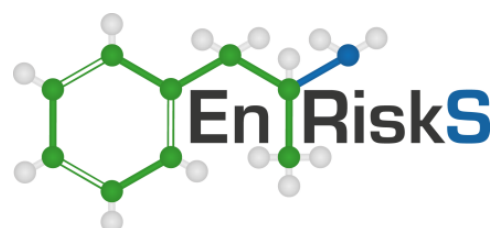




Bromelton Energy and Resource Centre: Human Health Risk Assessment

Prepared for: Cleanaway Operations Pty Ltd

17 February 2026





Document History and Status

Report Reference	CLEAN/25/BERC001
Revision	D – Final
Date	17 February 2026
Previous Revisions	A – Draft (24 September 2025) B – Final (14 November 2025) C – Final (17 December 2025)

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Glossary of Terms and Abbreviations

Term	Definition
ABS	Australian Bureau of Statistics
Acute exposure	Contact with a substance that occurs once or for only a short time (up to 14 days)
Absorption	The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs
Adverse health effect	A change in body function or cell structure that might lead to disease or health problems
ATSDR	US Agency for Toxic Substances and Disease Register
AAQ	Ambient air quality
ANZECC	Australia and New Zealand Environment and Conservation Council
Background level	An average or expected amount of a substance or material in a specific environment, or typical amounts of substances that occur naturally in an environment.
Biodegradation	Decomposition or breakdown of a substance through the action of micro-organisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).
Body burden	The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.
Carcinogen	A substance that causes cancer.
CCME	Canadian Council of Ministers of the Environment
Chronic exposure	Contact with a substance or stressor that occurs over a long time (more than one year) [compare with acute exposure and intermediate duration exposure].
CO	Carbon monoxide
CV	Calorific value
DCCEEW	Australian Department of Climate Change, Energy, the Environment and Water
DEFRA	UK Department for Environment, Food & Rural Affairs
DEH	Australian Department of Environment and Heritage (<i>acronym for the Commonwealth Department of Environment under a previous name – used here in reference to guidance published at that time. The Department acronym is now DCCEEW</i>)
Dose	The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An 'exposure dose' is how much of a substance is encountered in the environment. An 'absorbed dose' is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.
Exposure	Contact with a substance by swallowing, breathing, or touching the skin or eyes. Also includes contact with a stressor such as noise or vibration. Exposure may be short term [acute exposure], of intermediate duration, or long term [chronic exposure].
Exposure assessment	The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

Term	Definition
Exposure pathway	The route a substance takes from its source (where it began) to its endpoint (where it ends), and how people can come into contact with (or get exposed) to it. An exposure pathway has five parts: a source of contamination (such as chemical substance leakage into the subsurface); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.
Genotoxic carcinogen	These are carcinogens that have the potential to result in genetic (DNA) damage (gene mutation, gene amplification, chromosomal rearrangement). Where this occurs, the damage may be sufficient to result in the initiation of cancer at some time during a lifetime.
Guideline value	Guideline value is a concentration in soil, sediment, water, biota or air (established by relevant regulatory authorities such as the NSW Department of Environment and Conservation (DEC) or institutions such as the National Health and Medical Research Council (NHMRC), Australia and New Zealand Environment and Conservation Council (ANZECC) and World Health Organization (WHO)), that is used to identify conditions below which no adverse effects, nuisance or indirect health effects are expected. The derivation of a guideline value utilises relevant studies on animals or humans and relevant factors to account for inter and intra-species variations and uncertainty factors. Separate guidelines may be identified for protection of human health and the environment. Dependent on the source, guidelines would have different names, such as investigation level, trigger value and ambient guideline.
HI	Hazard Index
IARC	International Agency for Research on Cancer
Inhalation	The act of breathing. A hazardous substance can enter the body this way [see route of exposure].
Intermediate exposure Duration	Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].
LGA	Local Government Area
LHV	Lower heating value
LOR	Limit of Reporting
Metabolism	The conversion or breakdown of a substance from one form to another by a living organism.
mg/Nm ³	milligrams per normal cubic metre (i.e. at 273.15 degrees Kelvin and 101.325 kPa)
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
NHMRC	National Health and Medical Research Council
NO ₂	Nitrogen dioxide
NO _x	Nitrogen oxides
NSW	New South Wales
NSW EPA	NSW Environment Protection Authority
OEH	NSW Office of Environment and Heritage
OEHHA	Office of Environmental Health Hazard Assessment, California Environment Protection Agency (Cal EPA)
PAHs	Polycyclic aromatic hydrocarbons
PCBs	Polychlorinated biphenyls (some chemicals in this group are included in the dioxin-like compounds)
PCDD/PCDF	Polychlorinated dibenzo- <i>p</i> -dioxin and polychlorinated dibenzofuran (collectively known as dioxin-like compounds)

Term	Definition
PFAS	Per- or Poly-fluoroalkyl Substances – these are chemicals that are made of carbon and fluorine atoms. They are a large group of synthetic chemicals. They have been in widespread use since 1940s. Some of the individual chemicals that are part of this group and which are commonly investigated include: PFOS = Perfluorooctanesulfonic Acid PFBS = Perfluorobutanesulfonic Acid PFPeS = Perfluoropentanesulfonic Acid PFHxS = Perfluorohexanesulfonic Acid PFHpS = Perfluoroheptanesulfonic Acid PFDS = Perfluorodecane sulfonic Acid PFOA = Perfluorooctanoic Acid PFBA = Perfluorobutanoic Acid PFPeA = Perfluoropentanoic Acid PFHxA = Perfluorohexanoic Acid PFHpA = Perfluoroheptanoic Acid PFNA = Perfluorononanoic Acid PFDA = Perfluorodecanoic Acid PFUnDA = Perfluoroundecanoic Acid PFDaA = Perfluorododecanoic Acid PFTrDA = Perfluorotridecanoic Acid PFTeDA = Perfluorotetradecanoic Acid
PM	Particulate matter
PM _{2.5}	Particulate matter of aerodynamic diameter 2.5 µm and less
PM ₁₀	Particulate matter of aerodynamic diameter 10 µm and less
Point of exposure	The place where someone can come into contact with a substance present in the environment [see exposure pathway].
Population	A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).
Receptor population	People who could come into contact with hazardous substances [see exposure pathway].
Risk	The probability that something would cause injury or harm.
Route of exposure	The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].
SDA	State Development Area
SEIFA	Socio-Economic Index for Areas
SO ₂	Sulfur dioxide
TCEQ	Texas Commission on Environmental Quality
TEQ	Total equivalents (method of risk assessment applied to groups of chemicals, such as dioxin-like compounds, that act via the same mechanism which allows the concentration of each individual chemical to be converted to the “equivalent” concentration of the primary member of the group so they can be all added together and the risk posed by the mixture of the chemicals in the group to be fully assessed)
Toxicity	The degree of danger posed by a substance to human, animal or plant life.
Toxicity data	Characterisation or quantitative value estimated (by recognised authorities) for each individual chemical substance for relevant exposure pathway (inhalation, oral or dermal), with special emphasis on dose-response characteristics. The data are based on available toxicity studies relevant to humans and/or animals and relevant safety factors.

Term	Definition
Toxicological profile	An assessment that examines, summarises, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.
Toxicology	The study of the harmful effects of substances on humans or animals.
TSP	Total suspended particulates
TVOC	Total volatile organic compounds
UK	United Kingdom
US	United States
USEPA	United States Environmental Protection Agency
VOC	Volatile organic compounds
WHO	World Health Organization
$\mu\text{g}/\text{m}^3$	Micrograms per cubic metre
$\mu\text{g}/\text{Nm}^3$	Micrograms per normal cubic metre (i.e. at 273.15 degrees Kelvin and 101.325 kPa)

Executive Summary

Introduction

Environmental Risk Sciences Pty Ltd (enRiskS) has been engaged by Cleanaway Operations Pty Ltd (Cleanaway) to undertake a Human Health Risk Assessment (HHRA) for an Energy from Waste (EfW) facility in Bromelton, Queensland.

Cleanaway is an Australian waste management, recycling, and industrial services company. Cleanaway is developing an EfW facility in Queensland known as the Bromelton Energy and Resource Centre (BERC) (the Project). The Project will be located within the Special Industry Precinct of the Bromelton State Development Area (SDA), west of Beaudesert within the Scenic Rim Regional Council local government area.

The BERC has been designed to thermally treat a nominal capacity of 760,000 tonnes per annum (tpa) of waste feedstock via two 140 megawatt thermal (MWth) processing lines when operating at the design calorific value (CV) of 10.6 MJ/kg (LHV) and assuming 8,000 hours per annum for boiler availability. The waste feedstock is expected to consist of a mix of residual Municipal Solid Waste (MSW) and residual commercial and industrial waste (C&I), which is waste that would otherwise be sent to landfill. Waste feedstock processed by the BERC will be subject to a Waste Acceptance Protocol to determine eligibility and suitability for processing wastes both prior to arrival and upon arrival on-site. The BERC will also incorporate maturation and processing of incinerator bottom ash (IBA) to recover recyclable metals, with the intent to utilise the remaining ash as an aggregate in construction.

This HHRA has addressed potential impacts to community health from air emissions using a quantitative approach and potential impacts to community health from other matters (including noise, water management, stormwater management, dangerous goods storage and transport, flooding, bushfire) using a qualitative approach. This is because potential impacts to community health from emissions to air are the primary issue for an energy from waste facility.

Assessment of emissions to air

Location of receptors and types of exposure

The human health risk assessment has evaluated the potential for exposure to air emissions from the facility in a quantitative fashion. This report describes the assessment of the potential for people to be exposed to contaminants in air via:

- Inhalation of air containing the emissions
- Deposition of particles onto soil followed by:
 - direct contact with that soil
 - uptake into and consumption of home grown fruit and vegetables grown in this soil
 - uptake into and consumption of home grown eggs where chickens have access to this soil
 - uptake into and consumption of home grown meat where cattle have access to this soil
 - uptake into and consumption of home grown milk where cattle have access to this soil
- Deposition of particles onto a roof, collection into household rainwater tanks and consumption of tank water for domestic purposes including drinking.

It is noted that the pathways related to consumption of fruit, vegetables, eggs, meat or milk refer to home grown produce – i.e. wholly consumed by those living on a property where the maximum impacts are predicted for their entire lives where they consume all these types of produce every day (i.e. worst case).

Exposure scenarios evaluated

The following exposure scenarios have been assessed:

- Off-site location with the maximum modelled ground level concentrations – exposures via inhalation and direct contact with soil
- Maximum residential location (location that is currently used for residential land use that has the maximum ground level concentrations for any residential site) – exposure via inhalation, direct contact with soil and consumption of home grown fruit, vegetables and eggs and use of a rainwater tank
- Maximum residential location (location that is currently used for residential land use that has the maximum ground level concentrations for any residential site should its land use be changed to a farm) – exposure via inhalation, direct contact with soil and consumption of home grown fruit, vegetables and eggs and use of a rainwater tank and, in addition, consumption of home grown milk and meat
- Maximum commercial/industrial location (location that is currently used for commercial/industrial land use that has the maximum ground level concentrations for any commercial/industrial site) – exposure via inhalation and direct contact with soil
- Maximum commercial/industrial location (as residential) – assumes this location may change land use to residential at some time in the future
- Maximum on-site location – exposure via inhalation for workers and direct contact with soil at the Visitor and Education Centre (this will also be protective for visitors to the centre who will be present at the centre for less time than those that work there).

Modelling

Emissions from the facility were modelled by Katestone in line with Queensland Department of Environment, Tourism, Science and Innovation (DETSI) requirements. The facility has been designed to meet the daily average and spot sample emission concentration limits (upper) set out in the BREF 2019 (EU 2019) (this document is the most recent update on best practice technologies for such facilities developed in Europe), and the half-hour average emission concentration limits set out in the EU Industrial Emissions Directive (2010) for short-term emission rates.

The modelling estimated concentrations at ground level for assessing both short and long term exposures. The potential for impacts on human health due to short term exposures were assessed using the maximum 1 hour concentrations at ground level for the facility assuming normal operations at maximum thermal input and with all pollutants simultaneously at their expected maximum emission concentration limits. The potential for impacts on human health due to long term exposure scenarios were assessed using annual average ground level concentrations assuming normal operations. Cumulative impacts on air quality have also been assessed considering existing air quality in the area, estimated emissions from this proposed facility and background levels in diet and water across Australia.

The air quality modelling and the risk calculations have assumed the following mitigation measures will be in place during operation of such a facility:

- proper operation and maintenance of the facility
- detailed monitoring of emissions (including continuous monitoring for relevant pollutants and plant conditions (which indicate appropriate conditions for destruction/minimisation of those pollutants which cannot be monitoring continuously))
- monitoring of the proper operation of pollution control/flue gas equipment using sensors to detect breakage in the baghouse
- automated doors that rapidly open and close for entry to and exit from the waste reception tipping hall
- transport of waste to the site using enclosed trucks
- waste always unloaded within the building housing the tipping bays and storage bunker.

Results

Criteria pollutants

Criteria air pollutants are those that are targeted by the National Environment Protection (Ambient Air Quality) Measure (NEPM) (NEPC 2021b). They are common air pollutants arising from many sources (e.g. vehicles, windblown dust, fires) that are always present in the air people breathe and which need to be managed well to maintain acceptable air quality. These pollutants include sulfur dioxide, nitrogen dioxide, carbon monoxide and particles (PM₁₀, PM_{2.5}).

Air quality modelling was undertaken by Katestone to estimate the ground level concentrations at all relevant locations around the proposed facility. The results for the maximum off-site location (the location with the maximum modelled ground level concentrations outside the boundary of the facility) have been listed in **Table ES-1**. All other locations around the facility will have lower concentrations than those listed here.

Table ES-1: Criteria pollutants (maximum off-site location) (µg/m³)

Scenario	Sulfur dioxide	Nitrogen dioxide	Carbon monoxide	PM _{2.5}	PM ₁₀
Guideline (NEPC 2021a)	52	28	10,000	7	25
Guideline EPP (Air) (Queensland Government 2019b)	229	62	11,000	8	25
Averaging period	24 hour average	Annual average	8 hour average	Annual average	Annual average
Contribution from Project	3.1	0.3	16	0.01	0.01
% contribution from Project (compared to NEPM guideline value)	6%	1%	0.2%	0.1%	0.04%
Cumulative case	18	13	870	6.7	17

For all of these pollutants, levels contributed by this EfW facility are low.

For all of the pollutants, while the increments for this facility are low, the cumulative concentrations (those including background) are higher but still remain below the relevant national guidelines.

For particles, particularly those that are very small (PM_{2.5}), levels contributed by this facility are extremely low. When adding the levels from the Project to the existing levels in the atmosphere in the area (i.e. cumulative case), the overall concentrations remain in compliance with the national guidelines – the same as is currently the case.

The existing levels of particles come from the many sources of particles to the atmosphere including wood heaters, gas fired BBQs, gas cooking, candles, all types of vehicles, bushfires, power stations, windblown dust and degradation/breakdown of vegetation.

PM_{2.5} sized particles (i.e. those less than 2.5 µm) are always present in air, particularly in urban areas. National health authorities have developed guidelines for particles based on understanding all of this background.

Regardless, more detailed evaluation has been undertaken in this assessment to confirm risks to community health due to exposure to this small additional contribution of PM_{2.5} will be negligible (i.e. increased risk of 1×10^{-7}).

Other pollutants

Short term exposures

The assessment of short term exposure for all other air pollutants used the maximum 1 hour average ground level concentration at the worst-case location and compared that value to public health based guidelines for exposure over 1 hour. The worst-case location is the maximum off-site location (the location with the maximum modelled ground level concentrations outside the boundary of the facility). This location varies depending on the chemical (gases versus those attached to particles). It will be located just outside the site boundary or on the roads surrounding the facility.

The risk quotient is the ratio between the maximum 1 hour average ground level concentration and the public health based guideline for each chemical that might be present in the emissions (i.e. how much lower the estimated concentration is than the guideline which public health authorities have deemed to be acceptable (and based on no effects)).

For this assessment, the individual risk quotients were between 1.5 times and 5,000 times lower than the relevant guideline based on short term exposure in air. In addition, all the risk quotients were summed to get an overall consideration of short term risk. This risk index was also below 1 (i.e. 0.3 or 0.7 depending on which chemical was assumed to be relevant for assessing total volatile organic chemicals).

Short term exposures at the most affected location pose a negligible risk based on guidelines recommended by Australian health authorities. All other locations will have lower short term concentrations and lower short term risks.

Long term exposures

The assessment of long term exposures to emissions has been assessed for the types of exposures noted above. The assessment used the annual average ground level concentrations.

The risk calculations involved determining exposure for each of the various chemicals that might be present in the emissions via:

- inhalation (breathing it in)
- ingestion (eating or drinking it – direct or indirect)
- dermal (absorbing it through the skin).

Inhalation has been directly assessed for gases and for particles in air (with chemicals attached).

Chemicals attached to particles may also:

- deposit onto the ground and mix into soil where people might come into direct contact with the soil
- deposit onto the ground and mix into soil where various types of produce like vegetables or eggs might be grown in backyards or on farms or where cattle may graze and then be used for milk or meat
- deposit onto the roofs and mix into rainwater tanks.

Ingestion and dermal exposure via all these pathways have been assessed for the chemicals attached to particles.

There are 2 mechanisms by which chemicals are presumed to act to cause effects:

- Threshold
- Non-threshold.

Threshold chemicals are those that have a threshold below which no effects will occur (i.e. no risk at low exposures) while non-threshold chemicals have a low risk at low exposures because of the way they cause damage.

Risks (using risk quotients and risk indices etc) have been calculated for each exposure scenario and each type of exposure. An overall summary of the findings for the risk calculations is provided in **Table ES-2** for chemicals that act via a mechanism with a threshold and **Table ES-3** for chemicals that act via a mechanism with no threshold.

Table ES-2: Calculated Risks – threshold chemicals

Scenario	Risk (inhalation)	Risk (deposition – i.e. accumulation in soil or produce)	Risk (rainwater tank)	Risk (total)
Maximum off-site	0.03	0.0002	NA	0.03
Maximum residential	0.02	0.0003	0.000004	0.02
Maximum residential (as farm)	0.02	0.0005	0.000004	0.02
Maximum commercial	0.003	0.0001	0.000003	0.003
Maximum on-site	0.003	0.000004	NA	0.003
Guideline	≤1			

Table ES-3: Calculated Risks – non-threshold chemicals

Scenario	Risk (inhalation)	Risk (deposition – i.e. accumulation in soil or produce)	Risk (rainwater tank)	Risk (total)
Maximum off-site	8×10^{-8}	3×10^{-16}	NA	8×10^{-8}
Maximum residential	5×10^{-8}	3×10^{-16}	6×10^{-18}	5×10^{-8}
Maximum residential (as farm)	5×10^{-8}	1×10^{-15}	6×10^{-18}	5×10^{-8}
Maximum commercial	5×10^{-9}	5×10^{-17}	4×10^{-18}	5×10^{-9}
Maximum on-site	6×10^{-9}	1×10^{-17}	NA	6×10^{-9}
Guideline	≤1×10^{-5}			

For chemicals that act via a threshold mechanism, risks calculated across all the various scenarios evaluated for this facility are at least 30 times below the relevant guideline at the locations where ground level concentrations are expected to be highest (i.e. maximum off-site) and between 50 times and 300 times lower than the relevant guideline for all the other worst-case locations (maximum residential or commercial/industrial location).

For chemicals that act via a non-threshold mechanism, risks are at least 125 times below the relevant guideline for the combination of chemicals at the maximum off-site location and between 200 and 2,000 times lower than the relevant guideline for the other worst-case locations.

Assessment of other matters

To assess the potential for impacts to community health arising due to other aspects of the Project such as noise, water management, bushfire potential or chemical hazards, information from other technical studies prepared for this planning assessment has been used. The approach taken in this assessment was to determine if those technical studies have included consideration of potential impacts to human health (in addition to the range of issues they must address) and whether those matters have the potential to cause any changes to the current situation.

For matters related to noise, the Project will comply with relevant Queensland guidelines as well as guidelines from the World Health Organisation (WHO) which are targeted at limiting impacts to community health due to changes in noise levels.

For matters related to water, the Project has been designed to have appropriate stormwater management and to not change the current flooding potential for the site – i.e. little change to the current situation.

For matters related to chemical hazards, the Project will comply with all relevant Queensland guidance in relation to storage and handling of such materials which will ensure such hazards are appropriately managed.

Overall Conclusions

Based on the assessment undertaken in relation to this facility and the potential for changes to community health, the following has been concluded:

■ Air quality

With consideration of the air quality guidance adopted and the assessment of potential changes in air quality arising due to this proposed facility, where the facility is designed to meet the specifications identified, there will be no changes in air quality that could impact on community health in the area around the proposed facility. In particular, this assessment has shown:

- No unacceptable risks for criteria pollutants (NO_x , SO_x , CO , $\text{PM}_{2.5}$, PM_{10})
- No unacceptable risks for short term exposures from the proposed facility at the maximum off-site location – all other locations will have lower concentrations and so risks will be lower
- No unacceptable risks for relevant exposure scenarios considering long term exposures (both via inhalation and after deposition onto soil and uptake into home grown produce) at:
 - Maximum off-site location
 - Maximum residential location (and maximum commercial/industrial and maximum other places if land use changes to residential)
 - Maximum commercial/industrial location
 - Maximum on-site location
- No unacceptable risks for relevant exposure scenarios for rainwater tanks.

The following conservative, best practice HHRA assumptions have been incorporated into the assessment:

- risks via exposure to chemicals attached to particles have been assessed assuming the particles may deposit onto the soil around the facility for 70 years without removal (i.e. more than the lifetime of the facility).
 - risks via inhalation are based on the worst-case locations and assuming people will be present at those locations 24 hours/day for 365 days per year for the lifetime of the facility.
- Potential for PFAS (per/poly fluoroalkyl substances) to be present in emissions from this facility has been considered in this assessment. Using worst case data from a Swedish facility, these chemicals make an insignificant contribution to the overall risk estimates for this Project. Given the likely decreasing levels of these chemicals in the residual waste to be used as fuel for this facility due to phasing out/bans in the use of these chemicals as well as the control measures built into the facility (i.e. temperatures in the combustion chamber, use of activated carbon and other chemicals in the pollution control equipment and the baghouse), it is expected that levels of PFAS in the emissions will be very low to negligible/not measurable.

■ Noise

With consideration of the noise limits adopted and the assessment of potential changes in noise levels for the Project, where the Project is designed to meet the noise specifications identified (i.e. predicted levels), and where the identified noise mitigation measures are implemented, there will be no changes in noise sufficient to cause changes in community health for the off-site community.

■ Other matters (water, soil contamination, dangerous goods/chemical hazards)

Based on the evaluations provided and ensuring that government regulations about storage and transport of hazardous chemicals are followed, there are no changes in stormwater management, flooding potential, handling of dangerous goods or bushfire hazard that would be sufficient to cause changes in community health for the off-site community.

■ Drinking water catchment

Given the very low levels of emissions from this facility, it is not possible for these emissions to make a noticeable change in the concentrations of the relevant chemicals in raw water within the drinking water catchment (Logan Albert Catchment) due to deposition of particles from this facility. This has been confirmed by comparison of worst case air concentrations to existing air quality likely in urban areas and by comparison of estimated water concentrations in rainwater tanks to Australian drinking water guidelines. In both cases, the estimated levels from this facility are thousands of times lower, if not, millions of times lower than relevant air or drinking water guidelines. The emissions will not change the existing air quality in a way that is measurable or noticeable.

Section 1. Introduction

1.1 Background

Environmental Risk Sciences Pty Ltd (enRiskS) has been engaged by Cleanaway Operations Pty Ltd (Cleanaway) to undertake a Human Health Risk Assessment (HHRA) for an Energy from Waste (EfW) facility in Bromelton, Queensland.

Cleanaway is an Australian waste management, recycling, and industrial services company. Cleanaway is developing an EfW facility in Queensland known as the Bromelton Energy and Resource Centre (BERC) (the Project). The Project will be located within the Special Industry Precinct of the Bromelton State Development Area (SDA), west of Beaudesert within the Scenic Rim Regional Council local government area.

The BERC has been designed to thermally treat a nominal capacity of 760,000 tonnes per annum (tpa) of waste feedstock via two 140 megawatt thermal (MWth) processing lines when operating at the design calorific value (CV) of 10.6 MJ/kg (LHV) and assuming 8,000 hours per annum for boiler availability. The waste feedstock is expected to consist of a mix of residual Municipal Solid Waste (MSW) and residual commercial and industrial waste (C&I), which is waste that would otherwise be sent to landfill. Waste feedstock processed by the BERC will be subject to a Waste Acceptance Protocol to determine eligibility and suitability for processing wastes both prior to arrival and upon arrival on-site. The BERC will also incorporate maturation and processing of incinerator bottom ash (IBA) to recover recyclable metals, with the intent to utilise the remaining ash as an aggregate in construction.

1.2 Objectives

This HHRA is being undertaken to evaluate the potential for the Project to cause impacts on human health for the off-site community, with consideration of existing and proposed land uses via air emissions, noise emissions, water issues or hazardous substances handling.

The objectives of this assessment are:

- Provision of a human health risk assessment to assess and evaluate potential community exposures to air emissions from the proposed facility including inhalation of criteria pollutants from combustion and specific air toxics, and multi-pathway exposure to persistent and bioaccumulative chemicals via deposition to soil and water and uptake into produce (where relevant).
- Provision of a qualitative assessment of issues relating to community health for noise, water and chemicals management.

The HHRA has been based on the proposed waste to be used as fuel and the proposed combustion and emissions control technology. The HHRA has considered emissions scenarios relevant to the operation of the facility, with impacts associated with regulatory emissions limits being addressed. The assessment has only evaluated risks to community health.

The assessment has not included a quantitative assessment of risk based on occupational exposures for workers at the facility during construction or operation as this is covered by Work Health and Safety legislation. A qualitative discussion has been provided in **Sections 5.2 and 5.3**.

Risks to ecosystems are considered in other parts of the planning documents related to the proposed facility and are not covered in a Human Health Risk Assessment.

1.3 Approach

The HHRA has been undertaken in accordance with the following guidance (and associated references as relevant):

- enHealth, 2012. Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a)
- enHealth 2012. Australian Exposure Factors Guide (enHealth 2012b).

Consideration has also been given to the following guidance documents on the conduct of health risk assessments associated with air emissions from point sources:

- NEPC 2021. National Environment Protection (Ambient Air Quality) Measure (NEPC 2021b)
- NEPC 2011. National Environmental Protection (Air Toxics) Measure (NEPC 2011a)
- Environmental Protection (Air Policy) 2019 (Air EPP).

In addition, where relevant, the HHRA has considered the requirements of the following regulations and policies issued in Queensland:

- *Environmental Protection Act 1994*
- *Environmental Protection Regulation 2019 (including Environmental Protection Policy (Air)) (Queensland Government 2019b)*
- *Energy from Waste Policy (the EfW Policy) 2021 (Queensland Government 2021a)*
- *Energy from Waste Guideline (the EfW Guideline) 2021 (Queensland Government 2021b)*
- *DES Guideline, Application requirements for activities with impacts to air – ESR/2015/1840, 2021*
- *DES Guideline, Assessment requirements for making a decision for an EA for an environmentally relevant activity – ESR/2015/1725, 2022*
- *Bromelton State Development Area Development Scheme 2017*

To address the above, the HHRA has presented the following:

- Description of the Project (**Section 2**)
- Identification of the community of concern – this is the location and characteristics of the population surrounding the site (**Section 3**)
- Description of methodology for assessing health impacts from air emissions (**Section 4**)
- Assessment of health impacts from air emissions – this is a quantitative assessment of potential community health impacts from changes in air quality as a result of the operation of the facility (**Section 5**)
- Assessment of health impacts from noise emissions (**Section 6**)
- Assessment of health impacts for other aspects of the Project including water, soil contamination, chemicals management, groundwater (**Section 7**)
- Conclusions (**Section 8**).

1.4 Definitions

The following definitions are relevant for the conduct of the HHRA and should be considered when reading this report.

Health

The World Health Organisation defines health as “*a (dynamic) state of complete physical, mental and social wellbeing and not merely the absence of disease or infirmity*”.

Hence the assessment of health should include both the traditional/medical definition that focuses on illness and disease as well as the more broad social definition that includes the general health and wellbeing of a population.

Health Hazard

These are aspects of a project, or specific activities that present a hazard or source of negative risk to health or well-being.

In relation to the HHRA, these hazards may be associated with specific aspects of the proposed development/construction or operational activities, incidents or circumstances that have the potential to directly affect health. In addition, some activities may have a flow-on effect that results in some effect on health. Hence health hazards may be identified on the basis of the potential for both direct and indirect effects on health.

Health Outcomes

These are the effects of the activity on health. These outcomes can be negative (such as injury, disease or disadvantage), or positive (such as good quality of life, physical and mental wellbeing, reduction in injury, diseases or disadvantage).

It is noted that where health effects are considered these are also associated with a time or duration with some effects being experienced for a short period of time (acute) and other for a long period of time (chronic). The terminology relevant to acute and chronic effects is most often applied to the assessment of negative/adverse effects as these are typically the focus of technical evaluations of various aspects of the Project.

Likelihood

This refers to how likely it is that an effect or health outcome will be experienced. It is often referred to as the probability of an impact occurring.

Risk

This is the chance of something happening that will have an impact on objectives. In relation to the proposed Project and the conduct of the HHRA, the concept of risk more specifically relates to the chance that some aspect of the project will result in a reduction or improvement in the health and/or well-being of the local community. The assessment of risk has been undertaken on a quantitative basis for air, water and noise emissions and a qualitative basis for all other impacts. This is in line with the methods and levels of evidence currently available to assess risk.

Equity

Equity relates to the potential for the Project to lead to impacts that are differentially distributed in the surrounding population. Population groups may be advantaged or disadvantaged based on age,

gender, socioeconomic status, geographic location, cultural background, aboriginality, and current health status and existing disability.

1.5 Available information

This HHRA has been undertaken on the basis of relevant Project specific information which is available in the following reports:

- Arup (2025a). Bromelton Energy and Recovery Centre, Technical process description. Dated 19 December 2025.
- Katestone (2026), Bromelton Energy and Recovery Centre, Air quality assessment. Rev C Dated February 2026.
- Air Quality Modelling Output Spreadsheets provided by Katestone (received 15/09/25).
- Arup (2025b), Bromelton Energy and Resource Centre, Noise and Vibration Technical Report. Rev C Dated 19 December 2025.
- Douglas Partners (2025). Report on Preliminary Site Investigation for Contamination Bromelton Energy and Resource Centre (BERC) Lot 1 Beaudesert Boonah Road, Bromelton, QLD. Dated 19 December 2025.
- Arup (2026a). Bromelton Energy and Resource Centre (BERC). Hazards and risk. Rev C Dated 22 January 2026.
- Arup (2026b). Stormwater management plan and flood risk assessment, Bromelton Energy and Resource Centre (BERC). DRAFT Dated 4 February 2026.
- Arup (2025c). Bromelton Energy and Resource Centre, Bushfire Hazard Assessment. Dated 19 December 2025.
- Arup (2026c). Socio-economic impact assessment, Bromelton Energy and Resource Centre (BERC). Rev C Dated 6 February 2026.

1.6 Legislative background

1.6.1 General

The legislation, policies, and guidelines relevant to the human health risk assessment for this facility are summarised in **Tables 1.1** and **1.2**. National guidance documents provide more specific guidance about the conduct of human health assessments for specific developments.

1.6.2 Australian Government

Table 1.1 summarises the national legislation, policy and guideline documents relating to the conduct of human health assessments in Australia, with some providing more specific guidance for assessing potential health impacts associated with changes in air quality.

Table 1.1: Current legislation, policy and guidelines for assessing health impacts – Australian Government

Legislation, policy, or guideline	Overview and implications for this Project
<i>National Environment Protection Council Act 1994</i>	The <i>National Environment Protection Council Act 1994</i> relates to the establishment and operation of the National Environment Protection Council (NEPC) to meet the objectives that:

Legislation, policy, or guideline	Overview and implications for this Project
	<i>people enjoy the benefits or equivalent protection from air, water or soil pollution and from noise, wherever they live in Australia; and decisions of the business community are not distorted, and markets are not fragmented by variations between participating jurisdictions in relation to the adoption or implementation of major environment protection measures.</i> The Act provides for the NEPC to make and vary or revoke National Environment Protection Measures (NEPM) and assess the implementation of the NEPM. Some of the more relevant NEPM documents are referenced below. The Act does not specifically address the assessment of health impacts for individual projects.
enHealth Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a)	The enHealth Environmental Health Risk Assessment Guidelines provide an outline of the national approach adopted for the assessment of environmental health risks. These assessments focus on situations where a more detailed assessment of exposure, toxicity and health risk is required, and can be undertaken. This may include exposure to environmental stressors such as changes in air quality, noise, water quality and/or soil/waste. The guide provides the Australian framework and approach for the quantitative assessment of these risks.
enHealth Australian Exposure Factors Guide (enHealth 2012b)	The enHealth Australian Exposure Factors Guide is a compendium to the enHealth environmental Health Risk Assessment Guidelines (2012a). The document provides a review of quantitative exposure factors that may be used in the conduct of a quantitative health risk assessment.
NEPC National Environment Protection (Assessment of Site Contamination) Amendment Measure 2013 (No.1) (NEPC 2013)	The NEPC National Environment Protection Amendment Measure provides the national guidance in relation to the assessment of contaminated land in Australia. This guidance is provided in Schedules B1 to B9. This includes the derivation of health-based guidelines for the assessment of contaminated land (NEPC 1999 amended 2013b, 1999 amended 2013c).
NEPC National Environment Protection (Ambient Air Quality) Measure (2016 and as updated 2021) (NEPC 2021b)	The NEPC National Environment Protection Measure is implemented under section 14 of the <i>National Environment Protection Council Act 1994</i>. The Measure provides national environment protection protocols for the sampling and reporting of ambient air quality and sets national environment protection standards and goals for carbon monoxide, nitrogen dioxide, photochemical oxidants (as ozone), sulfur dioxide, lead, and particulates as PM₁₀ and PM_{2.5}. The desired environmental outcome of the Measure is ambient air quality that minimises the risk of adverse health impacts from exposure to air pollution. The Measure was amended and approved in 2021. Compliance with the goals and standards in this Measure (as relevant to the Project) would facilitate management of ambient air quality that is protective of health and wellbeing.
NEPC National Environmental Protection (Air Toxics) Measure (NEPC 2011a)	The NEPC National Environment Protection Measure is implemented under section 14 of the <i>National Environment Protection Council Act 1994</i>. This Measure provides national environment protection protocols for the sampling and reporting of specific toxic chemicals in ambient air quality and sets monitoring investigation levels relevant to these chemicals. The Measure addresses benzene, benzo(a)pyrene, formaldehyde, toluene and xylenes in ambient air in Australia. The purpose of the Measure is to improve the information base regarding ambient air toxics in Australia to facilitate the development of standards. The Measure provides for guidelines for monitoring that are based on the management of human health and wellbeing. Compliance with the monitoring investigation levels in this Measure (as relevant to the Project) would facilitate management of ambient air toxics that is protective of health and wellbeing.

Legislation, policy, or guideline	Overview and implications for this Project
National Environment Protection Council (NEPC) Schedule B8 Guideline on Community Consultation and Risk Communication, National Environment Protection (Assessment of Site Contamination) Measure	The NEPC Guideline of Community Consultation and Risk Communication Measure set out an approach for effective community engagement and risk communication relating to the assessment of environmental health risk issues.

1.6.3 Queensland Government

Table 1.2 summarises the state legislation and guidance that broadly relate to assessing the potential impacts of developments in regard to protection of health.

Table 1.2: Current legislation, policy and guidelines for assessing health impacts – Queensland Government

Legislation, policy, or guideline	Overview and implications for this Project
<i>Environmental Protection Act 1994</i>	This Act (and related regulations and related environmental protection policies) sets the basis for environmental protection in Queensland – both in relation to impacts on people or the environment. The objective of the Act is <i>to protect Queensland's environment while allowing for development that improves the total quality of life in a way that maintains the ecological processes on which life depends</i>. It outlines the General Environmental Duty relevant in QLD and provides the requirements for seeking Environmental Authorities (EA) to undertake Environmentally Relevant Activities (ERA). It also provides the basis for the environmental protection policies which also apply to assessing new developments. There are no specific references to the assessment or protection of community health in relation to environmental authorities. However, to demonstrate that the environmental values relevant to the Project are considered, it is important to consider potential for impacts on human health for the people living or working around a proposed facility.
<i>Bromelton State Development Area, December 2017</i>	State Development Areas identify an area targeted by the Queensland Government for a specific type of development. In this case, the Bromelton SDA has been identified as an area where industrial development of regional, state or national significance is to be located. The SDA also notes that sensitive land uses that would be inconsistent with the type of industrial development being targeted should not be permitted within the designated area. The SDA Development Scheme provides SDA wide assessment criteria which include, amongst other things, a requirement that development avoid or minimise adverse impacts from air, noise and other emissions that will affect the health and safety, wellbeing and amenity of communities and individuals.
<i>Environmentally relevant activities (ERAs), State Code 22</i>	Environmentally relevant activities are industrial activities etc where there is potential to release emissions which may impact on the environment and surrounding land uses. For any industrial development, the appropriate ERA classification(s) need to be identified. For sites undertaking an environmentally relevant activity, an Environmental Authority must be obtained as part of the planning application process. Where emissions from an activity may impact on the environment and surrounding land uses, the potential for impacts on human health must be considered.
<i>Environmental Protection (Air) Policy 2019</i>	The purpose of EPP (Air) is show how to: identify environmental values that need to be protected or enhanced in relation to the air environment

Legislation, policy, or guideline	Overview and implications for this Project
	- determine indicators and objectives that enhance/protect relevant environmental values in relation to the air environment - apply a framework for decision making about the air environment to ensure consistent, equitable and informed decisions.
<i>Environmental Protection (Noise) Policy 2019</i>	The purpose of EPP (Noise) is show how to: - identify environmental values that need to be protected or enhanced in relation to noise - determine indicators and objectives that enhance/protect relevant environmental values in relation to noise - apply a framework for decision making about noise to ensure consistent, equitable and informed decisions.
<i>Health Considerations – Environmental Impact Statement (guidelines for proponents) (2016)</i>	Queensland Department of Health has provided guidance on what sort of health considerations are required in environmental impact statements for new projects. This guidance is designed to: - ensure that a proponent identifies relevant environmental hazards that impact on human health and wellbeing - provide guidance to proponents on how to demonstrate that risks to human health have been minimised. Potential for impacts on community health due to changes in air quality, noise, water quality, land management etc should be considered in such an assessment. Such an assessment should be undertaken in accordance with the enHealth guideline for the conduct of such assessments. This is the guidance document used for this risk assessment.
<i>Queensland Energy from Waste Policy and Guideline (2021)</i>	These documents outline the specific requirements for an energy from waste facility in Queensland. These documents note that a proposal for such a facility must document how the facility will protect the waste hierarchy, demonstrate that it will perform as expected, that only residual waste will be used to produce energy, that the facility can adapt to changes in waste streams, how energy will be recovered from the process, how the community will be consulted and how the facility will ensure the environment is protected (including protection of people). It is noted that such facilities must ensure that they limit the risk of harm to people and the environment from air emissions and other environmental releases. The guideline document provides specific information about how air emissions from the facility are to be assessed (including a list of pollutants and requirements for estimating mass emission rates). The human health risk assessment uses the air emission modelling data, so these requirements are important for this assessment as well as the air quality impact assessment.

Section 2. Project description

2.1 Overview

Cleanaway Operations Pty Ltd (Cleanaway) is an Australian waste management, recycling, and industrial services company. Cleanaway is developing an Energy from Waste (EfW) facility in Queensland known as the Bromelton Energy and Resource Centre (BERC) (the Project). The Project will be located within the Special Industry Precinct of the Bromelton State Development Area (SDA), west of Beaudesert within the Scenic Rim Regional Council local government area.

The Project has been designed to thermally treat approximately 760,000 tonnes per annum (tpa) of waste feedstock, consisting of residual Municipal Solid Waste (MSW) and residual Commercial and Industrial (C&I) waste. Residual waste is waste that is not technically, environmentally and economically practical (TEEP) to reuse or recycle, which would otherwise be sent to landfill for disposal. The facility's actual annual throughput will vary depending on the calorific value of the feedstock and plant availability. To account for variability in feedstock energy content, the facility will be designed for a maximum processing capacity of up to 10% above the nominal figure (836,000 tpa). The EfW process is summarised in **Figure 2.1**.

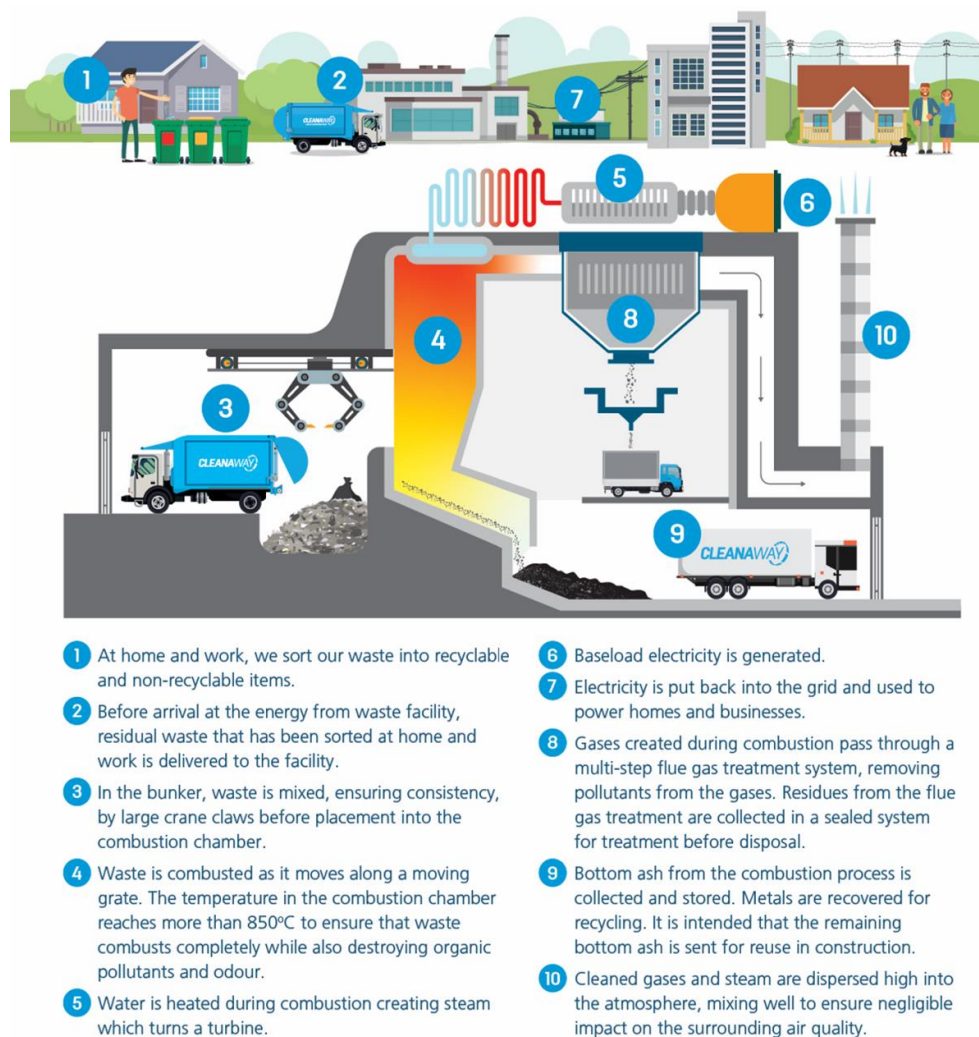


Figure 2.1: Energy from Waste Process

2.2 Proponent

Cleanaway, an Australian owned company, has operated for more than 50 years, supporting industry, government, communities and households across Australia. Its mission is to make a sustainable future possible, achieved through action across all levels of the waste hierarchy. Waste generation is minimised through extensive education programs, with Cleanaway's 50 education officers reaching over 120 businesses, 30 councils, and almost 33,000 school participants in the last financial year.

Cleanaway invests heavily in recycling, operating several Material Recovery Facilities nationwide and has partnered with Pact Group, Asahi Beverages and Coca-Cola to deliver two large-scale PET (polyethylene terephthalate) recycling facilities and a HDPE (high density polyethylene) recycling facility with Pact Group. In addition, in collaboration with Viva Energy, Cleanaway is developing a soft plastics recycling plant. EfW is recognised by Cleanaway as an important part of the future waste management framework, enabling recovery of energy and other resources from residual waste that cannot otherwise be recycled.

2.3 Policy Alignment

The BERC aligns with the Queensland EfW Policy and the Queensland EfW Guideline (2021) by diverting residual waste from landfill and delivering economic and social benefits through employment and local business opportunities in South East Queensland.

In accordance with Policy outcome 7 of the Queensland EfW Policy, the BERC will be designed to meet the best practice emission standards and requirements of the European Industrial Emissions Directive (IED) (2010) and the associated Best Available Techniques Reference (BREF) Document for Waste Incineration (2019), which sets the European Union environmental standards for waste incineration.

The Project also supports the Queensland Waste Strategy contributing to its 2050 targets to reduce household waste by 25%, recycle 75% of all waste, and divert 90% from landfill. In doing so, the BERC will play a key role in advancing Queensland's transition to a circular economy and reducing long-term reliance on landfill.

2.4 Key Components

The key components of the Project are summarised in **Table 2.1**.

Table 2.1: Key Project Components

Project Component	Detail
Address	Lot 1 Beaudesert Boonah Road, Bromelton, Queensland, 4285 (Lot 1 on RP45234). Refer Figure 2.2
Site Area	63 hectares
Summary of proposed works	The project includes the construction of a main EfW facility for waste processing, incinerator bottom ash (IBA) maturation and processing facility and an air pollution control residue (APCr) facility. It also includes all on-site infrastructure needed for the EfW facility, such as site utilities, internal roads, weighbridges, parking and hardstand areas, stormwater infrastructure, fencing and landscaping. The Project will include a visitor centre to help educate and inform the community on circular economy principles, the waste hierarchy, including waste avoidance, recycling, resource recovery, the benefits of landfill diversion and the EfW process. The intent behind this education is to drive a shift in community awareness and attitudes toward waste management and resource recovery.

Project Component	Detail
Technology Used	The proposed technology is moving grate combustion technology. This has been used globally for over 100 years and is common in many of the 500 similar operational examples across Europe.
Facility Outputs	Approximately 73.2 MW (577,600 MWh/year, corrected for ambient temperature) exported to grid as baseload electricity. Potential to supply energy in the form of heat and/or process steam to local industrial users. Residual material include IBA and APCr. It is intended that the IBA will be mechanically processed on-site to create incinerator bottom ash aggregate (IBAA). During the process, both ferrous and non-ferrous metals are recovered for recycling and sale. It is intended that the remaining material is re-purposed for use as an aggregate in construction. The Project includes a facility to stabilise APCr before removal and disposal at a licensed landfill.
Key Infrastructure	EfW facility including administration and visitors centre IBA maturation and processing facility (2 x pads and 2 x sheds) APCr facility (1 x pad, 1 x shed) Transformer yard and switchyard De-coupling area for trucks Weighbridges Internal road network and carparking 2 x combined storm water treatment and detention basin 1 x lined retention basin Access from Beaudesert Boonah Road via a signalised intersection with Recycling Street
Hours of Operation	24 hours per day, seven days per week (365 days per year)
Operational Staff	Approximately 60 staff

Figure 2.1: Site Description



2.5 Additional information relevant to the HHRA

2.5.1 Air pollution control equipment

The concentrations of some pollutants in the gas leaving the combustion chamber, such as nitrogen oxide, nitrogen dioxide (together referred to as NO_x), carbon monoxide (CO), volatile organic compounds (VOCs), and dioxin-like compounds, are directly linked to the performance of the combustion system as well as the composition of the waste.

The primary ways emissions are controlled include:

- Mixing of the waste in the waste bunker prior to feeding each combustion system
- Automatic combustion control system which continuously adjusts aspects like rate of waste feedstock entering the system, movement of the grate and air flows in order to maintain the desired rate of steam production
- Ensuring the gas temperature in the combustion chamber remains above 850 degrees Celsius for at least two seconds residence time as required in EU regulation (EU 2019).

In addition, gases from the combustion chamber must be cleaned before being released through the stack. It is not possible to bypass the air pollution control equipment, including in the event of an emergency shutdown.

As noted in Katestone (2026):

The design of the emission control and flue gas treatment systems does not include the ability to bypass emission control or flue gas treatment devices. The EU Industrial Emissions Directive Article 50(4) requires waste incineration plants to operate an automatic system to prevent waste feed: (a) at start-up, before the minimum combustion temperature is achieved, (b) if the high temperature residence time criteria of the combustion chamber cannot be maintained during operation or (c) if the continuous emissions monitoring system shows that an emission limit value is exceeded due to a failure or disturbance of a flue gas cleaning device. It is possible to bypass an individual cell within the bag filter to enable maintenance (e.g. bag replacement), however, the bag filter is overengineered so that the operating performance is maintained while one cell is offline for maintenance.

The air pollution control system will include:

- Selective Non-Catalytic Reduction (SNCR) – Ammonia or urea solution is injected into the combustion chamber (when the flue gas temperature is in the range 900-1,050°C). Either of these chemicals will react with NO_x to create nitrogen and water, both of which are not harmful to people or the environment.
- Semi-dry flue gas treatment – lime and activated carbon are injected into the reactor after the boiler. Lime reacts with HCl, HF and SO₂ to remove these acid gases. Metals and organics such as dioxins/furans are adsorbed onto the surface of activated carbon particles lowering the levels of these chemicals in the gases.
- Baghouse filter – the particles of lime and activated carbon (post reaction with acid gases or after adsorption of metals and organics) as well as particles of ash from the combustion process are collected by the filter bags within the baghouse. The particles collected within the bags form the air pollution control residues. Removing these particles removes the

particles and the contaminants attached to the particles from the gas which is then emitted from the stack.

This facility will be capable of cleaning the flue gases in line with the emissions limits as set out in the Industrial Emissions Directive (IED) and the associated Best Available Techniques Reference (BREF) document for waste incineration as published on 3rd December 2019 (EU 2010, 2019).

2.5.2 Ash management

Combustion of solid fuel (including waste) contains an incombustible fraction and this will always create ash that must be managed.

The Project will produce two solid residues:

■ Incinerator Bottom Ash (IBA)

- IBA is the inert, non-combustible component of the waste that is left on the grate at the end of the combustion process and is collected at the bottom of the grate. This material is somewhat similar to the ash left in the fireplace of a wood fired heater, a BBQ or a campfire.

IBA for this facility will be collected from below the grate using a wet ash conveyor system. IBA will be transported via a long conveyor to the IBA Treatment area where it will undergo maturation and sorting including the removal for recovery of ferrous and non-ferrous metals. Sorting and metals recovery will occur indoors, after a period of maturation.

After maturation and metals recovery, the IBA aggregate (IBAA) is typically considered (after suitable testing and confirmation during commissioning) to be non-hazardous. Recovered IBAA can then be used as an aggregate in construction (such as road bases). This is common practice for recycled IBAA in Europe and the UK. This project will repurpose the IBAA in accordance with QLD EfW legislation (Katestone 2026).

Figure 2.3 shows the process for managing IBA and IBAA proposed for this facility.

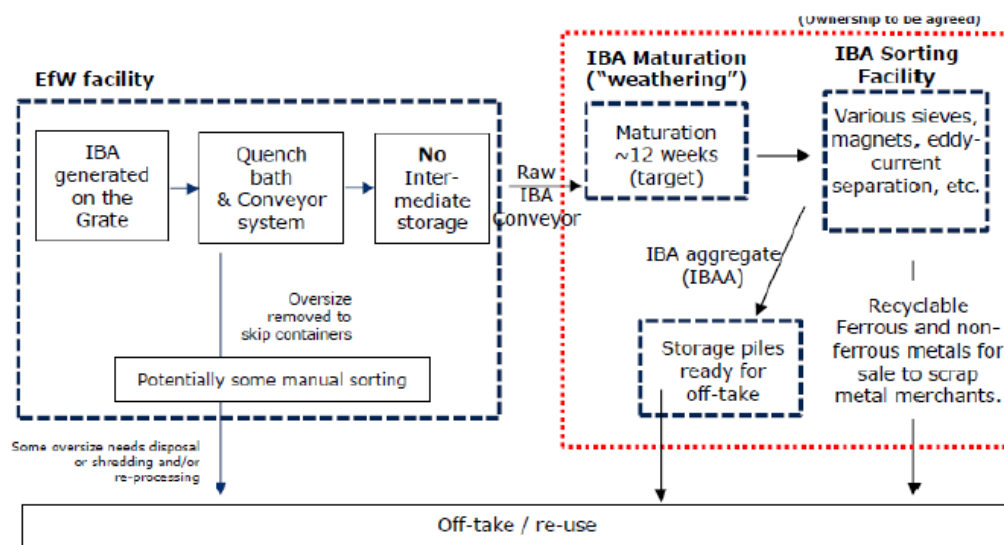


Figure 2.3: IBA/IBAA processing

■ Air pollution control residues

- Air pollution control residues (APCr) – APCr is the name given to any residues that are extracted from the process after the addition of flue gas treatment reagents. APCr are a combination of spent reagents (such as lime and activated carbon) and the entrained ash within the flue gases that did not fall out of the air as it moved through the boiler section. APCr are collected from the gas as it moves through the baghouse filter section of the plant. This material is considered hazardous and must be treated to minimise leaching of hazardous chemicals. Treatment involves mixing the material with cement and water to form solid concrete blocks which will be disposed to a relevant landfill.

Section 3. Community profile

3.1 General

This section provides an overview of the community in the vicinity of the proposed Project.

The site is located wholly within the Scenic Rim Local Government Area (LGA). Local government areas Ipswich City Council, Lockyer Valley City Council, Logan City Council and Gold Coast City Council adjoin Scenic Rim to the north and east. Other more rural areas adjoin this LGA to the south and west.

The community profile for the study area has been established by drawing upon the census data from the Australian Bureau of Statistics (ABS) for the relevant local government areas and statistical area 2 groupings (i.e. SA2s). SA2 groups are medium sized areas with similar characteristics/populations while local government areas are defined by the relevant state government. For this assessment, statistics for Scenic Rim and Logan City LGAs as well as Boonah and Beaudesert SA2s have been considered.

The key focus of this health risk assessment is the local community surrounding the site and has been informed, in part, by:

- Arup (2026c). Socio-economic impact assessment, Bromelton Energy and Resource Centre (BERC). Rev C Dated 6 February 2026.

3.2 Land uses

Surrounding characteristics and land uses are consistent with the Project, as most adjacent lots are designated for industrial uses and are within a State Development Area (SDA). Therefore, the Project is unlikely to impact the amenity of the surrounding landscape especially as sensitive receivers are at least 1.4 km away. Most businesses within the study area are also focused around industrial or agricultural uses (Arup 2026c).

Beaudesert is the closest urban centre to the study area (approximately 6.5 km east) and hosts several medical services including a hospital, child care and aged care facilities, schools, and many recreational facilities including a parks and a golf course. Active businesses within the study area comprise of mainly Agriculture, Forestry and Fishery, Construction, and Rental, Hiring and Real Estate Services. The Project would encourage a more diverse economy within the region, and also has the potential to utilise existing businesses in the construction and operational phases (Arup 2026c).

Historical photography indicates the site has primarily been used in the past 100 years for grazing and agricultural purposes (Arup 2026c).

Figure 3.1 shows the regional context for land use.

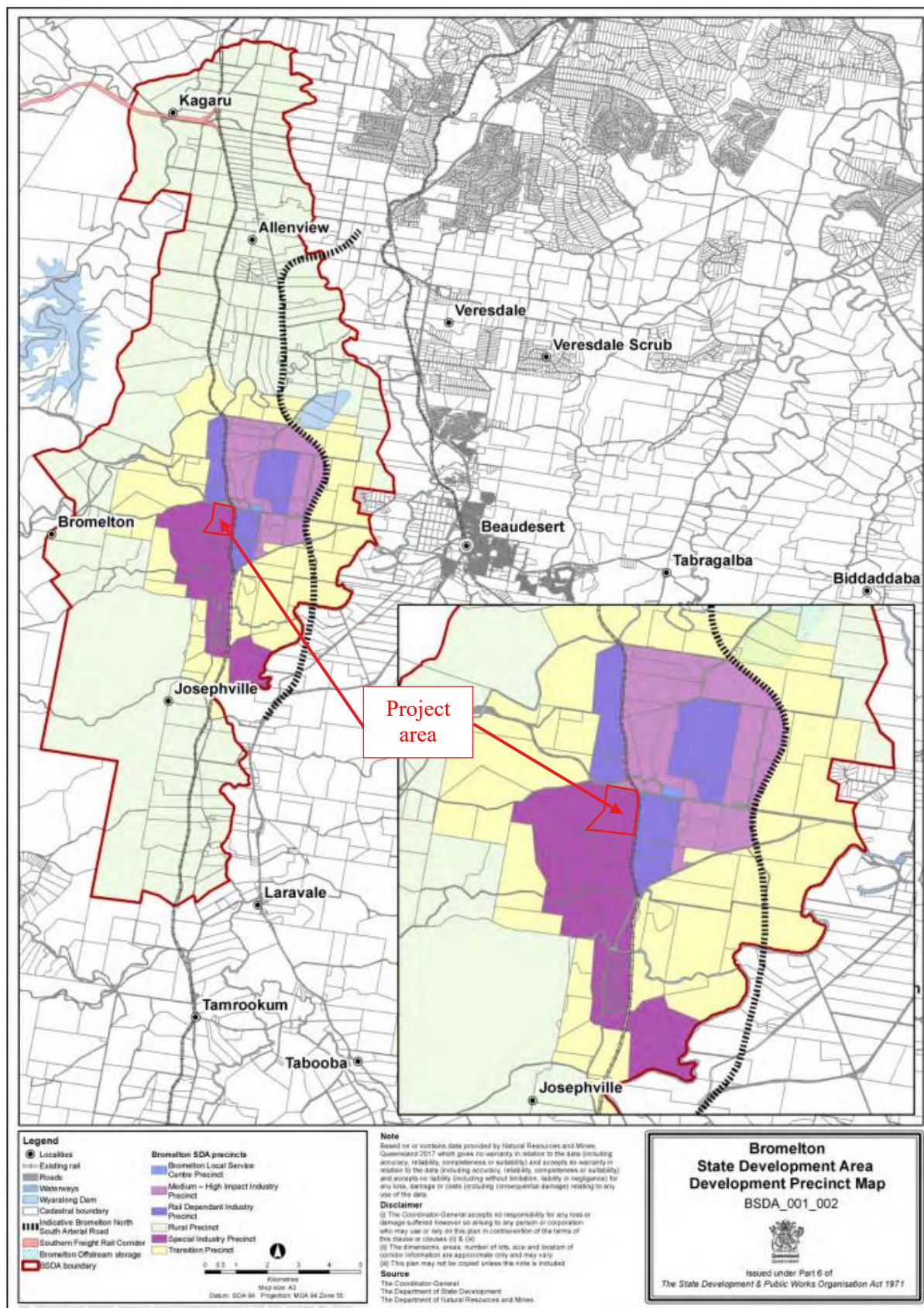


Figure 3.1

3.3 Population

Table 3.1 presents a summary of the populations in the relevant LGAs. Population data have been sourced from 2021 Census QuickStats¹ and 2021 Socio-Economic Indexes² from the Australian Bureau of Statistics. Unemployment data are taken from the latest Labour Force statistics for Australia and Queensland³ (August 2023) and the Small Area Labour Statistics⁴ (June 2022).

Census data are only collected every 5 years so the most recent is that from 2021. The next census is scheduled for August 2026 and it is assumed that data from that will not be available until 2027⁵.

Data are compared to data for Queensland and Australia as a whole.

Table 3.1: Summary of populations surrounding the proposed Project site (ABS Census 2021)

Indicator	Local Government Areas		Statistical Areas (SA2)		Queensland	Australia
	Scenic Rim LGA	Logan City Council LGA	Boonah	Beaudesert		
Total population	42,984	345,098	12,617	14,674	5,156,140	25,422,789
Population 0 - 4 years	5% (2,072)	7% (24,941)	5% (603)	5% (736)	5.7% (292,452)	5.8% (1,463,817)
Population 5 - 19 years	19% (8,097)	22% (75,768)	18% (2,322)	19% (2,828)	19% (985,377)	18% (4,632,001)
Population 20 - 64 years	54% (23,097)	58% (198,493)	53% (6,684)	53% (7,775)	58% (3,002,708)	59% (14,948,880)
Population 65 years and over	23% (9,718)	13% (45,911)	24% (3,006)	23% (3,332)	17% (875,603)	17% (4,378,088)
Median age	46	34	47	45	38	38
Household size	2.6	2.9	2.5	2.5	2.5	2.5
Unemployment	5.3%	7.1%	3.7%	9.5%	4.1%	3.7%
Tertiary education	13%	17%	12%	11%	21%	23%
SEIFA IRSD	983	959	987	924	--	--
SEIFA IRSD quintile	3	2	2	1	--	--
Indigenous	3.7%	4.2%	2.5%	6.4%	4.6%	3.2%
Born overseas	21.9%	34.3%	16.9%	19.4%	28.6%	33.1%

Notes:

SEIFA IRSD = index of socioeconomic disadvantage, Quintile is related to rank within Australia and ranges from

1 = most disadvantaged to 5 = least disadvantaged

Shading relates to comparison against statistics for Queensland overall: more vulnerable less vulnerable

Based on the population data presented in **Table 3.1** for the Scenic Rim local government area, this community generally has:

- Higher proportion of people over 65 than QLD overall
- Higher median age than QLD overall
- Similar proportion of people under 19 (including 0-4 year) compared to QLD overall
- Similar proportion of indigenous people compared to QLD as a whole
- Higher unemployment than QLD as a whole
- Lower proportion of people born overseas than QLD as a whole
- Similar household size to QLD as a whole

¹ <https://abs.gov.au/census/find-census-data/search-by-area>

² <https://www.abs.gov.au/ausstats/abs@.nsf/mf/2033.0.55.001>

³ <https://www.abs.gov.au/statistics/labour/employment-and-unemployment/labour-force-australia/latest-release>

⁴ <https://www.nationalskillscommission.gov.au/topics/small-area-labour-markets>

⁵ <https://www.abs.gov.au/census/about-census/australian-census>

- Lower proportion of people with tertiary education compared to QLD as a whole

Using all of the community breakdowns listed in Table 3.1, these communities range from somewhat disadvantaged (Boonah and Beaudesert Statistical Area 2, Logan LGA) to around the average socioeconomic advantage/disadvantage (Scenic Rim LGA) compared to Australia overall.

The indicators outlined in **Table 3.1** are chosen to provide some insight into the vulnerability of a community – including its ability to adapt to environmental stresses. It is important to consider these matters when evaluating a project to ensure some consideration of equity. **Sections 5.1, 5.4, 5.5.3** and **5.5.3.2** provide information about how community vulnerabilities have been considered in this assessment.

This Project will be implemented in proximity to a community that has potential for increased vulnerabilities based on higher unemployment levels and larger proportions of older people but it is noted that improving employment opportunities in this area (such as will occur as a result of this Project) will have a significant benefit for community health.

3.4 Population health

The health of the community is influenced by a complex range of interactive factors including age, socio-economic status, social capital, behaviours, beliefs and lifestyle, life experiences, country of origin, genetic predisposition and access to health and social care. The health indicators available and reviewed in this report (**Table 3.2**) generally reflect a wide range of these factors.

The population in the area surrounding the Project site is relatively small and health data are not available that specifically relates to this population.

Data for populations in Queensland are available for Local Government Areas – Scenic Rim and Logan LGA have been summarised in the table below.

Table 3.2 presents a summary of the general population health considered relevant to the area. The table presents available information on health-related behaviours (i.e. key factors related to lifestyle and behaviours known to be of importance to health) and indicators for the burden of disease within the community compared to Queensland.

Table 3.2: Summary of baseline data on health and wellbeing data

Parameter/measure	Scenic Rim LGA	Logan LGA	Queensland	Greater Brisbane	Rest of QLD
Health behaviours					
Met vegetable consumption guidelines – adults ²	10.2%	6.3%	8.7%	7.7-8.5%	--
Met fruit consumption guidelines – adults ¹ (2017/18)	49.0%	48.7%	50.6%	50.8%	50.5%
Increased risk of alcohol-related harm – adult ¹ (Consume >2 std drinks per day on average) (2017/18)	19.7%	14.8%	18.2%	16%	20.3%
Adult population defined as overweight but not obese ¹ (2017/18)	34.2%	32.7%	34.6%	34.5%	34.7%
Adult population defined as obese ¹ (2017/18)	35.4%	39.1%	32.7%	31.9%	33.5%
Adults - current smokers ¹ (2017/18)	13.4%	17.4%	15.8%	13.8%	17.5%
Sufficient levels of physical activity – adults ² (2019/20)	61.8%	50.8%	58.3%	--	--
Baseline health (as age standardised rate per 100,000)					
Premature mortality - all causes (2016-2020) ¹	266.2	342.7	246.1	232.8	253.6
Premature mortality – cardiovascular (2016-2020) ¹	51.2	61.8	43.9	41.5	44.6
Premature mortality – respiratory disease (2016-2020) ¹	16.7	22.3	16.2	14.8	17.1
Hospitalisations – cardiovascular disease (2019/20) ¹	1,329.2	1,959.1	1,668.8	1,587.3	1,734.8
Hospitalisations – respiratory disease (2019/20) ¹	1,484.0	2,473.1	1,683.1	1,723.0	1,647.5
Adults ever diagnosed with asthma ¹	12.6	12.6	11.8	11.4	12.2

Notes:

1 Age standardised ratio - data from the Social Health Atlas of Australia, Queensland: <http://phidu.torrens.edu.au/social-health-atlases>

2 Data from 2022 Chief Health Officer Report https://public.tableau.com/views/QSAS_Regional_By_Region/Adult?:embed=y&display_count=no&showShareOptions=false&showVizHome=no

Shading relates to comparison against Queensland (potential): more vulnerable, less vulnerable. If a cell is not shaded, it is not considered to be sufficiently different from the data for Queensland.

Review of the data presented in **Table 3.2** indicates that the population in the Scenic Rim LGA, who are assumed to be representative of the population potentially impacted by the Proposal, are generally similar to the larger Queensland population.

The Scenic Rim LGA data indicate a community that has:

- Higher rates of vegetable consumption (potential for lower vulnerability than Queensland overall)
- Lower rates of smoking (potential for lower vulnerability than Queensland overall)
- Higher rates of people who have adequate exercise (potential for lower vulnerability than Queensland overall)
- Lower rates of hospitalisations for cardiovascular or respiratory issues (potential for lower vulnerability than Queensland overall)
- Slightly higher levels of overweight/obese people (potential for higher vulnerability than Queensland overall)
- Slightly lower levels of fruit consumption (potential for higher vulnerability than Queensland overall)
- Slightly higher rates of asthma in adults (potential for higher vulnerability than Queensland overall)
- Slightly higher premature mortality rates (potential for higher vulnerability than Queensland overall)

Sections 5.1, 5.4, 5.5.3 and 5.5.3.2 provide information about how community vulnerabilities have been considered in this assessment.

Section 4. Assessment of health impacts – air: methodology

4.1 Approach

The primary type of emissions that need to be assessed for this risk assessment are emissions to air.

This section explains the methodology adopted to assess the potential for health impacts related to emissions to air relevant to the construction and operation of the facility.

The assessment presented has relied on the information provided in the air quality assessment prepared for the Proposal:

- Katestone (2026), Bromelton Energy and Recovery Centre, Air quality assessment. Rev C Dated February 2026.
- Air Quality Modelling Output Spreadsheets provided by Katestone (received 15/09/25).

The overall approach is shown in **Chart 4.1**.

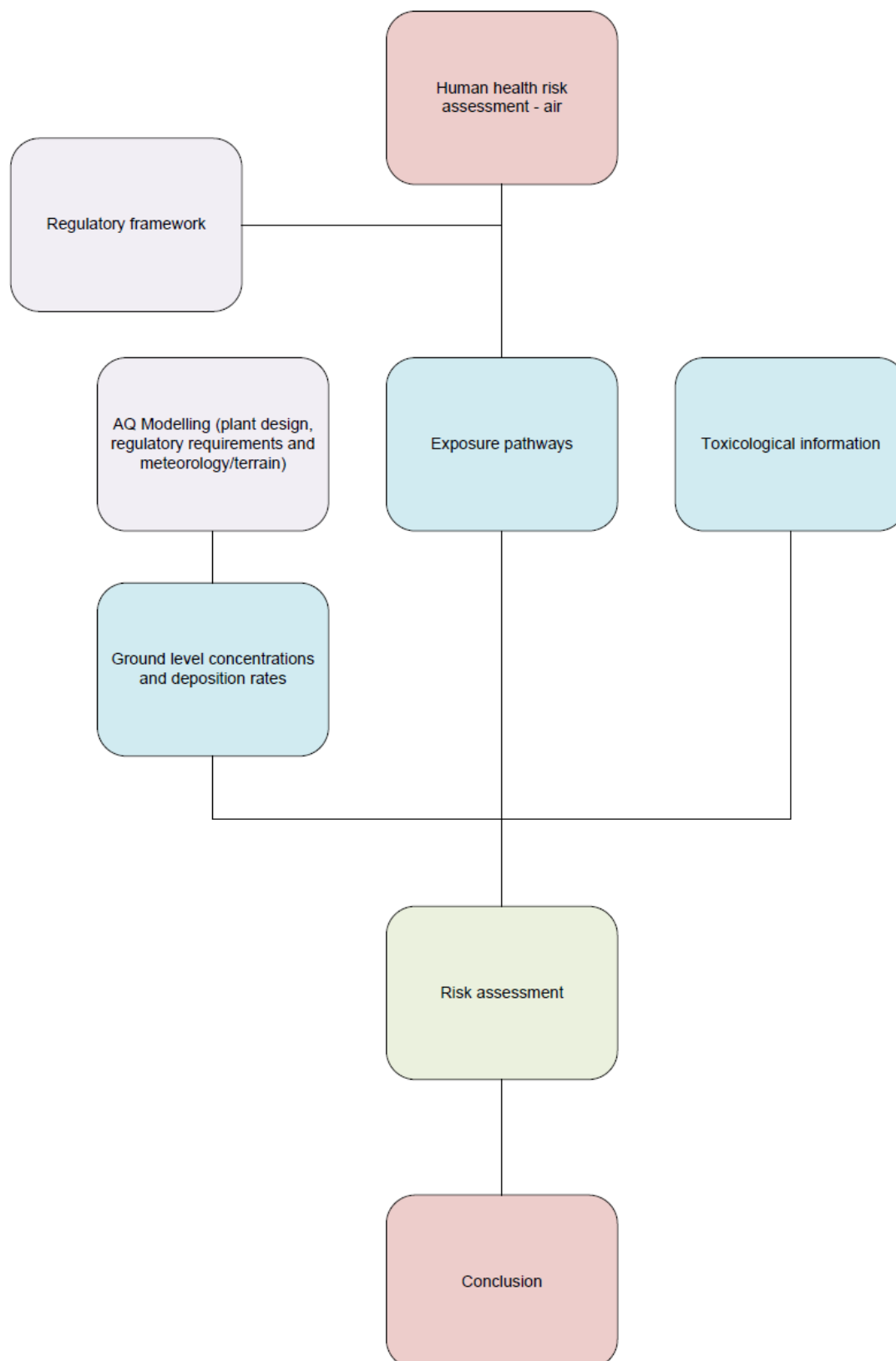


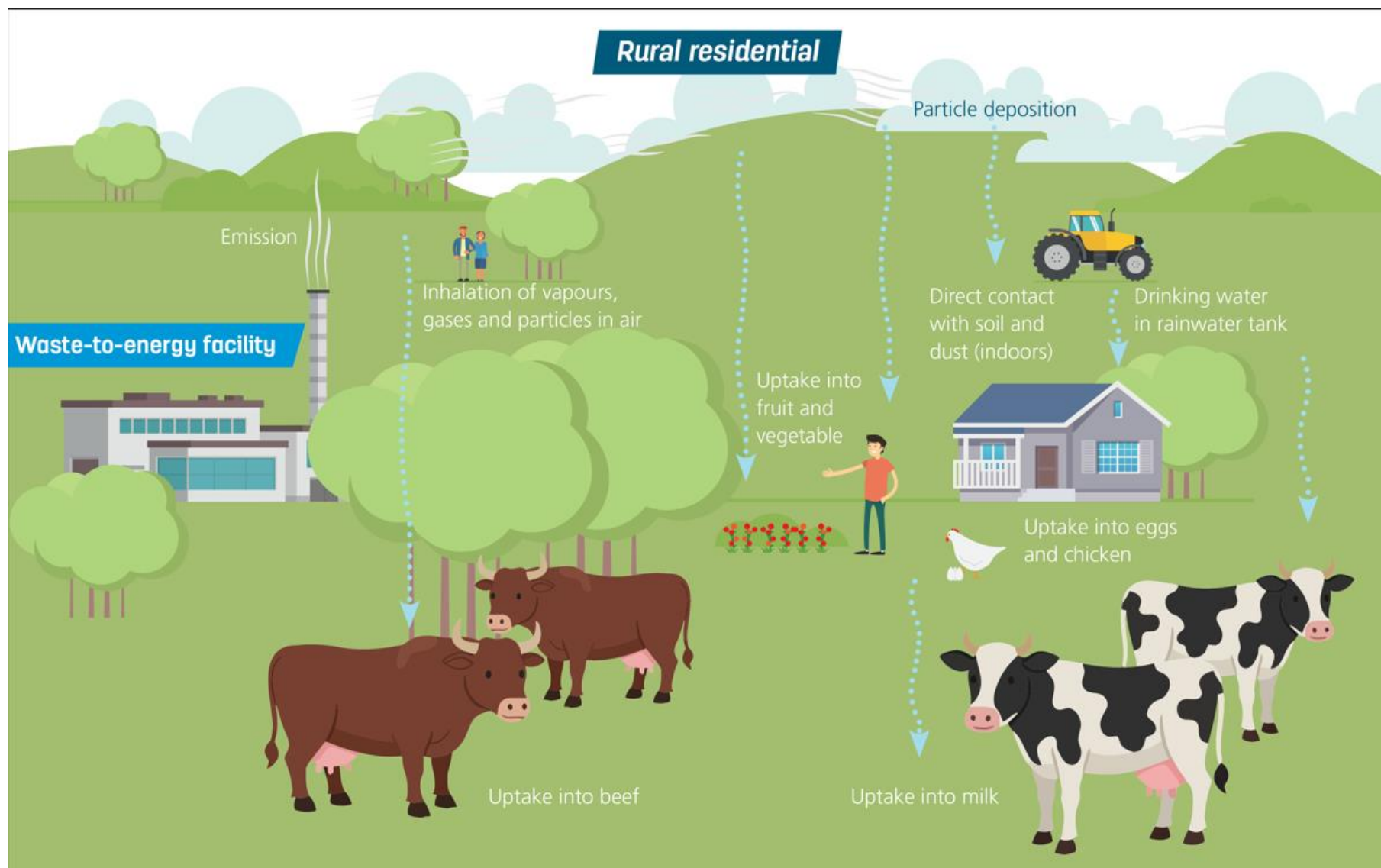
Chart 4.1: Approach to quantitative risk assessment for air emissions

4.2 Conceptual site model

Understanding how a community member may come into contact with pollutants released in air emissions from the proposed EfW facility is a vital step in assessing potential health risk from these emissions.

A conceptual site model provides a holistic view of these exposures, outlining the ways a community may come in contact with these pollutants. An illustration of the conceptual site model for this assessment is provided in **Figure 4.1**.

Figure 4.1: Conceptual site model (illustrative only, not to scale)



There are three main ways a person may be exposed to a chemical substance emitted from the proposed EfW facility (or any other industrial facility):

- inhalation (breathing it in)
- ingestion (eating or drinking it – direct or indirect)
- dermally (absorbing it through the skin).

The inhalation pathway is the first to be assessed. There are a few steps in determining whether such pollutants would be present in the area surrounding the proposed facility:

- Estimating the levels in the stack prior to discharge
- Combining the stack concentrations with the local weather (wind, rain) to work out how much could reach the local neighbourhood.

The following figures (**Figures 4.2 and 4.3**) illustrate how the emissions get mixed into the atmosphere.

Gases (and fine particles) are emitted at around 130°C from the stack and they are pushed out of the stack using fans (at some speed), so these gases (and fine particles) rise up into the air from the top of the stack. This is because gases that are hot and travelling at a faster speed rise above the air surrounding the stack. This can be seen in **Figure 4.2**.

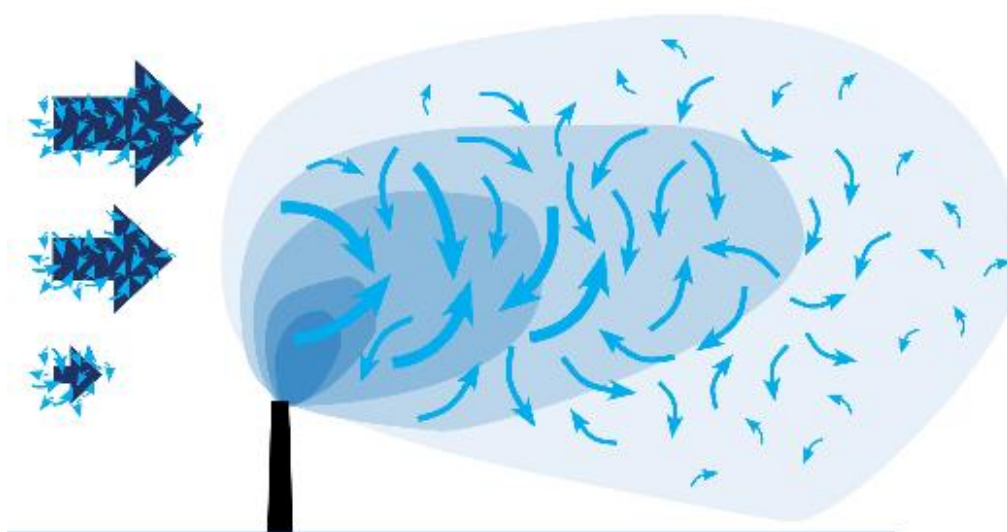
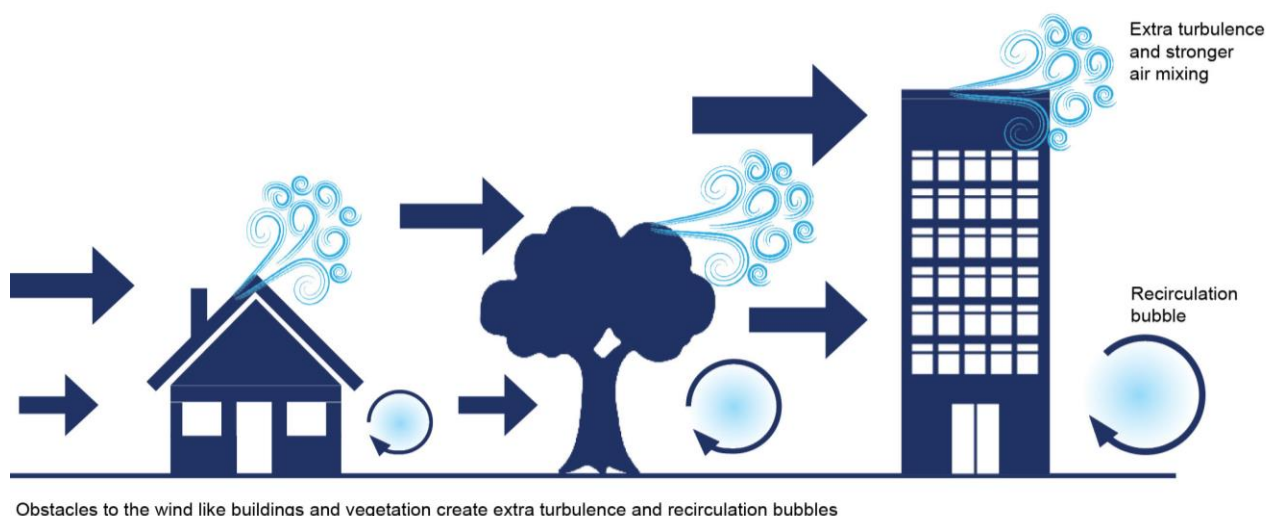


Figure 4.2: Turbulence in the air, how it mixes and dilutes pollutants (NSW Chief Scientist 2018)

As the gases (and fine particles) cool and slow down they begin to interact with the wind above the stack (95 m high). This mixes the gases (and fine particles) into the atmosphere decreasing the actual concentration present in any particular location. **Figure 4.2** shows most of the pollutants remain up in the atmosphere away from where people could breathe them in. However, small amounts do eventually reach ground level (the small arrows at the bottom of the plume near the ground).



Obstacles to the wind like buildings and vegetation create extra turbulence and recirculation bubbles

Figure 4.3: Turbulence in the air and how it is affected by buildings and vegetation (NSW Chief Scientist 2018)

The air dispersion modelling determines what proportion of the amount in the stack could reach ground level. Such modelling looks at worst-case weather characteristics as well as the turbulence due to buildings and terrain (as shown in **Figure 4.3**). The approach taken in the modelling ensures that the amount that could reach ground level in areas where people live or work neighbouring the proposed facility is not underestimated. It is the ground level concentration of each of the relevant pollutants that is then used to assess potential for health impacts.

For some of the chemicals emitted from the proposed EfW facility, inhalation is the only route of exposure. This is due to the substance's chemical properties, which make the other pathways inconsequential. Gases such as NO₂, SO₂, HCl, HF and CO can be considered in this category.

Other chemicals are attached to particles. Fine particulate matter is normally classified as particles less than 10 micrometres (i.e. PM₁₀) and particles less than 2.5 micrometres (i.e. PM_{2.5}). For combustion sources like the proposed facility, most of the particles that could be released would be classified as PM_{2.5}. Particles of this size are most likely to stay suspended in the air. However, it has been assumed that these particles may also fall from the air onto the ground. These particles (and anything attached to them) can then mix with soil. Once being present in soil such contaminants can then be ingested either directly through incidental consumption of soil/dust or indirectly through food grown or raised in the soil (fruit, vegetables and eggs, meat or milk). Skin contact with the soil is also possible.

