From:	Close, David
To:	fracking inquiry
Cc:	[redacted]
Subject:	Submission: Independent risk assessment of Beetaloo hydraulic fracturing fluids (including flowback fluid)
Date:	Tuesday, 12 September 2017 11:22:44 AM
Attachments:	image001.png Submission letter, Chem and HFS RA, 12 Sep 2017.pdf 170908_HFRA - Beetaloo Amungee NW-1H-FINAL.PDF

Dear Justice Pepper.

Please find attached correspondence regarding this submission of the hydraulic fracturing fluid risk assessment referenced by Origin in our appearances before the Inquiry Panel and in our April 30 written submission.

Regards,
David

David Close Ph.D.
Unconventional Exploration Manager

Origin
Level 5, 135 Coronation Drive, Milton QLD 4064
[redacted]

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12 September 2017

The Hon. Justice Pepper
Hydraulic Fracturing Taskforce
GPO Box 4396
Darwin, NT 0801, Australia

Dear Justice Pepper,

RE: Independent risk assessment of Beetaloo hydraulic fracturing fluids (including flowback fluid)

Please find attached the "Beetaloo Project Hydraulic Fracturing Risk Assessment", completed under contract by AECOM. This assessment provides a comprehensive overview of individual and collective risks associated with fluids used in hydraulic fracturing operations and recovered during flowback operations at the Amungee NW-1H well in 2016. We have previously referenced this risk assessment in our panel appearances and written submissions.

Please advise if the Inquiry Panel or Taskforce need any clarification regarding this submission. If you require any further information then please contact myself or Stephanie Stonier [redacted]

Yours sincerely,

David Close
Unconventional Exploration Manager
Origin
[redacted]

Attachments:
1. 170908_HFRA - Beetaloo Amungee NW-1H-FINAL.pdf

AECOM Imagine it. Delivered.

Origin Energy Resources Limited
08-Sep-2017

Beetaloo Project Hydraulic Fracturing Risk Assessment

Amungee NW-1H

Beetaloo Project Hydraulic Fracturing Risk Assessment

Amungee NW-1H

Client: Origin Energy Resources Limited

ABN: 66007845338

Prepared by

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Quality Information

Document Beetaloo Project Hydraulic Fracturing Risk Assessment

Ref 60542121

Date 08-Sep-2017

Prepared by Cindy Cheung, Tony Jong, Hayden Seear

Reviewed by Mark Chapman, Mark Stewart

Revision History

Rev	Revision Date	Details	Authorised	
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0	08-Sep-2017	Final	Hayden Seear Project Manager	

Table of Contents

Executive Summary i
1.0 Introduction 8
1.1 Background 8
1.2 Regulatory Considerations 8
1.3 Risk Assessment Process 8
1.4 Overall Approach 9
1.5 Structure of the Risk Assessment 10
2.0 Conceptual Site Model and Issue Identification 12
2.1 Conceptual Site Model 12
2.2 Issue Identification 12
3.0 Human Health Risk Assessment 14
3.1 Exposure Assessment 14
3.1.1 Potential Exposure Pathways and Receptors 15
3.1.2 Quantification of Exposure 17
3.2 Derivation of Oral Reference Dose and Drinking Water Guideline Values 19
3.2.1 Oral Toxicity Reference Value derivation 19
3.2.2 Determination of drinking water guideline values 19
3.3 Identification of Human Health COPC in Flowback Water 20
3.3.1 Empirical Dataset 20
3.3.2 Theoretical Dataset 21
3.4 Toxicity Assessment 25
3.4.1 Selection of Toxicity Values 25
3.4.2 Summary of Toxicity Reviews 26
3.5 Human Health Risk Characterisation 30
3.5.1 Risks to Human Health 30
3.5.2 Summary of Risk 31
3.6 Conclusions 33
4.0 Environmental Risk Assessment 34
4.1 Persistence, Bioaccumulation and Environmental Toxicity Assessment 34
4.2 Exposure Assessment 43
4.2.1 Potential Exposure Pathways and Receptors 43
4.2.2 Quantification of Exposure 48
4.3 Toxicity Assessment 49
4.3.1 General 49
4.3.2 Concentration Based TRV 49
4.3.3 Calculation of PNEC for Freshwater 49
4.4 Environmental Risk Characterisation 52
4.4.1 Estimation of Risk 52
4.4.2 Summary of Risk 53
4.5 Interpretation of Environmental Significance 61
4.6 Direct Toxicity Assessments 61
4.7 Conclusions 62
5.0 Non-Chemical Risks Assessment 63
5.1 Noise and Vibration 63
5.2 Radiological Exposure 63
5.2.1 Recommendations 64
6.0 Uncertainty Assessment 65
6.1 Sampling and Analysis Uncertainties 65
6.2 Exposure Uncertainties 65
6.3 Toxicity Uncertainties 65
7.0 Conclusions 66
7.1 Summary of Assessed Risks and Mitigation Measures 66
7.2 Summary of Findings 73
8.0 References 75

Appendix A
Site Conceptual Model A

Appendix B
Hydraulic Fracturing Fluid Mass Balance B

Appendix C
Risk Calculations C

Appendix D
Hydraulic Fracturing Fluid Chemical Toxicological Assessments D

Appendix E
Naturally Occurring Radioactive Materials Risk Assessment E

Executive Summary

Origin Energy (Origin) commissioned AECOM Australia Pty Ltd (AECOM) to perform a Human Health and Environmental Risk Assessment of hydraulic fracturing conducted for the Beetaloo shale gas project, specifically the hydraulic fracturing operation that was performed at shale gas well Amungee NW-1H.

Located approximately 650 km (by road) south-east of Darwin, the Amungee NW-1H well is within the Beetaloo Project exploration permits, which comprise of 18,512 km^2 of pastoral lease on the Sturt Plain, part of the Barkly Tableland.

The risk assessment was conducted in accordance with the Australian guidelines for human health and environmental risk assessments and Australian industry standard practice for hydraulic fracturing risk assessments.

Specifically, this report includes a qualitative and quantitative assessment of the potential human health and environmental risks associated with the injection of hydraulic fracturing fluids into the shale formations in the Beetaloo Basin and the management and storage of hydraulic fracturing fluid above grade.

The approach taken to the assessment of human health risks is in accordance with guidelines/ protocols provided by enHealth (2012a) and Amended National Environment Protection (Assessment of Site Contamination) Measure (NEPM) Schedule B4, Site-specific health risk assessment methodology (NEPC 2013). The framework for an environmental risk assessment is similar to the one adopted for the human health risk assessment and is consistent with guidance provided in the Amended ASC NEPM Guideline on Environmental Risk Assessment", Schedule B5, 2013 and in the Australian and New Zealand Guidelines for Fresh and Marine Water Quality (ANZECC) 2000.

This approach provides for a review of the nature and extent of potential contamination, identification of issues that may give rise to exposure, and identification of the key chemicals, exposure pathways and receptors that may be relevant. In addition, the assessment utilises site-specific data with exposure parameters and toxicity data (obtained from Australian sources where available) to enable the quantification of risk to human and environmental health.

On the basis of the risk assessment undertaken, with consideration of the uncertainties inherent in the assessment, the following is concluded:

The toxicity of the chemicals used in the hydraulic fracturing process has been assessed for persistence, bioaccumulation and aquatic toxicity (PBT), terrestrial toxicity and human health toxicity. Through this assessment methodology it has been determined that the chemicals used in hydraulic fracturing fluid systems can be generally characterized as neither persistent, bioaccumulative or toxic to aquatic and human receptors, however some chemicals (in undiluted form) have been associated with potential risks to aquatic receptors. The chemicals used in the hydraulic fracturing process do not contain benzene, toluene, ethylbenzene, xylene (BTEX) or polycyclic aromatic hydrocarbons (PAH) as additives, compliant with the legislative requirements for hydraulic fracturing fluid.

The risk assessment evaluated the toxicity of the individual chemicals, and characterised the cumulative risks of the total fluid mixture to human receptors and environmental terrestrial and aquatic receptors. The methodology incorporated an assessment of potential exposures to human and environmental receptors, with the following identified as the only potentially complete exposure pathways:

- Incidental ingestion and dermal contact of flowback fluid by human trespassers at the flowback fluid storage ponds; and
- Potential releases of flowback fluid to aquatic environments

Based on the risk mitigation measures Origin has in place, no potentially complete exposure pathway was identified for hydraulic fracturing chemicals to impact groundwater that is used for beneficial use in the project area.

The risk of chemical exposure to workers involved in the operation of hydraulic fracturing activities (including the transportation of chemicals) was not quantitatively evaluated as this risk is addressed

through the implementation of appropriate occupational health and safety procedures and management plans.

Potential exposures to hydraulic fracturing chemicals 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 ponds for evaporation or transport to an off-site water treatment facility.

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. Exposure point concentrations were also based on empirical data obtained from laboratory analysis of flowback water from Amungee NW-1H. Geogenic components were analysed as part of the empirical data set.

For the assessment of the overall potential for adverse human health effects posed by simultaneous exposure to multiple chemicals, the actual uptake of Chemicals Of Potential Concern (COPC) by 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). Similarly potentially environmental risks to aquatic receptors were estimated by comparing the EPC to reference values to provide a HQ, which was summed for a total HI of the mixture.

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 likely but the evaluation requires further evaluation to define the risk characterisation process.

On the basis of the risk evaluation, no unacceptable risk to human receptors (trespassers) was identified. The contribution of risk via geognenic components measured in the flowback water was considered insignificant, with the resultant risk several orders of magnitude below the threshold value of 1. Conservative risk scenarios assessed included regular access to the uncovered holding ponds, where swimming type activities could be undertaken, with exposures to high theoretical concentrations of COPC in the flowback water.

The risk assessment identified potential impacts to aquatic receptors could occur if substantial (acute) releases of flowback water occur and result in overland flow to impact upon adjacent surface water bodies. This conservative assessment does not take into account ephemeral surface water dilution that is expected to occur where flowback water is discharged to a surface water body in the event of a significant spill or leak. In addition, processes such as sorption and biodegradation have not been considered. During normal operation procedures there are no discharges to surface water bodies. The likelihood that any of these chemicals may be discharged to an environment where any level of exposure may occur is managed via the implementation of existing operational management measures, which are understood to be consistent with API guidance and ‘Best Practice’ specifications for the design and implementation of all hydraulic fracturing operations. Considering this, the identified level of risk is effectively negligible or low.

The overall risk to human health and environment associated with the chemicals involved in hydraulic fracturing is low. It is understood that Origin will further minimise the potential for exposures and risk by implementing effective operational controls, including:

- All relevant Australian standards are met during the drilling the wells.
- Unless otherwise authorised under a specific Approval, petroleum activities that require earthworks, vegetation clearing and/or placing fill, other than that associated with the construction of linear infrastructure, is not permitted in or within a specified buffer distance from any wetland, lake or spring and the high bank of any other watercourse. Typical buffer distance is 100 m.
- Implementation of spill containment procedures during operations to prevent migration of and exposure to chemicals, and if a spill was to occur, rectification measures would be taken immediately and notification to regulators of any unauthorised releases will be made.
- Engineered design of the fluid containment facility.
- Routine holding pond inspections to check for leakage of flowback fluid.

- Freeboard buffer included above high fluid level automatic pump shut-off system, in the event of heavy rainfall.
- Fencing and signage around the flowback storage ponds.
- Routine operational and security patrols to prevent trespassing.

The operational controls will be routinely evaluated by Origin and modifications and revisions made, where necessary. These operational and management controls can be further supported by the following recommendations:

- Maintain compliance with Origin's *Beetaloo Exploration Drilling Campaign 1 – Groundwater Monitoring Plan (NT-2050-15-MP-0011)*, which has been designed to provide baseline information about water level(s) and water quality across the area of the Permits.
- Maintain compliance with the *Amungee NW-1H Hydraulic Stimulation and Well Testing Environmental Plan (CDN/ID NT-2050-35-PH-0018)*, which has been designed to ensure the environmental risks of conducting hydraulic fracturing on Amungee NW-1H are understood and mitigated.

ABBREVIATIONS

Abbreviation	Description
ADI	Acceptable Daily Intake
ADWG	Australian Drinking Water Guidelines 2011 (updated 2016)
AEA	Australian Environmental Agency
AECOM	AECOM Australia Pty Ltd
ANZECC	Australian and New Zealand Environment and Conservation Council
API	American Petroleum Institute
APLNG	Australia Pacific LNG Project
ARMCANZ	Agriculture and Resource Management Council of Australia and New Zealand
ARPANSA	Australian Radiation Protection and Nuclear Safety Agency
ASC	Assessment of Site Contamination
ATSDR	Agency for Toxic Substances and Disease Registry
B	Bioaccumulation
BAF	Bio-accumulation Factor
BCF	Bio-concentration Factor
Beetaloo	Beetaloo Basin
Bq	Becquerel
Bq/kg	Becquerel per kilogram
Bq/L	Becquerel per litre
Bq/m^3	Becquerel per cubic metre
BOM	Bureau of Meteorology
CBL	Cement Bond Log
CCL	Casing Collar Locator
CG	Coordinator General
cm	centimetre
COPC	Chemical of Potential Concern
COPEC	Chemical of Potential Ecological Concern
CRC CARE	Cooperative Research Centre for Contamination Assessment and Remediation of the Environment
DotE	Department of the Environment
EA	Environmental Authority
EC	Electrical Conductivity
ECHA	European Chemicals Agency
EC(L)50	Median effective concentration
ErC50	EC50 in terms of reduction growth rate after exposure
enHealth	Environmental Health Standing Committee
EPBC	Environmental Protection and Biodiversity Conservation Act
EPC	Exposure Point Concentration
ERA	Environmental Risk Assessment
EU	European Union

Abbreviation	Description
GA	Geoscience Australia
GL	Gigalitre
GR	Gamma Ray
HHRA	Human Health Risk Assessment
HI	Hazard Index
hr	hour
HQ	Hazard Quotients
JMPR	Joint Meeting on Pesticide Residues
IARC	International Agency for Research of Cancer
IESC	Independent Expert Scientific Committee
IRIS	Integrated risk information system
K	Potassium
K_h	Horizontal hydraulic conductivity
kg	Kilogram
km	Kilometre
K_v	Vertical hydraulic conductivity
L	Litres
LC_{50}	Lethal Concentration for 50% of tested animals
LOAEL	Lowest Observed Adverse Effect Level
LOR	Limit of Reporting
m	metres
mg/L	milligrams per litre
ML	mega Litre
mSv	milliSievert
Mt	million tonnes
NEPM	National Environmental Protection Measures
NHMRC	National Health and Medical Research Council
NOAEL	No observed adverse effect level
NOEC	No Observed Effect Concentration
NORM	naturally occurring radioactive material
NRC	National Research Council
NT	Northern Territory
NWQMS	National Water Quality Management Strategy
OECD	Organisation for Economic Co-operation and Development
ORNL/RAIS	Oak Ridge National Laboratory/Risk Assessment Information System
Origin	Origin Energy Resources Limited
Ra	radium
RHSAC	Radiation Health and Safety Advisory Council
Rn	radon
P	Persistence

Abbreviation	Description
PAEC	potential alpha energy concentration
PBT	Persistence, Bioaccumulation, Toxicity
PGA	Peak Ground Acceleration
PNEC	Predicted No Effects Concentration
QSAR	Quantitative structure-activity relationship
REACH	Registration, Evaluation, Authorisation, and Restriction of Chemicals
RfD	Reference Dose
RME	Reasonable Maximum Exposure
S	Storativity
SDS	Safety Data Sheet
SIAR	SID Initial Assessment Reports
SIDS	Standard Information Data Sheets
Ss	Specific Storage
Sv	Sievert
T	Transmissivity or Toxicity
TBeq	Terabecquerel
TDI	Tolerable Daily Intake
TDS	Total Dissolved Solids
Th	thorium
TRV	Topological Reference Values
U	uranium
UF	Uncertainty Factor
USEPA	United States Environmental Protection Agency
WHO	World Health Organisation
VDL	Variable Density Log
^{40}K	potassium-40
^{208}Tl	thallium-208
^{210}Pb	lead-210
^{210}Po	polonium-210
^{212}Bi	bismuth-212
^{212}Pb	lead-212
^{212}Po	polonium-212
^{214}Bi	bismuth-214
^{214}Pb	lead-214
^{214}Po	polonium-214
^{216}Po	polonium-216
^{218}Po	polonium-218
^{220}Ra	radium-220
^{220}Rn	radon-220 (‘thoron’)
^{222}Rn	radon-222 (‘radon’)

Abbreviation	Description
^{224}Ra	radium-224
^{226}Ra	radium-226
^{228}Ra	radium-228
^{228}Th	thorium-228
^{232}Th	thorium-232
^{238}U	uranium-238

1.0 Introduction

1.1 Background

Origin Energy Resources Limited (Origin) commissioned AECOM Australia Pty Ltd (AECOM) to perform a Human Health and Environmental Risk Assessment of hydraulic fracturing conducted for the Beetaloo shale gas project, specifically the hydraulic fracturing operation that was performed at shale gas well Amungee NW-1H.

Located approximately 650 km (by road) south-east of Darwin, the Amungee NW-1H well is within the Beetaloo Project exploration permits, which comprise of 18,512 km^2 of pastoral lease on the Sturt Plain, part of the Barkly Tableland.

1.2 Regulatory Considerations

In accordance with the Northern Territory *Petroleum (Environment) Regulations (2016) (the Regulations)*, an Environmental Management Plan (EMP) must be approved by the Department of Primary Industry and Resources (formerly the Department of Minerals and Energy) prior to commencing well stimulation (hydraulic fracturing) activities, as they are classified a 'regulated activity'.

The EMP must include a robust environmental risk assessment process to ensure:

- Onshore oil and gas activities are carried out in a manner consistent with the principles of ecologically sustainable development; and
- Environmental impacts and risks associated with onshore oil and gas development activities are reduced to a level that is as low as reasonably practicable (ALARP) and acceptable.

It is noted the Environmental Plan for the Amungee NW-1H hydraulic fracturing operation (*Amungee NW-1H Hydraulic Stimulation and Well Testing Environmental Plan (CDN/ID NT-2050-35-PH-0018))* was developed and approved prior to the commencement of this regulation.

The Regulations specify that it is the responsibility of the proponent to demonstrate the environmental sources of risk and consequential impacts are identified and can be managed to avoid, reduce or mitigate environmental harm. The Regulations reference the Australian standard for risk assessments, AS/NZS ISO 31000:2009, as a guide for carrying out environmental risk assessments. The Regulations support the use of industry standard models to quantify environmental risks.

1.3 Risk Assessment Process

The methodology adopted for the Amungee NW-1H hydraulic fracturing risk assessment is in accordance with human health and environmental risk assessment industry standard practice.

The framework adopted for the human health risk assessment is consistent with guidance provided in:

- The protocols / guidelines recommended by enHealth "Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards", 2012; and
- The Amended National Environment Protection (Assessment of Site Contamination) Measure 1999 (Amended ASC NEPM); Schedule B4, Site-specific health risk assessment methodology, 2013.

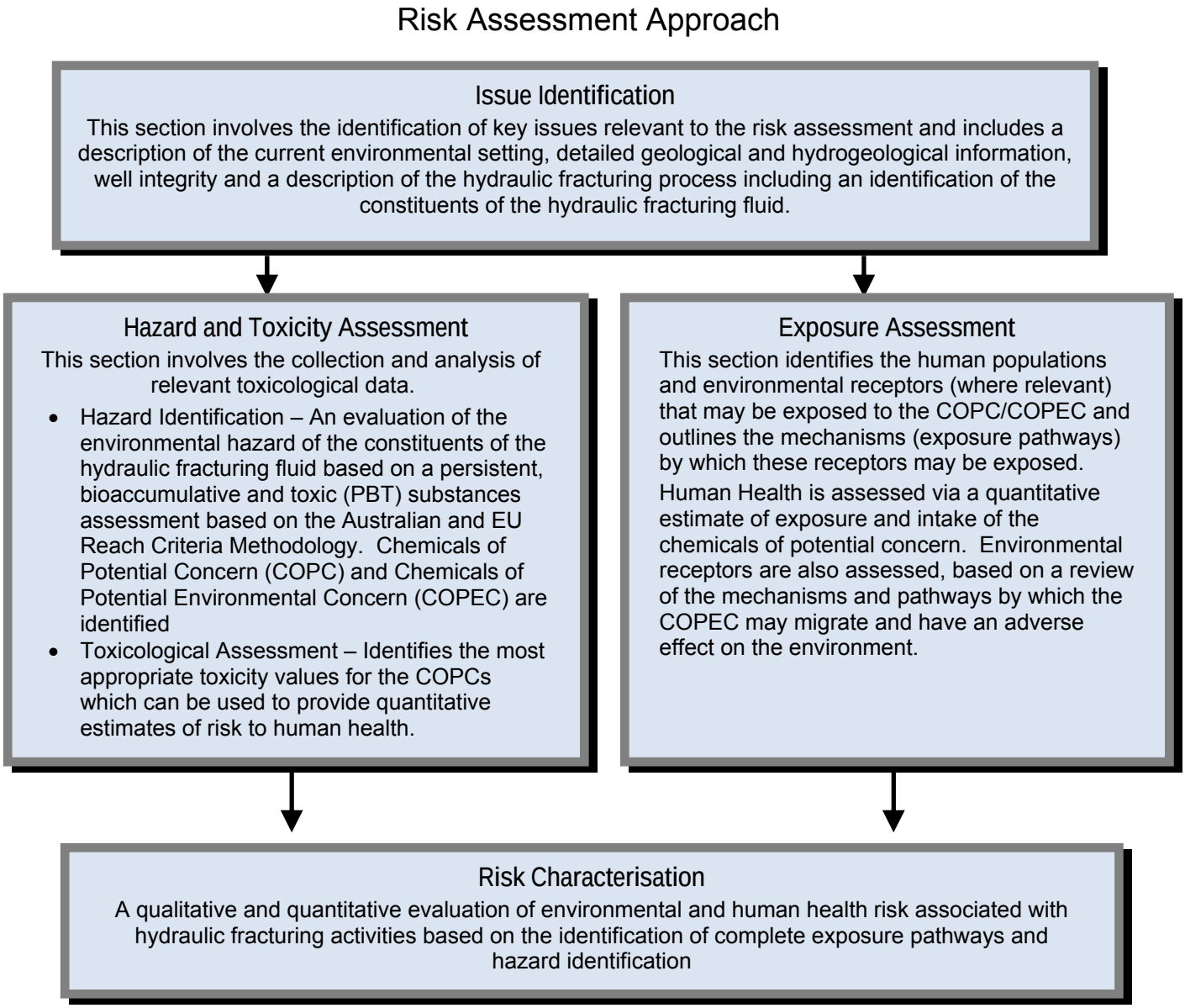
The environmental risk assessment framework is similar to that adopted for the human health risk assessment, and is consistent with guidance provided in:

- the Amended ASC NEPM Guideline on Environmental Risk Assessment", Schedule B5, 2013; and
- the Australian and New Zealand Guidelines for Fresh and Marine Water Quality (ANZECC) 2000.

The risk assessment also considered the guidance presented in National Health and Medical Research Council, Australian Drinking Water Guidelines (NHMRC 2011) for the derivation of drinking water guidelines.

1.4 Overall Approach

The following Risk Assessment Approach diagram illustrates the key activities associated with the environmental risk assessment and how each of these tasks fits into the overall assessment of risks. Key site-specific summary information is provided in the inserts within the flow chart, to provide an overview of the risk assessment presented herein.



1.5 Structure of the Risk Assessment

In order to adhere to the environmental risk assessment process, whilst addressing all the regulatory conditions, the report has been structured as follows:

Section 1.0: Describes the regulatory background for the risk assessment and the details the risk assessment process.

Section 2.0: Identifies the potential risks to conducting hydraulic fracturing in the project area.

Section 3.0: Assesses the potential human health risks associated with the hydraulic fracturing activities.

Section 4.0: Assesses the potential environmental risks associated with the hydraulic fracturing activities.

Section 5.0: Discusses potential environmental risks of hydraulic fracturing not related to the use of chemicals, these risks include: noise and vibration and exposure to radioactive substances.

Section 6.0: Discusses the remaining uncertainties from this risk assessment

Section 7.0: Summarises the risks of hydraulic fracturing performed at well Amungee NW-1H.

Appendix A: Details the conceptual site model and hydraulic fracturing process used by Origin, including: hydraulic fracturing location and schedule, the receiving environment, integrity of the gas well, hydraulic fracturing process, operation, fluid systems, and monitoring and assessment.

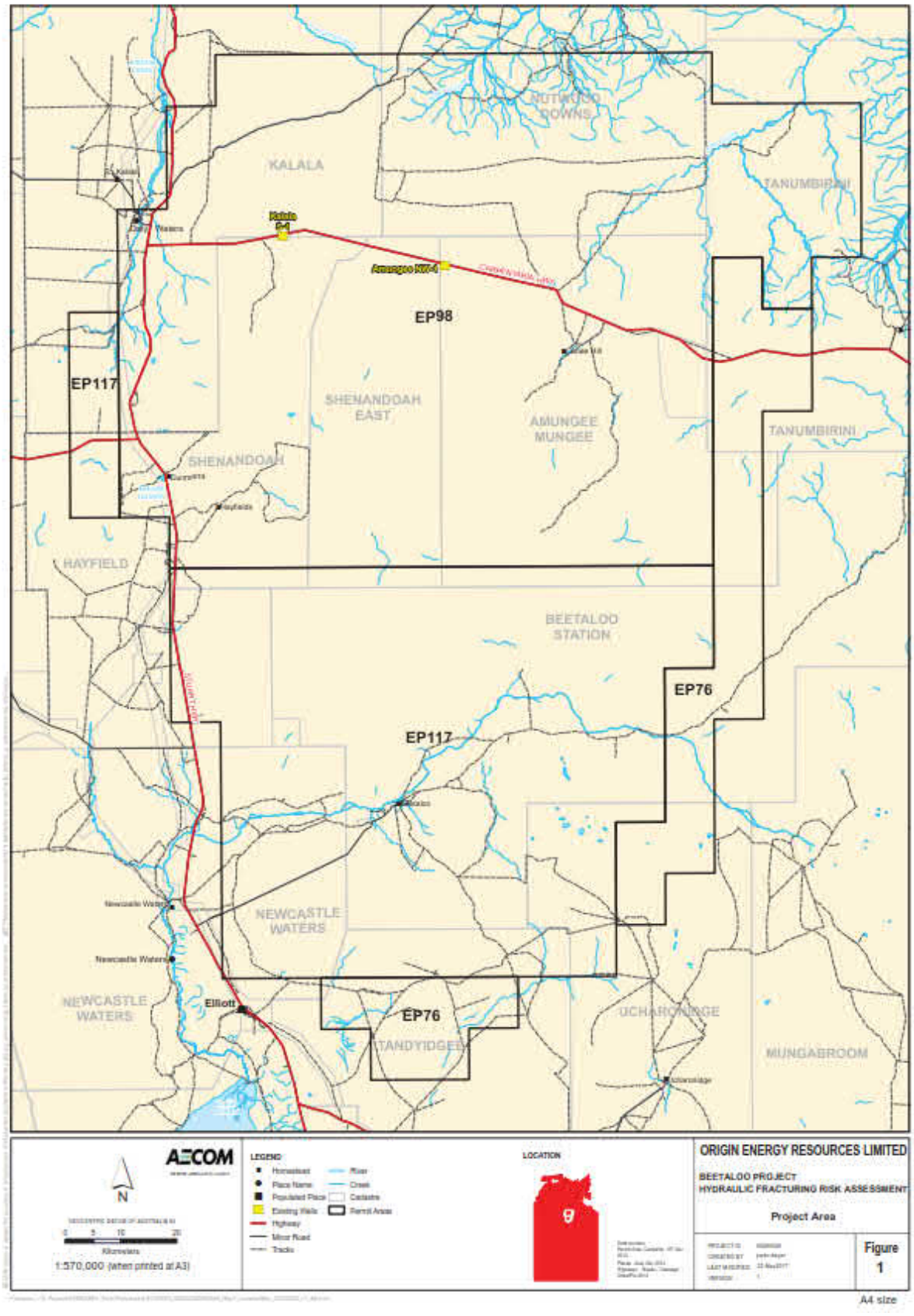
Appendix B: Presents the mass-balance of chemicals used in the hydraulic fracturing fluids

Appendix C: Collates the quantitative risk assessment calculations

Appendix D: Collates the ecotoxicological profiles prepared for chemicals used in the hydraulic fracturing fluids

Appendix E: Risk assessment of naturally occurring radioactive material present in the hydraulic fracturing flowback fluid

Figure 1 Project Area



2.0 Conceptual Site Model and Issue Identification

2.1 Conceptual Site Model

The Conceptual Site Model, which informs the Issue Identification Process, is presented in **Appendix A**.

The Conceptual Site Model comprises the following components:

- Location and Schedule (**Section A1.1**)
- Receiving Environment (**Section A1.2**)
- Well Integrity (**Section A1.3**)
- Hydraulic Fracturing (**Section A1.4**), inclusive of the hydraulic fracturing chemicals mass balance (**Appendix B**)

2.2 Issue Identification

As described in NEPC (2013), the problem formulation stage establishes the goals and focus of the assessment. Additional products of the problem formulation stage are a conceptual model that identifies the stressors, sources, receptors, exposure pathways and potential adverse human health effects that the risk assessment will evaluate, and a work plan that outlines the approaches that will be used in the risk assessment.

In 2016, the United States Environmental Protection Agency (US EPA) released the findings of a landmark study of hydraulic fracturing and its potential to impact upon drinking water resources (US EPA, 2016). The study is considered to define current international best practice for hydraulic fracturing risk assessments. The study identified six key conditions under which impacts of hydraulic fracturing activities can be more frequent or severe:

1. Water withdrawals for hydraulic fracturing in times or areas of low water availability, particularly in areas with limited or declining groundwater resources;
2. Spills during the handling of hydraulic fracturing fluids and chemicals or produced water that result in large volumes or high concentrations of chemicals reaching groundwater resources;
3. Injection of hydraulic fracturing fluids into wells with inadequate mechanical integrity, allowing gases or liquids to move to groundwater resources;
4. Injection of hydraulic fracturing fluids directly into groundwater resources;
5. Discharge of inadequately treated hydraulic fracturing wastewater to surface water; and
6. Disposal or storage of hydraulic fracturing wastewater in unlined pits resulting in contamination of groundwater resources.

Based on the information provided in the Amungee NW-1H Conceptual Site Model developed in **Appendix A, Section A1.2** (Receiving Environment), **Section A1.3** (Well Integrity) and **Section A1.4** (Hydraulic Fracturing) and considering the six key issues identified by the US EPA, the potential hazards of conducting hydraulic fracturing at Amungee NW-1H comprise:

- Flow of fluids out of the production zone
- Fracture overgrowth out of the production zone
- Migration of fluid via fractures intersecting geological features
- Migration of fluid via fractures intersecting with offset wells
- The loss of containment of chemicals used for hydraulic fracturing during hydraulic fracturing
- Alteration of groundwater quality in aquifers containing beneficial use groundwater overlying or underlying the target shale formation

- The loss of containment of hydraulic fracturing flowback water or produced water after hydraulic fracturing (inclusive of geogenic/naturally-occurring radioactive material)

Based on the information presented in the sections above, the objective of this assessment will consist of a qualitative and quantitative assessment of the potential human health and environmental risks associated with the injection of hydraulic fracturing fluids into the shale formations in the Beetaloo Basin and the management and storage of flowback fluids above grade.

The potential complete exposure pathways will be identified through the qualitative evaluation process, and quantitative assessments will then be completed only on those potentially complete exposure scenarios. Through the process of both qualitative and quantitative risk assessment, appropriate management controls will be identified for the management of potential risks.

3.0 Human Health Risk Assessment

The human health risk assessment (HHRA) was undertaken in general accordance with the following protocols and guidance:

- Amended National Environment Protection (Assessment of Site Contamination) Measure 1999 (Amended ASC NEPM); Schedule B4, Site-specific health risk assessment methodology (NEPC 2013);
- Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a). These guidelines draw on and are supplemented by those provided by a number of other Australian and international agencies, with some of the key supporting documents listed below:
  - Methodology for setting air quality standards in Australia (NEPC 2011); and
  - Science and decisions: Advancing risk assessment (NRC 2008).

Human health risk assessment can be divided into the following four tasks:

- Issue Identification
- Hazard/Toxicity Assessment
- Exposure assessment.
- Risk characterisation

Guidance provided by enHealth (and detailed by NEPC) is utilised as the primary source. However, where only general guidance for the completion of risk assessment tasks (above) is provided, more detailed protocols and guidelines developed by the National Research Council (NRC 2008) and United States Environment Protection Agency (US EPA) (1989, 1991, 2002, 2005 and 2009) have been used as supplementary guidance.

The HHRA approach evaluates the toxicity of the individual substances and characterises the cumulative risks of the total effluent toxicity and ecotoxicity, which is consistent with the National Water Quality Management Strategy (NWQMS) and the principles outlined in the ANZECC & ARMCANZ (2000) guidelines.

3.1 Exposure Assessment

This section identifies the human populations (receptors) who may be exposed to the COPC identified for the site, and outlines the exposure pathways by which these receptors may be exposed. The exposure assessment also provides a quantitative estimate of exposure and intake of the COPC.

For an exposure pathway to be considered to be complete there must be all of the following:

- Source of COPC - how the chemical got into the environment;
- A transport media - how the chemical moves or migrates through the environment (soil, water or air);
- An exposure point - how human and environmental receptors can come into contact with the chemicals; and
- An exposure route - how the chemical could enter the body of the receptor (e.g. inhalation, ingestion or dermal contact).

If any one of these steps (source, transport media, exposure point or route) is not present, then the exposure pathway is incomplete and, hence, further assessment of risks is not required.

In the cases where the exposure pathways are complete, or have the potential to be complete, then the pathways can be considered significant or less significant. The significance of the exposure pathway depends on the nature of the impact present and the evaluation of the likely exposure concentrations that may be associated with the pathway.

3.1.1 Potential Exposure Pathways and Receptors

A summary of the decision tree used to evaluate receptors and pathways is presented in **Table 1**. Shaded cells indicate potentially complete exposure pathway, assessed as part of this HHRA.

Table 1 Identification of Potential Human Receptors and Pathways

Source	Exposure Scenario	Receptors (On and Off-site)	Pathway	Pathway Potentially Complete?	Comment
Hydraulic Fracturing Chemicals	Handling of chemicals either on or off-site (i.e. during transportation of chemicals to site), spills and leaks.	On and off-site workers	Ingestion, dermal	No	OH&S procedures and spill containment adequately address this exposure.
Hydraulic Fracturing Fluids	Fracturing fluid escapes into aquifer (well casing failure, fault, and fracture, unconformity in seam or strata) and enters a water bore used for beneficial use.	Off-site residents	Ingestion, dermal	No	Origin has detailed operational procedures that are designed to contain hydraulic fracturing fluids within the shale formation. No direct connection is understood to exist between groundwater in the shale formation and surface-water, aquifers utilised for the purpose of extraction (refer **Appendix A, Section A1.2.4**).
Flowback Water	Deliberate entry into Flexiponds	Trespassers	Ingestion, dermal	Yes	Trespassers entry is limited via fencing and signage around the drill pad areas. In addition, the walls of the Flexiponds are 2 m high. Access ladders on the side of the Flexiponds exist. As such, this pathway has been evaluated as part of this assessment.
	Deliberate entry near the Flexiponds, within the vicinity of mist fallout from the evaporator.	Trespassers	Inhalation, dermal (of flowback water as mist from evaporator)	No	Most flowback water COPC are not volatile, and no COPC are sufficiently volatile and present at high enough concentrations to be of concern in relation to inhalation exposures. Dermal absorption and ingestion of COPC in flowback water (as mist) may occur, however the pathway is not considered to be of significance, due to the short exposure period. The Evaporator is programmed to automatically shut down in unsafe conditions (i.e. changes in wind speed or wind conditions not within the approved operating envelope), to ensure the plume mist is contained within the Flexipond.
	Working with the Flexiponds (i.e. drainage)	Workers	Ingestion, dermal	No	OH&S procedures limit workers exposure to flowback water.
	Flexipond overflow/ failure/leaks	Workers	Ingestion, dermal	No	OH&S procedures limit workers exposure to flowback water. Origin control measures include regular pond inspections

Source	Exposure Scenario	Receptors (On and Off-site)	Pathway	Pathway Potentially Complete?	Comment
					to identify level and/or faults.

In reviewing and considering the potential significance of exposure pathways (presented in **Table 1**), the following points have been considered:

- Origin has in place detailed operational procedures which are designed to contain the hydraulic fracturing fluids within the target shale formation. Providing these operational procedures are followed, there is no potential for connectivity between beneficial use groundwater and the hydraulic fracturing operation. On this basis, no further assessment of beneficial use groundwater is warranted.
- Potential risks to workers involved with the hydraulic fracturing process, where the COPC are handled on and off-site (i.e. during transportation of the COPC to the site), have not been considered as detailed Health and Safety (H&S) procedures are employed to manage these exposures.
- All flowback fluid will be stored on location in lined, above ground Flexiponds to allow Origin to assess the quality and chemistry of the flowback fluid to accurately determine the appropriate management of that fluid. Above ground contained storage also prevents access to the fluid by livestock and other ground dwelling fauna. While it is expected that the Flexiponds are not accessible to the public, exposure may occur if trespassers enter the Flexiponds, or if there is a leak in the pipeline or structure, and water from the infrastructure is released to the local environment.
- There is potential for flowback water mist (as created by the evaporator) to be distributed onto the adjacent surrounding areas (dependant on the wind strength), as such, trespassers may be exposed to the COPCs via inhalation or direct contact of the mist. However, most of the COPC identified in flowback water are not volatile, and no COPC are sufficiently volatile and present at high enough concentrations to be of concern in relation to inhalation exposures. Dermal absorption and ingestion of COPC in flowback water (as mist) may occur, however the pathways are not considered to be of significance, due to the short exposure period (further, the risk of ingestion of flowback water will be assessed via the conservative scenario of trespassers access into the Flexiponds, where swimming type activities will be undertaken). In addition, AECOM understand the enhanced evaporator unit is programed only to operate in optimal weather conditions to ensure the plume of mist generated remains within the extent of the Flexipond. A weather station is setup nearby to monitor wind intensity and direction. If there is a sudden change in wind direction or an increase in wind speed, the evaporator automatically shuts down and will not restart until conditions return to within the approved operating envelope. Prior to being used to evaporate flowback water, the evaporator was commissioned using potable water. On this basis, the risk to trespassers direct contact with flowback water (as mist) has not been further assessed.

Based on the available information, the key human receptors (and all complete exposures that are considered significant) that may be exposed to the COPC identified on and off the site include the following:

<u>Trespassers</u>

- Direct contact (ingestion and dermal contact) with COPC identified in flowback water contained within the above-ground Flexiponds, where recreational activities (i.e. swimming) will be undertaken.

3.1.2 Quantification of Exposure

When calculating chemical intake or exposure, the risk assessment process focuses on exposure occurring over a prolonged period; that is, chronic exposure that occurs over years and possibly a lifetime. The following steps have been followed to estimate chemical intake:

- Calculation of ***intake factors*** for each of the identified exposure pathways and receptors. An intake factor is a site-specific and receptor-specific value which, when multiplied by the concentration of each COPC, provides an estimate of the daily chemical intake of the COPC for each receptor and pathway. Some examples include inhalation rate, exposure frequency (i.e. hours per day or days per year) and exposure duration (e.g. number of years as a resident) and body weight; and

- Estimation of the ***chemical concentration*** in each medium relevant to the receptor groups and exposure pathways. This involves the use of relevant data collected from the site and surrounds.

The assessment presented has addressed potential worst-case exposure to COPC, and exposure has been calculated for a ***Reasonable Maximum Exposure (RME)*** scenario estimated by using the intake factors and chemical concentrations that define the highest exposure that is reasonably likely to occur in the area assessed. The RME is likely to provide a conservative estimate of total exposure and therefore health risk.

3.1.2.1 Exposure Parameters

Exposure parameters which are considered representative of RME have been selected for the receptor group evaluated; namely trespassers. Where available, exposure parameters have been obtained from Australian sources (CRC CARE 2011; enHealth 2012; NEPC 2013). As required, this has been supplemented with data from the US EPA (2008 and 2009).

The exposure assumptions adopted are consistent with the typical exposure assumptions utilised by enHealth (2012) for recreational exposure. The exposure parameters adopted also partially reflect that storage of flowback water at individual well pad sites will be only a temporary activity and only during pilot operations, with operational controls and activities at the well sites likely limiting the occurrence of trespasser entry and exposures.

The following **Table 2** presents an overview of the parameters selected. These parameters, as well as relevant references, are utilised in the exposure models presented in **Appendix C** .

Table 2 Exposure Parameters – Trespassers

Exposure Factor	Units	Trespasser	Reference
Exposure Frequency	Days/year	1 day within the time period when the flowback pond is on-site during the pilot phase.	Professional judgement
Exposure Duration	Year	6 months for adults and children, the maximum duration a flowback pond will be on-site during the pilot phase.	Professional judgement
Exposure Time Outdoors	Hours/day	1 hour	Professional judgement
Surface Area	cm^2	20,000 cm^2 for adults, 15,900 cm^2 for children. Exposure is assumed to be equal to that of swimming, where the whole body gets wet	enHealth 2012
Body Weight	kg	78 kg for adults, 39 kg for children	enHealth 2012

3.1.2.2 Exposure Equations

The following equations were used for calculating the intake of COPC:

Ingestion of water:

$$Daily\ Chemical\ Intake\ (iw) = Cw\ x\ \frac{IRw\ x\ EF\ x\ ED}{BW\ x\ AT}$$

Dermal contact with water:

$$Daily\ Chemical\ Intake\ (dw) = Cw\ x\ \frac{SAw\ x\ ET\ x\ DP\ x\ CF\ x\ EF\ x\ ED}{BW\ x\ AT}$$

Where:

CW = concentration in water (mg/l)
ET = exposure time (hr/day)
EF = exposure frequency (day/yr)
ED = exposure duration (years)
CF = correction factor (0.001 L/cm^3)

AT = Averaging time (days)
IR = Ingestion rate (L/hr)

BW = body weight (kg)
SA = skin surface area (cm^2/d)
DP = dermal permeability factor (cm/hr)

The calculations undertaken to estimate intake via ingestion and dermal absorption are presented in **Appendix C**.

3.1.2.3 Exposure Point Concentrations

Exposure Point Concentrations (EPC) of COPC requiring further assessment, as identified on the basis of theoretical or empirical data are presented in **Section 3.3**.

3.2 Derivation of Oral Reference Dose and Drinking Water Guideline Values

3.2.1 Oral Toxicity Reference Value derivation

Where required, the oral reference dose was calculated using the following equation:

$$Oral\ TRV = \frac{NOAEL\ (or\ LOAEL)}{UF_{Total}}$$

Where:

NOAEL = No Observed Adverse Effect Level
LOAEL = Lowest Observed Adverse Effect Level
UF_{Total} = Total uncertainty factors

When dosing regimens were 5 days/week, the NOAEL was adjusted to 7 days/week by multiplying the NOAEL by 5/7.

Uncertainty factors of 10 were used to account for the following (USEPA, 2002):

- Variation in sensitivity among members of the human population, i.e., inter-individual variability:
- Uncertainty in extrapolating animal data to humans, i.e., interspecies uncertainty;
- Uncertainty in extrapolating data obtained in a study with less-than-lifetime exposures, i.e., extrapolating from subchronic to chronic exposure; and.
- Uncertainty in extrapolating from a LOAEL rather than from a NOAEL.

3.2.2 Determination of drinking water guideline values

The ADWG (2016) values were used when applicable. The WHO, and then USEPA, were also consulted for any additional drinking water standards when there were no ADWGs.

For those chemicals where there are no regulatory drinking water guidelines, and for which a threshold exists, the guideline value was derived from animal toxicity data using the following equation (ADWG, 2016):

$$Drinking\ Water\ Criteria = \frac{NOAEL\ (or\ LOAEL) x\ Body\ Weight\ x\ Proportion\ intake\ from\ water}{volume\ of\ water\ consumed\ x\ UF_{Total}}$$

Where:

NOAEL = No Observed Adverse Effect Level
LOAEL = Lowest Observed Adverse Effect Level
Body weight = 78 kg
Proportion of intake from water = 10%
Volume of water consumed = 2 L/day
UF_{Total} = Total Uncertainty Factors

3.3 Identification of Human Health COPC in Flowback Water

COPC have been identified as those which are known or suspected to be present at concentrations high enough to warrant inclusion in an assessment of human health risks. The identification of COPC is based on the assessment of the nature of these chemicals in the environment on-site. Two datasets were evaluated in this HHRA: a theoretical and an empirical dataset

3.3.1 Empirical Dataset

The empirical dataset was obtained from the laboratory analysis of the following three samples of flowback water collected from the Amungee NW1-H site in October 2016.

- Sample BET-TK001, collected from Flexipond at Amungee NW1-H
- Sample BET-TK002, collected from Flexipond at Amungee NW1-H
- Sample BET-TK003, collected from Flexipond at Amungee NW1-H

Samples were analysed for Total Petroleum Hydrocarbons (TPH), Benzene, Toluene, Ethylbenzene, Xylenes (BTEX), Inorganics, heavy metals, Polycyclic aromatic Hydrocarbons (PAH)/Phenols, Volatile Organic Carbons (VOCs), and radionuclides.

The determination of COPC from the empirical dataset was based on a comparison of the analytical results with human health-based screening level guidelines. It should be noted that the presence of chemicals at concentrations higher than the screening level guidelines does not indicate an unacceptable risk; rather, it indicates that potential exposures to these chemicals should be evaluated in greater detail, taking into account site-specific pathways of exposure. The screening criteria considered in this assessment include:

- Australian Drinking Water Guidelines (ADWG), 2016; The National Health and Medical Research Council (NHMRC).
- Resource Management Council of Australia and New Zealand have developed the Australian Drinking Water Guidelines, updated in 2016. The guidelines provide health-based and aesthetic values for a range of micro-organisms, physical quality, inorganic chemicals, organic chemicals, radiological quality and pesticides. The health-based guideline values, which were used to identify COPC in the flowback water, are concentrations, which, based on present knowledge, do not result in any significant risk to the health of a consumer of the water over a lifetime. These guidelines are recognised within the Amended ASC NEPM (Schedule B(1) Guideline on Investigation Levels for Soil and Groundwater, 2013) as relevant Groundwater Investigation Levels (GILs) for the assessment of human health issues at the point of extraction (for use as drinking water – protection of human health issues associated with use of water as domestic supply within households). These guidelines are more current and extensive than the ANZECC 2000 Guidelines for Recreational Water Quality and Aesthetics (relevant to lower levels of exposure than drinking water), and, hence, were used in preference to the recreational guidelines.
- World Health Organisation Drinking Water Guidelines (WHO DWG), 2008 and rolling revisions. The WHO has also developed drinking water guidelines using the same approach as in the ADWG. The health-based guideline values, which were used to identify COPCs in the groundwater, are concentrations, which, based on present knowledge, do not result in any significant risk to the health of a consumer of the water over a lifetime.
- USEPA Regional Screening Levels 2016. Where no guideline value was available from the above sources, the USEPA Regional Screening Levels (RSLs) were used. The RSLs are conservative, risk-based values for soil, tap water and air. It should be noted that the RSLs are currently not recognised in Australia. However, they were used in this assessment to provide a screening level for the purpose of identifying the COPC for further assessment.

  Table 3 presents a summary of the flowback water data reviewed with a comparison against the relevant screening levels for determination of COPC requiring further assessment in this HHRA. Only chemicals above the LOR are assessed for screening purposes.

Table 3 Identification of empirical COPC in flowback water on-site during operations

Analyte Detected in Flowback Water	Max Concentration Reported in 2016 (mg/L)*	Adopted Screening Level Guideline (mg/L)		Selected as COPC?
		Value	Source	
Arsenic	0.084	0.007	ADWG	Yes
Barium	36.4	2	ADWG	Yes
Boron	66	4	ADWG	Yes
Chromium (III+VI)	0.055	0.1	USEPA RSL	No
Cobalt	0.011	0.006	USEPA RSL	Yes
Manganese	2.24	0.5	ADWG	Yes
Nickel	0.04	0.02	ADWG	Yes
3-&4-methylphenol	0.762	1.5	USEPA RSL	No
Phenol	0.0185	5.8	USEPA RSL	No
TRH C6 - C10	0.05	15	WHO	No
TRH C10 - C16	0.49	0.1	WHO	Yes
TRH C16 - C34	0.49	0.09	WHO	Yes
Benzene	0.004	0.001	ADWG	Yes
Toluene	0.003	0.8	ADWG	No
Xylenes	0.002	0.6	ADWG	No

Notes:
*2016 data from BET-PW001, BET-TK001, BET-TK002 and BET-TK003, sourced from Origin.
ADWG Australian Drinking Water Guidelines (NHMRC 2011) updated in 2016)
WHO Guidelines for Drinking Water Quality (2011)
USEPA Regional Screening Levels (May 2016)
Shaded cells indicate COPC assessed as part of this HHRA

Review of the data presented in **Table 3** indicates that in relation to the assessment of risks to human health a number of the compounds detected are well below the adopted screening level criteria. These compounds will not significantly contribute to any risk issues evaluated at the site and will not be further evaluated in this assessment. Only those compounds detected in flowback water that exceed the available guidelines are considered further in this assessment.

3.3.2 Theoretical Dataset

The theoretical dataset calculated exposure point concentrations (EPCs) using the mass balance data for each hydraulic fracturing fluid system (refer to **Appendix A**, **Section A1.4.7**). Two fluid systems (Slickwater and a Crosslinked Gel - Slickwater Hybrid) were used during the 2016 hydraulic fracturing program. For the purpose of this assessment, it was conservatively assumed that 100% of the mass of chemicals injected into the well will be present in the flowback water. The theoretical concentrations of the frac chemicals in flowback water are presented in **Table 4** and **Table 5** below.

The determination of COPC from the theoretical dataset was based on a comparison of the estimated concentrations with human health-based screening level guidelines. Where available, the Australian Drinking Water Guidelines (ADWG 2016) (i.e. concentrations in water that are safe for all members of the population to consume every day for a lifetime) have been identified and considered in this assessment. Where these guidelines are not available and the available published reviews have identified a suitable Tolerable Daily Intake (TDI) or No Observed Adverse Effects Level (NOAEL) this value has been presented and used to derive a preliminary drinking water guideline following guidance provided by the National Health and Medical Research Council (refer to **Section 4.4.2** for further discussion).

Table 4 Theoretical concentrations of COPC in flowback water – Amungee NW-1H Stage 1 Slickwater and Gel Flush

COPC	Mass injection in hydraulic fracturing fluid (mg/L)	Flowback Concentration 100% mass recovery (mg/L)	Adopted Screening Level Guideline (mg/L)	Selected as COPC?
Stage 1 Slickwater and Gel Flush				
Urea	6.86	6.86	NA	No
Tetrasodium ethylenediaminetetraacetate	0.22	0.22	0.25 as EDTA (A)	No
2-hydroxy-N,N,N-trimethylethanaminium chloride	1499.41	1499.41	248 (D)	Yes
Propan-2-ol	1.71	1.71	16.38 (D)	No
2-Propenamid (acrylamide)	0.32	0.32	0.0002 (A)	Yes
Ethylene Glycol	107.60	107.60	0.59 (D)	Yes
2,2"-oxydiethanol (diethylene glycol)	1.07	1.07	1.17 (D)	No
Dodecamethylcyclohexasiloxane	0.11	0.11	0.39 (D)	No
Decamethyl cyclopentasiloxane	0.11	0.11	0.39 (D)	No
Octamethylcyclotetrasiloxane	0.11	0.11	0.39 (D)	No
Sodium hydroxide (caustic soda)	0.54	0.54	180 (A)	No
2-methyl-2h-isothiazol- 3-one (Methylisothiazolinone)	1.18	1.18	0.078 (D)	Yes
Potassium chloride	0.32	0.32	71 (D)	No
Non-crystalline silica	1.61	1.61	NA	No
Hydrochloric acid	579.57	579.57	NA	No
Sodium chloride	20.35	20.35	NA	No
Sodium sulfate	1.61	1.61	180 as sodium (A)	No
Copper(II) sulfate	0.11	0.11	2 as copper (A)	No
Ammonium sulfate	142.19	142.19	250 as sulfate (A)	No
Magnesium chloride	3.74	3.74	250 (A)	No
Guar gum	62.85	62.85	49 (D)	Yes
Calcium chloride	10.28	10.28	NA	No
Magnesium nitrate	7.60	7.60	50 as nitrate (A)	No
Cristobalite	0.74	0.74	NA	No
5-chloro-2-methyl-2h-isothiazolol-3-one (Methylchloroisothiazolinone)	4.08	4.08	0.078 (D)	Yes
Polyethylene glycol monohexyl ether	203.75	203.75	1.95 (D)	Yes
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid,	104.93	104.93	NA	No

COPC	Mass injection in hydraulic fracturing fluid (mg/L)	Flowback Concentration 100% mass recovery (mg/L)	Adopted Screening Level Guideline (mg/L)	Selected as COPC?
sodium salt polymer				
Dicoco dimethyl quaternary ammonium chloride	8.45	8.45	0.39 (D)	Yes
Dimethyl siloxanes and silicones	0.11	0.11	5.85 (D)	No
Siloxanes and silicones, dimethyl, reaction products with silica	0.11	0.11	5.85 (D)	No
Diatomaceous earth, calcined	37.79	37.79	NA	No
2-Propenoic acid, polymer with sodium phosphinate	100.65	100.65	NA	No
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate	11.25	11.25	NA	No

Notes: D – Derived; A – ADWG 2016; NA – Not applicable, *refer to **Appendix C** for more detail; mg/L – milligram per litre

Table 5 Theoretical concentrations of COPC in flowback water – Amungee NW-1H Stage 7 Cross-Linked Gel/Slickwater Hybrid

COPC	Mass injection in hydraulic fracturing fluid (mg/L)	Flowback Concentration 100% mass recovery (mg/L)	Adopted Screening Level Guideline (mg/L)	Selected as COPC?
Stage 7 Cross Linked Gel/Slickwater Hybrid				
Urea	2.60	2.60	NA	No
Tetrasodium ethylenediaminetetraacetate	0.14	0.14	0.25 as EDTA (A)	No
2-hydroxy-N,N,N-trimethylethanaminium chloride	1692.76	1692.76	248 (D)	Yes
Propan-2-ol	1.73	1.73	16.38 (D)	No
2-Propenamid (acrylamide)	0.14	0.14	0.0002 (A)	Yes
Ethylene Glycol	360.58	360.58	0.59 (D)	Yes
2,2"-oxydiethanol (diethylene glycol)	1.01	1.01	1.17 (D)	No
Dodecamethylcyclohexasiloxane	0.14	0.14	0.39 (D)	No
Decamethyl cyclopentasiloxane	0.14	0.14	0.39 (D)	No
Octamethylcyclotetrasiloxane	0.14	0.14	0.39 (D)	No
Sodium hydroxide (caustic soda)	200.81	200.81	180 (A)	Yes
2-methyl-2h-isothiazol- 3-one (Methylisothiazolinone)	1.16	1.16	0.078 (D)	Yes
Potassium chloride	0.36	0.36	71 (D)	No
Non-crystalline silica	5.78	5.78	NA	No
Hydrochloric Acid	459.50	459.50	NA	No

COPC	Mass injection in hydraulic fracturing fluid (mg/L)	Flowback Concentration 100% mass recovery (mg/L)	Adopted Screening Level Guideline (mg/L)	Selected as COPC?
Sodium chloride	19.37	19.37	NA	No
Sodium sulfate	0.58	0.58	180 as sodium (A)	No
Copper(II) sulfate	0.14	0.14	2 as copper (A)	No
Ammonium sulfate	52.89	52.89	250 as sulfate (A)	No
Magnesium chloride	3.61	3.61	250 (A)	No
Guar gum	9.83	9.83	49 (D)	No
Calcium chloride	9.83	9.83	NA	No
Magnesium nitrate	7.23	7.23	50 as nitrate (A)	No
Cristobalite	0.65	0.65	NA	No
5-chloro-2-methyl-2h-isothiazolol-3-one (Methylchloroisothiazolinone)	3.83	3.83	0.078 (D)	Yes
Polyethylene glycol monohexyl ether	208.54	208.54	1.95 (D)	Yes
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer	38.95	38.95	NA	No
Dicoco dimethyl quaternary ammonium chloride	8.74	8.74	0.39 (D)	Yes
Dimethyl siloxanes and silicones	0.14	0.14	5.85 (D)	No
Siloxanes and silicones, dimethyl, reaction products with silica	0.14	0.14	5.85 (D)	No
Diatomaceous earth, calcined	36.06	36.06	NA	No
2-Propenoic acid, polymer with sodium phosphinate	96.11	96.11	NA	No
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate	4.19	4.19	NA	No
Fumaric acid	11.71	11.71	23.4 (D)	No
Boronatrocalcite	445.34	445.34	3.5 as boron (A)	Yes
Sodium tetraborate	37.50	37.50	3.5 as boron (A)	Yes
Monosodium fumarate	11.71	11.71	23.4 (D)	No
Sodium bromate	149.58	149.58	0.02 as bromate (A)	Yes
Boric acid	11.71	11.71	3.5 as boron (A)	Yes
Diutan	0.58	0.58	3.9 (D)	No

Notes: D – Derived; A – ADWG 2016; NA – Not applicable, *refer to **Appendix C** for more detail; mg/L – milligram per litre

3.4 Toxicity Assessment

The objective of the toxicity assessment is to identify toxicity values for the COPC that can be used to quantify risks to human health associated with the calculated intake. The quantification of risk requires identification of toxicity values for the COPC identified as well as quantification of potential exposure.

Toxicity can be defined as *"the quality or degree of being poisonous or harmful to plant, animal or human life"* (NEPC 2013).

The steps involved in this process include the following:

- Obtain relevant qualitative and quantitative toxicity information on the COPC relevant to the significant exposure pathways being assessed.
- Identify the appropriate toxicity values for assessing both threshold[1] effects and non-threshold carcinogenic[2] effects.

3.4.1 Selection of Toxicity Values

The identification of toxicity values undertaken in this risk assessment has followed ANZECC (1992) guidance, which is in accordance with the NEPC (1999) policy. enHealth (2004) provides a list of toxicological data sources. These are classified as Level 1 or 2 data, with Level 1 sources recommended. In order of preference, the Level 1 sources are:

1. National Health and Medical Research Council documents and documents from other joint Commonwealth, State and Territory organisations.
2. ADI List from the Therapeutic Goods Administration.
3. World Health Organisation (WHO) documents.
4. enHealth Council documents.
5. National Environmental Health Forum documents.
6. International Agency for Research on Cancer (IARC) monographs.
7. WHO/FAO Joint Meeting on Pesticides (JMPR) monographs.
8. NICNAS Priority Existing Chemical (PEC) reports.
9. US Agency for Toxic Substances and Disease Registry (ATSDR) documents.
10. National Toxicology Program (NTP) carcinogenicity appraisals.
11. OECD Standard Information Data Sets (SIDS) and SID Initial Assessment Reports (SIAR).
12. USEPA Reference Doses.

Level 2 sources include peer-reviewed journals and industry publications and reference to Level 2 sources is considered warranted where Level 1 sources do not provide applicable criteria.

The following types of toxicity values may therefore be applicable:

- Acceptable or Tolerable Daily Intakes (ADIs or TDIs), for assessment of non-cancer effects (ANZECC, NHMRC and WHO);
- Benchmark doses, for assessment of cancer effects (ANZECC);

[1] Threshold toxicity effects are assessed on the basis that there is a dose of the chemical below which toxic effects will not occur (i.e., the threshold).

[2] Non-threshold carcinogenic effects assume that, for some chemicals classified as carcinogenic, there is no threshold below which there will be no increased risk of a toxic effect. Hence, assessment of these chemicals is based on the use of a slope factor, which assumes that any exposure to the chemicals will result in an increased incremental risk or probability of developing cancer over a lifetime.

- Reference Concentrations (RfC), for inhalation assessment of non-cancer effects (USEPA);
- Reference Doses (RfD), for oral assessment of non-cancer effects (USEPA); and
- Cancer slope factors, for assessment of cancer effects (WHO and USEPA).

Potential threshold effects are characterised by comparing the estimated chemical intakes with the ADIs, TDIs or RfDs, that represent the threshold intake for adverse health effects. Threshold toxicity effects are assessed on the basis that there is a dose of the chemical below which toxic effects will not occur (i.e., the threshold).

Potential non-threshold carcinogenic effects are the estimated incremental probabilities that an individual will develop cancer over a lifetime as a result of the estimated exposure to the COPC. When a carcinogenic slope factor is used to evaluate health risk, it is assumed that any exposure to the chemical will, in theory, result in an increased risk or probability of developing cancer. The higher the carcinogenic slope factor, the more potent the chemical, and the greater the calculated cancer risk for a given exposure.

In reviewing relevant toxicity information for the COPC identified in this HHRA, the following has also been considered in the approach adopted:

- **Assessment of Dermal Exposures**: No dermal toxicity values are available for the COPC identified. While it is not considered appropriate to undertake route-route extrapolation, the adoption of oral toxicity values (with relevant consideration of bioavailabilities) has been identified and considered appropriate by USEPA (2004) in the absence of dermal data. Hence, dermal exposures have been assessed on the basis of oral data, where available
- **Assessment of Oral Exposures**: The oral Toxicity Reference Value (TRV) is utilised for the purpose of establishing drinking water (or recreational water) guidelines. Existing oral TRVs and drinking water guideline values were used when applicable. Otherwise, information on mammalian toxicity was acquired primarily from reported data that had already been through a screening process such as the OECD SIDS program, the U.S. Cosmetics Ingredient Review or the EU Scientific Committee on Consumer Safety (SCCS). The data from these programs were considered sufficiently reviewed as to not require further evaluation. Data reported as part of other equivalent peer reviewed risk assessment programs (e.g., HHRA (www.HHRAproject.com/); USEPA HPV Chemical Challenge Program) were also considered in a similar fashion, although a certain level of expert judgment was required to evaluate the quality of these programs. Toxicity information was also obtained via the ECHA CHEM database. This database provides electronic public access to information on chemical substances manufactured or imported in Europe. The information originates from the registration dossiers submitted by companies to ECHA in the framework of REACH Regulation. If no data were available from the above sources or the available data were considered insufficient for the determination of oral RfDs and drinking water guideline values, then toxicity information on MSDSs were used, as well as read-across from available experimental data on a structurally related substances, and predicted values from QSAR models. With exception to the USEPA integrated risk information system (IRIS) values, the most appropriate or reliable NOAEL or LOAEL, if a NOAEL was unavailable, and the critical effect were identified.

3.4.2 Summary of Toxicity Reviews

Quantitative toxicity values identified/derived and the drinking water guidelines identified/derived for site related COPC were adopted in accordance with guidance available from enHealth (2012), ADWG (2016) and NEPM (2013). **Appendix D** provides further toxicity information on each of the COPC. **Table 6** provides a summary of the quantitative toxicity values for oral and dermal exposures and the drinking water criteria for the COPC identified.

Table 6 Toxicity Data for Chronic Oral/Dermal Exposures and Drinking Water Screening Criteria for COPC

CAS	Chemical	Non-Threshold Slope Factor (mg/kg/day)$^{-1}$	Threshold Chronic TDI (mg/kg/day)		Dermal Permeability (cm/hr)	Drinking Water Guideline (mg/L)	
COPC in Hydraulic Fracturing Fluid Injected into Well							
57-13-6	Urea	Low chronic toxicity					
64-02-8	Tetrasodium ethylenediaminetetraacetate	--	0.190	A	2.40E-14	0.25 (health as EDTA)	A
67-48-1	2-hydroxy-N,N,N-trimethylethanaminium chloride	--	71	D	--	248	D
67-63-0	Propan-2-ol	--	4	D	7.78E-04	16.38	D
79-06-1	2-Propenamid (acrylamide)	--	0.0002	A	--	0.0002 (health)	A
107-21-1	Ethylene Glycol	--	1.50	D	9.00E-05	0.59	D
111-46-6	2,2"-oxydiethanol (diethylene glycol)	--	0.3	D	4.17E-05	1.17	D
540-97-6	Dodecamethylcyclohexasiloxane	--	0.1	D	--	0.39	D
541-02-6	Decamethyl cyclopentasiloxane	--	0.1	D	--	0.39	D
556-67-2	Octamethylcyclotetrasiloxane	--	0.1	D	--	0.39	D
1310-73-2	Sodium hydroxide (caustic soda)	Acute toxicity only (irritant and corrosive), not systemically available in body					
2682-20-4	2-methyl-2h-isothiazol- 3-one (Methylisothiazolinone)	--	0.020	D	1.00E-04	0.078	D
7447-40-7	Potassium chloride	--	18.20	D	--	71	D
7631-86-9	Non-crystalline silica	Not toxic via oral exposure as not absorbed via GI tract					
7647-01-0	Hydrochloric Acid	Acute toxicity only					
7647-14-5	Sodium chloride	Low chronic toxicity					

CAS	Chemical	Non-Threshold Slope Factor $(mg/kg/day)^{-1}$	Threshold Chronic TDI (mg/kg/day)		Dermal Permeability (cm/hr)	Drinking Water Guideline (mg/L)	
7757-82-6	Sodium sulfate	Low chronic toxicity					
7758-98-7	Copper(II) sulfate		0.5	A	--	2 (health as copper)	A
7783-20-2	Ammonium sulfate	Low chronic toxicity					
7786-30-3	Magnesium chloride	Low chronic toxicity					
9000-30-0	Guar gum		12.5	D	--	49	D
10043-52-4	Calcium chloride	No toxic effects shown in available studies					
10377-60-3	Magnesium nitrate		14.3	A	9.30E-05	50 (health as nitrate)	A
14464-46-1	Cristobalite	Not toxic via oral exposure as not absorbed via GI tract					
26172-55-4	5-chloro-2-methyl-2h-isothiazolol-3-one (Methylchloroisothiazolinone)		0.020	D	1.40E-04	0.078	D
31726-34-8	Polyethylene glycol monohexyl ether		0.5	D		1.95	D
38193-60-1	Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer	Low chronic toxicity					
61789-77-3	Dicoco dimethyl quaternary ammonium chloride		0.1	D	--	0.39	D
63148-62-9	Dimethyl siloxanes and silicones		1.5	J	--	5.85	J
67762-90-7	Siloxanes and silicones, dimethyl, reaction products with silica		1.5	J	--	5.85	J
91053-39-3	Diatomaceous earth, calcined	Not toxic via oral exposure as not absorbed via GI tract					
129898-01-7	2-Propenoic acid, polymer with sodium phosphinate	Low chronic toxicity					
136793-29-8	Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate	Low chronic toxicity					

CAS	Chemical	Non-Threshold Slope Factor (mg/kg/day)$^{-1}$		Threshold Chronic TDI (mg/kg/day)		Dermal Permeability (cm/hr)	Drinking Water Guideline (mg/L)	
110-17-8	Fumaric acid			6.00	J	--	23.4	J
1319-33-1	Boronatrocalcite			0.130	A	1.00E-03	3.5	A (boron)
1330-43-4	Sodium tetraborate			0.130	A	1.00E-03	3.5	A (boron)
7704-73-6	Monosodium fumarate			6.00	J	--	23.4	J
7789-38-0	Sodium bromate			0.003	A	--	0.02 (health as bromate)	A
10043-35-3	Boric acid			0.130	A	1.00E-03	3.5	A (boron)
595585-15-2	Diutan			1.00	D	--	3.9	D
COPC identified in Flowback Water Analysis								
7440-38-2	Arsenic	--		0.002	N	1.00E-03	0.01	A
7440-42-8	Boron	--		0.2	N	1.00E-03	4	A
7439-96-5	Manganese	--		0.14	N	1.00E-03	0.5	A
7440-02-0	Nickel	--		0.012	N	2.00E-04	0.02	A
7440-39-3	Barium	--		0.2	U	1.00E-03	2	A
7440-48-4	Cobalt	--		0.0014	N	4.00E-04	0.0006	U
	TPH C10-C16 Aromatic	--		0.04	T	6.94E-02	0.1	W
	TPH C10-C16 Aliphatic	--		0.1	T	6.94E-02	0.3	W
	TPH C16+ Aromatic	--		0.03	T	3.24E-01	0.09	W
	TPH C16+ Aliphatic	--		2	T	3.24E-01	0.3	W
71-43-2	Benzene	3.50E-02	W	0.004	U	1.49E-02	0.001	A

Notes: D – Derived; A – ADWG 2016; W – WHO 2011; J-JECFA; ; N – NEPM 2013; T – TPHCWG; --- Not available

3.5 Human Health Risk Characterisation

3.5.1 Risks to Human Health

Risk characterisation is the final step in a quantitative risk assessment. It involves the incorporation of the exposure assessment and toxicity assessment to provide a quantitative assessment of non-threshold carcinogenic risk and threshold risk. In the assessment presented, evaluation of exposures to the COPC involves an assessment of threshold and non-threshold risks.

The calculation of risks has been undertaken using an in-house spreadsheet model, RiskE (version 2011). The equations utilised within RiskE follow risk assessment methodology as outlined in Section 6.1 following protocols established by ANZECC, NHMRC, NEPM and CRC CARE. The output from this model has been incorporated into the tables presented, and calculation sheets are presented in **Appendix C**.

3.5.1.1 Hazard Index for Threshold Effects

The potential for adverse threshold effects, resulting from exposure to an individual COPC, has been evaluated by comparing an exposure level, expressed as a daily chemical intake, with the ADI/TDI or RfD. The resulting ratio is referred to as the hazard quotient, and is derived in the following manner for oral/dermal and inhalation exposures:

$$Hazard\ Quotient\ (oral\ or\ dermal) = \frac{Daily\ Chemical\ Intake}{(ADI, TDI, RfD - Background)}$$

Where

ADI = acceptable daily intake (mg/kg/day)
TDI = tolerable daily intake (mg/kg/day)
RfD = Reference dose (mg/kg/day)

If the daily chemical intake for the individual COPC exceeds the ADI, TDI or RfD (i.e. if the hazard quotient exceeds one), then this would indicate potentially unacceptable chemical intakes. The hazard quotient does not represent a statistical probability of an effect occurring.

To assess the overall potential for adverse health effects posed by simultaneous exposure to multiple chemicals, the hazard quotients for each chemical and exposure pathway have been summed. The resulting sum is referred to as the hazard index (HI). The HI approach assumes that multiple sub-threshold exposures to several chemicals could result in a cumulative adverse health effect, and exposures are summed over all intake routes.

Acceptable Risk

An “acceptable” risk in this assessment has been defined as a Hazard Index of 1 (as per risk assessment industry practice, supported by protocols outlined in NEPM (2013) and USEPA guidance.

If the Hazard Index is less than one, cumulative exposure to the site chemicals is judged unlikely to result in an adverse effect. If the index is greater than one, a more detailed and critical evaluation of the risks (including consideration of specific target organs affected and mechanisms of toxic action of the chemicals of concern) would be required to ascertain if the cumulative exposure would, in fact, be likely to harm exposed individuals. All hazard quotient and hazard index calculations are presented in **Appendix C**.

3.5.1.2 Non-Threshold Carcinogenic Risk

The potential for unacceptable non-threshold carcinogenic risks associated with exposure to COPC have been evaluated using US EPA (1989 and 2009) methodology.

Non-threshold carcinogenic risks are estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to a potential non-threshold carcinogen. The numerical estimate of excess lifetime cancer risk is calculated as follows for oral/dermal and inhalation exposures:

$$Carcinogenic\ Risk\ (oral\ or\ dermal) = Daily\ Chemical\ Intake\ x\ Cancer\ Slope\ Factor$$

The total non-threshold carcinogenic risk is the sum of the risk for each chemical for each pathway.

Australian guidance related to the significance of non-threshold cancer risk estimates currently specifies that risks of less than 10^{-5} per lifetime is generally accepted as indicating conditions that do not warrant specific management or remedial action.

Acceptable Risk

The adopted acceptable risk is defined as a risk of less than 1×10^{-5} incremental lifetime risk of cancer.

3.5.2 Summary of Risk

A summary of the calculated risks for the identified receptor group (trespassers) that are relevant to the detailed assessment of potential exposure to COPCs on-site, based on the available data is presented **Table 7** and **Table 8**.

Table 7 Risk associated with potential exposure to trespassers – Amungee NW-1H Stage 1 Slickwater and Gel Flush (theoretical and empirical data)

Receptor and Pathway	Non-Threshold Carcinogenic Risk	Threshold Hazard Index
	100% Mass Return	
Amungee NW-1H Stage 1 Slickwater and Gel Flush - COPC associated with theoretical data		
Adult Trespassers		
Ingestion of chemicals via incidental contact with flowback water	NA	0.0031
Dermal exposure to chemicals via incidental contact with flowback water	NA	0.000038
Total Risk	**NA**	**0.003**
Child Trespassers		
Ingestion of chemicals via incidental contact with flowback water	NA	0.0061
Dermal exposure to chemicals via incidental contact with flowback water	NA	0.000061
Total Risk	**NA**	**0.006**
Amungee NW-1H Stage 1 Slickwater and Gel Flush - COPC associated with empirical data		
Adult Trespassers		
Ingestion of chemicals via incidental contact with flowback water	1.8E-12	0.0011
Dermal exposure to chemicals via incidental contact with flowback water	1.0E-11	0.0027
Total Risk	**1E-11**	**0.004**
Child Trespassers		
Ingestion of chemicals via incidental contact with flowback water	3.5E-12	0.0021
Dermal exposure to chemicals via incidental contact with flowback water	1.7E-11	0.0043
Total Risk	**2E-11**	**0.006**

Notes:
Risk values have been rounded to two significant figures with totals rounded to one significant figure; hence, the sum of individual risks may not add up exactly to the total presented.
NA = Not Assessed as there are no non-threshold COPC in the media of concern

The following can be noted from the table above:

- The calculated risks associated with potential exposure to COPC identified in flowback water, where Slickwater and Gel Flush is used and assuming 100% mass recovery are below the targets of 1×10^{-5} and 1, respectively. Hence, chronic health risks are considered to be low and acceptable.

- The calculated risk to trespassers associated with exposure to COPC that may be present in flowback water, as determined from empirical data obtained from existing operations, is below the targets of 1 x 10^{-5} and 1, respectively. Hence, chronic health risks are considered to be low and acceptable.

Table 8 Risk associated with potential exposure to trespassers – Amungee NW-1H Stage 7 Cross-Linked Gel/Slickwater Hybrid (theoretical and empirical data)

Receptor and Pathway	Non-Threshold Carcinogenic Risk	Threshold Hazard Index
	100% Mass Return	
Amungee NW-1H Stage 7 Cross-Linked Gel/Slickwater Hybrid - COPC associated with theoretical data		
Adult Trespassers		
Ingestion of chemicals via incidental contact with flowback water	NA	0.097
Dermal exposure to chemicals via incidental contact with flowback water	NA	0.0027
Total Risk	**NA**	**0.1**
Child Trespassers		
Ingestion of chemicals via incidental contact with flowback water	NA	0.19
Dermal exposure to chemicals via incidental contact with flowback water	NA	0.0043
Total Risk	**NA**	**0.2**
Amungee NW-1H Stage 7 Cross-Linked Gel/Slickwater Hybrid - COPC associated with empirical data		
Adult Trespassers		
Ingestion of chemicals via incidental contact with flowback water	1.8E-12	0.0011
Dermal exposure to chemicals via incidental contact with flowback water	1.0E-11	0.0027
Total Risk	**1E-11**	**0.004**
Child Trespassers		
Ingestion of chemicals via incidental contact with flowback water	3.5E-12	0.0021
Dermal exposure to chemicals via incidental contact with flowback water	1.7E-11	0.0043
Total Risk	**2E-11**	**0.006**

Notes:
Risk values have been rounded to two significant figures with totals rounded to one significant figure; hence, the sum of individual risks may not add up exactly to the total presented.
NA = Not Assessed as there are no non-threshold COPC in the media of concern

The following can be noted from the table above:

- The calculated risks associated with potential exposure to COPC identified in flowback water, where Cross-Linked Gel/Slickwater Hybrid is used and assuming 100% mass recovery are below the targets of 1 x 10^{-5} and 1, respectively. Hence, chronic health risks are considered to be low and acceptable.
- The calculated risk to trespassers associated with exposure to COPC that may be present in flowback water, as determined from empirical data obtained from existing operations, is below the targets of 1 x 10^{-5} and 1, respectively. Hence, chronic health risks are considered to be low and acceptable.

3.6 Conclusions

The quantification of potential risks to human health associated with the use of chemicals in well simulation activities conducted by Origin within the project area has involved the assessment of potential exposures to compounds used or formed in the simulation activities. Based on the conceptual site model, operational controls, security and management practices implemented by Origin, the only potentially complete exposure pathway identified and assessed in the risk assessment was the incidental ingestion of and dermal contact with flowback fluids by trespassers at the flowback fluid Flexiponds.

On the basis of the conservative assessment undertaken, it can be concluded that there are no unacceptable potential health risks to trespassers in relation to potential exposures to flowback water from well hydraulic fracturing activities. Further consideration of the uncertainties associated with the risk assessment is presented in **Section 5.2.1**.

Effective operational controls and management implemented by Origin further minimises potential exposures to trespassers. These operational controls include:

- Fencing and signage around the Flexiponds
- Routine operational and security patrols to prevent trespassing

4.0 Environmental Risk Assessment

The objective of undertaking an Environmental Risk Assessment (ERA) is to investigate the potential risks to the environment associated with the concentrations of chemicals of potential environmental concern (COPEC) that may be present in the flowback water (in the event there is a leak or spill from aboveground infrastructure).

With regards to the assessment of potential environmental risks, the requirements of the Amended ASC NEPM Schedule B5a, Guideline on Ecological Risk Assessment (NEPC 2013) were considered.

Similar to the HHRA, the ERA consists of the four basic components:

- Issue Identification/Problem Formulation:
  - Identification of COPECs;
  - Identification of receptors of concern; and
  - Identification of operable exposure pathways warranting quantification of risk.
- Toxicity Assessment:
  - Present the doses or concentrations of COPECs below which risks are assumed to be negligible or low (toxicological reference values, TRVs);
- Exposure Assessment:
  - Determination of exposure concentrations for the site's various receptors (i.e. concentrations used to quantify risks); and
  - Estimation of contaminant intake (dose entering body, mg/kg-day) for operable exposure pathways.
- Risk Characterisation:
  - Quantify risks as magnitude of exceedance of the TRVs (i.e. hazard quotients, dose divided by TRV) and derivation of species sensitivity distributions.

It is noted that the ERA approach presented in this report provides a methodology for assessment of the risks posed by mixtures that meet the requirement in the EA consent conditions. This approach evaluates the toxicity of the individual substances and characterises the cumulative risks of the total effluent toxicity and ecotoxicity, which is consistent with the National Water Quality Management Strategy (NWQMS) and the principles outlined in the ANZECC & ARMCANZ (2000) guidelines. The NEPC (2013) also addresses the mixture of constituents and has adopted the hazard quotient approach for the Guideline for ERA.

Flowback water from the hydraulic fracturing process potentially contains hydraulic fracturing chemicals and chemical constituents naturally present within the shale formations. Similar to the human health risk assessment approach, the estimated exposure to the flowback water was based on a theoretical and empirical EPC that was developed either from knowledge of the chemicals and quantities used in the hydraulic fracturing process, or the results of laboratory analysis of return water from existing operations.

4.1 Persistence, Bioaccumulation and Environmental Toxicity Assessment

This semi qualitative assessment is an evaluation of the environmental hazard of the constituents of the hydraulic fracturing fluid based on their persistence (P), bioaccumulation (B) and toxicity (T). The PBT assessment has been conducted according to guidance developed by the Australian Government Department of Health National Industrial Chemicals Notification and Assessment Scheme (NICNAS). The criteria considered are presented in **Table 9**.

