation, mulch or sealed surfaces;
(b) past land management practices;
(c) natural conditions.

WARNINGS:

A *Sediment Basin* will usually be required if the disturbed area exceeds 0.25ha (2500m^2) within any sub-catchment (i.e. land flowing to one outlet point).

COMMENTS:

For soil disturbances greater than 0.25ha, the revegetation phase should be staged to minimise the duration for which soils are exposed to wind, rain and concentrated runoff.

[6] **REQUIREMENTS:**

All developments that involve earthworks or construction within a natural watercourse (whether that watercourse is in a natural or modified condition) must submit a conceptual ESCP to the regulatory authority for review and/or approval (as required by the regulatory authority) during planning negotiations.

Permits and/or licences may be required from the State Government, including possible submission of the ESCP to the relevant Government department.

[7] **REQUIREMENTS:**

No areas of soil disturbance shall be left exposed to rainfall or dust-producing winds at the end of a development without an adequate degree of protection and/or an appropriate action plan for the establishment of at least 70% cover.

COMMENTS:

Grass seeding without the application of a light mulch cover is considered the least favourable revegetation technique. A light mulch cover is required to protect the soil from raindrop impact, excessive temperature fluctuations, and the loss of essential soil moisture.

[8] **COMMENTS:**

All receiving waters can be adversely affected by unnatural quantities of sediment-laden runoff. Freshwater ecosystems are generally more susceptible to ecological harm resulting from the inflow of fine or dispersible clays than saline water bodies. The further inland a land disturbance is, the greater the potential for the released sediment to cause environmental harm as this sediment travels towards the coast.

For the purpose of this clause it is assumed that all sediment-laden runoff will eventually flow into saline waters. Thus, sediment-laden discharges that flow first into freshwater are likely to adversely affect both fresh and saline water bodies and are therefore considered potentially more damaging to the environment.

This clause does **not** imply that sediment-laden runoff will not cause harm to saline waters.

[9] **COMMENTS:**

This clause refers to subsoils exposed during the construction phase either as a result of past land practices or proposed construction activities. The exposure of subsoils resulting from the excavation of minor service trenches should not be considered.

[10] **WARNINGS:**

The greater the extent of external catchment, the greater the need to divert up-slope stormwater runoff around any soil disturbance.

COMMENTS:

The ability to separate “clean” (i.e. external catchment) stormwater runoff from “dirty” site runoff can have a significant effect on the size, efficiency and cost of the temporary drainage, erosion, and sediment control measures.

[11] **REQUIREMENTS:**

Permission must be obtained from the owner of a road reserve before placing any erosion and sediment control measures within the road reserve.

WARNINGS:

Few sediment control techniques work efficiently when placed on a road and/or around roadside stormwater inlets. Great care must be taken if sediment control measures are located on a public roadway, specifically:

- safety issues relating to road users;
- the risk of causing flooding on the road or within private property.

The construction of roads (whether temporary or permanent) will usually modify the flow path of stormwater runoff. This can affect how "dirty" site runoff is directed to the sediment control measures.

COMMENTS:

"On-road" sediment control devices are at best viewed as secondary or supplementary sediment control measures. Only in special cases and/or on very small projects (e.g. kerb and channel replacement) might these controls be considered as the "primary" sediment control measure.

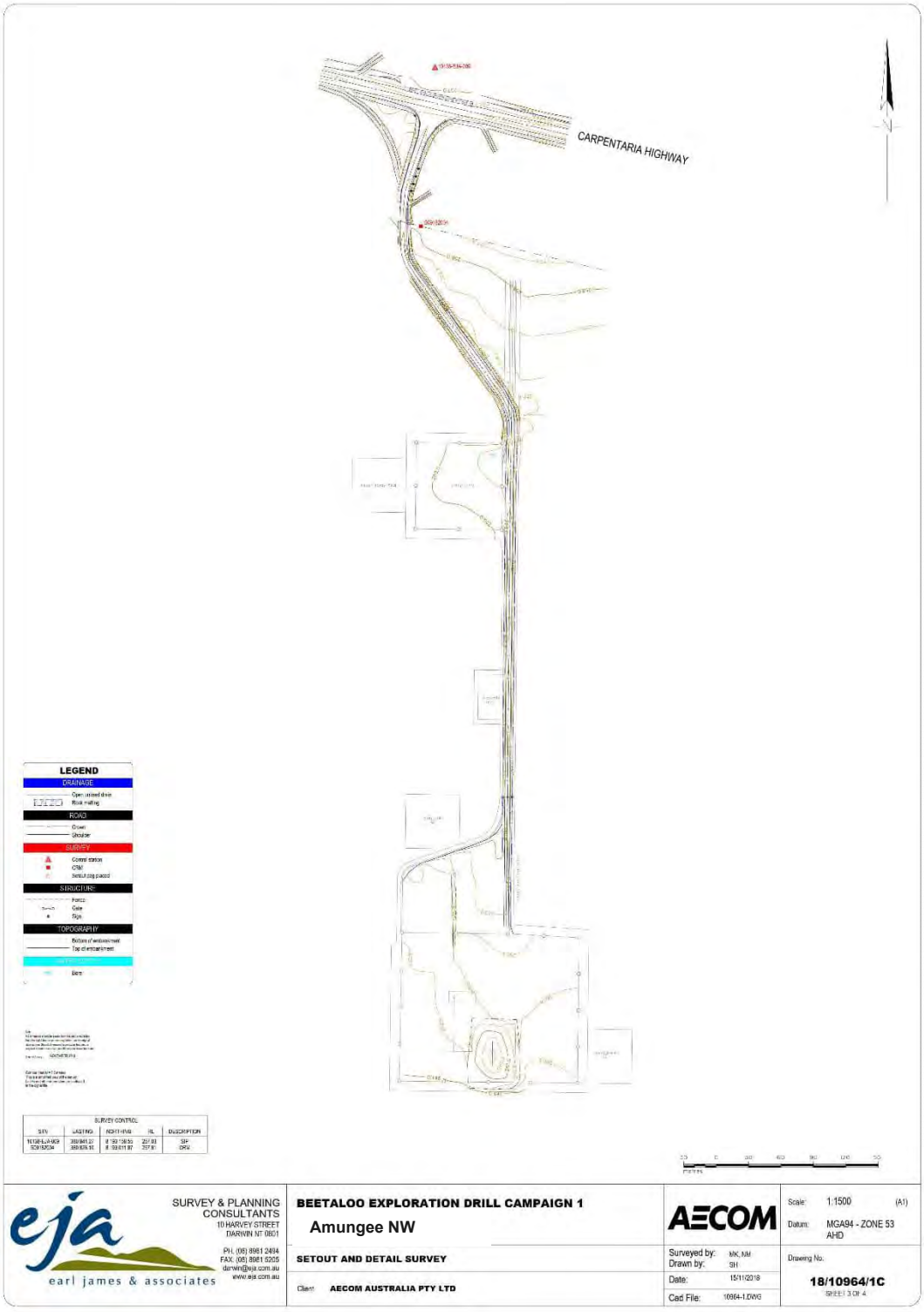
[12] **WARNINGS:**

Soils with a pH less than 5.5 or greater than 8 will usually require treatment in order to achieve satisfactory revegetation. Soils with a pH of less than 5 (whether naturally acidic or in acid sulfate soil areas) may also limit the choice of chemical flocculants (e.g. Alum) for use in the flocculation of *Sediment Basins*.

[13] **REQUIREMENTS:**

A preliminary ESCP must be submitted to the local government for approval during the planning phase for any development that obtains a total point score of 17 or greater or when any trigger value is scored or exceeded.

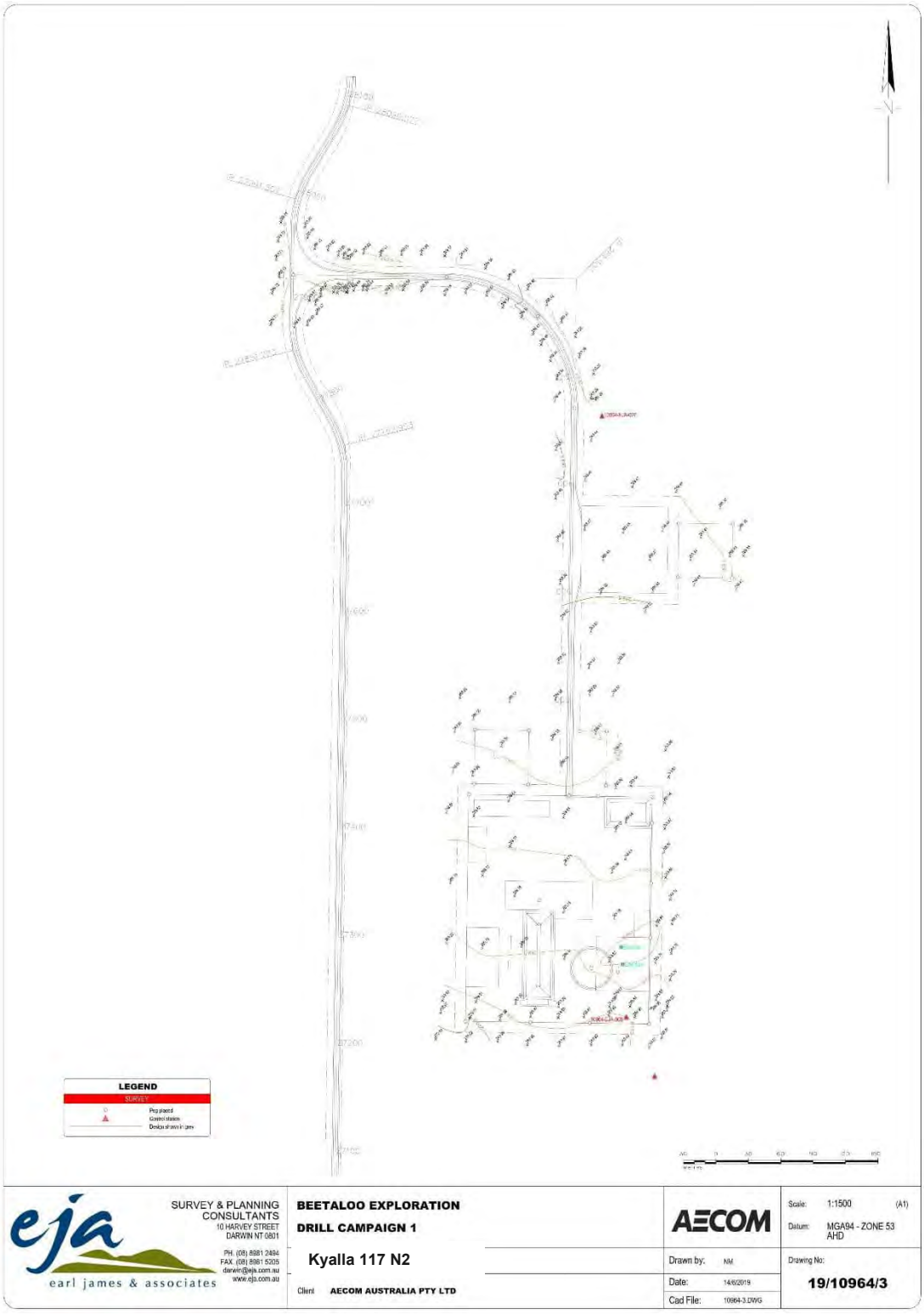
Appendix B Lease Pad and Stuart Highway Topographical Survey



Review due: 13/09/2025

Erosion and Sediment Control Plan

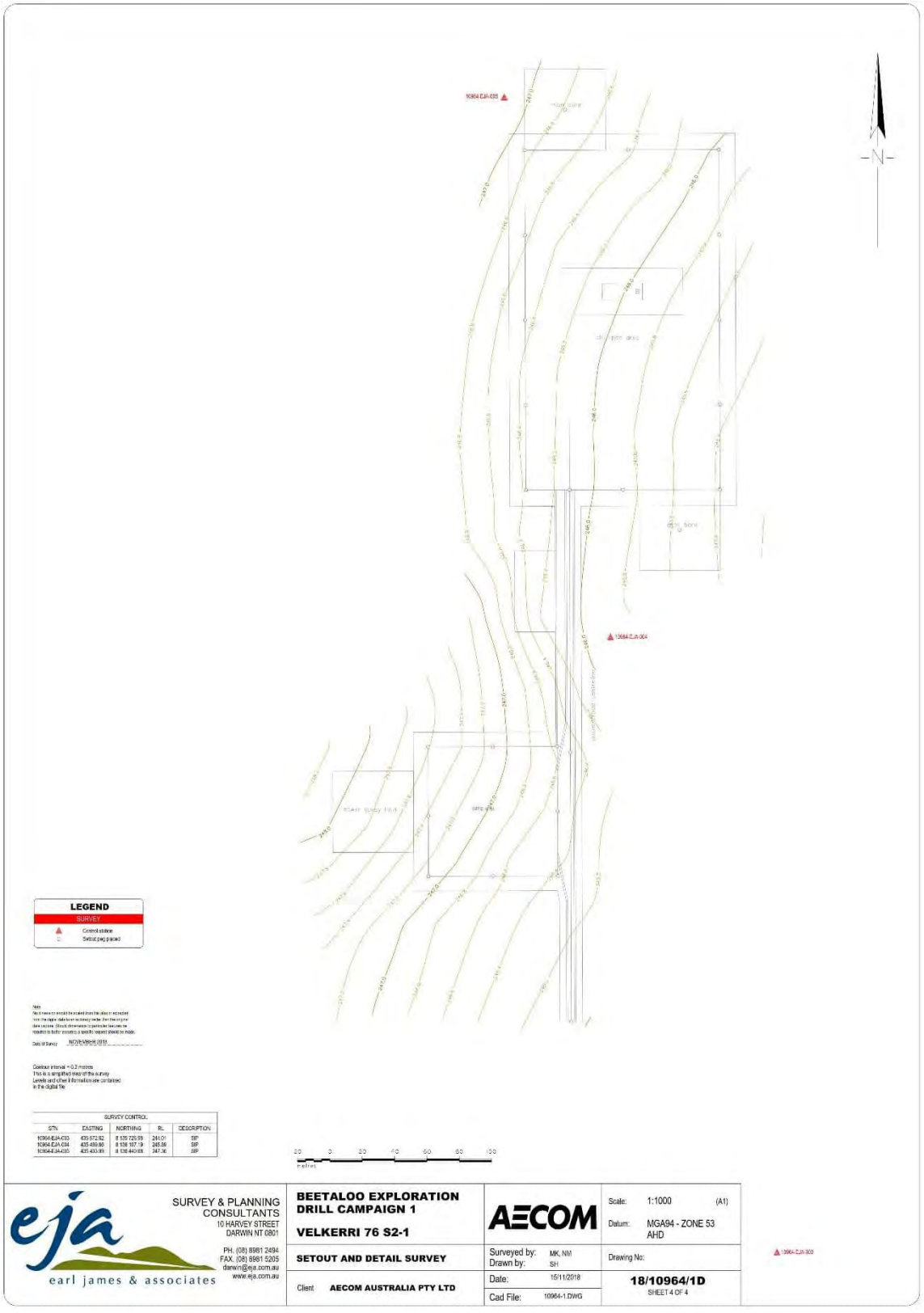
NT-2050-15-MP-019.



Review due: 13/09/2025

Erosion and Sediment Control Plan

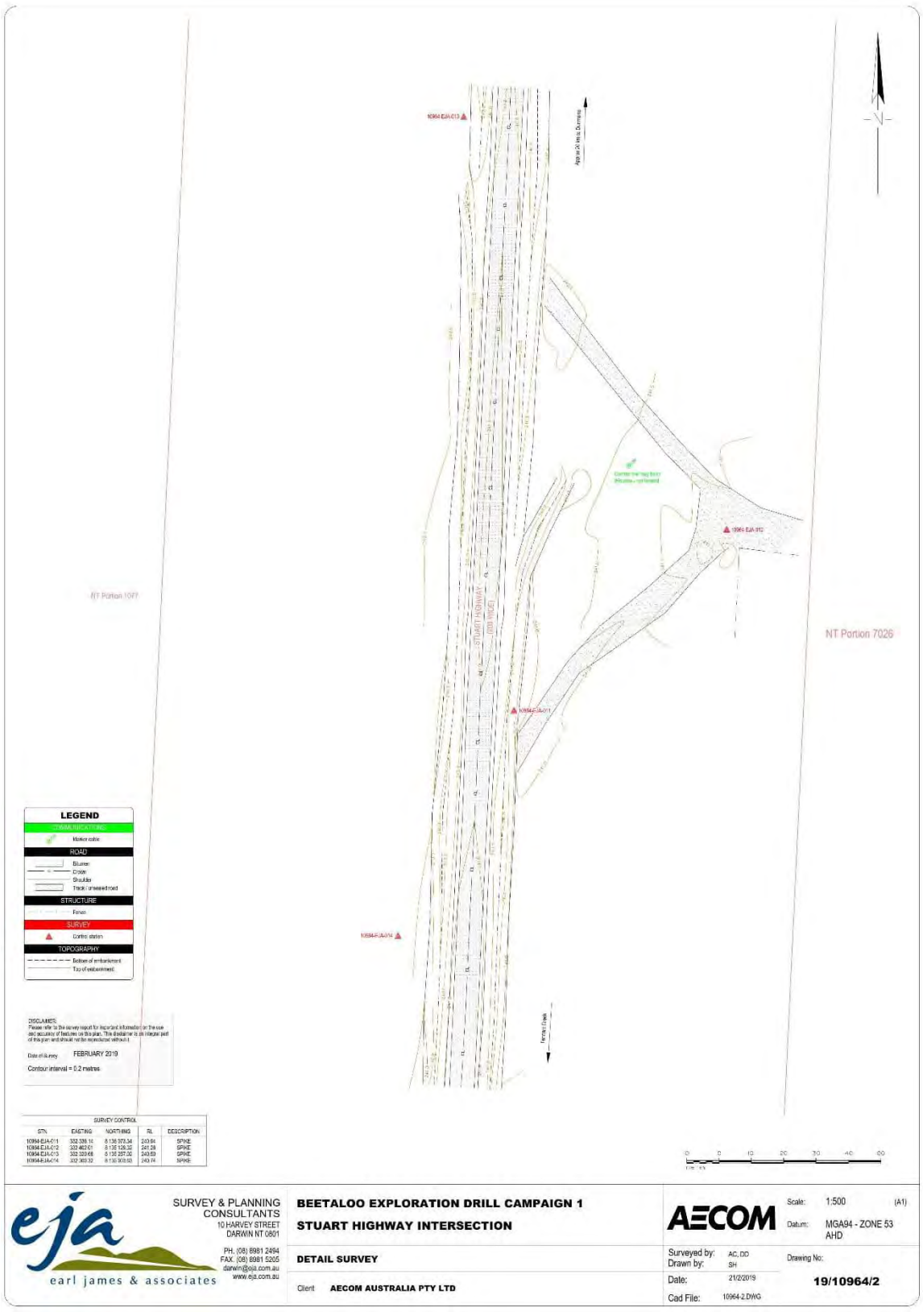
NT-2050-15-MP-019.



Review due: 13/09/2025

Erosion and Sediment Control Plan

NT-2050-15-MP-019.