To allow these aspects to be considered, the air dispersion modelling estimates the rate at which particles in the emissions could fall out of the sky (due to gravity) or get washed out of the sky (due to rain). It is this deposition rate that is then used to estimate the amount of each chemical that may be attached to particles which could get into soil or water around the facility.

The deposition rate allows calculation of potential accumulation in soil which people might come into contact with or where they might grow fruit and vegetables. This covers the other two pathways of exposure – ingestion or dermal contact (i.e. skin contact).

The deposition rate also allows calculation of potential runoff of particles from a roof into a rainwater tank for a house nearby.

It is noted that this assessment assumes that these particles (with attached chemicals) will be present in the atmosphere where people can breathe them in and that they will fall out of the atmosphere onto the ground where they mix into the soil or water. This is a conservative approach as it does result in double counting (i.e. where some of the particles are assessed as both being in the air people breathe and falling onto the ground to mix into the soil or water).

In this instance, metals and organics that are bound to the particulate matter that may fall out and deposit onto the ground have been assessed for inhalation, ingestion and dermal contact with soil and ingestion and dermal contact with water.

Table 4.1 lists the substances considered in the EfW emissions and the exposure pathway/s of potential concern for each one. These substances have been evaluated in this assessment because they have been identified in guidance in Europe as those that are relevant for such facilities (EC 2006; EU 2019; JRC 2018).

Studies of combustion processes show that criteria air pollutants such as NO₂, SO₂, CO and PM are emitted from all such processes. Burning (i.e. combustion) of many fuels also results in particles with metals attached. Metals are present in fuels such as coal, natural gas, petrol, diesel as well as wastes, as will be used as fuel in this facility. When such fuels are burned, the metals can attach to the fine particles of ash that are formed. Pollutants like acid gases (hydrogen chloride and hydrogen fluoride) are also commonly present in emissions from combustion because fuels contain chlorine, fluorine, or chloride ions or fluoride ions (i.e. salts like sodium chloride are commonly present in soils, rocks, and fuels like coal, natural gas, petrol, diesel as well as waste plastics, such as PVC). Dioxin-like compounds are also commonly formed in all combustion processes (both in nature (e.g. bushfires) and man-made (e.g. fireworks, combustion processes)) due to the presence of larger organic molecules (for example, proteins from plants and animals) and chloride ions in the fuels which combine during the burning process.

Table 4.1: Substances and routes of exposure

Substance	Route of exposure
Nitrogen dioxide (NO ₂)	Inhalation only as these are gases
Sulfur dioxide (SO ₂)	
Hydrogen chloride (HCl)	
Hydrogen fluoride (HF)	
Carbon monoxide (CO)	
Total volatile organic compounds (TOC) (as benzene or as formaldehyde)	
Ammonia	Inhalation only as these particulates are very small and will remain suspended in air. It is noted that other exposure pathways have been assessed for the individual chemical substances bound to these particles. Exposure to the individual chemical substances that may reach the ground attached to these particles needs to be assessed. Exposure to the particles themselves via direct contact is not an important pathway as it is similar to direct contact with soil or sand.
PM ₁₀	
PM _{2.5}	
Antimony	Inhalation of these pollutants adhered to fine particles Ingestion and dermal contact with these pollutants when particles with them adhered are deposited to soil or water
Arsenic	
Cadmium	
Chromium	

Substance	Route of exposure
Cobalt	Ingestion of produce grown in soil potentially impacted by these pollutants (i.e. homegrown fruit and vegetables and eggs – where the pollutants can be taken up/bioaccumulated into plants and animals). As the area surrounding the site has some farms, the raising of livestock for meat or milk is permitted and has also been assessed.
Copper	
Lead	
Manganese	
Mercury	
Nickel	
Thallium	
Vanadium	
Dioxins-like compounds	
Per/polyfluoroalkyl substances (PFAS)	
Polycyclic aromatic hydrocarbons (PAHs)	

The full set of exposure pathways considered in this assessment is shown in **Figure 4.1**.

The assessment has relied on the Katestone Air Quality Assessment (AQA) (Katestone 2026). This assessment has relied on the data provided directly from Katestone.

The characterisation of risk follows the general principles outlined in the enHealth document Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a).

4.3 Existing Air Quality

Katestone (2026) provides a detailed assessment of local air quality relevant for the site.

The closest air quality monitoring stations (operated by DETSI) relevant for this proposed facility are Flinders View and Springwood, which are located approximately 23 km to the north of the site (**Figure 4.4**). Information about the air quality monitoring stations is available at the DETSI website⁶.

These stations collect data for NO_x, SO₂ and particulate matter (PM₁₀, PM_{2.5}). These stations also collect meteorological data. CO is not monitored at these stations. Data for CO from the South Brisbane and Woolloongabba stations have been used for this assessment.

⁶ <https://apps.des.qld.gov.au/air-quality/>

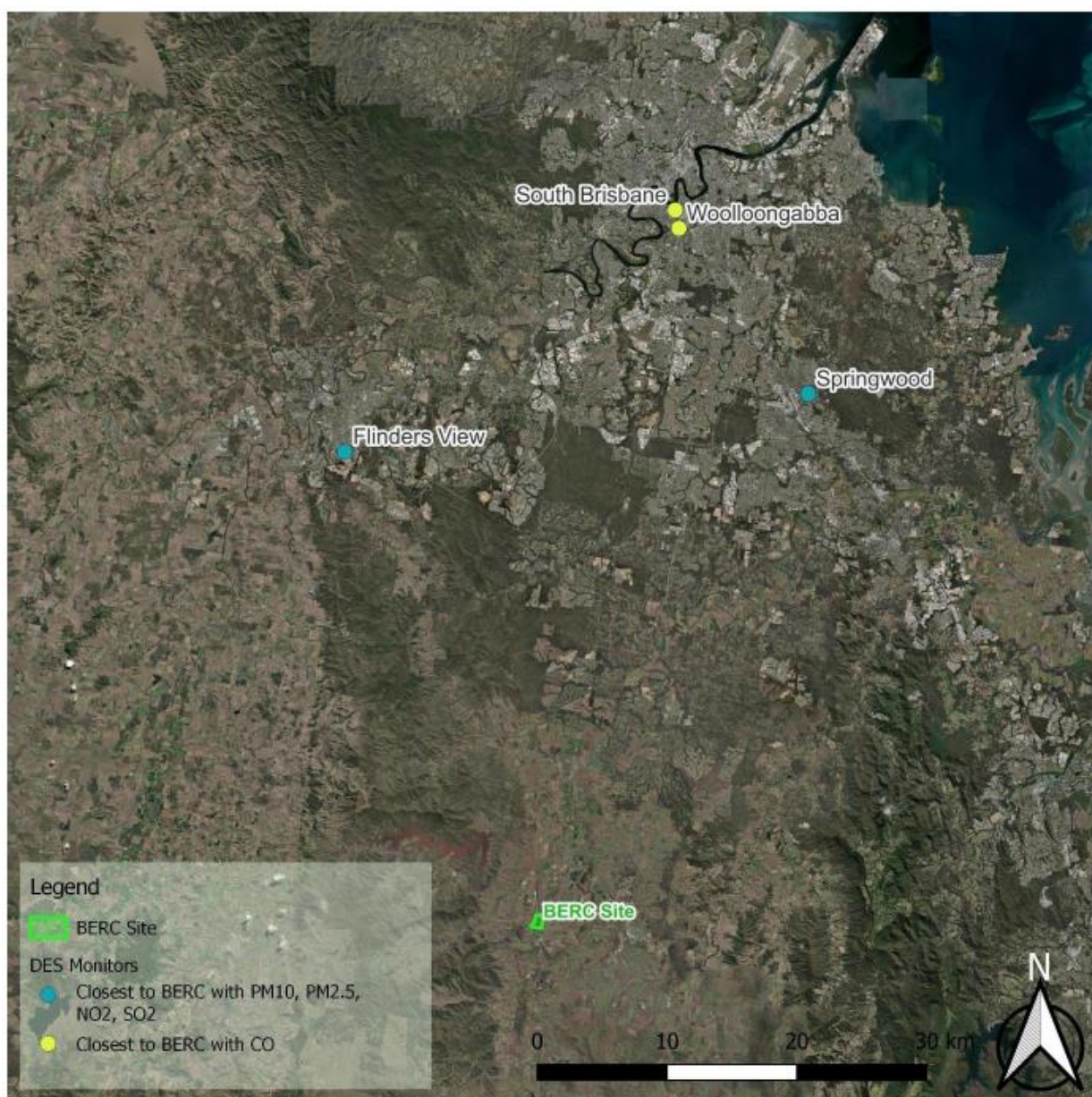


Figure 4.4: Monitoring station location

It is important to note that these criteria pollutants are always present in ambient air in cities, rural areas and in remote areas. There are many sources of such chemicals from human activities. All combustion processes will generate such pollutants. Consequently, establishing existing concentrations without the newly proposed plant is critical to interpreting the potential for impact of the new facility.

These background concentrations as measured by government monitoring stations are used to ensure that all relevant existing sources (whether large facilities or from vehicles or homes) are considered in the assessment.

Background concentrations for the area adopted within Katestone (2026) are listed in **Table 4.2**. The highest concentration for each air pollutant over the five years (2017-2022) (relevant to each

averaging period) has been used as the background value in this assessment (unless otherwise stated).

Table 4.2: Background air concentrations for criteria air pollutants.

Pollutant	Averaging period	Criteria ($\mu\text{g}/\text{m}^3$)	Background concentration used in this assessment ($\mu\text{g}/\text{m}^3$)
PM ₁₀	24 hour	50	20.6
	Annual	25	19.9
PM _{2.5}	24 hour	25	6.7
	Annual	8	5.9
NO ₂	1 hour	250	18.5
	Annual	62	15.4
SO ₂	1 hour	286/570	2.9
	24 hour	57/229	3.8
	Annual	57	3.1
CO	8 hour	11,000	375

4.4 Modelling

4.4.1 General

Air dispersion modelling was undertaken to predict the concentration of emissions from the proposed EfW facility. The modelling was undertaken using the CALPUFF model (version 7.2.1). The modelling covered a 20 km x 20 km domain to ensure that dispersion of the emissions across a wide area was considered (Katestone 2026).

The model uses the following matters to predict the ground level concentrations and deposition of pollutants everywhere within the defined study area (the modelling grid), and also at all the discrete receptor locations (Katestone 2026):

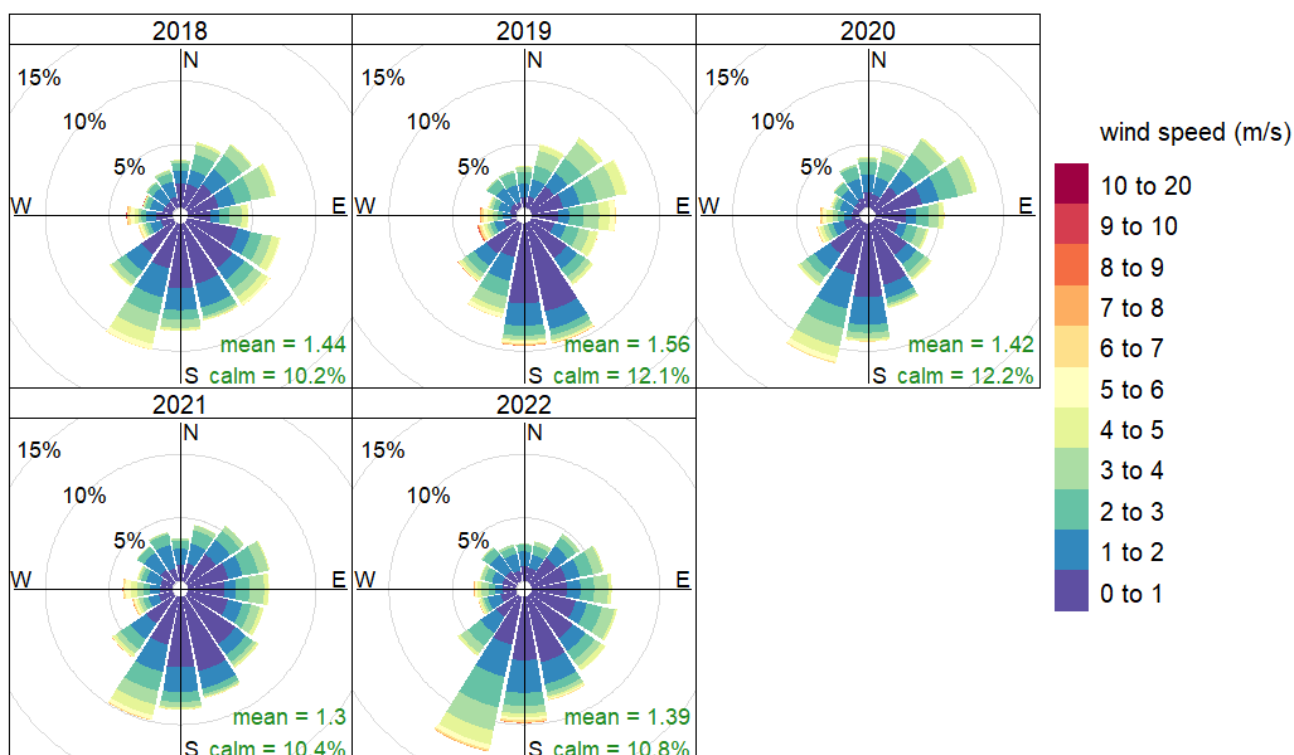
- local terrain (topography and buildings)
- meteorological data
- plant design (for example, stack location and height)
- air emissions estimates (stack concentrations).

Modelling evaluated potential concentrations for relevant chemicals at ground level across a grid of approximately 3 km x 3 km centred on the facility. The grid used a 25 m resolution.

4.4.2 Meteorology

The information describing this grid in the model included all the relevant local terrain (i.e. low and high points in the land surrounding the proposed facility) and meteorological data.

Wind patterns around the facility were described by the wind roses shown in **Figure 4.5**. The windroses show that there is little variation in the pattern of wind in this area with dominant winds being those that blow toward the south.



Frequency of counts by wind direction (%)

Figure 4.5: Annual windroses for Drumley Street, Beaudesert (2018-2022) (BOM Met Station)

Katestone (2026) reported that the most relevant meteorological data for this assessment was that for 2018. The calendar year 2018 has been used for the modelling as it is judged to be the year with the least variation from the mean when averaged across all of the parameters considered.

Both TAPM and CALMET were used to generate the meteorological data for use in the air dispersion modelling for this assessment.

4.4.3 Stack Parameters

The stack parameters (i.e. plant design parameters) considered in the dispersion modelling scenarios are summarised below in **Table 4.3**.

The engineering of the stack and the way the combustion chamber will operate are key considerations in the modelling of the emissions from the plant. How the emissions disperse into the atmosphere is affected by how high the stack is, how fast the air moves out of the stack and the temperature of the emissions. Design of the stack has been engineered to optimise mixing.

The stack parameters used in the modelling are shown in **Table 4.3**.

Table 4.3: Stack parameters assumed for modelling emissions

Parameter	Value	Units
Single line		
Stack height	95	m
Exhaust diameter	2.67	m
Exhaust temperature	130	°C
Exhaust velocity	20	m/s
Moisture content	18	% by volume
Oxygen content	7	% by volume (wet)
Flow rate	403,955	m ³ per hour (wet, actual)
Flow rate (dry, NTP, 11% O ₂ with 10% additional flow rate)	308,289	Nm ³ per hour (@ 11% O ₂ , dry)
Two lines (single stack)		
Exhaust diameter (effective)	3.8	m
Exit velocity	20	m/s
Flow rate (dry, NTP, 11% O ₂ with 10% additional flow rate)	616,579	Nm ³ per hour (@ 11% O ₂ , dry)

The plant will have two lines and each will have a stack. Each stack has a diameter of 2.67 m and a height of approximately 95 m.

How the combustion chamber is operated will also affect how the flue gas moves through the plant. The waste fuel will have different heat generating capacities depending on what is present in the waste, or how wet it is. It is this calorific value which changes how much and how quickly the waste can move through the chamber. If it has a large calorific value, it can easily generate heat and will burn more quickly, and so the material can move through the chamber a little more quickly and vice versa.

The characteristics of the waste and how it moves through the combustion chamber (i.e. calorific value etc) determine the load point in the chamber at any moment in time. The system will be designed to control the calorific value/amount of waste/speed of movement through the chamber to ensure the system operates at or near 100% thermal input and within the optimal zone (i.e. at or near to Load Point 1, LP1) for the majority of time. The combustion chamber will automatically shut down if a situation arises where the calorific value for the waste causes the chamber to run too hot or too cold or requires the waste to move through the system too fast or too slow. Such facilities are designed to operate within a design envelope with built in shutdown triggers.

The operation of the combustion chamber is kept within the optimal range (for calorific value, speed of the conveyor and amount of waste) as much as is possible (referred to as Load Point 1 (LP1) in Katestone (2026)).

The differences in stack parameters between the range of load points at which the combustion chamber can operate arise because the flow rate through the plant depends on the energy content of the waste being burned at a particular time.

Almost all of the time, the plant will burn waste within the allowable calorific value range and material will move through the chamber at the target speed and volume to generate the target temperature and allow electricity to be generated. However, at times the energy content of the waste will be a bit lower than average and this will result in the automatic combustion control system requesting a higher flow rate of waste for combustion in order to maintain the thermal input and a steady boiler steam generation rate. This will also result in a slightly higher flue gas flow rate. At other times, the energy content of the waste will be a bit higher than average and this will result in the automatic combustion control system requesting a lower flow rate of waste for combustion in

order to maintain the thermal input and a steady boiler steam generation rate. This will also result in a slightly lower flue gas flow rate.

The plant will be designed to initiate auxiliary fuel firing or a shutdown if combustion chamber operation within these load points cannot be maintained. Overall, it is expected that the plant will operate around LP1 for around 90% of the time, and between LP1 and the other valid load points the rest of the operational time. The average case modelling assumes operation around LP1.

Shutdowns are needed from time-to-time for to allow for routine maintenance. The plant will maintain airflow through the combustion chamber and will continue to operate the air pollution control systems during a shutdown. An emergency diesel generator will be installed to ensure this will occur even in a blackout. Also, during a shutdown, the first step is to stop feeding waste into the combustion chamber so emissions from waste do not continue at the normal rate as the amount of waste in the chamber rapidly decreases.

This means that, even during a shutdown, the emissions from the facility should not be more than modelled in this assessment as the pollution control equipment will continue to operate unless there is a component failure within the air pollution control system itself, which has instigated the shutdown of the impacted boiler line.

In regard to the start-up phase, no waste will be permitted to enter the combustion chamber until the chamber has reached the minimum required operating temperature of at least 850 degrees Celsius for at least 2 seconds residence time after final combustion air injection.

4.4.4 Stack Concentrations

The final step in the process for modelling ground level concentrations in relevant locations is to determine how to estimate what concentrations of each chemical will be assumed to be in the stack to allow the calculation of emissions and ground level concentrations after mixing in the atmosphere.

There are a number of ways to estimate what stack concentrations may be relevant for a facility that has not yet been constructed including:

- Assuming stack concentrations will be at the levels specified in legislation or policy relevant for the location where the plant is proposed to be constructed (i.e. worst-case)
- Assuming stack concentrations will be similar to those measured at an operating facility that uses similar waste types for fuel and which has similar pollution control technology.

In this case, the legislation/policies relevant in Queensland include:

- *Environmental Protection Act* 1994
- *Environmental Protection Regulation* 2019 (including Environmental Protection Policy (Air)) (Queensland Government 2019b)
- Energy from Waste Policy (the EfW Policy) 2021 (Queensland Government 2021a)
- Energy from Waste Guideline (the EfW Guideline) 2021 (Queensland Government 2021b)
- European Union Industrial Emissions Directive (EU IED) (EU 2010)
- European Union Best Available Techniques for Waste Incineration (BREF) (EU 2019)

For this assessment, the stack concentrations used in air quality modelling were those based on the most recent European values as required by the EfW Policy in Queensland. This is a conservative approach as, when a plant operates, the actual stack concentrations will be lower than these limit

values so emissions from the facility into the surrounding environment will be lower than assessed here.

This approach is also preferable, as it means that the plant design and technology can be specified per recent European plant designs that are proven to meet the suite of stringent requirements when burning such materials and the operator can be confident that the facility will operate in line with expectation.

For this facility, the stack concentrations for this facility have been estimated based on an understanding of the engineering of all the equipment that will be installed, the requirements of all the legislation/policies and the type of waste that will be used as fuel. The plant design stack emission concentrations that have been assumed in the air quality modelling for the Project are outlined in **Table 4.4**. The stack concentrations for 1-hour and 24-hour averaging periods are generally similar to the **IED (2010)** and the **BREF-WI (2019)** values as these are the relevant sources for legislation/policy based stack concentrations and meeting these requirements is the benchmark used for plant design as required in the EfW Policy (EU 2010, 2019).

Table 4.4: Modelled stack emission concentrations (EU 2019)

Pollutant	Units @ 11% O ₂ dry	Design/modelled concentrations – LP1	
		Daily average	Short term (1/2 hour)
CO ⁽³⁾	mg/Nm ³	50	100
TOC (VOC) ⁽⁴⁾	mg/Nm ³	10	20
Dust/TSP	mg/Nm ³	5	30
HCl	mg/Nm ³	6	60
HF	mg/Nm ³	1	4
SO _x	mg/Nm ³	30	200
NO _x (calculated as NO ₂)	mg/Nm ³	120	400
NH ₃	mg/Nm ³	10	NA
Hg	mg/Nm ³	0.02	NA
Cd+Tl	mg/Nm ³	0.02	NA
Sb + As + Pb + Cr + Co + Cu + Mn + Ni + V	mg/Nm ³	0.3	NA
Dioxin-like compounds (I-TEQs) (dioxins and furans only)	ng/Nm ³	0.04	NA
Dioxin-like compounds (I-TEQs) (dioxins and furans + dioxin-like PCBs)	ng/Nm ³	0.06	0.1
Polycyclic aromatic hydrocarbons (PAHs) as BaP equivalents	µg/Nm ³	0.2	NA
Per/polyfluoroalkyl substances (PFAS) ¹	ng/Nm ³	9.5	NA

Notes:

NA = not applicable

1 = stack concentrations for PFAS are based on Bjorklund et al. 2024 as discussed in **Section 5.7**

The emissions considered in the AQA include the following pollutants:

- oxides of nitrogen (NO_x)
- sulfur dioxide (SO₂)
- carbon monoxide (CO)
- particulates as PM₁₀ and PM_{2.5}
- hydrogen fluoride (HF)
- hydrogen chloride (HCl)
- ammonia (NH₃)

- metals (cadmium, thallium, mercury, antimony, arsenic, lead, chromium (assumed to be 100% chromium VI as this is the most toxic form), cobalt, copper, manganese, nickel, vanadium)
- volatile organic compounds (assumed to all be present as benzene or all present as formaldehyde)
- dioxins and furans (i.e. dioxin-like compounds)
- polycyclic aromatic hydrocarbons.

Combining the assumed stack concentrations and the engineering parameters for the stacks allows an emission rate to be calculated for each pollutant. These values for LP1 conditions are shown in **Table 4.5**.

Table 4.5: Modelled emission rates based on stack concentrations and stack parameters

Pollutant	Daily average (g/s)	Short term (1/2 hour) (g/s)
CO	8.564	12.85
TOC (VOC) (as benzene)	1.713	2.569
TOC (VOC) (as formaldehyde)	1.713	2.569
Dust/TSP	0.856	2.997
HCl	1.028	5.652
HF	0.171	0.428
SO _x	5.14	19.7
NO _x (calculated as NO ₂)	20.55	44.53
NH ₃	1.713	NA
Hg	0.00343	NA
Cd	0.00203	0.00303
Tl	0.0014	0.00209
As	0.00158	0.00192
Co	0.00128	0.00141
Cr (total)	0.00516	0.00683
Cu	0.00452	0.0076
Mn	0.00772	0.0111
Ni	0.00622	0.00658
Pb	0.0206	0.0489
Sb	0.00136	0.00498
V	0.00064	0.000771
Dioxin-like compounds (I-TEQs) (dioxins and furans only)	0.00000000685	NA
Dioxin-like compounds (I-TEQs) (dioxins and furans + dioxin-like PCBs)	0.0000000103	NA
Polycyclic aromatic hydrocarbons (PAHs) as BaP equivalents	0.0000000343	NA
Per/polyfluoroalkyl substances (PFAS)	0.0000013	NA

Notes:

NA = not applicable

1 = emission rates for PFAS are based on concentrations measured in Bjorklund et al. 2024 as discussed in **Section 5.7** and the stack parameters etc for the proposed facility as discussed in **Section 4.4.3**

In regard to oxides of nitrogen, the most important form that needs to be considered in this risk assessment is NO₂. Katestone (2026) notes that the modelling assumed 30% conversion of NO_x to NO₂.

As noted above, the scenarios assessed in this health risk assessment use the maximum stack concentrations based on legislation/policy only, whereas in practice the actual emissions will be less. This is because, to comply with licence limits set based on legislation/policy, the equipment must be operated below those limits – otherwise ability to comply may be compromised.

There are no scenarios for which risk calculations have been undertaken based on measured stack concentrations from reference facilities. This is because the stack concentrations based on legislation/policy limit values will always be higher than the actual measured/expected concentrations, so if risks are acceptable for the legislation/policy limit based concentrations then they will also be acceptable (and lower) for the actual measured/expected concentrations.

There are hundreds of energy from waste facilities in Europe. These facilities all use a mix of commercial/industrial waste and residual from domestic waste which is what is proposed for this facility. No unusual wastes that could add extra amounts of a particular pollutant or additional pollutants are to be used as fuel at this site.

Ramboll (Technical Advisor to Cleanaway on Energy from Waste responsible for engineering design for this facility) has provided data to work out the proportions of each metal likely to be present in the emissions for such a plant. These proportions have then been used to determine concentrations for each metal individually for use in the air quality modelling and these risk calculations. The proportions are shown in **Table 4.6**.

Table 4.6: Metal distribution provided by Ramboll

Metal	Clean gas		Fly ash	
	Cd and TI	Other metals	Cd and TI	Other metals
Antimony (Sb)	--	7%	--	0.053%
Arsenic (As)	--	3%	--	0.013%
Cadmium (Cd)	59%	--	59%	--
Cobalt (Co)	--	2%	--	0.005%
Chromium (Cr)	--	10%	--	0.065%
Copper (Cu)	--	9%	--	0.12%
Lead (Pb)	--	40%	--	1.1%
Manganese (Mn)	--	15%	--	0.13%
Nickel (Ni)	--	12%	--	0.014%
Thallium (TI)	41%	--	41%	--
Vanadium (V)	--	1%	--	0.005%

4.4.5 Scenarios

As already discussed, the air quality modelling has been undertaken using the following information to allow the calculation of ground level concentrations for each pollutant type at each point on the grid (i.e. every 25 m) across a 10 km² area (i.e. approximate 3 km x 3 km) and at each discrete sensitive location as shown on **Figure 4.6**:

- Meteorological data for the area
- Grid across the relevant area (i.e. 20 km x 20 km with cross overs at 0.2 km spacing)
- Stack parameters (stack design and loading information)
- Stack concentrations

The combination of all these different sets of information allows the estimation of ground level concentrations for the various pollutants at the cross over points of the modelling grid (i.e. every 200

m going north south and going east west) as well as at each of the locations of homes or businesses shown in **Figure 4.6**.

The assumed concentrations in the stack for each pollutant, as discussed in **Section 4.4.4**, were combined with the engineering information about the design of the stack (size, exit velocity as per **Section 4.4.3**) for the various scenarios outlined below to determine the emission rates for each chemical. These emission rates were then used in the air quality model to determine ground level concentrations across the grid and at the relevant receptor locations.

The average case or LP1 is the normal, optimal or design load point for the operation and would be expected to occur 90% of the time.

Operation at any of the other load points would be brief but could potentially occur for several hours before returning to the design load point. To consider this, the air quality assessment developed a conservative, hypothetical maximum case.

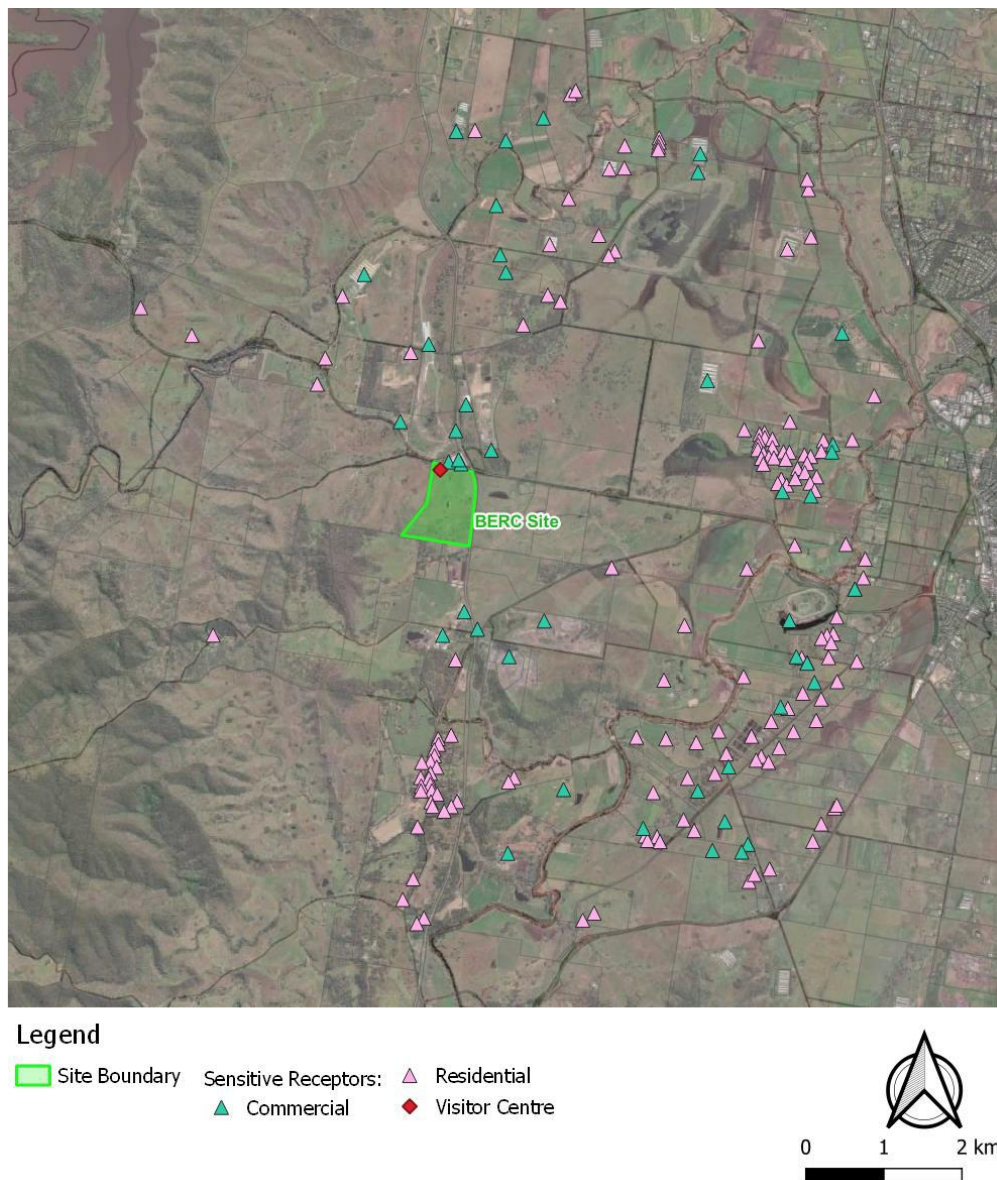


Figure 4.6: Sensitive locations evaluated in the air quality modelling

Modelling included assessing ground level concentrations for three different scenarios. The different scenarios were primarily based on the different averaging periods that are necessary for use in these assessments.

The scenarios include:

- Scenario 1 – this scenario is based on daily average operations at LP1. It uses the following information:
 - Nominal waste throughput
 - BREF (2019) upper *daily average or spot sample average* stack concentration limits
 - Assumes both lines are operating at these stack concentrations
 - Relevant meteorological conditions across the year
 - Stack parameters as discussed in **Section 4.4.3**.

This scenario is used to estimate the **annual average ground level concentrations** for each pollutant type. It takes the in-stack limit concentrations from the European best practice guidance and assumes those concentrations are emitted every hour of the year when the proposed facility is operating in accordance with the average loading (LP1). The modelling of this scenario generates concentrations for all the grid locations and all the discrete receptor locations. These ground level concentrations have been assessed in detail in this risk assessment.

- Scenario 2 – this scenario is used to assess short term non-routine operations. It uses the following information:
 - Nominal waste throughput
 - One line at IED (2010) 100% *short term (30 minute)* stack concentration limits and the second line at BREF (2019) BAT-AEL *upper daily average or spot sample average* stack concentration limits"
 - Assumes both lines are operating with one line at these short term concentrations and the other line at the daily average stack concentrations
 - Assumes this could occur for up to 30 minutes at a time
 - Relevant meteorological conditions across the year
 - Stack parameters as discussed in **Section 4.4.3**.

The modelling of this scenario generates the theoretical upper maximum **1 hour average ground level concentrations** at every grid location and at all discrete receptor locations. These ground level concentrations have been assessed in detail in this risk assessment for the short term exposure situation.

- Scenario 3 – LP2 – this scenario is used to show how concentrations may differ from Scenario 1 for differing load points. It uses the following information:
 - Maximum waste throughput
 - BREF (2019) *upper daily average or spot sample average* stack concentration limits
 - Assumes both lines are operating at these stack concentrations
 - Relevant meteorological conditions across the year
 - Stack parameters as discussed in **Section 4.4.3**.

Emissions from truck movements and exhausts are not expected to have any meaningful impact on overall emissions, with releases from the flue gas stack likely to dominate. Therefore, these non-stack sources have not been considered further (Katestone 2026).

The most relevant information in regard to health impacts from this facility is that related to longer term average concentrations – i.e. annual averages. The emissions expected for the plant over the longer term are those for Scenario 1. The estimated ground level concentrations for Scenario 1 (annual averages) have been assessed for all pathways in **Section 5**. The estimated ground level concentrations for Scenario 2 have been used to assess short term inhalation exposures as per **Section 5.5.3.1**.

Understanding the potential ground level concentrations from this facility in the context of what is already likely to be present in a location is also important. This concept of cumulative exposure has been considered in this assessment. For the criteria pollutants (NO_x, SO_x, CO, PM), government stations provide data on regional air quality – these ambient background levels have been incorporated into the modelling.

Katestone (2026) noted the location of a number of industrial facilities that could contribute pollutants to air in this vicinity. These included Boral Quarries Purga, Bromelton Quarry, Blue Rock Quarry, Gelita Australia and LMS Energy Bromelton (Beauesert) Facility. The Boral and Blue Rock Quarries are the largest emitters from this group but both are located at least 30 km from the site for this facility.

Background concentrations for the most important pollutants (PM, NO_x, SO_x and CO) have been conservatively accounted for by explicitly modelling nearby industry and adding ambient backgrounds from the DETSI ambient air monitoring stations at Springwood and Woolloongabba, both of which are significantly closer to major population centres and industry pollution sources than the Project site (Katestone 2026).

The assessment of the potential for any impacts on the surrounding community has considered the location where maximum impacts from the Project may occur. This location is usually just outside a proposed facility – at the boundary fence or on the road outside.

In addition, impacts in the wider region have also been considered. This assessment has used the estimated ground level concentrations (1 hour and annual average) at the following locations:

- maximum anywhere across the grid in the off-site area (i.e. at the boundary fence/road outside)
- maximum at any residential location
- maximum at any commercial/retail/industrial location
- maximum on-site for visitors to the facility (i.e. visitors to the education centre).

These ground level concentrations have been used in the calculations to determine risks.

Using the location in each category where the ground level concentrations are the highest results in an estimate of risks for the worst-case in each situation. All other locations in each category will have **lower risks** than these “maximum” locations as the concentrations will be lower.

Demonstrating that the risks for these “maximum” locations are acceptable confirms that the risks at all locations in each category are acceptable/negligible.

4.4.6 IBA and APCr residues

The potential for emissions of dust from the IBA/IBAA storage areas were included in the air dispersion modelling undertaken by Katestone (2026).

There are a range of controls proposed to be included in the project to minimise this potential including:

- During the maturation phase, the maturing IBA stockpiles will be contained within a three sided building to limit the potential for fugitive dust emissions from raw IBA and limit the interaction of rainwater with the maturing IBA. The IBA maturation stockpiles will be sprayed with water to optimise the required chemical reactions and limit dust formation (if necessary).
- IBA sorting will occur within the fully enclosed IBA Hall (Sorting Building) to control noise, water and dust emissions. Dust management techniques will include a combination of managing the IBA moisture content (if necessary) and the use of enclosed conveyors. This building would have the following controls in place to manage dust emissions:
 - A fugitive dust management system including a baghouse or cartridge filter type dust collector (with an induced draft fan and vent stack) – to be confirmed during detailed design depending on known dust emissions sources and types
 - The discharge point will be via a short vent stack at the exist of the baghouse / air filter to release cleaned air at an elevation above the IBA sorting plant roof. The proposed emission limit for dust in the cleaned air (i.e. 5 mg/Nm³ dry, 11% O₂) is in accordance with BAT 26 of the BREF-WI (2019)
 - Discharges would be intermittent (i.e. only during plant operation of 10-12 hours per day). Emissions would be insignificant relative to the EfW process stack emissions, and therefore, not considered a licensable discharge point.
- Inert IBAA stockpiles still have a moisture content of ~10% and will be temporarily stored outside and sprayed with rainwater collected from the IBA area to limit dust formation (subject to detailed design, noting that mains water or excess process water would be used in the case that there is no available stormwater).
- Most of the IBAA will have particle sizes ranging from 0.1 mm to 50 mm, depending on the end use of the material in the construction industry (e.g. for making concrete and / or forming a road base). IBAA may contain smaller 'sand' like particles, but it is unlikely to contain any fine particulates (PM₁₀ and PM_{2.5}) that can be carried longer distances. With water sprays to suppress fugitive dust emissions, IBAA is not expected to be carried significant distances.
- Dust suppression on sealed roadways in IBA/IBAA storage area.

The air dispersion modelling included estimates of emissions to air based on handling of these materials from stockpile management and loading trucks as well as wind erosion of stockpiled material even with the expected effectiveness of all of the control measures. This is expected to be extremely conservative.

The results of this aspect of the modelling were incorporated into the overall modelling for the site. The results have been considered in **Section 5**.

Section 5. Assessment of health impacts – air: results

5.1 General

This section provides the assessment of potential health risks from emissions to air from the proposed facility.

The assessment of risk has relied on air modelling presented in the Air Quality Report (Katestone 2026) and follows the risk assessment principles detailed in guidance referenced in **Section 1.3**. This approach requires assessment of:

- how people may be exposed to the emissions to air over short-term (acute) and long-term (chronic) (i.e. exposure assessment)
- the hazards posed by (or toxicity of) the chemicals present in the emissions (i.e. hazard or toxicity assessment)
- calculation of potential risks to health or risk characterisation.

The exposure assessment is designed to be conservative. This is done by making worst case assumptions about how people may be exposed in each of the calculations.

The toxicity assessment is also designed to be conservative. This is done by using toxicity reference values that are relevant for sensitive subpopulations as well as robust adults.

Figure 5.1 presents an overview of the assessment approach detailed in the following sections.

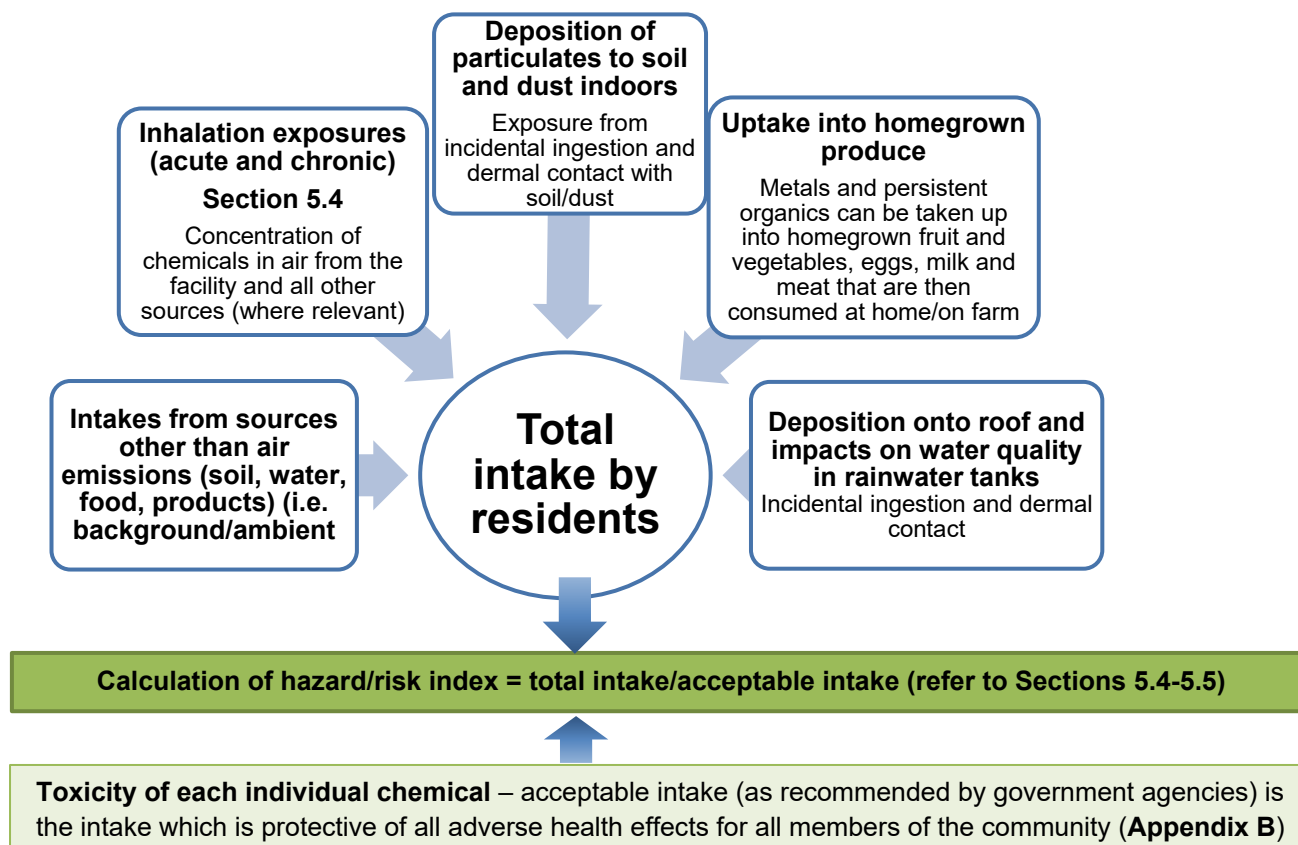


Figure 5.1: Overview of health risk assessment

5.2 Construction

As with most construction projects (residential, industrial, commercial), emissions of dust during earthworks or while the ground is cleared of vegetation as well as emissions from vehicles used on-site are the main issues relevant to health risks during constructions. Such activities and emissions are temporary and sporadic during such projects, so the potential for such emissions can be difficult to predict.

Katestone (2026) undertook a review of the potential for such emissions of dust during construction.

The assessment noted that there was potential for people to be present within 350 m of the boundary of the site and/or 50 m of the route by which construction vehicles would travel within 500 m of the Project site. As a result, a more detailed assessment was triggered.

The more detailed assessment indicated that there was no need for demolition at the site but dust could be generated by the earthworks, construction activities and heavy vehicle movement that would occur during the construction of the facility.

The sensitivity of the area to dust was judged to be medium based on the presence of two industrial facilities within 350 m of the Project site (low sensitivity for people) and the presence of a number of sensitive ecological locations within 250 m of the Project site (medium sensitivity for ecosystems).

A range of mitigation measures have been proposed to be included in the Construction Environmental Management Plan (CEMP) to ensure dust is well managed during construction. These include:

- Construction dust will be managed through a Dust Management Plan integrated with the CEMP which may include the following measures:
 - Record all dust and air quality complaints, identify cause(s), take appropriate measures to reduce emissions in a timely manner, and record the measures taken.
 - Make the complaints log available upon request from a regulating authority.
 - Record any exceptional incidents that cause dust and/or air emissions, either on- or offsite, and the action taken to resolve the situation in the log book.
 - Plan site layout so that machinery and dust causing activities are located away from sensitive receptors, as far as reasonably practicable.
 - Erect solid screens or barriers around dusty activities or the entire Project site that are at least as high as any stockpiles on site.
 - Avoid site runoff of water or mud.
 - Remove materials that have a potential to produce dust from site as soon as possible, unless being re- used on- site. If they are being re- used on- site cover as described below.
 - Cover, seed or fence stockpiles to prevent wind whipping.
 - Only use cutting, grinding or sawing equipment fitted or in conjunction with suitable dust suppression techniques such as water sprays or local extraction, e.g. suitable local exhaust ventilation systems.
 - Ensure an adequate water supply on the site for effective dust/particulate matter
 - Suppression/mitigation, using non- potable water where possible and appropriate.
 - Bonfires and burning of waste materials should not be permitted.
 - Covering or watering exposed stockpiles when not in use
 - Revegetating disturbed areas as soon as is feasible

- Ensuring all fine powder materials such as cement are delivered in closed tankers and stored in silos
- Ensure sand and other aggregates are stored in bunded areas
- Avoid scabbling (roughening of concrete surfaces) if possible
- Minimise drop heights from loading shovels and other loading or handling equipment, use enclosed chutes and cover skips
- Hosing down truck tyres before exiting the site
- Avoiding dry sweeping of large areas, instead using hoses, sprays, or water trucks.
- Use water- assisted dust sweeper(s) on the access and local roads, to remove, as necessary, any material tracked out of the site.
- Undertake daily on- site and off- site inspection, where receptors (including roads) are nearby, to monitor dust, record inspection results, and make the log available to regulatory authority when asked.
- Carry out regular site inspections to monitor compliance with the DMP, record inspection results, and make an inspection log available upon request from a regulating authority.
- Increase the frequency of site inspections by the person accountable for air quality and dust issues on site when activities with a high potential to produce dust are being carried out (such as major excavation works) and during prolonged dry or windy conditions.
- Consideration will also be given to ensuring best practice management of emissions from plant and vehicles during construction including:
 - Ensure all vehicles switch off engines when stationary. There should be no idling vehicles.
 - Minimise the use of diesel or petrol powered generators if possible and use mains electricity or battery powered equipment where practicable.
 - Impose and signpost a maximum speed limit of 25 km/h on surfaced and 15 km/h on unsurfaced haul roads and work areas.

These are mitigation measures which are common for large construction projects and will ensure that dust emissions and other air emissions from the site are well controlled.

5.3 Workers at proposed facility

Assessing the potential for impacts to workers within the proposed facility from emissions from the stack is not particularly relevant.

These workers will be primarily exposed to emissions from the wastes themselves within the Waste reception hall (tipping hall) or during maintenance on the bunker or cranes, as the wastes are prepared for introduction into the combustion chamber. Since the Waste reception hall and waste bunker are held under negative air pressure during normal operation, odours are preferentially drawn into the combustion chamber of each operating boiler where they are destroyed by the high temperature combustion process. This normal operating practice will help to minimise the exposure of workers to odours and other air emissions associated with the waste within the waste bunker area.

It is noted that worker health and safety laws apply to all workplaces at all times. All employers must provide a safe workplace for their employees. One of the ways to demonstrate that a workplace is safe is compliance with all relevant workplace exposure standards (concentrations of chemicals in

air within a workplace – these standards are set by SafeWork Australia). In addition, both Safework Australia and WorkSafe QLD have a range of relevant policies which must be complied with including those for safe manual handling, working in confined spaces, working at heights.

Cleanaway are experienced in providing safe workplaces in operational facilities with many different types of controls which are tailored to suit the site in question. Facilities operated by Cleanaway that would have similar workplace requirements include:

- Cleanaway Solid Waste Services (e.g. Ashmore, Bundaberg East, Craiglie, Doonan, Gympie, Innisfail, Morningside, Narangba, New Chum, Northgate, Oxley, Parkhurst, Cairns)
- Cleanaway Industrial Waste Services (e.g. Burleigh Heads, Darra, Gladstone, Roma)
- Cleanaway Liquid Waste Services (e.g. Bundaberg, Crestmead, Emerald, Moranbah, Mt Isa, Narangba, Mackay, Rockhampton, Stratford, Toowoomba)
- Cleanaway Daniels Medical Waste Services (e.g. Yatala)

All of these facilities currently require appropriate standard operating procedures and appropriate building conditions (such as ventilation rates) to ensure provision of a safe workplace for employees. Similar approaches would be adopted at this facility to ensure the workplace will be safe.

In regard to assessing air quality for such workers when outdoors (i.e. air that might be impacted by the emissions from the stack), the assessment for air quality for visitors to the site is also relevant for the staff working within the plant (See **Section 5.5.3.5**). This assessment for site visitors used public health guidelines rather than workplace exposure standards. Workplace exposure standards are often 10-100 times higher than public health guidelines so the assessment for visitors is conservative when applied to the plant workers.

5.4 Criteria Pollutants

5.4.1 General

Criteria air pollutants are those that are targeted by the National Environment Protection (Ambient Air Quality) Measure (NEPC 2021b). They are common air pollutants that need to be managed well to maintain acceptable air quality.

There are many sources of these air pollutants including all combustion sources – fires, bushfires, cooking, cigarettes, vehicles, wood fired heaters, open fireplaces, ship engines and power stations – and other sources like windblown dust and salt spray.

The pollutants included are PM₁₀, PM_{2.5}, NO₂, SO₂, CO (NEPC 2021b). The ambient air quality NEPM also contains guidance about lead. It was included in these criteria or principal pollutants when lead was used in petrol. Lead is now included with the other air pollutants in **Section 5.5**.

The most recent published version of the National Environment Protection (Ambient Air Quality) Measure was the version published in April 2021. This version includes an update to the goals for NO₂ and SO₂ as well as the updated guidelines for particulates that come into effect in 2025 (NEPC 2021b). The criteria established for these pollutants in Australia include data from epidemiological studies of whole city populations so include consideration of sensitive subpopulations like children, the elderly and the infirm.

The ground level concentrations of these criteria pollutants have been estimated in the air quality modelling (Katestone 2026).

The assessment has evaluated the following:

- Incremental changes in ground level concentrations of these criteria pollutants from this facility for the LP1 case (i.e. normal operations).
- Cumulative ground level concentrations for the combination of this facility and the existing ambient background.

Sections 5.4.2 to 5.4.5 discuss each of these criteria pollutants and provide the modelled ground level concentrations in each of the relevant locations and the relevant Australian guidelines for these pollutants – the guidelines from the National Environment Protection (Ambient Air Quality) Measure (NEPC 2021b).

The results for the criteria pollutants have been assessed for:

- maximum off-site location
- maximum residential location
- maximum commercial/industrial location
- maximum on-site location

5.4.2 Sulfur oxides (SO_x)

Sulfur oxides are formed during combustion when chemicals containing sulfur present in fuels (such as coal, gas, petrol) react with oxygen to form sulfur oxides. Burning of coal in power stations in Europe resulted in acid rain affecting forests. The acid rain was primarily a result of the formation of sulfur oxides as the coal was burnt. Sulfur oxides are also released from volcanos. Wildfires and other types of fires are also sources to the atmosphere of these chemicals (USEPA 2018b).

Sulfur dioxide (SO₂) is the main sulfur oxide that can have impacts on people. Exposure to elevated levels can result in irritation of the respiratory system and can make breathing difficult. The most affected by exposure to these chemicals are people with asthma (USEPA 2018b).

Guidelines are available from NEPC (NEPC 2021b) which indicate concentrations of sulfur dioxide considered to be acceptable by national health authorities. These guidelines are based on protection from adverse health effects following both short term (acute) and longer term (chronic) exposure for all members of the population including sensitive populations like asthmatics, children and the elderly.

Guidelines are also available in the Queensland Government Environmental Protection Policy for air (Queensland Government 2019b). The listed values for SO₂ are the values that were listed in the ambient air NEPM at the time the EPP (air) was published. They have not been updated using the more recent values listed in the NEPM. The NEPM values have been used as the focus for this assessment as they are more stringent.

Table 5.1 shows the comparison of modelled SO₂ levels and the relevant NEPM guidelines for the facility alone and, for the cumulative case, including this Project + existing background.

Table 5.1: SO₂ impacts from the Project

Parameter	SO ₂ (µg/m ³)		
	1-hour average	24-hour average	Annual average
Guideline (NEPC 2021b)	214 (75 ppb)	57 (20 ppb)	Guideline removed
QLD EPP (Air) (Queensland Government 2019b)	570 (200 ppb)	229 (80 ppb)	57 (20 ppb)
Maximum off-site location			
Contribution from Project (increment)	35	3.1	0.08
% contribution of Project to AAQ NEPM	16%	6%	--
Cumulative case (Project + background)	200	18	4
Maximum residential location			
Contribution from Project (increment)	16	0.9	0.05
% contribution of Project to AAQ NEPM	7%	1.7%	--
Cumulative case (Project + background)	106	9.4	3.4
Maximum commercial location			
Contribution from Project (increment)	35	3.1	0.06
% contribution of Project to AAQ NEPM	16%	6%	--
Cumulative case (Project + background)	164	12	3.5
Maximum on-site location			
Contribution from Project (increment)	23	2	0.03
% contribution of Project to AAQ NEPM	11%	3.8%	--
Cumulative case (Project + background)	39	6.6	3.3

On this basis, the operation of the facility is not expected to significantly contribute to, or change ambient levels of SO₂. In addition, the cumulative ground level concentrations (i.e. from this facility, from other nearby facilities and from background existing air quality in the area) are in compliance with the most recent national guidelines.

It is noted that these national guidelines are not actually intended for application as regulatory standards at a single facility. They are designed for assessment of regional air quality within air sheds (i.e. across Brisbane as a whole, for example) (NEPC 2021a).

The cumulative data indicate that there are significant other sources of sulfur oxide in the region around this proposed facility – ones that are not related to this proposed facility. Regardless of these other sources, the cumulative ground level concentrations are in compliance with those specified in the AAQ NEPM – i.e. national guidelines for air in Australia.

5.4.3 Nitrogen Oxides (NO_x)

Nitrogen oxides (NO_x) refer to a collection of highly reactive gases containing nitrogen and oxygen, most of which are colourless and odourless. Nitrogen oxide gases form when fuel is burnt including when waste is used as fuel. Motor vehicles, along with industrial, commercial and residential (e.g. gas heating, cigarettes or cooking) combustion sources, are primary producers of nitrogen oxides.

In terms of health effects, nitrogen dioxide is the only oxide of nitrogen that may be of concern (WHO 2000a). Nitrogen dioxide is a colourless and tasteless gas with a sharp odour. Nitrogen dioxide can cause inflammation of the respiratory system and increase susceptibility to respiratory infection. Exposure to elevated levels of nitrogen dioxide has also been associated with increased mortality, particularly related to respiratory disease, and with increased hospital admissions for asthma and heart disease patients (WHO 2013a). Asthmatics, the elderly and people with existing cardiovascular and respiratory disease are particularly susceptible to the effects of elevated nitrogen dioxide (Morgan, Broom & Jalaludin 2013; NEPC 2010).

Guidelines are available from NEPC (NEPC 2021b) which indicate concentrations of nitrogen dioxide considered to be acceptable by national health authorities. These guidelines are based on protection from adverse health effects following both short term (acute) and longer term (chronic) exposure for all members of the population including sensitive populations like asthmatics, children and the elderly.

Guidelines are also available in the Queensland Government Environmental Protection Policy for air (Queensland Government 2019b). The listed values for NO₂ are the values that were listed in the ambient air NEPM at the time the EPP (air) was published. They have not been updated using the more recent values listed in the NEPM. The NEPM values have been used as the focus for this assessment as they are more stringent.

Table 5.2 shows the comparison of modelled NO₂ levels and the relevant NEPM guidelines for the facility alone and, for the cumulative case, including this Project + existing background.

Table 5.2: NO₂ impacts from the Project

Parameter	NO ₂ (µg/m ³)	
	1-hour average	Annual average
Guideline (NEPC 2021b)	164 (0.08 ppm)	31 (0.015 ppm)
QLD EPP (Air) (Queensland Government 2019b)	250 (0.12 ppm)	62 (0.03 ppm)
Maximum off-site location		
Contribution from Project (increment)	42	0.3
% contribution of Project to AAQ NEPM	28%	1%
Cumulative case (Project + background)	59	13
Maximum residential location		
Contribution from Project (increment)	19	0.2
% contribution of Project to AAQ NEPM	13%	0.7%
Cumulative case (Project + background)	37	13
Maximum commercial location		
Contribution from Project (increment)	42	0.2
% contribution of Project to AAQ NEPM	28%	0.7%
Cumulative case (Project + background)	59	13
Maximum on-site location		
Contribution from Project (increment)	27	0.1
% contribution of Project to AAQ NEPM	18%	0.4%
Cumulative case (Project + background)	44	13

On this basis, the operation of the facility is not expected to change ambient levels of NO₂ in the area. In addition, the cumulative ground level concentrations (i.e. from this facility, from other nearby facilities and from background existing air quality in the area) are in compliance with the most recent national guidelines.

It is noted that these national guidelines are not actually intended for application as regulatory standards at a single facility. They are designed for assessment of regional air quality within air sheds (i.e. across Brisbane as a whole, for example) (NEPC 2021a).

The cumulative data for the maximum off-site location highlights that there are other sources of nitrogen oxides in the region around this proposed facility. The annual average concentration increment from this facility is 0.3 µg/m³ while the cumulative total (i.e. increment from this facility plus existing ambient concentrations and concentrations arising from other industrial facilities nearby) is 13 µg/m³ (i.e. around 40 fold higher). Taking into account existing air quality, air quality changes due to other sources in the area and potential air quality changes due to this facility, the modelled cumulative ground level concentrations (i.e. the sum of concentrations already existing in

the area plus those from nearby industrial facilities and the increment from this facility) is in compliance with those specified in the AAQ NEPM – i.e. national guidelines for air in Australia.

5.4.4 Carbon monoxide

Carbon monoxide is produced during combustion when there is a limited supply of oxygen. This facility is designed to optimise the oxygen available in the combustion zone, so the production of carbon monoxide should be very low. Motor vehicles are the dominant source of carbon monoxide in air (DECCW 2009).

It is well known that excess levels of carbon monoxide in enclosed spaces can cause significant impacts. This may occur when indoor gas or other types of heaters are not operating correctly and are left on overnight. Potential effects that can be expected due to exposure to excess levels of CO are those linked with carboxyhaemoglobin (COHb) in blood – i.e. where CO replaces oxygen in the blood preventing oxygen from being transported around the body. In addition, association between exposure to carbon monoxide and cardiovascular hospital admissions and mortality, especially in the elderly for cardiac failure, myocardial infarction and ischemic heart disease; and some birth outcomes (such as low birth weights) have been identified (NEPC 2010).

Guidelines are available from NEPC (NEPC 2021b) which indicate concentrations of carbon monoxide considered to be acceptable by national health authorities for the Australian population.

Guidelines are also available in the Queensland Government Environmental Protection Policy for air (Queensland Government 2019b). The listed value for CO is the value listed in the ambient air NEPM – the difference is just due to rounding.

Table 5.3 shows the comparison of modelled CO levels and the relevant NEPM guidelines for the facility alone and for the cumulative case which includes this Project + existing background.

Table 5.3: CO impacts from the Project

Parameter	CO ($\mu\text{g}/\text{m}^3$)
	8-hour average
Guideline (NEPC 2021b)	10,000 (9 ppm)
QLD EPP (Air) (Queensland Government 2019b)	11,000 (9 ppm)
Maximum off-site location	
Contribution from Project (increment)	16
% contribution of Project to AAQ NEPM	0.2%
Cumulative case (Project + background)	870
Maximum residential location	
Contribution from Project (increment)	4.4
% contribution of Project to AAQ NEPM	0.04%
Cumulative case (Project + background)	700
Maximum commercial location	
Contribution from Project (increment)	16
% contribution of Project to AAQ NEPM	0.2%
Cumulative case (Project + background)	740
Maximum on-site location	
Contribution from Project (increment)	10
% contribution of Project to AAQ NEPM	0.1%
Cumulative case (Project + background)	630

On this basis, the operation of the facility is not expected to significantly contribute to, or change ambient levels of CO. In addition, the cumulative ground level concentrations (i.e. from this facility, from other nearby facilities and from background existing air quality in the area) are in compliance with the most recent national guidelines.

It is noted that these national guidelines are not actually intended for application as regulatory standards at a single facility. They are designed for assessment of regional air quality within air sheds (i.e. across Brisbane as a whole, for example) (NEPC 2021a).

The cumulative data indicate that there are significant other sources of carbon monoxide in the region around this proposed facility – ones that are not related to this proposed facility. Regardless of these other sources, the cumulative ground level concentrations are in compliance with those specified in the AAQ NEPM – i.e. national guidelines for air in Australia.

5.4.5 Particles

Particles or particulate matter (PM) is a widespread air pollutant with a mixture of physical and chemical characteristics that vary by location (and source). Particles are always present in the air.

Unlike many other pollutants, particles comprise a broad class of diverse materials and substances, with varying shape, chemical, physical and thermodynamic properties, with sizes that vary from less than 0.005 microns (μm) to greater than 100 microns (μm).

Sources of particles include bushfires, other types of fires, cooking (gas and wood fired), BBQs (gas and wood fired), vehicle emissions, wood fired heaters (and gas fired), windblown dust, salt spray (when near ocean) as well as large facilities using combustion, like power stations and waste-to-energy facilities.

The most important aspect to consider, in regard to health/exposure to particles, is the difference (if any) in the concentration of particles in air in the local area due to the facility.

The main focus of studies about health effects due to particles in air is the smaller particles. These fine particles are small enough to reach deep into the lungs, so they are the most relevant for assessing potential health effects.

Particles are measured as those particles less than 10 micron in size (PM_{10}) or those that are less than 2.5 micron in size ($\text{PM}_{2.5}$).

It is important to note that PM_{10} includes all the particles that are less than 2.5 microns in size as well as the ones that are larger than 2.5 microns but less than 10 microns. The same goes for $\text{PM}_{2.5}$ – it includes all the ultrafine particles (those less than 1 micron or 0.1 micron) as well as those between 1 and 2.5 microns.

This means ultrafine particles are included in the health effects assessments even if not specifically mentioned. In this case the predominant particles being emitted by this facility are those that are less than (or equal to) 2.5 microns ($\text{PM}_{2.5}$).

Numerous epidemiological studies⁷ have reported significant positive associations between particulate air pollution measured as PM₁₀ or PM_{2.5} and adverse health outcomes. Effects noted in large studies undertaken in cities in Europe and the US include mortality as well as a range of adverse cardiovascular and respiratory effects (USEPA 2012, 2018a; WHO 2013c). In particular, the links between levels of PM_{2.5} and health effects have been shown to be clear and robust. The health effects for both PM₁₀ and PM_{2.5} covered by these studies on populations including the young, the elderly and those with respiratory issues were all considered in the derivation of the NEPM air guidelines for particles.

Guidelines are available from NEPC (NEPC 2021b) which indicate concentrations of particles considered to be acceptable by national health authorities for the Australian population.

The recent review of the guidelines by NEPC identified additional supporting studies⁸ for the evaluation of potential adverse health effects (Golder 2013; NEPC 2010). The review recommended an amendment to the guidelines for particles which came into effect in January 2025. The change is that the 24 hour average for PM_{2.5} dropped from 25 to 20 µg/m³ and the annual average dropped from 8 to 7 µg/m³ (NEPC 2021b).

Guidelines are also available in the Queensland Government Environmental Protection Policy for air (Queensland Government 2019b). The listed values for PM₁₀ and PM_{2.5} are the values that were listed in the ambient air NEPM at the time the EPP (air) was published. They have not been updated using the more recent values listed in the NEPM (only relevant for PM_{2.5}). The NEPM values have been used as the focus for this assessment as they are more stringent.

Tables 5.4 (PM_{2.5}) and 5.5 (PM₁₀) show the comparison of modelled particle levels and the relevant NEPM guidelines for the facility alone and, for the cumulative case, which includes this Project + existing background.

The assessment of background air quality has used a contemporaneous assessment for particles. This type of assessment is undertaken when the existing or background levels are close to or above relevant guideline values. Contemporaneous assessment calculates the cumulative levels (i.e. the emissions from the proposed facility plus the background levels) of PM_{2.5} and PM₁₀ separately for every hour of the year – matching the meteorological conditions for each hour. This allows an evaluation of whether the proposed facility makes any difference to the total number of days per year that PM_{2.5} and PM₁₀ might exceed guideline values in the region.

Table 5.4: PM_{2.5} impacts from the Project

Parameter	PM _{2.5} (µg/m ³)	
	24-hour average	Annual average
Guideline (NEPC 2021b)	20	7
QLD EPP (Air) (Queensland Government 2019b)	25	8
Maximum off-site location		
Contribution from Project (increment)	0.5	0.01

⁷ Epidemiology is the study of diseases in populations. Epidemiological evidence can only show that this risk factor is associated (correlated) with a higher incidence of disease in the population exposed to that risk factor. The higher the correlation the more certain the association. Causation (ie that a specific risk factor actually causes a disease) cannot be proven with only epidemiological studies. For causation to be determined a range of other studies need to be considered in conjunction with the epidemiology studies.

⁸ Many of the more current studies are epidemiology studies that relate to a mix of urban air pollutants (including particulate matter) where it is more complex to determine the effects that can be attributed to carbon monoxide exposure only.

Parameter	PM _{2.5} (µg/m ³)	
	24-hour average	Annual average
% contribution of Project to AAQ NEPM	2.5%	0.1%
Cumulative case (Project + background)	8.9	6.7
Maximum residential location		
Contribution from Project (increment)	0.15	0.009
% contribution of Project to AAQ NEPM	0.8%	0.1%
Cumulative case (Project + background)	7.8	6.7
Maximum commercial location		
Contribution from Project (increment)	0.5	0.01
% contribution of Project to AAQ NEPM	2.5%	0.1%
Cumulative case (Project + background)	8.1	6.7
Maximum on-site location		
Contribution from Project (increment)	0.3	0.004
% contribution of Project to AAQ NEPM	1.5%	0.05%
Cumulative case (Project + background)	7.5	6.6

Table 5.5: PM₁₀ impacts from the Project

Parameter	PM ₁₀ (µg/m ³)	
	24-hour average	Annual average
Guideline (NEPC 2021b)	50	25
QLD EPP (Air) (Queensland Government 2019b)	50	25
Maximum off-site location		
Contribution from Project (increment)	0.5	0.01
% contribution of Project to AAQ NEPM	1%	0.04%
Cumulative case (Project + background)	23	17
Maximum residential location		
Contribution from Project (increment)	0.15	0.009
% contribution of Project to AAQ NEPM	0.3%	0.04%
Cumulative case (Project + background)	21	17
Maximum commercial location		
Contribution from Project (increment)	0.5	0.01
% contribution of Project to AAQ NEPM	1%	0.04%
Cumulative case (Project + background)	21	17
Maximum other places location		
Contribution from Project (increment)	0.3	0.004
% contribution of Project to AAQ NEPM	0.6%	0.02%
Cumulative case (Project + background)	19	17

On this basis, the operation of the facility is not expected to significantly contribute to, or change ambient levels of particles. In addition, the cumulative ground level concentrations (i.e. from this facility, from other nearby facilities and from background existing air quality in the area) are in compliance with the most recent national guidelines.

It is noted that these national guidelines are not actually intended for application as regulatory standards at a single facility. They are designed for assessment of regional air quality within air sheds (i.e. across Brisbane as a whole, for example) (NEPC 2021a).

The cumulative data indicate that there are significant other sources of particles in the region around this proposed facility – ones that are not related to this proposed facility. Regardless of these other sources, the cumulative ground level concentrations are in compliance with those specified in the AAQ NEPM – i.e. national guidelines for air in Australia.

To refine this assessment, exposure-response relationships developed by the World Health Organisation (WHO) to estimate the incremental individual risk associated with the change in PM_{2.5} from the facility have also been used.

This calculation has been undertaken on the basis of the most significant health indicator, namely mortality, for which changes in PM_{2.5} have been identified to have a causal relationship. The relationship for PM₁₀ is not as robust, hence the focus is on exposures to PM_{2.5}. Focusing on PM_{2.5} is also protective for PM₁₀ as the exposure-response relationships based on PM₁₀ provide a similar or lower risk estimate (i.e. even low risks).

The air quality modelling for this assessment has assumed that all PM₁₀ is in the PM_{2.5} size fraction, so the approach taken here is assessing PM₁₀ as if it were all PM_{2.5} – i.e. the fraction which has higher risks.

Using this health indicator also covers a wide range of other health effects associated with PM_{2.5} such as those for hospitalisations for respiratory or cardiovascular issues. The exposure-response relationships for these other effects give the same or lower levels of effect (i.e. similar or lower estimated risks) for the same change in particle levels.

The calculation in this assessment specifically considers the baseline mortality rate for Scenic Rim LGA – i.e. the local area around the Project location. This is the mortality rate that currently exists in these areas without this facility. The rate in Scenic Rim LGA is 266.2 per 100,000 for 2016/20 (all ages and all causes) (refer to **Table 3.2**).

These baseline rates are used along with the incremental change in PM_{2.5} due to the facility as inputs to the relevant exposure-response relationship developed by the WHO for assessing all-cause mortality due to exposure to PM_{2.5}.

Further details and calculations are presented in **Appendix A**. These calculations assume that a person is present at the relevant location for 24 hours a day, every day of the year.

For the incremental level of PM_{2.5} at the maximum residential location, the maximum individual risk is 1×10^{-7} (i.e. 1 in 10 million or 0.1 in 1 million). Values for any location further from the facility than this location will have lower risks than this value.

This risk level (1×10^{-7}) is considered to be very low and is lower than the mortality risk criteria considered to indicate negligible risk outlined by AAQ NEPM and in other literature (Capon & Wright 2019; NEPC 2011b). On this basis, changes in PM_{2.5} (and, therefore, PM₁₀) in the local area due to this proposed facility are considered to have a negligible impact on the health of the community (including sensitive subpopulations).

5.5 Other Pollutants

5.5.1 Approach

The quantitative assessment of potential risks to human health for any chemical requires the consideration of:

- the types of effects seen in laboratory studies
- the dose or concentration related to those effects
- where possible, mechanisms of action (i.e. how the chemical caused the effect).

Most chemicals have a threshold below which there are no adverse effects. That means there is a dose or concentration below which no adverse effects are seen because there is not enough of the chemical in the body to cause enough of a change in some system in the body or enough damage to cells.

The threshold is determined by taking the lowest dose that did not cause effects in a study (typically from animal or human (e.g. occupational) studies) to which a number of uncertainty factors are then applied (based on type of study, how many studies are available etc). This value is termed the toxicity reference value (TRV). When intakes / exposures are lower than the TRV, they are considered to not be associated with an adverse health risk (NHMRC 1999).

Some other chemicals have the potential to cause cancer by directly damaging DNA. These chemicals are described as non-threshold as it is assumed that they could cause some level of effect even at low doses. While this can be interpreted to mean that there is no safe dose, this is a policy choice/assumption built into the calculations to ensure risks are not underestimated. It actually means the risk of effects due to exposure to such a chemical increases with increasing dose or exposure – i.e. at low exposure, risk is low or even negligible and at high exposure, risk is much higher.

The difference between threshold and non-threshold chemicals is chemicals that act via a threshold mechanism of action have no risk when exposure is to levels below the threshold and that risks will increase with increasing exposure once the dose/concentration is above the threshold while chemicals that act via a non-threshold mechanism of action have a low/negligible risk at low levels of exposure and higher risks at higher levels of exposure.

Dose-response values (threshold or non-threshold), i.e. TRVs, that are considered relevant to the characterisation of potential health effects associated with exposure have been selected for this assessment from credible peer-reviewed sources in accordance with the current approved national guidance from enHealth and NEPC. These are discussed in **Appendix B** (enHealth 2012a; NEPC 1999 amended 2013a).

For the other pollutants (i.e. not criteria/principal pollutants discussed in **Section 5.4**), inhalation exposures have considered for both short-term/acute exposures (based on estimated worst case conditions) as well as chronic exposures (based on long term normal operating conditions).

The AQA prepared by Katestone (2026) included modelling emissions over a grid of 20 km x 20 km.

The results for the 1 hour, 24 hour and annual averages for each of the pollutants assessed at the grid maximum (i.e. the maximum anywhere across the grid) as well as at the maximum locations for residential, commercial/industrial and on the site itself were provided by Katestone for use in this assessment (Katestone 2026).

This assessment has included evaluation of risks when people are exposed just via inhalation for short term and long term exposure.

In addition, risks due to direct contact with soil onto which particles being emitted from the facility have deposited or via consumption of home grown produce grown in such soil have also been assessed.

Specifically, all of the following exposure pathways have been considered, where relevant:

- Inhalation at ground level

- Deposition onto soil and direct contact with that soil
- Deposition onto soil and uptake into fruit and vegetables grown in that soil
- Deposition onto soil and uptake into eggs
- Deposition onto soil and uptake into meat
- Deposition onto soil and uptake into milk
- Deposition onto roof and collection in household rainwater tanks.

It is noted that the pathways related to consumption of fruit, vegetables, eggs, meat or milk refer to home grown produce rather than food from the commercial food supply (i.e. foods purchased from supermarkets).

The calculations for fruit, vegetables and eggs are designed to assess consumption of these types of produce by people who grow them in their backyards.

The calculations for meat and milk are designed to assess consumption of meat and milk by the people living or working at a farm where cattle are kept. The calculations are not relevant for the production of meat or milk for the commercial food supply. This is because the home grown type calculations assume a person consumes meat or milk from the same location all year round. When meat or milk are included in the commercial food supply, consumption by a single person of meat or milk from the same animals or from animals from the same farm all year round is not possible. The risks for meat or milk in the commercial food supply will be much lower than those calculated in this assessment and, therefore, the results of this HHRA are considered to be conservative.

Details of the methodology and equations used to undertake this assessment are provided in **Appendices B** and **C**. Also included in **Appendices B** and **C** are the assumed values for the parameters used in these calculations.

The approach adopted is in line with national guidance including:

- enHealth, 2012. Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards.
- enHealth 2012. Australian Exposure Factors Guide.
- NEPC 1999 amended 2013. Schedule B4 of National Environment Protection (Assessment of Site Contamination) Measure.

International guidance from the World Health Organisation and the US Environmental Protection Agency has also been considered where relevant.

5.5.2 Identification of Complete Exposure Pathways

The receptors and exposure pathways considered in this assessment are shown in **Table 5.6**.

Table 5.6: Summary of Key Exposure Groups and Pathways

Receptor	Media	Exposure Pathways									
		Inhalation	Incidental Ingestion Soil	Dermal Contact Soil	Ingestion of eggs	Ingestion of fruit & vegetables	Ingestion of fruit	Ingestion of Milk	Ingestion of Meat	Ingestion of water from rainwater tank	Dermal contact of water from rainwater tank
Maximum Off-site	Gases	✓	x	x	x	x	x	x	x	x	x
	Particles	✓	✓	✓	x	x	x	x	x	✓	✓
Maximum Residential	Gases	✓	x	x	x	x	x	x	x	x	x
	Particles	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Maximum Commercial/Industrial	Gases	✓	x	x	x	x	x	x	x	x	x
	Particles	✓	✓	✓	x	x	x	x	x	✓	✓
Maximum On-site	Gases	✓	x	x	x	x	x	x	x	x	x
	Particles	✓	✓	✓	x	x	x	x	x	✓	✓

Notes:

- ✓ Exposure pathway complete
 x Incomplete exposure pathway

The specific aspects that have been assessed for the exposure scenarios outlined in **Section 4.4.5** include:

- maximum off-site location – exposures via inhalation and direct contact with soil are the only ones relevant. This location is on the boundary of the facility or on the roads immediately outside the boundary.
- maximum residential location – exposure via inhalation, direct contact with soil and consumption of home grown fruit, vegetables and eggs and use of a rainwater tank.
- maximum residential location assuming farming occurred – exposure via inhalation, direct contact with soil and consumption of home grown fruit, vegetables, meat, milk and eggs and use of a rainwater tank.
- maximum commercial/industrial location – exposure via inhalation and direct contact with soil only.
- maximum commercial/industrial location should such a site change land use to residential – exposure via inhalation, direct contact with soil and consumption of home grown fruit, vegetables and eggs and use of a rainwater tank.
- maximum on-site location – exposure via inhalation for workers/visitors to the education centre.

Short term exposures via inhalation only have been assessed using data from modelling of Scenario 2 (i.e. LP1 based on short term stack concentrations – i.e. peak concentrations).



Long term exposures via inhalation and deposition have been assessed using data from modelling of Scenario 1 – i.e. normal operations (LP1).

The specific assumptions made in this assessment in regard to how people can be exposed are listed in **Table 5.7**.

Table 5.7: Exposure parameter assumptions

Parameter		Residential		Farm		Visitor and Education Centre Staff
		Young children	Adults	Young children	Adults	Adult
BW	Body weight	15	70	15	70	70
		((enHealth 2012b) and ASC NEPM (NEPC 1999 amended 2013c))				
EF	Exposure frequency (days/year)	365	365	365	365	240
		All year				Working year
ED	Exposure duration (years)	6	29	6	29	30
		As per (enHealth 2012b) and ASC NEPM				
AT	Averaging time (days)	Threshold = ED x 365 days/year; Non-threshold = 70 years x 365 days/year				
C _M	Concentration of chemical substance in media or relevance (soil, fruit and vegetables, eggs, meat, milk) (mg/kg)	Calculations undertaken on the basis of the maximum predicted impacts relevant to areas where multi-pathway exposures may occur				
IR _M	Ingestion rate of media					
	Soil (mg/day)	100	50	100	50	25 (where relevant)
		Ingestion rate of outdoor soil and dust (tracked or deposited indoors) as per enHealth (enHealth 2012b)				
	Fruit and vegetables (kg/day)	0.28	0.4	0.28	0.4	Not applicable
		85% from aboveground crops 16% from root crops (Total fruit and vegetable intakes per day as per ASC NEPM (NEPC 1999 amended 2013c))	73% from aboveground crops 27% from root crops (Total fruit and vegetable intakes per day as per ASC NEPM (NEPC 1999 amended 2013c))	85% from aboveground crops 16% from root crops (Total fruit and vegetable intakes per day as per ASC NEPM (NEPC 1999 amended 2013c))	73% from aboveground crops 27% from root crops (Total fruit and vegetable intakes per day as per ASC NEPM (NEPC 1999 amended 2013c))	
	Eggs (kg/day)	0.036	0.059	0.036	0.059	Not applicable
		Ingestion rate of eggs per day – P90 consumption for consumers from FSANZ (FSANZ 2017)				

Parameter		Residential		Farm		Visitor and Education Centre Staff
		Young children	Adults	Young children	Adults	Adult
	Meat (kg/day)	NR	0.085	0.16	Not applicable	
			Ingestion rate of beef per day – P90 consumption for consumers from FSANZ (FSANZ 2017) Relevant for consumer for home slaughtered meat			
	Milk (kg/day)	NR	1.097	1.295	Not applicable	
			Ingestion rate of milk per day – P90 consumption for consumers from FSANZ (FSANZ 2017) Relevant for consumer of on farm produced milk			
FI	Fraction of media ingested derived from impacted media, or fraction of produce consumed each day derived from the property					
	Soil	100%	100%	100%	100%	100%
		Assume all soil contact that occurs during a day comes from the one property				
	Fruit and vegetables	10%	10%	35%	35%	Not applicable
		Standard conservative assumption for backyard production (NEPC 1999 amended 2013c))		Standard conservative assumption for rural residential production		
	Eggs	100%	100%	100%	100%	Not applicable
		Assume all eggs consumed are home-produced				
	Meat	NR	35%	35%	Not applicable	
			Assume significant portion of meat consumed on farm is produced at the farm (i.e. home slaughtered)			
	Milk	NR	100%	100%	Not applicable	
Assume all milk consumed on farm is produced at the farm						
B	Bioavailability or absorption of chemical substance via ingestion	100%	100%	100%	100%	100%
		Conservative assumption – maximum possible				
SA	Surface area of body exposed to soil per day (cm ² /day)	2,700	6,300	2,700	6,300	3,800
		Exposed skin surface area relevant to adults/children and workplaces as per ASC NEPM (NEPC 1999 amended 2013c)				

Parameter		Residential		Farm		Visitor and Education Centre Staff
		Young children	Adults	Young children	Adults	Adult
AF	Adherence factor, amount of soil that adheres to the skin per unit area which depends on soil properties and area of body (mg/cm ² per event)	0.5	0.5	0.5	0.5	0.5
		Default (conservative) value from ASC NEPM (NEPC 1999 amended 2013c)				
ABSd	Dermal absorption fraction (unitless)	Chemical specific Refer to Tables B3 and B4				
CF	Conversion factor					
	Soil	1x10 ⁻⁶ to convert mg to kg (Conversion of units relevant to soil ingestion and dermal contact)				

5.5.3 Inhalation

5.5.3.1 General

Exposure to chemicals present in the facility emissions via inhalation of air at ground level has been assessed for both short term exposure and long term exposure – i.e. 1 hour average concentrations and annual average concentrations.

The concentrations arising due to the proposed facility have been compared to national or international guidelines for each chemical in air. These are generally based on studies which estimate the concentration which has no observable effects on laboratory animals for small/subtle types of effect or for sensitive life stages (e.g. very young). These no observable effects concentrations are then made more conservative by adjustment using a range of uncertainty factors to account for unavoidable limits in knowledge. These guidelines are, therefore, considered to be protective for sensitive subpopulations like the young and the elderly as well as the population in general.

5.5.3.2 Modelled concentrations and existing background concentrations

The air quality modelling undertaken by Katestone has allowed the estimation of concentrations of the relevant chemicals from the proposed WtE facility over short periods (1 hour averages) as well as over the long term (annual averages).

These ground level concentrations are calculated by the computer model used in the air quality modelling. This does not imply that these concentrations could generate noticeable changes in air quality at a location or that they are actually measurable. They can, however, be calculated using the equations that form the basis of the dispersion modelling recommended by Qld regulators – i.e. the maths allows them to be calculated.