Table 9 PBT criteria adopted by NICNAS

Criterion	PBT criteria		
P	For PBT purposes a chemical is considered persistent in a particular media if its half-life in the media exceeds the following: Half-life ($T_{1/2}$) >2 months in water Half-life ($T_{1/2}$) >6 months in soil Half-life ($T_{1/2}$) >6 months in sediment Half-life ($T_{1/2}$) >2 days in air		
B	For PBT purposes a chemical is considered to be bioaccumulative if it has a BCF/BAF of >2000, or in its absence of any BCF/BAF measurement a log K_{ow} >4.2.		
T	For PBT purposes, in respect of aquatic toxicity, a chemical may be considered toxic under the following circumstances (corresponding to criteria for GHS chronic category 1:		
	Non-rapidly degradable substances for which there are adequate chronic toxicity data available	Chronic NOEC or EC_x (for fish)	<0.1 mg/L and/or
		Chronic NOEC or EC_x (for crustacea)	<0.1 mg/L and/or
		Chronic NOEC or EC_x (for algae or other aquatic plants)	<0.1 mg/L
	Rapidly degradable substances for which adequate chronic toxicity data are available	Chronic NOEC or EC_x (for fish)	<0.01 mg/L and/or
		Chronic NOEC or EC_x (for crustacea)	<0.01 mg/L and/or
		Chronic NOEC or EC_x (for algae or other aquatic plants)	<0.01 mg/L
	Substances for which adequate chronic toxicity data are not available (providing criteria for P and B are met)	96 h LC_{50} (for fish)	<1 mg/L and/or
		48 h EC_{50} (for crustacea)	<1 mg/L and/or
		72 or 96 ErC_{50} (for algae or other aquatic plants)	<1 mg/L
		And the substance is not rapidly degradable and/or the experimentally determined BCF is >500 (or, if absent, the log K_{ow} is >4.2	
	Toxicity to other (terrestrial) organisms	Should be considered on a case by cases basis, compared with the highly toxic classifications DotE has developed for ag/vet chemicals	
	Long term toxicity or evidence such as endocrine disruption effects	Should be considered on a case-by-case basis.	

The screening criteria developed by the EU for REACH (ECHA, 2008) can also be used as surrogate information to decide whether a substance may potentially fulfil the PBT criteria. A summary of these screening criteria is provided in **Table 10**.

Table 10 EU REACH screening criteria for persistent (P), very persistent (vP), bio-accumulative (B), very bio-accumulative (vB) and toxicity (T)

Type of Data	Criterion	Screening Assignment
Persistence		
Ready biodegradability test	Readily biodegradable	Not P and not vP
Enhanced ready biodegradability test	Readily biodegradable	Not P and not vP
Specified tests on inherent biodegradability		
Zahn-Wellens (OECD 302B)	>70% mineralization (DOC removal) within 7 days; log phase no longer than 3 days; removal before degradation occurs below 15%; no pre-adapted	Not P

Type of Data	Criterion	Screening Assignment
	inoculum	
MITI II test (OECD 302C)	>70% mineralization (O_2 uptake) within 14 days; log phase no longer than 3 days; no pre-adapted inoculum	Not P
Biowin 2 (non-linear model prediction) and Biowin 3 (ultimate biodegradation time) or	Does not biodegrade fast (probability) and ultimate biodegradation timeframe prediction: >months (value <2.2) Or	P
Biowin 6 (MITI non-linear model prediction) and Biowin 3 (Ultimate biodegradation time)	Does not biodegrade fast (probability <0.5) and ultimate biodegradation timeframe prediction: >months (value <2.2)	P
Bioaccumulation		
Convincing evidence that a substance can biomagnify in the food chain (e.g., field data).	e.g., BMF >1	B or vB, definitive assignment possible
Octanol-water partition coefficient (experimentally derived or estimated by valid QSAR)	Log K_{ow} < 4.5	Not B and not vB
Toxicity		
Short-term toxicity aquatic toxicity (algae, daphnia, fish)	EC_{50} or LC_{50} <0.01 mg/L	T, criterion considered to be definitely fulfilled
Short-term toxicity aquatic toxicity (algae, daphnia, fish)	EC_{50} or LC_{50} <0.1 mg/L	T
Avian toxicity (subchronic or chronic toxicity or toxic for reproduction)	NOEC <30 mg/kilogram(kg) food	T

On the basis of this approach, the chemicals proposed to be used in hydraulic fracturing fluids have been evaluated, with the outcomes of the assessment conducted summarised in **Table 11** and in individual toxicity profiles in **Appendix D**.

Table 11 PBT Assessment of the Hydraulic Fracturing Chemicals Used in the Project Area

Substance	P/vP Criteria Fulfilled	B/vB Criteria Fulfilled	T Criteria Fulfilled?	Overall Conclusion
Aluminium sulfate	No. Not applicable, inorganic salt. Biodegradation is not applicable to aluminium sulfate.	No. Aluminium sulfate is not expected to bioaccumulate.	No. The chronic NOEC value in fish for aluminium is <0.1 mg/L; thus, the dissolved aluminium from aluminium sulfate meets the screening criteria for toxicity	Not PBT
Calcium chloride	No. Not applicable, inorganic salt, ionic species ubiquitous in environment.	No. Not applicable, inorganic salt, ionic species ubiquitous in environment.	No chronic toxicity data exist on calcium chloride; however, the acute EC (L) 50s are >0.1 mg/L in fish, invertebrates and algae. Thus, calcium chloride does not meet the screening criteria for toxicity.	Not PBT
Cristobite, silica and diatomaceous earth	No. Not applicable, inorganic substance, ubiquitous in environment.	No. Not applicable, inorganic substance, ubiquitous in environment.	No. Long term data not available (acute data >0.1 mg/L).	Not PBT
Guar gum	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	The molecular weight of guar gum is high ranging 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	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	Not PBT
Hydrochloric acid	No. 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.	Hydrogen and chloride ions are essential to all living organisms and their intracellular and extracellular concentrations are actively regulated. Thus, hydrochloric acid is not expected to bioaccumulate.	No chronic toxicity data exist on hydrochloric acid; 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.	Not PBT
Magnesium chloride	Magnesium chloride is an organic salt that dissociates completely to magnesium and chloride ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions; both magnesium and chloride ions are also ubiquitous and are present in most water, soil and sediment. Thus, the persistent	Magnesium and chloride ions are essential to all living organisms and their intracellular and extracellular concentrations are actively regulated. Thus, magnesium chloride is not expected to bioaccumulate.	No chronic toxicity data exist on magnesium chloride; however, the acute EC(L)50s are >0.1 mg/L in fish, invertebrates and algae. Thus, magnesium chloride does not meet the adopted screening criteria for toxicity.	Not PBT

Substance	P/vP Criteria Fulfilled	B/vB Criteria Fulfilled	T Criteria Fulfilled?	Overall Conclusion
	criterion is not considered applicable to this inorganic salt.			
Magnesium nitrate	Magnesium nitrate is an organic salt that dissociates completely to magnesium and nitrate ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions; both magnesium and nitrate ions are also ubiquitous and are present in most water, soil and sediment. Thus, the persistent criterion is not considered applicable to this inorganic salt	Magnesium ions are essential to all living organisms and its intracellular and extracellular concentrations are actively regulated. Nitrates are ions that are very soluble in aqueous solutions. Thus, magnesium nitrate is not expected to bioaccumulate.	No chronic toxicity data exist on magnesium nitrate; however, the acute EC(L)50s are >0.1 mg/L in fish, invertebrates and algae. Thus, magnesium nitrate does not meet the screening criteria for toxicity.	Not PBT
Methylisothiazolinone (MIT) and Methylchloroisothiazolinone (CMIT)	No. The half-lives of CMIT and MIT in a river water-sediment system were 17.3 hours and 9.1 hours, respectively. Thus, CMIT/MIT do not meet the criteria for persistence	No. The experimental BCF for CMIT is 67-114 in bluefish sunfish, and the BCF for MIT was determined to be 2.3. Thus, CMIT/MIT do not meet the criteria for bioaccumulation.	No chronic toxicity data are available for CMIT/MIT; however, the acute E(L)C50 values for algae are <0.1 mg/L. Thus, CMIT/MIT meet the screening criteria for toxicity.	Not P or B. Potentially T.
Non crystalline silica	No. Not applicable, inorganic substance, ubiquitous in environment.	No. Not applicable, inorganic substance, ubiquitous in environment.	No. Chronic toxicity data not available. Acute data >0.1 mg/L in fish, invertebrates and algae, hence does not meet the screening criteria for toxicity.	Not PBT
Potassium chloride	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.	Potassium and chloride ions are essential to all living organisms. Thus, potassium chloride is not expected to bioaccumulate.	No chronic toxicity data exist on potassium chloride; however, the acute EC(L)50s are >0.1 mg/L in fish, invertebrates and algae. Thus, potassium chloride does not meet the screening criteria for toxicity	Not PBT
Sodium chloride	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	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.	No chronic toxicity data exist on sodium chloride; the acute EC(L)50s are expected to be >0.1 mg/L in fish, invertebrates and algae based on aquatic toxicity studies involving sodium and	Not PBT

Substance	P/vP Criteria Fulfilled	B/vB Criteria Fulfilled	T Criteria Fulfilled?	Overall Conclusion
	present in most water, soil and sediment. The persistent criteria is not considered applicable to this inorganic salt.		chloride ions. Thus, sodium chloride does not meet the screening criteria for toxicity.	
Sodium hydroxide	Not applicable (inorganic salt, ionic species ubiquitous in environment)	Not applicable. Biodegradation is not applicable to these inorganic ions; sodium and hydroxide ions are ubiquitous and are present in most water, soil and sediment.	Not applicable. Chronic toxicity data not available (acute data >0.1 mg/L in fish, invertebrates and algae), thus sodium hydroxide does not meet the screening criteria for toxicity.	Not PBT
Ethylene Glycol	Ethylene glycol is readily biodegradable both aerobically and anaerobically and as such not persistent in the environment.	Based on the measured log Kow of -1.36 and a measured BCF of 10, Ethylene glycol is not bioaccumulative.	The acute aquatic toxicity of Ethylene glycol is > 0.01 mg/L. Hence the substance does not fulfil the screening criteria for toxicity.	Not PBT
Fumaric acid	Fumaric acid is readily biodegradable and as such not persistent in the environment.	Based on the measured log Kow of <3 Fumaric acid is not bioaccumulative.	The acute aquatic toxicity of Fumaric acid is > 0.01 mg/L. Hence the substance does not fulfil the screening criteria for toxicity.	Not PBT
Boronatrocalcite (Boric acid and Boron as surrogate)	For the purposes of this PBT assessment, the persistent criterion is not considered applicable to this inorganic substance.	For the purposes of this PBT assessment, the bioaccumulation criterion is not considered applicable to this inorganic substance.	For the purposes of this PBT assessment, the toxicity criterion is not considered applicable to this inorganic substance.	Not PBT
Sodium tetraborate (Boric acid and Boron as surrogate)	For the purposes of this PBT assessment, the persistent criterion is not considered applicable to this inorganic substance.	For the purposes of this PBT assessment, the bioaccumulation criterion is not considered applicable to this inorganic substance.	For the purposes of this PBT assessment, the toxicity criterion is not considered applicable to this inorganic substance.	Not PBT
Monosodium fumarate (Fumaric acid as surrogate)	Monosodium fumarate is readily biodegradable and as such not persistent in the environment.	Based on the measured log Kow of <3 Monosodium fumarate is not bioaccumulative.	The acute aquatic toxicity of Monosodium fumarate is > 0.01 mg/L. Hence the substance does not fulfil the screening criteria for toxicity.	Not PBT
Boric acid	For the purposes of this PBT assessment, the persistent criterion is not considered applicable to this inorganic substance.	For the purposes of this PBT assessment, the bioaccumulation criterion is not considered applicable to this inorganic substance.	For the purposes of this PBT assessment, the toxicity criterion is not considered applicable to this inorganic substance.	Not PBT

Substance	P/vP Criteria Fulfilled	B/vB Criteria Fulfilled	T Criteria Fulfilled?	Overall Conclusion
Diutan	Diutan expected to readily biodegrade. Thus, it is not expected to meet the screening criteria for persistence	The molecular weight of Diutan is > 1,000,000 g/mol, and it is also water soluble. Thus, Diutan is not expected to meet the criteria for bioaccumulation	The acute aquatic toxicity of Diutan is >0.1 mg/L. Thus, Diutan is not expected to meet the screening criteria for toxicity	Not PBT
Urea	Urea is biodegradable and as such not persistent in the environment.	Based on the measured log Kow of -1.59, Urea is not bioaccumulative.	The acute aquatic toxicity of Urea is > 0.01 mg/L. Hence the substance does not fulfil the screening criteria for toxic (T)	Not PBT
Tetrasodium ethylenediaminetetraacetates (EDTA)	EDTAs are not readily biodegradable and as such are persistent in the environment.	EDTAs have a low potential for bioaccumulation	The acute aquatic toxicity of EDTAs are > 0.01 mg/L. Hence the substances do not fulfil the screening criteria for toxic (T)	Potentially P. Not B. Not T.
2-hydroxy-N,N,N-trimethylethanaminium chloride (Choline Chloride)	Choline chloride is readily biodegradable and thus it does not meet the screening criteria for persistence.	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.	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.	Not PBT
Propan-2-ol (Isopropanol)	Isopropanol is readily biodegradable and thus it does not meet the screening criteria for persistence.	Based on a measured log Kow of 0.05 and a calculated BCF of 1, isopropanol does not meet the screening criteria for bioaccumulation..	The chronic toxicity data on isopropanol show NOECs of >0.01 mg/L. Thus, isopropanol does not meet the screening criteria for toxicity.	Not PBT
2-Propenamid (acrylamide)	Acrylamide is readily biodegradable and as such not persistent in the environment.	Based on the measured log Kow of -1.0 and an estimated BCFs of 0.26 - 2.53, Acrylamide is not bioaccumulative.	The acute aquatic toxicity of Acrylamide is > 0.01 mg/L. Hence the substance does not fulfil the screening criteria for toxic (T)	Not PBT
2,2''-oxydiethanol (diethylene glycol DEG)	DEG is readily biodegradable and as such not persistent in the environment.	An estimated BCF of 3 suggests the potential for bioconcentration in aquatic organisms is low	The acute aquatic toxicity of DEG is > 0.01 mg/L. Hence the substance does not fulfil the screening criteria for toxic (T)	Not PBT
Dodecamethylcyclohexasiloxane (D6),Decamethylcyclopentasiloxane (D5), Octamethylcyclotetrasiloxane (D4)	Cyclicdimethylpolysiloxane compounds are not readily biodegradable and are persistent in the environment.	These compounds are bioaccumulative, based on a log Kow >3.	The chronic aquatic toxicity of D4 is < 0.1 mg/L. Hence the substance does fulfil the screening criteria for toxic (T)	Potentially P. Potentially B. Potentially T.
Sodium sulfate	Sodium sulphate is an organic salt that dissociates completely to sodium and sulphate ions in aqueous solutions. The persistent	The BCF of sodium sulfate is very low (0.5) and therefore bioconcentration is not expected.	The acute aquatic toxicity of sodium sulfate is > 0.01 mg/L. Hence the substance does not fulfil the screening	Not PBT

Substance	P/vP Criteria Fulfilled	B/vB Criteria Fulfilled	T Criteria Fulfilled?	Overall Conclusion
	criterion is not considered applicable to this inorganic salt.		criteria for toxic (T)	
Copper(II) sulfate	Not applicable, inorganic substance, ubiquitous in environment.	The bioaccumulative criterion is not considered applicable to inorganic copper compounds.	The acute aquatic toxicity of copper sulfate is < 0.01 mg/L. Hence the substance is considered toxic to aquatic organisms.	Not P. Not B. Potentially T
Polyethylene glycol monohexyl ether	Polyethylene glycol monohexyl ether is readily biodegradable and as such not persistent in the environment	Based on a log Kow value greater than 3, and a maximum BCF value of under 800., Polyethylene glycol monohexyl ether is not bioaccumulative.	The acute aquatic toxicity of Polyethylene glycol monohexyl ether is > 0.01 mg/L. Hence the substance does not fulfil the screening criteria for toxic (T)	Not PBT
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer and Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate	These polymers are not readily biodegradable; hence they meet the screening criteria for persistence.	These polymers are expected to have a very high molecular weight and poor water solubility. They are not expected to be bioavailable; hence these polymers do not meet the criteria for bioaccumulation.	There are no aquatic toxicity studies on these polymers. They are expected to have low concern for aquatic toxicity because of their very high molecular weight and poor water solubility. As such, the polymers do not meet the criteria for toxicity.	Potentially P. Not B. Not T.
Dicoco dimethyl quaternary ammonium chloride	Dicoco dimethyl quaternary ammonium chloride is readily biodegradable, and as such not persistent in the environment	Based on the available measured bioconcentration data, Dicoco dimethyl quaternary ammonium chloride is Not Bioaccumulative.	Based on available acute ecotoxicity values below 1 mg/L and/or chronic ecotoxicity values below 0.1 mg/L, Dicoco dimethyl quaternary ammonium chloride is categorised as Toxic.	Not P. Not B. Potentially T.
Dimethyl siloxanes and silicones	Dimethyl siloxanes and silicones are not readily biodegradable and as such are persistent in the environment.	Due to their molecular size, Dimethyl siloxanes and silicones are not bioaccumulative.	The acute aquatic toxicity of Dimethyl siloxanes and silicones are > 0.01 mg/L. Hence the substance does not fulfil the screening criteria for toxic (T)	Potentially P. Not B. Not T.
2-Propenoic acid, polymer with sodium phosphinate	The polymer is not readily biodegradable, hence it meets the screening criteria for persistence	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.	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 criteria for toxicity.	Potentially P. Not B. Not T.

Substance	P/vP Criteria Fulfilled	B/vB Criteria Fulfilled	T Criteria Fulfilled?	Overall Conclusion
Sodium bromate	Not applicable. Sodium bromate is an inorganic substance which dissociates to bromate ion which is ubiquitous in environment.	Sodium bromate is not bioaccumulative based on published information and experimental evidence for metabolic transformation.	The acute aquatic toxicity of sodium bromate is > 0.01 mg/L. Hence the substance does not fulfil the screening criteria for toxic (T)	Not PBT

Shaded cells indicate substances that meet the screening criteria for persistence, bioaccumulation or aquatic toxicity.
For chemical and physical properties not readily available from MSDS or the Hazardous Substances Databank (HSDB), modelled data was obtained from USEPA (2009) EPISUITE™.

As the **Table 11** indicates, most of the compounds (with the exception of MIT, CMIT, tetrasodium EDTA, cyclicdimethylpolysiloxane compounds, acrylamide polymers, dimethyl siloxanes and silicones, copper sulfate and dicoco dimethyl quaternary ammonium chloride) utilised in hydraulic fracturing fluids are not considered to be persistent, bioaccumulative or toxic in the environment. If these compounds are present in flowback water, they will readily degrade or dissociate in the environment and will not bioaccumulate in terrestrial or aquatic species. Polymers, EDTA, siloxanes and silicones have limited biodegradation potential; however are not bioaccumulative and are essentially non-toxic to aquatic species. While MIT and CMIT, copper sulfate and dicoco dimethyl quaternary ammonium chloride meet the screening criteria for toxicity, qualified by their acute toxicity capacity, adverse chronic outcomes are not evident based on their reactivity and lack of persistence and bioaccumulation in the environment.

Cyclicdimethylpolysiloxane compounds meet the criteria for being persistent, bioaccumulative and toxic in the environment. They are considered poorly soluble in water, volatile and will adsorb strongly to organic matter in sediment and soil. It is not expected that the PBT properties of cyclicdimethylpolysiloxane compounds would be realised in the flowback water. A quantitative evaluation of the environmental risk posed by the COPCs above is presented in **Section 4.4**.

4.2 Exposure Assessment

The exposure setting provides important information regarding the current site use and physical attributes of the site. The information herein will form the basis for determining applicable exposure pathways between sources of contamination and receptors of concern (assessment endpoints). The objective of the environmental risk assessment is to assess the typical potential exposures that may occur in any of the operational areas in the CSG fields. As such, the exposure assumptions for the environmental receptors were based on a typical operational layout and adjacent land use that includes pastoral land and a flowing surface water body.

For the purpose of this ERA, the study area is divided into the operational area and the offsite pastoral land.

The operational area is considered a highly modified system (i.e. the site has been extensively cleared, with gravel and compacted soil covering the well pad and around infrastructure) that has minimal wildlife habitat to support a functioning aquatic or terrestrial ecosystem including foraging and breeding habitat. In addition, the operational controls, security and management practices implemented by Origin, such as the installation and maintenance of fences around the operational site, would limit the exposure of environmental receptors to the hydraulic fracturing fluids considered in this assessment. It is noted that there may be occasional incursions of wildlife into the operational area. However, as the flowback water will be contained inside Flexiponds (with 2 m high walls), there would be minimal opportunity for environmental receptors to come into contact with the with the flowback water.

The adjacent areas comprise of unimproved pastures and remnant native vegetation (woodlands), both of which are used for cattle grazing purposes. A range of surface water systems that include streams, creeks dams and rivers also exist nearby. Despite that fact that the clearing and farming practices has diminished the overall habitat quality, the adjacent areas are still suitable to support indigenous flora and fauna.

4.2.1 Potential Exposure Pathways and Receptors

Environmental surveys were conducted for Origin that identified flora and fauna that may inhabit the local and regional environments associated with the hydraulic fracturing sites. As presented in the conceptual site model, the potential environmental receptors include domesticated livestock, agricultural and native plants, local and regional wildlife, and aquatic fish and invertebrates. However, as previously noted, the objective of this ERA is to evaluate the potential risks from a typical hydraulic fracturing site; therefore, the selection of environmental receptors for this ERA is based on understanding that the selected environmental receptors all have an equal opportunity to be exposed to residual hydraulic fracturing flowback water at any of the hydraulic fracturing sites within the project area.

For an exposure pathway to be considered to be complete there must be all of the following:

- Source of COPC - how the chemical got into the environment;
- A transport media - how the chemical moves or migrates through the environment (soil, water or air);
- An exposure point - how human and environmental receptors can come into contact with the chemicals; and
- An exposure route - how the chemical could enter the body of the receptor (e.g. inhalation, ingestion or dermal contact).

If any one of these steps (source, transport media, exposure point or route) is not present, then the exposure pathway is incomplete and, hence, further assessment of risks is not required.

In the cases where the exposure pathways are complete, or have the potential to be complete, then the pathways can be considered significant or less significant. The significance of the exposure pathway depends on the nature of the impact present and the evaluation of the likely exposure concentrations that may be associated with the pathway.

A summary of the decision tree used to evaluate the environmental receptors and pathways is presented in **Table 12**.

Table 12 Identification of Potential Environmental Receptors and Pathways

Source	Exposure Scenario	Receptors (on and off-site)	Pathway	Pathway Potentially Complete?	Comment
Hydraulic Fracturing Chemicals	Spills and leaks occur while handling of chemicals either on or off-site (i.e. during transportation of chemicals to the site)	Indigenous fauna and flora	Ingestion, dermal, uptake	No	OH&S procedures and spill containment adequately address this exposure.
Flowback Water	Leaks, above ground Flexipond overflow system failure	Aquatic invertebrates, fish, Indigenous fauna	Ingestion, dermal	Yes	There is the potential for a release of stored hydraulic fracturing flowback water to surface water systems located near the hydraulic fracturing operational areas in the situation of an accidental release, such as from piping, leaks or failure of overflow system in the event of heavy rainfall. However, this exposure scenario unlikely due to robust holding Flexipond design and overflow controls implemented by Origin. Nevertheless, this pathway has been evaluated as part of this assessment.
		Indigenous fauna and flora, stock animals	Ingestion, dermal, uptake	No	The potential for an accidental release of flowback fluid from leaking Flexipond or piping does exist (although is considered unlikely due to the operational controls and robust designs implemented by Origin), however as the operational area is considered a highly modified environment (normal operational activities, infrastructure, gravel and compacted soil cover on the well pad sites and around infrastructure would significantly inhibit the establishment/function of wildlife habitat in the immediate vicinity), the exposure pathway to indigenous flora is deemed incomplete for minor leaks and spills. The probability of such an accidental release is further reduced due to the temporary duration that the flowback fluids are stored in the Flexiponds. In considering this, the likelihood that exposure to spilled or leaked flowback fluid to domestic livestock and mammalian wildlife is considered insignificant, as their exposure is not habitat driven (i.e. they would not normally forage and

Source	Exposure Scenario	Receptors (on and off-site)	Pathway	Pathway Potentially Complete?	Comment
					breed in the operational areas) and operational controls (i.e. fences) and activities at the site limiting the occurrence of livestock and wildlife entry and exposures. The potential for terrestrial receptors obtaining drinking water from leaked flowback fluid only on occasions were they to inadvertently enter onto the operational area is considered highly unlikely (i.e. the exposure frequency and duration is deemed insignificant) and hence this exposure pathway is considered effectively incomplete.
	Entry into Flexiponds	Indigenous fauna	Ingestion, dermal	No	Entry is limited via fencing around the operational areas. In addition, the walls of the Flexiponds are at least 2 m high, which prevents access into the holding ponds.
Flowback Water	Entry near the Flexiponds, within the vicinity of the mist fallout from the evaporator	Stock animals, indigenous fauna	Ingestion, inhalation, dermal	No	Entry is limited via fencing around the operational areas. The physchem and environmental fate properties of the COPC limit their uptake by terrestrial receptors. Most of the COPCs do not readily sorb to soil particles, and no COPCs are sufficiently volatile and present at concentrations to be of concern in relation to inhalation exposures. Dermal absorption of COPC (as mist) is not considered to be of significance, due to the thick skins/coats of livestock and the short exposure period. In addition Evaporator control procedures are in place to ensure the plume mist is contained within the Flexipond (refer to)
Hydraulic Fracturing Fluids	Fracturing fluid escapes into aquifer (well casing failure, fault, fracture, unconformity in seam or strata) used for stock water supply and discharges to surface water.	Stock animals, Aquatic invertebrates, fish	Ingestion, dermal	No	Origin has detailed operational procedures that are designed to contain hydraulic fracturing fluids within the shale formations. No direct connection is understood to exist between groundwater in the shale formations and surface-water, aquifers utilised for the purpose of extraction (refer **Section 2.2.4**).

Shaded cells indicate potentially complete exposure pathway, assessed as part of this ERA.

In reviewing and considering the potential significance of exposure pathways, the following points have been considered:

- No further assessment is considered warranted for groundwater as there are no potentially complete exposure pathways. Detailed operational procedures have been provided that are designed to contain the hydraulic fracturing fluids within the shale formations, and no connection exists between groundwater in the shale formations and surface-water, aquifers utilised for the purpose of extraction (for any beneficial use), or springs;
- The flowback water is contained in aboveground Flexiponds. Each modular above ground storage pond will have both geotextile and polyethylene linings (i.e. multiple barriers) in addition to monitoring to detect any leakage through the primary barrier. Terrestrial receptors identified to exist near the site and surrounds include domesticated livestock (cattle), large mammalian wildlife (kangaroo) and small mammalian wildlife (dingos). Notwithstanding the fences in place to deter terrestrial wildlife from entering the hydraulic fracturing operational area, the height of the above ground Flexiponds (approximately 1.99 m high) will effectively limit their access to the flowback water.
- To enhance the evaporation of the flowback water, floating evaporator units are deployed in the Flexiponds. The evaporators use high speed fan blade rotation which creates optimum water droplet distributions for evaporation. There is potential for flowback water mist (as created by the evaporator) to be distributed onto the ground surface or nearby surrounds (dependant on the wind strength), as such livestock or wildlife may be exposed to the COPCs via incidental soil ingestion, inhalation or direct contact of the mist. However, most of the COPC identified in flowback water do not readily sorb to soil particles, and hence few COPC would remain in the surface soil or sediments long enough to be of concern. In addition livestock typically only ingest soil or sediments where grazing. No grazing pastures were present near the hydraulic fracturing operational area, hence there is not potential for this pathway to be of concern. No COPC are sufficiently volatile and present at high enough concentrations to be of concern in relation to inhalation exposures. Dermal absorption of COPC in flowback water (as mist) may occur where livestock or wildlife are in close proximity to the Flexiponds. While this may occur, the pathway is not considered to be of significance, due to the thick skins/coats of livestock (CCME 2008) and the short exposure period. Furthermore, evaporator operation control procedures are in place to effectively contain the plume mist within the Flexipond. On this basis, and considering the physical deterrences in place (fences, height of ponds) that effectively limit livestock and wildlife access to the hydraulic fracturing operational area, the risk to terrestrial receptors has not been further assessed.
- Whilst considered minimal, there is the potential for a release of stored hydraulic fracturing flowback water to surface water systems located near the well pads in the situation of an accidental release, such as from piping, leaks or failure of overflow system in the event of heavy rainfall.

Based on the available information, the key receptors (and exposures that are considered significant) who may be exposed to the COPEC identified on and off the site includes the following:

<u>Aquatic invertebrates and fish</u>

- Direct contact (ingestion and dermal contact) with residual hydraulic fracturing chemicals identified in surface water in the event of a release of stored flowback fluid. While this is a highly conservative scenario, as the closest surface water receptor is over 1km from a HSF site, and the concentrations of residual COPECs in the flowback fluid is assumed to be 100% of the injected mass, this scenario has nevertheless been assessed. As aquatic receptors are most sensitive to the flowback fluid COPECs, the assessment of this scenario has therefore been assumed to be protective of terrestrial effects.

The assessment endpoints, or environmental values selected was the survival and reproduction of aquatic invertebrates. The assessment endpoints are sensitive to the residual hydraulic fracturing flowback water COPEC identified, and have the toxicological and life history databases to support their use as representatives of the study area ecosystem.

Invertebrates live on or in the sediments and surface waters depending on the substrate and trophic level. The exposure of invertebrates would result from direct contact with surface waters. Fish live in the surface waters (benthic and pelagic) and may move up and down stream with seasonal flow and to forage. Fish are exposed in the surface water through uptake via the gills, and through ingestion of invertebrates and other food items. It is noted that the assessment of potential impacts to the aquatic environment has also considered impacts to algae, a major food source for other aquatic species.

4.2.2 Quantification of Exposure

4.2.2.1 Exposure Pathways

As discussed in **Section 4.2.1**, the key environmental receptors (and exposures that are considered significant) that may be exposed to the COPEC identified on and off the site were aquatic invertebrates and fish.

4.2.2.2 Exposure Point Concentrations

Similar to the human health risk assessment, the exposure point concentrations for the environmental risk assessment was based on a theoretical data set (assuming 100% mass recovery in flowback water) and an empirical dataset (based on the results of laboratory analysis of flowback water from existing operations). The theoretical COPECs are presented in **Table 4** and **Table 5**. The empirical COPEC identified within the flowback water is presented in **Table 13** below.

Table 13 Identification of empirical COPEC in flowback water on-site

Analyte Detected in Flowback Water	Max Concentration Reported in 2016 (mg/L)	Adopted Screening Level Guideline (mg/L)		Selected as COPC?
Arsenic	0.084	0.024	ANZECC	Yes
Boron	66	0.37	ANZECC	Yes
Manganese	2.24	1.9	ANZECC	Yes
Nickel	0.04	0.011	ANZECC	Yes
Barium	36.4	0.004	USEPA	Yes
Chromium	0.055	0.001	ANZECC	Yes
Cobalt	0.011	0.09	ANZECC	No
3-&4-methylphenol	0.762	0.32	ANZECC	Yes
Phenol	0.0185	0.32	ANZECC	No
TPH C6-C10	0.05	0.18*	ANZECC	No
TPH C10-C34	0.98	0.7	ANZECC	Yes
Benzene	0.004	0.95	ANZECC	No
Toluene	0.003	0.18	ANZECC	No
Xylenes	0.002	0.2	ANZECC	No

Notes:
2016 data from BET-PW001, BET-TK001, BET-TK002 and BET-TK003, sourced from Origin.
*No specific guideline available for TPH C6-C10, used toluene as surrogate
Shaded cells indicate COPEC assessed as part of this ERA.
ANZECC 2000 Freshwater Quality Guidelines ; USEPA Region 3 Biological Technical Assistance Group Freshwater Screening Benchmarks.

4.3 Toxicity Assessment

4.3.1 General

The purpose of toxicity assessment is to identify concentrations or doses of COPECs at which adverse effects may occur to receptors.

The risk characterisation performed in this ERA relies on toxicity data and screening level guidelines as toxicity reference values (TRVs). The TRVs are derived based on COPC levels that imply no adverse effects or levels that represent the lowest concentrations at which adverse effects may occur. To evaluate the concentration of the COPEC in the surface water and direct exposure by the aquatic environmental receptor, a concentration based TRV was derived.

4.3.2 Concentration Based TRV

The existing water quality guidelines for protection of aquatic life were researched and compiled using the following sources of ecotoxicological information:

- Australian and New Zealand Environment and Conservation Council (ANZEEC) guidelines for fresh and marine water quality.
- Western Australia (WA) Department of Environment and Conservation. Assessment Levels for Soil, Sediment and Water. February 2010. Contaminated Site Management Series.
- Department of Environmental and Resource Management. Approval of Coal Seam Gas Water for Beneficial Use. Environmental Protection (Waste Management) Regulation 2000. Queensland Government. March 2010.
- International Guidance (USEPA, American Petroleum Institute, Oak Ridge National Laboratory/Risk Assessment Information System (ORNL/RAIS).

If water quality guidelines were not available, information on aquatic toxicity was acquired from reported values that had already been through a screening process such as the following:

- OECD-SIDS program
- EU existing substances risk assessment
- HHRA (www.HHRAproject.com)
- USEPA HPVC Challenge Program
- ECHA CHEM database

The data from these programs were considered sufficiently reviewed as to not require further evaluation. If no data were available from the above sources or the available data were considered insufficient for TRV determinations, then toxicity information on MSDSs were used, as well as read-across from available experimental data on a structurally related substances, and predicted values from QSAR models.

4.3.3 Calculation of PNEC for Freshwater

The determination of TRVs for freshwater was conducted according to the predicted no-effects concentration (PNEC) guidance in the Environmental Risk Assessment Guidance Manual for Industrial Chemicals prepared by the Australian Environmental Agency (AEA, 2009).

A PNEC is calculated using toxicity test data such as LC50, EC50, other L(E)Cx values, NOEC (no observed effect concentration) and LOEC (lowest observed effect concentration). Derivation of a PNEC commonly relies on using assessment or safety factors. Assessment factors reflect the following uncertainties inherent in most datasets, and the extrapolations that can be required:

1. Intra-species and inter-species variations
2. The extrapolation of short-term toxicity towards long-term toxicity
3. The extrapolation of laboratory results towards the field.

Table 14 lists the assessment factors used to adjust the effect concentration and to estimate a PNEC.

Table 14 Summary of assessment factors for estimating a PNEC (AEA 2009)

Data	Data Available	Range of assessment factors	Comments
When only acute toxicity data are available, an assessment factor of between 100 and 1000 is applied to the lowest L(E)C50			
Acute	• EC50 algae (72h) • EC50 *Daphnia* (24-48 h acute test) • LC50 fish (96h)	100-1000	A factor of 1000 is a conservative and protective factor and applied when only limited data are available. A factor of 100 can be applied if the following evidence is available: • Availability of data from a wide variety of species including those which are considered to represent sensitive species • Information from structurally similar compounds or QSAR, to suggest that the acute to chronic ratio is likely to be low • Information to suggest that the chemical acts in a non-specific or narcotic manner, with little inter-species variation in toxicity • Information to suggest that the release of the chemical is short-term or intermittent, and that the chemical would not be persistent in the environment.
When chronic toxicity data are available in addition to acute data, often an assessment factor of between 10 and 100 is applied to the lowest NOEC			
Chronic	• NOEC *Daphnia* (14-21d chronic toxicity test) • NOEC algae (72h) NOEC fish (chronic toxicity test)	10-100	• If a chronic NOEC is available from one or two species representing one or two trophic levels (i.e. fish, *Daphnia* or algae), a factor of 100 or 50 is applied to the lowest NOEC. In this case, a PNEC value derived from chronic data should be compared to that derived from the lowest acute data. It is then the lowest value that is used in the assessment. • If chronic NOECs are available from three species representing three trophic levels (i.e. fish, *Daphnia* and algae), a factor of 10 is applied to the lowest NOEC. If there is convincing evidence that the most sensitive species for which acute toxicity data are available have been tested chronically, a factor of 10 may also be applied to the lowest NOEC from two species representing two trophic levels (i.e. fish and/or *Daphnia* and/or algae).

It should be noted that in the case of algae studies, which are actually multigenerational studies, it is generally accepted that a 72-hour (or longer) EC50 value may be considered as equivalent to a short-term result and that a 72-hour (or longer) EC10 or NOEC value can be considered as a long-term results.

Table 15 below presents the summary of derived TRVs for the Protection of Aquatic Species (PNECs). **Appendix D** presents the environmental reviews of the COPEC identified in the hydraulic

fracturing fluids and includes further details on the endpoints selected and PNEC values calculated for each COPC.

Table 15 Summary of derived TRVs for protection of aquatic species (PNECs)

CAS #	Chemical	Endpoint*	E(L)C50 or NOEC (mg/L)	Assessment Factor (AF)	PNEC Aquatic (mg/L)**
57-13-6	Urea	L(E)C50 (algae)	29	100	0.29
64-02-8	Tetrasodium ethylenediaminetetraacetate	21 day NOEC (Daphnia)	22	10	2.2
67-48-1	2-hydroxy-N,N,N-trimethylethanaminium chloride	NOEC (Daphnia)	32	10	3.2
67-63-0	Propan-2-ol	NOEC (Daphnia)	30	100	0.3
79-06-1	2-Propenamid (impurity)	72-h EC50 (freshwater algae)	33.85	1000	0.03385
107-21-1	Ethylene glycol	NOEC	3469	10	346.9
111-46-6	2,2"-oxydiethanol (impurity)	NOEC (algae)	100	50	2
540-97-6	Dodecamethylcyclohexasiloxane	NOEC	0.0046	No AF	0.0046
541-02-6	Decamethyl cyclopentasiloxane	NOEC	0.0046	No AF	0.0046
556-67-2	Octamethylcyclotetrasiloxane	NOEC	0.0046	No AF	0.0046
1310-73-2	Sodium hydroxide (impurity)	NA. Not a PBT substance.			
2682-20-4	2-methyl-2h-isothiazol-3-one	72-hr EC50 (algae)	0.027	1,000	0.000027
7447-40-7	Potassium chloride (impurity)	72-hr EC50 (algae)	100	1000	0.1
7631-86-9	Non-crystalline silica (impurity)	NA. Not a PBT substance.			
7647-01-0	Hydrochloric acid	NA. Not a PBT substance.			
7647-14-5	Sodium chloride	NA. Not a PBT substance.			
7757-82-6	Sodium sulfate	EC50 120h (Daphnia)	1,900	1000	1.9
7758-98-7	Copper(II) sulfate	ANZECC Fresh Water Guidelines			0.0014
7783-20-2	Ammonium sulfate	ANZECC Fresh Water Guidelines			0.9
7786-30-3	Magnesium chloride	72-hr EC50 (algae)	100	1000	0.1
9000-30-0	Guar gum	48-hr EC50 (Daphnia)	42	1,000	0.042
10043-52-4	Calcium Chloride	Chronic Daphnia	160	50	3.2
10377-60-3	Magnesium nitrate	ANZECC Fresh Water Guidelines			0.7
14464-46-1	Cristobalite	NA. Not a PBT substance.			

CAS #	Chemical	Endpoint*	E(L)C50 or NOEC (mg/L)	Assessment Factor (AF)	PNEC Aquatic (mg/L)**
26172-55-4	5-chloro-2-methyl-2h-isothiazolol-3-one	72-hr EC50 (algae)	0.027	1000	0.000027
31726-34-8	Polyethylene glycol monohexyl ether	Chronic Daphnia	26.3	10	2.63
38193-60-1	Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer	NA. Polymer is non-toxic to aquatic receptors.			
61789-77-3	Dicoco dimethyl quaternary ammonium chloride	96 h EC50 (algae)	0.014	100	0.0028***
63148-62-9	Dimethyl siloxanes and silicones	NA. Substance is non-toxic to aquatic receptors.			
67762-90-7	Siloxanes and silicones, dimethyl, reaction products with silica	NA. Substance is non-toxic to aquatic receptors.			
91053-39-3	Diatomaceous earth, calcined	NA. Not a PBT substance.			
129898-01-7	2-Propenoic acid, polymer with sodium phosphinate	NA. Polymer is non-toxic to aquatic receptors.			
136793-29-8	Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate	NA. Polymer is non-toxic to aquatic receptors.			

NA – data not available or not applicable

** refer to **Appendix C** for further information on the endpoints selected and PNEC values for each COPC.

*** PNEC multiplied by a factor of 20 to account for the 5% bioavailable fraction in environmental waters (EPHC 2009).

4.4 Environmental Risk Characterisation

Risk characterisation is the process of quantifying and characterising risk using the selected measurement endpoints or lines of evidence. The NEPC (2013) and USEPA (1997) have recommended protection goals for interpreting the risks associated with individual hazard quotients (HQ) and toxicity test results.

The use of HQs has been common practice in human health risk assessments and has been carried over and used in most screening level ERAs. In this screening level ERA for aquatic receptors, calculation for HQs was used in assessing the risk of COPECs where limited species specific toxicity data exists.

4.4.1 Estimation of Risk

The results of the exposure assessment (EPCs) are compared to TRVs (or PNEC aquatic) to estimate the potential for adverse environmental effects. When exposure is greater than TRVs there is a potential for adverse impacts. To assess potential adverse environmental effects HQs were calculated. Hazard quotient calculations were calculated using the derived TRVs. HQs were calculated as follows:

$$\text{HQ aquatic} = \frac{\text{EPC}}{\text{TRV (or PNEC aquatic)}}$$

Where:

HQaquatic = Hazard Quotient
EPC = COPEC exposure point concentration in surface water (mg/L)
TRV = Concentration based TRV (or PNEC aquatic) (mg/L)

To assess the overall potential for adverse environmental effects posed by simultaneous exposure to multiple chemicals, the hazard quotients for each chemical and exposure pathway have been summed. The resulting sum is referred to as the hazard index (HI).

The risk classification system used to qualify the potential for environmental risk based on HI results was as follows:

- HI less or equal to 1 indicates acceptable low or extremely low risk;
- HI greater than 1 indicates potential risk or adverse effects and further evaluation is required to refine the calculation of potential risk to environmental receptors.

This HI system was considered appropriate given the conservative approach to estimating environmental exposure levels using the maximum measured COPEC concentrations and provide a tool by which the potential for impacts in surface water can be assessed. All hazard quotient and hazard index calculations are presented in **Appendix C**.

4.4.2 Summary of Risk

A summary of the cumulative hazard indexes for both the theoretical and empirical scenarios for the identified receptor group (aquatic invertebrates and fish) are presented in **Table 16** to **Table 17**. It is noted that a conservative approach has been adopted to estimate potential “worst case scenario” environmental risk. This worst case scenario is based on a significant spill or release event directly to an adjacent surface water body, with no operational or spill management controls. The calculations presented below also do not take into account the significant dilution that is expected to occur where flowback water is discharged to a surface water body in the event of a significant spill or leak. In addition, processes such as sorption and biodegradation have not been considered.

Table 16 Summary of calculated HQ for aquatic risk – 100% mass return (theoretical) and empirical data for hydraulic fracturing fluid Stage 1 Slickwater and Gel Flush

CAS	Chemical	Endpoint	E(L)C50 or NOEC	Assessment Factor	PNEC Aquatic	EPC	Calculated HQ
						100% Mass Return	
Stage 1 Slickwater and Gel Flush							
57-13-6	Urea	L(E)C50 (algae)	29	100	0.29	6.86	23.6
64-02-8	Tetrasodium ethylenediaminetetraacetate	21 day NOEC (Daphnia)	22	10	2.2	0.22	0.1
67-48-1	2-hydroxy-N,N,N-trimethylethanaminium chloride	NOEC (Daphnia)	32	10	3.2	1499.41	468.6
67-63-0	Propan-2-ol	NOEC (Daphnia)	30	100	0.3	1.71	5.7
79-06-1	2-Propenamid (impurity)	72-h EC50 (freshwater algae)	33.85	1000	0.03385	0.32	9.4
107-21-1	Ethylene glycol	NOEC	3469	10	346.9	107.60	0.3
111-46-6	2,2"-oxydiethanol (impurity)	NOEC (algae)	100	50	2	1.07	0.5
540-97-6	Dodecamethylcyclohexasiloxane	NOEC	0.0046	No AF	0.0046	0.11	23.9
541-02-6	Decamethyl cyclopentasiloxane	NOEC	0.0046	No AF	0.0046	0.11	23.9
556-67-2	Octamethylcyclotetrasiloxane	NOEC	0.0046	No AF	0.0046	0.11	23.9
1310-73-2	Sodium hydroxide (impurity)	NA. Not a PBT substance.					
2682-20-4	2-methyl-2h-isothiazol-3-one	72-hr EC50 (algae)	0.027	1,000	0.000027	1.18	43850.3
7447-40-7	Potassium chloride (impurity)	72-hr EC50 (algae)	100	1000	0.1	0.32	3.2
7631-86-9	Non-crystalline silica (impurity)	NA. Not a PBT substance.					
7647-01-0	Hydrochloric acid	NA. Not a PBT substance.					
7647-14-5	Sodium chloride	NA. Not a PBT substance.					
7757-82-6	Sodium sulfate	EC50 120h (Daphnia)	1,900	1000	1.9	1.61	0.8

CAS	Chemical	Endpoint	E(L)C50 or NOEC	Assessment Factor	PNEC Aquatic	EPC	Calculated HQ
						100% Mass Return	
Stage 1 Slickwater and Gel Flush							
7758-98-7	Copper(II) sulfate	ANZECC Fresh Water Guidelines			0.0014	0.11	78.7
7783-20-2	Ammonium sulfate	ANZECC Fresh Water Guidelines			0.9	142.19	158.0
7786-30-3	Magnesium chloride	72-hr EC50 (algae)	100	1000	0.1	3.74	37.4
9000-30-0	Guar gum	48-hr EC50 (Daphnia)	42	1,000	0.042	62.85	1496.3
10043-52-4	Calcium Chloride	Chronic Daphnia	160	50	3.2	10.28	3.2
10377-60-3	Magnesium nitrate	ANZECC Fresh Water Guidelines			0.7	7.60	10.9
14464-46-1	Cristobalite	NA. Not a PBT substance.					
26172-55-4	5-chloro-2-methyl-2h-isothiazolol-3-one	72-hr EC50 (algae)	0.027	1000	0.000027	4.08	150926.5
31726-34-8	Polyethylene glycol monohexyl ether	Chronic Daphnia	26.3	10	2.63	203.75	77.5
38193-60-1	Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer	NA. Polymer is non-toxic to aquatic receptors					
61789-77-3	Dicoco dimethyl quaternary ammonium chloride	96 h EC50 (algae)	0.014	100	0.0028	8.45	3019
63148-62-9	Dimethyl siloxanes and silicones	NA. Polymer is non-toxic to aquatic receptors.					
67762-90-7	Siloxanes and silicones, dimethyl, reaction products with silica	NA. Polymer is non-toxic to aquatic receptors.					
91053-39-3	Diatomaceous earth, calcined	NA. Not a PBT substance.					
129898-01-7	2-Propenoic acid, polymer with sodium phosphinate	NA. Polymer is non-toxic to aquatic receptors.					

CAS	Chemical	Endpoint	E(L)C50 or NOEC	Assessment Factor	PNEC Aquatic	EPC	Calculated HQ
						100% Mass Return	
Stage 1 Slickwater and Gel Flush							
136793-29-8	Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate	NA. Polymer is non-toxic to aquatic receptors.					
						Total HI	**200242**
COPC identified in flowback water analysis from current operations							
	Arsenic	ANZECC (2000) Freshwater quality guideline			0.024	0.084	3.5
	Boron	ANZECC (2000) Freshwater quality guideline			0.37	66	178.4
	Manganese	ANZECC (2000) Freshwater quality guideline			1.9	2.24	1.2
	Nickel	ANZECC (2000) Freshwater quality guideline			0.011	0.04	3.6
	Barium	USEPA Region 3 Biological Technical Assistance Group Freshwater Screening Benchmarks 2006			0.004	36.4	9100
	Chromium	ANZECC (2000) Freshwater quality guideline			0.001	0.055	55
	3-&4-methylphenol	ANZECC (2000) Freshwater quality guideline (as phenol)			0.32	0.762	2.4
	TPH C10-C34	ANZECC (2000) Freshwater quality guideline			0.7	0.98	1.4
						Total HI	**9345**

The following can be noted from the table above:

- The calculated hazard index results for the theoretical and empirical worst case exposure scenarios show all COPECs exceeding the acceptable risk threshold of 1.0 by 4 to 5 orders of magnitude, indicating potentially unacceptable risks to the receiving aquatic environment. It is to be noted that the calculated results are based on worst case and highly conservative exposure scenarios which are expected to overestimate the actual risk. Refer to **Section 4.5** for the interpretation of this low likelihood event.

Table 17 Summary of calculated HI for aquatic risk – 100% mass return (theoretical) and empirical data for hydraulic fracturing fluid Stage 7 Cross-Linked Gel/Slickwater Hybrid

CAS	Chemical	Endpoint	E(L)C50 or NOEC	Assessment Factor	PNEC Aquatic	EPC	Calculated HQ
						100% Mass Return	
Stage 7 Cross-Linked Gel/Slickwater Hybrid							
57-13-6	Urea	L(E)C50 (algae)	29	100	0.29	2.60	9.0
64-02-8	Tetrasodium ethylenediaminetetraacetate	21 day NOEC (Daphnia)	22	10	2.2	0.14	0.1
67-48-1	2-hydroxy-N,N,N-trimethylethanaminium chloride	NOEC (Daphnia)	32	10	3.2	1692.76	529.0
67-63-0	Propan-2-ol	NOEC (Daphnia)	30	100	0.3	1.73	5.8
79-06-1	2-Propenamid (impurity)	72-h EC50 (freshwater algae)	33.85	1000	0.03385	0.14	4.3
107-21-1	Ethylene glycol	NOEC	3469	10	346.9	360.58	1.0
111-46-6	2,2"-oxydiethanol (impurity)	NOEC (algae)	100	50	2	1.01	0.5
540-97-6	Dodecamethylcyclohexasiloxane	NOEC	0.0046	No AF	0.0046	0.14	31.4
541-02-6	Decamethyl cyclopentasiloxane	NOEC	0.0046	No AF	0.0046	0.14	31.4
556-67-2	Octamethylcyclotetrasiloxane	NOEC	0.0046	No AF	0.0046	0.14	31.4
1310-73-2	Sodium hydroxide (impurity)	NA. Not a PBT substance.					
2682-20-4	2-methyl-2h-isothiazol-3-one	72-hr EC50 (algae)	0.027	1,000	0.000027	1.16	42820.7
7447-40-7	Potassium chloride (impurity)	72-hr EC50 (algae)	100	1000	0.1	0.36	3.6
7631-86-9	Non-crystalline silica (impurity)	NA. Not a PBT substance.					
7647-01-0	Hydrochloric acid	NA. Not a PBT substance.					
7647-14-5	Sodium chloride	NA. Not a PBT substance.					

CAS	Chemical	Endpoint	E(L)C50 or NOEC	Assessment Factor	PNEC Aquatic	EPC	Calculated HQ
						100% Mass Return	
Stage 7 Cross-Linked Gel/Slickwater Hybrid							
7757-82-6	Sodium sulfate	EC50 120h (Daphnia)	1,900	1000	1.9	0.58	0.3
7758-98-7	Copper(II) sulfate	ANZECC Fresh Water Guidelines			0.0014	0.14	103.2
7783-20-2	Ammonium sulfate	ANZECC Fresh Water Guidelines			0.9	52.89	58.8
7786-30-3	Magnesium chloride	72-hr EC50 (algae)	100	1000	0.1	3.61	36.1
9000-30-0	Guar gum	48-hr EC50 (Daphnia)	42	1,000	0.042	9.83	234.0
10043-52-4	Calcium Chloride	Chronic Daphnia	160	50	3.2	9.83	3.1
10377-60-3	Magnesium nitrate	ANZECC Fresh Water Guidelines			0.7	7.23	10.3
14464-46-1	Cristobalite	NA. Not a PBT substance.					
26172-55-4	5-chloro-2-methyl-2h-isothiazolol-3-one	72-hr EC50 (algae)	0.027	1000	0.000027	3.83	141843.5
31726-34-8	Polyethylene glycol monohexyl ether	Chronic Daphnia			2.63	208.54	79.3
38193-60-1	Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer	NA. Polymer is non-toxic to aquatic receptors.					
61789-77-3	Dicoco dimethyl quaternary ammonium chloride	96 h EC50 (algae)	0.014	100	0.0028	8.74	3122.7
63148-62-9	Dimethyl siloxanes and silicones	NA. Polymer is non-toxic to aquatic receptors.					
67762-90-7	Siloxanes and silicones, dimethyl, reaction products with silica	NA. Polymer is non-toxic to aquatic receptors.					
91053-39-3	Diatomaceous earth, calcined	NA. Not a PBT substance.					
129898-01-7	2-Propenoic acid, polymer with sodium	NA. Polymer is non-toxic to aquatic receptors.					

CAS	Chemical	Endpoint	E(L)C50 or NOEC	Assessment Factor	PNEC Aquatic	EPC	Calculated HQ
						100% Mass Return	
Stage 7 Cross-Linked Gel/Slickwater Hybrid							
	phosphinate						
136793-29-8	Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate	NA. Polymer is non-toxic to aquatic receptors.					
110-17-8	Fumaric acid	72 hr EC50 (algae)	41	1000	0.041	11.71	285.5
1319-33-1	Boronatrocalcite	Canadian Water Quality Guidelines			1.5	445.34	296.9
1330-43-4	Sodium tetraborate	Canadian Water Quality Guidelines			1.5	37.50	25.0
7704-73-6	Monosodium fumarate	72 hr EC50 (algae)	41	1000	0.041	11.71	285.5
7789-38-0	Sodium bromate	7-day LC50	1.093	100	0.01093	149.58	13685.1
10043-35-3	Boric acid	Canadian Water Quality Guidelines			1.5	11.71	7.8
595585-15-2	Diutan gum	72 hr EC50 (algae)	100	100	1	0.58	0.6
Total HI							**203546**
COPC identified in flowback water analysis from current operations							
	Arsenic	ANZECC (2000) Freshwater quality guideline			0.024	0.084	3.5
	Boron	ANZECC (2000) Freshwater quality guideline			0.37	66	178.4
	Manganese	ANZECC (2000) Freshwater quality guideline			1.9	2.24	1.2
	Nickel	ANZECC (2000) Freshwater quality guideline			0.011	0.04	3.6
	Barium	USEPA Region 3 Biological Technical Assistance Group Freshwater Screening Benchmarks 2006			0.004	36.4	9100
	Chromium	ANZECC (2000) Freshwater quality guideline			0.001	0.055	55

CAS	Chemical	Endpoint	E(L)C50 or NOEC	Assessment Factor	PNEC Aquatic	EPC	Calculated HQ
						100% Mass Return	
Stage 7 Cross-Linked Gel/Slickwater Hybrid							
	3-&4-methylphenol	ANZECC (2000) Freshwater quality guideline (as phenol)			0.32	0.762	2.4
	TPH C10-C34	ANZECC (2000) Freshwater quality guideline			0.7	0.98	1.4
						Total HI	**9345**

The following can be noted from the table above:

- The calculated hazard index results for the theoretical and empirical worst case exposure scenarios show all COPECs exceeding the acceptable risk threshold of 1.0 by 4 to 5 orders of magnitude, indicating potentially unacceptable risks to the receiving aquatic environment. It is to be noted that the calculated results are based on worst case and highly conservative exposure scenarios which are expected to overestimate the actual risk. Refer to **Section 4.5** for the interpretation of this low likelihood event.

4.5 Interpretation of Environmental Significance

The estimated environmental risks have been qualitatively evaluated via a hazard index approach to determine their environmental significance for the hydraulic fracturing operations evaluated in this assessment. The HQ results show all COPECs from the theoretical data set exceed the acceptable risk threshold of 1.0 for aquatic receptors by 4 to 5 orders of magnitude.

It is noted that this estimated potential risk is based on a significant spill or release event directly to an adjacent surface water body. The closest surface water body is 24 km to the east Amungee NW-1H. The calculations presented above do not take into account the significant dilution that is expected to occur where flowback water is discharged to a surface water body in the event of a significant spill or leak. In addition, processes such as sorption and biodegradation have not been considered.

During normal operation procedures there are no discharges to adjacent surface water bodies. Furthermore, the lack of a robust aquatic toxicological database resulted in aquatic screening values for the theoretical exposure scenario COPEC to be highly conservative, which is expected to significantly overestimate the actual risk.

Notwithstanding this conservative approach to estimating environmental exposure levels, risk management measures are routinely implemented by Origin which effectively minimise the risk of accidental spills and releases of flowback fluid. These include:

- Implementation of spill containment procedures during operations to prevent migration of and exposure to chemicals;
- Routine holding tank inspections to check for leakage of flowback fluid;
- Freeboard buffer included above high fluid level automatic pump shut-off system, in the event of heavy rainfall;
- Locked perimeter fencing with warning signage.

Based on the implementation of a range of risk management measures by Origin, accidental releases of flowback fluid are considered to be unlikely. No such events have occurred during operations (including APLNG) conducted to date, supporting the unlikely nature of these events.

4.6 Direct Toxicity Assessments

While no direct toxicity assessment was conducted for the Beetaloo shale gas project, the monitoring methodology and results from the coal seam gas (CSG) work program may provide some insight into the toxicity of hydraulic fracturing fluid chemicals.

For context, the Origin's APLNG Project EPBC approval (2009/4974) requires the assessment of; *the constituent components of any hydraulic fracturing agents and any other reinjected fluid and their toxicity as total effluent ecotoxicity*. In order to meet this requirement, APLNG engaged consultant Hydrobiology to develop an ecotoxicity testing program to assess the incremental toxicity of representative hydraulic fracturing fluids, in the context of the natural ecotoxicity of CSG groundwater to surface freshwater organisms. A brief summary of the Origin results are provided below.

Direct toxicity assessments were conducted to assess the ecotoxicity of the hydraulic fracturing fluids in the context of CSG waters (coal seam water pre- hydraulic fracturing) and flowback waters (coal seam water post hydraulic fracturing) from within the Surat Basin, as such, ecotoxicity testing of the hydraulic fracturing fluids was performed using whole mixtures rather than the individual components. The program of works consisted of three phases:

Phase 1 – Background ecotoxicity of CSG water. The aim was to determine the background ecotoxicity of representative CSG water, collected from a non-hydraulically stimulated well (RC-PW06J) within the Reedy Creek tenement.

Phase 2 – Ecotoxicity of representative hydraulic fracturing fluids. The aim was to assess the response of freshwater species to two hydraulic fracturing fluid types (cross-linked fluid RAM24 and nitrogen foam cross-linked fluid RAM23) mixed with source water from the Talinga water treatment facility.

Phase 3 – Ecotoxicity of flowback waters. The aim was to determine the hazard potential of flowback waters in comparison with pre-stimulated CSG waters (Phase 1) and a representative hydraulic fracturing fluid (Phase 2). Flowback waters from the Ramyard tenement were selected (RAM23 and RAM24), and stimulated with fluids tested in Phase 2, and a late stage flowback from the Horse creek tenement (HCK16) was also tested, stimulated using similar cross-linked mixture compared with the RAM24 hydraulic fracturing fluid.

The direct toxicity testing was conducted in accordance with standard methods based on OECD and USEPA protocols and consistent with ANZECC/ARMCANZ (2000) guidelines for direct toxicity assessment.

The results indicated similar toxicity between the two Ramyard flowback waters (RAM23 and RAM24). Flowback from HCK16, collected at a later stage flowback (considered produced water) return volume of 1605%, had a slightly lower ecotoxicity than the Ramyard flowback waters (that were collected at earlier stages of flowback return volumes). The flowback water samples were comparable with Trigger Values observed for the background CSG water, despite the much higher toxicity for the hydraulic fracturing fluid. This indicates that the hazard from flowback waters is not increased above the non-stimulated CSG waters.

The direct toxicity assessment results demonstrate that the flowback water is of similar toxicity to the background CSG water to the species tested. This is likely due to the high salinity of the existing coal seam groundwater in the Surat Basin. Direct contact of hydraulic fracturing fluids to the freshwater species tested are highly unlikely due to distance, operational controls and management measures understood to be implemented by Origin, which minimises the risk of accidental spills and releases of hydraulic fracturing fluids and produced waters.

Currently Origin is in the process of collaborating with other CSG operators to compile a scientifically robust and industry representative report, in response to the Commonwealth review. Once this document has been assessed and approved by the Commonwealth it will be publicly released and can be provided as an appendix to this report.

4.7 Conclusions

The evaluation of potential risks to the environment associated with the use of chemicals in well simulation activities conducted by Origin within the project area has involved the assessment of potential exposures to compounds used or formed in the simulation activities. Based on the operational controls, security and management practices implemented by Origin and the design of the Flexiponds, the only potentially complete exposure pathway was from the accidental release of flowback water from the Flexiponds to adjacent aquatic environments.

On the basis of the assessment undertaken with consideration of the uncertainties identified in **Section 7**, the following can be concluded in relation to potential exposures to flowback water from well simulation activities:

- Risks to the environment are considered to be negligible or low. While potential impacts to aquatic receptors could occur if releases of flowback water reach adjacent surface water bodies, the likelihood that any of these chemicals may be discharged to an environment where any level of exposure may occur is considered low, due to the implementation of existing operational management measures. The identified level of risk is supported by review of historic and existing operations where no accidental releases of flowback water from the Flexiponds have occurred.

5.0 Non-Chemical Risks Assessment

5.1 Noise and Vibration

Noise and vibrations generated by the pumps, blenders, generators during the hydraulic fracturing operation may have posed a hazard to residents, were they located sufficiently close to Amungee NW-1H.

No residents are located within the vicinity of Amungee NW-1H. If residents were located near a well to be hydraulically fractured, AECOM understand that Origin would perform noise modelling and, if deemed warranted by the model outcomes, offer temporary relocation or compensation to the residents.

5.2 Radiological Exposure

As hydraulic fracturing fluid passes through the shale, it releases substances trapped naturally in the rock. Consequently, the flowback water can be highly saline, contain dissolved and suspended solids, heavy metals and naturally occurring radioactive material (NORM) of varying concentrations (Edmiston et al. 2011; Warner et al. 2013). High concentrations of NORM such as uranium (U), thorium (Th) and radium (Ra) can be found in many sedimentary shale formations (Walter et al. 2012).

The radiation risk associated with NORM in flowback water has been assessed based on the data acquired during operations at Amungee NW-1H. The volumes of flowback water and the concentrations of radionuclides that could be expected in flowback water if the Beetaloo shale gas project proceeds (under large scale development scenario) were considered in the context of other fluxes of radionuclides derived from local groundwater, other shale gas basins, and those generated and managed by various Australian industries. The assessment is presented in **Appendix D** and summarised below.

The B-shale within the Velkerri Formation of the Proterozoic Roper Group of the Beetaloo Basin is naturally enriched in NORM, but their absolute concentrations are not remarkably high. Their concentrations are comparable to levels found in post-Archean average Australian shale (Taylor and McLennan 1985); North American shale composite (Gromet *et al.* 1984); average shale (Wedepohl 1971); and shale gas formations in the United States (Chermak and Schreiber 2014).

Based on a radon concentration of 225 Bq/m^3, the annual effective radiation dose from the inhalation radon progeny was calculated to be 0.70 mSv for site workers. This effective dose is below the annual Australian limits for public (1 mSv) and occupational (20 mSv) exposure to ionizing radiation (ARPANSA 2002). The annual effective dose from randon progeny is expected to be <0.70 mSv for the general public (e.g. unauthorised access) because of lower exposure times.

Generally lower radioactivity and radionuclide concentrations have been measured in the flowback from Amungee NW-1H in the Beetaloo compared to levels reported for Silurian Shale (Baltic Basin and Lublin Trough), and Marcellus Shale, and Bowland Basin. Compared to produced water from conventional oil and gas production, the concentrations of radionuclides are significantly lower in the flowback water from Amungee NW-1H.

The predicted radioactive flux in flowback water from Beetaloo is significantly lower than those predicted for the Marcellus, Bowland and Silurian shale basins and produced water from conventional oil and gas. The radioactive flux in the flowback water from Beetaloo is marginally higher than that predicted if local groundwater is pumped in the same quantities as forecasted for hydraulic fracturing operations in Beetaloo. This increased activity from flowback water above 'background' water is much less significant when viewed relative to other shale gas basins from overseas and produced water from conventional oil and gas production.

The largest contributors to the radioactivity flux among the modelled Australian industries include produced water from conventional oil and gas production, bauxite mining and associated red mud tailings disposal, sand mining and processing, iron ore smelting, phosphate fertiliser production and coal and coal waste (fly ash and bottom ash). These industries would contribute > 99% of the total radioactive flux generated over the next 24 years (the forecasted timeframe for hydraulic fracturing operations in Beetaloo under a large scale development scenario). The comparative analysis

demonstrates that Australia not only generates and manages greater volumes of products or waste than could be expected from hydraulic fracturing operations in the Beetaloo, but also currently manages greater volumes of proportionately more radioactive materials. The radioactive flux in flowback under the modelled conditions is comparable to those found in sediment /sludge waste generated by typical Australian metropolitan drinking water treatment plants.

The inhalation of radon progeny was the dominant source that contributes more than 96% of the Total Effective Dose, followed by ^{226}Ra (2.1%) and ^{228}Ra (1.1%). The calculated annual Total Effective Dose of 0.72 mSv, based on the available data and modelled exposure conditions, does not exceed the Australian (ARPANSA 2011) annual effective dose limits of 1 mSv for general public and 20 mSv for occupational workers. Therefore, radiation exposure and associated health risks of NORM in flowback water from the hydraulic fracturing operations conducted at Amungee NW-1H are minimal for site-workers and general public (unauthorised access).

Technologies available for treatment and reuse of flowback water (Boschee 2014) can be broadly categorised into conventional treatment (e.g. flocculation, coagulation, sedimentation, filtration, and lime softening) and advanced treatment technology (e.g. reverse osmosis, ion-exchange, and thermal distillation, evaporation and/or crystallization processes). Treatments such as flocculation, reverse osmosis, ion exchange or lime softening tend to remove radium (and dissolved uranium) quite efficiently (ARPANSA 2008b). Chemical treatment in the form of sulfate precipitation for barium removal also results in the co-precipitation of ^{226}Ra with barium sulfate ($BaSO_4$) (Zhang *et al.* 2014).

For treatment processes where the volume of waste generated is much smaller than the volume of treated water (e.g. reverse osmosis and thermal distillation/evaporation) pre-concentration of radionuclides can occur and disposal of such wastes may require careful management to minimise exposures. The levels of these potential radionuclides concentrate and their impacts will be assessed as part of the Beetaloo shale gas project pre-feasibility and design phase.

5.2.1 Recommendations

AECOM recognises that the Beetaloo shale gas project is in its infancy and recommends that the following be considered as the project progresses to confirm the findings of this assessment and to increase the knowledge base on the distribution and levels of NORM in shale gas plays in the Beetaloo Basin:

- Further testing of radionuclides, including gross alpha and beta activities, ^{238}U, ^{232} Th, ^{40}K and ^{226}Ra and ^{228}Ra, in flowback water, drill cuttings from target formations and bottom solids/sediment samples collected from flowback water storage facilities:
  - Flowback water is to be sampled at selected hydraulically fractured wells to capture the initial return of the water flowed back and over the following 4 weeks on a weekly basis then monthly for a further two months to assess the temporal changes in radioactivity.
  - Drill cuttings from target shale formations and other major formations such as the Cambrian Limestone Aquifer is to be sampled.
  - Bottom solids/sediment samples are to be collected from flowback water storage facilities. The intention of the sampling is to obtain samples representative of the entire footprint of each storage facility.
- Conduct Toxicity Characterisation Leaching Procedure (TCLP) on selected bottom solids/sediment samples collected from flowback water storage facilities. Such testing should be conducted within the context of the National Environmental Protection (Assessment of Site Contamination) Measure 1999 (ASC NEPM) as amended in 2013 (NEPC 2013). Testing is to be conducted using both acetic acid and deionised water extractions to provide information in relation to landfill disposal options and simulate leaching due to natural infiltration by meteoric water.
- Targeted and selective testing of gas samples for radon at the well head and ambient air in the vicinity of the flowback water storage facilities.
- Update the NORM assessment report based on the outcomes of the above testing program.

6.0 Uncertainty Assessment

In general, the uncertainties and limitations of the human health and environmental risk assessment can be classified into the following categories:

- Sampling and analysis;
- Receptor exposure assessment; and
- Toxicological assessment.

The risk assessment process, following both Australian and international guidance, provides a systematic means for organising, analysing, and presenting information on the nature and magnitude of risks to public health posed by chemical exposures. Despite the advanced state of the current risk assessment methodology, uncertainties and limitations are inherent in the risk assessment process. The uncertainties associated with each of these categories are discussed in the sections above.

6.1 Sampling and Analysis Uncertainties

Variability in environmental chemistry sampling and analysis error can stem from the sampling and analysis procedures, and from the heterogeneity of the matrix being sampled. In relation to the assessment presented in this report, a number of uncertainties exist in the assessment of potential exposures to COPC based on empirical data. Limited empirical data is available for flowback water from existing operations, and from the use of the range of hydraulic fracturing fluids proposed. In addition the concentrations of COPC in regional groundwater (within the target shale formation) are not known. Hence assuming that all of the detected compounds (inorganic and organic) are associated with the well hydraulic fracturing activities is highly conservative and may not be representative of impacts that may be only associated with these activities. It is recommended that once additional data is available on flowback water quality (and regional water quality) that these potential risk issues are further reviewed.

6.2 Exposure Uncertainties

Risk assessments require the adoption of several assumptions in order to assess potential human exposure. This risk assessment includes assumptions about general characteristics and patterns of human exposure relevant to the receptor groups. These assumptions are conservative and developed to provide an estimate of maximum possible exposures rather than the actual exposures. For example, the location and activity of the human receptors assume that the trespassers will be in contact with the flowback fluid in the holding ponds over a period of 10 years. The typical period of construction (drilling, completion and work over) is 30 days, with drilling comprising a 3 to 7 day period, and as such this approach is expected to overestimate the risks.

Further, the presence and concentrations of COPC assumed in the calculations are based on a worst-case assumption that the COPC present in hydraulic fracturing fluids do not degrade or sorb significantly in the sub-surface, and that these chemicals may all be present in the water to which trespassers, aquatic receptors and terrestrial species are exposed.

6.3 Toxicity Uncertainties

In general, the available scientific information is insufficient to provide a thorough understanding of all of the potential toxic properties of chemicals to which humans may be exposed. It is necessary, therefore, to extrapolate these properties from data obtained under other conditions of exposure which generally involve experimental laboratory animals.

Although there may be interspecies differences in chemical absorption, metabolism, excretion and toxic response, there may also be uncertainties concerning the relevance of animal studies using exposure routes that differ from human exposure routes. In addition, the frequent necessity to extrapolate results of short term or subchronic animal studies to humans exposed over a lifetime has inherent uncertainty. In order to adjust for these uncertainties, published toxicity values generally incorporate safety factors that may vary from 10 to 1000. The uncertainties inherent in the toxicological values adopted are considered likely to result in an overestimation of actual risk.

7.0 Conclusions

7.1 Summary of Assessed Risks and Mitigation Measures

Table 18, below discusses the potential hazards identified in **Section 2.0,** the mechanisms by which adverse impacts are mitigated, the residual risk and recommendations to address residual risk. The hazards are classified based on the six key conditions under which the US EPA considers the impacts of hydraulic fracturing activities can be more frequent or severe (see **Section 2.2**).

Table 18 Summary of Risks, Mitigation Measures and Recommendations

US EPA Nominated Key Activities and Factors for Hydraulic Fracturing Risk Assessment	Identified Potential Hazard	Hazard Causes and Consequences	Risk Mitigation Measures	Residual Risk	Recommended Mitigation to Address Residual Risk
Water withdrawals for hydraulic fracturing in times or areas of low water availability, particularly in areas with limited or declining groundwater resources	Assessment of the adequacy of regional water resources to service hydraulic fracturing operations is beyond the scope of this risk assessment. Origin has forecast an increase of 2.6% of the sustainable yield of the CLA is the maximum groundwater supply requirement during peak field development, with an average increase of 1.2% over the course of the development. Origin has committed to making impacted stakeholders whole if they are affected by Origin's activities. If a landholder's business or well-being is adversely impacted, Origin has committed to remediate and/or compensate for the financial loss or loss of amenity experienced. Source - *Origin Submission to the Inquiry into Hydraulic Fracturing in the Northern Territory #153 Part 2*.				
Spills during the management of hydraulic fracturing fluids and chemicals or produced water that result in large volumes or high concentrations of chemicals reaching groundwater resources	**The loss of containment of chemicals used for hydraulic fracturing (or flowback water) during hydraulic fracturing**	Exposure of workers, residents or the environment to potentially hazardous hydraulic fracturing chemicals, resulting from loss of containment occurring: • Whilst in transit to site; or • During the mixing of hydraulic fracturing fluid on site; or • (In diluted form) during the storage and transport of well flowback fluid.	It is understood Origin implements a range of engineering and operational control measures to ensure the risk of hydraulic fracturing fluid spills are minimised during hydraulic fracturing operations, including during transport to and from the wellsite and during the flowback of hydraulic fracturing chemicals from the well. These control measures include but are not limited to: • Adherence to the material storage and transport requirements as stipulated on all the chemical Safety Data Sheets (SDS). • Adherence to the Transport of Dangerous Goods by Road Regulations (for chemicals classified as 'Dangerous Goods'). • Ensuring chemicals remain in their original containers and are not decanted into smaller containers which may not be fit for purpose. • The use of secondary containment beneath all pipework connections and equipment holding or pumping chemicals, inclusive of the flowback Flexipond (i.e. doubled lined) during hydraulic fracturing operations, Using fit for purpose flowback fluid storage ponds. Conducting regular integrity checks of fluid storage ponds, during commissioning and use. Ensure adequate freeboard is maintained at all times.	The evaluation of the risk to workers involved in the operation of hydraulic fracturing activities (including transportation of the COPC to the site) is not considered warranted in the subsequent sections of this risk assessment, as it is understood that Origin's hydraulic fracturing contractors implement appropriate occupational health and safety procedures and management plans. Exposures by workers involved in the operation of hydraulic fracturing activities have not been evaluated as their exposures are addressed through the implementation of appropriate occupational health and safety procedures and management plans. The PBT assessment of the chemicals indicated that most of the compounds (with the exception of MIT, CMIT, tetrasodium EDTA, cyclicdimethylpolysiloxane compounds, acrylamide polymers, dimethyl siloxanes and silicones , copper sulfate and dicoco dimethyl quaternary ammonium chloride) utilised in hydraulic fracturing fluids are not considered to be persistent, bioaccumulative or toxic in the environment. As such, the compounds are expected to degrade in the sub-surface and would not be expected to remain in flowback water extracted from the well. Where these compounds are present in flowback water, they will readily degrade or dissociate in the environment and will not bioaccumulate in terrestrial or aquatic species. Polymers, EDTA, siloxanes and silicones have limited biodegradation potential; however, are not bioaccumulative and are essentially non-toxic to aquatic species. While MIT and CMIT, copper sulfate and dicoco dimethyl quaternary ammonium chloride meet the screening criteria for toxicity, (qualified by their acute toxicity capacity), adverse chronic outcomes are not evident based on their reactivity and lack of persistence and bioaccumulation in the environment.	Operational controls which could further minimise the potential for exposures include: • the installation of fencing and signage around the flowback storage ponds; • routine operational and security patrols to prevent trespassing; • Implementation of spill containment procedures; • routine gathering network inspections and evaluation of production rate data; • routine holding pond inspections to check for possible leakage of flowback fluid; • freeboard buffer included above high fluid level automatic pump shut-off system, in the event of heavy rainfall.

US EPA Nominated Key Activities and Factors for Hydraulic Fracturing Risk Assessment	Identified Potential Hazard	Hazard Causes and Consequences	Risk Mitigation Measures	Residual Risk	Recommended Mitigation to Address Residual Risk
Spills during the management of hydraulic fracturing fluids and chemicals or produced water that result in large volumes or high concentrations of chemicals reaching groundwater resources	**The loss of containment of chemicals used for hydraulic fracturing (or flowback water) during hydraulic fracturing**			Cyclicdimethylpolysiloxane compounds meet the criteria for being persistent, bioaccumulative and toxic in the environment. They are considered poorly soluble in water, volatile and will adsorb strongly to organic matter in sediment and soil. Nevertheless, these COPCs were included in the quantitative evaluation of environmental risks (refer to **Section 5.0**). The risk assessment was conducted using a combination of theoretical calculations and the results of flowback water sampling (site specific data). Based on the conceptual site model, the potentially complete exposure pathways identified in the risk assessment comprised the following: • Incidental ingestion and dermal contact by trespassers at the flowback fluid holding tanks; and • Potential releases of water to the environment. Providing the risk mitigation measures understood to be implemented by Origin are followed, the results of the risk assessment indicated the following: • No unacceptable risks to trespassers were identified. • Risks to the environment are considered negligible or low. The likelihood that any of these COPCs may be discharged to an environment where potential environmental harm may occur is considered highly unlikely, due to the implementation of existing operational management measures. • The identified level of risk is supported by review of historic and existing operations where no accidental release of flowback fluid from the holding ponds have occurred.	These operational and management controls will further support the monitoring recommendations included in: Origin's *Groundwater Management Plan (NT-2050-15-MP-0011 Beetaloo GMP Rev3)* and *Amungee NW-1H Hydraulic Stimulation and Well Testing Environmental Plan (CDN/ID NT-2050-35-PH-0018),* which have been complied to monitor the potential impacts and mitigate the potential risks of hydraulic fracturing in the project area.
	The loss of containment of chemicals used for casing perforation	Exposure of workers, residents or the environment to potentially hazardous chemicals, resulting from loss of containment occurring whilst in transit to site.	The perforation chemicals are transported to site in pre-packed, sealed 'charges', before being loaded into the guns on site.	Given the perforation chemicals are transported to site in a pre-packed form and not directly handled in transit or on site the risk of loss of containment is commensurate with the transport of any containerised chemical product, hence the risk is not specific to hydraulic fracturing and does not warrant further quantitative risk assessment.	Not required.

US EPA Nominated Key Activities and Factors for Hydraulic Fracturing Risk Assessment	Identified Potential Hazard	Hazard Causes and Consequences	Risk Mitigation Measures	Residual Risk	Recommended Mitigation to Address Residual Risk
Injection of hydraulic fracturing fluids into wells with inadequate mechanical integrity, allowing gases or liquids to move to groundwater resources ***and*** ***Injection of hydraulic fracturing fluids directly into groundwater resources***	**Alteration of groundwater quality in aquifers containing beneficial use groundwater overlying or underlying the target gas-bearing formation;** **i.e. water quality alteration occurring via leakage of hydraulic fracturing fluid directly from the well or migration of the fluid from the target shale gas formation into beneficial-use aquifers.**	Contamination of groundwater in beneficial-use aquifers overlying or underlying the target shale gas formation with hydraulic fracturing chemicals due to: • Hydraulic fracturing creating a hydraulic connection between the target shale gas formation and overlying or underlying aquifers; • Failure of the integrity of the shale gas well, resulting in a connection forming between fluid inside the well and the beneficial-use aquifers overlying the target shale. • Loss of hydraulic fracturing fluids into the over- or under-lying aquifers. • Migration of residual hydraulic fracturing fluid left within the hydraulically generated fractures into beneficial-use aquifers	Regional landholder bores source groundwater from the aquifers containing beneficial-use groundwater, which are located at least 2190 m above the horizontal section of Amungee NW-1H (the hydraulically fractured interval).No usable aquifers are recognised below the target shale. Origin installs and cements in-place two strings of casing through the beneficial-use aquifers interval (three strings in the CLA), which are a physical barrier preventing the leakage of hydraulic fracturing fluid from within the well into the beneficial-use aquifers. The geological formations targeted by Origin hydraulic fracturing operations are separated by low permeability aquitards, made of high strength rock (see **Appendix A**, **Section A1.2.3** and **A1.4.3**). The modelled fracture half-length and fracture height at Amungee NW-1H are 150m and 50m respectively. The fracture height is contained within the B shale due to the stark contrast in mechanical properties between the organic rich zone and the formation above (i.e. interburden). Hence there would be at least 2190 m vertical separation between the leading edge of the hydraulic fracture and the beneficial-use aquifers (i.e. CLA). This separation interval comprises a sequence of low permeability formations (see **Appendix A**), which precludes the migration of hydraulic fracturing fluid from within the fractures upwards towards beneficial-use aquifers. Hydraulic fracturing operations are designed so as not to propagate outside of the target formation; through the design of a safe injection pressure operating envelope (see **Appendix A, Section A.1.4.4**). This operating envelope is developed based on consideration of: • Pressure testing • Mechanical earth model (see **Appendix A, Section A1.4.3**) • Hydraulic fracturing models (see **Appendix A, Section A1.4.3**) • Relevant legislation and internal and external standards and operating procedures Further, the low permeability of the target shale limits the capacity for hydraulic fracturing fluid to migrate in the formation beyond the leading edge of the hydraulically-generated fractures.	Based on the hydrogeological setting within the project area (**Appendix A, Sections A1.2.3** and **A1.2.4**), and the understanding that Origin adhere to the protocols which they have put in place regarding well integrity (**Appendix A, Section A1.3**) and hydraulic fracturing process control (**Appendix A, Section A1.4.4**), there is no complete exposure pathway between groundwater in the aquifers containing beneficial use groundwater overlying the target formation.	Groundwater monitoring within beneficial use aquifers in accordance with Origin's *Beetaloo Exploration Drilling Campaign 1 – Groundwater Monitoring Plan (NT-2050-15-MP-0011)*.