Review due: 13/09/2025

Appendix C Geotechnical Laboratory Results

Material Test Report

Douglas Partners
Douglas Partners Pty Ltd
Darwin Laboratory
Unit 2/14 Caryota Crt COCONUT GROVE NT 0810
Phone: (08) 8948 6800
Fax: (08) 8948 6899
Email: dave.millard@douglaspartners.com.au
Accredited for compliance with ISO/IEC 17025 - Testing

NATA

Approved Signatory: Dave Millard
Engineering Geologist
NATA Accredited Laboratory Number: 828

Report Number: 677612.00-1
Issue Number: 2 - *This version supersedes all previous issues*
Date Issued: 29/01/2019
Client: AECOM Australia Pty Ltd
PO Box 73, Hunter Region NSW 2310
Contact: Jace Emberg
Project Number: 677612.00
Project Name: 60480548 - Beetaloo soil samples
Project Location: Beetaloo, NT
Work Request: 1466
Sample Number: 19-1466F
Date Sampled: 13/12/2018
Sampling Method: AS1289 1.2.1 6.4 - Sampling from layers in earthworks or pavement - uncompacted/compacted
Sample Location: **KYALLA01 (0.0 - 0.4m)**
Material: SAND, with silt

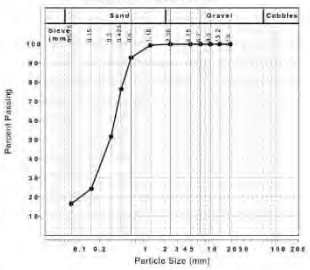
Particle Distribution (AS1289 3.6.1)			
Sieve	Passed %	Passing Limits	
19 mm	100		
13.2 mm	100		
9.5 mm	100		
6.7 mm	100		
4.75 mm	100		
2.36 mm	100		
1.18 mm	99		
0.6 mm	93		
0.425 mm	76		
0.3 mm	52		
0.15 mm	24		
0.075 mm	16		

Atterberg Limit (AS1289 3.1.2 & 3.2.1 & 3.3.1)		Min	Max
Sample History	Air Dried		
Preparation Method	Dry Sieve		
Liquid Limit (%)	Not Obtainable		
Plastic Limit (%)	Not Obtainable		
Plasticity Index (%)	**Non Plastic**		

Linear Shrinkage (AS1289 3.4.1)		Min	Max
Linear Shrinkage (%)	0.0		
Cracking Crumbling Curling	None		

Moisture Content (AS 1289 2.1.1)	
Moisture Content (%)	1.9

Particle Size Distribution



Material Test Report

Douglas Partners
Douglas Partners Pty Ltd
Darwin Laboratory
Unit 2/14 Caryota Crt COCONUT GROVE NT 0810
Phone: (08) 8948 6800
Fax: (08) 8948 6899
Email: dave.millard@douglaspartners.com.au
Accredited for compliance with ISO/IEC 17025 - Testing

NATA

Approved Signatory: Dave Millard
Engineering Geologist
NATA Accredited Laboratory Number: 828

Report Number: 677612.00-1
Issue Number: 2 - *This version supersedes all previous issues*
Date Issued: 29/01/2019
Client: AECOM Australia Pty Ltd
PO Box 73, Hunter Region NSW 2310
Contact: Jace Emberg
Project Number: 677612.00
Project Name: 60480548 - Beetaloo soil samples
Project Location: Beetaloo, NT
Work Request: 1466
Sample Number: 19-1466G
Date Sampled: 13/12/2018
Sampling Method: AS1289 1.2.1 6.4 - Sampling from layers in earthworks or pavement - uncompacted/compacted
Sample Location: **KYALLA02 (0.0 - 0.4m)**
Material: Silty SAND

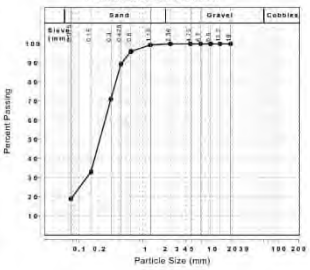
Particle Distribution (AS1289 3.6.1)			
Sieve	Passed %	Passing Limits	
19 mm	100		
13.2 mm	100		
9.5 mm	100		
6.7 mm	100		
4.75 mm	100		
2.36 mm	100		
1.18 mm	99		
0.6 mm	96		
0.425 mm	89		
0.3 mm	71		
0.15 mm	33		
0.075 mm	19		

Atterberg Limit (AS1289 3.1.2 & 3.2.1 & 3.3.1)		Min	Max
Sample History	Air Dried		
Preparation Method	Dry Sieve		
Liquid Limit (%)	14		
Plastic Limit (%)	13		
Plasticity Index (%)	**1**		

Linear Shrinkage (AS1289 3.4.1)		Min	Max
Linear Shrinkage (%)	0.0		
Cracking Crumbling Curling	Cracking		

Moisture Content (AS 1289 2.1.1)	
Moisture Content (%)	2.3

Particle Size Distribution



Material Test Report

Douglas Partners
Douglas Partners Pty Ltd
Darwin Laboratory
Unit 2/14 Caryota Crt COCONUT GROVE NT 0810
Phone: (08) 8948 6800
Fax: (08) 8948 6899
Email: dave.millard@douglaspartners.com.au
Accredited for compliance with ISO/IEC 17025 - Testing

NATA

Approved Signatory: Dave Millard
Engineering Geologist
NATA Accredited Laboratory Number: 828

Report Number: 677612.00-1
Issue Number: 2 - *This version supersedes all previous issues*
Date Issued: 29/01/2019
Client: AECOM Australia Pty Ltd
PO Box 73, Hunter Region NSW 2310
Contact: Jace Emberg
Project Number: 677612.00
Project Name: 60480548 - Beetaloo soil samples
Project Location: Beetaloo, NT
Work Request: 1466
Sample Number: 19-1466H
Date Sampled: 13/12/2018
Sampling Method: AS1289 1.2.1 6.4 - Sampling from layers in earthworks or pavement - uncompacted/compacted
Sample Location: **KYALLA03 (0.0 - 0.4m)**
Material: Silty SAND

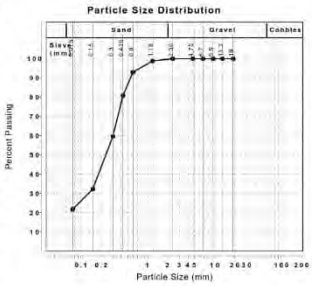
Particle Distribution (AS1289 3.6.1)			
Sieve	Passed %	Passing Limits	
19 mm	100		
13.2 mm	100		
9.5 mm	100		
6.7 mm	100		
4.75 mm	100		
2.36 mm	100		
1.18 mm	99		
0.6 mm	93		
0.425 mm	81		
0.3 mm	60		
0.15 mm	32		
0.075 mm	22		

Atterberg Limit (AS1289 3.1.2 & 3.2.1 & 3.3.1)		Min	Max
Sample History	Air Dried		
Preparation Method	Dry Sieve		
Liquid Limit (%)	15		
Plastic Limit (%)	13		
Plasticity Index (%)	**2**		

Linear Shrinkage (AS1289 3.4.1)		Min	Max
Linear Shrinkage (%)	1.0		
Cracking Crumbling Curling	Cracking		

Moisture Content (AS 1289 2.1.1)	
Moisture Content (%)	3.1

Particle Size Distribution



Material Test Report

Douglas Partners
Douglas Partners Pty Ltd
Darwin Laboratory
Unit 2/14 Caryota Crt COCONUT GROVE NT 0810
Phone: (08) 8948 6800
Fax: (08) 8948 6899
Email: dave.millard@douglaspartners.com.au
Accredited for compliance with ISO/IEC 17025 - Testing

NATA

Approved Signatory: Dave Millard
Engineering Geologist
NATA Accredited Laboratory Number: 828

Report Number: 677612.00-1
Issue Number: 2 - *This version supersedes all previous issues*
Date Issued: 29/01/2019
Client: AECOM Australia Pty Ltd
PO Box 73, Hunter Region NSW 2310
Contact: Jace Emberg
Project Number: 677612.00
Project Name: 60480548 - Beetaloo soil samples
Project Location: Beetaloo, NT
Work Request: 1466
Sample Number: 19-1466I
Date Sampled: 13/12/2018
Sampling Method: AS1289 1.2.1 6.4 - Sampling from layers in earthworks or pavement - uncompacted/compacted
Sample Location: **KYALLA04 (0.0 - 0.4m)**
Material: Silty SAND

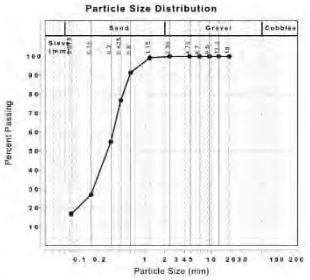
Particle Distribution (AS1289 3.6.1)			
Sieve	Passed %	Passing Limits	
19 mm	100		
13.2 mm	100		
9.5 mm	100		
6.7 mm	100		
4.75 mm	100		
2.36 mm	100		
1.18 mm	99		
0.6 mm	91		
0.425 mm	77		
0.3 mm	55		
0.15 mm	27		
0.075 mm	17		

Atterberg Limit (AS1289 3.1.2 & 3.2.1 & 3.3.1)		Min	Max
Sample History	Oven Dried / Air Dried / Natural		
Preparation Method	Wet Sieve / Dry Sieve / Both Sieves		
Liquid Limit (%)	15		
Plastic Limit (%)	13		
Plasticity Index (%)	**2**		

Linear Shrinkage (AS1289 3.4.1)		Min	Max
Linear Shrinkage (%)	0.5		
Cracking Crumbling Curling	Cracking		

Moisture Content (AS 1289 2.1.1)	
Moisture Content (%)	2.0

Particle Size Distribution



Erosion and Sediment Control Plan

NT-2050-15-MP-019.

Material Test Report

Douglas Partners
Douglas Partners Pty Ltd
Darwin Laboratory
Unit 2/14 Caryota Crt COCONUT GROVE NT 0810
Phone: (08) 8948 6800
Fax: (08) 8948 6899
Email: dave.millard@douglaspartners.com.au
Accredited for compliance with ISO/IEC 17025 - Testing

Approved Signatory: Dave Millard
Engineering Geologist
NATA Accredited Laboratory Number: 828

Report Number:	**677612.00-1**
Issue Number:	2 - *This version supersedes all previous issues*
Date Issued:	29/01/2019
Client:	AECOM Australia Pty Ltd PO Box 73, Hunter Region NSW 2310
Contact:	Jace Emberg
Project Number:	677612.00
Project Name:	60480548 - Beetaloo soil samples
Project Location:	Beetaloo, NT
Work Request:	1466
Sample Number:	19-1466A
Date Sampled:	13/12/2018
Sampling Method:	AS1289 1.2.1 6.4 - Sampling from layers in earthworks or pavement - uncompacted/compacted
Sample Location:	**VELKERRI01 (0.0 - 0.4m)**
Material:	Silty SAND, trace gravel

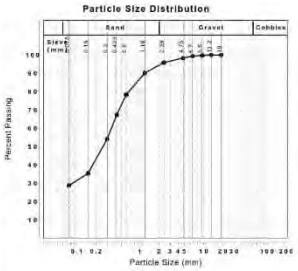
Particle Distribution (AS1289 3.6.1)			
Sieve	Passed %	Passing Limits	
19 mm	**100**		
13.2 mm	**100**		
9.5 mm	**100**		
6.7 mm	**99**		
4.75 mm	**98**		
2.36 mm	**96**		
1.18 mm	**90**		
0.6 mm	**78**		
0.425 mm	**67**		
0.3 mm	**54**		
0.15 mm	**35**		
0.075 mm	**29**		

Atterberg Limit (AS1289 3.1.2 & 3.2.1 & 3.3.1)		Min	Max
Sample History	Air Dried		
Preparation Method	Dry Sieve		
Liquid Limit (%)	25		
Plastic Limit (%)	15		
Plasticity Index (%)	**10**		

Linear Shrinkage (AS1289 3.4.1)		Min	Max
Linear Shrinkage (%)	4.5		
Cracking Crumbling Curling	Cracking		

Moisture Content (AS 1289 2.1.1)	
Moisture Content (%)	8.8

Particle Size Distribution



Material Test Report

Douglas Partners
Douglas Partners Pty Ltd
Darwin Laboratory
Unit 2/14 Caryota Crt COCONUT GROVE NT 0810
Phone: (08) 8948 6800
Fax: (08) 8948 6899
Email: dave.millard@douglaspartners.com.au
Accredited for compliance with ISO/IEC 17025 - Testing

Approved Signatory: Dave Millard
Engineering Geologist
NATA Accredited Laboratory Number: 828

Report Number:	**677612.00-1**
Issue Number:	2 - *This version supersedes all previous issues*
Date Issued:	29/01/2019
Client:	AECOM Australia Pty Ltd PO Box 73, Hunter Region NSW 2310
Contact:	Jace Emberg
Project Number:	677612.00
Project Name:	60480548 - Beetaloo soil samples
Project Location:	Beetaloo, NT
Work Request:	1466
Sample Number:	19-1466B
Date Sampled:	13/12/2018
Sampling Method:	AS1289 1.2.1 6.4 - Sampling from layers in earthworks or pavement - uncompacted/compacted
Sample Location:	**VELKERRI02 (0.0 - 0.4m)**
Material:	Silty SAND

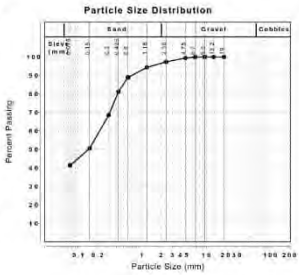
Particle Distribution (AS1289 3.6.1)			
Sieve	Passed %	Passing Limits	
19 mm	**100**		
13.2 mm	**100**		
9.5 mm	**100**		
6.7 mm	**100**		
4.75 mm	**99**		
2.36 mm	**97**		
1.18 mm	**94**		
0.6 mm	**89**		
0.425 mm	**81**		
0.3 mm	**68**		
0.15 mm	**50**		
0.075 mm	**41**		

Atterberg Limit (AS1289 3.1.2 & 3.2.1 & 3.3.1)		Min	Max
Sample History	Air Dried		
Preparation Method	Dry Sieve		
Liquid Limit (%)	18		
Plastic Limit (%)	13		
Plasticity Index (%)	**5**		

Linear Shrinkage (AS1289 3.4.1)		Min	Max
Linear Shrinkage (%)	3.5		
Cracking Crumbling Curling	Cracking		

Moisture Content (AS 1289 2.1.1)	
Moisture Content (%)	5.6

Particle Size Distribution



Material Test Report

Douglas Partners
Douglas Partners Pty Ltd
Darwin Laboratory
Unit 2/14 Caryota Crt COCONUT GROVE NT 0810
Phone: (08) 8948 6800
Fax: (08) 8948 6899
Email: dave.millard@douglaspartners.com.au
Accredited for compliance with ISO/IEC 17025 - Testing

Approved Signatory: Dave Millard
Engineering Geologist
NATA Accredited Laboratory Number: 828

Report Number:	**677612.00-1**
Issue Number:	2 - *This version supersedes all previous issues*
Date Issued:	29/01/2019
Client:	AECOM Australia Pty Ltd PO Box 73, Hunter Region NSW 2310
Contact:	Jace Emberg
Project Number:	677612.00
Project Name:	60480548 - Beetaloo soil samples
Project Location:	Beetaloo, NT
Work Request:	1466
Sample Number:	19-1466C
Date Sampled:	13/12/2018
Sampling Method:	AS1289 1.2.1 6.4 - Sampling from layers in earthworks or pavement - uncompacted/compacted
Sample Location:	**VELKERRI03 (0.0 - 0.4m)**
Material:	Silty SAND

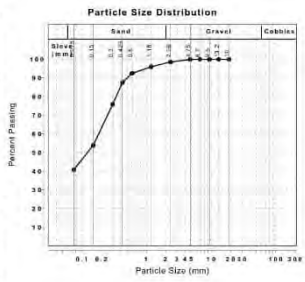
Particle Distribution (AS1289 3.6.1)			
Sieve	Passed %	Passing Limits	
19 mm	**100**		
13.2 mm	**100**		
9.5 mm	**100**		
6.7 mm	**100**		
4.75 mm	**100**		
2.36 mm	**99**		
1.18 mm	**96**		
0.6 mm	**92**		
0.425 mm	**88**		
0.3 mm	**76**		
0.15 mm	**54**		
0.075 mm	**41**		

Atterberg Limit (AS1289 3.1.2 & 3.2.1 & 3.3.1)		Min	Max
Sample History	Air Dried		
Preparation Method	Dry Sieve		
Liquid Limit (%)	19		
Plastic Limit (%)	12		
Plasticity Index (%)	**7**		

Linear Shrinkage (AS1289 3.4.1)		Min	Max
Linear Shrinkage (%)	3.5		
Cracking Crumbling Curling	Cracking		

Moisture Content (AS 1289 2.1.1)	
Moisture Content (%)	4.9

Particle Size Distribution



Material Test Report

Douglas Partners
Douglas Partners Pty Ltd
Darwin Laboratory
Unit 2/14 Caryota Crt COCONUT GROVE NT 0810
Phone: (08) 8948 6800
Fax: (08) 8948 6899
Email: dave.millard@douglaspartners.com.au
Accredited for compliance with ISO/IEC 17025 - Testing

Approved Signatory: Dave Millard
Engineering Geologist
NATA Accredited Laboratory Number: 828

Report Number:	**677612.00-1**
Issue Number:	2 - *This version supersedes all previous issues*
Date Issued:	29/01/2019
Client:	AECOM Australia Pty Ltd PO Box 73, Hunter Region NSW 2310
Contact:	Jace Emberg
Project Number:	677612.00
Project Name:	60480548 - Beetaloo soil samples
Project Location:	Beetaloo, NT
Work Request:	1466
Sample Number:	19-1466D
Date Sampled:	13/12/2018
Sampling Method:	AS1289 1.2.1 6.4 - Sampling from layers in earthworks or pavement - uncompacted/compacted
Sample Location:	**VELKERRI04 (0.0 - 0.4m)**
Material:	Silty SAND

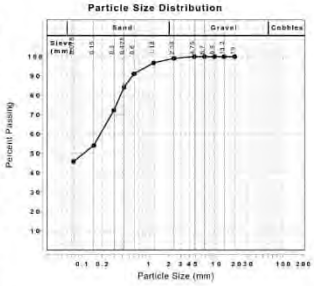
Particle Distribution (AS1289 3.6.1)			
Sieve	Passed %	Passing Limits	
19 mm	**100**		
13.2 mm	**100**		
9.5 mm	**100**		
6.7 mm	**100**		
4.75 mm	**100**		
2.36 mm	**99**		
1.18 mm	**97**		
0.6 mm	**91**		
0.425 mm	**84**		
0.3 mm	**72**		
0.15 mm	**54**		
0.075 mm	**46**		

Atterberg Limit (AS1289 3.1.2 & 3.2.1 & 3.3.1)		Min	Max
Sample History	Air Dried		
Preparation Method	Dry Sieve		
Liquid Limit (%)	24		
Plastic Limit (%)	13		
Plasticity Index (%)	**11**		

Linear Shrinkage (AS1289 3.4.1)		Min	Max
Linear Shrinkage (%)	4.5		
Cracking Crumbling Curling	Cracking		

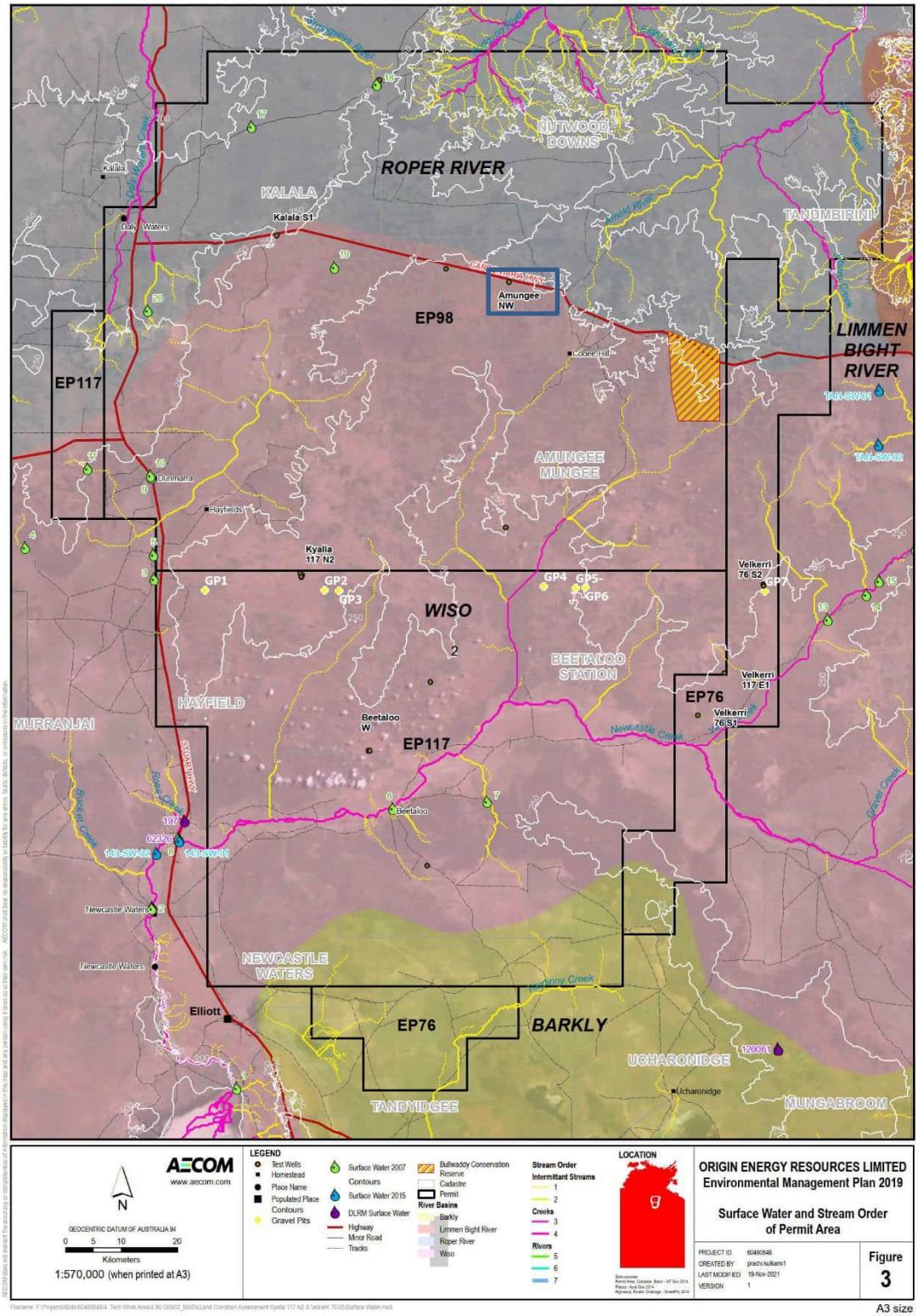
Moisture Content (AS 1289 2.1.1)	
Moisture Content (%)	5.2