To illustrate how small these modelled concentrations are, this section provides a comparison of the modelled ground level concentrations with measured concentrations in air in Australia in a study by NSW EPA (NSW DEC 2003, 2004a, 2004b; NSW EPA 2003). Similar studies in Queensland have not been undertaken.

Modelled ground level concentrations have been determined by Katestone for the proposed EfW facility alone assuming normal operations.

Annual average concentrations (long term) (facility alone) at ground level for the maximum residential location closest to the proposed EfW facility are listed in **Table 5.8**. This table shows how low the modelled concentrations are for these various chemicals.

Table 5.8: Modelled annual average ground level concentrations at the maximum residential location

Pollutants	Maximum ground level concentration ($\mu\text{g}/\text{m}^3$)
	EfW facility only
Hydrogen chloride (HCl)	0.01
Hydrogen fluoride (HF)	0.002
Ammonia	0.02
Benzene	0.02
Formaldehyde	0.02
Cadmium	0.00002
Thallium	0.00001
Mercury	0.00004
Antimony	0.00004
Arsenic	0.00002

Pollutants	Maximum ground level concentration ($\mu\text{g}/\text{m}^3$)
	EfW facility only
Lead	0.0002
Chromium (Cr VI assumed)	0.00005
Cobalt	0.00001
Copper	0.00005
Manganese	0.00008
Nickel	0.00006
Vanadium	0.000007
Dioxin-like compounds	0.0000000001
PAHs	0.0000000004
PFAS	0.00000001

The 1 hour average ground level concentrations at the maximum off-site location and at the maximum residential location are listed in **Table 5.9**.

Table 5.9: 1 hour average ground level concentrations

Pollutants	Modelled Air Concentration ($\mu\text{g}/\text{m}^3$) EfW facility only	
	Maximum off-site	Maximum residential location
Hydrogen chloride (HCl)	40	18
Hydrogen fluoride (HF)	3	1
Ammonia	12	5
Cadmium	0.02	0.01
Mercury	0.02	0.01
Antimony	0.04	0.02
Arsenic	0.01	0.006
Chromium (VI)	0.05	0.02
Cobalt	0.001	0.005
Copper	0.05	0.02
Manganese	0.08	0.04
Nickel	0.05	0.02
Vanadium	0.005	0.002
Benzene	18	8
Formaldehyde	18	8

These results show that modelling allows the estimation of ground level concentrations that are well below concentrations that could be routinely measured.

As noted above, the NSW EPA undertook a large study around 2000, monitoring the levels of metals, PAHs and dioxin-like compounds in air in a number of locations around Sydney, Newcastle and Wollongong and in some more remote NSW locations for some chemicals (NSW DEC 2003, 2004a, 2004b; NSW EPA 2003). This study was undertaken to provide a baseline for the levels of these chemicals in urban air in NSW.

The locations that were monitored for many of the metals, PAHs and dioxin-like compounds included:

- Metals (samples collected over 24 hours on each occasion)
 - Sydney – Blacktown, Earlwood, Richmond and Rozelle
 - Newcastle – CBD
 - Illawarra – Albion Park and CBD
- Dioxin-like compounds (samples collected using high volume sampler over 12 days for the urban areas and 24 days at Siding Spring – each sample required sampling for these extended periods)

- Sydney – Westmead
- Wollongong – Warrawong
- Regional/remote area – Siding Spring Observatory
- PAHs (samples collected over 24 hours on each occasion)
 - Sydney – CBD, Rozelle, Earlwood, Blacktown, Richmond, Lindfield
 - Newcastle – CBD, Wallsend, Beresfield
 - Wollongong – CBD, Albion Park, Warrawong, Kembla Grange, Kembla Street, Wattle Street and Military Road
 - Regional areas – Orange, Lithgow, Tumut, Cooma, Nowra and Armidale

Table 5.10 lists the average concentrations reported for each of the pollutants relevant for this assessment at the various locations monitored by NSW EPA in their study.

Table 5.10: Average ground level concentrations reported in NSW EPA monitoring

Pollutants	Average concentrations ($\mu\text{g}/\text{m}^3$)			
	Sydney	Newcastle	Wollongong	Regional/remote
Cadmium	0.06-0.28	0.41	0.04-0.11	NA
Thallium	NA	NA	NA	NA
Mercury	NA	NA	NA	NA
Antimony	0.1-2.2	2.8	0.22-0.28	NA
Arsenic	0.4-1.3	0.84	0.18-0.22	NA
Lead	8.9-68	30	3.4-24	NA
Chromium (as VI)	NA	NA	NA	NA
Cobalt	0.13-0.26	0.12	0.1	NA
Copper	3.9-22	6.2	3.1-4.9	NA
Manganese	4.4-7.4	66	4.6-5.9	NA
Nickel	0.95-6.2	1.6	ND	NA
Vanadium	0.26-13	1	0.48-0.66	NA
PAHs as BaP ¹	0.00003-0.00046	0.00003-0.00016	0.00006-0.0003	0.00002-0.0042
Dioxin-like compounds	0.000000014	NA	0.00000001	0.00000000064

Notes:

- 1 range reported is average concentration in summer and in winter – concentrations of PAHs in winter are higher than in summer due to the use of wood fired heating
- NA no sampling was undertaken at this type of location
- ND not detected above the limit of reporting

Since the time of this study, reductions in the concentrations of some pollutants and increases in some others are likely to have occurred due to changes in industrial emissions (e.g. closure of steelworks near Newcastle CBD and ongoing removal of lead from fuels and paints).

Comparing the range of concentrations for each chemical from the NSW EPA data to the modelled concentrations at the maximum residential location is shown in **Table 5.11**.

Table 5.11: Comparison of concentrations reported in NSW EPA monitoring with those for the proposed EfW facility

Pollutants	Concentrations ($\mu\text{g}/\text{m}^3$)		
	NSW EPA data	1 hour average – EfW only	Annual average – EfW only
Cadmium	0.04-0.41	0.01	0.00002
Antimony	0.1-2.8	0.02	0.00004
Arsenic	0.18-1.3	0.006	0.00002
Lead	3.4-68	--	0.0002
Cobalt	0.1-0.26	0.005	0.00001

Pollutants	Concentrations ($\mu\text{g}/\text{m}^3$)		
	NSW EPA data	1 hour average – EfW only	Annual average – EfW only
Copper	3.1-22	0.02	0.00005
Manganese	4.4-66	0.04	0.00008
Nickel	0.95-6.2	0.02	0.00006
Vanadium	0.26-13	0.002	0.000007
PAHs as BaP	0.00003-0.0042	--	0.0000000004
Dioxin-like compounds	0.000000014	--	0.0000000001
PFAS	--	--	0.00000001

This comparison shows that the modelled annual average ground level concentrations (increment) for the proposed EfW facility at the closest residential location are between 140 and >1,000,000 times lower than the concentrations of these chemicals measured in urban air by NSW EPA depending on the chemical.

Even the 1 hour average ground level concentrations for this facility are smaller than the low end of the range of concentrations measured by NSW EPA.

The NSW EPA data are predominantly 24 hour average ground level concentrations. It would be expected that 1 hour average concentrations would generally be higher than 24 hour average concentrations if measured at the same location under the same conditions/sources with just the averaging time varied.

This comparison shows that these small amounts of these chemicals arising from the proposed EfW facility cannot make a measurable difference to the concentrations of these chemicals already present in air in this location.

5.5.3.3 Acute/short term

The assessment of acute exposures is based on comparing the maximum predicted 1-hour average concentration (at the grid maximum (i.e. maximum location for all receptors off the site)) with health-based criteria relevant to an acute or short-term exposure, also based on a 1-hour average exposure time.

In addition, the other types of land use locations in the off-site area have also been compared with health-based criteria relevant to an acute or short-term exposure, also based on a 1-hour average exposure time. These calculations are provided in **Appendix D**. However, it is noted that every other location will have lower concentrations in air at ground level than at the maximum off-site location which means the risks will be lower for all other locations than those indicated in **Table 5.12**.

The predicted ground level concentrations used in this short term assessment are those derived from considering the maximum loading for the operation of the facility. These are the values determined for Scenario 2 as described in **Section 4.5.5**.

Information about the relevant short term health based criteria adopted for each chemical from reputable sources is provided in **Appendix B**.

For this assessment, the acute toxicity reference values (TRVs) have been selected on the basis of the following approach:

- Acute guidelines relevant to a 1-hour average exposure period are preferred.
- The TRVs have been selected on the basis of the following hierarchy:
 - Texas Commission on Environmental Quality (TCEQ) Acute Reference Value (Acute ReV), i.e. based on a target HI/RI of 1 (i.e. hazard index/risk index), consistent with the target HI/RI adopted in the derivation of guidelines in Australia (enHealth 2012a; NEPC 1999 amended 2013c, 2004) by the WHO (WHO 2000d, 2000e, 2010b). These are used as the primary source of acute guidelines as they specifically relate to and consider studies relevant to a 1-hour exposure and they have undergone the most recent detailed review process.
 - California Office of Environmental Health Hazard Assessment (OEHHA) acute Reference Exposure Level (REL), which are all based on a target HI/RI of 1 with RELs relevant to 1-hour average exposures adopted.
 - Other robust international sources including ATSDR.

Some pollutants are not considered to be acute toxicants, which means that they have a very low acute or short term toxicity – i.e. would require exposure to very high amounts for any effects to be seen. As a result, there are no suitable and robust acute inhalation guidelines available. For these pollutants, the assessment of chronic exposure is of most importance, which is evaluated on the basis of appropriate chronic toxicity values (discussed below). The pollutants where acute inhalation exposures have not been quantified are lead, dioxin-like compounds, thallium and PAHs.

An Environmental Protection Policy was published in Queensland in relation to air pollutants in 2019 (EPP (Air)) (Queensland Government 2019b). This includes air quality objectives for a range of pollutants that may be present in air. For many pollutants, objectives in this policy are based on chronic/long-term exposure (annual averages) and these are considered in the following sections.

Formaldehyde is the only pollutant relevant for this assessment that has a short term objective listed in the policy (other than the criteria pollutants – NO_x, SO_x, CO and PM).

A 30 minute average objective of 0.109 mg/m³ is listed for formaldehyde. This value is higher than the value recommended by TCEQ for a 1 hour average, so the more stringent value from TCEQ (i.e. 0.05 mg/m³) has been adopted for this assessment (compliance with this more stringent value will also mean compliance with the air quality objective from the EPP (Air)).

The ratio of the maximum predicted concentration to the acute guideline for each individual chemical is termed a hazard or risk quotient (RQ). When the maximum predicted concentration for an individual chemical is less than the guideline value that results in a risk quotient less than 1.

To deal with exposure to mixtures of chemicals, all risk quotients for the individual chemicals are summed to give a risk index (RI). When the total risk index for all chemical quotients added together is less than 1, it means the concentrations of each chemical must be well below each of their individual guideline values. This is a conservative approach to adopt.

Table 5.12 presents a summary of the relevant health-based guidelines, the predicted maximum 1-hour average concentration and the calculated RQ/RI for the maximum location in the off-site area. The assessment for other locations (maximum residential, maximum commercial, maximum other or

the various zones) are presented in **Appendix D**. All of these will pose lower risks than those listed in **Table 5.12**.

Table 5.12: Review of acute exposures and risks (maximum off-site – 1 hour average) – LP2

Pollutants	Acute air guideline (1-hour average) (mg/m ³)	Modelled Air Concentration		Calculated RQ
		(µg/m ³)	(mg/m ³)	
Hydrogen chloride (HCl)	0.66 ¹	40	0.04	0.06
Hydrogen fluoride (HF)	0.06 ¹	3	0.003	0.05
Cadmium	0.018 ¹	0.02	0.00002	0.001
Antimony	0.001 ³	0.03	0.00003	0.04
Arsenic	0.0099 ¹	0.0001	0.00001	0.001
Chromium (VI)	0.0013 ¹	0.05	0.00005	0.04
Cobalt	0.00069 ¹	0.01	0.00001	0.01
Copper	0.1 ²	0.05	0.00005	0.0005
Manganese	0.0091 ¹	0.08	0.00008	0.009
Nickel	0.0011 ¹	0.05	0.00005	0.04
Vanadium	0.03 ²	0.005	0.000005	0.0002
Benzene#	0.58 ¹	20	0.02	0.03
Formaldehyde#	0.05 ¹	20	0.02	0.4
Total Risk Index (VOCs as benzene)				0.3
Total Risk Index (VOCs as formaldehyde)				0.7
Target (acceptable Risk Index)				≤1

Notes:

References for health-based acute air guidelines (1-hour average)

- 1 = Guideline from the Texas Commission on Environmental Quality (TCEQ)
<https://www.tceq.texas.gov/toxicology/dsd/final>
- 2 = Guideline from California Office of Environmental Health Hazard Assessment (OEHHA)
<https://oehha.ca.gov/air/general-info/oehha-acute-8-hour-and-chronic-reference-exposure-level-summary>
- 3 = Guideline from ATSDR
- # = It is common practice to assume that all the volatile organic compounds (TVOCs) will be present as one of the more toxic compounds that are normally included in this group – often the choice has been benzene. This ensures a worst case assessment of risk. Both benzene and formaldehyde are compounds that are important to understand so for this assessment it has been assumed that all of the TVOCs will be present as benzene and also that they all will be present as formaldehyde to allow the risks to be estimated and to ensure there is no underestimation of risks.

Based on the assessment presented in **Table 5.12**, all the individual RQs as well as the total RI are less than 1 which is the value not to be exceeded as recommended by national health authorities. In this case, the individual risk quotients are between 2.5 times and 5,000 times lower than the relevant guideline based on short term exposure in air. The risk index is 1.4 (assuming all VOCs are present as formaldehyde) and 3 (assuming all VOCs are present as benzene) times lower than the value not to be exceeded as recommended by national health authorities (enHealth 2012a).

It is also noted that this assessment has assumed the maximum level of TVOCs (as per the European BREF requirements) will be discharged across the worst hour for dispersion and that all of these volatile chemicals will be either all benzene or all formaldehyde. This is a conservative approach as it is unlikely the maximum level of TVOCs will be reached and it will be definitely the case that 100% of the volatiles will be benzene or formaldehyde. The other chemicals that could be measured for this parameter (TVOCs) have screening guidelines for acute exposure that are higher than those for benzene or formaldehyde (i.e. less stringent) and so this approach is conservative.

On this basis, there are no acute/short term risk issues of concern in relation to inhalation exposures for the potential for health effects in the area surrounding the proposed facility.

5.5.3.4 Chronic exposures – approach

For the assessment of chronic exposures via inhalation, almost all the pollutants evaluated have a threshold guideline value that enables the predicted annual average concentration to be compared with a health based, or acceptable, guideline (i.e. reference concentrations).

The assessment has considered potential intakes of these chemical substances from background sources such as food in the commercial food supply, reticulated drinking water and urban air.

A small number of pollutants require assessment using a non-threshold approach. For these pollutants, health authorities determine a unit risk to be applied.

Information about the reference doses and unit risks adopted for each chemical from reputable sources is provided in **Appendix B**. Included in this appendix are more detailed toxicological profiles for some of the chemicals being assessed for this facility – the key pollutants that contribute most to the estimated risks and/or those that require more supporting information to explain how the reference doses were determined. As discussed above, the reference doses/concentrations and unit risks are designed to be conservative and include consideration of sensitive subpopulations.

To assess risk via inhalation, the long term average concentration of each pollutant from this facility is compared to the reference concentration recommended by health authorities. These reference concentrations pose negligible risk as discussed in **Appendix B**.

For threshold pollutants, the chronic exposure based quotients and index are calculated as follows:

$$\text{Risk quotient (RQ)} = \frac{\text{exposure concentration}}{\text{health based criterion adjusted for background levels}}$$

Where:

Exposure concentration = concentration in air relevant to the exposure period – annual average (mg/m³)

Health based criterion or tolerable concentration (TC) = health-based threshold protective of all health effects for the community (mg/m³)

Background = proportion of the TC to which people are already exposed from other sources/exposures such as water, soil or products (%). This part of the calculation means that if people are normally exposed to some amount of this pollutant, that exposure a person already has is taken into account.

$$\text{Risk index (RI)} = \sum_{\text{all chemicals and all pathways}} \text{RQs}$$

For non-threshold pollutants, the increased risk of cancer is calculated using the following approach:

$$\begin{aligned} \text{"Carcinogenic Risk" ("inhalation")"} \\ = \text{Adjusted exposure concentration} * \text{Inhalation Unit Risk} \end{aligned}$$

Appendix D presents all the calculations undertaken for inhalation exposures for the various land use types including maximum off-site, residential, commercial/industrial and on-site.

The Environmental Protection Policy in relation to air pollutants was published in Queensland in 2019 (Queensland Government 2019b). This includes air quality objectives for a range of pollutants that may be present in air. For many pollutants other than the criteria pollutants discussed in **Section 5.4**, the listed values in this policy are based on chronic/long-term exposure (annual

averages). They are, however, not always based on protection of human health (they may be based on odours etc).

Katestone (2026) undertook a comparison of the predicted annual average concentrations at the relevant maximum locations with the EPP (Air) values. This comparison has not been repeated here. Katestone (2026) found that predicted maximum concentrations for all pollutants were less than the value recommended in the EPP (Air) (i.e. in compliance).

This assessment has focused on guidelines that are specifically based on the protection of human health and include the use of up to date effects data wherever possible – i.e. have included recent review of the literature. It also includes consideration of exposures via inhalation and exposures via indirect pathways due to deposition of particles to the ground. This is designed to provide additional support to the view that this facility will make a minimal/negligible change in the air quality in the off-site area around the proposed facility and that any predicted change will pose a negligible risk to human health.

Sections 5.5.3.3 to 5.5.3.5 present the calculated individual substance risk quotients and risk indices relevant to the assessment of chronic inhalation exposures for both the threshold and non-threshold pollutants for all relevant receptors.

Potential risks due to exposures to all listed gases and chemical substances attached to fine particles have been assumed to be additive and the total RI (the sum of all individual substance RQ's) is also presented.

5.5.3.5 Chronic exposures – residential

Exposure to people living at a location and being home 24 hours a day, 365 days per year has been assessed for the modelled ground level concentration at:

- maximum off-site location
- maximum residential location (i.e. where houses are currently located)
- maximum commercial/industrial location (potential future location of a residence).

These modelled concentrations have been assessed for the emissions assumed for normal operations – i.e. LP1. The assessment is shown in **Tables 5.13** (threshold chemicals)/**5.14** (non-threshold chemicals).

The maximum off-site location will be a location close to the boundary of the facility – i.e. on the road to the north or along the boundary fence. It is not likely to be a location where houses might be constructed so assuming people could be present at this location 24 hours per day/365 days per year is a conservative worst case assessment.

These calculations have assumed that a person might be present at the relevant location 24 hours per day. Calculations have also been undertaken assuming that a person could live at the maximum off-site location and that the maximum commercial location could be transferred to residential zoning and that a person could live at that location. An additional conservative assumption included in these calculations is that all particles (and chemicals that are attached to them) are assumed to be small enough to reach down into the lungs – i.e. no adjustment for particles that only reach the throat has been included.

Table 5.13: Calculated chronic risks for inhalation exposures – Residential

Pollutants	Calculated RQ – Maximum off-site	Calculated RQ – Maximum residential	Calculated RQ – Maximum commercial/ industrial
Hydrogen chloride (HCl)	0.0006	0.0004	0.0005
Hydrogen fluoride (HF)	0.00009	0.00006	0.00007
Ammonia	0.00008	0.00006	0.00006
Cadmium	0.008	0.005	0.006
Thallium	0.00003	0.00002	0.00003
Mercury	0.0003	0.0002	0.0002
Antimony	0.0002	0.0001	0.0001
Arsenic	0.0004	0.0002	0.0003
Lead	0.0006	0.0004	0.0005
Chromium (Cr VI assumed)	0.02	0.01	0.01
Cobalt	0.0002	0.0001	0.0001
Copper	0.0000004	0.0000002	0.0000003
Manganese	0.001	0.0007	0.0007
Nickel	0.002	0.001	0.001
Vanadium	0.0001	0.00007	0.00007
Dioxin-like compounds	0.00004	0.00003	0.00003
PFAS (assumed as PFOS)	0.0000003	0.0000002	0.0000002
Benzene	0.001	0.0007	0.0007
Formaldehyde	0.003	0.0009	0.003
Total RI (VOCs as benzene)	0.03	0.02	0.02
Total RI (VOCs as formaldehyde)	0.035	0.02	0.03
Negligible risk		≤1	

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

Cadmium, chromium and formaldehyde have the highest individual risk quotients for normal operations. The maximum concentrations for these chemicals are 50 to 500 times lower than the guideline issued by national health authorities protective for continuous exposure (i.e. 24 hours a day, 365 days a year). The other pollutants range from more than 1,000 fold to 5 million fold lower than the relevant guidelines from national health authorities protective for continuous exposure (enHealth 2012a; NEPC 1999 amended 2013b).

The sum of all the risk quotients for the various chemicals indicates that the overall risk index is at least 30 times lower than the not to be exceeded value recommended by national health authorities (enHealth 2012a; NEPC 1999 amended 2013b).

Table 5.14: Non-threshold chronic risks for inhalation exposures – Residential land use

Pollutants	Calculated Risk – Maximum off-site	Calculated Risk – Maximum residential	Calculated RQ – Maximum commercial/industrial
PAHs	2×10^{-13}	1×10^{-13}	1×10^{-13}
Benzene	8×10^{-8}	5×10^{-8}	6×10^{-8}
TOTAL	8×10^{-8}	5×10^{-8}	6×10^{-8}
Negligible risk		$\leq 1 \times 10^{-5}$	

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

This assessment has assumed all total volatile organic carbon (TVOC) emitted by the facility will be present as benzene. This is a conservative assessment as these emissions (measured as TVOC or TOC) will actually be a mix of chemicals. Benzene has been used to assess this mix as it has the most sensitive guideline value so assuming all TVOC is benzene results in a conservative assessment. The maximum concentration of benzene anywhere in the off-site area is approximately

125 fold lower than the guideline issued by national health authorities protective for continuous exposure (i.e. 24 hours a day 365 days a year) (enHealth 2012a; NEPC 1999 amended 2013b). The risks at the other locations are even lower.

The maximum concentration of PAHs anywhere in the off-site area is more than 1 million times lower than the guideline issued by national health authorities protective for continuous exposure (i.e. 24 hours a day 365 days a year). The risks at the other locations are even lower (enHealth 2012a; NEPC 1999 amended 2013b).

5.5.3.6 Chronic exposures – commercial/industrial

Exposure to people working at a location for 10 hours a day, 240 days per year has been assessed for the ground level concentrations at the maximum off-site location and the maximum current commercial/industrial location. These modelled concentrations have been assessed for the emissions assumed for normal operations (i.e. Scenario 1). The results are shown in **Tables 5.15** (threshold chemicals)/**5.16** (non-threshold chemicals).

Table 5.15: Calculated chronic risks for inhalation exposures – commercial/industrial

Pollutants	Calculated RQ – Maximum off-site	Calculated RQ – Maximum commercial/industrial
Hydrogen chloride (HCl)	0.0002	0.0001
Hydrogen fluoride (HF)	0.00002	0.00002
Ammonia	0.00002	0.00002
Cadmium	0.002	0.002
Thallium	0.000009	0.000007
Mercury	0.00007	0.00005
Antimony	0.00005	0.00004
Arsenic	0.0001	0.00007
Lead	0.0002	0.0001
Chromium (Cr VI assumed)	0.005	0.004
Cobalt	0.00005	0.00004
Copper	0.0000001	0.00000007
Manganese	0.0003	0.0002
Nickel	0.0005	0.0004
Vanadium	0.00003	0.00002
Dioxin-like compounds	0.00001	0.000009
PFAS (assumed as PFOS)	0.00000008	0.00000006
Benzene	0.0005	0.0004
Formaldehyde	0.0009	0.0007
Total RI (VOCs as benzene)	0.009	0.007
Total RI (VOCs as formaldehyde)	0.01	0.007
Negligible risk	≤1	

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

Chromium, cadmium and formaldehyde have the highest individual risk quotients. These maximum concentrations, however, are still 200 to 1,000 times lower than the guideline issued by national health authorities for the protection of the general public adjusted to be relevant for worker exposure (i.e. 10 hours a day, 240 days a year). These general public based guidelines are relevant for such an assessment when the chemical is not resulting from the activities in a workplace (i.e. public health guidelines used rather than occupational health guidelines). The other pollutants range from 2,000 to 12 million times lower than the relevant guidelines for each chemical individually (enHealth 2012a; NEPC 1999 amended 2013b).

This assessment has also listed the sum of the individual risk quotients for all of the chemicals (i.e. risk index). This assumes all the chemicals act via the same mechanism in the body (i.e. additive risks). In this case, the sum is at least 100 times lower than the not to be exceeded value recommended by national health authorities in Australia (enHealth 2012a; NEPC 1999 amended 2013b).

Table 5.16: Non-threshold chronic risks for inhalation exposures – commercial/industrial

Pollutants	Calculated Risk – Maximum off-site	Calculated Risk – Maximum commercial/industrial
PAHs	4×10^{-14}	3×10^{-14}
Benzene	2×10^{-8}	1×10^{-8}
TOTAL	2×10^{-8}	1×10^{-8}
Negligible risk	$\leq 1 \times 10^{-5}$	

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

This assessment has also assumed all total organic carbon (TVOC) emitted by the facility will be present as benzene. This is a conservative assessment as these emissions (measured as TVOC) will actually be a mix of chemicals. Benzene has been used to assess this mix as it has the most sensitive guideline values (i.e. worst case). The highest estimated concentration of benzene anywhere in the off-site area is more than 500 times lower than the guideline issued by national health authorities for the protection of the general public adjusted for worker exposure (i.e. 10 hours a day 240 days a year) (enHealth 2012a; NEPC 1999 amended 2013b).

The maximum concentration of PAHs anywhere in the off-site area is more than 1 million times lower than the guideline issued by national health authorities for the protection of the general public adjusted for worker exposure (i.e. 10 hours a day 365 days a year). The risks at the other locations are even lower (enHealth 2012a; NEPC 1999 amended 2013b).

5.5.3.7 Chronic exposures – on-site

The potential for risks to workers and visitors to the visitor and education centre at the site has been assessed using the maximum on-site ground level concentrations. This scenario has been assessed based on assuming a person could work at the site in the visitor and education centre for 10 hours per day on 240 days per year. It is acknowledged that such workers will not be outside for much of the time they will be at work because they will primarily remain within the visitor and education centre but a conservative approach has been adopted for this assessment.

For visitors to the visitor and education centre, it would be expected that they would only be present for up to half a school day (i.e. 4 hours) on 1 day per year for the students and up to 12 days per year for teachers (assuming 1 teacher may bring multiple groups across the year). This would result in a much lower level of exposure over a year (48 hours compared to 2,400 hours for a person working in the education centre). If risks for a worker in the visitor and education centre are negligible, then the risks for visitors to the visitor and education centre will also be negligible. Again, it is acknowledged that visitors will not be outside for much of the time they will be at the site because they will primarily remain within the visitor and education centre but a conservative approach has been adopted for this assessment.

The results are shown in **Tables 5.17** (threshold chemicals)/**5.18** (non-threshold chemicals).

Table 5.17: Calculated chronic risks for inhalation exposures – on-site

Pollutants	Calculated RQ – Education centre worker
Hydrogen chloride (HCl)	0.00005
Hydrogen fluoride (HF)	0.000008
Ammonia	0.000007
Cadmium	0.0007
Thallium	0.000003
Mercury	0.00002
Antimony	0.00002
Arsenic	0.00003
Lead	0.00006
Chromium (Cr VI assumed)	0.002
Cobalt	0.00002
Copper	0.00000003
Manganese	0.00009
Nickel	0.0002
Vanadium	0.000009
Dioxin-like compounds	0.000004
PFAS (assumed as PFOS)	0.00000003
Benzene	0.00009
Formaldehyde	0.0003
Total RI (VOCs as benzene)	0.003
Total RI (VOCs as formaldehyde)	0.003
Negligible risk	≤1

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

Cadmium, chromium, nickel and formaldehyde have the highest individual risk quotients. These maximum concentrations, however, are still more than 500 times lower than the guideline issued by national health authorities for the protection of the general public. The other pollutants range from 10,000 to more than 30 million times lower than the relevant guidelines for each chemical individually (enHealth 2012a; NEPC 1999 amended 2013b).

This assessment has also listed the sum of the individual risk quotients (i.e. risk index). This assumes all the chemicals act via the same mechanism in the body (i.e. additive risks). In this case, the sum is at least 300 times lower than the maximum acceptable risk index recommended by national health authorities in Australia (enHealth 2012a; NEPC 1999 amended 2013b).

Table 5.18: Non-threshold chronic risks for inhalation exposures – on-site

Pollutants	Calculated Risk – Visitors Centre
PAHs	1×10^{-14}
Benzene	6×10^{-9}
TOTAL	6×10^{-9}
Negligible risk	$\leq 1 \times 10^{-5}$

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

This assessment has assumed all total organic carbon (TVOC) emitted by the facility is present as benzene. This is a conservative assessment as these emissions (measured as TVOC) will actually be a mix of chemicals. The concentration of benzene at the maximum on-site location is more than 1,600 times lower than the guideline issued by health authorities relevant for people working within the education centre.

The concentration of PAHs at the maximum on-site location is more than 10 million times lower than the guideline issued by national health authorities for the protection of the general public adjusted for worker exposure (i.e. 10 hours a day 365 days a year).

5.5.3.8 Chronic exposures – summary

Risks associated with chronic exposures by inhalation are considered to be negligible when the individual RQ's and total RI's are less than or equal to 1 for all exposure scenarios or when non-threshold risks are less than or equal to 1×10^{-5} for all exposure scenarios.

Based on the assessment presented in these tables (and in the spreadsheet images in **Appendix D**), all the individual RQs and total RI's for inhalation exposures at the worst case locations that may occur in residential locations or in commercial/industrial areas or on the site for the education centre are all less than 1 (and non-threshold risks are less than 1×10^{-5}).

On this basis, chronic risks are negligible in relation to inhalation exposures for all of the scenarios.

5.5.4 Multi pathway evaluation

5.5.4.1 General

Where pollutants may be bound to particulates, are persistent in the environment and have the potential to bioaccumulate in plants or animals, it is relevant to also assess potential exposures that may occur as a result of particles depositing onto soil where a range of other exposures may then occur.

These exposure pathways include:

- Incidental ingestion and dermal contact with soil (and dust indoors that is derived from outdoor soil or deposited particles).
- Ingestion of homegrown fruit and vegetables where particles may deposit onto the plants and may also be present in the soil where the plants are grown, and where pollutants bound to these particles are taken up into these plants (backyard veggie patch).
- Ingestion of eggs where particles may deposit onto the ground and be present in soil which the chickens come into contact with and incidentally ingest resulting in the pollutants bound to these particles being taken up into the eggs (backyard chickens).
- Ingestion of milk where particles may deposit onto the ground and be present in soil (which the pasture/feed grows in and animals also ingest when feeding), and the pollutants bound to these particles are taken up into milk (consumed on farm).
- Ingestion of meat where particles may deposit onto the ground and be present in soil (which the pasture/feed grows in and animals also ingest when feeding), and the pollutants bound to these particles are taken up into meat (consumed on farm).

These exposures are only relevant over the longer term, so this assessment has used the annual average deposition rates determined in the air quality modelling and has assumed that deposition into the soil occurs for at least 70 years. It is then the concentration in that soil for each pollutant which is assessed for uptake into fruit, vegetables, eggs, milk or meat.

The calculation of risks posed by multiple pathway exposures only relates to pollutants that are bound to the particles, not to pollutants only present as vapours or gases. Consequently, non-threshold risks for benzene or formaldehyde are not assessed for these other pathways. Both of these chemicals are present as a gas not attached to a particle. In addition, hydrogen chloride, hydrogen fluoride and ammonia are not relevant for this part of the assessment as they are always present as gases. The chemicals that are considered in this part of the assessment are the various

metals/metalloids, dioxin-like compounds, polycyclic aromatic hydrocarbons and per/polyfluoroalkyl substances.

Appendix C includes the equations and assumptions adopted for the assessment of potential exposures via these exposure pathways, with the calculation of risk for each of these exposure pathways presented in **Appendix D**.

Once exposure for each pathway has been assessed, the predicted daily intake of a pollutant from all pathways (i.e. inhalation, direct contact with soil and intake from consumption of home grown produce) can be compared to the reference dose recommended by health authorities as posing negligible risk.

Information about the reference doses adopted for each chemical from reputable sources is provided in **Appendix B**. Included in this appendix are more detailed toxicological profiles for some of the chemicals being assessed for this facility – the key pollutants that contribute most to the estimated risks and/or those that require more supporting information to explain how the reference doses were determined.

Sections 5.5.4.2 to 5.5.4.6 present the calculated risks associated with these multiple pathway exposures relevant to both adults and children for the various combinations relevant for each land use. These risks have been calculated on the basis of the maximum predicted deposition rate at the maximum relevant location for each land use type for normal operations. Consideration of short term upset conditions is not relevant for this part of the assessment as these do not contribute significantly to any change in the annual average deposition rate.

Each table presents the total RI for each exposure pathway separately, calculated as the sum over all the pollutants evaluated. The table also includes the calculated RI associated with inhalation exposures provided in **Section 5.5.3**, as these exposures are additive to the other exposure pathways. As noted above, these exposure pathways for deposition of particles are not relevant for benzene or formaldehyde (i.e. TOC).

In regard to this part of the assessment, the only group of chemicals that has a non-threshold mechanism of action are PAHs, so these chemicals have been assessed using a non-threshold approach.

5.5.4.2 Maximum off-site

The location in the off-site area with the maximum concentrations will be just outside the boundary of the site.

The maximum off-site location has been assessed based on assuming a person could live at that location breathing the air and having direct contact with soil for up to 35 years (6 as a child and 29 as an adult) and they can come into contact with soil on a daily basis. Home grown produce is not included in this part of the assessment.

The results are shown in **Tables 5.19** (threshold chemicals)/**5.20** (non-threshold chemicals).

Table 5.19: Summary of threshold risks for multiple pathway exposures – Maximum off-site

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	0.03	0.03
Soil ingestion (SI)	0.00002	0.0002
Soil dermal contact (SD)	0.000001	0.000003
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.03	0.03
Negligible risk		≤1

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the maximum off-site location, the total risk for all relevant exposure pathways is at least 30 fold lower than the value national health authorities indicate poses a negligible risk.

Table 5.20: Non-threshold chronic risks for multiple pathway exposures – Maximum off-site

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	8×10^{-8}	8×10^{-8}
Soil ingestion (SI)	1×10^{-16}	3×10^{-16}
Soil dermal contact (SD)	6×10^{-16}	2×10^{-16}
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	8×10^{-8}	8×10^{-8}
Negligible risk		≤1×10^{-5}

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the maximum off-site location, the total risk for all relevant exposure pathways is more than 125 times lower than the value national health authorities indicate poses a negligible risk.

5.5.4.3 Residential

General

The residential exposure scenario has been assessed for 3 location types:

- Maximum location which is currently used for residential purposes (**Table 5.21/5.22**)
- Maximum location which is currently used for commercial/industrial purposes but could be changed to a residential land use at some point in the future (**Table 5.23/5.24**)
- Maximum residential location which may be used as a farm at some point in the future (**Table 5.25/5.26**)

Residential

For the maximum residential locations, it has been assumed that a person may live there for up to 35 years (6 as a child and 29 as an adult) breathing the air, coming into contact with soil and growing and consuming home grown produce (fruit, vegetables, eggs).

In relation to potential farming sites, it has been assumed that a person may live there for up to 35 years (6 as a child and 29 as an adult) breathing the air, coming into contact with soil and growing and consuming home grown produce (fruit, vegetables, eggs) as well as that a location may also be used for on farm consumption of meat and milk.

For each of these locations, exposure (and risk) has been assessed for normal operations.

Table 5.21: Summary of threshold risks for multiple pathway exposures – current maximum residential

Exposure pathway	Calculated RI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	0.02	0.02
Soil ingestion (SI)	0.00002	0.0002
Soil dermal contact (SD)	0.000001	0.000002
Ingestion of homegrown fruit and vegetables (F&V)	0.00005	0.0001
Ingestion of homegrown eggs (E)	0.000006	0.00002
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.02	0.02
I + SI + SD + F&V	0.02	0.02
I + SI + SD + E	0.02	0.02
All pathways combined	0.02	0.02
Negligible risk		≤1

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the current maximum residential location in the area surrounding the proposed facility, the risk index is approximately 50 times lower than the value national health authorities indicate poses a negligible risk.

Table 5.22: Non-threshold chronic risks for multiple pathway exposures – current maximum residential

Exposure pathway	Calculated RI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	5×10^{-8}	5×10^{-8}
Soil ingestion (SI)	1×10^{-17}	2×10^{-17}
Soil dermal contact (SD)	5×10^{-17}	2×10^{-17}
Ingestion of homegrown fruit and vegetables (F&V)	3×10^{-16}	3×10^{-16}
Ingestion of homegrown eggs (E)	1×10^{-19}	6×10^{-20}
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	5×10^{-8}	5×10^{-8}
I + SI + SD + F&V	5×10^{-8}	5×10^{-8}
I + SI + SD + E	5×10^{-8}	5×10^{-8}
All pathways combined	5×10^{-8}	5×10^{-8}
Negligible risk		≤1×10^{-5}

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the current maximum residential location in the area surrounding the proposed facility, the total risk is more than 200 times lower than the value national health authorities indicate poses a negligible risk.

Commercial as residential

For the maximum commercial location, it has been assumed that a person may live there for up to 35 years (6 as a child and 29 as an adult) breathing the air, coming into contact with soil and growing and consuming home grown produce (fruit, vegetables, eggs).

Table 5.23: Summary of threshold risks for multiple pathway exposures– current maximum commercial/industrial assuming residential could occur at that location

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	0.03	0.03
Soil ingestion (SI)	0.00001	0.0001
Soil dermal contact (SD)	0.0000007	0.000001
Ingestion of homegrown fruit and vegetables (F&V)	0.00001	0.00003
Ingestion of homegrown eggs (E)	0.000004	0.00001
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.03	0.03
I + SI + SD + F&V	0.03	0.03
I + SI + SD + E	0.03	0.03
All pathways combined	0.03	0.03
Negligible risk		≤1

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the current maximum commercial/industrial location, if this location should change to residential land use, the total risk is more than 30 fold lower than the value national health authorities indicate poses a negligible risk.

Table 5.24: Non-threshold chronic risks for multiple pathway exposures – current maximum commercial/industrial assuming residential could occur at that location

Exposure pathway	Calculated RI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	6×10^{-8}	6×10^{-8}
Soil ingestion (SI)	7×10^{-17}	1×10^{-16}
Soil dermal contact (SD)	3×10^{-16}	1×10^{-16}
Ingestion of homegrown fruit and vegetables (F&V)	7×10^{-17}	5×10^{-17}
Ingestion of homegrown eggs (E)	2×10^{-19}	1×10^{-19}
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	6×10^{-8}	6×10^{-8}
I + SI + SD + F&V	6×10^{-8}	6×10^{-8}
I + SI + SD + E	6×10^{-8}	6×10^{-8}
All pathways combined	6×10^{-8}	6×10^{-8}
Negligible risk		≤1×10^{-5}

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the current maximum commercial/industrial location if this location should change to residential land use, the total risk is approximately 150 fold lower than the value national health authorities indicate poses a negligible risk.

Residential as farming land

The proposed site for this facility is within the declared State Development Area, so it is some distance from rural zoned areas. However, an exposure scenario for a farm has been assessed for the maximum residential location. This location is within the transitional zone in the State Development Area.

It is assumed that a person may live at the farm for up to 35 years (6 as a child and 29 as an adult) breathing the air, coming into contact with soil, growing and consuming home grown produce (fruit, vegetables, eggs) and, in addition, keeping livestock for on farm consumption of meat and milk.

The results are shown in **Tables 5.25** (threshold chemicals)/**5.26** (non-threshold chemicals).

Table 5.25: Summary of threshold risks for multiple pathway exposures – current maximum residential – assuming a farm could occur at that location

Exposure pathway	Calculated RI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	0.02	0.02
Soil ingestion (SI)	0.00002	0.0002
Soil dermal contact (SD)	0.000001	0.000002
Ingestion of homegrown fruit and vegetables (F&V)	0.00005	0.0001
Ingestion of homegrown eggs (E)	0.000006	0.00002
Ingestion of meat/beef (B)	0.00003	0.00006
Ingestion of milk (M)	0.00003	0.0001
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.02	0.02
I + SI + SD + F&V	0.02	0.02
I + SI + SD + E	0.02	0.02
I + SI + SD + B	0.02	0.02
I + SI + SD + M	0.02	0.02
All pathways combined	0.02	0.02
Negligible risk		≤1

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the current maximum residential location in the area surrounding the proposed facility, the risk index is approximately 50 times lower than the value national health authorities indicate poses a negligible risk if this location was to be used as a farm and produce grown on site is consumed year round by those living at the farm.

Table 5.26: Non-threshold chronic risks for multiple pathway exposures – current maximum residential – assuming a farm could occur at that location

Exposure pathway	Calculated RI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	5×10^{-8}	5×10^{-8}
Soil ingestion (SI)	1×10^{-17}	2×10^{-17}
Soil dermal contact (SD)	5×10^{-17}	2×10^{-17}
Ingestion of homegrown fruit and vegetables (F&V)	3×10^{-16}	3×10^{-16}
Ingestion of homegrown eggs (E)	1×10^{-19}	6×10^{-20}
Ingestion of meat/beef (B)	7×10^{-17}	4×10^{-17}
Ingestion of milk (M)	1×10^{-15}	9×10^{-16}
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	5×10^{-8}	5×10^{-8}
I + SI + SD + F&V	5×10^{-8}	5×10^{-8}
I + SI + SD + E	5×10^{-8}	5×10^{-8}
I + SI + SD + B	5×10^{-8}	5×10^{-8}
I + SI + SD + M	5×10^{-8}	5×10^{-8}
All pathways combined	5×10^{-8}	5×10^{-8}
Negligible risk		≤1×10^{-5}

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the current maximum residential location in the area surrounding the proposed facility, the non-threshold risk is approximately 200 times lower than the value national health authorities indicate poses a negligible risk, if this location was to be used as a farm and produce grown on site (including milk and meat) is consumed year round by those living at the farm.

Addition of exposure via home grown fruit, vegetables, eggs, meat or milk does not make a noticeable difference to the total risk estimates for all pathways combined. The risks estimated by exposure via inhalation alone drive the consideration of risks for this Project.

5.5.4.4 Commercial/Industrial

The maximum commercial/industrial location in the off-site area has been assessed based on assuming a person could work at that location breathing the air 240 days per year for up to 30 years as an adult. They are also assumed to potentially come into contact with soil where particles may have deposited. Exposure as a child has also been included in case children could regularly visit a workplace. Home grown produce is not included in this part of the assessment.

The results are shown in **Tables 5.27** (threshold chemicals)/**5.28** (non-threshold chemicals).

Table 5.27: Summary of threshold risks for multiple pathway exposures – current maximum commercial/industrial

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	0.003	0.003
Soil ingestion (SI)	0.00001	0.0001
Soil dermal contact (SD)	0.0000008	0.000002
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.003	0.003
Negligible risk		≤1

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

Table 5.28: Non-threshold chronic risks for multiple pathway exposures – current maximum commercial/industrial

Exposure pathway	Calculated RI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	5×10^{-9}	5×10^{-9}
Soil ingestion (SI)	9×10^{-18}	2×10^{-17}
Soil dermal contact (SD)	4×10^{-17}	2×10^{-17}
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	5×10^{-9}	5×10^{-9}
Negligible risk		≤1×10^{-5}

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the maximum commercial/industrial location in the area surrounding the proposed facility, the total risk for chemicals with a threshold is at least 300 times lower than the value national health authorities indicate poses a negligible risk and 2,000 times lower than the value national health authorities indicate poses a negligible risk for chemicals that are classified as non-threshold.

5.5.4.5 On-site

The maximum on-site location has been assessed based on assuming a person could work at that location breathing the air 240 days per year for up to 30 years as an adult. They are also assumed to potentially come into contact with soil where particles may have deposited. Exposure as a child has also been included as they may visit the education centre although that is likely to only occur once or twice per year for an individual. Home grown produce has not been considered for this part of the assessment as produce will not be grown at the facility site.

The results are shown in **Tables 5.29** (threshold chemicals)/**5.30** (non-threshold chemicals).

Table 5.29: Summary of threshold risks for multiple pathway exposures – on-site education centre

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	0.003	0.003
Soil ingestion (SI)	0.000004	0.00004
Soil dermal contact (SD)	0.0000002	0.0000005
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.003	0.003
Negligible risk		≤1

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the maximum commercial/industrial location in the area surrounding the proposed facility, the total risk is at least 300 fold lower than health authorities indicate is acceptable/negligible.

Table 5.30: Non-threshold chronic risks for multiple pathway exposures – on-site education centre

Exposure pathway	Calculated RI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	6×10^{-9}	6×10^{-9}
Soil ingestion (SI)	3×10^{-18}	6×10^{-18}
Soil dermal contact (SD)	1×10^{-17}	5×10^{-18}
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	6×10^{-9}	6×10^{-9}
Negligible risk		≤1×10^{-5}

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the maximum on-site location, the total risk for chemicals with a threshold is at least 300 times lower than the value national health authorities indicate poses a negligible risk and 1,500 times lower than the value national health authorities indicate poses a negligible risk for chemicals that are classified as non-threshold.

5.5.4.6 Rainwater tanks

The potential for impacts on rainwater tanks in the off-site area has been estimated using the maximum deposition rate for each chemical at the maximum location for residential and commercial/industrial land uses.

QLD Department of Health has issued guidance about the use of rainwater tanks (see **Appendix E**). Regulations in QLD require mosquito proofing of all tanks and that a first flush diverter must be fitted. First flush diverters are designed to exclude the initial debris that may be present on a roof

from entering the tank during the next rain event and this includes most of any particles that may settle onto a roof from the atmosphere.

For tanks that are used to collect water used for drinking, the requirements in QLD are that the roof and gutters are kept clean and that water quality in the tank is monitored on a regular basis. QLD Health recommends the use of town water supplies for drinking, showering and food preparation/cooking.

Rainwater tank maintenance should include:

- Check first flush diverter is working properly every 3 months
- Check the screens and other parts of the system to control mosquitos every 3 months (repair where necessary)
- Inspect and clean the roof and gutters every 6 months
- Leaf filters – check, clean and repair every 6 months
- Water quality – check for odour, colour and sediment every 6 months
- Internal inspection – check for evidence of animal intrusion into the tank (including mosquitos) every 6 months – if present identify and close access points
- Accumulated sediment – check inside the tank and remove accumulated sediment where necessary every 2 years.

The most reliable drinking water supply in urban areas will be the reticulated public water supply. In urban areas, the quality of water in rainwater tanks can be less reliable. The public water supply is filtered and disinfected as well as regularly monitored to ensure it is of appropriate quality. The use of rainwater tanks for the supply of water for non-drinking uses is useful for maximising water saving opportunities.

This advice indicates that water in rainwater tanks should not be used for potable purposes without appropriate, regular controls. Regardless, the estimated concentrations in a rainwater tank have been assessed assuming all dust on the roof can wash into the tank (i.e. no first flush device) and considering both ingestion and direct contact exposure pathways in a similar fashion to the calculations for contact with soil. The assumptions used in the calculations are those relevant to the Australian Drinking Water Guidelines from NHMRC (NHMRC 2011 updated 2025). This assessment has been undertaken for Scenario LP1 – i.e. normal operation.

Drinking water guidelines are used to define water of a quality that is suitable for uses around the home, so these guidelines are considered appropriate to assess water used for various domestic uses including showering, cooking, cleaning and irrigation.

Appendix C includes the equations and assumptions adopted for the assessment of potential exposures via deposition into a rainwater tank, with the calculation of risk presented in **Appendix D**.

The rainwater tank exposure scenario has been assessed for:

- Maximum location which is currently used for residential purposes (**Table 5.31/5.32**)
- Maximum location which is currently used for commercial/industrial purposes (**Table 5.33/5.34**)

Table 5.31: Summary of threshold risks for rainwater tanks – maximum residential location

Exposure pathway	Calculated RI	
	Adults	Children
Individual exposure pathways		
Water ingestion	0.000004	0.000004
Water dermal contact	0.00000003	0.00000008
Total	0.000004	0.000004
Negligible risk		≤1

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the maximum residential location, the total threshold risk related to using water from a rainwater tank is at least 250,000 fold lower than the value national health authorities indicate poses a negligible risk.

Table 5.32: Non-threshold chronic risks for rainwater tanks – maximum residential location

Exposure pathway	Calculated RI	
	Adults	Children
Individual exposure pathways		
Water ingestion	1×10^{-18}	2×10^{-19}
Water dermal contact	5×10^{-18}	2×10^{-18}
Total	6×10^{-18}	2×10^{-18}
Negligible risk		≤1×10^{-5}

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the maximum residential location, the total non-threshold risk related to using water from a rainwater tank is more than 10 million times lower than the value national health authorities indicate poses a negligible risk.

Table 5.33: Summary of threshold risks for rainwater tanks – maximum commercial/ industrial location

Exposure pathway	Calculated RI	
	Adults	Children
Individual exposure pathways		
Water ingestion	0.000003	0.000003
Water dermal contact	0.00000002	0.00000006
Total	0.000003	0.000003
Negligible risk		≤1

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the maximum commercial location, the total threshold risk related to using water from a rainwater tank (should a rainwater tank be present) is 300,000 fold lower than the value national health authorities indicate poses a negligible risk.

Table 5.34: Non-threshold chronic risks for rainwater tanks – maximum commercial/ industrial location

Exposure pathway	Calculated RI	
	Adults	Children
Individual exposure pathways		
Water ingestion	8×10^{-19}	2×10^{-19}
Water dermal contact	3×10^{-18}	2×10^{-18}
Total	4×10^{-18}	2×10^{-18}
Negligible risk		$\leq 1 \times 10^{-5}$

Notes:

Refer to **Appendices B, C and D** for detail on health based criteria and risk calculations

At the maximum commercial location, the total non-threshold risk related to using water from a rainwater tank is more than 10 million times lower than the value national health authorities indicate poses a negligible risk.

5.5.4.7 Drinking water catchment

BERC is located in the Logan-Albert catchment (Refer **Figure 5.2**). The Logan-Albert Catchment Action Plan 2017-2020 states that:

The Logan-Albert catchment is the third largest in SEQ with a combined area of approximately 3,800 km²... Several large dams, weirs and water treatment plants (WTP) supply water for irrigation, industry and urban use in the Logan-Albert catchment. There are currently four WTPs that provide water to local towns:

- Rathdowney WTP – draws water from Rathdowney Weir on the Logan River
- Koorabyn WTP – the Koorabyn WTP draws from the Logan River
- Canungra WTP – draws water from Canungra Creek
- Beaudesert WTP – water sourced from Bromelton Weir and Beaudesert Weir both on the Logan River.

Seqwater are planning for a new water treatment plant downstream from the existing Wyaralong Dam and a connection to the SEQ Water Grid as an additional drinking water source.

This facility will be located within the catchment of the Eastern Logan River sub-catchment as shown in **Figure 5.2**.

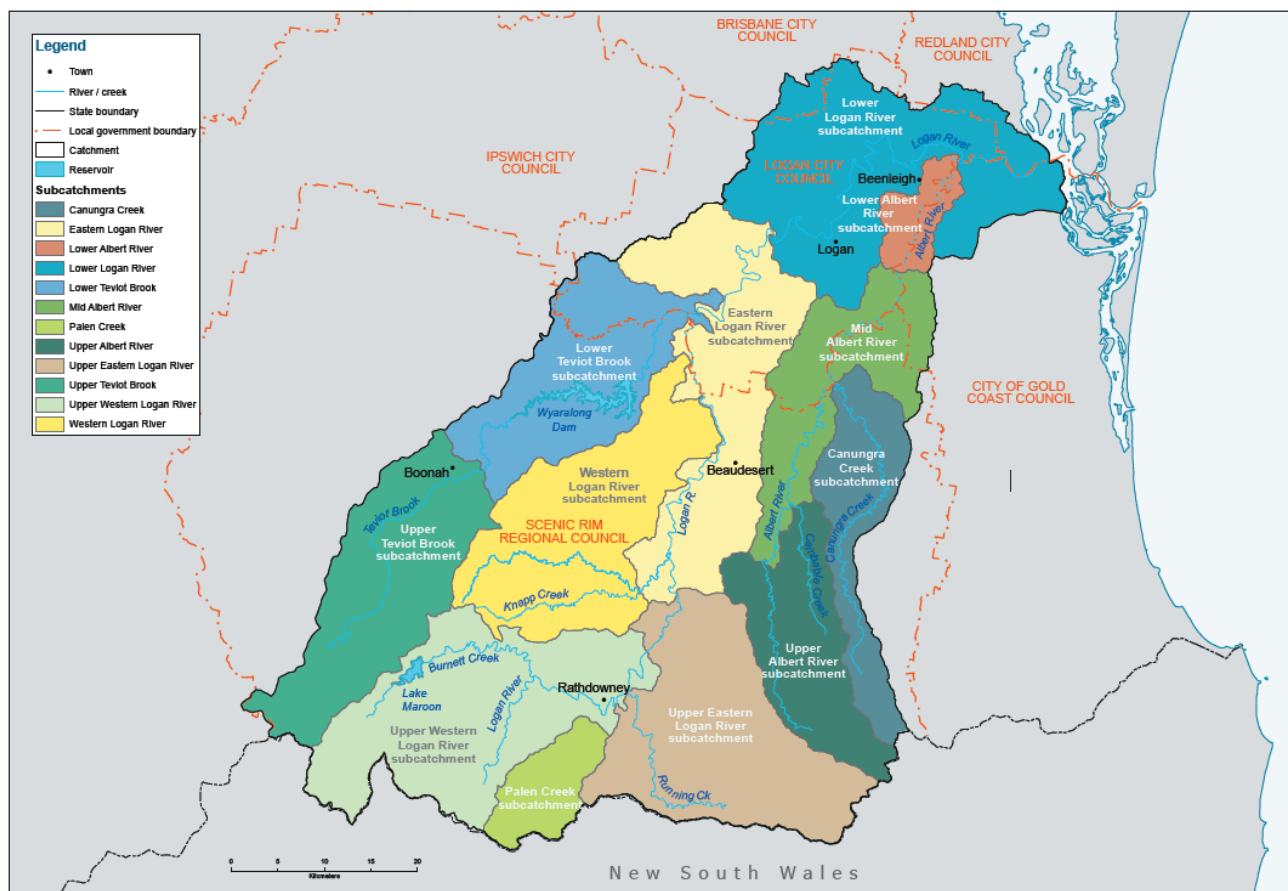


Figure 5.2: Logan-Albert Catchment

Logan River is around 5 km to the east of the site and Canungra Creek is around 20 km to the south east of the site. The Wyalong Dam is more than 5 km to the northwest of the proposed facility.

The strongest prevailing winds in this area blow to the north and northeast – i.e. away from Wyalong Dam but toward Logan River.

The commercial land uses closest to the facility are located immediately across the Beaudesert Boonah Road to the north of the facility – around 100 m. The residential land uses closest to the facility are located to the north/northwest of the facility at around 1.6 km, to the north/northeast of the facility at around 2.1 km and to the south of the facility at around 2 km.

Section 5.5.4.7 provides an assessment of how these worst case estimates of emissions from this facility at the closest relevant locations could impact on water quality in a rainwater tank. This assessment shows that potential concentrations in such tanks will be >200,000 times lower than the relevant guideline from national health authorities. This assessment looks at deposition of particles onto a roof and then assumes those particles will wash into the rainwater tank.

This is also considered a reasonable assessment of potential for this facility to impact on drinking water catchments like the Logan-Albert catchment for the following reasons:

- Characteristics of catchment vs rainwater tank
 - Volume of water in the catchment is much larger than that present in a rainwater tank.
 - Particles on a roof more easily wash off into a tank compared to particles on the ground or vegetation so a higher percentage of what will fall onto a roof will end up in the tank.
 - Water in Logan River is flowing so is continuously moving through the system whereas the rainwater tank assessment assumes the average rainfall across a year will be collected into the tank and no water will leave the tank.
- Emissions of particles from this facility
 - Deposition of particles onto the ground from this facility (based on worst case estimates of emissions) decreases with increasing distance from the facility so the rainwater tank assessment is conservative, given that Logan River is around 5 km to the east of the facility.
- Other sources of particles already in the catchment
 - Combustion sources like vehicles, wood fired heaters, power stations, BBQs etc all emit particles similar to those from this facility which are already present in the catchment – these particles contain similar chemicals as the ones from this facility as fuel used for burning (whether waste such as for this facility, or petrol, diesel, coal, wood, gas, etc) all generate the same sorts of chemicals attached to particles).
 - There are many other sources of particles in air such as windblown dust, salt from sea spray, breakdown of vegetation etc – these particles contain similar chemicals as the ones from this facility particular metals as they are present in soil, dust, vegetation etc.

Figures 5.3 and 5.4 shows how much particles from wood heaters changes the concentrations of particles in air in winter to emphasise the existing situation without this facility being present.

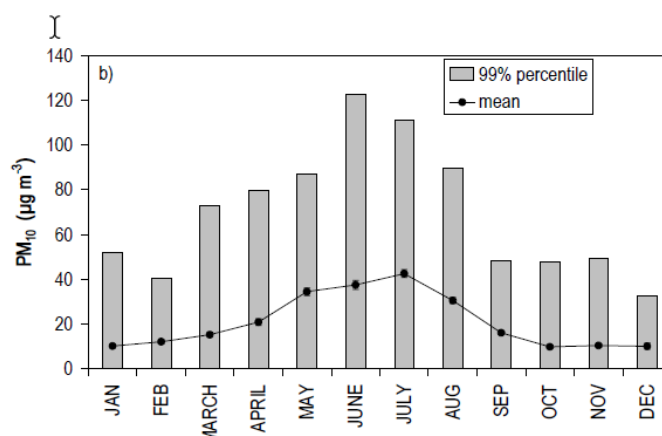


Figure 5.3: Variation in PM₁₀ concentrations across a year for Launceston (averages for each month for data from 1997 to 2005⁹)

⁹ <https://nla.gov.au/nla.obj-1382362253/view>

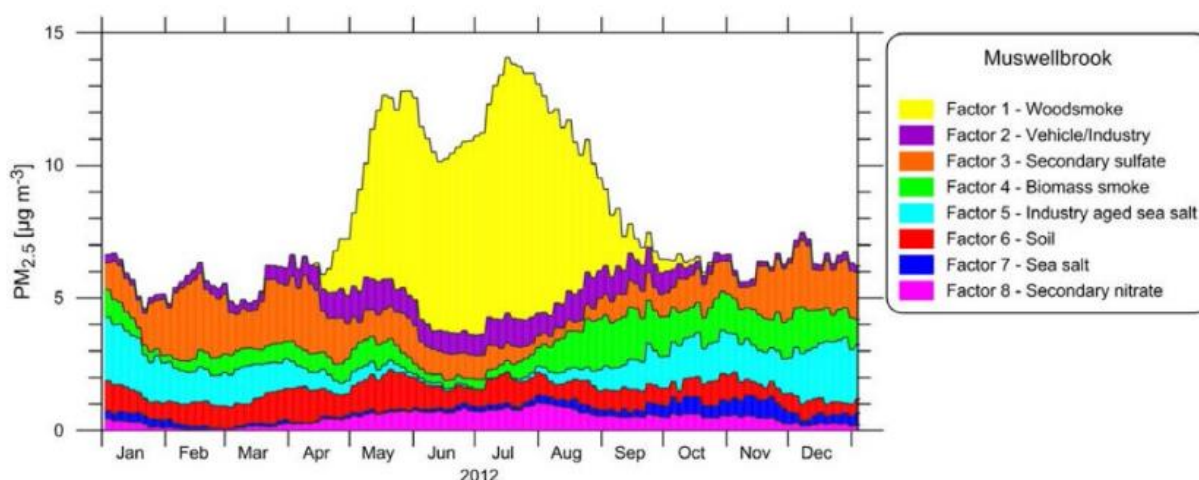


Figure 5.4: Changes in PM_{2.5} across a year at Muswellbrook, NSW¹⁰

While it is acknowledged that these data are for locations that would be much colder in winter than the area around this facility, these figures highlight how existing sources (many of which are not well controlled/regulated) can impact on particle concentrations in air (and, therefore, deposition to waterways).

It is not straightforward to do a similar assessment for the Logan River, given the flow in the river and that information about how many particles that might deposit on the ground could wash off into the River.

Instead, the estimated ground level air concentrations at the closest commercial and residential locations are listed in **Table 5.35** to show how small the concentrations of these chemicals are estimated to be.

Table 5.35: Ground level concentrations estimated at closest commercial and residential locations

Key chemicals	Concentration mg/m ³	
	Maximum residential	Maximum commercial
Cadmium (Cd)	0.0000000212	0.0000000234
Thallium (Tl)	0.0000000145	0.0000000161
Mercury (Hg)	0.0000000357	0.0000000395
Antimony (Sb)	0.0000000377	0.0000000416
Arsenic (As)	0.0000000165	0.0000000182
Lead (Pb)	0.000000215	0.000000238
Chromium (Cr VI assumed)	0.0000000538	0.0000000594
Cobalt (Co)	0.0000000133	0.0000000147
Copper (Cu)	0.0000000471	0.0000000520
Manganese (Mn)	0.0000000805	0.0000000889
Nickel (Ni)	0.0000000648	0.0000000715
Vanadium (V)	0.0000000067	0.0000000074
Dioxin-like compounds	0.000000000001	0.000000000001
PAHs	0.0000000000004	0.0000000000004
PFAS (assumed as PFOS)	0.00000000001	0.00000000002

¹⁰ <https://www.environment.nsw.gov.au/publications/upper-hunter-valley-particle-characterization-study-final-report>

These locations are around 100 m from the proposed facility (commercial) or around 1.6-2 km from the facility (residential). These results indicate how small the levels of these chemicals are even this close to the facility. The Logan River is more than 5 km from the facility.

For the metals, these concentrations are much less than 0.001 mg/m^3 . Measuring these metals in air is usually based on a limit of reporting around 0.001 mg/m^3 which means, even at the most impacted locations, the change in concentrations of metals in air due to this facility will not be measurable – with a margin of at least 5,000. The estimated concentrations at these worst case locations range from 0.0000002 to $0.00000007 \text{ mg/m}^3$.

For dioxin-like compounds, the air concentrations for this facility are estimated to be in the order of $0.000000000001 \text{ mg/m}^3$ (i.e. 1 femtogram/ m^3). Dioxin-like compounds are measured using high volume samplers which can collect air over multiple days. NSW EPA measured dioxin-like compounds around 2000 in urban areas and at Siding Spring Observatory – a remote area (NSW EPA 2002). At Siding Spring Observatory, it took 24 days of collecting the sample to have sufficient material to analyse for the dioxin-like compounds. The concentration in air at this remote location was on average 0.6 femtogram per m^3 while concentrations in urban areas were around 10-15 femtogram per m^3 . The estimated increment from this facility will be similar to that measured at the fairly remote location. It will not be measurably different from existing concentrations in urban areas as it will be less than 10% of the existing concentrations.

For PAHs, the limit of reporting in the studies by NSW EPA was around 0.0000001 mg/m^3 . Concentrations reported in urban areas by NSW EPA were on average between 0.0000002 and 0.00002 mg/m^3 (NSW EPA 2002). The increment estimated for this facility of around $0.000000000004 \text{ mg/m}^3$ will not be measurable with a margin of 500,000.

For PFAS, there are few methods for analysing in air but there are 2 USEPA methods for use in stack testing – OTM-45 and OTM-50. The reporting limit for OTM-45 is in the range 0.0000005 mg/m^3 . The increment estimated for this facility is around $0.00000000002 \text{ mg/m}^3$. Given the limit of reporting of this method, this increment will not be measurable with a margin of 25,000.

If the incremental concentrations are not measurable by relevant sampling and analysis methods and are not actually different from commonly found levels of these chemicals in existing urban air, even at the worst case locations around this facility where people live and work, then it would not be possible for emissions from this proposed facility to make a noticeable change in concentrations of these chemicals normally present in surface water – Logan River or the Dam.

Accordingly, emissions from the BEREC are not anticipated to cause a measurable or discernible impacts on water quality within the Logan-Albert Catchment, and are, therefore, not expected to pose a risk to current or future drinking water supplies.

To provide further evidence to support this conclusion, Table 5.36 lists the estimated concentrations in a rainwater tank at the worst case locations around the facility (where people live or work) and compares them to the Australian drinking water guideline (NHMRC 2011 updated 2025) (or equivalent – USEPA Regional Screening Levels for tapwater have been adopted where necessary (USEPA 2024)) for each relevant chemical.

Table 5.36: Concentrations in rainwater tanks at maximum residential location compared to Australian drinking water guidelines

Key chemicals	Concentration mg/L	
	Maximum residential	Australian drinking water guideline
Cadmium (Cd)	0.000000004	0.002
Thallium (Tl)	0.000000008	0.0002 (USEPA RSL)
Mercury (Hg)	0.000000007	0.001
Antimony (Sb)	0.000000001	0.003
Arsenic (As)	0.000000009	0.01
Lead (Pb)	0.000000004	0.005
Chromium (Cr VI assumed)	0.000000004	0.05
Cobalt (Co)	0.000000004	0.006 (USEPA RSL)
Copper (Cu)	0.000000002	2
Manganese (Mn)	0.000000002	0.1
Nickel (Ni)	0.000000002	0.02
Vanadium (V)	0.0000000001	0.09
Dioxin-like compounds	0.0000000000000000003	0.00000001 (USEPA RSL)
PAHs	0.000000000000000004	0.00001
PFAS (assumed as PFOS)	0.000000000003	0.000008 (as PFOS)

This comparison shows that the estimated concentrations in a rainwater tank due to emissions from this facility are at least 25,000 times lower than the relevant drinking water guideline. In most cases, the estimated concentrations are more than 1 million times lower than the guideline.

A rainwater tank holds only a small volume of water (compared to a drinking water catchment). This assessment washes particles from an entire roof into that smaller volume of water and assumes that no water leaves the tank across the year. The comparison in **Table 5.36** based on these assumptions about tanks is considered a reasonable estimate for concentrations in water across the drinking water catchment because:

- these are the concentrations at the worst case location for a residential area
- deposition of particles in areas closer to the Logan River etc will be lower than what occurs at the maximum residential location
- wash off from landscape is not as easy as from a roof – particles get stuck in grass and other vegetation (and are restricted by buildings, roads etc) so fewer particles per area will wash off into the Logan River etc during rain than for a roof/rainwater tank combination
- all of these chemicals are also present in particles from many other sources and the extra amount from this facility will not change the deposition of particles into the drinking water catchment from the current situation.

5.5.5 Cumulative risk for these other pollutants

In addition to assessing the potential for incremental changes in air quality due to this proposed facility (i.e. changes just due to emissions for this facility), an assessment of cumulative changes in air quality has also been undertaken for those chemicals for which there are data. These data include emissions from other facilities in the area and existing air quality in this region. Existing air quality arises due to emissions from everyday activities of people including driving vehicles, cooking, BBQ'ing, gardening, construction, demolition, etc as well as a range of naturally occurring actions like windblown dust, bushfires etc.

In relation to the criteria pollutants (NO₂, SO₂, CO, particles), cumulative impacts on air quality covering both existing air quality, emissions from other facilities and potential emissions from this facility are discussed in **Section 5.4**.

For the other pollutants, existing concentrations were available for a subset of chemicals relevant for this assessment – arsenic, chromium, copper, manganese, nickel and benzene. Comparison of the ground level concentrations of these chemicals due to the increment related to this facility and that which is already existing in the area is provided in **Table 5.36**.

Table 5.36: Comparison – incremental vs cumulative air quality

Chemical	Maximum off-site (mg/m ³)		Maximum residential (mg/m ³)		Maximum commercial (mg/m ³)	
	Increment for this facility	Cumulative	Increment for this facility	Cumulative	Increment for this facility	Cumulative
Arsenic	0.00000002	0.00004	0.00000002	0.00002	0.00000002	0.00003
Chromium	0.00000008	0.00008	0.00000005	0.00005	0.00000006	0.00006
Copper	0.00000007	0.0001	0.00000005	0.00006	0.00000005	0.00007
Manganese	0.00000001	0.0001	0.00000008	0.00008	0.00000009	0.00009
Nickel	0.00000001	0.0001	0.00000006	0.00007	0.00000007	0.00007
Benzene	0.00003	3.5	0.00002	3.5	0.00002	3.5

These results show that the existing air quality in the area has concentrations for these chemicals that are more than 1,000 times higher than those that will be added by this facility. Such a small increment will not result in a noticeable (or measurable) change in the air quality in the area.

5.6 Summary

This assessment has shown:

- No unacceptable risks for criteria pollutants (NO_x, SO_x, CO, PM_{2.5}, PM₁₀)
- No unacceptable risks for short term exposures from the proposed facility at the maximum off-site location – all other locations will have lower concentrations and so risks will be lower
- No unacceptable risks for relevant exposure scenarios considering long term exposures (both via inhalation and after deposition onto soil and uptake into home grown produce) at:
 - Maximum off-site location
 - Maximum residential location (and maximum commercial/industrial and maximum other places if land use changes to residential)
 - Maximum commercial/industrial location
 - Maximum on-site location
- No unacceptable risks for relevant exposure scenarios for rainwater tanks.

5.7 Additional Considerations – PFAS

A group of chemicals that has been of concern to communities are the per- and polyfluoroalkyl substances (PFAS) which have been discussed in the media for sites where fire fighting foams may have been used (Defence bases and airports, in particular).

PFAS are a family of man-made fluorine-containing chemicals. They do not occur naturally in the environment. They have unique properties that make materials stain- and water-resistant. These unique properties also make them persistent in the environment and highly mobile in soil and water (i.e. they readily leach into groundwater). These chemicals are highly water soluble (and often

present as ions in solution) and most of the commonly present substances are not volatile (HEPA 2025).

These chemicals have been used in a wide range of products including:

- Fire fighting foams
- Packaging materials for food
- Waterproofing or stainproofing agents (e.g. scotchguard)
- Non-stick products (e.g. Teflon)
- Polishes
- Waxes
- Paints
- Cleaning products
- Surfactants used in chrome plating or electronics manufacture (HEPA 2025).

Concerns regarding this group of chemicals were raised internationally around 2000 when it first became possible to measure these chemicals in water and soil. A number of chemicals in this group have since been included on the list of chemicals regulated by the Stockholm Convention – an international treaty to which Australia is a party that requires uses of listed chemicals (long lived/persistent ones) to be reduced or eliminated.

Since 2000 many uses of these chemicals have been phased out. Such reductions are expected to continue, given the listing of these chemicals on the Stockholm Convention.

It is likely that low levels may be present in residual household waste due to their widespread use in products used in the general community and around homes (HEPA 2025). PFAS have been reported to be present in leachate from some landfills in Australia (Gallen et al. 2017). The presence of these chemicals in current and future residual household waste would be expected to continue to decrease and to already be much lower than the levels currently discussed in the scientific literature for existing landfills.

Recent studies have shown that stormwater runoff from urban areas contributes these chemicals to local waterways to levels above those being contributed by wastewater discharges like those from sewage treatment plants or landfills (Lewis et al. 2025). This is because these chemicals are present in many products that we use around our homes.

Methods for the analysis of these chemicals in air are not routinely available (HEPA 2025). There is also no requirement for analysis of these chemicals in emissions from similar plants in Europe. This is because it is difficult to measure these chemicals in air and also because they are only expected at low levels. These chemicals have not been added to the European BREF regulation even though it was most recently updated in 2019 – a time when PFAS might have been considered (EU 2019). There are also very few reports providing monitoring data.

Regardless of these difficulties, PFAS have been added to the air dispersion modelling undertaken for this Project in Katestone (2026).

Data to allow this modelling was taken from studies in Sweden (Björklund 2024; Björklund, Weidemann & Jansson 2023, 2024). These studies evaluated the potential presence of PFAS in flue gas from an EfW facility in Sweden combusting municipal solid waste (60%) and industrial waste (40%).

The air dispersion modelling for this proposed facility assumed that the concentrations measured by Bjorklund et al. 2024 could be present in the stack. The value chosen from this study to use for this assessment was the maximum concentration measured on any occasion in the untreated flue gas. This was a sample of gas collected at the exit of the boiler. It was taken from a location after activated carbon injection and after the baghouse filter, but before the acid gas scrubbing system. Gas from this location was sampled and analysed on 3 occasions. This is a conservative choice as it does not take into account the full effects of the flue gas treatment processes to reduce the concentrations of these chemicals in the final emissions from this facility. This is, however, quite appropriate, given the limited size of the existing dataset.

In addition, monitoring data are available for a facility in Denmark (Norfors Usserod Power Station) (see <https://norfors.dk/ikke-fundet-pfas-i-roeggassen>). These data were collected for samples from 2 of the lines at this facility in March 2023. Both samples did not show detections for any of the PFAS covered by the analytical method used supporting the use of data from Bjorklund et al. (2024) as conservative. The method used in the Danish monitoring included the normal set of individual PFAS compounds measured by Australian laboratories as well as 2-3 additional ones and was also a similar list to that covered in the Björklund et al. (2023, 2024) study discussed above. The limits of reporting used by the laboratory for this work were appropriate for consideration.

The air pollution control technology proposed for this Project can address the presence of these chemicals in the following processes:

- Combustion chamber – PFAS are present in materials that could be in the waste as mixtures. Within those mixtures, some of this group of chemicals are readily degradable at temperatures easily reached in the chamber. Some do require higher temperatures to breakdown. It is noted that much of the chamber will have temperatures in excess of 1,000°C and these temperatures along with sufficient oxygen will allow for effective combustion/breakdown of these chemicals.
- Acid gas treatment (injection of lime) – the air pollution control technology proposed includes a process for removing acid gases from the air. This treatment process will also assist in the removal of the breakdown products from the destruction of PFAS.
- Activated carbon treatment – activated carbon is added to the waste gases to remove metals and a range of other chemicals. This technology will also assist in removing PFAS.
- Baghouse filter – chemicals attached to particles (including activated carbon particles) are captured within the filter bags in the baghouse. This will include particles to which PFAS are attached.

These studies found that individual PFAS reported in these samples included PFBA, PFHxA, PFHpA, PFOA, PFDA, PFBS and PFOS. The largest contributors to the sum of PFAS in these samples were PFBA and PFHxA. These 2 chemicals are not bioaccumulative and their toxicity is less than PFOS (the individual PFAS considered to be most toxic) (Brendel et al. 2018; Conder et al. 2008). These chemicals (PFBA and PFHxA) are, therefore, not chemicals that would accumulate in soil, water or agricultural produce in the community.

Regardless, to ensure this assessment was appropriately conservative in relation to this group of chemicals the following matters were included in the modelling:

- Maximum concentration in the Swedish studies for flue gas that had not been fully treated was adopted

- Maximum concentration used in the calculations was the sum of all PFAS detected in the study was assumed to be PFOS rather than PFBA and PFHxA.

It is, therefore, considered that this assessment is conservative and appropriate. Results for ground level concentrations for PFAS have been included in all the calculations in **Sections 5.5.3 and 5.5.4**. The contribution made by PFAS in these risk calculations was extremely low so risks due to the presence of the expected very low to negligible levels of these chemicals within residual waste to be combusted at this facility are also expected to be negligible.

5.8 Uncertainties

The characterisation of potential health risks related to exposures to emissions to air from the proposed EfW facility has utilised data from the air quality modelling as well as a number of assumptions. The following presents further discussion on the data and parameters, the level of uncertainty in these values and whether changes in these values will change the outcome of the assessment presented.

Air modelling

The modelling of air emissions has been undertaken by Katestone (2026) using a regulatory approved model, which utilises meteorological and terrain data for the local area. The emissions data used in the assessment were based on the maximum permissible levels as provided in Queensland regulation/EU regulation (EU 2010, 2019). It is expected that the modelled ground level concentrations are appropriately conservative.

Inhalation exposures

Residential exposures

The assessment has assumed that residents are home 24 hours per day, every day of the year for as long as they live at their home. This is an overestimate as most people spend time away from home at childcare, school, work or other activities and for holidays away from the home. When they are away from their houses, they will breathe the air (and whatever it contains) in the location where they are, not that present at their homes. As a result, the risks calculated for inhalation exposures are considered conservative.

In particular, it has been assumed that someone might be present at the worst case location anywhere off-site for 24 hours a day, 365 days per year. Even at this location (which will be on the road outside the facility or around the boundary of the site), the total risk is at least 100 fold less than the maximum acceptable value for this facility alone. It is not possible for anyone to live at this location.

In addition, the potential risks via inhalation have been assessed for the maximum residential as well as the maximum commercial/industrial locations assuming people could one day live at this location. This covers the situation if land use changes at a site closer to the facility at some time in the future. Such land use changes are considered unlikely in this location as the proposed facility will be located within an area that the Queensland Government has declared as a State Development Area.

Industrial workers

For workers at commercial/industrial sites around the proposed facility, it has been assumed that they are present at those locations for 10 hours a day, 240 days of the year. This scenario is slightly more conservative than the commercial/industrial scenario used to generate guidelines for such land in the National Environment Protection (Assessment of Site Contamination) Measure where workers are assumed to be present for 8 hours per day.

Multi-pathway exposures

These have been calculated on the basis of modelled dust deposition rates. It is noted that, due to presence of extensive flue gas treatment equipment in the proposed facility, the deposition rate is estimated to be very low. The potential for deposition to increase concentrations in soil for the relevant chemicals that could be present in the emissions from the proposed facility has still been estimated.

In addition, it is expected that the majority of particles to be emitted by this facility are likely to be in the PM_{2.5} size fraction (i.e. 2.5 micrometre or less). The ambient air NEPM notes that particles within the PM_{2.5} size fraction act as gases not particles (NEPC 2021b). This means that deposition of such particles will not occur under dry conditions as the particles are too small and too light to actually fall onto the ground. There will be some deposition during rain events.

The modelling for this assessment has assumed all the particles are in the PM₁₀ fraction and that they do deposit to the ground under wet and dry conditions. This results in a conservative assessment as the risks from these chemicals have been assessed via deposition and via remaining in the atmosphere (i.e. no deposition). So, the risks have actually been double counted but this allows a worst case assessment for chemicals that could deposit to land or be present in air attached to particles. This worst case assessment ensures risks have not been underestimated.

In addition, the assessment related to deposition assumes that the particles have deposited for 70 years prior to determining the concentration of a chemical that might be present in soil and available for uptake into produce. The soil concentrations likely to arise from deposition of particles for less than 70 years will be lower than those estimated in this assessment for 70 years of deposition.

The quantification of potential intakes via ingestion of soil, fruit and vegetables, milk, meat and/or eggs, and dermal contact with soil, has adopted a number of assumptions relating to how the dust mixes in with soil, how much accumulates in fruit and vegetables, milk, meat and eggs, and how people may be exposed. These assumptions have used conservative models and uptake factors that are likely to overestimate the accumulation of pollutants in soil, fruit and vegetables, milk, meat and eggs. In addition, default exposure parameters have been adopted assuming exposures occur all day every day, which is conservative. For example, ingestion rates for each of the food types have been based on guidance from FSANZ about a high end consumer – i.e. when they surveyed people the values chosen for use in these calculations are the amounts a person will consume of that food type based on what the top 10% of people consuming that food type said they ate on the survey day.

Overall, the approach taken will have overestimated actual exposures and risks. Changes in the assumptions to those more representative of actual exposures will result in lower levels of risk, rather than higher levels of risk.

5.9 Outcomes of health risk assessment

Table 5.37 presents a summary of the outcomes of the assessment undertaken in relation to the impacts on community health of changes in air quality, associated with the proposed Project.

Table 5.37: Summary of health impacts – air quality

Air quality	
Benefits	Provision of sufficient infrastructure to manage waste effectively in an urban area is a benefit to the health of the community.
Impacts	Negligible impacts to community health are predicted during both construction and operations in regard to changes in air quality arising from this Project.
Mitigation	Standard mitigation measures to control dust generated by construction projects will be considered for this Project including: • Record all dust and air quality complaints, identify cause(s), take appropriate measures to reduce emissions in a timely manner, and record the measures taken. • Make the complaints log available upon request from a regulating authority. • Record any exceptional incidents that cause dust and/or air emissions, either on- or offsite, and the action taken to resolve the situation in the log book. • Plan site layout so that machinery and dust causing activities are located away from sensitive receptors, as far as reasonably practicable. • Erect solid screens or barriers around dusty activities or the entire Project site that are at least as high as any stockpiles on site. • Avoid site runoff of water or mud. • Remove materials that have a potential to produce dust from site as soon as possible, unless being re- used on- site. If they are being re- used on- site cover as described below. • Cover, seed or fence stockpiles to prevent wind whipping. • Only use cutting, grinding or sawing equipment fitted or in conjunction with suitable dust suppression techniques such as water sprays or local extraction, e.g. suitable local exhaust ventilation systems. • Ensure an adequate water supply on the site for effective dust/particulate matter • Suppression/mitigation, using non- potable water where possible and appropriate. • Bonfires and burning of waste materials should not be permitted. • Covering or watering exposed stockpiles when not in use • Revegetating disturbed areas as soon as is feasible • Ensuring all fine powder materials such as cement are delivered in closed tankers and stored in silos • Ensure sand and other aggregates are stored in bunded areas • Avoid scabbling (roughening of concrete surfaces) if possible • Minimise drop heights from loading shovels and other loading or handling equipment, use enclosed chutes and cover skips • Hosing down truck tyres before exiting the site • Avoiding dry sweeping of large areas, instead using hoses, sprays, or water trucks. • Use water- assisted dust sweeper(s) on the access and local roads, to remove, as necessary, any material tracked out of the site. • Undertake daily on- site and off- site inspection, where receptors (including roads) are nearby, to monitor dust, record inspection results, and make the log available to regulatory authority when asked. • Carry out regular site inspections to monitor compliance with the DMP, record inspection results, and make an inspection log available upon request from a regulating authority. • Increase the frequency of site inspections by the person accountable for air quality and dust issues on site when activities with a high potential to produce dust are being

Air quality	
	carried out (such as major excavation works) and during prolonged dry or windy conditions. • Consideration will also be given to ensuring best practice management of emissions from plant and vehicles during construction including: • Ensure all vehicles switch off engines when stationary. There should be no idling vehicles. • Minimise the use of diesel or petrol powered generators if possible and use mains electricity or battery powered equipment where practicable. • Impose and signpost a maximum speed limit of 25 km/h on surfaced and 15 km/h on unsurfaced haul roads and work areas. Air pollution treatment processes will be included in this facility, in line with government and best practice requirements. No further mitigation measures are required for this facility, given the negligible risks identified in this assessment.