US EPA Nominated Key Activities and Factors for Hydraulic Fracturing Risk Assessment	Identified Potential Hazard	Hazard Causes and Consequences	Risk Mitigation Measures	Residual Risk	Recommended Mitigation to Address Residual Risk
	Alteration of the quality of groundwater (i.e. formation water) in the target shale formation via the injection hydraulic fracturing fluid, which in turn is accessed by landholder bores screened in the target shale-gas formation	This risk is not applicable to the development of shale gas in the Beetaloo. The Velkerri and Kyalla formations are not aquifers and no water is sourced from these formations.			Not required.
	Hydraulically fracturing the incorrect geological formation	The risk of hydraulically fracturing the incorrect geological formation is non-applicable to horizontal wells. This would require the operator to land the horizontal section in the incorrect formation. Given the thickness and continuity of the shale formations the risk is non-existent.			Not required.
Discharge of inadequately treated hydraulic fracturing wastewater to surface water resources	**The loss of containment of flowback fluid via the enhanced evaporation system**	Flowback fluid mist (created by the evaporator) is distributed onto the adjacent surrounding areas (dependant on the wind strength), which in turn is carried into watercourses with rainfall runoff or makes dermal contact with trespassers or livestock.	AECOM understands the enhanced evaporation unit is programed only to operate in optimal weather conditions to ensure the plume of mist generated remains within the extent of the Flexipond. A weather station is setup nearby to monitor wind intensity and direction. If there is a sudden change in wind direction or an increase in wind speed, the evaporator automatically shuts down and will not restart until conditions return to within the approved operating envelope. Prior to being used to evaporate flowback fluid, the evaporator was commissioned using potable water.	On this basis the control measures put in place by Origin to prevent loss of containment of the flowback fluid via the enhanced evaporation misters are followed and function as intended, further consideration of the risk is not warranted.	Not required.
Disposal or storage of hydraulic fracturing wastewater in unlined pits, resulting in contamination of groundwater resources	Origin does not discharge hydraulic fracturing wastewater (flowback water) into unlined pits. Flowback water is held in above ground, double lined 'Flexipond' tanks, from which it is evaporated or trucked off site for disposal at an appropriately licenced waste disposal facility.				

US EPA Nominated Key Activities and Factors for Hydraulic Fracturing Risk Assessment	Identified Potential Hazard	Hazard Causes and Consequences	Risk Mitigation Measures	Residual Risk	Recommended Mitigation to Address Residual Risk
	Harm to residents or fauna due to noise and/or vibrations from hydraulic fracturing operations	Significant noise and vibrations can be created during hydraulic fracturing operations by the pumps, blenders, generators used to conduct the operation. This noise has the potential to harm residents or fauna in the short or long term.	No residents are located near Amungee NW-1H. If residents were located near a well to be hydraulically fractured, AECOM understand that Origin would perform noise modelling and, if deemed warranted by the model outcomes, offer temporary relocation or compensation to the residents.	Not applicable for Amungee NW-1H.	Risk mitigation achieved via existing Origin protocols (e.g. resident relocation).
	Harm to site-workers and general public (unauthorised access) from exposure to ionising radiation from NORM in flowback water	Internal exposure to NORM may result from the inhalation or incidental ingestion of radionuclides leading to exposure to ionising radiation. This may occur while working on or in hydraulic fracturing plant/equipment, handling flowback water.	Standard radiation protection measures in line with ARPANSA guidelines will be implemented during hydraulic fracturing operations. Restrictions on access to plant/equipment and occupancy time will be implemented to limit external exposure and annual doses to below guideline limits. Restrictions to site access by general public.	Uncertainty in the level and distribution of NORM concentrations across the Beetaloo Basin	Further monitoring and testing of gross alpha and beta activities and radionuclides (Ra-226, Ra-228, U-238, Th-232 and K-40) in flowback water and drill cuttings as project moves forward.
Miscellaneous	**Harm to workers and general public from exposure to ionising radiation from NORM in flowback water treatment waste residuals**	Internal exposure to NORM may result from direct contact and incidental ingestion of radionuclides leading to exposure to ionising radiation. This may occur during operations, maintenance, the transport of waste and contaminated equipment, the decontamination of equipment, and the processing and disposal of waste.	Standard radiation protection measures in line with ARPANSA guidelines and codes of practice will be implemented for the safe predisposal management and disposal of potentially 'radioactive' waste materials. Restrictions to site access by general public.	Uncertainty in the level and distribution of NORM concentrations in potential flowback water treatment waste residuals	Further testing of gross alpha and beta activities and radionuclides (Ra-226, Ra-228, U-238, Th-232 and K-40) in bottom sediments flowback water storage facilities and treatment waste residuals and their propensity to leach NORM as project progresses
	Hydraulic fracturing resulting in seismic activity due to the reactivation of geological faults or alteration of tectonic stresses	Opening of fractures within geological material in the surrounds of dormant geological faults, potentially resulting in fault reactivation.	Origin has a detailed understanding of the geological structures, faults and stress fields (**Appendix A, Section A 1.2.2.3**) within the project area, established using the following the techniques: • Development of a mechanical earth model (see **Appendix A, Section A1.4.3**), incorporating Origin's understanding of the local geological stress regime at each location to be hydraulically fractured. This understanding allows Origin to design hydraulic fracturing considerate of the local geological stress regime. • Seismic surveys (to inform understanding of subsurface geometric relationships and identification of geological structures).	No further assessment of risk is considered warranted, based on the risk mitigation measures in place. • The project area is one of the least seismically active areas on the Australian continent (see **Appendix A, Section A1.2.2.3**). • Geological faults within the area are considered to be dormant (see **Appendix A, Section A1.2.2.2**) • Each hydraulic fracturing operation is designed, considerate of the local geological stress regime.	Not required.

US EPA Nominated Key Activities and Factors for Hydraulic Fracturing Risk Assessment	Identified Potential Hazard	Hazard Causes and Consequences	Risk Mitigation Measures	Residual Risk	Recommended Mitigation to Address Residual Risk
	Harm to the environment due to the release of fugitive emissions from hydraulically fractured wells.	Release of potentially hazardous gases from the shale gas well at surface following the hydraulic fracturing of a well; e.g. if the hydraulic fracturing injection pressure were to open a micro annulus in the production casing annular cement seal, from the shale gas formation to surface.	Origin's Well Design protocols mandate the use of both surface and production casing sections. In the unlikely event that a micro annulus was opened in the production casing annular cement seal from the target shale to surface, any gas that migrated through that pathway would be contained by the wellhead which is fastened to the top of the surface casing, encapsulating the top of the production casing and production casing annular seal (see **Appendix A, Section A1.3.3**). To minimize the risk of stray gas migration and surface casing vent flows Origin designs its wells to have the surface casing landed below the CLA and the top of cement above the shallowest hydrocarbon bearing zone. Furthermore, all wells must comply to the integrity, maintenance and testing requirements outlined in Origin's Well Integrity Management System (WIMS). Annular pressures are monitored and reported as per WIMS requirements. Any surface casing vent flows would be identified by monitoring annular pressures.	Origin has in place procedures which: • Reduce the likelihood that wells leak gas through cement seals • Eliminate the pathway for gas to leak from the target shale to surface via the production casing cement seal (Origins Well Design protocols). These measures mitigate the risk associated with gas leaking from target shale to surface as a result of hydraulic fracturing.	Not required.
	Harm to residents or fauna/flora due release of dust from hydraulic fracturing activities	During the hydraulic fracturing process, certain fluid additives may be present which pose a human health risk due to inhalation due to dust generation. Crystalline silica (sand) is an example of a potential risk additive.	Chemical blending operations are carried out in closed system tanks, minimising exposure to workers onsite and the potential for creating windblown dust, which could impact residents or flora/fauna. With this control in place, the risk for dust created by hydraulic fracturing to harm residents or fauna/flora is considered as low.	Origin's work procedures (notably the blending of chemicals in closed-system tanks) removes the exposure pathway to workers, residents, flora and fauna, hence no further hazard assessment is warranted.	Not required.

7.2 Summary of Findings

The potential risks to human health and the environment associated with hydraulic fracturing in the project area was undertaken in accordance with enHealth, NEPM and ANZECC guidance, with reference to peer-reviewed international guidance where relevant.

Detailed operational procedures have been developed by Origin that are designed to contain the hydraulic fracturing fluids within the target shale formations, such that groundwater utilised for the purpose of extraction (and/or for any beneficial use), or springs are not impacted by hydraulic fracturing chemicals.

The risk of chemical exposure to workers involved in the operation of hydraulic fracturing activities (including the transportation of chemicals) was not quantitatively evaluated as this risk is addressed through the implementation of appropriate occupational health and safety procedures and management plans.

Potential exposures to hydraulic fracturing chemicals were therefore assessed to be limited to the above ground storage and handling of flowback water. Management of hydraulic fracturing fluid involves temporary storage of flowback water in above ground Flexiponds, after which the fluid will be transported to a disposal facility off-site. Exposure point concentrations were developed for each of the hydraulic fracturing fluid systems using a combination of 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) and empirical data obtained from laboratory analysis of flowback water from existing operations.

The toxicity of the chemicals used in the hydraulic fracturing process has been assessed for persistence, bioaccumulation and aquatic toxicity (PBT), and human health toxicity. The chemicals used in the hydraulic fracturing process do not contain BTEX or polycyclic aromatic hydrocarbons as additives. It is noted that most of the identified compounds (with the exception of MIT, CMIT, Tetrasodium EDTA, cyclicdimethylpolysiloxane compounds, acrylamide polymers, Dimethyl siloxanes and silicones , copper sulfate and dicoco dimethyl quaternary ammonium chloride) utilised in hydraulic fracturing fluids are not considered to be persistent, bioaccumulative or toxic in the environment. As such, the compounds are expected to degrade in the sub-surface and would not be expected to remain in the flowback water. Where these compounds are present in flowback water, they will readily degrade or dissociate in the environment and will not bioaccumulate in terrestrial or aquatic species. Polymers, EDTA, siloxanes and silicones have limited biodegradation potential; however, they are not bioaccumulative and are essentially non-toxic to aquatic species. While MIT and CMIT, copper sulfate and dicoco dimethyl quaternary ammonium chloride meet the screening criteria for toxicity (qualified by their acute toxicity capacity), adverse chronic outcomes are not evident based on their reactivity and lack of persistence and bioaccumulation in the environment. Cyclicdimethylpolysiloxane compounds (D4, D5 and D6) meet the criteria for being persistent, bioaccumulative and toxic in the environment. They are considered poorly soluble in water, volatile and will adsorb strongly to organic matter in sediment and soil. While D5 and D6 are not toxic to aquatic organisms, D4 causes toxicity in fish over chronic exposure durations.

Based on the operational controls, security and management practices implemented by Origin and the design of the Flexiponds, the potentially complete exposure pathways identified and assessed in the risk assessment comprised the following:

- Incidental ingestion and dermal contact by trespassers at the flowback fluid holding ponds; and
- Potential releases of water to aquatic environments.

Based on the risk calculations and with the consideration of the uncertainties identified, the following can be concluded in relation to potential exposures to flowback water from well hydraulic fracturing activities:

- No unacceptable risks to trespassers were identified. Conservative risk scenarios assessed included regular access to the uncovered Flexiponds, where swimming type activities were undertaken, with exposures to high theoretical concentrations of COPC in the flowback water.

- Risks to the environment are considered to be negligible or low. The likelihood that any of these chemicals may be discharged to an environment where any level of exposure may occur is managed via the implementation of existing operational management measures consistent with API guidance and 'Best Practice' specifications for the design and implementation of all hydraulic fracturing operations that are conducted for the project areas. The identified level of risk is supported by review of historic and existing operations where no accidental releases of flowback water from the holding ponds have occurred.

Effective operational controls and management implemented by Origin further minimises the potential for exposures and risk. These operational controls include:

- All relevant Australian standards are met during the drilling component of these wells.
- No unauthorised personnel allowed on site during hydraulic fracturing and well testing.
- Implementation of spill containment procedures during operations to prevent migration of and exposure to chemicals, and if a spill was to occur, rectification measures would be taken immediately and notification to regulators of any unauthorised releases will be made.
- Routine holding pond inspections to check for leakage of flowback fluid.
- Freeboard buffer included above high fluid level automatic pump shut-off system, in the event of heavy rainfall.
- Fencing and signage around the flowback storage ponds.
- Routine operational and security patrols to prevent trespassing.
- Off-site disposal to a licenced waste facility of all regulated waste.

The operational controls will be routinely evaluated by Origin and modifications and revisions made, where necessary. These operational and management controls can be further supported by the following recommendations:

- Maintain compliance with Origin's *Beetaloo Exploration Drilling Campaign 1 – Groundwater Monitoring Plan (NT-2050-15-MP-0011)*, which has been addressed to monitor the impact of hydraulic fracturing in the project area.

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Appendix A

Site Conceptual Model

A1.0 Conceptual Site Model and Issue Identification

A1.1 Location and Schedule

The Amungee NW-1H well is located approximately 540 km south-east of Darwin (16° 20' 51.034"S, 133° 53' 4.403"E (GDA 94)).

Hydraulic fracturing operations were carried out on Amungee NW-1H from July to September 2016.

A1.2 Receiving Environment

To conduct a risk assessment of hydraulic fracturing activities within the project area, an understanding of the surface and sub-surface environmental conditions is required. A conceptual model of the existing environment within the project area is developed in the subsequent sections of this report, to facilitate the assessment of the risks associated with the proposed hydraulic fracturing.

Furthermore, an understanding of the existing environmental values allows for the development of appropriate risk management systems through impact mitigation and minimisation and for the design of a robust hydraulic fracturing verification and validation (monitoring) program.

A1.2.1 Surface Setting

A1.2.1.1 Climate

The climate of the permit areas can be described as arid to semi-arid, with rainfall decreasing in frequency and quantity from north to south. The climate is monsoon influenced, with a distinctive wet and dry season experienced through the year. The area experiences a wet season during the summer months between October and March, which is dominated by hot and wet conditions. During the winter months, known as the Dry Season, from May to August, mild days and cool nights are experienced (Holt and Bertram, 1981). September and April are transitional months, with occasional rainfall. The average annual rainfall in the north of the permit area is listed at 680 mm at Daly Waters. The southern portion of the permit area records an average annual rainfall of 535 mm at Newcastle Waters and 608 mm listed at Elliott. Approximately 90% of the rainfall occurs during the Wet Season, and annual totals show moderate variability from year to year. **Table 1** presents the climate averages for Daly Waters, Newcastle Waters and Elliot.

The maximum rainfall for the permit area occurs during January and February. Daly Waters experiences the highest rainfall in the region at this time, with 165 mm during each month, followed by Elliott (133-164 mm during each month) and Newcastle Waters (125-130 mm during each month). July and August experience the least amount of rainfall and are the driest months across all three weather monitoring sites, ranging from one to four mm of rainfall. The annual rainfall pattern within the area is highly variable, and becomes increasingly unpredictable with distance from the coast. Drought conditions are known to occur in the region once every ten years (Holt and Bertram, 1981).

The maximum temperature in the permit area exceeds 30ºC from August to May. November is usually the hottest month with the temperature reaching up to 40ºC (**Table 1**). Minimum monthly temperatures range from 11 to 24ºC, with July being the coldest month.

Relative humidity and evaporation are highest during the summer months. The 9am maximum average for relative humidity ranges from 68% to 73% and occurs during February across all three monitoring sites. The maximum mean daily evaporation level is 8.8 mm at Daly Waters, 9.6 mm at Newcastle Waters and 9.8 mm at Elliott. The evaporation rate is high because of the high temperatures, moderate humidity, wind and (indirectly) the high number of sunshine hours that are experienced in the area (Holt and Bertram, 1981).

The prevailing wind in the permit area is from the south east; with winds blowing velocities of at 6 to 9 km/hr. Daly Waters is the only weather station to experience a change in wind direction from October to December, with winds blowing from the north east and commonly up to 6 to 7 km/hr (BOM, 2014).

A weather impact study was carried out in early 2015 to provide a better understanding of the likely timing of the onset and contraction of the monsoon season in order to optimise planning of operations.

The study aimed to assist in identifying the timing associated with undertaking activities with respect to NT weather patterns, as well as the susceptibility of the drill sites and access to flooding and soil integrity deterioration under moist conditions. Although the weather impact study focused on 2015 there were some findings that are still of relevance to the program and have therefore been included below.

The following findings from the study are of general relevance to the project:

- The climate of Northern Australia is dominated by the dry and wet or monsoon seasons. Conducting activities during the wet seasons can be difficult if not impossible at times as a result of flooding and generally wet ground conditions that result from poor natural drainage throughout much of Northern Australia. Therefore, for transport operators, the timing of the onset of the monsoon can be critical for planning operations.
- Seasonal rainfall variation and variation in the onset date of the monsoon in the Beetaloo region was assessed. The long term average monsoon onset date was found to be 25 November, based on the commonly applied criteria. The influence of ENSO on the monsoon onset was investigated, and a general trend of early monsoon onset during La Niña episodes and late monsoon onset during El Niño episodes was observed. During La Niña the monsoon tends to start during early October to late November, whereas during El Niño the monsoon tends to start from late November to early January.
- The soil integrity at Amungee NW-1 is likely to deteriorate earlier in the monsoon than other sites due to the higher fines content in the soil at Amungee NW-1.
- Sustained rainfall of the monsoons, the soils may become saturated and result in significant soil integrity deterioration at all sites.
- The well site and the short access road for Amungee NW-1H is not likely to be impacted by flooding of adjacent major flow paths and creeks.

Table 1 Average Climate at Daly Waters, Newcastle Waters and Elliott

Month	Daily Maximum Temperature (ºC)			Daily Minimum Temperature (ºC)			Mean Monthly Rainfall (mm)			Relative Humidity (%)#			Mean Daily Evaporation (mm)			Wind Speed (e)#			Wind Direction#		
	DW	NW	E	DW	NW	E	DW	NW	E	DW	NW	E	DW	NW	E	DW	NW	E	DW	NW	E
January	36.6	37.3	37.6	24.0	24.1	24.1	165.4	125.5	133.1	69	63	65	7.1	8.2	7.9	5.9	8.8	5.5	NW	NW	NW
February	35.5	35.6	36.6	23.4	23.7	23.7	165.4	130.9	164.0	73	68	70	6.0	7.0	7.3	4.8	9.4	5.8	NW	E,SE	W
March	34.6	34.7	35.6	22.4	22.6	22.2	120.1	93.7	85.9	70	62	64	5.9	6.3	7.3	4.5	11.0	6.9	SE	E,SE	E
April	33.7	33.3	34.4	19.3	19.4	19.6	23.6	24.6	23.0	53	54	50	6.2	6.4	7.5	6.7	14.0	9.2	SE	E,SE	SE
May	31.4	30.0	31.3	15.8	16.1	15.9	5.0	9.3	7.2	50	51	47	5.9	5.1	6.5	7.8	16.7	10.4	SE	E,SE	SE
June	28.7	27.7	28.4	12.9	12.7	12.4	5.6	5.3	4.6	50	51	46	5.5	4.4	5.6	7.5	15.2	11.6	SE	E,SE	E
July	28.9	27.5	28.3	11.8	11.3	11.2	1.5	3.4	2.8	47	47	42	5.6	4.6	5.7	7.8	15.0	10.8	SE	E,SE	SE
August	31.9	30.8	31.3	13.5	13.7	13.2	1.7	1.0	1.1	44	42	37	6.6	6.2	6.7	6.6	15.3	11.2	SE	E,SE	SE
September	35.0	34.1	35.4	17.2	17.4	17.4	4.9	5.4	5.9	43	41	36	8.4	8.2	8.4	7.8	14.2	11.4	SE	E,SE	E
October	37.7	37.1	38.0	21.1	21.2	20.9	22.5	20.9	22.4	45	42	41	8.8	9.1	9.4	7.8	14.5	11.2	SE	N	N
November	38.4	38.4	39.1	23.5	23.3	23.3	59.4	35.7	49.0	52	43	45	8.5	9.2	9.5	6.7	12.5	8.6	NW	N	NW
December	38.2	38.5	38.6	24.0	24.1	24.4	110.0	77.3	95.7	59	51	56	8.1	9.1	8.7	5.5	10.1	6.5	NW	N	N
Annual							680.5	535.4	608.2												
Minimum	28.7	27.5	28.3	11.8	11.3	11.2	1.5	1.0	1.1	43	41	36	5.5	4.4	5.6	4.5	8.8	5.5			
Maximum	38.4	38.5	39.1	24.0	24.1	24.4	165.4	130.9	164.0	73	68	70	8.8	9.2	9.5	7.8	16.7	11.6			
Average	34.2	33.8	34.6	19.1	19.1	19.0	57.1	44.4	49.56	54	51	50	6.9	7.0	7.5	6.6	13.1	9.1			

DW – Daly Waters, NW – Newcastle Waters, E – Elliott, # data recorded at 9am daily, - No data recorded.
Data sourced from Bureau of Meteorology, *Climate Averages and Wind Frequency Analysis for Station 014618 Daly Waters recorded from 1873-2013, 015089 Newcastle Waters recorded from 1941-1980 and 015131 Elliott recorded from 1949-2014*.

A1.2.1.2 Topography

The Beetaloo project area is located within three main topographic zones. These are primarily made up of black soil plains in the south, laterite plains in the north and small sections of bedrock hills in the south west and north east of the permit areas (Tickell, 2003). **Figure 1** shows the topographic contours for the project area.

The black soil plains are primarily located within the Barkly Tablelands, which covers EP76 and a small section of EP98. The Barkly Tablelands are dominated by a gently undulating to flat landscape with relatively low relief. The lowest area, Lake Woods, lies outside of the south west limit of the permit area near Newcastle Waters, at approximately 200 m above sea level. Away from Lake Woods the plains gently increase to between 220 m and 250 m above sea level.

The laterite plains, which were formed by laterite capping on Cretaceous aged sedimentary rocks, are located within EP98 (Tickell, 2003). A small section of laterite plains extends from Beetaloo to Mungabroom and is separated from the northern laterite plains by a 6 to 8 km line of black soil plains. The northern laterite plains are approximately 250 m to 280 m above sea level and located along the drainage divide that separates inland drainage from the north flowing streams (Nutwood Downs), which lead into the Gulf of Carpentaria. From there, the land gently slopes towards the south and south west (Tickell, 2003).

The bedrock hills occur adjacent to Amungee Mungee in the eastern border of EP98, near the Carpentaria Highway. The maximum elevation in this area is slightly higher than 300 m above sea level and drains north into the Arnold River (Tickell, 2003).

The elevation of Amungee NW-1H is approximately 263 mAHD.

A1.2.1.3 Surface Water and Drainage

The Beetaloo project area is located within three main drainage basins (Randal, 1967). These include the Barkly, Roper and Wiso Basins (**Figure 2**). Inland drainage occurs in the Barkly Basin and parts of the Wiso Basin.

The Wiso Basin covers the majority of the permit area EP98 (southern half of area) and is drained by the Georgina River and its major tributaries (Ranken, James, Buckley and Woodroffe Rivers). The Georgina River flows south into Queensland and eventually reaches Lake Eyre in South Australia. These rivers only flow for short periods in the wet season. They are often braided when flowing through soft alluvia, but are restricted to one channel when crossing areas of resistant rock, such as the limestone country on Austral Downs (HLA, 2005).

The Barkly Basin covers the southern part of the study area within EP76 and EP117. Some areas in the southern desert country have little surface drainage. The remainder is drained by short streams which peter out or empty into local drainage basins, usually bluebush (*Chenopodium auricomum*) swamps. The major streams are the Buchanan, Gosse and Playford rivers and Creswell, Newcastle, Puzzle, Phillip, Tennant and Brunette Creeks. The streams flow for only short periods during the wet season. If the wet season is poor, the swamps often remain dry, whereas, during heavy wet seasons, large areas of the internal drainage systems are flooded. The streams area often lined with scattered small trees.

Most existing dams and potential dam sites lie on these internal drainage channels, which drain to Lake Woods, which is located south of Newcastle Waters Station. Lake Woods covers an area of inundation of approximately 50,000 ha in normal rainfall years, extending to 80,000 ha in exceptionally wet years, after which it can retain water for several years (HLA, 2005).

Surface water quality in the permit area has previously been investigated (HLA, 2006a; 2006b), and it was concluded that the local surface waters carry a dominance of calcium, magnesium carbonate and bicarbonate, which is in contrast to generally held views of inland waters, which generally show sodium chloride dominance of ionic composition. The 2006 monitoring programme also revealed a strong surface-aquifer influence on the persistence of ephemeral water-bodies within areas of the permit.

Figure 1 Topography

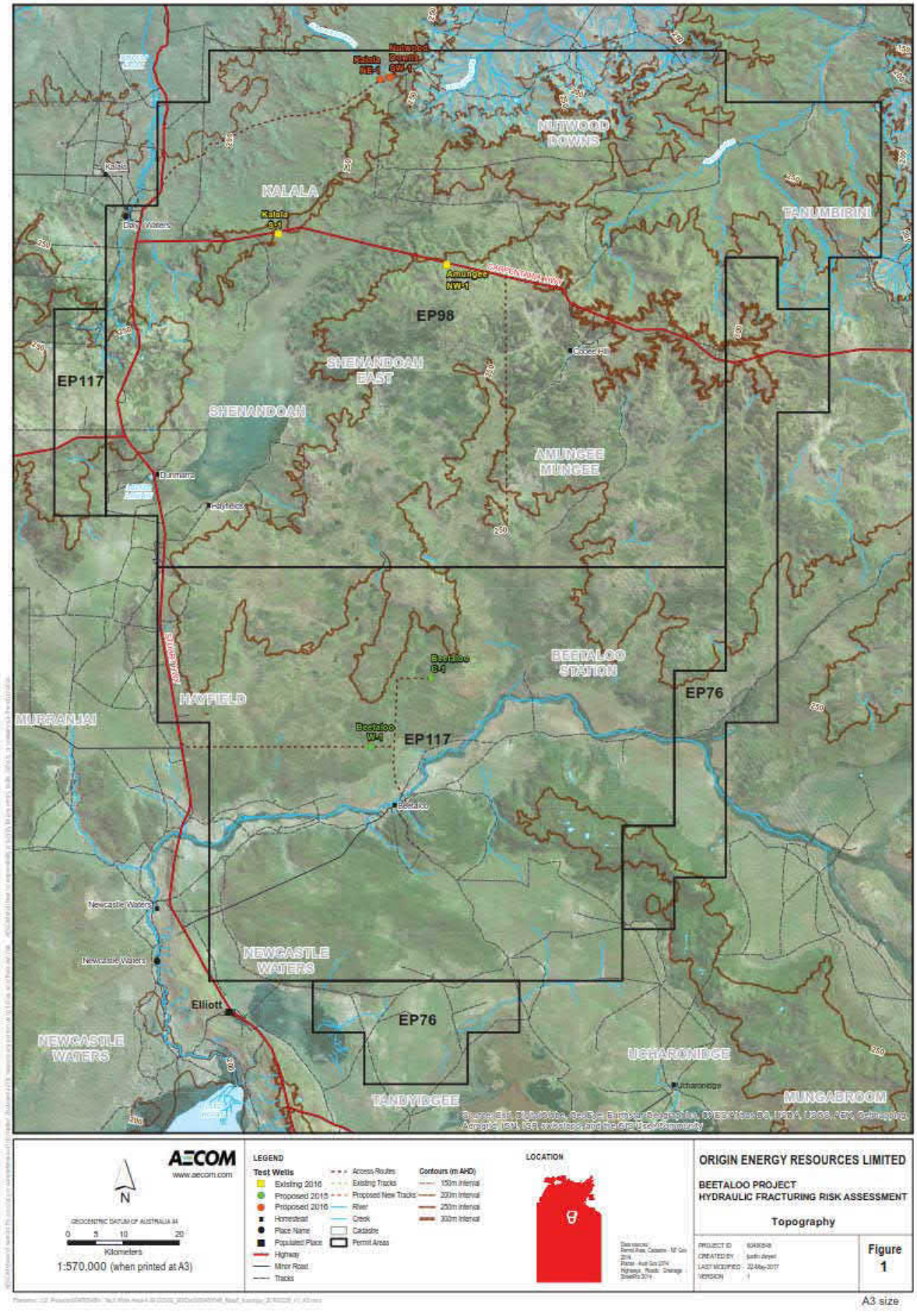
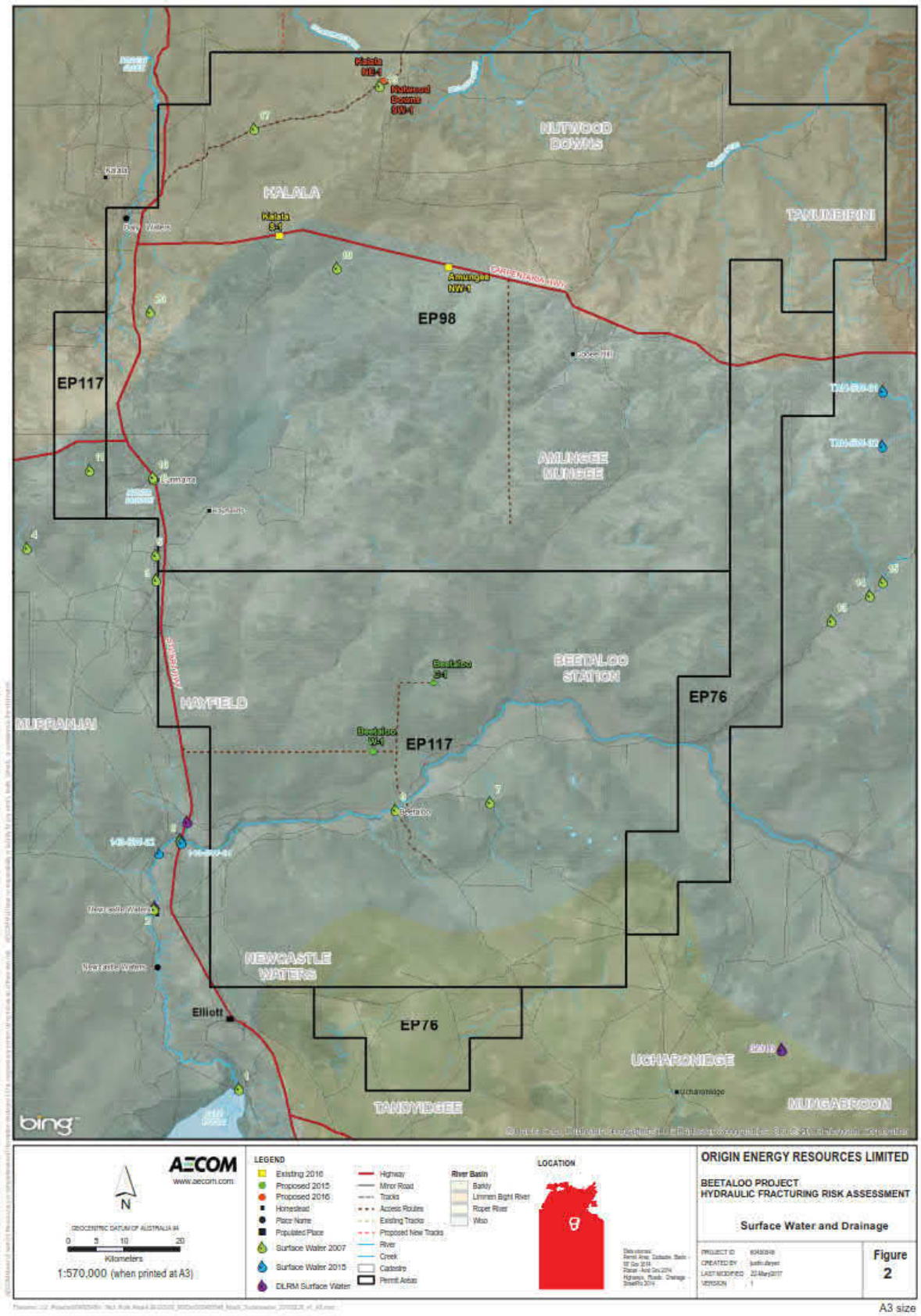


Figure 2 Surface Water and Drainage



A1.2.2 Geological Setting

A.1.2.2.1 Geology

The geology within and surrounding the Beetaloo project permit areas (**Figure 3**) was primarily formed over three main periods – the Precambrian (> 550 million years ago), the Cambrian (500 million years ago) and the Cretaceous (100 million years ago).

Pre-Cambrian rock formations, known as the Roper Group, are located at depth across the permit areas, beneath the younger formations, and are exposed only in the bedrock hills located to the north east of EP98 (Tickell, 2003).

Cambrian formations are expressed only in the south west region of the study area. They predominantly fall outside of the identified permit areas, and comprise of limestone, siltstone and sandstone. The rock formation is near flat, rarely cut by faults and forms distinct layers. The Cambrian sediments contain the sub-artesian water storage, pedocalcic soils, Cambrian dolomite, limestone, and tertiary alluvium (Tickell, 2003).

The majority of the permit area is located within the Beetaloo Sub-basin, a sub-basin of the McArthur Basin (**Figure 4**), which was formed during the Mesoproterozoic period, over 1,000 million years ago. Soft clays and sandstone are the primary rock formation in the basin and overlie the older Pre-Cambrian and Cambrian rocks. Small and patchy occurrences of freshwater limestone accumulations, formed during the Miocene Period (15 million years ago) when erosion and the gradual sinking of some areas produce isolated fresh-water lakes.

Following the deposition of Cretaceous sediments, a period of geomorphic activity occurred during the Tertiary period. This resulted in the area, being gently folded and warped, which exposed it to a long period of erosional forces (Christian et al., 1951). These forces resulted in the area being dominated by undulating plains that contain extensive swampland and lakes.

Following a period of lateritization during the end of the Tertiary period, rivers were at grade and erosion was reduced to a state that allowed deep stable soil profiles to be established and be preserved (Christian et al., 1951), resulting in the ‘black’ soil clay plains and the lateritic and non-lateritic rises that are in the region today (Randal, 1967). With the onset of a more arid climate during the post–Miocene period, lakes and swamps dried up, resulting in high concentrations of lime and silica deposits that were leached from the lateritic soils into the ground and surface waters, which in turn formed a number of Tertiary limestone outcrops within the permit area. During the Quaternary period, which occurred less than 2 million years ago, the minor alluvial and lake deposits throughout the permit area were formed.

The Beetaloo Sub-basin stratigraphic column is presented in **Plate 1**.

Plate 1 Beetaloo Sub-Basin Stratigraphic Column

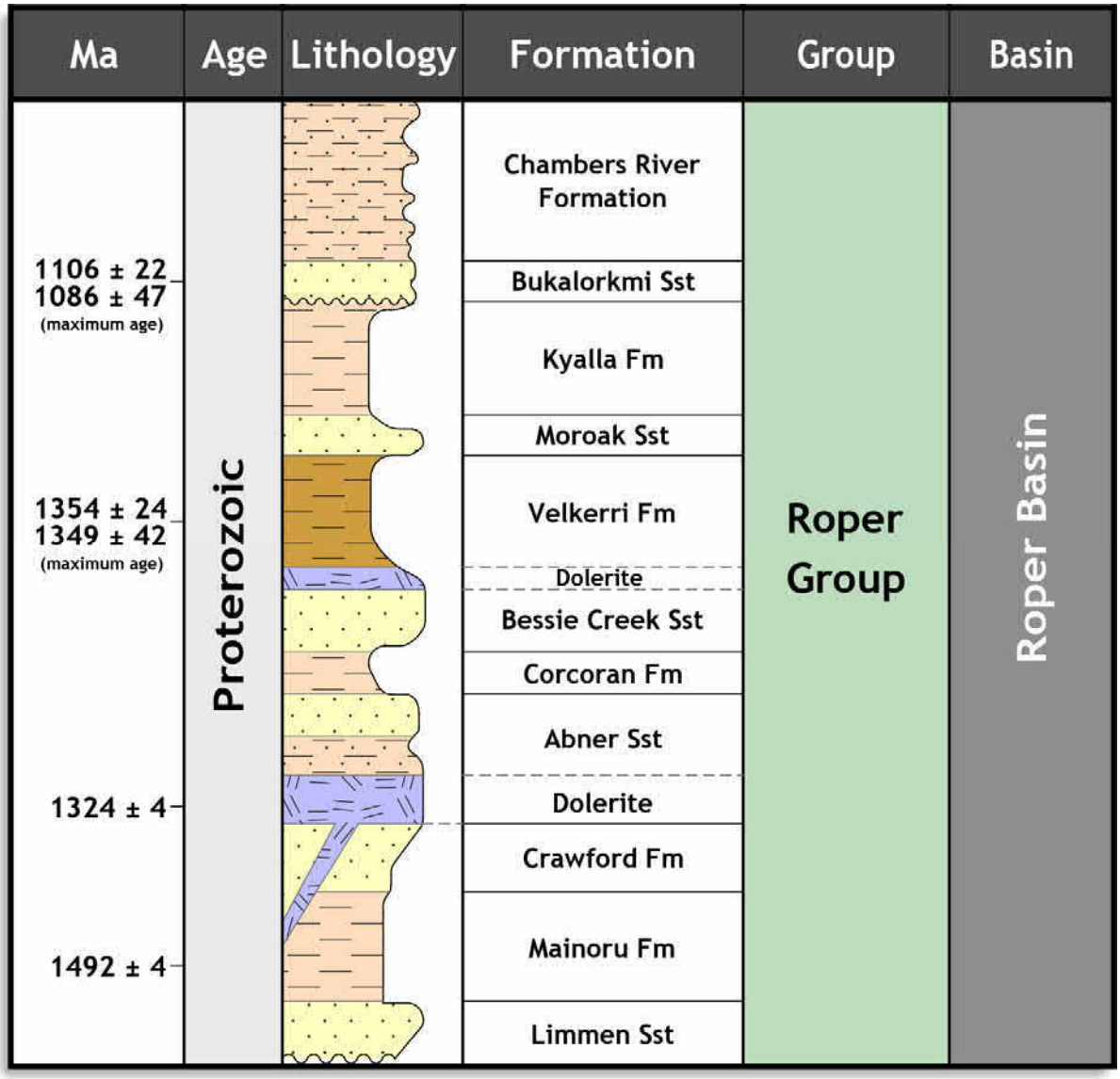


Figure 3 Surface Geology

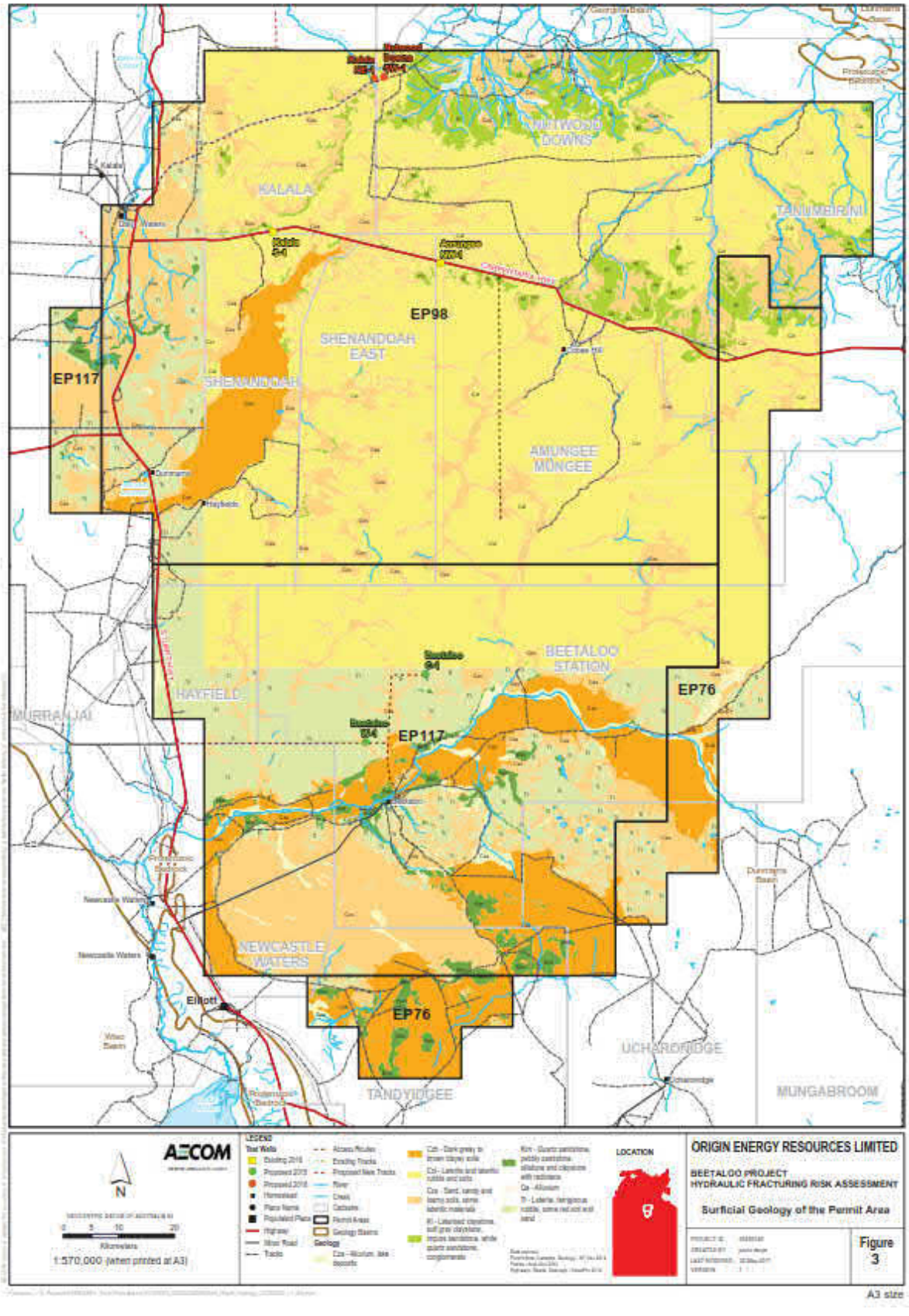
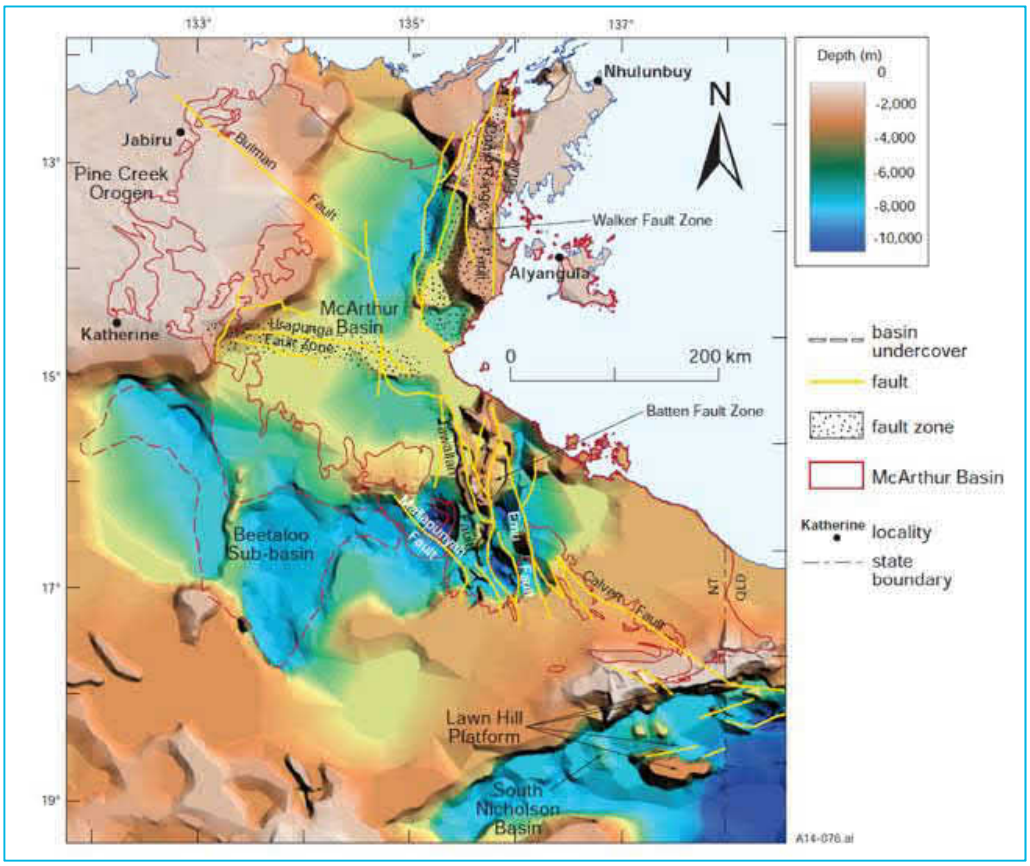


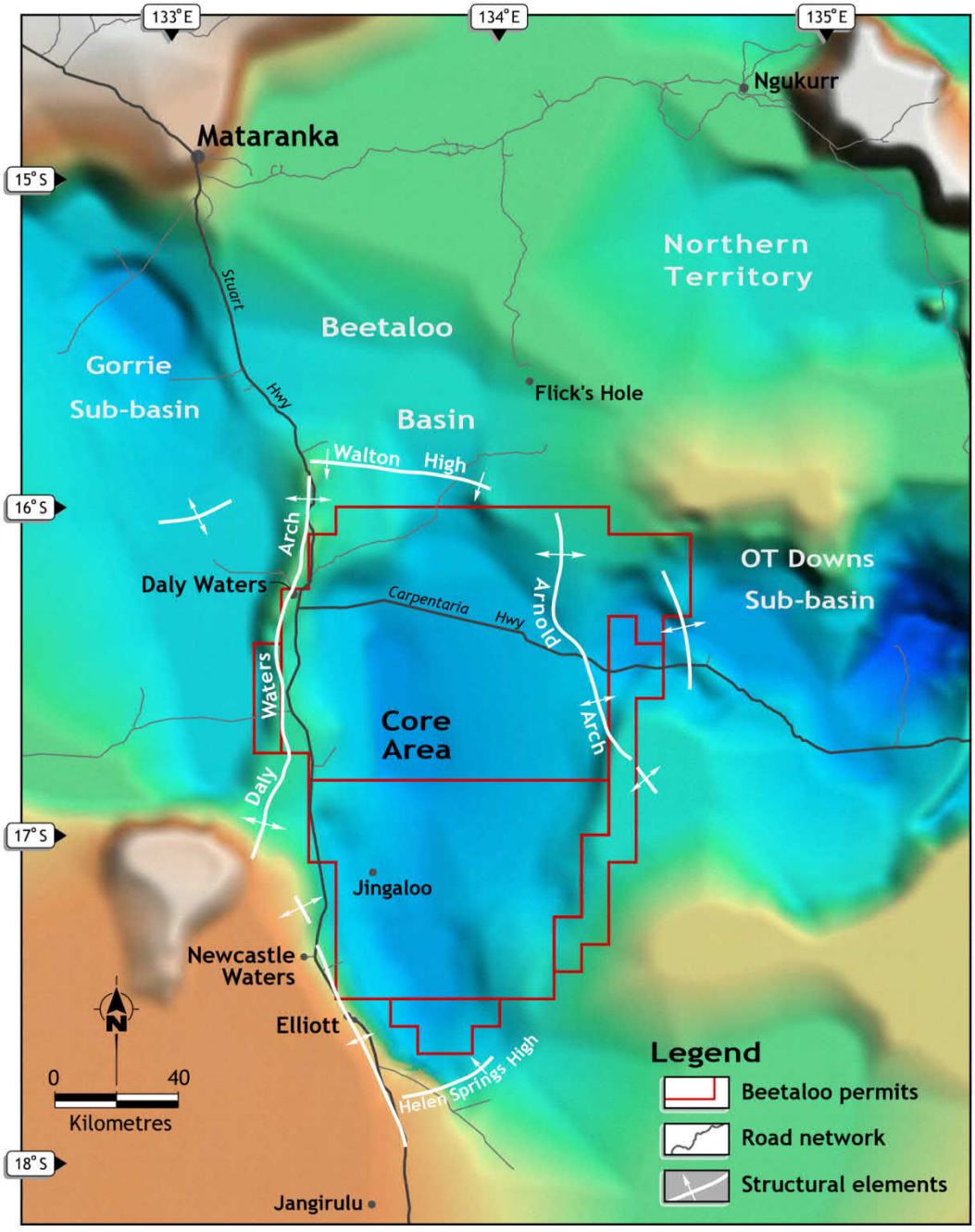
Figure 4 Interpreted Beetaloo Sub-Basin within the McArthur Basin based on OZ SEEBASETM depth-to-basement image (Revie, D. 2017)



A1.2.2.2 Structural Geology

The Beetaloo Sub-basin is structurally subdivided into three geographical areas and two major structural highs. The north-south trending, structurally complex Daly Waters Arch (west) and structurally benign Arnold Arch (east) divide the area in three major depocenters, referred to here as the Gorrie Sub-basin, the Core area and the OT Downs Sub-basin from west to east respectively (**Figure 5**). Early compression and strike-slip movement are thought to be responsible for the development of these structures (Silverman et al., 2007).

Figure 5 Structural divisions within the Beetaloo Sub-Basin based on OZ SEEBASETM depth-to-basement image (figure provided by Origin)



A1.2.2.3 Seismic Geophysics History of the Project Area

In the context of the McArthur Basin, the strata within the Beetaloo Basin appears to have undergone only mild tectonism after deposition as evidenced by the flat-lying character of the upper Roper Group in seismic profiles (**Figure 6** to **Figure 9**) and the faults mapped within the region (**Figure 10**). The nature and timing of structural events during and postdating Roper Group deposition appear dominated by two major compressional/transpressional episodes defined by truncation beneath the base of the Bukalorkmi Sandstone and base of the Cambrian Unconformity; the former more evident over structural highs such as the Arnold Arch whereas the latter shows a flatter and regionally extensive character. Truncation at the base of the Bukalorkmi Sandstone followed by the Precambrian/Cambrian unconformity suggests the present day shape of the Beetaloo Basin is a large post-depositional feature which contains the remnant of an original, more extensive basin. Throughout

the Phanerozoic, tectonic/structural activity in the area was limited to regional events including broad tilting and relative uplift towards the north of the basin (Lanigan et al., 1994).

Faulting and folding occurs primarily within discrete zones up to 10 km wide likely as a result of reactivation of deep-seated faults and subsequent alignment of younger structures. Fault zones largely consist of wrench-related strike-slip and reverse faults producing en echelon, antiformal (positive) flower structures (Lanigan et al., 1994). Due to limited regional tectonic activity during the Cambrian period, the Antrim Plateau Volcanics and Gum Ridge Formation formed a flat-lying drape over the more structured Proterozoic strata. Exceptions occur within the north-western and eastern margins of the Basin where faults extend into the Cambrian sequence (Fulton and Knapton, 2015).

Figure 6 North to South seismic composite section through the Core area of the Beetaloo Basin (Origin, 2016)

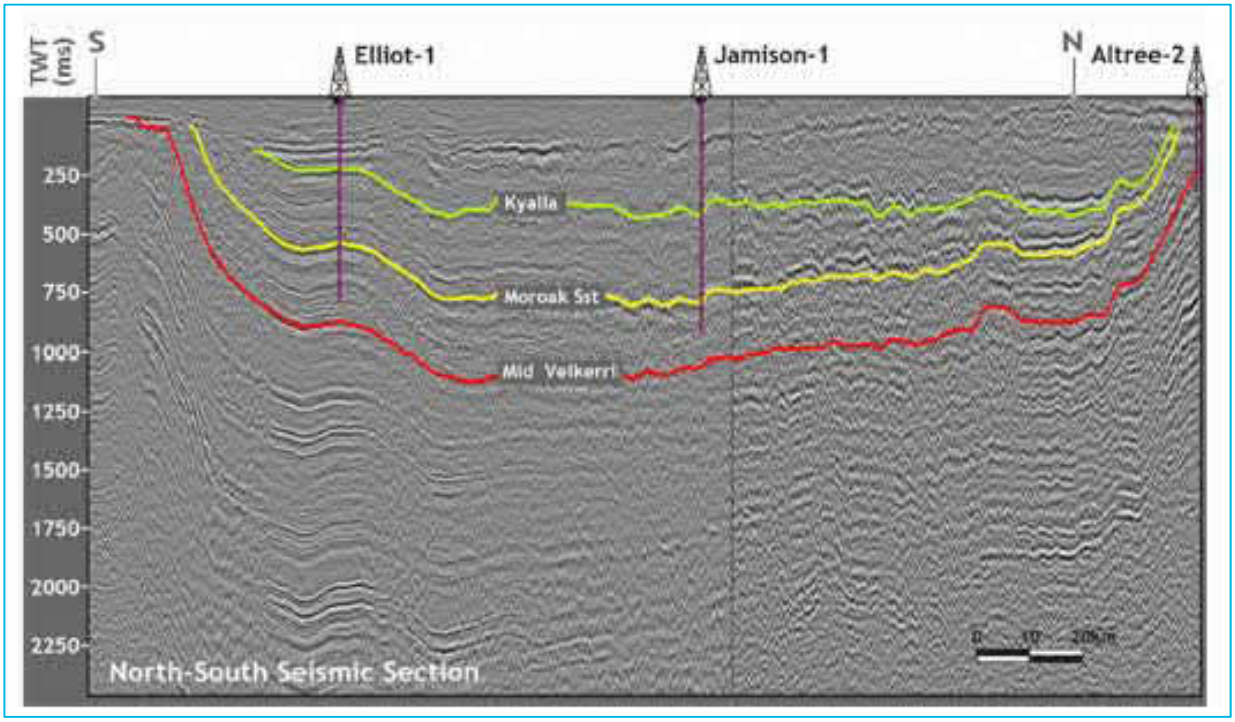


Figure 7 North to South geological cross section through the Core area of the Beetaloo Basin (Fulton and Knapton, 2015)

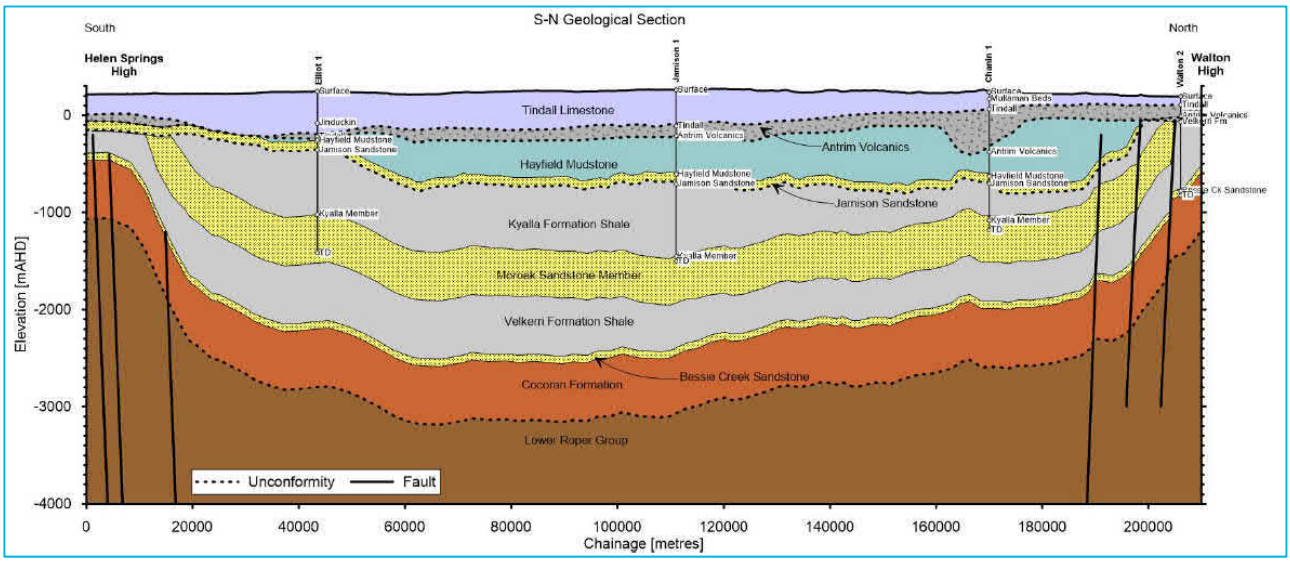


Figure 8 West to East seismic composite section through the Core area of the Beetaloo Basin (Origin, 2016)

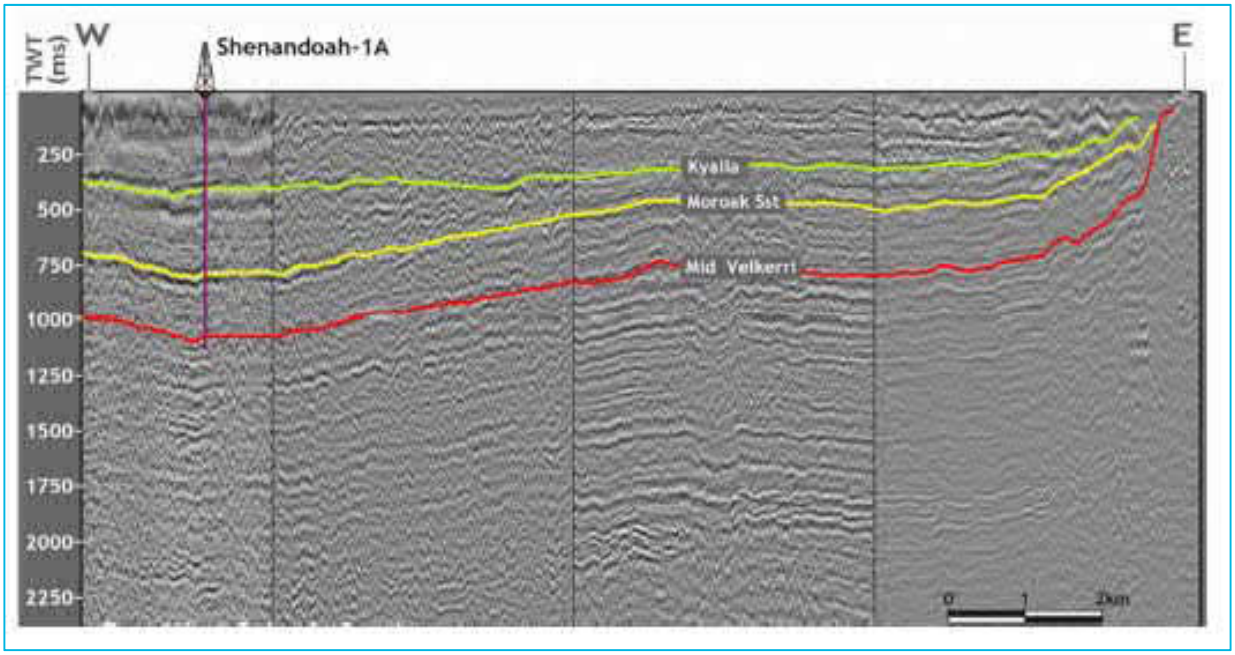


Figure 9 West to East seismic composite section through the Core area of the Beetaloo Basin (Fulton and Knapton, 2015)

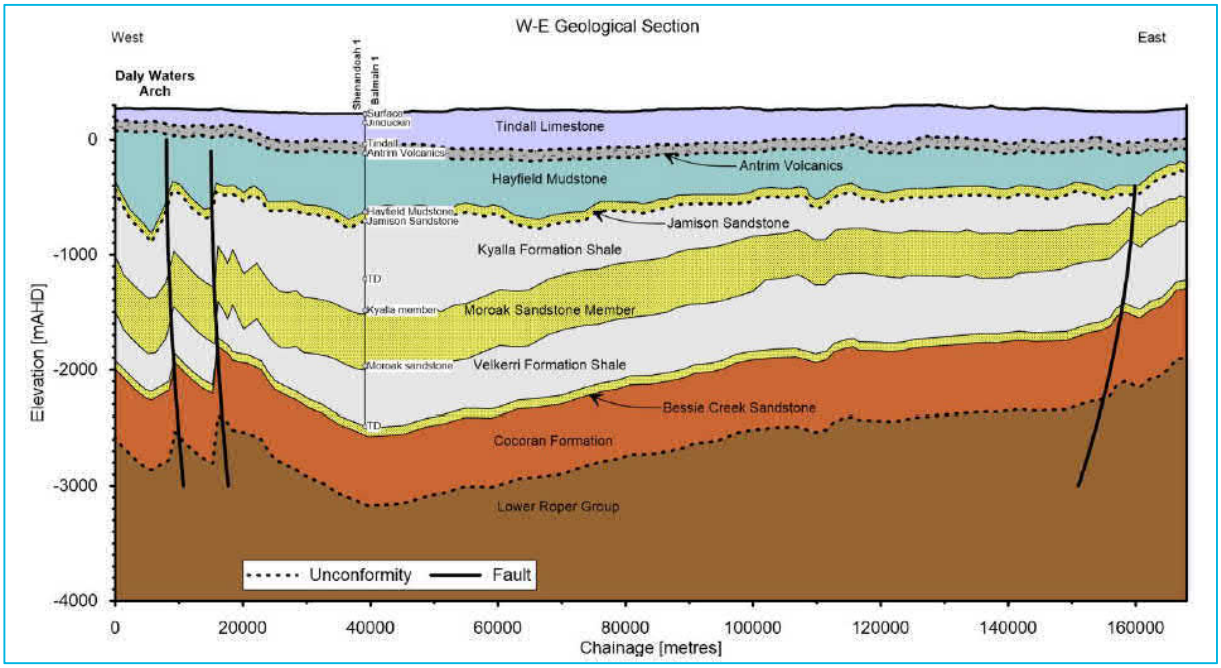
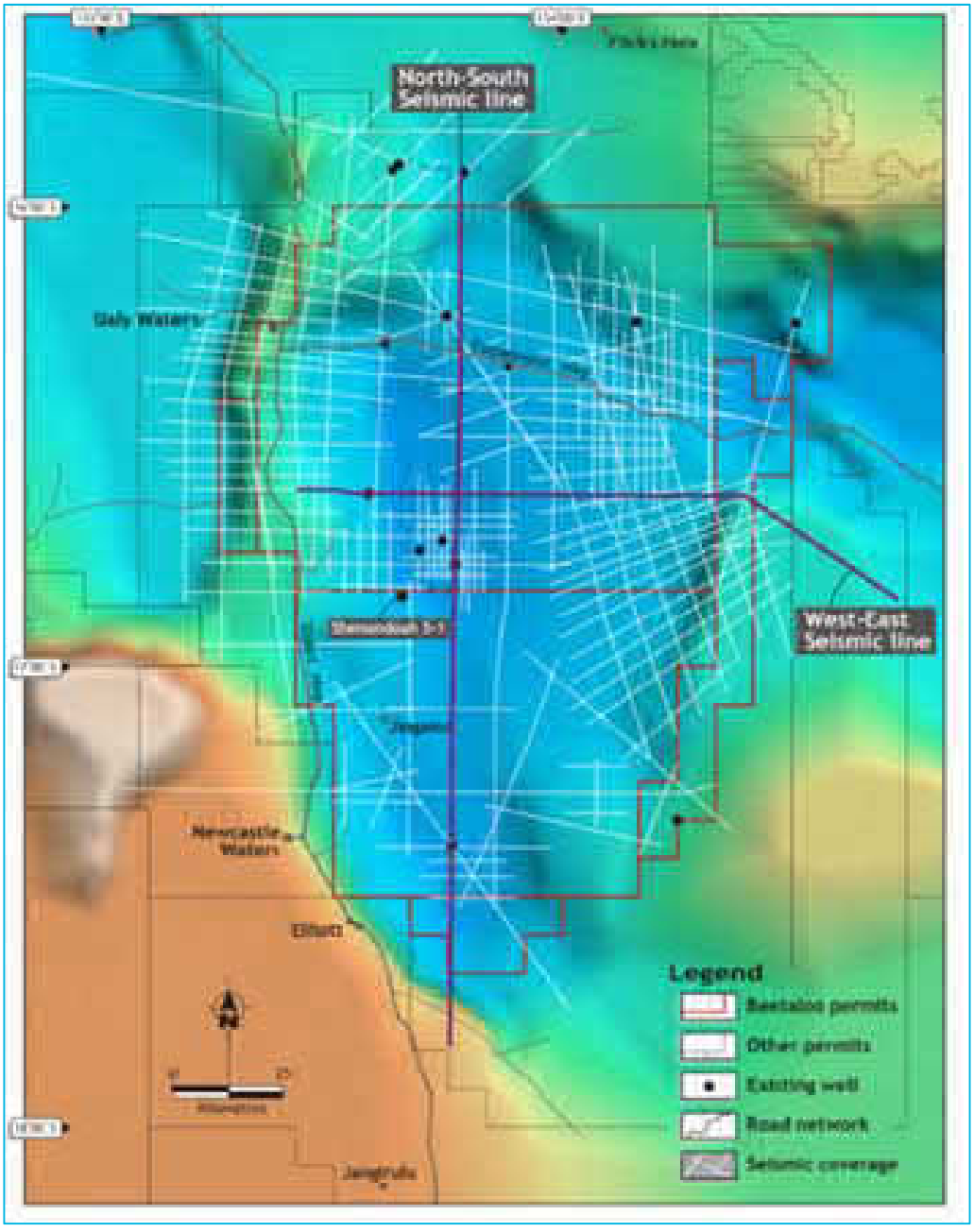


Figure 10 Map showing location of 2D seismic composite sections from Figure 6andFigure 8



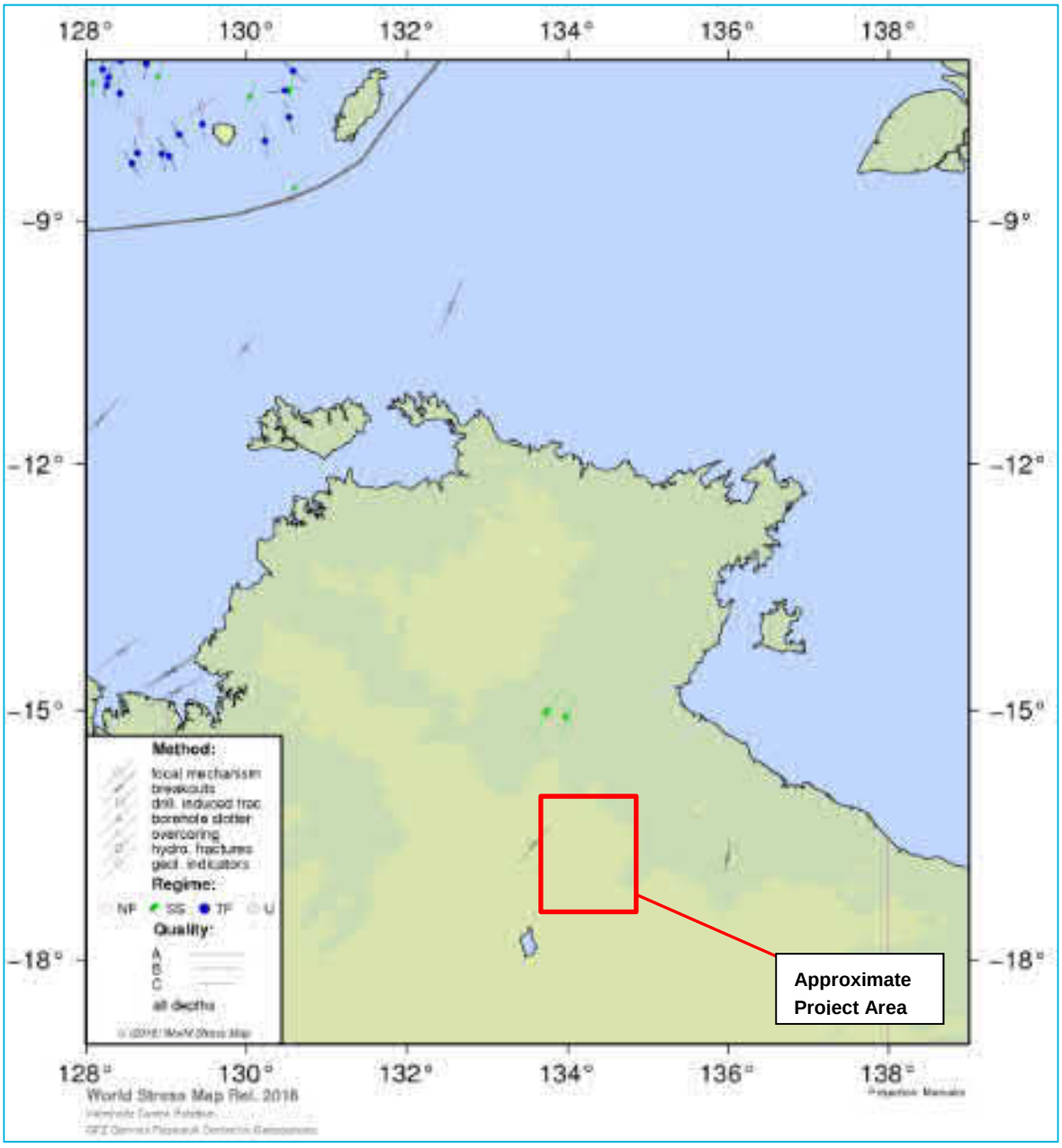
A1.2.2.4 Stress-Field Setting

At depth, geological formations are compressed by the weight of overlying strata and laterally constricted by the adjacent geological material. These compressive forces create stresses within the formation in three dimensions; defining a major stress field direction, an intermediate stress field direction, and minor stress field direction. The orientation and magnitude of the stress fields is of relevance to hydraulic fracturing as they influence the direction of hydraulically-induced fracture propagation.

On a regional scale, Australia forms part of the Indo - Australian tectonic plate which undergoes absolute movement of approximately 7 cm per year north-northeast (N-NE). This is reflected in the general N-NE to S-SW orientation of the regional major horizontal stress field in the Northern Territory, as depicted on the Australasian Stress Map, **Figure 11**. The minor horizontal stress field is approximately perpendicular to the major horizontal stress direction; that is, regionally in the Northern Territory the minor horizontal stress is typically oriented in an N-NW to S-SE direction.

On the project area scale, stress fields within in the targeted shale (Velkerri Formation) are affected by the local structural geology such as faults and folds. The propensity of geological material to fracture under stress (either natural or applied) is dependent on the physical properties of the rock material.

Figure 11 Project Area Stress Map



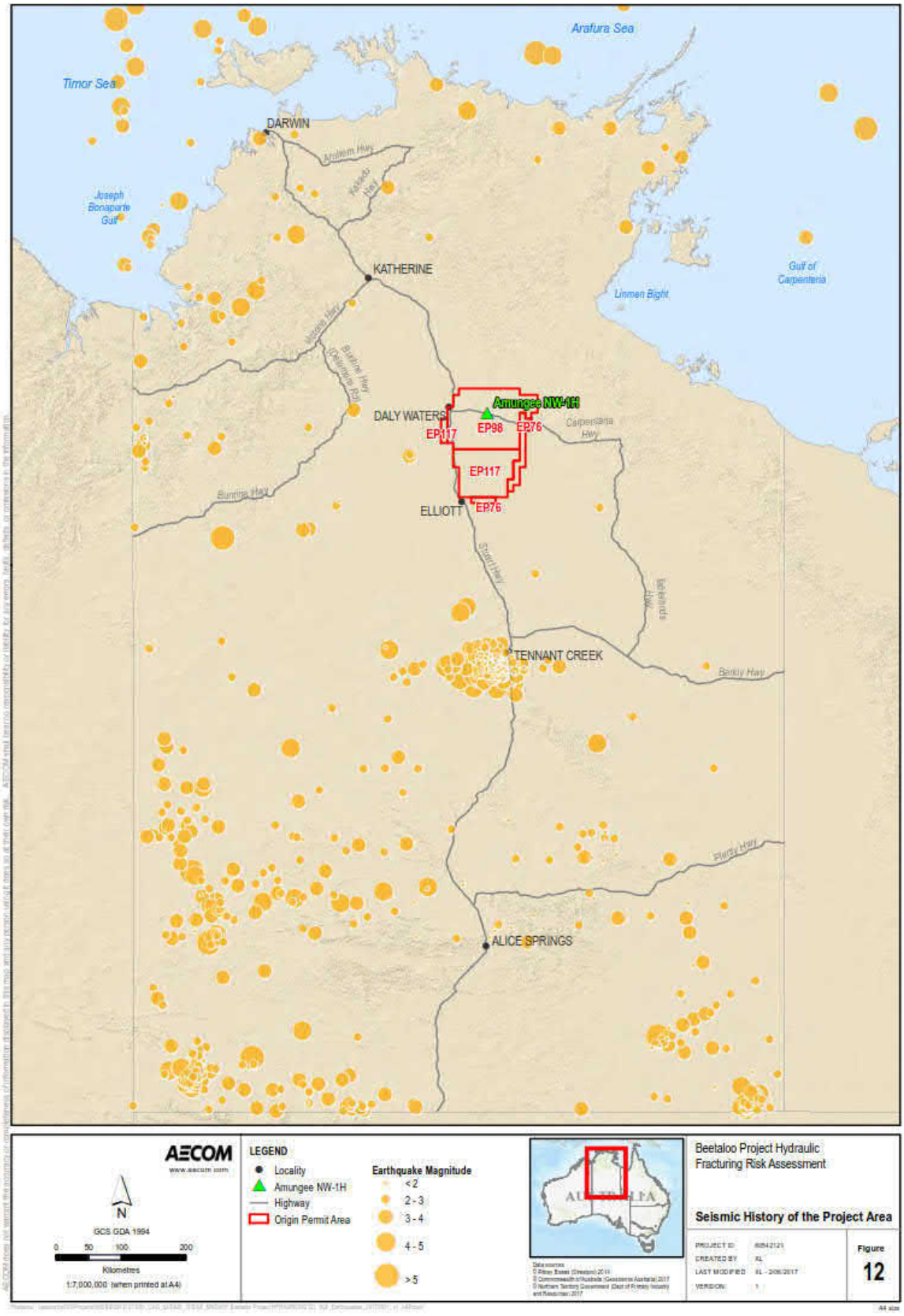
A1.2.3 Regional Seismic History

Earthquakes in the northern Australia are generally caused by the release of built-up stress in the Indo-Australian continental plate, as a result of its drift N-NE.

The minimal number and magnitude of seismic events in the surrounds of the project area (see **Figure 11**) suggest the area currently has low seismic activity. No seismic events have been recorded within the Beetaloo Project permit areas.

Within the vicinity of the project area, there are no neotectonic geological faults (GA, 2017), which are faults which have been active within the 5-10 million years and are therefore considered capable of hosting future displacement/re-activation.

Figure 12 Seismic History of the Project Area



A1.2.4 Hydrogeological Setting

The Beetaloo Basin comprises a thick sequence of flat-lying mudstone and sandstone formations (Roper Group) that were deposited between 1,500 and 1,430 million years ago (**Table 2**). The Roper Group is estimated to reach of 5,000 m in thickness in the centre of the basin and with the exception of the north and eastern margins occurs at an average depth of about 500 m. The Roper Group is overlain by the Georgina Basin (630 – 497 Ma), which includes widespread basalts and a thick limestone sequence that forms the Cambrian Limestone Aquifer (CLA), a significant water supply aquifer. The Georgina Basin is capped by Cretaceous mudstone and sandstone (145 – 66 Ma) and recent alluvial and laterite deposits.

Table 2 Summary of Beetaloo Basin Hydrostratigraphy (Fulton and Knapton, 2015)

Province	Period/Age	Formation		Aquifer Status	Reported Thickness (m)	Yield (L/s)	Ave EC (µs/cm)	Transmissivity (m^2/day)
CARPENTARIA BASIN	CRETACEOUS 145 – 66 Ma	Undifferentiated		*Local Aquifer*	0 - 130	0.3 - 4	1,800	41
GEORGINA BASIN	CAMBRIAN 497-630 Ma	Cambrian Limestone Aquifer (CLA)	Anthony Lagoon Beds	REGIONAL AQUIFER	0 – 200	1 - 10	1,600	13-1400
			Gum Ridge Formation	REGIONAL AQUIFER	0 – 300	0.3 - >20	1,400	3-3377
		Antrim Plateau Volcanics		REGIONAL AQUITARD	0 – 440	0.3 - 5	900	0.8-17
		Bukalara Sandstone		*Local Aquifer (not regionally connected)*	0 – 75	0.3 - 5	1,000	17-78
BEETALOO BASIN (ROPER GROUP)	NOT KNOWN	Hayfield Mudstone		REGIONAL AQUITARD	0 – 450	-	32,000	-
		Jamison Sandstone		*Local Aquifer (not regionally connected)*	0 – 150	-	138,000	-
	MESO-PROTEROZOIC 1,430-1,500 Ma	Kyalla Formation		REGIONAL AQUITARD	0 – 800	-	-	-
		Moroak Sandstone		*Local Aquifer (not regionally connected)*	0 – 500	0.5 - 5	131,000	-
		Velkerri Formation		REGIONAL AQUITARD	700 – 900	-	-	-
		Bessie Ck Sandstone		*Local Aquifer (not regionally connected)*	450	0.5 - 5	-	-

The following formation descriptions have been taken from the *Beetaloo Basin Hydrogeological Assessment* (Fulton and Knapton, 2015).

A1.2.4.1 Undifferentiated Cretaceous Sediments

In the Beetaloo Basin the Carpentaria Basin is represented by an undifferentiated sedimentary sequence, previously referred to as the Mullaman Beds but now not formally named (Ahmed et al, 2013). The sequence is divided into three informal units. The basal “A” unit comprises massive, quartz sandstone which reaches a maximum thickness of 37 m (Browne and Randal, 1969). Unit “B” consist of a thin fossiliferous siltstone layer which is overlaid by a fine grained sandstone, the total thickness of the unit is approximated at 10 m (Skwarko, 1966). The uppermost unit “C” comprises claystone with minor lenses of sand, it has a reported thickness of up to 60 m (Browne, 1969). Drillhole data from water bores and exploration wells indicate the total thickness of the Cretaceous sequence within the Beetaloo Basin ranges from 0 - 132 m with an average thickness of 50 m. The undifferentiated Cretaceous sequence forms a continuous veneer across the Beetaloo Basin with the exception of the south west margin. The sequence unconformably underlies undifferentiated Quaternary and Tertiary units.

A1.2.4.2 Cambrian Limestone Formation (Gum River Formation and Anthony Lagoon Beds)

Gum River Formation

The Gum Ridge Formation and Anthony Lagoon Beds are broadly grouped as the Cambrian Limestone Aquifer (CLA) (Fulton, 2009a), a significant regional aquifer system comprising fractured and karstic rocks that extends across the Georgina, Wiso and Daly Basins. Yin Foo (2002) describes the Gum Ridge Formation and Anthony Lagoon Beds as distinct hydrostratigraphic units in the Sturt Plateau region, however the recent construction of a nested piezometer site (RN36471) by DLRM south of Dunmarra and aquifer testing completed in the Helen Springs region (Rooke, 2002) suggest there is a level of vertical connection across the Cambrian Limestone sequence.