Particle Size Distribution



Review due: 13/09/2025

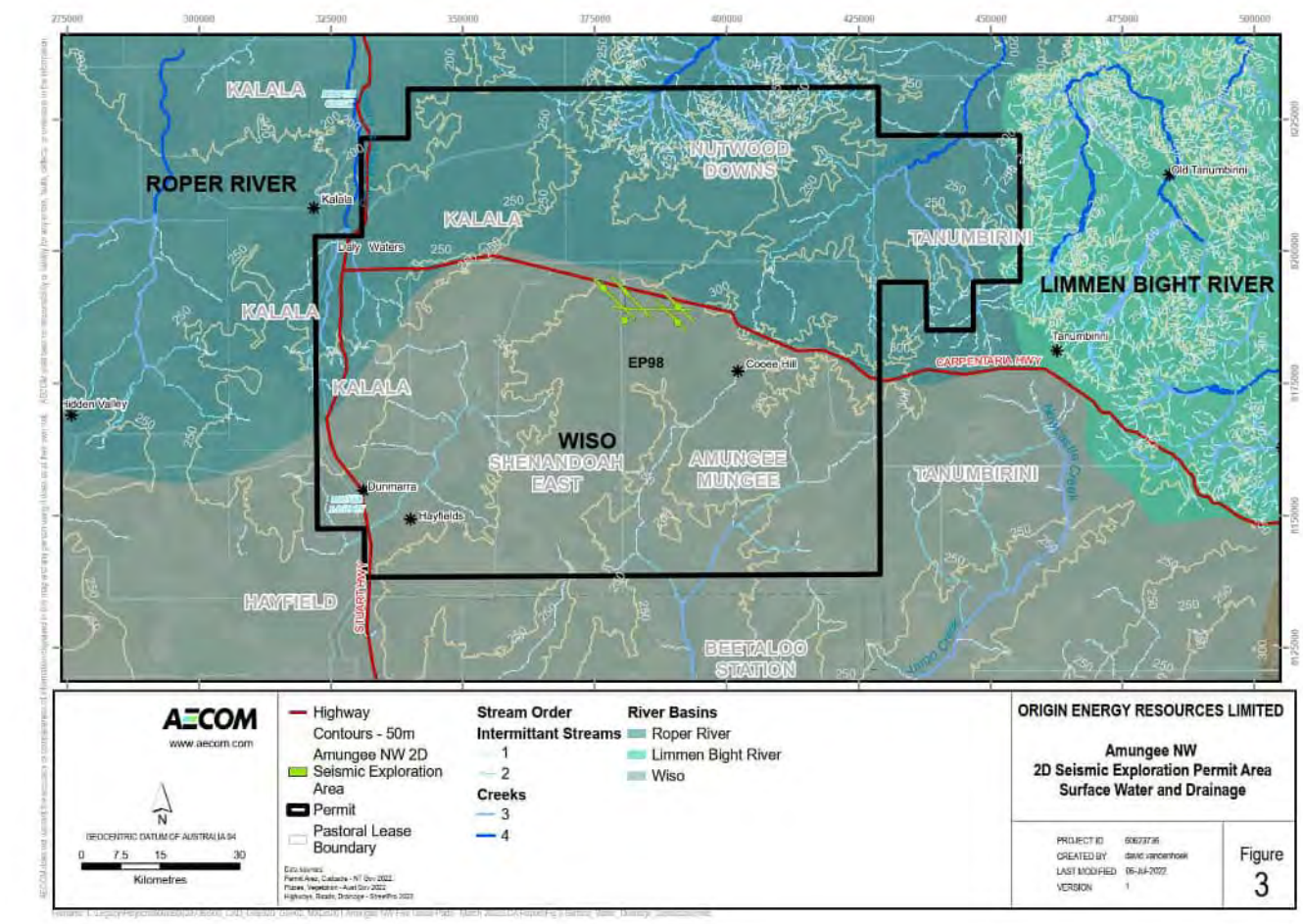
Appendix D Permit Area Surface Water



Review due: 13/09/2025

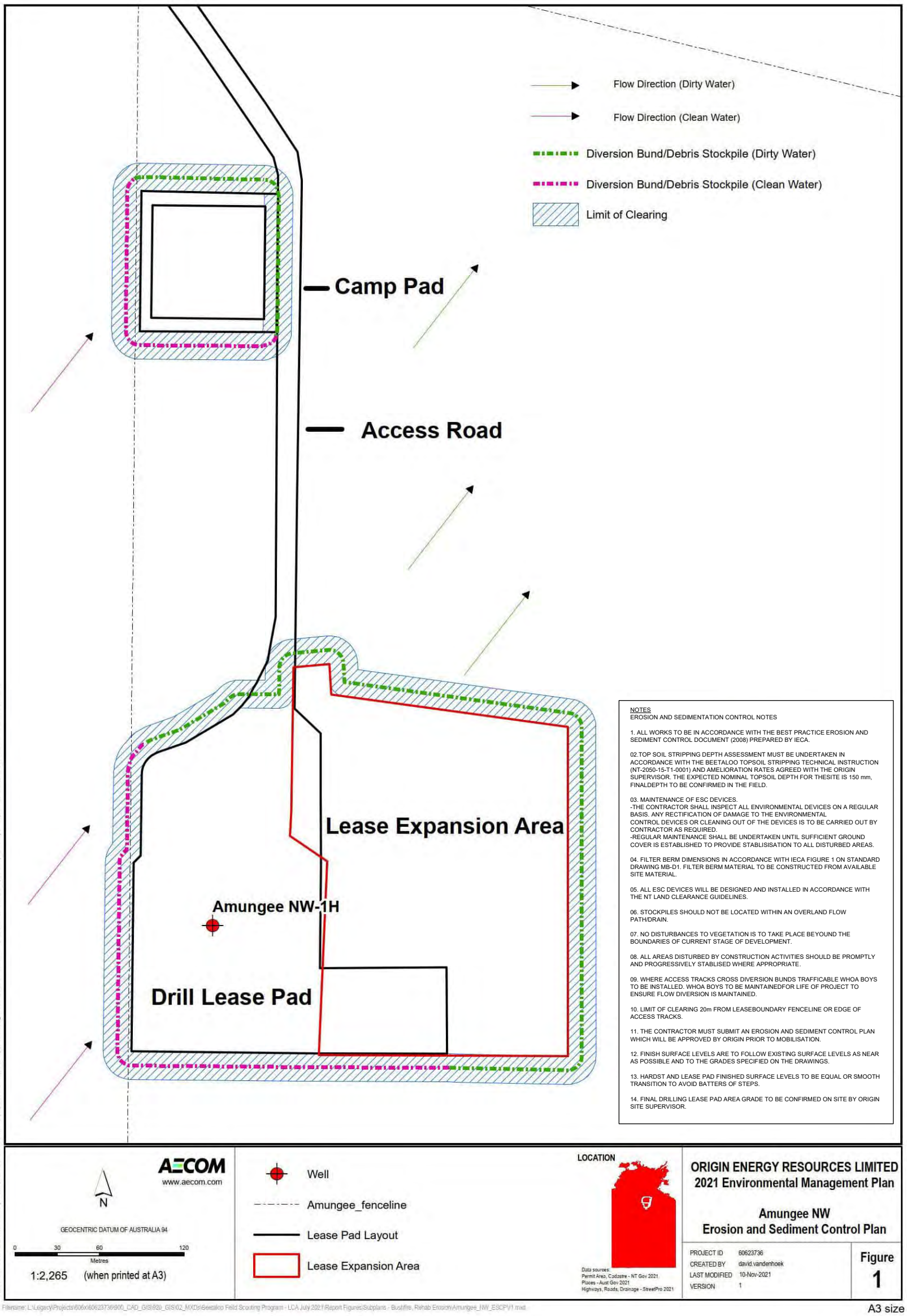
Erosion and Sediment Control Plan

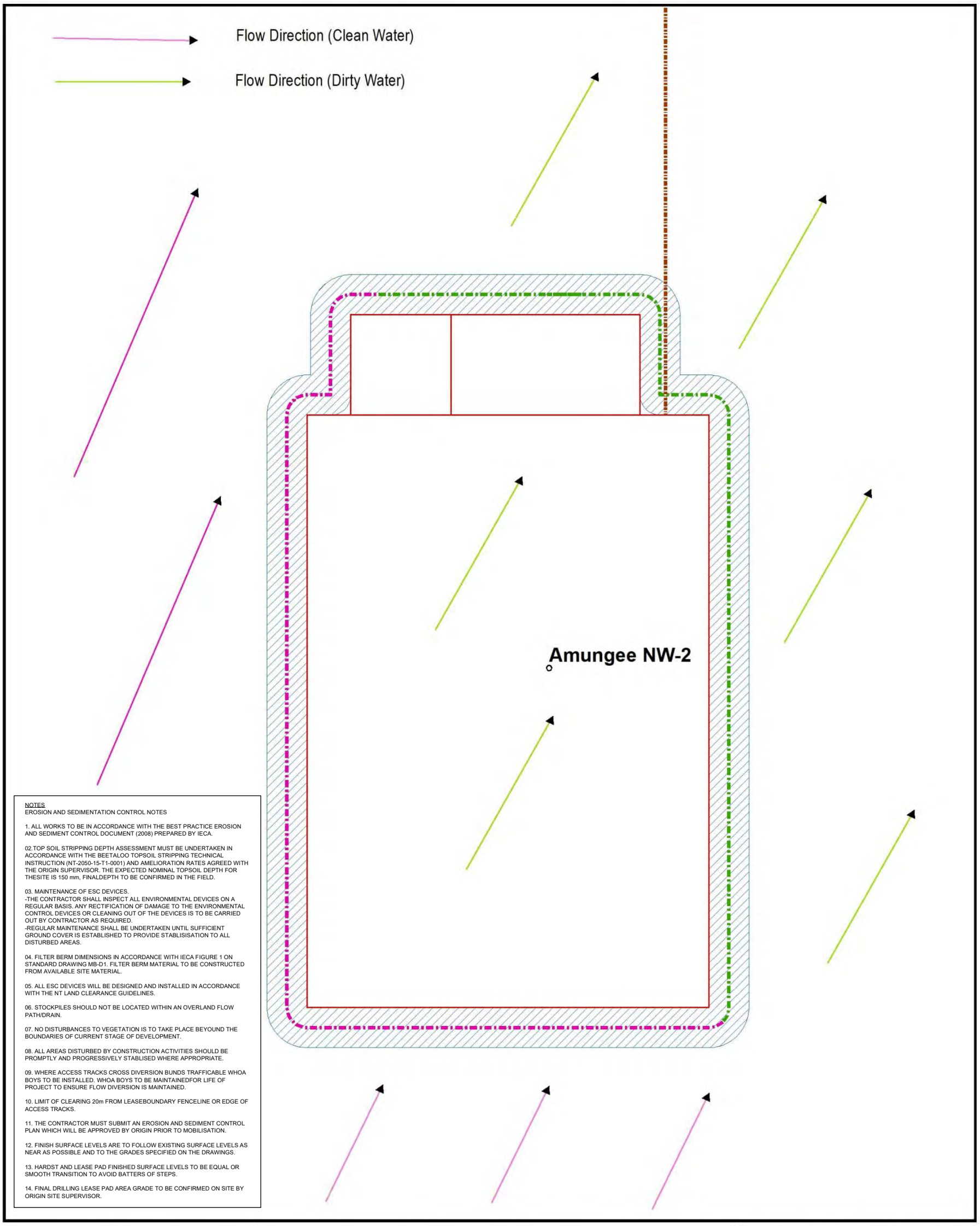
NT-2050-15-MP-019.



Review due: 13/09/2025

Appendix E Erosion and Sediment Control Plan for Amungee NW, Amungee NW-2, Amungee NW-3, Amungee NW-4 and Amungee NW-5





NOTES
EROSION AND SEDIMENTATION CONTROL NOTES

1. ALL WORKS TO BE IN ACCORDANCE WITH THE BEST PRACTICE EROSION AND SEDIMENT CONTROL DOCUMENT (2008) PREPARED BY IECA.

02.TOP SOIL STRIPPING DEPTH ASSESSMENT MUST BE UNDERTAKEN IN ACCORDANCE WITH THE BEETALOO TOPSOIL STRIPPING TECHNICAL INSTRUCTION (NT-2050-15-T1-0001) AND AMELIORATION RATES AGREED WITH THE ORIGIN SUPERVISOR. THE EXPECTED NOMINAL TOPSOIL DEPTH FOR THESITE IS 150 mm, FINALDEPTH TO BE CONFIRMED IN THE FIELD.

03. MAINTENANCE OF ESC DEVICES.
-THE CONTRACTOR SHALL INSPECT ALL ENVIRONMENTAL DEVICES ON A REGULAR BASIS. ANY RECTIFICATION OF DAMAGE TO THE ENVIRONMENTAL CONTROL DEVICES OR CLEANING OUT OF THE DEVICES IS TO BE CARRIED OUT BY CONTRACTOR AS REQUIRED.
-REGULAR MAINTENANCE SHALL BE UNDERTAKEN UNTIL SUFFICIENT GROUND COVER IS ESTABLISHED TO PROVIDE STABLISISATION TO ALL DISTURBED AREAS.

04. FILTER BERM DIMENSIONS IN ACCORDANCE WITH IECA FIGURE 1 ON STANDARD DRAWING MB-D1. FILTER BERM MATERIAL TO BE CONSTRUCTED FROM AVAILABLE SITE MATERIAL.

05. ALL ESC DEVICES WILL BE DESIGNED AND INSTALLED IN ACCORDANCE WITH THE NT LAND CLEARANCE GUIDELINES.

06. STOCKPILES SHOULD NOT BE LOCATED WITHIN AN OVERLAND FLOW PATH/DRAIN.

07. NO DISTURBANCES TO VEGETATION IS TO TAKE PLACE BEYOUND THE BOUNDARIES OF CURRENT STAGE OF DEVELOPMENT.

08. ALL AREAS DISTURBED BY CONSTRUCTION ACTIVITIES SHOULD BE PROMPTLY AND PROGRESSIVELY STABLISED WHERE APPROPRIATE.

09. WHERE ACCESS TRACKS CROSS DIVERSION BUNDS TRAFFICABLE WHOA BOYS TO BE INSTALLED. WHOA BOYS TO BE MAINTAINEDFOR LIFE OF PROJECT TO ENSURE FLOW DIVERSION IS MAINTAINED.

10. LIMIT OF CLEARING 20m FROM LEASEBOUNDARY FENCELINE OR EDGE OF ACCESS TRACKS.

11. THE CONTRACTOR MUST SUBMIT AN EROSION AND SEDIMENT CONTROL PLAN WHICH WILL BE APPROVED BY ORIGIN PRIOR TO MOBILISATION.

12. FINISH SURFACE LEVELS ARE TO FOLLOW EXISTING SURFACE LEVELS AS NEAR AS POSSIBLE AND TO THE GRADES SPECIFIED ON THE DRAWINGS.

13. HARDST AND LEASE PAD FINISHED SURFACE LEVELS TO BE EQUAL OR SMOOTH TRANSITION TO AVOID BATTERS OF STEPS.

14. FINAL DRILLING LEASE PAD AREA GRADE TO BE CONFIRMED ON SITE BY ORIGIN SITE SUPERVISOR.

AECOM
www.aecom.com

N

Projection: GDA94 MGA Zone 53

0 25 50 100
Meters

Proposed Tracks
Clean Water
Dirty Water
Infrastructure
Water Diversion Line

Data sources:
Co-ordinate system: GDA_1994_MGA_Zone_53
Permit Area, Cadastre - NT Gov 2019.
Places, Vegetation - Aust Gov 2019

ORIGIN ENERGY RESOURCES LIMITED
2022 Amungee NW Exploraiton Well Pads

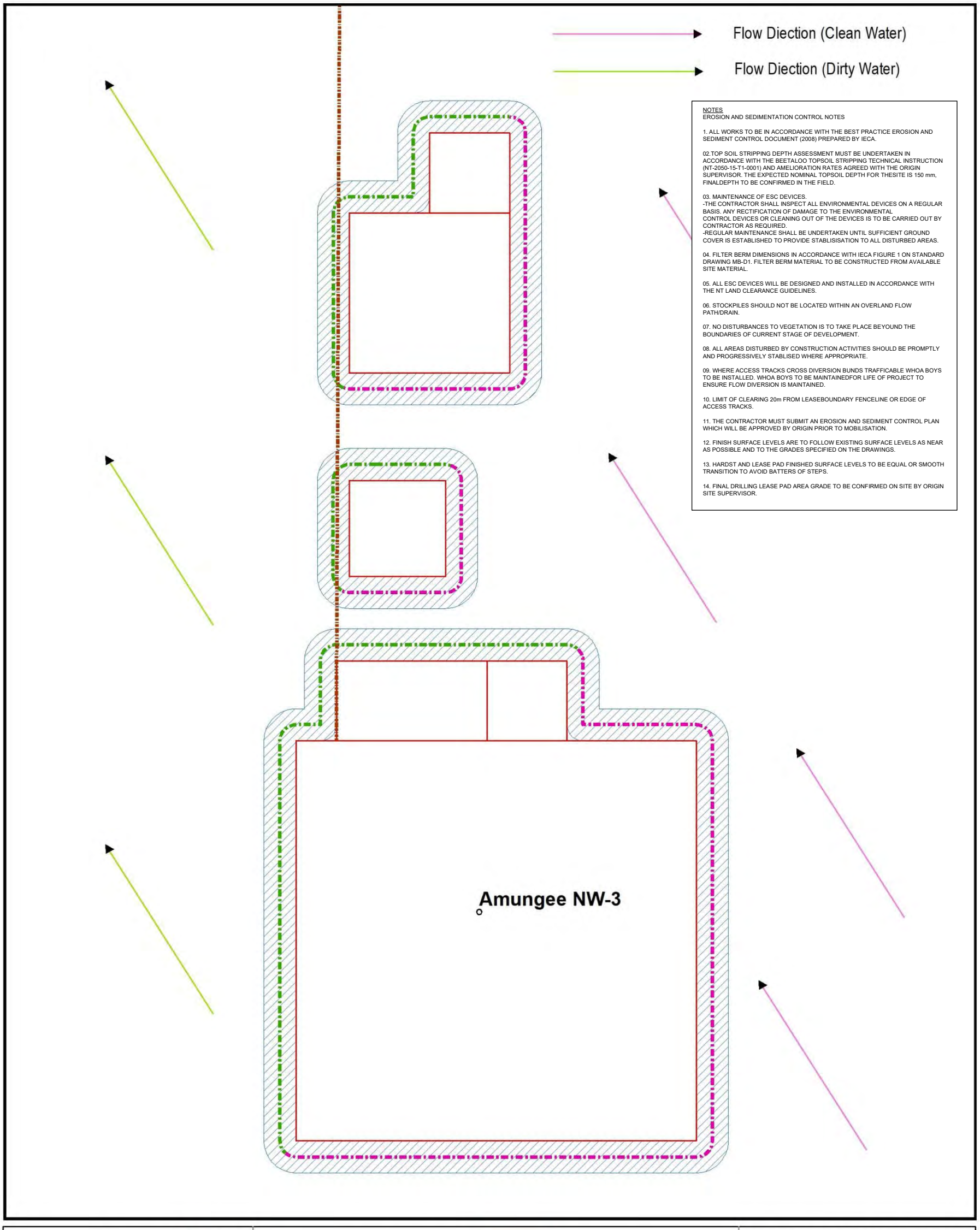
Amungee NW-2 Lease Pad Areas

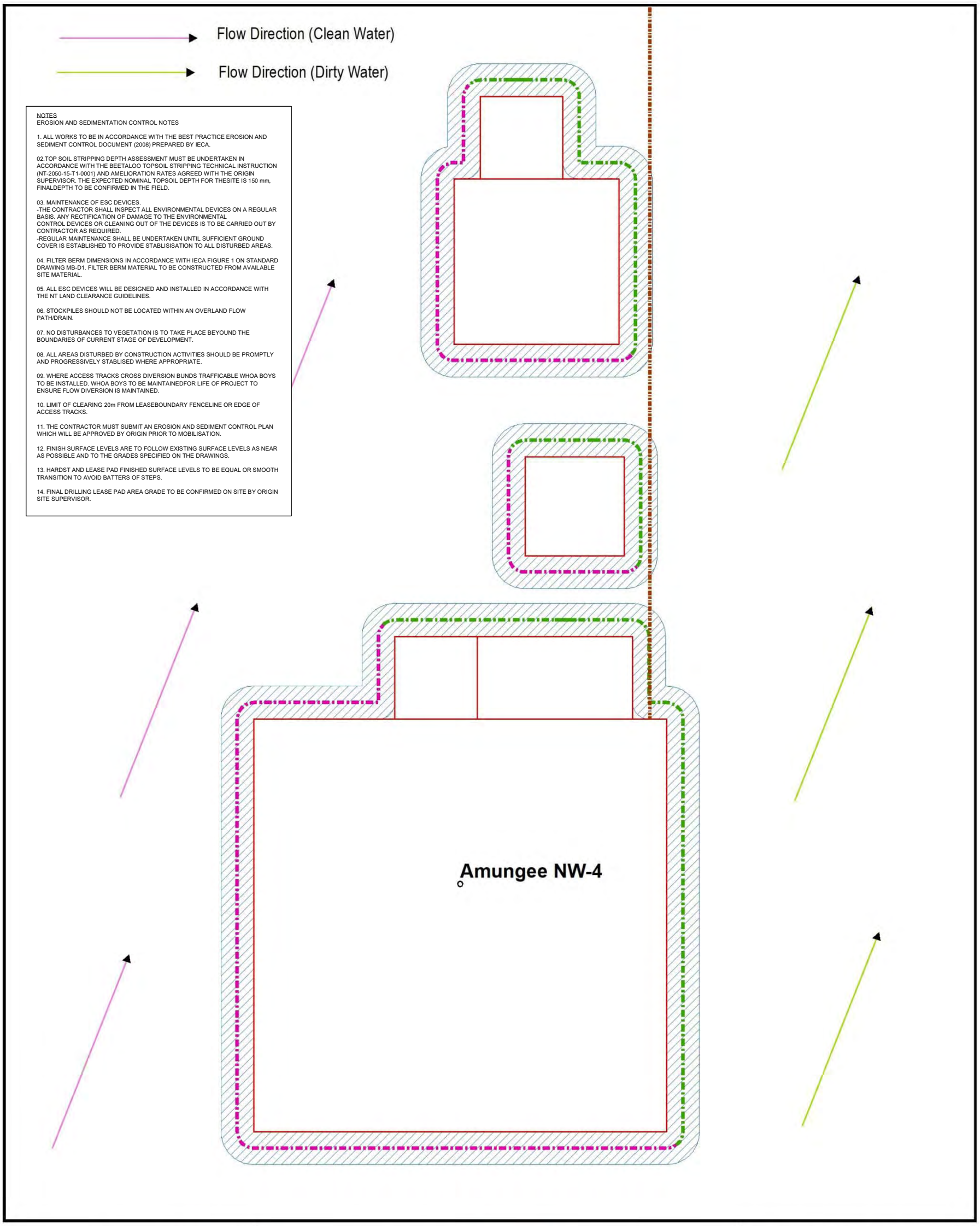
PROJECT ID	60480548
CREATED BY	david.vandenhoek
LAST MODIFIED	08-Jul-2022
VERSION	1