Section 6. Assessment of health impacts – noise

6.1 Approach

This section presents a review and further assessment of impacts on health associated with noise, relevant to the operation of the facility.

The assessment presented has relied on the information provided in the following noise and vibration (NV) report:

- Arup (2025b), Bromelton Energy and Resource Centre, Noise and Vibration Technical Report. Rev C Dated 19 December 2025.

6.2 Health impacts associated with noise

Environmental noise has been identified as a growing concern in urban areas because it has negative effects on quality of life and well-being and it has the potential to cause harmful physiological health effects. With increasingly urbanised societies, impacts of noise on communities have the potential to increase over time.

Sound is a natural phenomenon that only becomes noise when it has some undesirable effect on people or animals. Unlike chemical pollution, noise energy does not accumulate either in the body or in the environment, but it can have both short-term and long-term adverse effects on people.

These health effects include (WHO 1999a, 2011c):

- sleep disturbance (sleep fragmentation that can affect psychomotor performance, memory consolidation, creativity, promote risk-taking behaviour and increase risk of accidents)
- annoyance
- cardiovascular health
- hearing impairment and tinnitus
- cognitive impairment (effects on reading and oral comprehension, short and long-term memory deficits, attention deficit).

Other effects for which evidence of health impacts exists, but for which the evidence is weaker, include:

- effects on quality of life, well-being and mental health (usually in the form of exacerbation of existing issues for vulnerable populations rather than direct effects)
- adverse birth outcomes (pre-term delivery, low birth weight and congenital abnormalities)
- metabolic outcomes (type 2 diabetes and obesity).

Within a community, the severity of the health effects of exposure to noise and the number of people who may be affected are schematically illustrated in **Figure 6.1**.

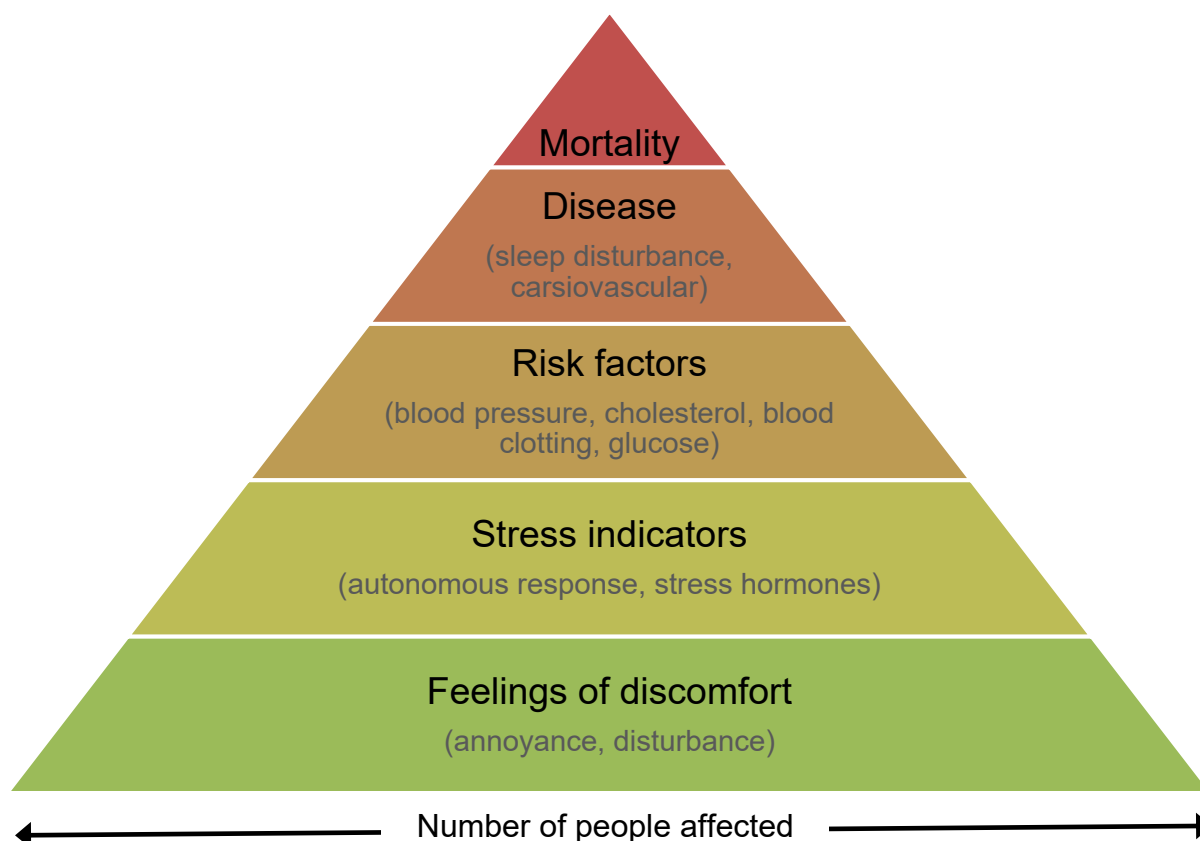


Figure 6.1: Schematic of severity of health effects of exposure to noise and the number of people affected (WHO 2011c)

Often, annoyance is the major consideration because it reflects the community's dislike of noise and their concerns about the full range of potential negative effects, and it affects the greatest number of people in the population.

There are many possible reasons for noise annoyance in different situations. Noise can interfere with speech communication or other desired activities. Noise can contribute to sleep disturbance, which has the potential to lead to other long-term health effects. Sometimes noise is just perceived as being inappropriate in a particular setting without there being any objectively measurable effect at all. In this respect, the context in which sound becomes noise can be more important than the sound level itself (I-INCE 2011; WHO 2011c, 2018).

Different individuals have different sensitivities to types of noise and this reflects differences in expectations and attitudes more than it reflects differences in underlying auditory physiology. A noise level that is perceived as reasonable by one person in one context (for example, in their kitchen when preparing a meal) may be considered completely unacceptable by that same person in another context (for example, in their bedroom when they are trying to sleep). In this case, the annoyance relates, in part, to the intrusion from the noise. Similarly, a noise level, which is considered to be completely unacceptable by one person, may be of little consequence to another, even if they are in essentially the same room. In this case, the annoyance depends almost entirely on the personal preferences, lifestyles and attitudes of the listeners concerned (I-INCE 2011; WHO 2011c, 2018).

Perceptible vibration (e.g., from construction activities) also has the potential to cause annoyance or sleep disturbance and adverse health outcomes in the same way as airborne noise. However, the health evidence available relates to occupational exposures or the use of vibration in medical treatments. No data is available to evaluate health effects associated with community exposures to perceptible vibrations (I-INCE 2011; WHO 2011c, 2018).

It is against this background that an assessment of potential noise impacts of the Project on health was undertaken.

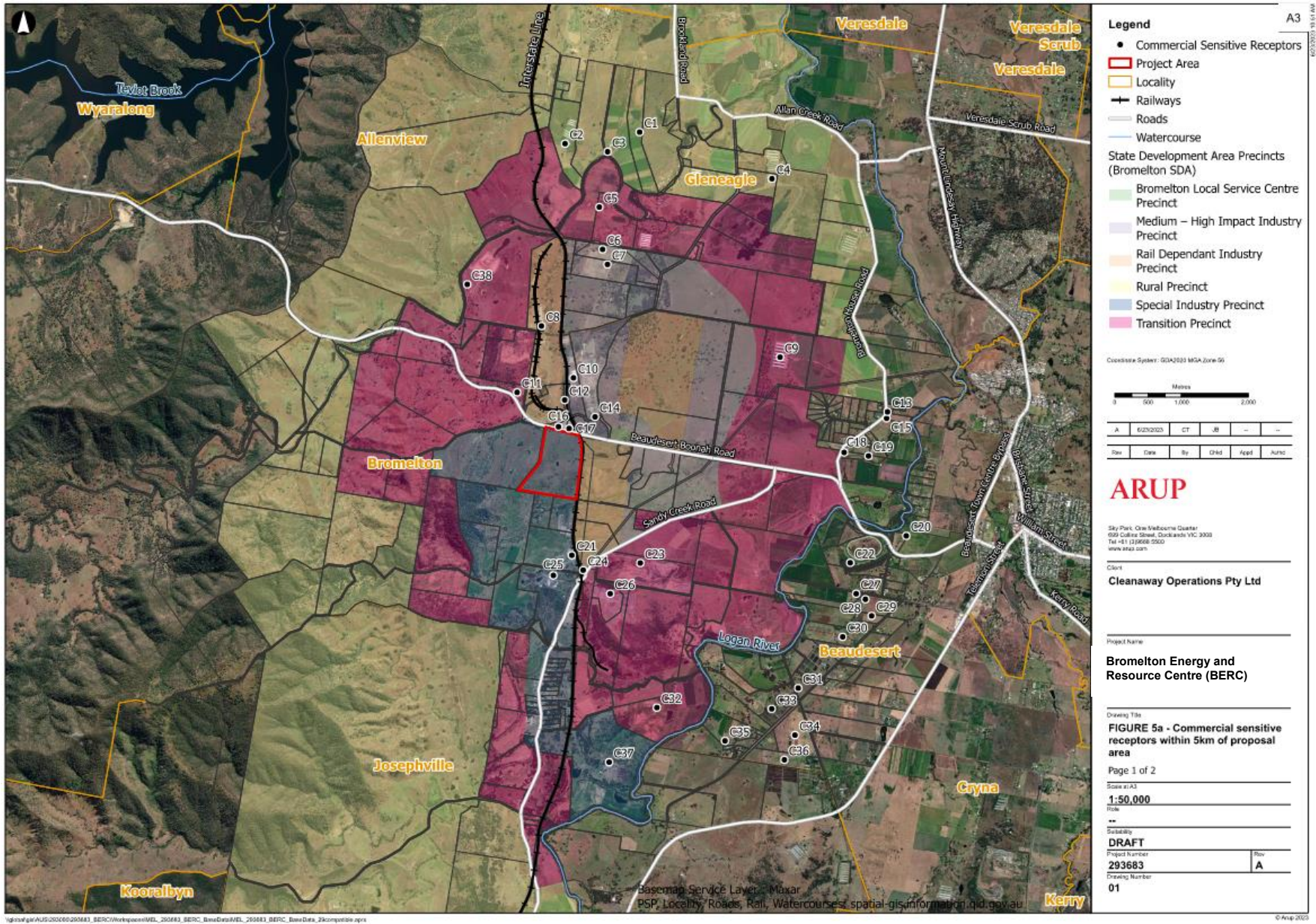
In relation to the available noise guidelines, the most recent review of noise by the WHO (WHO 2018) provides an update in relation to environmental noise guidelines (and targets) that more specifically relate to transportation (road, rail and air), wind turbines and leisure noise sources. The more comprehensive guideline levels for noise (related to all sources) remain the older WHO guidelines (WHO 1999a) and night noise guidelines (WHO 2009).

6.3 Summary of noise assessment

6.3.1 Background noise levels

Existing noise at the location of any proposed new project needs to consider the existing noise environment at that location. **Figure 6.2** shows the existing industrial, road and rail noise sources around the site proposed for this facility (Arup 2025b).

Figure 6.2: Existing industrial facilities and roads and rail noise sources surrounding the Project area



Legend

- Project Area
- Study Area
- Cadastre
- Noise sensitive receptors
- Industrial receptors

Noise Monitoring Locations

- A# Attended Monitoring Location
- L# Unattended Logger Location (plus attended)

Coordinate System: GDA2020 MGA Zone 56

Scale: 0 250 500 1,000

ARUP

4100 (Shelburne) Rd
Horseshoe Valley QLD 4000
Tel: +61 7 5500 5000
www.arup.com

Client:
Cleanaway Operations Pty Ltd

Project Name:
Bromelton Energy and Resource Centre (BERC)

Drawing Title:
Figure 2: Study Area with Sensitive Receptors and Noise Monitoring Locations

Scale of A3:
1:20,000

Issue:
ISSUE

Project Number:
293683

Drawing Number:
01

Revision:
A

The NV assessment (Arup 2025b) has evaluated background noise levels at 6 attended monitoring locations around the site (at rural residential locations in the main) and 3 unattended monitoring locations. One of the attended locations (A6) was within the Project site. Two of the unattended monitoring locations (L2 and L3) were on the northern and southern boundary of the Project site. The monitoring locations are shown on **Figure 6.3** (see above).

The nearest residential locations are listed in **Table 6.1**. They are all more than 1 km from the Project site (Arup 2025b).

Table 6.1: Nearest noise sensitive receivers (Arup 2025b)

ID	Noise sensitive receiver address	Distance to subject site boundary (m)	Rating background level (dB(A) $L_{A90, 1hr}$)		
			Day	Evening	Night
R16	356 Brabazon Road, Gleneagle	1,994	34	29	29
R19	140 Tilley Road, Bromelton	1,438	34	29	29
R21	2430 Beadesert Boonah Road, Bromelton	1,797	35	32	30
R56	149 Sandy Creek Road, Bromelton	1,843	31	29	29
R66	358 Sandy Creek Road, Bromelton	1,466	39	36	34

Monitoring was undertaken in June 2023. The dominant sources of noise noted by those undertaking the monitoring were road traffic, birds and an occasional light plane overhead.

The rating background level is the median value for each time period for which monitoring was undertaken. These values are reported in terms of L_{A90} which indicates it is the noise level that occurred for more than 90% of the 1 hour period over which measurement occurred.

Noise criteria adopted for assessing noise impacts in Arup (2025b) utilised the rating background levels as listed in **Table 6.1**.

6.3.2 Construction noise guidelines

Noise limits were determined to manage impacts from noise during construction using the Queensland Department of Transport and Main Roads (TMR) Transport Noise Management Code of Practice: Volume 2 – Construction Noise and Vibration (CoPV2) (2023). This guidance indicates that the construction noise criteria are developed by allowing noise levels 5 or 10 dB(A) above the rating background level depending on the time of day. A minimum lower limit of 45 dB(A) $_{Aeq,adj,15min}$ applies to times outside standard hours and 50 dB(A) $_{Aeq,adj,15min}$ applies as the minimum lower limit during standard work hours (Arup 2025b).

Using the background noise levels and this guidance gives the limits listed in **Table 6.2**.

Table 6.2: Construction noise limits (Arup 2025b)

Time of day	Management level (dB(A) $L_{Aeq, adj, 15 min}$)	
	Lower limit	Upper limit
Day (standard work hours) – residential locations (R16, R19, R21, R56)	45 or 50 depending on the length of time over which construction will occur	65
Day (standard work hours) – industrial locations (R66)	49 or 50 depending on the length of time over which construction will occur	65
Day/evening (non-standard hours) – all locations	45	45
Night (non-standard hours) – all locations	45	45

These are the limits to be applied during construction for this facility. These construction noise criteria indicate that existing background noise levels at this site are quite low and the lower limits and upper limits that are to be applied are the minimum specified in state guidance.

Once the criteria to be applied to the Project were established, noise modelling was undertaken to assess noise at the site during construction to determine if the Project could comply with these criteria. Information from relevant Australian and British Standards as well as Transport for NSW guidance was used to determine appropriate sound power levels for the equipment expected to be used at the site (Arup 2025b).

Noise modelling estimated that noise levels during construction at the identified nearby locations – i.e. worst case locations (Arup 2025b). The values estimated are shown in **Table 6.3**.

Table 6.3: Predicted noise levels during construction at worst case locations (Arup 2025b)

Work period	ID of relevant locations (worst case)	External noise level as per Table 6.2	Predicted noise levels during construction	
			Phase 1 of Project	Phase 2-4 of Project
Standard hours – i.e. DAY	R16	45	43	44
	R19		38	40
	R21		38	39
	R56		38	39
	R66	49	37	38
Non-standard hours – i.e. DAY/EVENING	R16	45	43	44
	R19		38	40
	R21		38	39
	R56		38	39
	R66		37	38
Non-standard hours – i.e. NIGHT	R16	45	43	44
	R19		38	40
	R21		38	39
	R56		38	39
	R66		37	38

These predicted noise levels have made assumptions about the types of equipment to be used on-site during construction. The predicted noise levels have been compared to the lower limit for permissible noise due to construction. In all cases, the predicted noise levels comply with the lower limits developed in line with Queensland guidance.

This means the potential for risks to community health in the off-site area due to changes in noise during construction is expected to be negligible.

6.3.3 Operational noise guidelines

Potential noise from the proposed facility has been assessed on the basis of guidance in the Environmental Protection Policy (Noise) (Queensland Government 2019a).

The EP Act lists obligations and duties to prevent environmental harm, nuisances, and contamination. One of the primary duties that applies is the general environmental duty (i.e. GED). This duty means a person must not carry out any activity that causes or is likely to cause, environmental harm unless all reasonable and practicable measures to prevent or minimise the harm have been taken (Arup 2025b).

Noise creating activities during operations are those generally expected in an urban area – things like vehicle movements etc. Most of the activities at the site will be undertaken within the main building or within structures. This means potential changes in the noise environment in the local area due to this new facility will be small.

The EPP (Noise) provides noise limits for people living nearby – the acoustic quality objectives. These criteria provided by Queensland government are to be achieved at the closest residence to a development. The values recommended for this facility are the values listed in terms of LA_{eq} as these were the most stringent of the options in the guidance. The values are listed in **Table 6.4**.

Table 6.4: Acoustic quality objectives for this Project (Arup 2025b)

	Time of day	Acoustic quality objective (dB(A) $LA_{eq,adj,1hr}$)
Residence – outdoors	Daytime and evening	50
Residence – indoors	Daytime and evening	35
	Night	30

The guidance also notes that an adjustment of 7 dB(A) to convert indoor values to outdoor values is permissible based on what is understood about typical homes in Queensland.

This means the acoustic quality objectives to be applied for this Project are:

- 42 dB(A) $LA_{eq,adj,1hr}$ applied to the external area (i.e. outside the house) for daytime/evening
- 37 dB(A) $LA_{eq,adj,1hr}$ applied to the external area (i.e. outside the house) for night.

These values are more stringent than the outdoor value listed above (i.e. 50 dB(A) $LA_{eq,adj,1hr}$) so are the most appropriate to apply.

6.4 Review and assessment of health impacts from noise

6.4.1 Review of proposed noise limits

Night

The WHO review of night time noise identified the following (WHO 2009):

- there is no sufficient evidence of biological effects that are harmful to health at noise levels below 40 dB $L_{night, outside}$
- adverse effects, however, have been observed at levels above 40 dB $L_{night, outside}$ such as self-reported sleep disturbance, environmental insomnia and increased use of drugs that assist with sleep.

The acoustic quality objective for nighttime adopted for the Project was 37 dB(A), as $LA_{eq,1hr}$, when applied to the outside a building.

The WHO guideline of 40 dB $L_{night, outside}$ applies to the whole nighttime period not just the average over 1 hour. Conversion of a noise level for the whole night to a value relevant as an average over 1 hour during the night can be undertaken by adding 3 dB(A) to the $LA_{eq,night}$ value. This means the relevant night criteria based on the WHO guideline is 43 dB(A) reported as $LA_{eq,1hr}$. This means the acoustic quality objectives proposed for this Project are definitely protective of health impacts.

The noise modelling for this Project indicates that the noise levels during the night period will be in compliance with relevant acoustic quality objectives for nighttime (i.e. equal to or less than 37 dB(A) as $LA_{eq,1hr}$). A value of 37 dB(A) $LA_{eq,1hr}$ is smaller than the guideline for nighttime recommended by

WHO (i.e. 43 dB(A) as $LA_{eq,1hr}$) so if the site will comply with the project acoustic quality objectives, it will also comply with the WHO guidance. This means the potential for risks to community health (e.g. sleep disturbance) in the off-site area due to changes in noise during operation is expected to be negligible in relation to noise at night.

Day

During the day, noise guidelines from WHO that are protective of moderate levels of annoyance in outdoor living areas are 50 dB(A) as LA_{eq} (outdoors) and 35 dB(A) (indoors). The indoor value is based on protecting conversations and learning for children (for day or evening periods) (WHO 1999a). These values apply to the whole day or the whole day and evening and are considered by WHO as protective for health effects from noise.

As discussed above, conversion of a noise level for the whole day to a value relevant as an average over 1 hour during the day can be undertaken by adding 3 dB(A) to the $LA_{eq,night}$ value. This means the relevant day/evening criteria from the WHO guidance is 53 dB(A) as $LA_{eq,1hr}$ for outdoors or 45 dB(A) as $LA_{eq,1hr}$ when the indoor value is converted to an outdoor/external value and also converted from a whole day or day/evening value to a 1 hour average value (i.e. use of 7 dB(A) to convert indoor to outdoor relevant values and use of 3 dB(A) to convert from all day/evening value to a 1 hour average. As noted above, the acoustic quality objective proposed for this Project of 50 dB(A) (outdoor) and 35 dB(A) (indoor) as 1 hour averages will be protective of community health impacts.

The noise modelling for this Project (without inclusion of any mitigation measures) indicates that the noise levels during the day once the facility is operational will comply with the acoustic quality objectives (i.e. equal to or less than 42 dB(A) as $LA_{eq,1hr}$) for all scenarios except for evening and night at R19.

Given that the WHO based guideline (i.e. 45 dB(A) as $LA_{eq,1hr}$) is higher than the project based acoustic quality objectives based on the EPP (Noise) regulation, then, if the noise impacts from this Project comply with the QLD guidance, the changes in noise will also comply with the WHO guidelines. The minor exceedances (when everything is unmitigated) at R19 are still in compliance with the WHO guidelines so, even under this scenario, impacts on community health due to changes in noise will be negligible. When mitigation measures such as those listed in **Section 6.4.3** are included in the modelling, changes in noise under worst case conditions comply with the project specific objectives and the WHO guidelines for the protection of community health at all locations.

6.4.2 Mitigation measures – construction

Although the risk of noise exceedances during construction is considered low based on the assessment, it has been still recommended that typical ‘industry best practice’ construction noise mitigation measures be put in place to minimise noise during construction (Arup 2025b).

A range of mitigation measures have also been recommended to ensure noise is well managed (to a greater extent than that used in the modelling) during construction. If these measures are included as well as those assumed in the modelling, then risks to health from changes in noise during construction will be even smaller.

The measures recommended include:

- A detailed Construction Noise and Vibration Management Plan (CNVMP) will be prepared. This plan will include:
 - Roles and responsibilities
 - Noise sensitive receiver locations/ areas of potential impacts (modelling indicates there are no locations where impacts are expected)
 - Noise mitigation strategy
 - Monitoring methods
 - Community engagement strategy
 - Minimum safe working distances for vibration intensive plant (to be planned and monitored as part of the CNVMP)
 - Safe working distances and equipment selections / mitigated work methods to prevent vibration impacts to rail corridor
 - Consultation with the utility provider of the telecoms line running near/along the northern boundary of the site to determine site specific vibration assessment requirements and whether any safe working or other recommended setback distances are required.
 - A Construction Transport Management Plan (including a Supply Chain Haul Route Management Strategy by the construction contractor as per TMR MRTS02) will be prepared to outline mitigation measures to avoid noise and vibration impacts from construction vehicles.

This document will feed into the overall site wide Construction Environmental Management Plan (CEMP).

Other general practices to reduce noise levels are likely to be required and could include:

- Adherence to standard approved working hours – as outlined in application
- Where work is required to be undertaken outside standard approved working hours – detailed management of noise to ensure such works are well controlled.
- Locating stationary plant as far as possible from sensitive locations
- Using site sheds and other structures/screens to limit noise leaving the site where possible (i.e. placing such structures between noise generating equipment and sensitive locations in the off-site area)
- Use of equipment that generates only low levels of noise to the extent practicable
- Modifications to equipment or timing of works when noisy works need to be undertaken
- Consultation with the local community throughout the construction process – providing early notice for noisy works and or disruption of travel etc around the site and including site

managers occasionally checking with nearby sensitive locations to see if noise levels are being managed appropriately

- Avoiding shouting, talking loudly or slamming vehicle doors
- Avoiding the use of radios or stereos in outdoor areas where the noise might carry
- Turning off equipment when not in use
- Training workers regularly to use equipment appropriately to minimise noise
- Avoiding the use of any public address systems.

As noted in **Section 6.3.2**, levels of noise at sensitive locations closest to the site (i.e. worst case locations) are not expected to exceed the lower limits as set in accordance with Queensland guidance. It is also noted that, given the area is relatively quiet, the limits for this Project set in accordance with Queensland guidance are the most stringent specified in that guidance. If these worst case locations are not unduly impacted by any changes in noise during construction of this facility, then all other locations that are further away will also not be affected.

Implementation of these monitoring and mitigation measures will give confidence in ensuring noise does not pose a risk to health in locations around the proposed facility while the facility is under construction.

6.4.3 Mitigation measures – operations

A range of mitigation measures have also been recommended to ensure noise is well managed (to a greater extent than that used in the modelling which did not include mitigation measures) during operation of this proposed facility.

If these measures are included as well as those assumed in the modelling, then risks to health from changes in noise during operation will be even smaller than estimated in the noise modelling.

The measures listed in the application include:

- Upgrade of roofing to ensure minimal noise impacts.
- Installing acoustically rated louvres at roof vents etc.
- Enclosing the IBA conveyor belt motor.
- Addition of an upgraded silencer on the stack flue exhaust.
- Installation of a noise barrier to the west, north and east sides of the main site transformer – extending to a height of 2.5 m above the height of the transformer (i.e. likely to be 6.5 m in height) and installation of sound absorbing material on the inside of those walls – including on the south side of the transformer (the wall of the main building).
- Attenuation added to mechanical ventilation fans relevant to the IBA area.

When all of these mitigation measures are included in the noise modelling for this Project, all locations – including R19 (evening and night) – are in compliance with the project specific acoustic quality objectives, confirming negligible risks to community health in the local area due to changes in noise during operation of this Project.

6.5 Outcomes of health risk assessment

Table 6.5 presents a summary of the outcomes of the assessment undertaken in relation to the impacts on community health of changes in noise, associated with the proposed Project.

Table 6.5: Summary of health impacts – noise

Noise	
Benefits	Provision of sufficient infrastructure to manage waste effectively in an urban area is a benefit to the health of the community.
Impacts	Negligible impacts to community health are predicted during both construction and operations in regard to changes in the noise environment arising from this Project. This conclusion is based on modelling which did not include noise mitigation measures.
Mitigation	Mitigation during construction involves normal/common practice matters like choosing low noise equipment, reminding workers regularly to use equipment in ways that minimise noise etc. This is in line with requirements at most construction sites – whether industrial facilities or homes. Mitigation during operations involves adding some additional noise mitigation measures into the design of the facility – applying enclosures around motors and transformers as well as applying low noise fittings to the stack and vents on buildings.

Section 7. Assessment of health impacts – water, soil contamination, chemicals management, groundwater

7.1 Approach

This section provides a summary of any potential health impacts associated with other aspects of the proposed Project, such as water, traffic or contamination. This review has relied on information available in the following reports:

- Douglas Partners (2025). Report on Preliminary Site Investigation for Contamination Bromelton Energy and Resource Centre (BERC) Lot 1 Beaudesert Boonah Road, Bromelton, QLD. Dated 19 December 2025.
- Arup (2026a). Bromelton Energy and Resource Centre (BERC). Hazards and risk. Rev C Dated 22 January 2026.
- Arup (2026b). Stormwater management plan and flood risk assessment, Bromelton Energy and Resource Centre (BERC). DRAFT Dated 4 February 2026.
- Arup (2025c). Bromelton Energy and Resource Centre, Bushfire Hazard Assessment. Dated 19 December 2025.

These aspects have been assessed in line with Queensland requirements for consideration of the potential for a community to be exposed to health or safety hazards over the short or long term due to emissions to air, water, noise or chemical hazards from a proposed facility.

7.2 Contamination

Douglas Partners (2023) describes a desktop assessment of the history of the site to identify potential for soil contamination, a site walkover and a limited program of soil and groundwater sampling. Surface water sampling in the dams to the south of the site was also undertaken (Douglas Partners 2023).

The site history indicates the site has been used for agricultural purposes since at least 1944. The main area where contamination could be present is the location of a dip site/spray race. There is limited information about the exact locations of these activities and for how long such activities occurred. The site has not been listed on the QLD registers for contaminated land. It is noted that a Notifiable Activity Notification Form was submitted to DETSI on 15 December 2025 providing evidence of the historical presence of the spray race on the site (Douglas Partners 2023).

Soil samples did not show contamination above national guidelines in any sample, but the sampling was limited in extent. In particular, the metals concentrations in soil samples were quite low and there were no detectable levels of indicators of petroleum hydrocarbon contamination (i.e. benzene, toluene, TRH etc) (Douglas Partners 2023).

Groundwater samples showed elevated levels of conductivity, turbidity and some metals (chromium, copper, lead, mercury and zinc). The metals were present at concentrations within 10 fold of the national guidelines for the protection of ecosystems and well below national drinking water guidelines (i.e. protective of human health). pH was also just outside the normal range. TRH was also detected in 2 of the groundwater wells – indicative of potential petroleum hydrocarbons. It is

noted that decaying vegetation and other naturally occurring sources can result in chemicals that are measured as part of TRH (Douglas Partners 2023).

Similar issues were present in the surface water samples from the dams with the addition of elevated nutrient levels (Douglas Partners 2023).

Soil conditions at the site typically comprise a 0.2 m thick layer of topsoil underlain by stiff to very stiff clays which extend to 9.7 m deep in some parts of the site. Bedrock is located beneath the clays and are a mix of siltstone, mudstone and sandstone. Depth to groundwater was in the order of 11 to 15 m below ground level (Douglas Partners 2023).

The construction of the proposed facility is not expected to impact on existing soil quality. Existing soil quality is in compliance with national guidelines so moving soil around the site during construction should not result in any impacts or change in impacts. It is noted that evaluation of the area where the dip site was located may still be required prior to construction commencing to ensure that material is handled appropriately (Douglas Partners 2023).

There are no matters related to soil contamination that could change the risks to community health for the off-site community.

7.3 Hazards/Chemicals management

During construction, it is likely that transport and storage of dangerous goods at the site will be limited to materials such as diesel (fuel for the equipment) or construction related materials. Spills of such materials could occur during construction.

A Construction Environmental Management Plan (CEMP) will be prepared in accordance with the requirements of the Environment Protection Act. This plan will include procedures to ensure all vehicles transporting hazardous materials to the site during construction are in compliance with all legislative requirements and procedures to ensure all hazardous chemicals stored at the site during construction are stored appropriately within bunds (as required) and maintaining separation distances (where required) (Arup 2026a).

In relation to operation of the facility once construction is complete, a detailed review has been undertaken of potential hazards resulting from dangerous goods transport and storage as well as the specific activities undertaken at the facility.

The review identified a number of materials that may be used at the site that have specific storage and transport requirements as outlined in government guidance. These include:

- Hydraulic oil
- Lubrication oil
- Activated carbon
- Ammonia (this may not be used at the site – the initial design is proposing use of urea instead of ammonia in the treatment system to control nitrogen oxides but the alternative is ammonia solution and would use a 25% aqueous ammonia solution)
- Propane/acetylene (if necessary for welding)
- Sodium hydroxide
- Hydrochloric acid

- Air pollution control residue – this is the material collected in the baghouse part of the air pollution control processes (treatment will occur at the proposed facility so only treated material will be transported from the site)
- Diesel (Arup 2026a).

Issues that may arise during transport and storage of these items are well understood and requirements of the Dangerous Goods Code provide guidance outlining appropriate management. This Code usually provides sufficient guidance to ensure all relevant risks to health from the transport and storage of such materials are appropriately managed (Arup 2026a).

Of these listed materials, it is likely that only diesel will be present in quantities triggering notification of DETSI under the EP Act.

The amounts of diesel, ammonia (if used), sodium hydroxide and hydrochloric acid that may be stored at the site may be sufficient to require appropriate signage at the entry of the site or nearby (Arup 2026a).

Queensland legislation requires that an emergency plan be prepared for the site and submitted to Queensland Fire and Emergency Services for review. The plan is required to address evacuation procedures, medical treatments (where needed) and communication protocols to ensure all workers at the site understand what is occurring in such circumstances and know what to do should such conditions arise (Arup 2026a).

Other hazards that might arise during operation of the proposed facility include:

- Issues around managing the waste used as fuel for the facility (including potential for a fire in the waste bunker and potential for inappropriate hazardous wastes (e.g. LPG containers) to be received at the site)
- Issues related to spills or leaks of dangerous goods stored at the site (including the potential for a dust explosion for activated carbon or a spill/release of diesel or ammonia (if ammonia is to be used at the site)) (Arup 2026a).

An operational environmental management plan will be developed. The plan will outline procedures to ensure correct storage, handling and use of dangerous goods and diesel in accordance with relevant standards and best practice guidelines. This plan will include a specific hazardous materials management plan and all necessary safety data sheets for chemicals held at the site (Arup 2026a).

An emergency response plan will also need to be developed for this facility. This plan will outline procedures and equipment in place at the facility to reduce the risks posed by any emergency situations. The plan will be reviewed by Queensland Fire and Emergency Services (QFES). This plan will also include procedures for managing firewater should such a situation arise. The plan will be designed in line with government requirements (Arup 2026a).

There are no matters related to dangerous good storage and transport that could change the risks to community health for the off-site community.

7.4 Water management

Under normal operations, the facility does not generate a process wastewater stream requiring discharge. Wastewater is produced from the site toilets and kitchens and these will be addressed via an on-site septic system.

The focus of the assessment is, therefore, what changes are necessary (compared to the current situation) to manage stormwater at the site once the facility has been constructed (and during construction) as well as whether it is necessary to include any specifics into the design to ensure the facility can appropriately manage flooding (Arup 2026b).

The subject site is currently steeply sloping pastoral land comprising grasses with low density shrubs and trees. It is bounded to the north by Boonah Beaudesert Road and a state owned railway corridor to the east. There are no formal watercourses within the site boundary, however, a natural overland flow path exists through the centre of the site. Three cascading farm dams are positioned along this overland flow path which conveys water to an existing 1.8 m high brick culvert below the railway corridor. This overland flow path is to be retained and is not impacted by the development of the facility apart from the addition of discharge from 1 of the proposed stormwater detention basins into this flow path (Arup 2026b).

The site is open farmland at this time and no formal piped stormwater management infrastructure is present. The dams present along the overland flow path do allow some sediment to be removed from stormwater as flows slow when they reach these dams in large events (Arup 2026b).

Modelling using the MUSIC suite of models has been undertaken to understand the risk of flooding at the site and to inform the design of stormwater control and treatment devices that could be required.

Management of stormwater has been based on relevant guidance from state and local government. Appropriate management of stormwater is a requirement for all industrial/commercial sites and involves ensuring adequate infrastructure to manage flows is included in any site design and that such infrastructure is appropriately connected into the region wide system (Arup 2026b).

Runoff from the developed portion of the site will be managed via minor and major stormwater infrastructure which will convey water to bioretention (i.e. lined basins with plants to take up nutrients) and on-site detention basins (i.e. dams). The size of these basins will be based on the requirements of government guidance (i.e. sized to manage a 10 year ARI event).

It is important to note that most areas that could impact on the quality of stormwater will be contained within the buildings at the proposed facility (e.g. bunker where waste will be stored) so stormwater will not come into contact with such materials. The rest of the site will remain undeveloped, so stormwater management and quality from these areas will not change from the current situation (Arup 2026b).

The basis of the site stormwater management design is:

- Retain existing farm dams serving the southern part of the central catchment allowing undeveloped areas of this catchment to maintain existing drainage regime.
- Maintain existing drainage regime within the southern catchment of the site.
- Generally, maintain pre-development catchment distribution.
- Retain current stormwater discharge locations and flow rates via use of attenuation basins.
- Provide catchment wide stormwater quality improvement measures at centralised locations.
- Collect rainwater and re-use to offset high process demands.
- Provide specific measures locally to serve areas of higher risk (Arup 2026b).

Rainwater falling on the roofs of the key buildings at the site will be collected in tanks for reuse. Water that falls on the roads and hardstand areas will be collected via inlet pits and piped to the various basins (Arup 2026b).

Runoff from the IBA treatment area will be segregated with a dedicated stormwater system and retention basin and treatment as required. Water collected in this area will be reused in the IBA maturation process. Physical separation in the form of grading and upstands will be used to separate the IBA maturing hardstand from adjacent roadways (Arup 2026b).

Modelling of the system as designed using TUFLOW demonstrates that the risks related to flooding will not change noticeably from the current situation. This is due to the use of basins to detain peak flows (Arup 2026b).

Potable water from the reticulated water supply is to be used for drinking, sanitary purposes (toilets, showers), fire water (sprinklers and hydrants). This is in line with normal uses of potable water and such water will be discharged to the sewage system (Arup 2026b).

There are no matters related to stormwater management and flooding potential that could change the risks to community health for the off-site community.

7.5 Bushfire

An assessment of the potential for the project area to be impacted by bushfire was undertaken as part of the overall evaluation of the Project (Arup 2025c). This work was undertaken in line with the requirements of QLD guidance. This includes the following steps:

- Mapping of bushfire prone locations within the area relevant for a project.
- Field investigations in the relevant area to ground truth the mapping including all land within 150 m of the Project site. This includes verifying vegetation communities, land slope and location of adjacent bushland, fire breaks and access roads.
- Determine the need for asset protection zones.

The site for this Project is within a bushfire prone area as per government mapping (Arup 2025c). The relevant high, very high and medium bushfire prone areas on and around this site are shown on **Figure 7.1**.

A full assessment has found the following:

- Patches of vegetation (bushfire prone types) in the rail corridor and along the roadsides are too small for full fire development and so do not need to be considered further.
- Other vegetation within 150 m of the proposed facility does not qualify as bushfire prone so further assessment is not required.
- The assessment has found the site is not subject to bushfire hazard in regard to slope and other aspects and complies with the relevant assessment benchmarks as well as State Planning Policy 2017 and the Bromelton SDA Development Scheme.
- The development has been designed to ensure efficient access should evacuation or fire fighting be required (Arup 2025c).

There are no matters related to bushfire potential that could change the risks to community health for the off-site community.

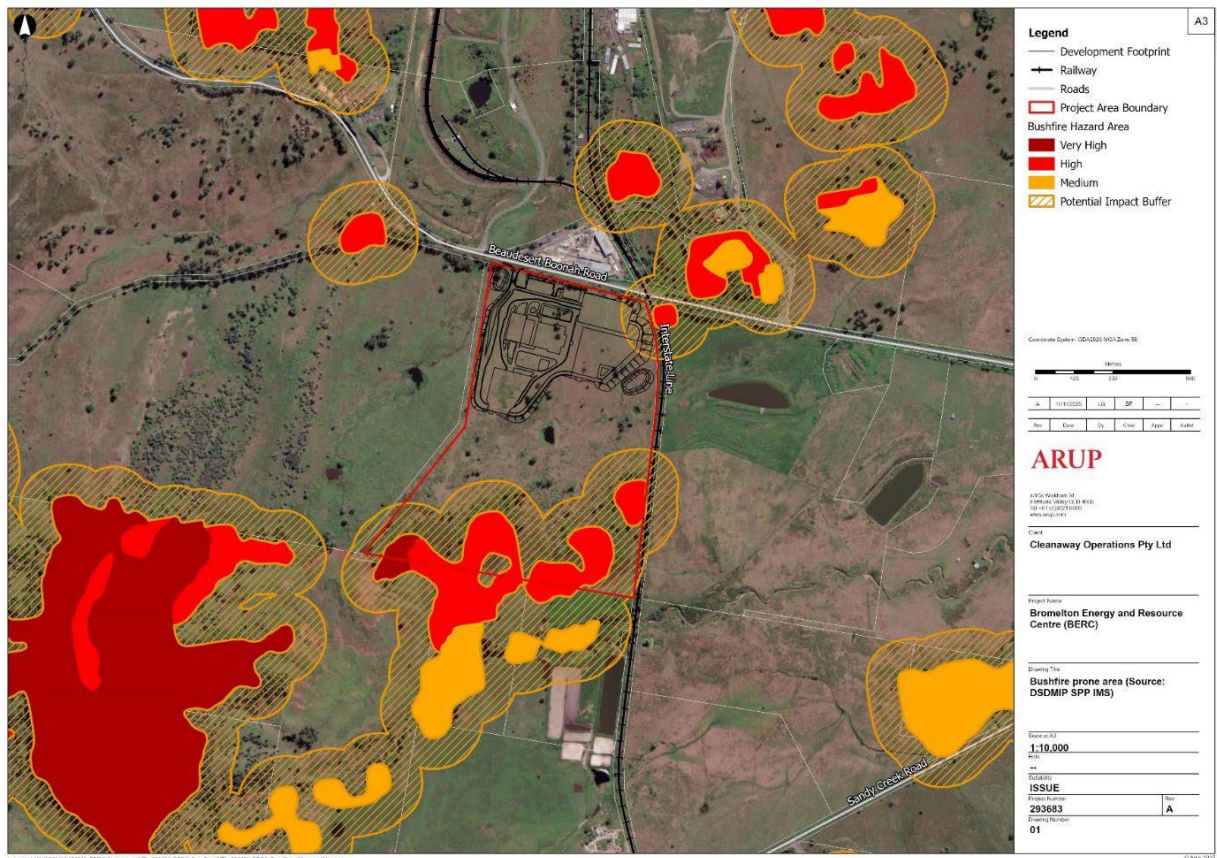


Figure 7.1: Bushfire potential assessment outcomes

7.6 Outcomes of health risk assessment

Table 7.1 presents a summary of the outcomes of the assessment undertaken in relation to the impacts on community health of changes in relation to other matters (e.g. flooding, bushfire potential, water management or handling of dangerous goods) associated with the proposed Project.

Table 7.1: Summary of health impacts – other matters

Other matters	
Benefits	Provision of sufficient infrastructure to manage waste effectively in an urban area is a benefit to the health of the community.
Impacts	Negligible impacts to community health are predicted during both construction and operations in regard to handling of hazardous substances, soil contamination, water management, flooding or bushfire potential.
Mitigation	Existing legal requirements for the soil contamination, storage and transport of hazardous substances, bushfire and stormwater management and flooding potential must be complied with at the proposed facility. Particular control measures to be included at the proposed facility include: - Design of the treatment plant to comply with design requirements to ensure no change in flooding potential. - Inclusion of appropriate access to allow the plant to be accessed or evacuated during floods.

Other matters	
	• Design of the proposed facility to meet all the requirements in requirements for dangerous goods storage and transport. • Ensuring an appropriate stormwater management is designed and installed for the site.

Section 8. Conclusions

Environmental Risk Sciences Pty Ltd (enRiskS) has been engaged by Cleanaway to undertake a Human Health Risk Assessment (HHRA) for a waste-to-energy facility in Bromelton, Queensland.

Based on the assessment undertaken in relation to this facility and the potential for changes to community health that could arise, the following has been concluded:

■ Air quality

With consideration of the air quality guidance adopted and the assessment of potential changes in air quality arising due to this proposed facility, where the facility is designed to meet the specifications identified, there will be no changes in air quality that could impact on community health in the area around the proposed facility. In particular, this assessment has shown:

- No unacceptable risks for criteria pollutants (NO_x , SO_x , CO, $\text{PM}_{2.5}$, PM_{10})
- No unacceptable risks for short term exposures from the proposed facility at the maximum off-site location – all other locations will have lower concentrations and so risks will be lower
- No unacceptable risks for relevant exposure scenarios considering long term exposures (both via inhalation and after deposition onto soil and uptake into home grown produce) at:
 - Maximum off-site location
 - Maximum residential location (and maximum commercial/industrial and maximum other places if land use changes to residential)
 - Maximum commercial/industrial location
 - Maximum on-site location
- No unacceptable risks for relevant exposure scenarios for rainwater tanks.

The following conservative, best practice HHRA assumptions have been incorporated into the assessment:

- risks arising from exposure to chemicals emitted to air attached to particles have been assessed assuming the particles to which the chemicals are attached remain in the air and can be inhaled by people (i.e. 100% available for inhalation pathway) AND that the particles are assumed to settle onto the ground where they can impact on soil quality and be taken up into plants or other food types – essentially double counting which is a conservative approach.
- risks via inhalation are based on the worst-case locations and assuming people will be present at those locations 24 hours/day for 365 days per year for the lifetime of the facility.
- risks via exposure to chemicals attached to particles have been assessed assuming the particles may deposit onto the soil around the facility for 70 years without removal (i.e. more than the lifetime of the facility).

- Potential for PFAS (per/poly fluoroalkyl substances) to be present in emissions from this facility has been considered in this assessment. Using worst-case measured data from a Swedish facility, these chemicals make an insignificant contribution to the overall risk estimates for this Project. Given the likely decreasing levels of these

chemicals in residual waste to be used as fuel for this facility due to phasing out and bans on these chemicals as well as the control measures built into this facility (i.e. temperatures in the combustion chamber, use of activated carbon and other chemicals in the pollution control equipment and the baghouse), it is expected that levels of PFAS in the emissions will be very low to negligible and definitely at levels that are not measurable.

■ Noise

With consideration of the noise limits adopted and the assessment of potential changes in noise levels for the Project, where the Project is designed to meet the noise specifications identified (i.e. predicted levels), and where the identified noise mitigation measures are implemented, there will be no changes in noise sufficient to cause changes in community health for the off-site community.

■ Other matters (water, soil contamination, dangerous goods/chemical hazards)

Based on the evaluations provided and ensuring that government regulations about storage and transport of hazardous chemicals are followed, there are no changes in stormwater management, flooding potential, handling of dangerous goods or bushfire hazard that would be sufficient to cause changes in community health for the off-site community.

■ Drinking water catchment

Given the very low levels of emissions from this facility, it is not possible for these emissions to make a noticeable change in the concentrations of the relevant chemicals in raw water within the drinking water catchment (Logan Albert Catchment) due to deposition of particles from this facility. This has been confirmed by comparison of worst case air concentrations to existing air quality likely in urban areas and by comparison of estimated water concentrations in rainwater tanks to Australian drinking water guidelines. In both cases, the estimated levels from this facility are thousands of times lower, if not, millions of times lower than relevant air or drinking water guidelines. The emissions will not change the existing air quality in a way that is measurable or noticeable.

Section 9. References

Site Specific References

Arup (2025a). Bromelton Energy and Recovery Centre, Technical process description. Dated 19 December 2025.

Katestone (2026), Bromelton Energy and Recovery Centre, Air quality assessment. Rev C Dated February 2026.

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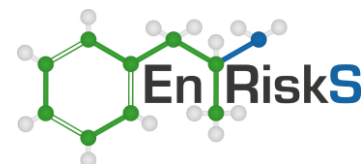
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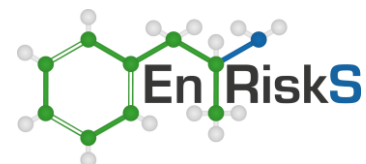
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Appendix A Calculation of risks from PM_{2.5}

Calculation of risk: PM_{2.5}

A quantitative assessment of risk for particles uses a mathematical relationship between an exposure concentration (i.e. concentration in air) and a response (namely a health effect). This relationship is termed an exposure-response relationship. Such relationships are relevant for the range of health effects (or endpoints) identified as relevant (to the nature of the emissions assessed) and robust (as determined by the World Health Organisation review).

An exposure-response relationship can have a threshold (i.e. where there is a safe level of exposure, below which there are no adverse effects) or the relationship can have no threshold (and is regarded as linear) where there is some potential for adverse effects at any level of exposure.

In relation to the health effects associated with exposure to particulate matter, no threshold has been identified. This means WHO has adopted non-threshold approaches to develop the exposure-response relationships. These non-threshold based relationships have been identified for the health endpoints considered relevant for particles.

This assessment has focused on PM_{2.5} as this fraction has been shown to be more consistently related to health effects from exposure to particles in the large epidemiological studies that WHO has drawn on to develop these exposure-response relationships. The exposure response relationships based on PM₁₀ are a little less consistent/robust than those for PM_{2.5} and it is considered that the effects relate to the amount of PM_{2.5} within the PM₁₀ being assessed. Given that this assessment, has assumed that all of the PM₁₀ that would be emitted from this facility is in the PM_{2.5} size fraction, doing this additional assessment for the most sensitive and most robust exposure response relationship for PM_{2.5} is relevant for both PM_{2.5} and PM₁₀.

Risk calculations relevant to exposures of the community to PM_{2.5} have been undertaken utilising concentration-response functions relevant to the most significant health effect associated with exposure to PM_{2.5}, namely mortality (all cause). In this case, the concentration in air that people breathe is the measure of exposure for the exposure-response relationship.

These exposure-response relationships are derived from large city wide studies with multiple cities combined together to look at potential effects due to regional levels of particles in air. The use of such relationships for evaluation of a point source (and, more particularly, for the worst case location around a point source) is not entirely appropriate or applicable. However, it has been adopted in assessments like this to provide some additional understanding of the potential risks due to the facility.

The assessment of potential risks associated with exposure to particles involves the calculation of a relative risk (RR). For the purpose of this assessment the shape of the exposure-response function used to calculate the relative risk is assumed to be linear¹¹. The calculation of a relative risk based

¹¹ Some reviews have identified that a log-linear exposure-response function may be more relevant for some of the health endpoints considered in this assessment. Review of outcomes where a log-linear exposure-response function has been adopted (Ostro 2004) for PM_{2.5} identified that the log-linear relationship calculated slightly higher relative risks compared with the linear relationship within the range 10–30 micrograms per cubic metre, (relevant for evaluating potential impacts associated with air quality goals or guidelines) but lower relative risks below and above this range. For this assessment (where impacts from a particular project are being evaluated) the impacts assessed relate to concentrations of PM_{2.5} that are well below 10 micrograms per cubic metre and hence use of the linear relationship is expected to provide a more conservative estimate of relative risk.

on the change in exposure concentration from baseline/existing (i.e. based on incremental impacts from the project) can be calculated on the basis of the following equation (Ostro 2004):

Equation 1 $RR = \exp[\beta(X-X_0)]$

Where:

R = relative risk

$X-X_0$ = the change in particulate matter concentration to which the population is exposed ($\mu\text{g}/\text{m}^3$)

β = regression/slope coefficient, or the slope of the exposure-response function which can also be expressed as the per cent change in response per 1 $\mu\text{g}/\text{m}^3$ increase in particulate matter exposure.

Based on this equation, where the published studies have derived relative risk values that are associated with a 10 micrograms per cubic metre increase in exposure, the β coefficient can be calculated using the following equation:

Equation 2
$$\beta = \frac{\ln(RR)}{10}$$

Where:

RR = relative risk for the relevant health endpoint as published ($\mu\text{g}/\text{m}^3$)

10 = increase in particulate matter concentration associated with the RR (where the RR is associated with a 10 $\mu\text{g}/\text{m}^3$ increase in concentration).

The assessment of health impacts for a particular population associated with exposure to particles has been undertaken utilising the methodology presented by the WHO (Ostro 2004)¹² where the exposure-response relationships identified have been directly considered on the basis of the approach outlined below.

¹² For regional guidance, such as that provided for Europe by the WHO WHO 2006a, *Health risks or particulate matter from long-range transboundary air pollution*, World Health Organisation Regional Office for Europe, regional background incidence data for relevant health endpoints are combined with exposure-response functions to present an impact function, which is expressed as the number/change in incidence/new cases per 100,000 population exposed per microgram per cubic metre change in particulate matter exposure. These impact functions are simpler to use than the approach adopted in this assessment, however, in utilising this approach it is assumed that the baseline incidence of the health effects is consistent throughout the whole population (as used in the studies) and is specifically applicable to the sub-population group being evaluated. For the assessment of exposures in the areas evaluated surrounding the project it is more relevant to utilise local data in relation to baseline incidence rather than assume that the population is similar to that in Europe (where these relationships are derived).

An additional risk can be calculated as:

Equation 3 **Risk = $\beta \times \Delta X \times B$**

Where:

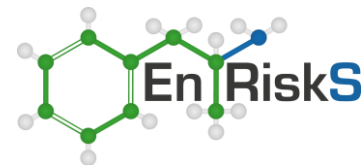
β = slope coefficient relevant to the per cent change in response to a 1 $\mu\text{g}/\text{m}^3$ change in exposure

ΔX = change (increment) in exposure concentration in $\mu\text{g}/\text{m}^3$ relevant to the project at the point of exposure

B = baseline incidence of a given health effect per person (e.g. annual mortality rate)

The calculation of the incremental individual risk for relevant health endpoints associated with exposure to particulate matter as outlined by WHO (Ostro 2004) has considered the following four elements:

- Estimates of the changes in particulate matter exposure levels (i.e. incremental impacts) due to the project for the relevant modelled scenarios – these have been modelled for the proposed project, with the maximum change overall addressed. For this assessment, the change in $\text{PM}_{2.5}$ relates to the change in annual average air concentrations at the maximum residential location which is close to the site. The value considered in this assessment is 0.009 $\mu\text{g}/\text{m}^3$.
- Public health discussions in the literature have noted that a change in particles concentration of 0.02 $\mu\text{g}/\text{m}^3$ or less is generally considered negligible. This assessment has still been undertaken (Capon & Wright 2019).
- The calculation has considered the baseline mortality rate for Scenic Rim LGA. The rate in Scenic Rim LGA is 266.2 per 100,000 for 2016/20 (all ages and all causes) (refer to **Table 3.2**).
- The baseline incidence is, therefore, 0.002662 for Scenic Rim LGA.
- Exposure-response relationships expressed as a percentage change in health endpoint per microgram per cubic metre change in particles exposure, where a relative risk (RR) is determined (refer to Equation 1).
- The concentration response function used in this report is that recommended in a NEPC published report (Jalaudin & Cowie 2012). It was derived from a study in the United States which examined the health outcomes of hundreds of thousands of people living in cities all over the United States. These people were exposed to many different concentrations of $\text{PM}_{2.5}$ (Pope et al. 2002).
- The study found a relative risk (RR) of all-cause mortality of 1.06 per 10 $\mu\text{g}/\text{m}^3$ change in $\text{PM}_{2.5}$, and that this risk relationship was in the form of an exponential function. Based on a RR of 1.06 per 10 $\mu\text{g}/\text{m}^3$ change in $\text{PM}_{2.5}$, this results in a $\beta = 0.0058$. It is noted that the exposure response relationship established in this study was re-affirmed in a follow-up study (that included approximately 500,000 participants in the US) (Krewski et al. 2009) and is consistent with findings from California (Ostro et al. 2006). The relationship is also more conservative (i.e. estimates a higher risk at the same concentration) than a study undertaken in Australia and New Zealand (EPHC 2010).



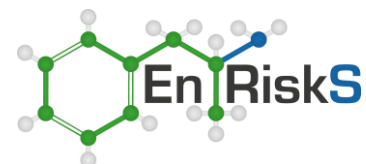
The above approach (while presented slightly differently) is consistent with that presented in Australia (Burgers & Walsh 2002), US (OEHHA 2002; USEPA 2005b, 2010) and Europe (Martuzzi et al. 2002; Sjoberg et al. 2009).

Based on the air quality modelling data and the population health statistics in the area, the calculated incremental individual risk is:

$$\begin{aligned}\text{Risk} &= \beta \times \Delta X \times B \\ &= 0.0058 \times 0.009 \times 0.002662 \\ &= 1 \times 10^{-7}\end{aligned}$$

The use of a risk criteria of 1 in 1,000,000 or 1 in 100,000 to indicate negligible or acceptable risk was included in a range of large infrastructure projects which have been approved in Australia including North East Link, NorthConnex, WestConnex and others. A detailed discussion of these criteria is also provided in (Capon & Wright 2019).

In this case, the calculations for this proposed facility result in risks that are below 1 in 1,000,000 risk (i.e. 1×10^{-6}) and so are considered negligible.



Appendix B Toxicological information

B1 Approach

The quantitative assessment of potential risks to human health for any substance requires the consideration of the health end-points and where carcinogenicity is identified; the mechanism of action needs to be understood. This will determine whether the chemical substance is considered a threshold or non-threshold chemical substance. A threshold chemical has a concentration below which health effects are not considered to occur. A non-threshold chemical substance is believed to theoretically cause health effects at any concentration, and it is the level of health risk posed by the concentration of the chemical substance that is assessed. The following paragraphs provide further context around these concepts.

For chemical substances that are not carcinogenic, a threshold exists below which there are no adverse effects (for all relevant end-points). The threshold typically adopted in risk calculations (a tolerable daily intake [TDI] or tolerable concentration [TC]) is based on the lowest no observed adverse effect level (NOAEL), typically from animal or human (e.g., occupational) studies, and the application of a number of safety or uncertainty factors. Intakes/exposures lower than the TDI/TC is considered safe, or not associated with an adverse health risk (NHMRC 1999).

Where the chemical substance has the potential for carcinogenic effects the mechanism of action needs to be understood as this defines the way that the dose-response is assessed. Carcinogenic effects are associated with multi-step and multi-mechanism processes that may include genetic damage, altering gene expression and stimulating proliferation of transformed cells. Some carcinogens have the potential to result in genetic (DNA) damage (gene mutation, gene amplification, chromosomal rearrangement) and are termed genotoxic carcinogens. For these carcinogens it is assumed that any exposure may result in one mutation or one DNA damage event that is considered sufficient to initiate the process for the development of cancer sometime during a lifetime (NHMRC 1999). Hence no safe-dose or threshold is assumed, and assessment of exposure is based on a linear non-threshold approach using slope factors or unit risk values.

For other (non-genotoxic) carcinogens, while some form of genetic damage (or altered cell growth) is still necessary for cancer to develop, it is not the primary mode of action for these chemical substances. For these chemical substances carcinogenic effects are associated with indirect mechanisms (that do not directly interact with genetic material) where a threshold is believed to exist.

In the case of particulate matter (PM_{10} or $PM_{2.5}$), current health evidence has not been able to find a concentration below which health impacts do not exist. Thus, the quantification of risk for $PM_{2.5}$ follows a non-threshold approach as described in **Appendix A**.

B2 Values adopted for the assessment of acute exposures

The assessment of potential acute exposures relates to inhalation exposures only. The assessment is based on the maximum predicted 1-hour average air concentration. Hence the selection of relevant and appropriate acute toxicity reference values (TRVs) has focused on guidelines that relate to a peak 1-hour exposure. There are other guidelines available that can be termed acute or short-term, however, these relate to exposure periods longer than 1-hour, e.g., an 8-hour average or averaging periods up to 14 days (as is adopted by ATSDR). Guidelines for averaging periods longer than 1-hour are not preferred as the assessment would not then be comparing exposure concentrations and guidelines on the same basis.

The acute TRVs are protective of all adverse health effects for all members of the community including sensitive groups, such as children and the elderly.

For this assessment, the acute TRVs have been selected on the basis of the following approach:

- Acute guidelines relevant to a 1-hour average exposure period are preferred.
- The TRVs have been selected on the basis of the following hierarchy:
 1. Texas Commission on Environmental Quality (TCEQ) Acute Reference Value (Acute ReV), which is based on a target HI of 1, consistent with the target HI adopted in the derivation of guidelines in Australia (enHealth 2012a; NEPC 1999 amended 2013c, 2004) by the WHO (WHO 2000d, 2000e, 2010b). These are used as the primary source of acute guidelines as they specifically relate to and consider studies relevant to a 1-hour exposure and they have undergone the most recent detailed review process.
 2. California Office of Environmental Health Hazard Assessment (OEHHA) acute Reference Exposure Level (REL), which are all based on a target HI of 1 with RELs relevant to 1-hour average exposures adopted.
 3. Other robust international sources including ATSDR.

Some pollutants are not considered to be acute toxicants, which means that they have a very low acute toxicity – i.e. would require exposure to very high amounts for any effects to be seen. As a result, there are no suitable and robust acute inhalation guidelines available. For these pollutants, the assessment of chronic exposure is of most importance, which is evaluated on the basis of appropriate chronic toxicity values (discussed below). The pollutants where acute inhalation exposures have not been quantified are lead, dioxin-like compounds, thallium and PAHs.

Based on the above, the acute TRVs listed in **Table B1** have been adopted in this assessment.

An Environmental Protection Policy was published in Queensland in relation to air pollutants in 2019 (Queensland Government 2019b). This includes air quality objectives for a range of pollutants that may be present in air. For many pollutants, objectives in this policy are based on chronic/long-term exposure (annual averages) and these are considered below (see **Table B2**).

Formaldehyde is the only pollutant relevant for this assessment that has a short term objective listed in the policy (other than the criteria pollutants – NO_x, SO_x, CO and PM). A 30 minute average objective of 0.109 mg/m³ is listed for formaldehyde. This value is higher than the value recommended by TCEQ for a 1 hour average, so the more stringent value from TCEQ (i.e. 0.05 mg/m³) has been adopted for this assessment (compliance with this more stringent value will also mean compliance with the air quality objective from the EPP (air)).

Table B1: Acute TRVs adopted in this assessment

Chemicals evaluated	Acute air guideline (1-hour average) (mg/m ³)	Key health effects
Gases		
Hydrogen chloride (HCl)	0.66	HCl gas is a strong irritant, causing irritation of the eye, nose, and throat. Inhalation of HCl gas at sufficiently high concentrations can also produce acute tracheobronchitis (characterized by cough, sore throat, chest pain, and light-headedness); bronchoconstriction; and pulmonary oedema. The listed air guideline is protective of these acute effects, with respiratory effects in individuals with asthma being the most sensitive effect (TCEQ 2015a).
Hydrogen fluoride (HF)	0.06	The upper respiratory tract is the most sensitive target of acute toxicity of F and HF exposure. HF gas is corrosive to the eyes and mucous membranes of the respiratory tract. Acute inhalation exposure to F or HF in humans has resulted in eye, nose and respiratory irritation, and inflammation of the airways. Exposure to high concentrations of HF can cause severe irritation, pulmonary oedema, pulmonary haemorrhagic oedema, tracheobronchitis, or death. The results of acute human and animal studies show that humans might be more sensitive than rats to the irritation effects of HF or F, approximately by an order of magnitude. The listed air guideline is based on short term exposure leading to increased airway inflammation in human studies (TCEQ 2015d).
Ammonia	0.59 (TCEQ 2014c)	The available studies (occupational and experimental) indicate that acute exposure to low to moderate concentrations of ammonia (less than 100 ppm) can cause sensory irritation (discomfort in the eyes and/or nose) in humans but are not related to functional respiratory deficits. In general, the acute health effects reported in animals following short-term inhalation of ammonia include oral, nasal and eye irritation, respiratory tract irritation, decreased respiratory rate, increased respiratory depth, reduced body weight, and lethargy. In humans, the health effects of acute exposure are similar to those reported in animals and include oral, nasal and eye irritation, respiratory tract irritation, and increased respiratory depth. Effects on tissues and organs distant from the entry point have not been observed because of the scrubbing mechanism of the nasopharyngeal region. Ammonia is highly water soluble and as such readily dissolves in the mucous membrane layer of the cornea and upper airway. This "scrubbing" protects the lower respiratory tract and has been shown to be concentration and time dependent. The acute air guideline is based on the most sensitive effects, namely mild, transient effects in respiratory system and CNS effects in human studies (TCEQ 2014c).
VOCs as 100% benzene	0.58	The group of volatile organic compounds (VOCs) has been assessed on the assumption that 100% of the VOCs present comprise benzene, one of the most toxic VOC compounds likely to be present in VOCs released from the facility. The actual proportion of benzene in VOCs is expected to be very small and hence this approach is highly conservative. The key health effects associated with exposure to benzene relate to chronic exposures. Both animal and human data indicate the most sensitive noncarcinogenic health effect of acute and chronic exposure is haematotoxicity (i.e. bone marrow depression: leukopenia, pancytopenia, granulocytopenia, lymphocytopenia, thrombocytopenia, aplastic anaemia) as well as CNS excitation and depression and neurological effects. The acute air guideline is based on decreased lymphocytes in an animal study (TCEQ 2015c). The study used by TCEQ is the same adopted by ATSDR (ATSDR 2007b) in establishing their acute air guideline (noting the ATSDR review is more dated).

Chemicals evaluated	Acute air guideline (1-hour average) (mg/m ³)	Key health effects
VOCs as 100% formaldehyde	0.05	The group of volatile organic compounds (VOCs) has been assessed on the assumption that 100% of the VOCs present comprise formaldehyde, another of the most toxic VOC compounds likely to be present in VOCs released from the facility. The actual proportion of formaldehyde in VOCs is expected to be very small and hence this approach is highly conservative. The key health effects associated with exposure to formaldehyde relate to irritation in the eyes, nose and throat. The irritation occurs from short term and long term exposure. Short term exposures of people to formaldehyde resulted in irritation which was reversible once exposure was removed. For chronic exposures, irritation is also the key effect along with associated respiratory symptoms (TCEQ 2015b). The acute air guideline is based on eye and nose irritation in people (volunteers) (TCEQ 2015b). ATSDR has also published a short term/acute guideline – 0.04 ppm (i.e. 0.05 mg/m³) and this value is based on one of the studies used by TCEQ. The TCEQ review is more recent than that from ATSDR.
Inorganics and organics bound to particulates (where acute effects are relevant)		
Antimony	0.001 (ATSDR 2019a)	The most sensitive effects related to acute inhalation exposures to antimony have been identified as respiratory effects, with effects on the cardiovascular system less sensitive (ATSDR 2019a). Acute air guideline adopted is based on respiratory effects (epithelium effects at base of epiglottis) in an animal study (ATSDR 2019a).
Arsenic	0.0099 (TCEQ 2012)	Short-term exposures to arsenic have been reported to result in severe irritation to both the upper and lower parts of the respiratory system, followed by symptoms of cough, dyspnea, and chest pain. In addition, exposure to arsenic dust has been reported to cause laryngitis, bronchitis, and/or rhinitis. Further, exposure to arsenic via inhalation and/or ingestion can also cause gastrointestinal symptoms such as garlic-like breath, vomiting, and diarrhea. The available occupational and epidemiological studies have not identified developmental or reproductive effects; however, these effects have been observed in animals but only at doses exceeding maternal toxicity. Acute air guideline adopted is based on the most sensitive effect, namely maternal effects in a reproductive study in animals.
Cadmium	0.018 (TCEQ 2016)	The toxicity of cadmium in air is dependent on the form of cadmium. The toxicity is higher with the more soluble cadmium compounds. Acute inhalation exposure to cadmium may cause destruction of lung epithelial cells, resulting in decreased lung function, pulmonary oedema, tracheobronchitis, and pneumonitis in both humans and animals if concentrations are high enough. Other effects identified in animal studies include decreased immune response, erosion of the stomach, decreased body weight gain and tremors (ATSDR 2012e). Acute air guideline is based on immunological effects in animals (most sensitive effect identified).
Chromium (Cr VI assumed)	0.0013 (TCEQ 2014a)	The assessment of chromium exposures has assumed that it comprises 100% chromium VI, which is the most toxic form of chromium. The toxicity is higher for soluble forms of Cr VI than insoluble forms. The respiratory system is the most sensitive health effect for both forms (TCEQ 2014a). Acute air guideline is based on respiratory effects (increased lung weight) in animals.
Cobalt	0.00069	The key health effect associated with inhalation exposures to cobalt in humans and animals are respiratory effects (TCEQ 2017d). Acute air guideline adopted is based on the protection of respiratory inhalation in a human study.
Copper	0.1 (OEHHA)	Copper is an essential element and hence health effects occur as a result of deficiency as well as excess levels. Acute inhalation value is based on occupational exposures to copper fume (unlikely to be representative of copper bound to particulates). In

Chemicals evaluated	Acute air guideline (1-hour average) (mg/m ³)	Key health effects
		the absence of any other acute guidelines, this value has been conservatively adopted in this assessment as other forms of copper are likely to be less toxic for short term exposures.
Manganese	0.0091 (TCEQ 2017c)	Manganese is an essential element and hence health effects occur as a result of deficiency as well as excess. The neurological effects of inhaled manganese have been well documented in humans chronically exposed to elevated levels in the workplace. The syndrome known as “manganism” is caused by exposure to very high levels of manganese dusts or fumes and is characterized by a “Parkinson-like syndrome”, including weakness, anorexia, muscle pain, apathy, slow speech, monotonous tone of voice, emotionless “masklike” facial expression and slow, clumsy movement of the limbs. In general, these effects are irreversible (WHO 2017). The most sensitive effect relevant to acute exposures, are respiratory effects. The acute air guideline is based on protection of respiratory effects in an animal study.
Mercury (as inorganic and elemental)	0.0006 (OEHHA)	This assessment has assumed that mercury in air comprises 100% elemental mercury vapour, which will result in a conservative assessment of inhalation exposures for inorganic mercury attached to particulates as this form of mercury is less toxic than the elemental form. Acute exposure to high concentrations of mercury vapour has been associated with chest pains, haemoptysis, breathlessness, cough and impaired lung function with the lung identified as the main target following acute exposure (ATSDR 1999b). The central nervous system is generally the most sensitive indicator of toxicity of metallic mercury vapour. Data on neurotoxic effects are available from many occupational studies. Acute air guideline is based on protection of central nervous system (CNS) effects in an animal study.
Nickel	0.0011	The respiratory system is the primary site of toxicity for inhaled nickel in both humans and laboratory animals. Effects seen in occupationally exposed workers include chronic bronchitis, emphysema, reduced vital capacity and asthma (UK EA 2009b). In relation to acute exposures respiratory effects are the most sensitive. The acute air guideline is based on protection of respiratory effects from an occupational study with nickel sulfate aerosols.
Vanadium	0.03 (OEHHA)	Data relevant to inhalation exposures to vanadium relate to vanadium pentoxide, with the most significant and most sensitive health effect identified as respiratory effects. The acute air guideline is based on the protection of these effects.

Notes:

TCEQ = Acute reference exposure value (Acute ReV) available from the Texas Commission on Environmental Quality as referenced, also available from: <https://www.tceq.texas.gov/toxicology/dsd/final.html>

OEHHA = Guideline available from California Office of Environmental Health Hazard Assessment (OEHHA) <https://oehha.ca.gov/air/general-info/oehha-acute-8-hour-and-chronic-reference-exposure-level-rel-summary>

ATSDR = Guideline available from the Agency for Toxic Substances and Disease Registry (ATSDR), as an acute air guideline (relevant to exposures from 1 hour to 14 days) <https://www.atsdr.cdc.gov/mrls/index.html>

B3 Values adopted for the assessment of chronic exposures

Approach

The quantitative assessment of potential risks to human health for any substance requires the consideration of the health end-points and, where carcinogenicity is identified; the mechanism of action needs to be understood. This will determine whether the chemical substance is considered a threshold or non-threshold chemical substance. The risks for these two different mechanisms are

calculated differently so it is important to recognise the relevant health endpoints/mechanisms correctly.

Chronic toxicity reference values (TRVs) associated with inhalation, ingestion and dermal exposures have been adopted from credible peer-reviewed sources as detailed in the NEPM (NEPC 1999 amended 2013b) and enHealth (enHealth 2012a). The identification of the most appropriate and robust TRVs has followed guidance from Australia (enHealth 2012a).

The concentrations arising due to the proposed facility have been compared to national or international guidelines for each chemical in air. These are generally based on studies which estimate the concentration which has no observable effects on laboratory animals for small/subtle types of effect or for sensitive life stages (e.g. very young). These no observable effects concentrations are then made more conservative by adjustment using a range of uncertainty factors to account for unavoidable limits in knowledge. These guidelines are, therefore, considered to be protective for sensitive subpopulations like the young and the elderly as well as the population in general. This type of approach is also adopted when developing toxicity reference values relevant for guidelines related to protection of human health in relation to ingestion or dermal exposure.

Threshold

A threshold chemical has a concentration below which health effects are not considered to occur. This is because insufficient amounts of the chemical reach relevant parts of the body to trigger the adverse effect (i.e. to disrupt cell processes, interact with receptors etc) for exposures below the threshold.

The threshold typically adopted in risk calculations (a tolerable daily intake [TDI] or tolerable concentration [TC]) is based on the lowest no observed adverse effect level (NOAEL), typically from animal or human (e.g. occupational) studies, and the application of a number of safety or uncertainty factors to calculate the reference dose or tolerable intake. Intakes/exposures lower than the TDI/TC are considered safe, or not associated with an adverse health risk (NHMRC 1999).

Non-threshold

Where the chemical substance has the potential for carcinogenic effects, the mechanism of action needs to be understood as this defines the way that the dose-response should be assessed. Carcinogenic effects are associated with multi-step and multi-mechanism processes that may include genetic damage, altering gene expression and stimulating proliferation of transformed cells. Some carcinogens have the potential to result in genetic (DNA) damage (gene mutation, gene amplification, chromosomal rearrangement) and are termed genotoxic carcinogens. For these carcinogens, it is assumed that any exposure may result in one mutation or one DNA damage event and that may be sufficient to initiate the process for the development of cancer sometime during a lifetime (NHMRC 1999). Such chemicals are termed genotoxic. Hence no safe-dose or threshold is assumed, and assessment of risk is based on a linear non-threshold approach using slope factors or unit risk values.

For other (non-genotoxic) carcinogens, while some form of genetic damage (or altered cell growth) is still necessary for cancer to develop, it is not the primary mode of action for these chemical substances. For these chemical substances, carcinogenic effects are associated with indirect mechanisms (that do not directly interact with genetic material) and, for these other mechanisms, a

threshold is believed to exist, so these chemicals are assessed in the same fashion as the threshold chemicals described above.

For this assessment, the following pollutants have been classified as class 1 carcinogens by the International Agency for Research on Cancer (IARC), and a review has been undertaken on the mechanism of action relevant to the way in which they cause cancer as follows:

- Arsenic – the mechanism by which cancer is caused does not appear to be mutagenic with a threshold mode of action identified for the assessment of cancer (where damage to cells and sufficient exposure to result in cancer proliferation required) (NEPC 1999 amended 2013c). Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.
- Cadmium – the available data suggests only weak evidence of genotoxicity and review by NEPC indicates that a threshold mode of action is relevant for the assessment of cancer. Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.
- Chromium VI – the available data suggests the compound may have some genotoxic potential, however, review by NEPC (NEPC 1999 amended 2013c) indicates that carcinogenicity is likely to act on the basis of a threshold mode of action. Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.
- Formaldehyde – the available data suggests that it may be weakly genotoxic, however, where cancer has been observed it occurs at the point of contact (e.g. nose) and only occurs where exposure is sufficient for cytotoxicity occurs (i.e. cell death) (IARC 2006; UNEP 2002; WHO 2002). A threshold mode of action is, therefore, considered appropriate. Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.
- Nickel – the available data indicates that the compound may be genotoxic, however, the mechanism of action is not well understood. WHO (WHO 1991b) indicates that very high concentrations of nickel are required to produce genotoxic effects (after cell damage/death) and hence a threshold mode of action is considered appropriate (NEPC 1999 amended 2013c). Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.
- Dioxin-like compounds (as 2,3,7,8-TCDD) – review of carcinogenicity by NHMRC (NHMRC 2002) and WHO (FAO/WHO 2018; WHO 2019) indicates that TCDD is not genotoxic and hence a threshold approach is considered appropriate. Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.

In the case of particulate matter (PM_{10} or $PM_{2.5}$), current health evidence has not been able to find a concentration below which health impacts do not exist. The health effects relevant for particles are cardiovascular and respiratory effects rather than cancer. However, due to the lack of a threshold, quantification of risk for $PM_{2.5}$ follows a non-threshold approach as described in **Appendix A**.

All chronic TRVs adopted for the assessment of chronic exposures are protective of adverse health effects for all members of the community including sensitive groups such as children and the elderly.

For the gaseous pollutants considered in this assessment, only inhalation TRVs have been adopted. For inorganics (i.e. metals) as well as dioxin-like compounds and PAHs, TRVs relevant to all exposure pathways have been adopted. Background intakes of these pollutants have been estimated on the basis of existing available information as noted.

Table B2 provides an overview of the hazards identified in relation to potential chronic exposures to the pollutants considered in this assessment. This table simply provides a summary of the hazards or health effects identified in relation to these chemicals. As with all chemicals, it is the exposure that determines whether there is sufficient exposure for the health effects that have been identified to actually occur.

Tables B3 and **B4** present the TRVs adopted for the assessment of chronic health effects associated with exposure to the other pollutants (i.e. all pollutants other than the criteria ones – i.e. other than NO_x, SO_x, CO, PM) considered in this assessment. **Table B3** presents the TRVs for chemicals that act via a mode of action that has a threshold, while **Table B4** presents the TRVs for chemicals that act via a mode of action that does not have a threshold (i.e. genotoxic carcinogens).

Table B2: Summary of hazards – chronic exposures

Pollutant evaluated	Summary of chronic health effects
Gases	
Hydrogen chloride (HCl)	The key hazards associated with HCl, relate to acute effects, where the respiratory system is the most sensitive health effect (refer to Table B1). Few human studies are available on the chronic effects of HCl exposure. Occupational studies have reported bleeding of the nose and gums and ulceration of the mucous membranes after repeated exposure to HCl mist at high (but unquantified) concentrations, work impairment and dental erosion following exposure to acid mists. IARC has not assessed HCl in relation to carcinogenicity. The available data do not support that HCl is carcinogenic. Chronic inhalation air guidelines are based on the most sensitive health effect, being hyperplasia of the nasal mucosa, larynx and trachea in animals (rat study) (TCEQ 2015a). Ambient or background levels of HCl in air are expected to be negligible.
Hydrogen fluoride (HF)	The key hazards associated with HF, relate to acute effects, where the respiratory system is the most sensitive health effect (refer to Table B1). In relation to chronic inhalation exposures, the key adverse health effects are skeletal fluorosis and respiratory effects. HF is not considered to be carcinogenic, with IARC and the USEPA not having evaluated carcinogenicity due to inadequate data. Some genotoxicity has been identified, however, only at doses that are highly toxic to cells (TCEQ 2015d). Chronic air guideline adopted is based on the most sensitive effect, namely skeletal fluorosis, based on an occupational study (TCEQ 2015d). Ambient or background levels of HF in air are expected to be negligible (DEFRA 2008).
Ammonia	The key hazards associated with ammonia, relate to acute effects, where the respiratory and CNS systems are the most sensitive health effects (refer to Table B1). In relation to chronic exposures, there are few studies addressing long-term inhalation exposures to low concentrations. The key health effects identified in occupational studies relate to respiratory irritation, including cough, chest tightness, stuffy/runny nose, sneezing, phlegm, wheezing, dyspnea, chronic bronchitis, and asthma. Studies have shown acclimation of effects (ATSDR 2004a; TCEQ 2014c). Ammonia has not been classified as a human carcinogen and is not considered carcinogenic in animals. The chronic air guideline adopted is based on the most sensitive effect identified, namely respiratory effects (lung function) in an occupational study (TCEQ 2014c). The guideline adopted from TCEQ reflects the most current evaluation of effects and studies and is similar to the reference concentration available from the USEPA (USEPA IRIS). Ambient or background levels of ammonia (away from specific sources) in air are expected to be negligible, however, it is noted that ammonia is produced endogenously (i.e. produced by the body). The studies used to develop the chronic air guideline are occupational studies and relate to an air concentration to which a range of individuals are exposed (where endogenous ammonia is already accounted for).
VOCs as benzene	As noted in Table B1, the assessment of VOCs assuming it comprises 100% benzene is conservative. Chronic exposure to benzene results primarily in haematotoxicity, including aplastic anaemia, pancytopenia, or any combination of anaemia, leukopenia, and thrombocytopenia. Chronic

Pollutant evaluated	Summary of chronic health effects
	benzene exposure is associated with an increased risk of leukaemia. In chronic exposures, benzene metabolites are considered the toxic agents, not the parent compound. The relative contribution of different benzene metabolic pathways may be dose related, with more toxic agents produced by high affinity low capacity pathways (WHO 1993). Benzene is classified as a “known” human carcinogen (Category A) by the USEPA for all routes of exposure based upon convincing human evidence as well as supporting evidence from animal studies. IARC has classified benzene in Group 1 (known human carcinogen) (IARC 2012b; USEPA 2005b, 2005a). Benzene is carcinogenic via oral and inhalation routes of exposure (ATSDR 2007b; IARC 2012b; UK EA 2009c; WHO 1993) indicates that the overall results of available studies show that it is appropriate to consider benzene (and/or its metabolites) as genotoxic (though the genotoxic profile is considered unusual (Baars et al. 2001)). The assessment of benzene toxicity needs to consider carcinogenic effects where a non-threshold dose-response approach is appropriate. Threshold or noncarcinogenic effects are also considered to ensure the additivity of these effects with other chemicals in air is adequately addressed, and to ensure that using benzene as a surrogate for VOCs addresses all potential health effects. The non-threshold toxicity reference value adopted (refer to Table B4) is from the WHO , noting the evaluations provided by the WHO, USEPA and TCEQ are similar. The threshold value for assessing non-threshold effects adopted for this assessment is from the USEPA (USEPA 2002), which provides a similar value as adopted in the more recent evaluation from TCEQ (TCEQ 2015c). Ambient or background exposures are only relevant for the threshold assessment. The available data on ambient levels in air (NSW EPA 2013; NSW Health 2004) suggest background intakes may comprise around 10% of the air guideline.
VOCs as formaldehyde	As noted in Table B1, the assessment of VOCs assuming it comprises 100% formaldehyde is also conservative. The actual proportion of formaldehyde in VOCs emitted from this facility is expected to be very small and hence this approach is highly conservative. Chronic exposures to formaldehyde result in irritation and associated respiratory symptoms (TCEQ 2015b). The critical effects used in developing the guideline were elevated rates of eye, nose and airway discomfort in people (i.e. studies were undertaken in people rather than laboratory animals). Formaldehyde is a naturally occurring chemical within our bodies – part of everyday metabolism and causes us no harm in that situation. Formaldehyde is also present in many household items and situations (e.g. produced in gas cookers, present in particle board/plywood, cosmetics, medicines, dishwashing liquids etc.) (ATSDR 1999a). People are always exposed to formaldehyde, this assessment has looked at the potential for additional exposure. The International Agency for Research on Cancer (IARC 2012b) categorised formaldehyde in group 1 (carcinogenic to humans). The USEPA has classified the carcinogenicity of formaldehyde as B1 – probable human carcinogen based on limited evidence (USEPA IRIS). Information from WHO and IARC indicate that formaldehyde is only weakly genotoxic (if at all) and so a threshold approach to assessing risk is appropriate – particularly as where cancer has been seen it occurs at the site of initial contact with the chemical (e.g. nose) and only occurs when exposure is sufficient to cause the death of cells (i.e. cytotoxicity). Longer term/chronic guidelines are based on studies in people. Background intakes have been considered (where relevant).
Inorganics and organics bound to particulates	
Antimony	Antimony is one of the oldest known remedies used in medicine. Data on side effects and toxicity of antimony and compounds have identified that the most sensitive effects relate to the respiratory tract, heart, gastrointestinal tract, serum glucose, and developmental effects. The International Agency for Research on Cancer (IARC 2015) categorized antimony trioxide in group 2B (possibly carcinogenic to humans) and antimony trisulfide in group 3 (not classifiable as to its carcinogenicity to humans). The USEPA have not classified the carcinogenicity of antimony. In relation to chronic exposures, the most sensitive health effects identified relate to the respiratory system (inhalation exposures); and the gastrointestinal tract, liver, and serum glucose levels (oral exposures). The chronic air guideline adopted in this assessment is based on respiratory effects (lung inflammation) in animals from ATSDR (ATSDR 2019a), noting no other chronic inhalation guidelines are available.