The Gum Ridge Formation and its lateral equivalents in the Wiso Basin, the Montejinni Limestone and Tindall Limestone, comprise an extensive aquifer system that forms the principle water resource in the northern half of the Beetaloo Basin; in the south of the basin the Gum Ridge Formation occurs at depth beneath the Anthony Lagoon Beds. The Gum Ridge aquifer is locally unconfined where it abuts outcropping Proterozoic Formations in the north8east of the Beetaloo Basin and also in the northwest on Avago, Sunday Creek and Birdum stations where the limestone sequence thins and has limited saturated thickness over the Antrim Plateau Volcanics. The Gum Ridge is confined by Cretaceous mudstones in the north and central Beetaloo Basin and by lower permeability layers within the Anthony Lagoon Beds in the south.

The aquifer comprises massive grey limestone beds that are heavily fractured and commonly karstic. Formation permeability is highly dependent on the development of these dissolution and fracture features. A review of water bores that intersect cavities or record circulation loss during drilling suggests that the karst development is widespread across the Beetaloo Basin and that aquifer permeability is generally not spatially correlated.

Within the study area over 415 operational and abandoned water bores screen the Gum Ridge Formation (and equivalents), with bore depths ranging from 34 – 221 m (average 105 m). Reported airlift yields range from 0.3 – 20 l/s (average 3.5 l/s), though this range generally reflects the bore water requirement which being largely for stock watering is modest. Pumping test results and yields from irrigation bores drilled in the analogous Daly Basin Tindall Limestone suggest that yields of up to 100 l/s may be possible in specifically constructed bores. The standing water level (SWL) in the Gum Ridge Formation ranges from 23 to 155 metres below ground level (mBGL). The shallowest water levels (<50 mBGL) occur along the north8east margin and in the north8west of the Beetaloo Basin. The deepest groundwater levels (>125 mBGL) occur along the Carpentaria Highway on Amungee Mungee and Tanumbirini stations.

Results from 21 pumping tests undertaken by WRD in the study area report a Transmissivity (T) range of 3 – 3377 m2/day for the Gum Ridge and Montejinni Limestone aquifer. The lowest T values (<50 m2/day) occur in the northwest of the basin where the Gum Ridge Formation has very limited saturated thickness and aquifer development is restricted to the unconformity with the underlying Antrim Plateau Volcanics (Yin Foo, 2002). Five test reports did not provide an estimate of T due to

negligible (< 10 cm) drawdown, the transmissivity for these bores likely to be at the higher end of the stated range. Most pumping tests identified were single well bore performance tests and do not provide an estimate of storativity. The exception is the Helen Springs groundwater investigation (Paul, 2003) which estimated the Gum Ridge storativity at 5.6 x 10-4.

Anthony Lagoon Beds

The Anthony Lagoon Beds form the upper unit of the Cambrian Limestone sequence within the Georgina Basin. The formation contains the principle water resource and uppermost aquifer system in the south of the study area but thins progressively to the north pinching out around the Carpentaria Highway. The formation is absent west of the Ashburton Ranges but a similar sequence of interbedded siltstones, dolomite and limestone is reflected in the upper section of the Montejinni Limestone. The Anthony Lagoon Beds is unconfined where it outcrops on the southern edge of the Beetaloo Basin and along the eastern flank of the Ashburton Ranges. Outside these areas the aquifer is confined by the overlying Cretaceous sequence.

The Anthony Lagoon Beds is a fractured rock aquifer comprising silicified limestone, dolomitic siltstone, fine grained sandstone and dolomite. Similar to the Gum Ridge Formation the Anthony Lagoon Beds displays dissolution and karst features that significantly enhance the permeability of the formation. The distinction between the two Cambrian Limestone Formations is not clear in many water bore logs due to circulation loss during drilling, consequently some crossover between the bore and water quality statistics for these formations is expected.

Over 78 bores have been constructed in the Anthon Lagoon Beds with completion depths from 63 to 175 m (average 109 m). Estimates of bore yields from air lifting range from 1 to 10 l/s (average 2.9 l/s) and are consistent with pump test yields (10 l/s) at Elliot where the Anthony Lagoon Beds is tapped for town water supply. The standing water level in the aquifer ranges from 42 – 115 mBNS (average 76 mBNS) and is closest to the surface where the aquifer outcrops in the south of the basin. Results from 11 pump tests display significant variation in aquifer transmissivity which ranges from 13 – 1400 m^2/day, most of these tests were undertaken around Elliot and in the Helen Springs region (Paul, 2003) adjacent the southern edge of the study area. Storage coefficients for the Anthony Lagoon Beds are reported at between 2.5 x 1083 and 9.7 x 1085 (Paul, 2003).

A1.2.4.3 Antrim Volcanics

The Antrim Plateau Volcanics form a continuous base beneath the Cambrian limestone aquifer across the northern and central Beetaloo Basin but is absent in the south and east of the basin. Thicker sequences of the massive basalt flows have negligible primary porosity and where present the Antrim Plateau Volcanics are considered to form a regional basement to the overlying sedimentary sequence (Tickell, 2005, Yinfoo, 2002). At a local scale the Antrim Plateau Volcanics can form a viable aquifer and the basalt provides a water supply for a number of communities in the greater region including Lajamanu, Minyerri and Pigeon Hole. Localised aquifer development is commonly dependent on enhanced secondary porosity derived from fault related fracturing (Yin Foo, 2002).

In the Beetaloo Basin, the Antrim Plateau Volcanics forms a localised, fractured rock aquifer where it sub-crops at shallow depths beneath the Cambrian Limestone sequence in the north-west of the basin. The formation outcrops on the northern edge of the basin on Nutwood Downs Station, in this area the volcanics are competent with limited aquifer development

A1.2.4.4 Bukalara Sandstone

The Bukalara Sandstone forms a fractured and weathered aquifer with a local scale flow system where it outcrops just beyond the northern margin of the Beetaloo Basin (Fulton and Zaar, 2009). The formation is also present at depth in the north and centre of the basin but is discontinuous between these areas. The Bukalara Sandstone comprises quartz sandstone with visible porosity (Lanigan et al, 1994) and minor shale interbeds, secondary porosity is likely to be enhanced by evident jointing within the sandstone outcrop.

Twenty-two bores have been constructed in the Bukalara Sandstone on Nutwood Downs and Hodgson River stations where it provides a stock and domestic water supply. Most bores penetrate the overlying Antrim Plateau Volcanics, which forms a local confining layer over the sandstone aquifer of varying in thickness between 10 and 140 m (Fulton and Zaar, 2009). To the north-east of the volcanic exposures the Bukalara Sandstone outcrops and is likely to be unconfined, though there are

very few bores drilled in the outcrop area. Completion depths for bores screening the Bukalara Sandstone range from 32 – 150 m (average 89 m), while airlift estimates of bore yield range from 0.3 – 5 l/s (average 2.6 l/s). Pumping tests have been completed on two production bores and report an aquifer transmissivity of 18 – 79 m2/day, estimates of storage coefficient are not available. Groundwater levels in the Bukalara Sandstone range from 3 – 34 mBGL (average 17 mBGL) and groundwater flow direction is to the north (Fulton and Zaar, 2009).

A1.2.4.5 Hayfield Mudstone

The Hayfield Mudstone is a fine grained mudstone and claystone sequence with minor sandstone intervals that has anaverage thickness of 320 m. The formation is restricted to the Beetaloo Basin and is extensive except north of the Walton High and in the south of the basin. Silverman (2007) notes that the Hayfield Mudstone is likely to act as a seal for the underlying Jamison Sandstone, where present it is considered to act as an aquitard separating the Roper Group from the overlying Cambrian sequence. Due to its relatively younger time of deposition the Hayfield Mudstone has not experienced the same level of deformation as older Roper Group sediments and a number of deeper seated faults do not penetrate the formation. Minor sandstone interbeds occur within the greater mudstone sequence and more significant layers may behave as local aquifers. One sandstone inlier, located around 60 m above the base of the formation has a thickness of 58 15 m, porosities of 5 – 15 % and a permeability of up to 20 mD (0.02 m/day).

A1.2.4.6 Jamison Sandstone

The Jamison Sandstone comprises a very fine to coarse grained quartz sandstone that is moderately to well sorted and displays an upwards coarsening trend. The top third of the formation characteristically contains a cleaner sand unit, 10 – 20 m thick, that becomes progressively shalier and grades into the overlying Hayfield Mudstone (Silverman et al, 2007). The Jamison Sandstone can be distinguished from underlying Roper Group sandstones by the presence of feldspathic and lithic grains and higher clay content (Lanigan et al, 1994). Unlike the underlying Roper Group formations, which are also present in the southern McArthur Basin, the occurrence of the Jamison Sandstone is restricted to the Beetaloo Basin where it is absent on the Walton High but otherwise extensive. The formation has a minimum thickness of 73 m at Chanin81 in the west of the basin and thickens from west to east increasing to 160 m at Burdo81. The Jamison Sandstone forms a gradational contact with the overlying Hayfield Mudstone.

A1.2.4.7 Kyalla Formation

The Kyalla Formation is continuous across the basin except north of the Walton High. Silverman (2007) notes the Kyalla Formation has significant potential to act as a seal for the underlying Moroak Sandstone. It obtains a thickness of over 700 m in the centre of the basin and where substantial mudstone sequences are encountered it is likely to operate as an aquitard and retard vertical flow. The potential exception is over the Arnold Arch, where the upper section of the formation is eroded leaving a thinner basal sequence of Kyalla Formation (170 m) that has a higher proportion of sandstone interbeds and may display enhanced fracturing associated with the underlying structural feature.

A1.2.4.8 Moroak Sandstone

The Moroak Sandstone comprises a sequence of fine to coarse grained cemented quartz sandstones, which in the top section of the formation are massive and display a coarsening upwards trend. The Moroak Sandstone displays characteristic fracturing, which contributes to the permeability of the sandstone (Silverman, 2007). The Moroak Sandstone is absent in drill holes on the north of the Walton High (Alltree81, Walton82) where the formation has been eroded but is otherwise extensive in the Beetaloo Basin. The formation thickens towards the centre of the basin attaining a maximum recorded thickness of 483 m in Shenandoah81. The Moroak Sandstone conformably underlies the Kyalla Formation.

1.2.4.9 Velkerri Formation

The Velkerri Formation represents a substantial shale and siltstone sequence of up to 879 m that is extensive across the Beetaloo Basin. Silverman (2007) suggests that the Velkerri Formation is likely to provide a competent seal for the underlying Bessie Creek Sandstone and the formation is inferred to operate as a regional aquitard within the Beetaloo Basin isolating aquifers in the underlying Roper Group from the overlying Cambrian and Mesozoic systems.

A1.2.4.10 Beneficial-use aquifers and proximity to target shale formation

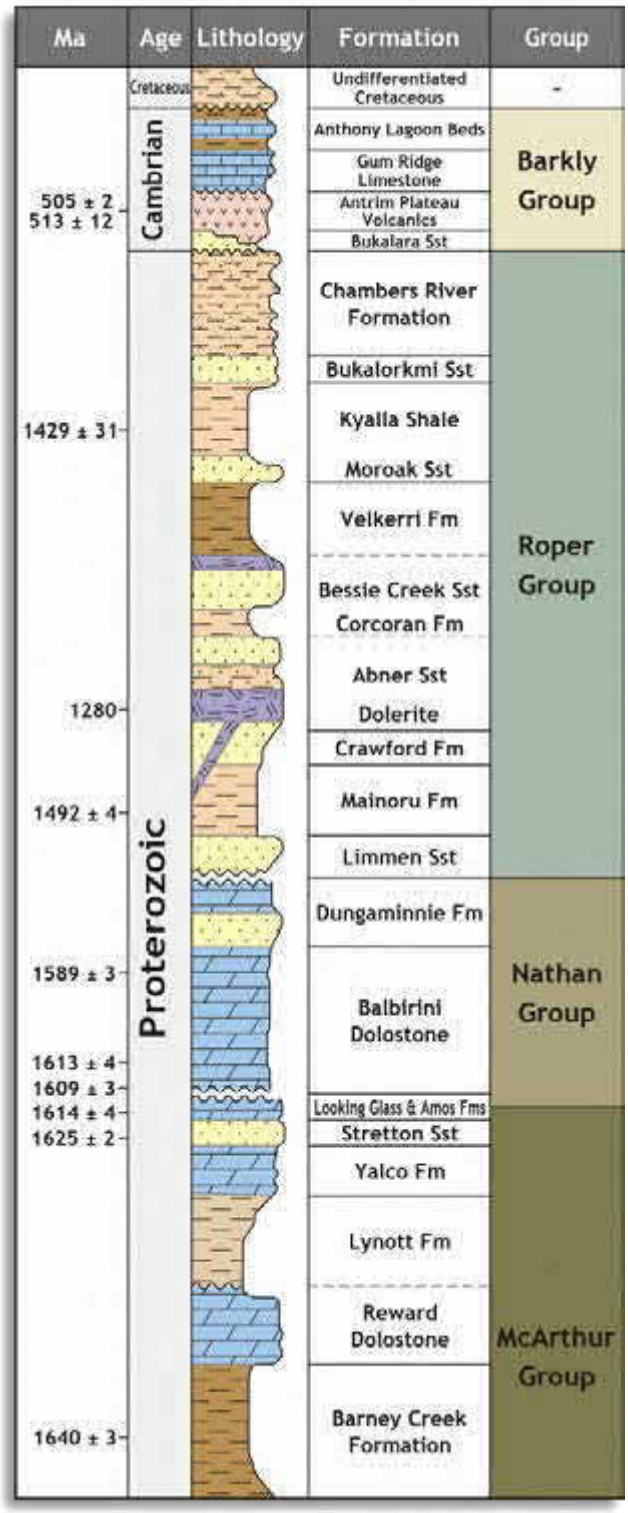
On the basis of the quality of groundwater yielded (low salinity) and formation transmissivity, the following units are considered major aquifers at Amungee NW-1H:

- Undifferentiated Cretaceous Sediments
- Cambrian Limestone Formation (Gum River Formation and Anthony Lagoon Beds)
- Bukalara Sandstone

These beneficial-use aquifers are separated from the shale gas target formation (Velkerri Formation) by alternating layers of low permeability formations (Kyalla Formation and Hayfield Mudstone) and aquifers not considered to be of beneficial use due to bearing hypersaline groundwater (Moroak Sandstone and Jamison Sandstone).

The vertical separation distance between the beneficial use aquifers and the target shale formation (Velkerri Formation) at Amungee NW-1H is illustrated in **Figure 13**.

Figure 13 Stratigraphic Profile at Amungee NW-1H



A1.2.5 Groundwater Resources

A1.2.5.1 Groundwater Flow, Recharge and Discharge

The regional groundwater flow direction in the CLA is north-west toward Mataranka, where the aquifer discharges into the Roper River and supports significant groundwater dependent ecosystems including the Roper River at Elsey National Park and Red Lily/57 Mile Waterhole. These discharge features occur around 100 km north-west of the Beetaloo Basin. Dry season flow in the Roper River has been gauged at 95,000 – 126,000 ML/yr and provides an estimate of the magnitude groundwater discharge from the CLA. Large decadal changes in the discharge to the Roper River suggest that most recharge input occurs close to the discharge zone (i.e. beyond the Beetaloo Basin region). Groundwater recharge mechanisms to the CLA are poorly characterised but are likely to be dominated by infiltration through sinkholes and preferential recharge through soil cavities.

The potential for fluid migration through faults between the CLA and adjacent formations is limited because most faults don't extend to the shallow formations.

A1.2.5.2 Groundwater Use

The CLA contains a significant but largely undeveloped groundwater resource with the sustainable yield from the Georgina Basin estimated to be in the order of 100,000 ML/year (NALWTF, 2009). Existing groundwater use in the Beetaloo Basin is estimated at 6,000 ML/year.

Unconventional gas exploration in the Beetaloo basin is at a very early stage and the volume of water required to develop any potential resource is uncertain. However a first order estimate of the water required to develop potential gas resources on Origin tenements is 1,000 ML/year over the development phase. Combined, current groundwater extraction and projected demand for gas development in the Beetaloo Basin represents 7% of the estimated water resource available from the CLA in the Georgina Basin.

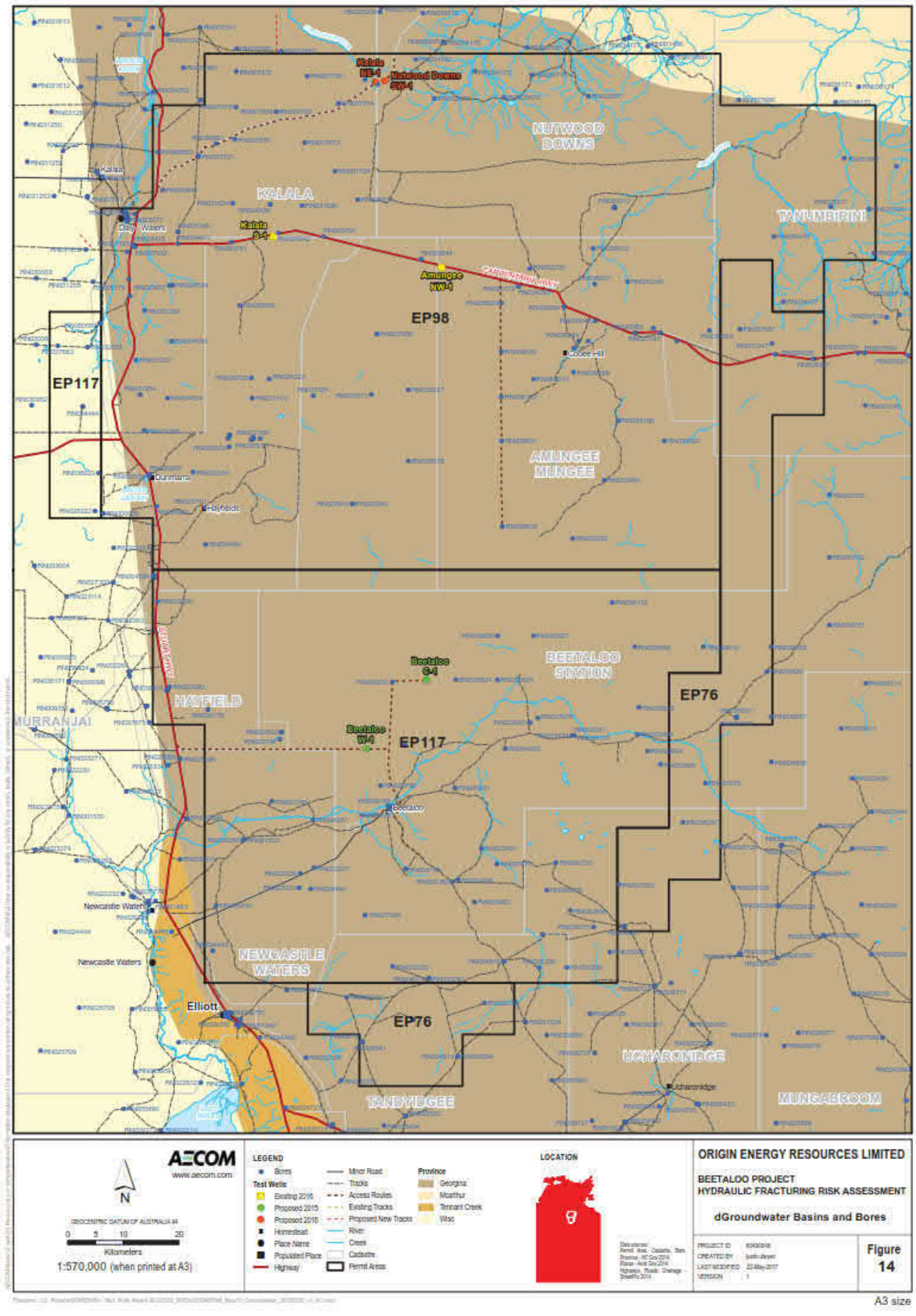
A1.2.5.3 Groundwater Bores in the Surrounds of Amungee NW-1H

Figure 14 shows the location of the groundwater bores in the surrounds of Amungee NW-1H, and **Table 3** summarises construction and aquifer information for those bores that are closest to Amungee NW-1H. **Table 3** has been compiled from the NT government groundwater database and exploration-related field activities.

Table 3 Summary of Groundwater Bores near Amungee NW-1H

Proximity to Amungee-NW1H	Bore Reference	Installation Date	Purpose	Total Depth	SWL (mbTOC)	Yield (L/s)	Physical Parameters
3.9 km West along Carpentaria Highway	RN005844	01/05/1967	ND	124.0	108.8	2.5	pH 7.8 EC 1,085 µS/cm
12.3 km South East Amungee Mungee Station	RN036920	01/05/2010	Stock Bore	165.0	126.0	3.0	pH 6.84
~14 km South East Amungee Mungee Station	RN038630	06/07/2014	Stock Bore	149.95	95.0	5.0	pH 8.08
~18 km South East Amungee Mungee Station	RN038109	03/07/2014	Stock Bore	162.15	97.0	1.0	pH 8.59
~21 km South East Amungee Mungee Station	RN038631	07/07/2014	Stock Bore	148.85	93.0	6.0	pH 8.16
~24 km South East Amungee Mungee Station	RN038632	09/07/2014	Stock Bore	140.1	97.0	3.5	pH 8.36
27 km South East Amungee Mungee Station	RN006877	22/08/1969	Homestead Bore	140.0	114.3	1.89	pH 7.4 EC 900 µS/cm

Figure 14 Groundwater Bores in the surrounds of Amungee NW-1H



A1.3 Well Integrity

To inform the assessment of risks associated with hydraulic fracturing within the project area, the mechanical integrity of Origin shale gas wells must be understood.

The mechanical integrity of a well describes its ability to isolate fluids (both hydraulic fracturing and production fluids) within the well from the surface and sub-surface zones exterior to the well during its working life and beyond, including after it has been formally abandoned by the mandated well abandonment processes; this includes the prevention of cross-flow of fluid between geological formations (i.e. zonal isolation).

The mechanical integrity of a well is essential for managing the risks associated with hydraulic fracturing, given the need to isolate groundwater resources from the hydraulic fracturing fluid; the increase in hydraulic pressure used to conduct hydraulic fracturing; and the formation pressure within the producing formation during subsequent production.

It is the robustness of the well design, construction, and eventual abandonment that ensures that well integrity is maintained at all times. Origin's internal policies and procedure comply with international best practice for well integrity, recognised to be the specifications of the American Petroleum Institute (API).

The Origin procedure *Integrity Management Plan: Conventional Wells Operate and Maintain Phase (INT 1000 ENG PLN cDN/ID 7726350)* mandates the testing and maintenance procedures which are to be employed to ensure that the integrity of well and wellhead equipment is maintained throughout all stages of the well lifecycle.

Throughout the well design and installation process Origin employs stringent quality control procedures and testing, to ensure the integrity of each well before it is hydraulically fractured. Aspects of the well design and construction phases which develop and maintain well integrity include:

- Design of the well construction to account for the local sub-surface conditions;
- Conducting a down-hole geophysical survey (wireline logging) of the drilled borehole, prior to the installation of well casing;
- Constructing the well using casing materials suitable for the pressures to which the well will be subjected during in its lifetime;
- Utilising cementing materials and techniques in accordance with international best practice (API specifications);
- Pressure testing of the casing (above the maximum pressure required to perform hydraulic fracturing) prior to casing perforation; and
- Conducting down-hole surveying (a wireline cement bond log (CBL)) of the well casing following cementation, to confirm integrity.
- These procedures are discussed in the following sections of this report

A1.3.1 Well Design

The primary principle of all shale gas production wells is to maintain the mechanical integrity of the well at all times. The well design phase is critical to meeting this objective.

Origin has developed internal guidance documents to ensure that well designs and installation procedures meet regulatory frameworks and international best practices. The relevant documents are:

- Drilling & Completions Basis of Design (NT-2050-35-PH-0020 Beetaloo Exploration Campaign Amungee NW-1H Outline Stimulation Program and NT-205035-TR-0002- Amungee NW-1H Casing Design Rev2)
- Well Design Study Report (NT-2050-35-TR-0003- Beetaloo Exploration Concept Select 2016)
- Integrated Gas Level 1 and 2 HSE Risk Register Drilling and Completions (Beetaloo Drilling Operations Hazard Register, Hydraulic Fracturing Specific Risk Registers)
- Integrated Gas Well Design & Construction Operations Assurance Plan (INT-1000-35-AP-001)

- Drilling and Completions Standards (Barriers *INT-1000-35-TS-002*, Casing & Tubing *INT-1000-35-TS-003*, Cementing *INT-1000-35-TS-004*, Well Integrity *INT-1000-35-TS-013*)

Origin designed and constructed Amungee NW-1H to isolate the interior of the well from the surrounding geological formations that were not the resource target of the well (i.e. all formations above the Velkerri Formation). The well was also designed to prevent cross-flow between geological formations (i.e. ensure zonal isolation).

Multiple physical isolation barriers were constructed between the interior of the well and the shallow beneficial-use aquifers, by installing, cementing and pressure testing steel casing in these intervals, prior to drilling ahead into the underlying formations which bear hyper-saline groundwater and shale gas (see **Section A1.2.5**).

A1.3.2 Drilling and Casing

As part of the well design process, casing material types and depths are considered to ensure that complete well integrity is obtained. Origin wells are designed with multiple sections of casing that become sequentially smaller in size as the well is drilled deeper. The reasons for installing multiple sections of casing include; borehole stability, isolation of non-targeted formations and well control (containment of gas and artesian flow of groundwater).

The Amungee NW-1H well was drilled and completed with four casing strings referred to as the conductor, surface, intermediate and production sections (see **Table 4**). The surface, intermediate and production casing sections are cemented and pressure tested to ensure zonal isolation is achieved.

- The conductor section is installed to protect the well from shallow groundwater intrusion and ground surface subsidence during drilling.
- Surface and Intermediate Sections are used to isolate non targeted formations and to provide the well with well control integrity. Origin designed Amungee NW-1H such that the surface casing is set below the deepest aquifer.
- The Production Section forms the conduit for the hydraulic fracturing fluid to be pumped into the target shale formation and for gas and produced water to be extracted.

A final completion string is installed within the production casing which may include tubing or pressure monitoring equipment to aid production from the well.

Table 4 Summary of casing properties for Amungee NW- 1H well

Casing String	Setting Depth	Well Section	Diameter (inches)	Weight (lbs/ft)	Grade	Thread
Conductor	113 mTVD	Vertical	16	42	X42	Threaded and Coupled
Surface	276 mTVD	Vertical	10-3/4	40.5	K55	Threaded and Coupled
Intermediate	1427 mTVD	Vertical	7-5/8	29.7	P110	Premium Gas Tight Connection
Production	3803 mMD 2428 mTVD	Horizontal	4-1/2	15.1	P110	Premium Gas Tight Connection

mMD: m Metres Drilled, mTVD: m Ture Vertical Depth

Amungee NW-1H was installed and cased in accordance with:

- Origin Casing and Tubing Standard (INT-1000-35-TS-002)
- Origin Corrosion and Materials Selection Standard (INT-1000-35-TS-006)

A1.3.3 Surface Completion

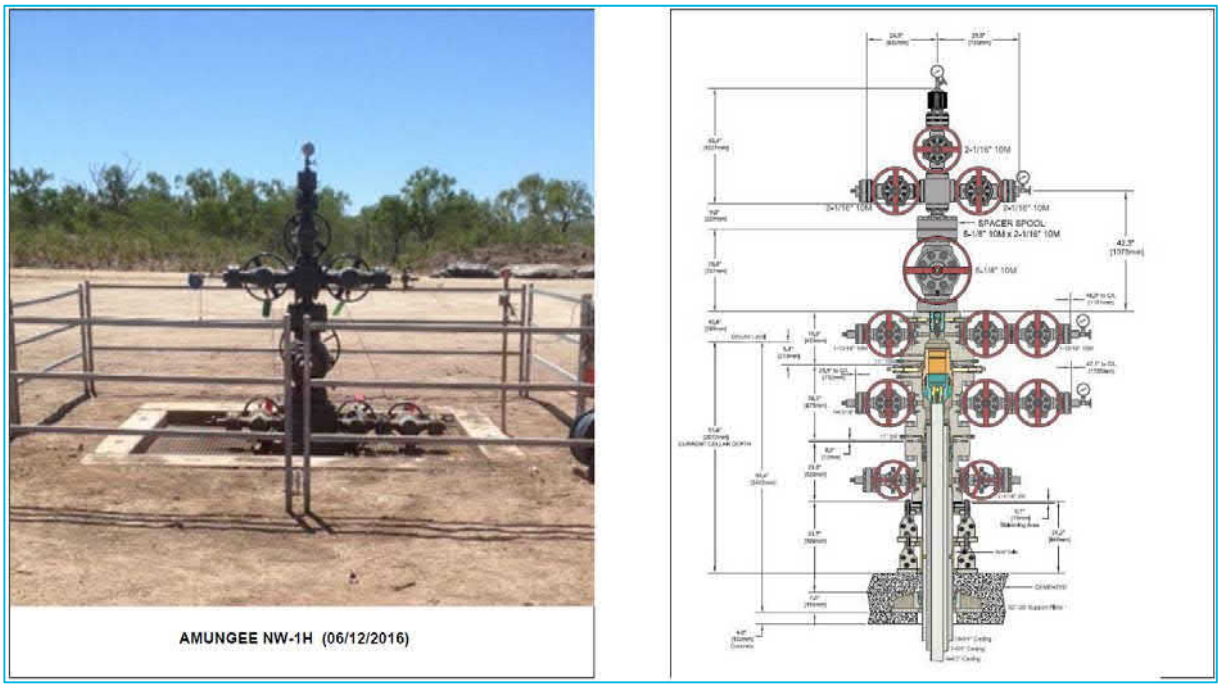
Wellheads are equipment that is installed on top of the casing to provide a seal at the surface. The wellhead is critical in maintaining well integrity at surface. Origin uses wellheads which meet API specifications. The Amungee NW-1H wellhead is divided into multiple sections:

- A Section – Intermediate Casing Spool
- B section – Production Casing Spool
- C Section – Tubing Head Spool
- D Section – Master Valve
- E Section – Production Tree

Refer to **Figure 15** for a schematic of the Amungee NW-1H wellhead.

Each section serves as a point to isolate the well interior from the surface. Origin and their suppliers follow stringent pressure testing procedures (API Spec 6A) to ensure that the wellheads are adequate barriers from down-hole conditions.

Figure 15 Amungee NW-1H Wellhead



A1.3.4 Casing Selection and Installation Procedures

Origin drilling engineers following international best practice (API standards) and drilling engineering principles when designing their shale gas wells to ensure the well is capable of withstanding the compressive, tensile and bending forces associated with the construction, operation and abandonment of the well. Further the well is designed and constructed to minimise corrosion from fluids encountered interior and exterior to the well during the life of the well. The steel casing used is manufactured to comply with API standards 5B and 5CT, for which casing must meet strict tension, compression, collapse and burst resistance requirements.

Selection of the most appropriate casing in terms of its composition, weight, strength, and connection type is primarily a function of the expected forces and stresses or load that must be withstood during the well lifecycle. The Origin Casing Design Standard (INT-1000-35-TS-002) stipulates which load cases must be considered when designing a well and are based on the forces that could be exerted on each casing string through the entirety of the well's life. The logic of a typical casing design (and that of the Amungee NW- 1H well is that as the well gets progressively deeper the pressures

encountered are likely to be higher and therefore the casing must be stronger: It is also important that the final casing string (the production string) be robust enough to withstand the loads that will be applied during any stimulation and production activities, when higher internal pressures will be experienced.

Table 5 Summary of casing strength for different strings in the Amungee NW-1H well

Casing String	Burst Pressure	Collapse Resistance	Yield Strength
Surface	3,130	1,580	629 klbs
Intermediate	9.470	5,340	940 klbs
Production	14,420	14,320	485 klbs

Casing is manufactured in a range of standard diameters and weights defined by the American Petroleum Institute (API), each of these casing types has individual ratings for compressive and shear strength (see **Table 5**). Ratios of the yield or failure thresholds for a given casing type define minimum design factors which cannot be exceeded during well operations. The ratio of the modelled or required force a casing string will experience divided by the yield thresholds must exceed the minimum design factors to be acceptable under the Casing Design Standard (see **Table 6**) - if one of the failure type criteria are not met higher strength casing is required.

Table 6 Summary of modelled loads for each casing string at Amungee NW-1H

Casing String	Pressure – Burst	Pressure – Collapse	Axial	Triaxial
Minimum Design Factor	1.1	1.0	Static: 1.3 Dynamic: 1.6	1.25
Surface	3.24 (Pressure Test / Leak-off Test)	2.57 (Complete Evacuation	2.10 (230 klb Overpull)	2.10 (230 klb Overpull)
Intermediate	1.43 (Production Casing Leak)	2.33 (Complete Evacuation)	3.17 (170 klb Overpull)	1.75 (Production Casing Leak)
Production	1.17 (Injection Screen-out)	2.57 (Full Evacuation)	1.84 (Overpull 150 klb)	1.25 (Injection Screen-out)

Following the installation and cementing of the casing, Origin performs casing pressure tests. The purpose of these tests is to ensure that the casing can withstand pressure which may be encountered during the life of the well. The pressure tests are completed following the installation of each string of casing.

During the installation of a string of casing in the borehole, each individual length of casing (referred to as a casing joint) is connected to the previous length of casing using API approved casing grease (referred to as ‘pipe dope’), then tightened to within the recommended range of torque or in accordance with the torque-marker indicator on the casing. API guidance documents highlight that too little torque may results in leaky connections; whilst over-torqueing the connections may results in failures in the casing threads (stripped threads). The casing used by Origin is threaded at each end and are designed to be screwed together using the drilling rigs mechanical jaws. Casing requiring welded connections is not used by Origin.

Origin drilling contractors and well site supervisors complete onsite audits and supervision of the casing install process. The Origin operating company representative and drilling rig manager conduct quality control over the casing installation process, this includes: confirming the quality of the casing construction by checking the inner diameter, outer diameter and grade of the casing, measuring the length of each individual length of casing (referred to as ‘strapping’ the casing), inspecting the casing and casing threads, reviewing the casing tally to ensure the correct installation depth and overseeing the casing installation process.

A1.3.5 Cementing

The pressure cementing of gas production wells is a highly controlled and engineered process. For each string of casing pressure cemented, on every well installed, a tailored cementing program is designed by specialised well cementing service companies. The cement program varies the density and volume of the cement used to match the purpose and design of the well and local geology. Origin conducts an independent review of each cement program and approves the design, prior to commencement of each cementing event (referred to as a cement 'job').

Well cementing is performed in accordance with Origin's Cementing Standard (INT-1000-35-TS-004).

API standards 10A and API RP 10B-2 are followed by Origin and their cementing contractor when designing and testing the cement mixes.

The cement slurry design used is laboratory-tested by the cementing contractor prior to their arrival on site, to confirm suitability and ascertain curing times.

The cement job consists of mixing water with cement on site 'on-the-fly', i.e.: cement is blended with water in the cementing unit just prior to being pumped down the well. This allows the cementer to control the density of the cement, as well as the pumping rate and the pumping pressure as the cement job proceeds.

During a cement job, cement slurry is pumped from surface, down the inside of the well casing; upon reaching the casing shoe, the cement slurry exits the casing and begins to return to surface from the bottom of the well, up the annulus between the casing and the geological formation around the casing. Once the volume of cement required to fill the annulus between the well casing and the geological formation has been pumped (including excess cement volume for contingency/losses of cement to formation) the cement is displaced from the interior of the casing with a brine fluid. Cementing 'wiper plugs' are pumped down the well at various stages of the cementing operation, to both separate the cement slurry from the displacement brine, and to clear remnant cement from the interior wall of the casing.

Once the wiper plug reaches the casing float (a one-way valve located close to the bottom of the casing string), it forms a seal at the bottom of the casing string, blocking the flow of fluid out of base of the casing sting. Following this 'bumping of the plug', pumping of the displacement brine into the casing is continued at a slower rate, this builds pressure inside the casing string. Pressure is raised inside the casing until the desired pressure is achieved for conducting a casing pressure test, at which point the casing is sealed at surface, 'shutting-in' the pressure in the casing. The pressure test consists of holding the pressure in the casing for a specified duration to confirm that the casing is capable of holding pressure, thus confirming the casing integrity; the ability of the casing to isolate pressure within the casing from the surrounding geological formations. A typical pressure cement job setup is illustrated in **Figure 16**.

During the cement job an Origin representative supervises the cementing contractor, to witness the actions of the cementer and confirm that the cement job proceeds without incident. Data from the cementing unit including injection pressures, pumping rates and cement density is recorded throughout the cementing process. Samples of cement are taken at various times during the cement job as a quality control procedure to confirm the cement density and the required curing time.

The properly conducted application of cement will:

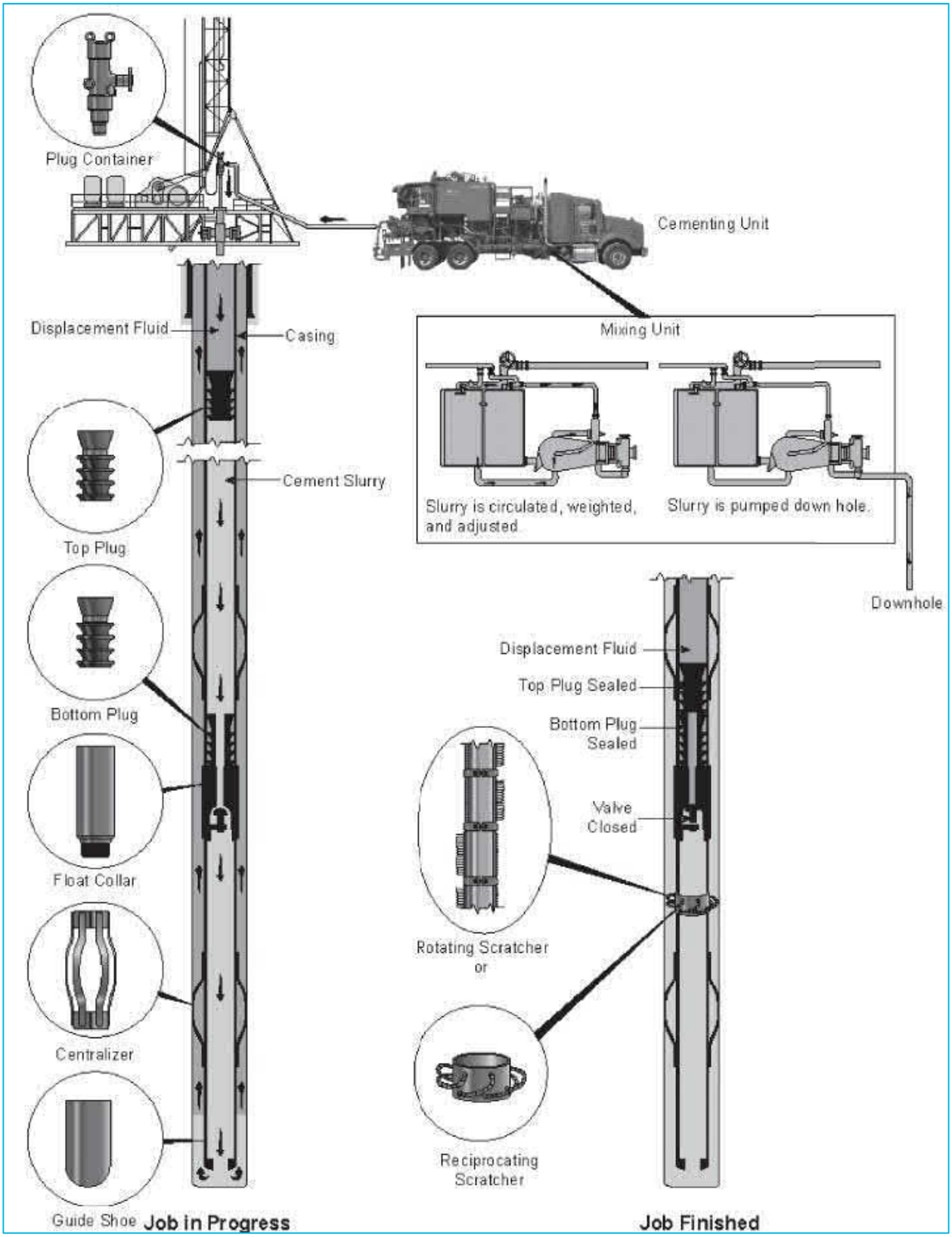
1. Evenly distribute cement in the annulus between the casing and formation/s
2. Prevent migration paths and isolate the targeted zone from other formations
3. Protect groundwater resources from contamination
4. Maintain aquifer pressures and quality
5. Obtain and maintain well integrity
6. Protect the casing from corrosion

Origin adheres to API standards (industry best practices) for cement operators. The key cement application API standards that are used by Origin and their contractors for performing cementing of wells are:

- API Spec 10A – Well Cements
- API RP 10F – Cement Float Equipment
- API 10B-2 - Well Cement Testing
- API RP 65 – Part 2 Isolation of Potential Flow Zones
- API RP 10D-2 – Centraliser placement and stop collar testing
- API TR 10TR4 – Selection of centralisers

Due to the geology of the CLA (limestone formation), it is difficult to cement the Surface Casing to ground surface. For this reason, Origin designed Amungee NW-1H such that the Intermediate and the Production Casing strings were cemented to ground surface to ensure isolation between the hydrocarbon bearing formations and the aquifers.

Figure 16 Pressure cement job equipment and setup (API, 2009)



A1.3.6 Well Integrity Testing

Following the drilling, casing and cementing of the well, isolation between each aquifer intersected by the well must be achieved to prevent both the leakage of groundwater between geological formations via the annulus of the well casing and the connection of the target shale formation from any overlying aquifers. This zonal isolation is achieved by ensuring sufficient cement is present throughout the annulus of the well casing, particularly in the depth intervals where natural aquitards are present. Evaluation is performed in accordance with Origin's Well Integrity Standard (INT-1000-35-TS-013).

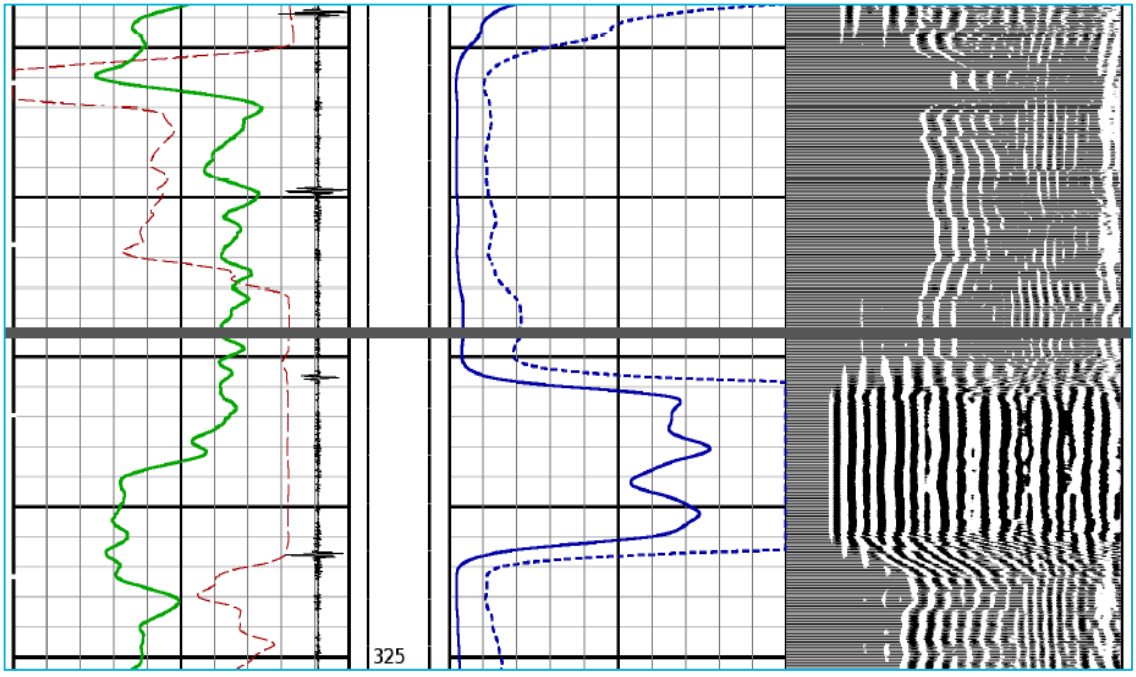
To verify the effectiveness of the casing cement job, a down-hole survey of the well is performed on all Origin shale wells to be stimulated using a cement bond and variable density logging tool (CBL-VDL). Typically a gamma ray (GR) and Casing Collar Locator (CCL) tool is run concurrently with the CBL-VDL survey, so that the results can be correlated with any open-hole geophysical surveys performed. If following the CBL-VDL log the cement job is deemed to be inadequate the hydraulic fracturing will not be conducted. A secondary (remedial) cement job may then be performed on the well, prior to any further works (including hydraulic fracturing) being conducted on the well.

The CBL tool is an acoustic device, the output of which can be used to infer quality of the bond between the casing and the surrounding cement; the cement bond quality can vary from a 'good' to 'non-existent' (free pipe). The tool transmits a continuous sound or vibration signal inside the casing and records the amplitude of the reflected signal. Casing that has a poor bond with the surrounding cement sheath produces a high amplitude returned acoustic signal, as most of the acoustic energy is reflects off the wall of the casing and surrounding fluid. Casing that has a good bond with the surrounding cement sheath will have a much smaller amplitude signal reflect to the tool, as a higher proportion of the acoustic energy is absorbed by the surrounding geological formation after it is transmitted via the better cement bond.

The VDL tool also provides a graphical representation of a reflected sonic waveform; however the transmitter and receiver are located at an increased spacing on the logging tool compared to the CBL transmitter/receiver. This allows the VDL to interpret the quality of the bond between the cement sheath and the surrounding geological formation.

The CBL and VDL tools are complementary and always performed concurrently. An example of a CBL-VDL output is presented in **Figure 17**. In this figure, the green line is a natural gamma plot (relevant to indicate permeability of the geology with respect to depth), the blue line is the CBL and the plot on the right is the VDL log, intervals of poor cement bond are represented by high peaks amplitude the blue plot and the thick black lines.

Figure 17 Example of a Cement Bond Log, an integrity assessment technique



A1.4 Hydraulic Fracturing

A1.4.1 Background

Shale gas is natural gas (predominately methane) which, in suitable geological conditions, is found within hard, dense, fine grained sedimentary rock, referred to as shale. Under natural conditions the low intrinsic permeability of the shale traps this methane.

Hydraulic fracturing is a technique employed to increase the permeability of shale by opening fractures within the shale. This liberates the methane from the rock, which in-turn flows to ground surface via the gas well.

A1.4.2 Process

The hydraulic fracturing process is a series of operations performed on and oil or gas well, to increase the permeability of the target hydrocarbon-bearing reservoir formation in the immediate surrounds of the well.

The process consists of pumping fluid at a high pressure down the wellbore then out into the target geological formation; the pressure of the fluid initiates fractures within the formation. As the pressure is sustained the fractures propagate from the well, through the formation. Once optimal fracture propagation has been achieved, uniform fine grained sand or other propping material, referred to as “proppant” is pumped down the well and into the open fractures. The hydraulic fracturing fluid carries the proppant in suspension. Once the pumping of fluid ceases, the proppant is deposited in the fractures. The pressure is removed and the fractures are held open by the proppant, thus the permeability within the targeted formation is increased. This allows both liquid and gas to flow more readily, improving the efficiency of gas recovery from the well.

A1.4.2.1 In-Situ Differential Stresses

In-situ stress and the relative stress difference between geologic strata are important influences on fracture development. An underground geological formation is bound vertically by the weight of overlying strata and laterally by surrounding geological material; this creates stress within the formation (referred to in-situ stress). These stresses occur in three dimensions, defining a maximum stress direction, intermediate stress direction, and minimum stress direction. These stresses are normally compressive and vary in magnitude throughout geological formations, particularly in the vertical direction (from layer to layer).

The magnitude and direction of the stresses is important because they control the pressure required to create and propagate a fracture, the shape and vertical extent of the fracture, and the direction of the fracture. To initiate and propagate a fracture, the minimum stress must be overcome by the pressure of the fracturing fluid. In a uniform medium, the plane of a fracture is perpendicular to the direction of the minimum stress; i.e. fractures propagate parallel to the maximum stress direction, opening against the direction of minimum stress.

A1.4.3 Containment of Hydraulic Fracturing

A strong understanding of the in-situ stress regime is required to understand fracture geometry. Despite the relative homogeneity of shale formations (compared to thinner and more variable formations like coal seams), there are still stress / geological barriers within shale that determine fracture height growth. Low stress rock provides a preferential fluid ‘sink’ during fluid injection, whereas high stress rock are barriers to fluid flow, i.e. the fluid/pressure wave is arrested at high stress barriers.

High and low stress rocks can be readily identified from dipole-sonic wireline logs, which are run prior to hydraulic fracturing. The dipole-sonic logs are calibrated using rock strength and/or closure pressure data from DFIT (diagnostic fracture infectivity tests) or leak off tests. The calibrated dipole sonic logs are used to build a mechanical earth model is generated to identify stress barriers. The process of generating a mechanical earth model and equations is described in Brooke-Barnett et al. (2015) and Flottmann et al. (2013).

The identification of stress barriers in combination with the controlled hydraulic fracture initiation depth (casing perforation interval) in pressure sink (low in-situ stress) lithologies are used in hydraulic fracture design modelling software packages (e.g. StimPlan). Using stress barriers and initiation points

as inputs the hydraulic fracturing is then designed to limit pressures and the injected fluid volume for each hydraulic fracturing stage, to contain height growth into the target formations, thereby avoiding the potential for inadvertent injection into overlying or underlying aquifers.

A secondary safeguard is in place during the actual pumping, at the stage when pressure diagnostics indicate excessive height growth (e.g. related to small faults), that stage of hydraulic fracturing is stopped.

A1.4.4 Design and Safe Operating Limits

For each hydraulic fracturing event, Origin engineers determine a safe operating envelope for injection pressure. The safe operating envelope is selected based on the following considerations:

- CBL results (see **Section A1.3.1**)
- Well design (see **Section A1.3**)
- Mechanical earth model (see **Section A1.4.3**)
- Hydraulic fracturing models (see **Section A1.4.3**)
- Relevant legislation
- Internal and external standards

For the Amungee NW-1H hydraulic fracturing operation, the maximum injection pressure was limited to 9250 psi.

If at any stage during the process there is potential for deviation from the established pressure envelope, operations are terminated and re-evaluated.

A1.4.5 Control

The hydraulic fracturing approach used to stimulate the shale gas wells will differ across the project area due to local characteristics of geology and depth, effectiveness, production characteristics, or other factors. The fracturing approach affects the development (height, length, and width) of hydraulically induced fractures.

The choice of fracturing fluid (e.g. water or gel based) also affects fracture dimensions. The effects of these fluid-controlled actions interact with and are influenced by the physical properties, depths, and in-situ stress of the geologic formations being fractured (as discussed above). For example, if a hydraulically induced fracture has a relatively constant height due to a geologic layer acting as a barrier to fracture propagation, and the fracture is forced to grow and increase in volume (through an increased volume of fracturing fluid), the fracture will mainly grow in length.

The design principles used by Origin aim to maximise the zone of influence of each hydraulic fracturing operation. Due to the vertical stress distribution along the Middle Velkerri member, the fracture propagation is anticipated to grow downwards to the lower stressed region, towards the base of the B Shale. For the Amungee NW-1H hydraulic fracturing operation, each hydraulic fracturing stage has been designed to propagate 150 m into formation (referred to as the fracture half-length).

A1.4.6 Operation

The process of an Origin hydraulic fracturing program is conducted in the following sequential stages:

i. Site mobilisation and setup
ii. Casing pressure test to confirm integrity
iii. Well casing perforation
iv. Hydraulic fracturing fluid injection
v. Well flowback
vi. Zone isolation (by setting a drillable plug in the casing)
vii. Repeat steps iii-viii for all zones
viii. Suspend well ready for completion

It is understood that Origin hydraulic fracturing specialists continuously monitor the progress of each hydraulic fracturing operation, to ensure that work is being conducted in line with Origin policies and procedures. Monitoring is in real-time from a dedicated hydraulic fracturing room in Origin's Brisbane office; in addition site visits are conducted throughout the campaign.

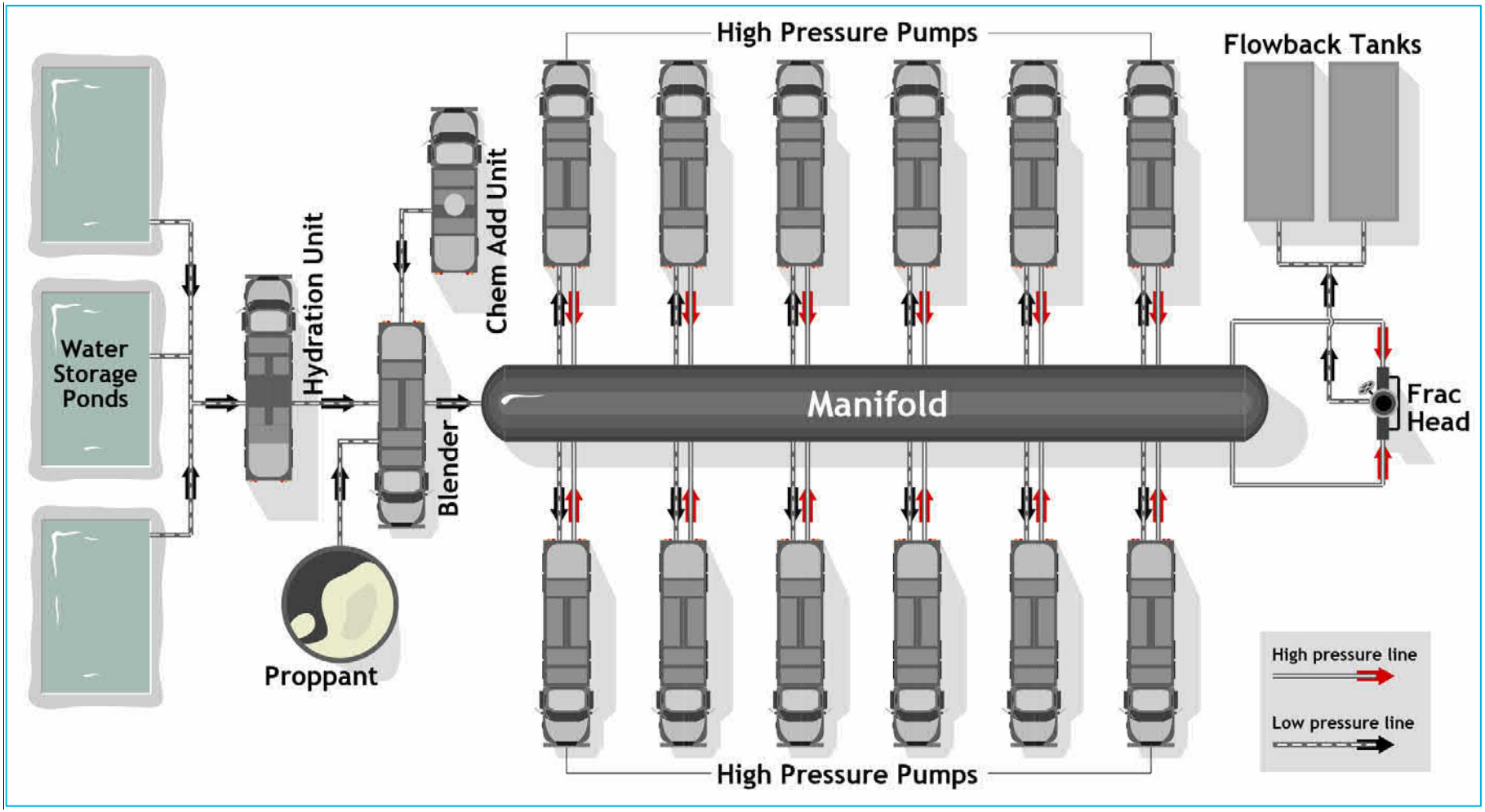
A1.4.6.1 Site Setup

The hydraulic fracturing operation requires various pieces of equipment, these may include:

- Data Unit – The control room from where the hydraulic fracturing contractor and Origin supervisor manage and monitor all aspects of the hydraulic fracturing operation including: surface and downhole pressures, injection flow rates and volumes, injected fluid parameters and proppant concentrations.
- Temporary Above Ground Tank(s) – Open top tanks with up to 15 ML storage capacity used to store water and supply it to the hydraulic fracturing operation as required. Panel tanks may also be used to temporarily store hydraulic fracturing flowback fluid from pilot hydraulic fracturing projects, prior to its treatment in water treatment plants.
- Proppant Trailer: a large sand storage and delivery trailer that holds the proppant prior to mixing in the Blender Unit.
- Hydration Units: pumps and tanks used to hydrate guar gum power with water to form a gel, this forms the basis of fluid viscosity in gel based hydraulic fracturing fluid.
- Blender Unit: pumps which mix the water, proppant and hydraulic fracturing chemicals on surface to achieve the required fluid consistency, prior to its injection into the well. Fluid is mixed "on-the-fly" as required throughout pumping procedures.
- High Pressure Pumps: pumps which inject the hydraulic fracturing fluid mixtures from the Blender Unit down the well, via the surface lines and hydraulic fracturing wellhead or coiled tubing unit.
- Hydraulic Fracturing Wellhead and Surface Lines: a network of pipes and pressure control valves that connect to the high pressure pumps and coiled tubing unit to the well.
- Coiled Tubing Unit : a large length of coiled steel pipe that can be temporarily installed in the well to convey hydraulic fracturing fluid from surface to the target depth interval to be fractured. The coil can use a number of bottom hole assemblies (BHAs) to deploy the hydraulic fracturing.
- Flowback tanks: temporary holding tanks with approximately 20,000 to 80,000 Litre storage capacity which are used to temporarily hold all fluid and proppant that flows back from the well following the hydraulic fracturing procedure. These tanks may be open-topped or sealed, depending on the requirements of the hydraulic fracturing operation.

A typical hydraulic fracturing operation site setup is presented in **Figure 18**.

Figure 18 Example of a Hydraulic Fracturing Operations Site Setup (supplied by Origin)

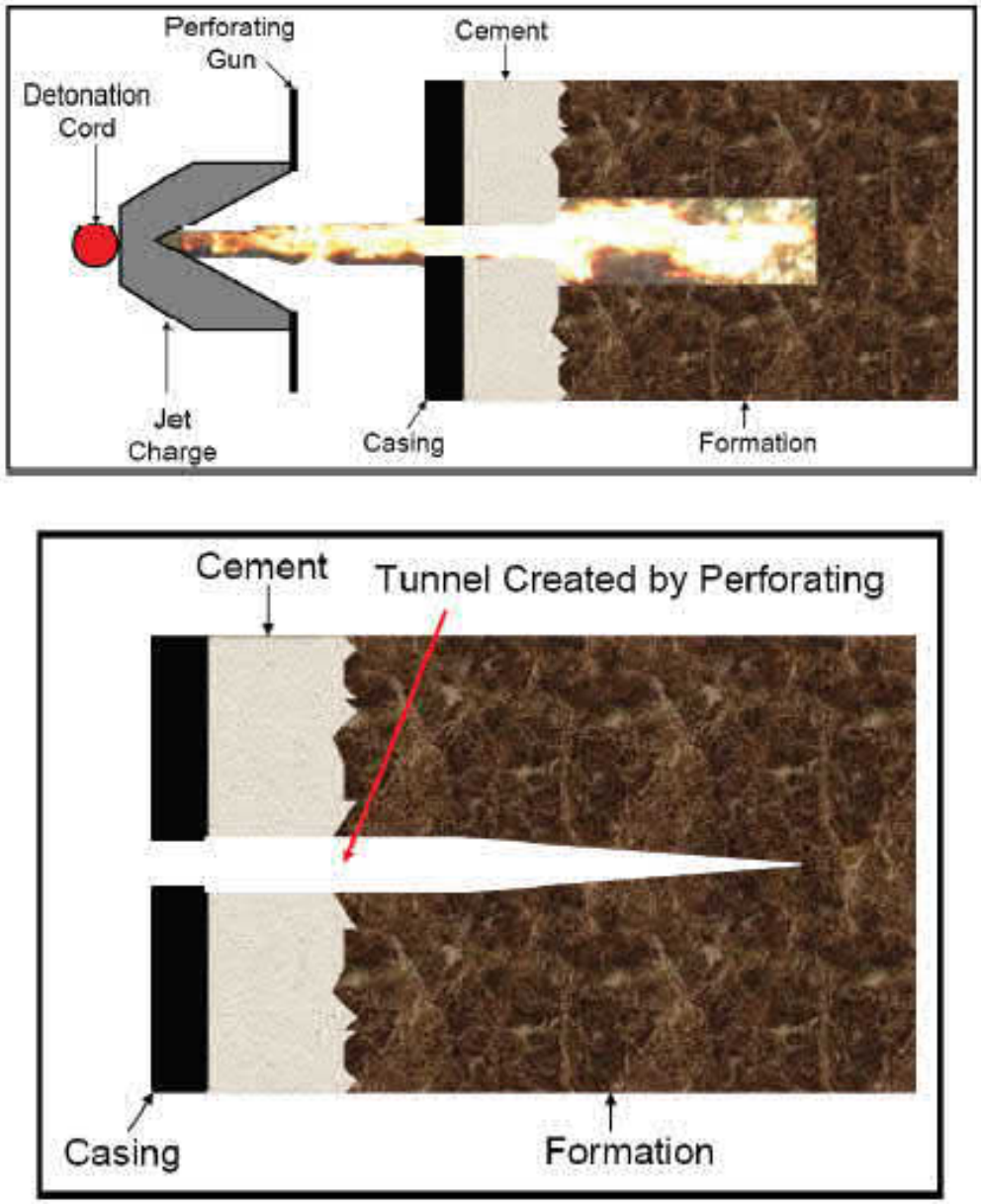


A1.4.6.2 Well Casing Perforation

The perforation of well casing is the process by which perforations (holes) are created in the casing of the well at the specific depth intervals, which correspond with the target formation interval to be hydraulically fractured. The well casing perforation process hydraulically connects the well to the target formation, within the zone of perforation. The total length of the perforation interval is designed based on the specifics of the hydraulic fracturing program.

Origin uses two different methods for the perforation of well casing for their hydraulic fracturing operations, depending on the requirements of the hydraulic fracturing program. The first method uses a perforating gun tool that is loaded with specialised explosive charges. The tool is lowered into the well to the desired perforation interval on a wireline and detonated remotely from surface (this method is illustrated in **Figure 19**). Alternatively, Origin also use the method of hydraulic jetting, whereby a high pressure jet of water and fine grained sand is used to cut holes in the casing.

Figure 19 Perforation gun charge (API, 2009)



A1.4.6.3 Fluid Injection Stages

Once the production casing of the well has been perforated in the depth interval that is to be hydraulically fractured, a series of fluid injection stages are performed.

A1.4.6.3.1 Injection and Isolation Techniques

Following perforation of the target zone, injection of fluid commences. The injection pressure, injection rates, slurry volumes, fluid viscosity, and proppant concentration are monitored in real-time during each injection, to confirm the success of the procedure and if necessary make adjustments to one of the monitored parameters.

In addition to monitoring data from the surface equipment, downhole pressure information is also recorded and reviewed during the hydraulic fracturing operation. Down-hole pressures are calculated based on wellhead pressure, fluid density, casing diameter and the depth to the target formation. When a coiled tubing unit is used as part of the operation pressure is monitored: inside the casing above the top packer of the tool, inside the coiled tubing delivering the fluid and inside the wellhead at surface.

Once the predetermined volume of fluid has been injected into the well the operation advances to the next stage of injection.

Between each hydraulic fracturing process, the perforation zones are isolated by either a coiled tubing unit with packers, a bridge plug set by a wireline operator, or by a baffle and ball-drop system. In the case of bridge plugs and baffles, once the entire hydraulic fracturing operation is completed for a well, a completion rig drills-out the bridge plugs or baffles that were installed for the hydraulic fracturing operation, to clear them from the well.

A1.4.6.3.2 Hydraulic Fracturing Fluid Injection

The hydraulic fracturing fluid injection stage comprises pumping hydraulic fracturing fluid at high pressure and high flow velocity into the perforated interval of the well.

The hydraulic fracturing fluid is designed, such that its ability to entrain particulate matter (referred to as ‘proppant’) is maximised. This is achieved by using a high viscosity fluid or a high fluid injection velocity. Based on the brittle nature of the target Velkerri Formation, high fluid injection velocity “slick water” and “hybrid gel” fluid designs were selected for Amungee NW-1H. The proppant material for Amungee NW-1H was comprised of graded sand.

A small volume acid spear head (pH stabilised 15% hydrochloric acid) was pumped ahead of the main fracturing fluids to decrease the pumping pressures by dissolving any remnant cement around the perforation holes.

To conduct the hydraulic fracturing process, the hydraulic fracturing fluid, inclusive of the proppant material is injected to the well at up to 50 barrels per minute (132 L/s). This flow rate was limited by the pipe-friction constraints of the 4-1/2” 15.1 lb/ft casing used in the Amungee NW-1H well and the requirement to maintain laminar flow. The injection of the fluid at high pressure opens and injects fluid into fractures within the target formation, at the interval in which the casing has been perforated. Once the designed volume of proppant has been injected into fractures, the injection pressure is reduced, at which point the aperture of the fractures reduces (due to formation in-situ compressional forces), which traps the proppant in the fractures. The proppant holds the fractures open, thus increasing the permeability of the fractured zone.

The design and propagation distance of the fractures is discussed in **Section A1.4.3**

The chemicals used by Origin in the hydraulic fracturing fluid and the concentrations at which they are used is detailed in **Section A1.4.7** and **Appendix B**.

A1.4.6.4 Initial Well Flowback

At the cessation of the hydraulic fracturing operation, hydraulic fracturing fluid is flowed back from the well to ground surface, which induces the flow of gas into the well. At Amungee NW-1H, the ‘flowback’ process comprised the purging of the weighted hydraulic fracturing fluid from the well by using the gas-lifting technique. This comprised using a coiled tubing unit to pump nitrogen gas into the well,

lowering the hydrostatic column in the wellbore, which in turn induced hydraulic fracturing fluid to flow from the fractured formation, back into the well and to ground surface.

Gas-lifting of the well was continued until sufficient fluid has been displaced from the well such that the wellbore could sustain a stabilised flow, driven by reservoir pressure.

All recovered flowback fluid (inclusive or particulate matter) is piped from the well into flowback tanks, used to settle-out any solids (i.e. proppant / sand) recovered to surface. Recovered fluid is then transferred into a double lined, surface tank referred to as a 'Flexipond' (see **Figure 20**). Flexiponds, provided by a third party company (Grizzly Energy Services) are engineered and certified to meet Australian standards. Each modular above ground storage pond has both geotextile and polyethylene linings (i.e. multiple barriers) in addition to monitoring to detect any leakage through the primary barrier. The Flexipond used at Amungee NW-1H had a capacity of 19 ML.

Once there was indication of hydrocarbon flow to surface, flow is directed through production testing equipment, which is similar arrangement to that used for the initial flowback, but includes a gas separator and flare.

The flowback fluid is predominantly comprised of hydraulic fracturing fluid and sand utilised in the hydraulic fracturing process. The amount of formation water (i.e. groundwater) in flowback is minimal on account of the limited groundwater bearing capacity of the target shale.

Flowback fluid is allowed to evaporate from the Flexipond; enhanced evaporation is used to speed the process. Enhanced evaporation comprises pumping water through high-speed fan blades which vaporises the water molecules. An example of an enhanced evaporation unit in use is presented in **Figure 21**. AECOM understand the enhanced evaporator unit is programed only to operate in optimal weather conditions to ensure the plume of mist generated remains within the extent of the Flexipond. A weather station is setup nearby to monitor wind intensity and direction. If there is a sudden change in wind direction or an increase in wind speed, the evaporator automatically shuts down and will not restart until conditions return to within the approved operating envelope. Prior to being used to evaporate flowback water, the evaporator was commissioned using potable water.

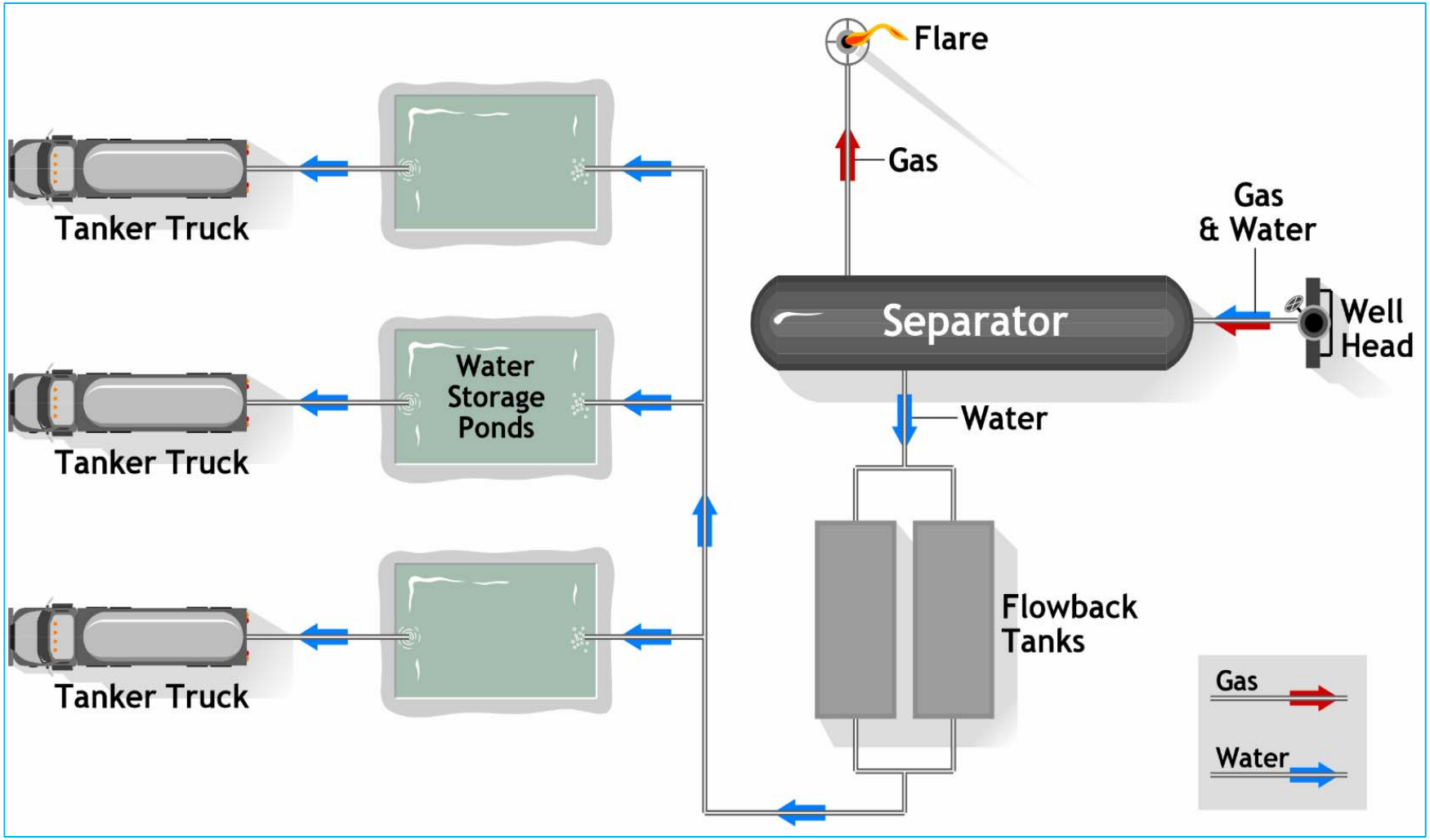
All residual fluid and solids (including that collected in the initial flowback tanks) is trucked off location and disposed at a facility in Queensland licenced to accept the material.

The flowpath of flowback fluid from Amungee NW-1H is presented in **Figure 22**.

Figure 20 Flexiponds in use during a hydraulic fracturing operation in the Northern Territory (provided by Origin)

Figure 21 Example of an enhanced evaporation system (provided by Origin)

Figure 22 Flowpath of fluid flowed back from Amungee NW-1H (provided by Origin)



A1.4.7 Fluid Systems

Hydraulic fracturing fluid is typically around 97% (v/v) water and proppant and approximately 3% (v/v) chemical additives introduced to improve the process of hydraulic fracturing. The composition of fracturing fluids varies in order to meet the specific needs of each hydraulic fracturing operation, based on local geology. Chemical additives may consist of acids, surfactants, biocides, bactericides, pH stabilisers, gel breakers, clay, corrosion and scale inhibitors, however not all additives types are used in every fracturing job.

Origin selected Schlumberger to be the hydraulic fracturing contractor for the Amungee NW-1H well.

Schlumberger successfully completed hydraulic fracturing of eleven (11) discrete intervals of the horizontal section of Amungee NW-1H. Schlumberger alternated between two (2) hydraulic fracturing fluid recipes, in order to assess the efficacy of using a slick-water versus a hybrid cross linked gel/slick-water based recipe. An inventory of chemicals in both recipes is provided in **Table 7**.

The chemical components added to the fluid recipe are summarised as follows:

- The primary additive to a slick water fluid system is the friction reducer polymer. This is added to the fluid system to reduce friction pressures generated from the interaction between the high rate fluids and the casing. It effectively reduces the fluid flow inside pipe from a turbulent flow to a laminar flow regime.
- A surfactant is a surface tension reducer, and is added to help induce fluid flowback after the hydraulic fracturing operation to clean up the reservoir.
- Clay stabilisers are added to the hydraulic fracturing fluid system to prevent clay particles from swelling and plugging off flow paths in water sensitive formations.
- Biocides are used to control bacteria growth in the water used for hydraulic fracturing. This is implemented to prevent any reservoir contamination from foreign bacteria.
- Scale Inhibitors are designed to inhibit the formation of calcium carbonate and/or barium/strontium/calcium sulphate scale.
- Guar gum is used as a thickening agent for the base fluid
- Cross linkers are used to create a gelled fluid system that increase the overall viscosity of the fluid
- Breakers are used to break down the crosslink bonds after the fluid is in the formation, returning the hydraulic fracturing fluid to its liquid state.

Table 7 Amungee NW-1H Hydraulic Fracturing Fluid Systems

Product	CAS Number	Amungee NW-1H Stage 1 Slickwater and Gel Flush	Amungee NW-1H Stage 7 Cross-Linked Gel /Slickwater Hybrid
Water	7732-18-5	✓	✓
Silicon dioxide (quartz / sand)	14808-60-7	✓	✓
Urea	57-13-6	✓	✓
Tetrasodium ethylenediaminetetraacetate	64-02-8	✓	✓
2-hydroxy-N,N,N-trimethylethanaminium chloride	67-48-1	✓	✓
Propan-2-ol	67-63-0	✓	✓
2-Propenamid (impurity)	79-06-1	✓	✓
Ethylene glycol	107-21-1	✓	✓
2,2"-oxydiethanol (impurity)	111-46-6	✓	✓
Dodecamethylcyclohexasiloxane	540-97-6	✓	✓
Decamethyl cyclopentasiloxane	541-02-6	✓	✓
Octamethylcyclotetrasiloxane	556-67-2	✓	✓
Sodium hydroxide (impurity)	1310-73-2	✓	✓
2-methyl-2h-isothiazol-3-one	2682-20-4	✓	✓
Potassium chloride (impurity)	7447-40-7	✓	✓

Product	CAS Number	Amungee NW-1H Stage 1 Slickwater and Gel Flush	Amungee NW-1H Stage 7 Cross-Linked Gel /Slickwater Hybrid
Non-crystalline silica (impurity)	7631-86-9	✓	✓
Hydrochloric acid	7647-01-0	✓	✓
Sodium chloride	7647-14-5	✓	✓
Sodium sulfate	7757-82-6	✓	✓
Copper(II) sulfate	7758-98-7	✓	✓
Ammonium sulfate	7783-20-2	✓	✓
Magnesium chloride	7786-30-3	✓	✓
Guar gum	9000-30-0	✓	✓
Calcium Chloride	10043-52-4	✓	✓
Magnesium nitrate	10377-60-3	✓	✓
Cristobalite	14464-46-1	✓	✓
5-chloro-2-methyl-2h-isothiazolol-3-one	26172-55-4	✓	✓
Polyethylene glycol monohexyl ether	31726-34-8	✓	✓
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer	38193-60-1	✓	✓
Dicoco dimethyl quaternary ammonium chloride	61789-77-3	✓	✓
Dimethyl siloxanes and silicones	63148-62-9	✓	✓
Siloxanes and silicones, dimethyl, reaction products with silica	67762-90-7	✓	✓
Diatomaceous earth, calcined	91053-39-3	✓	✓
2-Propenoic acid, polymer with sodium phosphinate	129898-01-7	✓	✓
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate	136793-29-8	✓	✓
Fumaric acid	110-17-8		✓
Boronatrocalcite	1319-33-1		✓
Sodium tetraborate	1330-43-4		✓
Monosodium fumarate	7704-73-6		✓
Sodium bromate	7789-38-0		✓
Boric acid	10043-35-3		✓
Diutan gum	595585-15-2		✓

A1.4.7.1 Mass Balance

A mass balance of all fluid systems currently used for hydraulic fracturing operations is included in **Appendix B**.

The mass balance describes the concentration of the chemical components of each fluid system, in terms of weight and volume. The concentration of each chemical injected into the target shale formation is calculated, in terms of:

- Weight for weight (w/w), i.e. kilogram of chemical per kilogram of fluid (kg/kg)
- Volume for volume (v/v), i.e. litre of chemical per litre of fluid (L/L)
- Weight per volume (w/v), i.e. milligram of chemical per Litre of fluid (mg/L)

For the (w/w) and (v/v) calculation the weight/volume of the proppant material is included in the total weight/volume of the fluid system. In the (w/v) calculation, the volume of the proppant is not included in the total volume, so as to not under-represent the concentration of each chemical relative to the overall liquid fluid system; instead the total volume used to calculate chemical concentration (w/v) includes only liquid components of the fluid in the total volume.

The mass balance also estimates the concentration of each chemical that will be returned to surface during the flowback of the hydraulically fractured well, based on an assumption of 100% hydraulic fracturing fluid recovery. This conservative scenario was selected to ensure the quantitative assessment carried out in subsequent sections of this report considered a maximal-case scenario.

1.4.8 Monitoring and Assessment

Part of the hydraulic fracturing design process includes the demarcation of an acceptable safe operating pressure envelope. The key information provided in this envelope is the minimum pressures and maximum rates acceptable. If the operating pressure surpasses these parameters it is possible that a loss of fracture integrity has occurred.

During the hydraulic fracturing operation the injection pressure and fluid volume is continuously monitored to immediately identify a condition that would indicate the loss of integrity of the well casing (see **Section A1.4.5**). In the event a potential loss of integrity is observed the operation would be ceased immediately.

In addition to this monitoring, Origin monitor the pressure, flowrate and the chemistry of water flowed back from gas wells throughout their production phase for operational reasons.

A1.4.8.1 Production Well Pressure and Water Quality Monitoring

During the hydraulic fracturing operation the injection pressure is continuously monitored to immediately identify a condition that would indicate the loss of integrity of the well casing or overburden formation. The injection pressure is the principal indicator of a loss of the integrity of the well casing.

An unexpected drop in injection pressure could be indicative of a failure of the well casing or a new fracture being propagated inside the target formation.

The determination of whether a pressure drop is indicative of a loss of containment is based on the safe operating pressure envelope, determined by Origin for each well. If the fracturing pressure drops below the safe operating pressure envelope, hydraulic fracturing is immediately ceased.

Following the completion of the hydraulic fracturing operation and the commissioning of the production well, formation pressure, flowrates and the quality (chemistry) of water flowed back from gas wells is continually monitored. A change in pressure or flow rate is used as a primary indicator of potential loss of containment warranting further investigation. A change in water chemistry may take some time to become apparent in the groundwater flowing back from the well, hence changes in water chemistry is used as a secondary indicator warranting further investigation.

A1.4.9 Comparison to International Best Practice

The American Petroleum Institute (API) documents developed over this period provide the industry best practice guidance for the hydraulic fracturing operations and the management of hydraulic fracturing fluids. The API is the primary distributor of industry standards and guidance, primarily through their recommended practises, technical reports and standards publications. The API specifications relevant to hydraulic fracturing are presented in **Figure 23**.