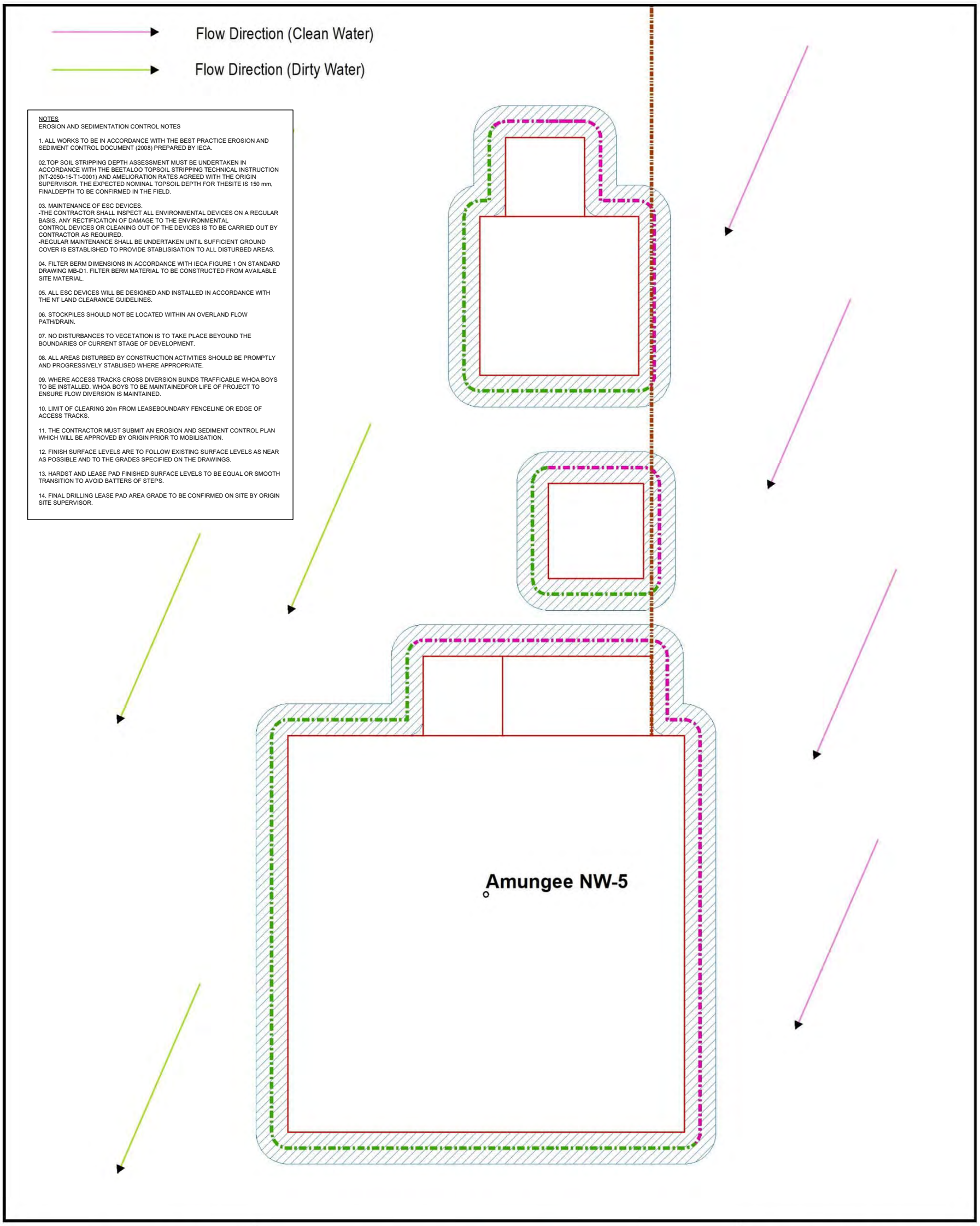
Filename: L:\Legacy\Projects\606x\60623736\900_CAD_GIS\920_GIS\02_MXDs\001 Amungee NW Five Lease Pads - March 2022\LCA Report\Fire, rehab and erosion\Amungee NW-2 ESCP.mxd

Erosion and Sediment Control Plan

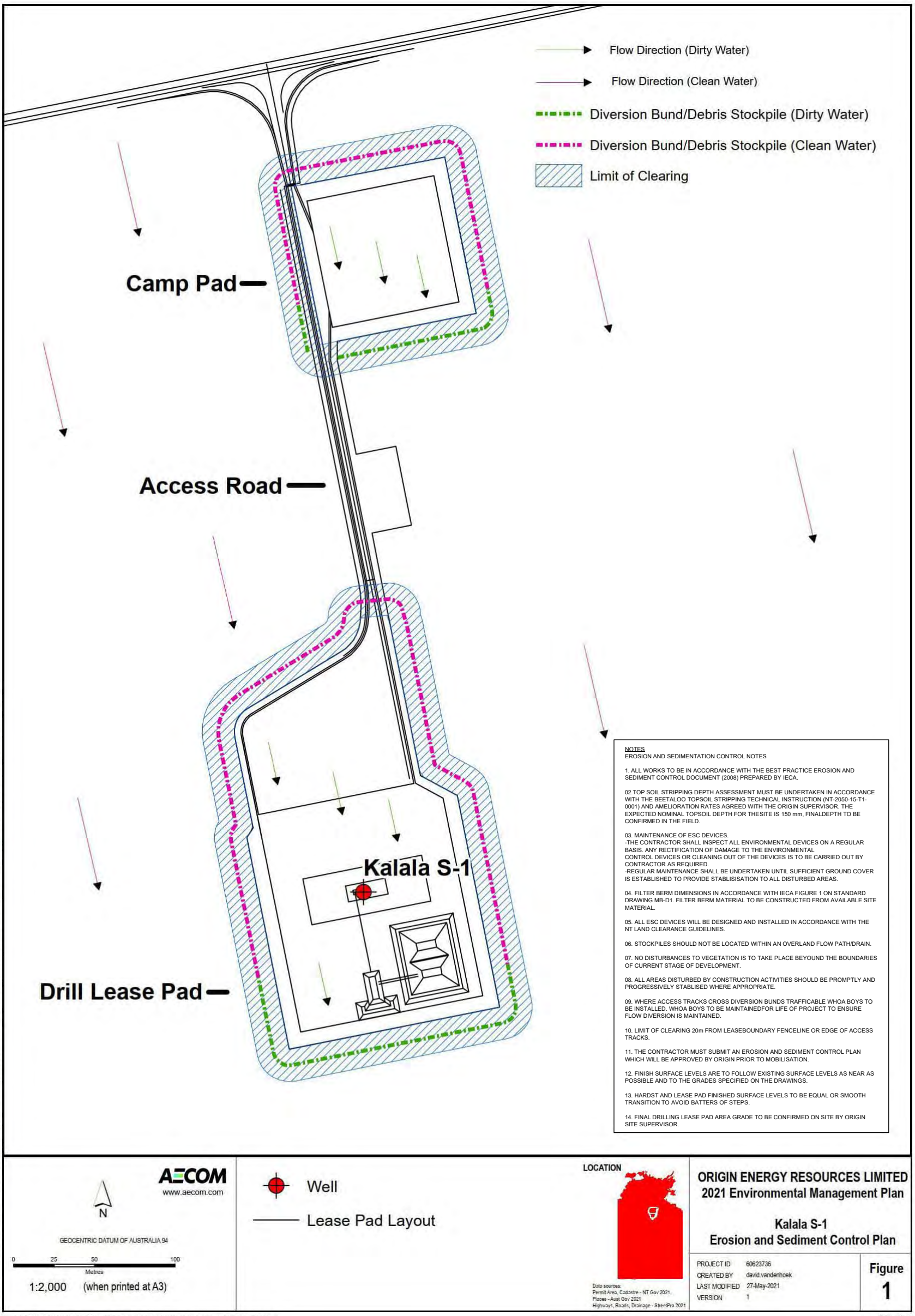
NT-2050-15-MP-019.





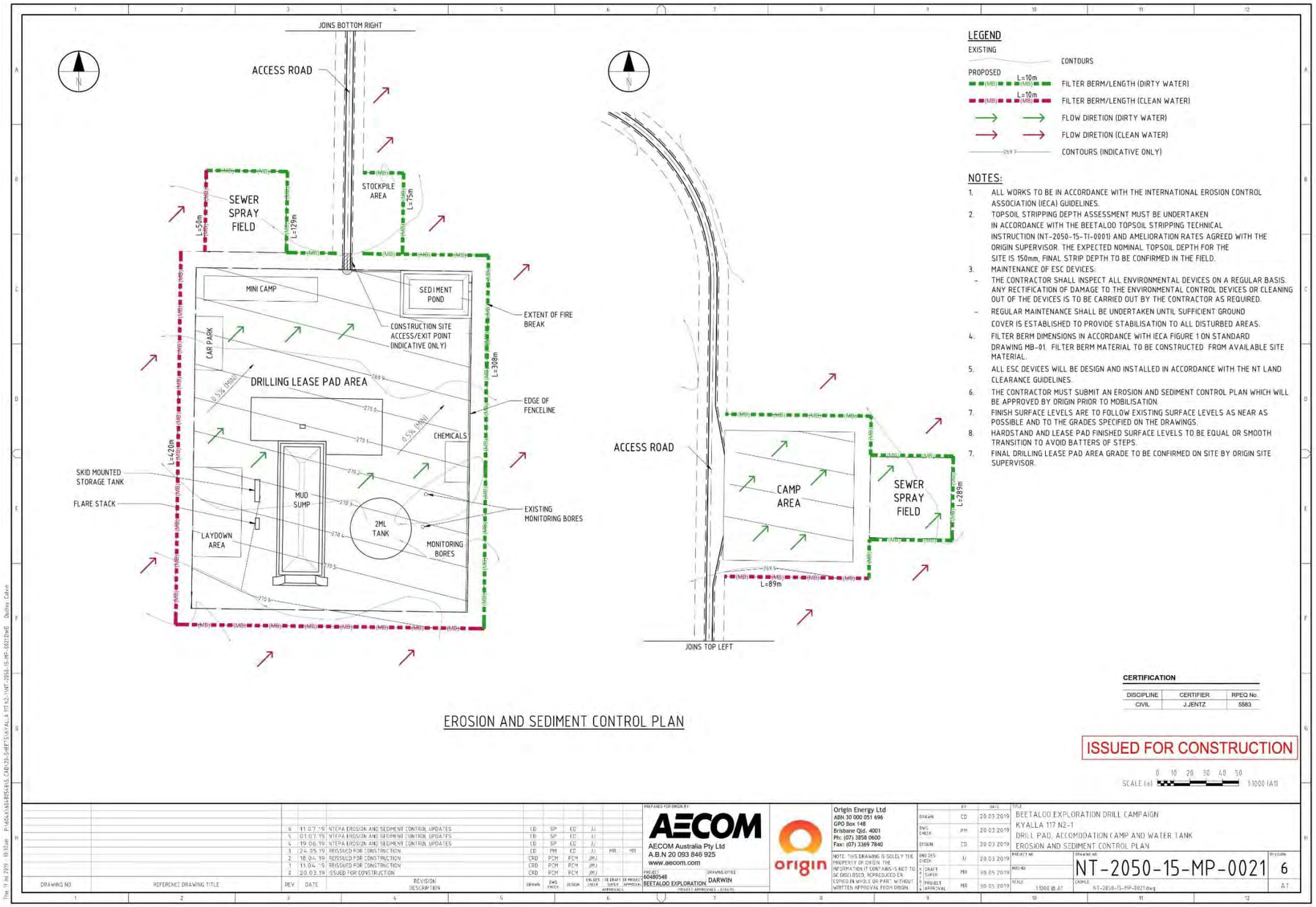


Appendix F Erosion and Sediment Control Plan for Kalala S1

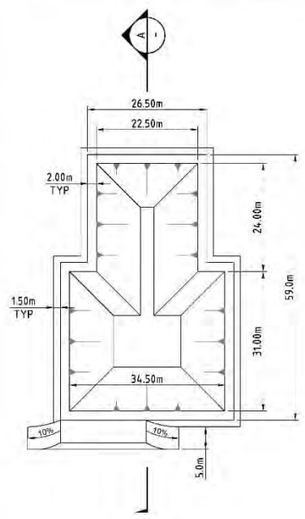


Review due: 13/09/2025

Appendix G Erosion and Sediment Control Plan for Kyalla 117-N2

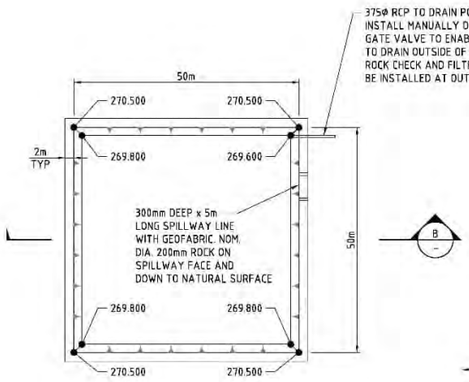


Review due: 13/09/2025



PLAN - MUD SUMP

Scale N.T.S.



PLAN - SECONDARY CONTAINMENT

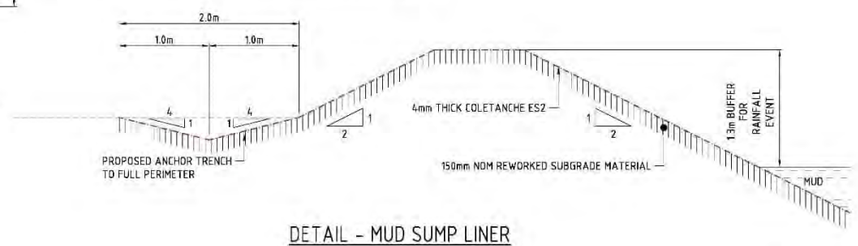
Scale N.T.S.

NOTES:

1. ALL UNITS ARE IN MILLIMETRES (mm) UNLESS NOTED OTHERWISE.
2. GROUND CONDITIONS AND SUITABLE MATERIALS TO BE CONFIRMED ON SITE BY SUPERINTENDENT.
3. REFER TO SPECIFICATION FOR COMPACTION REQUIREMENTS.
4. REFER TO SPECIFICATION FOR GENERAL REQUIREMENTS.
5. THE CONTRACTOR IS TO NOTIFY THE ORIGIN SUPERVISOR IF AREAS OF SOFT GROUND ARE ENCOUNTERED OR IF THE SPECIFICATION CANNOT BE ACHIEVED.
6. REFER TO DRAWING NT-2050-20-DA-0022 FOR SETOUT DETAILS OF MUD SUMP.
7. FAUNA LADDERS TO BE INSTALLED TO MUD SUMP.
8. CONTRACTOR TO PROVIDE CATTLE PANELS FOR FENCING AROUND SUMP WHERE DEPTH IS GREATER THAN 1.5m.
9. FENCE OR BARRICADE TO BE INSTALLED AT SUMP LOAD OUT LOCATION TO ENSURE VEHICLES DO NOT COMPROMISE SUMP WALL/ENTER SUM.
10. MUD SUMP VEHICLE OFFLOAD AREA TO BE COMPACTED TO 200 KPa

NOTES:

1. GEOSYNTHETICS ARE DRAWN EXAGGERATED FOR CLARITY.



DETAIL - MUD SUMP LINER

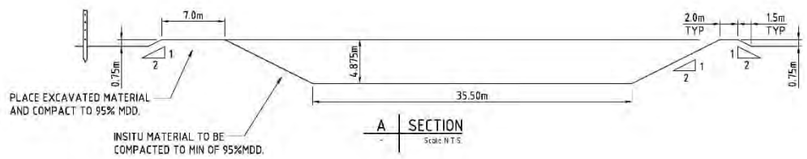
Scale N.T.S.

DETAIL - BASE LINER

Scale N.T.S.

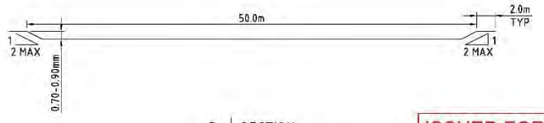
CERTIFICATION

DISCIPLINE	CERTIFIER	RPEQ No.
CIVIL	J.JENTZ	5583



A | SECTION

Scale N.T.S.



B | SECTION

Scale N.T.S.

ISSUED FOR CONSTRUCTION

REV	DATE	REVISION DESCRIPTION	DRAWN	CHK	DESIGN	ENG DISC CHECK	GE DRAFT ES PROJECT APPROVAL
1	18.04.19	RESSUED FOR CONSTRUCTION	CRD	PCM	PCM	JMJ	
0	11.04.19	ISSUED FOR CONSTRUCTION	CRD	PCM	PCM	JMJ	

DRAWING NO.	REFERENCE DRAWING TITLE
NT-2050-20-DA-0022	GENERAL ARRANGEMENT PLAN

PREPARED FOR ORIGIN BY:

AECOM

AECOM Australia Pty Ltd
A.B.N 20 093 846 925
www.aecom.com

PROJECT: 60480548 | DRAWING OFFICE: DARWIN
BEETALOO EXPLORATION
PROJECT APPROVALS - OTHERS

Origin Energy Ltd
ABN 30 000 051 696
GPO Box 148
Brisbane Qld. 4001
Ph: (07) 3858 0600
Fax: (07) 3369 7840

NOTE: THIS DRAWING IS SOLELY THE PROPERTY OF ORIGIN. THE INFORMATION IT CONTAINS IS NOT TO BE DISCLOSED, REPRODUCED OR COPIED IN WHOLE OR PART WITHOUT WRITTEN APPROVAL FROM ORIGIN.

	BY	DATE
DRAWN	CRD	20.03.2019
DWG CHECK	PCM	20.03.2019
DESIGN	PCM	20.03.2019
ENGINEERS CHECK	JMJ	20.03.2019
C DRAFT ES PROJECT APPROVAL		
A PROJECT APPROVAL		