Pollutant evaluated	Summary of chronic health effects
Arsenic	Oral (and dermal) exposures have been assessed on the basis of the tolerable daily intake adopted by NHMRC and WHO in deriving drinking water guidelines (NHMRC 2011 updated 2022; WHO 2017). Background intakes have been considered (where relevant). Arsenic is a known human carcinogen, based on human epidemiological studies that show skin and internal cancers (in particular bladder, liver and lung) associated with chronic exposures to arsenic in drinking water. The International Agency for Research on Cancer (IARC) has classified arsenic and inorganic arsenic compounds as Group 1 'carcinogenic to humans' (IARC 2012a). The mechanism of action in relation to carcinogenicity is not clear and remains debated (IARC 2012a; Sams et al. 2007), with the weight of evidence indicating that a threshold approach is appropriate, noting effects on DNA occur through indirect mechanisms and at high levels of exposure. A threshold toxicity reference value, consistent with that adopted in the ASC NEPM, which is protective of carcinogenic and non-carcinogenic health effects has been adopted for the assessment of oral and dermal exposures to arsenic. Less data are available with respect to inhalation exposures to arsenic, though trivalent arsenic has been shown to be carcinogenic via inhalation exposures (with lung cancer as the end point). Data relevant to carcinogenic effects relate to exposures within and near copper smelters, however, there are insufficient data to indicate that these effects are not of importance in other types of exposures. Further the mechanism of action is also not clear with key agencies suggesting a non-threshold approach is appropriate. The chronic air guideline adopted for the assessment of arsenic is from the TCEQ (TCEQ 2012) evaluation, which is based on a non-threshold approach and is protective of both carcinogenic and non-carcinogenic effects. Background intakes have been considered (where relevant).
Cadmium	Numerous studies examining the toxicity of cadmium in workers have identified the respiratory tract, the kidney and bone as sensitive targets of toxicity. Other effects identified include developmental and reproductive effects, hepatic effects, haematological effects and immunological effects (ATSDR 2012e). IARC has classified cadmium and cadmium compounds as a Group 1 agent (i.e., carcinogenic to humans) based on additional evidence of carcinogenicity in humans and animals. The USEPA has classified cadmium as a probable human carcinogen via inhalation. There are conflicting data on the genotoxicity of cadmium. Based on the available information, assessment of oral and dermal exposures has adopted the threshold toxicity value from the WHO (WHO 2010a) which is consistent with the approach and value adopted by the NHMRC to determine a drinking water guideline for Australia (NHMRC 2011 updated 2022). Sufficient data are available to conclude cadmium is carcinogenic via inhalation exposures. The inhalation air guideline adopted (WHO 2000e) is based on the most sensitive effect, namely kidney toxicity, which is also protective of carcinogenic effects. Background or ambient intakes have also been considered (where relevant).
Chromium (Cr VI assumed)	The assessment of chromium exposures has assumed that it comprises 100% chromium VI, which is the most toxic form of chromium for people. In the environment, Cr VI changes into the less toxic form (Cr III) in the presence of oxidizable organic matter and hence assuming that Cr VI remains following long-term deposition to land is highly conservative. It is more likely to be present as Cr III. Cr VI is unstable in the body and is reduced to Cr V, Cr IV and ultimately to Cr III by many substances, including ascorbate and glutathione (naturally occurring substances that perform a range of tasks to ensure appropriate operation of the body). It is believed that the toxicity of Cr VI compounds results from damage to cellular components during this process (WHO 2013b). Chronic exposure to Cr VI via inhalation has been found (in occupational studies) to result in respiratory tract and eye irritation, and cancer (respiratory tract and lung cancer) (WHO 2013b). Oral exposures to Cr VI can cause gastrointestinal effects (most sensitive) and haematological effects. Oral exposures have not demonstrated an association with cancer in humans, however, animal studies have shown carcinogenic potential. Dermal exposure to Cr VI can result in ulcers and allergic contact dermatitis (WHO 2013b). IARC (IARC 2012a) has classified Cr VI compounds as Group 1 carcinogens: carcinogenic to humans. Chromium is classified by the USEPA as a Group A: known human carcinogen by the inhalation route, with carcinogenicity by the oral route of exposure noted to be Group D: not classified (USEPA 1998a).

Pollutant evaluated	Summary of chronic health effects
	Assessment of oral and dermal exposures is undertaken on the basis of a threshold (noting limited data to support carcinogenicity), where the current value from ASTDR (ATSDR 2012d) is most appropriate. Inhalation exposures need to be assessed on the basis of data that are protective of non-carcinogenic and carcinogenic effects, with a non-threshold approach relevant for the assessment of carcinogenic effects. The most current review is available from TCEQ, where an air guideline has been adopted that is protective of all effects, which are dominated by the assessment of carcinogenicity (using a non-threshold approach). Background or ambient intakes are only relevant for oral and dermal exposures, where 10% has been adopted.
Cobalt	Indicators of adverse health effects in humans for cobalt include cardiomyopathy and decreased iodine uptake by the thyroid. Cobalt is a sensitizer in humans by any route of exposure. Sensitized individuals may react to inhalation of cobalt by developing asthma; ingestion or dermal contact with cobalt may result in development of dermatitis. Respiratory effects, including respiratory irritation, wheezing, asthma, pneumonia and fibrosis, have been widely reported in humans exposed to cobalt by inhalation. Epidemiology studies show decreased pulmonary function in workers exposed to inhaled cobalt (USEPA 2008). IARC has classified cobalt metal, cobalt sulfate and other soluble cobalt (II) salts as Group 2B: possible human carcinogen. The USEPA has determined cobalt sulfate (soluble) is described as “likely to be carcinogenic to humans by the inhalation route”. The available data, however, suggest a non-genotoxic mechanism for carcinogenicity. Oral and dermal exposures have been assessed on the basis of a threshold value from RIVM (Baars et al. 2001) while inhalation exposures have been assessed on the basis of the evaluation from the WHO (WHO 2006b) which is considered protective of all adverse health effects. Background or ambient intakes have also been considered.
Copper	Copper is an essential element and as such adverse effects may occur as a result of deficiency as well as excess intakes resulting from contamination. Liver and gastrointestinal effects are the most sensitive health effects from exposure to high levels of copper (ATSDR 2022; MfE 2011a), particularly in sensitive subpopulations. Copper is not considered to be carcinogenic. Exposure to copper has been evaluated on the basis of a toxicity reference value derived from a tolerable upper limit, with background intakes determined on the basis of information on dietary intakes (the key source of copper exposure).
Lead	The key health effects associated with exposure to lead are chronic. There is a large amount of information available about the health effects of lead, with information and data from epidemiological studies being the major lines of evidence. The health effects of lead are the same regardless of the route of exposure (ATSDR 2019b). Health effects associated with exposure to inorganic lead and compounds include, but are not limited to: neurological, renal, cardiovascular, haematological, immunological, reproductive, and developmental effects. Neurological effects of Pb are of greatest concern because effects are observed in infants and children and may result in life-long decrements in neurological function. The most sensitive targets for lead toxicity are the developing nervous system in children; and effects on the haematological and cardiovascular systems, and the kidney in adults. However, due to the multi-modes of action of lead in biological systems, lead could potentially affect any system or organs in the body. The effects of lead exposure have often been related to the blood lead content, which is generally considered to be the most accurate means of assessing exposure (MfE 2011a). Children and pregnant women are particularly sensitive to lead exposure, and low lead exposure studies have focused on a range of health outcomes including on neurological (such as cognitive and behavioural functioning), cardiovascular and reproductive and developmental health endpoints (Armstrong et al. 2014). The International Agency for Research on Cancer (IARC 2006) has classified inorganic lead as Group 2A: probably carcinogenic to humans. While it is appropriate to utilise a blood lead model to evaluate exposure to lead, toxicity reference values have been developed using blood lead models that are protective of adverse health effects in adults and children (UK DEFRA & EA 2014). This assessment has adopted these values as well as information about background lead exposures (principally from the diet).
Manganese	Manganese is an essential element and hence health effects occur as a result of deficiency as well as toxicity. Exposures via inhalation have the potential to result in respiratory effects as

Pollutant evaluated	Summary of chronic health effects
	well as neurological effects (refer to Table B1). By the oral route, manganese is regarded as one of the least toxic elements, however, there is some concern that the neurological effects observed from inhalation exposures also occur with oral exposures. Manganese is not considered to be carcinogenic. The chronic inhalation guideline is based on based on protection of neurological effects. The oral value is based on a tolerable upper intake for the element, with background intakes considered (principally from the diet).
Mercury (as inorganic and elemental)	This assessment has assumed that mercury in air comprises 100% elemental mercury vapour, which will result in a conservative assessment of inhalation exposures in relation to inorganic mercury attached to particles. The central nervous system is generally the most sensitive indicator of toxicity of metallic mercury vapour. Data on neurotoxic effects are available from many occupational studies. Chronic exposure to metallic mercury may result in kidney damage with occupational studies indicating an increased prevalence of proteinuria. Elemental and inorganic mercury are not considered to be carcinogenic. Inhalation exposures have been assessed on the basis of a toxicity value from the WHO (WHO 2003) based on the protection of CNS effects. The value is consistent with guidance from other organisations. Oral and dermal exposures have assumed the form of mercury in the environment is inorganic mercury, where the kidney is the key health effect. Other health effects identified in relation to inorganic mercury include neurological effects and reproductive and developmental effects. Oral and dermal exposures have been assessed on the basis of a provisional tolerable weekly intake established for mercury intakes in the diet (JECFA 2011), with background intakes considered.
Nickel	The respiratory system is the primary site of toxicity for inhaled nickel in both humans and laboratory animals. Nickel and compounds have been established as carcinogenic via inhalation and the compounds are generally considered to be genotoxic, however, the mechanism of action is not well understood. An air guideline has been adopted that is protective of all adverse health effects, including non-carcinogenic and carcinogenic (based on a linear/non-threshold approach) effects. The most sensitive health effects relate to respiratory effects and lung cancer. Nickel is a potent skin sensitiser and ingestion of nickel can result in skin reactions in sensitised individuals. Other health effects associated with ingestion include the potential for kidney and developmental effects. There is no substantial evidence that nickel is carcinogenic via oral or dermal exposures and hence these exposures are assessed on the basis of a threshold toxicity value that is protective of all adverse health effects. Background intakes have been considered where relevant.
Thallium	Thallium is a highly toxic trace element. Acute (non-fatal) exposures have the potential to cause gastrointestinal effects, with alopecia occurring within 2 weeks of elevated exposures. Chronic exposures include hair loss, neurological effects (the most significant adverse health effect), as well as polyneuritis, encephalopathy, tachycardia and degenerative changes of the heart, liver and kidneys. While limited data are available, thallium has not been determined to be carcinogenic. There are limited studies available to establish quantitative toxicity reference values. All available values are based on the same key study, with the value adopted for this assessment being that developed by RIVM (Janssen et al. 1998) and recommended following more recent review (Pearson & Ashmore 2020), with background intakes also considered.
Vanadium	Vanadium exposures have the potential to result in respiratory effects along with gastrointestinal effects, haematological effects and reproductive effects. Most of the available data on this compound relate to vanadium pentoxide which is considered to have carcinogenic potential. For other vanadium compounds (more likely to be present), the carcinogenic potential is not known. Assessment of chronic oral and dermal exposures has adopted available and relevant toxicity values protective of all adverse health effects for vanadium compounds. Assessment of chronic inhalation exposures has adopted the most current guideline value for vanadium pentoxide. Background intakes of vanadium are expected to be negligible.
Dioxin-like compounds	Dioxins and furans are widely present in the environment, some occurring naturally - mostly as unwanted by-products of combustion (e.g. bushfires or volcanos). They also occur due to combustion used in human activities (vehicle emissions, power stations, cooking, heating, cigarettes etc). These compounds are persistent and accumulate in the body. Human exposure to dioxins and dioxin-like substances has been associated with a range of toxic effects,

Pollutant evaluated	Summary of chronic health effects
	including chloracne; reproductive, developmental and neurodevelopmental effects; immunotoxicity; and effects on thyroid hormones, liver and tooth development. Dioxins are also carcinogenic with IARC classifying them as Group 1. Dioxins and furans, however, are not considered to be genotoxic. In addition, the dose required to result in carcinogenic effects is greater than the dose required for more sensitive effects such as developmental and reproductive effects. Developmental effects in males are the most sensitive reproductive health end-point, making children, particularly breastfed infants, a population at elevated risk. Dioxin-like compounds are listed on the Stockholm Convention on Persistent Organic Pollutants. The assessment of exposure, from all pathways, has been undertaken on the basis of a threshold toxicity value established by NHMRC (NHMRC 2002), which is consistent with the WHO evaluation (FAO/WHO 2018; WHO 2019), with background intakes considered based on available data.
PAHs as BaP	PAHs are a large group of organic compounds with two or more fused aromatic rings made up of carbon and hydrogen atoms. There are several hundred PAHs, including derivatives of PAHs. The best known (and studied) is benzo[a]pyrene (BaP), which is also the most toxic. This assessment has assumed that 100% of the PAHs are present as BaP or are as toxic as BaP. This is conservative as there are a range of toxicity equivalent factors established for a number of key PAHs, most of which are significantly less toxic than BaP. A key health effect of exposure to BaP is carcinogenicity. BaP is classified by IARC as a human carcinogen (Group 1). BaP is considered to be a genotoxic carcinogen (USEPA 2017; WHO 1998). The key health effects of exposure to BaP relate to developmental (including developmental neurotoxicity), reproductive, and immunological effects in animal studies. Epidemiology studies (i.e. studies in people) have shown exposure to BaP is associated with adverse birth outcomes (including reduced birth weight, postnatal body weight, and head circumference), neurobehavioral effects, and decreased fertility (USEPA 2017). In regard to cancer, studies have shown that benzo[a]pyrene is carcinogenic at multiple tumour sites (alimentary tract, liver, kidney, respiratory tract, pharynx, and skin) by all routes of exposure in animals. BaP is also a skin irritant and dermal sensitiser. Where there is dermal exposure to coal tars, there is the potential for skin cancers. These effects are not relevant for non-coal tar exposures. The assessment of exposure to BaP is undertaken on the basis of a non-threshold approach, as carcinogenicity is the key health effect associated with exposure. Age-dependent adjustment factors have also been incorporated to ensure exposures as a child (where effects on development increases the risk of cancer) are incorporated.

Table B3: Summary of chronic TRVs adopted for pollutants – threshold effects

Pollutant	Inhalation TRV (mg/m³)	Oral/dermal TRV (mg/kg/day)	GI absorption factor*	Dermal absorption*	Dermal permeability (cm/hr)	Background intakes (as percentage of TRV)	
						Oral/dermal**	Inhalation**
Hydrogen chloride (HCl)	0.026 ^T	NA (gaseous pollutant)				NA	0%
Hydrogen fluoride (HF)	0.029 ^T	NA (gaseous pollutant)				NA	0%
Ammonia	0.32 ^T	NA (gaseous pollutant)				NA	0%
VOCs as 100% benzene	0.03 ^U	NA (gaseous pollutant)				NA	10%
VOCs as 100% formaldehyde	0.011 ^T	NA (gaseous pollutant)				NA	30%
Antimony	0.0003 ^A	0.00086 ^{NH}	15%	0	0.001	0%	0%
Arsenic	0.000067 ^T	0.002 ^N	100%	0.005	0.001	50%	0%
Cadmium	0.000005 ^W	0.0008 ^W	2.5%	0.001	0.001	60%	20%
Chromium (Cr VI assumed)	0.0000043 ^T	0.0009 ^A	2.5%	0	0.002	10%	0%
Cobalt	0.0001 ^W	0.0014 ^D	100%	0	0.0004	20%	0%
Copper	0.49 ^R	0.14 ^W	100%	0	0.001	60%	60%
Lead	0.0005 ^N	0.0006 ^X	100%	0	0.001	50%	0%
Manganese	0.00015 ^W	0.14 ^A	100%	0	0.001	50%	20%
Mercury (as inorganic and elemental)	0.0002 ^W	0.0006 ^W	7%	0	0.001	60%	0%
Nickel	0.000059 ^T	0.012 ^W	4%	0	0.0002	60%	10%
Thallium	0.0007 ^R	0.0002 ^D	100%	0	0.001	10%	10%
Vanadium	0.0001 ^A	0.002 ^D	2.6%	0	0.001	0%	0%
Dioxin-like compounds	8.05E-09 ^R	2.3E-09 ^{NH}	100%	0.03	0.81	54%	54%

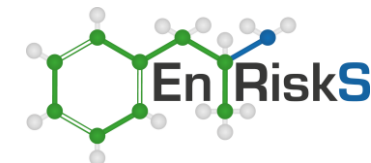
Table B4: Summary of chronic TRVs adopted for pollutants – non-threshold effects

Pollutant	Inhalation TRV (mg/m ³) ⁻¹	Oral/dermal TRV (mg/kg/day) ⁻¹	GI absorption factor*	Dermal absorption*	Dermal permeability (cm/hr)	Background intakes
VOCs as 100% benzene	0.006 ^U	NA (Gaseous pollutant)				NA for non-threshold risk calculations
PAHs as BaP#	0.6 ^U	0.233	100%	0.06	0.71	

Notes for Table B3 and B4:

* GI factor and dermal absorption values adopted from RAIS (accessed in 2023) (RAIS), except for BaP where 6% has been adopted (MfE 2011a)

** Background intakes relate to intakes from inhalation, drinking water and food products. The values adopted based on information provided in the ASC NEPM (NEPC 1999 amended 2013c) and relevant sources as noted for the TRVs. Gaseous pollutant background intakes are not known and hence for this assessment they have been assumed to be negligible except for benzene and formaldehyde as there are monitoring data available for these 2 chemicals.



Age-dependent adjustment factors considered in the quantification of exposure to address exposures as a child

- R = No inhalation-specific TRV available, hence inhalation exposures assessed on the basis of route-extrapolation from the oral TRV, as per USEPA guidance (USEPA 2009)
- A = TRV available from ATSDR, relevant to chronic intakes (ATSDR 2012c, 2012a, 2012b)
- D = TRV available from Dutch authorities – RIVM (Baars et al. 2001; Janssen et al. 1998; van Vlaardingen, Posthumus & Posthuma-Doodeman 2005)
- N = Inhalation guideline adopted for lead from the ambient air quality NEPM (NEPC 2021b), arsenic oral/dermal value and BaP oral/dermal value as adopted in ASC NEPM (NEPC 1999 amended 2013c)
- NH = Dioxin value (and background intakes, which includes natural soil) adopted from NHMRC (NHMRC 2002) and Environment Australia (DEH 2005; EPHC 2005), and antimony and lead values consistent with that adopted by NHMRC to assess intakes in drinking water (NHMRC 2011 updated 2022)
- T = TRV available from TCEQ, relevant to chronic inhalation exposures (and HI=1) (TCEQ 2013a, 2014c, 2015a, 2015d, 2017a, 2017b)
- U = TRV available from the USEPA IRIS (current database) (USEPA IRIS)
- W = TRV available from the WHO, relevant to chronic inhalation exposures (WHO 1999b, 2000e, 2006b, 2017), noting inhalation value adopted for mercury is for elemental mercury (WHO 2003)
- X = TRV derived to be protective of all adverse health effects (lower value is relevant to blood pressure effects in adults and also protective of IQ effects in children), from blood lead modelling (UK DEFRA & EA 2014)

B4 Toxicological profiles

Arsenic

Background

Several comprehensive reviews of arsenic in the environment and toxicity to humans are available (ATSDR 2007a; NRC 2001; UK EA 2009d, 2009e; WHO 2001).

Arsenic is a metalloid which can exist in four valence states (-3, 0, +3 and +5) and forms a steel gray, brittle solid in elemental form (ATSDR 2007a). Under reducing conditions arsenite (AsIII) is the dominant form and in well oxygenated environments, arsenate (AsV) predominates (WHO 2001). Arsenic is the 20th most commonly occurring element in the earth's crust occurring at an average concentration of 3.4 ppm (ATSDR 2007a).

Review of current information from Australia with respect to arsenic indicates the following:

- The most recent Australian Total Diet Survey (ATDS) that addresses arsenic in food was published by FSANZ in 2011 (FSANZ 2011). Based on data presented in this report, dietary intake of arsenic for children aged 2-5 years ranges from a mean of 1.2 µg/kg/day to a 90th percentile of 2.8 µg/kg/day. These intakes are based on total arsenic in produce, rather than inorganic arsenic.
- Review of background intakes from food, water, air, soil and contact with play equipment based on available Australian data presented by (APVMA 2005) suggests background intakes of inorganic arsenic by young children may be on average 0.62 µg/kg/day. Further review of inorganic arsenic intakes by the Joint FAO/WHO Expert Committee on Food Additives indicated that for populations (not located in areas of arsenic contaminated groundwater) intakes by young children ranged from 0.14 to 1.39 µg/kg/day (WHO 2011a). On the basis of the range of intake estimations available, a reasonable estimation of 50% of the oral toxicity reference value (TRV) from sources other than soil has been assumed.
- Intakes from inhalation exposures are low (around 0.0017 µg/kg/day (APVMA 2005)), comprising <1% of the inhalation TRV adopted.

For this assessment, intakes from all other sources have been calculated separately based on available information on the existing environment.

With respect to arsenic toxicity and the identification of appropriate toxicity reference values a number of issues need to be considered. These include: the relevance of non-threshold carcinogenic values for the assessment of oral exposures; identification of an appropriate oral toxicity value; and identification of an appropriate approach and value for inhalation exposures. These are discussed further below.

Classification

The International Agency for Research on Cancer (IARC) has classified arsenic and inorganic arsenic compounds as Group 1 'carcinogenic to humans' (IARC 2012a).

Identification of Toxicity Reference Values

Oral

Arsenic is a known human carcinogen, based on human epidemiological studies that show skin and internal cancers (in particular bladder, liver and lung) associated with chronic exposures to arsenic

in drinking water. The research available on arsenic carcinogenicity is dominated by epidemiological studies (which have limitations) rather than animal studies which differs from carcinogenic assessments undertaken on many other chemicals. The principal reason for the lack of animal studies is because arsenic has not been shown to cause cancer in rodents (most common species used in animal tests) due to interspecies differences between rodents and humans.

Review of arsenic by (IARC 2012a) has concluded the following:

- For inorganic arsenic and its metabolites, the evidence points to weak or non-existent direct mutagenesis (genotoxicity), which is seen only at highly cytotoxic concentrations.
- Long-term, low-dose exposures to inorganic arsenic (more relevant to human exposure) is likely to cause increased mutagenesis as a secondary effect of genomic instability. While the mechanism of action (MOA) is not fully understood it is suggested by (IARC 2012a) that it may be mediated by increased levels of reactive oxygen species, as well as co-mutagenesis with other agents. The major underlying mechanisms observed at low concentrations include the rapid induction of oxidative DNA damage and DNA-repair inhibition, and slower changes in DNA-methylation patterns, aneuploidy, and gene amplification.
- Inhibition of DNA repair leads to co-carcinogenicity.

The WHO guidelines on drinking water (WHO 2017) adopted a practical value based on the analytical limit of reporting rather than based on a dose-response approach. The oral slope factor derived by the USEPA has not been used to derive a guideline as the slope factor is noted by the WHO as likely to be an overestimate.

USEPA reviews have retained the use of a non-threshold approach based on sufficient supporting evidence associated with increased rates of bladder and lung cancer (for inhalation exposures (USEPA 2001). The USEPA approach adopted follows a review by the (NRC 2001) which concluded that “... *internal cancers are more appropriate as endpoints for risk assessment than non-melanoma skin cancers*”. Slope factors relevant for the assessment of these end points range from 0.4 to 23 (mg/kg/day)⁻¹. The use of a non-threshold approach (slope factor), however, is more by default through following the USEPA Carcinogenic Guidelines (USEPA 2005b) as there remains uncertainty on the carcinogenic MOA for arsenic (Sams et al. 2007). Further research is required to define and review the MOA prior to the USA revising the dose-response approach currently adopted. Inherent in the current US approach (where a non-threshold slope factor is derived) are some key uncertainties that likely result in an overestimate of risk, which include:

- the choice of the cancer endpoint
- the choice of the mathematical model used to estimate risk (shape of the dose-response curve at low doses) as there is no clear biological basis for extrapolation
- the assumptions used to estimate exposure from studies (primarily epidemiological studies) (Boyce et al. 2008; Brown 2007; Chu & Crawford-Brown 2006; Lamm & Kruse 2005; SAB 2005).

Review of recent studies presented by (Boyce et al. 2008) has indicated that for carcinogenic effects associated with arsenic exposure a linear (or non-threshold) dose-response is not supported (also note discussion by (Clewell et al. 2007). This is based on the following:

- Epidemiological studies (worldwide) that have repeatedly demonstrated that cancers associated with inorganic arsenic ingestion are observed only in populations exposed to arsenic concentrations in drinking water that are greater than 150 µg/L. In the US, exposures

to concentrations in drinking water have only been associated with carcinogenic effects where mean concentrations are greater than 190 µg/L (Schoen et al. 2004).

- Mechanistic information on how arsenic affects the cellular processes associated with carcinogenicity. This includes consideration that arsenic and its metabolites may modify DNA function through more indirect mechanisms such as inhibition of DNA repair, induction of dysfunctional cell division, perturbation of DNA methylation patterns, modulation of signal transduction pathways (leading to changes in transcriptional controls and the over-stimulation of growth factors), and generation of oxidative stress and that evidence for the indirect mechanisms for genotoxicity identified in in vitro studies have nearly all been at concentrations that are cytotoxic (Klein et al. 2007).

Hence the default approach adopted by the USEPA in adopting a non-threshold approach to the assessment of the carcinogenic effects associated with arsenic exposure is not well supported by the available data. This is consistent with the most recent Australian review available (APVMA 2005). The review conducted considered current information on arsenic carcinogenicity and genotoxicity which noted the following:

“Although exposure to high concentrations of inorganic arsenic results in tumour formation and chromosomal damage (clastogenic effect), the mechanism by which these tumours develop does not appear to involve mutagenesis. Arsenic appears to act on the chromosomes and acts as a tumour promoter rather than as an initiator ...”. “Furthermore, the epidemiological evidence from occupational exposure studies indicates that arsenic acts at a later stage in the development of cancer, as noted with the increased risk of lung cancer mortality with increasing age of initial exposure, independent of time after exposure...”. “Hence arsenic appears to behave like a carcinogen which exhibits a threshold effect. This would also be conceptually consistent with the notion that humans have ingested food and water containing arsenic over millennia and so the presence of a threshold seems likely. Nevertheless the mechanism by which tumour formation develops following arsenic exposure has been and still continues to be a source of intensive scientific investigation.”

On the basis of the above the use of a threshold dose-response approach for the assessment of carcinogenic effects associated with arsenic exposure is considered.

The review of arsenic by the New Zealand Ministry for the Environment (MfE 2011a) noted that while there is general consensus that arsenic is likely to act indirectly on DNA in a sub-linear or threshold manner, it is considered that there is insufficient data available to determine a “well-defined non-linear dose-response”. For this reason, the derivation of the New Zealand soil guideline values has adopted a non-threshold (linear) approach for arsenic (i.e. adopting a default non-threshold approach similar to that adopted by default by the USEPA). This differs from the approach adopted in Australia.

Assessment of End-Points – Oral Exposures

Existing Oral Dose-Response Approaches – Australia

Oral intakes of arsenic were considered in Australia in (Langley 1991) and the Australian Drinking Water Guidelines (ADWG) (NHMRC 2011 updated 2022). The following can be noted from these guidelines:

- The derivation of the previous HIL for arsenic was dated and considers all intakes of arsenic on the basis of a threshold PTWI established by the WHO in 1983, and reconfirmed in 1988 (Langley 1991; WHO 1989). The PTWI adopted was 15 $\mu\text{g/kg/week}$. In setting the PTWI it was noted that there is “a narrow margin between the PTWI and intakes reported to have toxic effects in epidemiological studies” (WHO 1989). The PTWI was withdrawn by JECFA (WHO 2011a) following further review (refer to discussion below).
- The previous ADWG (NHMRC 2004) derived a guideline of 7 $\mu\text{g/L}$ for inorganic arsenic in drinking water based on the former WHO PTWI (noted above) converted to a daily intake (provisional maximum tolerable daily intake) of 2 $\mu\text{g/kg/day}$. The current ADWG (NHMRC 2011 updated 2022) has adopted a guideline of 10 $\mu\text{g/L}$ based on a “practicable achievable” approach supported by contemporary epidemiological studies in which elevated cancer risks and other adverse effects are not demonstrable at arsenic concentrations around 10 $\mu\text{g/L}$. It is noted that this level is equivalent to an adult (70 kg) intake of 0.28 $\mu\text{g/kg/day}$.

A review of arsenic toxicity was conducted by the APVMA (APVMA 2005) where a threshold approach was considered appropriate (noted above). A threshold value of 3 $\mu\text{g/kg/day}$ was derived by the Australian and New Zealand Food Authority (ANZFA now Food Standards Australia New Zealand (FSANZ)) in 1999, and considered in the APVMA (APVMA 2005) review. The review considered that skin cancers appear to be the most sensitive indicator of carcinogenicity of inorganic arsenic in humans and based on epidemiological studies a threshold of 2.9 $\mu\text{g/kg/day}$ (rounded to 3 $\mu\text{g/kg/day}$) can be obtained. This threshold is the value adopted as a provisional tolerable daily intake (PTDI) by FSANZ (FSANZ 2003), similar to the former PTWI available from the WHO (noted above). This approach has been considered by APVMA for all intakes of arsenic (oral, dermal and inhalation). The evaluation has not been further updated.

Oral Dose-Response Approaches - International

Evaluation of arsenic by JECFA considered the available epidemiological data in relation to the increased incidence of lung cancer and urinary tract cancer associated with exposure to arsenic in water and food. Using the data associated with these endpoints, JECFA derived a benchmark dose lower confidence limit for a 0.5% increased incidence ($\text{BMDL}_{0.5}$) of lung cancer (most sensitive endpoint) of 3 $\mu\text{g/kg/day}$ (ranging from 2-7 $\mu\text{g/kg/day}$). Uncertainties associated with the assumptions related to total exposure, extrapolation of the $\text{BMDL}_{0.5}$ and influences of the existing health status of the population were identified. Given the uncertainties and that the $\text{BMDL}_{0.5}$ was the essentially equal to the PTWI (WHO 1989), the PTWI was withdrawn. No alternative threshold values were suggested by JECFA as the application of the BMDL needs to be addressed on a regulatory level, including when establishing guideline levels.

The review conducted by JECFA is generally consistent with that conducted by the European Food Safety Authority (EFSA) Panel on Contaminants in the Food Chain (CONTAM) (EFSA 2010). The review concluded that the PTWI was “no longer appropriate as data are available that shows inorganic arsenic causes cancer of the lung and bladder in addition to skin, and that the range of adverse effects had been reported at exposures lower than those reviewed by the JECFA” in establishing the PTWI. Modelling conducted by EFSA considered the available epidemiological studies and selected a benchmark response (lower limits) of 1% extra risk (BMBL_{01}). BMBL_{01} range from 0.3 to 8 $\mu\text{g/kg/day}$ for cancers of the lung, bladder and skin. The CONTAM Panel (EFSA 2010) concluded that the overall range of BMDL_{01} values of 0.3 to 8 $\mu\text{g/kg/day}$ should be used for the risk characterisation of inorganic arsenic rather than a single reference point, primarily due to the

number of uncertainties associated with the possible dose-response relationships considered. On this basis it would not be appropriate to consider just one value in the range presented.

The assessment completed by New Zealand (MfE 2011a) acknowledges the debate relating to the mechanism of action in relation to carcinogenicity. However they have adopted a linear or non-threshold approach to the assessment of carcinogenic effects, as they consider there is insufficient data to define a threshold. The approach adopted for the quantification of the most sensitive effect, carcinogenicity, is to adopt a risk-specific dose of 0.0086 $\mu\text{g/kg/day}$, which is noted to represent a negligible risk by Canadian agencies. Background intakes are not relevant as the risk index is based on a non-threshold approach.

The determination of an appropriate TRV requires a single value that can be used in a quantitative assessment, rather than a wide range of values, that is considered adequately protective of the population potentially exposed. The determination of an appropriate TRV for arsenic in soil in Australia has, therefore, considered the following:

- The studies considered in the derivation of the different ranges of BMDL values (EFSA 2010; WHO 2011a, 2017) are based on drinking water studies. No studies considered are derived from other sources including soil. There are uncertainties inherent in the epidemiological studies considered by the WHO and EFSA (EFSA 2010; WHO 2011a, 2017). These uncertainties include limitations or absence of information on levels of individual exposure or arsenic intake (from drinking water), limited quantification of arsenic intakes from other sources including food, size of the studies (variable) and the assumption that arsenic intake is the single cause of all endpoints identified.
- The drinking water studies are primarily associated with populations that have poorer nutritional status (i.e. Taiwan and Bangladesh). Studies (as summarised by EFSA (EFSA 2010)) have shown that populations with poor nutrition (and health status) are more susceptible to the prevalence and severity of arsenic-related health effects.
- The largest of the studies conducted was within rural Asian populations which differ from Australian populations with respect to generic lifestyle factors.

In view of the above, consideration of the lower end of the range of BMDL values available from WHO and EFSA (EFSA 2010; WHO 2011a, 2017) is not considered appropriate for the Australian population.

Based on the above considerations, a TRV of 2 $\mu\text{g/kg/day}$ has been adopted. The TRV has been selected on the basis of the following:

- The TRV is at the lower end of the range derived from JECFA, and also lies within, but is not at the lower end of the range presented by EFSA (EFSA 2010; WHO 2011a);
- The value is within the range of no observable adverse effect levels (NOAELs) identified by RIVM (Baars et al. 2001), USEPA (USEPA IRIS) and ATSDR that are associated with non-carcinogenic effects (and derived from drinking water studies in Taiwan and Bangladesh) of 0.8 to 8 $\mu\text{g/kg/day}$. Consistent with the approach discussed above in relation to the range of TRVs relevant to a cancer endpoint, it is not considered appropriate that the most conservative end of this range is adopted for the Australian population.

Due to the level of uncertainty in relation to determining a single TRV for the assessment of arsenic exposures, the oral TRV utilised is not considered to be a definitive value but is relevant for the

current assessment. The approach adopted is based on developing science that should be reviewed in line with further developments in both science and policy.

The dermal absorption factor adopted for arsenic in the ASC NEPM 2013 is 0.005 (NEPC 1999 amended 2013c).

Inhalation

Less data are available with respect to inhalation exposures to arsenic, though trivalent arsenic has been shown to be carcinogenic via inhalation exposures (with lung cancer as the end point). Review of the relevant mechanisms for carcinogenicity by RIVM (Baars et al. 2001) suggests that the mechanism for arsenic carcinogenicity is the same regardless of the route of exposure. Hence a threshold is also considered relevant for the assessment of inhalation exposures. This is consistent with the approach adopted in the derivation of the previous arsenic HIL (Langley 1991) and in the review undertaken by APVMA (APVMA 2005). While NEPC (previous HIL) and APVMA adopted the oral PTWI as relevant for all routes of exposure, RIVM has derived an inhalation-specific threshold value. (Baars et al. 2001) identified that the critical effect associated with chronic inhalation exposures in humans was lung cancer. The lowest observable adverse effect concentration (LOAEC) for trivalent arsenic associated with these effects is $10 \mu\text{g}/\text{m}^3$ (based on the review (ATSDR 2007a)). Applying an uncertainty factor of 10 to address variability in human susceptibility, a tolerable concentration (TC) in air of $1 \mu\text{g}/\text{m}^3$ was derived.

Given the above, there is some basis for the assessment of inhalation exposures to arsenic to adopt an appropriate threshold value, but the available epidemiological studies associated with exposures in copper smelters suggest a linear or non-threshold approach may be relevant. The WHO review of arsenic (WHO 2000b) also suggested the use of a linear (non-threshold) approach to the assessment of inhalation exposures to arsenic. The assessment presented is limited and essentially adopts the US approach with no discussion or consideration of the relevance of the linear model adopted. The later review by WHO (WHO 2001) with respect to inhalation exposures and lung cancer provides a more comprehensive review and assessment. The review presented identified that a linear dose–response relationship is supported by the occupational and epidemiological studies. The three key studies associated with copper smelters in Tacoma, Washington (USA), Anaconda, Montana (USA) and Ronnskar (Sweden) (as summarised in (WHO 2001)) demonstrate a statistically significant excess risk of lung cancer at cumulative exposure levels of approximately $\geq 750 \mu\text{g}/\text{m}^3$ per year.

The relevance of inhalation values derived from studies near smelters to the assessment of contaminated arsenic in soil in areas away from smelters, or in areas where exposures are significantly lower than from the smelters evaluated is not well founded. Hence it is recommended that a threshold approach is considered for the assessment of inhalation exposures associated with arsenic in soil, or where multipathway exposures are being evaluated. The threshold TC derived by RIVM (Baars et al. 2001) of $1 \mu\text{g}/\text{m}^3$ is lower than the cumulative exposure value identified by WHO (WHO 2001) of $750 \mu\text{g}/\text{m}^3$ per year as statistically associated with an increase in lung cancer. The values are considered reasonably comparable if the exposure occurs over a period of 40 years and appropriate uncertainty factors are applied to convert from a lowest observable adverse effect level (LOAEL) to a NOAEL. In addition, the TC is consistent with the TC05 value derived by Health Canada (Health Canada 1993) associated with lung cancer in humans and an incremental lifetime risk of 1 in 100 000. The value adopted is lower than the recommended PTDI adopted for the

assessment of oral intakes (when the TC is converted to a daily intake). Hence use of the RIVM TC has been considered appropriate and adequately protective of all health effects associated with inhalation exposures, including carcinogenicity.

New Zealand (MfE 2002) has adopted an air guideline of $0.0055 \mu\text{g}/\text{m}^3$ (as an annual average) based on the use of inhalation unit risk (non-threshold values) from the USEPA and OEHHa and an acceptable risk of 1 in 100,000. This value is more conservative than the more recently published air guidelines from TCEQ (as below) which also address carcinogenicity using a non-threshold approach.

TCEQ (TCEQ 2012) conducted a review of inhalation toxicity relevant to arsenic. The assessment identified the following:

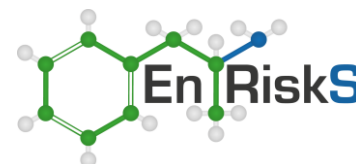
- an acute reference exposure level of $0.0099 \text{ mg}/\text{m}^3$ relevant to assessing 1 hour average exposures was determined based on maternal toxicity in rats exposed via an inhalation study (NOEL_{HEC} of $3.89 \text{ mg}/\text{m}^3$ for arsenic trioxide, application of a 300 fold uncertainty factor and conversion to arsenic)
- long-term exposures to arsenic in occupational environments have been linked to increased risk of lung cancer. The mechanisms of action for carcinogenicity have not been clearly identified, however, as noted above, there are sufficient data to support a genotoxic mechanism of action, and the use of a linear dose-response assessment for evaluating inhalation exposures to arsenic
- based on the available studies on respiratory and lung cancer in occupational workers, TCEQ determined a linear (non-threshold) dose response relationship, with an inhalation unit risk of $0.00015 (\mu\text{g}/\text{m}^3)^{-1}$
- application of the inhalation unit risk along with an incremental carcinogenic risk of 1 in 100,000 resulted in establishing a chronic air guideline of $0.067 \mu\text{g}/\text{m}^3$
- no threshold TRV was established by TCEQ in relation to inhalation exposures.

Based on the above, the more recent evaluation by TCEQ is appropriate for assessing inhalation exposures to arsenic. However, the most appropriate approach for assessing oral and dermal exposures is consistent with the ASC NEPM, where a threshold approach is adopted. When undertaking multipathway exposure assessments, it is difficult to mix non-threshold and threshold approaches in the calculations as multiple exposure pathways would not be considered additive. Hence inhalation exposures have also been assessed on the basis of a threshold, however, the threshold adopted is the chronic air guideline developed by TCEQ that is protective of lung cancer effects (based on a non-threshold approach). This approach ensures all adverse effects are appropriately addressed and risks from multipathway exposures added.

Adopted toxicity reference values

On the basis of the discussion above, the following toxicity reference values (TRVs) have been adopted for arsenic:

- Oral TRV = $0.002 \text{ mg}/\text{kg}/\text{day}$ for oral and dermal intakes
- Inhalation TRV = $0.000067 \text{ mg}/\text{m}^3$ (based on air guideline from TCEQ developed on the basis of non-threshold approach)
- Oral Bioavailability of 100% assumed



- Background Intakes from other sources (as % of TRV) = 50% for oral and dermal, and 0% for inhalation.

Benzene

General

Benzene found in the environment is from both human activities and natural processes. Benzene was first discovered and isolated from coal tar in the 1800s. Today, benzene is made mostly from petroleum sources. Benzene, also known as benzol is a volatile, colourless liquid with a characteristic "aromatic" odour. Benzene evaporates into air very quickly and dissolves slightly in water. Benzene is highly flammable (ATSDR 2007b).

Exposure of the general population to benzene may occur in all urban areas, as motor vehicle emissions are a contributor to benzene levels. Inhalation is the primary route of exposure in industrial and everyday settings. Cigarette smoke contains benzene and is a significant exposure for active smokers. Other exposures include furnishings, solvents, adhesives, pumping petrol and residential areas near chemical manufacturing sites. Trace amounts are typically found in food and water (ATSDR 2007b).

Exposure, absorption, health effects

There is no clinical disease which is unique to benzene toxicity. However, the effects on the haematopoietic and immune systems are well recognised. Data from animal and human studies indicates that benzene is rapidly absorbed through the lungs. Definitive scientific data on the rate of absorption after ingestion of benzene in humans are not available. However, case studies of accidental or intentional poisoning indicate that it is absorbed readily. Benzene can be absorbed through the skin, however the rate of absorption is much lower than that for inhalation (ATSDR 2007b).

Once absorbed, benzene partitions to lipid-rich tissues due to the lipophilic nature of the chemical with total uptake dependant on fat content and metabolism. Benzene accumulates in the adipose tissue, bone marrow and brain. The metabolism of benzene is rapid with water-soluble metabolites excreted within 2 hrs of exposure. A substantial proportion of absorbed benzene is eliminated unchanged in exhaled air, with the remainder eliminated in the urine, principally as metabolites. Benzene is metabolised primarily in the liver and to a lesser extent, in the bone marrow. There is no evidence that the route of administration has any substantial effect on subsequent metabolism of benzene in humans or animals (ATSDR 2007b).

Acute benzene exposure produces central nervous system excitation and depression. Acute exposure to high concentrations of benzene in air results in neurological toxicity and may sensitize the myocardium to endogenous catecholamines. Acute ingestion of benzene causes gastrointestinal and neurological toxicity (WHO 1993).

Chronic exposure to benzene results primarily in haematotoxicity, including aplastic anaemia, pancytopenia, or any combination of anaemia, leukopenia, and thrombocytopenia. Chronic benzene exposure is associated with an increased risk of leukaemia. In chronic exposures, benzene metabolites are considered the toxic agents, not the parent compound. The relative contribution of different benzene metabolic pathways may be dose related, with more toxic agents produced by high affinity low capacity pathways (WHO 1993).

Classification

Benzene is classified as a “known” human carcinogen (Category A) by the USEPA for all routes of exposure based upon convincing human evidence as well as supporting evidence from animal studies. IARC has classified benzene in Group 1 (known human carcinogen) (IARC 2012b; USEPA 2005b, 2005a).

NICNAS (NICNAS 2001) has classified benzene as a “Carcinogen, Category 1” and “Toxic: Danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed”. In addition, benzene is classified as “Irritating to eyes, respiratory system and skin” and as a mutagenic substance in Category 3 “Possible risks of irreversible effects”.

Benzene is carcinogenic via oral and inhalation routes of exposure (ATSDR 2007b; IARC 2012b; UK EA 2009c; WHO 1993) indicates that the overall results of available studies show that it is appropriate to consider benzene (and/or its metabolites) as genotoxic (though the genotoxic profile is considered unusual (Baars et al. 2001)).

Quantitative toxicity values

On the basis that benzene is considered a genotoxic carcinogen (and not mutagenic) it is appropriate that carcinogenic endpoints are assessed on the basis of a non-threshold approach. In addition there is the potential for mixtures of benzene with toluene, ethylbenzene and xylenes to result in additive effects associated with non-carcinogenic/neurological effects (ATSDR 2004b). Hence both threshold and non-threshold endpoints require quantification with respect to potential exposures to benzene, where also present with TEX. The following non-threshold and threshold chronic values are available from Level 1 Australian and International sources:

Source	Value	Basis/Comments
ADWG (NHMRC 2011 updated 2022)	SF = 0.035 (mg/kg/day)⁻¹	A drinking water guideline was derived on the basis of the WHO evaluation (see below) with consideration of a 1 in 1,000,000 lifetime risk level.
NEPM (NEPC 2004)	No unit risk presented Investigation level = 0.003ppm	A regional air investigation level of 0.003 ppm (chronic yearly exposures) is recommended as an 8-year goal. The basis for the air guideline value is not clear from the supporting information, however the value is intended to be used as an ambient goal associated with all sources and is not directly relevant for the assessment of exposures from one source.
New Zealand (MfE 2002)	Air GV = 0.03 mg/m ³	Air guideline value (as an annual average) based on the WHO upper value inhalation unit risk (as below) and an acceptable carcinogenic risk between 1 in 10,000 and 1 in 100,000.
WHO	SF = 0.035 (mg/kg/day)⁻¹ UR = 6x10⁻⁶ (ug/m³)⁻¹	Oral SF derived (WHO 2011b) based on route extrapolation of the data considered in the derivation of the inhalation UR. Consideration of oral data from a 2-year gavage study on rats and mice with a linearised multistage model resulted in a similar oral slope factor as derived on the basis of the epidemiological data (from inhalation studies). Inhalation UR derived based on data on leukaemia from epidemiological inhalation studies where a range of unit risk values were derived (4.4-7.5x10 ⁻⁶ (ug/m ³) ⁻¹). The geometric mean value was adopted for the purpose of deriving an air guideline.
UK (UK EA 2009c)	Derived index doses	Oral index dose derived on the basis of USEPA approach and a lifetime cancer risk of 10 ⁻⁵ . Inhalation index dose based on WHO approach and adopting an air guideline of 3.2 ug/m ³ equivalent to a lifetime cancer risk of 1x10 ⁻⁵ , and consideration of a range of non-cancer effects that would be relevant at concentrations above 1.7-3.2 ug/m ³ .

Source	Value	Basis/Comments
Texas (TCEQ 2013b)	Chronic ESL (non-threshold) = 0.0045 mg/m^3 , UR = $2.2 \times 10^{-6} (\text{ug/m}^3)^{-1}$ Chronic ESL (threshold) = 0.084 mg/m^3 Acute ESL (health) = 0.17 mg/m^3 Acute ESL (odor) = 8.7 mg/m^3	Chronic ESL for threshold effects based on an occupational exposure study with decreased ALC as the critical effect, uncertainty factor of 30 and HI of 0.3 (to account for mixture exposures) Chronic ESL for threshold effects consistent with the derivation present by the USEPA.
ATSDR (ATSDR 2007b)	Inh MRL = 0.0098 mg/m^3	Chronic inhalation MRL has been derived on the basis of a benchmark dose (lower limit 0.25 sd) of 0.098 mg/m^3 associated with decreased lymphocyte counts in humans and an uncertainty factor of 10.
USEPA (USEPA 1998b, 1999) (non-threshold)	SF = 0.015 to 0.055 $(\text{mg/kg/day})^{-1}$ UR = 2.2 to $7.8 \times 10^{-6} (\text{ug/m}^3)^{-1}$	Oral SF (last reviewed in 2000) derived on the basis of route extrapolation of the data considered in the derivation of the inhalation UR. The range presented is consistent with that considered by the WHO where the same approach was used. Inhalation UR based on a linear model from data on leukaemia from epidemiological inhalation studies (same studies considered by the WHO).
USEPA (USEPA 2002) (threshold)	Oral RfD = 0.004 mg/kg/day Inhal RfC = 0.03 mg/m^3	Non-carcinogenic threshold values are available from the USEPA. The oral RfD and Inhalation RfC are derived on the basis of a benchmark dose (lower limit) of 1.2 mg/kg/day associated with decreased lymphocyte counts in human studies (inhalation study) and an uncertainty factor of 300.

The non-threshold values available from the WHO and USEPA are derived from the same studies and consider similar approaches. The USEPA provides a range of values while the WHO has adopted the geometric mean. As the two approaches are similar the values derived by the WHO (also adopted in the ADWG, NHMRC 2022) have been adopted for the quantification of carcinogenic risks.

Where relevant, the quantification of non-carcinogenic chronic effects, the threshold values available from the USEPA are current and appropriate for use in this assessment:

- For inhalation exposures a chronic RfC = 0.03 mg/m^3 has been adopted.
- For oral/dermal exposures a chronic RfD = 0.004 mg/kg/day has been adopted.

It is noted that where risk-based criteria that may be derived for benzene will be dominated by the calculation of criteria based on non-threshold effects. Hence, only non-threshold TRVs are adopted for the derivation of such criteria.

No quantitative data are available to assess dermal exposures; therefore the oral value has been adopted for the purpose of assessing both oral and dermal exposures. Dermal permeability and other physical/chemical properties relevant to the quantification of volatilisation have been obtained from RAIS website (RAIS).

Background Intake

Background intakes of benzene relevant for urban and rural areas are based on inhalation exposure being the major contributor. The major source of benzene in air in urban areas is benzene in petrol. Emissions from vehicles will, therefore, include benzene.

NSW EPA reported benzene concentrations in air in Sydney (NSW EPA 2004) for the period 1996 to 2001 at a number of locations. The average concentrations included 0.0074 mg/m^3 for the Sydney CBD, 0.0035 mg/m^3 in Rozelle (inner city area) and 0.00128 mg/m^3 in western Sydney (St Marys). These concentrations comprise between 4% and 25% of the adopted TRV (i.e. 0.03 mg/m^3). Concentrations of benzene in other cities (Perth (average 0.0008 mg/m^3), Brisbane (range

in 2019/2020 – 0.001-0.006 mg/m³) and Melbourne (average 0.0018 mg/m³) are similar to those in Sydney.

The data reported by NSW EPA (NSW EPA 2004) for 1996 to 2001 are dated and do not reflect more current benzene levels in urban air in Australia. This is because national cleaner fuel standards required refineries to reduce benzene levels in petrol from around four per cent to less than one per cent (Fuel Standard (Petrol) Determination 2001) from 2006. This has resulted in lower levels of benzene in ambient air in all cities in Australia.

Monitoring of benzene at 5 locations in Sydney in 2006, and 2 sites in 2008-2009 reported lower levels of benzene in the range 0.0006 to 0.0016 mg/m³ (NSW EPA 2013) (consistent with the lowering of benzene content in fuel). These levels are lower than those reported for 1996 to 2001 and are more relevant to current levels of benzene in urban air. These concentrations are around 5% of the TRV. The most recent data available for Brisbane (Springwood monitoring site) also indicate levels are lower than in the late 90s (range in 2019/2020 – 0.001-0.006 mg/m³).

It is noted that, when soil quality guidelines for use in contaminated land assessments were determined for various petroleum hydrocarbons (including benzene), the authors (CRC CARE 2011) adopted the maximum background level of 20% from the earlier study in Sydney CBD (NSW EPA 2004). For this assessment, a background intake of 10% of the TRV has been considered where relevant as this is based on a conservative interpretation of more current data based on lower concentrations of benzene in fuel as required by regulation.

Cadmium

General

Several comprehensive reviews of cadmium in the environment and toxicity to humans are available (ATSDR 2012e; UK EA 2009f; WHO 2004a).

Pure cadmium is a silver-white, lustrous and malleable metal, is a solid at room temperature, is insoluble in water, and has a relatively low melting point and vapour pressure. The most common oxidation state of cadmium is 2+. Naturally occurring cadmium is commonly found in the earth's crust associated with zinc, lead, and copper ores. Whereas pure cadmium and cadmium oxides are insoluble in water, some cadmium salts including cadmium chloride, cadmium nitrate, cadmium sulfate and cadmium sulfide are soluble in water (ATSDR 2012e).

Cadmium is found naturally in mineral forms (primarily sulfide minerals) in association with zinc ores, zinc-bearing lead ores, and complex copper-lead-zinc ores. Due to its corrosion-resistant properties, a wide range of commercial and industrial applications have been developed involving cadmium-containing compounds and alloys that are used in a wide range of materials and products including batteries, pigments, metal coatings and platings, stabilisers for plastics, nonferrous alloys and solar cell devices (ATSDR 2012e).

Cadmium is toxic to a wide range of organs and tissues, and a variety of toxicological endpoints (reproductive toxicity, neurotoxicity, carcinogenicity) have been observed in experimental animals and subsequently investigated in human populations (MfE 2011a).

The toxicity of cadmium in air is dependent on the form of cadmium. The toxicity is higher with the more soluble cadmium compounds. Acute inhalation exposure to cadmium at concentrations may cause destruction of lung epithelial cells, resulting in decreased lung function, pulmonary oedema,

tracheobronchitis, and pneumonitis in both humans and animals. Other effects identified in animal studies include decreased immune response, erosion of the stomach, decreased body weight gain and tremors (ATSDR 2012e).

Numerous studies examining the toxicity of cadmium in workers have identified the respiratory tract, the kidney and bone as sensitive targets of toxicity. Other effects identified include developmental and reproductive effects, hepatic effects, haematological effects and immunological effects (ATSDR 2012e).

Background

The WHO review of cadmium included food intakes provided by FSANZ of 0.1 µg/kg/day (FSANZ 2003; WHO 2004a). Intakes for a young child aged 2-5 years from the 23rd Australian Food Survey ranged from a mean of 0.32 µg/kg/day to a 90th percentile of 0.44 µg/kg/day (FSANZ 2011). These intakes are similar to those estimated in New Zealand (MfE 2011a), which are 0.41 µg/kg/day for children and 0.26 µg/kg/day for adults. While the WHO (2004) review notes that intakes of cadmium from food can exceed the adopted toxicity reference value, data from FSANZ (2011) does not suggest this is the case. Based on the available data from FSANZ (2011), intakes from food comprise up to 60% of the recommended oral TRV.

Cadmium was detected in air samples collected from urban and rural areas in NSW (NSW DEC 2003). The average concentration reported was 0.17 ng/m³, ranging from 0.3 to 1 ng/m³. These concentrations constitute <5% to 20% of the recommended inhalation TRV in air (also considered as an international target in the DEC document). Background levels for cadmium in air can be conservatively assumed to comprise 20% of the recommended inhalation TRV.

Classification

IARC has classified cadmium and cadmium compounds as a Group 1 agent (i.e., carcinogenic to humans) based on additional evidence of carcinogenicity in humans and animals. It is noted that there is limited evidence of carcinogenicity in experimental animals following exposure to cadmium metal (IARC 2012a). The USEPA has classified cadmium as a probable human carcinogen via inhalation.

Review of Available Values/Information

The following has been summarised from the review of cadmium presented by MfE (MfE 2011a):

- Cadmium is primarily toxic to the kidney, especially to the proximal tubular cells where it accumulates over time and may cause renal dysfunction. Loss of calcium from the bone and increased urinary excretion of calcium are also associated with chronic cadmium exposure. Recent studies have reported the potential for endocrine disruption in humans as a result of exposure to cadmium. Notably, depending on the dosage, cadmium exposure may either enhance or inhibit the biosynthesis of progesterone, a hormone linked to both normal ovarian cyclicity and maintenance of pregnancy. Exposure to cadmium during human pregnancy has also been linked to decreased birth weight and premature birth.
- While cadmium has been classified as known human carcinogen (based on inhalation data from occupational inhalation data), there is no evidence of carcinogenicity via the oral route of exposure.

- There are conflicting data on the genotoxicity of cadmium. Some studies indicate that chromosomal aberrations occur as a result of oral or inhalation exposures in humans, while others do not. Studies in prokaryotic organisms largely indicate that cadmium is weakly mutagenic. In animal studies genetic damage has been reported, including DNA strand breaks, chromosomal damage, mutations and cell transformations (ATSDR 2012e). IARC (2012) concluded that ionic cadmium causes genotoxic effects in a variety of eukaryotic cells, including human cells, although positive results were often weak and/or seen at high concentrations that also caused cytotoxicity. Based on the weight of evidence, MfE considered there to be weak evidence for the genotoxicity of cadmium.

On the basis of the available information, TRVs relevant for oral (and dermal) intakes and inhalation intakes have been considered separately.

Oral (and Dermal) Intakes

Insufficient data are available to assess carcinogenicity via oral intakes and, therefore, the oral TRV has been based on a threshold approach with renal tubular dysfunction considered to be the most sensitive endpoint. The following are available for oral intakes from Level 1 Australian and International sources:

Source	Value	Basis/Comments
ADWG (NHMRC 2011 updated 2022)	TDI = 0.0007 mg/kg/day	The threshold oral value available from the ADWG (NHMRC 2011 updated 2022) of 0.0007 mg/kg/day is derived from a WHO/JECFA evaluation in 2000. The JECFA summary provided in 2004 noted that a PTWI of 0.007 mg/kg was established in 1988. This differs from that referenced (not cited) and considered in the ADWG. It is noted however that the WHO may have rounded the TDI adapted as both values are similar.
MfE (MfE 2011a)	TDI = 0.0008 mg/kg/day	Adopted the toxicity value from the WHO review (as below).
JECFA (WHO 2010a)	PTMI = 0.025 mg/kg (equivalent to PTDI = 0.0008 mg/kg/day)	Review of cadmium by JECFA in 2010 withdrew the previous PTWI (noted below). The review considered more recent epidemiological studies where cadmium-related biomarkers were reported in urine following environmental exposures. They identified that in view of the long half-life of cadmium in the body, dietary intakes should be assessed over months and tolerable intakes assessed over a period of at least a month. Hence the committee established a PTMI of 0.025 mg/kg. While established over a month, use of the value in the methodology adopted for establishing HILs requires a daily value. Exposures assessed in the HILs are chronic and hence, while used as a daily value, it relates to long term exposures to cadmium. The former JECFA (WHO 2005) review provided a PTWI of 0.007 mg/kg for cadmium in reviews available from 1972 to 2005. This is equivalent to an oral PTDI of 0.001 mg/kg/day. This is based on review by JECFA where renal tubular dysfunction was identified as the critical health outcome with regard to the toxicity of cadmium. The PTWI is derived on the basis of not allowing cadmium levels in the kidney to exceed 50 mg/kg following exposure over 40-50 years. This PTDI is adopted by FSANZ (2003), the current WHO DWG (2011) and was used in the derivation of the current HIL (Langley 1991).
WHO DWG (WHO 2017)	PTMI = 0.025 mg/kg (equivalent to PTDI = 0.0008 mg/kg/day)	Based on JECFA review noted above
RIVM (Baars et al. 2001)	TDI = 0.0005 mg/kg/day	Value derived on the same basis as JECFA (WHO 2005) however RIVM has included an additional uncertainty factor of 2 to address potentially sensitive populations.

Source	Value	Basis/Comments
ATSDR (ATSDR 2012e)	Oral MRL = 0.0001 mg/kg/day	The MRL is based on the BMDL ₁₀ for low molecular weight proteinuria estimated from a meta-analysis of environmental exposure data (from ATSDR).
USEPA (USEPA IRIS)	RfD = 0.0005 mg/kg/day for intakes from water and RfD = 0.001 mg/kg/day for intakes from food	Cadmium was last reviewed by the USEPA in 1994. The RfD for intakes from water derived on the same basis as considered by ATSDR. RfD derived for intakes from food on the basis of a NOAEL of 0.01 mg/kg/day from chronic human studies and an uncertainty factor of 10.

The available toxicity reference values or oral intakes are similar from the above sources with the PTMI established by JECFA (WHO 2010) providing the most current review of the available studies. This value has, therefore, been recommended for use and is consistent with that adopted in the ADWG (NHMRC 2011 updated 2022).

Inhalation Exposures

Inhalation of cadmium has been associated with carcinogenic effects (as well as others). Sufficient evidence is available (IARC 1993) to conclude that cadmium can produce lung cancers via inhalation (IARC 2012a). While cadmium is thought to be potentially genotoxic, the weight of evidence is not clear. In addition, epidemiology studies associated with lung cancer have confounding issues that limit useful interpretation (WHO 2000e). It is noted that the USEPA derived their inhalation unit risk on the basis of the same study that the WHO dismissed due to confounding factors. In particular, a lot of the epidemiological data available also includes co-exposures with zinc and in some cases both zinc and lead.

Cadmium is not volatile and hence inhalation exposures are only relevant to dust intakes. These are not likely to be significant for soil contamination and hence the consideration of carcinogenic effects (where the mode of action is not clear) using a non-threshold approach is not considered appropriate. It is appropriate to consider intakes on the basis of a threshold approach associated with the most significant end-point. This is consistent with the approach noted by RIVM (2001) and considered by the WHO (2000) and UK EA (2009) where a threshold value for inhalation based on the protection of kidney toxicity (the most significant endpoint) has been considered. The value derived was then reviewed (based on the US cancer value) and considered to be adequately protective of lung cancer effects. On this basis, the WHO (2000) derived a guideline value of 0.005 $\mu\text{g}/\text{m}^3$ and the UK EA (2009) derived an inhalation TDI of 0.0014 $\mu\text{g}/\text{kg}/\text{day}$ (which can be converted to a guideline value of 0.005 $\mu\text{g}/\text{m}^3$ – the same as the WHO value).

The review by TCEQ (TCEQ 2016) indicated that multiple mechanisms (e.g., aberrant gene expression, inhibition of DNA damage repair, induction of oxidative stress/reactive oxygen species and genomic instability, inhibition of apoptosis) appear to be involved in cadmium-induced carcinogenesis. The approach adopted for the derivation of a chronic air guideline was to consider noncarcinogenic effects (kidney effects most sensitive) and carcinogenic effects using a linear (non-threshold) approach. The air guideline derived based on protection of kidney effects 0.011 $\mu\text{g}/\text{m}^3$ was lower than that derived for carcinogenic effects (0.02 $\mu\text{g}/\text{m}^3$). Both of these values are higher than the WHO air guideline adopted. Hence the value adopted for assessing inhalation exposures is considered protective of all adverse health effects.

Adopted toxicity reference values

On the basis of the discussion above, the following toxicity reference values (TRVs) have been adopted for cadmium:

- Oral TRV (TRV_o) = 0.0008 mg/kg/day (WHO 2010a)
- Dermal absorption (DAF) = negligible (0%)
- Inhalation TRV (TRV_i) = 0.000005 mg/m³ (WHO 2000e)
- Background intakes from other sources:
 - BI_o = 60% for oral intakes
 - BI_i = 20% for inhalation

Chromium VI

General

Several comprehensive reviews of chromium VI (Cr VI) in the environment and toxicity to humans are available (APVMA 2005; ATSDR 2012d; UK DEFRA & EA 2002).

Cr VI is less stable than the commonly occurring trivalent chromium but can be found naturally in the rare mineral crocoite. Cr VI typically exists as strongly oxidizing species such as CrO_3 and CrO_4^{2-} . Some Cr VI compounds, such as chromic acid and the ammonium and alkali metal salts (e.g., sodium and potassium) of chromic acid are readily soluble in water. The Cr VI compounds are reduced to the trivalent form in the presence of oxidisable organic matter. However, in natural waters where there is a low concentration of reducing materials, Cr VI compounds are more stable (ATSDR 2012d).

Chromium is of fundamental use in a wide range of industries including the metallurgical (to produce stainless steels, alloy cast irons and nonferrous alloys), refractory (to produce linings used for high temperature industrial furnaces) and chemical industries. In the chemical industry, Cr VI is used in pigments, metal finishing and in wood preservatives (ATSDR 2012d).

The soil chemistry and toxicity of chromium is complex and hence the form of chromium in soil is of importance. In general soil chromium is present as Cr III, however, the distribution of Cr III and Cr VI depends of factors such as redox potential, pH, presence of oxidising or reducing compounds and formation of Cr complexes and salts (ATSDR 2012d).

Cr VI can readily pass through cell membranes and be absorbed by the body. Inside the body, Cr VI is rapidly reduced to Cr III. This reduction reaction can act as a detoxification process when it occurs at a distance from the target site for toxic or genotoxic effect. Similarly if Cr VI is reduced to Cr III extracellularly, this form of the metal is not readily transported into cells and so toxicity is not observed (ATSDR 2012d). However, if Cr VI is transported into cells, and close to the target site for toxic effect, under physiological conditions it can be reduced. This reduction reaction produces reactive intermediates, which can attack DNA, proteins, and membrane lipids, thereby disrupting cellular integrity and functions (ATSDR 2012d).

The toxicity is higher for soluble forms of Cr VI than insoluble forms. The respiratory system is the most sensitive health effect for both forms (TCEQ 2014a).

In the environment, Cr VI less toxic form Cr III in the presence of oxidizable organic matter and hence assuming that Cr VI remains following long-term deposition to land is highly conservative. It is more likely to be present as Cr III.

Cr VI is unstable in the body and is reduced to Cr V, Cr IV and ultimately to Cr III by many substances, including ascorbate and glutathione. It is believed that the toxicity of Cr VI compounds results from damage to cellular components during this process (WHO 2013b).

Chronic exposure to Cr VI via inhalation has been found (in occupational studies) to result in respiratory tract and eye irritation, and cancer (respiratory tract and lung cancer) (WHO 2013b).

Oral exposures to Cr VI have not demonstrated an association with cancer in humans, however animal studies have shown carcinogenic potential. Dermal exposure to Cr VI can result in ulcers and allergic contact dermatitis (WHO 2013b).

Background

Review of current information from Australia with respect to chromium indicates the following:

- Intakes of total chromium were addressed in the FSANZ 22nd Australian Total Diet Survey (FSANZ 2008). Estimated dietary intakes of chromium (total) for infants and 2-3 year old's ranged from 14 µg/day to 26 µg/day, and for adults ranged from 14 µg/day to 53 µg/day for males 19-30 years. The average values reported are consistent with intakes reported from Germany and US by APVMA (APVMA 2005). Dietary intakes of total chromium may comprise a significant portion of the TDI for Cr VI. However, it is noted that the most common form of chromium in fresh produce is Cr III. If Cr VI comprised 10% of the total Cr intake from the diet (based on data from bread analyses, (Soares, Vieira & Bastos Mde 2010) then background intakes may comprise 0.09 to 0.17 µg/kg/day for young children aged 2-3 years. It is considered reasonable that an average intake be adopted given additional intakes from plant uptake are included in addition to these intakes, resulting in some doubling up of intakes from food sources. The average intake of Cr VI is estimated to be 0.13 µg/kg/day for 2-3 year olds, approximately 10% of the recommended oral TRV.
- In New Zealand a higher level of background intake has been identified, at 1.2 µg/kg/day for children and 0.53 µg/kg/day for adults.
- No data on Cr VI in air are available for Australia. Intakes of Cr VI from air may comprise up to 30% of total chromium (Baars et al. 2001), which has been reported up to 1.5 ng/m³ (Baars et al. 2001) to 3 ng/m³ (UK DEFRA & EA 2002). It is noted that concentrations of Cr VI in Europe and the UK are expected to be higher than in Australia due to the potential for long-range atmospheric transport from a greater proportion of industry in these general regions. Based on the recommended TRV for particulate phase Cr VI, these conservative air concentrations comprise less than 1% of the TC and are assumed negligible.

Classification

IARC (IARC 2012a) has classified Cr VI compounds as Group 1 carcinogens: carcinogenic to humans based on: sufficient evidence in humans for the carcinogenicity of Cr VI compounds as encountered in the chromate production, chromate pigment production and chromium plating industries.

Chromium is classified by the USEPA as a Group A: known human carcinogen by the inhalation route, with carcinogenicity by the oral route of exposure noted to be Group D: not classified (USEPA 1998a).

Review of Available Values/Information

Oral

There are limited data available regarding the carcinogenic potential of ingested Cr VI. Cr VI compounds appear to be genotoxic and some reviews (Baars et al. 2001) suggest that a non-threshold approach is relevant to all routes of exposure. Some drinking water studies (NTP 2008) are available that show a statistically significant increase in tumours in rats and mice. However, there are currently no peer-reviewed data available to determine a quantitative non-threshold value for ingestion of Cr VI compounds (note a value has been recently published by (OEHHA 2011) using a non-threshold approach). There is also some suggestion (De Flora et al. 1997; Jones 1990) that there may be a threshold for the carcinogenicity of Cr VI based on hypothesis that it is a high dose phenomenon where the dose must exceed the extracellular capacity to reduce Cr VI to Cr III.

The following are available for oral intakes:

Source	Value	Basis/Comments
ADWG (NHMRC 2011 updated 2022)	No evaluation available	The ADWG does not specifically derive a guideline; however it references the WHO DWG assessment, where the basis for derivation is not clear. No quantitative toxicity values can be obtained from these sources.
New Zealand (MfE 2011a)	0.003 mg/kg/day	Adopted the RfD from the USEPA evaluation.
WHO DWG (WHO 2017)	No evaluation available	Current guideline based on limit of detection as no adequate toxicity studies were available to provide the basis for a NOAEL. It is noted that chromium is included in the plan of work of rolling revisions to the WHO DWG (2011).
UK DEFRA & EA (UK DEFRA & EA 2002)	TDI = 0.003 mg/kg/day	Adopted oral RfD from the USEPA.
RIVM (Baars et al. 2001)	TDI = 0.005 mg/kg/day	RIVM has adopted a provisional threshold TDI of 0.005 mg/kg/day based on a 1-year drinking water study in rats as used in the derivation of the former and current USEPA RfD (with a small difference in the application of uncertainty factors).
ATSDR (ATSDR 2012d)	MRL = 0.0009 mg/kg/day	The chronic oral MRL is based on a BMDL ₁₀ of 0.09 mg/kg/day for non-neoplastic lesions of the duodenum in a 2-year drinking water study in rats and mice (NTP 2008) and an uncertainty factor of 90. The study considered by ATSDR was not available when the other organisations (USEPA) reviewed Cr VI.
USEPA IRIS (USEPA 1998a)	RfD = 0.003 mg/kg/day	The USEPA IRIS entry (last reviewed in 1998) derived an oral RfD of 0.003 mg/kg/day based on a NOAEL of 2.5 mg/kg/day from a 1-year drinking water study in rats and an uncertainty factor of 300 and modifying factor of 3 to address uncertainties in the study. The confidence level in the study, database and RfD is noted to be low.

It is recommended that the lower value derived by (ATSDR 2012d) be adopted for the assessment of oral exposures to Cr VI as the assessment provides the most current comprehensive assessment of the available studies, including a more recent key study (NTP 2008) not available at the time of review by other organisations. The values adopted by RIVM and the UK are essentially the same, using the study considered by the USEPA (McKenzie et al. 1958) in the derivation of the RfD. It is noted that review by Health Canada (Health Canada 2004) considered the study used by the USEPA was of poor quality, however, it was utilised due to the lack of additional, better quality data.

Inhalation

Epidemiological studies have shown an association between exposure to Cr VI and lung cancer. These studies have involved chromate production, chromate pigment production and use, chromium plating, stainless steel welding, ferrochromium alloy production and leather tanning. Various Cr VI compounds have also been shown to be carcinogenic via inhalation in experimental animals. Cr VI has also been shown to be genotoxic. As noted by UK DEFRA & EA (UK DEFRA & EA 2002), there is some suggestion that chromium-induced cancer of the respiratory tract may be exclusively a high-dose phenomenon with a threshold approach relevant to low-dose exposures but quantitative data is lacking.

Chromium is not volatile and hence inhalation exposures are only relevant to dust intakes. These are not likely to be significant for soil contamination and hence the consideration of carcinogenic effects using a non-threshold approach may not be appropriate. It is appropriate to consider intakes on the basis of a threshold approach associated with the most significant end-point. In addition, inhalation exposures relating to soil contamination (dust) are expected to differ from the occupation studies from which the non-threshold criteria are derived (where inhalation of fine dust and chromic acid mists occurs). These issues were considered by ITER (ITER 1998) in the derivation of an RfC that is relevant for environmental exposures only, not to occupational exposures associated with mists and aerosols, and USEPA (USEPA 1998a) in the derivation of an RfC.

The following are available for inhalation exposures for Cr VI particulates or dust:

- No Australian guideline values are available for Cr VI.
- The WHO (WHO 2013b) has derived a tolerable concentration of $0.03 \mu\text{g}/\text{m}^3$ based on non-carcinogenic respiratory effects in humans for Cr VI salts (not the acid form). To protect against lung cancer effects an air guideline of $0.00025 \mu\text{g}/\text{m}^3$ (based on lifetime exposures and 1 in 100,000 risk). This is based on the WHO (WHO 2000b, 2013b) inhalation unit risk of $0.04 (\mu\text{g}/\text{m}^3)^{-1}$ derived from the mean of a number of occupational studies.
- The USEPA (USEPA 1998a) derived an inhalation RfC of $0.0001 \text{ mg}/\text{m}^3$ or $0.1 \mu\text{g}/\text{m}^3$ for Cr VI particulates based on lower respiratory effects in a subchronic rat study. The USEPA review of particulate exposures indicated chromium inhalation induced pneumocyte toxicity and suggested that inflammation is essential for the induction of most chromium inhalation effects and may influence the carcinogenicity of Cr VI compounds. The USEPA has also derived a separate RfC (lower) for exposure to chromic acid mists and dissolved Cr VI aerosols, which would be relevant for the assessment of an occupational environment.
- ITER (ITER 1998) derived an inhalation RfC of $0.0003 \text{ mg}/\text{m}^3$ or $0.3 \mu\text{g}/\text{m}^3$ for Cr VI particulates based on the same study as USEPA considered but the value derived was on the basis of an arithmetic average of benchmark concentrations for the pulmonary inflammation end point.
- New Zealand (MfE 2002) has adopted an air guideline for Cr VI of $0.0011 \mu\text{g}/\text{m}^3$ as an annual average. This is based on adopting a non-threshold approach, an acceptable risk of 1 in 100,000 and US unit risk factors (derivation is not provided).
- WHO has derived a range of air guideline values based on an inhalation unit risk of $0.04 (\mu\text{g}/\text{m}^3)^{-1}$ derived from the mean of a number of occupational studies.
- USEPA (USEPA 1998a) also derived a unit risk of $0.012 (\mu\text{g}/\text{m}^3)^{-1}$ derived from one occupational study (also considered by WHO).

- TCEQ (TCEQ 2014a) has derived a noncarcinogenic air guideline of $0.22 \mu\text{g}/\text{m}^3$ based on changes in lung weight in rats, and a carcinogenic air guideline of $0.0043 \mu\text{g}/\text{m}^3$ based on lung cancer in industrial workers and use of a linear (non-threshold approach) and 1 in 100,000 risk level.
- UK DEFRA & EA (UK DEFRA & EA 2002) has derived an index dose of $0.001 \mu\text{g}/\text{kg}/\text{day}$ for Cr VI based on occupational inhalation studies based on a lung cancer end point, consideration of the WHO non-threshold approach and a target risk level of 10^{-4} .
- RIVM (Baars et al. 2001) has adopted a cancer risk value of $0.0025 \mu\text{g}/\text{m}^3$ based on occupational inhalation studies based on a lung cancer end point, consideration of the WHO non-threshold approach and a target risk level of 10^{-4} . It is noted that a 10^{-4} target risk level is used for inhalation guidelines by (UK DEFRA & EA 2002) and RIVM (Baars et al. 2001). The value results in guidelines that address background levels of Cr VI reported in ambient air, which range up to 30% of total chromium reported (up to $0.0015\text{--}0.0025 \mu\text{g}/\text{m}^3$).
- ATSDR (ATSDR 2012d) has derived a chronic inhalation MRL for Cr VI aerosols and mists but this is not considered relevant to the derivation of toxicity reference values for Cr VI bound to particulates.

Based on the above there are a range of values available, with mixed guidance as to the most appropriate approach to adopt for assessing inhalation exposures to Cr VI bound to particulates. To be sufficiently conservative the more recent values from TCEQ (TCEQ 2014a) have been adopted in this assessment.

Adopted toxicity reference values

On the basis of the discussion above the following toxicity reference values (TRVs) have been adopted for Cr VI:

- Oral TRV (TRV_o) = $0.0009 \text{ mg}/\text{kg}/\text{day}$ (ATSDR 2012d)
- Inhalation TRV (TRV_i) = $0.0043 \mu\text{g}/\text{m}^3$ (TCEQ 2014a)
- Background intakes from other sources (as % of TRV) = 10% for oral/dermal intakes and 0% for inhalation.

Dioxin-like compounds

General

The assessment of dioxins utilises the information and evaluations undertaken by the NHMRC (NHMRC 2002) and the Australian Government (DEH 2005; EPHC 2005; FSANZ 2004), both of which reference the evaluations conducted by the WHO (Van den Berg et al. 2006; WHO 2000f) (JECFA 2002; WHO 2019). These are the principal sources of information presented in this review as the evaluations provided in these guidance remain current (FAO/WHO 2018; WHO 2019) relevant for the assessment of dioxin exposures in Australia. The following provides a summary of the available information relevant to the characterisation of health effects.

The term “dioxins and dioxin-like substances” describes a group of organic chemicals that remain in the environment for a long time. There are several hundred of these compounds that are members of three closely related families: polychlorinated dibenzodioxins (PCDDs), polychlorinated dibenzofurans (PCDFs or furans) and certain co-planar polychlorinated biphenyls (PCBs). They are two- or three-ring structures that can be chlorinated to varying degrees. PCBs can have up to 10

chlorine atoms substituting for hydrogen atoms, and PCDDs and PCDFs can have up to eight. The term dioxins is commonly used to refer to all three families together.

The compounds often have similar toxicity profiles and common mechanisms of action, and are generally considered together as a group to set guidelines, using toxicity equivalent factors (TEFs) to get a toxic equivalent (TEQ) concentration. The TEFs relate the toxicity of the individual dioxin and dioxin-like compounds to the most well studied compound 2,3,7,8-TCDD. The current approach is to use TEFs available from the 2005 WHO review (Van den Berg et al. 2006), resulting in the reporting of concentrations as a WHO₀₅ TEQ.

The National Dioxins Program (NDP) has focused on the 29 most toxic individual chemicals within these groups which are recognised internationally as being most harmful to humans and animals.

Sources and exposures

PCDDs and PCDFs are widely present in the environment, occurring naturally, but mainly as unwanted by-products of combustion and of various industrial processes. PCDFs were major contaminants of PCBs, but neither PCDDs nor PCDFs have ever been deliberately manufactured for use for commercial purposes other than for scientific research.

PCBs are not natural substances but were globally manufactured and used in the past. Although PCB manufacture is now prohibited under the Stockholm Convention, their release into the environment still occurs from the disposal of large-scale electrical equipment and waste, from metallurgical uses, and some chemical manufacture and processing. The Stockholm Convention also requires the phase-out of the use of PCBs in equipment by 2025 and the final elimination of PCBs by 2028.

Mixtures of the substances with different numbers and positions of chlorine substitution are found in the environment. The degree of chlorination of dioxin mixtures released into the environment through incineration is determined by the source material and the amount of chlorine available.

PCDDs and PCDFs are by-products of industrial processes, particularly waste incineration, cement kilns firing hazardous waste, chlorine bleaching of pulp, and thermal processes in the metallurgical industry, as well as the manufacture of chlorophenols and phenoxy herbicides. They can also be generated by natural events, such as volcanic eruptions and forest fires. PCBs were previously manufactured for use as dielectric insulating fluids (with low electrical conductivity) in larger-scale electrical products such as transformers and capacitors, in heat transfer and hydraulic systems, and in industrial oils and lubricants. PCDFs were common contaminants of commercial PCB mixtures.

The National Dioxins Program in Australia involved the assessment of dioxins in the environment as a result of various different sources (DEH 2004, 2005; EPHC 2005; FSANZ 2004). The following is a summary:

Sources

- Dioxin-like compounds are mainly unintended by-products of combustion processes. It has been estimated that 96 per cent of dioxin-like compounds in the environment are from emissions to air.
- The new inventory estimates that total emissions to air in Australia are between 160–1,788 g TEQ/year with a best estimate being 500 g. Uncontrolled combustion, which includes bushfires, waste burning and accidental fires, is estimated to contribute nearly 65 per cent of

total emissions to air and over 80 per cent of total emissions to land, with most being emitted from grass fires.

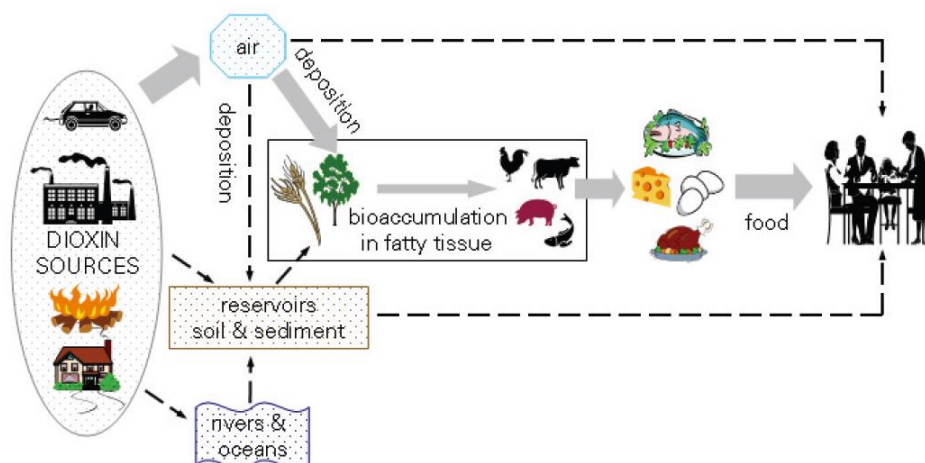
- Dioxin-like compounds from vehicle emissions account for less than 2 per cent of total dioxins emissions to air.
- Disposal and landfilling are estimated to be the largest source of dioxin emissions to water, contributing over 75 per cent of total emissions.