The API has also published three guidance documents that outline best practices to minimise environmental impacts associated with hydraulic fracturing. These are:

1. ***Hydraulic Fracturing Operations – Well Construction and Integrity G*uidelines**

   This document focuses on groundwater protection related to drilling and hydraulic fracturing operations.

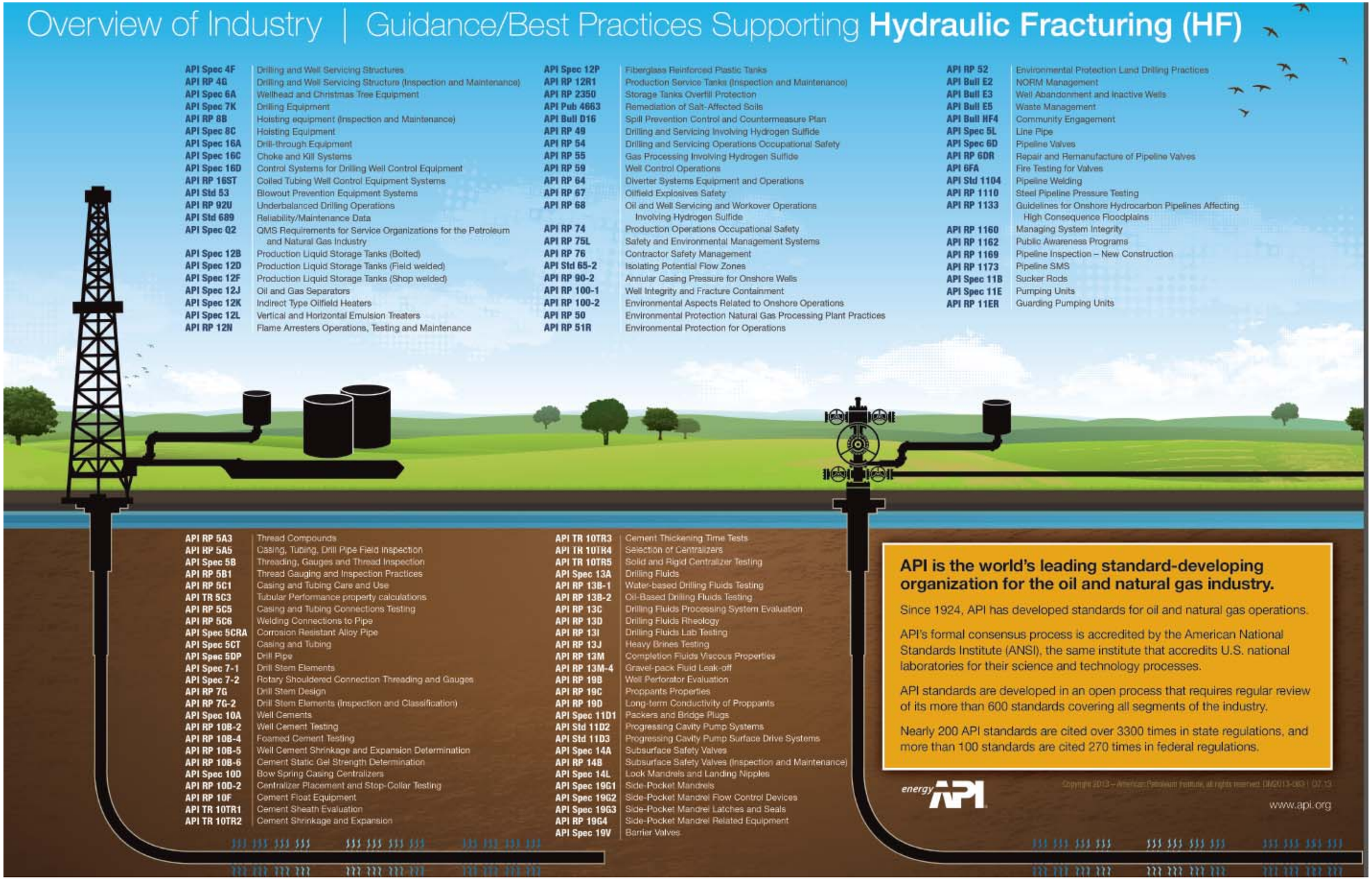
2. ***Water Management Associated with Hydraulic Fracturing***

   This document focuses on the management of water used for hydraulic fracturing.

3. ***Practices for Mitigating Surface Impacts Associated with Hydraulic Fracturing***

   This document outlines potential impacts to surface waters, soils, and other ecosystems and describes best practises to mitigate these environmental impacts. It is understood that Origin and Origin contractors adhere to the API guidance and ‘Best Practice’ specifications for the design and implementation of all hydraulic fracturing operations that are conducted for the Beetaloo Project. In addition, Origin complies with all regulatory directives regarding hydraulic fracturing process.

Figure 23 API Guidance/Best Practices supporting Hydraulic Fracture (2013, API)



Appendix B

Hydraulic Fracturing Fluid Mass Balance

		Schlumberger Amundgee NW-1H - Stage 1 (Slickwater with Gel Flush)							Schlumberger Amundgee NW-1H - Stage 7 (Cross-Linked Gel / Slickwater Hybrid)						
Chemical Name	**CAS Number**	**Volume of Chemical (L)**	**Volume Fraction (%v/v)**	**Chemical Mass in Fluid (kg)**	**Mass Fraction (% w/w)**	**Concentration in Flowback Fluid assuming 100% Recovery (mg/L)**	**Flowback Concentration Assuming 20% Mass Recovery (mg/L)**	**Flowback Concentration Assuming 60% Mass Recovery (mg/L)**	**Volume of Chemical (L)**	**Volume Fraction (%v/v)**	**Chemical Mass in Fluid (kg)**	**Mass Fraction (% w/w)**	**Concentration in Flowback Fluid assuming 100% Recovery (mg/L)**	**Flowback Concentration Assuming 20% Mass Recovery (mg/L)**	**Flowback Concentration Assuming 60% Mass Recovery (mg/L)**
Urea	57-13-6	5.79	0.0008%	4.98	0.00064%	7	1.37	4.11	4.2	0.0003%	#	0.00023%	3	0.52	1.56
Tetrasodium ethylenediaminetetraacetate	64-02-8	0.12	0.0000%	0.16	0.00002%	0	0.04	0.13	0.1	0.0000%	#	0.00001%	0	0.03	0.09
2-hydroxy-N,N,N-trimethylethanaminium chloride	67-48-1	990.13	0.1327%	1,089.14	0.14003%	1499	299.88	899.65	2,129.60	0.1465%	#	0.14963%	1693	338.55	1015.66
Propan-2-ol	67-63-0	1.58	0.0002%	1.24	0.00016%	2	0.34	1.02	3	0.0002%	#	0.00015%	2	0.35	1.04
2-Propenamid (impurity)	79-06-1	0.21	0.0000%	0.23	0.00003%	0	0.06	0.19	0.1	0.0000%	#	0.00001%	0	0.03	0.09
Ethylene glycol	107-21-1	87.92	0.0118%	78.16	0.01005%	108	21.52	64.56	561.3	0.0386%	#	0.03187%	361	72.12	216.35
2,2''-oxydiethanol (impurity)	111-46-6	0.48	0.0001%	0.78	0.00010%	1	0.21	0.64	0.9	0.0001%	#	0.00009%	1	0.20	0.61
Dodecamethylcyclohexasiloxane	540-97-6	0.04	0.0000%	0.08	0.00001%	0	0.02	0.07	0.1	0.0000%	#	0.00001%	0	0.03	0.09
Decamethyl cyclopentasiloxane	541-02-6	0.04	0.0000%	0.08	0.00001%	0	0.02	0.07	0.1	0.0000%	#	0.00001%	0	0.03	0.09
Octamethylcyclotetrasiloxane	556-67-2	0.04	0.0000%	0.08	0.00001%	0	0.02	0.07	0.1	0.0000%	#	0.00001%	0	0.03	0.09
Sodium hydroxide (impurity)	1310-73-2	0.18	0.0000%	0.39	0.00005%	1	0.11	0.32	130.5	0.0090%	#	0.01775%	201	40.16	120.49
2-methyl-2h-isothiazol-3-one	2682-20-4	0.68	0.0001%	0.86	0.00011%	1	0.24	0.71	1.3	0.0001%	1.6	0.00010%	1	0.23	0.69
Potassium chloride (impurity)	7447-40-7	0.12	0.0000%	0.23	0.00003%	0	0.06	0.19	0.2	0.0000%	0.5	0.00003%	0	0.07	0.22
Non-crystalline silica (impurity)	7631-86-9	0.49	0.0001%	1.17	0.00015%	2	0.32	0.97	3.3	0.0002%	8	0.00051%	6	1.16	3.47
Hydrochloric acid	7647-01-0	350.82	0.0470%	420.99	0.05413%	580	115.91	347.74	530	0.0365%	635.9	0.04062%	460	91.90	275.70
Sodium chloride	7647-14-5	6.83	0.0009%	14.78	0.00190%	20	4.07	12.21	12.4	0.0009%	26.8	0.00171%	19	3.87	11.62
Sodium sulfate	7757-82-6	0.59	0.0001%	1.17	0.00015%	2	0.32	0.97	0.4	0.0000%	0.8	0.00005%	1	0.12	0.35
Copper(II) sulfate	7758-98-7	0.04	0.0000%	0.08	0.00001%	0	0.02	0.07	0.1	0.0000%	0.2	0.00001%	0	0.03	0.09
Ammonium sulfate	7783-20-2	102.26	0.0137%	103.28	0.01328%	142	28.44	85.31	72.5	0.0050%	73.2	0.00468%	53	10.58	31.74
Magnesium chloride	7786-30-3	1.17	0.0002%	2.72	0.00035%	4	0.75	2.25	2.2	0.0002%	5	0.00032%	4	0.72	2.17
Guar gum	9000-30-0	65.22	0.0087%	45.65	0.00587%	63	12.57	37.71	9.5	0.0007%	13.6	0.00087%	10	1.97	5.90
Calcium Chloride	10043-52-4	5.22	0.0007%	7.47	0.00096%	10	2.06	6.17	#	0.0007%	13.6	0.00087%	10	1.97	5.90
Magnesium nitrate	10377-60-3	6.21	0.0008%	5.52	0.00071%	8	1.52	4.56	#	0.0008%	10	0.00064%	7	1.45	4.34
Cristobalite	14464-46-1	0.21	0.0000%	0.54	0.00007%	1	0.15	0.45	#	0.0000%	0.9	0.00006%	1	0.13	0.39
5-chloro-2-methyl-2h-isothiazolol-3-one	26172-55-4	2.36	0.0003%	2.96	0.00038%	4	0.82	2.45	#	0.0003%	5.3	0.00034%	4	0.77	2.30
Polyethylene glycol monohexyl ether	31726-34-8	144.82	0.0194%	148	0.01903%	204	40.75	122.25	#	0.0194%	288.6	0.01843%	209	41.71	125.13
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer	38193-60-1	57.18	0.0077%	76.22	0.00980%	105	20.99	62.96	# #	0.0028%	53.9	0.00344%	39	7.79	23.37
Dicoco dimethyl quaternary ammonium chloride	61789-77-3	6.14	0.0008%	6.14	0.00079%	8	1.69	5.07	#	0.0008%	12.1	0.00077%	9	1.75	5.25
Dimethyl siloxanes and silicones	63148-62-9	0.08	0.0000%	0.08	0.00001%	0	0.02	0.07	#	0.0000%	0.2	0.00001%	0	0.03	0.09
Siloxanes and silicones, dimethyl, reaction products with silica	67762-90-7	0.1	0.0000%	0.08	0.00001%	0	0.02	0.07	# #	0.0000%	0.2	0.00001%	0	0.03	0.09
Diatomaceous earth, calcined	91053-39-3	58.41	0.0078%	27.45	0.00353%	38	7.56	22.67	106.1	0.0073%	49.9	0.00319%	36	7.21	21.63
2-Propenoic acid, polymer with sodium phosphinate	129898-01-7	28.12	0.0038%	73.11	0.00940%	101	20.13	60.39	51.2	0.0035%	133	0.00850%	96	19.22	57.66
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate	136793-29-8	4.19	0.0006%	8.17	0.00105%	11	2.25	6.75	3	0.0002%	5.8	0.00037%	4	0.84	2.51
Fumaric acid	110-17-8								9.9	0.0007%	16.2	0.00103%	12	2.34	7.02
Boronatrocalcite	1319-33-1								289.4	0.0199%	616.3	0.03937%	445	89.07	267.20
Sodium tetraborate	1330-43-4								21.9	0.0015%	51.9	0.00332%	38	7.50	22.50
Monosodium fumarate	7704-73-6								7.7	0.0005%	16.2	0.00103%	12	2.34	7.02
Sodium bromate	7789-38-0								62	0.0043%	207	0.01322%	150	29.92	89.75
Boric acid	10043-35-3								11.3	0.0008%	16.2	0.00103%	12	2.34	7.02
Diutan gum	595585-15-2								0.3	0.0000%	0.8	0.00005%	1	0.12	0.35
Proppants															
Quartz, Crystalline silica	14808-60-7	19,706.36	2.64%	51,236.60	6.58730%	70537	14107.43	42322.30	69,481.60	9.31%	180,652.30	23.22579%	130539	26107.83	78323.50
Water															
Water (inclusive of mix water and water in additives)	7732-18-5	724449.6	97.10%	724450.4	93.13989%				1,379,507.90	94.92%	1379509.4	88.11641%			
Total Chemical Additivies		1928		2,122					4386		5,392				
Total Proppant		19706		51237					69482		180652				
Total Water		724449.6		724450					1379507.9		1379509				
% Chemical Additivies		0.26%		0.27%					0.30%		0.34%				
% Proppant		2.64%		6.59%					4.78%		11.54%				
% Water (in additives and make up water)		97.10%		93.14%					94.92%		88.12%				

Appendix C

Risk Calculations

Ingestion of Chemicals via Incidental Contact with Flowback Water (100% Mass Return and consideration of empirical data) - Stage 1 Slickwater and Gel Flush

Adult Trespassers

General Data/ Equations	Units	Exposure Calculations (RME) Flowback Water (100% return)	
Exposure Parameters			
Exposure Frequency (EF)	days/year	1	Assume direct contact with flowback water occurs over 1 day within the time period that the flow back pond is on-site during the pilot phase
Exposure Duration (ED)	years	0.5	Maximum duration of a flow back pond on-site during the pilot phase.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 2009
Averaging Time - Threshold (ATn)	days	182.5	USEPA 2009
Ingestion Rate (IRw)	L/day or L/hr	0.05	Assume incidental ingestion of 50 mL
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**1.3E-08** **1.8E-06**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Intake Factor
NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake 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)
Urea		-						--	
Tetrasodium ethylenediaminetetraacetate		1.9E-01		1.9E-01				--	--
2-hydroxy-N,N,N-trimethylethanaminium chloride		7.1E+01		7.1E+01	1692.76	2.1E-05	3.0E-03	--	0.000042
Propan-2-ol		4.0E+00		4.0E+00				--	--
2-Propenamid (acrylamide)		2.0E-04		2.0E-04	0.14	1.8E-09	2.5E-07	--	0.001269
Ethylene Glycol		1.5E+00		1.5E+00	360.58	4.5E-06	6.3E-04	--	0.000422
2,2''-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01				--	--
Dodecamethylcyclohexasiloxane		1.0E-01		1.0E-01				--	--
Decamethyl cyclopentasiloxane		1.0E-01		1.0E-01				--	--
Octamethylcyclotetrasiloxane		1.0E-01		1.0E-01				--	--
Sodium hydroxide (caustic soda)					200.81	2.5E-06	3.5E-04	--	--
2-methyl-2h-isothiazol- 3-one (Methylisothiazolinone)		2.0E-02		2.0E-02	1.16	1.5E-08	2.0E-06	--	0.000102
Potassium chloride		1.8E+01		1.8E+01				--	--
Non-crystalline silica								--	--
Hydrochloric Acid								--	--
Sodium chloride								--	--
Sodium sulfate								--	--
Copper(II) sulfate		5.0E-01		5.0E-01				--	--
Ammonium sulfate								--	--
Magnesium chloride								--	--
Guar gum		1.3E+01		1.3E+01	62.85	7.9E-07	1.1E-04	--	0.000009
Calcium chloride								--	--
Magnesium nitrate		1.4E+01		1.4E+01				--	--
Cristobalite								--	--
5-chloro-2-methyl-2h-isothiazolol-3-one (Methylchloroisothiazolinone)		2.0E-02		2.0E-02	3.83	4.8E-08	6.7E-06	--	0.000336
Polyethylene glycol monohexyl ether		5.0E-01		5.0E-01	208.54	2.6E-06	3.7E-04	--	0.000732
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer								--	--
Dicoco dimethyl quaternary ammonium chloride		1.0E-01		1.0E-01	8.74	1.1E-07	1.5E-05	--	0.000154
Dimethyl siloxanes and silicones		1.5E+00		1.5E+00				--	--
Siloxanes and silicones, dimethyl, reaction products with silica		1.5E+00		1.5E+00				--	--
Diatomaceous earth, calcined								--	--
2-Propenoic acid, polymer with sodium phosphinate								--	--
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate								--	--
							Total Risk (mixture)		**0.003066**
COPC identified in Empirical Data									
Arsenic		2.0E-03		2.0E-03	0.084	1.1E-09	1.5E-07	--	0.000074
Boron		2.0E-01		2.0E-01	66	8.3E-07	1.2E-04	--	0.000580
Manganese		1.4E-01		1.4E-01	2.24	2.8E-08	3.9E-06	--	0.000028
Nickel		1.2E-02		1.2E-02	0.04	5.0E-10	7.0E-08	--	0.000006
Barium		2.0E-01		2.0E-01	36.4	4.6E-07	6.4E-05	--	0.000320
Cobalt		1.4E-03		1.4E-03	0.011	1.4E-10	1.9E-08	--	0.000014
TPH C10-C16 Aromatic		4.0E-02		4.0E-02	0.245	3.1E-09	4.3E-07	--	0.000011
TPH C10-C16 Aliphatic		1.0E-01		1.0E-01	0.245	3.1E-09	4.3E-07	--	0.000004
TPH C16+ Aromatic		3.0E-02		3.0E-02	0.245	3.1E-09	4.3E-07	--	0.000014
TPH C16+ Aliphatic		2.0E+00		2.0E+00	0.245	3.1E-09	4.3E-07	--	0.000000
Benzene	3.5E-02	4.0E-03		4.0E-03	0.004	5.0E-11	7.0E-09	1.8E-12	0.000002
							Total Risk (mixture)	**1.8E-12**	**0.00105**

Dermal Exposure to Chemicals via Incidental Contact with Flowback Water (100% mass return and consideration of empirical data) - Stage 1 Slickwater and Gel Flush

Adult Trespassers

General Data/ Equations	Units	Exposure Calculations (RME) Flowback Water (100% return)	
Exposure Parameters			
Exposure Frequency (EF)	days/year	1	Assume direct contact with flowback water occurs over 1 day within the time period that the flow back pond is on-site during the pilot phase
Exposure Duration (ED)	years	0.5	Maximum duration of a flow back pond on-site during the pilot phase.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - Threshold (ATn)	days	182.5	USEPA 2009
Surface Area (SAw)	cm^2	20000	Whole adult body surface area as per enHealth 2012
Exposure Time (ET)	hr/day	1	Assume adult gets completely wet for 1 hour each time
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)	**5.0E-06** **7.0E-04**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor
NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake 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)	Dermal Permeability	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)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
Urea		-			-				--	--
Tetrasodium ethylenediaminetetraacetat		1.9E-01		1.9E-01	2.4E-14				--	--
2-hydroxy-N,N,N-trimethylethanaminium		7.1E+01		7.1E+01		1692.76			--	--
Propan-2-ol		4.0E+00		4.0E+00	7.8E-4				--	--
2-Propenamid (acrylamide)		2.0E-04		2.0E-04		0.14			--	--
Ethylene Glycol		1.5E+00		1.5E+00	9.0E-5	360.58	1.6E-07	2.3E-05	--	0.0000152
2,2"-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01	4.2E-5				--	--
Dodecamethylcyclohexasiloxane		1.0E-01		1.0E-01					--	--
Decamethyl cyclopentasiloxane		1.0E-01		1.0E-01					--	--
Octamethylcyclotetrasiloxane		1.0E-01		1.0E-01					--	--
Sodium hydroxide (caustic soda)					-	200.81			--	--
2-methyl-2h-isothiazol- 3-one (Methylisothiazolinone)		2.0E-02		2.0E-02	1.0E-4	1.16	5.8E-10	8.1E-08	--	0.0000041
Potassium chloride		1.8E+01		1.8E+01					--	--
Non-crystalline silica					-				--	--
Hydrochloric Acid					-				--	--
Sodium chloride					-				--	--
Sodium sulfate					-				--	--
Copper(II) sulfate		5.0E-01		5.0E-01					--	--
Ammonium sulfate					-				--	--
Magnesium chloride					-				--	--
Guar gum		1.3E+01		1.3E+01		62.85			--	--
Calcium chloride					-				--	--
Magnesium nitrate		1.4E+01		1.4E+01	9.3E-5				--	--
Cristobalite					-				--	--
5-chloro-2-methyl-2h-isothiazolol-3-one (Methylchloroisothiazo		2.0E-02		2.0E-02	1.4E-4	3.83	2.7E-09	3.8E-07	--	0.0000188
Polyethylene glycol monohexyl ether		5.0E-01		5.0E-01		208.54			--	--
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodiu					-				--	--
Dicoco dimethyl quaternary ammonium chloride		1.0E-01		1.0E-01		8.74			--	--
Dimethyl siloxanes and silicones		1.5E+00		1.5E+00					--	--
Siloxanes and silicones, dimethyl, reaction products with silica		1.5E+00		1.5E+00					--	--
Diatomaceous earth, calcined					-				--	--
2-Propenoic acid, polymer with sodium phosphinate					-				--	--
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium					-				--	--
								Total Risk (mixture)		**0.0000381**
COPC identified in Empirical Data										
Arsenic		2.0E-03		2.0E-03	1.0E-3	0.084	4.2E-10	5.9E-08	--	0.0000295
Boron		2.0E-01		2.0E-01	1.0E-3	66	3.3E-07	4.6E-05	--	0.0002318
Manganese		1.4E-01		1.4E-01	1.0E-3	2.24	1.1E-08	1.6E-06	--	0.0000112
Nickel		1.2E-02		1.2E-02	2.0E-4	0.04	4.0E-11	5.6E-09	--	0.0000005
Barium		2.0E-01		2.0E-01	1.0E-3	36.4	1.8E-07	2.6E-05	--	0.0001279
Cobalt		1.4E-03		1.4E-03	4.0E-4	0.011	2.2E-11	3.1E-09	--	0.0000022
TPH C10-C16 Aromatic		4.0E-02		4.0E-02	6.9E-2	0.245	8.5E-08	1.2E-05	--	0.0002986
TPH C10-C16 Aliphatic		1.0E-01		1.0E-01	6.9E-2	0.245	8.5E-08	1.2E-05	--	0.0001194
TPH C16+ Aromatic		3.0E-02		3.0E-02	3.2E-1	0.245	4.0E-07	5.6E-05	--	0.0018588
TPH C16+ Aliphatic		2.0E+00		2.0E+00	3.2E-1	0.245	4.0E-07	5.6E-05	--	0.0000279
Benzene	3.5E-02	4.0E-03		4.0E-03	1.5E-2	0.004	3.0E-10	4.2E-08	1.0E-11	0.0000105
							Total Risk (mixture)		**1.0E-11**	**0.00272**

Ingestion of Chemicals via Incidental Contact with Flowback Water (100% mass return and consideration of empirical data) - Stage 1 Slickwater and Gel Flush

Older Children Trespassers (5-15 yrs)

General Data/ Equations	Units	Exposure Calculations (RME) Flowback Water (100% return)	
Exposure Parameters			
Exposure Frequency (EF)	days/year	1	Assume direct contact with flowback water occurs over 1 day within the time period that the flow back pond is on-site during the pilot phase
Exposure Duration (ED)	years	0.5	Maximum duration of a flow back pond on-site during the pilot phase.
Body Weight (BW)	kg	39	Mean body weight (from 4-7, 8-11 and 12-15 year old data) as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 2009
Averaging Time - Threshold (ATn)	days	182.5	USEPA 2009
Ingestion Rate (IRw)	L/day or L/hr	0.05	Assume incidental ingestion of 50 mL
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**2.5E-08** **3.5E-06**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Intake Factor
NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake 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)
Urea		-						--	
Tetrasodium ethylenediaminetetraacetate		1.9E-01		1.9E-01				--	--
2-hydroxy-N,N,N-trimethylethanaminium chloride		7.1E+01		7.1E+01	1692.76	4.2E-05	5.9E-03	--	0.00008
Propan-2-ol		4.0E+00		4.0E+00				--	--
2-Propenamid (acrylamide)		2.0E-04		2.0E-04	0.14	3.6E-09	5.1E-07	--	0.00254
Ethylene Glycol		1.5E+00		1.5E+00	360.58	9.0E-06	1.3E-03	--	0.00084
2,2"-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01				--	--
Dodecamethylcyclohexasiloxane		1.0E-01		1.0E-01				--	--
Decamethyl cyclopentasiloxane		1.0E-01		1.0E-01				--	--
Octamethylcyclotetrasiloxane		1.0E-01		1.0E-01				--	--
Sodium hydroxide (caustic soda)					200.81	5.0E-06	7.1E-04	--	--
2-methyl-2h-isothiazol- 3-one (Methylisothiazolinone)		2.0E-02		2.0E-02	1.16	2.9E-08	4.1E-06	--	0.00020
Potassium chloride		1.8E+01		1.8E+01				--	--
Non-crystalline silica								--	--
Hydrochloric Acid								--	--
Sodium chloride								--	--
Sodium sulfate								--	--
Copper(II) sulfate		5.0E-01		5.0E-01				--	--
Ammonium sulfate								--	--
Magnesium chloride								--	--
Guar gum		1.3E+01		1.3E+01	62.85	1.6E-06	2.2E-04	--	0.00002
Calcium chloride								--	--
Magnesium nitrate		1.4E+01		1.4E+01				--	--
Cristobalite								--	--
5-chloro-2-methyl-2h-isothiazolol-3-one (Methylchloroisothiazolinone)		2.0E-02		2.0E-02	3.83	9.6E-08	1.3E-05	--	0.00067
Polyethylene glycol monohexyl ether		5.0E-01		5.0E-01	208.54	5.2E-06	7.3E-04	--	0.00146
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer								--	--
Dicoco dimethyl quaternary ammonium chloride		1.0E-01		1.0E-01	8.74	2.2E-07	3.1E-05	--	0.00031
Dimethyl siloxanes and silicones		1.5E+00		1.5E+00				--	--
Siloxanes and silicones, dimethyl, reaction products with silica		1.5E+00		1.5E+00				--	--
Diatomaceous earth, calcined								--	--
2-Propenoic acid, polymer with sodium phosphinate								--	--
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate								--	--
							Total Risk (mixture)		**0.0061**
COPC identified in Empirical Data									
Arsenic		2.0E-03		2.0E-03	0.084	2.1E-09	3.0E-07	--	0.0001475
Boron		2.0E-01		2.0E-01	66	1.7E-06	2.3E-04	--	0.0011591
Manganese		1.4E-01		1.4E-01	2.24	5.6E-08	7.9E-06	--	0.0000562
Nickel		1.2E-02		1.2E-02	0.04	1.0E-09	1.4E-07	--	0.0000117
Barium		2.0E-01		2.0E-01	36.4	9.1E-07	1.3E-04	--	0.0006393
Cobalt		1.4E-03		1.4E-03	0.011	2.8E-10	3.9E-08	--	0.0000276
TPH C10-C16 Aromatic		4.0E-02		4.0E-02	0.245	6.1E-09	8.6E-07	--	0.0000215
TPH C10-C16 Aliphatic		1.0E-01		1.0E-01	0.245	6.1E-09	8.6E-07	--	0.0000086
TPH C16+ Aromatic		3.0E-02		3.0E-02	0.245	6.1E-09	8.6E-07	--	0.0000287
TPH C16+ Aliphatic		2.0E+00		2.0E+00	0.245	6.1E-09	8.6E-07	--	0.0000004
Benzene	3.5E-02	4.0E-03		4.0E-03	0.004	1.0E-10	1.4E-08	3.5E-12	0.0000035
							Total Risk (mixture)	**3.5E-12**	**0.0021**

Dermal Exposure to Chemicals via Incidental Contact with Flowback Water (100% mass return and consideration of empirical data) - Stage 1 Slickwater and Gel Flush

Older Children Trespassers (5-15 yrs)

General Data/ Equations	Units	Exposure Calculations (RME) Flowback Water (20% return)	
Exposure Parameters			
Exposure Frequency (EF)	days/year	1	Assume direct contact with flowback water occurs over 1 day within the time period that the flow back pond is on-site during the pilot phase
Exposure Duration (ED)	years	0.5	Maximum duration of a flow back pond on-site during the pilot phase.
Body Weight (BW)	kg	39	Mean body weight (from 4-7, 8-11 and 12-15 year old data) as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	Averaging of exposure over 70yr lifetime as per USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	182.5	Averaging of exposure over exposure duration as per USEPA 1989 and CSMS 1996
Surface Area (SAw)	cm^2	15900	Wholebody surface area (child 11-15 yrs) as per enhealth 2012
Exposure Time (ET)	hr/day	1	Assume child gets completely wet for 1 hour each time
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)	**8.0E-06** **1.1E-03**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor
NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity Data: Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% Chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)	Dermal Permeability	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)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
Urea		-			-				--	--
Tetrasodium ethylenediaminetetraacetate		1.9E-01		1.9E-01	2.4E-14				--	--
2-hydroxy-N,N,N-trimethylethanaminium chloride		7.1E+01		7.1E+01		1692.760317			--	--
Propan-2-ol		4.0E+00		4.0E+00	7.8E-4				--	--
2-Propenamid (acrylamide)		2.0E-04		2.0E-04		0.144519791			--	--
Ethylene Glycol		1.5E+00		1.5E+00	9.0E-5	360.5768797	2.6E-07	3.6E-05	--	0.0000242
2,2"-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01	4.2E-5				--	--
Dodecamethylcyclohexasiloxane		1.0E-01		1.0E-01					--	--
Decamethyl cyclopentasiloxane		1.0E-01		1.0E-01					--	--
Octamethylcyclotetrasiloxane		1.0E-01		1.0E-01					--	--
Sodium hydroxide (caustic soda)					-	200.8102502			--	--
2-methyl-2h-isothiazol- 3-one (Methylisothiazolinone)		2.0E-02		2.0E-02	1.0E-4	1.156158332	9.2E-10	1.3E-07	--	0.0000065
Potassium chloride		1.8E+01		1.8E+01					--	--
Non-crystalline silica					-				--	--
Hydrochloric Acid					-				--	--
Sodium chloride					-				--	--
Sodium sulfate					-				--	--
Copper(II) sulfate		5.0E-01		5.0E-01					--	--
Ammonium sulfate					-				--	--
Magnesium chloride					-				--	--
Guar gum		1.3E+01		1.3E+01		62.84611742			--	--
Calcium chloride					-				--	--
Magnesium nitrate		1.4E+01		1.4E+01	9.3E-5				--	--
Cristobalite					-				--	--
5-chloro-2-methyl-2h-isothiazolol-3-one (Methylchloroisothiazolinone)		2.0E-02		2.0E-02	1.4E-4	3.829774473	4.3E-09	6.0E-07	--	0.0000299
Polyethylene glycol monohexyl ether		5.0E-01		5.0E-01		208.5420591			--	--
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer					-				--	--
Dicoco dimethyl quaternary ammonium chloride		1.0E-01		1.0E-01		8.743447382			--	--
Dimethyl siloxanes and silicones		1.5E+00		1.5E+00					--	--
Siloxanes and silicones, dimethyl, reaction products with silica		1.5E+00		1.5E+00					--	--
Diatomaceous earth, calcined					-				--	--
2-Propenoic acid, polymer with sodium phosphinate					-				--	--
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate					-				--	--
								Total Risk (mixture)		**0.000061**
COPC identified in Empirical Data										
Arsenic		2.0E-03		2.0E-03	1.0E-3	0.084	6.7E-10	9.4E-08	--	0.0000469
Boron		2.0E-01		2.0E-01	1.0E-3	66	5.3E-07	7.4E-05	--	0.0003686
Manganese		1.4E-01		1.4E-01	1.0E-3	2.24	1.8E-08	2.5E-06	--	0.0000179
Nickel		1.2E-02		1.2E-02	2.0E-4	0.04	6.4E-11	8.9E-09	--	0.0000007
Barium		2.0E-01		2.0E-01	1.0E-3	36.4	2.9E-07	4.1E-05	--	0.0002033
Cobalt		1.4E-03		1.4E-03	4.0E-4	0.011	3.5E-11	4.9E-09	--	0.0000035
TPH C10-C16 Aromatic		4.0E-02		4.0E-02	6.9E-2	0.245	1.4E-07	1.9E-05	--	0.0004748
TPH C10-C16 Aliphatic		1.0E-01		1.0E-01	6.9E-2	0.245	1.4E-07	1.9E-05	--	0.0001899
TPH C16+ Aromatic		3.0E-02		3.0E-02	3.2E-1	0.245	6.3E-07	8.9E-05	--	0.0029555
TPH C16+ Aliphatic		2.0E+00		2.0E+00	3.2E-1	0.245	6.3E-07	8.9E-05	--	0.0000443
Benzene	3.5E-02	4.0E-03		4.0E-03	1.5E-2	0.004	4.8E-10	6.7E-08	1.7E-11	0.0000166
								Total Risk (mixture)	**1.7E-11**	**0.0043**

Summary of Risk to Trespassers - Stage 1 Slickwater and Gel Flush
Exposure fo Target Chemicals - Theoretical and Empirical Data

Receptor/Exposure Pathway	Calculated Non-Threshold Risk 100% Mass Return	Calculated HI 100% Mass Return
Use of Stimulation Fluid in Hydraulic Fracturing		
Adult Trespassers		
Ingestion of Chemicals via Incidental Contact with Flowback Water		0.0031
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water		0.000038
Total Risk		**0.003**
Child Trespassers		
Ingestion of Chemicals via Incidental Contact with Flowback Water		0.0061
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water		0.000061
Total Risk		**0.006**
COPC associated with Empirical Data		
Adult Trespassers		
Ingestion of Chemicals via Incidental Contact with Flowback Water	1.8E-12	0.0011
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water	1.0E-11	0.0027
Total Risk	**1E-11**	**0.004**
Child Trespassers		
Ingestion of Chemicals via Incidental Contact with Flowback Water	3.5E-12	0.0021
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water	1.7E-11	0.0043
Total Risk	**2E-11**	**0.006**

Aquatic Toxicity Endpoints - Risk Calculations- Stage 1 Slickwater and Gel Flush

General Equations

PNEC Aquatic mg/L = E(L)C50 or NOEC / Assessment Factor

HQ = EPC / PNEC Aquatic

PNEC Aquatic = Predicted No Effects Concentration (Aquatic Receptors)
E(L)C50 = Median effective concentration
NOEC = No Observed Effect Concentration
HQ = Hazard Quotient
EPC = Exposure Point Concentration
Total HI = Hazard Index (Sum of HQ)

Chemical	Endpoint	E(L)C50 or NOEC mg/L	Assessment Factor	PNEC Aquatic mg/L	EPC 100% Mass Return mg/L	HI 100% Mass Return mg/L
Stage 1 Slickwater and Gel Flush						
Urea	L(E)C50 (algae)	29	100	0.29	6.86	23.6
Tetrasodium ethylenediaminetetraacetate	21 day NOEC (Daphnia)	22	10	2.2	0.22	0.1
2-hydroxy-N,N,N-trimethylethanaminium chloride	NOEC (Daphnia)	32	10	3.2	1499.41	468.6
Propan-2-ol	NOEC (Daphnia)	30	100	0.3	1.71	5.7
2-Propenamid (impurity)	72-h EC50 (freshwater algae)	33.85	1000	0.03385	0.32	9.4
Ethylene glycol	NOEC	3469	10	346.9	107.60	0.3
2,2''-oxydiethanol (impurity)	NOEC (algae)	100	50	2	1.07	0.5
Dodecamethylcyclohexasiloxane	NOEC	0.0046	No AF	0.0046	0.11	23.9
Decamethyl cyclopentasiloxane	NOEC	0.0046	No AF	0.0046	0.11	23.9
Octamethylcyclotetrasiloxane	NOEC	0.0046	No AF	0.0046	0.11	23.9
Sodium hydroxide (impurity)	NA. Not a PBT substance.					
2-methyl-2h-isothiazol-3-one	72-hr EC50 (algae)	0.027	1,000	0.000027	1.18	43850.3
Potassium chloride (impurity)	72-hr EC50 (algae)	100	1000	0.1	0.32	3.2
Non-crystalline silica (impurity)	NA. Not a PBT substance.					
Hydrochloric acid	NA. Not a PBT substance.					
Sodium chloride	NA. Not a PBT substance.					
Sodium sulfate	EC50 120h (Daphnia)	1,900	1000	1.9	1.61	0.8
Copper(II) sulfate	ANZECC Fresh Water Guidelines			0.0014	0.11	78.7
Ammonium sulfate	ANZECC Fresh Water Guidelines			0.9	142.19	158.0
Magnesium chloride	72-hr EC50 (algae)	100	1000	0.1	3.74	37.4
Guar gum	48-hr EC50 (Daphnia)	42	1,000	0.042	62.85	1496.3
Calcium Chloride	Chronic Daphnia	160	50	3.2	10.28	3.2
Magnesium nitrate	ANZECC Fresh Water Guidelines			0.7	7.60	10.9
Cristobalite	NA. Not a PBT substance.					
5-chloro-2-methyl-2h-isothiazolol-3-one	72-hr EC50 (algae)	0.027	1000	0.000027	4.08	150926.5
Polyethylene glycol monohexyl ether	Chronic Daphnia	26.3	10	2.63	203.75	77.5
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer	NA. Polymer is non-toxic to aquatic receptors.					
Dicoco dimethyl quaternary ammonium chloride	96 h EC50 (algae)	0.014	100	0.0028	8.45	3018.9
Dimethyl siloxanes and silicones	NA. Polymer is non-toxic to aquatic receptors.					
Siloxanes and silicones, dimethyl, reaction products with silica	NA. Polymer is non-toxic to aquatic receptors.					
Diatomaceous earth, calcined	NA. Not a PBT substance.					
2-Propenoic acid, polymer with sodium phosphinate	NA. Polymer is non-toxic to aquatic receptors.					
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate	NA. Polymer is non-toxic to aquatic receptors.					
					Total HI	**200242**
COPC identified in flowback water analysis from current operations						
Arsenic	ANZECC (2000) Freshwater quality guideline			0.024	0.084	3.5
Boron	ANZECC (2000) Freshwater quality guideline			0.37	66	178.4
Manganese	ANZECC (2000) Freshwater quality guideline			1.9	2.24	1.2
Nickel	ANZECC (2000) Freshwater quality guideline			0.011	0.04	3.6
Barium	USEPA Region 3 Biological Technical Assistance Group Freshwater Screening Benchmarks			0.004	36.4	9100.0
Chromium	ANZECC (2000) Freshwater quality guideline			0.001	0.055	55.0
3-&4-methylphenol	ANZECC (2000) Freshwater quality guideline (as phenol)			0.32	0.762	2.4
TPH C10-C34	ANZECC (2000) Freshwater quality guideline			0.7	0.98	1.4
					Total HI	**9345**

Ingestion of Chemicals via Incidental Contact with Flowback Water (100% Mass Return and consideration of empirical data) - STAGE 7 Cross-Linked Gel/Slickwater Hybrid

Adult Trespassers

General Data/ Equations	Units	Exposure Calculations (RME) Flowback Water (100% return)	
Exposure Parameters			
Exposure Frequency (EF)	days/year	1	Assume direct contact with flowback water occurs over 1 day within the time period that the flow back pond is on-site during the pilot phase
Exposure Duration (ED)	years	0.5	Maximum duration of a flow back pond on-site during the pilot phase.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 2009
Averaging Time - Threshold (ATn)	days	182.5	USEPA 2009
Ingestion Rate (IRw)	L/day or L/hr	0.05	Assume incidental ingestion of 50 mL
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**1.3E-08** **1.8E-06**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Intake Factor
NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake 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)
Urea		-						--	
Tetrasodium ethylenediaminetetraacetate		1.9E-01		1.9E-01				--	--
2-hydroxy-N,N,N-trimethylethanaminium chloride		7.1E+01		7.1E+01	1692.76	2.1E-05	3.0E-03	--	0.000042
Propan-2-ol		4.0E+00		4.0E+00				--	--
2-Propenamid (acrylamide)		2.0E-04		2.0E-04	0.14	1.8E-09	2.5E-07	--	0.001269
Ethylene Glycol		1.5E+00		1.5E+00	360.58	4.5E-06	6.3E-04	--	0.000422
2,2"-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01				--	--
Dodecamethylcyclohexasiloxane		1.0E-01		1.0E-01				--	--
Decamethyl cyclopentasiloxane		1.0E-01		1.0E-01				--	--
Octamethylcyclotetrasiloxane		1.0E-01		1.0E-01				--	--
Sodium hydroxide (caustic soda)					200.81	2.5E-06	3.5E-04	--	--
2-methyl-2h-isothiazol- 3-one (Methylisothiazolinone)		2.0E-02		2.0E-02	1.16	1.5E-08	2.0E-06	--	0.000102
Potassium chloride		1.8E+01		1.8E+01				--	--
Non-crystalline silica								--	--
Hydrochloric Acid								--	--
Sodium chloride								--	--
Sodium sulfate								--	--
Copper(II) sulfate		5.0E-01		5.0E-01				--	--
Ammonium sulfate								--	--
Magnesium chloride								--	--
Guar gum		1.3E+01		1.3E+01				--	--
Calcium chloride								--	--
Magnesium nitrate		1.4E+01		1.4E+01				--	--
Cristobalite								--	--
5-chloro-2-methyl-2h-isothiazolol-3-one (Methylchloroisothiazolinone)		2.0E-02		2.0E-02	3.83	4.8E-08	6.7E-06	--	0.000336
Polyethylene glycol monohexyl ether		5.0E-01		5.0E-01	208.54	2.6E-06	3.7E-04	--	0.000732
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer								--	--
Dicoco dimethyl quaternary ammonium chloride		1.0E-01		1.0E-01	8.74	1.1E-07	1.5E-05	--	0.000154
Dimethyl siloxanes and silicones		1.5E+00		1.5E+00				--	--
Siloxanes and silicones, dimethyl, reaction products with silica		1.5E+00		1.5E+00				--	--
Diatomaceous earth, calcined								--	--
2-Propenoic acid, polymer with sodium phosphinate								--	--
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate								--	--
Fumaric acid		6.0E+00		6.0E+00				--	--
Boronatrocalcite		1.3E-01		1.3E-01	445.34	5.6E-06	7.8E-04	--	0.006016
Sodium tetraborate		1.3E-01		1.3E-01	37.50	4.7E-07	6.6E-05	--	0.000507
Monosodium fumarate		6.0E+00		6.0E+00				--	--
Sodium bromate		3.0E-03		3.0E-03	149.58	1.9E-06	2.6E-04	--	0.087565
Boric acid		1.3E-01		1.3E-01	11.71	1.5E-07	2.1E-05	--	0.000158
Diutan		1.0E+00		1.0E+00				--	--
							Total Risk (mixture)		**0.097303**
COPC identified in Empirical Data									
Arsenic		2.0E-03		2.0E-03	0.084	1.1E-09	1.5E-07	--	0.000074
Boron		2.0E-01		2.0E-01	66	8.3E-07	1.2E-04	--	0.000580
Manganese		1.4E-01		1.4E-01	2.24	2.8E-08	3.9E-06	--	0.000028
Nickel		1.2E-02		1.2E-02	0.04	5.0E-10	7.0E-08	--	0.000006
Barium		2.0E-01		2.0E-01	36.4	4.6E-07	6.4E-05	--	0.000320
Cobalt		1.4E-03		1.4E-03	0.011	1.4E-10	1.9E-08	--	0.000014
TPH C10-C16 Aromatic		4.0E-02		4.0E-02	0.245	3.1E-09	4.3E-07	--	0.000011
TPH C10-C16 Aliphatic		1.0E-01		1.0E-01	0.245	3.1E-09	4.3E-07	--	0.0000043
TPH C16+ Aromatic		3.0E-02		3.0E-02	0.245	3.1E-09	4.3E-07	--	0.000014
TPH C16+ Aliphatic		2.0E+00		2.0E+00	0.245	3.1E-09	4.3E-07	--	0.00000022
Benzene	3.5E-02	4.0E-03		4.0E-03	0.004	5.0E-11	7.0E-09	1.8E-12	0.0000018
							Total Risk (mixture)	**1.8E-12**	**0.0011**

Dermal Exposure to Chemicals via Incidental Contact with Flowback Water (100% mass return and consideration of empirical data) - Stage 7 Cross-Linked Gel/Slickwater Hybrid

Adult Trespassers

General Data/ Equations	Units	Exposure Calculations (RME) Flowback Water (100% return)	
Exposure Parameters			
Exposure Frequency (EF)	days/year	1	Assume direct contact with flowback water occurs over 1 day within the time period that the flow back pond is on-site during the pilot phase
Exposure Duration (ED)	years	0.5	Maximum duration of a flow back pond on-site during the pilot phase.
Body Weight (BW)	kg	78	Average male and female adults as per enHealth 2012
Averaging Time - Threshold (ATn)	days	182.5	USEPA 2009
Surface Area (SAw)	cm^2	20000	Whole adult body surface area as per enHealth 2012
Exposure Time (ET)	hr/day	1	Assume adult gets completely wet for 1 hour each time
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)	**5.0E-06** **7.0E-04**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor
NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake 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)	Dermal Permeability	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)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
Urea		-			-				--	--
Tetrasodium ethylenediaminetetraaceta		1.9E-01		1.9E-01	2.4E-14				--	--
2-hydroxy-N,N,N-trimethylethanaminium		7.1E+01		7.1E+01		1692.76			--	--
Propan-2-ol		4.0E+00		4.0E+00	7.8E-4				--	--
2-Propenamid (acrylamide)		2.0E-04		2.0E-04		0.14			--	--
Ethylene Glycol		1.5E+00		1.5E+00	9.0E-5	360.58	1.6E-07	2.3E-05	--	0.0000152
2,2''-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01	4.2E-5				--	--
Dodecamethylcyclohexasiloxane		1.0E-01		1.0E-01					--	--
Decamethyl cyclopentasiloxane		1.0E-01		1.0E-01					--	--
Octamethylcyclotetrasiloxane		1.0E-01		1.0E-01					--	--
Sodium hydroxide (caustic soda)					-	200.81			--	--
2-methyl-2h-isothiazol- 3-one (Methylisothiazolinone)		2.0E-02		2.0E-02	1.0E-4	1.16	5.8E-10	8.1E-08	--	0.0000041
Potassium chloride		1.8E+01		1.8E+01					--	--
Non-crystalline silica					-				--	--
Hydrochloric Acid					-				--	--
Sodium chloride					-				--	--
Sodium sulfate					-				--	--
Copper(II) sulfate		5.0E-01		5.0E-01					--	--
Ammonium sulfate					-				--	--
Magnesium chloride					-				--	--
Guar gum		1.3E+01		1.3E+01					--	--
Calcium chloride					-				--	--
Magnesium nitrate		1.4E+01		1.4E+01	9.3E-5				--	--
Cristobalite					-				--	--
5-chloro-2-methyl-2h-isothiazolol-3-one (Methylchloroisothiaz		2.0E-02		2.0E-02	1.4E-4	3.83	2.7E-09	3.8E-07	--	0.0000188
Polyethylene glycol monohexyl ether		5.0E-01		5.0E-01		208.54			--	--
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sod					-				--	--
Dicoco dimethyl quaternary ammonium chloride		1.0E-01		1.0E-01		8.74			--	--
Dimethyl siloxanes and silicones		1.5E+00		1.5E+00					--	--
Siloxanes and silicones, dimethyl, reaction products with silic		1.5E+00		1.5E+00					--	--
Diatomaceous earth, calcined					-				--	--
2-Propenoic acid, polymer with sodium phosphinate					-				--	--
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodiu					-				--	--
Fumaric acid		6.0E+00		6.0E+00					--	--
Boronatrocalcite		1.3E-01		1.3E-01	1.0E-3	445.34	2.2E-06	3.1E-04	--	0.0024065
Sodium tetraborate		1.3E-01		1.3E-01	1.0E-3	37.50	1.9E-07	2.6E-05	--	0.0002027
Monosodium fumarate		6.0E+00		6.0E+00					--	--
Sodium bromate		3.0E-03		3.0E-03		149.58			--	--
Boric acid		1.3E-01		1.3E-01	1.0E-3	11.71	5.9E-08	8.2E-06	--	0.0000633
Diutan		1.0E+00		1.0E+00					--	--
								Total Risk (mixture)		**0.0027105**
COPC identified in Empirical Data										
Arsenic		2.0E-03		2.0E-03	1.0E-3	0.084	4.2E-10	5.9E-08	--	0.0000295
Boron		2.0E-01		2.0E-01	1.0E-3	66	3.3E-07	4.6E-05	--	0.0002318
Manganese		1.4E-01		1.4E-01	1.0E-3	2.24	1.1E-08	1.6E-06	--	0.0000112
Nickel		1.2E-02		1.2E-02	2.0E-4	0.04	4.0E-11	5.6E-09	--	0.0000005
Barium		2.0E-01		2.0E-01	1.0E-3	36.4	1.8E-07	2.6E-05	--	0.0001279
Cobalt		1.4E-03		1.4E-03	4.0E-4	0.011	2.2E-11	3.1E-09	--	0.0000022
TPH C10-C16 Aromatic		4.0E-02		4.0E-02	6.9E-2	0.245	8.5E-08	1.2E-05	--	0.0002986
TPH C10-C16 Aliphatic		1.0E-01		1.0E-01	6.9E-2	0.245	8.5E-08	1.2E-05	--	0.0001194
TPH C16+ Aromatic		3.0E-02		3.0E-02	3.2E-1	0.245	4.0E-07	5.6E-05	--	0.0018588
TPH C16+ Aliphatic		2.0E+00		2.0E+00	3.2E-1	0.245	4.0E-07	5.6E-05	--	0.0000279
Benzene	3.5E-02	4.0E-03		4.0E-03	1.5E-2	0.004	3.0E-10	4.2E-08	1.0E-11	0.0000105
							Total Risk (mixture)		**1.0E-11**	**0.00272**

Ingestion of Chemicals via Incidental Contact with Flowback Water (100% mass return and consideration of empirical data) - STAGE 7 Cross-Linked Gel/Slickwater Hybrid

Older Children Trespassers (5-15 yrs)

General Data/ Equations	Units	Exposure Calculations (RME) Flowback Water (100% return)	
Exposure Parameters			
Exposure Frequency (EF)	days/year	1	Assume direct contact with flowback water occurs over 1 day within the time period that the flow back pond is on-site during the pilot phase
Exposure Duration (ED)	years	0.5	Maximum duration of a flow back pond on-site during the pilot phase.
Body Weight (BW)	kg	39	Mean body weight (from 4-7, 8-11 and 12-15 year old data) as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	USEPA 2009
Averaging Time - Threshold (ATn)	days	182.5	USEPA 2009
Ingestion Rate (IRw)	L/day or L/hr	0.05	Assume incidental ingestion of 50 mL
Bioavailability (B)	-	100%	Assume 100% bioavailability via ingestion of chemicals in water.
Intake Factor = IRw*ET*B*EF*ED / BW*AT	L/kg/day	**2.5E-08** **3.5E-06**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Intake Factor
NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake 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)
Urea		-						--	
Tetrasodium ethylenediaminetetraacetate		1.9E-01		1.9E-01				--	--
2-hydroxy-N,N,N-trimethylethanaminium chloride		7.1E+01		7.1E+01	1692.76	4.2E-05	5.9E-03	--	0.00008
Propan-2-ol		4.0E+00		4.0E+00				--	--
2-Propenamid (acrylamide)		2.0E-04		2.0E-04	0.14	3.6E-09	5.1E-07	--	0.00254
Ethylene Glycol		1.5E+00		1.5E+00	360.58	9.0E-06	1.3E-03	--	0.00084
2,2"-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01				--	--
Dodecamethylcyclohexasiloxane		1.0E-01		1.0E-01				--	--
Decamethyl cyclopentasiloxane		1.0E-01		1.0E-01				--	--
Octamethylcyclotetrasiloxane		1.0E-01		1.0E-01				--	--
Sodium hydroxide (caustic soda)					200.81	5.0E-06	7.1E-04	--	--
2-methyl-2h-isothiazol- 3-one (Methylisothiazolinone)		2.0E-02		2.0E-02	1.16	2.9E-08	4.1E-06	--	0.00020
Potassium chloride		1.8E+01		1.8E+01				--	--
Non-crystalline silica								--	--
Hydrochloric Acid								--	--
Sodium chloride								--	--
Sodium sulfate								--	--
Copper(II) sulfate		5.0E-01		5.0E-01				--	--
Ammonium sulfate								--	--
Magnesium chloride								--	--
Guar gum		1.3E+01		1.3E+01				--	--
Calcium chloride								--	--
Magnesium nitrate		1.4E+01		1.4E+01				--	--
Cristobalite								--	--
5-chloro-2-methyl-2h-isothiazolol-3-one (Methylchloroisothiazolinone)		2.0E-02		2.0E-02	3.83	9.6E-08	1.3E-05	--	0.00067
Polyethylene glycol monohexyl ether		5.0E-01		5.0E-01	208.54	5.2E-06	7.3E-04	--	0.00146
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer								--	--
Dicoco dimethyl quaternary ammonium chloride		1.0E-01		1.0E-01	8.74	2.2E-07	3.1E-05	--	0.00031
Dimethyl siloxanes and silicones		1.5E+00		1.5E+00				--	--
Siloxanes and silicones, dimethyl, reaction products with silica		1.5E+00		1.5E+00				--	--
Diatomaceous earth, calcined								--	--
2-Propenoic acid, polymer with sodium phosphinate								--	--
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate								--	--
Fumaric acid		6.0E+00		6.0E+00				--	--
Boronatrocalcite		1.3E-01		1.3E-01	445.34	1.1E-05	1.6E-03	--	0.01203
Sodium tetraborate		1.3E-01		1.3E-01	37.50	9.4E-07	1.3E-04	--	0.00101
Monosodium fumarate		6.0E+00		6.0E+00				--	--
Sodium bromate		3.0E-03		3.0E-03	149.58	3.8E-06	5.3E-04	--	0.17513
Boric acid		1.3E-01		1.3E-01	11.71	2.9E-07	4.1E-05	--	0.00032
Diutan		1.0E+00		1.0E+00				--	--
							Total Risk (mixture)		**0.1946**
COPC identified in Empirical Data									
Arsenic		2.0E-03		2.0E-03	0.084	2.1E-09	3.0E-07	--	0.0001475
Boron		2.0E-01		2.0E-01	66	1.7E-06	2.3E-04	--	0.0011591
Manganese		1.4E-01		1.4E-01	2.24	5.6E-08	7.9E-06	--	0.0000562
Nickel		1.2E-02		1.2E-02	0.04	1.0E-09	1.4E-07	--	0.0000117
Barium		2.0E-01		2.0E-01	36.4	9.1E-07	1.3E-04	--	0.0006393
Cobalt		1.4E-03		1.4E-03	0.011	2.8E-10	3.9E-08	--	0.0000276
TPH C10-C16 Aromatic		4.0E-02		4.0E-02	0.245	6.1E-09	8.6E-07	--	0.0000215
TPH C10-C16 Aliphatic		1.0E-01		1.0E-01	0.245	6.1E-09	8.6E-07	--	0.0000086
TPH C16+ Aromatic		3.0E-02		3.0E-02	0.245	6.1E-09	8.6E-07	--	0.0000287
TPH C16+ Aliphatic		2.0E+00		2.0E+00	0.245	6.1E-09	8.6E-07	--	0.0000004
Benzene	3.5E-02	4.0E-03		4.0E-03	0.004	1.0E-10	1.4E-08	3.5E-12	0.0000035
							Total Risk (mixture)	**3.5E-12**	**0.0021**

Dermal Exposure to Chemicals via Incidental Contact with Flowback Water (100% mass return and consideration of empirical data) - Stage 7 Cross-Linked Gel/Slickwater Hybrid

Older Children Trespassers (5-15 yrs)

General Data/ Equations	Units	Exposure Calculations (RME) Flowback Water (20% return)	
Exposure Parameters			
Exposure Frequency (EF)	days/year	1	Assume direct contact with flowback water occurs over 1 day within the time period that the flow back pond is on-site during the pilot phase
Exposure Duration (ED)	years	0.5	Maximum duration of a flow back pond on-site during the pilot phase.
Body Weight (BW)	kg	39	Mean body weight (from 4-7, 8-11 and 12-15 year old data) as per enHealth 2012
Averaging Time - NonThreshold (ATc)	days	25550	Averaging of exposure over 70yr lifetime as per USEPA 1989 and CSMS 1996
Averaging Time - Threshold (ATn)	days	182.5	Averaging of exposure over exposure duration as per USEPA 1989 and CSMS 1996
Surface Area (SAw)	cm^2	15900	Wholebody surface area (child 11-15 yrs) as per enhealth 2012
Exposure Time (ET)	hr/day	1	Assume child gets completely wet for 1 hour each time
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)	**8.0E-06** **1.1E-03**	**NonThreshold** **Threshold**

Daily Intake from Water = Concentration in Water x Dermal Permeability x Intake Factor
NonThreshold Risk = Daily Intake for NonThreshold Effects x Slope Factor
Hazard Quotients = (Daily Intake for Threshold Effects/ADI)

Chemical	Toxicity Data: Non-Threshold Slope Factor	Chronic Threshold TDI	Background Intake (% Chronic TDI)	Chronic TDI Allowable for Assessment (TDI-Background)	Dermal Permeability	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)	(cm/hr)	(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
Urea		-			-				--	--
Tetrasodium ethylenediaminetetraacetate		1.9E-01		1.9E-01	2.4E-14				--	--
2-hydroxy-N,N,N-trimethylethanaminium chloride		7.1E+01		7.1E+01		1692.760317			--	--
Propan-2-ol		4.0E+00		4.0E+00	7.8E-4				--	--
2-Propenamid (acrylamide)		2.0E-04		2.0E-04		0.144519791			--	--
Ethylene Glycol		1.5E+00		1.5E+00	9.0E-5	360.5768797	2.6E-07	3.6E-05	--	0.0000242
2,2"-oxydiethanol (diethylene glycol)		3.0E-01		3.0E-01	4.2E-5				--	--
Dodecamethylcyclohexasiloxane		1.0E-01		1.0E-01					--	--
Decamethyl cyclopentasiloxane		1.0E-01		1.0E-01					--	--
Octamethylcyclotetrasiloxane		1.0E-01		1.0E-01					--	--
Sodium hydroxide (caustic soda)					-	200.8102502			--	--
2-methyl-2h-isothiazol- 3-one (Methylisothiazolinone)		2.0E-02		2.0E-02	1.0E-4	1.156158332	9.2E-10	1.3E-07	--	0.0000065
Potassium chloride		1.8E+01		1.8E+01					--	--
Non-crystalline silica					-				--	--
Hydrochloric Acid					-				--	--
Sodium chloride					-				--	--
Sodium sulfate					-				--	--
Copper(II) sulfate		5.0E-01		5.0E-01					--	--
Ammonium sulfate					-				--	--
Magnesium chloride					-				--	--
Guar gum		1.3E+01		1.3E+01					--	--
Calcium chloride					-				--	--
Magnesium nitrate		1.4E+01		1.4E+01	9.3E-5				--	--
Cristobalite					-				--	--
5-chloro-2-methyl-2h-isothiazolol-3-one (Methylchloroisothiazolinone)		2.0E-02		2.0E-02	1.4E-4	3.829774473	4.3E-09	6.0E-07	--	0.0000299
Polyethylene glycol monohexyl ether		5.0E-01		5.0E-01		208.5420591			--	--
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer					-				--	--
Dicoco dimethyl quaternary ammonium chloride		1.0E-01		1.0E-01		8.743447382			--	--
Dimethyl siloxanes and silicones		1.5E+00		1.5E+00					--	--
Siloxanes and silicones, dimethyl, reaction products with silica		1.5E+00		1.5E+00					--	--
Diatomaceous earth, calcined					-				--	--
2-Propenoic acid, polymer with sodium phosphinate					-				--	--
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate					-				--	--
Fumaric acid		6.0E+00		6.0E+00					--	--
Boronatrocalcite		1.3E-01		1.3E-01	1.0E-3	445.3377373	3.6E-06	5.0E-04	--	0.0038264
Sodium tetraborate		1.3E-01		1.3E-01	1.0E-3	37.50288588	3.0E-07	4.2E-05	--	0.0003222
Monosodium fumarate		6.0E+00		6.0E+00					--	--
Sodium bromate		3.0E-03		3.0E-03		149.5779841			--	--
Boric acid		1.3E-01		1.3E-01	1.0E-3	11.70610311	9.3E-08	1.3E-05	--	0.0001006
Diutan		1.0E+00		1.0E+00					--	--
								Total Risk (mixture)		**0.004310**
COPC identified in Empirical Data										
Arsenic		2.0E-03		2.0E-03	1.0E-3	0.084	6.7E-10	9.4E-08	--	0.0000469
Boron		2.0E-01		2.0E-01	1.0E-3	66	5.3E-07	7.4E-05	--	0.0003686
Manganese		1.4E-01		1.4E-01	1.0E-3	2.24	1.8E-08	2.5E-06	--	0.0000179
Nickel		1.2E-02		1.2E-02	2.0E-4	0.04	6.4E-11	8.9E-09	--	0.0000007
Barium		2.0E-01		2.0E-01	1.0E-3	36.4	2.9E-07	4.1E-05	--	0.0002033
Cobalt		1.4E-03		1.4E-03	4.0E-4	0.011	3.5E-11	4.9E-09	--	0.0000035
TPH C10-C16 Aromatic		4.0E-02		4.0E-02	6.9E-2	0.245	1.4E-07	1.9E-05	--	0.0004748
TPH C10-C16 Aliphatic		1.0E-01		1.0E-01	6.9E-2	0.245	1.4E-07	1.9E-05	--	0.0001899
TPH C16+ Aromatic		3.0E-02		3.0E-02	3.2E-1	0.245	6.3E-07	8.9E-05	--	0.0029555
TPH C16+ Aliphatic		2.0E+00		2.0E+00	3.2E-1	0.245	6.3E-07	8.9E-05	--	0.0000443
Benzene	3.5E-02	4.0E-03		4.0E-03	1.5E-2	0.004	4.8E-10	6.7E-08	1.7E-11	0.0000166
								Total Risk (mixture)	**1.7E-11**	**0.0043**

Summary of Risk to Trespassers - STAGE 7 Cross-Linked Gel/Slickwater Hybrid Exposure fo Target Chemicals - Theoretical and Empirical Data

Receptor/Exposure Pathway	Calculated Non-Threshold Risk 100% Mass Return	Calculated HI 100% Mass Return
Use of Stimulation Fluid in Hydraulic Fracturing		
Adult Trespassers		
Ingestion of Chemicals via Incidental Contact with Flowback Water		0.097
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water		0.0027
Total Risk		**0.1**
Child Trespassers		
Ingestion of Chemicals via Incidental Contact with Flowback Water		0.19
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water		0.0043
Total Risk		**0.2**
COPC associated with Empirical Data		
Adult Trespassers		
Ingestion of Chemicals via Incidental Contact with Flowback Water	1.8E-12	0.0011
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water	1.0E-11	0.0027
Total Risk	**1E-11**	**0.004**
Child Trespassers		
Ingestion of Chemicals via Incidental Contact with Flowback Water	3.5E-12	0.0021
Dermal Exposure to Chemicals via Incidental Contact with Flowback Water	1.7E-11	0.0043
Total Risk	**2E-11**	**0.006**

Aquatic Toxicity Endpoints - Risk Calculations-STAGE 7 Cross-Linked Gel/Slickwater Hybrid

General Equations

PNEC Aquatic mg/L = E(L)C50 or NOEC / Assessment Factor

HQ = EPC / PNEC Aquatic

PNEC Aquatic = Predicted No Effects Concentration (Aquatic Receptors)
E(L)C50 = Median effective concentration
NOEC = No Observed Effect Concentration
HQ = Hazard Quotient
EPC = Exposure Point Concentration
Total HI = Hazard Index (Sum of HQ)

Chemical	Endpoint	E(L)C50 or NOEC mg/L	Assessment Factor	PNEC Aquatic mg/L	EPC 100% Mass Return mg/L	HI 100% Mass Return mg/L
STAGE 7 Cross-Linked Gel/Slickwater Hybrid						
Urea	L(E)C50 (algae)	29	100	0.29	2.60	9.0
Tetrasodium ethylenediaminetetraacetate	21 day NOEC (Daphnia)	22	10	2.2	0.14	0.1
2-hydroxy-N,N,N-trimethylethanaminium chloride	NOEC (Daphnia)	32	10	3.2	1692.76	529.0
Propan-2-ol	NOEC (Daphnia)	30	100	0.3	1.73	5.8
2-Propenamid (impurity)	72-h EC50 (freshwater algae)	33.85	1000	0.03385	0.14	4.3
Ethylene glycol	NOEC	3469	10	346.9	360.58	1.0
2,2"-oxydiethanol (impurity)	NOEC (algae)	100	50	2	1.01	0.5
Dodecamethylcyclohexasiloxane	NOEC	0.0046	No AF	0.0046	0.14	31.4
Decamethyl cyclopentasiloxane	NOEC	0.0046	No AF	0.0046	0.14	31.4
Octamethylcyclotetrasiloxane	NOEC	0.0046	No AF	0.0046	0.14	31.4
Sodium hydroxide (impurity)	-	-	-	-	200.81	
2-methyl-2h-isothiazol-3-one	72-hr EC50 (algae)	0.027	1,000	0.000027	1.16	42820.7
Potassium chloride (impurity)	72-hr EC50 (algae)	100	1000	0.1	0.36	3.6
Non-crystalline silica (impurity)	-	-	-	-	5.78	
Hydrochloric acid	-	-	-	-	459.50	
Sodium chloride	-	-	-	-	19.37	
Sodium sulfate	EC50 120h (Daphnia)	1,900	1000	1.9	0.58	0.3
Copper(II) sulfate	ANZECC Fresh Water Guidelines			0.0014	0.14	103.2
Ammonium sulfate	ANZECC Fresh Water Guidelines			0.9	52.89	58.8
Magnesium chloride	72-hr EC50 (algae)	100	1000	0.1	3.61	36.1
Guar gum	48-hr EC50 (Daphnia)	42	1,000	0.042	9.83	234.0
Calcium Chloride	Chronic Daphnia	160	50	3.2	9.83	3.1
Magnesium nitrate	ANZECC Fresh Water Guidelines			0.7	7.23	10.3
Cristobalite	-	-	-	-	0.65	
5-chloro-2-methyl-2h-isothiazolol-3-one	72-hr EC50 (algae)	0.027	1000	0.000027	3.83	141843.5
Polyethylene glycol monohexyl ether	Chronic Daphnia			2.63	208.54	79.3
Acrylamide, 2-acrylamido-2-methylpropanesulfonic acid, sodium salt polymer	-	-	-	-	38.95	
Dicoco dimethyl quaternary ammonium chloride	96 h EC50 (algae)	0.014	100	0.0028	8.74	3122.7
Dimethyl siloxanes and silicones	-	-	-	-	0.14	
Siloxanes and silicones, dimethyl, reaction products with silica	-	-	-	-	0.14	
Diatomaceous earth, calcined					36.06	
2-Propenoic acid, polymer with sodium phosphinate	-	-	-	-	96.11	
Polymer of 2-acrylamido-2-methylpropanesulfonic acid sodium salt and methyl acrylate	-	-	-	-	4.19	
Fumaric acid	72 hr EC50 (algae)	41	1000	0.041	11.71	285.5
Boronatrocalcite	Canadian Water Quality Guidelines			1.5	445.34	296.9
Sodium tetraborate	Canadian Water Quality Guidelines			1.5	37.50	25.0
Monosodium fumarate	72 hr EC50 (algae)	41	1000	0.041	11.71	285.5
Sodium bromate	7-day LC50	1.093	100	0.01093	149.58	13685.1
Boric acid	Canadian Water Quality Guidelines			1.5	11.71	7.8
Diutan gum	72 hr EC50 (algae)	100	100	1	0.58	0.6
					Total HI	**203546**
COPC identified in flowback water analysis from current operations						
Arsenic	ANZECC (2000) Freshwater quality guideline			0.024	0.084	3.5
Boron	ANZECC (2000) Freshwater quality guideline			0.37	66	178.4
Manganese	ANZECC (2000) Freshwater quality guideline			1.9	2.24	1.2
Nickel	ANZECC (2000) Freshwater quality guideline			0.011	0.04	3.6
Barium	USEPA Region 3 Biological Technical Assistance Group Freshwater Screening Benchma			0.004	36.4	9100.0
Chromium	ANZECC (2000) Freshwater quality guideline			0.001	0.055	55.0
3-&4-methylphenol	ANZECC (2000) Freshwater quality guideline (as phenol)			0.32	0.762	2.4
TPH C10-C34	ANZECC (2000) Freshwater quality guideline			0.7	0.98	1.4
					Total HI	**9345**

Appendix D

Hydraulic Fracturing Fluid Chemical Toxicological Assessments

2-Propenoic acid, polymer with sodium phosphinate

2-Propenoic acid, polymer with sodium phosphinate	
Chemical and Physical Properties[1,2]	
CAS number	129898-01-7
Molecular formula	(C3H4O2.H3O2P.Na)x.xNa
Molecular weight	Likely >1000 MW
Solubility in water	No information available.
pH	No information available.
Melting point	No information available.
Boiling point	No information available.
Colour/Form	No information available.
Vapour pressure	No information available.
Henrys law constant	No information available.
Explosive potential	No information available.
Flammability potential	No information 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.
Environmental Fate[1]	
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 information available.
Carcinogenicity	No information available.
Mutagenicity/Genotoxicity	No information available.
Reproductive/developmental Toxicity	No information available.
Acute Toxicity	No information available.
Irritation	No information available.
Sensitisation	No information available.
Key Study/Critical Effect for Screening Criteria	No information available.
Ecological Toxicity[1]	
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.
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	No data found
PBT Assessment[1]	

2-Propenoic acid, polymer with sodium phosphinate

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 a PBT substance

References

1. Categorization Results from the Canadian Domestic Substance List, 2-Propenoic acid, polymer with sodium phosphinate
2. 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

2-hydroxy-N,N,N-trimethylethanaminium

2-hydroxy-N,N,N-trimethylethanaminium (Choline Chloride)	
Chemical and Physical Properties[1,2,3,4]	
CAS number	67-48-1
Molecular formula	C5H14NOCl
Molecular weight	139.63 g/mole
Solubility in water	Very sol in water and alcohol.
pH	4.7 (10% solution in water)
Melting point	247°C
Boiling point	Decomposition upon heating
Colour/Form	white crystalline solid
Vapour pressure	0.0000000657 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.
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 signaling, 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.
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 tumor 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 keep 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 tumors, leukemia nor other tumors 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*

2-hydroxy-N,N,N-trimethylethanaminium

	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/developmental Toxicity	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/fetal absorptions showed that there was no All fetuses were resorbed in the 20,000 mg/kg group. Embryonic/fetal 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).
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.
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure	No data found

2-hydroxy-N,N,N-trimethylethanaminium

Limits	
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).

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Propan-2-ol (Isopopranol)

Propan-2-ol (Isopopranol)	
Chemical and Physical Properties[1,3,4,5,6]	
CAS number	67-63-0
Molecular formula	C3H8O
Molecular weight	60.10 g/mol
Solubility in water	100 vol% at 20 °C (miscible)
pH	No data found
Melting point	-88.5 C
Boiling point	82.5 C
Colour/Form	Colourless liquid with a slight alcohol odour
Vapour pressure	45.4 mm Hg at 25oC
Henrys law constant	7.52 x 10-6 atm m3/mole
Explosive potential	Is classified as explosive. The vapours may form an explosive mixture with air.
Flammability potential	Flammable liquid and vapour.
Overview	Isopropanol (IPA) is a high production volume chemical which has wide use as an industrial solvent and as a component in numerous industrial and consumer products. It has a potential for widespread exposure to both workers and consumers. Based upon physical and chemical properties, isopropanol is not expected to persist in the environment. Aerobic biodegradation of isopropanol occurs rapidly. IPA is not expected to persist in soil due to low soil adsorption and rapid evaporation to air. In the air, isopropanol is subject to rapid oxidation by hydroxyl radical attack. IPA has a low order of toxicity to aquatic organisms and plants, and bioconcentration in aquatic organisms is not expected to occur.
Environmental Fate[1,4,5,6]	
Soil/Water/Air	Based on calculated results from a Level I fugacity model, isopropanol is expected to partition primarily to the aquatic compartment (77.7%) with the remainder to the air (22.3%) (OECD, 1977a,b). Aerobic biodegradation of isopropanol has been shown to occur rapidly under nonacclimated conditions, based on a result of 49% biodegradation from a 5-day BOD test (Bridie *et al.*, 1979). Additional biodegradation data developed using standardized test methods show that isopropanol is readily biodegradable in both freshwater and saltwater media (72 to 78% biodegradation in 20 days) (Price *et al.*, 1974). Bioconcentration of isopropanol in aquatic organisms is not expected to occur based on a measured log n-octanol/water partition coefficient (log Kow) of 0.05, a calculated bioconcentration factor of 1 for a freshwater fish, and the unlikelihood of constant, longterm exposures (OECD 1977a,b).
Human Health Toxicity Summary[1,2,3,4,5,6]	
Chronic Repeated Dose Toxicity	Considering the lowest observed adverse effect levels (LOAELs) available from a 12-week rat study (1390 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. Male Wistar rats were administered the chemical at concentrations of 0, 1, 2, 3, or 5 % (0, 870, 1390, 1700, or 2500 mg/kg bw/day) in drinking water for 12 weeks. The top dose was reduced to 4 % due to unpalatability after two weeks. Significantly decreased bodyweights were seen at the two highest doses and dose-related increases in relative liver and kidney weights were also significant at 1390 mg/kg bw/day and above. Relative adrenal weights were also significantly increased at the two highest doses; increased testis weight was noted only at the top dose. A dose-dependent increase of hyaline casts and hyaline droplet formation in the proximal tubules of the kidneys was also noted. The no observed adverse effect level (NOAEL) was determined to be 870 mg/kg bw/day, based on liver and kidney effects observed at the LOAEL of 1390 mg/kg bw/day (OECD, 2002; EFSA, 2005). In another repeated dose study, rats (strain not specified) were administered the chemical in drinking water at doses of 600 or 2300 mg/kg bw/day for males and 1000 or 3900 mg/kg bw/day for females for 27 weeks. Male rats showed decreased bodyweight gain during the first 13 weeks and increased bodyweight gain for the remainder of the treatment. Female rats showed decreased bodyweight gain throughout the dosing period. No other effects

Propan-2-ol (Isopopranol)

	were reported. The NOAELs were 2300 and 1000 mg/kg bw/day for males and females, respectively. The LOAEL in females was 3900 mg/kg bw/day but could not be established in males (OECD, 2002). Several repeated dose inhalation studies were available in rats and mice. Considering the no observed adverse effect concentrations (NOAECs) available from these studies (500 ppm), and based on the treatment-related effects reported, the chemical is not considered to cause serious damage to health from repeated inhalation exposure. The kidney appears to be the target organ with kidney lesions and changes in urine chemistry indicative of impaired kidney function observed at doses ≥2500 ppm in animals exposed to the chemical for □78 weeks (effects not observed in 13-week studies). Transient signs of narcosis were observed for both mice and rats at doses ≥1500 ppm (OECD, 2002; REACH; US EPA, 1986). Although limited information is available, it has been reported that oral intake of low doses of the chemical (2.6 or 6.4 mg/kg bw/day) by groups of eight men for six weeks had no effect on their blood cells, serum or urine and also produced no clinical symptoms (HSDB).
Carcinogenicity	Based on available data, the chemical is not considered to be carcinogenic (OECD, 2002; WHO, 1990a; EFSA, 2005; REACH). The International Agency for Research on Cancer (IARC) has concluded that there is inadequate evidence for the carcinogenicity of isopropanol in laboratory animals and humans, placing the chemical in Group 3 (Not classifiable as to its carcinogenicity to humans) (IARC, 1999). Although there are no carcinogenicity studies available for the chemical by oral exposure, studies are available for inhalation exposure in rats and mice. In a carcinogenicity study (OECD TG 451), F344 rats were exposed (whole-body) through inhalation to vapours of the chemical at concentrations of 0, 500, 2500, and 5000 ppm for six hours a day, five days a week for two years. The only neoplastic lesion found was stated to be increased frequency of interstitial (Leydig) cell adenoma of the testis (77.3, 86.7 and 94.7 % at low, mid and top dose groups, respectively). The authors did not consider the tumours to be treatment related as testicular adenomas are a common finding in aged male rats and that incidence of this spontaneous tumour reported for the control group (64.9 %) of this study was lower than the historical incidence (88 %) of control F344 rats of numerous two-year National Toxicology Program (NTP) carcinogenicity studies. In a similar carcinogenicity study, CD-1 mice were also exposed (whole-body) through inhaltion to vapours of the chemical at concentrations of 0, 500, 2500, and 5000 ppm for six hours a day, five days a week for 18 months. No increased frequency of neoplastic changes was reported in any of the treated groups (OECD, 2002; EFSA, 2005; REACH).
Mutagenicity/Genotoxicity	The chemical does not show specific reproductive or developmental toxicity. Any reproductive and developmental effects were only observed secondary to maternal toxicity.
Reproductive/developmental Toxicity	The chemical does not show specific reproductive or developmental toxicity. Any reproductive and developmental effects were only observed secondary to maternal toxicity. Several one or two-generation reproductive toxicity studies (rats) and developmental studies (rats and rabbits) were available. Other than a statistically significant reduction in the male mating index observed in a recent two generation study (high dose, 1000 mg//kg bw/day second generation males), there were no other effects on reproductive indices, including fertility and gestational indices and histopathology of the reproductive organs. The NOAELs for reproductive toxicity were reported as ≥500 mg/kg bw/day. A benchmark dose (BMD) assessment was conducted for the study's developmental and reproductive findings (Shipp et al., 1996). For the offspring developmental effects, BMD dosages (BMDL5) of 449 and 418 mg/kg/day were estimated for the F1 and F2 generations, respectively. Based upon the decrease in male mating index observations in the P2 males, a BMDL10 of 407 mg/kg/day was estimated for reproductive effects (OECD, 2002; EFSA, 2005; REACH). Developmental effects, including a reduction in postnatal survival and decreased foetal bodyweights, occurred only at maternally toxic doses. No accompanying malformations

Propan-2-ol (Isopopranol)

	were observed. In a developmental toxicity study (US EPA TSCA Guidelines), pregnant Sprague Dawley (SD) rats were administered the chemical by gavage at 0, 400, 800 or 1200 mg /kg bw/day on gestational days 6–15. In the same study, pregnant New Zealand white rabbits were dosed orally with the chemical at 0, 120, 240 or 480 mg/kg bw/day during gestational days 6–18. There was no evidence of developmental toxicity in rats and rabbits at any tested dose. There was mortality of two dams (8%) at 1200 mg/kg and one dam (4%) at 800 mg/kg. Reduced maternal gestational weight gain associated with significantly reduced gravid uterine weights was noted in the higher dose group. The NOAEL for maternal toxicity in rats was reported to be 400 mg/kg bw/day. The NOAEL for developmental toxicity in rats was established as 400 mg /kg bw/day, based on significantly reduced foetal litter body weights at the 800 and 1200 mg/kg dose levels. The NOAEL for maternal toxicity in rabbits was determined to be 240 mg/kg bw/day, based on decreased maternal bodyweight and profound clinical signs (peripheral vasodilatation, cyanosis, lethargy, laboured respiration) of toxicity seen at the top dose. There was no evidence of any developmental toxicity and the NOAEL for developmental toxicity was established as the highest dose: 480 mg/kg bw/day. There was no evidence of any teratogenicity in either studies in rats and rabbits (US EPA, 1995; OECD, 2002; EFSA, 2005; HSDB; REACH).
Acute Toxicity	The chemical was of low acute toxicity in animal tests following oral exposure. The median lethal dose (LD50) in rats is greater than 2000 mg/kg bw. Observed effects included irritation and respiratory arrest while under narcosis (OECD, 2002; WHO, 1990a; HSDB). The chemical was of low acute toxicity in an animal test following dermal exposure. The median lethal dose (LD50) in rats is greater than 2000/kg mg/kg bw. Observed effects were not reported (OECD, 2002; WHO, 1990a; HSDB). IThe chemical was of low acute toxicity in animal tests following inhalation exposure with reported median lethal concentrations (LC50) >20 mg/L in rats (OECD, 2002; HSDB). Observed effects included severe irritation of the mucous membranes and central nervous system depression as indicated by ataxia, prostration and narcosis. The chemical is currently classified with the risk phrase ‘Vapours may cause drowsiness and dizziness (R67)’ in Australia (Safe Work Australia—HSIS). In an acute inhalation toxicity study (OECD TG 403), Fischer 344 (F344) rats were exposed (whole-body exposure) to the chemical at 500, 1500, 5000, and 10000 ppm for six hours (instead of the standard four hours). Transient concentration-related narcosis and/or central nervous system sedation was noted in the study and the motor activity was decreased at 1500 ppm (males only), 5000 ppm (both sexes). Severe central nervous system depression was seen in the 10000 ppm group. After one and six hours exposure at 10000 ppm, prostration, severe ataxia, decreased arousal, slowed or laboured respiration, decreased neuromuscular tone, hypothermia, and loss of reflex function was observed (OECD, 2002; REACH). Acute intoxication incidents in humans with the chemical have been reported (WHO, 1990b; OCED, 2002; HSDB). Ingestion and inhalation are the common routes of poisoning in humans. Acute intoxication of the chemical has a rapid onset (30–60 minutes) following ingestion, and reported symptoms included drowsiness, poor coordination, abdominal pain, cramps, nausea, vomiting and diarrhoea, with unconsciousness and death following massive exposure. Inhaling high concentrations of the chemical can cause nausea, headache, light headedness, drowsiness, ataxia and deep narcosis (WHO, 1990b; OECD, 2002; HSDB).
Irritation	Isopropanol applied to the intact or abraded skin of rabbits and guinea pigs produced negligible irritation (Nixon *et al.*, 1975). Liquid isopropanol is moderately irritating to the eyes of rabbits (Griffith *et al.*, 1980; WHO, 1990). Isopropanol produced little irritation when tested on the skin of six human subjects (Bevan, 2012).The chemical is classified as hazardous with the risk phrase 'Irritating to eyes' (Xi; R36) in HSIS (Safe Work Australia).