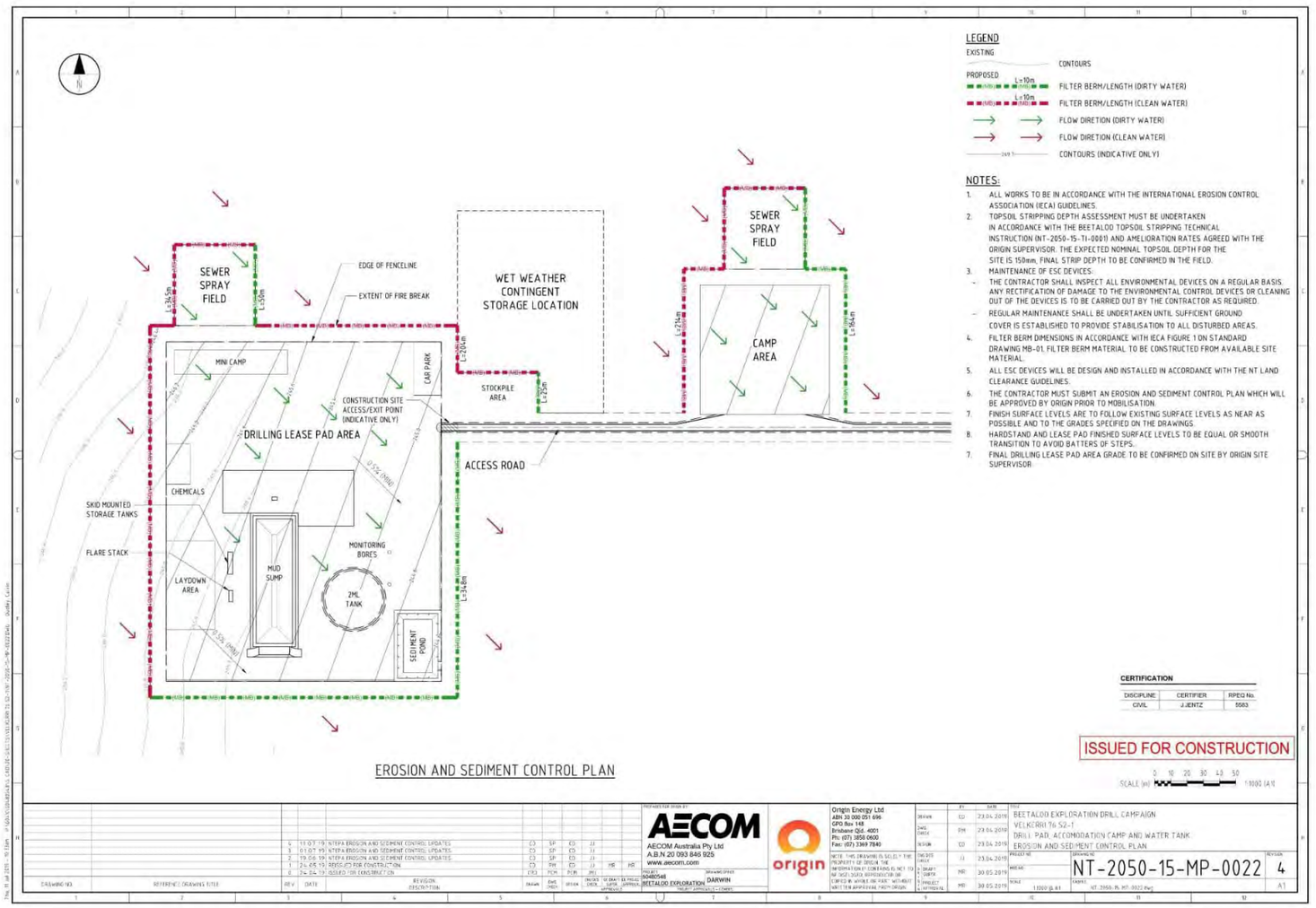
TITLE
BEETALOO EXPLORATION DRILL CAMPAIGN
KYALLA 117 N2-1
MUD SUMP DETAILS

PROJECT NO.
PDE NO.
DRAWING NO. NT-2050-20-DD-0030
REVISION 1
SCALE 1:1000 @ A1
DATAFILE NT-2050-20-DD-0030.dwg
A1

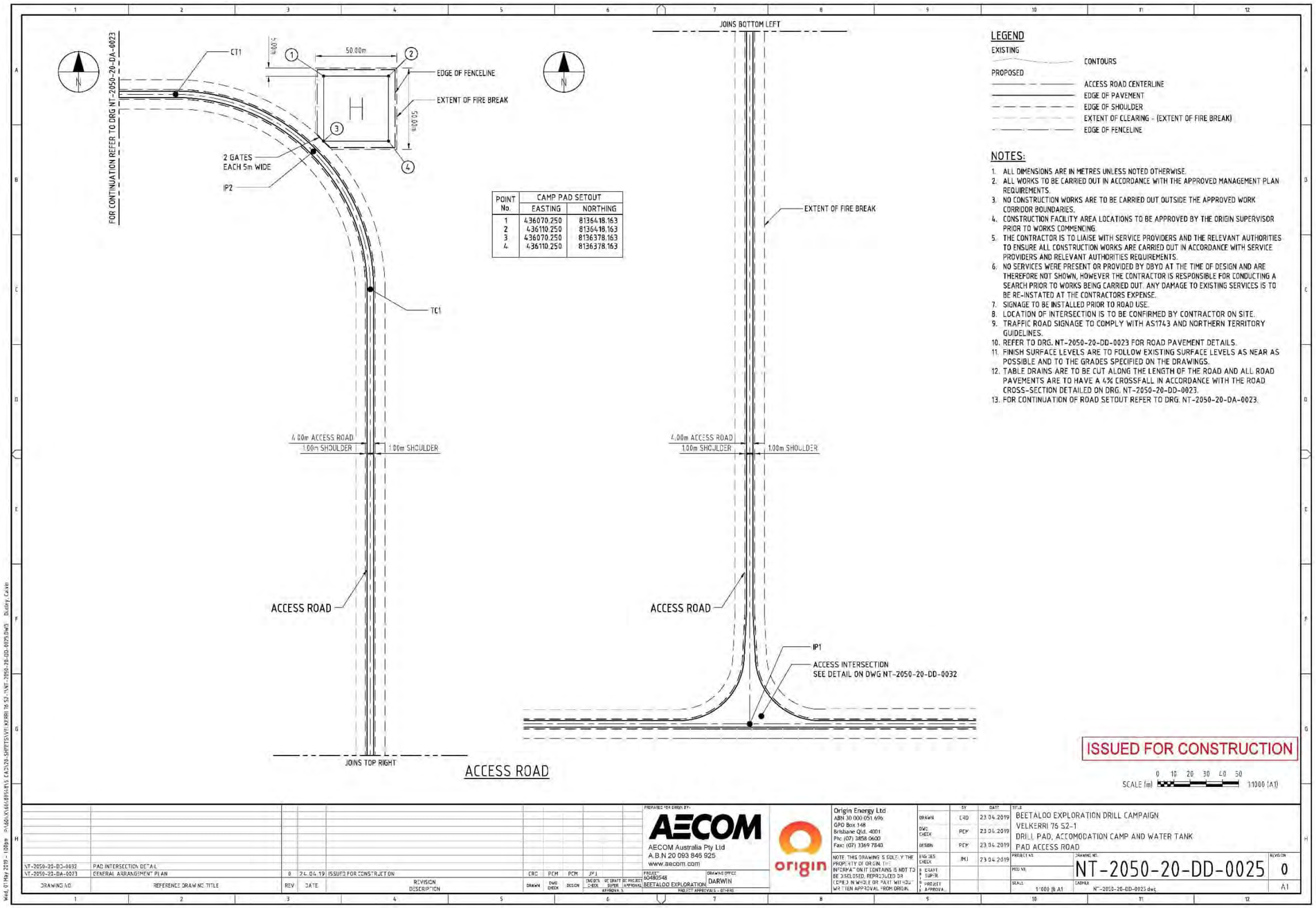
Wed 01 May 2019 - 1:18pm - P:\GAS\KYALLA\PROJECTS\KYALLA 117 N2-1\NT-2050-20-DD-0030.DWG - Dudley, Calvin

Review due: 13/09/2025

Appendix H Erosion and Sediment Control Plan for Velkerri 76 S2



Review due: 13/09/2025



POINT No.	CAMP PAD SETOUT	
	EASTING	NORTHING
1	436070.250	8136418.163
2	436110.250	8136418.163
3	436070.250	8136378.163
4	436110.250	8136378.163

LEGEND

EXISTING

CONTOURS

PROPOSED

ACCESS ROAD CENTERLINE

EDGE OF PAVEMENT

EDGE OF SHOULDER

EXTENT OF CLEARING - (EXTENT OF FIRE BREAK)

EDGE OF FENCELINE

NOTES:

1. ALL DIMENSIONS ARE IN METRES UNLESS NOTED OTHERWISE.
2. ALL WORKS TO BE CARRIED OUT IN ACCORDANCE WITH THE APPROVED MANAGEMENT PLAN REQUIREMENTS.
3. NO CONSTRUCTION WORKS ARE TO BE CARRIED OUT OUTSIDE THE APPROVED WORK CORRIDOR BOUNDARIES.
4. CONSTRUCTION FACILITY AREA LOCATIONS TO BE APPROVED BY THE ORIGIN SUPERVISOR PRIOR TO WORKS COMMENCING.
5. THE CONTRACTOR IS TO LIAISE WITH SERVICE PROVIDERS AND THE RELEVANT AUTHORITIES TO ENSURE ALL CONSTRUCTION WORKS ARE CARRIED OUT IN ACCORDANCE WITH SERVICE PROVIDERS AND RELEVANT AUTHORITIES REQUIREMENTS.
6. NO SERVICES WERE PRESENT OR PROVIDED BY DBYD AT THE TIME OF DESIGN AND ARE THEREFORE NOT SHOWN, HOWEVER THE CONTRACTOR IS RESPONSIBLE FOR CONDUCTING A SEARCH PRIOR TO WORKS BEING CARRIED OUT. ANY DAMAGE TO EXISTING SERVICES IS TO BE RE-INSTATED AT THE CONTRACTORS EXPENSE.
7. SIGNAGE TO BE INSTALLED PRIOR TO ROAD USE.
8. LOCATION OF INTERSECTION IS TO BE CONFIRMED BY CONTRACTOR ON SITE.
9. TRAFFIC ROAD SIGNAGE TO COMPLY WITH AS1743 AND NORTHERN TERRITORY GUIDELINES.
10. REFER TO DRG. NT-2050-20-DD-0023 FOR ROAD PAVEMENT DETAILS.
11. FINISH SURFACE LEVELS ARE TO FOLLOW EXISTING SURFACE LEVELS AS NEAR AS POSSIBLE AND TO THE GRADES SPECIFIED ON THE DRAWINGS.
12. TABLE DRAINS ARE TO BE CUT ALONG THE LENGTH OF THE ROAD AND ALL ROAD PAVEMENTS ARE TO HAVE A 4% CROSSFALL IN ACCORDANCE WITH THE ROAD CROSS-SECTION DETAILED ON DRG. NT-2050-20-DD-0023.
13. FOR CONTINUATION OF ROAD SETOUT REFER TO DRG. NT-2050-20-DA-0023.

ISSUED FOR CONSTRUCTION

SCALE (m) 0 10 20 30 40 50 1:1000 (A1)

DRAWING NO	REFERENCE DRAWING TITLE
NT-2050-20-DD-0032	PAD INTERSECTION DETAIL
NT-2050-20-DA-0023	GENERAL ARRANGEMENT PLAN

REV	DATE	REVISION DESCRIPTION	CRG	PCM	PCM	JM1
			DRAWN	DWG CHECK	DESIGN	ORIGIN'S CHECK / DC DRAFT SUPER APPROVAL / DC PROJECT APPROVAL
0	24.04.19	ISSUED FOR CONSTRUCTION				

PREPARED FOR ORIGIN BY:
AECOM
AECOM Australia Pty Ltd
A.B.N 20 093 846 925
www.aecom.com
PROJECT 60480548
DRAWING OFFICE DARWIN
BEETALOO EXPLORATION
PROJECT APPROVALS - OTHERS

Origin Energy Ltd
ABN 30 000 051 696
GPO Box 148
Brisbane Qld. 4001
Ph: (07) 3858 0600
Fax: (07) 3369 7840

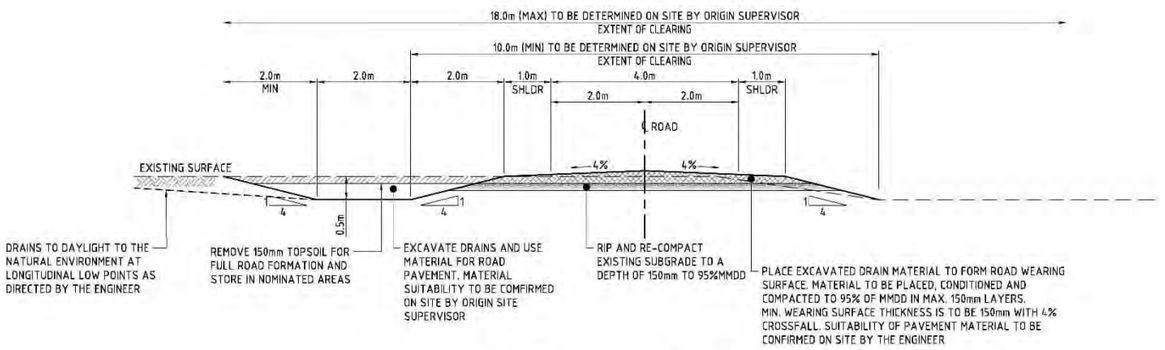
NOTE: THIS DRAWING IS SOLELY THE PROPERTY OF ORIGIN. THE INFORMATION IT CONTAINS IS NOT TO BE DISCLOSED, REPRODUCED OR COPIED IN WHOLE OR PART WITHOUT WRITTEN APPROVAL FROM ORIGIN.

	BY	DATE
DRAWN	CAD	23.04.2019
DWG CHECK	PCM	23.04.2019
DESIGN	PCM	23.04.2019
ENGINEERS CHECK	JM1	23.04.2019
ORIGIN'S SUPER		
PROJECT APPROVAL		

TITLE
BEETALOO EXPLORATION DRILL CAMPAIGN
VELKERRI 76 S2-1
DRILL PAD, ACCOMODATION CAMP AND WATER TANK
PAD ACCESS ROAD

PROJECT NO.
PKG NO.
DRAWING NO. NT-2050-20-DD-0025
REVISION 0
SCALE 1:1000 @ A1
CADFILE NT-2050-20-DD-0025.dwg
A1

Wed, 01 May 2019 - 1:08pm P:\560\X\560489\815 EA\520-SHEETS\VF\KERRI 76 S2-1\NT-2050-20-DD-0025.DWG Dudley Calvin



TYPICAL CROSS SECTION - ACCESS ROAD

SCALE (m) 0 0.5 1.0 1.5 2.0 2.5 — 1:50 (A1)

NOTES:

1. ENSURE THAT TABLE DRAINS ARE FREE DRAINING.
2. GROUND CONDITIONS AND SUITABLE MATERIALS TO BE CONFIRMED ON SITE BY SUPERINTENDENT.
3. CLEARED VEGETATION TO BE STORED ALONGSIDE NEW ROAD FOR FUTURE REHABILITATION.
4. REFER TO SPECIFICATION FOR COMPACTION REQUIREMENTS.
5. STRIPPED TOPSOIL TO BE STORED IN BERMS ALONGSIDE ROAD.
6. REFER TO SPECIFICATION FOR GENERAL REQUIREMENTS.
7. THE CONTRACTOR IS TO NOTIFY THE ORIGIN SUPERVISOR IF AREAS OF SOFT GROUND ARE ENCOUNTERED OR IF THE SPECIFICATION CANNOT BE ACHIEVED.
8. CLEAR, STRIP 150mm TOPSOIL AND STOCKPILE IN NOMINATED BUFFER ZONE TO ESTABLISH BORROW PITS.
9. THIS DRAWING MAY BE USED FOR TEMPORARY ACCESS APPLICATION TO NT ROADS.
10. TYPICAL CROSS SECTION DESIGNED IN ACCORDANCE WITH NORTHERN TERRITORY STANDARD DRAWING C(S)3003.

ISSUED FOR CONSTRUCTION

DRAWING NO.	REFERENCE DRAWING TITLE
NT-2050-20-DA-0023	GENERAL ARRANGEMENT PLAN

REV	DATE	REVISION DESCRIPTION	DRAWN	DWG CHECK	DESIGN	ENG DES CHECK	IE DRAFT SUPER	IE REVIEW APPROVAL
0	24.04.19	ISSUED FOR CONSTRUCTION	CRD	PCM	PCM	JM J		

PREPARED FOR ORIGIN BY:
AECOM
AECOM Australia Pty Ltd
A.B.N 20 093 846 925
www.aecom.com

PROJECT: 60480548
DRAWING OFFICE: DARWIN
BEETALOO EXPLORATION
PROJECT APPROVALS - ETHERS

Origin Energy Ltd
ABN 30 000 051 696
GPO Box 148
Brisbane Qld. 4001
Ph: (07) 3858 0600
Fax: (07) 3369 7840

NOTE: THIS DRAWING IS SOLELY THE PROPERTY OF ORIGIN. THE INFORMATION IT CONTAINS IS NOT TO BE DISCLOSED, REPRODUCED OR COPIED IN WHOLE OR PART WITHOUT WRITTEN APPROVAL FROM ORIGIN

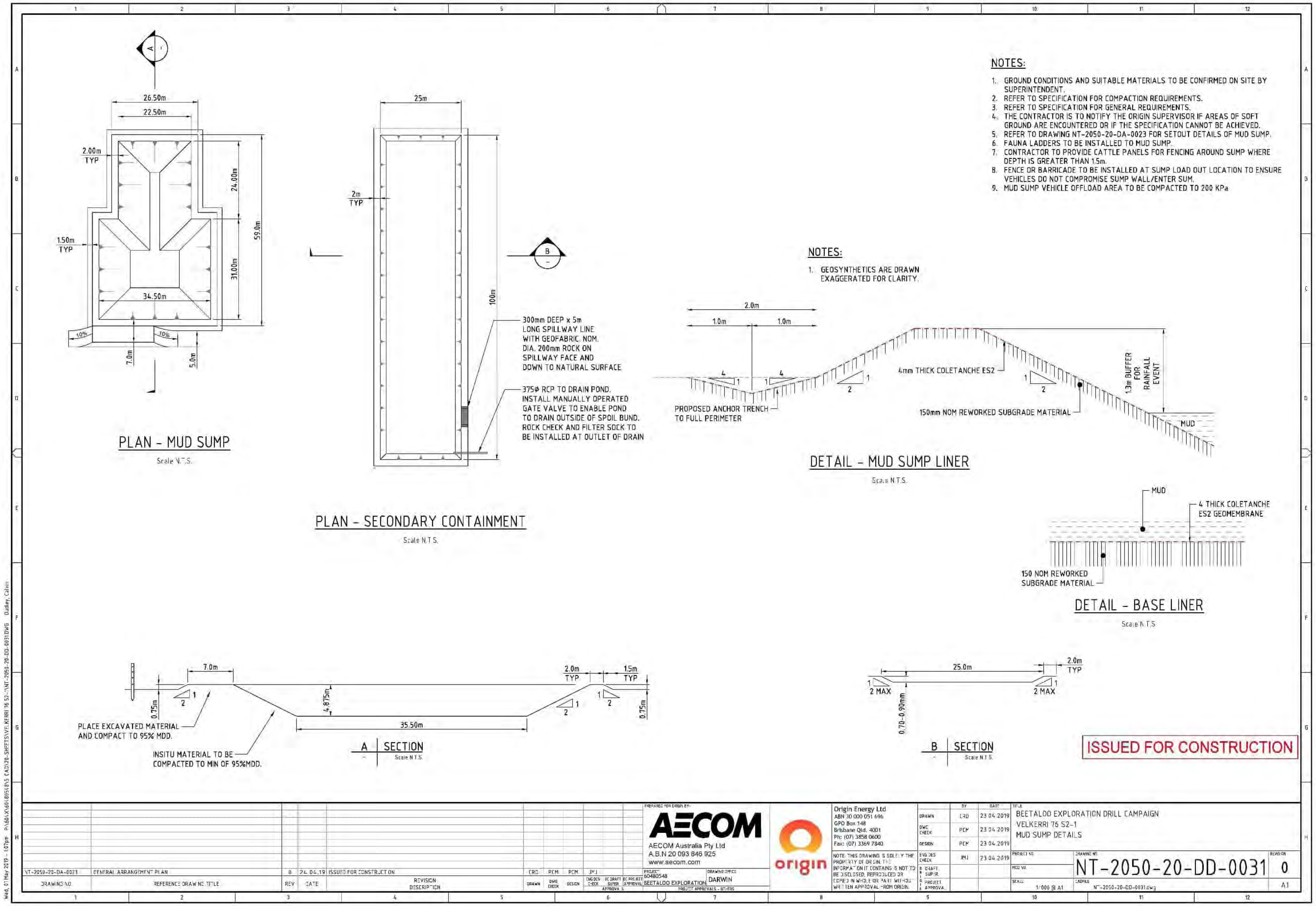
	BY	DATE
DRAWN	CRD	23.04.2019
DWG CHECK	PCM	23.04.2019
DESIGN	PCM	23.04.2019
ENG DES CHECK	JM J	23.04.2019
IE DRAFT SUPER		
IE PROJECT APPROVAL		

TITLE: BEETALOO EXPLORATION DRILL CAMPAIGN
VELKERRI 76 S2-1
DESIGN DETAILS

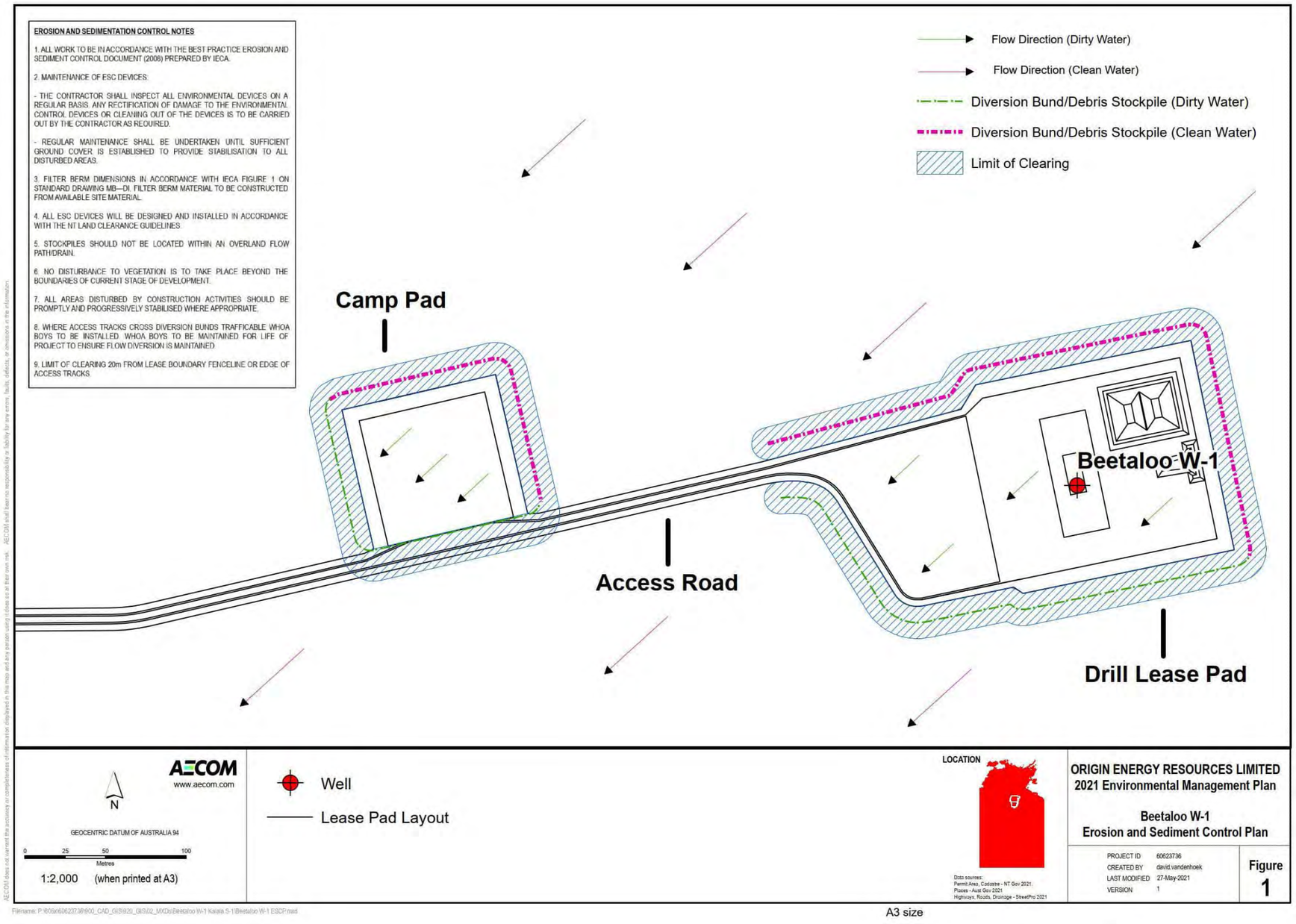
PROJECT NO.
PCM NO.
DRAWING NO.: NT-2050-20-DD-0023
REVISION: 0
SCALE: 1:1000 @ A1
CADFILE: NT-2050-20-DD-0023.dwg
A1