Body burden

- Blood serum levels of dioxin-like compounds were presented for the Australian population. The levels reported were considered very low by international standards with a mean of 10.9 pg TEQ/g lipid. The data showed increasing levels with age, related to on-going lifetime intakes of dioxin-like compounds.
- Dioxin-like compounds were also detected in breastmilk with a mean of 9 pg TEQ/g lipid. While breast milk contains low levels of these chemicals because of its fat content, all babies are exposed to dioxin-like compounds whether breastfed or not. This is because other foods such as infant formula also contain dioxin-like compounds because of their fat content. Breast feeding is still the normal and most appropriate method for feeding infants as supported by the Australian health authorities.

Background intakes

- The program included the collection of data to evaluate dioxin-like compounds levels in air, soil, water and our diet. This was used to determine the range of likely background intakes of dioxin-like compounds for Australians.
- For the general population, over 95 per cent of exposure to dioxin-like compounds is through the diet, with foods of animal origin such as meat, dairy products and fish being the main sources. These intakes of dioxin-like compounds into the human body are illustrated below.
- Based on the dietary study of dioxins, the intake of dioxin-like compounds for the Australian population is lower than in most other countries.
- The risk assessment (DEH 2005) found that for Australians aged 2 years or older, the monthly intake of dioxin-like compounds was between 3.9–15.8 pg TEQ/kg bw/month.
- Estimates of intake based on serum concentrations suggests that during approximately the last 25 years the average intake was probably close to 1.3 pg WHO-TEQ/kg bw/day. Where this intake is considered, this comprises 54% of the adopted tolerable intake.
- Intakes are lower in females than males for the same age, and levels decline with age in both sexes, the most rapid decline occurring after puberty. Infants and toddlers had a higher intake.



Pathway for dioxin-like compounds entering our bodies (DEH 2004)

Background intakes for New Zealand populations have been estimated (MfE 2011a) to be 10 pg/kg/month (i.e., 33% of the tolerable monthly intake adopted in New Zealand) based on the dietary intake of adult males, assumed to be also relevant to children.

Health effects

These compounds are persistent in the environment and tend to accumulate in biological systems. One of the most extensively studied PCDD congeners, 2,3,7,8-tetrachlorodibenzo-*p*-dioxin (2,3,7,8-TCDD), exhibits a broad range of toxic effects in laboratory animals, some at very low doses.

Human exposure to dioxins and dioxin-like substances has been associated with a range of toxic effects, including chloracne; reproductive, developmental and neurodevelopmental effects; immunotoxicity; and effects on thyroid hormones, liver and tooth development. Dioxins are also carcinogenic. Developmental effects in males are the most sensitive reproductive health end-point, making children – particularly breastfed infants – a population at elevated risk.

In 1997, IARC classified TCDD as Group 1: carcinogenic to humans, based on evidence from occupationally exposed workers and animal studies. The overall evaluation concluded:

- 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD) is carcinogenic to humans (Group 1).
- Other polychlorinated dibenzo-*p*-dioxins are not classifiable as to their carcinogenicity to humans (Group 3).
- Dibenzo-*p*-dioxin is not classifiable as to its carcinogenicity to humans (Group 3).
- Polychlorinated dibenzofurans are not classifiable as to their carcinogenicity to humans (Group 3).

The USEPA has not classified TCDD in relation to carcinogenicity.

It can be concluded that TCDD is not a genotoxic carcinogen, but a multi-site carcinogen in experimental animals. This has been demonstrated using several lines of evidence to act through a mechanism involving the Ah receptor. This receptor is highly conserved in an evolutionary sense and functions the same way in humans as in experimental animals (Tiesjema & Baars 2009). The dose required to be of concern in relation to carcinogenic effects is greater than those relevant to reproductive and developmental effects (the most sensitive non-carcinogenic effects).

Dioxins and dioxin-like substances are persistent organic pollutants (POPs) covered by the Stockholm Convention on Persistent Organic Pollutants; they can travel long distances from the emission source and can bioaccumulate in food chains. Human exposure occurs mainly through consumption of contaminated food, but higher levels of exposure can occur in occupational settings. Public health and regulatory actions are needed to reduce emissions of these substances, as required by the Stockholm Convention, and to reduce human exposure, particularly for children.

Toxicity reference values

Tolerable daily intake adopted for Australian assessments

Based on an analysis of various international hazard assessments and relevant literature published between 1999 and late 2003, it is considered that the Australian Tolerable Monthly Intake (TMI) of 70 pg/kg bw/month (or 2.3 pg/kg/day where long term exposures are assessed on the basis of a daily intake) as recommended by the NHMRC and the TGA's Office of Chemical Safety in 2002 (NHMRC 2002) should be adequately protective of the general population with respect to effects of dioxin-like compounds. This value is the same as that set by the WHO/FAO Joint Expert Committee on Food Additives and Contaminants (JECFA) in 2002 (JECFA 2002), which has been retained by the WHO (FAO/WHO 2018; WHO 2019).

The JECFA TMI is based on a LOEL of 25 ng/kg/day for TCDD from a reproductive study in rats and a NOEL of 13 ng/kg/day for TCDD for another reproductive study on rats (with effects on sperm and prostate weights the sensitive effects identified). These were converted to a human equivalent monthly intake of 630 pg/kg and 330 pg/kg (accounting for background body burden, 1st order kinetics at low doses and absorption of 50% and systematic half-life in humans of 7.6 years). Uncertainty factors of 9.6 and 3.2 were applied to these studies respectively to account for intraspecies variability and the use of a LOEL (for the first study). This results in a range of tolerable intakes between 40 and 100 pg/kg/month, with the mid-point of 70 pg/kg/month adopted.

Tolerable daily intake adopted for New Zealand assessments

Review of dioxins and dioxin-like polychlorinated biphenyls (PCBs), i.e. dioxin-like compounds, by MfE (MfE 2011a) also concluded that TCDD is not a genotoxic carcinogen, with developmental effects identified as the most sensitive health endpoint, which is also protective of carcinogenicity. The review acknowledges there is general agreement between the various expert committees that a threshold, or tolerable intakes are appropriate for assessing dioxin like compounds, where the monthly intake value of 70 pg/kg/month is appropriate. Given the long half-lives of dioxins, and thus the likely lack of effect of small excursions of a daily or even weekly intake, it is recommended that a monthly intake toxic-equivalent dose (TEQ) is used.

The Ministry of Health, however, established a maximum monthly intake of 30 pg/kg/month (or 1 pg/kg/day where long term exposures are evaluated on the basis of a daily intake), based on the lower end of the range of tolerable intakes determined by the older WHO (1998) review. The MfE has retained use of this lower tolerable monthly intake, which has been adopted for assessments completed in New Zealand.

Other assessments

The DEH Risk Assessment (DEH 2005) on dioxins provides a review of the other international assessments available at the time of the publication. These support the approach outlined above.

The review completed by RIVM (Tiesjema & Baars 2009) identified a provisional TDI of 2 pg/kg/day based on the JECFA evaluation.

The USEPA conducted a review of dioxins over a long period of time.

The USEPA evaluation conducted in 2000 (USEPA 2000) concluded that although dioxins can initiate biochemical and biological events potentially leading to a range of cancer types and non-cancer effects in animals and humans, *'there is currently no clear indication of increased disease in the general population attributable to dioxin-like compounds'*. However, the USEPA stated that the lack of a clear indication of disease could not be taken as evidence that dioxins were having no effect. This review also identified that it was appropriate to assess carcinogenic effects, however no oral slope factor was derived.

The final re-assessment of dioxin-like compounds was released by the USEPA in 2012. This review focused on the non-carcinogenic health endpoints. This identified and utilised data from 2 more recent human studies (published in 2008 from the Seveso incident in 1976), where LOAELs were identified for reproductive and developmental effects. One study showed that men exposed in childhood had a reduced sperm count and motility. The other study related to elevated levels of thyroid-stimulating hormone (TSH) in neonates. A PBPK model was used (as the studies reported LOAELs as pg/g fat or TSH and dioxin levels in blood) to derive an oral RfD of 0.7 pg/kg/day. This RfD is listed as an estimate with uncertainty spanning perhaps an order of magnitude. Given this level of uncertainty, the RfD calculated by the USEPA should not be considered to be sufficiently different to that derived by JECFA and adopted in Australia.

Adopted toxicity reference values

On the basis of the discussion above, the following toxicity reference values (TRVs) have been adopted for dioxin-like compounds:

- TRV for all pathways of exposure = 70 pg/kg/month (equivalent to 2.3 pg/kg/day for continuous exposures) (NHMRC 2002)
- Background intakes from other sources (as % of TRV) = 54%.

Formaldehyde

General

Formaldehyde is a colourless, highly flammable gas that is sold commercially as 30 to 50% (by weight) aqueous solutions. Formaldehyde enters the environment from natural sources and from direct human sources, such as automotive and other fuel combustion and industrial onsite uses. Secondary formation also occurs, by the oxidation of natural and anthropogenic organic compounds present in air. Motor vehicles are the largest direct human source of formaldehyde in the environment with releases from industrial processes generally lower (WHO 2002).

When formaldehyde is released to or formed in air, most of it degrades, and a very small amount moves into water. Formaldehyde does not persist in the environment, but its continuous release and formation result in long-term exposure near sources of release and formation (WHO 2002).

The reported odour threshold for formaldehyde is variable, listed in the range 0.02 to 1 ppm (ACGIH 2017; DECOS 2003; NICNAS 2006; WHO 2010b). Human subjects in chamber tests have been reported to readily detect formaldehyde concentrations of 0.01 ppm in air (U.S. NRC 1980; 1981).

Exposure, absorption and health effects

Formaldehyde is formed endogenously (i.e. naturally in the body) during the metabolism of amino acids and xenobiotics, with much of it bound to macromolecules. In addition, formaldehyde is a metabolite of methanol (WHO 2002). Studies have indicated that exposure to 2.5 mg/m³ or less does not increase blood formaldehyde concentrations and exposure to 0.5 mg/m³ or less does not increase the presence of metabolites of formaldehyde in urine (WHO 2010b).

Formaldehyde is a highly reactive gas that is soluble and absorbed quickly at the point of contact. It is rapidly metabolised, such that exposure to high concentrations (up to 15 ppm in rats) does not result in increased blood concentrations. More than 90% of inhaled formaldehyde gas is absorbed and rapidly metabolized to formate in the upper respiratory tract. Repeated formaldehyde exposure caused toxic effects only in the tissues of direct contact after inhalation, oral or dermal exposure characterised by local cytotoxic destruction and subsequent repair of the damage.

Sensory irritation of the eyes and respiratory tract by formaldehyde has been observed consistently in clinical studies and epidemiological surveys in occupational and residential environments. At concentrations higher than those generally associated with sensory irritation, formaldehyde may also contribute to the induction of generally small, reversible effects on lung function (WHO 2002).

Amounts of formaldehyde which produce marked toxic effects at the portal of entry, do not lead to an appreciable systemic dose and thus do not produce systemic toxicity. This is consistent with formaldehyde's high reactivity with many cellular nucleophiles and its rapid metabolic degradation. The available data does not indicate any reproductive or developmental effect (UNEP 2002).

Classification

Epidemiological studies taken as a whole do not provide strong evidence for a causal association between formaldehyde exposure and human cancer, although the possibility of increased risk of respiratory cancers, particularly those of the upper respiratory tract, cannot be excluded on the basis of available data. Therefore, based primarily upon data derived from laboratory studies, the inhalation of formaldehyde under conditions that induce cytotoxicity and sustained regenerative proliferation is considered to present a carcinogenic hazard to humans.

IARC (IARC 2006) has classified formaldehyde as Group 1 (carcinogenic to humans) with the older review completed by the USEPA (in 1991) classifying it as B1 (probable human carcinogen).

There is sufficient evidence that formaldehyde causes nasal cancer in animals and nasopharyngeal cancer in humans with a non-linear, biphasic concentration/dose-response relationship. Carcinogenicity studies in rats, mice and hamsters do not show a consistent association between formaldehyde and lymphohaematopoietic malignancies. Associations between exposure to formaldehyde and nasopharyngeal malignancies and leukaemia in humans are limited to high exposure concentrations. Increased cell proliferation due to cell damage is considered a key mechanism for the development of nasal malignancies following exposure to formaldehyde, where a threshold has been identified (WHO 2010b).

Review of formaldehyde genotoxicity by the WHO and UNEP (UNEP 2002; WHO 2002) indicates that it is considered to be weakly genotoxic, with good evidence of an effect at site of contact, but less convincing evidence at distal sites. Further review by IARC (IARC 2006) determined that both genotoxicity and cytotoxicity play important roles in the carcinogenicity of formaldehyde in nasal

tissues, with the dose-response relationships identified to be non-linear and bi-phasic. Hence, a threshold approach to setting a guideline for cancer effects is appropriate (WHO 2010b).

Overall, inhalation exposure effects of formaldehyde are expected to be limited to the site of contact, generally the nasal and upper airways and guidelines derived on the basis of a threshold that consider both non-cancer and carcinogenic endpoints are relevant to consider.

Background sources

As discussed above, formaldehyde is found ubiquitously in the environment, from natural sources including biomass combustion (forest and bush fires), decomposition and volcanoes. Direct human sources, such as automotive and other fuel combustion and industrial onsite uses, also contribute formaldehyde to the environment, with motor vehicles considered to be the largest direct human source (releases from industrial processes are generally lower) (WHO 2002). Formaldehyde is also formed in the atmosphere due to the breakdown of hydrocarbons in the troposphere (HSDB database).

The indoor air concentration depends somewhat on the types of materials used in construction (ATSDR 1999a, 2010). The major sources of formaldehyde in indoor air are combustion processes (e.g. smoking cigarettes, heating, cooling, candle burning) and building materials and consumer products including (WHO 2010b):

- furniture and wooden products containing formaldehyde-based resins such as particleboard, plywood and medium-density fibreboard
- insulating materials
- textiles
- do-it-yourself products such as paints, wallpapers, glues, adhesives, varnishes and lacquers
- household cleaning products such as detergents, disinfectants, softeners, carpet cleaners and shoe products
- cosmetics such as liquid soaps, shampoos, nail varnishes and nail hardeners
- electronic equipment, including computers and photocopiers
- other consumer items such as insecticides and paper products.

Studies investigating formaldehyde concentrations in indoor air areas such as bedrooms have reported average formaldehyde concentrations in the range 0.009 to 0.03 ppm. This includes a study of 185 homes in Perth, where indoor air formaldehyde concentrations ranged between 0.002 and 0.11 ppm (consistent with data from other studies) (WHO 2010b). The ATSDR indicates that indoor air concentrations range from 0.08 to 0.03 ppm in most homes (ATSDR 1999a, 2010), with concentrations in some mobile homes reported up to 0.8 ppm (Paustenbach et. al. 1997). One study in China reported a mean formaldehyde concentration of 0.19 ppm in recently refurbished houses. It is also noted that indoor air concentrations can reach more than 0.16 to 0.27 ppm close to someone who is smoking cigarettes in the room (Paustenbach et. al. 1997; WHO 2010b).

Formaldehyde concentrations in outdoor air are generally much lower than indoor air concentrations. Study data indicates that outdoor air concentrations in US cities are in the range 0.0008 to 0.01 ppm, with concentrations reported up to 0.09 to 0.15 ppm in areas with photochemical smog and heavy traffic (Paustenbach et. al. 1997). The mean outdoor air concentration from a study across Brazil, Canada, Germany, Italy, Mexico, the Netherlands and the US was 0.006 ppm (range of 0.001 to 0.01 ppm). The mean outdoor air concentration in China was

0.01 ppm (WHO 2010b). The greatest sources of formaldehyde in outdoor air in the Sydney metropolitan area are residential wood heaters and non-road diesel equipment and transport. Monitoring of outdoor air at 5 sites in 2006 and 2 sites in 2008 to 2009 reported concentrations of 0.002 to 0.003 ppm (NSW EPA 2013).

Toxicity reference values – inhalation exposures

The following table provides an overview of the available TRVs relevant to the assessment of inhalation exposures for formaldehyde.

Source	Value	Basis/Comments
WHO (WHO 2002)	0.1 mg/m ³ relevant to 30-minute exposure	Guideline protective of nose and throat irritation in humans. No chronic guideline has been developed by the WHO
ATSDR (ATSDR 1999a)	Acute MRL = 0.05 mg/m ³ Chronic MRL = 0.01 mg/m ³	The acute MRL is based on a range of clinical symptoms in humans. The chronic MRL is based on histological evidence of mild damage to the nasal epithelial tissue for workers in an occupational environment and application of a 30 fold uncertainty factor.
OEHHA (OEHHA 2013)	Acute REL = 0.055 mg/m ³ for 1-hour average Acute REL = 0.009 mg/m ³ for 8-hour average Chronic REL = 0.009 mg/m ³	OEHHA RELs based on protection of eye irritation in humans (acute REL) and respiratory effects (8-hour and chronic guideline)
TCEQ (TCEQ 2013a, 2014b)	Acute = 0.05 mg/m ³ as 1-hour average Acute = 0.05 mg/m ³ as 24 hour average Chronic = 0.011 mg/m ³	Acute inhalation guideline based on protection of short-term eye and nose irritation in humans. It is noted that the odour threshold for formaldehyde is 0.62 mg/m ³ . Chronic inhalation guideline based on elevated rates of eye, nasal and lower airway discomfort in humans. The TCEQ evaluation considered both threshold effects and non-threshold effects (specifically nasopharyngeal cancer), with the guideline established on the basis of a threshold being the driving (lower) health endpoint/approach. Hence the threshold guideline has been adopted.
USEPA IRIS (USEPA 2022)	Chronic RfC = 0.007 mg/m ³	The USEPA evaluation is based on outcomes from a range of studies relating to asthma, decreased pulmonary function, allergic conditions and sensory irritation in humans and application of a 3 or 10 fold uncertainty factor. This threshold value is not considered different to that derived by TCEQ.

Based on the available data, the acute and chronic guidelines established by TCEQ have been adopted for the assessment of inhalation exposures to formaldehyde. For the public, not workers or smokers, background intakes of formaldehyde have been assumed to be a small proportion of the chronic TRV adopted.

Mercury

General

Several comprehensive reviews of mercury in the environment and toxicity to humans are available and should be consulted for more detailed information (ATSDR 1999b; CCME 1999; JECFA 2011; UK EA 2009a; USEPA 1997a; WHO 1991a, 2000c, 2003). The following provides a summary of the key aspects of mercury.

Mercury is a heavy metal which exists in three oxidation states: 0 (elemental), +1 (mercurous) and +2 (mercuric). As well as the common mercurous and mercuric inorganic salts, mercury can also

bind covalently to at least one carbon atom. Thus the most commonly encountered exposures associated with mercury are with elemental mercury, inorganic mercuric compounds and methyl mercury.

Mercury occurs naturally as a mineral is widely distributed by natural and anthropogenic processes. The most significant natural source of atmospheric mercury is the degassing of the Earth's crust and oceans and emissions from volcanoes. Man-made sources such as mining, fossil fuel combustion and industrial emissions generally contribute less on a global scale, but more on a local scale. Wet and dry deposition to land and surface water result in mercury sorption to soil and sediments.

Uses of mercury include use in the electrical and chlor-alkali industry (lamps, batteries and as cathodes in the electrolysis of sodium chloride to produce caustic soda and chloride), industrial and domestic instruments, laboratory and medical instruments and dental amalgam (mixed in proportion of 1:1 with a silver-tin alloy).

Mercury in the environment, including groundwater, exhibits complex behaviour that affects both its mobility and potential toxicity. Mercury has a low solubility in water; however, it also has the potential to form multiple species in the environment, which can lead to increased total mercury concentrations in aqueous systems. The relative toxicity of mercury is also dependent on the form in which it occurs, which, is dependent on: biogeochemical processes, partitioning between solids, and complexation with dissolved organic and inorganic ligands.

On the basis of the potential for long-range transport, persistence in water, soil and sediment, bioaccumulation, toxicity and ecotoxicity, mercury is considered persistent and is addressed in the 1998 UN-ECE Convention on Long-Range Transboundary Air Pollution on Heavy Metals (UNECE 1998). The United Nations Environment Programme (UNEP) Governing Council concluded, at its 22nd session in February 2003, after considering the key findings of the Global Mercury Assessment report, that there is sufficient evidence of significant global adverse impacts from mercury to warrant further international action to reduce the risks to humans and wildlife from the release of mercury to the environment.

Potential for exposure

Ingestion of soil and dust is considered the most significant pathway of exposure for inorganics in soil. The consideration of bioavailability and other exposure pathways has been further reviewed as noted below:

Oral Bioavailability

The bioavailability of different forms of mercury, by different routes of exposure, are expected to vary considerably (Imray & Neville 1996) with oral bioavailabilities reported in the range 2% – 15% for inorganic mercury and 80% to 100% for methyl mercury. Insufficient data are available to adequately define the bioavailability of the different forms of mercury from soil. On this basis a default approach of assuming 100% oral (and inhalation) bioavailability has been adopted. It is noted that site-specific assessment of bioavailability can be considered where required.

Dermal absorption:

Review of dermal absorption by MfE (MfE 2011a) has noted that *“Mercury reacts with skin proteins, and as a result penetration does not increase commensurably with increasing exposure concentration but rather approaches a plateau value. Mercury has a permeability coefficient in the*

order of 10^{-5} cm/h (Guy et al., 1999), which compares to permeability coefficients in the order of 10^{-4} cm/h for lead.”

ATSDR (ATSDR 1999b) note that absorption of mercurous salts in animals can occur through the skin, however no quantitative data are available, hence a default value of 0.1% has been adopted based on the lower end of the range for metals presented by USEPA (USEPA 1995).

ATSDR also noted no information was identified for absorption of methylmercury via dermal absorption. The UK (UK EA 2009a) notes that dermal absorption of methyl mercury is reported to be similar to that of inorganic mercury. Hence the value adopted for inorganic mercury has also been adopted for methyl mercury. It is noted that dermal absorption of dimethylmercury has been reported to be of potential significance and may need to be considered in a site-specific assessment if identified as the key form of mercury in soil.

The USEPA (USEPA 2004) has recommended the use of a gastrointestinal absorption factor (GAF) of 7% for inorganic mercury based on mercuric chloride and other soluble mercury salt studies used in the derivation of the oral RfD. The GAF is used to modify the oral toxicity reference value to a dermal value in accordance with the USEPA (USEPA 2004) guidance provided.

Inhalation of Dust:

Inorganic mercury and methyl mercury are not volatile and inhalation exposures associated with particulates outdoors and indoors are expected to be of less significance than ingestion of soil. Note that if elemental mercury is present then vapour phase issues need to be considered on a site-specific basis.

Exposure to elemental mercury:

Limited data is available concerning the absorption of elemental mercury. Inhaled mercury vapour by humans indicates approximately 80% of the vapour crosses the alveolar membranes into the blood. Ingested elemental mercury is poorly absorbed from the gastrointestinal tract (with approximately 0.01% absorbed, WHO 2003) unless there is an unusual delay in passage through the gastrointestinal tract or a gastrointestinal abnormality. This is partly due to the formation of sulfur laden compounds on the surface of the metal which prevents absorption. The processes of absorption in the gastrointestinal tract via sorption of mercury vapour (following partitioning in the GI tract to a vapour phase) have not been demonstrated in the available studies or case studies associated with accidental ingestion of elemental mercury. When evaluating exposures to elemental mercury, absorption following ingestion is too low to be of significance as the vapour inhalation pathway is of most importance (EA 2002, 2009).

Dermal absorption of mercury vapour is limited and may only contribute approximately 2.5% of absorbed mercury following inhalation exposures. No data are available concerning dermal absorption of liquid metallic mercury (ATSDR 1999b).

Absorbed mercury is lipophilic and rapidly distributed to all tissues and able to cross the blood-brain and foetal barriers easily. Mercury is oxidised in the red blood cells by catalase and hydrogen peroxide to divalent ionic mercury. Approximately 7-14% of inhaled mercury vapour is exhaled within a week after exposure. The rest of the elemental mercury is either excreted via sweat and saliva, or is excreted as mercuric mercury. Approximately 80% is excreted as mercuric mercury via faeces and urine. Half-life elimination is approximately 58 days (ATSDR 1999b).

Acute exposure to high concentrations of mercury vapour has been associated with chest pains, haemoptysis, breathlessness, cough and impaired lung function with the lung identified as the main target following acute exposure.

The central nervous system is generally the most sensitive indicator of toxicity of metallic mercury vapour. Data on neurotoxic effects are available from many occupation studies.

Chronic exposure to metallic mercury may result in kidney damage with occupational studies indicating an increased prevalence of proteinuria.

Exposure to inorganic mercury:

Limited data is available concerning the absorption of inhaled mercury compounds; however it is expected to be determined by the size and solubility of the particles. Absorption of ingested inorganic mercury has been estimated to be approximately 5 to 10% with absorption be children greater than for adults.

Review of dermal absorption by New Zealand (MfE 2011b) has noted that “*Mercury reacts with skin proteins, and as a result penetration does not increase commensurably with increasing exposure concentration but rather approaches a plateau value. Mercury has a permeability coefficient in the order of 10^{-5} cm/h (Guy et al., 1999), which compares to permeability coefficients in the order of 10^{-4} cm/h for lead.*” ATSDR (1999) note that absorption of mercurous salts in animals can occur through the skin, however no quantitative data are available, hence a default value of 0.1% has been adopted based on the lower end of the range for metals (USEPA 1995).

The USEPA (USEPA 2004) has recommended the use of a gastrointestinal absorption factor (GAF) of 7% for inorganic mercury based on mercuric chloride and other soluble mercury salt studies used in the derivation of the oral RfD. The GAF is used to modify the oral toxicity reference value to a dermal value in accordance with the USEPA (2004) guidance provided.

Inorganic mercury compounds are rapidly distributed to all tissues following absorption. The fraction that crosses the blood-brain and foetal barriers is less than for elemental mercury due to poor lipid solubility. The major site of systemic deposition of inorganic mercury is the kidney. Most inorganic mercury is excreted in the urine or faeces.

Acute exposure to high concentrations of ingestion of inorganic mercury has been associated with gastrointestinal damage, cardiovascular damage, acute renal failure and shock.

The kidney is the critical organ associated with chronic exposure to inorganic mercury compounds. The mechanism for the end toxic effect on the kidney, namely autoimmune glomerulonephritis, is the same for inorganic mercury compounds and elemental mercury and results in a condition sometimes known as nephrotic syndrome.

There is some evidence that inorganic mercury may cause neurological effects, particularly associated with studies of mercuric chloride. Reproductive and developmental effects have been observed in rats given mercuric chloride.

Exposure to methylmercury:

Limited data are available concerning the absorption of inhaled methylmercury compounds, however studies on rats indicates rapid and almost complete absorption of inhaled methylmercury vapour. Ingested methylmercury is almost completely absorbed. ATSDR (1999) also noted no

information was identified for absorption of methylmercury via dermal absorption. The UK (EA 2009) notes that dermal absorption of methylmercury is reported to be similar to that of inorganic mercury. Hence the value adopted for inorganic mercury has also been adopted for methylmercury.

Methylmercury is distributed via the blood to all tissues. It can cross into the brain and foetus. The major site of systemic deposition of methylmercury is the kidney. Hair levels are typically used as an index of exposure to mercury and there is a proportional relationship between mercury intake, blood mercury and hair mercury. Methylmercury is converted to mercuric mercury in animals and humans, though less readily than for elemental mercury.

The key target of methylmercury in humans is the CNS, particularly the brain. Evidence from animal and human studies indicates that the embryo and foetus are more sensitive to methylmercury than adults.

Other effects associated with methylmercury include damage to other tissues and organs including the lung, cardiovascular system, liver and kidney. In animals, the most sensitive indicator of damage other than CNS effects, are renal effects.

Plant Uptake:

A detailed review of the plant uptake of mercury (primarily inorganic mercury) is presented by The UK (UK EA 2009a). This review considered studies that are based in the uptake of mercury into green vegetables, root vegetables, tuber vegetables, herbaceous fruit, shrub fruit and tree fruit. The review provides recommendations on soil to plant uptake factors that are relevant for these types of produce. The recommendations from this review are summarised below for the range of crops considered:

Produce Group	Plant Uptake Factors (mg/kg produce fresh weight per mg/kg soil) (UK EA 2009a)
Green vegetables	0.0038
Root vegetables	0.0069
Tuber vegetables	0.0042
Tree fruit	0.001

It is noted that the inclusion of home-grown produce results in some double counting of intakes from fruit and vegetable produce (also included in background intakes). To address this, half the intake estimated to be derived from home-ground produce is assumed to be already accounted for in the total background intake (noted below).

No plant uptake values are reviewed or recommended for methyl mercury. UK EA notes that methylated mercury compounds are likely to be more toxic to plants compared with ionic forms, however no specific data are provided. Review by the USEPA (USEPA 1997b) suggests that methyl mercury complexes in soil are available for plant uptake and translocation. In addition, plants have some mercury methylation ability and hence the percentage of methyl mercury in plants may not originate from methyl mercury uptake from soil. Due to the level of uncertainty involved in the estimation of plant uptake of methyl mercury from soil, including the potential for phytotoxicity, it is expected that the conservative approach to the consideration of intakes from dietary sources adequately addresses potential intakes that may be derived from the consumption of 10% home grown produce.

Intakes from Other Sources – Background

For inorganic and elemental mercury, review of current information from Australia indicates the following:

- Mercury levels are reported in the 25th Australian Total Diet Survey (FSANZ 2019). Mean dietary intakes of total mercury (which includes organic mercury in seafood) ranged from 0.16 to 0.38 µg/kg/day for toddlers (aged 2-5 years). For adults, intakes from food comprise between 0.06 and 0.16 µg/kg/day. For inorganic mercury intakes range from 0.027 to 0.3 µg/kg/day for toddlers (aged 2-5 years), and 0.008 to 0.14 µg/kg/day for adults.
- Typical concentrations of mercury reported in the ADWG (NHMRC 2011 updated 2022) are less than 0.0001 mg/L, resulting in an intake (1 L/day and body weight of 15.5 kg) by toddlers of 0.0073 µg/kg/day. It is noted that the diet surveys include consumption of water.
- Review of intakes associated with amalgam fillings in Australian children and adults (based on average number of fillings of 0.5 and 8 respectively) provides a reasonable estimate of daily mercury absorption per person of about 0.3 µg for children and 3.5 µg for adults. The estimate for children is expected to be conservative as mercury dental amalgams is declining with advice provided to minimise use in children and pregnant and breastfeeding women.
- Based on the above, background intakes by young children may be up to 0.4 µg/kg/day from oral intakes (dietary, dental and water). These intakes comprise approximately 60% of the recommended oral TRV. Adult intakes may comprise up to approximately 30% of the oral TRV. These are higher than intakes of 0.1 µg/kg/day from RIVM (Baars et al. 2001), 0.037 µg/kg/day from the UK ((UK EA 2009a), for a 20kg child) and 0.05 µg/kg/day for a child and 0.065 µg/kg/day for an adult from New Zealand.
- Levels of inorganic mercury in air are not available for Australia with estimates from the WHO (WHO 2003) for mercury in air ranging from 2 ng/m³ (rural) to 10 to 20 ng/m³ (urban areas) with no indication on speciation between elemental and inorganic. Where elemental mercury is measured levels are low approximately 1 to 3 ng/m³ (EU 2002), which is negligible when compared with the adopted TRV. Worst-case modelling of outdoor air concentrations of elemental mercury indicates levels should be approximately 100 times lower than this measured value (WHO 2003). Hence for this assessment background intakes are assumed to be negligible.

For methyl mercury review of current information from Australia indicates the following:

- Methyl mercury levels are reported in the 25th Australian Total Diet Survey (FSANZ 2019). Dietary intakes of methyl mercury ranged up to 0.24 µg/kg/day for toddlers (aged 2 years) and 0.09 µg/kg/day for adults.
- The most recent review of methyl mercury by JECFA (WHO 2004b) included a review of estimated dietary intakes from a number of countries. The review references previous total diet surveys (from 1992 and 1995) and indicates that the mean intake of methyl mercury for the population is approximately 0.7 µg/kg/week. It is noted that the 95th percentile intake estimated exceeds the recommended PTWI adopted by JECFA (WHO 2004b). This is a conservative estimate, but it suggests intakes may be a significant proportion of the recommended PTWI.
- Reviews of background intakes of methyl mercury by the UK and RIVM (Baars et al. 2001) suggest intakes ranging from 8% to 20% of the adopted TDI (similar to the recommended

TRV). Data from Australia suggests intakes may be higher and hence a value of 80% is recommended to address the potential for a significant proportion of the recommended oral TRV to be derived from background intakes.

It is noted that the potential for intakes in excess of the recommended oral TRV may occur in populations with high intakes of seafood. This may need to be considered on a site-specific basis.

Classification

The International Agency for Research on Cancer (IARC) has classified methyl mercury as Group 2B: possibly carcinogenic to humans. IARC has classified metallic mercury and inorganic mercury compounds as Group 3: not classifiable.

It is noted that the USEPA has classified methyl mercury as Class C: possible human carcinogen. In addition, the USEPA has classified mercuric chloride as Group C: possible human carcinogen based on increased incidence of squamous cell papillomas of the forestomach and marginally increased incidence of thyroid follicular cell adenomas and carcinomas from long term oral studies in rats.

Identification of Toxicity Reference Values

Inorganic and elemental mercury

Most information on the toxicity of inorganic mercury compounds comes from studies of mercuric chloride. As the water solubility and bioavailability of many other inorganic compounds, notably mercurous compounds, are much less than those of mercuric chloride, such compounds are likely to be less toxic. These issues should be considered further in a site-specific assessment, where relevant.

Carcinogenicity studies in experimental animals are available for mercuric chloride where no carcinogenic effect was observed in mice or female rats, however marginal increases in the incidence of thyroid follicular adenomas and carcinomas and forestomach papillomas were observed in male rats exposed orally. Mercuric chloride binds to DNA and induces clastogenic effects *in vitro*; *in vivo*, both positive and negative results have been reported, without a clear-cut explanation of the discrepancy. The overall weight of evidence is that mercuric chloride possesses weak genotoxic activity but does not cause point mutations (WHO 2017). The USEPA (USEPA IRIS) evaluation of mercuric chloride indicates that a linear low-dose extrapolation is not appropriate as kidney tumour seen in mice occurred at doses that were also nephrotoxic.

On this basis a threshold approach is considered appropriate based on the most sensitive effect associated with mercury exposure. The following threshold values are available from Level 1 Australian and International sources:

Source	Value	Basis/Comments
Inorganic mercury		
ADWG (NHMRC 2011 updated 2022)	Guideline established on the basis of methyl mercury	
FSANZ (FSANZ 2003)	PTWI = 0.003 mg/kg/week	Value for total mercury referenced from JECFA 1989, based on methyl mercury.
New Zealand (MfE 2011a)	TDI = 0.002 mg/kg/day	MfE adopted the TDI from the RIVM evaluation (noted below), which is also consistent with the TDI adopted in the derivation of the WHO drinking water guideline.

Source	Value	Basis/Comments
MfE (MfE 2002)	Air GV = 0.00033 mg/m ³	Air guideline value (as annual average) for inorganic mercury based on occupational health standards for inorganic mercury and the US values.
WHO DWG (WHO 2017)	TDI = 0.002 mg/kg/day	The current WHO DWG (consistent with the review conducted in 2003 and 2011) has derived a guideline of 0.006 mg/L based on a TDI of 0.002 mg/kg/day derived from a NOAEL of 0.23 mg/day associated with kidney effects in a 26-week study in rats and an uncertainty factor of 100. A similar TDI was derived on the basis of a LOAEL of 1.9 mg/kg/day associated with renal effects in a 2-year rat study and an uncertainty factor of 1000.
JECFA (JECFA 2011)	PTWI = 0.004 mg/kg (equivalent to PTDI = 0.0006 mg/kg/day)	Review of mercury by JECFA indicated that the predominant form of mercury indoors, other than fish and shellfish, is inorganic mercury and while data on speciation is limited the toxicological database on mercury (II) chloride was relevant for establishing a PTWI for foodborne inorganic mercury. A PTWI was established on the basis of a benchmark dose approach, where the BMDL ₁₀ of 0.06 mg/kg/day for relative kidney weight increases in male rats was considered as the point of departure. A 100-fold uncertainty factor was applied.
WHO (WHO 2003)	TDI = 0.002 mg/kg/day TC = 0.0002 mg/m ³	TDI derived as noted in the DWG above. A TC in air was also derived for elemental mercury in air (0.0002 mg/m ³) associated with CNS effects in workers exposed to elemental mercury. The relevance of this value to inorganic compounds is not discussed. The TC is considered relevant to inhalation exposures to elemental vapour.
UK (UK EA 2009a)	TDI = 0.002 mg/kg/day TC = 0.0002 mg/m ³	TDI referenced from the WHO and WHO DWG (WHO 2017). Inhalation value (converted to a dose by the UK) is based on the WHO value and has been assumed to be relevant to inorganic mercury in air.
RIVM (Baars et al. 2001)	TDI = 0.002 mg/kg/day	Derived on the same basis as WHO. No inhalation value is derived for inorganic mercury.
ATSDR (ATSDR 1999b)	No chronic MRLs derived	No chronic duration MRLs have been derived for inorganic mercury. An intermediate duration oral MRL of 0.002 mg/kg/day was derived.
USEPA (USEPA IRIS)	RfD = 0.0003 mg/kg/day	RfD (last reviewed in 1995) based on a LOAEL of 0.226 mg/kg/day associated with autoimmune effects in a subchronic rat feeding study and an uncertainty factor of 1000. No RfC is available for inorganic mercury.
Elemental mercury		
WHO (WHO 2000b)	TC = 0.001 mg/m ³	TC or guideline value derived on the basis of a LOAEL derived from occupational studies on elemental vapour. The WHO note that this value is expected to be adequately protective of renal effects associated with exposure to inorganic mercury.
WHO (WHO 2003)	TC = 0.0002 mg/m³	A TC in air was also derived for elemental mercury in air (0.0002 mg/m ³) associated with CNS effects in workers exposed to elemental mercury. The relevance of this value to inorganic compounds is not discussed. The TC is considered relevant to inhalation exposures to elemental vapour.
ATSDR (ATSDR 1999b)	TC = 0.0002 mg/m ³	A chronic inhalation MRL was derived based on a LOAEL of 0.026 mg/m ³ associated with effects on the nervous system in an occupational study. The value was adjusted for continual exposure with a 30 fold uncertainty factor adopted.
RIVM (Baars et al. 2001)	TC = 0.0002 mg/m ³	RIVM adopted the same air criteria as ATSDR (1999)
UK (UK EA 2009a)	TC = 0.0002 mg/m ³	TDI referenced from the WHO and WHO DWG (WHO 2017). Inhalation value (converted to a dose by the UK) is based on the WHO value and has been assumed to be relevant to inorganic mercury in air.
USEPA (USEPA IRIS) (OEHHA)	RfC = 0.0003 mg/m ³	USEPA review (conducted in 1995) utilised a LOAEL of 0.025 mg/m ³ for CNS effects from a number of occupational studies some of which included extrapolation from blood levels and biological monitoring. The value was adjusted for continual exposure and a 30 fold uncertainty factor was applied.

The PTWI derived for inorganic mercury available from JECFA (JECFA 2011) is considered to provide the most current review of the available studies in relation to exposure to inorganic mercury.

Inhalation values for mercury are derived from occupational studies associated with elemental mercury vapour. While the WHO (WHO 2000b) provides some comment on the potential relevance of the guideline value derived to the assessment of inorganic mercury in air, the available toxicity data does not specifically relate to the inhalation of inorganic mercury compounds likely to be present in soil contamination. The UK (UK EA 2009a) has adopted the lower guideline value (TC) for the assessment of inorganic mercury.

For the assessment of exposure to elemental mercury the value from WHO (WHO 2003) is of most relevance.

Methyl mercury

Long-term exposure to methyl mercury has induced renal tumours in mice, but only at doses at which significant nephropathy was also evident (WHO 2004b). Review by the USEPA (USEPA IRIS) concluded that methyl mercury is not a potent genotoxic agent and that methyl mercury induced tumours in mice were likely to have a non-genotoxic mode of action. On this basis a threshold approach is considered appropriate based on the most sensitive effect associated with methyl mercury exposure. The following threshold values are available from Level 1 Australian and International sources:

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 updated 2022)	TDI = 0.00047 mg/kg/day	Current ADWG derived a guideline of 0.001 mg/L on the basis of a PTWI of 0.0033 mg/kg derived from the older JECFA evaluation (see below).
FSANZ (FSANZ 2019)	PTWI = 0.0016 mg/kg/week (PTDI = 0.00023 mg/kg/day)	Value for methyl mercury from JECFA (WHO 2004b) review.
International		
WHO DWG (WHO 2017)	Not established for methyl mercury	The current WHO DWG has derived a guideline for inorganic mercury in drinking water only.
MfE (MfE 2002)	Air GV = 0.00013 mg/m ³	Air guideline value (as annual average) for organic mercury, scaled from the value adopted for inorganic mercury.
JECFA (WHO 2004b)	PTWI = 0.0016 mg/kg/week (PTDI = 0.00023 mg/kg/day)	The most current evaluation by JECFA derived a PTWI of 0.0016 mg/kg based on a steady state intake of 1.5 µg/kg/day (from review of mercury in hair and blood, a benchmark dose approach to assess the relationship between maternal hair concentrations and foetal neurotoxicity and a pharmacokinetic model). This intake is estimated to represent the exposure that would be expected to have no appreciable adverse effects on children and applying an uncertainty factor of 6.4. The PTWI was considered to be sufficient to protect developing foetuses, the most sensitive subpopulation identified. The previous evaluations by JECFA identified a PTWI of 0.0033 mg/kg methyl mercury based on review of oral intakes of mercury and hair and blood mercury levels. Subsequent reviewed of the PTWI by JECFA in 2000 identified that the value may not be adequately protective of foetuses and infants who are more sensitive than adults.
UK (UK EA 2009a)	PTWI = 0.0016 mg/kg/week (PTDI = 0.00023 mg/kg/day)	Value adopted is referenced from JECFA for all routes of exposure.
RIVM (Baars et al. 2001)	TDI = 0.0001 mg/kg/day	Derived on the basis of a NOAEL of 1.3 µg/kg/day for developmental effects in humans (and hair concentrations) and an uncertainty factor of 10.

Source	Value	Basis/Comments
ATSDR (ATSDR 1999b)	MRL = 0.0003 mg/kg/day	Chronic oral MRL derived on the basis of a NOAEL of 0.0013 mg/kg/day (adjusted) associated with CNS effects in humans (and hair concentrations) and an uncertainty factor of 4.5.
USEPA (USEPA IRIS)	RfD = 0.0001 mg/kg/day	RfD (last reviewed in 2001) based on a BMD of 0.0009 to 0.0015 mg/kg/day (adjusted) based on CNS effects in humans (and blood concentrations) and an uncertainty factor of 10.

The PTWI derived for methyl mercury from JECFA (WHO 2004b) is considered to be based on the most recent detailed review of available studies in relation to exposure to methyl mercury. The TRV established by JECFA is within the same range of values previously established by the USEPA and ATSDR and is recommended for use in the derivation of a soil HIL for methyl mercury. No dermal or inhalation specific data are available and hence the PTWI is recommended to be adopted for all routes of exposure.

Recommendation

On the basis of the discussion above the following toxicity reference values (TRVs) have been adopted for mercury:

Inorganic mercury:

- Oral TRV (TRV_o) = 0.0006 mg/kg/day (JECFA 2011) for oral and dermal routes of exposure
- Gastrointestinal absorption factor (GAF) = 0.07 (USEPA 2004)
- Dermal absorption factor (DAF) = 0.001 (or 0.1%) (USEPA 1995)
- Inhalation TRV (TRV_i) = 0.0002 mg/m³ (WHO 2003) – note this is for elemental mercury
- Background intakes from other sources (as % of TRV):
 - BI_o = 60% for oral and dermal intakes
 - BI_i = 0% for inhalation
- Uptake into edible produce may occur and should be evaluated where relevant.

Elemental mercury:

- Inhalation TRV (TRV_i) = 0.0002 mg/m³ (WHO 2003)
- Background intakes from other sources (as % of TRV): negligible

Methyl mercury:

- Oral TRV (TRV_o) = 0.00023 mg/kg/day (WHO 2004b) for all routes of exposure
- Dermal absorption factor (DAF) = 0.001 (or 0.1%) (as for inorganic mercury)
- Background intakes from other sources (as % of TRV):
 - BI_o = 80% for oral and dermal intakes

Nickel

General

Several comprehensive reviews of nickel in the environment and toxicity to humans are available (ATSDR 2005; UK EA 2009b; WHO 1991b).

Nickel is a silvery white metal that is stable under environmental conditions. It occurs naturally in the earth's crust. It is the 24th most abundant element and is primarily found as oxides or sulfides (ATSDR 2005). Nickel is extracted from mined ore via pyro- and hydrometallurgical refining processes. Most nickel is used for the production of stainless steel and other nickel alloys with high corrosion and temperature resistance. The primary sources of nickel emissions into the atmosphere

are the combustion of coal and oil for heat or power generation, the incineration of waste and sewage sludge, nickel mining and primary production, steel manufacture, electroplating and cement manufacturing (WHO 1991b).

The chemistry of nickel is complex, and the toxicological properties of the various compounds depend on physicochemical characteristics, surface chemistry, solubility, geological history. Hence it is important that any site specific assessment of nickel consider these issues.

Background

Review of current information from Australia indicates the following:

- Dietary intakes of nickel have been assessed in the 22nd Australian Total Diet Survey (FSANZ 2008), where mean intakes reported for children aged 2-3 years was reported to be 83-91 µg/day, or 6.2 to 6.9 µg/kg/day. Estimates provided by (ATSDR 2005) and UK (UK EA 2009b) suggest that adult intakes from food are 69-162 µg/day (up to 2.3 µg/kg/day) and 130 µg/day (1.9 µg/kg/day) respectively. Intakes for children (ATSDR 2005) range from 6.9 µg/kg/day (6-11 months old) to 9.5 µg/kg/day (children aged less than 18).
- Typical concentrations of nickel reported in the ADWG (NHMRC 2011 updated 2022) are less than 0.01 mg/L. resulting in an intake (1 L/day and body weight of 15.5 kg) by toddlers of 0.6 µg/kg/day.
- Based on intakes estimated from Australian data, background intakes by young children are approximately 7 µg/kg/day, up to 60% of the recommended oral TRV.
- Nickel was reported in ambient air data collected in (NSW DEC 2003) where concentrations (24-hour averages) in urban, regional and industrial areas assessed ranged from 0.86 to 20 ng/m³ (average of 3.5 ng/m³). Typical background concentrations in air have been reported by to be from 0.3 to 4.5 ng/m³, consistent with that reported by (NSW DEC 2003). These background concentrations comprise (based on average concentrations) approximately 7% of the recommended TC. A conservative background of 10% of the recommended inhalation TRV has been assumed for intakes from air.

Health effects

The following is noted in relation to the toxicity of nickel (UK EA 2009b):

“Nickel is a potent skin sensitiser, and as many as 1–4% of men and 8–20% of women in the general population may be nickel-sensitive. The threshold for initial induction of sensitisation is unknown. Oral ingestion of nickel can also produce skin sensitisation reactions in individuals who have been previously sensitised to nickel. Sensitised individuals have experienced skin reactions following ingestion of about 0.5–0.7 mg of nickel. In a volunteer study, an acute oral dose of 12 µg kg⁻¹ bw on an empty stomach induced hand eczema in women with an established skin sensitivity to nickel.

The other main concern for oral exposure to nickel is its developmental toxicity potential, which has been observed in experimental animal studies. In a two-generation rat study, a wide range of developmental effects were observed at doses of 2.2 mg nickel kg⁻¹ bw day⁻¹.

The respiratory system is the primary site of toxicity of inhaled nickel in both humans and laboratory animals. Effects seen in occupationally exposed workers include chronic bronchitis, emphysema, reduced vital capacity and asthma. Respiratory effects were seen in rodents chronically exposed to nickel sulphate at 60 µg m⁻³.

There is adequate evidence from occupational studies that soluble nickel salts and the mixture of sulphides and oxides present in nickel refinery dust are also carcinogenic to the lungs and/or nasal tissues in humans. Lifetime inhalation of nickel subsulphide or nickel oxide also led to lung tumours in rats, while a similar study on metallic nickel found increases in adrenal gland tumours but not respiratory tract cancers. Nickel sulphate showed no carcinogenic activity in lifetime studies in rats or mice exposed by inhalation, or in rats treated by gavage or via the diet. There is some evidence that occupational exposure to nickel compounds can induce chromosome aberrations, and nickel salts (especially the sulphate and chloride) have shown activity in a range of in vivo and in vitro screening tests for genotoxicity. Although the evidence is not clear, several expert groups have therefore assumed that the genotoxic character displayed by nickel could play a role in tumour development and, consequently, there might not be a threshold for the carcinogenicity of inhaled nickel. Other expert groups, however, have concluded that there will be a threshold. For oral exposure, nickel compounds tested thus far have shown no carcinogenic potential.”

Classification

(IARC 2012a) classified nickel compounds as Group 1: carcinogenic to humans. The IARC working group noted that the overall evaluation of nickel compounds as a group was undertaken on the basis of the combined results of epidemiological studies, carcinogenicity studies in experimental animals, and several types of other relevant data supported by the underlying assumption that nickel compounds can generate nickel ions at critical sites in their target cells.

It is noted that the USEPA has classified nickel refinery dust as Group A: human carcinogen.

Review of Available Values/Information

The toxicity of nickel is complex and appears to differ via the different routes of exposure and hence the following addresses oral exposures separately from inhalation exposures.

Oral

Review in (WHO 2011b) concluded that there was no substantial evidence that nickel compounds may produce cancers other than in the lung or nose in occupationally exposed persons. Limited animal studies on carcinogenic effects after oral exposures to nickel compounds did not show any significant increase in tumours. Review by the UK noted that while not all expert groups (WHO, USEPA, EU) have explicitly concluded that there is no carcinogenic concern from ingested nickel, none of those evaluating oral exposure concluded that a non-threshold approach should be undertaken. Hence the assessment of oral intakes on the basis of a threshold approach is reasonable. The following quantitative values are available from Level 1 Australian and International sources:

Source	Value	Basis/Comments
ADWG (NHMRC 2011 updated 2022)	TDI = 0.005 mg/kg/day	The ADWG derived a health based guideline of 0.02 mg/L based on NOEL of 5 mg/kg/day associated with organ-to-body-weight ratios in a 2-year rat study and an uncertainty factor of 1000. An additional factor of 10 was not included to address carcinogenicity as this was only relevant for inhalation exposures, not oral exposures.
WHO DWG (WHO 2017)	TDI = 0.012 mg/kg/day	The current WHO DWG, based on a review conducted in 2005, derived a guideline of 0.07 mg/L based on a TDI of 0.012 mg/kg/day derived from a LOAEL of 0.012 mg/day established from a study associated with hand eczema in nickel-sensitised

Source	Value	Basis/Comments
		volunteers who had fasted prior to administration of the nickel salt ((Nielsen et al. 1999)). This study (using fasted patients) was considered conservative and an uncertainty factor of 1 was adopted. The review also noted that a general guideline value of 0.13 mg/L could also be derived from a TDI of 0.022 mg/kg/day on the basis of a two-generation study in rats where a NOAEL of 2.2 mg/kg/day could be determined for all end-points studied and an uncertainty factor of 100.
RIVM (Baars et al. 2001)	TDI = 0.05 mg/kg/day	TDI derived on the basis of a NOAEL of 5 mg/kg/day (same study considered in the ADWG) and an uncertainty factor of 100.
UK EA (UK EA 2009b))	TDI = 0.012 mg/kg/day	Adopted the WHO evaluation presented in the WHO DWG.
TERA (TERA 1999)	RfD = 0.008 mg/kg/day	RfD derived for soluble nickel salts on the basis of a LOAEL of 7.6 mg/kg/day associated with kidney effects in rats and an uncertainty factor of 1000. The value derived was in addition to the diet rather than total intake.
ATSDR (ATSDR 2005)	No oral MRL derived	
USEPA (IRIS 2012)	RfD = 0.02 mg/kg/day	RfD (last reviewed in 1991) based on a NOAEL of 5 mg/kg/day (same study as considered in the ADWG) and an uncertainty factor of 300.

Inhalation

Inhalation exposures to nickel are complex, with the toxicity dependent on the form of nickel present. The most recent review of nickel toxicity by UK Environment Agency indicates the following with respect to the consideration of inhalation exposures:

- Nickel and compounds are established carcinogens via the inhalation route with tumours of the respiratory tract a consequence of occupational exposure to both soluble and insoluble nickel salts.
- Nickel compounds are generally considered to be genotoxic; however, the mechanism of action associated is not well understood. The lack of understanding has resulted in a conservative approach that genotoxicity is critical in the development of tumours and that a non-threshold may be appropriate.
- Non-threshold assessments of inhalation cancer risk have relied on occupational studies to derive a quantitative value (unit risk). These occupational studies relate to specific nickel compounds in the occupational environment including nickel subsulfide , nickel sulfate and nickel refinery dusts (USEPA).
- The WHO (WHO 1991b) notes that very high concentrations of nickel are required to produce teratogenic and genotoxic effects.
- Review by RIVM (Baars et al. 2001) suggested the mechanism of action suggests a cytotoxic effect and that a threshold was appropriate for inhalation exposure to nickel. Review by UK Environment Agency (UK EA 2009b) also suggested a non-genotoxic threshold mechanism of action and that a threshold can be considered.
- A threshold value can be adopted for inhalation exposure that is protective of both carcinogenic and non-carcinogenic effects. However, it is noted that the assessment of carcinogenic issues relies on the non-threshold values available and acceptance of a 1 in 100,000 excess lifetime cancer risk.

Nickel is not volatile and hence inhalation exposures are only relevant for dust intakes. Carcinogenic end points are expected to be of particular importance if they are derived from nickel refinery dust of nickel subsulfide, but dust generated from soil contamination is not likely to be significant and hence the consideration of carcinogenic effects using a non-threshold approach may not be appropriate. It is therefore appropriate to consider intakes on the basis of a threshold approach associated with the most significant end point which includes both carcinogenic and non-carcinogenic effects. These issues were considered by UK Environment Agency (UK EA 2009b), where a threshold value was recommended that was considered protective of both carcinogenic and non carcinogenic effects.

The following quantitative threshold values (including guideline values derived to be protective of carcinogenic effects) are available for the assessment of inhalation exposures from Australian and International sources:

Source	Value	Basis/Comments
Australian – No guidelines derived		
International		
WHO (WHO 2000b)	GV = 0.025 $\mu\text{g}/\text{m}^3$	Review by WHO established a range of air guideline values for nickel based on a non-threshold approach with a unit risk derived from occupational studies associated with nickel subsulfate. It has been assumed that the nickel ion is the active agent in the occupational studies and therefore the studies are relevant to all nickel exposures. The guideline value noted here is based on an excess lifetime cancer risk of 1 in 100 000.
TCEQ (TCEQ 2017a)	Acute ReV = 1.1 $\mu\text{g}/\text{m}^3$ Chronic ReV = 0.23 $\mu\text{g}/\text{m}^3$ Carcinogenic ReV = 0.059 $\mu\text{g}/\text{m}^3$	Acute inhalation value based on bronchial constriction in human volunteers with occupational asthma, and application of 30 fold uncertainty factor. Chronic air guidelines based on chronic lung inflammation and associated lesions in rats and a 30 fold uncertainty factor. Carcinogenic values based on non-threshold approach (based on UR = $1.7 (\mu\text{g}/\text{m}^3)^{-1}$) for lung cancer effects in industrial workers and 1 in 100,000 risk. TCEQ values are based on studies related to nickel sulfate, which is the soluble form of nickel, which is more toxic than insoluble forms. It was a science policy decision to use this as a surrogate for all inorganic forms of nickel.
Health Canada (Health Canada 1994)	TC = 0.0035 $\mu\text{g}/\text{m}^3$ TC05 = 0.07 mg/m^3	Tolerable concentration (TC) derived on the basis of a threshold approach from a LOAEC (HEC) of 0.0035 mg/m^3 associated with respiratory effects from nickel sulfate in rats, and an uncertainty factor of 1000. Health Canada also derived a tumorigenic concentration of 5%, TC05, based on epidemiology studies of exposed workers at two nickel refineries (based on nickel sulphate and nickel chloride), and derived from the non-threshold dose-response curves.
RIVM (Baars et al. 2001)	TC = 0.05 $\mu\text{g}/\text{m}^3$	Tolerable concentration (TC) derived on the basis of a threshold approach from a NOAEC (HEC) of 0.005 mg/m^3 associated with respiratory effects in rats, and an uncertainty factor of 100.
UK Air Quality Standards (UK Air Quality Standards 2010)	TC = 0.02 $\mu\text{g}/\text{m}^3$	TC derived assuming a threshold approach is appropriate, based on a LOAEL of 0.02 mg/m^3 associated with respiratory tract tumours in occupational nickel exposures, and an uncertainty factor of 1000. TC derived is similar to but slightly lower than that derived on the basis of inflammatory response in experimental animals.
UK EA (UK EA 2009b)	TC = 0.02 $\mu\text{g}/\text{m}^3$	Adopted evaluation of EPAQS, noting the value derived is protective of carcinogenic and non-carcinogenic effects.
OEHHA (OEHHA 2009)	Chronic REL = 0.014 $\mu\text{g}/\text{m}^3$	Chronic inhalation reference exposure level (REL) for nickel and nickel compounds (except nickel oxide where a higher REL is derived) based on a NOAEL (HEC) of 0.0016 mg/m^3 associated with respiratory/lung effects in a 104-week rat study, and an uncertainty factor of 30. OEHHA also provide a non-threshold unit risk for nickel and compounds.

Source	Value	Basis/Comments
TERA (TERA 1999)	RfC = 0.2 $\mu\text{g}/\text{m}^3$	RfC derived on the basis of a benchmark approach using a BMCL10 (HEC) of 0.0017 mg/m^3 associated with lung fibrosis from soluble nickel salts in a rat study and an uncertainty factor of 10. This is the same study as considered by the ATSDR.
ATSDR (ATSDR 2005)	Inhalation MRL = 0.09 $\mu\text{g}/\text{m}^3$	Chronic inhalation MRL derived on the basis of a NOAEL (HEC) of 0.0027 mg/m^3 associated with lung effects in rats, and an uncertainty factor of 30.
USEPA IRIS (USEPA)	GV = 0.04 $\mu\text{g}/\text{m}^3$	Review by the USEPA (last reviewed in 1991) established a range of air guideline values for nickel based on a non-threshold approach with a unit risk derived from occupational studies associated with nickel refinery dust. The guideline value noted here is based on an excess lifetime cancer risk of 1 in 100 000.

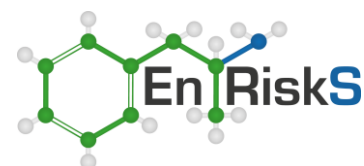
Adopted toxicity reference values

With respect to oral exposures, the more recent review by WHO (WHO 2017) is considered appropriate (and most current) and adequately protective of the most critical health effects. The threshold value recommended is considered adequately protective of hypersensitivity responses that may be associated with oral (and dermal) exposures.

With respect to inhalation exposures a number of evaluations are available that consider LOAELs/NOAELs that are similar, and also address carcinogenicity, with the application of different uncertainty factors. It is recommended that the guideline value (lower value protective of carcinogenic effects) provided by TCEQ (TCEQ 2017a) be adopted (which is protective of adverse health effects including carcinogenicity at an excess lifetime cancer risk level of 1 in 100 000).

On the basis of the discussion above, the following toxicity reference values (TRVs) have been adopted for nickel:

- Oral TRV (TRV_o) = 0.012 $\text{mg}/\text{kg}/\text{day}$ (WHO 2017) for oral and dermal routes of exposure
- Inhalation TRV (TRV_i) = 0.000059 mg/m^3 (TCEQ 2017a)
- Background intakes from other sources (as % of TRV) = 60% for oral and dermal intakes and 10% for inhalation intakes.



Appendix C Methodology and assumptions for quantification of inhalation and multi-pathway exposures

C1 Introduction

This appendix presents the methodology and assumptions adopted in the calculation of risk related to the assessment of chronic risks via inhalation or other pathways that may occur following deposition of chemical substances that are persistent.

C2 Quantification of inhalation exposure

Intakes via inhalation have been assessed on the basis of the inhalation guidance available from the USEPA and recommended for use in the ASC NEPM and enHealth (enHealth 2012a; NEPC 1999 amended 2013c; USEPA 2009).

This guidance requires the calculation of an exposure concentration which is based on the concentration in air and the time/duration spent in the area of impact. It is not dependent on age or body weight. The following equation outlines the calculation of an inhalation exposure concentration, and **Table C1** provides details on the assumptions adopted in this assessment:

$$\text{Inhalation Exposure Concentration} = C_{\text{air}} \times \frac{\text{ET} \times \text{FI} \times \text{RF} \times \text{EF} \times \text{ED}}{\text{AT}} \quad (\text{mg/m}^3)$$

Table C1: Inhalation exposure assumptions

Parameter		Value adopted	Basis
C _{air}	Concentration of chemical substance in air (mg/m ³)	Modelled for the Project, adopting the maximum predicted anywhere (all grid receptors) and the maximum from all discrete receptors	Calculations undertaken on the basis of the maximum predicted impacts
FI	Fraction inhaled from site	100%	All exposures occur at the same location
RF	Dust lung retention factor (unitless)	0.375 for pollutants bound to particles 1 for gases	Percentage of respirable dust as PM ₁₀ that is small enough to reach and be retained in the lungs (NEPC 1999 amended 2013c)
ET	Exposure time (dependent on activity) (hours/day)	Residents = 24 hours/day Workers = 10 hours/day	Assume residents exposed at the maximum location all day, every day of the year
EF	Exposure frequency (days/year)	Residents = 365 days Workers = 240 days	Assume workers exposed all day, every work day
ED	Exposure duration (years)	Residents = 35 years Workers = 30 years	Duration of residency and at a workplace as per enHealth (enHealth 2012b)
AT	Averaging time (hours)	Threshold = ED x 365 days/year x 24 hours/day Non-threshold = 70 years x 365 days/year x 24 hours/day	As per enHealth (enHealth 2012a) guidance

C3 Multiple pathway exposures

C3.1 Ingestion and dermal absorption

Chemical substances attached to particles that are deposited on the ground have the potential to be ingested either directly through accidental consumption of soil or indirectly through food grown or raised in the soil (fruit and vegetables, eggs, beef and milk) that is subsequently consumed.

The assessment of the potential ingestion of chemical substances has been undertaken using the approach presented by enHealth and the USEPA (enHealth 2012a; USEPA 1989). This approach is presented in the following equation, and parameters adopted in this assessment are presented in

Table C2:

$$\text{Daily chemical intake}_{\text{ingestion}} = C_m \times \frac{IR_m \times FI \times B \times CF \times EF \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Chemical substances attached to particles that are deposited on the ground have the potential to be absorbed through the skin when skin comes in contact with soil or dust.

The assessment of the potential dermal absorption of chemical substances has been generally undertaken using the approach presented by the USEPA (USEPA 1989, 2004). The USEPA define a simple approach to the evaluation of dermal absorption associated with soil contact. This is presented in the following equation and parameters adopted in this assessment are presented in

Table C2:

$$\text{Daily chemical intake}_{\text{dermal}} = C_m \times \frac{SA \times AF \times ABS_d \times CF \times EF \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Table C2: Ingestion and dermal exposure assumptions

Parameter	Value adopted		Basis
	Young children	Adults	
C_m	Concentration of chemical substance in media or relevance (soil, fruit and vegetables, eggs, beef or milk) (mg/kg)		Modelled based on deposition of particles to soil – maximum deposition rate for each of the maximum locations
IR_m	Ingestion rate of media		Calculations undertaken on the basis of the maximum predicted impacts relevant to areas where multi-pathway exposures may occur
	Soil (mg/day)	100 mg/day	50 mg/day
	Fruit and vegetables (kg/day)	0.28 kg/day 85% from aboveground crops 16% from root crops	0.4 kg/day 73% from aboveground crops 27% from root crops
	Eggs (kg/day)	0.036 kg/day	0.059 kg/day
			Ingestion rate of outdoor soil and dust (tracked or deposited indoors) as per enHealth (enHealth 2012b) and ASC NEPM (NEPC 1999 amended 2013c)
			Total fruit and vegetable intake per day as per ASC NEPM (NEPC 1999 amended 2013c)
			Ingestion rate of eggs per day – P90 intakes from FSANZ (FSANZ 2017)

Parameter		Value adopted		Basis
		Young children	Adults	
	Beef (kg/day)	0.085 kg/day	0.16 kg/day	Ingestion rate for adults aged 19 years and older (enHealth 2012b), also consistent with P90 intakes from FSANZ (FSANZ 2017), Values for children from FSANZ (2017)
	Milk (kg/day)	1.097 kg/day	1.295 kg/day	Ingestion rate P90 intakes from FSANZ (FSANZ 2017)
FI	Fraction of media ingested derived from impacted media, or fraction of produce consumed each day derived from the property			
	Soil	100%	100%	Assume all soil contact occurs on the one property
	Fruit and vegetables	35%	35%	Rate assumed for rural area (higher than the default of 10% for urban areas)
	Eggs	100%	100%	Assume all eggs consumed are from chickens kept at the site
	Beef	35%	35%	Rate assumed for on farm consumption for rural area
	Milk	100%	100%	Assume all milk consumed each day is from the property
B	Bioavailability or absorption of chemical substance via ingestion	100%	100%	Conservative assumption
SA	Surface area of body exposed to soil per day (cm ² /day)	2,700	6,300	Exposed skin surface area relevant to adults as per ASC NEPM (NEPC 1999 amended 2013c)
AF	Adherence factor, amount of soil that adheres to the skin per unit area which depends on soil properties and area of body (mg/cm ² per event)	0.5	0.5	Default (conservative) value from ASC NEPM (NEPC 1999 amended 2013c)
ABSd	Dermal absorption fraction (unitless)	Chemical specific		Refer to Tables B3 and B4
CF	Conversion factor			
	Soil	1x10 ⁻⁶ to convert mg to kg		Conversion of units relevant to soil ingestion and dermal contact
	Produce	1		No units conversion required for these calculations
BW	Body weight	70	15	As per ASC NEPM (NEPC 1999 amended 2013c)
EF	Exposure frequency (days/year)	365	365	Assume residents exposed every day
ED	Exposure duration (years)	6 years	29 years	Duration of residency as per enHealth (enHealth 2012b) and split between young children and adults as per ASC NEPM
AT	Averaging time (days)	Threshold = ED x 365 days/year Non-threshold = 70 years x 365 days/year		As per enHealth (enHealth 2012a) guidance

C3.2 Calculation of concentrations in various media

Potential Concentrations in Soil

The potential accumulation of persistent and bioaccumulative chemical substances in soil, which may be the result of deposition from a number of air emissions source, can be estimated using a soil accumulation model (OEHHA 2015; Stevens 1991).

The concentration in soil, which may be the result of deposition following emission of persistent chemical substances, can be calculated using the following equation, with assumptions adopted in this assessment presented in **Table C3**.

$$C_s = \frac{DR \cdot [1 - e^{-k \cdot t}]}{d \cdot \rho \cdot k} \cdot 1000 \quad (\text{mg/kg})$$

Table C3: Assumptions adopted to estimate soil concentrations

Parameter		Value adopted		Basis
		Surface soil*	Agricultural soil*	
DR	Particle deposition rate for accidental release (mg/m ² /year)	Modelled for the Project. Adopted deposition rate at relevant maximum location for each scenario		Relevant to areas where multi-pathway exposures may occur
k	Chemical-specific soil-loss constant (1/year) = $\ln(2)/T^{0.5}$	Calculated	Calculated	
T ^{0.5}	Chemical half-life in soil (years)	Chemical specific	Chemical specific	Default values adopted for pollutants considered as per OEHHA (2015)
t	Accumulation time (years)	70 years	70 years	Default value (OEHHA 2015)
d	Soil mixing depth (m)	0.01 m	0.15 m	Default values (OEHHA 2015)
ρ	Soil bulk-density (g/m ³)	1,600,000	1,600,000	Default for fill material (CRC CARE 2011)
1000	Conversion from g to kg	Default conversion of units		

* Surface soil values adopted for the assessment of direct contact exposures. All other exposures including produce and meat/milk intakes utilise soil concentrations calculated for agricultural intakes (OEHHA 2015)

Homegrown fruit and vegetables

Plants may become contaminated with persistent chemical substances via deposition directly onto the plant outer surface and following uptake via the root system. Both mechanisms have been assessed.

The potential concentration of persistent chemical substances that may be present within the plant following atmospheric deposition can be estimated using the following equation (Stevens 1991), with the parameters and assumptions adopted outlined in **Table C4**:

$$C_p = \frac{DR \cdot F \cdot [1 - e^{-k \cdot t}]}{Y \cdot k} \quad (\text{mg/kg plant – wet weight})$$

The potential uptake of persistent chemical substances into edible crops via the roots can be estimated using the following equation (OEHHA 2015; USEPA 2005c), with the parameters and assumptions adopted outlined in **Table C4**:

$$C_{rp} = C_s \cdot RUF \quad (\text{mg/kg plant – wet weight})$$

Table C4: Assumptions adopted to estimate concentration in fruit and vegetables

Parameter		Value adopted	Basis
DR	Particle deposition rate for accidental release (mg/m ² /day)	Modelled for the Project. Adopted maximum deposition rate for residential receptors	Relevant to areas where multi-pathway exposures may occur
F	Fraction for the surface area of plant (unitless) – also, called plant interception fraction	0.051	Relevant to aboveground exposed crops as per Stevens (1991) and OEHHA (OEHHA 2012)
k	Chemical-specific loss constant for particles on plants (1/days) = $\ln(2)/T^{0.5}$	calculated	
T ^{0.5}	Chemical half-life on plant (day)	14 days	Weathering of particulates on plant surfaces does occur and in the absence of measured data, it is generally assumed that organics deposited onto the outer portion of plant surfaces have a weathering half life of 14 days (Stevens, 1991)
t	Deposition time or length of growing season (days)	70 days	Relevant to aboveground crops based on the value relevant to tomatoes, consistent with the value adopted by Stevens (1991)
Y	Crop yield (kg/m ²)	2 kg/m ²	Value for aboveground crops (OEHHA 2015)
Cs	Concentration of pollutant in soil (mg/kg)	Calculated value for agricultural soil	Calculated as described above and assumptions in Table B5
RUF	Root uptake factor (unitless)	Chemical specific value adopted	Root uptake factors from RAIS (RAIS) (soil to wet weight of plant)

Eggs, beef and milk

The concentration of bioaccumulative pollutants in animal products is calculated on the basis of the intakes of these pollutants by the animal (chicken or cow) and the transfer of these pollutants to the edible produce. The approach adopted in this assessment has involved calculation of intakes from pasture, assumed to be grown on the property, and soil.

The concentration (C_P) calculated in eggs, beef or milk is calculated using the following equation (OEHHA 2015), with parameters and assumptions adopted presented in **Table C5**:

$$C_P = (FI \times IR_C \times C + IR_S \times C_s \times B) \times TF_P$$

Table C5: Assumptions adopted to estimate concentration in animal produce

Parameter		Value adopted	Basis
FI	Fraction of grain/crop ingested by animals each day derived from the property (unitless)	100%	Assume all pasture/crops ingested by chickens and cows are grown on the property
IR _c	Ingestion rate of pasture/crops by each animal considered (kg/day)		
	Chickens	0.12 kg/day	Ingestion rate from OEHHA (2015)
	Beef cattle	9 kg/day	Ingestion rate from OEHHA (2015)
	Lactating cattle	22 kg/day	Ingestion rate for lactating cattle from OEHHA (2015)
C	Concentration of pollutant in crops consumed by animals (mg/kg)	Assume equal to that calculated in aboveground produce	Calculated as described above with assumptions in Table B6
IR _s	Ingestion rate of soil by animals each day (kg/day)		
	Chickens	0.01 kg/day	As per OEHHA (2015) and advice from Ag Vic
	Beef cattle	0.45 kg/day	Based on data from OEHHA 2015 (5% total produce intakes from soil from pasture)
	Lactating cattle	1.1 kg/day	Based on data from OEHHA 2015 (5% total produce intakes from soil from pasture)
C _s	Concentration of pollutant in soil (mg/kg)	Calculated value for agricultural soil	Calculated as described above and assumptions in Table B5
B	Bioavailability of soil ingested (unitless)	100%	Conservative assumption
TF _P	Transfer factor for the produce of interest		
	Eggs	Chemical specific	Transfer factors adopted from OEHHA (2015), with the exception of chromium where the value was derived from an earlier OEHHA (OEHHA 2003) evaluation. Values for antimony and tin are the mean values for the transfer of heavy metals into eggs (Leeman, Van Den Berg & Houben 2007)
	Beef	Chemical specific	Transfer factors adopted from OEHHA (2015) and RAIS
	Milk	Chemical specific	Transfer factors adopted from OEHHA (2015) and RAIS

All calculations relevant to the estimation of pollutant concentrations in soil, fruit and vegetables as well as animal products are presented in **Appendix D**.

Rainwater tanks

The concentration in rainwater tanks depends on the deposition rate of dust, the size of the roof, the volume of rainfall each year and how much of the rain that falls onto the roof is captured in the tank. The concentration in rainwater for Project related emissions, which may be used for all household purposes is calculated as follows, where the parameters adopted for this assessment are detailed in **Table C6**:

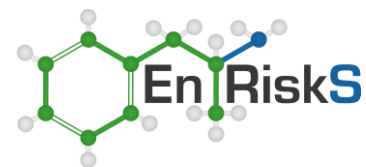
$$C_w = \frac{DM}{VR \times K_d \times \rho}$$

$$VR = \frac{R \times \text{Area} \times Rc}{1000}$$

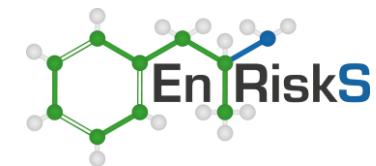
Table C6: Assumptions adopted to estimate concentration in rainwater tank

Parameter		Value adopted	Basis
DM	Mass of dust deposited on the roof each year (mg)	DR x Area	
DR	Particle deposition rate (mg/m ² /year)	Modelled in the Air Quality Assessment for each sensitive location	Relevant to areas where multi-pathway exposures may occur
Area	Area of the roof (m ²)	200	Based on the average roof size for a 4 bedroom house in Australia (refer to Footnote 1)
VR	Volume of water collected from the roof each year	calculated	Equation as above
R	Rainfall each year (mm)	900.5	Average rainfall at Beaudesert Drumley St Met Station for all years of records (2007 – 2023). No first flush device is assumed hence all rainfall is considered
Rc	Runoff coefficient	0.7	Assumes 30% loss in capture of water into the tank (Lizárraga-Mendiola et al. 2015)
1000	Conversion from mm to m		
Kd	Soil-water partition coefficient (cm ³ /g)	Chemical-specific	All values from RAIS (RAIS)
p	Soil bulk density (g/m ³)	0.5	Assumed for loose deposited dust on roof (upper end measured for powders)

1 - <https://www.nedlands.wa.gov.au/sites/default/files/Rainwater%20tank%20factsheet.pdf>



Appendix D Risk calculations



Acute/ short-term exposures (1 Hour Average)

Predicted ground level concentrations and screening assessment - acute exposures (LP2 - maximum capacity)

COPC	Acute air guideline (mg/m ³)	Air Concentration (mg/m ³)				Calculated RI/Hi			
		Grid Maximum Off-site	Maximum for commercial/ industrial locations	Maximum for residential locations	Maximum for Visitor centre	Grid Maximum Off-site	Maximum for commercial/ industrial locations	Maximum for residential locations	Maximum for Visitor centre
Hydrogen chloride (HCl)	0.66	0.03971958	0.03971958	0.01802753	0.02604233	0.0602	0.0602	0.0273	0.0395
Hydrogen fluoride (HF)	0.06	0.00300978	0.00300978	0.00136605	0.00197338	0.0502	0.0502	0.0228	0.0329
Cadmium (Cd)	0.018	0.00002131	0.00002131	0.00000967	0.00001397	0.001184	0.001184	0.000537	0.000776
Antimony (Sb)	0.001	0.00003498	0.00003498	0.00001588	0.00002294	0.0350	0.0350	0.0159	0.0229
Arsenic (As)	0.0099	0.00001347	0.00001347	0.00000611	0.00000883	0.001360	0.001360	0.000617	0.000892
Chromium (Cr VI assumed)	0.0013	0.00004800	0.00004800	0.00002179	0.00003147	0.0369	0.0369	0.0168	0.0242
Cobalt (Co)	0.00069	0.00000992	0.00000992	0.00000450	0.00000650	0.01438	0.01438	0.00653	0.00943
Copper (Cu)	0.1	0.00005343	0.00005343	0.00002425	0.00003503	0.000534	0.000534	0.000243	0.000350
Manganese (Mn)	0.0091	0.00007774	0.00007774	0.00003528	0.00005097	0.00854	0.00854	0.00388	0.00560
Nickel (Ni)	0.0011	0.00004621	0.00004621	0.00002097	0.00003030	0.0420	0.0420	0.0191	0.0275
Vanadium (V)	0.03	0.00000542	0.00000542	0.00000246	0.00000355	0.0001807	0.0001807	0.0000820	0.0001185
Benzene	0.58	0.01805188	0.01805188	0.00819321	0.01183580	0.0311	0.0311	0.0141	0.0204
Formaldehyde	0.05	0.01805188	0.01805188	0.00819321	0.01183580	0.3610	0.3610	0.1639	0.2367
Risk Index with VOCs as Benzene						0.3421	0.3403	0.1546	0.2226
Risk Index with VOCs as Formaldehyde						0.6721	0.6702	0.3043	0.4389



Chronic/ long term exposures



Maximum off-site

Predicted ground level concentrations - chronic exposures - increment

	Air Concentration - annual average (µg/m³)	Air Concentration - annual average (mg/m³)	Cumulative air concentrations (mg/m³)	Exposure pathways							
Key Chemicals	Maximum Off-site	Maximum Off-site	Maximum Off-site	inhalation	soil ingestion	soil - dermal	egg ingestion	fruit and vegetable ingestion	rainwater tank	meat ingestion	milk ingestion
Hydrogen chloride (HCl)	0.01578	0.00001578		✓	x	x	x	x	x	x	x
Hydrogen fluoride (HF)	0.00263	0.0000263		✓	x	x	x	x	x	x	x
Ammonia	0.02630	0.00002630		✓	x	x	x	x	x	x	x
Cadmium (Cd)	0.0000312	0.000000312		✓	✓	✓	x	x	✓	x	x
Thallium (Tl)	0.0000214	0.000000214		✓	✓	✓	x	x	✓	x	x
Mercury (Hg)	0.0000527	0.000000527		✓	✓	✓	x	x	✓	x	x
Antimony (Sb)	0.0000555	0.000000555		✓	✓	✓	x	x	✓	x	x
Arsenic (As)	0.0000243	0.000000243	0.0000371760	✓	✓	✓	x	x	✓	x	x
Lead (Pb)	0.0003169	0.000003169		✓	✓	✓	x	x	✓	x	x
Chromium (Cr VI assumed)	0.0000792	0.000000792	0.0000815410	✓	✓	✓	x	x	✓	x	x
Cobalt (Co)	0.0000197	0.000000197		✓	✓	✓	x	x	✓	x	x
Copper (Cu)	0.0000694	0.000000694	0.0000999580	✓	✓	✓	x	x	✓	x	x
Manganese (Mn)	0.0001186	0.0000001186	0.0001329600	✓	✓	✓	x	x	✓	x	x
Nickel (Ni)	0.0000954	0.000000954	0.0001037200	✓	✓	✓	x	x	✓	x	x
Vanadium (V)	0.0000098	0.000000098		✓	✓	✓	x	x	✓	x	x
Dioxin-like compounds	1.58E-10	1.58E-13		✓	✓	✓	x	x	✓	x	x
PAHs	5.27E-10	5.27E-13		✓	✓	✓	x	x	✓	x	x
PFAS (assumed as PFOS)	2.00E-08	2.00E-11		✓	✓	✓	x	x	✓	x	x
Benzene	0.02630	0.00002630	3.52629969	✓	x	x	x	x	x	x	x
Formaldehyde	0.02630	0.00002630		✓	x	x	x	x	x	x	x

	Deposition Rate - annual average (ng/m²/year)	Deposition Rate - annual average (mg/m²/year)
Key Chemicals	Maximum Off-site	Maximum Off-site
Hydrogen chloride (HCl)	NR	NR
Hydrogen fluoride (HF)	NR	NR
Ammonia	NR	NR
Cadmium (Cd)	138.99	0.0001390
Thallium (Tl)	95.85	0.00009585
Mercury (Hg)	234.84	0.00023484
Antimony (Sb)	247.85	0.00024785
Arsenic (As)	108.18	0.00010818
Lead (Pb)	1410.44	0.00141044
Chromium (Cr VI assumed)	353.29	0.00035329
Cobalt (Co)	87.64	0.00008764
Copper (Cu)	309.47	0.00030947
Manganese (Mn)	528.57	0.00052857
Nickel (Ni)	425.87	0.00042587
Vanadium (V)	43.82	0.00004382
Dioxin-like compounds	7.05E-04	7.05E-10
PAHs	2.35E-03	2.35E-09
PFAS (assumed as PFOS)	8.94E-02	8.94E-08
Benzene	NR	NR
Formaldehyde	NR	NR

Inhalation - gases and particulates

Grid maximum for this facility for residential land use

$$\text{Inhalation Exposure Concentration} = Ca * \frac{ET * FI * EF * ED}{AT} \quad (\text{mg/m}^3) \text{ for gases}$$

$$\text{Inhalation Exposure Concentration} = Ca * \frac{ET * FI * DRF * EF * ED}{AT} \quad (\text{mg/m}^3) \text{ for chemicals attached to particles}$$

Parameters Relevant to Quantification of Community Exposures - Residents		
Exposure Time at Home (ET, hr/day)	24	Assume residents at home or on property 24 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Assume resident at the same property
Dust lung retention factor (unitless)	0.375	Percentage of respirable dust that is small enough to reach and be retained in the lungs (ASC NEPM) - NA for gases
Exposure Frequency - normal conditions (EF, days/yr)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	35	As per ASC NEPM
Averaging Time - NonThreshold (Atc, hours)	613200	ASC NEPM
Averaging Time - Threshold (Atn, hours)	306600	ASC NEPM

It is noted that because particles (and attached chemicals) from an WtE facility are very small, it has been assumed that all particles are small enough to reach down into the lungs - i.e. this factor has not been included in the calculation.