Propan-2-ol (Isopopranol)

	The available data support this classification (OECD, 2002; WHO, 1990a; REACH).
Sensitisation	There have been reports of isolated cases of dermal irritation and/or skin sensitization (Bevan, 2012). Except for three case reports, the positive reactions were observed on patch testing patients with contact dermatitis due to ethanol. These patients also had a positive reaction to ethanol. The chemical does not contain a structural alert for skin sensitisation (OECD Toolbox).
Key Study/Critical Effect for Screening Criteria	The benchmark dose level of 420 mg/kg/day from the two-generation reproductive toxicity study will be used to derive drinking water guidance for IP. Uncertainty factors: 10 (interspecies variability); 10 (intraspecies variability) Oral Reference Dose = 420/100 = 0.42 mg/kg/day Drinking water = 16.38 mg/L
Ecological Toxicity [2,4,5]	
Aquatic Toxicity	The 96-hour LC50 in *Pimephales promelas* is 9,640 mg/L (Veith *et al.*, 1983). The 24- hour EC50 in *Daphnia magna* is >10,000 mg/L (Brinkmann and Kuehn, 1977). Chronic aquatic toxicity has also been shown to be of low concern, based on 16- and 21-day NOEC values of 141 and 30 mg/L, respectively, for the freshwater invertebrate *Daphnia magna* (Hermens *et al.*, 1985); OECD, 1977a,b). Toxicity of isopropanol to plants is expected to be low, based on a 7-day toxicity threshold value of 1,800 mg/L for freshwater algae (Bringmann and Kuehn, 1980).
Determination of PNEC aquatic	PNECaquatic: Experimental results are available for three trophic levels. Acute E(L)C50 values are available for fish (9,640 mg/L) and invertebrates (>10,000 mg/L). Results from chronic studies are available for invertebrates (16- and 21-day NOECs for Daphnia are 141 and 30 mg/L, respectively). On the basis that the data consists of a chronic study on one trophic level, an assessment factor of 100 has been applied to the lowest reported NOEC of 30 mg/L for Daphnia. The PNECaquatic is 0.3 mg/L.
Screening Criteria[7]	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	The chemical has an exposure standard of 983 mg/m³ (400 ppm) time weighted average (TWA) and 1230 mg/m³ (500 ppm) short-term exposure limit (STEL).
PBT Assessment[4,5]	
P/vP Criteria fulfilled?	Isopropanol 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 0.05 and a calculated BCF of 1, isopropanol does not meet the screening criteria for bioaccumulation.
T criteria fulfilled?	The chronic toxicity data on isopropanol show NOECs of >0.01 mg/L. Thus, isopropanol does not meet the screening criteria for toxicity.
Overall conclusion	Not a PBT substance (based on screening data).

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2,2''-oxydiethanol (Diethylene glycol)

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
pH	No data found
Melting point	-10°C
Boiling point	245°C
Colour/Form	odourless, colourless, viscous and hygroscopic liquid with a sharply sweetish taste
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 deg C
Explosive potential	Not explosive
Flammability potential	Combustible.
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 modeling 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 modeling 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

2,2''-oxydiethanol (Diethylene glycol)

	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/developmental Toxicity	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 andmice. 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

2,2''-oxydiethanol (Diethylene glycol)

	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 occuring 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.
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 (subchronic 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.
Screening Criteria [6]	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	TWA (time weighted average) = 100 mg/m^3 (Safe Work Australia).
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).

References

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2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS), 2009. Diethylene glycol (DEG)—hazard assessment
3. National Industrial Chemicals Notification and Assessment Scheme (NICNAS), 2013. Human Health Tier II assessment for Ethanol, 2,2'-oxybis-
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.

Sodium Sulfate

Sodium Sulfate	
Chemical and Physical Properties[1,3,4,5]	
CAS number	7757-82-6
Molecular formula	Na2SO4
Molecular weight	142.04 g/mol
Solubility in water	161 g/l at 20 °C
pH	No data found.
Melting point	884 °C
Boiling point	Decomposition occurs above 884°C.
Colour/Form	Solid
Vapour pressure	Expected to be extremely low
Henrys law constant	No data found.
Explosive potential	No data found.
Flammability potential	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 s 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/developmental Toxicity	Limited data of poor validity did not provide an indication of toxicity to reproduction.

Sodium Sulfate

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.
Screening Criteria[2]	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	The Australian Drinking Water Guideline for sulphate is 250 mg/L (aesthetic) and sodium is 180 mg/L (aesthetic).
Occupational Exposure Limits	No data found
PBT Assessment[3,4,5]	
P/vP Criteria fulfilled?	Sodium sulphate is an organic 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

Copper (II) Sulfate

Copper (II) Sulfate	
Chemical and Physical Properties[1,2,3,4,6]	
CAS number	7758-98-7
Molecular formula	$CuSO_4.5H_2O$
Molecular weight	159.6 g/mol
Solubility in water	266 g/L
pH	No data found.
Melting point	590 °C
Boiling point	Decomposition to CuO occurs at 650°C.
Colour/Form	White hygroscopic crystals
Vapour pressure	Negligible
Henrys law constant	No data found.
Explosive potential	Not explosive, however reacts violently with hydroxylamine. This generates fire hazard. Reacts with magnesium. This produces flammable/explosive gas
Flammability potential	Not flammable. Gives off irritating or toxic fumes/gases in a fire.
Overview	Copper is an essential nutrient for humans and non-human organisms and, therefore, low concentrations may lead to deficiency while high concentrations of copper ions may lead to copper toxicity. Once released to the environment, copper ions have more than one oxidation state. The principal ionic forms are cuprous (Cu(I), Cu+) and cupric (Cu(II), Cu2+). The trivalent form (Cu(III), Cu3+) occurs but is relatively unimportant in physical and biological systems. Cu+ is unstable in aqueous media and Cu1+-ions readily trans-form into Cu2+-ions. Depending on the chemistry of the receiving environment, soluble and/or insoluble copper compounds are formed. Hence Cu1+-ions are, due to their instability, considered as a source of Cu2+ ions for environmental and systemic toxicity. For the environmental endpoints and systemic health endpoints, the copper ion is considered as the reactive functional group within the category. The counter-ions of the copper salts (i.e. oxygen, sulphate, chloride and hydroxide), are due to their ubiquitous presence in environmental media and human fluids not considered to contribute to the environmental nor systemic human toxicity of copper salts. Copper is an essential trace element for humans. It is estimated that adult requirements are about 2–3 mg per person per day. High doses of copper (above 50 mg/kg bodyweight) can be lethal. This substance is used in the following products: adsorbents, pH regulators and water treatment products, polymers, coating products, fertilisers, adhesives and sealants, fillers, putties, plasters, modelling clay, lubricants and greases, photo-chemicals, polishes and waxes, inks and toners, leather treatment products, textile treatment products and dyes, washing & cleaning products, cosmetics and personal care products, metal surface treatment products, laboratory chemicals, nonmetal-surface treatment products, water treatment chemicals, extraction agents, metals and water softeners. NICNAS has performed an IMAP environment Tier 1 summary which concluded that copper sulfate could be used in cosmetics but only at low concentrations.
Environmental Fate[1,3,4,5]	
Soil/Water/Air	Copper is a naturally occurring element that can be found at background levels in water, sediment and soil Hydrolysis, biodegradation and phototransformation are not applicable endpoints for copper and the inorganic copper salts. The occurrence of various copper species will depend on the characteristics of the receiving environment. 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. Scientific information on copper bioaccumulation factors (BCF, BAF, TTF) does not support the use of BAF or BCF values when they are used as traditional generic threshold criteria for the hazard potential since they are not an intrinsic

Copper (II) Sulfate

	property for copper. Therefore, for inorganic copper compounds, bioaccumulation factors should be used with caution
Human Health Toxicity Summary[1,3,4,5,6]	
Chronic Repeated Dose Toxicity	The copper sulphate studies indicate that there is no evidence to indicate that copper or copper compounds present a hazard for repeat dose oral toxicity. A NOAEL of 16.7 mgCu/kg/bw/day was determined for male and female rats in a subchonic 92 day study, where copper sulfate pentalhydrate was administrated in the feed. Symptoms included hyperplasia of the squamous, liver inflammation, altered clinical chemistry and urinary parameters, increased cytoplasmic droplets. A NOAEL of 97.2 mgCu/kg/bw/day for male rats and a NOAEL of 125.7 mgCu/kg/bw/day for female rats, was determined for rats in a subchronic 92 day oral study.
Carcinogenicity	Dietary copper/copper compounds have been administered orally to rats in long-term studies. None of the studies presented below meets exactly the requirements of the International Guidelines, but they do show conclusively that copper has no carcinogenic activity. Studies performed include investigative studies demonstrating no tumour formation or long term effects even at very high dose levels, and co-administration with known carcinogens to demonstrate that copper is effective at reducing the incidence and delaying the onset of tumours.
Mutagenicity/Genotoxicity	Copper and copper compounds are not genotoxic
Reproductive/developmental Toxicity	Copper has no reproductive or developmental toxicity potential.
Acute Toxicity	The acute oral effects - LD50 , oral, rat- observed for copper sulphate pentahydrate: 481 mg/kg. Oral: the release of copper ions drive the acute oral toxicity. Dicopper oxide, copper sulphate pen-tahydrate, dicopper chloride hydroxide and coated copper flakes present a hazard by the oral route. Copper oxide, copper powder and copper massive are not hazardous by the oral route. Dermal: all tested substances within the category are not hazardous by the dermal route. Considering the lower solubility and bioaccessability of copper powders/massives compared to the tested copper substances, copper powders/massive forms do not present a hazard by the dermal route. Inhalation: Dicopper oxide, dicopper chloride hydroxide and copper flakes are considered hazardous by the inhalation route. Copper sulphate pentahydrate, copper oxide and copper powder/massive are not hazardous by inhalation route
Irritation	A key OECD 406 guideline study for irritation (skin, eye and respiratory tract) on rabbits indicated that copper sulphate presents severe eye irritation. Copper sulphate is considered a mild irritant to intact skin, and contact dermatitis has been reported. Very small ingestion (milligrams) are likely to cause only nausea and vomiting.
Sensitisation	Copper sulphate is not a skin sensitiser
Key Study/Critical Effect for Screening Criteria	The Australian Drinking Water Guidelines for copper (2 mg/L) and sulphate (250 mg/L) may apply.
Ecological Toxicity [1,3,4,5]	
Aquatic Toxicity	Copper and copper compounds may present a hazard for the environment depending on the re-lease/bioaccessibility of copper ions and on the conditions of the receiving environment (pH, hardness, presence and type of organic matter, anions and competing cations) High quality, acute L(E)C50 values were retained from short term standard freshwater tests for 10 standard fish, invertebrate and algae test species (*Pseudokirchneriella subcapitata, Chlamydomonas reinhardtii*, *Chlorella vul-garis*, *Ceriodaphnia dubia*, *Daphnia magna, Oncorhynchus mykiss*, *Pimephales promelas, Lepomis macrochirus, Brachydanio rerio* and *Cyprinus carpio)*. Reliable values range between 3 and 9,150 µg Cu/L. The variability has been attributed to species-specific differences in sensitivity as well as water-characteristics such as pH, DOC and hardness. Species-specific geometric mean values range between 34 µg Cu/L (*Ceriodaphnia dubia*) and 2837 µg Cu/L (*Lepomis macrochirus*).

Copper (II) Sulfate

	High quality, chronic NOEC(s) / EC10 values were retained from standard freshwater tests for 9 standard fish, in-vertebrate and aquatic plant test species (*Pseudokirchneriella subcapitata, Chlorella vulgaris, Chlamydomonas reinhardti* and *Lemna minor, Ceriodaphnia dubia, Daphnia magna, Oncorhynchus mykiss, Pimephales promelas* and *Salvelinus fontinalis*). Reliable values range between 2 and 337 µg Cu/L. The variability has been attributed to species and endpoint specific differences in sensitivity as well water-characteristics such as pH, DOC and hardness. Species-specific geometric mean values range between 12 µg Cu/L (*Ceriodaphnia dubia and On-corhynchus mykiss*) and 137 µg Cu/L (*Chlorella vulgaris*) Copper is very toxic to the aquatic organisms. There was no consistent pattern in differences in toxicity between the different copper variants. Organisms are considered to be exposed to dissolved copper in the aquatic phase. Therefore, the lowest observed toxicity endpoints for all copper variants were used in the risk assessment. The lowest endpoints on the acute time scale were 96h LC50 = 0.008 mg Cu (dissolved)/L for fish, EC50 = 0.0266 mg Cu (dissolved)/L for *D. magna*. The lowest endpoints on a long-term time scale were a NOEC (growth) of 0.0017 mg Cu (dissolved)/L for fish and a NOEC (reproduction) of 0.0076 mg Cu (total)/L for *D. magna*. The lowest EbC50 for algae was 0.00939 mg Cu (total)/L.
Determination of PNEC aquatic	The ANZECC 2000 Freshwater Quality Guidelines for 95% protection for copper (1.4 ug/L) may apply.
Screening Criteria[7,8,9]	
Aquatic Toxicity Guidelines	ANZECC freshwater guidelines 95% protection for copper is 1.4 ug/L
Water Quality Guidelines	The Australian Drinking Water Guideline for sulphate is 250 mg/L (aesthetic) and copper is 2 mg/L (health) and 1 mg/L (aesthetics).
Occupational Exposure Limits	Safe work Australia TWA for Copper (fume) is 0.2 mg/m^3 and the TWA for copper, dusts and mists (as Cu) is 1 mg/m^3.
PBT Assessment[3,4]	
P/vP Criteria fulfilled?	The persistent criterion is not considered applicable to copper sulfate.
B/vB criteria fulfilled?	The Bioaccumulative criterion is not considered applicable to inorganic copper compounds.
T criteria fulfilled?	The acute aquatic toxicity of copper sulfate is < 0.01 mg/L. Hence the substance is considered toxic to aquatic organisms.
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
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4. OECD Categorization Results from the Canadian Domestic Substance List – Sulfuric acid copper (2+) salt (1:1), CAS# 7758-98-7
5. European Chemicals Agency, Voluntary Risk Assessment Reports – Copper and Copper Compounds, 2008
6. IPCS INCHEM, Copper Sulfate (anhydrous) CAS# 7758-98-7
7. 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.
8. Australian and New Zealand Environment and Conservation Council, 2000. Australian and New Zealand Guidelines for Fresh and Marine Water Quality
9. Safe Work Australia 2011. Workplace Exposure Standards for Airborne Contaminants.

Ammonium Sulfate

Ammonium Sulfate	
Chemical and Physical Properties[2,4,5,6,7]	
CAS number	7783-20-2
Molecular formula	H8N2O4S
Molecular weight	132.14 g/mol
Solubility in water	764 g/l at 20°C
pH	5 - 6
Melting point	150-280°C
Boiling point	Decomposition occurs above 280°C.
Colour/Form	White solid
Vapour pressure	4.053×10^{-7} Pa (partial pressure of ammonia over solid (NH4)2SO4 at 25°C)
Henrys law constant	No data found.
Explosive potential	Non-explosive.
Flammability potential	Non-combustible, substance itself does not burn but may decompose upon heating to produce corrosive and/or toxic fumes
Overview	Ammonium salts and sulfates are abundant in the environment. Ammonium sulfate is a neutralization product of ammonia and sulfuric acid (WHO, 1986). Sulfate results from the oxidation of elemental sulfur, sulfide minerals, and organic sulfur, e.g. through the combustion of sulfur-containing fuels. Sulfates are found almost universally in natural waters at concentrations ranging from a few tenths to several thousand mg/l (EPA, 2002). In aqueous solution, ammonium sulfate is completely dissociated into the ammonium ion (NH4 +) and the sulfate anion (SO4 2-). In aqueous media, ammonium sulfate dissociates in the ammonium and sulfate ions (NH4 +, SO4 2-). These can be taken up into the body by the oral and respiratory routes. Absorbed ammonium is transported to the liver and there metabolised to urea and excreted via the kidneys. Ammonium is also an endogenous substance that serves a major role in the maintenance of the acid-base balance. Minor amounts of ammonium nitrogen are incorporated in the physiological N-pool. Sulfate is a normal intermediate in the metabolism of endogenous sulfur compounds, and is excreted unchanged or in conjugated form in urine. Ammonium sulphate has been assessed by National Industrial Chemicals Notification and Assessment Scheme (NICNAS). The assessment concluded that ammonium 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[2,5,6,7]	
Soil/Water/Air	Based on the physico-chemical properties of ammonium sulfate, water is expected to be the main target compartment. Because of the chemical structure of ammonium sulfate, the Henrys Law Constant is not assignable. Based on the high water solubility a low geoaccumulation potential and high mobility in soil is to be expected. However, due to ion-ion interactions it is to be expected that mobility in soil is significantly reduced. Ammonium sulfate will not volatilise from soil. In unsterilized soil, ammonium sulfate is mineralized fairly rapidly, and subsequently nitrified. Nitrification and de-nitrification processes also occur naturally in streams and rivers, as well as in many secondary sewage treatment processes (WHO, 1986). Based on a log Kow of –5.1 (measured; BASF AG, 1988), bioaccumulation is not expected.
Human Health Toxicity Summary[2,3,5,6,7]	
Chronic Repeated Dose Toxicity	A 14-day inhalation study on rats exposed to 300 mg/m^3, the only tested dose, did not report histopathological changes in the lower respiratory tract. As the respiratory tract is the target organ for inhalation exposure, the NOEL for toxicity to the lower respiratory tract is 300 mg/m^3. The NOAEL after feeding diets containing ammonium sulfate for 13 weeks to rats was 886 mg/kg bw/day. The only toxicity sign found was diarrhea in male animals of the high-dose group (LOAEL: 1792 mg/kg bw/day).
Carcinogenicity	High doses of ammonium sulfate may have the capability of tumor promotion in the rat stomach; it is, however, much less potent than sodium chloride when tested under identical conditions. Aluminium sulfate is not listed as a carcinogen under IARC.

Ammonium Sulfate

Mutagenicity/Genotoxicity	Ammonium sulfate was not mutagenic in bacteria (Ames test) and yeasts with and without metabolic activation systems. It did not induce chromosomal aberrations in mammalian or human cell cultures. No in vivo genotoxicity tests are available. Based on the negative results from in vitro studies and the negative results in the micronucleus test in vivo with ammonium chloride a mutagenic activity of ammonium sulfate in vivo is unlikely.
Reproductive/developmental Toxicity	There are no valid studies available on the effects of ammonium sulfate on fertility and development. Based on data from a similar ammonium compound (diammonium phosphate), which has been tested up to 1500 mg/kg bw in a screening study according to OECD TG 422 in rats it can be concluded that ammonium ions up to the dose tested have no negative effects on fertility. In the 13-week feeding study of ammonium sulfate with rats, no histological changes of testes were observed up to 1792 mg/kg bw. The ovaries were not examined. Fully valid studies with sulfate on fertility are not available. In a limited study (pretreatment time short, low number of animals, no fertility indices measured) where female mice were treated with up to ca. 6550 mg sulfate/kg bw (as sodium sulfate) no effects on litter size were found. Studies of developmental toxicity for ammonium sulfate are not available. In the screening study according to OECD TG 422 with up to 1500 mg diammonium phosphate/kg bw no effects on development have been detected in rats. In another limited screening study with exposure of mice to a single dose of 2800 mg sodium sulfate/kg bw no macroscopic effects or adverse effects on body weight gain have been detected in the pups. In both studies foetuses were not examined histopathologically.
Acute Toxicity	Ammonium sulfate is of relatively low acute toxicity (LD50, oral, rat: 2000 - 4250 mg/kg bw; LD50 dermal, rat/mouse > 2000 mg/kg bw; 8-h LC50, inhalation, rat > 1000 mg/m^3). Clinical signs after oral exposure included staggering, prostration, apathy, and laboured and irregular breathing immediately after dosing at doses near to or exceeding the LD50 value. In humans, inhalation exposure to 0.1 – 0.5 mg ammonium sulfate/m³ aerosol for two to four hours produced no pulmonary effects. At 1 mg ammonium sulfate/m^3 very slight pulmonary effects in the form of a decrease in expiratory flow, in pulmonary flow resistance and dynamic lung compliance were found in healthy volunteers after acute exposure.
Irritation	Ammonium sulfate is not irritating to the skin and eyes.
Sensitisation	No data available.
Key Study/Critical Effect for Screening Criteria	The Australian Drinking Water Guidelines for ammonia (0.5 mg/L aesthetic) and sulphate may apply 250 mg/L (aesthetic). Ammonia concentrations above 0.5 mg/L may attack copper pipes and fittings, or result in nuisance growths of microorganisms. Concentrations of ammonia that may cause health effects are unlikely to occur in drinking water supplies; accordingly, no health-based guideline is set.
Ecological Toxicity[1,4,5,6,7]	
Aquatic Toxicity	The toxicity of ammonia to aquatic organisms is highly dependent on physicochemical factors, most notably pH. The acute toxicity of ammonia is also influenced to a lesser degree by temperature, carbon dioxide, dissolved oxygen, and salinity. It is the un-ionized ammonia which is generally considered to be the primary cause of toxicity in aquatic systems. Un-ionized ammonia is more toxic to aquatic organisms than the ammonium ion because the un-ionized form is readily soluble in the lipid of the cell membrane and is rapidly absorbed by the gill. In contrast, the charged ion is not easily passed through the charged-line hydrophobic space in the membrane. Under most environmental conditions, the un-ionized ammonia concentration is the primary driver of toxicity. Information on acute toxicity in aquatic freshwater systems is available from all three trophic levels, fish, invertebrates, and algae. Fish are the most sensitive trophic level, with the most sensitive species being juvenile *Salmo gairdneri* (Thurston and Russo, 1983), with an LC50 (96 h) of ca. 173 mg/l. For invertebrates, the lowest LC50 (24 h)of 393 mg/l is observed for juveniles of the freshwater snail *Helisoma trivolyis* (Tchounwou, Englande and Malek, 1991). Some other results (e.g. Daphnia) are > 100 mg/l. For the algal species *Chlorella vulgaris* (Tam and Wong, 1996) an EC50 (18d, cell count) of ca. 2700 mg/l can be calculated. In aquatic freshwater systems, chronic toxicity information is available for fish, the trophic level shown to be the most sensitive in acute tests. The greatest sensitivity to ammonium sulfate was shown by alevins of *Oncorhynchus gorbuscha* before complete yolk absorption, with a NOEC (61 d) of 11 mg/l (NH4)2SO4 for effects on the length and weight of fry at emergence (Rice and Bailey, 1980).

Ammonium Sulfate

Determination of PNEC aquatic	ANZECC Fresh Water Guidelines 95% protection for Ammonia (900 ug/L) may apply.
Screening Criteria[1,3]	
Aquatic Toxicity Guidelines	ANZECC Fresh Water Guidelines 95% protection for Ammonia (900 ug/L)
Water Quality Guidelines	The Australian Drinking Water Guideline for ammonia is 0.5 mg/L (aesthetic) and suphate is 250 mg/L (aesthetic). There are no existing drinking water guideline values for ammonium ions.
Occupational Exposure Limits	No data found
PBT Assessment[2,5,6,7]	
P/vP Criteria fulfilled?	Ammonium sulphate is an organic salt that dissociates completely to ammonium ion and sulphate anions in aqueous solutions. The persistent criterion is not considered applicable to this inorganic salt.
B/vB criteria fulfilled?	Based on a log Kow of –5.1, bioaccumulation is not expected
T criteria fulfilled?	The acute aquatic toxicity of ammonium 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. Australian and New Zealand Environment and Conservation Council, 2000. Australian and New Zealand Guidelines for Fresh and Marine Water Quality
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. 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.
4. National Industrial Chemicals Notification and Assessment Scheme (NICNAS), IMAP Environment Tier I Summary all tranches, 2016.
5. OECD (2004a) Screening Information Dataset (SIDS) Initial Assessment Report for Ammonium Sulfate, CAS Number 7783-20-2, UNEP Publications
6. OECD (2004b) SIDS Initial Assessment Profile for Ammonium Sulfate, CAS Number 7783-20-2, UNEP Publications
7. OECD Categorisation Results from the Canadian Domestic Substance List, Sulfuric acid diammonium salt, CAS# 7783-20-2

Sodium bromate

Sodium bromate	
Chemical and Physical Properties[1,2,4,6]	
CAS number	7789-38-0
Molecular formula	BrHO3.Na
Molecular weight	150.90 g/mol
Solubility in water	36.4 g/100 mL at 20 C
pH	Aqueous solution is neutral
Melting point	350 C
Boiling point	Decomposes at 381 C
Colour/Form	Colourless crystals
Vapour pressure	Negligible
Henrys law constant	Negligible
Explosive potential	Risk of fire and explosion on contact with combustible substances or reducing agents.
Flammability potential	Not combustible but enhances combustion of other substances. Gives off irritating or toxic fumes (or gases) in a fire.
Overview	The toxicological effects of these chemicals are mediated primarily through the bromate ion. Following dissociation in water, sodium (Na+) cations are released, which are naturally occurring species and do not contribute to toxicity. Sodium bromate is used in cleaning/washing agents, surface treatments, paints, lacquers and varnishes, and in cosmetics as an oxidising agent. These chemicals dissociate in water and bromate ion is rapidly absorbed from the gastrointestinal tract, at least in part unchanged. It is distributed throughout the body appearing in plasma and urine unchanged and in other tissues as bromide. Bromate is reduced to bromide in several body tissues. Most bromate is excreted in the urine either as bromate or bromide, but some may leave the body in the faeces. Bromine has been detected in adipose tissue of mice following long-term treatment with bromate (US EPA, 2001; REACHb). Sodium bromate and potassium bromate produce similar effects and these chemicals are roughly equivalent in the delivery of bromate ions. Information on potassium bromate has been included in this toxicological profile.
Environmental Fate[1,2,5,6]	
Soil/Water/Air	Sodium bromate can be assumed to have a negligible vapour pressure, and it is therefore not expected to partition to air. Similar to many inorganic salts, sodium bromate is highly soluble in water and dissociates rapidly (primarily ionic bonds) to release the bromate ion. The bromate ion is expected to have high mobility in water and relatively little bromate is expected to partition to sediments and soils. Bromate ions found in sediments and soils are expected to be mobile in these compartments. Butler et al. (2005a) indicated that bromate is persistent in water even if this ion is thermodynamically unstable (e.g., Takeno 2005) and subject to slow biological reduction under natural conditions. In aqueous solution, bromate is highly stable at room temperature, does not volatilize and is not removed by boiling (Butler et al. 2005a). A number of studies have demonstrated that bromate can be reduced to bromide in soil, using enriched microbial communities and an appropriate carbon source (Rodgers 1980; Butler et al. 2005b). Furthermore, Rodgers (1980) observed 60% to nearly 100% conversion of BrO3− to Br− following 14-day incubation, at 25°C, of aerobic and anaerobic soils, both amended and unamended with glucose. These results suggest that natural attenuation of bromate in soil is possible. Considering published information and experimental evidence for metabolic transformation, potassium bromate does not meet the bioaccumulation criteria (BAF, BCF ≥5000)

Sodium bromate

Human Health Toxicity Summary[1,2,3,4,5,6,7]	
Chronic Repeated Dose Toxicity	A number of repeated dose oral toxicity studies in animals indicate that the kidney is the major target organ of bromate-associated toxicity, leading to carcinogenicity. Specific non-cancer effects included degenerative, necrotic, nephropathic, and regenerative changes in the kidney. In a repeated dose toxicity study, potassium bromate was administered in the drinking water at concentrations of 0, 150, 300, 600, 1250, 2500, 5000, or 10000 mg/L to male and female Fischer 344 (F344) rats (10/sex/group) for 13 weeks. All animals exposed to >1250 mg/L died within seven weeks. Significant inhibition of body weight gain was observed in males exposed to 600 or 1250 mg/L. Various-sized droplets and regenerative changes were observed in the renal tubules of treated males. A no observed adverse effect level (NOAEL) of 300 mg/L was determined (US EPA, 2001; NTP, 2007; REACHb). In a chronic toxicity/carcinogenicity study, potassium bromate was administered at 0, 250, and 500 ppm concentrations to F344 rats (53/sex/group) for 110 weeks. Daily intake of potassium bromate was equivalent to 12.5 and 27.5 mg/kg bw/day in males and 12.5 and 25.5 mg/kg bw/day in females, respectively. As the growth of males in the high dose group was severely inhibited, the concentration in this group was reduced to 400 ppm at week 60. Body weight gain was significantly reduced in high-dose males, but not in the other treated groups. Survival was reduced in high-dose males by about week 60 and in low-dose males by about week 100. No effect on survival was observed in treated female rats. A variety of non-cancer effects were reported, including: degenerative, necrotic, and regenerative changes in renal tubules; formation of hyaline droplets; thickening of transitional epithelium of the renal pelvis; papillary hyperplasia; and papillary growth. It was noted that the lesions were more extensive in degree and distribution in treated rats compared with controls, especially males. However, in the absence of information on the incidence of these lesions or on the statistical significance of these findings, a NOAEL for non-cancer effects could not be determined (US EPA, 2001; Health Canada, 2010). In another chronic study, potassium bromate was administered to male F344 rats and male B6C3F1 mice in drinking water at concentrations of 0, 0.02, 0.1, 0.2, and 0.4 g/L and 0, 0.08, 0.4, and 0.8 g/L, respectively, for 100 weeks. The doses were equal to 0, 1.5, 7.9, 16.9, and 37.5 mg/kg bw/day and 0, 9.1, 42.4, and 77.8 mg/kg bw/day, respectively, for rats and mice. In male rats, a statistically significant decrease in the mean body weight and survival was noted at the termination of the study at 0.4 g/L. The decrease in survival and body weight was attributed to an excessive mesothelioma burden. The effects on survival and body weight in rats indicate that the maximum tolerated dose (MTD) was reached in this study. A significant dose-dependent increase in the incidence of urothelial hyperplasia was noted in rats in the 0.1 g/L and higher dose groups. Foci of mineralisation of the renal papilla and eosinophilic droplets in the proximal tubule epithelium were also noted, without any information on dose levels. There were no other treatment-related non-neoplastic effects observed in any other tissue examined. On the basis of kidney effects in male rats, a NOAEL of 0.02 g/L (20 ppm; 1.5 mg/kg bw/day) was determined (US EPA, 2001; Health Canada, 2010). These results also indicate that male B6C3F1 mice may be less sensitive to the effects of bromate exposure than rats. Bromate in drinking water had no effect on the body weights and survival of male mice. There was no increased incidence of non-neoplastic lesion in any tissue examined. Therefore, the highest tested dose of 0.8 g/L (77.8 mg/kg bw/day) is a NOAEL for male mice (US EPA, 2001; Health Canada, 2010).
Carcinogenicity	Potassium bromate is currently classified as hazardous as a Category 2 carcinogen with the risk phrase ‘May cause cancer’ (T; R45) in the HSIS (Safe Work Australia). The available data support this classification (Health Canada, 1999; US EPA, 2001; WHO, 2005; REACHa). Considering that potassium bromate and sodium bromate will produce similar effects through bromate ions, a classification similar to the above is also recommended for sodium bromate. This is supported by the classification of 'bromate moiety' as a carcinogen by other regulatory agencies (Health Canada, 1999; US EPA, 2001; WHO, 2005). The International Agency for Research on Cancer (IARC) has concluded that there is inadequate evidence in humans for the carcinogenicity of potassium bromate. However, there is sufficient evidence in experimental animals for its carcinogenicity and it is classified as possibly carcinogenic to humans (Group 2B) (IARC, 1999). Health Canada has classified the bromate moiety as 'probably carcinogenic to humans, based on sufficient evidence in animals and no data in humans' (Health Canada, 1999). The US EPA has also classified the bromate moiety as a 'probable human carcinogen based on no evidence in humans, but adequate evidence of carcinogenicity in male and female rats' (Group B2 carcinogen) under previous guidelines and as a 'likely human carcinogen by the oral route of exposure, insufficient data for evaluation by the inhalation route' under current guidelines (US EPA,

Sodium bromate

	2001). Recently, the World Health Organization (WHO) evaluated the bromate moiety under the WHO Guidelines for Drinking-water Quality and stated that 'the weight of evidence from rat bioassays clearly indicates that bromate has the potential to be a human carcinogen' (WHO, 2005). Several studies have been conducted in animals by oral administration to evaluate the carcinogenic effects of potassium bromate. The kidney is the major target organ of bromate-associated toxicity, rats are more sensitive than mice to bromate treatment and specific non-cancer effects include degeneration, necrosis, nephropathic, and regenerative changes in kidneys. The chemical produced tumours in kidneys (renal tubular tumours - adenomas and carcinomas) and the thyroid (follicular cell adenomas and carcinomas) and peritoneal mesotheliomas in males rats. However, only kidney tumours were developed in female rats and these were observed in the absence of the significant toxicity observed in the male rats. The chemical also produced a low incidence of renal cell tumours in male mice and the incidence of renal tubular tumours was marginally increased in male Syrian hamsters (IRIS, 2001; US EAP, 2001; WHO, 2005; Health Canada, 2010). The exact mode of action for induction of tumours is not clear. However, considering the detection of 8-hydroxydeoxyguanosine in kidneys of rodents, the role of oxidative stress has been suggested in the formation of kidney tumours. The evidence is insufficient to establish lipid peroxidation and free radical production as key events responsible for the induction of kidney tumours. Even though the role of cell proliferation has also been proposed in the induction of tumours, the mechanism involving cell proliferation remains to be elucidated. Although bromate is mutagenic in bacteria and causes chromosomal aberrations, the role of mutation in the induction of tumours has also been questioned. The US EPA has suggested the predominant mode of action is DNA reactivity at low doses, considering the detection of tumours at relatively early time points and the positive response of bromate in a variety of genotoxicity assays (US EPA, 2001; WHO, 2005; Health Canada, 2010).
Mutagenicity/Genotoxicity	Although potassium bromate has been found to be genotoxic in a variety of assays (in vitro, in vivo), results were not sufficient to support its classification. The genotoxicity of potassium bromate has recently been linked to oxidative stress (US EPA, 2001; Health Canada, 2001; REACHa; REACHb).
Reproductive/developmental Toxicity	Limited data are available on the reproductive or developmental effects. However, the available information indicated that these chemicals are not likely to have specific reproductive or developmental effects.
Acute Toxicity	<u>Oral</u> Potassium bromate is classified as hazardous with the risk phrase ‘Toxic if swallowed’ (T; R25) in the Hazardous Substances Information System (HSIS) (Safe Work Australia). The available data (median lethal dose—LD50—157 mg/kg bw) support this classification (REACHa). Data are not available for sodium bromate. Considering that both chemicals will produce similar effects through bromate ions, a classification similar to the above is also recommended for sodium bromate (NTP, 2007; Health Canada, 2010; REACHb). <u>Dermal</u> No data are available. <u>Inhalation</u> No data are available. <u>Observation in humans</u> A number of cases of acute bromate toxicity have been reported in humans following accidental or intentional ingestion of permanent hair wave neutralising solution. These products usually contain either 2 % potassium bromate or 10 % sodium bromate. Bromate intoxication leads to gastrointestinal symptoms (abdominal pain, nausea, vomiting, diarrhoea), central nervous system depression, renal failure, and hearing loss. Although these effects are usually reversible, death from renal failure may ensue if medical intervention is not successful. Hearing loss is usually irreversible (US EPA, 2001; NTP, 2007; HSDB; REACHb).
Irritation	<u>Skin Irritation</u> Although limited data are available, the available information indicates that these chemicals are not likely to be corrosive. The purpose of the available study was to identify potential of potassium bromate for skin corrosion using an in vitro method. The study was conducted

Sodium bromate

	according to Organisation for Economic Co-operation and Development (OECD) Test Guideline (TG) 431, using a human skin model. The study consisted of a topical exposure of potassium bromate to a human reconstructed model followed by a cell viability test. Potassium bromate was not considered to possess a corrosive potential (REACHa). Eye Irritation Although limited data are available, the available information indicates that these chemicals are not likely to be eye irritants. An eye irritation study was conducted according to OECD TG 437: Bovine Corneal Opacity and Permeability Test Method for Identifying Ocular Corrosives and Severe Irritants. In this test, the damage is assessed by quantitative measurements of changes in corneal opacity and permeability with an opacitometer and a visible light spectrophotometer, respectively. Potassium bromate caused weak opacity but no permeability of the cornea compared with the results of the negative control group. The chemical was considered to be a mild eye irritant (REACHa).
Sensitisation	Skin Sensitisation The available data on potassium bromate indicate that these chemicals are not likely to be skin sensitisers. In a skin sensitisation study conducted according to OECD TG 429 (local lymph node assay—LLNA), potassium bromate (CAS No. 7758-01-2) at 1.25 %, 2.5 %, and 7.5 % (w/v) concentration was applied topically at the dorsum of each ear of female CBA mice once daily on three consecutive days. A further group of mice was treated with the positive control item and a control group of mice was also treated with the vehicle only. Stimulation Indices (S.I.) of 0.90, 0.53, and 0.64 were determined with the test item at concentrations of 1.25, 2.5, and 7.5 % (w/v), respectively. The EC3 value could not be calculated, since none of the tested concentrations induced an S.I. of greater than three. Potassium bromate was not considered to be a skin sensitiser (REACHa).
Key Study/Critical Effect for Screening Criteria	The Australian Drinking Water guideline for Bromate (0.02 mg/L health) may apply.
Ecological Toxicity[1,5,6]	
Aquatic Toxicity	Ecotoxicological data of bromate toxicity to aquatic biota are available for a range of aquatic organisms including freshwater algae, invertebrates, fish and estuarine and marine crustaceans and bivalves. Hutchinson et al. (1997) summarized the results from seven studies which indicate that the median effective(lethal) concentrations (E(L)C50 values) spanned over 4 orders of magnitude. The lowest reported values, ranging from 0.05 to -100 mg/L in a 48-hour EC50 embryo study on the oyster, (*Crassostrea gigas*) were markedly lower than E(L)C50 values obtained with the other species, which ranged from 31 to 100 mg/L bromate. In an attempt to reproduce the findings of the oyster study, Hutchinson et al. (1997) repeated twice the embryo development test using a similar protocol but was unable to reproduce the results and instead obtained a 24-hour EC50 of 170 mg/L as bromate for this endpoint. Given the lack of reproducibility of the test, the next most sensitive results were considered. The National Water Research Institute (NWRI) of Environment Canada in Burlington, Ontario, performed a suite of acute toxicity tests on metallic and non-metallic elements using *Hyalella azteca* (Environment Canada 2007). The objective of this experiment was to compare the relative toxicity of a number of inorganic ions in a reasonable worst-case situation using water chemistry representative of the diluted waters of the Canadian Shield (10% Lake Ontario water with low ionic strength and low dissolved organic carbon). Exposure lasted 7 days, and temperature was maintained between 24°C and 25ºC. A 7-day LC50 of 1.093 mg/L was estimated for bromate based on nominal concentrations. Because bromate is stable in water, nominal concentrations can be considered a good estimate of exposure concentration. This study was determined to have a high degree of reliability (see robust study summary in Appendix 2) and is therefore considered a key line of evidence. These toxicity data indicate that potassium bromate generally has only a low to moderate potential for toxicity to aquatic organisms; however, it may be highly hazardous to some sensitive organisms (e.g., *Hyalella azteca*).
Determination of PNEC aquatic	A predicted no-effect concentration (PNEC) was derived from the lowest acceptable toxicity value identified for a freshwater organism—an acute LC50 for *Hyalella azteca* of 1.093 mg/L. An assessment factor of 100 was applied to account for uncertainties associated with inter- and intra-species variability and extrapolation from a laboratory LC50 to a chronic no-effect value in the field. This calculation resulted in a PNEC of 0.011 mg/L.
Screening Criteria[7]	

Sodium bromate

Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	Australian Drinking Water guideline for Bromate is 0.02 mg/L (health)
Occupational Exposure Limits	No data found
PBT Assessment[1,5,6]	
P/vP Criteria fulfilled?	Not applicable. Sodium bromate is an inorganic substance which dissociates to bromate ion which is ubiquitous in environment
B/vB criteria fulfilled?	Sodium bromate is not bioaccumulative based on published information and experimental evidence for metabolic transformation.
T criteria fulfilled?	The acute aquatic toxicity of sodium bromate 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 Industrial Chemicals Notification and Assessment Scheme (NICNAS), 2015. Accelerated Assessment of Industrial Chemicals in Australia (IMAP), Human Health Tier II Assessment for Bromates CAS # 7758-01-2 and 7789-38-0
3. IARC (International Agency for Research on Cancer) (1986). IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: some naturally occurring and synthetic food components furocoumarins, ultraviolet radiation and potassium bromate. World Health Organization, IARC, 40.
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7. 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.

Polydimethylsiloxanes

Polydimthylsiloxanes	
Chemical and Physical Properties[1,2,3]	
CAS number	63148-62-9 - Dimethyl siloxanes and silicones 67762-90-7 - Siloxanes and silicones, dimethyl, reaction products with silica
Molecular formula	$(C_2H_6OSi)_n$
Molecular weight	Range from 1,125 to 74,000 g/mol
Solubility in water	<1ng/L
pH	No data available
Melting point	Decomposes at >250C
Boiling point	>65 deg C
Colour/Form	clear, colourless, odourless and viscous fluid
Vapour pressure	Not volatile
Henrys law constant	Not volatile
Explosive potential	Not applicable
Flammability potential	Not applicable
Overview	Linear polydimthylsilxoanes (PDMSs) are polymeric organosilicon substances commonly referred to as 'silicones'. At ambient temperature and pressure, PDMSs are clear, colourless, odourless and viscous fluids and have no detectable vapour pressure. They exhibit stability to chemical and oxidative degradation and radiation and are essentially insoluble in water. PDMSs strongly sorb to particulate matter in water and soil and are essentially immobile in the soil and sediment compartments of the environment. PDMSs have a wide range of industrial, consumer, food (as a antifoaming and anticaking agent) and medicinal or pharmaceutical applications. The Joint FAO/WHO expert committee on food additives (JECFA) established the ADI of 1.5 mg/kg bw for PDMS.
Environmental Fate[1,2]	
Soil/Water/Air	PDMSs are non volatile and are insoluble in water. Any PDMS released into the environment will strongly sorb to particulate matter in water and soil. PDMSs are immobile in soil and sediment, but will break down slowly to dimethylsilanediol, which is soluble in water and can biodegrade to carbon dioxide, water and inorganic silicate. Due to its molecular size, bioconcentration of PDMS is very unlikely.
Human Health Toxicity Summary[1,2,3]	
Chronic Repeated Dose Toxicity	The combined chronic/carcinongenicity feeding study was conducted with doses ranging from 1 to 1000 mg/kgbw/d. No test material pre-neoplastic or plastic changes were observed and the NOAEL for this study was >1,000 mg/kgbw/d.
Carcinogenicity	PDMS is not considered to be a carcinogen. The NOAEL from a carcinogenic oral study was >1,000 mg/kgbw/d.
Mutagenicity/Genotoxicity	PDMS was not considered to be mutagenic in a Bacterial Reverse Mutation Assay.
Reproductive/developmental Toxicity	No effects were seen in reproductive or developmental studies with PDMS. The NOAEL for these studies was >1,000 mg/kgbw/d.
Acute Toxicity	In acute oral studies the LD50 ranged from > 2 g/kgbw and > 5 g/kgbw. The acute dermal LD50 was >2 g/kgbw and in an acute aerosol inhalation study the LC50s ranged from >696 mg/m3 to >11,582 mg/m^3. No clinical signs of toxicity and no microscopic lesions were observed in subacute feeding studies. The NOAEL for the studies was >100,000 ppm.
Irritation	PDMS was shown to be essentially non-irritating in both dermal and eye irritation studies.
Sensitisation	PDMS is considered a non-sensitiser in a Guinea pig maximisation test.
Key Study/Critical Effect for Screening Criteria	PDMS has a very low order of acute, subacute, subchronic, reproductive/developmental, chronic/carcinogenicity and immunological toxicity. The NOAELs in these studies have been shown to be greater than the highest dose tested. Likewise, these materials are non-

Polydimethylsiloxanes

	irritating and are non-sensitisers. The Joint FAO/WHO expert committee on food additives (JECFA) established the ADI of 1.5 mg/kg bw for PDMS.
Ecological Toxicity [1,2]	
Aquatic Toxicity	PDMS has no effects when tested on aquatic organisms (fish, daphnia, algae). The median lethal concentration (LC50) values determined from acute fish toxicty testing were generally >1,000 mg PDMS/L. Adverse effects were not observed at concentrations up to and exceeding the aqueous solubility of PDMS. Therefore no environmental realistic NOEC can be derived for free swimming organisms. Most PDMS that enters the surface water is sorbed into particulate matter. As a result aquatic organisms have the highest potential for exposure to PDMS through sediment. Acute studies on PDMS in sediment swelling marine and estuarine organisms result in LC50s ranging from >1020 mg/L (mussel) to >10,000 mg/kg (polychaete worm). Sub chronic and chronic studies on PDMS in sediment dwelling organims result in NOECs ranging from >560 to >2590 mg/kg. The available data support the conclusions that PDMS has no effect on sediment living organisms.
Determination of PNECaquatic	Since no NOEC could be derived for aquatic organisms, no PNEC can be determined.
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	No data found
PBT Assessment[1,2]	
P/vP Criteria fulfilled?	PDMS is not readily biodegradable and as such is persistent in the environment.
B/vB criteria fulfilled?	Due to PDMS molecular size, PDMS is not bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of PDMS 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. European Centre for Ecotoxicology and Toxicology of Chemicals (1994) Linear polydimethylsiloxanes (CAS No. 63148-62-9). Brussels, Belgium, European Centre for Ecotoxicology and Toxicology of Chemicals, Joint Assessment of Commodity Chemicals, September (JACC Report No. 26).
3. World Health Organisation, 2011, Seventy Fourth report of the Joint FAO/WHO Expert Committee on Food Additives, Evaluation of Certain Food Additives and Contaminants, WHO Technical Report Series 966

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]	
CAS number	38193-60-1 and 136793-29-8
Molecular formula	38193-60-1: (C7H13NO4S.C3H5NO.Na)x 136793-29-8: C11H18NNaO6S
Molecular weight	Likely >1000 MW
Solubility in water	No information available.
pH	No information available.
Melting point	No information available.
Boiling point	No information available.
Colour/Form	No information available.
Vapour pressure	No information available.
Henrys law constant	No information available.
Explosive potential	No information available.
Flammability potential	No information 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.
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/developmental Toxicity	No information 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.
Key Study/Critical Effect for Screening Criteria	No information 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.

Acrylamide Polymers

Determination of PNEC aquatic	No PNEC values were calculated.
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	No data found
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

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

2-Propenamid (Acrylamide)

2-Propenamid (Acrylamide)	
Chemical and Physical Properties[2,4]	
CAS number	79-06-1
Molecular formula	$CH_2 = CH - CONH_2$
Molecular weight	71.09 g/mol
Solubility in water	2.16 g/mL at 30 C
pH	5.0-6.5 /50% aqueous solution
Melting point	84.5 C
Boiling point	192.6 deg C
Colour/Form	White crystalline solid, acrylamide sublimes slowly at room temperature
Vapour pressure	0.9 Pa at 25 deg C
Henrys law constant	1.7X10-9 atm-cu m/mol at 25 deg C
Explosive potential	Finely dispersed particles form explosive mixtures in air
Flammability potential	Combustible. Gives off irritating or toxic fumes/gases in a fire.
Overview	Acrylamide is highly water soluble and rapidly polymerises in an exothermic reaction to form water soluble polymers. These high molecular weight polymers are produced to give non-ionic, cationic and anionic properties for specific uses. The largest uses of acrylamide are in wastewater treatment, paper and pulp processing and mineral processing. Liquid or powder partially-hydrolysed polyacrylamides are used as additives to water- based drilling muds to provide a lubricating film and reduce friction at the drill bit, impart stability to shales and clays and increase viscosity. Limited information on toxicokinetics of acrylamide in humans is available. Animal studies indicate that acrylamide is readily absorbed by oral, dermal and inhalation routes. Absorbed acrylamide is distributed rapidly and throughout the body. Acrylamide is metabolised via conjugation with glutathione and via oxidative metabolism to the epoxide metabolite, glycidamide. Glutathione conjugation of glycidamide also occurs. Between 50 and 70% of administered dose is eliminated in 24hours, with the majority via urinary route and a small amount in faeces. Studies show that acrylamide and glycidamide bind to DNA, RNA and a wide range of proteins. In addition, both acrylamide and glycidamide form adducts with blood haemoglobin. The critical adverse effect in humans and in animals from acute and repeated exposure is neurototoxicity, with effects being seen in CNS and PNS. Chronic oral studies in rats indicate that acrylamide is clearly carcinogenic, although no firm conclusions regarding carcinogenicity in humans could be drawn from available epidemiological studies. Similarly, no conclusions could be drawn regarding the mode of action for tumourigenicity in animals and relevance to humans, as evidence indicates both genotoxic and epigenetic effects. Acrylamide has also been shown to have effects on fertility in rats and mice and to be a skin irritant and sensitiser.
Environmental Fate[1,2,4,5]	
Soil/Water/Air	Acrylamide is a powder (normally supplied as a 30-60% w/w aqueous solution) with a vapour pressure of 0.9 Pa at 25 °C, a water solubility of 2,155 g/l at 30 °C and a log octanol-water partition coefficient (log KOW) of ~ -1.0. It does not polymerise significantly at temperatures up to its melting point (~84°C) in the absence of light. However, above its melting point it can polymerise rapidly and exothermically. Hydrolysis and photolysis in water are environmentally insignificant but it is considered readily biodegradable meeting the 10-day test window. The log KOW value implies a low bioaccumulation potential in aquatic species (confirmed by fish bioconcentration factors (BCFs) of 0.26 - 2.53) and low adsorption to soils and sediments, thus will be relatively mobile in most soil types. The substance chiefly partitions to water.
Human Health Toxicity Summary[1,2,3,4,5]	
Chronic Repeated Dose Toxicity	The key toxicological endpoints for repeated exposure to acrylamide are neurotoxicity, genotoxicity, carcinogenicity and reproductive effects, although only neurotoxicity has been reported/observed in humans. No firm conclusions could be drawn from available human cohort mortality studies on potential carcinogenicity of acrylamide to humans. From the available data, there are no clear species differences in sensitivity to neurotoxic effects of acrylamide, however lower (critical) NOAELs were determined for rats. A critical

2-Propenamid (Acrylamide)

	NOAEL of 0.2 mg/kg/d was determined for F-344 rats in a 90-day drinking water study, with a LOAEL of 1 mg/kg/d, where slight changes in nerve tissue were seen by electron microscopy (Burek et al 1980). A chronic study NOAEL of 0.5 mg/kg/d was determined in the same species/strain, based on lack of histopathological effects in tibial nerve (Johnson et al 1986). It was reported that neurological investigation in this study was not as extensive as that carried out in the Burek et al study. For reproductive effects, seen in both rats and mice, a critical NOAEL of 2 mg/kg/d was determined in F-344 rats in two-generation reproduction (drinking water studies), with a LOAEL of 5 mg/kg/d (Tyl 1987, Tyl 2000). Acrylamide produced an increase in incidence of a number of benign and malignant tumours, in a number of organs in F-344 rats (Johnson et al 1986; Friedman et al 1995) and was genotoxic in a number of in vivo studies. Despite the fact that statistical significance was only apparent at high doses (lowest LOAEL = 0.5 mg/kg/d for scrotal mesothelioma), it was not possible to identify a clear NOAEL from the available carcinogenicity data.
Carcinogenicity	Acrylamide is carcinogenic in animals producing increased incidences in a number of benign and malignant tumours identified in a variety of organs (eg. thyroid, adrenals, testicular mesothelioma). In rats administered acrylamide in drinking water, there was clear evidence of an increase in tumour incidence in several organs in both sexes, although bioassays in mice indicate that acrylamide may not act as a complete carcinogen (NICNAS). Acrylamide meets the National Occupational Health and Safety Commission (NOHSC) Approved Criteria (NOHSC 1999a) for classification as a Category 2 carcinogen (Risk Phrase R45 – May cause cancer). The International Agency for Research on Cancer (IARC) has classified the chemical as category Group 2A (probably carcinogenic to humans), as there is sufficient evidence in experimental animals for the carcinogenicity of acrylamide.
Mutagenicity/Genotoxicity	Acrylamide meets the NOHSC Approved Criteria for classification as a Category 2 mutagen - May cause heritable genetic damage (R46). Overall, the weight of evidence from in vitro and in vivo studies indicates that acrylamide is genotoxic in both somatic and germ cells.
Reproductive/developmental Toxicity	Acrylamide meets the NOHSC Approved Criteria for classification as a Category 3 reprotoxicant – May impair fertility (R60). In summary, there is sufficient evidence to indicate that fertility effects seen in either male or female animals are not secondary to either generalised or specific toxicity, such as neurotoxicity or hormonal effects. In addition, many of these fertility effects have been demonstrated at relatively low exposure levels. Evidence indicates that such effects may be primarily seen in males. Although the mode of action is not known, acrylamide has been shown to reduce sperm counts in some studies (NICNAS).
Acute Toxicity	Acrylamide has been shown to cause skin and eye irritation in both humans and animals. Animal evidence also indicates a skin sensitisation potential. In rats, acrylamide is toxic and harmful by oral and dermal routes of administration respectively, and neurotoxic effects were reported in one human case report of accidental ingestion. Doses of acrylamide causing acute systemic effects, including neuropathy, in animal studies were around two orders of magnitude greater than those eliciting effects in repeated dose studies (NICNAS).
Irritation	Evidence from human exposures indicate that acrylamide meets the NOHSC Approved Criteria for skin irritant effects and is classified as irritating to skin (R38). Acrylamide meets the NOHSC Approved Criteria for eye irritant effects and is classified as Irritating to eyes (R36). Dermatological effects have been reported in humans exposed to acrylamide and acrylamide solutions. Whether effects, particularly skin peeling, were due to primary irritant effects is unclear. However, other effects such as rash and ulcerations on hands and feet have been reported in workers using acrylamide. Eye irritation is apparently the most frequently reported adverse effect in workers using acrylamide grouts. Tests in rabbits on acrylamide applied dermally under occlusive or semi-occlusive conditions indicate a lack of skin irritation potential. Tests in rabbits found acrylamide to be an eye irritant when applied in powdered form or in aqueous solutions (>40% acrylamide). All signs of irritation were normal seven days post-application (NICNAS).

2-Propenamid (Acrylamide)

Sensitisation	Acrylamide meets the NOHSC Approved Criteria for skin sensitisation and is classified as May cause skin sensitisation by skin contact (R43).
Key Study/Critical Effect for Screening Criteria	The Australian Drinking Water Guideline for Acrylamide is 0.0002 mg/L.
Ecological Toxicity [4]	
Aquatic Toxicity	Acrylamide is slightly toxic to aquatic plants (lowest EC50=33.8 mg/L) and organisms (lowest Daphnia EC50=98 mg/L and lowest fish EC50=85 mg/L) (NICNAS).
Determination of PNEC aquatic	Valid acute LC50 values are reported for fish (lowest 96-hour LC50=100 mg/L, Lepomis macrochirus), aquatic invertebrates (the lowest 48-hour EC50=98 mg/L, Daphnia magna), and freshwater algae (the lowest 72-hour EC50=33.85 mg/L, Selenastrum capricornutum). Since there is valid acute toxicity data for three trophic levels and only a valid long-term (72-hour) NOEC for freshwater algae, an assessment factor of 1000 is used (in accordance with EU guidance). Based on the72-h EC50 for freshwater algae (the most sensitive species in short term tests), the aquatic PNEC is 33.85 ug/L (NICNAS).
Screening Criteria [4,6,7]	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	The Australian Drinking Water Guideline for Acrylamide is 0.0002 mg/L.
Occupational Exposure Limits	Acrylamide has an exposure standard of 0.03 mg/m3 time weighted average (TWA)
PBT Assessment [1,2,4,5]	
P/vP Criteria fulfilled?	Acrylamide is readily biodegradable and as such not persistent in the environment.
B/vB criteria fulfilled?	Based on the measured log Kow of -1.0 and an estimated BCFs of 0.26 - 2.53, Acrylamide is not bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of Acrylamide 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. European Commission Joint Research Centre 2002, European Union Risk Assessment Report, Acrylamide, CAS no. 79-06-1.
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3. International Agency for Research on Cancer (IARC) Monographs Volume 60, Acrylamide
4. National Industrial Chemicals Notification and Assessment Scheme (NICNAS), 2002. Acrylamide, Priority Existing Chemical Assessment Report No. 23
5. OECD (2001) SIDS Initial Assessment Profile for Acrylamide, CAS Number 76-06-1
6. Safe Work Australia 2011. Workplace Exposure Standards for Airborne Contaminants.
7. 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.

Boron Compounds: Boric Acid/Sodium Tetraborate/ Boronatrocalcite

Boron Compounds: Boric Acid/Sodium Tetraborate/ Boronatrocalcite	
Chemical and Physical Properties[1,3,5,8]	
CAS number	Boric Acid: 10043-35-3 Sodium Tetraborate: 1330-43-4 Boronatrocalcite: 1319-33-1
Molecular formula	Boric acid: H_3Bo_3 Sodium Tetraborate: $Na_2B_4O_7$ Boronatrocalcite: $CaNaH_{12}(BO_3)5.2H_2O$
Molecular weight	Boric acid: 61.833 g/mol Sodium Tetraborate: 201.220 g/mol Boronatrocalcite: 405.23 g/mol
Solubility in water	Boric acid: 49.20 g/l @ 20± 0.5 C Sodium Tetraborate: 3.1% at 25 C Boronatrocalcite: no data found
pH	Boric acid: 6.1 in a 0.1% (wt) solution Sodium Tetraborate: 9.3 at 20 deg C (3% solution) Boronatrocalcite: no data found
Melting point	Boric Acid: 170.9 C Sodium Tetraborate: 743 C Boronatrocalcite: no data found
Boiling point	Boric Acid: 300 C Sodium Tetraborate: 1,575 C (decomposes) Boronatrocalcite: no data found
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.
Vapour pressure	Boric acid: 9.9×10^{-6} Pa @ 25 C Sodium Tetraborate: Negligible at 20 C Boronatrocalcite: no data found
Henrys law constant	No data found
Explosive potential	Not explosive
Flammability potential	Not flammable
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

Boron Compounds: Boric Acid/Sodium Tetraborate/ Boronatrocalcite

	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	These forms of boron are highly soluble and not easily removed from solution by natural mechanisms. Borate and boric acid are in equilibrium depending on the pH of the water. At an acidic pH, boron exists in solution mainly as undissociated boric acid, whereas at alkaline pH it is present as borate ions. Boric acid is a persistent molecule, mobile in soil and sediment, not subject to hydrolysis, photodegradation or biodegradation. Other borates yield boric acid upon dissolution in water (or borate anion in higher pH conditions).
Human Health Toxicity Summary [2,3,4,8]	
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/developmental Toxicity	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. 17.5 mg boron /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 B/kg (54 mg boric acid/kg/day; 85 mg disodium tetraborate decahydrate).
Acute Toxicity	Boric acid is of low acute toxicity. LD50 oral rat > 2000 mg/kg; LD50 dermal rat > 2000 mg/kg; LC50 inhalation rat > 2 mg/l.
Irritation	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.
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 B/kg/day (54 mg boric acid/kg/day; 85 mg disodium tetraborate decahydrate), 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

Boron Compounds: Boric Acid/Sodium Tetraborate/ Boronatrocalcite

Ecological Toxicity [3]	
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).
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).
Screening Criteria	
Aquatic Toxicity Guidelines	For boron: 90 ug/L (ANZECC 2000 99% Freshwater)
Water Quality Guidelines	For boron: 4 mg/L (ADWG 2011 drinking water)
Occupational Exposure Limits	No data found.
PBT Assessment	
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?	For the purposes of this PBT assessment, the toxicity criteria is not considered applicable to this inorganic substance.
Overall conclusion	Not PBT

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 European Chemicals Agency, Draft Support Document for the Identification of Boric Acid, 2010.
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

CALCIUM CHLORIDE

CALCIUM CHLORIDE	
Chemical and Physical Properties[1]	
CAS number	10043-52-4
Molecular formula	$CaCl_2$
Product name	--
Molecular weight	110.99 g/mol
Solubility in water	745 g/L at 20^{o}C
pH	Salt
Melting point	772^{o}C
Boiling point	1670 ^{o}C
Colour/Form	White cubic crystals or powder
Vapour pressure	NA
Henrys law constant	NA
Explosive potential	NA
Flammability potential	Forms flammable gases and evolves hydrogen when reacted with zinc.
Overview	Calcium chloride is easily dissociated into calcium and chloride ions in water. Both ions are essential elements in animals and humans. Calcium is essential for the formation of skeletal structure, neural transmission, muscle contraction, coagulation of the blood, and a range of other physiological functions. Chloride is required for regulating intracellular osmotic pressure and buffering.
Environmental Fate[2,3]	
Soil/Water/Air	Calcium chloride is soluble in water and its vapour pressure is negligible. When released into the environment calcium chloride is distributed into the water in the form of calcium and chloride ions. Calcium chloride is not expected to be absorbed in soil due to its dissociation properties and high water solubility. The chloride ion is mobile in soil and eventually drains into surface water because it is readily dissolved in water. Calcium chloride is not expected to undergo photolysis or biodegradation. Considering its dissociation properties, calcium chloride is not expected to accumulate in living organisms
Human Health Toxicity Summary [2,3]	
Chronic Repeated Dose Toxicity	Rats (40-day old) were fed 20 mg/g calcium chloride in the diet for 12 months. There were no treatment-related effects on mortality, weight gain, or daily food consumption. In addition, no neoplastic lesions were observed in gastrointestinal tract, urinary tract, liver, heart, brain or spleen of the animals. Calcium and chloride are both essential nutrients for humans as well as other animal species. As for healthy humans, the tolerable upper intake level for calcium is set at 2,500 mg per day (equivalent to 6.9 g CaCl2 per day) The establishment of the ADI for calcium chloride has not been deemed necessary by JECFA. The substance has also been considered a GRAS substance by the U.S. FDA. It is thus very unlikely that calcium chloride taken orally as food additives adversely affects human health
Carcinogenicity	No data available.
Mutagenicity/Genotoxicity	Genetic toxicity of calcium chloride was reported as negative in bacterial mutation tests and the mammalian chromosome aberration test.
Reproductive Toxicity	No data available.
Developmental Toxicity/Teratogenicity	A developmental toxicity study showed no toxic effects on dams or fetuses at doses up to 189 mg/kg/day (mice), 176 mg/kg/day (rats) and 169 mg/kg/day (rabbits).
Acute Toxicity	The oral LD50 values were 2,045 (male) and 1,940 mg/kg (female) in mice, 3,798 (male) and 4,179 mg/kg (female) in rats, and 500-1,000 mg/kg in male rabbits. Acute oral toxicity of the substance is attributed to the severe irritating property of the original compound or its high-concentration solutions to the gastrointestinal tract, causing perforation and ulceration of the contact area of the tract. The dermal LD50 value in male/female rabbits was >5,000 mg/kg.
Irritation	Calcium chloride is non to slightly irritating to skin of rabbits; it is, however, severely irritating to eyes of rabbits. Irritating effect of calcium chloride has been observed in human skin

CALCIUM CHLORIDE

	injuries caused by the incidental contact with the substance or its high-concentration solutions.
Sensitisation	No data available.
Key Study/Critical Effect for Screening Criteria	The drinking water guidelines for chloride and hardness (as calcium carbonate) may apply to calcium chloride.
Ecological Toxicity [2,3]	
Aquatic Toxicity	Several studies on acute toxicity to fish have been reported. The lowest 96-hr LC50 value was 4,630 mg/L in fathead minnow (Pimephales promelas). No chronic toxicity studies on fish conducted under standard guidelines have been reported. There are seven acute toxicity data available for Daphnia. Two of these studies were conducted according to international or national guidelines, giving the 48-hr EC50 of 2,400 mg/L for Daphnia magna and the 48-hr LC50 of 1,830 mg/L for Ceriodaphnia sp. The lowest 48-hr EC50 was 1,062 mg/L for Daphnia magna. The chronic effect of 21-day exposure on reproduction of Daphnia magna has been investigated as a long-term study. The concentration required for 16% and 50% inhibition of reproduction (EC16 and EC50) were 320 and 610 mg/L, respectively. The NOEC = EC16/2 = 320/2 = 160 mg/L. There is one study with fresh water algae, Selenastrum capricornutum, which was conducted according to OECD TG 201. The 72-hr EC50 and EC20 obtained on the basis of growth rate from the study were >4,000 and 2,700 mg/L, respectively. The 72-hr EC50 and EC20 obtained on the basis of biomass from the study were 2,900 and 1,000 mg/L, respectively. The NOECs are calculated as EC20/2, which corresponds to 1,350 and 500 mg/L for growth rate and biomass, respectively.
Determination of PNEC aquatic	Experimental results are available for three trophic levels. Acute E(L)C50 values are available for fish (4,630 mg/L), Daphnia (1,062 mg/L), and algae (2,900 mg/L). Results from a chronic Daphnia study (NOEC = 160 mg/L) and algae study (NOECs = 1,350 and 500 mg/L for growth rate and biomass, respectively) are also available. On the basis that the data consists of short-term results from three trophic levels and chronic studies on Daphnia and algae, an assessment factor of 50 has been applied to the lowest reported NOEC of 160 mg/L for Daphnia.
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	No data found
PBT Assessment	
P/vP Criteria fulfilled?	No. Not applicable, inorganic salt, ionic species ubiquitous in environment.
B/vB criteria fulfilled?	No. Not applicable, inorganic salt, ionic species ubiquitous in environment.
T criteria fulfilled?	No chronic toxicity data exist on calcium chloride; however, the acute EC(L)50s are >0.1 mg/L in fish, invertebrates and algae. Thus, calcium chloride does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT

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. IUCLID (2002) IUCLID Data Set for Calcium chloride (CASRN 10043-52-4), UNEP Publications.
3. OECD-SIDS (2002) Screening Information Dataset (SIDS) Initial Assessment Report for Calcium chloride (CASRN 10043-52-4), UNEP Publications.

Crystalline Silica-Cristobite, Crystalline Silica-Quartz, and Diatomaceous Earth

CRISTOBITE, SILICA, DIATOMACEOUS EARTH	
Chemical and Physical Properties[1]	
CAS number	Crystalline Silica (Cristobite) : 14464-46-1 Crystalline Silica (Quartz): 14808-60-7 Diatomacous Earth: 91053-39-3
Molecular formula	Crystalline Silica (Cristobite): SiO_2 Crystalline Silica (Quartz): SiO_2 Diatomacous Earth: SiO_2
Product name	
Molecular weight	60.09 g/mol
Solubility in water	-
pH	-
Melting point	1710^{o}C
Boiling point	2230 ^{o}C
Colour/Form	Transparent crystals
Vapour pressure	NA
Henrys law constant	NA
Explosive potential	Not explosive
Flammability potential	Not flammable
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]	
Carcinogenicity	The USEPA has classified Crystalline Silica as a suspected human carcinogen A2, as there is sufficient evidence in humans for the carcinogenicity of inhaled crystalline silica in the form of quartz from occupational sources.
Systemic toxicity/organ effects	Respirable quartz dust particles can be inhaled and deposited in the lungs. There are many epidemiological cohort studies of workers exposed to respirable quartz dust. Silicosis, lung cancer and pulmonary tuberculosis are associated with occupational exposure to quartz dust.
Mutagenicity/Genotoxicity	No data available.
Reproductive Toxicity	No data available.
Developmental Toxicity/Teratogenicity	No data available.
Acute Toxicity	No data available.
Irritation	No data available.
Sensitisation	No data available.
Key Study/Critical Effect for Screening Criteria	Not applicable.