Wed, 01 May 2019 - 1:06pm P:\561\X\NT\605615 CAD\20_SHEETS\VELKERRI 76 S2-1\NT-2050-20-DD-0023.DWG Dudley, Calvin

Review due: 13/09/2025



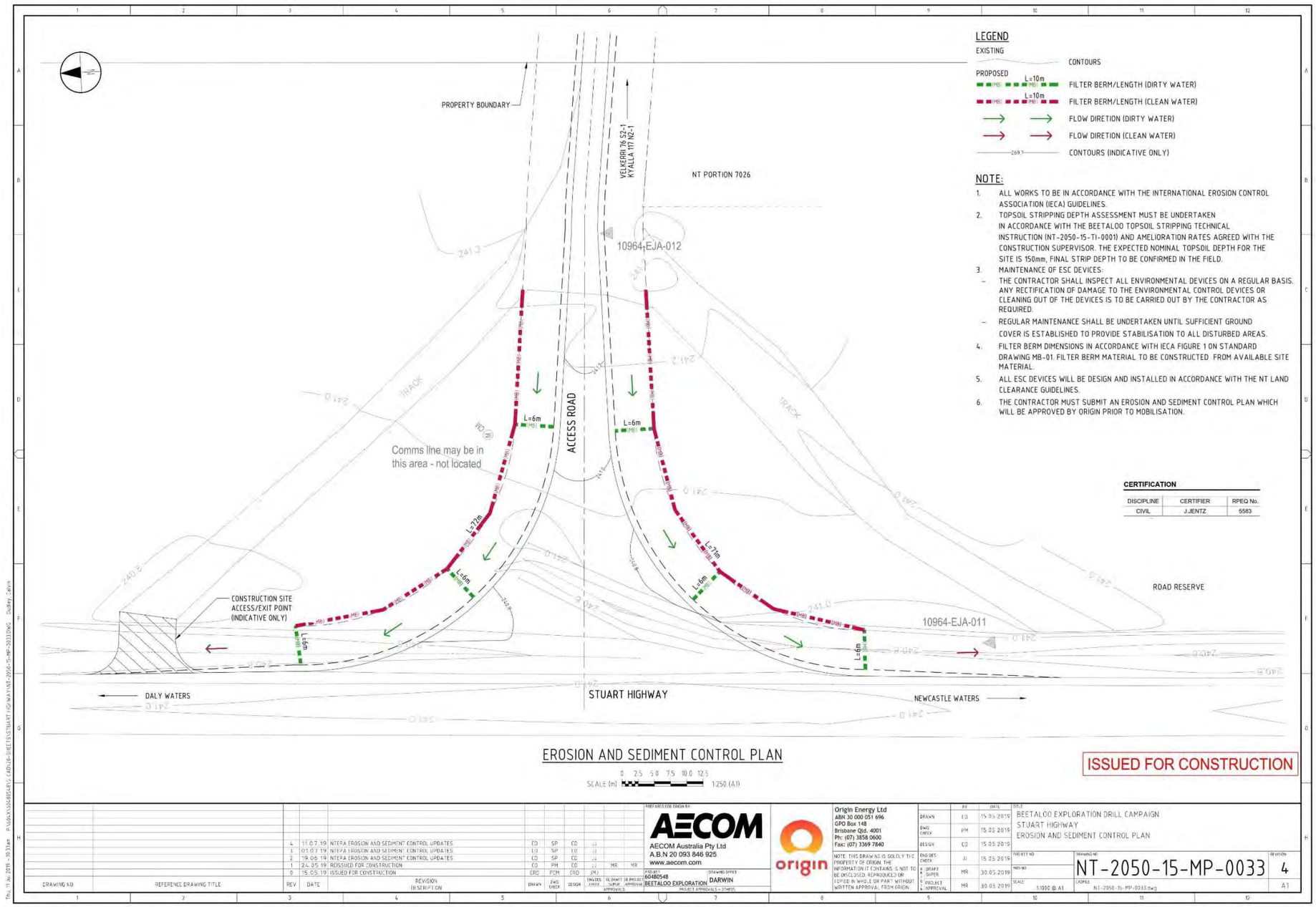
Appendix I Erosion and Sediment Control Plan for Beetaloo W



Filename: P:\605x\60623736\900_CAD_GIS\920_GIS\02_MXDs\Beetaloo W-1 Kalala S-1\Beetaloo W-1 ESCP.mxd

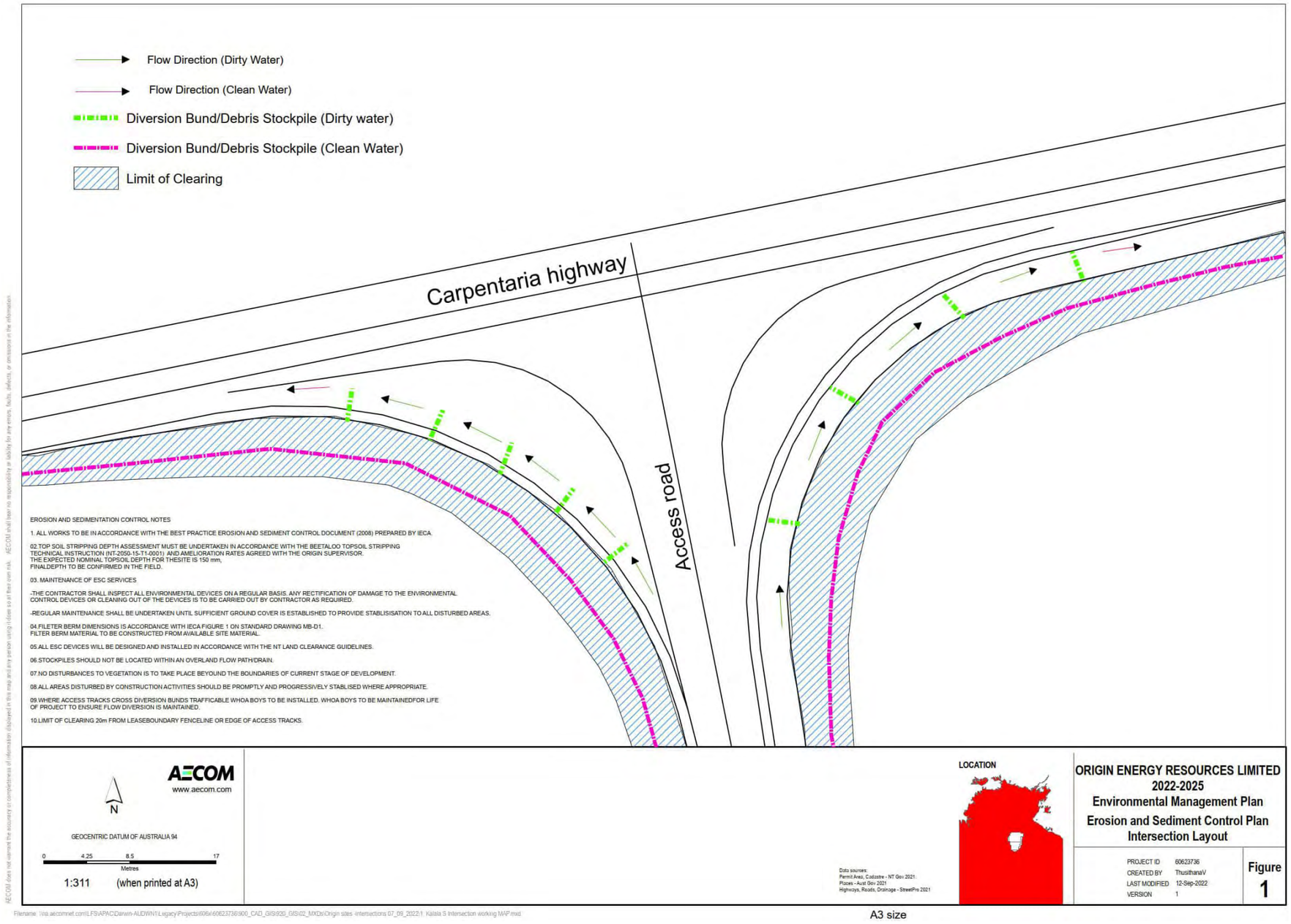
Review due: 13/09/2025

Appendix J Erosion and Sediment Control Plan for Stuart Highway Intersection



Review due: 13/09/2025

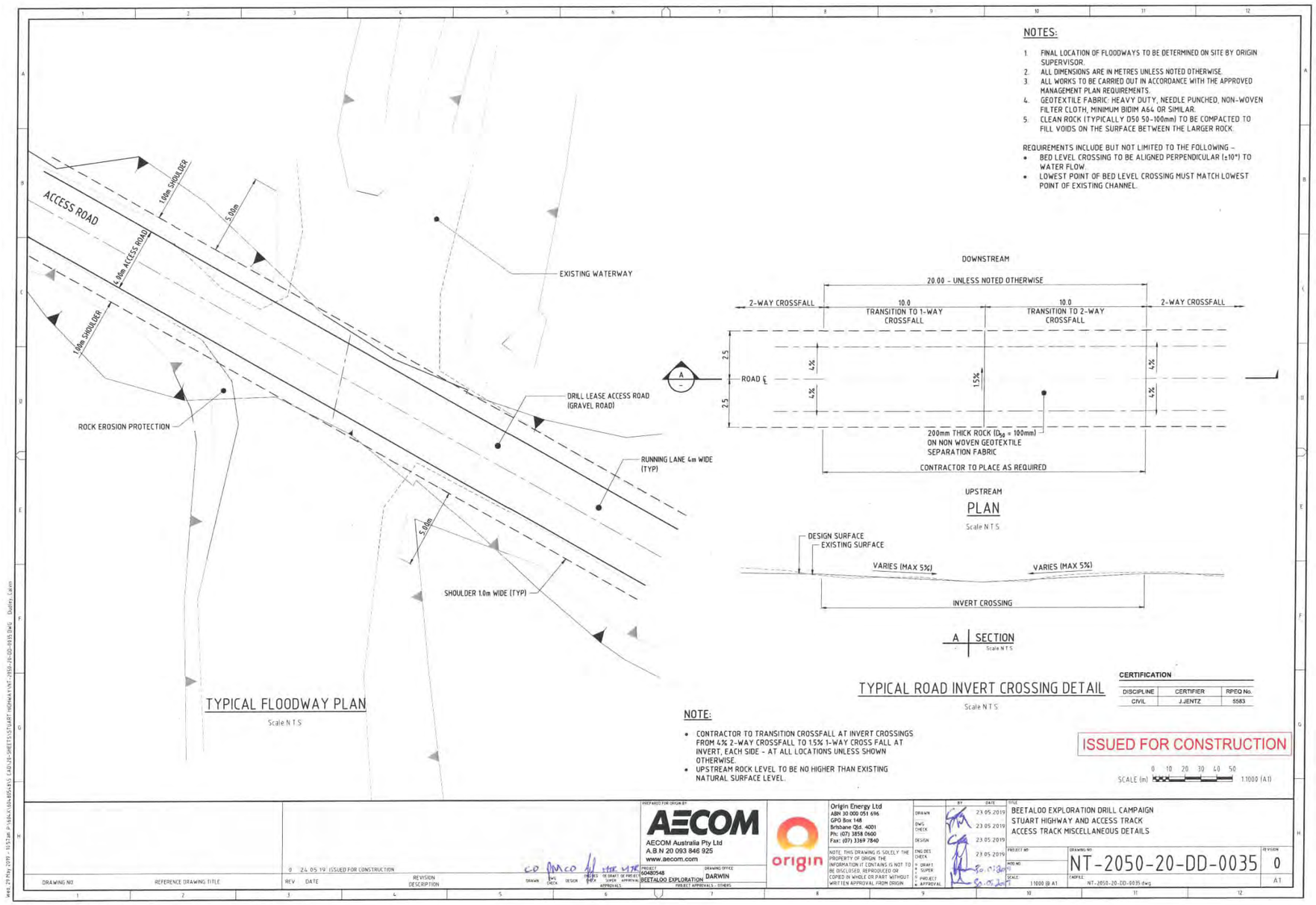
Appendix K Erosion and Sediment Control Plan for Carpentaria Highway Intersection



Filename: \\na.aecomnet.com\LFS\APAC\Darwin-AUDWN1\Legacy\Projects\606x\60623736\900_CAD_GIS\920_GIS\02_MXDs\Origin sites -Intersections 07_09_2022\1 Kalala S Intersection working MAP.mxd

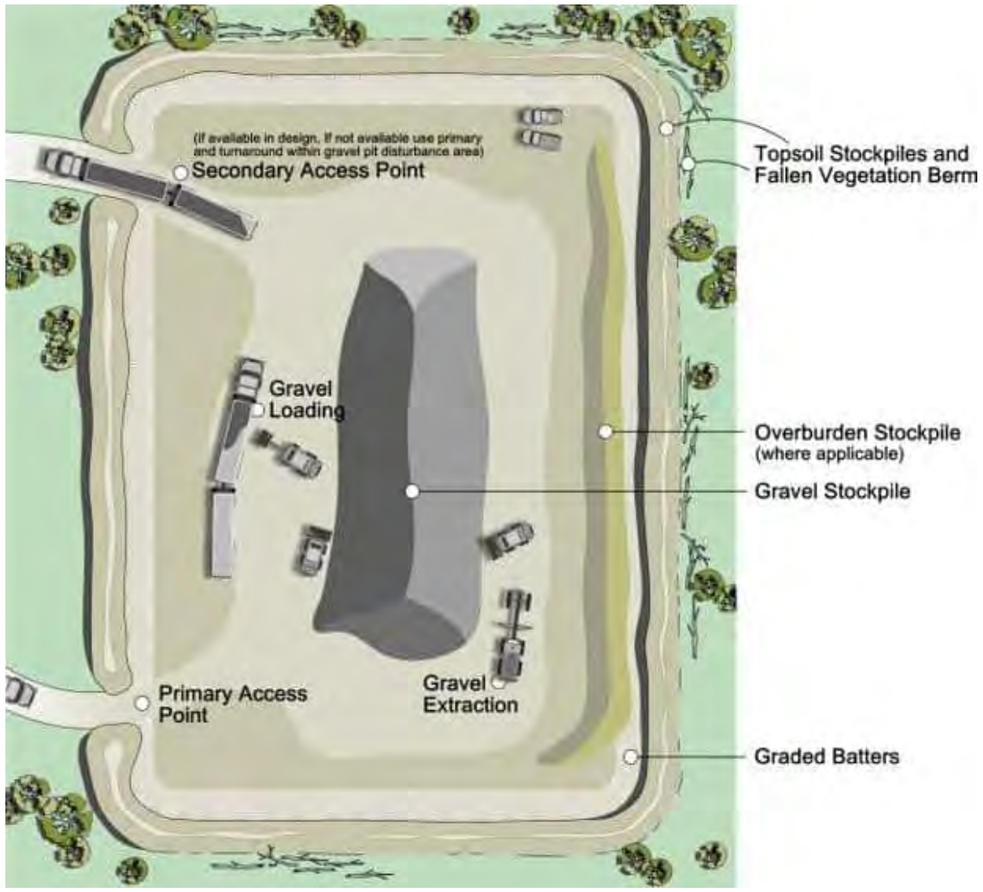
Review due: 13/09/2025

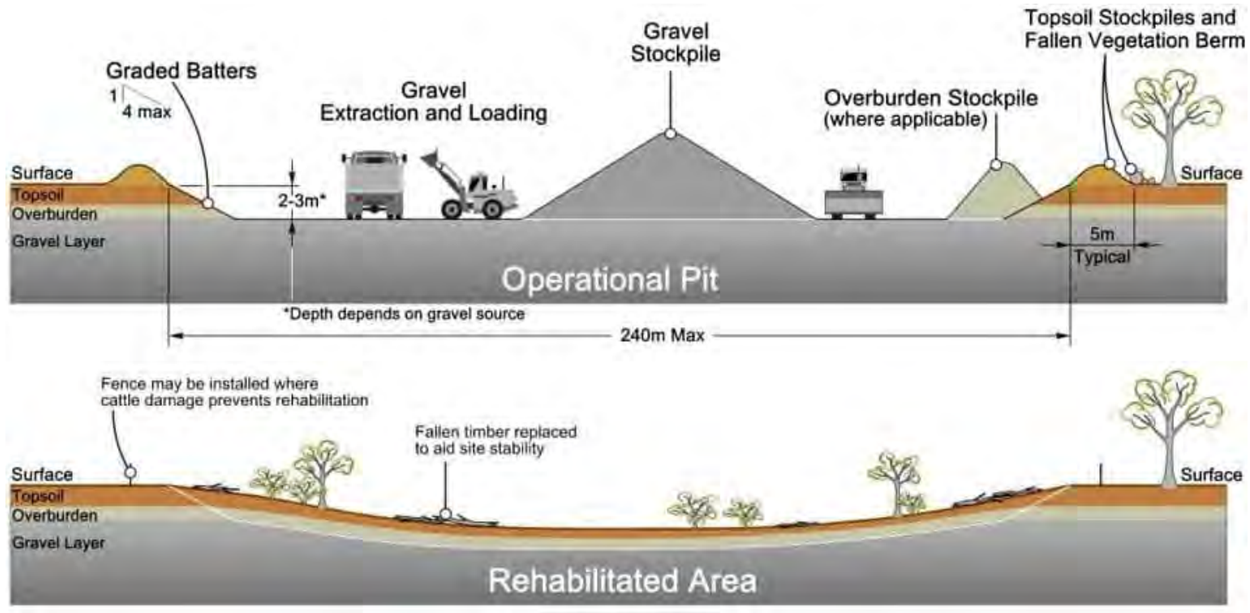
Appendix L Erosion and Sediment Control Plan for Typical Road Invert Crossing



Review due: 13/09/2025

Appendix M Erosion and Sediment Control Schematic for Typical Gravel Pit





Appendix N Other IECA Standard Specifications (as required)

MATERIALS

(i) MULCH MUST COMPLY WITH THE REQUIREMENTS OF AS4454.

(ii) MAXIMUM SOLUBLE SALT CONCENTRATION OF 5dS/m.

(iii) MOISTURE CONTENT OF 30 TO 50% PRIOR TO APPLICATION.

INSTALLATION

1. REFER TO APPROVED PLANS FOR LOCATION AND EXTENT. IF THERE ARE QUESTIONS OR PROBLEMS WITH THE LOCATION, EXTENT, MATERIAL TYPE, OR METHOD OF INSTALLATION CONTACT THE ENGINEER OR RESPONSIBLE ON-SITE OFFICER FOR ASSISTANCE.

2. WHEN SELECTING THE LOCATION OF A MULCH FILTER BERM, TO THE MAXIMUM DEGREE PRACTICAL, ENSURE THE BERM IS LOCATED:

(i) TOTALLY WITHIN THE PROPERTY BOUNDARIES;

(ii) ALONG A LINE OF CONSTANT ELEVATION (PREFERRED, BUT NOT ALWAYS PRACTICAL);

(iii) AT LEAST 1m, IDEALLY 3m, FROM THE TOE OF A FILL EMBANKMENT;

(iv) AWAY FROM AREAS OF CONCENTRATED FLOW.

3. ENSURE THE BERM IS INSTALLED IN A MANNER THAT AVOIDS THE CONCENTRATION OF FLOW ALONG THE BERM, OR THE UNDESIRABLE DISCHARGE OF WATER AROUND THE END OF THE BERM.

4. ENSURE THE BERM HAS BEEN PLACED SUCH THAT PONDING UP-SLOPE OF THE BERM IS MAXIMISED.

5. ENSURE BOTH ENDS OF THE BERM ARE ADEQUATELY TURNED UP THE SLOPE TO PREVENT FLOW BYPASSING PRIOR TO WATER PASSING OVER THE BERM.

6. ENSURE 100% CONTACT WITH THE SOIL SURFACE.

7. WHERE SPECIFIED, TAKE APPROPRIATE STEPS TO VEGETATE THE BERM.

MAINTENANCE

1. DURING THE CONSTRUCTION PERIOD, INSPECT ALL BERMS AT LEAST WEEKLY AND AFTER ANY SIGNIFICANT RAIN. MAKE NECESSARY REPAIRS IMMEDIATELY.

2. REPAIR OR REPLACE ANY DAMAGED SECTIONS.

3. WHEN MAKING REPAIRS, ALWAYS RESTORE THE SYSTEM TO ITS ORIGINAL CONFIGURATION UNLESS AN AMENDED LAYOUT IS REQUIRED OR SPECIFIED.

4. REMOVE ACCUMULATED SEDIMENT IF THE SEDIMENT DEPOSIT EXCEEDS A DEPTH OF 100mm OR 1/3 THE HEIGHT OF THE BERM.

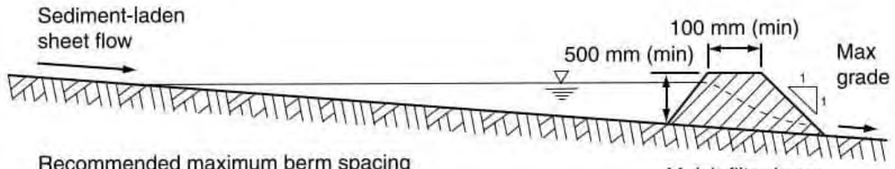
5. DISPOSE OF SEDIMENT IN A SUITABLE MANNER THAT WILL NOT CAUSE AN EROSION OR POLLUTION HAZARD.