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk	Chronic TC Air	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background)	Estimated Concentration in Air - Maximum anywhere (Ca)	Inhalation Exposure Concentration - NonThreshold	Inhalation Exposure Concentration - Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/m ³) ⁻¹	(mg/m ³)		(mg/m ³)	(mg/m ³)	(mg/m ³)	(mg/m ³)	(unitless)		(unitless)	
Hydrogen chloride (HCl)		2.6E-02	0%	2.6E-02	1.6E-05	7.9E-06	1.6E-05	--		0.000607	2%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	2.6E-06	1.3E-06	2.6E-06	--		0.0000907	0%
Ammonia		3.2E-01	0%	3.2E-01	2.6E-05	1.3E-05	2.6E-05	--		0.0000822	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	3.1E-08	1.6E-08	3.1E-08	--		0.00779	24%
Thallium (Tl)		7.0E-04	10%	6.3E-04	2.1E-08	1.1E-08	2.1E-08	--		0.0000340	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	5.3E-08	2.6E-08	5.3E-08	--		0.0002633	1%
Antimony (Sb)		3.0E-04	0%	3.0E-04	5.6E-08	2.8E-08	5.6E-08	--		0.0001851	1%
Arsenic (As)		6.7E-05	0%	6.7E-05	2.4E-08	1.2E-08	2.4E-08	--		0.000363	1%
Lead (Pb)		5.0E-04	0%	5.0E-04	3.2E-07	1.6E-07	3.2E-07	--		0.000634	2%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	7.9E-08	4.0E-08	7.9E-08	--		0.01843	57%
Cobalt (Co)		1.0E-04	0%	1.0E-04	2.0E-08	9.8E-09	2.0E-08	--		0.0001966	1%
Copper (Cu)		4.9E-01	60%	2.0E-01	6.9E-08	3.5E-08	6.9E-08	--		0.000000354	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	1.2E-07	5.9E-08	1.2E-07	--		0.000988	3%
Nickel (Ni)		5.9E-05	10%	5.3E-05	9.5E-08	4.8E-08	9.5E-08	--		0.001797	6%
Vanadium (V)		1.0E-04	0%	1.0E-04	9.8E-09	4.9E-09	9.8E-09	--		0.0000983	0%
Dioxin-like compounds		8.1E-09	54%	3.7E-09	1.6E-13	7.9E-14	1.6E-13	--		0.0000426	0%
PAHs	6.0E-01	0.0E+00	0%	0.0E+00	5.3E-13	2.6E-13	5.3E-13	1.6E-13	--	--	
PFAS (assumed as PFOS)		7.0E-05	0%	7.0E-05	2.0E-11	1.0E-11	2.0E-11	--		0.000000286	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	2.6E-05	1.3E-05	2.6E-05	7.9E-8		0.000974	3%
Formaldehyde		1.1E-02	30%	7.7E-03	2.6E-05	1.3E-05	2.6E-05	--		0.00342	10%

Risk Index with VOCs as Benzene	7.9E-08	0.0326
Risk Index with VOCs as Formaldehyde	1.6E-13	0.0350

Inhalation - gases and particulates

Grid maximum for this facility for commercial land use

$$\text{Inhalation Exposure Concentration} = Ca * \frac{ET * FI * EF * ED}{AT} \quad (\text{mg/m}^3) \text{ for gases}$$

$$\text{Inhalation Exposure Concentration} = Ca * \frac{ET * FI * DRF * EF * ED}{AT} \quad (\text{mg/m}^3) \text{ for chemicals attached to particles}$$

Parameters Relevant to Quantification of Community Exposures - Commercial/Industrial

Exposure Time at Home (ET, hr/day)	10	Assume workers at work site 10 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Only exposed at the work site
Dust lung retention factor (unitless)	0.375	Percentage of respirable dust that is small enough to reach and be retained in the lungs (ASC NEPM) - NA for gases
Exposure Frequency - normal conditions (EF, days/yr)	240	Days at work (ASC NEPM)
Exposure Duration (ED, years)	30	ASC NEPM
Averaging Time - NonThreshold (Atc, hours)	613200	ASC NEPM
Averaging Time - Threshold (Atn, hours)	262800	ASC NEPM

It is noted that because particles (and attached chemicals) from an W/E facility are very small, it has been assumed that all particles are small enough to reach down into the lungs - i.e. this factor has not been included in the calculation.

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk		
	Inhalation Unit Risk (mg/m ³) ⁻¹	Chronic TC Air (mg/m ³)	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background) (mg/m ³)	Estimated Concentration in Air - Maximum anywhere (Ca) (mg/m ³)	Inhalation Exposure Concentration - NonThreshold (mg/m ³)	Inhalation Exposure Concentration - Threshold (mg/m ³)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)
Hydrogen chloride (HCl)		2.6E-02	0%	2.6E-02	1.6E-05	1.9E-06	4.3E-06	--		0.000166
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	2.6E-06	3.1E-07	7.2E-07	--		0.0000248
Ammonia		3.2E-01	0%	3.2E-01	2.6E-05	3.1E-06	7.2E-06	--		0.0000225
Cadmium (Cd)		5.0E-06	20%	4.0E-06	3.1E-08	3.7E-09	8.5E-09	--		0.002135
Thallium (Tl)		7.0E-04	10%	6.3E-04	2.1E-08	2.5E-09	5.9E-09	--		0.00000932
Mercury (Hg)		2.0E-04	0%	2.0E-04	5.3E-08	6.2E-09	1.4E-08	--		0.0000721
Antimony (Sb)		3.0E-04	0%	3.0E-04	5.6E-08	6.5E-09	1.5E-08	--		0.0000507
Arsenic (As)		6.7E-05	0%	6.7E-05	2.4E-08	2.9E-09	6.7E-09	--		0.0000993
Lead (Pb)		5.0E-04	0%	5.0E-04	3.2E-07	3.7E-08	8.7E-08	--		0.0001737
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	7.9E-08	9.3E-09	2.2E-08	--		0.00505
Cobalt (Co)		1.0E-04	0%	1.0E-04	2.0E-08	2.3E-09	5.4E-09	--		0.0000539
Copper (Cu)		4.9E-01	60%	2.0E-01	6.9E-08	8.1E-09	1.9E-08	--		0.000000970
Manganese (Mn)		1.5E-04	20%	1.2E-04	1.2E-07	1.4E-08	3.2E-08	--		0.000271
Nickel (Ni)		5.9E-05	10%	5.3E-05	9.5E-08	1.1E-08	2.6E-08	--		0.000492
Vanadium (V)		1.0E-04	0%	1.0E-04	9.8E-09	1.2E-09	2.7E-09	--		0.0000269
Dioxin-like compounds		8.1E-09	54%	3.7E-09	1.6E-13	1.9E-14	4.3E-14	--		0.00001167
PAHs	6.0E-01	NA	0%	NA	5.3E-13	6.2E-14	1.4E-13	3.7E-14		0%
PFAS (assumed as PFOS)		7.0E-05	0%	7.0E-05	2.0E-11	2.4E-12	5.5E-12	--		0.000000784
Benzene	6.0E-03	3.0E-02	54%	1.4E-02	2.6E-05	3.1E-06	7.2E-06	1.9E-8		0.000522
Formaldehyde		1.1E-02	30%	7.7E-03	2.6E-05	3.1E-06	7.2E-06	--		0.000936

Risk Index with VOCs as Benzene	1.9E-08	0.00918
Risk Index with VOCs as Formaldehyde	3.7E-14	0.00959

Calculation of Concentrations in Soil

$$C_s = \frac{DR \cdot [1 - e^{-k \cdot t}]}{d \cdot \rho \cdot k} \cdot 1000 \quad (\text{mg/kg}) \quad \text{ref: Stevens B. (1991)}$$

where:

DR= Particle deposition rate (mg/m²/year)

K = Chemical-specific soil-loss constant (1/year) = ln(2)/T_{0.5}

T_{0.5} = Chemical half-life in soil (years)

t = Accumulation time (years)

d = Soil mixing depth (m)

ρ = Soil bulk-density (g/m³)

1000 = Conversion from g to kg

General Parameters		Surface (for direct contact)	Depth (for agricultural pathways)	
Soil bulk density (ρ)	g/m ³	1600000	1600000	Default for fill materials
General mixing depth (d)	m	0.01	0.15	As per OEHHHA (2015) guidance
Duration of deposition (T)	years	70	70	As per OEHHHA (2015) guidance

Chemical-specific Inputs and calculations - maximum anywhere					
Chemical	Half-life in soil years	Loss constant (K) per year	Deposition Rate (DR) mg/m ² /year	Surface Concentration in Soil mg/kg	Agricultural Concentration in Soil mg/kg
Cadmium (Cd)	273973	2.5E-06	1.39E-04	6.1E-04	4.1E-05
Thallium (Tl)	273973	2.5E-06	9.59E-05	4.2E-04	2.8E-05
Mercury (Hg)	273973	2.5E-06	2.35E-04	1.0E-03	6.8E-05
Antimony (Sb)	273973	2.5E-06	2.48E-04	1.1E-03	7.2E-05
Arsenic (As)	273973	2.5E-06	1.08E-04	4.7E-04	3.2E-05
Lead (Pb)	273973	2.5E-06	1.41E-03	6.2E-03	4.1E-04
Chromium (Cr VI assumed)	273973	2.5E-06	3.53E-04	1.5E-03	1.0E-04
Cobalt (Co)	273973	2.5E-06	8.76E-05	3.8E-04	2.6E-05
Copper (Cu)	273973	2.5E-06	3.09E-04	1.4E-03	9.0E-05
Manganese (Mn)	273973	2.5E-06	5.29E-04	2.3E-03	1.5E-04
Nickel (Ni)	273973	2.5E-06	4.26E-04	1.9E-03	1.2E-04
Vanadium (V)	273973	2.5E-06	4.38E-05	1.9E-04	1.3E-05
Dioxin-like compounds	15	4.6E-02	7.05E-10	9.2E-10	6.1E-11
PAHs	1.18	6.9E-02	2.35E-09	2.1E-09	1.4E-10
PFAS (assumed as PFOS)	273973	2.5E-06	8.94E-08	3.9E-07	2.6E-08

Half-life in soil for dioxins: 9-15 years in surface soils; 25-100 years in subsurface soils (ATSDR 1998, DEH 2004)

Half-life in soil for metals, PAHs: OEHHHA 2015

Half-life in soil for PFAS assumed same as metals - i.e. no degradation at all but these chemicals do degrade slowly - this is conservative

Exposure to Chemicals via Incidental Ingestion of Soil - Grid Maximum Off-site

$$\text{Daily Chemical Intake}_{IS} = C_S \cdot \frac{IR_S \cdot FI \cdot CF \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults

Ingestion Rate (IRs, mg/day)	50	ASC NEPM
Fraction Ingested from Source (FI, unitless)	100%	All of daily soil intake occurs from site
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	29	Time at one residence as adult as per ASC NEPM
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	6.1E-04	1.8E-10	4.3E-10	--		1.4E-06	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	4.2E-04	1.2E-10	3.0E-10	--		1.7E-06	7%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.0E-03	3.0E-10	7.3E-10	--		3.1E-06	12%
Antimony (Sb)		8.6E-04		8.6E-04	100%	1.1E-03	3.2E-10	7.7E-10	--		9.0E-07	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	4.7E-04	1.4E-10	3.4E-10	--		3.4E-07	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	6.2E-03	1.8E-09	4.4E-09	--		1.5E-05	60%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	1.5E-03	4.6E-10	1.1E-09	--		1.4E-06	6%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	3.8E-04	1.1E-10	2.7E-10	--		2.4E-07	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.4E-03	4.0E-10	9.7E-10	--		1.7E-08	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	2.3E-03	6.8E-10	1.7E-09	--		2.4E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.9E-03	5.5E-10	1.3E-09	--		2.8E-07	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	1.9E-04	5.7E-11	1.4E-10	--		6.8E-08	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	9.2E-10	2.7E-16	6.5E-16	--		6.2E-07	3%
PAHs	2.3E-01				100%	2.1E-09	6.2E-16	1.5E-15	1.4E-16		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	100%	3.9E-07	1.2E-13	2.8E-13	--		1.7E-08	0%
TOTAL									1.4E-16		0.0000246	

Exposure to Chemicals via Incidental Ingestion of Soil - Grid Maximum Off-site

$$\text{Daily Chemical Intake}_{IS} = C_S \cdot \frac{IR_S \cdot FI \cdot CF \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Young Children

Ingestion Rate (IRs, mg/day)	100	ASC NEPM
Fraction Ingested from Source (FI, unitless)	100%	All of daily soil intake occurs from site
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	6.1E-04	3.5E-10	4.1E-09	--		1.27E-05	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	4.2E-04	2.4E-10	2.8E-09	--		1.55E-05	7%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.0E-03	5.9E-10	6.8E-09	--		2.85E-05	12%
Antimony (Sb)		8.6E-04		8.6E-04	100%	1.1E-03	6.2E-10	7.2E-09	--		8.41E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	4.7E-04	2.7E-10	3.2E-09	--		3.15E-06	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	6.2E-03	3.5E-09	4.1E-08	--		1.37E-04	60%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	1.5E-03	8.8E-10	1.0E-08	--		1.27E-05	6%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	3.8E-04	2.2E-10	2.6E-09	--		2.28E-06	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.4E-03	7.7E-10	9.0E-09	--		1.61E-07	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	2.3E-03	1.3E-09	1.5E-08	--		2.20E-07	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.9E-03	1.1E-09	1.2E-08	--		2.59E-06	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	1.9E-04	1.1E-10	1.3E-09	--		6.39E-07	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	9.2E-10	5.2E-16	6.1E-15	--		5.77E-06	3%
PAHs	2.3E-01				100%	2.1E-09	1.2E-15	1.4E-14	2.8E-16		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	100%	3.9E-07	2.2E-13	2.6E-12	--		1.63E-07	0%
TOTAL									2.8E-16		0.000230	

Dermal Exposure to Chemicals via Contact with Soil - Grid Maximum Off-site

$$\text{Daily Chemical Intake}_{DS} = C_S \cdot \frac{SA_S \cdot AF \cdot FE \cdot ABS \cdot CF \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults		
Surface Area (SAs, cm ²)	6300	Exposed skin surface area for adults as per ASC NEPM
Adherence Factor (AF, mg/cm ²)	0.5	Default as per ASC NEPM
Fraction of Day Exposed	1	Assume skin is washed after 24 hours
Conversion Factor (CF)	1.E-06	Conversion of units
Dermal absorption (ABS, unitless)	Chemical-specific (as below)	
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	29	Time at one residence as adult as per ASC NEPM
Body Weight (BW, kg)	70	For male and females combined (ASC NEPM)
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

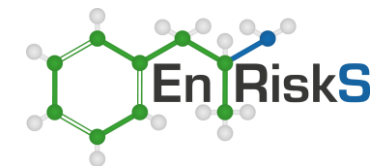
Key Chemical	Toxicity Data					Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)	Dermal Absorption (ABS)		Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	6.1E-04	1.1E-11	2.7E-11	--		8.6E-08	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04		4.2E-04			--		--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		1.0E-03			--		--	
Antimony (Sb)		8.6E-04		8.6E-04		1.1E-03			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	4.7E-04	4.4E-11	1.1E-10	--		1.1E-07	8%
Lead (Pb)		6.0E-04	50%	3.0E-04		6.2E-03			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		1.5E-03			--		--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		3.8E-04			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		1.4E-03			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		2.3E-03			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		1.9E-03			--		--	
Vanadium (V)		2.0E-03		2.0E-03		1.9E-04			--		--	
Dioxin-like compounds		2.3E-09	54%	1.1E-09	0.03	9.2E-10	5.1E-16	1.2E-15	--		1.2E-06	86%
PAHs	2.3E-01				0.06	2.1E-09	2.4E-15	5.7E-15	5.5E-16		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05		3.9E-07			--		--	
TOTAL									5.5E-16		1.4E-06	

Dermal Exposure to Chemicals via Contact with Soil - Grid Maximum Off-site

$$\text{Daily Chemical Intake}_{DS} = C_S \cdot \frac{SA_s \cdot AF \cdot FE \cdot ABS \cdot CF \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Young Children		
Surface Area (SAs, cm ²)	2700	Exposed skin surface area for young children as per ASC NEPM
Adherence Factor (AF, mg/cm ²)	0.5	Default as per ASC NEPM
Fraction of Day Exposed	1	Assume skin is washed after 24 hours
Conversion Factor (CF)	1.E-06	Conversion of units
Dermal absorption (ABS, unitless)	Chemical-specific (as below)	
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data					Soil Concentration (mg/kg)	Daily Intake		Calculated Risk		
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)	Dermal Absorption (ABS)		Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	6.1E-04	4.7E-12	5.5E-11	--	1.7E-07	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04		4.2E-04			--	--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		1.0E-03			--	--	
Antimony (Sb)		8.6E-04		8.6E-04		1.1E-03			--	--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	4.7E-04	1.8E-11	2.1E-10	--	2.1E-07	8%
Lead (Pb)		6.0E-04	50%	3.0E-04		6.2E-03			--	--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		1.5E-03			--	--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		3.8E-04			--	--	
Copper (Cu)		1.4E-01	60%	5.6E-02		1.4E-03			--	--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		2.3E-03			--	--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		1.9E-03			--	--	
Vanadium (V)		2.0E-03		2.0E-03		1.9E-04			--	--	
Dioxin-like compounds		2.3E-09	54%	1.1E-09	0.03	9.2E-10	2.1E-16	2.5E-15	--	2.3E-06	86%
PAHs	2.3E-01				0.06	2.1E-09	9.7E-16	1.1E-14	2.3E-16	--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05		3.9E-07			--	--	
TOTAL									2.3E-16	2.7E-06	



Current Maximum Residential Location

Predicted ground level concentrations - chronic exposures - increment

Key Chemicals	Air Concentration - annual average (µg/m³)	Air Concentration - annual average (mg/m³)	Cumulative Air Concentration (mg/m³)	Exposure pathways							
	Maximum Residential	Maximum Residential	Maximum Residential	inhalation	soil ingestion	soil - dermal	egg ingestion	fruit and vegetable ingestion	rainwater tank	meat ingestion	milk ingestion
Hydrogen chloride (HCl)	0.01071	0.00001071		✓	*	*	*	*	*	*	*
Hydrogen fluoride (HF)	0.00178	0.00000178		✓	*	*	*	*	*	*	*
Ammonia	0.01785	0.00001785		✓	*	*	*	*	*	*	*
Cadmium (Cd)	0.0000212	0.0000000212		✓	✓	✓	✓	✓	✓	✓	✓
Thallium (Tl)	0.0000145	0.0000000145		✓	✓	✓	✓	✓	✓	✓	✓
Mercury (Hg)	0.0000357	0.0000000357		✓	✓	✓	✓	✓	✓	✓	✓
Antimony (Sb)	0.0000377	0.0000000377		✓	✓	✓	✓	✓	✓	✓	✓
Arsenic (As)	0.0000165	0.0000000165	0.0000219075	✓	✓	✓	✓	✓	✓	✓	✓
Lead (Pb)	0.0002151	0.0000002151		✓	✓	✓	✓	✓	✓	✓	✓
Chromium (Cr VI assumed)	0.0000538	0.0000000538	0.0000542528	✓	✓	✓	✓	✓	✓	✓	✓
Cobalt (Co)	0.0000133	0.0000000133		✓	✓	✓	✓	✓	✓	✓	✓
Copper (Cu)	0.0000471	0.0000000471	0.0000599121	✓	✓	✓	✓	✓	✓	✓	✓
Manganese (Mn)	0.0000805	0.0000000805	0.0000848103	✓	✓	✓	✓	✓	✓	✓	✓
Nickel (Ni)	0.0000648	0.0000000648	0.0000666221	✓	✓	✓	✓	✓	✓	✓	✓
Vanadium (V)	0.0000067	0.0000000067		✓	✓	✓	✓	✓	✓	✓	✓
Dioxin-like compounds	1.07E-10	1.07E-13		✓	✓	✓	✓	✓	✓	✓	✓
PAHs	3.57E-10	3.57E-13		✓	✓	✓	✓	✓	✓	✓	✓
PFAS (assumed as PFOS)	1.36E-08	1.36E-11		✓	*	*	*	*	*	*	*
Benzene	0.01785	0.00001785	3.51784826	✓	*	*	*	*	*	*	*
Formaldehyde	0.01785	0.00001785		✓	*	*	*	*	*	*	*

Shaded entries indicate pathways included in the farming scenario only

Key Chemicals	Deposition Rate - annual average (ng/m²/year)	Deposition Rate - annual average (mg/m²/year)
	Maximum Residential	Maximum Residential
Hydrogen chloride (HCl)	NR	NR
Hydrogen fluoride (HF)	NR	NR
Ammonia	NR	NR
Cadmium (Cd)	99.36	0.0000994
Thallium (Tl)	68.52	0.00006852
Mercury (Hg)	167.88	0.00016788
Antimony (Sb)	177.18	0.00017718
Arsenic (As)	77.33	0.00007733
Lead (Pb)	1008.25	0.00100825
Chromium (Cr VI assumed)	252.55	0.00025255
Cobalt (Co)	62.65	0.00006265
Copper (Cu)	221.23	0.00022123
Manganese (Mn)	377.85	0.00037785
Nickel (Ni)	304.43	0.00030443
Vanadium (V)	31.32	0.00003132
Dioxin-like compounds	5.04E-04	5.04E-10
PAHs	1.68E-03	1.68E-09
PFAS (assumed as PFOS)	6.39E-02	6.39E-08
Benzene	NR	NR
Formaldehyde	NR	NR

Inhalation - gases and particulates

Maximum residential location

$$\text{Inhalation Exposure Concentration} = Ca * \frac{ET * FI * EF * ED}{AT} \quad (\text{mg/m}^3) \text{ for gases}$$

$$\text{Inhalation Exposure Concentration} = Ca * \frac{ET * FI * DRF * EF * ED}{AT} \quad (\text{mg/m}^3) \text{ for chemicals attached to particles}$$

Parameters Relevant to Quantification of Community Exposures - Residents

Exposure Time at Home (ET, hr/day)	24	Assume residents at home or on property 24 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Assume resident at the same property
Dust lung retention factor (unitless)	0.375	Percentage of respirable dust that is small enough to reach and be retained in the lungs (ASC NEPM) - NA for gases
Exposure Frequency - normal conditions (EF, days/yr)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	35	As per ASC NEPM
Averaging Time - NonThreshold (Atc, hours)	613200	ASC NEPM
Averaging Time - Threshold (Atn, hours)	306600	ASC NEPM

It is noted that because particles (and attached chemicals) from an W/E facility are very small, it has been assumed that all particles are small enough to reach down into the lungs - i.e. this factor has not been included in the calculation.

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk	Chronic TC Air	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC- Background)	Estimated Concentration in Air Maximum anywhere (Ca)	Inhalation Exposure Concentration - NonThreshold	Inhalation Exposure Concentration - Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/m ³) ⁻¹	(mg/m ³)		(mg/m ³)	(mg/m ³)	(mg/m ³)	(mg/m ³)	(unitless)		(unitless)	
Hydrogen chloride (HCl)		2.6E-02	0%	2.6E-02	1.1E-05	5.4E-06	1.1E-05	--		0.000412	2%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	1.8E-06	8.9E-07	1.8E-06	--		0.0000615	0%
Ammonia		3.2E-01	0%	3.2E-01	1.8E-05	8.9E-06	1.8E-05	--		0.0000558	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	2.1E-08	1.1E-08	2.1E-08	--		0.00529	24%
Thallium (Tl)		7.0E-04	10%	6.3E-04	1.5E-08	7.3E-09	1.5E-08	--		0.0000231	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	3.6E-08	1.8E-08	3.6E-08	--		0.000179	1%
Antimony (Sb)		3.0E-04	0%	3.0E-04	3.8E-08	1.9E-08	3.8E-08	--		0.000126	1%
Arsenic (As)		6.7E-05	0%	6.7E-05	1.6E-08	8.2E-09	1.6E-08	--		0.000246	1%
Lead (Pb)		5.0E-04	0%	5.0E-04	2.2E-07	1.1E-07	2.2E-07	--		0.000430	2%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	5.4E-08	2.7E-08	5.4E-08	--		0.0125	57%
Cobalt (Co)		1.0E-04	0%	1.0E-04	1.3E-08	6.7E-09	1.3E-08	--		0.000133	1%
Copper (Cu)		4.9E-01	60%	2.0E-01	4.7E-08	2.4E-08	4.7E-08	--		0.000000240	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	8.0E-08	4.0E-08	8.0E-08	--		0.000671	3%
Nickel (Ni)		5.9E-05	10%	5.3E-05	6.5E-08	3.2E-08	6.5E-08	--		0.00122	6%
Vanadium (V)		1.0E-04	0%	1.0E-04	6.7E-09	3.3E-09	6.7E-09	--		0.0000667	0%
Dioxin-like compounds		8.1E-09	54%	3.7E-09	1.1E-13	5.4E-14	1.1E-13	--		0.0000289	0%
PAHs	6.0E-01				3.6E-13	1.8E-13	3.6E-13	1.1E-13		--	
PFAS (assumed as PFOS)		7.0E-05	0%	7.0E-05	1.4E-11	6.8E-12	1.4E-11	--		0.000000194	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	1.8E-05	8.9E-06	1.8E-05	5.4E-8		0.000661047	3%
Formaldehyde		1.1E-02	30%	7.7E-03	1.8E-05	8.9E-06	6.7E-06	--		0.000869233	4%

Risk Index with VOCs as Benzene	5.4E-08	0.0221
Risk Index with VOCs as Formaldehyde	1.1E-13	0.0223

Calculation of Concentrations in Soil

$$C_s = \frac{DR \cdot [1 - e^{-k \cdot t}]}{d \cdot \rho \cdot k} \cdot 1000 \quad (\text{mg/kg}) \quad \text{ref: Stevens B. (1991)}$$

where:

DR= Particle deposition rate (mg/m²/year)
 K = Chemical-specific soil-loss constant (1/year) = ln(2)/T_{0.5}
 T_{0.5} = Chemical half-life in soil (years)
 t = Accumulation time (years)
 d = Soil mixing depth (m)
 ρ = Soil bulk-density (g/m³)
 1000 = Conversion from g to kg

General Parameters		Surface (for direct contact)	Depth (for agricultural pathways)	
Soil bulk density (ρ)	g/m ³	1600000	1600000	Default for fill materials
General mixing depth (d)	m	0.01	0.15	As per OEHH (2015) guidance
Duration of deposition (T)	years	70	70	As per OEHH (2015) guidance

Chemical-specific Inputs and calculations

Chemical	Half-life in soil years	Degradation constant (k) per year	Deposition Rate (DR) mg/m ² /year	Surface Concentration in Soil mg/kg	Agricultural Concentration in Soil mg/kg
Cadmium (Cd)	273973	2.5E-06	9.9E-05	4.3E-04	2.9E-05
Thallium (Tl)	273973	2.5E-06	6.9E-05	3.0E-04	2.0E-05
Mercury (Hg)	273973	2.5E-06	1.7E-04	7.3E-04	4.9E-05
Antimony (Sb)	273973	2.5E-06	1.8E-04	7.8E-04	5.2E-05
Arsenic (As)	273973	2.5E-06	7.7E-05	3.4E-04	2.3E-05
Lead (Pb)	273973	2.5E-06	1.0E-03	4.4E-03	2.9E-04
Chromium (Cr VI assumed)	273973	2.5E-06	2.5E-04	1.1E-03	7.4E-05
Cobalt (Co)	273973	2.5E-06	6.3E-05	2.7E-04	1.8E-05
Copper (Cu)	273973	2.5E-06	2.2E-04	9.7E-04	6.5E-05
Manganese (Mn)	273973	2.5E-06	3.8E-04	1.7E-03	1.1E-04
Nickel (Ni)	273973	2.5E-06	3.0E-04	1.3E-03	8.9E-05
Vanadium (V)	273973	2.5E-06	3.1E-05	1.4E-04	9.1E-06
Dioxin-like compounds	15.00	4.6E-02	5.0E-10	6.5E-10	4.4E-11
PAHs	1.18	5.9E-01	1.7E-09	1.8E-10	1.2E-11
PFAS (assumed as PFOS)	273973	2.5E-06	6.4E-08	2.8E-07	1.9E-08

Half-life in soil for dioxins: 9-15 years in surface soils; 25-100 years in subsurface soils (ATSDR 1998, DEH 2004)

Half-life in soil for metals, PAHs: OEHH 2015

Half-life in soil for PFAS assumed same as metals - i.e. no degradation at all but these chemicals do degrade slowly - this is conservative

Exposure to Chemicals via Incidental Ingestion of Soil - Maximum residential

$$\text{Daily Chemical Intake}_{IS} = C_S \cdot \frac{IR_S \cdot FI \cdot CF \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults

Ingestion Rate (IRs, mg/day)	50	As per ASC NEPM
Fraction Ingested from Source (FI, unitless)	100%	All of daily soil intake occurs from site
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	29	Time at one residence as adult as per ASC NEPM
Body Weight (BW, kg)	70	For male and females combined ASC NEPM
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	4.3E-04	1.3E-10	3.1E-10	--		9.70E-07	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	3.0E-04	8.9E-11	2.1E-10	--		1.19E-06	7%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	7.3E-04	2.2E-10	5.2E-10	--		2.19E-06	12%
Antimony (Sb)		8.6E-04		8.6E-04	100%	7.8E-04	2.3E-10	5.5E-10	--		6.44E-07	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	3.4E-04	1.0E-10	2.4E-10	--		2.42E-07	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	4.4E-03	1.3E-09	3.2E-09	--		1.05E-05	60%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	1.1E-03	3.3E-10	7.9E-10	--		9.74E-07	6%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	2.7E-04	8.1E-11	2.0E-10	--		1.75E-07	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	9.7E-04	2.9E-10	6.9E-10	--		1.23E-08	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.7E-03	4.9E-10	1.2E-09	--		1.69E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.3E-03	3.9E-10	9.5E-10	--		1.98E-07	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	1.4E-04	4.1E-11	9.8E-11	--		4.89E-08	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	6.5E-10	1.9E-16	4.7E-16	--		4.42E-07	3%
PAHs	2.3E-01				100%	1.8E-10	5.3E-17	1.3E-16	1.2E-17		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	100%	2.8E-07	8.3E-14	2.0E-13	--		1.25E-08	0%
TOTAL									1.2E-17		0.0000176	

Exposure to Chemicals via Incidental Ingestion of Soil - Maximum residential

$$\text{Daily Chemical Intake}_{IS} = C_S \cdot \frac{IR_S \cdot FI \cdot CF \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Young Children

Ingestion Rate (IRs, mg/day)	100	Assumed daily soil ingestion rate for young children, ASC NEPM
Fraction Ingested from Source (FI, unitless)	100%	All of daily soil intake occurs from site
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		0.0008	60%	3.2E-04	100%	4.3E-04	2.5E-10	2.9E-09	--		9.06E-06	6%
Thallium (Tl)		0.0002	10%	1.8E-04	100%	3.0E-04	1.7E-10	2.0E-09	--		1.11E-05	7%
Mercury (Hg)		0.0006	60%	2.4E-04	100%	7.3E-04	4.2E-10	4.9E-09	--		2.04E-05	12%
Antimony (Sb)		0.00086		8.6E-04	100%	7.8E-04	4.4E-10	5.2E-09	--		6.01E-06	4%
Arsenic (As)		0.002	50%	1.0E-03	100%	3.4E-04	1.9E-10	2.3E-09	--		2.26E-06	1%
Lead (Pb)		0.0006	50%	3.0E-04	100%	4.4E-03	2.5E-09	2.9E-08	--		9.80E-05	60%
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04	100%	1.1E-03	6.3E-10	7.4E-09	--		9.09E-06	6%
Cobalt (Co)		0.0014	20%	1.1E-03	100%	2.7E-04	1.6E-10	1.8E-09	--		1.63E-06	1%
Copper (Cu)		0.14	60%	5.6E-02	100%	9.7E-04	5.5E-10	6.5E-09	--		1.15E-07	0%
Manganese (Mn)		0.14	50%	7.0E-02	100%	1.7E-03	9.4E-10	1.1E-08	--		1.57E-07	0%
Nickel (Ni)		0.012	60%	4.8E-03	100%	1.3E-03	7.6E-10	8.9E-09	--		1.85E-06	1%
Vanadium (V)		0.002		2.0E-03	100%	1.4E-04	7.8E-11	9.1E-10	--		4.57E-07	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	6.5E-10	3.7E-16	4.4E-15	--		4.13E-06	3%
PAHs	0.233				100%	1.8E-10	1.0E-16	1.2E-15	2.4E-17		--	
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05	100%	2.8E-07	1.6E-13	1.9E-12	--		1.16E-07	0%
TOTAL									2.4E-17		0.000164	

Dermal Exposure to Chemicals via Contact with Soil - Maximum residential

$$\text{Daily Chemical Intake}_{DS} = C_S \cdot \frac{SA_S \cdot AF \cdot FE \cdot ABS \cdot CF \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults		
Surface Area (SAs, cm ²)	6300	Exposed skin surface area for adults as per ASC NEPM
Adherence Factor (AF, mg/cm ²)	0.5	Default as per ASC NEPM
Fraction of Day Exposed	1	Assume skin is washed after 24 hours
Conversion Factor (CF)	1.E-06	Conversion of units
Dermal absorption (ABS, unitless)	Chemical-specific (as below)	
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	29	Time at one residence as adult as per ASC NEPM
Body Weight (BW, kg)	70	For male and females combined ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

Key Chemical	Toxicity Data					Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)	Dermal Absorption (ABS)		Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		0.0008	60%	3.2E-04	0.001	4.3E-04	8.1E-12	2.0E-11	--		6.1E-08	6%
Thallium (Tl)		0.0002	10%	1.8E-04		3.0E-04			--		--	
Mercury (Hg)		0.0006	60%	2.4E-04		7.3E-04			--		--	
Antimony (Sb)		0.00086		8.6E-04		7.8E-04			--		--	
Arsenic (As)		0.002	50%	1.0E-03	0.005	3.4E-04	3.2E-11	7.6E-11	--		7.6E-08	8%
Lead (Pb)		0.0006	50%	3.0E-04		4.4E-03			--		--	
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04		1.1E-03			--		--	
Cobalt (Co)		0.0014	20%	1.1E-03		2.7E-04			--		--	
Copper (Cu)		0.14	60%	5.6E-02		9.7E-04			--		--	
Manganese (Mn)		0.14	50%	7.0E-02		1.7E-03			--		--	
Nickel (Ni)		0.012	60%	4.8E-03		1.3E-03			--		--	
Vanadium (V)		0.002		2.0E-03		1.4E-04			--		--	
Dioxin-like compounds		2.3E-09	54%	1.1E-09	0.03	6.5E-10	3.7E-16	8.8E-16	--		8.4E-07	86%
PAHs	0.233				0.06	1.8E-10	2.0E-16	4.8E-16	4.7E-17			
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05		2.8E-07			--		--	
TOTAL									4.7E-17		9.7E-07	

Dermal Exposure to Chemicals via Contact with Soil - Maximum residential

$$\text{Daily Chemical Intake}_{DS} = C_S \cdot \frac{SA_S \cdot AF \cdot FE \cdot ABS \cdot CF \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Young Children		
Surface Area (SAs, cm ²)	2700	Exposed skin surface area for young children as per ASC NEPM
Adherence Factor (AF, mg/cm ²)	0.5	Default as per ASC NEPM
Fraction of Day Exposed	1	Assume skin is washed after 24 hours
Conversion Factor (CF)	1.E-06	Conversion of units
Dermal absorption (ABS, unitless)	Chemical-specific (as below)	
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data					Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)	Dermal Absorption (ABS)		Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		0.0008	60%	3.2E-04	0.001	4.3E-04	3.4E-12	3.9E-11	--		1.2E-07	6%
Thallium (Tl)		0.0002	10%	1.8E-04		3.0E-04			--		--	
Mercury (Hg)		0.0006	60%	2.4E-04		7.3E-04			--		--	
Antimony (Sb)		0.00086		8.6E-04		7.8E-04			--		--	
Arsenic (As)		0.002	50%	1.0E-03	0.005	3.4E-04	1.3E-11	1.5E-10	--		1.5E-07	8%
Lead (Pb)		0.0006	50%	3.0E-04		4.4E-03			--		--	
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04		1.1E-03			--		--	
Cobalt (Co)		0.0014	20%	1.1E-03		2.7E-04			--		--	
Copper (Cu)		0.14	60%	5.6E-02		9.7E-04			--		--	
Manganese (Mn)		0.14	50%	7.0E-02		1.7E-03			--		--	
Nickel (Ni)		0.012	60%	4.8E-03		1.3E-03			--		--	
Vanadium (V)		0.002		2.0E-03		1.4E-04			--		--	
Dioxin-like compounds		2.3E-09	54%	1.1E-09	0.03	6.5E-10	1.5E-16	1.8E-15	--		1.7E-06	86%
PAHs	0.233				0.06	1.8E-10	8.3E-17	9.6E-16	1.9E-17		--	
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05		2.8E-07			--		--	
TOTAL									1.9E-17		1.9E-06	

Calculation of Concentrations in Plants

ref: Stevens B. (1991)

Uptake Due to Deposition in Aboveground Crops $C_p = \frac{DR \cdot F \cdot [1 - e^{-k \cdot t}]}{Y \cdot k} \quad (\text{mg/kg plant - wet weight})$ where: DR= Particle deposition rate for accidental release (mg/m²/day) F= Fraction for the surface area of plant (unitless) k= Chemical-specific soil-loss constant (1/years) = ln(2)/T_{0.5} T_{0.5}= Chemical half-life as particulate on plant (days) t= Deposition time (days) Y= Crop yield (kg/m²)	Uptake via Roots from Soil $C_{rp} = C_s \cdot RUF \quad (\text{mg/kg plant - wet weight})$ where: C_s = Concentration of persistent chemical in soil assuming 15cm mixing depth within gardens, calculated using Soil Equation for each chemical assessed (mg/kg) RUF = Root uptake factor which differs for each Chemical (unitless)
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General Parameters	Units	Value
Crop		Edible crops
Crop Yield (Y)	kg/m ²	2
Deposition Time (t)	days	70
Plant Interception fraction (F)	unitless	0.051

Chemical-specific Inputs and calculations - Maximum residential							
Chemical	Half-life on plant (T _{0.5})#	Loss constant (k) &	Deposition Rate (DR)	Aboveground Produce Concentration via Deposition	Root Uptake Factor (RUF)\$	Soil Concentration (C _s)	Below Ground Produce Concentration
	days	per day	mg/m ² /day	mg/kg ww	unitless	mg/kg	mg/kg ww
Cadmium (Cd)	14	0.05	2.7E-07	1.4E-07	0.125	2.9E-05	3.6E-06
Thallium (Tl)	14	0.05	1.9E-07	9.4E-08	0.001	2.0E-05	2.0E-08
Mercury (Hg)	14	0.05	4.6E-07	2.3E-07	0.225	4.9E-05	1.1E-05
Antimony (Sb)	14	0.05	4.9E-07	2.4E-07	0.05	5.2E-05	2.6E-06
Arsenic (As)	14	0.05	2.1E-07	1.1E-07	0.01	2.3E-05	2.3E-07
Lead (Pb)	14	0.05	2.8E-06	1.4E-06	0.01	2.9E-04	2.9E-06
Chromium (Cr VI assumed)	14	0.05	6.9E-07	3.5E-07	0.00188	7.4E-05	1.4E-07
Cobalt (Co)	14	0.05	1.7E-07	8.6E-08	0.005	1.8E-05	9.1E-08
Copper (Cu)	14	0.05	6.1E-07	3.0E-07	0.1	6.5E-05	6.5E-06
Manganese (Mn)	14	0.05	1.0E-06	5.2E-07	0.0625	1.1E-04	6.9E-06
Nickel (Ni)	14	0.05	8.3E-07	4.2E-07	0.015	8.9E-05	1.3E-06
Vanadium (V)	14	0.05	8.6E-08	4.3E-08	0.00137	9.1E-06	1.3E-08
Dioxin-like compounds	14	0.05	1.4E-12	6.9E-13	0.000876	4.4E-11	3.8E-14
PAHs	14	0.05	4.6E-12	2.3E-12	0.00214	1.2E-11	2.5E-14
PFAS (assumed as PFOS)	14	0.05	1.8E-10	8.7E-11	0.011	1.9E-08	2.0E-10

\$ Root uptake factors from RAIS (soil to wet weight of plant)

& Loss constant is 1/half life

Half life on plant taken from Stevens 1991 which notes that particles deposit onto the surface of plants but then over time are lost due to weathering (wind, rain etc) - the half life for the amount of time these particles remain on the surface of the plant (and so may be present in the produce) is 14 days

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables - Maximum residential

$$\text{Daily chemical intake} = C_A \times \frac{IR_P \times \%A \times FI \times ME \times EF \times ED}{BW \times AT} + C_R \times \frac{IR_P \times \%R \times FI \times ME \times ED \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Produce (IRp) (kg/day)	0.4	Total fruit and vegetable consumption rate for adults as per ASC NEPM
Proportion of total intake from aboveground crops (%A)	73%	Proportions as per ASC NEPM
Proportion of total intake from root crops (%R)	27%	Proportions as per ASC NEPM
Fraction ingested that is homegrown (%)	35%	Relevant to rural fringe
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	29	Time at one residence as adult as per ASC NEPM
Body Weight (BW, kg)	70	For male and females combined ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration (mg/kg wet weight)	Root crops concentrations (mg/kg wet weight)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)				NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		0.0008	60%	3.2E-04	100%	1.4E-07	3.6E-06	8.9E-10	2.2E-09	—		6.7E-06	13%
Thallium (Tl)		0.0002	10%	1.8E-04	100%	9.4E-08	2.0E-08	6.1E-11	1.5E-10	—		8.2E-07	2%
Mercury (Hg)		0.0006	60%	2.4E-04	100%	2.3E-07	1.1E-05	2.6E-09	6.3E-09	—		2.6E-05	52%
Antimony (Sb)		0.00086		8.6E-04	100%	2.4E-07	2.6E-06	7.2E-10	1.7E-09	—		2.0E-06	4%
Arsenic (As)		0.002	50%	1.0E-03	100%	1.1E-07	2.3E-07	1.1E-10	2.8E-10	—		2.8E-07	1%
Lead (Pb)		0.0006	50%	3.0E-04	100%	1.4E-06	2.9E-06	1.5E-09	3.6E-09	—		1.2E-05	24%
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04	100%	3.5E-07	1.4E-07	2.4E-10	5.8E-10	—		7.1E-07	1%
Cobalt (Co)		0.0014	20%	1.1E-03	100%	8.6E-08	9.1E-08	7.2E-11	1.7E-10	—		1.6E-07	0%
Copper (Cu)		0.14	60%	5.6E-02	100%	3.0E-07	6.5E-06	1.6E-09	3.9E-09	—		7.0E-08	0%
Manganese (Mn)		0.14	50%	7.0E-02	100%	5.2E-07	6.9E-06	1.9E-09	4.5E-09	—		6.4E-08	0%
Nickel (Ni)		0.012	60%	4.8E-03	100%	4.2E-07	1.3E-06	5.5E-10	1.3E-09	—		2.8E-07	1%
Vanadium (V)		0.002		2.0E-03	100%	4.3E-08	1.3E-08	2.9E-11	6.9E-11	—		3.5E-08	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	6.9E-13	3.8E-14	4.3E-16	1.0E-15	—		9.7E-07	2%
PAHs	0.233				100%	2.3E-12	2.5E-14	1.4E-15	3.4E-15	3.2E-16		—	
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05	100%	8.7E-11	2.0E-10	9.9E-14	2.4E-13	—		1.5E-08	0%
TOTAL										3.2E-16		0.0000503	

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables - Maximum residential

$$\text{Daily chemical intake} = C_A \times \frac{IR_P \times \%A \times FI \times ME \times EF \times ED}{BW \times AT} + C_R \times \frac{IR_P \times \%R \times FI \times ME \times ED \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Young children

Ingestion Rate of Produce (IRp) (kg/day)	0.28	Total fruit and vegetable consumption rate for children as per ASC NEPM
Proportion of total intake from aboveground crops (%A)	84%	Proportions as per ASC NEPM
Proportion of total intake from root crops (%R)	16%	Proportions as per ASC NEPM
Fraction ingested that is homegrown (%)	35%	Relevant to rural fringe
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration (mg/kg wet weight)	Root crops concentrations (mg/kg wet weight)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)				NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		0.0008	60%	3.2E-04	100%	1.4E-07	3.6E-06	3.9E-10	4.5E-09	—		1.4E-05	12%
Thallium (Tl)		0.0002	10%	1.8E-04	100%	9.4E-08	2.0E-08	4.6E-11	5.3E-10	—		3.0E-06	2%
Mercury (Hg)		0.0006	60%	2.4E-04	100%	2.3E-07	1.1E-05	1.1E-09	1.3E-08	—		5.3E-05	45%
Antimony (Sb)		0.00086		8.6E-04	100%	2.4E-07	2.6E-06	3.5E-10	4.0E-09	—		4.7E-06	4%
Arsenic (As)		0.002	50%	1.0E-03	100%	1.1E-07	2.3E-07	7.0E-11	8.2E-10	—		8.2E-07	1%
Lead (Pb)		0.0006	50%	3.0E-04	100%	1.4E-06	2.9E-06	9.1E-10	1.1E-08	—		3.5E-05	30%
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04	100%	3.5E-07	1.4E-07	1.7E-10	2.0E-09	—		2.5E-06	2%
Cobalt (Co)		0.0014	20%	1.1E-03	100%	8.6E-08	9.1E-08	4.8E-11	5.7E-10	—		5.0E-07	0%
Copper (Cu)		0.14	60%	5.6E-02	100%	3.0E-07	6.5E-06	7.2E-10	8.4E-09	—		1.5E-07	0%
Manganese (Mn)		0.14	50%	7.0E-02	100%	5.2E-07	6.9E-06	8.6E-10	1.0E-08	—		1.4E-07	0%
Nickel (Ni)		0.012	60%	4.8E-03	100%	4.2E-07	1.3E-06	3.2E-10	3.7E-09	—		7.7E-07	1%
Vanadium (V)		0.002		2.0E-03	100%	4.3E-08	1.3E-08	2.1E-11	2.5E-10	—		1.2E-07	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	6.9E-13	3.8E-14	3.3E-16	3.8E-15	—		3.6E-06	3%
PAHs	0.233				100%	2.3E-12	2.5E-14	1.1E-15	1.3E-14	2.5E-16		—	
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05	100%	8.7E-11	2.0E-10	5.9E-14	6.9E-13	—		4.3E-08	0%
TOTAL										2.5E-16		0.000119	

Calculation of Concentrations in Eggs

Uptake in to chicken eggs

$$C_E = (FI \times IR_C \times C + IR_S \times C_S \times B) \times TFE \quad (\text{mg/kg egg - wet weight})$$

where:

FI = Fraction of pasture/crop ingested by chickens each day (unitless)

IR_C = Ingestion rate of pasture/crop by chicken each day (kg/day)

C = Concentration of chemical in grain/crop eaten by chicken (mg/kg)

IR_S = Ingestion rate of soil by chickens each day (kg/day)

C_S = Concentration in soil the chickens ingest (mg/kg)

B = Bioavailability of soil ingested by chickens (%)

TFE = Transfer factor from ingestion to eggs (day/kg)

General Parameters	Units	Value
FI (fraction of crops ingested from property)		1
IR _C (ingestion rate of crops)	kg/day	0.12
IR _S (ingestion rate of soil)	kg/day	0.01
B (bioavailability)	%	100%

Assume 100% of crops consumed by chickens is grown in the same soil

Assumed ingestion rate from OEHHa 2015 (assume concentration the same as predicted for aboveground crops)

USEPA (2005) (Ag Victoria recommendation)

Chemical-specific Inputs and calculations - Maximum residential				
Chemical	Concentration in crops ingested by chickens mg/kg ww	Soil Concentration - Agriculture (C _S) mg/kg	Transfer factor to eggs day/kg	Egg Concentration mg/kg ww
Cadmium (Cd)	1.4E-07	2.9E-05	1.0E-02	3.1E-09
Thallium (Tl)	9.4E-08	2.0E-05	3.8E-02	8.0E-09
Mercury (Hg)	2.3E-07	4.9E-05	8.0E-01	4.1E-07
Antimony (Sb)	2.4E-07	5.2E-05	1.7E-01	9.3E-08
Arsenic (As)	1.1E-07	2.3E-05	7.0E-02	1.7E-08
Lead (Pb)	1.4E-06	2.9E-04	4.0E-02	1.2E-07
Chromium (Cr VI assumed)	3.5E-07	7.4E-05	3.8E-02	3.0E-08
Cobalt (Co)	8.6E-08	1.8E-05	3.8E-02	7.3E-09
Copper (Cu)	3.0E-07	6.5E-05	3.8E-02	2.6E-08
Manganese (Mn)	5.2E-07	1.1E-04	3.8E-02	4.4E-08
Nickel (Ni)	4.2E-07	8.9E-05	2.0E-02	1.9E-08
Vanadium (V)	4.3E-08	9.1E-06	3.8E-02	3.7E-09
Dioxin-like compounds	6.9E-13	4.4E-11	1.0E+01	5.2E-12
PAHs	2.3E-12	1.2E-11	3.0E-03	1.2E-15
PFAS (assumed as PFOS)	8.7E-11	1.9E-08	7.0E-02	1.4E-11

OEHHa (2012)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

OEHHa (2012)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

OEHHa (2012)

OEHHa (2012)

OEHHa (2012)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

OEHHa (2012)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

OEHHa (2012)

OEHHa (2012)

RAIS (2024)

Transfer factors from OEHHa 2012 unless otherwise noted

Exposure to Chemicals via Ingestion of Eggs - Maximum residential

$$\text{Daily chemical intake} = C_E \times \frac{IR_E \times FI \times ME \times EF \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults

Ingestion Rate of Eggs (IRE) (kg/day)	0.059	Ingestion rate of eggs relevant for adults as per FSANZ (2017) (P90 for consumers - i.e. high end consumer)
Fraction ingested that is homegrown (%)	100%	Assume all eggs consumed are from backyard chickens
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	29	Time at one residence as adult as per ASC NEPM
Body Weight (BW, kg)	70	ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration (mg/kg wet weight)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		0.0008	60%	3.2E-04	100%	3.1E-09	1.1E-12	2.6E-12	--		8.1E-09	0%
Thallium (Tl)		0.0002	10%	1.8E-04	100%	8.0E-09	2.8E-12	6.8E-12	--		3.8E-08	1%
Mercury (Hg)		0.0006	60%	2.4E-04	100%	4.1E-07	1.4E-10	3.5E-10	--		1.5E-06	24%
Antimony (Sb)		0.00086		8.6E-04	100%	9.3E-08	3.2E-11	7.8E-11	--		9.1E-08	1%
Arsenic (As)		0.002	50%	1.0E-03	100%	1.7E-08	5.8E-12	1.4E-11	--		1.4E-08	0%
Lead (Pb)		0.0006	50%	3.0E-04	100%	1.2E-07	4.3E-11	1.0E-10	--		3.5E-07	6%
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04	100%	3.0E-08	1.0E-11	2.5E-11	--		3.1E-08	1%
Cobalt (Co)		0.0014	20%	1.1E-03	100%	7.3E-09	2.6E-12	6.2E-12	--		5.5E-09	0%
Copper (Cu)		0.14	60%	5.6E-02	100%	2.6E-08	9.0E-12	2.2E-11	--		3.9E-10	0%
Manganese (Mn)		0.14	50%	7.0E-02	100%	4.4E-08	1.5E-11	3.7E-11	--		5.3E-10	0%
Nickel (Ni)		0.012	60%	4.8E-03	100%	1.9E-08	6.5E-12	1.6E-11	--		3.3E-09	0%
Vanadium (V)		0.002		2.0E-03	100%	3.7E-09	1.3E-12	3.1E-12	--		1.5E-09	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	5.2E-12	1.8E-15	4.4E-15	--		4.1E-06	67%
PAHs	0.233				100%	1.2E-15	4.1E-19	1.0E-18	9.6E-20		--	
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05	100%	1.4E-11	4.8E-15	1.2E-14	--		7.3E-10	0%
TOTAL									9.6E-20		0.00000613	

Exposure to Chemicals via Ingestion of Eggs - Maximum residential

$$\text{Daily chemical intake} = C_E \times \frac{IR_E \times FI \times ME \times EF \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Young children

Ingestion Rate of Eggs (IRE) (kg/day)	0.036	Ingestion rate of eggs relevant for young children as per FSANZ (2017) (P90 for consumers - i.e. high end consumer)
Fraction ingested that is homegrown (%)	100%	Assume all eggs consumed are from backyard chickens
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration (mg/kg wet weight)	Daily Intake		Calculated Risk		
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)
Cadmium (Cd)		0.0008	60%	3.2E-04	100%	3.1E-09	6.3E-13	7.3E-12	--		2.3E-08
Thallium (Tl)		0.0002	10%	1.8E-04	100%	8.0E-09	1.7E-12	1.9E-11	--		1.1E-07
Mercury (Hg)		0.0006	60%	2.4E-04	100%	4.1E-07	8.5E-11	9.9E-10	--		4.1E-06
Antimony (Sb)		0.00086		8.6E-04	100%	9.3E-08	1.9E-11	2.2E-10	--		2.6E-07
Arsenic (As)		0.002	50%	1.0E-03	100%	1.7E-08	3.4E-12	4.0E-11	--		4.0E-08
Lead (Pb)		0.0006	50%	3.0E-04	100%	1.2E-07	2.6E-11	3.0E-10	--		9.9E-07
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04	100%	3.0E-08	6.1E-12	7.1E-11	--		8.8E-08
Cobalt (Co)		0.0014	20%	1.1E-03	100%	7.3E-09	1.5E-12	1.8E-11	--		1.6E-08
Copper (Cu)		0.14	60%	5.6E-02	100%	2.6E-08	5.3E-12	6.2E-11	--		1.1E-09
Manganese (Mn)		0.14	50%	7.0E-02	100%	4.4E-08	9.1E-12	1.1E-10	--		1.5E-09
Nickel (Ni)		0.012	60%	4.8E-03	100%	1.9E-08	3.9E-12	4.5E-11	--		9.4E-09
Vanadium (V)		0.002		2.0E-03	100%	3.7E-09	7.5E-13	8.8E-12	--		4.4E-09
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	5.2E-12	1.1E-15	1.2E-14	--		1.2E-05
PAHs	0.233				100%	1.2E-15	2.4E-19	2.8E-18	5.7E-20		--
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05	100%	1.4E-11	2.8E-15	3.3E-14	--		2.1E-09
TOTAL									5.7E-20		0.0000175

Calculation of Concentrations in Homegrown Beef

Uptake in to beef meat

$$C_E = (FI \times IR_C \times C + IR_S \times C_S \times B) \times TFE_B \quad (\text{mg/kg beef – wet weight})$$

where:

FI = Fraction of grain/crop ingested by cattle each day (unitless)

IRc = Ingestion rate of grain/crop by cattle each day (kg/day)

C = Concentration of chemical in grain/crop eaten by cattle (mg/kg)

IRs = Ingestion rate of soil by cattle each day (kg/day)

Cs = Concentration in soil the cattle ingest (mg/kg)

B = Bioavailability of soil ingested by cattle (%)

TFE = Transfer factor from ingestion to beef (day/kg)

General Parameters	Units	Value
FI (fraction of crops ingested from property)		1
IRc (ingestion rate of crops)	kg dw/day	9
IRs (ingestion rate of soil)	kg/day	0.45
B (bioavailability)	%	100%

Assume 100% of pasture consumed by cattle is grown in the same soil

Assumed ingestion rate from OEHHHA 2015 (assume concentration the same as predicted for aboveground crops)
OEHHHA (2015) Table 5.4 (soil ingestion = 5% of pasture ingestion (as dry weight))

Chemical-specific Inputs and calculations - Maximum residential

Chemical	Concentration in crops ingested by cattle mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to beef day/kg	Beef Concentration mg/kg ww	
Cadmium (Cd)	1.4E-07	2.9E-05	5.5E-04	7.8E-09	RAIS (2023)
Thallium (Tl)	9.4E-08	2.0E-05	4.0E-02	3.9E-07	RAIS (2023)
Mercury (Hg)	2.3E-07	4.9E-05	2.5E-01	6.0E-06	RAIS (2023)
Antimony (Sb)	2.4E-07	5.2E-05	1.0E-03	2.5E-08	RAIS (2023)
Arsenic (As)	1.1E-07	2.3E-05	2.0E-03	2.2E-08	RAIS (2023)
Lead (Pb)	1.4E-06	2.9E-04	4.0E-04	5.8E-08	RAIS (2023)
Chromium (Cr VI assumed)	3.5E-07	7.4E-05	5.5E-03	2.0E-07	RAIS (2023)
Cobalt (Co)	8.6E-08	1.8E-05	2.0E-02	1.8E-07	RAIS (2023)
Copper (Cu)	3.0E-07	6.5E-05	1.0E-02	3.2E-07	RAIS (2023)
Manganese (Mn)	5.2E-07	1.1E-04	4.0E-04	2.2E-08	RAIS (2023)
Nickel (Ni)	4.2E-07	8.9E-05	6.0E-03	2.6E-07	RAIS (2023)
Vanadium (V)	4.3E-08	9.1E-06	2.5E-03	1.1E-08	RAIS (2023)
Dioxin-like compounds	6.9E-13	4.4E-11	1.6E-01	4.1E-12	RAIS (2023)
PAHs	2.3E-12	1.2E-11	3.4E-02	8.8E-13	RAIS (2023)
PFAS (assumed as PFOS)	8.7E-11	1.9E-08	2.0E-03	1.8E-11	RAIS (2023)

Exposure to Chemicals via Ingestion of Beef - Maximum residential

$$\text{Daily chemical intake} = C_B \times \frac{IR_B \times FI \times ME \times EF \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults

Ingestion Rate of Beef (IRB) (kg/day)	0.16	Ingestion rate of beef for adults >19 years (enHealth 2012, noted to be the same as P90 from FSANZ 2017)
Fraction ingested that is homegrown (%)	35%	Assume 35% beef intakes from home-sourced meat
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per ASC NEPM
Body Weight (BW, kg)	70	ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration (mg/kg wet weight)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		0.0008	60%	3.2E-04	100%	7.8E-09	2.6E-12	6.3E-12	--		2.0E-08	0%
Thallium (Tl)		0.0002	10%	1.8E-04	100%	3.9E-07	1.3E-10	3.1E-10	--		1.7E-06	7%
Mercury (Hg)		0.0006	60%	2.4E-04	100%	6.0E-06	2.0E-09	4.8E-09	--		2.0E-05	79%
Antimony (Sb)		0.00086		8.6E-04	100%	2.5E-08	8.4E-12	2.0E-11	--		2.4E-08	0%
Arsenic (As)		0.002	50%	1.0E-03	100%	2.2E-08	7.4E-12	1.8E-11	--		1.8E-08	0%
Lead (Pb)		0.0006	50%	3.0E-04	100%	5.8E-08	1.9E-11	4.6E-11	--		1.5E-07	1%
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04	100%	2.0E-07	6.6E-11	1.6E-10	--		2.0E-07	1%
Cobalt (Co)		0.0014	20%	1.1E-03	100%	1.8E-07	6.0E-11	1.4E-10	--		1.3E-07	1%
Copper (Cu)		0.14	60%	5.6E-02	100%	3.2E-07	1.1E-10	2.5E-10	--		4.5E-09	0%
Manganese (Mn)		0.14	50%	7.0E-02	100%	2.2E-08	7.2E-12	1.7E-11	--		2.5E-10	0%
Nickel (Ni)		0.012	60%	4.8E-03	100%	2.6E-07	8.7E-11	2.1E-10	--		4.4E-08	0%
Vanadium (V)		0.002		2.0E-03	100%	1.1E-08	3.7E-12	9.0E-12	--		4.5E-09	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	4.1E-12	1.4E-15	3.3E-15	--		3.1E-06	12%
PAHs	0.233				100%	8.8E-13	2.9E-16	7.0E-16	6.8E-17		--	
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05	100%	1.8E-11	6.1E-15	1.5E-14	--		9.2E-10	0%
TOTAL									6.8E-17		0.0000255	

Exposure to Chemicals via Ingestion of Beef - Maximum residential

$$\text{Daily chemical intake} = C_B \times \frac{IR_B \times FI \times ME \times EF \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Children

Ingestion Rate of Beef (IRB) (kg/day)	0.085	Ingestion rate of beef by children aged 2-6 years (P90 value) FSANZ (2017)
Fraction ingested that is homegrown (%)	35%	Assume 35% beef intakes from home-sourced meat
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration (mg/kg wet weight)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		0.0008	60%	3.2E-04	100%	7.8E-09	1.3E-12	1.6E-11	--		4.9E-08	0%
Thallium (Tl)		0.0002	10%	1.8E-04	100%	3.9E-07	6.7E-11	7.8E-10	--		4.3E-06	7%
Mercury (Hg)		0.0006	60%	2.4E-04	100%	6.0E-06	1.0E-09	1.2E-08	--		5.0E-05	79%
Antimony (Sb)		0.00086		8.6E-04	100%	2.5E-08	4.3E-12	5.0E-11	--		5.9E-08	0%
Arsenic (As)		0.002	50%	1.0E-03	100%	2.2E-08	3.8E-12	4.4E-11	--		4.4E-08	0%
Lead (Pb)		0.0006	50%	3.0E-04	100%	5.8E-08	9.8E-12	1.1E-10	--		3.8E-07	1%
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04	100%	2.0E-07	3.4E-11	4.0E-10	--		4.9E-07	1%
Cobalt (Co)		0.0014	20%	1.1E-03	100%	1.8E-07	3.1E-11	3.6E-10	--		3.2E-07	1%
Copper (Cu)		0.14	60%	5.6E-02	100%	3.2E-07	5.4E-11	6.3E-10	--		1.1E-08	0%
Manganese (Mn)		0.14	50%	7.0E-02	100%	2.2E-08	3.7E-12	4.3E-11	--		6.1E-10	0%
Nickel (Ni)		0.012	60%	4.8E-03	100%	2.6E-07	4.5E-11	5.2E-10	--		1.1E-07	0%
Vanadium (V)		0.002		2.0E-03	100%	1.1E-08	1.9E-12	2.2E-11	--		1.1E-08	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	4.1E-12	6.9E-16	8.1E-15	--		7.7E-06	12%
PAHs	0.233				100%	8.8E-13	1.5E-16	1.7E-15	3.5E-17		--	
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05	100%	1.8E-11	3.1E-15	3.6E-14	--		2.3E-09	0%
TOTAL									3.5E-17		0.0000633	

Calculation of Concentrations in Dairy Milk

Uptake in to milk (dairy cows)

$$C_E = (FI \times IR_C \times C + IR_S \times C_S \times B) \times TF_B \quad (\text{mg/kg beef – wet weight})$$

where:

FI = Fraction of grain/crop ingested by cattle each day (unitless)

IR_C = Ingestion rate of grain/crop by cattle each day (kg/day)

C = Concentration of chemical in grain/crop eaten by cattle (mg/kg)

IR_S = Ingestion rate of soil by cattle each day (kg/day)

C_S = Concentration in soil the cattle ingest (mg/kg)

B = Bioavailability of soil ingested by cattle (%)

TF_E = Transfer factor from ingestion to milk (day/kg)

General Parameters	Units	Value
FI (fraction of crops ingested from property)		1
IR _C (ingestion rate of crops)	kg (dw)/day	22
IR _S (ingestion rate of soil)	kg/day	1.1
B (bioavailability)	%	100%

Assume 100% of pasture consumed by cattle is grown in the same soil

Assumed ingestion rate from OEHHA 2015 for lactating cattle (assume concentration the same as predicted for aboveground crops)

OEHHA (2015) Table 5.4 (soil ingestion = 5% of pasture ingestion (as dry weight))

Chemical-specific Inputs and calculations - Maximum residential					
Chemical	Concentration in crops ingested by cattle mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to milk day/kg	Milk Concentration mg/kg ww	
Cadmium (Cd)	1.4E-07	2.9E-05	5.0E-06	1.7E-10	OEHHa (2015)
Thallium (Tl)	9.4E-08	2.0E-05	2.0E-03	4.8E-08	RAIS (2023)
Mercury (Hg)	2.3E-07	4.9E-05	7.0E-05	4.1E-09	OEHHa (2015)
Antimony (Sb)	2.4E-07	5.2E-05	1.0E-04	6.2E-09	RAIS (2023)
Arsenic (As)	1.1E-07	2.3E-05	5.0E-05	1.4E-09	OEHHa (2015)
Lead (Pb)	1.4E-06	2.9E-04	6.0E-05	2.1E-08	OEHHa (2015)
Chromium (Cr VI assumed)	3.5E-07	7.4E-05	9.0E-06	8.0E-10	OEHHa (2015)
Cobalt (Co)	8.6E-08	1.8E-05	2.0E-03	4.4E-08	RAIS (2023)
Copper (Cu)	3.0E-07	6.5E-05	1.5E-03	1.2E-07	RAIS (2023)
Manganese (Mn)	5.2E-07	1.1E-04	3.5E-04	4.6E-08	RAIS (2023)
Nickel (Ni)	4.2E-07	8.9E-05	3.0E-05	3.2E-09	OEHHa (2015)
Vanadium (V)	4.3E-08	9.1E-06	2.0E-05	2.2E-10	RAIS (2023)
Dioxin-like compounds	6.9E-13	4.4E-11	2.0E-02	1.3E-12	OEHHa (2015)
PAHs	2.3E-12	1.2E-11	1.0E-02	6.4E-13	OEHHa (2015)
PFAS (assumed as PFOS)	8.7E-11	1.9E-08	5.0E-05	1.1E-12	OEHHa (2015)

Exposure to Chemicals via Ingestion of Milk - Maximum residential

$$\text{Daily chemical intake} = C_M \times \frac{IR_M \times FI \times ME \times EF \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults

Ingestion Rate of Milk (IRM) (kg/day)	1.295	Ingestion rate of cows milk for adults (P90 value from FSANZ 2017)
Fraction ingested that is homegrown (%)	100%	Assume all milk consumed is from the dairy farm
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per ASC NEPM
Body Weight (BW, kg)	70	ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration (mg/kg wet weight)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		0.0008	60%	3.2E-04	100%	1.7E-10	1.3E-12	3.2E-12	--		1.0E-08	0%
Thallium (Tl)		0.0002	10%	1.8E-04	100%	4.8E-08	3.7E-10	8.9E-10	--		4.9E-06	17%
Mercury (Hg)		0.0006	60%	2.4E-04	100%	4.1E-09	3.2E-11	7.6E-11	--		3.2E-07	1%
Antimony (Sb)		0.00086		8.6E-04	100%	6.2E-09	4.8E-11	1.2E-10	--		1.3E-07	0%
Arsenic (As)		0.002	50%	1.0E-03	100%	1.4E-09	1.0E-11	2.5E-11	--		2.5E-08	0%
Lead (Pb)		0.0006	50%	3.0E-04	100%	2.1E-08	1.6E-10	3.9E-10	--		1.3E-06	4%
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04	100%	8.0E-10	6.1E-12	1.5E-11	--		1.8E-08	0%
Cobalt (Co)		0.0014	20%	1.1E-03	100%	4.4E-08	3.4E-10	8.1E-10	--		7.3E-07	2%
Copper (Cu)		0.14	60%	5.6E-02	100%	1.2E-07	8.9E-10	2.2E-09	--		3.8E-08	0%
Manganese (Mn)		0.14	50%	7.0E-02	100%	4.6E-08	3.6E-10	8.6E-10	--		1.2E-08	0%
Nickel (Ni)		0.012	60%	4.8E-03	100%	3.2E-09	2.5E-11	5.9E-11	--		1.2E-08	0%
Vanadium (V)		0.002		2.0E-03	100%	2.2E-10	1.7E-12	4.1E-12	--		2.0E-09	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	1.3E-12	9.7E-15	2.3E-14	--		2.2E-05	75%
PAHs	0.233				100%	6.4E-13	4.9E-15	1.2E-14	1.1E-15		--	
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05	100%	1.1E-12	8.6E-15	2.1E-14	--		1.3E-09	0%

TOTAL **1.1E-15** **0.0000296**

Exposure to Chemicals via Ingestion of Milk - Maximum residential

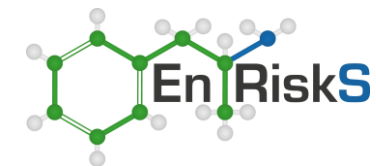
$$\text{Daily chemical intake} = C_M \times \frac{IR_M \times FI \times ME \times EF \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Children

Ingestion Rate of Milk (IRM) (kg/day)	1.097	Ingestion rate of cows milk for children aged 2-6 years (P90 value from FSANZ 2017)
Fraction ingested that is homegrown (%)	100%	Assume all milk consumed is from the dairy farm
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration (mg/kg wet weight)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		0.0008	60%	3.2E-04	100%	1.7E-10	1.1E-12	1.3E-11	--		4.0E-08	0%
Thallium (Tl)		0.0002	10%	1.8E-04	100%	4.8E-08	3.0E-10	3.5E-09	--		2.0E-05	17%
Mercury (Hg)		0.0006	60%	2.4E-04	100%	4.1E-09	2.6E-11	3.0E-10	--		1.3E-06	1%
Antimony (Sb)		0.00086		8.6E-04	100%	6.2E-09	3.9E-11	4.5E-10	--		5.3E-07	0%
Arsenic (As)		0.002	50%	1.0E-03	100%	1.4E-09	8.5E-12	9.9E-11	--		9.9E-08	0%
Lead (Pb)		0.0006	50%	3.0E-04	100%	2.1E-08	1.3E-10	1.6E-09	--		5.2E-06	4%
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04	100%	8.0E-10	5.0E-12	5.8E-11	--		7.2E-08	0%
Cobalt (Co)		0.0014	20%	1.1E-03	100%	4.4E-08	2.8E-10	3.2E-09	--		2.9E-06	2%
Copper (Cu)		0.14	60%	5.6E-02	100%	1.2E-07	7.3E-10	8.5E-09	--		1.5E-07	0%
Manganese (Mn)		0.14	50%	7.0E-02	100%	4.6E-08	2.9E-10	3.4E-09	--		4.8E-08	0%
Nickel (Ni)		0.012	60%	4.8E-03	100%	3.2E-09	2.0E-11	2.3E-10	--		4.9E-08	0%
Vanadium (V)		0.002		2.0E-03	100%	2.2E-10	1.4E-12	1.6E-11	--		8.0E-09	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	1.3E-12	7.9E-15	9.2E-14	--		8.7E-05	75%
PAHs	0.233				100%	6.4E-13	4.0E-15	4.7E-14	9.3E-16		--	
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05	100%	1.1E-12	7.0E-15	8.2E-14	--		5.1E-09	0%

TOTAL	9.3E-16	0.000117
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Maximum Commercial/Industrial Location

Predicted ground level concentrations - chronic exposures - increment

Key Chemicals	Air Concentration - annual average (µg/m ³)	Air Concentration - annual average (mg/m ³)	Cumulative Air Concentration (mg/m ³)	Exposure pathways							
	Maximum Commercial	Maximum Commercial	Maximum Commercial	inhalation	soil ingestion	soil - dermal	egg ingestion	fruit and vegetable ingestion	rainwater tank	meat ingestion	milk ingestion
Hydrogen chloride (HCl)	0.01183	0.00001183		✓	x	x	x	x	x	x	x
Hydrogen fluoride (HF)	0.00197	0.00000197		✓	x	x	x	x	x	x	x
Ammonia	0.01971	0.00001971		✓	x	x	x	x	x	x	x
Cadmium (Cd)	0.0000234	0.0000000234		✓	✓	✓	✓	✓	✓	x	x
Thallium (Tl)	0.0000161	0.0000000161		✓	✓	✓	✓	✓	✓	x	x
Mercury (Hg)	0.0000395	0.0000000395		✓	✓	✓	✓	✓	✓	x	x
Antimony (Sb)	0.0000416	0.0000000416		✓	✓	✓	✓	✓	✓	x	x
Arsenic (As)	0.0000182	0.0000000182	0.0000256258	✓	✓	✓	✓	✓	✓	x	x
Lead (Pb)	0.0002376	0.0000002376		✓	✓	✓	✓	✓	✓	x	x
Chromium (Cr VI assumed)	0.0000594	0.0000000594	0.0000601121	✓	✓	✓	✓	✓	✓	x	x
Cobalt (Co)	0.0000147	0.0000000147		✓	✓	✓	✓	✓	✓	x	x
Copper (Cu)	0.0000520	0.0000000520	0.0000693803	✓	✓	✓	✓	✓	✓	x	x
Manganese (Mn)	0.0000889	0.0000000889	0.0000935615	✓	✓	✓	✓	✓	✓	x	x
Nickel (Ni)	0.0000715	0.0000000715	0.0000741726	✓	✓	✓	✓	✓	✓	x	x
Vanadium (V)	0.0000074	0.0000000074		✓	✓	✓	✓	✓	✓	x	x
Dioxin-like compounds	1.18E-10	1.18E-13		✓	✓	✓	✓	✓	✓	x	x
PAHs	3.95E-10	3.95E-13		✓	✓	✓	✓	✓	✓	x	x
PFAS (assumed as PFOS)	1.50E-08	1.50E-11		✓	✓	✓	✓	✓	✓	x	x
Benzene	0.01971	0.00001971	3.51971277	✓	x	x	x	x	x	x	x
Formaldehyde	0.01971	0.00001971		✓	x	x	x	x	x	x	x

Shaded locations only relevant if commercial site land use changes to residential

Key Chemicals	Deposition Rate - annual average (ng/m ² /year)	Deposition Rate - annual average (mg/m ² /year)
	Maximum Commercial	Maximum Commercial
Hydrogen chloride (HCl)	NR	NR
Hydrogen fluoride (HF)	NR	NR
Ammonia	NR	NR
Cadmium (Cd)	68.04	0.0000680
Thallium (Tl)	46.93	0.00004693
Mercury (Hg)	114.97	0.00011497
Antimony (Sb)	121.34	0.00012134
Arsenic (As)	52.96	0.00005296
Lead (Pb)	690.49	0.00069049
Chromium (Cr VI assumed)	172.96	0.00017296
Cobalt (Co)	42.90	0.00004290
Copper (Cu)	151.51	0.00015151
Manganese (Mn)	258.77	0.00025877
Nickel (Ni)	208.49	0.00020849
Vanadium (V)	21.45	0.00002145
Dioxin-like compounds	3.45E-04	3.45E-10
PAHs	1.15E-03	1.15E-09
PFAS (assumed as PFOS)	4.37E-02	4.37E-08
Benzene	NR	NR
Formaldehyde	NR	NR

Inhalation - gases and particulates

Maximum commercial

$$\text{Inhalation Exposure Concentration} = Ca * \frac{ET * FI * EF * ED}{AT} \quad (\text{mg/m}^3) \text{ for gases}$$

$$\text{Inhalation Exposure Concentration} = Ca * \frac{ET * FI * DRF * EF * ED}{AT} \quad (\text{mg/m}^3) \text{ for chemicals attached to particles}$$

Parameters Relevant to Quantification of Community Exposures - Commercial/Industrial		
Exposure Time at Home (ET, hr/day)	10	Assume workers at work site 10 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Only exposed at the work site
Dust lung retention factor (unitless)	0.375	Percentage of respirable dust that is small enough to reach and be retained in the lungs (ASC NEPM) - NA for gases
Exposure Frequency - normal conditions (EF, days/yr)	240	Days at work (ASC NEPM)
Exposure Duration (ED, years)	30	ASC NEPM
Averaging Time - NonThreshold (Atc, hours)	613200	ASC NEPM
Averaging Time - Threshold (Atn, hours)	262800	ASC NEPM

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk	Chronic TC Air	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC- Background)	Estimated Concentration in Air - Maximum anywhere (Ca)	Inhalation Exposure Concentration - NonThreshold	Inhalation Exposure Concentration - Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/m ³) ⁻¹	(mg/m ³)		(mg/m ³)	(mg/m ³)	(mg/m ³)	(mg/m ³)	(unitless)		(unitless)	
Hydrogen chloride (HCl)		2.6E-02	0%	2.6E-02	1.18E-05	1.4E-06	3.2E-06	--		0.000125	2%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	1.97E-06	2.3E-07	5.4E-07	--		0.0000186	0%
Ammonia		3.2E-01	0%	3.2E-01	1.97E-05	2.3E-06	5.4E-06	--		0.0000169	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	2.34E-08	2.7E-09	6.4E-09	--		0.001600	23%
Thallium (Tl)		7.0E-04	10%	6.3E-04	1.61E-08	1.9E-09	4.4E-09	--		0.0000698	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	3.95E-08	4.6E-09	1.1E-08	--		0.0000541	1%
Antimony (Sb)		3.0E-04	0%	3.0E-04	4.16E-08	4.9E-09	1.1E-08	--		0.0000380	1%
Arsenic (As)		6.7E-05	0%	6.7E-05	1.82E-08	2.1E-09	5.0E-09	--		0.0000745	1%
Lead (Pb)		5.0E-04	0%	5.0E-04	2.38E-07	2.8E-08	6.5E-08	--		0.0001302	2%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	5.94E-08	7.0E-09	1.6E-08	--		0.00378	55%
Cobalt (Co)		1.0E-04	0%	1.0E-04	1.47E-08	1.7E-09	4.0E-09	--		0.0000404	1%
Copper (Cu)		4.9E-01	60%	2.0E-01	5.20E-08	6.1E-09	1.4E-08	--		0.000000727	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	8.89E-08	1.0E-08	2.4E-08	--		0.000203	3%
Nickel (Ni)		5.9E-05	10%	5.3E-05	7.15E-08	8.4E-09	2.0E-08	--		0.000369	5%
Vanadium (V)		1.0E-04	0%	1.0E-04	7.37E-09	8.7E-10	2.0E-09	--		0.0000202	0%
Dioxin-like compounds		8.1E-09	54%	3.7E-09	1.18E-13	1.4E-14	3.2E-14	--		0.00000875	0%
PAHs	6.0E-01				3.95E-13	4.6E-14	1.1E-13	2.8E-14			0%
PFAS (assumed as PFOS)		7.0E-05	0%	7.0E-05	1.50E-11	1.8E-12	4.1E-12	--		0.000000588	0%
Benzene	6.0E-03	3.0E-02	54%	1.4E-02	1.97E-05	2.3E-06	5.4E-06	1.4E-8		0.000391	6%
Formaldehyde		1.1E-02	30%	7.7E-03	1.97E-05	2.3E-06	5.4E-06	--		0.000701	10%

Risk Index with VOCs as Benzene	1.4E-08	0.00688
Risk Index with VOCs as Formaldehyde	2.8E-14	0.00719

Calculation of Concentrations in Soil

$$C_s = \frac{DR \cdot [1 - e^{-k \cdot t}]}{d \cdot \rho \cdot k} \cdot 1000 \quad (\text{mg/kg}) \quad \text{ref: Stevens B. (1991)}$$

where:

DR= Particle deposition rate (mg/m²/year)

K = Chemical-specific soil-loss constant (1/year) = ln(2)/T_{0.5}

T_{0.5} = Chemical half-life in soil (years)

t = Accumulation time (years)

d = Soil mixing depth (m)

ρ = Soil bulk-density (g/m³)

1000 = Conversion from g to kg

General Parameters		Surface (for direct contact)	Depth (for agricultural pathways)	
Soil bulk density (ρ)	g/m ³	1600000	1600000	Default for fill materials
General mixing depth (d)	m	0.01	0.15	As per OEHHHA (2015) guidance
Duration of deposition (T)	years	70	70	As per OEHHHA (2015) guidance

Chemical-specific Inputs and calculations					
Chemical	Half-life in soil (years)	Loss constant (K) per year	Deposition Rate (DR) mg/m ² /year	Surface Concentration in Soil mg/kg	Agricultural Concentration in Soil mg/kg
Cadmium (Cd)	273973	2.5E-06	6.80E-05	3.0E-04	2.0E-05
Thallium (Tl)	273973	2.5E-06	4.69E-05	2.1E-04	1.4E-05
Mercury (Hg)	273973	2.5E-06	1.15E-04	5.0E-04	3.4E-05
Antimony (Sb)	273973	2.5E-06	1.21E-04	5.3E-04	3.5E-05
Arsenic (As)	273973	2.5E-06	5.30E-05	2.3E-04	1.5E-05
Lead (Pb)	273973	2.5E-06	6.90E-04	3.0E-03	2.0E-04
Chromium (Cr VI assumed)	273973	2.5E-06	1.73E-04	7.6E-04	5.0E-05
Cobalt (Co)	273973	2.5E-06	4.29E-05	1.9E-04	1.3E-05
Copper (Cu)	273973	2.5E-06	1.52E-04	6.6E-04	4.4E-05
Manganese (Mn)	273973	2.5E-06	2.59E-04	1.1E-03	7.5E-05
Nickel (Ni)	273973	2.5E-06	2.08E-04	9.1E-04	6.1E-05
Vanadium (V)	273973	2.5E-06	2.15E-05	9.4E-05	6.3E-06
Dioxin-like compounds	15	4.6E-02	3.45E-10	4.5E-10	3.0E-11
PAHs	1.18	6.9E-02	1.15E-09	1.0E-09	6.9E-11
PFAS (assumed as PFOS)	273973	2.5E-06	4.37E-08	1.9E-07	1.3E-08