Ecological Toxicity [1,2]	
Aquatic Toxicity	No data available.
Determination of PNEC aquatic	Not applicable.
Screening Criteria	
Aquatic Toxicity Guidelines	TBD
Water Quality Guidelines	TBD
Occupational Exposure Limits	TWA – 0.1 mg/m^3
PBT Assessment	
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	Not PBT

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Cyclic dimethyl polysiloxane compounds

cyclic dimethyl polysiloxane compounds	
Chemical and Physical Properties[1,2,3,4,5,6,7]	
CAS number	540-97-6 – Dodecamethylcyclohexasiloxane (known as D6) 541-02-6 - Decamethylcyclopentasiloxane (known as D5) 556-67-2 – Octamethylcyclotetrasiloxane (known as D4)
Molecular formula	D6 - C12H36O6Si6 D4 - C8H24O4Si4 D5 - C10H30O5Si5
Molecular weight	D5 - 370.78 g/mol D4 - 296.62 g/mol D6- 444.93 g/mol
Solubility in water	D4- 0.056 mg/L at 23 °C D5- 0.017 mg/L at 23 °C D6 - 0.00513 mg/L at 23 °C
pH	No data found
Melting point	D6 -3 C
Boiling point	D6 - 245 ºC (at 1013 hPa)
Colour/Form	Clear siloxane fluids
Vapour pressure	D4 - 132 Pa at 25 °C D5 - 33.2 Pa at 25 °C D6 - 4.6 Pa at 25 ºC
Henrys law constant	D4- 1.21 × 106 Pa.m^3/ mol D5 - 3.34 × 106 Pa.m^3/ mol D6 - 2.25 x 10^6 Pa-m^3/mol at 23.7 deg C
Explosive potential	No data found
Flammability potential	Flammable
Overview	Chemicals Dodecamethylcyclohexasiloxane (known as D6), Decamethylcyclopentasiloxane (known as D5) and Octamethylcyclotetrasiloxane (known as D4) are components of cyclomethicone (cyclosiloxanes, dimethyl; CAS No. 69430-24-6), which is a mixture of cyclic dimethyl polysiloxane compounds consisting of 3–7 [–Si(CH3)2O–]X base units. Cyclomethicone is widely used in cosmetics. The major use of these substances is as a feedstock for the production of silicone polymers. These silicone fluids, elastomers, gels and resins in turn have a very wide range of uses, including as rubber; elastomers for coatings and sealants; antifoams; flow and/or gloss improvers in alkyd paints and varnishes; softening, waterproofing and wetting agents in textile manufacturing; components of polishes and other surface treatment formulations; lubricants, greases, anti-adhesion coatings and mould release agents; paper coatings; hydraulic, dielectric and heat transfer fluids; and consumer products such as personal, household, and automotive care products (Ashford, 1994; Chandra, 1997).
Environmental Fate[1,2,4,5]	
Soil/Water/Air	D4 and D5 are poorly soluble in water, volatile and adsorb strongly to organic matter, sediment and soil. They are not rapidly degraded, and have long environmental half-lives in sediment in particular. Wastewater treatment plants are usually efficient at removing them from waste water (especially if there is an aeration step, when a significant portion of the removal will be to air). Once in the atmosphere, they can be transported long distances, and have been detected in remote regions. However, they are expected to remain in the atmosphere until degraded, and their potential for deposition to surface water and land is generally very low. Due to its low water solubility, volatility and partitioning properties, D6 released into air or soil is expected to remain in that compartment, while D6 released into water is expected to partition primarily to the sediment. Results of multimedia modelling demonstrate that the fate, distribution and persistence of D6 are strongly influenced by the source of entry into the environment. For example, if D6 is emitted exclusively to air, the predicted distribution

Cyclic dimethyl polysiloxane compounds

	is 93.2% in air, 0.0% in water, 6.7% in soil, and 0.1% in sediment. Because of its low water solubility, volatility, and partitioning properties D6 released to air or soil is expected to remain in the compartment of emission where degradation is expected to occur. If released to water D6 is expected to partition to the sediment compartment where degradation is expected to occur. D6 is not readily biodegradable. Degradation of D6 has been demonstrated in dry soils (with half-lives ranging from hours to several months depending on soil type). D6 will ultimately degrade to inorganic silicate (sand), water, and carbon dioxide. Slow degradation is expected and volatilization is the more likely removal mechanism (as has been demonstrated for D4 and D5) Bioaccumulation studies with freshwater fish (*Pimephales promelas)* and the aquatic invertebrate, *Daphnia magna,* resulted in BCF values of 1160 and 2400 L/kg, respectively. D6 is not readily biodegradable and has the potential to bioaccumulate. D4 and D5- Both chemicals meet the European Chemicals Agency (ECHA) Annex XIII criteria for being persistent, bioaccumulative, and toxic (PBT) and very persistent very bioaccumulative (vPvB).
Human Health Toxicity Summary[1,2,3,4,5,6,7]	
Chronic Repeated Dose Toxicity	<u>Oral</u> D6 - In a combined repeated dose and reproductive/developmental toxicity study, conducted in accordance with OECD TG 422 (REACH), the chemical was administered to Crl:CD rats (n = 10/sex/dose) at oral doses of 0, 100, 330 or 1000 mg/kg bw/day, for 28 days. There were no effects observed on mortality, body weight and weight gain, food consumption, clinical chemistry and gross pathology. Reported effects included: relative organ weight increases in the liver and kidneys of both sexes and in the adrenals of females at all doses and a dose-related increase in the liver weights of females. However, these increases were reported to be within the historical control values. In males, prothrombin time (measuring the time blood takes to clot) was prolonged at the two highest doses, suggesting impaired liver function. There was an increase in periportal lipidosis (accumulation of lipids due to abnormal lipid metabolism) in the liver of females at all doses. A lowest observed adverse effect level (LOAEL) of 100 mg/kg bw/day was determined based on the increased liver weight and periportal lipidosis (OECD, 2009; REACH). In another oral gavage repeated-dose toxicity study, rats were exposed to 1500 mg/kg bw/day D6 in distilled water for 28 days. The study revealed no treatment-related effects in either sex. Based on no effects observed, the NOAEL in this study was considered to be 1500 mg/kg bw/day. D5- In a repeated dose toxicity study (OECD TG 408) in Wistar rats (n = 10/sex/dose), animals were administered the chemical by oral gavage at 0, 100, 330 or 1000 mg/kg bw/day for 13 weeks. Liver weights were significantly increased by 42–62 % in females exposed at □100 mg/kg bw/day compared with controls. This was not considered to be an adverse health effect, as there were no histopathological changes associated with the increased liver weights (Government of Canada, 2008; EA, 2009; SCCS, 2010; Dekant & Klaunig, 2016; REACH). D4- In two separate 14-day studies, the chemical (purity >98 %) in 0.5 % (w/v) methylcellulose vehicle was administered by gavage daily for two weeks to SD rats (8/sex/dose) at doses of 0, 25, 100, 400, or 1600 mg/kg bw/day or female NZW rabbits (n=6) at doses of 0, 500, or 1000 mg/kg bw/day. No overt signs of toxicity were observed in either species. Treatment-related effects in rats include decreased bodyweight at 1600 mg/kg bw/day (sex not specified) and increased liver weights in both sexes at 400 and 1600 mg/kg bw/day (SCCP, 2005; SCCS, 2010). Morphometric and electron microscopic examination of the liver showed that the increased liver weights were due to hepatocellular hyperplasia (Johnson et al., 2011). All treated rabbits exhibited significant decreases in food consumption and bodyweight. Changes in the spleen and thymus were also observed in the rabbits but were reportedly not dose-dependent (SCCP, 2005; SCCS, 2010; Johnson et al., 2011). <u>Dermal</u>

	D5- In a 28-day repeated dose dermal toxicity study (similar to OECD TG 410), SD rats (n = 10/sex/dose) were exposed (occlusively) to the chemical on shaved skin at 0, 200, 800 or 1600 mg/kg bw/day for six hours per day. Urinalysis confirmed that the chemical was absorbed and metabolised; no statistically or biologically significant local or systemic toxicity effects were reported (Government of Canada, 2008; EA, 2009; SCCS, 2010; Johnson et al., 2011; REACH). D4- In a study conducted similarly to OECD TG 410, the neat chemical (as Baysilone; 99.8 % purity) was applied to the shaved intact flank skin of NZW rabbits (5/sex/dose) at doses of 0, 0.1, 0.3, or 1.0 mL/kg bw/day (approximately equivalent to 0, 96, 190, or 960 mg/kg bw/day) five days a week for three weeks. The open application of the chemical was followed by a two-week recovery period. No treatment-related effects on clinical signs, survival, bodyweight gain, food consumption, haematology, clinical chemistry, urinalysis, and histopathology were observed (SCCP, 2005; SCCS, 2010; REACH). D6- No studies found <u>Inhalation</u> D6- In a subchronic toxicity study conducted in accordance with OECD TG 413 (REACH), groups of Sprague Dawley (SD) rats (n = 10/sex/dose) were exposed (whole body) to vapours of the chemical at concentrations of 0, 1, 10 or 30 ppm (equivalent to 0, 18.2, 182 and 546 mg/m3, respectively), six hours per day, seven days per week, for 13 weeks. Additional groups of rats were treated similarly and given another 28 days without treatment before sacrifice. There were no effects observed on mortality, body weight and weight gain, food consumption, clinical chemistry, organ weights, haematology or gross pathology. Microscopic findings were observed at 10 and 30 ppm in nasal tissues, liver and lung. At the mid and high doses, males and females exhibited increased incidence and severity of subacute inflammation and hyperplasia of nasal tissues, along with increased mucous cell hyperplasia Slight increased incidence of periportal vacuolation in the liver was observed in the high-dose female group. Minimally increased incidence of alveolar macrophages in the lung was seen in the mid- and high-dose female groups. While the effects in the liver and the lung resolved within 28 days postexposure, effects persisted in the nasal tissues. Based on the local effects seen in nasal tissues, a NOAEC of 1 ppm (18.2 mg/m^3) was determined (REACH). D5- In a combined chronic toxicity/carcinogenicity study (according to EPA OPPTS 870.4300), F344 rats were exposed (whole body) to the chemical vapour at: 0, 10, 40 or 160 ppm (0, 150, 600 and 2420 mg/m3) for six hours per day, five days per week for 26 weeks (n = 6/sex/dose); 52 weeks (n = 10/sex/dose); 52 weeks followed by a 52 week recovery period (n = 20/sex/dose); or 106 weeks (n = 60/sex/dose) The no observed adverse effect concentration (NOAEC) was ≥160 ppm. Mortality in the recovery group (rats exposed for 52 weeks, followed by a 52 week recovery period) ranged from 15–75 % for males and 5–30 % for females. There was a significant positive trend in mortality for males with increasing exposure concentration. Body weight was similar in all groups, irrespective of the treatment regimen. There was an increased incidence of hyaline inclusions in the nasal olfactory epithelium of rats exposed to the highest concentration in all treatment regimens; females were more affected than males. The hyaline inclusions were not associated with overt inflammatory responses or tissue damage. Red blood cell count was reduced in males exposed at ≥40 ppm for 26 weeks, but changes were deemed minimal and there was no evidence of increased haematopoiesis. Liver weights were significantly increased in females exposed to the chemical for 26 or 52 weeks, but not in a concentration- or time dependent manner. The liver weight changes were associated with altered serum metabolic parameters including decreased urea concentration, increased cholesterol, increased triglyceride, and increased gamma glutamyl transferase following 52 weeks' exposure (US EPA, 2005; Government of Canada, 2008; EA, 2009; SCCS, 2010; Dekant & Klaunig, 2016; Jean et al., 2016; REACH). D4- In a study conducted in accordance with OECD TG 412, F344 rats (10/sex/dose) were exposed to the chemical (purity >95 %) by nose-only inhalation for 6 hours/day, 5 days/week for four weeks. The actual concentrations used were 0, 2.78, 5.13, 8.62, and 14.21 mg/L (days 1-5) and 13.25 mg/L (days 6-29). The chemical was in vapour phase at

Cyclic dimethyl polysiloxane compounds

	the three lower doses and 20 % liquid aerosol at the highest two doses since its saturated vapour concentration was determined to be 130 mg/L. Dose-dependent clinical signs, which include hunched posture, stiff or abnormal gait, head tilt, and ruffled fur, were observed at ≥ 5.13 mg/L. Treatment-related increase in absolute and relative liver weights in all treated animals, and decrease in absolute and relative thymus weights (males at 14.21 mg/L dose group; females at 13.25 and 14.21 mg/L dose groups) were observed. Histopathological changes observed in all exposed groups include minimal to slight lung alveolar inflammation, ultrastructural changes in hepatocytes, vacuolation of the zona fasciculata of the adrenal cortex, and decreased relative mitochondria volume. Increased vaginal mucification and decrease in the mean corpora lutea score were seen in all exposed females. The no observed adverse effect concentration (NOAEC) established in this study was 2.78 mg/L (SCCP, 2005; SCCS, 2010; Johnson et al., 2011; REACH).
Carcinogenicity	Based on the available information, the chemicals are not considered to be carcinogenic.
Mutagenicity/Genotoxicity	Based on the available data, the chemicals are not considered to be genotoxic.
Reproductive/developmental Toxicity	D4 - is classified as hazardous—toxic to reproduction, category 3—with the risk phrase 'Possible risk of impaired fertility' (Xn; R62) in the HSIS (Safe Work Australia). The available data support this. The chemical does not show specific developmental toxicity, with developmental effects observed secondary to maternal toxicity. Several reproductive toxicity studies are available which include one-generation, dose range-finding, and "phased-female" inhalation studies (wholebody and nose-only) in F344 and SD rats at concentrations up to 898 ppm. The extensive evaluation of these studies by the European Commission SCCS (previously the Scientific Committee on Consumer Products (SCCP)) (SCCP, 2005; SCCS, 2010), the Government of Canada (Government of Canada, 2008), and the CIR Expert Panel (Johnson et al., 2011) indicated that the chemical is toxic to reproduction. The findings from these studies showed that the chemical does not affect male rat fertility. The NOAEC for female fertility effects was established at 300 ppm based on the following observations on female rat fertility parameters consistently reported in these evaluations: reduced numbers of ovulated eggs from "phased-female" studies; decreased corpora lutea, number of uterine implantation sites, total number of pups born, and mean live litter size at high exposures in onegeneration reproductive toxicity studies; and fertility effects seen in the absence of maternal toxicity. In the two-generation reproductive and developmental toxicity study described above, no treatment-related effects were observed in male reproductive parameters. The following effects on female reproduction were observed: statistically significant decrease in mating and fertility indices in F1, and mean live litter size and mean number of pups in F0 and F1 at doses ≥500 ppm; extended parturition and dystocia in F0 at doses ≥500 ppm; and increased oestrus cycle length, reduced corpora lutea, and decreased number of pregnancies in F1 at 700 ppm (SCCP, 2005; Government of Canada, 2008; SCCS, 2010; Johnson et al., 2011; REACH). D5- Based on the available data, the chemical is not considered to cause reproductive or developmental toxicity following inhalation exposure at concentrations up to the maximum reproducible vapour pressure of approximately 160 ppm. D6- Based on the available data, the chemical is not expected to have reproductive or developmental toxicity.
Acute Toxicity	Based on the available data, the cyclic dimethyl polysiloxane compounds have low acute oral toxicity. <u>Oral</u> D4 - has low acute toxicity following oral exposure based on results from earlier animal studies (predating GLP). Although the details of these studies are lacking in the publicly available literature, the reported median lethal dose (LD50) values were > 2000 mg/kg bw in rats (SCCP, 2005; SCCS, 2010; Johnson et al., 2011; REACH). D5- In an acute oral toxicity study (similar to OECD TG 401), the median lethal dose (LD50) in Wistar rats was >4800 mg/kg bw (Government of Canada, 2008; SCCS, 2010; Johnson et al., 2011). In addition, other LD50 values were reported as >61,440 mg/kg bw in male

	Wistar rats, >20 000 mg/kg bw in SD rats and >24 134 mg/kg bw in rats (strain not specified) (REACH; RTECS). D6- The median lethal dose (LD50) in Wistar rats is >2000 mg/kg bw. No mortality or sub-lethal effects were observed (Government of Canada, 2008; OECD, 2009). <u>Dermal</u> D6- The LD50 in Wistar rats is >2000 mg/kg bw. No mortality or sub-lethal effects were observed (Government of Canada, 2008; OECD, 2009). D5 - The reported dermal LD50 values are >2000 mg/kg bw in New Zealand White rabbits; >15 360 mg/kg bw in NZW rabbits; and >2400 mg/kg bw in rats (strain not specified) (REACH). D4- LD50 values were reported as > 2000 mg/kg bw in rats and rabbits (SCCP, 2005; SCCS, 2010; Johnson et al., 2011; REACH). The studies in rats were reportedly conducted similarly to the OECD TG 402 (REACH). <u>Inhalation</u> D4 - The median lethal concentration (LC50) in rats is 36 mg/L. D5 - In an acute inhalation toxicity study conducted according to OECD TG 403, the median lethal concentration (LC50) was 8.67 mg/L (560 ppm) in F344 rats exposed (nose-only) to the aerosolised chemical for four hours. Observed sub-lethal effects included rigid gait, hunched posture, ruffled fur, restlessness and tachypnoea (rapid breathing) (SCCS, 2010; Johnson et al., 2011; REACH D6- no acute inhalation studies available
Irritation	Based on the available data, the chemicals are not a skin irritant. D6- In a skin irritation study conducted in accordance with OECD Test Guideline (TG) 404 (REACH), three New Zealand White (NZW) rabbits were applied 0.5 mL of the undiluted chemical under semi-occlusive conditions, for four hours. Signs of skin irritation were not observed at one, 24, 48 or 72 hours following exposure. The mean primary dermal irritation index (PDII) was reported to be zero (OECD, 2009; REACH). D5- In a dermal irritation study (similar to OECD TG 404) in NZW rabbits (n = 3/sex), animals were exposed (semi-occlusively) to the neat chemical on shaved sites (intact or abraded) for 24 hours and observed up to 72 hours. There were no signs of erythema (redness) or oedema (swelling), and the animals continued to gain weight during the study (REACH). D4- In a skin irritation study conducted similarly to OECD TG 404 (non-GLP), 0.5 mL of neat chemical was applied to intact and abraded skin of New Zealand White (NZW) rabbits (n=6; sex not specified). No skin reactions were seen in the intact skin. Desquamation was observed in abraded skin but fully resolved within 72 hours. No other details were provided (REACH). Based on the available data, the compounds are considered to be mild respiratory irritants. D4- The chemical caused local respiratory irritation at high concentrations in acute inhalation toxicity studies and in nose-only inhalation studies in rats. The effects in the nose and lungs were considered to be adaptive responses and not related to the treatment of the chemical (SCCP, 2005; Government of Canada, 2008; SCCS, 2010). D5- In several repeated dose inhalation toxicity studies (according or similar to OECD TG 412 or 413), F344 or SD rats exposed (nose only or whole body) to the chemical for 28 or 90 days showed increased incidence and severity of nasal cavity goblet cell (mucus-producing cells) proliferation and/or minimal to light lung interstitial inflammation. Effects

Cyclic dimethyl polysiloxane compounds

	were generally reversed after two or four week recovery periods, when there was no chemical exposure. These local morphological changes were considered to be of little or no relevance to human exposure (Government of Canada, 2008; EA, 2009; SCCS, 2010; Johnson et al., 2011; Danish EPA, 2014; Dekant & Klaunig, 2016; HSDB; REACH). D6-no studies found. Based on the available data, the chemicals are not considered to be eye irritants. D5- In a standard Draize test (similar to OECD TG 405) in NZW rabbits (n = 3/sex), 0.1 mL neat chemical was instilled into the conjunctival sac of one eye and animals were observed up to 72 hours. There were no signs of irritation (SCCS, 2010; REACH). D6- In an eye irritation study conducted in accordance with OECD TG 405 (REACH), three NZW rabbits were exposed to a volume of 0.1 mL of the undiluted chemical, instilled into one eye, which was observed at one, 24, 48 and 72 hours following exposure. Redness of the conjunctivae was observed but resolved within 24 hours (scores were not provided). The mean overall irritation score was reported to be zero (OECD, 2009; REACH). D4- In a study conducted in accordance with OECD TG 405, neat chemical (purity not specified) was instilled into the conjunctival sac of female NZW rabbits (n=3). The other eye was used as control. The treatment-related effects on the cornea, iris, and conjunctivae were evaluated at the following timepoints after instillation: 1, 24, 48, and 72 hours; and 7 days. The mean eye irritation score at the 24-, 48-, and 72-hour observation periods was 0.3 for conjunctival effects. No effects were seen on the cornea and iris (REACH).
Sensitisation	The compounds are not expected to be a skin sensitiser.
Key Study/Critical Effect for Screening Criteria	The critical effect level for repeated-dose toxicity is considered to be 100 mg/kg-bw/day via the oral route. This is based on increased periportal lipidosis and dose-related increases in liver weights and thyroid follicular cell hypertrophy in a 4-week rat study. The oral RfD is thus based on the NOAEL of 100 mg/kg/day. Uncertainty factors: 10 (interspecies variability); 10 (intraspecies variability); 10 (subchronic to chronic) Oral RfD = 100/1000 = 0.1 mg/kg/day Drinking water guideline value = 0.39 mg/L
Ecological Toxicity [1,5,6,7]	
Aquatic Toxicity	Toxicity tests at three trophic levels have been conducted (fish, algae, and daphnia) for D5 and D6. Test results in each study have shown that D5 and D6 are not toxic to aquatic organisms via water exposure to concentrations at or approaching the limit of water solubility. D4 is not toxic to fish when they are exposed for short durations (e.g. up to 96 hours) at concentrations up to the solubility limit in the test media. However, it causes toxicity in fish over longer exposure durations. In a prolonged acute toxicity test, 20 per cent mortality was observed at an exposure concentration of 6.9 µg/L, and the 14-day NOEC was 4.4 µg/L. Additional studies indicated that smaller fish appear to be more sensitive to D4 than larger fish. In contrast, a NOEC of ≥4.4 µg/L was obtained from a long-term (93-day) fish early life stage toxicity study (this was the highest concentration tested and no adverse effects were observed). It cannot be ruled out that effects might have been observed at higher concentrations (as suggested by the prolonged acute test). D4 is not toxic to aquatic invertebrates when they are exposed for short durations at concentrations up to the solubility limit in the test media. Following longer exposure, D4 does cause toxicity. The key study is: · Sousa *et al*. (1995) carried out a 21-day reproduction study with *Daphnia magna* using a flow-through system with no head space (to minimise loss of D4 through volatilisation). The D4 tested was >99 per cent pure and stock solutions of the substance were prepared by slow-stirring dilution water with a floating layer (approximately 6 mm thick) of D4. This method of stock-solution preparation gives reproducible results and can achieve a maximum concentration of ca. 15 µg/L in hard freshwater. Five exposure concentrations were used (measured concentrations were 1.7, 1.8, 4.2, 7.9, and 15 µg/L). This study showed a statistically significant (p = 0.05) reduction in the 32 survival at the highest concentration tested (survival in the 15 µg/L was 77 per

Cyclic dimethyl polysiloxane compounds

	cent) compared with the control population (survival was 93 per cent) after 21 days. The 21-day NOEC survival was therefore 7.9 µg/L. For the reproduction endpoint, the mean cumulative number of offspring per female daphnid was 111 in the control, 107, 92, 123, 151, and 167 in the 1.7, 1.8, 4.2, 7.9 and 15 µg/L treatment groups, respectively. There were no statistically significant (p = 0.05) differences between the control response and the treatment response in the 1.7, 1.8, and 4.2 µg/L groups, but the mean cumulative number of offspring per female was significantly higher in the 7.9 µg/L treatment group than in the control groups (the data for the 15 µg/L treatment group were not included in the statistical analysis as a reduction in daphnid survival occurred in this group). Therefore it is concluded that concentrations of D4 ≤7.9 µg/L do not adversely affect the reproduction of *D. magna*. No reliable laboratory data are available on the toxicity of D4 to algae but consideration of QSAR data in EA (2009a) suggests that algae should not be more sensitive to D4 than fish or invertebrates. D4 has a long-term fish NOEC of around 4 – 6 µg/L (although there is some uncertainty in this value) and a long-term NOECsurvival of 7.9 µg/L for *Daphnia magna*. Significant toxicity to invertebrates is also apparent in sediment organism studies. In addition, it is classified as toxic to reproduction category 2. Therefore it can therefore be concluded that D4 meets the Annex XIII criteria for toxicity (T) based on both aquatic and mammalian end points. (ECHA 2016) Chronic toxicity studies with freshwater fish (*Pimephales promelas,* 49 days) and the water flea (*Daphnia magna,* 21 days) showed no observed effects (NOEC) at the limits of water solubility (4.4 and 4.6 µg/L, respectively); based on total radioactivity using radiolabeled 14C-D6. D6 also showed no adverse effects (NOEC ≥ 2 µg/L) on the yield or growth rate of the freshwater alga, *Pseudokirchneriella subcapitata* in a closed bottle study at the functional limit of water solubility (2 µg/L). Likewise, no toxicity to *Lumbriculus variegatus* was observed during the sediment BAF study at the two concentrations tested, 28 and 484 mg/kg (dry weight; measured concentrations).
Determination of PNEC aquatic	In the aquatic compartment, two experimental chronic toxicity studies—one with *Daphnia magna* and the other with fathead minnow—were critically reviewed and accepted. D6 exhibited no adverse effects at 0.0044 to 0.0046 mg/L, a concentration that is very close to the water solubility limit of the substance (~ 0.005 mg/L). No application factor was applied to the chronic NOEC of 0.0046 mg/L. The PNEC is therefore 0.0046 mg/L.
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	No data found
PBT Assessment[1,5,6,7]	
P/vP Criteria fulfilled?	The compounds are not readily biodegradable and are persistent in the environment.
B/vB criteria fulfilled?	The compounds are bioaccumulative, based on measured fish BCF>10,000L/kg.
T criteria fulfilled?	The chronic aquatic toxicity of D4 is < 0.1 mg/L. Hence the substance does fulfill the screening criteria for toxic (T)
Overall conclusion	A PBT substance (based on screening data).

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Dicoco dimethyl quaternary ammonium chloride

Dicoco dimethyl quaternary ammonium chloride	
Chemical and Physical Properties[2]	
CAS number	61789-77-3
Molecular formula	C26H56CIN
Molecular weight	418.18 g/mol
Solubility in water	40-5040 mg/L
pH	No data found
Melting point	94 C
Boiling point	135 C
Colour/Form	Solid
Vapour pressure	Low
Henrys law constant	No data found
Explosive potential	No data found
Flammability potential	No data found
Overview	Dicoco dimethyl quaternary ammonium chloride is from a subgroup of quaternary ammonium salts that are derived from chemicals that have a biological origin. The substance represented by CAS# 61789-77-3 is expected to be a mixture of discrete chemicals with two alkyl chains of six to 18 carbons derived from coconut oil. Commercially available quaternary ammonium surfactants are often prepared indirectly from natural fats and oils. Natural fats derived from the fatty tissue of sheep or cattle, oil obtained from the kernel of the seed of *Cocos nucifera* (coconut), and seeds of *Glycine soja* (soybean) are used to prepare tallow alkyl-, coconut oil alkyl-, and soybean oil alkyl-ammonium compounds, respectively (Ash and Ash, 2004a; b). These surfactants have carbon chains with even numbers of carbon atoms, as fatty acid biosynthesis occurs mainly through addition of two carbon units in the form of acetyl-CoA (Voet and Voet, 1990). The major process for transforming fats and oils of biological origins into oleochemicals is the hydrolysis of natural triglycerides into glycerine and mixed fatty acids (Corma, et al., 2007). Reaction of these fatty acids and ammonia followed by hydrogenation produces fatty amines (Corma, et al., 2007), which are then alkylated at the nitrogen atom by reaction with chloromethane (de Oude, 1992). Alternatively, the fatty acids may be reacted with trimethylamine followed by hydrogenation to form quaternary ammonium compounds (Qadir, et al., 2014). Chemicals in this group are a source of cationic surfactants that have a wide range of industrial applications reported internationally. They are used in cleaning and washing agents as well as cosmetics, such as hair conditioners, hand soaps and deodorants. Due to their biocidal activity, they are used in agricultural and non-agricultural pesticides, disinfectants and preservatives (Nordic Council of Ministers, 2015; US EPA, 2015). There is also some indication of use as algaecides, indicating potential water treatment uses (US EPA, 2015; US NLM, 2011).
Environmental Fate[2]	
Soil/Water/Air	The chemicals in this group are all salts of quaternary ammonium surfactants and are therefore expected to have low volatility (de Oude, 1992). The water solubility values reported were determined at the critical micelle concentrations (CMCs), as is appropriate for surface-active substances. CMCs decrease with increasing alkyl chain lengths, and di-alkyl quaternary ammonium compounds have lower CMCs compared to mono-alkyl quaternary ammonium compounds with comparable alkyl chain lengths (Tezel, 2009). The octanol-water partition coefficient parameter (K) of the chemicals in this group is not considered to provide a reliable indicator of the partitioning behaviour of surface active substances in the environment (McWilliams and Payne, 2001; Shorts, et al., 2010), and therefore has not been reported. The quaternary ammonium cations from substances in this group partition between water and sediment, or remain in soil when released from industrial uses. The chemicals in this group are quaternary ammonium salts. If discharged into natural waters, the chemicals are expected to dissociate and release their quaternary ammonium cations. The quaternary

Dicoco dimethyl quaternary ammonium chloride

	ammonium cations can adsorb to clays and natural organic materials, such as humic substances (de Oude, 1992). They are expected to remain in soil as they are strongly adsorbed and immobile (Zhang, et al., 2015). The quaternary ammonium cations from substances in this group are biodegradable. Di-alkyl quaternary ammonium cations are also found to be rapidly biodegradable in water, undergoing 79 to 80% degradation after 2 days for those with C alkyl chains (CAS RNs 61789-80-8 and 61789-77-3) (US EPA, 2016). The quaternary ammonium cations from substances in this group have low to moderate bioaccumulation potential in aquatic organisms. The chemicals in this group are not expected to undergo long-range transport based on their low volatility and their biodegradability in the environment. Quaternary ammonium cations adsorbed to clays, sediment and soil containing organic carbon (de Oude, 1992; Ivankovic and Hrenovic, 2010) are strongly bound and immobile (Zhang, et al., 2015). Limited human health toxicity information is available for Dicocodimethylquaternaryammonium chloride, as such, information for dioctadecyldimethylammonium chloride (DODMAC) has been included below.
Human Health Toxicity Summary[1,3]	
Chronic Repeated Dose Toxicity	Following repeated oral exposure of 500 mg/kg bw/d of DODMAC to rats degeneration of adrenal cortex was induced. No adverse effects were reported up to 100 mg/kg bw/d DODMAC (NOAEL). After repeated dermal application to rabbits, local irritation but no systemic toxic effects were observed up to 40 mg/kg bw/d (NOAEL). A systemic LOAEL was not determined. There is no information on effects after prolonged inhalation exposure to rodents.
Carcinogenicity	No data is available on carcinogenic effects of DODMAC. There are no data from mutagenicity studies which give concern regarding carcinogenicity of both substances.
Mutagenicity/Genotoxicity	DODMAC showed negative results in bacterial mutation tests and in an *in vitro* chromosomal aberration test. There is no evidence of a genotoxic potential of the substance
Reproductive/developmental Toxicity	In an oral study on rats according to OECD Guideline 421 a dose of 500 mg/kg bw/d led to impaired reproductive performance in combination with clear signs of general toxicity. Based on the reduced mating, fertility and gestation indices a NOAEL for reproductive toxicity of 125 mg/kg/d can be estimated.
Acute Toxicity	In rats, the substance exhibited only low acute toxicity with oral LD50 > 2000 mg/kg bw, dermal LD50 > 200 mg/kg bw and inhalation LC50 > 180 mg/l/1 hour
Irritation	Pure DODMAC causes serious damage to the eyes but only moderate irritation to the skin of rabbits. Data on respiratory irritation is not available. Technical grade DODMAC, however, has proven to be corrosive to the skin of rabbits because of a high content of isopropanol
Sensitisation	DODMAC enhances the allergic potency of other chemical substances, but does not seem to cause skin sensitization by itself as judged on the basis of tests with relevant concentrations of DODMAC.
Key Study/Critical Effect for Screening Criteria	The key study chosen for the determination of a drinking water guidance value is the subacute oral rat study, where the NOAEL was 100 mg/kg/d. The oral RfD for Dicoco dimethyl quaternary ammonium chloride is thus based on the NOAEL of 100 mg/kg/day. Uncertainty factors: 10 (interspecies variability); 10 (intraspecies variability); 10 (subchronic to chronic). Oral RfD: 100/1000 = 0.1 mg/kg/day Drinking water guideline value = 0.39 ppm
Ecological Toxicity [2]	
Aquatic Toxicity	The following measured median lethal concentration (LC50) and median effective concentration (EC50) values for model organisms across three trophic levels were reported in the Screening-Level Hazard Characterisation conducted by the United States Environmental Protection Agency (US EPA, 2008), the European Union Risk Assessment Report (IHCP, 2009), and the databases included in the OECD QSAR Toolbox (LMC, 2013) Fish *Lepomis macrochirus* (Bluegill) 96 h LC50 = 1.04 mg/L Invertabrate Dap*hnia magna* (Water flea) 48 h LC50 = 0.16 mg/L Algae *Pseudokirchneriella subcapitata* (Green algae) 96 h EC50 = 0.46 mg/L

Dicoco dimethyl quaternary ammonium chloride

	The following no-observed effect concentration (NOEC) values for model organisms across two trophic levels were reported in the European Union Risk Assessment Report (IHCP, 2009) and the databases included in the OECD QSAR Toolbox (LMC, 2013) Invertebrates *Daphnia magna* (Water flea) 21 d NOEC = 0.38 mg/L Algae *Pseudokirchneriella subcapitata* (Green algae) 96 h NOEC = 0.16 mg/L While the chemicals in this group can be very toxic to aquatic organisms, they are efficiently removed from wastewater in sewage treatment plants and they typically undergo rapid biodegradation in water and soil.
Determination of PNEC aquatic	The calculated PNEC for mono-alkyl quaternary ammonium compounds with C alkyl chains is 3.6 µg/L based on the 72 h NOEC of 0.0018 mg/L for algae. The laboratory endpoint value for algae was divided by an assessment factor of 10 to account for interspecies variation and the derived value was then multiplied by a factor of 20 to account for the 5% bioavailable fraction in environmental waters. The calculated PNEC for di-alkyl quaternary ammonium compounds with C alkyl chains is 2.8 µg/L based on the 96 h EC50 of 0.014 mg/L for algae. This value was calculated by a similar procedure as applied to the mono-alkyl quaternary ammonium compound, but using an assessment factor of 100 in accordance with standard methodology for deriving PNECs from acute toxicity endpoint values (EPHC, 2009).
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	No data found
PBT Assessment[2]	
P/vP Criteria fulfilled?	Not Persistent (Not P). Based on results obtained from biodegradation studies, all chemicals in this group are categorised as Not Persistent.
B/vB criteria fulfilled?	Not Bioaccumulative (Not B). Based on the available measured bioconcentration data, all chemicals in this group are categorised as Not Bioaccumulative.
T criteria fulfilled?	Toxic (T). Based on available acute ecotoxicity values below 1 mg/L and/or chronic ecotoxicity values below 0.1 mg/L, all chemicals in this group are categorised as Toxic.
Overall conclusion	Not P, Not B, T. The chemicals in this group are not PBT substances according to domestic environmental hazard criteria.

References

1. European Commission Joint Research Centre 2009, European Union Risk Assessment Report, Dioctadecyldimethylammonium chloride, CAS no. 107-64-2.
2. National Industrial Chemicals Notification and Assessment Scheme (NICNAS), 2017. IMAP, Environmental Tier II Assessment for Mono- and Di-Alkyl Quaternary Ammonium Surfactants
3. OECD (1996) SIDS Initial Assessment Profile for Dioctadecyldimethylammonium chloride, CAS Number 107-64-2

Diutan

Diutan (D-Glucurono-6-deoxy-L-manno-D-glucan, acetate, calcium magnesium potassium sodium salt)	
Chemical and Physical Properties[1]	
CAS number	595585-15-2
Molecular formula	(C6H12O6. C6H12O5. C6H10O7)x.xC2H4O2. xCa.xK.xMg.xNa
Molecular weight	> 1,000,000 g/mol
Solubility in water	6.3 g/L at pH 1 @ 200C > 40 g/L at pH range 7 and 10 @ 200C
pH	No data found.
Melting point	Duitan decomposed from approximately 175 ± 0.5C without melting.
Boiling point	No data found.
Colour/Form	White to tan powder
Vapour pressure	≈ 0.1 kPa at 25 C
Henrys law constant	NA
Explosive potential	Not explosive
Flammability potential	Not flammable
Overview	The polymer Diutan is suitable for a wide variety of thickening and suspending applications. Diutan is likely to be used in the following categories of application: cementitious packaged products, viscosifier for spacer fluids, and viscosifier for oil field drilling fluid, oil field cementing, firefighting foams, concrete, tyre /pneumatic application sealants, cleaners and coatings. There is limited toxicological data available for Diutan. The following information is obtained from the National Industrial Chemicals Notification and Assessment Scheme (NICNAS). Based on the available data, the notified polymer is not classified as a hazardous substance in accordance with the NOHSC Approved Criteria for Classifying Hazardous Substances (NOHSC 2004). Available data on the polymer indicates that it is of low toxicity, however there are concerns about possible adverse effects on lungs after inhalation exposure. The hydrophilic nature of the notified polymer in powder form can contribute to mechanical irritation and collection in the eyes, on the skin or in the airways when dust is generated.
Environmental Fate[1]	
Soil/Water/Air	The polymer, Duitan is expected to be highly mobile in solids and was found to be readily biodegradable via biotic and abiotic processes under the OECD TG 301 B Ready Biodegradability: CO2 Evolution Test. Based on the molecular weight, water solubility and Kow value (log Kow -2.76) the polymer is not expected to bioaccumulate.
Human Health Toxicity Summary [1]	
Chronic Repeated Dose Toxicity	In a 28-day oral repeat dose study in rats, a No Observed Effect Level (NOEL) was established as 1000 mg/kg bw/day, based on the absence of treatment related effects.
Carcinogenicity	Diutan not listed as an IARC carcinogen
Mutagenicity/Genotoxicity	The polymer was not mutagenic to bacteria and not clastogenic to human lymphocyte treated in vitro.
Reproductive/developmental Toxicity	No data found.
Acute Toxicity	The polymer is of low acute toxicity via the oral route. Dermal toxicity was not tested. An acute inhalation study in rats showed effects that were seen in both the test and control animals to a similar extent, and therefore cannot be attributed to the notified polymer. However the level of airborne dust achieved in this study (0.316 mg/L) was well below the cutoff of 5 mg/L for determining hazard classification for this endpoint. The U.S. Environmental Protection Agency (USEPA) identified concerns for lung effects

	from inhalation exposure to the notified polymer when it was assessed as a new chemical in the USA, based on structural analogues and submitted test data. The concern is that fine respirable particles of a high molecular weight substance, when inhaled deep into the lungs, would absorb water and cause congestion (communication from notifier). While the USEPA does not expect water-soluble polymers to exhibit lung toxicity because they are expected to rapidly clear the respiratory tract and therefore not cause an overloading effect, they require testing on new chemicals of this type under their exposure –based authority (USEPA, 2006). In this case the USEPA considered that significant inhalation exposure would not occur under the use conditions described for the USA, but that significant human exposure could occur under other scenarios. They have therefore recommended that a 90-day inhalation study with 60-day holding period be performed if additional applications for the chemical commence.
Irritation	Based on a study in rabbits the polymer is considered to be slightly irritating to the eyes, but not classifiable. A dermal irritation study was carried out on an analogue chemical containing the same monosaccharide units, but with a different molecular weight and branching structure. The protocol for this study was more severe than the OECD test method, as it used a 24 h rather than 4 h exposure time, abraded skin and occlusive covering. The test substance was not washed from the skin after the exposure period. Under the conditions of this test the analogue polymer was moderately irritating, with mild erythema and slight to moderate oedema. Additional information on the irritation potential of the polymer is available from the irritation effects of a 50% solution of the notified polymer in the guinea pig sensitisation study (24 h exposure time). In this study there was mild to moderate erythema, but oedema was absent. Based on the results of these two studies, it is considered that the notified chemical would not be classified as a skin irritant
Sensitisation	There was no evidence of sensitisation potential to the polymer in the guinea pig maximisation test. Therefore the notified polymer is considered not to be a potential skin sensitiser.
Key Study/Critical Effect for Screening Criteria	The NOEL of 1000 mg/kg bw/day, derived from a 28-day rat oral study will be used to derive a drinking water guidance value. Uncertainty factors: 10 (interspecies variability); 10 (intraspecies variability), 10 (subacute to chronic). Oral RfD = 1000/1000 = 1 mg/kg/day Drinking water guideline = 3.9 ppm
Ecological Toxicity [1]	
Aquatic Toxicity	The results of the aquatic toxicity tests conducted by NICNAS are listed below.

Organism	*Duration*	*End Point*	*mg/L*
Freshwater Fish	96 h	LC_{50}	> 100
Freshwater Daphnia	48 h	LC_{50}	> 100
Marine water Copepod	48 h	LC_{50}	250
Freshwater Algae	0-72 h	E_bC_{50}	> 100
		E_rC_{50}	> 100
Marine water Algae	0-72 h	E_bC_{50}	> 1000
		E_rC_{50}	> 1000

Determination of PNEC aquatic	Using the lowest value of > 100 mg/L for freshwater organism and a safety factor of 100 (based on 3 experimental results) for fish/Daphnia/algal acute toxicity endpoints, a Predicted No Effect Concentration (PNEC) for freshwater is > 1 mg/L.
Screening Criteria	
Aquatic Toxicity Guidelines	No data available
Water Quality Guidelines	No data available
Occupational Exposure Limits	No data available

Diutan

PBT Assessment[1]	
P/vP Criteria fulfilled?	Diutan expected to readily biodegrade. Thus, it is not expected to meet the screening criteria for persistence
B/vB criteria fulfilled?	The molecular weight of Diutan is > 1,000,000 g/mol, and it is also water soluble. Thus, Diutan 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, Diutan is not expected to meet the screening criteria for toxicity
Overall conclusion	Not a PBT substance.

References

1. National Industrial Chemicals Notification and Assessment Scheme (NICNAS), Full Public Report, Diutan Gum, 2006.

Disodium EDTA, Trisodium EDTA, Tetrasodium EDTA

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 deg C - Disodium EDTA
pH	5.3 - Disodium EDTA
Melting point	242 C - Disodium EDTA >300 C – Tetrasodium EDTA
Boiling point	252 C (decomposes) - Disodium EDTA
Colour/Form	Solid granular materials
Vapour pressure	Negligible
Henrys law constant	Negligible
Explosive potential	No data found.
Flammability potential	No data found.
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
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.

Disodium EDTA, Trisodium EDTA, Tetrasodium EDTA

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/developmental Toxicity	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.
Key Study/Critical Effect for Screening Criteria	The Australian Drinking Water Guideline (0.25 mg/L, health) may be used. for EDTA
Ecological Toxicity [1,3]	
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

Disodium EDTA, Trisodium EDTA, Tetrasodium EDTA

	PNECaqua of 2.2 mg/l.
Screening Criteria[4]	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	The Australian Drinking Water Guideline for EDTA is 0.25 mg/L.
Occupational Exposure Limits	No data found
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).

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.

Ethylene Glycol

Ethylene Glycol	
Chemical and Physical Properties[1,2]	
CAS number	107-21-1
Molecular formula	$C_2H_6O_2$
Molecular weight	62.07 g/mol
Solubility in water	Miscible with water.
pH	No data found
Melting point	-12.69 °C
Boiling point	197.3°C
Colour/Form	Colourless odourless liquid
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.
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.
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]	
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.

Ethylene Glycol

	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

Ethylene Glycol

	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/developmental Toxicity	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).
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/day Drinking water guideline value = 0.59 ppm
Ecological Toxicity [1,3,5]	
Aquatic Toxicity	Ethylene glycol has generally low toxicity to aquatic organisms. The 96-hour LC50 values to fry, juvenile, and subadult fathead minnows (Pimephales promelas) are 53,000, 49,000 and 57,000 mg/L, respectively (Mayes et al., 1983). The 96-hour LC50 values to fathead minnows (Pimephales promelas) were also reported to be 72,860 mg/L (Pillard, 1995). The 96-hour LC50 to rainbow trout (Oncorhynchus mykiss) is 22,810 mg/L (OECD, 2004b). In a 12-day chronic study, the NOEC to rainbow trout (Oncorhynchus mykiss) was reported to be 14,692 mg/L (OECD, 2004b). The 48-hour LC50 to Daphnia magna is 46,300 mg/L (Gersich et al., 1986). The 48-hour EC50 values to Ceriodaphnia dubia-affinis and Daphnia

Ethylene Glycol

	magna are 10,000 and 51,000 mg/L at 24oC, respectively; and 25,800 and 46,300 mg/L at 24oC, respectively (Cowgill. et al., 1985). The NOEC for a 7-day reproduction test to Ceriodaphnia dubia is 3,469 mg/L (OECD, 2004b). The 96-hour EC50 to green algae (Selenastrum capricornutum) is 10,940 mg/L based on cell growth; the NOEC is 10,000 mg/L (Pillard and DuFresne, 1999).
Determination of PNEC aquatic	PNECaquatic: Experimental results are available for three trophic levels. Acute E(L)C50 values are available for fish (22,810 mg/L), Daphnia (10,000 mg/L), and algae (10,940 mg/L). NOEC values are available for fish (14,692 mg/L), invertebrates (3,469 mg/L), and algae (10,000 mg/L). On the basis that the data consists of short-term and long-term results from three trophic levels, an assessment factor of 10 has been applied to the lowest reported NOEC of 3,469 mg/L for invertebrates. The PNECaquatic is 347 mg/L.
Screening Criteria[2]	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	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).
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).

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Fumaric Acid and Monosodium Fumarate

Fumaric Acid and Monosodium Fumarate	
Chemical and Physical Properties[2,3,5,8]	
CAS number	Fumaric Acid: 110-17-8 Monosodium Fumarate: 7704-73-6
Molecular formula	Fumaric Acid: C4H4O4 Monosodium Fumarate: C4H3NaO4
Molecular weight	Fumaric Acid: 116.07 g/mol Monosodium Fumarate: 138.06 g/mol
Solubility in water	Fumaric Acid: 7000 mg/L @ 25C Monosodium Fumarate: Soluble in water
pH	No data found
Melting point	287 C
Boiling point	522 C
Colour/Form	Fumaric Acid: Colourless odourless crystals or powder Monosodium Fumarate: Odourless, white crystalline powder
Vapour pressure	1.54X10-4 mm Hg at 25 deg C
Henrys law constant	No data found
Explosive potential	Dust presents explosion hazard
Flammability potential	Non flammable
Overview	Fumaric acid is an organic dicarboxylic acid naturally present in all organisms. It predominantly originates from the oxidation of succinate and is further converted to malic acid in the tricarboxylic acid cycle. Exogenous fumaric acid will be rapidly metabolised by well-recognised pathways, and neither fumarate nor its metabolites would be expected to accumulate in human or animal tissues. Fumaric acid is used primarily in liquid pharmaceutical preparations as an acidulant and flavoring agent. Fumaric acid is approved for use as a food additive in Australia, and use as a therapeutic agent in the treatment of psoriasis and other skin disorders, as wells as a feed additive for all animals without a maximum level. A Tier 1 human health risk assessment has been performed by the Australian Government Department of Health, National Industrial Chemicals Notification and Assessment Scheme (NICNAS), indicating the chemical is not considered to pose an unreasonable risk to the health of workers and public health. The highest category use is listed as Cosmetic and the data available on the function of the chemical indicate that it may be used in cosmetics but only at low concentrations. Monosodium fumarate is the sodium salt of fumaric acid, and is a food additive, used as a flavour enhancer and acidity regulator. The WHO JECFA has listed a group ADI of “not specified” for fumaric acid and its salts in 1999. Limited information is available for monosodium fumarate, and as such Fumaric acid has been used as its surrogate.
Environmental Fate[5]	
Soil/Water/Air	If released to soil, fumaric acid is expected to have very high mobility based upon an estimated Koc of 7. The pKa values of fumaric acid are 3.03 and 4.54, indicating that this compound will 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 is not expected because the acid exists as an anion and anions do not volatilize. Using a Warburg respirometer and a sewage inoculum, 5-day Theoretical BODs of 57-70% were reported, suggesting that biodegradation may be an important environmental fate process in soil. If released into water, fumaric acid is not expected to adsorb to suspended solids and sediment based upon the estimated Koc. The half-life of fumaric acid in various natural waters ranged from 1-15 days using river die-away studies, indicating that biodegradation is an important environmental fate process in water. Fumaric acid's pKa values indicate it 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. 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. Fumaric acid will be degraded in brightly sunlit natural waters by reaction with

Fumaric Acid and Monosodium Fumarate

	photochemically produced hydroxyl radicals with a half-life of 45 days.
Human Health Toxicity Summary[5,6]	
Chronic Repeated Dose Toxicity	Eight groups of 14 weanling rats were kept on diets containing 0, 0.1 and 1.0% fumaric acid and 1.38% sodium fumarate for one year (half the groups) or two years. No adverse effect was noted on rate of weight gain, haemoglobin, blood picture, calcium balance as shown by bone histology, or on the histology of liver, kidney, spleen and stomach (Levey et al., 1946). In another experiment five groups of 12 male and 12 female rats were fed diets containing 0, 0.1, 0.5, 0.8 and 1.2% of fumaric acid for two years without toxic effects on growth or food consumption. A further four groups of 12 male rats were kept for two years on diets containing 0, 0.5, 1.0 and 1.5% fumaric acid. Only at the 1.5% level was there a very slight increase in mortality rate and some testicular atrophy. Gross and microscopic examination of major organs revealed no abnormalities and tumour incidence was not significantly different between the groups (Fitzhugh & Nelson, 1947). Seventy-five chronically disabled subjects ranging in age from 29-91 years received 500 mg fumaric acid daily for one year without any toxic manifestations in haemoglobin level, RBC and WBC, nonprotein nitrogen level, creatinine level, bromosulfonphthalein excretion and phenolsulfonphthalein excretion (Levey et al., 1946).
Carcinogenicity	Based on the available data, fumaric acid is not considered to be a carcinogen. Fumaric acid has not been classified by International Agency for Research on Cancer (IARC) or the United States Environment Protection Agency (USEPA).
Mutagenicity/Genotoxicity	Fumaric acid is not considered to be a mutagen.
Reproductive/developmental Toxicity	No data found
Acute Toxicity	Fumaric acid has low acute toxicity via oral, inhalation, or dermal exposure. The LD50s for the oral administration of fumaric acid in rats range from 8,000 to 10,700 mg/kg bw and 3,600 to 4,800 mg/kg bw for rabbits. Inhalation LD50s for rats is reported to be 1,306 mg/L and a dermal LD50 of 20,000 mg/kg bw has been reported for rabbits.
Irritation	The available data show that fumaric acid is a mild irritant of the skin and may cause respiratory tract irritation. Furmaric acid is considered to cause serious eye irritation. Ingestion of fumaric acid may cause abdominal cramps, diarrhoea and nausea.
Sensitisation	The chemical is considered to be not sensitising.
Key Study/Critical Effect for Screening Criteria	WHO JECFA in 1975 derived an acceptable daily intake of 6 mg/kg bw for adults and children for use as a food additive. The key study chosen was the two-year rat feeding study by Fitzhugh & Nelson, (1947). No adverse chronic effects from fumaric acid dosing were seen in animals exposed below 1.2% (600 mg/kg bw). However it is to be noted that in 1989, the ADI was changed to 'not specified' when Fumaric Acid was evaluated as a flavouring agent by the JECFA. Drinking water guideline value = 2.3 ppm
Ecological Toxicity [3,5]	
Aquatic Toxicity	The EC50s for zebrafish was 245 mg/L for a 48 hour static test. The EC50 for Daphnia magna was 212 mg/L for a 48 hour static test. The EC50 for green algae was 41 mg/L for a 72 hour algal growth inhibition test.
Determination of PNEC $_{aquatic}$	PNECaquatic: Experimental results are available for three trophic levels. Acute E(L)C50 values are available for fish (245 mg/L), Daphnia (212 mg/L), and algae (41 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 41 mg/L for algae. The $PNEC_{aquatic}$ was calculated to be 0.041 mg/L.
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	No data found
PBT Assessment[3,5]	
P/vP Criteria fulfilled?	Fumaric acid is readily biodegradable and as such not persistent in the environment.
B/vB criteria fulfilled?	Based on the measured log Kow of <3 Fumaric acid is not bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of Fumaric acid is > 0.01 mg/L. Hence the substance does not fulfill the screening criteria for toxic (T)

Fumaric Acid and Monosodium Fumarate

Overall conclusion	Not a PBT substance (based on screening data).

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Guar Gum

Guar Gum	
Chemical and Physical Properties[1,2]	
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.
Colour/Form	solid
Vapour pressure	NA
Henrys law constant	NA
Explosive potential	NA
Flammability potential	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).
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	
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.

Guar Gum

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/developmental Toxicity	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	
Aquatic Toxicity	The 48-hour LC50 for Daphnia magna was reported to be 42 mg/L .
Determination of PNEC aquatic	PNECaquatic: Experimental results are available for one trophic level. An acute LC50 value is available for the invertebrate Daphnia (42 mg/L). 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 42 mg/L for Daphnia. The PNECaquatic is 0.042 mg/L.
Screening Criteria	
Aquatic Toxicity Guidelines	No data available
Water Quality Guidelines	No data available
Occupational Exposure Limits	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

Guar Gum

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
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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

HYDROCHLORIC ACID

HYDROCHLORIC ACID	
Chemical and Physical Properties[1,2]	
CAS number	7647-01-0
Molecular formula	HCl
Product name	Hydrochloric acid
Molecular weight	36.46 g/mol
Solubility in water	Soluble
Melting point	-114.22 $_{o}$C
Boiling point	-85.05$_{o}$C
Vapour pressure	35,424 mm Hg at 25 deg C
Henrys law constant	2.04 x10^{6} mol/L atm
Explosive potential	Reacts with most metals producing explosive hydrogen gas
Flammability potential	Not combustible
Colour/Form	liquid
Overview	HCl, hydrogen chloride the gas, and HCl, the aqueous acid (hydrochloric acid), have the same 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 [1,3,4]	
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 (Upotn 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 artifact 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.
Reproductive Toxicity	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

HYDROCHLORIC ACID

	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. Hydrogen chloride/hydrochloric acid is not expected to have developmental toxicity.
Developmental Toxicity/Teratogenicity	No data found.
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
Irritation	Concentrations >3.3% cause skin irritation, and concentrations >17% cause corrosion in animal studies.
Sensitisation	May cause dermatitis with frequent contact of aqueous solutions of hydrochloric acid.
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]	
Aquatic Toxicity	LC50 values of acute fish toxicity tests are highly variable. The hazard of hydrochloric acid for the environment is caused by the proton (pH effect). For this reason the effect of hydrochloric acid on the organisms depends on the buffer capacity of the aquatic ecosystem.
Determination of PNEC aquatic	PNECaquatic: It is not considered useful to calculate a PNEC for hydrochloric acid because factors such as the buffer capacity, the natural pH, and the fluctuation of the pH are very specific for a certain ecosystem. Based on the information above, a PNECaquatic was not derived for hydrochloric acid
Screening Criteria[7]	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	Peak limitation – 7.5 mg/m^3
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

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.

MAGNESIUM CHLORIDE

MAGNESIUM CHLORIDE	
Chemical and Physical Properties[1,3]	
CAS number	7786-30-3
Molecular formula	$MgCl_2$
Product name	M275
Molecular weight	95.21 g/mol
Solubility in water	56.g/100g at 20^{o}C
Melting point	714 ^{o}C
Boiling point	1,412^{o}C
Vapour pressure	No data found
Henrys law constant	No data found
Explosive potential	No data found
Flammability potential	No data found
Colour/Form	colourless crystalline powder
Overview	Magnesium chloride is an organic salt that dissociates completely to magnesium and chloride ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions; both magnesium and chloride ions are also ubiquitous and are present in most water, soil and sediment. Magnesium chloride is a component of fire extinguishers, ceramics, textile and paper manufacture and is also used in medication and disinfectants.
Environmental Fate[1,3]	
Soil/Water/Air	Given its high solubility in water, magnesium chloride will dissociate and release Mg2+ and Cl- ions. The dissociated Mg2+ cation can then transform and form complexes with dissolved ligands present in natural waters. Magnesium is widespread in living cells and does not bioconcentrate in aquatic organisms. Environmental fate analysis based on log Kow and log Koc and typical fugacity modelling is not applicable to magnesium chloride as it is an inorganic compound. Photodegradation and biodegradation are also not applicable to inorganic metal salts such as magnesium chloride.
Human Health Toxicity Summary [1,3]	
Chronic Repeated Dose Toxicity	In a repeated dose oral toxicity study in rats, there was no death observed in either sex. No dose-related changes in body weights, food consumption, sensory function, motor activity, urinalysis, hematology, blood chemistry, organ weights, and histopathology of tissues from a wide range of organs were noted at the highest dose level. However in 500 and 1,000 mg/kg bw/day main group, increased respiration was sporadically or frequently observed in most animals. Based on the effects on soft stool and abnormal gait and increased respiration in female 1,000 mg/kg bw group, the NOAEL for repeated dose oral toxicity was considered to be 1,000 (male) and 500 (female) mg/kg bw/day.
Carcinogenicity	Male and female mice were given in their feed 0, 0.5% or 2% magnesium chloride hexahydrate for 96 weeks, after which all animals received the control diet for 8 weeks and were then necropsied. Tumors were mainly found in the skin/subcutis, liver and lymphatic system. With the exception of a significant decrease in the incidence of liver tumors among the 2% males, there were no differences in the tumor incidences between the treated and control animals. Based on in vivo oral carcinogenicity study, magnesium chloride did not induce carcinogenic effects.
Mutagenicity/Genotoxicity	Based on the in vitro study results, magnesium chloride was not mutagenic in bacteria and did not cause chromosome aberration in hamster lung cells. And according to the in vivo study result, the test substance did not induce micronucleus formation in the femoral marrow cells in mice. In conclusion, magnesium chloride is considered to be non genotoxic under in vitro and in vivo conditions.
Reproductive/Developmental Toxicity	In an OECD 422 study, Wistar rats at dosages of 0, 250, 500 or 1,000 mg/kg showed no treatment related reproductive or developmental effects. The NOAEL for this study is 1,000 mg/kg/day.
Acute Toxicity	The acute oral LD50 value for female rats was between 300 and 2,000 mg/kg bw and was estimated to be 1,085 mg/kg bw. All animals of 2,000 mg/kg bw died without clinical signs after administration. Gastric filling with the test substance was observed in all the animals

MAGNESIUM CHLORIDE

	autopsied. The 24 hours dermal LD50 of magnesium chloride was in excess of 2,000 mg/kg bw in rats. Signs of toxicity such as diarrhea and watery diarrhea were observed at 2,000 mg/kg bw. According to the results, magnesium chloride showed moderate acute toxicity.
Irritation	No reliable studies available.
Sensitisation	Magnesium chloride hexahydrate was not a skin sensitizer when tested in guinea pigs.
Key Study/Critical Effect for Screening Criteria	The Australian drinking water guideline value for chloride may apply to magnesium chloride.
Ecological Toxicity [1,3]	
Aquatic Toxicity	In an acute toxicity test (OECD TG 203, the 96hr-LC50 and NOEC values for rice fish (Oryzias latipes) exposed to 100 mg/L magnesium chloride under static conditions were >95.2 mg/L and ≥95.2 mg/L, respectively. There were no mortalities and adverse effects observed in any of the fish exposed during the test. The 96-hour LC50 of magnesium chloride to Pimephales promelas is 2,119.3 mg of $MgCl_2$/L. The 48-hour LC50 for Daphnia magna is 548.4 mg of $MgCl_2$/L. In another study, the 48-hour LC50 to Daphnia magna and Ceriodaphnia dubia were 1328 and 881.4 mg of $MgCl_2$/L, respectively. The 72-hour EC50 of magnesium chloride for algae Scenedesmus subspicatus is >100 mg/L and the NOEC is 100 mg/L.
Determination of PNEC aquatic	$PNEC_{aquatic}$: Experimental results are available for three trophic levels. Acute $E(L)C_{50}$ values are available for fish (2,119 mg/L as $MgCl_2$), Daphnia (548 mg/L as $MgCl_2$), and algae (100 mg/L $MgCl_2$). 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 100 mg/L for algae. The $PNEC_{aquatic}$ was calculated to be 0.1 mg/L.
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	No data found
PBT Assessment[1]	
P/vP Criteria fulfilled?	Magnesium chloride is an organic salt that dissociates completely to magnesium and chloride ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions; both magnesium and chloride ions are also ubiquitous and are present in most water, soil and sediment. Thus, the persistent criterion is not considered applicable to this inorganic salt.
B/vB criteria fulfilled?	Magnesium and chloride ions are essential to all living organisms and their intracellular and extracellular concentrations are actively regulated. Thus, magnesium chloride is not expected to bioaccumulate.
T criteria fulfilled?	No chronic toxicity data exist on magnesium chloride; however, the acute EC(L)50s are >0.1 mg/L in fish, invertebrates and algae. Thus, magnesium chloride does not meet the adopted screening criteria for toxicity (refer to Section XX).
Overall conclusion	Not PBT

References

1. ECHA REACH database: http://apps.echa.europa.eu/registered/registered-sub.aspx
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. OECD-SIDS (2011) Screening Information Dataset (SIDS) Initial Assessment Report for Magnesium chloride (CASRN 7786-30-3), UNEP Publications.

MAGNESIUM NITRATE

MAGNESIUM NITRATE	
Chemical and Physical Properties[1,2]	
CAS number	10377-60-3
Molecular formula	$Mg(N0_3)_2$
Product name	M275
Molecular weight	148.31 g/mol
Solubility in water	71.2 g/100g at 25 deg $_0$C
Melting point	95$_0$C
Boiling point	330$_0$C
Colour/Form	White cubic crystals
Vapour pressure	0.494 mm Hg @ 25 deg C
Henrys law constant	No data found
Explosive potential	The substance is a strong oxidant and reacts with combustible and reducing materials, causing an explosion hazard.
Flammability potential	Magnesium nitrate itself is not combustible, but it enhances the combustion of other substances. It can give off irritating, corrosive and/or toxic fumes or gases in a fire.
Overview	Magnesium nitrate is an organic salt that dissociates completely to magnesium and nitrate ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions; both magnesium and nitrate ions are also ubiquitous and are present in most water, soil and sediment. Uses of magnesium nitrate include a catalyst in the manufacture of petrochemicals, as a desensitiser for lithographic plates or an alternative to sulphuric acid in the purification of nitric acid.
Environmental Fate[1,10]	
Soil/Water/Air	Magnesium nitrate is a water soluble inorganic salt that readily dissociate into the magnesium cation and the nitrate anion. Due to nitrate's high water solubility and weak retention by soil, nitrates are very mobile in soil, moving at approximately the same rate as water, and have a high potential to migrate to ground water. Because it does not volatilize, nitrate is likely to remain in water until consumed by plants or other organisms. Nitrate degradation is fastest in anaerobic conditions.
Human Health Toxicity Summary [4,6,7]	
Chronic Repeated Dose Toxicity	Repeated small doses of nitrates may cause weakness, headaches and mental impairment. The substance may cause effects on the blood when ingested, impairing its ability to carry oxygen, and resulting in the formation of methaemoglobin. The effects may be delayed. Magnesium nitrate breaks down into magnesium and nitrate once in the body. Magnesium is an essential element. Little information is available regarding the effects of long-term exposure to Magnesium nitrate and no LD/LC_{50} values were found. It was therefore decided to review information relating to the health effects of nitrates. In a 2-year study, rats were fed a diet containing 0, 0.1, 1, 5 or 10% sodium nitrate. At the 5% dose level a slight growth inhibition was observed, whereas inanition was noticed at the 10% dose level. No abnormalities or increased tumour incidence were found. The NOAEL was 1% (500 mg sodium nitrate/kg bw/day). Rabbits were given 0, 250 or 500 mg nitrate/l during 22 weeks. No detrimental effects on reproductive performances were identified after successive gestations. In a 7-month study, 15 female cows were fed a diet containing 445 or 665 mg NO_3^-/kg from 2 months of pregnancy until birth. No changes in pregnancy were observed. 20-50% incidence of methaemoglobinaemia was identified.
Carcinogenicity	Magnesium nitrate has not been reviewed by IARC. For 1 year, mice were fed diets containing 0, 25,000 or 50,000 mg sodium nitrate/kg of feed. No difference in tumour incidences was observed in the animals.
Mutagenicity/Genotoxicity	Magnesium nitrate hexahydrate is not mutagenic in the *Salmonella typhimurium* reverse mutation assay and in the *Escherichia coli* reverse mutation assay.
Reproductive/Developmental Toxicity	No data available
Acute Toxicity	The acute oral LD50 in rats is >5,000 mg/kg. A nuisance-causing concentration of airborne particles can be reached quickly when dispersed. May cause mechanical irritation to the eyes and the respiratory tract. Inhalation symptoms may include a cough, and/or sore throat. Eyes may become red and pain may be experienced. If ingested, symptoms may

MAGNESIUM NITRATE

	include abdominal pain, blue lips or fingernails, blue skin, confusion, convulsions, dizziness, headache, nausea, and unconsciousness. The concentration thresholds at which such symptoms would occur are not stated in the data source.
Irritation	No studies are available.
Sensitisation	Magnesium nitrate is not a skin sensitizer based on results of a Local Lymph Node Assay.
Key Study/Critical Effect for Screening Criteria	The Australian drinking water guidance value for nitrates may apply to magnesium nitrate.
Ecological Toxicity [1,3]	
Aquatic Toxicity	No data found for magnesium nitrate. Estimated acute toxicity data for nitrate using the USEPA ECOSAR database is as follows: LC50 for Fish is 2067.87 mg/L LC50 for Daphnid is 995.185 mg/L EC50 for Algae is 374.159 mg/L
Determination of PNEC aquatic	PNECaquatic: The ANZECC water quality guideline (2000) uses acute and chronic laboratory toxicity data for the derivation of "trigger values" for nitrate. The guideline summary for freshwaters is: *"A freshwater moderate reliability trigger value for nitrate toxicity as NO3 (nitrate) of 700 ug/L was calculated using the statistical distribution method 95% protection and the default ACR."*
Screening Criteria	
Aquatic Toxicity Guidelines	ANZECC 2000 Freshwater Quality Guideline - 700 ug/L for nitrate.
Water Quality Guidelines	ADWG drinking water guideline - 50 mg/L for nitrate.
Occupational Exposure Limits	No data available
PBT Assessment	
P/vP Criteria fulfilled?	Magnesium nitrate is an organic salt that dissociates completely to magnesium and nitrate ions in aqueous solutions. Biodegradation is not applicable to these inorganic ions; both magnesium and nitrate ions are also ubiquitous and are present in most water, soil and sediment. Thus, the persistent criterion is not considered applicable to this inorganic salt.
B/vB criteria fulfilled?	Magnesium ions are essential to all living organisms and its intracellular and extracellular concentrations are actively regulated. Nitrates are ions that are very soluble in aqueous solutions. Thus, magnesium nitrate is not expected to bioaccumulate.
T criteria fulfilled?	No chronic toxicity data exist on magnesium nitrate; however, the acute EC(L)50s are >0.1 mg/L in fish, invertebrates and algae. Thus, magnesium nitrate does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT

References

1. ECHA REACH database: http://apps.echa.europa.eu/registered/registered-sub.aspx
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. ANZECC & ARMCANZ (2000). Australian and New Zealand guidelines for fresh and marine water quality. National Water Quality Management Strategy Paper No 4, Australian and New Zealand Environment and Conservation Council & Agriculture and Resource Management Council of Australia and New Zealand, Canberra, Australia.
4. IPCS (International Programme on Chemical Safety) Inchem . (1999), International Chemical Safety Card 1041. http://www.inchem.org/documents/icsc/icsc/eics1041.htm
5. IARC (International Agency for Research on Cancer). (2011), Agents Classified by the IARC Monographs, Volumes 1 -102. http://www.iarc.fr
6. WHO JEFCA (Joint Expert Committee on Food Additives). (1996), WHO Food Additives Series, No.35. http://www.inchem.org/pages/jecfa.html
7. ECOTOX, US Environmental Protection Agency ECOTOX database
8. ECOSAR, US Environmental Protection Agency ECOSAR database , Ecological Structure Activity Relationships (ECOSAR) Class Program.
9. USEPA, Office of Prevention, Pesticides and Toxic Substances, Inert Tolerance Reassessment – Ammonium Nitrate, Magnesium Nitrate, Sodium Nitrate and Sodium Nitrite, January 2005

Methylisothiazolinone and Methylchloroisothiazolinone

Methylisothiazolinone and Methylchloroisothiazolinone	
Chemical and Physical Properties[1]	
CAS number	Methylisothiazolinone: 2682-20-4 Methylchloroisothiazolinone: 26172-55-4
Molecular formula	Methylisothiazolinone: C_4H_5NOS Methylchloroisothiazolinone: C_4H_4ClNOS
Product name	--
Molecular weight	Methylisothiazolinone: 115.15 g/mol Methylchloroisothiazolinone: 149.60 g/mol
Solubility in water	Methylisothiazolinone: 5.367 x 10^5 mg/L at 25^{o}C Methylchloroisothiazolinone: 1.487 x 10^5 mg/L at 25^{o}C
Melting point	Methylisothiazolinone: 47.48 ^{o}C Methylchloroisothiazolinone: 50-55^{o}C
Boiling point	Methylisothiazolinone: 237.75 ^{o}C Methylchloroisothiazolinone: 106.5 ^{o}C
Colour/Form	Liquid
Vapour pressure	Methylisothiazolinone: 0.031 mm Hg at 25^{o}C Methylchloroisothiazolinone: 20.8 hPa at 20^{o}C
Henrys law constant	Methylisothiazolinone: 4.96 x 10^{-8} atm m^3/mol Methylchloroisothiazolinone: 3.57 x 10^{-8} atm-m^3 /mol
Explosive potential	NA
Flammability potential	NA
Overview	The Isothiazoline derivatives have limited available toxicological data however, the information presented above highlights moderate short-term toxicity but high concerns for irritancy and sensitisation if contact with the substance occurs. There is a lack of data related to chronic toxicity but as persistence and bioaccumulation has not been determined as significant in the ecological assessments chronic exposures following environmental distribution is not anticipated. The moderate toxicity level of concern is thus more focussed towards acute occupational and environmental incidents during fracturing operations. Isothiazoline derivatives are effective biocides (antiseptic agents, preservatives, bactericides, slimicides, and fungicides). The biggest application is in the paint industry especially marine antifouling agent. 5-chloro-2-methyl-4-isothiazolin-3-one (CMIT), is used as a biodiesel biocide and is a high performance, broad spectrum antimicrobial agent based on isothiazolone chemistry. CMIT/MIT is very effective at very low concentrations in controlling microorganisms causing microbial induced spoilage. No other preservatives control a wider range of microorganisms over a wide range of pH at such low levels. They are also used in adhesives, cutting oils, water systems, cosmetics, household goods and wound protectant for pruning cuts. They are also used as pulp and wood impregnating agents as well as in leather, fur and polymer process.
Environmental Fate[1,2]	
Biodegradation	5-Chloro-2-methyl-4-isothiazolin-3-one and 2-methyl-4-isothiazolin-3-one, the active ingredients in DOW™ CMIT/MIT antimicrobial products, have a low bioconcentration potential (tendency to accumulate in the food chain) and a high potential for mobility in soil. They are completely soluble in water. In the environment these compounds would tend to partition into water where they would rapidly biodegrade to non-persistent substances which are several orders of magnitude less toxic than parent compound. They would also be removed by common biological wastewater treatment facilities
Human Health Toxicity Summary [1,2]	
Chronic Repeated Dose Toxicity	Chronic hazards are due to it causing sensitization by skin contact over the long-term. A 30 month skin painting study with mice using 400ppm isothiasoline three times per week showed no increased tumour frequency over control.
Carcinogenicity	Has not been reviewed by IARC. In a two-year rat chronic study, Kathon 886 is given as 14.2% active ingredient with a pH between 2-3. In the document with compiled batch information for Kathon the active ingredient is 13.2, [10.13 % CMI/ 3.85% MI] with 15.4%

	magnesium nitrate and 9.0% magnesium chloride. The dose levels were 0, 30, 100 and 300 ppm (equivalent to: 0, 2.0, 6.6, 17.2 mg a.i./kg/day in males and 0, 3.1, 9.8, 25.7 mg a.i./kg/day in females. There were no deaths. There were no treatment-related effects on body weight or body weight gain at doses or food consumption up to and including the mid dose group. A treatment related and concentration-dependent decrease in water consumption was seen in both sexes in all treated groups throughout the study. These decreases ranged from 0-22% at low dose 3-30% at mid dose and 15-40% at high dose. These decreases appear to be due to the unpalatability of the CMI/MI and not its inorganic stabilizer salts since the water consumption in the salt control was comparable to the tap water control throughout the study. Based on the average daily water consumption, the high dose was judged to be a maximum tolerated dose. The decreases in body weight and body weight gain were seen in high dose animals throughout the study and may be secondary to decreased water consumption. No treatment-related clinical effects were recorded. No treatment-related ophthalmic, hematological. biochemical or urinary changes were noted. Organ weights were comparable to the control. No effects on type or incidence of neoplasms were seen at up to and including the high dose (males: 17.2 a.i.; females 25.7 mg a.i./kg/day). Slight to moderate forestomach hyperplasia was seen at both mid and high dose groups. Gastric irritation was the primary effect observed. No adverse effects on the histopathology of any other tissues/organs were observed away from the site of dosing. No systemic effects were observed. The NOEL in this study was considered to be 30 ppm a.i. (2.0 to 3.1 mg a.i./kg/day), based primarily on gastric irritation of the stomach at 100 and 300 ppm a.i.. The NOAEL was considered to be 300 ppm a.i. (17.2 to 25.7mg/a.i./kg/day), since no evidence of systemic toxicity was observed at any dose and there was no adverse effects on the histopathology of any tissues/organs distant from the site of dosing at any dose.
Mutagenicity/Genotoxicity	The results of the *in vitro* mutagenicity tests were equivocal. In bacterial reverse mutation tests, CMI/MI was positive in *Salmonella* strain TA100 but was predominantly negative in other strains commonly tested strains. CMI/MI was mutagenic in the mouse lymphoma assay with and without metabolic activation. Effects at the Tk locus was seen both with and without S-9, but were enhanced with S9 mix. CMI/MI was not genotoxic in the *in vitro* unscheduled DNA synthesis (UDS) assay with primary rat hepatocytes or in an *in vitro* chromosomal aberration test with Chinese hamster lung cells. The *in vivo* studies indicated that CMI/MI does not have relevant mutagenic potential *in vivo*. The positive mutagenic effect of CMI/MI found in *in vitro* gene mutation assays was not confirmed in the sex-linked recessive lethal test in *Drosophila melanogaster* nor in two unscheduled DNA synthesis (UDS) studies in the rat. CMI/MI also did not show any increase in cells with micronuclei in mice nor did it induce chromosomal aberrations in rat bone marrow cells.
Reproductive/ Developmental Toxicity/Teratogenicity	A teratology study with rabbits and rats was negative using dosages of 1.5 to 15mg/kg isothiazoline.
Acute Toxicity	The substance causes burns to the eyes and skin and may cause sensitization by skin contact. It is corrosive to the respiratory system. It is corrosive and causes burns to the gastro-intestinal tract with effects such as nausea, vomiting and stomach pain. In severe cases blood may be vomited The oral LD50 of Kathon WT (1.5% a.i.) is >5,000 mg/kg (>75 mg a.i./kg) in male rats and >3,310 mg/kg (~49.6 mg a.i./kg) in female rats. The oral LD50 of Acticide 14 (14% a.i.) is 472-490 mg/kg (~66 to 69 mg a.i./kg) in rats (combined sexes); and 465 mg/kg (69 mg a.i./kg) in male rats and 393 mg/kg (59 mg a.i./kg) in female rats. The dermal LD50 of Kathon CG is >5,000 mg/kg (>75 mg a.i./kg) in female rabbits. The dermal LD50 of Acticide 14 (14% a.i.) is 1,008 mg/kg (141 mg a.i./kg) in rats (combined sexes). The inhalation LC50 of Kathon 886F (13.9% a.i.) in male and female rats is 2.36 mg/L (0.33 mg a.i./L).
Irritation	Undiluted Kathon MW (1.5% a.i.) and Acticide 14 (14% a.i.) were corrosive to rabbit skin. Undiluted Kathon CG was severely irritating to the eyes of rabbits. Kathon RH 886T at 100 ppm (0.01% a.i.) was non-irritating to the eyes of rabbits.
Sensitisation	CMI/MI has been tested in the guinea pig Buehler and Magnusson-Kligman tests and has been shown to be a strong skin sensitizer. It is also a strong contact allergen in humans.
Key Study/Critical Effect for Screening Criteria	A two-year dietary study was conducted in rats with CMI/MI (13.2% active ingredient). No systemic effects were observed. There was, however, gastric irritation of the stomach at 100 and 300 ppm. The NOEL for this study is 30 ppm (equivalent to 2.0 and 3.1 mg a.i./kg/day for males and females, respectively). The NOEL of 2.0 mg/kg/day will be used for deriving a drinking water guidance value. Uncertainty factors: 10 (interspecies variability); 10 (intraspecies variability) Oral Reference Dose = 2/100 = 0.02 mg/kg/day Drinking water guidance value = 0.078 ppm

Methylisothiazolinone and Methylchloroisothiazolinone

Ecological Toxicity [3]	
Aquatic Toxicity	The 96-hour LC50 for Bluegill sunfish (*Lepomis macrochirus*) is 0.28 mg/L, and the 96- hour LC50 for Rainbow trout (*Oncorhynchus mykiss*) is 0.188 mg/L. The 48-hour EC50 for *Daphnia magna* is 0.126 mg/L. The 72-hour ErC50 for algae (*Selenastrum capricornutum*) is 0.027 mg/L.
Determination of PNEC aquatic	PNECaquatic: Experimental results are available for three trophic levels. Acute E(L)C50 values are available for fish (0.188 mg/L), Daphnia (0.126 mg/L), and algae (0.027 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 0.027 mg/L for Daphnia. The PNECaquatic is 2.7 x 10^{-5} mg/L or 0.027ug/L
Screening Criteria	
Aquatic Toxicity Guidelines	No data available
Water Quality Guidelines	No data available
	No data available
PBT Assessment	
P/vP Criteria fulfilled?	No . The half-lives of CMIT and MIT is a river water-sediment system were 17.3 hours and 9.1 hours, respectively. Thus, CMIT/MIT do not meet the criteria for persistence.
B/vB criteria fulfilled?	No. The experimental BCF for CMIT is 67-114 in bluefish sunfish, and the BCF for MIT was determined to be 2.3. Thus, CMIT/MIT do not meet the criteria for bioaccumulation.
T criteria fulfilled?	No chronic toxicity data are available for CMIT/MIT; however, the acute E(L)C50 values for algae are >0.01 but <0.1 mg/L. Thus, CMIT/MIT meet the screening criteria for toxicity.
Overall conclusion	Not PBT

References

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2. EU SCCS (2009). Opinion on the mixture of 5-chloro-2-methyl-4-isothiazolin-3-one and 2-methyl-2H-isothiazol-3-one [3:1], Colipa No P56, Scientific Committee on Consumer Safety, SCCS/1238/09
3. Dow Chemicals MSDS for ROCIMA GT Dow Chemicals MSDS for ROCIMA 625 Dow Product Safety Assessment for DOW Methylisothiazoline (MIT) AntiMicrobial Products. DOW 5-Chloro-2-Methyl-2H-Isothiazol-3-one/2-Methyl-2H- Isothiazol-3- one (CMIT/MIT)-Based Antimicrobial Products

NON CRYSTALLINE SILICA

NON CRYSTALLINE SILICA	
Chemical and Physical Properties[1]	
CAS number	7631-86-9
Molecular formula	SiO_2
Product name	
Molecular weight	60.1 g/mol
Solubility in water	Insoluble
pH	Not applicable
Melting point	1710^{o}C
Boiling point	2230 ^{o}C
Colour/Form	Amorphous powder
Vapour pressure	NA
Henrys law constant	NA
Explosive potential	NA
Flammability potential	NA
Overview	Non crystalline silica (silica gel/amorphous silica) is silicon dioxide, an inorganic compound which is ubiquitous in the environment. Amorphous silica is incorporated in a variety of food products as anti-caking agent and as an excipient in pharmaceuticals.
Environmental Fate[2,3]	
Soil/Water/Air	Silicon oxides are the most abundant compounds in the earth's crust mass. Synthetic amorphous silica and silicates are released into the environment are expected to be distributed mainly into soils and sediments, weakly into water and probably not at all in the air due to their physico-chemical properties, particularly low water solubility and very low vapour pressure. Synthetic amorphous silica and silicates released into the environment are expected to combine indistinguishably with the soil or sediment due to their similarity with inorganic soil/sediment matter and will be subjected to natural processes under environmental conditions (cation exchange, dissolution, sedimentation). Biodegradation is not applicable to these inorganic substances. The bioavailable form of synthetic amorphous silica and silicates is the dissolved form which exists exclusively as monosilicic [Si(OH)4] acid under environmental pH. In analogy to the general chemical reaction of weak acids and salts of weak acids with water, the water-soluble fraction of silica acts as a weak acid and, therefore, will tend to lower the pH value, while that of a silicate acts as a base tending to bind protons and, thus, raise the pH value by forming hydroxyl ions. But pH shifts which are measurable at high loadings under laboratory conditions are not expected to occur from the anthropogenic deposition in the aquatic environment of synthetic amorphous silicas due to low aquatic releases and sufficient natural buffer capacities. Finally, these materials are supposed to combine indistinguishably with the soil layer or sediment due to their chemical similarity with inorganic soil matter. Dissolved silica can be actively assimilated by some marine and terrestrial organisms as normal natural processes mainly related to structural function.
Human Health Toxicity Summary [2,3]	
Chronic Repeated Dose Toxicity	Fischer rats and B6C3F1 mice were administered SAS in the diet at levels of 0, 1.25, 2.5, and 5% for 102 weeks. The animals were in good condition throughout and showed high survival. The tumour responses in all organs of SAS-fed rats and mice were not statistically significantly different from the controls. The NOAEL for rats and mice were 2,500 and 6,500 mg/kg/day, respectively.
Carcinogenicity	No data available.
Mutagenicity/Genotoxicity	There is no evidence for synthetic amorphous silica to induce mutations either *in vitro* or *in vivo* using standard genotoxicity tests.
Reproductive Toxicity	An early limited one-generation study on rats gave no evidence of adverse effects on reproduction performance at 500 mg/kg/day, the highest dose tested (NOAEL). But the reliability is poor due to the small group size of animals.

NON CRYSTALLINE SILICA

Developmental Toxicity/Teratogenicity	SAS was examined for embryotoxic and developmental effects during the gestation phase in various animals species, rat, mouse, rabbit and hamster, at oral doses up to 1,600 mg/kg/day. There were no significant signs of maternal or embryotoxic/developmental toxic effects in any species tested. The number of abnormalities seen in either soft or skeletal tissues of the test groups did not differ from the frequencies occurring spontaneously in the control animals.
Acute Toxicity	The acute oral administration of various forms of SAS (aqueous suspension or gel) failed to produce signs of toxicity or deaths in treated animals with LD50 values greater than the top doses applied, either by gavage: >3100 to >20000 mg/kg in mice and rats; or in the diet for 24 hours.
Irritation	Synthetic amorphous silicas are not irritating to the skin of rabbits exposed to 0.19 g (one case) or 0.5 g of dry or moistened test item under occlusive conditions for 4 or 24 hours. All products tested as a powder (0.1 g) have shown no or only weak and transient irritating effects on the conjunctivae of the eyes of rabbits with the iris and cornea not affected at all.
Sensitisation	No experimental data are available on the synthetic amorphous silicas. Medical surveillance records on workers gave no evidence of skin sensitization over decades of practical experience.
Key Study/Critical Effect for Screening Criteria	The lowest NOAEL from the two-year dietary study was 2,500 mg/kg/day for rats. Uncertainty factors: 10 (interspecies variability); 10 (intraspecies variability)
Ecological Toxicity [2,3]	
Aquatic Toxicity	Studies on fish, Daphnia and algae using excess loadings of SAS or NAS showed no acute toxicity, although physical effects on Daphnia were observed in tests using unfiltered test medium. Test results, based on loading rates, are as follows: 96hr-LL0 *(Brachydanio rerio)* is 10,000 mg/L for SAS and NAS; 24hr-EL50 *(Daphnia magna)* >10,000 mg/L for SAS; 72hr-NOEL *(Scenedesmus subspicatus)* is 10,000 mg/L for NAS. There are no chronic aquatic toxicity data, but due to the known inherent physico-chemical properties, absence of acute toxic effects as well as the ubiquitous presence of silica/silicates in the environment, there is no evidence of harmful long-term effects arising from exposure to synthetic amorphous silica/silicates.
Determination of $PNEC_{aquatic}$	Not applicable
Screening Criteria	
Aquatic Toxicity Guidelines	No data available
Water Quality Guidelines	No data available
Occupational Exposure Limits	No data available
PBT Assessment	
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. Chronic toxicity data not available. Acute data >0.1 mg/L in fish, invertebrates and algae, hence does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT

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2. IUCLID (2004) IUCLID Data Set for Synthetic Amorphous Silica and Silicates: Silicon Dioxide (CAS No. 7631-86-9, 112945-52-5, 112926-00-8; Silicic Acid, Aluminum Sodium Salt (CAS No. 1344-00-9); Silicic Acid, Calcium Salt (CAS No. 1344-95-2), UNEP Publications.
3. OECD-SIDS (2004) Screening Information Dataset (SIDS) Initial Assessment Report for Synthetic Amorphous Silica and Silicates: Silicon Dioxide (CAS No. 7631-86-9, 112945-52-5, 112926-00-8; Silicic Acid, Aluminum Sodium Salt (CAS No. 1344-00-9); Silicic Acid, Calcium Salt (CAS No. 1344-95-2), UNEP Publications.

Polyethylene glycol monohexyl ether

Polyethylene glycol monohexyl ether	
Chemical and Physical Properties[1,2]	
CAS number	31726-34-8
Molecular formula	(C2-H4-O)mult-C6-H14-O
Molecular weight	146.228 g/mol
Solubility in water	Soluble in water
pH	9-11
Melting point	5C
Boiling	High boiling points
Colour/Form	Clear yellow liquid with alcohol odour
Vapour pressure	Low vapour pressure
Henrys law constant	Low henrys law constant
Explosive potential	No information available
Flammability potential	Thermal decomposition can lead to release of irritating gases and vapours
Overview	Polyethylene glycol monohexyl ether (also known as poly(oxy-1,2-ethanediyl), α-hexyl-ω-hydroxy, hexan-1-ol, ethoxylated; and hexyl poly[oxyethylene] ether). The chemical is an Ethoxylated Alcohol (EA), a major class of non-ionic surfactants, used in oilfield applications, as solvents in cleaning fluids; in the manufacture of paper products; in adhesives and binding agents; in paints, lacquers, and varnishes; in surface treatments; in cosmetics; in nonagricultural pesticides and preservatives; in construction materials; in pharmaceuticals; as corrosion inhibitors; as antifreezing agents; in aerosol propellants; and in lubricants. Limited data is available for Polyethylene glycol monohexyl ether. Information on Alcohol Ethoxylates from the HERA report (2009) has been included in this toxicity profile.
Environmental Fate[2]	
Soil/Water/Air	EAs undergo rapid primary and biodegradation under both laboratory and field conditions. In surface water, sediment, and soil aerobic and anaerobic biodegradation will occur. In addition, EA may be taken up by plants or animals living in the surface water or soil The proposed half-lives in river water at 12C range from 4 to 24 hours (based on experimental data). EAs are not bioaccumulative, based on a log Kow value greater than 3, and a maximum BCF value of under 800. EAs are rather water soluble and the vapour pressures of EAs are relatively low, the Henry's law constants of EAs can be expected to be very low. As a result, volatilisation of surfactants can be expected to be negligible. Further work reported by Environment Canada and Health Canada (2006) has established that the degree of bioaccumulation expected from EA is well below the Canadian bioconcentration criterion of 5000. The sixteen measured BCF values for 15 EA homologues showed the lack of a linear relationship between alkyl or ethoxylate chain length and BCF, with the highest measured BCF value being under 800. Environment Canada (2006) concluded that it is evident that the EA metabolism rates prevent any significant accumulation. The data indicated that there may be an optimal structural combination of ethoxylate and alkyl chain lengths, at or around C14EO7, where BCF is maximized, but even the measured BCF for this chemical is well below the criterion of 5000. Thus Environment Canada (2006) concluded that ethoxylated aliphatic alcohols are not bioaccumulative.
Human Health Toxicity Summary[2]	
Chronic Repeated Dose Toxicity	In two chronic long-term toxicity studies which also investigated the carcinogenic potential of EAs, no adverse effects were observed up to a dose level of 50 mg/kg/day. In several dermal and oral subchronic studies over 90 days the range of NOELs/NOAELs was 50 to 700 mg/kg/day. Most of the 90-day oral feeding studies were in many respects similar to OECD test method 407. Two studies, one dermal and one oral repeated dose studies were conducted in compliance with GLP regulations. In the oral GLP-compliant study with C14-

Polyethylene glycol monohexyl ether

	15AE7, the NOEL was established at the 50 mg/kg bw/d exposure level. However, the same product was tested in a non-GLP 90-day oral feeding study and the NOAEL was determined to be at the highest exposure level of 700 mg/kg bw/d. C14-15AE7 was also examined in two 2-year feeding studies. Dose related body weight depressions in females in the upper two treatment levels were seen. At termination, elevated organ-to-body weight ratios were noted in the liver, kidney and heart. No effects have been observed on the organs of the reproductive system. Moreover, no treatment-related histopathology and no increase in tumour incidence were reported. It was concluded that the NOAEL should be established at the 0.5% level which converts to a dose of about 190 mg/kg bw/d for female rats. In the other long term study dose related body-weight depression were observed in females in the upper two treatment levels (*i.e.*, 100 and 250 mg/kg bw/d). Based on these findings, the NOAEL was established at the 50 mg/kg/d exposure level. In a 2-year feeding study with C12-14AE6.5 the NOAEL was established to be 50 mg/kg bw/d. At the higher dose levels (*i.e.*, 250 and 500 mg/kg bw/d) reduced food consumption and body weight gain was observed. At study termination, elevated organ-to-body weight ratios were noted for the liver, kidney and brain in females at the 250 and 500 mg/kg bw/d dose levels. These differences were not accompanied by histological changes in the organs examined. This study was not indicated to be GLP or OECD compliant but should be regarded as suitable as the study was conducted following the principles and procedures of the OECD guideline. A number of different alcohol ethoxylates with different structural characteristics were evaluated (*e.g.*, carbon chains raging in length from C9 to C14-16 and ethoxy unit length from 3 to 20). Despite differences in protocols and study periods the overall toxicological response was qualitatively and quantitatively similar and a contribution of structural characteristics to toxicity could not be established. No clear trends in the toxicity after repeated exposure with structural components of the test material could therefore be determined. Dermal treatment of 10 rats per sex per group for 90-days with 1%, 10% and 25% C9-11AE6 did not result in any significant compound related effects (Gingell and Lu, 1991). In-life observations included clinical observations for e.g., skin irritation, body weights, urine and blood collection and analysis. At necropsy organs and tissues collected were preserved in buffered formalin and histopathologically examined. Scores for signs of irritation at the application site throughout the study were zero but at 10% and 25% dry and flaky skin was noted. Relative kidney weights were increased in both sexes at the 25% treatment level, but no histological lesions could be determined. As a result of the observation of the increases in relative kidney weight, the NOAEL was established at the 10% level. This exposure level reflects a dose of about 80 mg/kg bw/d. This study followed the principles of the OECD procedure 411 and was GLP compliant. When given by gavage the most prominent finding was local irritation in the gastrointestinal tract. In repeated dose feeding studies the liver was the most prominent target organ. EAs induced increased relative liver weights and in some cases liver hypertrophy. This effect could however be related to an induction of liver metabolism and would normally considered an adaptive rather than an adverse effect. The NOAEL in the chronic toxicity studies is based on reduced body weight gain and increased relative organ weights only. The NOAEL of 50 mg/kg bw/d that is taken forward to the risk characterisation is based on the lowest NOAEL in a chronic oral feeding study in rats which was equal to the lowest NOAELs in subchronic feeding studies in rats.
Carcinogenicity	The carcinogenic potential of C14-15AE7 in rats has been evaluated in a one- to two-year oral feeding study (Procter and Gamble Ltd., 1979). C14-15AE7 was administered at dietary levels of 0, 0.1, 0.5 and 1% to four groups of Charles River rats (*i.e.*, 65 of each sex) for a period of one or two years. Fifteen males and females from the control and the 0.5% dose group, 15 males and 14 females from the 0.1% dose group, and 14 males and 15 females from the 1% dose group were sacrificed after an interim of 1 year exposure. The remaining animals were treated for the full 2-year period. Administration of C14-15AE7 for a period of 1 or 2 years did not produce any compound related changes in general behaviour and appearance. The survival rate of the test animals was comparable if not better than the controls. Body weights of females fed with 0.5% C14-15AE7 and males and females fed with 1% C14-15AE7 had significantly lower weight gains than the control. At necropsy, no compound related effects were observed in organ to body weight determinations. In conclusion, there was no evidence to indicate that treatment related changes of a carcinogenic nature were produced in rats by repeated ingestion of 0.1, 0.5 and 1% C14-15AE7.