REMOVAL (IF REQUIRED)

1. WHEN DISTURBED AREAS UP-SLOPE OF THE BERM ARE SUFFICIENTLY STABILISED TO RESTRAIN EROSION, THE BERM MAYBE REMOVED.

2. REMOVE ANY COLLECTED SEDIMENT AND DISPOSE OF IN A SUITABLE MANNER THAT WILL NOT CAUSE AN EROSION OR POLLUTION HAZARD.

3. REHABILITATE/REVEGETATE THE DISTURBED GROUND AS NECESSARY TO MINIMISE THE EROSION HAZARD.



Recommended maximum berm spacing

Land slope	Max spacing
< 2%	30 m
5%	25 m
10%	15 m
20%	8 m

Figure 1 - Typical placement of mulch filter berm

Drawn:	Date:		
GMW	Apr-10	Mulch Filter Berms	MB-01

Catchments & Creeks Pty Ltd

MATERIAL

ROCK MULCH: 25–75mm DURABLE, WEATHER RESISTANT AND EVENLY GRADED WITH 50% BY WEIGHT LARGER THAN THE SPECIFIED NOMINAL ROCK SIZE (IF SPECIFIED).

INSTALLATION

1. REFER TO APPROVED PLANS FOR LOCATION, EXTENT, AND APPLICATION DETAILS. IF THERE ARE QUESTIONS OR PROBLEMS WITH THE LOCATION, EXTENT, OR METHOD OF APPLICATION CONTACT THE ENGINEER OR RESPONSIBLE ON-SITE OFFICER FOR ASSISTANCE.

2. SPREAD ENOUGH ROCK TO COMPLETELY COVER THE SURFACE OF THE SOIL AT THE DENSITY OR THICKNESS SPECIFIED IN THE APPROVED PLANS. IF THE APPLICATION DENSITY IS NOT SUPPLIED, THEN APPLY AT A THICKNESS OF AT LEAST 50mm OR TWICE THE NOMINAL ROCK SIZE (WHICHEVER IS GREATER).

3. IF THE EXPOSED SOILS ARE DISPERSIVE, THEN ENSURE THESE SOILS ARE COVERED WITH A LAYER OF NON-DISPERSIVE SOIL (MINIMUM 200mm) BEFORE PLACEMENT OF ROCK.

4. MAKE ALL NECESSARY ADJUSTMENTS TO ENSURE ANY SURFACE FLOW IS ALLOWED TO PASS FREELY ACROSS THE TREATED AREA FOLLOWING ITS NATURAL DRAINAGE PATH.

MAINTENANCE

1. INSPECT ALL TREATED SURFACES FORTNIGHTLY AND AFTER RUNOFF-PRODUCING RAINFALL.

2. CHECK FOR RILL EROSION, OR DISLODGMENT OF THE ROCKS.

3. REPLACE ANY DISPLACED ROCKS TO MAINTAIN THE REQUIRED COVERAGE.

4. IF WASH-OUTS OCCUR, REPAIR THE SLOPE AND REINSTALL ROCK COVER.

5. IF THE ROCK MULCHING IS NOT EFFECTIVE IN CONTAINING THE SOIL EROSION IT SHOULD BE REPLACED, OR AN ALTERNATIVE EROSION CONTROL PROCEDURE ADOPTED.

Drawn:	Date:		
GMW	Dec-09	Rock Mulching	MR-01

PREPARATION

1. REFER TO APPROVED PLANS FOR LOCATION, EXTENT, AND DIMENSIONAL DETAILS. IF THERE ARE QUESTIONS OR PROBLEMS WITH THE LOCATION, OR EXTENT, CONTACT THE ENGINEER OR RESPONSIBLE ON-SITE OFFICER FOR ASSISTANCE.

2. TAKE ALL NECESSARY STEPS TO ENSURE DISTURBANCE TO THE BUFFER ZONE IS MINIMISED THROUGHOUT THE TIME IT IS USED AS A SEDIMENT TRAP.

3. TO THE MAXIMUM DEGREE PRACTICABLE, ENSURE FLOW PASSING THROUGH THE BUFFER ZONE IS NOT ALLOWED TO CONCENTRATE WITHIN DRAINAGE DEPRESSIONS, SWALES, RILLS OR WHEEL TRACKS.

4. WHERE NECESSARY, INSTALL APPROPRIATE DRAINAGE CONTROLS UP-SLOPE OF THE BUFFER ZONE TO DISTRIBUTE THE INFLOW ALONG THE FULLY LENGTH OF THE BUFFER ZONE AS 'SHEET FLOW'.

5. WHERE NECESSARY, INSTALL A COARSE SEDIMENT TRAP, SUCH AS A SEDIMENT FENCE, UP-SLOPE OF THE BUFFER ZONE TO REDUCE THE QUANTITY OF SEDIMENT PASSING ONTO THE GRASS. GENERALLY THIS IS REQUIRED IF LARGE QUANTITIES OF COARSE SEDIMENT ARE EXPECTED.

6. IF REQUIRED, INSTALL A LIGHT BARRIER FENCE TO CLEARLY IDENTIFY THE BUFFER ZONE AND HELP EXCLUDE CONSTRUCTION TRAFFIC.

MAINTENANCE

1. INSPECT THE BUFFER ZONE ON A REGULAR BASIS AND AFTER RUNOFF-PRODUCING RAINFALL.

2. ENSURE THAT THERE IS NO SOIL EROSION AND THAT SEDIMENT DEPOSITION IS NOT CAUSING THE CONCENTRATION OF FLOW THROUGH THE BUFFER ZONE, OR FLOW BYPASSING.

3. IF THE BUFFER ZONE HAS BEEN DISTURBED, TAKE NECESSARY STEPS TO RE-ESTABLISH SUITABLE SHEET FLOW CONDITIONS.

4. REMOVE EXCESSIVE ACCUMULATIONS OF SEDIMENT THAT MAY CAUSE THE CONCENTRATION OF FLOW. EXCESSIVE SEDIMENT SHOULD BE REMOVED AFTER EACH RUNOFF-PRODUCING RAINFALL EVENT, OR WHERE APPROPRIATE, EVENLY RAKED INTO THE SOIL. SEDIMENT SHOULD BE REMOVED IN A MANNER THAT AVOIDS DAMAGE TO THE BUFFER ZONE OR THE CREATION OF WHEEL TRACKS DOWN THE SLOPE.

5. EXCESSIVE SEDIMENT MAY BE DEFINED AS:

(i) ANY SEDIMENT THAT COVERS A PORTION OF THE GRASSED SURFACE; OR

(ii) SEDIMENT DEPOSITION SUCH THAT THE GRASS STRAND HEIGHT ABOVE THE SEDIMENT IS LESS THAN 50mm; OR

(iii) A DEPOSITION OF SEDIMENT IN EXCESS OF 750g/m^2 (APPROXIMATELY THE EQUIVALENT OF THREE 70mm DIAMETER BALLS OF DRY SOIL).

6. THE SOURCE OF ANY EXCESSIVE SEDIMENT SHOULD BE INVESTIGATED AND CONTROLLED WHERE PRACTICAL.

7. TAKE APPROPRIATE STEPS TO MAINTAIN AT LEAST 75% GRASS COVER OVER THE BUFFER ZONE.

8. WHERE PRACTICAL, MAINTAIN ANY GROUNDCOVER VEGETATION AT A HEIGHT GREATER THAN THE EXPECTED DEPTH OF WATER FLOW AND AT LEAST 50mm.

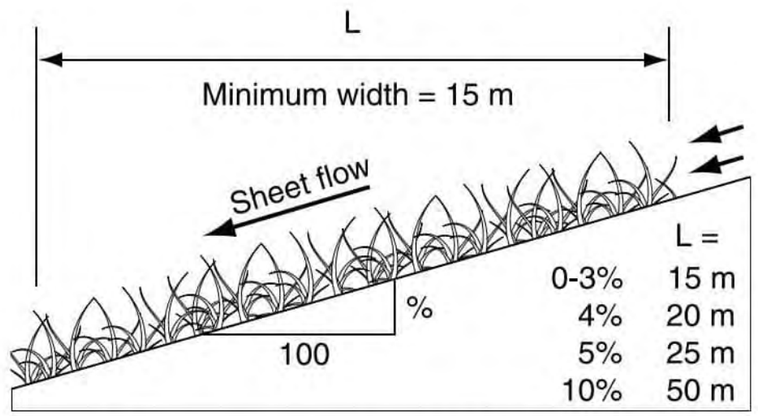
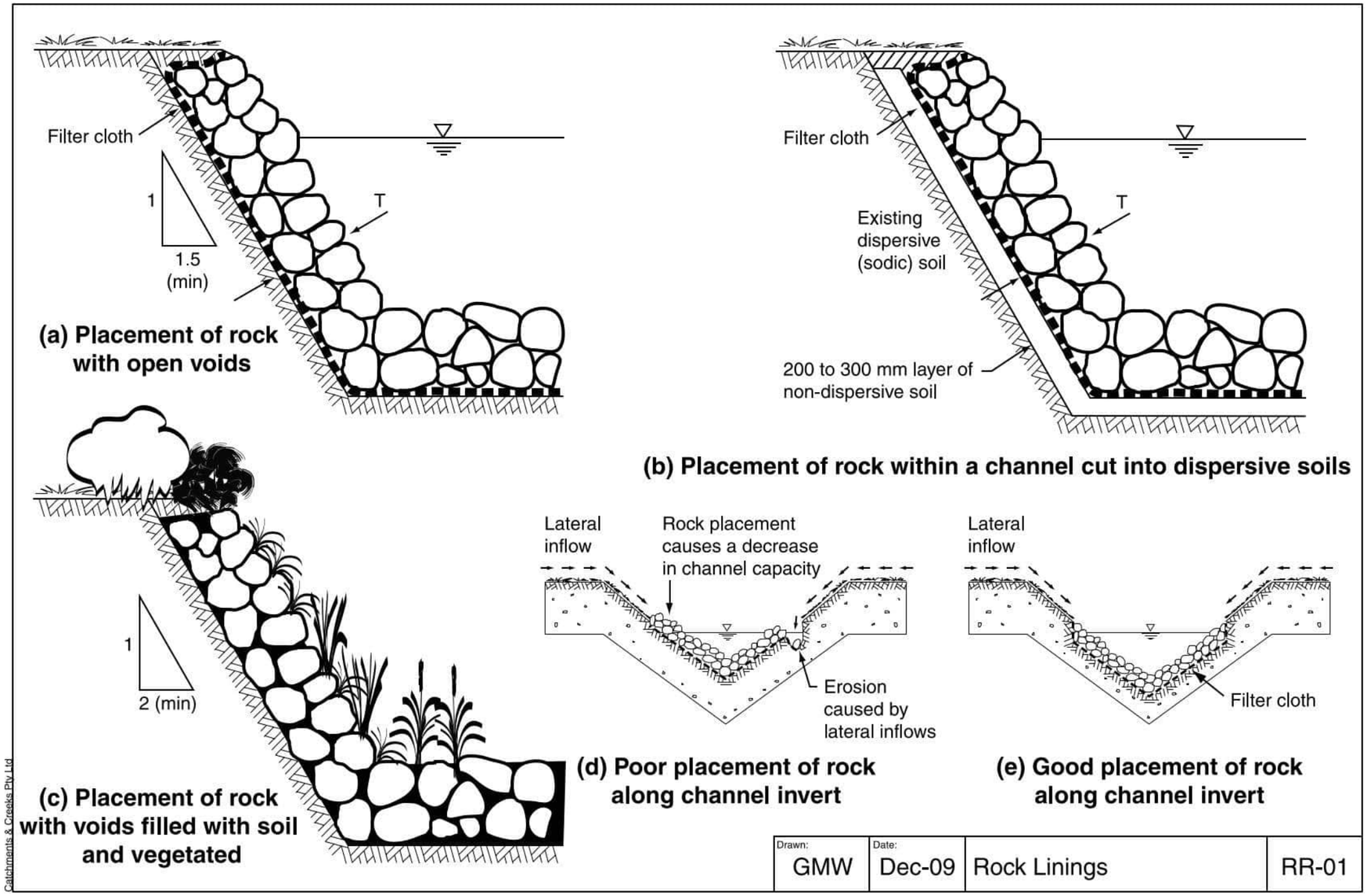


Figure 1 - Minimum dimensional requirements of a grassed buffer zone

Drawn:	Date:		
GMW	Apr-10	Buffer Zones (grassed)	BZ-01



Review due: 13/09/2025

MATERIALS

ROCK: HARD, ANGULAR, DURABLE, WEATHER RESISTANT AND EVENLY GRADED WITH 50% BY WEIGHT LARGER THAN THE SPECIFIED NOMINAL ROCK SIZE AND SUFFICIENT SMALL ROCK TO FILL THE VOIDS BETWEEN THE LARGER ROCK. THE DIAMETER OF THE LARGEST ROCK SIZE SHOULD BE NO LARGER THAN 1.5 TIMES THE NOMINAL ROCK SIZE. SPECIFIC GRAVITY TO BE AT LEAST 2.5.

GEOTEXTILE FABRIC: HEAVY-DUTY, NEEDLE-PUNCHED, NON-WOVEN FILTER CLOTH, MINIMUM BIDIM A24 OR EQUIVALENT.

INSTALLATION

1. REFER TO APPROVED PLANS FOR LOCATION, EXTENT AND INSTALLATION DETAILS. IF THERE ARE QUESTIONS OR PROBLEMS WITH THE LOCATION, EXTENT, OR METHOD OF INSTALLATION CONTACT THE ENGINEER OR RESPONSIBLE ON-SITE OFFICER FOR ASSISTANCE.

2. CLEAR THE PROPOSED CHANNEL AREA OF TREES, STUMPS, ROOTS, LOOSE ROCK, AND OTHER OBJECTIONABLE MATERIALS.

3. EXCAVATE THE CHANNEL TO THE LINES AND GRADES AS SHOWN ON THE PLANS. OVER-CUT THE CHANNEL TO A DEPTH EQUAL TO THE SPECIFIED DEPTH OF ROCK PLACEMENT SUCH THAT THE FINISHED ROCK SURFACE WILL BE AT THE ELEVATION OF THE SURROUNDING LAND.

4. ROCK MUST BE PLACED WITHIN THE CHANNEL AS SPECIFIED WITHIN THE APPROVED PLANS, INCLUDING THE PLACEMENT OF ANY SPECIFIED FILTER LAYER.

5. IF DETAILS ARE NOT PROVIDED ON THE ROCK PLACEMENT, THEN THE PRIMARY ARMOUR ROCK MUST BE EITHER PLACED ON:

(i) A FILTER BED FORMED FROM A LAYER OF SPECIFIED SMALLER ROCK (ROCK FILTER LAYER);

(ii) AN EARTH BED LINED WITH FILTER CLOTH;

(iii) AN EARTH BED NOT LINED IN FILTER CLOTH, BUT ONLY IF ALL VOIDS BETWEEN THE ARMOUR ROCK ARE TO BE FILLED WITH SOIL AND POCKET PLANTED IMMEDIATELY AFTER PLACEMENT OF THE ROCK.

6. IF A ROCK/AGGREGATE FILTER LAYER IS SPECIFIED, THEN PLACE THE FILTER LAYER IMMEDIATELY AFTER THE FOUNDATIONS ARE PREPARED. SPREAD THE FILTER ROCK IN A UNIFORM LAYER TO THE SPECIFIED DEPTH BUT A MINIMUM OF 150mm. WHERE MORE THAN ONE LAYER OF FILTER MATERIAL HAS BEEN SPECIFIED, SPREAD EACH LAYER SUCH THAT MINIMAL MIXING OCCURS BETWEEN EACH LAYER OF ROCK.

7. IF A GEOTEXTILE (FILTER CLOTH) UNDERLAY IS SPECIFIED, PLACE THE FABRIC DIRECTLY ON THE PREPARED FOUNDATION. IF MORE THAN ONE SHEET OF FABRIC IS REQUIRED TO OVER THE AREA, OVERLAP THE EDGE OF EACH SHEET AT LEAST 300mm AND PLACE ANCHOR PINS AT MINIMUM 1m SPACING ALONG THE OVERLAP.

8. ENSURE THE GEOTEXTILE FABRIC IS PROTECTED FROM PUNCHING OR TEARING DURING INSTALLATION OF THE FABRIC AND THE ROCK. REPAIR ANY DAMAGE BY REMOVING THE ROCK AND PLACING WITH ANOTHER PIECE OF FILTER CLOTH OVER THE DAMAGED AREA OVERLAPPING THE EXISTING FABRIC A MINIMUM OF 300mm.

9. WHERE NECESSARY, A MINIMUM 100mm LAYER OF FINE GRAVEL, AGGREGATE OR SAND SHOULD BE PLACED OVER THE FABRIC TO PROTECT IT FROM DAMAGE.

10. PLACEMENT OF ROCK SHOULD FOLLOW IMMEDIATELY AFTER PLACEMENT OF THE FILTER LAYER. PLACE ROCK SO THAT IT FORMS A DENSE, WELL-GRADED MASS OF ROCK WITH A MINIMUM OF VOIDS.

11. PLACE ROCK TO ITS FULL THICKNESS IN ONE OPERATION. DO NOT PLACE ROCK BY DUMPING THROUGH CHUTES OR OTHER METHODS THAT CAUSE SEGREGATION OF ROCK SIZES.

12. THE FINISHED SURFACE SHOULD BE FREE OF POCKETS OF SMALL ROCK OR CLUSTERS OF LARGE ROCKS. HAND PLACING MAY BE NECESSARY TO ACHIEVE THE PROPER DISTRIBUTION OF ROCK SIZES TO PRODUCE A RELATIVELY SMOOTH, UNIFORM SURFACE. THE FINISHED GRADE OF THE ROCK SHOULD BLEND WITH THE SURROUNDING AREA. NO OVERFALL OR PROTRUSION OF ROCK SHOULD BE APPARENT.

13. IMMEDIATELY UPON COMPLETION OF THE CHANNEL, VEGETATE ALL DISTURBED AREAS OR OTHERWISE PROTECT THEM AGAINST SOIL EROSION.

14. WHERE SPECIFIED, FILL ALL VOIDS WITH SOIL AND VEGETATE THE ROCK SURFACE IN ACCORDANCE WITH THE APPROVED PLAN.

MAINTENANCE

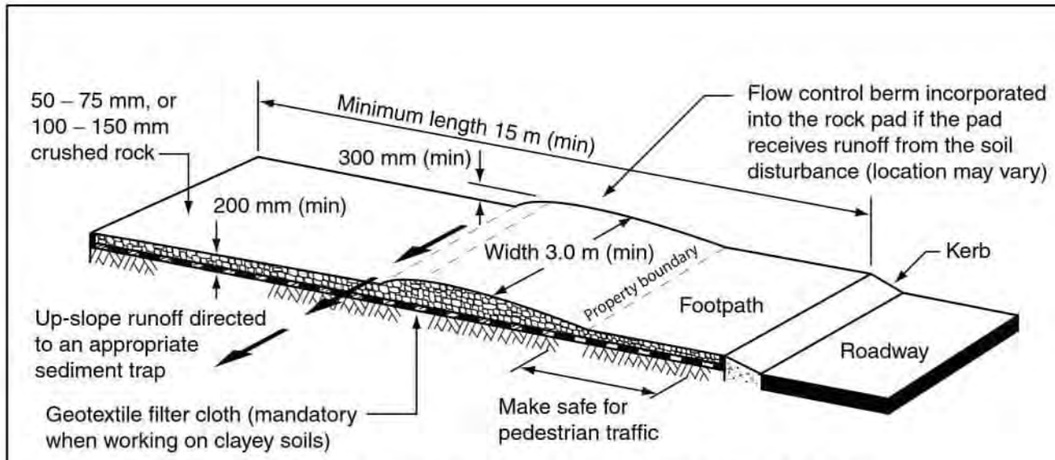
1. ROCK-LINED CHANNELS SHOULD BE INSPECTED PERIODICALLY AND AFTER SIGNIFICANT STORM EVENTS. CHECK FOR SCOUR OR DISLODGED ROCK. REPAIR DAMAGED AREAS IMMEDIATELY.

2. CLOSELY INSPECT THE OUTER EDGES OF THE ROCK PROTECTION. ENSURE WATER ENTRY INTO THE CHANNEL OR CHUTE IS NOT CAUSING EROSION ALONG THE EDGE OF THE ROCK PROTECTION.

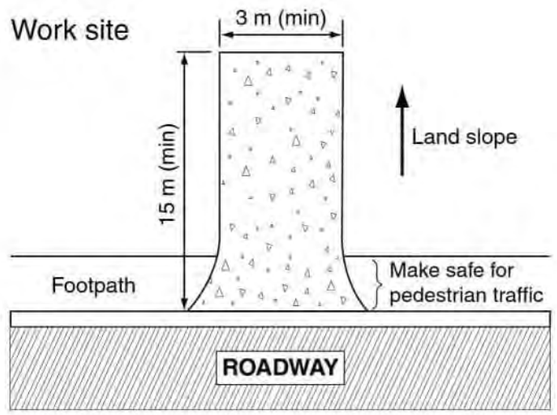
3. CAREFULLY CHECK THE STABILITY OF THE ROCK LOOKING FOR INDICATIONS OF PIPING, SCOUR HOLES, OR BANK FAILURES.