Half-life in soil for dioxins: 9-15 years in surface soils; 25-100 years in subsurface soils (ATSDR 1998, DEH 2004)

Half-life in soil for metals, PAHs: OEHHHA 2015

Half-life in soil for PFAS assumed same as metals - i.e. no degradation at all but these chemicals do degrade slowly - this is conservative

Exposure to Chemicals via Incidental Ingestion of Soil - Maximum commercial

$$\text{Daily Chemical Intake}_{IS} = C_S \cdot \frac{IR_S \cdot FI \cdot CF \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults

Ingestion Rate (IRs, mg/day)	50	ASC NEPM
Fraction Ingested from Source (FI, unitless)	100%	All of daily soil intake occurs from site
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	29	Time at one residence as adult as per ASC NEPM
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.0E-04	8.8E-11	2.1E-10	--		6.64E-07	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	2.1E-04	6.1E-11	1.5E-10	--		8.15E-07	7%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	5.0E-04	1.5E-10	3.6E-10	--		1.50E-06	12%
Antimony (Sb)		8.6E-04		8.6E-04	100%	5.3E-04	1.6E-10	3.8E-10	--		4.41E-07	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	2.3E-04	6.9E-11	1.7E-10	--		1.65E-07	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	3.0E-03	8.9E-10	2.2E-09	--		7.19E-06	60%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	7.6E-04	2.2E-10	5.4E-10	--		6.67E-07	6%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.9E-04	5.6E-11	1.3E-10	--		1.20E-07	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	6.6E-04	2.0E-10	4.7E-10	--		8.45E-09	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.1E-03	3.3E-10	8.1E-10	--		1.16E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	9.1E-04	2.7E-10	6.5E-10	--		1.36E-07	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	9.4E-05	2.8E-11	6.7E-11	--		3.35E-08	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	4.5E-10	1.3E-16	3.2E-16	--		3.03E-07	3%
PAHs	2.3E-01				100%	1.0E-09	3.0E-16	7.3E-16	7.1E-17		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	100%	1.9E-07	5.7E-14	1.4E-13	--		8.54E-09	0%
TOTAL									7.1E-17		0.0000121	

Exposure to Chemicals via Incidental Ingestion of Soil - Maximum commercial

$$\text{Daily Chemical Intake}_{IS} = C_S \cdot \frac{IR_S \cdot FI \cdot CF \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Young Children

Ingestion Rate (IRs, mg/day)	100	ASC NEPM
Fraction Ingested from Source (FI, unitless)	100%	All of daily soil intake occurs from site
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.0E-04	1.7E-10	2.0E-09	--		6.20E-06	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	2.1E-04	1.2E-10	1.4E-09	--		7.60E-06	7%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	5.0E-04	2.9E-10	3.4E-09	--		1.40E-05	12%
Antimony (Sb)		8.6E-04		8.6E-04	100%	5.3E-04	3.0E-10	3.5E-09	--		4.11E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	2.3E-04	1.3E-10	1.5E-09	--		1.54E-06	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	3.0E-03	1.7E-09	2.0E-08	--		6.71E-05	60%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	7.6E-04	4.3E-10	5.0E-09	--		6.23E-06	6%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.9E-04	1.1E-10	1.3E-09	--		1.12E-06	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	6.6E-04	3.8E-10	4.4E-09	--		7.89E-08	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.1E-03	6.5E-10	7.5E-09	--		1.08E-07	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	9.1E-04	5.2E-10	6.1E-09	--		1.27E-06	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	9.4E-05	5.4E-11	6.3E-10	--		3.13E-07	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	4.5E-10	2.6E-16	3.0E-15	--		2.83E-06	3%
PAHs	2.3E-01				100%	1.0E-09	5.9E-16	6.9E-15	1.4E-16		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	100%	1.9E-07	1.1E-13	1.3E-12	--		7.97E-08	0%
TOTAL									1.4E-16		0.000113	

Dermal Exposure to Chemicals via Contact with Soil - Maximum commercial

$$\text{Daily Chemical Intake}_{DS} = C_S \cdot \frac{SA_S \cdot AF \cdot FE \cdot ABS \cdot CF \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults		
Surface Area (SAs, cm ²)	6300	Exposed skin surface area for adults as per ASC NEPM
Adherence Factor (AF, mg/cm ²)	0.5	Default as per ASC NEPM
Fraction of Day Exposed	1	Assume skin is washed after 24 hours
Conversion Factor (CF)	1.E-06	Conversion of units
Dermal absorption (ABS, unitless)	Chemical-specific (as below)	
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	29	Time at one residence as adult as per ASC NEPM
Body Weight (BW, kg)	70	For male and females combined (ASC NEPM)
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

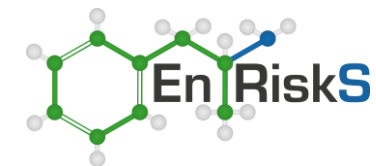
Key Chemical	Toxicity Data					Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)	Dermal Absorption (ABS)		Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	3.0E-04	5.5E-12	1.3E-11	--	--	4.2E-08	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04		2.1E-04			--	--	--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		5.0E-04			--	--	--	
Antimony (Sb)		8.6E-04		8.6E-04		5.3E-04			--	--	--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	2.3E-04	2.2E-11	5.2E-11	--	--	5.2E-08	8%
Lead (Pb)		6.0E-04	50%	3.0E-04		3.0E-03			--	--	--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		7.6E-04			--	--	--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		1.9E-04			--	--	--	
Copper (Cu)		1.4E-01	60%	5.6E-02		6.6E-04			--	--	--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		1.1E-03			--	--	--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		9.1E-04			--	--	--	
Vanadium (V)		2.0E-03		2.0E-03		9.4E-05			--	--	--	
Dioxin-like compounds		2.3E-09	54%	1.1E-09	0.03	4.5E-10	2.5E-16	6.1E-16	--	--	5.7E-07	86%
PAHs	2.3E-01				0.06	1.0E-09	1.2E-15	2.8E-15	2.7E-16	--	--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05		1.9E-07			--	--	--	
TOTAL									2.7E-16		6.7E-07	

Dermal Exposure to Chemicals via Contact with Soil - Maximum commercial

$$\text{Daily Chemical Intake}_{DS} = C_S \cdot \frac{SA_S \cdot AF \cdot FE \cdot ABS \cdot CF \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Young Children		
Surface Area (SAs, cm ²)	2700	Exposed skin surface area for young children as per ASC NEPM
Adherence Factor (AF, mg/cm ²)	0.5	Default as per ASC NEPM
Fraction of Day Exposed	1	Assume skin is washed after 24 hours
Conversion Factor (CF)	1.E-06	Conversion of units
Dermal absorption (ABS, unitless)	Chemical-specific (as below)	
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data					Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)	Dermal Absorption (ABS)		Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	3.0E-04	2.3E-12	2.7E-11	--		8.4E-08	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04		2.1E-04			--		--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		5.0E-04			--		--	
Antimony (Sb)		8.6E-04		8.6E-04		5.3E-04			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	2.3E-04	8.9E-12	1.0E-10	--		1.0E-07	8%
Lead (Pb)		6.0E-04	50%	3.0E-04		3.0E-03			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		7.6E-04			--		--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		1.9E-04			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		6.6E-04			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		1.1E-03			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		9.1E-04			--		--	
Vanadium (V)		2.0E-03		2.0E-03		9.4E-05			--		--	
Dioxin-like compounds		2.3E-09	54%	1.1E-09	0.03	4.5E-10	1.0E-16	1.2E-15	--		1.1E-06	86%
PAHs	2.3E-01				0.06	1.0E-09	4.8E-16	5.6E-15	1.1E-16		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05		1.9E-07			--		--	
TOTAL									1.1E-16		1.3E-06	



Assuming this location could be changed to residential

Inhalation - gases and particulates

Maximum commercial should land use be changed to residential

$$\text{Inhalation Exposure Concentration} = Ca * \frac{ET * FI * EF * ED}{AT} \quad (\text{mg/m}^3) \text{ for gases}$$

$$\text{Inhalation Exposure Concentration} = Ca * \frac{ET * FI * DRF * EF * ED}{AT} \quad (\text{mg/m}^3) \text{ for chemicals attached to particles}$$

Parameters Relevant to Quantification of Community Exposures - Residents		
Exposure Time at Home (ET, hr/day)	24	Assume residents at home or on property 24 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Assume resident at the same property
Dust lung retention factor (unitless)	0.375	Percentage of respirable dust that is small enough to reach and be retained in the lungs (ASC NEPM) - NA for gases
Exposure Frequency - normal conditions (EF, days/yr)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	35	As per ASC NEPM
Averaging Time - NonThreshold (Atc, hours)	613200	ASC NEPM
Averaging Time - Threshold (Atn, hours)	306600	ASC NEPM

Key Chemical	Toxicity Data				Concentration		Daily Exposure		Calculated Risk		
	Inhalation Unit Risk (mg/m ³) ⁻¹	Chronic TC Air (mg/m ³)	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background) (mg/m ³)	Estimated Concentration in Air - Maximum anywhere (Ca) (mg/m ³)	Inhalation Exposure Concentration - NonThreshold (mg/m ³)	Inhalation Exposure Concentration - Threshold (mg/m ³)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Hydrogen chloride (HCl)		2.6E-02	0%	2.6E-02	1.18E-05	5.9E-06	1.2E-05	--		0.000455	2%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	1.97E-06	9.9E-07	2.0E-06	--		0.0000680	0%
Ammonia		3.2E-01	0%	3.2E-01	1.97E-05	9.9E-06	2.0E-05	--		0.0000616	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	2.34E-08	1.2E-08	2.3E-08	--		0.00584	24%
Thallium (Tl)		7.0E-04	10%	6.3E-04	1.61E-08	8.0E-09	1.6E-08	--		0.00002549	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	3.95E-08	2.0E-08	3.9E-08	--		0.0001974	1%
Antimony (Sb)		3.0E-04	0%	3.0E-04	4.16E-08	2.1E-08	4.2E-08	--		0.0001387	1%
Arsenic (As)		6.7E-05	0%	6.7E-05	1.82E-08	9.1E-09	1.8E-08	--		0.000272	1%
Lead (Pb)		5.0E-04	0%	5.0E-04	2.38E-07	1.2E-07	2.4E-07	--		0.000475	2%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	5.94E-08	3.0E-08	5.9E-08	--		0.01381	57%
Cobalt (Co)		1.0E-04	0%	1.0E-04	1.47E-08	7.4E-09	1.5E-08	--		0.0001474	1%
Copper (Cu)		4.9E-01	60%	2.0E-01	5.20E-08	2.6E-08	5.2E-08	--		0.00000265	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	8.89E-08	4.4E-08	8.9E-08	--		0.000741	3%
Nickel (Ni)		5.9E-05	10%	5.3E-05	7.15E-08	3.6E-08	7.2E-08	--		0.001347	6%
Vanadium (V)		1.0E-04	0%	1.0E-04	7.37E-09	3.7E-09	7.4E-09	--		0.0000737	0%
Dioxin-like compounds		8.1E-09	54%	3.7E-09	1.18E-13	5.9E-14	1.2E-13	--		0.0000319	0%
PAHs	6.0E-01	0.0E+00	0%	0.0E+00	3.95E-13	2.0E-13	3.9E-13	1.2E-13		--	
PFAS (assumed as PFOS)		7.0E-05	0%	7.0E-05	1.50E-11	7.5E-12	1.5E-11	--		0.0000002146	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	1.97E-05	9.9E-06	2.0E-05	5.9E-8		0.000730	3%
Formaldehyde		1.1E-02	30%	7.7E-03	1.97E-05	9.9E-06	2.0E-05	--		0.002560	10%

Risk Index with VOCs as Benzene	5.9E-08	0.02442
Risk Index with VOCs as Formaldehyde	1.2E-13	0.0262

Calculation of Concentrations in Plants

ref: Stevens B. (1991)

Uptake Due to Deposition in Aboveground Crops	Uptake via Roots from Soil
$C_p = \frac{DR \cdot F \cdot [1 - e^{-k \cdot t}]}{Y \cdot k} \quad (\text{mg/kg plant - wet weight})$ where: DR= Particle deposition rate for accidental release (mg/m²/day) F= Fraction for the surface area of plant (unitless) k= Chemical-specific soil-loss constant (1/years) = ln(2)/T_{0.5} T_{0.5}= Chemical half-life as particulate on plant (days) t= Deposition time (days) Y= Crop yield (kg/m²)	$C_{rp} = C_s \cdot RUF \quad (\text{mg/kg plant - wet weight})$ where: Cs = Concentration of persistent chemical in soil assuming 15cm mixing depth within gardens, calculated using Soil Equation for each chemical assessed (mg/kg) RUF = Root uptake factor which differs for each Chemical (unitless)

General Parameters	Units	Value
Crop		Edible crops
Crop Yield (Y)	kg/m ²	2
Deposition Time (t)	days	70
Plant Interception fraction (F)	unitless	0.051

Chemical-specific Inputs and calculations - Maximum commercial							
Chemical	Half-life on plant (T _{0.5})#		Loss constant (k) & Deposition Rate (DR)	Aboveground Produce Concentration via Deposition	Root Uptake Factor (RUF)\$	Soil Concentration (Cs)	Below Ground Produce Concentration
	days	per day	mg/m ² /day	mg/kg ww	unitless	mg/kg	mg/kg ww
Cadmium (Cd)	14	0.05	1.9E-07	9.3E-08	0.125	2.0E-05	2.5E-06
Thallium (Tl)	14	0.05	1.3E-07	6.4E-08	0.001	1.4E-05	1.4E-08
Mercury (Hg)	14	0.05	3.1E-07	1.6E-07	0.225	3.4E-05	7.5E-06
Antimony (Sb)	14	0.05	3.3E-07	1.7E-07	0.05	3.5E-05	8.0E-06
Arsenic (As)	14	0.05	1.5E-07	7.2E-08	0.01	1.5E-05	7.7E-07
Lead (Pb)	14	0.05	1.9E-06	9.4E-07	0.01	2.0E-04	2.0E-06
Chromium (Cr VI assumed)	14	0.05	4.7E-07	2.4E-07	0.00188	5.0E-05	5.0E-07
Cobalt (Co)	14	0.05	1.2E-07	5.9E-08	0.005	1.3E-05	2.4E-08
Copper (Cu)	14	0.05	4.2E-07	2.1E-07	0.1	4.4E-05	2.2E-07
Manganese (Mn)	14	0.05	7.1E-07	3.5E-07	0.0625	7.5E-05	7.5E-06
Nickel (Ni)	14	0.05	5.7E-07	2.9E-07	0.015	6.1E-05	3.8E-06
Vanadium (V)	14	0.05	5.9E-08	2.9E-08	0.00137	6.3E-06	9.4E-08
Dioxin-like compounds	14	0.05	9.5E-13	4.7E-13	0.000876	3.0E-11	2.6E-14
PAHs	14	0.05	3.1E-12	1.6E-12	0.00214	6.9E-11	1.5E-13
PFAS (assumed as PFOS)	14	0.05	1.2E-10	6.0E-11	0.011	1.3E-08	1.4E-10

\$ Root uptake factors from RAIS (soil to wet weight of plant)

& Loss constant is 1/half life

Half life on plant taken from Stevens 1991 which notes that particles deposit onto the surface of plants but then over time are lost due to weathering (wind, rain etc) - the half life for the amount of time these particles remain on the surface of the plant (and so may be present in the produce) is 14 days

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables - Maximum commercial

$$\text{Daily chemical intake} = C_A \times \frac{IR_P \times \%A \times FI \times ME \times EF \times ED}{BW \times AT} + C_R \times \frac{IR_P \times \%R \times FI \times ME \times ED \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Produce (IRp) (kg/day)	0.4	Total fruit and vegetable consumption rate for adults as per ASC NEPM
Proportion of total intake from aboveground crops (%A)	73%	Proportions as per ASC NEPM
Proportion of total intake from root crops (%R)	27%	Proportions as per ASC NEPM
Fraction ingested that is homegrown (%)	10%	Relevant to rural fringe
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	29	Time at one residence as adult as per ASC NEPM
Body Weight (BW, kg)	70	For male and females combined ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration (mg/kg wet weight)	Root crops concentrations (mg/kg wet weight)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)				NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	9.3E-08	2.5E-06	1.7E-10	4.2E-10	—		1.32E-06	12%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	6.4E-08	1.4E-08	1.2E-11	2.9E-11	—		1.60E-07	1%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.6E-07	7.5E-06	5.1E-10	1.2E-09	—		5.12E-06	46%
Antimony (Sb)		8.6E-04		8.6E-04	100%	1.7E-07	8.0E-06	5.4E-10	1.3E-09	—		1.51E-06	13%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	7.2E-08	7.7E-07	6.2E-11	1.5E-10	—		1.49E-07	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	9.4E-07	2.0E-06	2.9E-10	7.0E-10	—		2.35E-06	21%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.4E-07	5.0E-07	7.3E-11	1.8E-10	—		2.18E-07	2%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	5.9E-08	2.4E-08	1.2E-11	2.8E-11	—		2.51E-08	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.1E-07	2.2E-07	5.0E-11	1.2E-10	—		2.15E-09	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.5E-07	7.5E-06	5.4E-10	1.3E-09	—		1.87E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.9E-07	3.8E-06	2.9E-10	7.1E-10	—		1.47E-07	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	2.9E-08	9.4E-08	1.1E-11	2.7E-11	—		1.34E-08	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	4.7E-13	2.6E-14	8.3E-17	2.0E-16	—		1.90E-07	2%
PAHs	2.3E-01				100%	1.6E-12	1.5E-13	2.8E-16	6.8E-16	6.5E-17		—	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	100%	6.0E-11	1.4E-10	1.9E-14	4.7E-14	—		2.9E-09	0%
TOTAL										6.5E-17		0.000011	

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables - Maximum commercial

$$\text{Daily chemical intake} = C_A \times \frac{IR_P \times \%A \times FI \times ME \times EF \times ED}{BW \times AT} + C_R \times \frac{IR_P \times \%R \times FI \times ME \times ED \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Young children

Ingestion Rate of Produce (IRp) (kg/day)	0.28	Total fruit and vegetable consumption rate for children as per ASC NEPM
Proportion of total intake from aboveground crops (%A)	84%	Proportions as per ASC NEPM
Proportion of total intake from root crops (%R)	16%	Proportions as per ASC NEPM
Fraction ingested that is homegrown (%)	10%	Relevant to rural fringe
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration (mg/kg wet weight)	Root crops concentrations (mg/kg wet weight)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)				NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	9.3E-08	2.5E-06	7.6E-11	8.9E-10	–		2.77E-06	11%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	6.4E-08	1.4E-08	9.0E-12	1.0E-10	–		5.82E-07	2%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.6E-07	7.5E-06	2.1E-10	2.5E-09	–		1.04E-05	40%
Antimony (Sb)		8.6E-04		8.6E-04	100%	1.7E-07	8.0E-06	2.3E-10	2.6E-09	–		3.07E-06	12%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	7.2E-08	7.7E-07	3.0E-11	3.4E-10	–		3.44E-07	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	9.4E-07	2.0E-06	1.8E-10	2.1E-09	–		6.94E-06	27%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.4E-07	5.0E-07	4.5E-11	5.2E-10	–		6.44E-07	2%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	5.9E-08	2.4E-08	8.5E-12	9.9E-11	–		8.84E-08	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.1E-07	2.2E-07	3.3E-11	3.9E-10	–		6.98E-09	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.5E-07	7.5E-06	2.4E-10	2.8E-09	–		4.01E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.9E-07	3.8E-06	1.4E-10	1.6E-09	–		3.30E-07	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	2.9E-08	9.4E-08	6.3E-12	7.4E-11	–		3.70E-08	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	4.7E-13	2.6E-14	6.4E-17	7.5E-16	–		7.07E-07	3%
PAHs	2.3E-01				100%	1.6E-12	1.5E-13	2.1E-16	2.5E-15	5.0E-17		–	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	100%	6.0E-11	1.4E-10	1.2E-14	1.4E-13	–		8.5E-09	0%
TOTAL										5.0E-17		0.00002598	

Calculation of Concentrations in Eggs

Uptake in to chicken eggs

$$C_E = (FI \times IR_C \times C + IR_S \times C_S \times B) \times TFE \quad (\text{mg/kg egg} - \text{wet weight})$$

where:

FI = Fraction of pasture/crop ingested by chickens each day (unitless)

IR_C = Ingestion rate of pasture/crop by chicken each day (kg/day)

C = Concentration of chemical in grain/crop eaten by chicken (mg/kg)

IR_S = Ingestion rate of soil by chickens each day (kg/day)

C_S = Concentration in soil the chickens ingest (mg/kg)

B = Bioavailability of soil ingested by chickens (%)

TFE = Transfer factor from ingestion to eggs (day/kg)

General Parameters	Units	Value
FI (fraction of crops ingested from property)		1
IR _C (ingestion rate of crops)	kg/day	0.12
IR _S (ingestion rate of soil)	kg/day	0.01
B (bioavailability)	%	100%

Assume 100% of crops consumed by chickens is grown in the same soil

Assumed ingestion rate from OEHHa 2015 (assume concentration the same as predicted for aboveground crops)

USEPA (2005) (Ag Victoria recommendation)

Chemical-specific Inputs and calculations - Maximum commercial

Chemical	Concentration in crops ingested by chickens mg/kg ww	Soil Concentration - Agriculture (C _S) mg/kg	Transfer factor to eggs day/kg	Egg Concentration mg/kg ww
Cadmium (Cd)	9.3E-08	2.0E-05	1.0E-02	2.1E-09
Thallium (Tl)	6.4E-08	1.4E-05	3.8E-02	5.5E-09
Mercury (Hg)	1.6E-07	3.4E-05	8.0E-01	2.8E-07
Antimony (Sb)	1.7E-07	3.5E-05	1.7E-01	6.4E-08
Arsenic (As)	7.2E-08	1.5E-05	7.0E-02	1.1E-08
Lead (Pb)	9.4E-07	2.0E-04	4.0E-02	8.5E-08
Chromium (Cr VI assumed)	2.4E-07	5.0E-05	3.8E-02	2.0E-08
Cobalt (Co)	5.9E-08	1.3E-05	3.8E-02	5.0E-09
Copper (Cu)	2.1E-07	4.4E-05	3.8E-02	1.8E-08
Manganese (Mn)	3.5E-07	7.5E-05	3.8E-02	3.0E-08
Nickel (Ni)	2.9E-07	6.1E-05	2.0E-02	1.3E-08
Vanadium (V)	2.9E-08	6.3E-06	3.8E-02	2.5E-09
Dioxin-like compounds	4.7E-13	3.0E-11	1.0E+01	3.6E-12
PAHs	1.6E-12	6.9E-11	3.0E-03	2.6E-15
PFAS (assumed as PFOS)	6.0E-11	1.3E-08	7.0E-02	9.4E-12

OEHHa (2012)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

OEHHa (2012)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

OEHHa (2012)

OEHHa (2012)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

OEHHa (2012)

Geometric mean transfer factor for metals, transfer to eggs (Leeman et al 2007)

OEHHa (2012)

OEHHa (2012)

RAIS (2024)

Transfer factors from OEHHa 2012 unless otherwise noted

Exposure to Chemicals via Ingestion of Eggs - Maximum commercial

$$\text{Daily chemical intake} = C_E \times \frac{IR_E \times FI \times ME \times EF \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults

Ingestion Rate of Eggs (IRE) (kg/day)	0.059	Ingestion rate of eggs relevant for adults as per FSANZ (2017) (P90 for consumers - i.e. high end consumer)
Fraction ingested that is homegrown (%)	100%	Assume all eggs consumed are from backyard chickens
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	29	Time at one residence as adult as per ASC NEPM
Body Weight (BW, kg)	70	ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration (mg/kg wet weight)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.1E-09	7.3E-13	1.8E-12	--		5.5E-09	0%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	5.5E-09	1.9E-12	4.6E-12	--		2.6E-08	1%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	2.8E-07	9.9E-11	2.4E-10	--		1.0E-06	24%
Antimony (Sb)		8.6E-04		8.6E-04	100%	6.4E-08	2.2E-11	5.4E-11	--		6.2E-08	1%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.1E-08	4.0E-12	9.6E-12	--		9.6E-09	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	8.5E-08	3.0E-11	7.2E-11	--		2.4E-07	6%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.0E-08	7.1E-12	1.7E-11	--		2.1E-08	1%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	5.0E-09	1.8E-12	4.2E-12	--		3.8E-09	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.8E-08	6.2E-12	1.5E-11	--		2.7E-10	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.0E-08	1.1E-11	2.6E-11	--		3.6E-10	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.3E-08	4.5E-12	1.1E-11	--		2.3E-09	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	2.5E-09	8.8E-13	2.1E-12	--		1.1E-09	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	3.6E-12	1.2E-15	3.0E-15	--		2.8E-06	67%
PAHs	2.3E-01				100%	2.6E-15	9.2E-19	2.2E-18	2.1E-19		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	100%	9.4E-12	3.3E-15	8.0E-15	--		5.0E-10	0%

TOTAL **2.1E-19** **0.000004200**

Exposure to Chemicals via Ingestion of Eggs - Maximum commercial

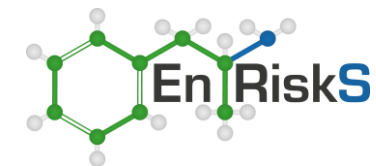
$$\text{Daily chemical intake} = C_E \times \frac{IR_E \times FI \times ME \times EF \times ED}{BW \times AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Young children

Ingestion Rate of Eggs (IRE) (kg/day)	0.036	Ingestion rate of eggs relevant for young children as per FSANZ (2017) (P90 for consumers - i.e. high end consumer)
Fraction ingested that is homegrown (%)	100%	Assume all eggs consumed are from backyard chickens
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per ASC NEPM
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration (mg/kg wet weight)	Daily Intake		Calculated Risk		
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.1E-09	4.3E-13	5.0E-12	--		1.6E-08
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	5.5E-09	1.1E-12	1.3E-11	--		7.3E-08
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	2.8E-07	5.8E-11	6.8E-10	--		2.8E-06
Antimony (Sb)		8.6E-04		8.6E-04	100%	6.4E-08	1.3E-11	1.5E-10	--		1.8E-07
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.1E-08	2.3E-12	2.7E-11	--		2.7E-08
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	8.5E-08	1.8E-11	2.0E-10	--		6.8E-07
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.0E-08	4.2E-12	4.9E-11	--		6.0E-08
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	5.0E-09	1.0E-12	1.2E-11	--		1.1E-08
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.8E-08	3.6E-12	4.3E-11	--		7.6E-10
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.0E-08	6.2E-12	7.3E-11	--		1.0E-09
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.3E-08	2.6E-12	3.1E-11	--		6.4E-09
Vanadium (V)		2.0E-03		2.0E-03	100%	2.5E-09	5.2E-13	6.0E-12	--		3.0E-09
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	3.6E-12	7.3E-16	8.5E-15	--		8.1E-06
PAHs	2.3E-01				100%	2.6E-15	5.4E-19	6.3E-18	1.3E-19		--
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	100%	9.4E-12	1.9E-15	2.3E-14	--		1.4E-09

TOTAL **1.3E-19** **0.000012**



On-site Education Centre Workers and Visitors

Predicted ground level concentrations - chronic exposures - increment

	Air Concentration - annual average ($\mu\text{g}/\text{m}^3$)	Air Concentration - annual average (mg/m^3)	Cumulative air concentration (mg/m^3)	Exposure pathways		
Key Chemicals	Maximum on-site	Maximum on-site	Maximum on-site	inhalation	soil ingestion	soil - dermal
Hydrogen chloride (HCl)	5.23E-03	5.23E-06		✓	*	*
Hydrogen fluoride (HF)	8.71E-04	8.71E-07		✓	*	*
Ammonia	8.71E-03	8.71E-06		✓	*	*
Cadmium (Cd)	1.03E-05	1.03E-08		✓	✓	✓
Thallium (Tl)	7.09E-06	7.09E-09		✓	✓	✓
Mercury (Hg)	1.74E-05	1.74E-08		✓	✓	✓
Antimony (Sb)	1.84E-05	1.84E-08		✓	✓	✓
Arsenic (As)	8.04E-06	8.04E-09	0.00350870765	✓	✓	✓
Lead (Pb)	1.05E-04	1.05E-07		✓	✓	✓
Chromium (Cr VI assumed)	2.62E-05	2.62E-08	0.00000002717	✓	✓	✓
Cobalt (Co)	6.51E-06	6.51E-09		✓	✓	✓
Copper (Cu)	2.30E-05	2.30E-08	0.0000000347	✓	✓	✓
Manganese (Mn)	3.93E-05	3.93E-08	0.0000000451	✓	✓	✓
Nickel (Ni)	3.16E-05	3.16E-08	0.0000000350	✓	✓	✓
Vanadium (V)	3.26E-06	3.26E-09		✓	✓	✓
Dioxin-like compounds	5.22E-11	5.22E-14		✓	✓	✓
PAHs	1.74E-10	1.74E-13		✓	✓	✓
PFAS (assumed as PFOS)	6.63E-09	6.63E-12				
Benzene	8.71E-03	8.71E-06	0.00350871	✓	*	*
Formaldehyde	8.71E-03	8.71E-06		✓	*	*

	Deposition Rate - annual average ($\text{ng}/\text{m}^2/\text{year}$)	Deposition Rate - annual average ($\text{mg}/\text{m}^2/\text{year}$)
Key Chemicals	Maximum on-site	Maximum on-site
Hydrogen chloride (HCl)	NR	NR
Hydrogen fluoride (HF)	NR	NR
Ammonia	NR	NR
Cadmium (Cd)	23.48	0.0000235
Thallium (Tl)	16.19	0.00001619
Mercury (Hg)	39.67	0.00003967
Antimony (Sb)	41.87	0.00004187
Arsenic (As)	18.28	0.00001828
Lead (Pb)	238.28	0.00023828
Chromium (Cr VI assumed)	59.69	0.00005969
Cobalt (Co)	14.81	0.00001481
Copper (Cu)	52.28	0.00005228
Manganese (Mn)	89.30	0.00008930
Nickel (Ni)	71.95	0.00007195
Vanadium (V)	7.40	0.00000740
Dioxin-like compounds	1.19E-04	1.19E-10
PAHs	3.97E-04	3.97E-10
PFAS (assumed as PFOS)	1.51E-02	1.51E-08
Benzene	NR	NR
Formaldehyde	NR	NR

Inhalation - gases and particulates Maximum on-site - visitors centre

$$\text{Inhalation Exposure Concentration} = Ca * \frac{ET * FI * EF * ED}{AT} \quad (\text{mg/m}^3) \text{ for gases}$$

$$\text{Inhalation Exposure Concentration} = Ca * \frac{ET * FI * DRF * EF * ED}{AT} \quad (\text{mg/m}^3) \text{ for chemicals attached to particles}$$

Parameters Relevant to Quantification of Community Exposures - Commercial/Industrial		
Exposure Time at Home (ET, hr/day)	10	Assume workers at work site 10 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Only exposed at the work site
Dust lung retention factor (unitless)	0.375	Percentage of respirable dust that is small enough to reach and be retained in the lungs (ASC NEPM) - NA for gasses
Exposure Frequency - normal conditions (EF, days/yr)	240	Days at work (ASC NEPM)
Exposure Duration (ED, years)	30	ASC NEPM
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	262800	US EPA 2009

Key Chemical	Toxicity Data				Concentration		Daily Exposure		Calculated Risk		
	Inhalation Unit Risk (mg/m ³) ⁻¹	Chronic TC Air (mg/m ³)	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background) (mg/m ³)	Estimated Concentration in Air - Maximum anywhere (Ca) (mg/m ³)	Inhalation Exposure Concentration - NonThreshold (mg/m ³)	Inhalation Exposure Concentration - Threshold (mg/m ³)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Hydrogen chloride (HCl)		2.6E-02	0%	2.6E-02	5.2E-06	6.1E-07	1.4E-06	--		5.51E-05	2%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	8.7E-07	1.0E-07	2.4E-07	--		8.23E-06	0%
Ammonia		3.2E-01	0%	3.2E-01	8.7E-06	1.0E-06	2.4E-06	--		7.46E-06	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	1.0E-08	1.2E-09	2.8E-09	--		7.07E-04	24%
Thallium (Tl)		7.0E-04	10%	6.3E-04	7.1E-09	8.3E-10	1.9E-09	--		3.08E-06	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	1.7E-08	2.0E-09	4.8E-09	--		2.39E-05	1%
Antimony (Sb)		3.0E-04	0%	3.0E-04	1.8E-08	2.2E-09	5.0E-09	--		1.68E-05	1%
Arsenic (As)		6.7E-05	0%	6.7E-05	8.0E-09	9.4E-10	2.2E-09	--		3.29E-05	1%
Lead (Pb)		5.0E-04	0%	5.0E-04	1.0E-07	1.2E-08	2.9E-08	--		5.75E-05	2%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	2.6E-08	3.1E-09	7.2E-09	--		1.67E-03	57%
Cobalt (Co)		1.0E-04	0%	1.0E-04	6.5E-09	7.6E-10	1.8E-09	--		1.78E-05	1%
Copper (Cu)		4.9E-01	60%	2.0E-01	2.3E-08	2.7E-09	6.3E-09	--		3.21E-08	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	3.9E-08	4.6E-09	1.1E-08	--		8.96E-05	3%
Nickel (Ni)		5.9E-05	10%	5.3E-05	3.2E-08	3.7E-09	8.7E-09	--		1.63E-04	6%
Vanadium (V)		1.0E-04	0%	1.0E-04	3.3E-09	3.8E-10	8.9E-10	--		8.92E-06	0%
Dioxin-like compounds		8.1E-09	54%	3.7E-09	5.2E-14	6.1E-15	1.4E-14	--		3.86E-06	0%
PAHs	6.0E-01				1.7E-13	2.0E-14	4.8E-14	1.2E-14		--	
PFAS (assumed as PFOS)		7.0E-05	0%	7.0E-05	6.6E-12	7.8E-13	1.8E-12	--		2.60E-08	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	8.7E-06	1.0E-06	2.4E-06	6.1E-9		8.84E-05	3%
Formaldehyde		1.1E-02	30%	7.7E-03	8.7E-06	1.0E-06	2.4E-06	--		3.10E-04	10%

Risk Index with VOCs as Benzene	6.1E-09	0.00295
Risk Index with VOCs as Formaldehyde	1.2E-14	0.00318

Calculation of Concentrations in Soil

$$C_s = \frac{DR \cdot [1 - e^{-k \cdot t}]}{d \cdot \rho \cdot k} \cdot 1000 \quad (\text{mg/kg}) \quad \text{ref: Stevens B. (1991)}$$

where:

DR= Particle deposition rate (mg/m²/year)

K = Chemical-specific soil-loss constant (1/year) = ln(2)/T_{0.5}

T_{0.5} = Chemical half-life in soil (years)

t = Accumulation time (years)

d = Soil mixing depth (m)

ρ = Soil bulk-density (g/m³)

1000 = Conversion from g to kg

General Parameters		Surface (for direct contact)	Depth (for agricultural pathways)	
Soil bulk density (ρ)	g/m ³	1600000	1600000	Default for fill materials
General mixing depth (d)	m	0.01	0.15	As per OEHA (2015) guidance
Duration of deposition (T)	years	70	70	As per OEHA (2015) guidance

Chemical-specific Inputs and calculations - maximum anywhere					
Chemical	Half-life in soil years	Loss constant (K) per year	Deposition Rate (DR) mg/m ² /year	Surface Concentration in Soil mg/kg	Agricultural Concentration in Soil mg/kg
Cadmium (Cd)	273973	2.5E-06	2.35E-05	1.0E-04	6.8E-06
Thallium (Tl)	273973	2.5E-06	1.62E-05	7.1E-05	4.7E-06
Mercury (Hg)	273973	2.5E-06	3.97E-05	1.7E-04	1.2E-05
Antimony (Sb)	273973	2.5E-06	4.19E-05	1.8E-04	1.2E-05
Arsenic (As)	273973	2.5E-06	1.83E-05	8.0E-05	5.3E-06
Lead (Pb)	273973	2.5E-06	2.38E-04	1.0E-03	6.9E-05
Chromium (Cr VI assumed)	273973	2.5E-06	5.97E-05	2.6E-04	1.7E-05
Cobalt (Co)	273973	2.5E-06	1.48E-05	6.5E-05	4.3E-06
Copper (Cu)	273973	2.5E-06	5.23E-05	2.3E-04	1.5E-05
Manganese (Mn)	273973	2.5E-06	8.93E-05	3.9E-04	2.6E-05
Nickel (Ni)	273973	2.5E-06	7.19E-05	3.1E-04	2.1E-05
Vanadium (V)	273973	2.5E-06	7.40E-06	3.2E-05	2.2E-06
Dioxin-like compounds	15	4.6E-02	1.19E-10	1.5E-10	1.0E-11
PAHs	1.18	5.9E-01	3.97E-10	4.2E-11	2.8E-12
PFAS (assumed as PFOS)	273973	2.5E-06	1.51E-08	6.6E-08	4.4E-09

Half-life in soil for dioxins: 9-15 years in surface soils; 25-100 years in subsurface soils (ATSDR 1998, DEH 2004)

Half-life in soil for metals: OEHA 2015

Half-life in soil for PFAS assumed same as metals - i.e. no degradation at all but these chemicals do degrade slowly - this is conservative

Exposure to Chemicals via Incidental Ingestion of Soil - Maximum commercial

$$\text{Daily Chemical Intake}_{IS} = C_S \cdot \frac{IR_S \cdot FI \cdot CF \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults

Ingestion Rate (IRs, mg/day)	50	As per NEPM 2013
Fraction Ingested from Source (FI, unitless)	100%	All of daily soil intake occurs from site
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.0E-04	3.0E-11	7.3E-11	--		2.29E-07	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	7.1E-05	2.1E-11	5.1E-11	--		2.81E-07	7%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.7E-04	5.1E-11	1.2E-10	--		5.17E-07	12%
Antimony (Sb)		8.6E-04		8.6E-04	100%	1.8E-04	5.4E-11	1.3E-10	--		1.52E-07	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	8.0E-05	2.4E-11	5.7E-11	--		5.71E-08	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.0E-03	3.1E-10	7.4E-10	--		2.48E-06	60%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.6E-04	7.7E-11	1.9E-10	--		2.30E-07	6%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	6.5E-05	1.9E-11	4.6E-11	--		4.13E-08	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.3E-04	6.8E-11	1.6E-10	--		2.92E-09	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.9E-04	1.2E-10	2.8E-10	--		3.99E-09	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.1E-04	9.3E-11	2.2E-10	--		4.68E-08	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	3.2E-05	9.6E-12	2.3E-11	--		1.16E-08	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	1.5E-10	4.6E-17	1.1E-16	--		1.05E-07	3%
PAHs	2.3E-01				100%	4.2E-11	1.2E-17	3.0E-17	2.9E-18		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	100%	6.6E-08	2.0E-14	4.7E-14	--		2.95E-09	0%

TOTAL **2.9E-18** **0.00000416**

Exposure to Chemicals via Incidental Ingestion of Soil - Maximum commercial

$$\text{Daily Chemical Intake}_{IS} = C_S \cdot \frac{IR_S \cdot FI \cdot CF \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Young Children

Ingestion Rate (IRs, mg/day)	100	Assumed daily soil ingestion rate for young children, enHealth (2012)
Fraction Ingested from Source (FI, unitless)	100%	All of daily soil intake occurs from site
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.0E-04	5.9E-11	6.8E-10	--		2.14E-06	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	7.1E-05	4.0E-11	4.7E-10	--		2.62E-06	7%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.7E-04	9.9E-11	1.2E-09	--		4.82E-06	12%
Antimony (Sb)		8.6E-04		8.6E-04	100%	1.8E-04	1.0E-10	1.2E-09	--		1.42E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	8.0E-05	4.6E-11	5.3E-10	--		5.33E-07	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.0E-03	6.0E-10	6.9E-09	--		2.32E-05	60%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.6E-04	1.5E-10	1.7E-09	--		2.15E-06	6%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	6.5E-05	3.7E-11	4.3E-10	--		3.86E-07	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.3E-04	1.3E-10	1.5E-09	--		2.72E-08	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.9E-04	2.2E-10	2.6E-09	--		3.72E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.1E-04	1.8E-10	2.1E-09	--		4.37E-07	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	3.2E-05	1.9E-11	2.2E-10	--		1.08E-07	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	1.5E-10	8.8E-17	1.0E-15	--		9.75E-07	3%
PAHs	2.3E-01				100%	4.2E-11	2.4E-17	2.8E-16	5.6E-18		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	100%	6.6E-08	3.8E-14	4.4E-13	--		2.75E-08	0%
TOTAL									5.6E-18		0.0000388	

Dermal Exposure to Chemicals via Contact with Soil - Maximum commercial

$$\text{Daily Chemical Intake}_{DS} = C_S \cdot \frac{SA_S \cdot AF \cdot FE \cdot ABS \cdot CF \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults		
Surface Area (SAs, cm ²)	6300	Exposed skin surface area for adults as per NEPM (2013)
Adherence Factor (AF, mg/cm ²)	0.5	Default as per NEPM (2013)
Fraction of Day Exposed	1	Assume skin is washed after 24 hours
Conversion Factor (CF)	1.E-06	Conversion of units
Dermal absorption (ABS, unitless)	Chemical-specific (as below)	
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

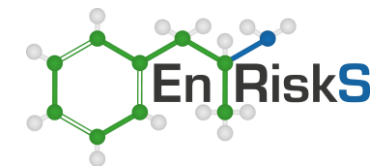
Key Chemical	Toxicity Data					Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)	Dermal Absorption (ABS)		Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	1.0E-04	1.9E-12	4.6E-12	--	--	1.4E-08	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04		7.1E-05			--	--	--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		1.7E-04			--	--	--	
Antimony (Sb)		8.6E-04		8.6E-04		1.8E-04			--	--	--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	8.0E-05	7.5E-12	1.8E-11	--	--	1.8E-08	8%
Lead (Pb)		6.0E-04	50%	3.0E-04		1.0E-03			--	--	--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		2.6E-04			--	--	--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		6.5E-05			--	--	--	
Copper (Cu)		1.4E-01	60%	5.6E-02		2.3E-04			--	--	--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		3.9E-04			--	--	--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		3.1E-04			--	--	--	
Vanadium (V)		2.0E-03		2.0E-03		3.2E-05			--	--	--	
Dioxin-like compounds		2.3E-09	54%	1.1E-09	0.03	1.5E-10	8.7E-17	2.1E-16	--	--	2.0E-07	86%
PAHs	2.3E-01				0.06	4.2E-11	4.7E-17	1.1E-16	1.1E-17			
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05		6.6E-08			--	--	--	
TOTAL									1.1E-17		2.3E-07	

Dermal Exposure to Chemicals via Contact with Soil - Maximum commercial

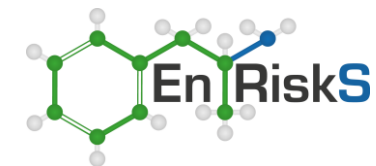
$$\text{Daily Chemical Intake}_{DS} = C_S \cdot \frac{SA_S \cdot AF \cdot FE \cdot ABS \cdot CF \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{mg/kg/day})$$

Parameters Relevant to Quantification of Exposure by Young Children		
Surface Area (SAs, cm ²)	2700	Exposed skin surface area for young children as per NEPM (2013)
Adherence Factor (AF, mg/cm ²)	0.5	Default as per NEPM (2013)
Fraction of Day Exposed	1	Assume skin is washed after 24 hours
Conversion Factor (CF)	1.E-06	Conversion of units
Dermal absorption (ABS, unitless)	Chemical-specific (as below)	
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Key Chemical	Toxicity Data					Soil Concentration (mg/kg)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)	Dermal Absorption (ABS)		Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	1.0E-04	7.9E-13	9.2E-12	--	--	2.9E-08	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04		7.1E-05			--	--	--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		1.7E-04			--	--	--	
Antimony (Sb)		8.6E-04		8.6E-04		1.8E-04			--	--	--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	8.0E-05	3.1E-12	3.6E-11	--	--	3.6E-08	8%
Lead (Pb)		6.0E-04	50%	3.0E-04		1.0E-03			--	--	--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		2.6E-04			--	--	--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		6.5E-05			--	--	--	
Copper (Cu)		1.4E-01	60%	5.6E-02		2.3E-04			--	--	--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		3.9E-04			--	--	--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		3.1E-04			--	--	--	
Vanadium (V)		2.0E-03		2.0E-03		3.2E-05			--	--	--	
Dioxin-like compounds		2.3E-09	54%	1.1E-09	0.03	1.5E-10	3.6E-17	4.2E-16	--	--	4.0E-07	86%
PAHs	2.3E-01				0.06	4.2E-11	2.0E-17	2.3E-16	4.6E-18			
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05		6.6E-08			--	--	--	
TOTAL									4.6E-18		4.6E-07	



Rainwater Tanks



Maximum Off-Site

Calculation of Concentrations in Rainwater tank

$$CW = DM / (VR \times Kd \times \rho) \quad (\text{mg/L})$$

where:

DM = Mass of dust deposited on roof each year (mg) = DR x Area
 DR = Deposition rate from model (mg/m²/year)
 Area = Area of roof (m²)
 VR = Volume of water collected from roof over year (L) = R x Area x Rc/1000
 R = Rainfall each year (mm)
 ρ = Soil bulk-density (g/m³)
 Rc = Runoff coefficient (unitless)
 Kd = Soil-water partition coefficient (cm³/g)
 1000 = Conversion from mm to m

General Parameters

Average rainfall	mm/year	900.5	Average rainfall at Beaudesert Drumley St Met Station for all years of records (2007 – 2023).
Roof area	m ²	200	4 bedroom australian home
Runoff coefficient	-	0.7	assumes 30% water loss in capture into tank
Volume of rainwater	m ³ /year	126.07	calculated
Volume of rainwater	L/year	126070	
Bulk density of deposited dust	g/cm ³	0.5	assumed for loose deposited dust on roof (similar to upper end measured for powders)

Chemical-specific Inputs and calculations - Grid Maximum Off-site

Chemical	PM10		Kd	Particulate Concentration in water mg/L	Dissolved Concentration in water mg/L
	Deposition Rate (DR) mg/m ² /year	Mass deposited each year (DM) mg			
Cadmium (Cd)	1.39E-04	0.0278	75.0	2.2E-07	5.9E-09
Thallium (Tl)	9.59E-05	0.0192	29	1.5E-07	1.0E-08
Mercury (Hg)	2.35E-04	0.0470	790	3.7E-07	9.4E-10
Antimony (Sb)	2.48E-04	0.0496	45	3.9E-07	1.7E-08
Arsenic (As)	1.08E-04	0.0216	29	1.7E-07	1.2E-08
Lead (Pb)	1.41E-03	0.2821	900	2.2E-06	5.0E-09
Chromium (Cr VI assumed)	3.53E-04	0.0707	19	5.6E-07	5.9E-08
Cobalt (Co)	8.76E-05	0.0175	45	1.4E-07	6.2E-09
Copper (Cu)	3.09E-04	0.0619	35	4.9E-07	2.8E-08
Manganese (Mn)	5.29E-04	0.1057	65	8.4E-07	2.6E-08
Nickel (Ni)	4.26E-04	0.0852	65	6.8E-07	2.1E-08
Vanadium (V)	4.38E-05	0.0088	1000	7.0E-08	1.4E-10
Dioxin-like compounds	7.05E-10	1.41E-07	63096	1.1E-12	3.5E-17
PAHs	2.35E-09	4.70E-07	12589	3.7E-12	5.9E-16
PFAS (assumed as PFOS)	8.94E-08	1.79E-05	7.4	1.4E-10	3.8E-11

Kd for dioxin-like compounds based on Log Kow of 6.8 and 1% organic carbon (0.01 Foc), Kd = Koc x Foc

Kd for BaP based on Log Kow of 6.1 and 1% organic carbon (0.01 Foc), Kd = Koc x Foc

Assume 1% OC

	Log Kow	Kow	Kd
Dioxin	6.8	6309573	63096 USEPA RSLs Nov 2024
BaP	6.1	1258925	12589 USEPA RSLs Nov 2024
PFOS	na	na	7.4 USEPA RSLs Nov 2024

Exposure to Chemicals via Incidental Ingestion of Water - Grid Maximum Off-site

$$\text{Daily Chemical Intake}_{IW} = C_W \cdot \frac{IR_W \cdot FI \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{L/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults

Ingestion Rate (Irw, L/day)	2	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	ASC NEPM
Body Weight (BW, kg)	70	ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10950	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)			(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	5.9E-09	7.2E-11	1.7E-10	—	5.25E-07	9%	
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	1.0E-08	1.3E-10	3.0E-10	—	1.66E-06	27%	
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	9.4E-10	1.2E-11	2.7E-11	—	1.12E-07	2%	
Antimony (Sb)		8.6E-04	0%	8.6E-04	100%	1.7E-08	2.1E-10	5.0E-10	—	5.81E-07	9%	
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.2E-08	1.4E-10	3.4E-10	—	3.38E-07	5%	
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	5.0E-09	6.1E-11	1.4E-10	—	4.74E-07	8%	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	5.9E-08	7.2E-10	1.7E-09	—	2.08E-06	34%	
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	6.2E-09	7.6E-11	1.8E-10	—	1.58E-07	3%	
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.8E-08	3.4E-10	8.0E-10	—	1.43E-08	0%	
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	2.6E-08	3.2E-10	7.4E-10	—	1.05E-08	0%	
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.1E-08	2.5E-10	5.9E-10	—	1.24E-07	2%	
Vanadium (V)		2.0E-03	0%	2.0E-03	100%	1.4E-10	1.7E-12	4.0E-12	—	1.99E-09	0%	
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	3.5E-17	4.3E-19	1.0E-18	—	9.58E-10	0%	
PAHs	2.3E-01				100%	5.9E-16	7.2E-18	1.7E-17	1.7E-18	—		
PFAS (assumed as PFOS)	0.0E+00	2.0E-05	20%	1.6E-05	100%	3.8E-11	4.7E-13	1.1E-12	—	6.84E-08	1%	

TOTAL **1.7E-18** **0.00000615**

Exposure to Chemicals via Incidental Ingestion of Water - Grid Maximum Off-site

$$\text{Daily Chemical Intake}_{IW} = C_W \cdot \frac{IR_W \cdot FI \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{L/kg/day})$$

Parameters Relevant to Quantification of Exposure by Children

Ingestion Rate (Irw, L/day)	0.4	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk		
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	5.9E-09	1.3E-11	1.6E-10	--		4.90E-07 9%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	1.0E-08	2.4E-11	2.8E-10	--		1.55E-06 27%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	9.4E-10	2.2E-12	2.5E-11	--		1.05E-07 2%
Antimony (Sb)		8.6E-04	0%	8.6E-04	100%	1.7E-08	4.0E-11	4.7E-10	--		5.42E-07 9%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.2E-08	2.7E-11	3.2E-10	--		3.16E-07 5%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	5.0E-09	1.1E-11	1.3E-10	--		4.42E-07 8%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	5.9E-08	1.3E-10	1.6E-09	--		1.94E-06 34%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	6.2E-09	1.4E-11	1.6E-10	--		1.47E-07 3%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.8E-08	6.4E-11	7.5E-10	--		1.34E-08 0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	2.6E-08	5.9E-11	6.9E-10	--		9.83E-09 0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.1E-08	4.8E-11	5.5E-10	--		1.15E-07 2%
Vanadium (V)		2.0E-03	0%	2.0E-03	100%	1.4E-10	3.2E-13	3.7E-12	--		1.85E-09 0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	3.5E-17	8.1E-20	9.5E-19	--		8.94E-10 0%
PAHs	2.3E-01				100%	5.9E-16	1.4E-18	1.6E-17	3.2E-19		--
PFAS (assumed as PFOS)	0.0E+00	2.0E-05	20%	1.6E-05	100%	3.8E-11	8.8E-14	1.0E-12	--		6.39E-08

TOTAL **3.2E-19** **0.00000574**

Dermal Exposure to Chemicals via Contact with Water - Grid Maximum Off-site

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Adults		
Surface Area (Saw, cm2)	20000	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	0.58	Reasonable maximum time spent showering or wet each day (USEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	ASC NEPM
Body Weight (BW, kg)	70	ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10950	ASC NEPM

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total H
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)	(cm/hr)			(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)	(unitless)
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.0E-03	5.9E-09	3.41E-15	4.2E-13	9.7E-13	--		3.04E-09	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	1.0E-03	1.0E-08	6.08E-15	7.4E-13	1.7E-12	--		9.65E-09	20%
Mercury (Hg)		6.0E-04	60%	2.4E-04	1.0E-03	9.4E-10	5.47E-16	6.7E-14	1.6E-13	--		6.51E-10	1%
Antimony (Sb)		8.6E-04		8.6E-04	1.0E-03	1.7E-08	1.01E-14	1.2E-12	2.9E-12	--		3.37E-09	7%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.0E-03	1.2E-08	6.86E-15	8.4E-13	2.0E-12	--		1.96E-09	4%
Lead (Pb)		6.0E-04	50%	3.0E-04	1.0E-04	5.0E-09	2.88E-16	3.5E-14	8.2E-14	--		2.75E-10	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	2.0E-03	5.9E-08	6.84E-14	8.4E-12	2.0E-11	--		2.41E-08	50%
Cobalt (Co)		1.4E-03	20%	1.1E-03	4.0E-04	6.2E-09	1.43E-15	1.8E-13	4.1E-13	--		3.66E-10	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.0E-03	2.8E-08	1.63E-14	2.0E-12	4.6E-12	--		8.30E-11	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.0E-03	2.6E-08	1.50E-14	1.8E-12	4.3E-12	--		6.11E-11	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.0E-04	2.1E-08	2.41E-15	3.0E-13	6.9E-13	--		1.44E-10	0%
Vanadium (V)		2.0E-03		2.0E-03	1.0E-03	1.4E-10	8.06E-17	9.9E-15	2.3E-14	--		1.15E-11	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	8.1E-01	3.5E-17	1.67E-20	2.0E-18	4.8E-18	--		4.50E-09	9%
PAHs	2.3E-01				7.1E-01	5.9E-16	2.44E-19	3.0E-17	7.0E-17	7.0E-18		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	4.69E-07	3.8E-11	1.04E-20	1.3E-18	3.0E-18	--		1.86E-13	0%

7.0E-18 0.0000000483

Dermal Exposure to Chemicals via Contact with Water - Grid Maximum Off-site

$$DA_{event} = K_p \times C_w \times t_{event}$$

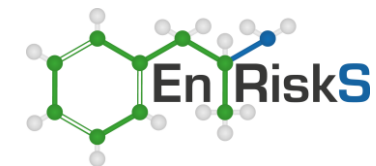
mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Children		
Surface Area (Saw, cm2)	6100	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	1	Reasonable maximum time spent showering or wet each day (USEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total H
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)	(cm/hr)			(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.0E-03	5.9E-09	5.88E-15	2.0E-13	2.4E-12	--		7.47E-09	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	1.0E-03	1.0E-08	1.05E-14	3.7E-13	4.3E-12	--		2.37E-08	20%
Mercury (Hg)		6.0E-04	60%	2.4E-04	1.0E-03	9.4E-10	9.43E-16	3.3E-14	3.8E-13	--		1.60E-09	1%
Antimony (Sb)		8.6E-04		8.6E-04	1.0E-03	1.7E-08	1.75E-14	6.1E-13	7.1E-12	--		8.26E-09	7%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.0E-03	1.2E-08	1.18E-14	4.1E-13	4.8E-12	--		4.81E-09	4%
Lead (Pb)		6.0E-04	50%	3.0E-04	1.0E-04	5.0E-09	4.97E-16	1.7E-14	2.0E-13	--		6.74E-10	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	2.0E-03	5.9E-08	1.18E-13	4.1E-12	4.8E-11	--		5.92E-08	50%
Cobalt (Co)		1.4E-03	20%	1.1E-03	4.0E-04	6.2E-09	2.47E-15	8.6E-14	1.0E-12	--		8.97E-10	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.0E-03	2.8E-08	2.81E-14	9.8E-13	1.1E-11	--		2.04E-10	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.0E-03	2.6E-08	2.58E-14	9.0E-13	1.0E-11	--		1.50E-10	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.0E-04	2.1E-08	4.16E-15	1.4E-13	1.7E-12	--		3.52E-10	0%
Vanadium (V)		2.0E-03		2.0E-03	1.0E-03	1.4E-10	1.39E-16	4.8E-15	5.7E-14	--		2.83E-11	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	8.1E-01	3.5E-17	2.87E-20	1.0E-18	1.2E-17	--		1.10E-08	9%
PAHs	2.3E-01				7.1E-01	5.9E-16	4.20E-19	1.5E-17	1.7E-16	3.4E-18		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	4.69E-07	3.8E-11	1.80E-20	6.3E-19	7.3E-18	--		4.57E-13	0%
										3.4E-18		0.000000118	



Maximum Residential

Calculation of Concentrations in Rainwater tank

$$CW = DM / (VR * Kd * \rho) \quad (\text{mg/L})$$

where:

DM = Mass of dust deposited on roof each year (mg) = DR x Area
 DR = Deposition rate from model (mg/m²/year)
 Area = Area of roof (m²)
 VR = Volume of water collected from roof over year (L) = R x Area x Rc/1000
 R = Rainfall each year (mm)
 ρ = Soil bulk-density (g/m³)
 Rc = Runoff coefficient (unitless)
 Kd = Soil-water partition coefficient (cm³/g)
 1000 = Conversion from mm to m

General Parameters

Average rainfall	mm/year	900.5	Average rainfall at Beaudesert Drumley St Met Station for all years of records (2007 – 2023).
Roof area	m ²	200	4 bedroom australian home
Runoff coefficient	-	0.7	assumes 30% water loss in capture into tank
Volume of rainwater	m ³ /year	126.07	calculated
Volume of rainwater	L/year	126070	
Bulk density of deposited dust	g/cm ³	0.5	assumed for loose deposited dust on roof (similar to upper end measured for powders)

Chemical-specific Inputs and calculations - Maximum residential

Chemical	PM10		Kd	Particulate Concentration in water	Dissolved Concentration in water
	Deposition Rate (DR) mg/m ² /year	Mass deposited each year (DM) mg			
Cadmium (Cd)	9.94E-05	1.99E-02	75.0	1.6E-07	4.2E-09
Thallium (Tl)	6.85E-05	1.37E-02	29	1.1E-07	7.5E-09
Mercury (Hg)	1.68E-04	3.36E-02	790	2.7E-07	6.7E-10
Antimony (Sb)	1.77E-04	3.54E-02	45	2.8E-07	1.2E-08
Arsenic (As)	7.73E-05	1.55E-02	29	1.2E-07	8.5E-09
Lead (Pb)	1.01E-03	2.02E-01	900	1.6E-06	3.6E-09
Chromium (Cr VI assumed)	2.53E-04	5.05E-02	19	4.0E-07	4.2E-08
Cobalt (Co)	6.26E-05	1.25E-02	45	9.9E-08	4.4E-09
Copper (Cu)	2.21E-04	4.42E-02	35	3.5E-07	2.0E-08
Manganese (Mn)	3.78E-04	7.56E-02	65	6.0E-07	1.8E-08
Nickel (Ni)	3.04E-04	6.09E-02	65	4.8E-07	1.5E-08
Vanadium (V)	3.13E-05	6.26E-03	1000	5.0E-08	9.9E-11
Dioxin-like compounds	5.04E-10	1.01E-07	63096	8.0E-13	2.5E-17
PAHs	1.68E-09	3.36E-07	12589	2.7E-12	4.2E-16
PFAS (assumed as PFOS)	6.39E-08	1.28E-05	7.4	1.0E-10	2.7E-11

Kd for dioxin-like compounds based on Log Kow of 6.8 and 1% organic carbon (0.01 Foc), Kd = Koc x Foc

Kd for BaP based on Log Kow of 6.1 and 1% organic carbon (0.01 Foc), Kd = Koc x Foc

Assume 1% OC

	Log Kow	Kow	Kd
Dioxin	6.8	6309573	63096 USEPA RSLs Nov 2024
BaP	6.1	1258925	12589 USEPA RSLs Nov 2024
PFOS	na	na	7.4 USEPA RSLs Nov 2024

Exposure to Chemicals via Incidental Ingestion of Water - Maximum residential

$$\text{Daily Chemical Intake}_{IW} = C_W \cdot \frac{IR_W \cdot FI \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{L/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults

Ingestion Rate (I _{rw} , L/day)	2	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	ASC NEPM
Body Weight (BW, kg)	70	ASC NEPM
Averaging Time - NonThreshold (A _{tc} , days)	25550	ASC NEPM
Averaging Time - Threshold (A _{tn} , days)	10950	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (C _w)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		0.0008	60%	3.2E-04	100%	4.2E-09	5.1E-11	1.2E-10	–		3.75E-07	9%
Thallium (Tl)		0.0002	10%	1.8E-04	100%	7.5E-09	9.2E-11	2.1E-10	–		1.19E-06	27%
Mercury (Hg)		0.0006	60%	2.4E-04	100%	6.7E-10	8.3E-12	1.9E-11	–		8.03E-08	2%
Antimony (Sb)		0.00086	0%	8.6E-04	100%	1.2E-08	1.5E-10	3.6E-10	–		4.15E-07	9%
Arsenic (As)		0.002	50%	1.0E-03	100%	8.5E-09	1.0E-10	2.4E-10	–		2.42E-07	5%
Lead (Pb)		0.0006	50%	3.0E-04	100%	3.6E-09	4.4E-11	1.0E-10	–		3.39E-07	8%
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04	100%	4.2E-08	5.2E-10	1.2E-09	–		1.49E-06	34%
Cobalt (Co)		0.0014	20%	1.1E-03	100%	4.4E-09	5.4E-11	1.3E-10	–		1.13E-07	3%
Copper (Cu)		0.14	60%	5.6E-02	100%	2.0E-08	2.5E-10	5.7E-10	–		1.02E-08	0%
Manganese (Mn)		0.14	50%	7.0E-02	100%	1.8E-08	2.3E-10	5.3E-10	–		7.53E-09	0%
Nickel (Ni)		0.012	60%	4.8E-03	100%	1.5E-08	1.8E-10	4.2E-10	–		8.85E-08	2%
Vanadium (V)		0.002	0%	2.0E-03	100%	9.9E-11	1.2E-12	2.8E-12	–		1.42E-09	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	2.5E-17	3.1E-19	7.2E-19	–		6.85E-10	0%
PAHs	0.233				100%	4.2E-16	5.2E-18	1.2E-17	1.2E-18			
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05	100%	2.7E-11	3.4E-13	7.8E-13	–		4.89E-08	1%

TOTAL **1.2E-18** **0.00000440**

Exposure to Chemicals via Incidental Ingestion of Water - Maximum residential

$$\text{Daily Chemical Intake}_{IW} = C_W \cdot \frac{IR_W \cdot FI \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{L/kg/day})$$

Parameters Relevant to Quantification of Exposure by Children

Ingestion Rate (Irw, L/day)	0.4	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)			(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		0.0008	60%	3.2E-04	100%	4.2E-09	9.6E-12	1.1E-10	—		3.50E-07	9%
Thallium (Tl)		0.0002	10%	1.8E-04	100%	7.5E-09	1.7E-11	2.0E-10	—		1.11E-06	27%
Mercury (Hg)		0.0006	60%	2.4E-04	100%	6.7E-10	1.5E-12	1.8E-11	—		7.49E-08	2%
Antimony (Sb)		0.00086	0%	8.6E-04	100%	1.2E-08	2.9E-11	3.3E-10	—		3.87E-07	9%
Arsenic (As)		0.002	50%	1.0E-03	100%	8.5E-09	1.9E-11	2.3E-10	—		2.26E-07	5%
Lead (Pb)		0.0006	50%	3.0E-04	100%	3.6E-09	8.1E-12	9.5E-11	—		3.16E-07	8%
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04	100%	4.2E-08	9.6E-11	1.1E-09	—		1.39E-06	34%
Cobalt (Co)		0.0014	20%	1.1E-03	100%	4.4E-09	1.0E-11	1.2E-10	—		1.05E-07	3%
Copper (Cu)		0.14	60%	5.6E-02	100%	2.0E-08	4.6E-11	5.3E-10	—		9.55E-09	0%
Manganese (Mn)		0.14	50%	7.0E-02	100%	1.8E-08	4.2E-11	4.9E-10	—		7.03E-09	0%
Nickel (Ni)		0.012	60%	4.8E-03	100%	1.5E-08	3.4E-11	4.0E-10	—		8.26E-08	2%
Vanadium (V)		0.002	0%	2.0E-03	100%	9.9E-11	2.3E-13	2.7E-12	—		1.33E-09	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	2.5E-17	5.8E-20	6.8E-19	—		6.39E-10	0%
PAHs	0.233				100%	4.2E-16	9.7E-19	1.1E-17	2.3E-19			
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05	100%	2.7E-11	6.3E-14	7.3E-13	—		4.56E-08	1%

TOTAL **2.3E-19** **0.00000411**

Dermal Exposure to Chemicals via Contact with Water - Maximum residential

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Adults		
Surface Area (Saw, cm2)	20000	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	0.58	Reasonable maximum time spent showering or wet each day (USEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	ASC NEPM
Body Weight (BW, kg)	70	ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10950	ASC NEPM

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk		
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.0E-03	4.2E-09	2.44E-15	3.0E-13	7.0E-13	--		2.18E-09
Thallium (Tl)		2.0E-04	10%	1.8E-04	1.0E-03	7.5E-09	4.35E-15	5.3E-13	1.2E-12	--		6.90E-09
Mercury (Hg)		6.0E-04	60%	2.4E-04	1.0E-03	6.7E-10	3.91E-16	4.8E-14	1.1E-13	--		4.66E-10
Antimony (Sb)		8.6E-04		8.6E-04	1.0E-03	1.2E-08	7.25E-15	8.9E-13	2.1E-12	--		2.41E-09
Arsenic (As)		2.0E-03	50%	1.0E-03	1.0E-03	8.5E-09	4.91E-15	6.0E-13	1.4E-12	--		1.40E-09
Lead (Pb)		6.0E-04	50%	3.0E-04	1.0E-04	3.6E-09	2.06E-16	2.5E-14	5.9E-14	--		1.96E-10
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	2.0E-03	4.2E-08	4.89E-14	6.0E-12	1.4E-11	--		1.73E-08
Cobalt (Co)		1.4E-03	20%	1.1E-03	4.0E-04	4.4E-09	1.02E-15	1.3E-13	2.9E-13	--		2.61E-10
Copper (Cu)		1.4E-01	60%	5.6E-02	1.0E-03	2.0E-08	1.16E-14	1.4E-12	3.3E-12	--		5.93E-11
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.0E-03	1.8E-08	1.07E-14	1.3E-12	3.1E-12	--		4.37E-11
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.0E-04	1.5E-08	1.72E-15	2.1E-13	4.9E-13	--		1.03E-10
Vanadium (V)		2.0E-03		2.0E-03	1.0E-03	9.9E-11	5.76E-17	7.1E-15	1.6E-14	--		8.23E-12
Dioxin-like compounds		2.3E-09	54%	1.1E-09	8.1E-01	2.5E-17	1.19E-20	1.5E-18	3.4E-18	--		3.22E-09
PAHs	2.3E-01				7.1E-01	4.2E-16	1.74E-19	2.1E-17	5.0E-17	5.0E-18		--
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	4.69E-07	2.7E-11	7.45E-21	9.1E-19	2.1E-18	--		1.33E-13

5.0E-18 0.0000000345

Dermal Exposure to Chemicals via Contact with Water - Maximum residential

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

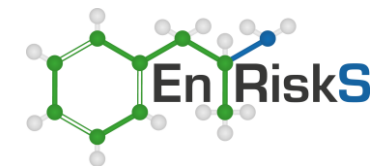
$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Children		
Surface Area (Saw, cm2)	6100	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	1	Reasonable maximum time spent showering or wet each day (USEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk		
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)
Cadmium (Cd)		0.0008	60%	3.2E-04	1.0E-03	4.2E-09	4.20E-15	1.5E-13	1.7E-12	--		5.34E-09
Thallium (Tl)		0.0002	10%	1.8E-04	1.0E-03	7.5E-09	7.50E-15	2.6E-13	3.0E-12	--		1.69E-08
Mercury (Hg)		0.0006	60%	2.4E-04	1.0E-03	6.7E-10	6.74E-16	2.4E-14	2.7E-13	--		1.14E-09
Antimony (Sb)		0.00086		8.6E-04	1.0E-03	1.2E-08	1.25E-14	4.4E-13	5.1E-12	--		5.91E-09
Arsenic (As)		0.002	50%	1.0E-03	1.0E-03	8.5E-09	8.46E-15	2.9E-13	3.4E-12	--		3.44E-09
Lead (Pb)		0.0006	50%	3.0E-04	1.0E-04	3.6E-09	3.55E-16	1.2E-14	1.4E-13	--		4.82E-10
Chromium (Cr VI assumed)		0.0009	10%	8.1E-04	2.0E-03	4.2E-08	8.43E-14	2.9E-12	3.4E-11	--		4.23E-08
Cobalt (Co)		0.0014	20%	1.1E-03	4.0E-04	4.4E-09	1.77E-15	6.2E-14	7.2E-13	--		6.42E-10
Copper (Cu)		0.14	60%	5.6E-02	1.0E-03	2.0E-08	2.01E-14	7.0E-13	8.2E-12	--		1.46E-10
Manganese (Mn)		0.14	50%	7.0E-02	1.0E-03	1.8E-08	1.84E-14	6.4E-13	7.5E-12	--		1.07E-10
Nickel (Ni)		0.012	60%	4.8E-03	2.0E-04	1.5E-08	2.97E-15	1.0E-13	1.2E-12	--		2.52E-10
Vanadium (V)		0.002		2.0E-03	1.0E-03	9.9E-11	9.94E-17	3.5E-15	4.0E-14	--		2.02E-11
Dioxin-like compounds		2.3E-09	54%	1.1E-09	8.1E-01	2.5E-17	2.05E-20	7.2E-19	8.4E-18	--		7.89E-09
PAHs	0.233				7.1E-01	4.2E-16	3.00E-19	1.0E-17	1.2E-16	2.4E-18		
PFAS (assumed as PFOS)		0.00002	20%	1.6E-05	4.69E-07	2.7E-11	1.28E-20	4.5E-19	5.2E-18	--		3.26E-13

2.4E-18	0.0000000847
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Maximum Commercial/Industrial

Calculation of Concentrations in Rainwater tank

$$CW = DM / (VR * Kd * \rho) \quad (\text{mg/L})$$

where:

DM = Mass of dust deposited on roof each year (mg) = DR x Area
 DR = Deposition rate from model (mg/m²/year)
 Area = Area of roof (m²)
 VR = Volume of water collected from roof over year (L) = R x Area x Rc/1000
 R = Rainfall each year (mm)
 ρ = Soil bulk-density (g/m³)
 Rc = Runoff coefficient (unitless)
 Kd = Soil-water partition coefficient (cm³/g)
 1000 = Conversion from mm to m

General Parameters

Average rainfall	mm/year	900.5	Average rainfall at Beaudesert Drumley St Met Station for all years of records (2007 – 2023). 4 bedroom australian home assumes 30% water loss in capture into tank calculated
Roof area	m ²	200	
Runoff coefficient	-	0.7	
Volume of rainwater	m ³ /year	126.07	
Volume of rainwater	L/year	126070	
Bulk density of deposited dust	g/cm ³	0.5	assumed for loose deposited dust on roof (similar to upper end measured for powders)

Chemical-specific inputs and calculations

Chemical	PM10		Kd	Particulate Concentration in water mg/L	Dissolved Concentration in water mg/L
	Deposition Rate (DR) mg/m ² /year	Mass deposited each year (DM) mg			
Cadmium (Cd)	6.80E-05	0.0136	75.0	1.1E-07	2.9E-09
Thallium (Tl)	4.69E-05	0.0094	29	7.4E-08	5.1E-09
Mercury (Hg)	1.15E-04	0.0230	790	1.8E-07	4.6E-10
Antimony (Sb)	1.21E-04	0.0243	45	1.9E-07	8.6E-09
Arsenic (As)	5.30E-05	0.0106	29	8.4E-08	5.8E-09
Lead (Pb)	6.90E-04	0.1381	900	1.1E-06	2.4E-09
Chromium (Cr VI assumed)	1.73E-04	0.0346	19	2.7E-07	2.9E-08
Cobalt (Co)	4.29E-05	0.0086	45	6.8E-08	3.0E-09
Copper (Cu)	1.52E-04	0.0303	35	2.4E-07	1.4E-08
Manganese (Mn)	2.59E-04	0.0518	65	4.1E-07	1.3E-08
Nickel (Ni)	2.08E-04	0.0417	65	3.3E-07	1.0E-08
Vanadium (V)	2.15E-05	0.0043	1000	3.4E-08	6.8E-11
Dioxin-like compounds	3.45E-10	6.90E-08	63096	5.5E-13	1.7E-17
PAHs	1.15E-09	2.30E-07	12589	1.8E-12	2.9E-16
PFAS (assumed as PFOS)	4.37E-08	8.75E-06	7.4	6.9E-11	1.9E-11

Kd for dioxin-like compounds based on Log Kow of 6.8 and 1% organic carbon (0.01 Foc), Kd = Koc x Foc

Kd for BaP based on Log Kow of 6.1 and 1% organic carbon (0.01 Foc), Kd = Koc x Foc

Assume 1% OC

	Log Kow	Kow	Kd
Dioxin	6.8	6309573	63096 USEPA RSLs Nov 2024
BaP	6.1	1258925	12589 USEPA RSLs Nov 2024
PFOS	na	na	7.4 USEPA RSLs Nov 2024

Exposure to Chemicals via Incidental Ingestion of Water - Maximum commercial

$$\text{Daily Chemical Intake}_{IW} = C_W \cdot \frac{IR_W \cdot FI \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{L/kg/day})$$

Parameters Relevant to Quantification of Exposure by Adults

Ingestion Rate (Irw, L/day)	2	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	ASC NEPM
Body Weight (BW, kg)	70	ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10950	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)			(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.9E-09	3.5E-11	8.2E-11	—		2.57E-07	9%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	5.1E-09	6.3E-11	1.5E-10	—		8.15E-07	27%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	4.6E-10	5.7E-12	1.3E-11	—		5.50E-08	2%
Antimony (Sb)		8.6E-04	0%	8.6E-04	100%	8.6E-09	1.0E-10	2.4E-10	—		2.84E-07	9%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	5.8E-09	7.1E-11	1.7E-10	—		1.66E-07	5%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	2.4E-09	3.0E-11	7.0E-11	—		2.32E-07	8%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.9E-08	3.5E-10	8.3E-10	—		1.02E-06	34%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	3.0E-09	3.7E-11	8.6E-11	—		7.72E-08	3%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.4E-08	1.7E-10	3.9E-10	—		7.01E-09	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.3E-08	1.5E-10	3.6E-10	—		5.16E-09	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.0E-08	1.2E-10	2.9E-10	—		6.06E-08	2%
Vanadium (V)		2.0E-03	0%	2.0E-03	100%	6.8E-11	8.3E-13	1.9E-12	—		9.72E-10	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	1.7E-17	2.1E-19	5.0E-19	—		4.69E-10	0%
PAHs	2.3E-01				100%	2.9E-16	3.5E-18	8.3E-18	8.3E-19		—	
PFAS (assumed as PFOS)	0.0E+00	2.0E-05	20%	1.6E-05	100%	1.9E-11	2.3E-13	5.4E-13	—		3.35E-08	1%

TOTAL **8.3E-19** **0.00000301**

Exposure to Chemicals via Incidental Ingestion of Water - Maximum commercial

$$\text{Daily Chemical Intake}_{IW} = C_W \cdot \frac{IR_W \cdot FI \cdot B \cdot EF \cdot ED}{BW \cdot AT} \quad (\text{L/kg/day})$$

Parameters Relevant to Quantification of Exposure by Children

Ingestion Rate (Irw, L/day)	0.4	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk		
	Non-Threshold Slope Factor (mg/kg-day) ⁻¹	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.9E-09	6.6E-12	7.7E-11	--		2.40E-07
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	5.1E-09	1.2E-11	1.4E-10	--		7.61E-07
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	4.6E-10	1.1E-12	1.2E-11	--		5.13E-08
Antimony (Sb)		8.6E-04	0%	8.6E-04	100%	8.6E-09	2.0E-11	2.3E-10	--		2.65E-07
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	5.8E-09	1.3E-11	1.5E-10	--		1.55E-07
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	2.4E-09	5.6E-12	6.5E-11	--		2.16E-07
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.9E-08	6.6E-11	7.7E-10	--		9.51E-07
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	3.0E-09	6.9E-12	8.1E-11	--		7.20E-08
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.4E-08	3.1E-11	3.7E-10	--		6.54E-09
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.3E-08	2.9E-11	3.4E-10	--		4.81E-09
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.0E-08	2.3E-11	2.7E-10	--		5.65E-08
Vanadium (V)		2.0E-03	0%	2.0E-03	100%	6.8E-11	1.6E-13	1.8E-12	--		9.08E-10
Dioxin-like compounds		2.3E-09	54%	1.1E-09	100%	1.7E-17	4.0E-20	4.6E-19	--		4.38E-10
PAHs	2.3E-01				100%	2.9E-16	6.6E-19	7.7E-18	1.5E-19		--
PFAS (assumed as PFOS)	0.0E+00	2.0E-05	20%	1.6E-05	100%	1.9E-11	4.3E-14	5.0E-13	--		3.13E-08

TOTAL **1.5E-19** **0.00000281**

Dermal Exposure to Chemicals via Contact with Water - Maximum commercial

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Adults		
Surface Area (Saw, cm2)	20000	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	0.58	Reasonable maximum time spent showering or wet each day (ESEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	ASC NEPM
Body Weight (BW, kg)	70	ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	10950	ASC NEPM

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total H
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)	(cm/hr)			(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.0E-03	2.9E-09	1.67E-15	2.0E-13	4.8E-13	--		1.49E-09	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	1.0E-03	5.1E-09	2.98E-15	3.6E-13	8.5E-13	--		4.73E-09	20%
Mercury (Hg)		6.0E-04	60%	2.4E-04	1.0E-03	4.6E-10	2.68E-16	3.3E-14	7.7E-14	--		3.19E-10	1%
Antimony (Sb)		8.6E-04		8.6E-04	1.0E-03	8.6E-09	4.96E-15	6.1E-13	1.4E-12	--		1.65E-09	7%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.0E-03	5.8E-09	3.36E-15	4.1E-13	9.6E-13	--		9.60E-10	4%
Lead (Pb)		6.0E-04	50%	3.0E-04	1.0E-04	2.4E-09	1.41E-16	1.7E-14	4.0E-14	--		1.34E-10	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	2.0E-03	2.9E-08	3.35E-14	4.1E-12	9.6E-12	--		1.18E-08	50%
Cobalt (Co)		1.4E-03	20%	1.1E-03	4.0E-04	3.0E-09	7.02E-16	8.6E-14	2.0E-13	--		1.79E-10	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.0E-03	1.4E-08	7.97E-15	9.8E-13	2.3E-12	--		4.06E-11	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.0E-03	1.3E-08	7.33E-15	9.0E-13	2.1E-12	--		2.99E-11	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.0E-04	1.0E-08	1.18E-15	1.4E-13	3.4E-13	--		7.03E-11	0%
Vanadium (V)		2.0E-03		2.0E-03	1.0E-03	6.8E-11	3.95E-17	4.8E-15	1.1E-14	--		5.64E-12	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	8.1E-01	1.7E-17	8.16E-21	1.0E-18	2.3E-18	--		2.20E-09	9%
PAHs	2.3E-01				7.1E-01	2.9E-16	1.19E-19	1.5E-17	3.4E-17	3.4E-18		--	
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	4.69E-07	1.9E-11	5.10E-21	6.2E-19	1.5E-18	--		9.11E-14	0%
										3.4E-18		0.0000000236	

Dermal Exposure to Chemicals via Contact with Water - Maximum commercial

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm² per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Children		
Surface Area (Saw, cm ²)	6100	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	1	Reasonable maximum time spent showering or wet each day (ESEPA)
Conversion Factor (CF, L/cm ³)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per ASC NEPM
Averaging Time - NonThreshold (Atc, days)	25550	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day) ⁻¹	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm ² per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.0E-03	2.9E-09	2.88E-15	1.0E-13	1.2E-12	--		3.66E-09	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	1.0E-03	5.1E-09	5.13E-15	1.8E-13	2.1E-12	--		1.16E-08	20%
Mercury (Hg)		6.0E-04	60%	2.4E-04	1.0E-03	4.6E-10	4.62E-16	1.6E-14	1.9E-13	--		7.82E-10	1%
Antimony (Sb)		8.6E-04		8.6E-04	1.0E-03	8.6E-09	8.56E-15	3.0E-13	3.5E-12	--		4.05E-09	7%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.0E-03	5.8E-09	5.79E-15	2.0E-13	2.4E-12	--		2.36E-09	4%
Lead (Pb)		6.0E-04	50%	3.0E-04	1.0E-04	2.4E-09	2.43E-16	8.5E-15	9.9E-14	--		3.30E-10	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	2.0E-03	2.9E-08	5.78E-14	2.0E-12	2.3E-11	--		2.90E-08	50%
Cobalt (Co)		1.4E-03	20%	1.1E-03	4.0E-04	3.0E-09	1.21E-15	4.2E-14	4.9E-13	--		4.39E-10	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.0E-03	1.4E-08	1.37E-14	4.8E-13	5.6E-12	--		9.97E-11	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.0E-03	1.3E-08	1.26E-14	4.4E-13	5.1E-12	--		7.34E-11	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.0E-04	1.0E-08	2.04E-15	7.1E-14	8.3E-13	--		1.72E-10	0%
Vanadium (V)		2.0E-03		2.0E-03	1.0E-03	6.8E-11	6.81E-17	2.4E-15	2.8E-14	--		1.38E-11	0%
Dioxin-like compounds		2.3E-09	54%	1.1E-09	8.1E-01	1.7E-17	1.41E-20	4.9E-19	5.7E-18	--		5.41E-09	9%
PAHs	2.3E-01				7.1E-01	2.9E-16	2.06E-19	7.2E-18	8.4E-17	1.7E-18			
PFAS (assumed as PFOS)		2.0E-05	20%	1.6E-05	4.69E-07	1.9E-11	8.80E-21	3.1E-19	3.6E-18	--		2.24E-13	0%

1.7E-18 0.0000000580