Polyethylene glycol monohexyl ether

	No carcinogenic effects were observed in a two-year study in which 100 Sprague-Dawley rats were fed with C12-13AE6.5 containing diet at doses up to 1% (*i.e.*, 500 mg/kg bw/d) (Exxon; Talmage, 1994). Reduced food consumption was noted at the higher dose levels (*i.e.*, 0.5 and 1% for females and 1% for males), resulting in a lower body weight gain compared to the control group. No treatment-related histopathology was found and no increase in tumour incidence was observed. Thus, on the basis of this study, C12-13AE6.5 is not considered to be carcinogenic No treatment-related lesions were observed when C12-13AE6.5 was applied to the backs of ICR Swiss mice three times a week at 0, 0.2, 1.0 or 5.0% for 18 month (Shell Chemicals Ltd., 2002; Talmage, 1994). On the basis of the information presented it can be concluded that alcohol ethoxylates are not carcinogenic.
Mutagenicity/Genotoxicity	In all available *in vitro* and *in vivo* genotoxicity assays, there was no indication of genetic toxicity of broad range of structurally different alcohol ethoxylates.
Reproductive/developmental Toxicity	In a two-generation study conducted in Charles River CD rats, the reproductive toxicity and developmental effects of C14-15AE7 were evaluated at dietary levels of 0.05%, 0.1% and 0.5% (*i.e.*, about 25, 50 and 250 mg/kg bw/d). No compound related differences were seen between control and treated rates with restpect to fertility, gestation or viablilty indices. No treatment-related changes in behaviour or appearance were observed in the parental rats or pups throughout the study. The reproductive toxicity and developmental effects of C12AE6 was evaluated in a feeding study using a similar experimental design as described above (Little, 1977; Shell Chemicals Ltd., 2002; Talmage, 1994). Rats were exposed in a two-generation study to the compound at dose levels of 25, 50 or 250 mg/kg bw/d. No treatment related effects in the parents or pups on general behaviour, appearance or survival were observed. Fertility of treated groups was comparable with the controls. The presented information indicates that the investigated EAs did not cause reproductive toxicity when applied orally or dermally.
Acute Toxicity	EAs are of low oral, dermal and inhalation toxicity. Alcohol ethoxylates have been shown to have a low to moderate order of acute oral toxicity in the rat with LD50 values ranging between 0.6 to more than 10 g/kg. The structure of the test compound influenced acute toxicity determined by the relative number of ethoxy units, whereas, carbon chain length was not correlated with the acute oral toxicity. The degree of ethoxylation of the EA appeared to be the only factor found to be of relevance in acute oral toxicity with the compounds with ethoxylate chains between 5 and 14 being more toxic by oral consumption than those with less than 4 or more than 21 ethoxy units. Clinical findings observed in the test animals after treatment were indicative of gastrointestinal irritation such as ulcerations of the stomach, pilo-erection, diarrhoea and lethargy and may be linked with administration of a bolus dose, in particular in cases where the test item was administered undiluted. There is further an apparent sex difference for a group ethoxylates with LD50 values below 2,000 mg/kg, with females being more susceptible to the acute oral toxicity than males. It should be noted that there is unpublished information suggesting that this is not a sex specific phenomenon, but an effect related to body weight; lighter animals being more susceptible than heavier animals. Alcohol ethoxylates are considered to be of low acute inhalation toxicity to rats with LC50 values exceeding the saturated vapour concentration in air. Acute toxic thresholds were reached only when animals were exposed to the undiluted test chemical in form of a respirable mist or aerosol. Alcohol ethoxylates were shown to have a low order of acute dermal toxicity in the rat and rabbit with LD50 values typically greater than the maximum applied dose, ranging from greater than 0.8 to greater than 5 g/kg in rats. LD50 values in rabbits were greater than 2 g/kg but less than 5 g/kg. There was no relationship between compound structure and dermal toxicity.
Irritation	High quality studies investigating the skin and eye irritation potential of alcohol ethoxylates have shown that the use of these compounds in household cleaning products is of low concern. When tested undiluted EAs were found to be slightly too severely irritating to skin in rabbits and rats and mildly to severely irritating to the rabbit eye. However, if the skin or eye irritation potential was investigated at in-use concentrations, EAs were only mildly

Polyethylene glycol monohexyl ether

	irritating to skin and eyes.
Sensitisation	EAs are not considered to be skin sensitizers.
Key Study/Critical Effect for Screening Criteria	EAs of different structures with regard to the length of the alkyl chain and the degree of ethoxylation were evaluated in oral and dermal repeated dose toxicity studies. The lowest NOAEL of the EAs for systemic toxicity was 50mg/kg/day in a 2-year oral feeding study in rats. Effects observed at the LOAEL were related to significantly elevated organ-to-body weight ratios for liver, kidney and heart. No adverse histopathological changes were observed at the LOAEL. Uncertainty factors: 10 (interspecicies variability); 10 (intraspecies variability) Oral RfD = 50/100 = 0.5 mg/kg/day Drinking water guidance value = 1.95 ppm
Ecological Toxicity [2]	
Aquatic Toxicity	The Danish EPA (2001) found that the acute toxicity of EA to fish varies, with LC50 values from 0.4 mg/l to more than 100 mg/l for linear EA and from 0.25 mg/l to 40 mg/l for branched EA. The Danish EPA (2001) found that the acute toxicity of EA to invertebrates varies, with EC50 values from 0.1 mg/l to more than 100 mg/l for linear EA and from 0.5 mg/l to 50 mg/l for branched EA. The Danish EPA (2001) found that the acute toxicity of linear and several branched EA to algae was in the same general range, with EC50 values from 0.05 to 50 mg/l.
Determination of PNEC aquatic	The chronic *Daphnia magna* QSAR developed by Boeije et al (2006) can be used, with an appropriate application factor, to determine the PNEC for each EA homologue. Of the three trophic level QSARs discussed here, the chronic *Daphnia magna* QSAR is the most robust. The algal QSAR work by Wind and Belanger (2006) has shown that EA toxicity to algae, daphnia, and fish are of similar magnitude, while the chronic EA toxicity data given in Table 4.33 of the HERA report indicates that *Daphnia magna* are among the most sensitive invertebrates. An application factor of 10 was applied to the lowest of three chronic test values, in order to generate the PNEC. Table 4.38 in the HERA report presents the PNEC values determined for each EA homologue using both the chronic Daphnia magna QSAR with an application factor of 10. The PNECs for the lower chained alcohol ethoxylates (C6-C10) range from 0.118-11.9 mg/L, the mean being 2.63 mg/L.
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	No data found
PBT Assessment[1,2]	
P/vP Criteria fulfilled?	EAs are readily biodegradable and as such not persistent in the environment.
B/vB criteria fulfilled?	Based on a log Kow value greater than 3, and a maximum BCF value of under 800. EAs are not bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of EAs are > 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).

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POTASSIUM CHLORIDE

POTASSIUM CHLORIDE	
Chemical and Physical Properties[1,2,3,8,9]	
CAS number	7447-40-7
Molecular formula	KCl
Product name	--
Molecular weight	74.55 g/mol
Solubility in water	34.20 at 20^{o}C
Melting point	771.00 ^{o}C
Boiling point	1500^{o}C
Colour/Form	White crystals or crystalline powder
Vapour pressure	No data found
Henrys law constant	No data found
Explosive potential	Not explosive
Flammability potential	Not flammable
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.
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 20^{0} 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

POTASSIUM CHLORIDE

	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/Developmental Toxicity	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.
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]	
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* were660 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).
Determination of PNEC $_{aquatic}$	PNECaquatic: Experimental results are available for three trophic levels. Acute E(L)C50 values are available for fish (720 mg/L), Daphnia (177 mg/L), and algae (>100 mg/L). Although chronic studies has been conducted on fish and invertebrates, these tests are not valid for deriving PNECs (ECHA REACH database). On the basis of the short-term results from three trophic levels, an assessment factor of 1,000 has been applied to the lowest reported effect concentration of 100 mg/L for algae. The PNECaquatic is determined to be 0.1 mg/L.
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure	No data found

POTASSIUM CHLORIDE

Limits	
PBT Assessment[1,8,9]	
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?	No chronic toxicity data exist on potassium chloride; however, the acute EC(L)50s are >0.1 mg/L in fish, invertebrates and algae. Thus, potassium chloride does not meet the screening criteria for toxicity
Overall conclusion	Not PBT

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.

SODIUM CHLORIDE

SODIUM CHLORIDE	
Chemical and Physical Properties[1]	
CAS number	7647-14-5
Molecular formula	Na-Cl
Product name	
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
Colour/Form	light brown liquid or colourless crystals
Vapour pressure	No data found
Henrys law constant	No data found
Explosive potential	No data found
Flammability potential	No data found
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).
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	No data available.
Developmental Toxicity/Teratogenicity	No data available.
Acute Toxicity	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.
Irritation	No data available.
Sensitisation	No data available.
Key Study/Critical Effect for Screening Criteria	The Australian drinking water guideline value for sodium and chloride may apply.

SODIUM CHLORIDE

Ecological Toxicity [2,3]	
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
Determination of PNEC $_{aquatic}$	Not applicable.
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	No data found
PBT Assessment	
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?	No chronic toxicity data exist on sodium chloride; the acute EC(L)50s are expected to be >0.1 mg/L in fish, invertebrates and algae based on aquatic toxicity studies involving sodium and chloride ions. Thus, sodium chloride does not meet the screening criteria for toxicity.
Overall conclusion	Not PBT

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

Sodium Hydroxide

Sodium Hydroxide	
Chemical and Physical Properties	
CAS number	1310-73-2
Molecular formula	Na-O-H
Molecular weight	40 g/mol
Solubility in water	1.11E+06 mg/L at 20C
pKa	13
Melting point	323 C
Boiling point	1388 C
Vapour pressure	1.82X10-21 mm Hg 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, dermal, inhalation or by other routes for 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	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.
Developmental Toxicity/Teratogenicity	No valid studies were identified regarding developmental 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). Caustic dusts are irritating to the upper respiratory system. Prolonged exposure to high concentrations may cause discomfort and ulceration of nasal passages.
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.
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.

Sodium Hydroxide

Ecological Toxicity [1,2,3]	
Aquatic Toxicity	The single-species acute toxicity tests with NaOH with fish resulted in acute LC50 values and toxic / lethal concentrations ranging from 35 to 189 mg/l. The results for invertebrates are very similar, with a range of 33 to 450 mg/l. There are no data for algae and higher aquatic plant species. For chronic toxicity of NaOH only one limited study is available, with fish (guppy) Lebistes reticulatus. This study showed effects on survival, growth and reproduction of fish at long-term exposure to NaOH concentrations of 25 mg/l and higher. The available data indicate that NaOH concentrations of 20 to 40 mg/l may be acutely toxic to fish and invertebrates. A change in pH of 1-2 pH units has also been shown to result in acute lethality to fish.
Determination of PNECs	Data not available for derivation of PNECs.
Screening Criteria[4]	
Aquatic Toxicity Guidelines	No data found
Water Quality 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 not available (acute data >0.1 mg/L in fish, invertebrates and algae), 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

Urea

Urea	
Chemical and Physical Properties[1, 2, 3,4,5]	
CAS number	57-13-6
Molecular formula	CH4N2O
Molecular weight	60.06 g/mol
Solubility in water	1080 g/L at 20C
pH	7.2 (10% aqueous solution)
Melting point	133C
Boiling point	135C decomposition
Colour/Form	Crystal prismatic or powder
Vapour pressure	80 Pa (0.6 mmHg) at 20C
Henrys law constant	1.74 × 10-12 atm-m3/mol at 25°C (estimated)
Explosive potential	Not explosive
Flammability potential	Non-flammable
Overview	Urea, also known as carbamide, is an endogenous product of protein and amino acid catabolism. It can also be produced synthetically by combining ammonia, carbon monoxide, and sulfur in methanol. It is used in a variety of applications including fertilizers, animal feed, plastics, flame-proofing agents, diesel-SCR, flavouring agent in foods, and in the manufacture of consumer goods such as liquid soaps, detergents, and household cleaning products. A Tier 1 Human Health Assessment for Urea has been conducted by NICNAS which concluded that urea was identified as low concern to human health by application of expert validated rules.
Environmental Fate[2,5]	
Soil/Water/Air	Urea is nonvolatile in solid form and highly water soluble. It is not expected to volatilize from moist or dry soil surfaces or to evaporate from water based on its Henry's law constant (1.74 × 10-12 atm-m3/mol at 25°C). Urea leaches from the soil into surface and groundwater due to its weak adsorption to the soil, high water solubility, and low soil-water partition coefficient. In both soil and water, urea is hydrolyzed quickly to ammonia and carbon dioxide by urease, an extracellular enzyme that originates from microorganisms and plant roots. It biodegrades rapidly and is not expected to bioaccumulate. In semi-continuous activated sludge, urea degraded, on average, 93–98% in a 24-hour cycle (NLM, 2008a; OECD SIDS, 2008). If urea is released into the air, it is expected to be found in both the vapor and particulate phases of the ambient atmosphere. Vapor phase urea is degraded by photochemical reaction with a half-life at 9.6 hours. Particulate-phase urea may be removed from the atmosphere by wet and dry deposition (NLM, 2008a; OECD SIDS, 2008).
Human Health Toxicity Summary[1,5,6]	
Chronic Repeated Dose Toxicity	Chronic toxicity and carcinogenicity screening studies in mice and rats fed with 4500, 9000 or 45000 ppm in diet (up to about 6750 mg/kg body weight/day for mice and about 2250 mg/kg body weight/day for rats) did not uncover any treatment-related toxic syndromes in the various organs studied. Neither was any weight depression noted at terminal necropsy for animals of either sex or species at any dose levels. Thus the NOAELs were about 6750 mg/kg body weight/day for mice and about 2250 mg/kg body weight/day for rats. Repeated dose toxicity studies with rats by skin application over 4 weeks and 25 weeks were conducted using urea ointment at 10%, 20% and 40% concentrations, and no consistent treatment-related toxic effects were found. The ointments were applied on a 20 cm2 area of the back skin but, unfortunately, the quantities of urea applied were not given. Therefore no value for NOAEL by dermal route can be determined. It can be concluded, however, that the repeated dose toxicity of urea by dermal route was low.
Carcinogenicity	The International Agency for Research on Cancer (IARC), has indicated that there is "inadequate information to assess the carcinogenic" potential of urea.
Mutagenicity/Genotoxicity	Urea has been negative in several appropriately conducted bacterial mutagenicity tests. Urea caused DNA single strand breaks in mammalian cells in vitro and was clastogenic for mammalian cells in vitro and in vivo but only at concentrations much beyond the physiological range (about 50-100 higher concentrations than found in human blood).The

Urea

	mechanism of genotoxicity is probably non-specific (e.g. difference in osmotic pressure across the cell membrane).
Reproductive/developmental Toxicity	The studies cited under repeated dose toxicity did not indicate any toxic effects on the reproductive organs of mice and rats. No adequate teratogenicity/developmental toxicity studies of urea with mammals were located. According to one rat study, 50 g/kg body weight/day administered by gavage in two doses 12 hours apart for an average of 14 days did not cause external teratogenicity; the mean birthweight of the newborn was lower but the litter size greater. Injection of urea into the air sack of eggs shows that urea is toxic to the development of chick embryo. No NOAEL can be given for the reproductive/developmental toxicity of urea because appropriate studies are lacking.
Acute Toxicity	The acute toxicity by urea is well delineated by the oral route. Toxicity is low in mammals other than ruminants, especially cattle, and sheep, in which the rumen micro-organisms contain urease activity and metabolize urea to ammonia at a high rate. In mice and rats, urea is of low toxicity even by the subcutaneous and intravenous route.
Irritation	Urea causes redness and irritation of skin and eyes. A 24-hour dermal exposure study on skin penetration enhancers evaluated their potential to induce skin irritation (Lashmar et al., 1989). Male MF l h nude mice (4 weeks old) were exposed to a 10% w/v urea solution in water (n = 3) or control vehicle (1% w/w Carbopol 940 neutralized with sodium hydroxide). The test chemical was filled into a polyvinyl chloride cup and fastened to the dorsum of the animal using surgical tape and Superglue then left in contact with the skin for 24 hours. Animals were sacrificed and specimens of the exposed area and an adjacent untreated skin area were taken for histological examination. Tissues were fixed in formalin, paraffin-embedded, and stained with hematoxylin and eosin. Treated skin samples from each animal were randomly selected and microscopically examined and scored using a standard scoring system. The authors reported that urea did not cause any significant change in skin histology after the 24-hour exposure period.
Sensitisation	Repeated or prolonged contact with skin may cause dermatitis.
Key Study/Critical Effect for Screening Criteria	Chronic toxicity and carcinogenicity screening studies of urea in diet with mice and rats suggested that the NOAELs are of the order 2000-6000 mg/kg body weight/day. In a human female patient ingestion of 470 mg/kg body weight/day of urea over 5 years did not cause adverse effects. The estimated dose of low concern (EDLC) from animal data, using 100 as the uncertainty factor, would give 20-60 mg/kg body weight/day but, as presented above, clinical experience shows that more than ten times higher dose levels in human patients have resulted in little or no adverse effects, even over relatively long periods of time. A further perspective to the EDLC assessment is the endogenous rate of urea formation (excretion), up to about 500 mg/kg body weight/day. Based on human observations, the EDLC appears to be about one order of magnitude higher than the proposed EHE.
Ecological Toxicity [1,4,5]	
Aquatic Toxicity	The 48 hour $LC_{50}$of urea in golden orfe is reported to be >10000 mg/l. This can also be considered as the NOEC. The results reported by the two laboratories were identical. The effects of urea on survival, food utilization and oxygen consumption of the fresh water fish *Oreochromis mossambicus* were studied. The percentage survival of *O. mossambicus* when exposed to different concentrations of urea at 24, 48, 72 and 96 h exposures was noted and it was found that 22000 and 38000 mg/L urea concentration were sublethal and lethal, respectively. The median lethal concentration, which killed 50% of the fish during 96 h exposure, was 28000 mg L−1. Rearing the fish in increasing sublethal concentrations of urea, it was found that the feeding rate decreased from 34.341 ± 7.067 mg g live fish−1 d−1(control) to 13.921 ± 2.315 mg g live fish−1 d−1at the highest concentration of urea (22,000 mg L−1). Growth rate was drastically reduced. The consumption of oxygen in *O. mossambicus* diminished from 0.962 ± 0.208 to 0.645 ± 0.118 mg g live fish−1 h−1 when reared in the highest sublethal concentration of urea. The 96 hour $LC_{50}$of urea to *B. barna*was > 9100 mg/l. The NOEL was 4961 ppm. The 96 hour acute $LC_{50}$of urea to golden orfe fish is reported in a further study to be >6810 mg/l. No long-term toxicity data are available: a waiver is proposed for this endpoint. Urea is of inherently low toxicity to fish species: it is a normal product of protein catabolism and therefore fish have evolved effective excretion mechanisms. Additionally, exposure will be limited by the action of microorganisms and incorporation of urea into the nitrogen cycle.

Urea

	The 24 hour EC50 for urea in Daphnia was reported to be >10000 mg/l; urea is not acutely toxic to daphnids. The 24 hour LC50 values for freshwater snail eggs, juveniles and adults were reported to be 14241 mg/l, 18255 mg/l and 22998 mg/l. Following 48 hours exposure, the LC_{50}value for adults was calculated to be 13477 mg/l. In another study, the 24 hour LC_{50}values for eggs, juvenile and adult snails were reported to be 13532 mg/l, 24504 mg/l and 26024 mg/l, respectively. Following 48 hours exposure, the LC_{50}value for adults was calculated to be 21412 mg/l. It is concluded that, under normal laboratory conditions, urea displays low molluscicidal activity. The 4 hour LC50 in mosquito (*Aedes aegypti*) larvae is reported to be 60000 mg/l. No long-term toxicity data are available: a waiver is proposed on exposure grounds. Urea is of inherently low toxicity to species of aquatic invertebrates and exposure will be limited by the action of microorganisms and incorporation of urea into the nitrogen cycle. The 192 hour toxicity threshold of blue-green algae urea was 47 mg/l. To some extent urea exhibits toxic action to *Microcystis aeruginosa*. The 7 day toxicity threshold of urea to *Scenedesmus quadricauda* was >10000 mg/l. The 72 hour toxicity threshold of *Entosiphon sulcatum* to urea was 29 mg/l, and the 16 hour toxicity threshold of urea to *Pseudomonas putida* was > 10000 mg/l.
Determination of PNEC aquatic	PNEC is calculated for the most sensitive species, protozoa flagellata (Entosiphon sulcatum) where the lowest aquatic effect concentration, toxicity treshold (72 hours) of 29 mg/L, has been recorded. An assessment factor 100 is used, because NOEC-values were not available and acute L(E)C50-values were available for fish and crustaceans. PNEC = 0.29 mg/L
Screening Criteria	
Aquatic Toxicity Guidelines	No data found
Water Quality Guidelines	No data found
Occupational Exposure Limits	No data found
PBT Assessment[1,2,5]	
P/vP Criteria fulfilled?	Urea is biodegradable and as such not persistent in the environment.
B/vB criteria fulfilled?	Based on the measured log Kow of -1.59, urea is not bioaccumulative.
T criteria fulfilled?	The acute aquatic toxicity of urea is > 0.01 mg/L. Hence the substance does not fulfill the screening criteria for toxicity.
Overall conclusion	Not a PBT substance (based on screening data).

References

1. ECHA Registered Dossier, Urea, CAS Number 57-13-6
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. IPCS Inchem, Urea, CAS Number 57-13-6
4. National Industrial Chemicals Notification and Assessment Scheme (NICNAS), 2002. IMAP, Human Health Tier 1 Assessment for Urea, CAS Number 57-13-6
5. OECD (2002) SIDS Initial Assessment Profile for Urea, CAS Number 57-13-6
6. USEPA Integrated Risk Information System (IRIS), 2011, Toxicological Review of Urea, CAS Number 57-13-6

Appendix E

Naturally Occurring Radioactive Materials Risk Assessment

Appendix E Naturally Occurring Radioactive Materials Risk Assessment

E1.0 Introduction

Origin Energy (Origin) is conducting an exploration program in the Beetaloo Basin (Beetaloo) of the Northern Territory (NT). The Beetaloo exploration permits (EP98, EP117 and EP76), covers ~18,512 km^2 of pastoral lease on the Sturt Plain, which is part of the Barkly Tableland.

The exploration program is focused on evaluating the shale gas potential of the Mesoproterozoic source rocks of the Roper Group, mainly the Velkerri and Kyalla formations. Origin completed drilling three vertical wells (Kalala S-1, Amungee NW-1 and Beetaloo W-1) and one horizontal well (Amungee NW-1H) as part of the exploration campaigns in 2015 and 2016 to appraise these formations. The Amungee NW-1H well, located approximately 540 km south-east of Darwin, was drilled from the production hole section of the Amungee NW-1 vertical well within EP98. The lateral or horizontal section of Amungee NW-1H, some 1200 metres (m) in length, was drilled through the 'B Shale' of the middle Velkerri Formation.

Shale is a common fine-grained, organic-rich, sedimentary rock and contains low concentrations of radionuclides which are adsorbed onto the organic material and clay minerals present (Smith 2011). Kerogen within the shale is converted to oil and gas during burial (Pepper and Corvit 1995; Barker 1980), some of which is expelled, but a proportion remains in pore spaces (Cokar *et al.* 2010) or adsorbed onto the organic matter. Some of this gas can subsequently be produced by hydraulic fracturing the rock (Keshavarzi *et al*. 2012).

The multi-stage hydraulic fracturing of the Amungee NW-1H lateral section was carried out between 25 August 2016 to 08 September 2016. A total of 11 hydraulic fracturing stages were completed using a total of 10.64 megalitres (ML) of water (Origin 2017b), which is comparable to typical volumes used for horizontal shale gas wells. This operation represents the first horizontal hydraulic fracturing operation in the Beetaloo. Typically horizontal hydraulically fractured shale gas wells requires between 7.6 and 19 ML of water, but this volume is dependent upon the length of the horizontal section of the well, depth and the number of fracturing stages (Rahm 2011). . After fracturing has been completed, the fracturing fluid invades the shale reservoir and then is returned to the surface through the wellbore (Anderson *et al.* 2010; Gregory *et al.* 2011)—this is referred to as 'flowback water' or 'flowback'. Generally, 10% to 40% of the injected fracturing fluid is generally recovered to the surface within 4 weeks (Zhang *et al.* 2015).

As fracturing fluid passes through the shale, it has the potential to dissolve substances naturally present in the rock. Consequently, flowback water can be highly saline, contain dissolved and suspended solids, heavy metals and naturally occurring radioactive material (NORM) of varying concentrations (Edmiston et al. 2011; Warner *et al.* 2013). High concentrations of NORM such as uranium (U), thorium (Th) and radium (Ra) can be found in many sedimentary shale formations (Walter et al. 2012). Radioactivity shale deposits is due to the presence of natural radionuclides (unstable atoms). Radioactivity (i.e. number of particles emitted per second) of a source is measured in units of Becquerel (Bq). These radionuclides are derived from natural radioactive decay of primarily U-238 (^{238}U), Th-232 (^{232}Th) and potassium K-40 (^{40}K) (Jodłowski *et al.* 2017). The number denotes the atomic weight of the isotope of that element.

The process of radioactive decay generates radiation, which relates to the energy or particles that are released. From a radiation protection point of view, the most important radionuclides produced from the radioactive decay of ^{238}U are radium Ra-226 (^{226}Ra), radon Rn-222 (^{222}Rn), lead Pb-210 (^{210}Pb) and polonium Po-210 (^{210}Po). The amount of time needed for half of each of these radionuclides to undergo decay (half-life, $t_{½}$) are 1,600 years, 3.8 days, 22.2 years and 138.4 days, respectively (Jodłowski *et al.* 2017). The most important radionuclides generated from the radioactive decay of ^{232}Th are Ra-228 (^{228}Ra), Th-228 (^{228}Th), Ra-224 (^{224}Ra) and Rn-220 (^{220}Ra), with half-lives of 5.75 years, 1.91 years, 3.6 days and 56 seconds, respectively (Jodłowski *et al*. 2017). The radioactive isotope ^{40}K is the third major source of radiation, with a natural abundance of 0.0119% (Jodłowski *et*

al. 2017). Potassium occurs in significant quantities in the Earth's upper continental crust, with a concentration of 2.80% (Taylor and McLennan 1995).

The total amount of radioactivity attributable to the radioactive decay of alpha-emitting radionuclides (e.g. ^{238}U ^{226}Ra and ^{210}Po) and beta-emitting radionuclides (e.g. ^{40}K, ^{228}Ra and ^{210}Pb) is commonly used as a screening method (gross alpha and beta activities, respectively) for drinking water and environmental matrices of potential presence of NORM.

The most abundant radionuclides in flowback water are typically radium-226 and radium-228 (Abdeen and Khalil 1995; Barbot *et al.* 2013). ^{40}K is also commonly found in flowback water (UK Environmental Agency 2011). Both radium and potassium are relatively more water-soluble than uranium and thorium isotopes and, thus, are more likely to be released or dissolved into adjacent pore water and fracturing fluid (Smith 1992, Abdeen and Khalil 1995) and can then flow back to the surface after hydraulic fracturing (Edmiston *et al.* 2011). Equally, Rn is commonly found in groundwater (e.g. Mullinger *et al.* 2009). The relatively short half-life of Rn (3.8 days to 56 seconds) generally explains why it is often not routinely analysed in flowback water.

Radionuclides are a source of ionising radiation, which is radiation with enough energy to strip or remove electrons from an atom resulting in the formation of a charged ion (negative or positive). Ionising radiation with enough energy cause chemical changes by breaking chemical bonds, and this effect has the potential to cause damage to living tissue. However, ionising radiation is common in the environment. The largest source of radiation exposure comes from external exposure to natural radioactivity in rocks and soil (terrestrial radiation) and inhalation of naturally occurring radon (Rn) gas that seeps from the ground into all buildings (Australian Radiation Protection and Nuclear Safety Agency (ARPANSA) 2015)). Other significant contributions occur from food and water, medical exposure and cosmic rays (ARPANSA 2015, World Health Organisation 2012).

The amount of background radiation present depends on many factors, such as the type of soil and rock present and diet. For example, natural radon exposure is particularly high in Cornwall, England, due to the area being largely underlain by granitic rocks that are relatively rich in uranium and radium (UK Health Protection Agency 2012). Exposure to radiation can thus be highly variable. The Sievert (Sv) is used to evaluate the biological impact of radiation on a living organism exposed to radioactivity. In Australia, the average person is exposed to 1.5 mSv of radiation each year from natural sources (ARPANSA 2015). This is about the same amount of radiation received from 75 chest X-rays. When radiation doses are high (>500 mSv), harmful cellular damage occur resulting in acute or deterministic effects (cell is killed and unviable) (ARPANSA 2015). At lower doses (up to 10 mSv), there is no direct evidence of human health effects from ionising radiation (ARPANSA 2015).

As part of a broader Human Health and Environmental Risk Assessment of hydraulic fracturing conducted for the Beetaloo shale gas project, Origin commissioned AECOM Australia Pty Ltd (AECOM) to assess the radiation risk associated with NORM in flowback water based on the data acquired from the hydraulic fracturing operations at Amungee NW-1H. The methodology and outcomes of this NORM assessment is the subject of this report.

E2.0 Legislative Context

E2.1 International

The recognised approach to managing NORM is typically based on published guidance and standards from the International Atomic Energy Agency (IAEA). The IAEA (2004) framework is based on a graded approach to regulation, which is dependent upon the actual risk and includes cut-off concentrations for radioactivity in materials, above which investigation of the material is required.

The following definitions are provided in the IAEA safety glossary (IAEA 2007) for materials to be classified as radioactive and, therefore, potentially subject to regulation:

- A material is defined as 'radioactive' when it is 'designated in national law or by a regulatory body as being subject to regulatory control because of its radioactivity'
- NORM is defined as material (irrespective of whether it is processed or not) that contains no significant amounts of radionuclides other than naturally occurring radionuclides.

- For naturally occurring radioactive material, moderate quantities of materials containing natural uranium (U_{nat}) and thorium (Th_{nat}) greater than 1 Becquerel per gram (Bq/g) are defined as radioactive material.

Once a material containing naturally occurring radioactivity exceeds the trigger level of 1 Bq/g, an assessment is to be undertaken to understand the actual risk and managed accordingly (IAEA 2004).

E2.2 Australian

The IAEA safety guides and standards for radiation protection is essentially adopted in Australia. The regulatory approach to NORM issues within Australia is guided by ARPANSA, which is the Australian Government's primary authority on radiation protection and nuclear safety.

All types of radioactive waste are classified, managed and disposed of in a manner consistent with the:

- Safety Guide for Classification of Radioactive Waste (ARPANSA 2010a), which provides a general scheme for classifying radioactive waste based primarily on considerations of long term safety and disposal of the waste.
- Code of practice for the near-surface disposal of radioactive waste in Australia (National Health and medical Research Council (NHMRC) 1992) (currently under revision) is applicable to bulk NORM residue disposal.
- Safety Guide for the Predisposal Management of Radioactive Waste (ARPANSA 2008a).
- National Directory for Radiation Protection (NDRP) (ARPANSA 2010b).

ARPANSA (2010b) has adopted the IAEA (2004) guidance of 1 Bq/g results in materials containing naturally occurring uranium and thorium (i.e. U_{nat} and Th_{nat}, respectively) being classified as 'radioactive' and therefore subject to regulation. The 1 Bq/g activity concentrations also apply to all of the naturally occurring radionuclide decay products.

Australian states and territories are ultimately responsible for developing and implementing regulations for radiation protection and monitoring and managing the use, transport and disposal of NORM. These regulations are generally based on ARPANSA's national codes of practice and guidance documents and cover similar aspects (e.g. exemption limits on the activity concentration of radioactive material to be regulated), but focus on the particular issues present in that jurisdiction.

E2.2.1 Northern Territory

Legislation which aim to protect the health and safety of people in the Northern Territory (NT) and their environment from the harmful effects of radiation include:

- Radiation Protection Act (the Act).
- Radiation Protection Regulations (the Regulations).

The Act applies to the manufacture, sale, acquisition, possession, use, storage, transport and disposal of a radiation source but can include any activity that is connected with radiation practices. Under the Act, a radioactive material is defined as material that:

- spontaneously emits ionising radiation as a consequence of nuclear transformations; and
- has or exceeds the activity or activity concentration prescribed by the Regulations.

A subordinate statutory instrument of the Act is the Regulations, which implement the Act by prescribing matters in relation to the content of radiation protection plans, the monitoring and recording of personal radiation exposure on mining sites, offences, infringement notices for infringement offences, and to fees payable under the Act.

Under the Regulations, the activity or activity concentration for:

- A radioactive material is the activity or activity concentration in the NDRP, Schedule 4, for the material; or

- Radioactive material that is a mixture of radioactive materials is the activity or activity concentration in the NDRP, Schedule 4, for each of the materials.

In the case of a mixture of radioactive materials, the mixture is defined as exempt (i.e. the Act does not apply) if:

- the sum of the fractions obtained by dividing the activity of each material present by the appropriate activity value from the NDRP, Schedule 4, does not exceed 1; or
- the sum of the fractions obtained by dividing the activity concentration of each material present by the appropriate activity concentration value from the NDRP, Schedule 4, does not exceed 1.

The dose limits for carrying out an activity relating to a radiation source that may result, whether or not intentionally, in exposing a person, animal or thing to radiation are the dose limits in the National Standard for Limiting Occupational Exposure to Ionizing Radiation (NOHSC:1013(1995)), Schedule 1. These occupational dose limits are the same as those provided in the Recommendations for Limiting Exposure to Ionizing Radiation (1995) (Guidance note [NOHSC:3022(1995)]), Schedule A, which also provides dose limits for the general public (Table E1).

Table E1 Summary of dose limits for ionizing radiation (ARPANSA 2002)

Application	Dose Limit[A]	
	Occupational	Public
Effective dose	20 mSv per year, averaged over a period of 5 consecutive calendar years[B]	1 mSv in a year[C]

Note A: The limits shall apply to the sum of the relevant doses from external exposure in the specified period and the 50-year committed dose (to age 70 years for children) from intakes in the same period.

Note B: With the further provision that the effective dose shall not exceed 50 mSv in any single year. In addition, when a pregnancy is declared by a female employee, the embryo or foetus should be afforded the same level of protection as required for members of the public.

Note C: In special circumstances, a higher value of effective dose could be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year.

E3.0 Methodology and Approach

The radiation risk associated with NORM in flowback water from the hydraulic fracturing operations conducted at Amungee NW-1H was assessed within the context of the legislative framework (see section E2.0), including:

- Evaluating the U, Th and K concentrations in shale recovered from the middle Velkerri Formation.
- Analysing Rn concentration in a gas sample collected at the wellhead, and evaluating the radiation dose due to the inhalation of radioactive decay products of radon.
- Analysing radionuclide concentrations in flowback water compared to three other shale gas basins.
- Conducting a comparative analysis on the volumes of flowback water and the concentrations of radionuclides that could be expected in flowback water if the Beetaloo shale gas project proceeds and place this in the context of the radioactive flux derived from other shale basins, local groundwater, and other industries in Australia.
- Evaluating the radiation exposure dose and health risks for general public (unauthorised access) and on-site workers associated with storage of flowback water in a typical containment facility (i.e. Flexipond) considering exposure from inhalation of airborne radionuclide (i.e. radon) and internal dose from ingestion of flowback water.
- Evaluating potential flowback water treatment options.

E3.1 U, Th and K concentrations in shale from Beetaloo

The U, Th and K concentrations in 13 shale samples recovered from B-Shale within the middle Velkerri Formation compared to:

- 'Reference' shales, including post-Archean average Australian shale (Taylor and McLennan 1985) and North American shale composite (Gromet *et al.* 1984).
- Average shale (Wedepohl 1971).
- Data from seven gas shale formations (Antrim, Bakken, Eagle Ford, Marcellus, New Albany, Utica and Woodford) in the United States (Chermak and Schreiber 2014).

E3.2 Radionuclides in flowback water

The radionuclide concentrations measured in flowback samples from Amungee NW-1H was compared to levels found in three other shale gas basins:

- Bowland Shale, UK.
- Silurian Shale, Poland.
- Marcellus Shale, USA.

Data compiled for Beetaloo was based on the information obtained from the exploration program and hydraulic fracturing operations at Amungee NW-1H. Concentrations of radionuclides in flowback water from Bowland Shale, Silurian Shale and Marcellus Shale basins were based upon the publically available values reported in the literature. All concentration data were or assumed to be for the aqueous (dissolved) rather than the adsorbed phase.

Further context to the level of radioactive flux in the flowback water from Beetaloo was assessed by comparing the expected radioactive flux if flowback from the other three shale gas formations, local groundwater and produced water from conventional oil and gas were pumped or recovered in the same quantities as flowback water from the Beetaloo assuming a large scale development scenario (5,090 ML; see Table E2). The radioactive flux under each scenario was based on the sum of gross alpha and gross beta activities ('total activity'). Where these parameters were not specifically measured, the sum of the activities of all measured radionuclides was used in calculations.

The radioactive flux is calculated as follows:

$$R_{flux} = \sum_i c_i V \qquad [1]$$

Where:

R_{flux} is the radioactive flux (Bq)

- c_i is the concentration of radionuclide j measured in flowback water (Bq/L)
- V is the volume of flowback water (5,090 ML).

The estimated number of wells and total volume of flowback water expected to be produced within each of the shale gas basins are summarised in Table E2.

Table E2 Compilation of number of wells and total volume of flowback water

Shale	No. of Wells	Total Flowback Volume (ML)[A]	Comments / Reference
Beetaloo, Australia	456	5,090[B]	If the Beetaloo shale gas project proceeds to a large scale development scenario, it would notionally involve 456 wells (Origin 2017c). The hydraulic stimulation of these wells would be staggered over ~24 years. The total volume of hydraulic fluid required for hydraulic fracturing of these wells is forecast at 5,090 ML based on the volume of fracturing fluid pumped into Amungee NW-1H (Origin 2017d).
Bowland Shale, UK	1-10	1.2-66	Shale gas development at the Bowland Basin is at its infancy and the number of wells planned for the development of the Bowland Basin is officially unclear. However, it is estimated that between 1 and 10 wells were to be drilled in 2013 (Almond *et al.* 2014). Based on the Preese Hall-1 well that has been drilled and hydraulically stimulated, the low-end estimate of the volume of flowback water for one well was 1.2 ML and the high estimate of 6.6 ML (Broderick *et al.* 2011).
Marcellus Shale, USA	8,307	75,600-245,060	As of 2015, there were 8,307 wells drilled in the Marcellus Shale (Kondash and Vengosh 2015). Total volumes of water used in hydraulic fracturing operations have been reported to range from 11 ML to more than 19 ML per well (Aminto and Olson, 2012). The New York State Department of Environmental Conservation (2011) estimates that a multi-stage fracturing operation for a well with a 1,220 m long lateral section typically involves between 8 and 13 stages, and use a total of 9.1 ML to 29.5 ML per well.
Silurian Shale, Poland	10-50	173-865	It is estimated that by 2021, 308 wells will have been developed across 110 of the 111 shale gas exploration licences granted in Poland. To date, 23 wells have been drilled, with another 47 drilling projects due for development before the end of 2013 (Almond *et al.* 2014). For this study, the minimum number of wells drilled is assumed to be 10, with a maximum of 50. Flowback volume for this basin is based on the Lubien LE-2H well that used a total of 17.3 ML of fracturing fluid for hydraulic fracturing operations.

Note A: Assumed that 100% of injected fracturing fluid is recovered.

Note B: Based on monitoring and modelling, it is conservatively estimated 30% of this volume would be recovered as flowback, but 100% has been used in the NORM assessment to derive conservative radioactive flux values.

E3.3 Comparative emissions

Further context is provided by comparing the total radioactive flux in the flowback water from Amungee NW-1H at Beetaloo compared to the radioactive flux derived from other industries in Australia, including:

- Produced water form conventional oil and gas production.
- Mining and mineral processing industries.
- Downstream processing of heavy minerals.
- Metal smelting.
- Titanium pigment production.
- Phosphate fertiliser production
- Coal and coal power generation.
- Water treatment/purification for drinking water.

Table E3 shows typical quantities and selected radioactivity concentrations in products or waste from various industries in Australia. Most of the data was derived from Cooper (2005), which was commissioned by the Radiation Health and Safety Advisory Council (RHSAC) (at the request of ARPANSA) as part of a discussion paper (RHSAC 2005) covering NORM in Australian industry processes and waste streams. The quantities and radioactivity concentrations associated with metropolitan drinking water sludge in Australia are based on the data reported in Kleinschmidt (2011).

Where radionuclide concentrations were not available for a product or waste stream from an Australian context, concentration data considered representative of 'typical operations' found in literature was used as first approximations. To this end, radionuclide concentrations in produced water from conventional oil and gas operations are derived from typical values reported by the International Atomic Energy Agency (2003).

The level of radioactive flux in the flowback water from Beetaloo was examined by comparing the expected radioactive flux if resources or waste products were generated over the same period forecasted for hydraulic fracturing operations in Beetaloo (24 years; see Table E2). Since the concentrations of NORM in products and waste streams were not all reported for the radionuclides chosen in the current assessment, the radionuclides that were most frequently reported were used in order that an internally consistent comparison could be made. As such, total radioactive flux in products and waste streams are based on the sum of ^{238}U and ^{232}Th. For flowback water, the sum of gross alpha and beta activities is used because ^{238}U and ^{232}Th were not detected. The sum of ^{226}Ra and ^{228}Ra was used for flowback water since radium is relatively more water-soluble than uranium and thorium isotopes and, thus, is considered to be a more representative of the true activity.

Table E3 Typical quantities and radioactivity concentrations in resources or waste from various industries in Australia

Industry / Type of NORM	Product / Waste	Quantity (Mt/yr)	^{238}U (Bq/kg)	^{232}Th (Bq/kg)	^{226}Ra (Bq/kg)	^{228}Ra (Bq/kg)	^{40}K (Bq/kg)
Conventional oil and gas production	Produced water	19.8[A, B]	0.0003-0.1[C]	0.0003-0.001[C]	0.002-1200[C]	0.3-180[C]	--
Mining and mineral processing	Bauxite ore	58	200	700	--	--	60
	Red mud tailings	>20	400	1400	--	--	160
	Tantalum ore	2.5	<60	<10	--	--	--
	Tantalum concentrate	0.0025	7500-75000[D]				
Sand mining	Ore	3.5	30-120	20-300			

Industry / Type of NORM	Product / Waste	Quantity (Mt/yr)	^{238}U (Bq/kg)	^{232}Th (Bq/kg)	^{226}Ra (Bq/kg)	^{228}Ra (Bq/kg)	^{40}K (Bq/kg)
and processing	Ilmenite concentrate	2	<100-600	200-2000	--	--	--
	Zircon concentrate	0.39	1000-4000	600-1200	--	--	--
	Monazite concentrate	0.08	6000-30000	40000-250000	--	--	--
	Primary tailings	30	<100	<200	--	--	--
	Secondary tailings	0.4	100-6000	1000-20000	--	--	--
	Dust from secondary separation	0.02	<100-300	<200-1500	--	--	--
Metal smelting	Iron ore	200	31	--	42	--	--
Titanium pigment production	Neutralised slurry waste	0.2	350	1200	--	--	--
	Effluent treatment solids	0.2	300-500	800-1400			
Phosphate fertiliser production	Phosphate rock	2	5-47	15-900			
	Phosphate fertiliser	4	1200-2200	10-60	100-1000		
	Phosphogypsum waste	0.25	10-20	<10	280-350		
Coal and coal power generation	Coal	119	10-50	5-50			10-500
	Fly ash and bottom ash	14	80-150	100-200	80-150	--	500-800
Water treatment / purification	Sediment/sludge waste	0.01658[E]	130-250	46-77	37-85	--	90-260

Note A: Value in gigalitres (GL).

Note B: Based on estimates for the year 2010 (Department of Environment 2014).

Note C: Based on typical values reported in International Atomic Energy Agency (2003). All values in Bq/L.

Note D: Value for U + Th.

Note E: Mean waste produced from metropolitan water treatment plants in Australia (Kleinschmidt 2011).

Note F: Based on values reported in Kleinschmidt (2011).

E.3.4 Dose from Inhalation of radon/thoron progeny

The immediate decay product (progeny) of ^{226}Ra and ^{224}Ra are ^{222}Rn (radon) and ^{220}Rn (thoron), respectively. The relevant radioactive decay products of ^{222}Rn ('radon progeny') are ^{218}Po, ^{214}Pb, bismuth-214 (^{214}Bi) and ^{214}Po, and the decay products of ^{220}Rn ('thoron progeny') are ^{216}Po, ^{212}Pb, ^{212}Bi, ^{212}Po and thallium-208 (^{208}Tl). The problems posed by ^{220}Rn are much less widespread than those posed by ^{222}Rn (Government of Western Australia 2010).

Since Rn (a gas) has a relatively short half-life (^{222}Rn and ^{220}Rn half-life are 3.8 days and 55.6 seconds respectively), the inhalation of radon gas itself does not contribute much to radiation dose since it is immediately exhaled. The main contribution to dose is thus from the inhalation of the radioactive decay products of Rn in air. Some of the inhaled radioactive decay products are retained in the soft tissue of the lung and a radiation dose is subsequently delivered to the respiratory system due to their alpha decay.

The radiation dose that may be received from the inhalation of radioactive decay products of radon in air is the potential alpha energy concentration (PAEC) (ARPANSA 2011, Government of Western Australia 2010). The PAEC provides a measure of the energy originating from the alpha decays of RDP per unit volume in air inhaled.

Potential alpha energy exposures of human receptors to radon progeny (P_{RnP}) is determined from the Rn concentration in air (ARPANSA 2011; Government of Western Australia 2010):

$$P_{RnP} = 5.56 \times 10^{-6} \times t \times F_{RnP} \times C_{Rn} \quad [1]$$

Where:

- P_{RnP} is the potential alpha energy exposure to radon progeny (mJh/m^3).
- t is the exposure time (hours). Assumed to be 2000 working hours (ARPANSA 2011).
- F_{RnP} is the equilibrium factor for radon progeny (typically 0.2 for outdoors).
- C_{Rn} is the radon gas concentration (Bq/m^3).

The effective radiation dose is then calculated by (ARPANSA 2011):

$$E_{RnD} = e_{RnD} \times P_{RnP} \quad [2]$$

where:

- E_{RnD} is the effective radiation dose from radon progeny (mSv)
- e_{RnD} is the dose conversion factor (1.4 mSv per mJh/m^3) for inhaled radon progeny or effective dose per unit exposure to radon progeny based on a breathing rate of 1.2 m^3/hr (ARPANSA 2011)
- P_{RnP} is the potential alpha energy exposure to radon progeny (mJh/m^3)

E3.5 Radiation dose and risk assessment

The effects of ionising radiation on living tissue are divided into stochastic and deterministic effects (ARPANSA 2002, Government of Australia 2010):

- Deterministic effects of radiation (cells are killed or made unviable) include radiation sickness, burns, grey cataract and foetal abnormalities.
- Stochastic (random) effects (cells are not killed but rather modified) include cancer and damage, which takes place in human cell and, if damage occurs in a cell whose function is to transmit genetic information to later generations, any resulting effects are passed on to descendants ('hereditary effects').

The objectives of radiation protection are to prevent deterministic (acute) radiation effects from occurring, and to reduce the probability that stochastic effect or risk of chronic effects to an acceptable level. The process of exposure to radiation requires (ARPANSA 2002):

- A source which emits radiation or releases radioactive materials.
- The transmission of the radiation or the translocation of the radioactive materials through the environment from the source to the exposed human receptor.
- The interaction of the radiation or radioactive materials with organs and tissues of the body.

For the current assessment, exposure of flowback water to site-workers and general public (unauthorised access) is considered to occur where external irradiation is reasonably uniform rather than targeting particular areas of the body.

It is considered that intakes of radionuclides may occur through inhalation or ingestion. The following pathways of radiation exposure are considered:

- Inhalation of radon and its radioactive decay products.
- Ingestion of radionuclides in flowback water.

The natural background level of exposure is taken to be zero because it is considered that intakes would be small and any consequent overestimation would result in conservative estimates of intake of radioactivity.

E3.5.1 Annual Total Effective Dose calculations

The method used to calculate the Total Effective Dose was based on established methods (ARPANSA 2011, Government of Western Australia 2010). The key assumptions used to calculate the Total effective dose are summarised in Table E4. The calculated Total Effective Dose is compared to the annual dose limits recommended by ARPANSA (2002) for the public (1 mSv) and occupational workers (20 mSv) to assess the potential risk associated with NORM in flowback water from the hydraulic fracturing operations conducted at Beetaloo.

The dose assessment is carried out by first calculating the intakes of radioactivity from monitoring result, and then converting to dose through published or established conversion coefficients. The annual total effective dose was calculated as follows:

$$E = E_{RnD} + \sum_{j} e_{j,ing} I_{j,ing} \qquad [3]$$

Where:

- E is the annual Total Effective Dose (mSv)
- $e_{j,ing}$ is the effective dose per unit intake by ingestion for radionuclide j (mSv/Bq)
- $I_{j,ing}$ is the intake of radionuclide j (Bq)
- E_{RnD} is the dose from inhalation exposure to radon progeny (mSV) (see section E3.4)

$$I_{j,ing} = c_j \times A_{wat} \qquad [4]$$

Where:

- c_j is the concentration of radionuclide j in flowback water (Bq/L)
- A_{wat} is the annual water consumption (L/year)

Table E4 Key assumptions and parameters used for estimating Total Effective Dose

Input Parameter	Unit	Value / Description	Comments / Reference
Pathways	--	Inhalation of radon progeny	Natural background level of exposure is taken to be zero (provides a layer of conservatism on calculations)
	--	Ingestion of flowback water	--
Radionuclide concentrations	Bq/L or Bq/kg	See section 2.1-2.5	References provided therein
Inhalation rate	m^3/hr	1.2	ARPANSA (2011)
Annual water consumption	L/year	7.3	Standard average consumption by adults is 730 L/year (i.e. 2 L/day) (WHO 2011, ARMCANZ and ANZECC 2000). However, flowback water is not used for drinking water. However, incidental ingestion may occur. As such, average consumption is set at 0.02 L/day (7.3 L/yr) or 10% of standard consumption rate, which results in a more realistic yet conservative range of risk calculations.

Input Parameter	Unit	Value / Description	Comments / Reference
Dose conversion factor for inhaled radon (^{222}Rn) progeny	mSv/ (mJh/m^3)	1.4	ARPANSA (2011)
Exposure time	hr	2000	ARPANSA (2011). Standard annual working hours for site worker
Equilibrium factor for radon progeny	--	0.2	Government of Western Australia (2010)
Effective dose per unit intake by ingestion factor	mSv/Bq	^{226}Ra 2.4×10^{-4} ^{228}Ra 6.7×10^{-4} ^{210}Po 2.4×10^{-4} / 1.2×10^{-3}	Government of Western Australia (2010). Value for ^{210}Po is for site-workers / general public. Values for Ra are for both receptors. ^{40}K occurs in a fixed ratio to stable potassium and is not considered a health risk because a constant level is maintained in the human body (WHO 2011).

E4.0 Results and Discussion

E4.1 U, Th and K concentration in source rocks

Based on the analysis of 13 drill cutting samples recovered from B-Shale within the middle Velkerri Formation at Amungee NW-1, the U, Th and K concentrations ranged from 10.2 mg/kg to 23 mg/kg, 9.9 mg/kg to 13.6 mg/kg and 1.21% to 1.54%, respectively (Table E5). These concentrations are comparable to levels found in post-Archean average Australian shale (Taylor and McLennan 1985); North American shale composite (Gromet *et al.* 1984); average shale (Wedepohl 1971); and shale gas formations in the United States (Chermak and Schreiber 2014).

The source rocks in the Proterozoic Roper Group of the Beetaloo (i.e. the Velkerri and Kyalla formations) are naturally enriched in NORM, but their absolute concentrations are not remarkably high. For example, many marine sedimentary black shale (and phosphorite) formations contain 0.01% to 0.02% uranium, and the Alum shale of Sweden contains as much as 0.5% (McKelvey and Nelson 2006).

Table E5 Comparison of U, Th and K concentrations in among shales

Shale	Uranium (mg/kg)	Thorium (mg/kg)	Potassium (%)	Reference
Beetaloo[A]	10.2-23.0	9.90-13.6	1.21-1.54	Origin (2017)[H]
PAAS[B]	3.10	14.6	3.07	Taylor and McLennan (1985)
NASC[C]	2.66	12.3	3.15	Gromet *et al.* (1984)
Average shale[D]	3.7	12	2.99	Wedepohl (1971)
USA gas shales[E]	39.9 (15.0, 203.6)[F]	12.4 (7.5, 37.0)[G]	--	Chermak and Schreiber (2014)

Note A: Based on data collected from 13 B-Shale samples, collected between 2,428 m and 2,463 m, within the middle Velkerri Formation at Amungee NW-1 (vertical hole).

Note B: Post-Archean average Australian shale is an average of 23 Australian shales of post-Archean age.

Note C: North American shale composite is based on 40 sediment samples (mainly shales).

Note D: Based on analysis of 277 samples.

Note E: Based on data from shale gas formations in the US (Antrim, Bakken, Barnett, Eagle Ford, Haynesville, Marcellus, New Albany, Utica and Woodford). Value is the geometric mean, with values in parenthesis representative of the 25th and 75th percentile values.

Note F: Based on 314 samples from Antrim, Bakken, Eagle Ford, Marcellus, New Albany and Utica shale formations.

Note G: Based on 179 samples data from Antrim, Bakken, Eagle Ford, Marcellus, New Albany, Utica and Woodford shale formations.

Note H: Data provided in 'Amungee_NW-1_ACC_XRF_Values_Final.xlsx' worksheet (C. Gamage, pers. comm., 04 August 2017).

The maximum total U, Th and K concentrations measured in drill cutting samples from Amungee NW-1 (vertical hole), including B-Shale from the middle Velkerri Formation, were 23.1 mg/kg, 44.9 mg/kg and 4.79%, respectively. The specific activity of a sample containing 1 mg/kg of U_{nat} is 0.01243 Bq/g, 1 mg/kg of Th_{nat} is 0.004109 Bq/g, and 1 mg/kg ^{40}K is 0.2593 Bq/g (Malczewski *et al.* 2004). Based on these conversion factors, the maximum total U, Th and K concentrations correspond to activity concentrations of 0.287 Bq/g, 0.184 Bq/g and 5.61 Bq/g, respectively. The calculated sum of the fractions obtained by dividing the activity concentration of each material present by the appropriate activity concentration value from Schedule 4 of the NDRP (ARPANSA 2010b) is 0.528, which does not exceed 1. Therefore, the drill cuttings from the Amungee NW-1 (vertical hole) are not considered to be radioactive materials under the Act and would be exempt from regulations.

Table E6 Calculated activity concentrations of drill cuttings from Beetaloo (Amungee NW-1 well)

Radionuclide	Activity Concentration[A] (Bq/g)	Conversion Factor[B]	Calculated Activity Concentrations[C] (Bq/g)
U_{nat}	1	1 mg/kg = 0.01243 Bq/g	0.287
Th_{nat}	1	1 mg/kg = 0.004109 Bq/g	0.184
^{40}K	100	1 mg/kg = 0.2593 Bq/g	5.60
Exemption Threshold[D]			**1**
Sum[E]			**0.528**
Classification under the Act[F]			**Exempt**

Note A: Activity concentration in Schedule 4 of the NDRP (ARPANSA 2010b).

Note B: Based on specific parent activity of a sample containing 1 mg/kg of U_{nat} is 0.01243 Bq/g, 1 mg/kg of Th_{nat} is 0.004109 Bq/g, and 1 mg/kg ^{40}K is 0.2593 Bq/g (Malczewski *et al.* 2004). ^{40}K abundance is 0.0117%.

Note C: Based on the maximum U (23.1 mg/kg), Th (44.9 mg/kg) and K (47,880 mg/kg) concentrations measured in drill cutting samples collected from Amungee NW-1 (vertical hole). Data provided in 'Amungee_NW-1_ACC_XRF_Values_Final.xlsx' worksheet (Origin 04 August 2017).

Note D: Exemption threshold for a mixture of radioactive materials under the Radiation Protection Regulations (NT).

Note E: Sum of the fractions obtained by dividing the activity concentration of each material present by the appropriate activity concentration value from Schedule 4 of the NDRP (ARPANSA 2010b).

Note F: Radiation Protection Act (NT).

E4.2 Radon and dose from inhalation of radon/thoron progeny

Based on the analysis of one gas sample collected at Amungee NW-1H, the Rn concentration was 225 Bq/m^3. This compares to an average annual Rn concentration of 39 $\pm$ 5 Bq/m^3 measured in Jabiru East (a former residential township), located within the Ranger Project Area, in the NT and is surrounded by the Kakadu National Park (Akber and Pfitzner 1994). The Australian Radiation Protection and Nuclear Safety Agency (ARPANSA) reported that the average concentration of Rn in Australian homes is about 10 Bq/m^3, based on a survey of more than 3300 homes, which is less than the global average indoor value of 40 Bq/m^3. The Rn concentration measured at Amungee NW-1H exceed the current Action Levels recommended by ARPANSA (2002) for Rn in households (200 Bq/m^3), but is well below the limits for workplaces (1,000 Bq/m^3).

Based on a radon concentration of 225 Bq/m^3, the annual effective radiation dose from the inhalation radon progeny was calculated to be 0.70 mSv for site workers. This effective dose is below the annual Australian limits for public (1 mSv) and occupational (20 mSv) exposure to ionizing radiation (ARPANSA 2002). The annual effective dose from randon progeny is expected to be <0.70 mSv for the general public (e.g. unauthorised access) because of lower exposure times.

Table E7 Dose from inhalation of radon progeny

Radon Concentration (Bq/m^3)	Effective Dose (site workers) (mSv)	Dose Limit[A]	
		Occupational	Public
225	0.70	20 mSv per year, averaged over a period of 5 consecutive calendar years[B]	1 mSv in a year[C]

Note A: The limits shall apply to the sum of the relevant doses from external exposure in the specified period and the 50-year committed dose (to age 70 years for children) from intakes in the same period.

Note B: With the further provision that the effective dose shall not exceed 50 mSv in any single year. In addition, when a pregnancy is declared by a female employee, the embryo or foetus should be afforded the same level of protection as required for members of the public.

Note C: In special circumstances, a higher value of effective dose could be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year.

E4.3 Radionuclides in flowback water

The concentrations of uranium (^{238}U) and thorium (^{228}Th, ^{230}Th and ^{232}Th) in flowback water from Amungee NW-1H (<0.026 Bq/L to 0.088 Bq/L) were minor compared to ^{226}Ra (8.74 Bq/L), ^{228}Ra (1.67 Bq/L) and ^{40}K (2.3 Bq/L) (Origin, pers. comm., 04 August 2017). The lower concentration of ^{228}Ra compared to ^{226}Ra is consistent with the low ^{232}Th/^{238}U activity ratio (0.64) in the basin lithology (see Section E4.1).

As radioactive isotopes decay by emitting alpha and beta particles, the sum of gross alpha and beta activities measured in flowback water samples are used as the primary indicators of potential NORM and total activity for comparison purposes. Since radium and potassium are typically the dominant radionuclides found in flowback water, ^{226}Ra, ^{228}Ra and ^{40}K concentrations were also used to provide further context to the levels of NORM measured in flowback water from each shale basin.

The concentrations of ^{226}Ra, ^{228}Ra and ^{40}K and total radioactivity measured in flowback water from each of shale gas basin and local groundwater are summarised in Table E8. It is recognised that while this is a relatively small data set, it does provide valuable information and context on the levels radionuclides measured in the flowback water from Amungee NW-1H. Table E8 shows that generally lower radioactivity and radionuclide concentrations have been measured in the flowback from Amungee NW-1H in the Beetaloo compared to levels reported for Silurian Shale (Baltic Basin and Lublin Trough), Marcellus Shale, and Bowland Basin. Compared to produced water from conventional oil and gas production, the concentrations of radionuclides are significantly lower in the flowback water from Amungee NW-1H.

Table E8 Concentrations of radionuclides and gross alpha and gross beta activities in flowback water

Basin	Total Activity (Bq/L)	^{226}Ra (Bq/L)	^{228}Ra (Bq/L)	^{40}K (Bq/L)	Reference
Produced water[H]	--	0.002-1200	0.3-180	--	International Atomic Energy Agency (2003)
Local Groundwater	0.06-0.73 (4)	--	--	--	Origin (2017e)
Beetaloo, Australia	<0.62[A] – 12.4 (11)[B]	8.74 (1)	1.67 (1)	2.3 (1)	Origin (2017)[G]
Bowland Shale, UK	13.1 – 247 (4)	14 – 90 (4)	--[D]	<1 – 3.5 (4)	UKEA (2011)
Marcellus Shale, USA	2.04 – 4551 (14)[C]	0.006 – 629 (34)	0.001 – 51.8 (30)	--[D]	Abualfaraj *et al*. (2016)
Silurian Shale, Poland[E]	9 – 651 (30)[F]	2 – 70 (30)	<2[A] – 36 (30)	5 – 545 (30)	Jodłowski *et al.* (2017)

Note A: Where values are below detection limits, the limit of detection was used for calculation purposes.

Note B: Values in parenthesis denote the number of samples.

Note C: Based on gross alpha activity.

Note D: Value not provided or measured.

Note E: Values from Baltic Basin and Lublin Trough (assumed to be Silurian Shale).

Note F: Based on sum of ^{226}Ra, ^{228}Ra and ^{40}K.

Note G: Data provided by Origin in 'Copy of Chemistry_Output_Table1 (003).xlsm' worksheet, 04 August 2017.

Note H: Produced water from conventional oil and gas.

If the Beetaloo shale gas project proceeds to a large scale development scenario, hydraulic fracturing operations would require a total of 5,090 ML of fracturing fluid. Based on this estimation, the predicted radioactive flux in flowback water from Beetaloo is estimated to range from 0.003-0.063 terabecquerel (TBq). These values are between two to three orders of magnitude lower than those predicted for the Marcellus, Bowland and Silurian shale basins and produced water from conventional oil and gas

(Table E9). The maximum radioactive flux from these latter four scenarios is calculated to range from 7.11 to 23.2 TBq.

Table E9 shows that the radioactive flux in the flowback water from Beetaloo is marginally higher than that predicted if local groundwater is pumped in the same quantities as forecasted for hydraulic fracturing operations in Beetaloo (5,090 ML). This increased activity from flowback water above 'background' water is much less significant when viewed relative to other shale gas basins from overseas and produced water from conventional oil and gas production.

Table E9 Radioactive fluxes for shale basins, local groundwater and produced water from conventional oil and gas

Basin	Radioactive flux (TBq)[A]
Produced water (conventional oil and gas)	0.002-7.02
Local groundwater	0.0003-0.0037
Beetaloo, Australia	0.003-0.063
Bowland Shale, UK	0.07-1.26
Marcellus Shale, USA	0.010-23.2
Silurian Shale, Poland	0.05-3.31

Note A: Based on 5,090 L of flowback and equivalent local groundwater and produced water.

E4.4 Comparative emissions

Table E10 shows estimated radioactivity flux in product and waste streams that would be generated by various industries in Australia over the next 24 years (timeframe forecasted for hydraulic fracturing operations in Beetaloo under a large scale development scenario). The estimated volume of produced water generated by conventional oil and gas production in 2010 was 19.8 gigalitres (GL) (Department of Environment 2014). This volume is considerably higher than those potentially generated (5.09 GL) by proposed hydraulic fracturing operations in Beetaloo. Even if the Beetaloo shale gas development was three times as large, the maximum volume of flowback (assuming all fracturing fluid injected is recovered) would be 15.27 GL. This hypothetical development scenario, although unlikely to occur, demonstrates that Australia not only currently generates and manages greater volumes of produced water for disposal than could be expected from hydraulic fracturing operations in the Beetaloo, but also manages greater volumes of proportionately more radioactive water.

The largest contributors to the radioactivity flux among the modelled Australian industries is bauxite mining and associated red mud tailings disposal, sand mining and processing, iron ore smelting, phosphate fertiliser production and coal and coal waste (fly ash and bottom ash). These industries would contribute >99% of the total radioactive flux generated over the same timeframe. Table E10 shows that the predicted total radioactive flux in flowback water is only minor compared to those produced and currently being managed by these established Australian industries. The radioactive flux in flowback under the modelled conditions is comparable to those found in sediment /sludge waste generated by Australian metropolitan drinking water treatment plants.

Table E10 Radioactive flux in Beetaloo flowback water and product/waste streams from various Australian industries

Industry / Type of NORM	Product / Waste	Radioactive Flux (TBq)[A]
Beetaloo, Australia	Flowback water	0.018-0.36
Conventional oil and gas production	Produced water	0.14-660
Mining and mineral processing	Bauxite ore	1250
	Red mud tailings	860

Industry / Type of NORM	Product / Waste	Radioactive Flux (TBq)[A]
	Tantalum ore	4.2
	Tantalum concentrate	0.003-0.063
Sand mining and processing	Ore	4.2-35.3
	Ilmenite concentrate	14.4-125
	Zircon concentrate	15.0-48.7
	Monazite concentrate	88.3-540
	Primary tailings	216
	Secondary tailings	10.6-250
	Dust from secondary separation	0.14-0.86
Metal smelting	Iron ore	149
Titanium pigment production	Neutralised slurry waste	7.44
	Effluent treatment solids	5.28-9.12
Phosphate fertiliser production	Phosphate rock	45.5
	Phosphate fertiliser	116-217
	Phosphogypsum waste	0.18
Coal and coal power generation	Coal	42.8-286
	Fly ash and bottom ash	60.5-118
Water treatment / purification	Sediment/sludge waste	0.070-0.13

Note A: Based on products and waste streams generated over 24 years (timeframe forecasted for hydraulic fracturing operations in Beetaloo under a large scale development scenario).

E4.5 Potential human health risks associated with flowback water

Table E11 shows that the inhalation of radon progeny was the dominant source contributing more than 96% of the Total Effective Dose, followed by ^{226}Ra (2.1%) and ^{228}Ra (1.1%). The calculated annual Total Effective Dose of 0.72 mSv, based on the available data and modelled exposure conditions (see Section E3.4 and E3.5), does not exceed the annual effective dose limits recommended by ARPANSA (2002) for the public (1 mSv) and occupational workers (20 mSv). Therefore, radiation exposure and associated health risks of NORM in flowback water from the hydraulic fracturing operations conducted at Amungee NW-1H are minimal for site-workers and general public (unauthorised access).

Table E11 Total Effective Dose due to exposure to radionuclides in flowback water from Amungee NW-1H

	^{228}Ra (mSv)	^{226}Ra (mSv)	^{226}Ra (mSv)	^{210}Po (mSv)[B]	E_{RnD} (mSV)	Annual Total Effective Dose (mSv)	
						Public	Site-worker
ARPANZA (2002)						**1**	**20[B]**
Beetaloo flowback water	0.015	0.0082	0.000015	0.000015 / 0.000073	0.70	0.72405	0.72411

Note A: Averaged over a period of 5 consecutive calendar years.

Note B: Value for ^{210}Po is for general public / site-workers.

Note C: The dose from inhalation exposure to radon progeny

Note D: In special circumstances, a higher value of effective dose could be allowed in a single year, provided that the average over 5 years does not exceed 1 mSv per year. With the further provision that the effective dose shall not exceed 50 mSv in any single year. In addition, when a pregnancy is declared by a female employee, the embryo or foetus should be afforded the same level of protection as required for members of the public.

Note E: Averaged over a period of 5 consecutive calendar years.

E4.6 Potential flowback water treatment options

Management options for flowback water include direct reuse without treatment (blending with freshwater for reuse in hydraulic fracturing operations), on-site treatment and reuse, off-site treatment and reuse, and off-site treatment and disposal. The latter method was used for hydraulic fracturing operations at Amungee NW-1H. However, if the Beetaloo shale gas project moves towards a development scenario or phase, water recycling and permanent fluid handling facilities will be constructed to manage the flowback water and produced water (Origin 2017c). Approximately 30% of the annual total water requirement is forecasted to be recycled if the Beetaloo shale gas project under a large scale development scenario (Origin 2017c).

The reuse of flowback water for subsequent hydraulic fracturing operations in the Marcellus Shale is typically the main management option (Vidic et al. 2013). The flowback water is typically stored in centralised impoundments or storage tanks prior to reuse to increase management flexibility and meet the drilling and hydraulic fracturing schedule.

Technologies available for treatment and reuse of flowback water (Boschee 2014) can be broadly categorised into two types:

- Conventional treatment, including flocculation, coagulation, sedimentation, filtration, and lime softening water treatment processes. Generally, these methods are effective in removing metals, non-dissolved constituents such as total suspended solids, oil and grease, and hardness compounds (calcium and magnesium).
- Advanced treatment technology, including reverse osmosis, ion-exchange, and thermal distillation, evaporation and/or crystallization processes. These methods are generally used to treat total dissolved solids (TDS).

Typically, conventional processes are used as a pre-treatment process to remove the non-dissolved constituents prior to the TDS treatment processes. The thermal and membrane processes generally require more energy than the conventional method (Boschee 2014).

The available data suggests that ^{226}Ra is the dominant radionuclide and is possibly an important proxy for the radioactivity and presence of NORM in Beetaloo flowback water. Treatments such as flocculation, reverse osmosis, ion exchange or lime softening tend to remove radium (and dissolved uranium) quite efficiently (ARPANSA 2008b). Chemical treatment in the form of sulfate precipitation for barium removal also results in the co-precipitation of ^{226}Ra with barium sulfate ($BaSO_4$) (Zhang *et al.* 2014).

Flowback water treatment and recovery by thermal processes is commercially practiced in a growing number of shale gas applications (Smistad 2013). To avoid scaling on heat transfer surfaces, influent flowback water is normally pre-treated to remove scale-causing ions such as iron and manganese as well as suspended solids and dissolved organics (Smistad 2013). These limitations are overcome by recent developments in water treatment technology such as the Ali-Jak, which is based on vacuum distillation methods.

For treatment processes where the volume of waste generated is much smaller than the volume of treated water (e.g. reverse osmosis and thermal distillation/evaporation) pre-concentration of radionuclides can occur and disposal of such wastes may require careful management to minimise exposures. The levels of these potential radionuclides concentrate and their impacts will be accessed as part of the Beetaloo shale gas project pre-feasibility and design phase.

E5.0 Conclusions

The radiation risk associated with NORM in flowback water based on the data acquired from the hydraulic fracturing operations at Amungee NW-1H. The volumes of flowback water and the concentrations of radionuclides that could be expected in flowback water if the Beetaloo shale gas project proceeds (under large scale development scenario) were considered in the context of other fluxes of radionuclides derived from local groundwater, other shale gas basins, and those generated and managed by various Australian industries.

The B-shale within the Velkerri Formation of the Proterozoic Roper Group of the Beetaloo Basin is naturally enriched in NORM, but their absolute concentrations are not remarkably high. Their concentrations are comparable to levels found in post-Archean average Australian shale (Taylor and McLennan 1985); North American shale composite (Gromet *et al.* 1984); average shale (Wedepohl 1971); and shale gas formations in the United States (Chermak and Schreiber 2014).

Based on a radon concentration of 225 Bq/m^3, the annual effective radiation dose from the inhalation radon progeny was calculated to be 0.70 mSv for site workers. This effective dose is below the annual Australian limits for public (1 mSv) and occupational (20 mSv) exposure to ionizing radiation (ARPANSA 2002). The annual effective dose from randon progeny is expected to be <0.70 mSv for the general public (e.g. unauthorised access) because of lower exposure times.

Generally lower radioactivity and radionuclide concentrations have been measured in the flowback from Amungee NW-1H in the Beetaloo compared to levels reported for Silurian Shale (Baltic Basin and Lublin Trough), Marcellus Shale, and Bowland Basin. Compared to produced water from conventional oil and gas production, the concentrations of radionuclides (are significantly lower in the flowback water from Amungee NW-1H.

The predicted radioactive flux in flowback water from Beetaloo is significantly lower than those predicted for the Marcellus, Bowland and Silurian shale basins and produced water from conventional oil and gas. The radioactive flux in the flowback water from Beetaloo is marginally higher than that predicted if local groundwater is pumped in the same quantities as forecasted for hydraulic fracturing operations in Beetaloo (5.090 GL). This increased activity from flowback water above 'background' water is much less significant when viewed relative to other shale gas basins from overseas and produced water from conventional oil and gas production.

The largest contributors to the radioactivity flux among the modelled Australian industries include produced water from conventional oil and gas production, bauxite mining and associated red mud tailings disposal, sand mining and processing, iron ore smelting, phosphate fertiliser production and coal and coal waste (fly ash and bottom ash). These industries would contribute >99% of the total radioactive flux generated over the next 24 years (the forecasted timeframe for hydraulic fracturing operations in Beetaloo under a large scale development scenario). The comparative analysis demonstrates that Australia not only generates and manages greater volumes of products or waste than could be expected from hydraulic fracturing operations in the Beetaloo, but also currently manages greater volumes of proportionately more radioactive materials. The radioactive flux in flowback under the modelled conditions is comparable to those found in sediment /sludge waste generated by typical Australian metropolitan drinking water treatment plants.

The inhalation of radon progeny was the dominant source that contributes more than 96% of the Total Effective Dose, followed by ^{226}Ra (2.1%) and ^{228}Ra (1.1%). The calculated annual Total Effective Dose of 0.72 mSv, based on the available data and modelled exposure conditions, does not exceed the Australian (ARPANSA 2011) annual effective dose limits of 1 mSv for general public and 20 mSv for occupational workers. Therefore, radiation exposure and associated health risks of NORM in flowback water from the hydraulic fracturing operations conducted at Amungee NW-1H are minimal for site-workers and general public (unauthorised access).

Technologies available for treatment and reuse of flowback water (Boschee 2014) can be broadly categorised into conventional treatment (e.g. flocculation, coagulation, sedimentation, filtration, and lime softening) and advanced treatment technology (e.g. reverse osmosis, ion-exchange, and thermal distillation, evaporation and/or crystallization processes). Treatments such as flocculation, reverse osmosis, ion exchange or lime softening tend to remove radium (and dissolved uranium) quite

efficiently (ARPANSA 2008b). Chemical treatment in the form of sulfate precipitation for barium removal also results in the co-precipitation of ^{226}Ra with barium sulfate ($BaSO_4$) (Zhang *et al.* 2014).

For treatment processes where the volume of waste generated is much smaller than the volume of treated water (e.g. reverse osmosis and thermal distillation/evaporation) pre-concentration of radionuclides can occur and disposal of such wastes may require careful management to minimise exposures. The levels of these potential radionuclides concentrate and their impacts will be accessed as part of the Beetaloo shale gas project pre-feasibility and design phase.

E6.0 Recommendations

AECOM recognises that the Beetaloo shale gas project is in its infancy and recommends that the following be considered as the project progresses to confirm the findings of this assessment and to increase the knowledge base on the distribution and levels of NORM in shale gas plays in the Beetaloo Basin:

- Further testing of radionuclides, including gross alpha and beta activities, ^{238}U, ^{232}Th, ^{40}K and ^{226}Ra and ^{228}Ra, in flowback water, drill cuttings from target formations and bottom solids/sediment samples collected from flowback water storage facilities:
  - Flowback water is to be sampled at selected hydraulic fractured wells to capture the initial return of the water flowed back and over the following 4 weeks on a weekly basis than monthly for a further two months to assess the temporal changes in radioactivity. All samples are to be filtered through 0.45 µm membrane to separate suspended solids and collected in laboratory suppled clean 1 L polypropylene bottles for gross alpha and beta activity analysis, two 1 L polypropylene bottles for ^{226}Ra and ^{228}Ra analysis, and two 1 L polypropylene bottles for ^{238}U, ^{232}Th, ^{40}K analysis.
  - Drill cuttings from target shale formations and other major formations such as the Cambrian Limestone Aquifer is to be sampled. All samples are to be collected in clean two 150 mL glass jars with Teflon lined lids.
  - Bottom solids/sediment samples are to be collected from flowback water storage facilities. The intention of the sampling is to obtain samples representative of the entire footprint of each storage facility. All samples are to be collected in clean two 150 mL glass jars with Teflon lined lids.
- Conduct Toxicity Characterisation Leaching Procedure (TCLP) on selected bottom solids/sediment samples collected from flowback water storage facilities. Such testing should be conducted within the context of the National Environmental Protection (Assessment of Site Contamination) Measure 1999 (ASC NEPM) as amended in 2013 (NEPC 2013). Testing is to be conducted using both acetic acid and deionised water extractions to provide information in relation to landfill disposal options and simulate leaching due to natural infiltration by meteoric water.
- Targeted and selective testing of gas samples for radon at the well head and ambient air in the vicinity of the flowback water storage facilities.
- Update the NORM assessment report based on the outcomes of the above testing program.

E7.0 References

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