4. REPLACE ANY DISPLACED ROCK WITH ROCK OF A SIGNIFICANTLY (MINIMUM 110%) LARGER SIZE THAN THE DISPLACED ROCK.

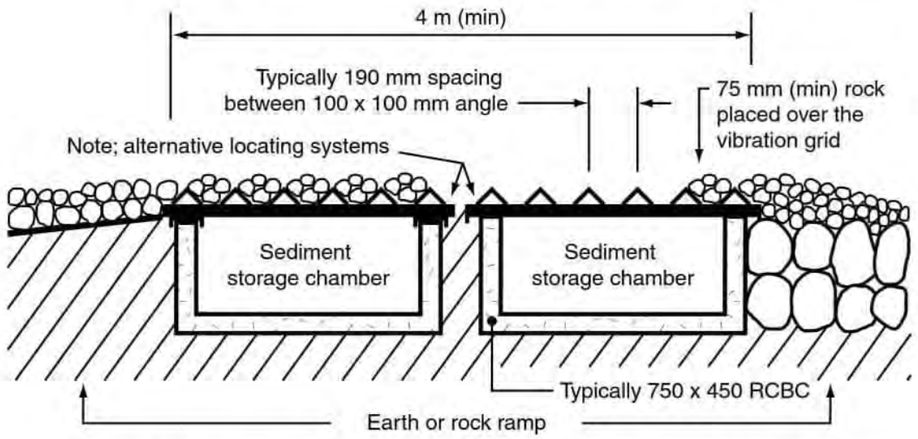
Drawn:	Date:		
GMW	May-10	Rock Linings	RR-02



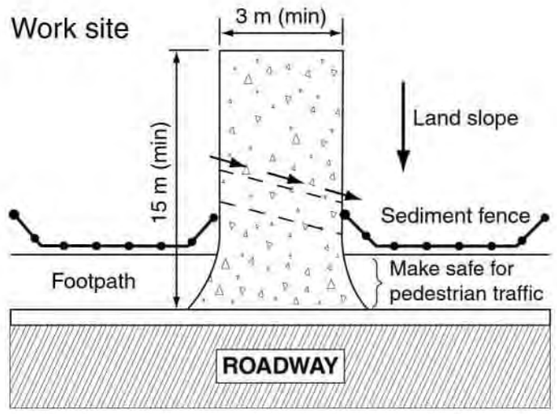
(a) Rock entry/exit pad for construction sites (refer to Standard Drawing Exit-03 for building sites)



(b) Rock pad sloping away from road



(c) Alternative low maintenance arrangement (still under development)



(d) Rock pad sloping towards the road

Drawn:	Date:		
GMW	Apr-10	Construction Exit - Rock Pad (construction sites only)	Exit-01

MATERIALS

ROCK: WELL GRADED, HARD, ANGULAR, EROSION RESISTANT ROCK, NOMINAL DIAMETER OF 50 TO 75mm (SMALL DISTURBANCES) OR 100 TO 150mm (LARGE DISTURBANCES). ALL REASONABLE MEASURES MUST BE TAKEN TO OBTAIN ROCK OF NEAR UNIFORM SIZE.

FOOTPATH STABILISING AGGREGATE: 25 TO 50mm GRAVEL OR AGGREGATE.

GEOTEXTILE FABRIC: HEAVY-DUTY, NEEDLE-PUNCHED, NON-WOVEN FILTER CLOTH ('BIDIM' A24 OR EQUIVALENT).

INSTALLATION

1. REFER TO APPROVED PLANS FOR LOCATION AND DIMENSIONAL DETAILS. IF THERE ARE QUESTIONS OR PROBLEMS WITH THE LOCATION, DIMENSIONS, OR METHOD OF INSTALLATION, CONTACT THE ENGINEER OR RESPONSIBLE ON-SITE OFFICER FOR ASSISTANCE.

2. CLEAR THE LOCATION OF THE ROCK PAD, REMOVING STUMPS, ROOTS AND OTHER VEGETATION TO PROVIDE A FIRM FOUNDATION SO THAT THE ROCK IS NOT PRESSED INTO SOFT GROUND. CLEAR SUFFICIENT WIDTH TO ALLOW PASSAGE OF LARGE VEHICLES, BUT CLEAR ONLY THAT NECESSARY FOR THE EXIT. DO NOT CLEAR ADJACENT AREAS UNTIL THE REQUIRED EROSION AND SEDIMENT CONTROL DEVICES ARE IN PLACE.

3. IF THE EXPOSED SOIL IS SOFT, PLASTIC OR CLAYEY, PLACE A SUB-BASE OF CRUSHED ROCK OR A LAYER OF HEAVY-DUTY FILTER CLOTH TO PROVIDE A FIRM FOUNDATION.

4. PLACE THE ROCK PAD FORMING A MINIMUM 200mm THICK LAYER OF CLEAN, OPEN-VOID ROCK.

5. IF THE ASSOCIATED CONSTRUCTION SITE IS UP-SLOPE OF THE ROCK PAD, THUS CAUSING STORMWATER RUNOFF TO FLOW TOWARDS THE ROCK PAD, THEN FORM A MINIMUM 300mm HIGH FLOW CONTROL BERM ACROSS THE ROCK PAD TO DIVERT SUCH RUNOFF TO A SUITABLE SEDIMENT TRAP.

6. THE LENGTH OF THE ROCK PAD SHOULD BE AT LEAST 15m WHERE PRACTICABLE, AND AS WIDE AS THE FULL WIDTH OF THE ENTRY OR EXIT AND AT LEAST 3m. THE ROCK PAD SHOULD COMMENCE AT THE EDGE OF THE OFF-SITE SEALED ROAD OR PAVEMENT.

7. FLARE THE END OF THE ROCK PAD WHERE IT MEETS THE PAVEMENT SO THAT THE WHEELS OF TURNING VEHICLES DO NOT TRAVEL OVER UNPROTECTED SOIL.

8. IF THE FOOTPATH IS OPEN TO PEDESTRIAN MOVEMENT, THEN COVER THE COARSE ROCK WITH FINE AGGREGATE OR GRAVEL, OR OTHERWISE TAKE WHATEVER MEASURES ARE NEEDED TO MAKE THE AREA SAFE.

MAINTENANCE

1. INSPECT ALL SITE ENTRY AND EXIT POINTS PRIOR TO FORECAST RAIN, DAILY DURING EXTENDED PERIODS OF RAINFALL, AFTER RUNOFF-PRODUCING RAINFALL, OR OTHERWISE AT FORTNIGHTLY INTERVALS.

2. IF SAND, SOIL, SEDIMENT OR MUD IS TRACKED OR WASHED ONTO THE ADJACENT SEALED ROADWAY, THEN SUCH MATERIAL MUST BE PHYSICALLY REMOVED, FIRST USING A SQUARE-EDGED SHOVEL, AND THEN A STIFF-BRISTLED BROOM, AND THEN BY A MECHANICAL VACUUM UNIT, IF AVAILABLE.

3. IF NECESSARY FOR SAFETY REASONS, THE ROADWAY SHALL ONLY BE WASHED CLEAN AFTER ALL REASONABLE EFFORTS HAVE BEEN TAKEN TO SHOVEL AND SWEEP THE MATERIAL FROM THE ROADWAY.

4. WHEN THE VOIDS BETWEEN THE ROCK BECOMES FILLED WITH MATERIAL AND THE EFFECTIVENESS OF THE ROCK PAD IS REDUCED TO A POINT WHERE SEDIMENT IS BEING TRACKED OFF THE SITE, A NEW 100mm LAYER OF ROCK MUST BE ADDED AND/OR THE ROCK PAD MUST BE EXTENDED.

5. ENSURE ANY ASSOCIATED DRAINAGE CONTROL MEASURES (e.g. FLOW CONTROL BERM) ARE MAINTAINED IN ACCORDANCE WITH THEIR DESIRED OPERATIONAL CONDITIONS.

6. DISPOSE OF SEDIMENT AND DEBRIS IN A MANNER THAT WILL NOT CREATE AN EROSION OR POLLUTION HAZARD.

REMOVAL

1. THE ROCK PAD SHOULD BE REMOVED ONLY AFTER IT IS NO LONGER NEEDED AS A SEDIMENT TRAP.

2. REMOVE MATERIALS AND COLLECTED SEDIMENT AND DISPOSE OF IN A SUITABLE MANNER THAT WILL NOT CAUSE AN EROSION OR POLLUTION HAZARD.
3. RE-GRADE AND STABILISE THE DISTURBED GROUND AS NECESSARY TO MINIMISE THE EROSION HAZARD.

Catchments & Creeks Pty Ltd

Drawn:	Date:		
GMW	Apr-10	Construction Exit - Rock Pad (construction sites only)	Exit-02

APPLICATION

1. REFER TO APPROVED PLANS FOR LOCATION, EXTENT, AND APPLICATION DETAILS. IF THERE ARE QUESTIONS OR PROBLEMS WITH THE LOCATION, EXTENT, OR METHOD OF APPLICATION CONTACT THE ENGINEER OR RESPONSIBLE ON-SITE OFFICER FOR ASSISTANCE.

2. FILL OR SUITABLY CONTOUR ANY EXISTING RUTTING, RILLING OR GULLIES.

3. SUITABLY DIVERT UP-SLOPE STORMWATER RUNOFF AROUND TREATED AREA AS DIRECTED WITHIN THE APPROVED PLANS, OR OTHERWISE AS DIRECTED BY THE SITE ENGINEER.

4. APPLY TREATMENT TO THE AREA TO THE DEPTH AND FREQUENCY (SPACING) SPECIFIED ON THE APPROVED PLANS, OR OTHERWISE AS DIRECTED BY THE SITE ENGINEER.

5. IMMEDIATELY SEED AND MULCH ROUGHENED AREAS TO OPTIMISE SEED GERMINATION AND GROWING CONDITIONS.

MAINTENANCE

1. DURING THE CONSTRUCTION PERIOD, INSPECT THE TREATED AREA PRIOR TO FORECAST RAINFALL, DAILY DURING EXTENDED PERIODS OF RAINFALL, AFTER SIGNIFICANT RUNOFF PRODUCING RAINFALL, OR OTHERWISE ON A WEEKLY BASIS.

2. FILL EROSION RILLS SLIGHTLY ABOVE THE ORIGINAL GRADE, OR REGRADE THE SLOPE AS DIRECTED TO REMOVE THE RILLS.

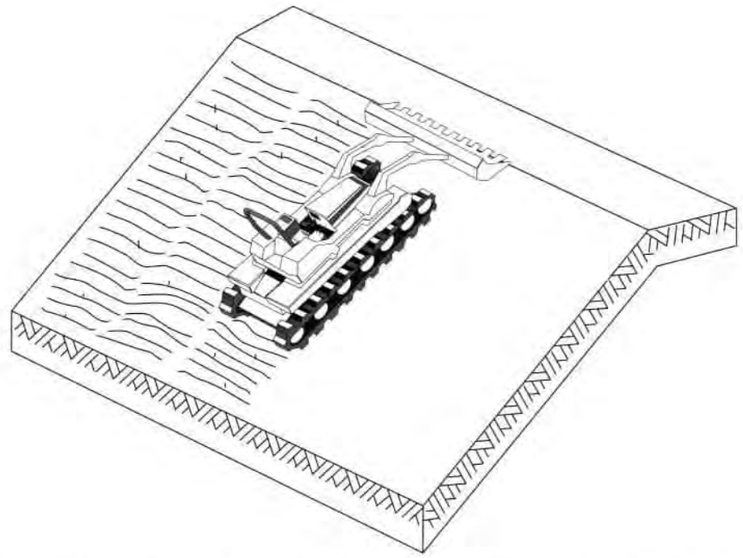


Figure 1 - Application of surface roughening on slope

Drawn:	Date:		
GMW	Dec-09	Surface Roughening	SR-01

Catchments & Creeks Pty Ltd

Appendix O Table 4.4.7 IECA Best Practice Land Clearing and Rehabilitation Requirements

Risk[1]	Best practice requirements
All cases	- All reasonable and practicable steps taken to apply best practice erosion control measures to completed earth works, or otherwise stabilise such works, prior to anticipated rainfall – including existing unstable, undisturbed, soil surfaces under the management or control of the building/construction works.
Very low	- Land clearing limited to 8 weeks of work if rainfall is reasonably possible. - Disturbed soil surfaces stabilised with minimum 60% cover[2] within 30 days of completion of works if rainfall is reasonably possible. - Unfinished earthworks are suitably stabilised if rainfall is reasonably possible, and disturbance is expected to be suspended for a period exceeding 30 days.
Low	- Land clearing limited to maximum 8 weeks of work. - Disturbed soil surfaces stabilised with minimum 70% cover[2] within 30 days of completion of works within any area of a work site. - Unfinished earthworks are suitably stabilised if rainfall is reasonably possible and disturbance is expected to be suspended for a period exceeding 30 days. - Appropriate protection of all planned garden beds is strongly recommended.
Moderate	- Land clearing limited to a maximum 6 weeks of work. - Disturbed soil surfaces stabilised with minimum 70% cover[2] within 20 days of completion of work within any area of a work site. - All planned garden beds protected with a minimum 75mm layer of organic *Mulching*, heavy *Erosion Control Blanket, Rock Mulching,* or the equivalent. - Staged construction and stabilisation of earth batters (steeper than 6H:1V) in maximum 3m vertical increments wherever reasonable and practicable. - The use of turf to form grassed surfaces given appropriate consideration. - Soil stockpiles and unfinished earthworks are suitably stabilised if disturbance is expected to be suspended for a period exceeding 10 days.
High	- Land clearing limited to a maximum 4 weeks of work. - Disturbed soil surface stabilised with minimum 75% cover[2] within 10 days of completion of works within any area of a work site. - All planned garden beds protected with a minimum 75mm layer of organic *Mulching*, heavy *Erosion Control Blanket, Rock Mulching,* or the equivalent. - Staged construction and stabilisation of earth batters (steeper than 6H:1V) in maximum 3m vertical increments wherever reasonable and practicable. - The use of turf to form grassed surfaces given appropriate consideration. - Soil stockpiles and unfinished earthworks are suitably stabilised if disturbance is expected to be suspended for a period exceeding 10 days.
Extreme	- Land clearing limited to maximum 2 weeks of work. - Disturbed soil surfaces stabilised with minimum 80% cover[2] within 5 days of completion of works within any area of a work site. - All planned garden beds protected with a minimum 75mm layer of organic *Mulching*, heavy *Erosion Control Blanket, Rock Mulching,* or the equivalent. - Staged construction and stabilisation of earth batters (steeper than 6H:1V) in maximum 2m vertical increments wherever reasonable and practicable. - High priority given to the use of turf to form grassed surfaces. - Soil stockpiles and unfinished earthworks are suitably stabilised if disturbance is expected to be suspended for a period exceeding 5 days.

1. Erosion risk based on monthly erosivity (Table 4.4.1), average monthly rainfall depth (Table 4.4.2), or soil loss rate (Table 4.4.3) as directed by the regulatory authority.
2. Minimum cover requirements may be redirected if the natural cover of the immediate land is less than the nominated value, for example in arid and semi-arid areas or on coastal sand dunes.

Appendix P Erosion and Sediment Control Treatment – Seismic Lines

Blade up erosion controls

The figure below shows the condition of land following blade up traverse of survey area.

No treatment required.

Figure 1 Typical condition 'blade up' treatment

Surface bladed by grader (including woodland areas)

Erosion control treatments as follows:

- A diversion bank shall be installed along sections of the survey lines where material has been stripped from the surface (refer Table 12).
- The bank shall be constructed as a cut and push operation. Lines shall be ripped across the area at a grade of 0.3%. A shallow channel should be cut along this line (approximately 0.6 metres deep). Excavated material is dumped on the down slope side of the channel then compacted and smoothed out to form a bank with even batters and a level top (refer Figure 4).
- To aid trafficability, an approach and departure ramp shall be shaped during construction of the bank.
- The bank should direct runoff into undisturbed vegetation or into an existing drain (care needs to taken to ensure that erosion does not occur where the water runs down into the drain).
- Ensure the diversion bank is not eroded by traffic.
- Undertake maintenance as necessary.

Table 12 Bank Spacing Requirements (m)

Slope		Diversion bank spacing (m)
%	Gradient	
0.5	1:200	170-180
1	1:100	120-130
2	1:50	90-100
3	1:33	70-80
4	1:25	60-70
5	1:20	55-60
6	1:17	40-45

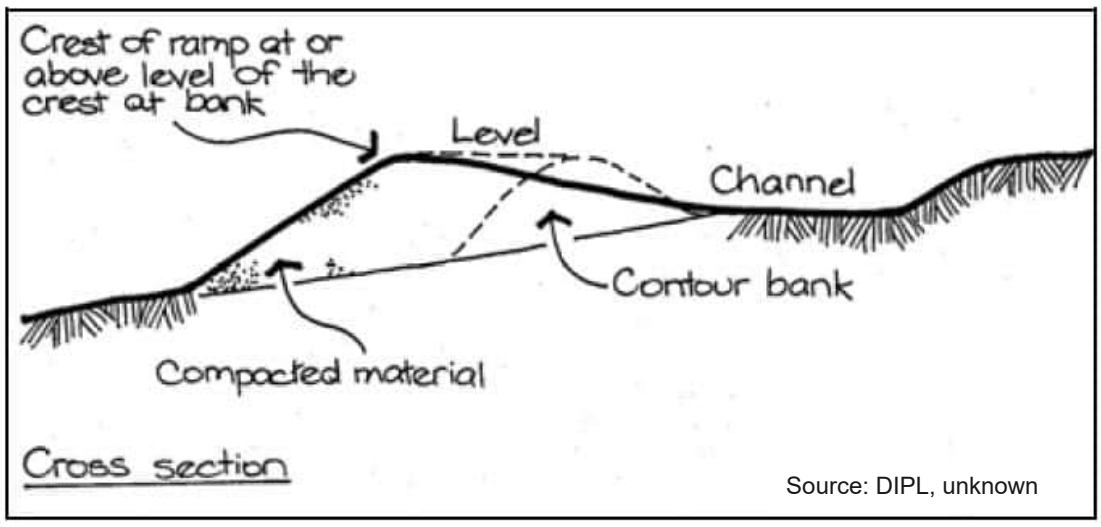


Figure 4 Whoa boys or roll over banks drawing

Woodland area erosion controls

The figure below shows the expected final rehabilitation treatment for woodland areas disturbed by the seismic survey activities. In the event of an expected significant rainfall event which will require the site to be abandoned, a similar treatment is to be adopted.

- Step 1. Respread windrowed topsoil of disturbed area and ripped into the soil surface.
- Step 2. Spread vegetation over top soiled area in an even layer.

Felled vegetation will be evenly spread over the top soiled area to provide additional protection against erosion.

Figure 2 Treatment for woodland areas

Typical Offlet Drain Detail for Access Tracks

- Construct access tracks with table drains that are free draining.
- Avoid road crowning to allow water to naturally cross the road.
- Form tracks to allow off-road drainage. Where track intercepts the direction of overland flow and re-directs this flow to a non-natural drainage line, install erosion control works to minimise potential erosion.
- The design and position of erosion control measures to be determined by experienced operator and site engineer, based on the site characteristics of the access track location.
- Where construction of table drains are deemed necessary, they should have a broad flat base at least 1 m wide and should not be graded to produce a 'V' shape. To minimise erosion, the slope should be no greater than 0.5% on erodible soils or 1% on stable soils.
- Where encounter dispersive / erosive soils they should be stabilised with gypsum or other stabiliser, as determined by laboratory analysis of soils.
- Where cut-out drains are required, they should be spaced based on the slope of the area i.e. 0.5% slope, allow for cut-out draining every 170-180 m or 1 % slope, allow for cut-out drainage every 120-130 m etc. (refer to NT Road Drainage Fact Sheet). It is noted that the recommended distance between turn-out drains is a guide and may not apply to all locations along the access track.
- Monitor road conditions to ensure deterioration does not occur. Assist in the maintenance and repair work on roads and tracks used.

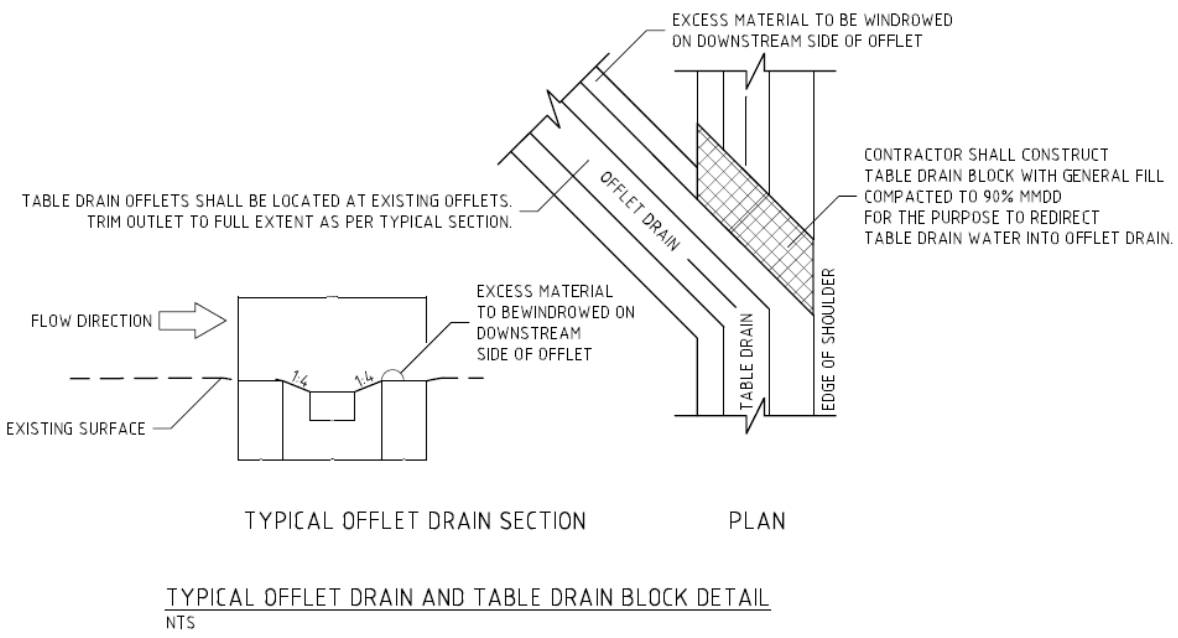


Figure 5 Typical offlet drain and table drain block detail