Attachment 13: Human Health Risk Assessment (enRiskS, 2023)

Melbourne Energy and Resource Centre: Human Health Risk Assessment

Prepared for: Cleanaway Operations Pty Ltd

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Document History and Status

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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	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
DECCW	NSW Department of Environment, Climate Change and Water
DEFRA	Department for Environment, Food & Rural Affairs
DEH	Australian Department of Environment and Heritage
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
LOR	Limit of Reporting
Metabolism	The conversion or breakdown of a substance from one form to another by a living organism.
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
NHMRC	National Health and Medical Research Council
NO_2	Nitrogen dioxide
NOx	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)
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).

Term	Definition
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].
SEIFA	Socio-Economic Index for Areas
SO_2	Sulfur dioxide
TCEQ	Texas Commission on Environmental Quality
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 based on available toxicity studies relevant to humans and/or animals and relevant safety factors.
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
UK	United Kingdom
US	United States
USEPA	United States Environmental Protection Agency
WHO	World Health Organization
$\mu g/m^3$	Micrograms per cubic metre

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 a waste-to-energy facility in Melbourne, Victoria.

Cleanaway, an Australian waste management, recycling, and industrial services company, is developing a waste-to-energy (WtE) facility in Victoria known as the Melbourne Energy and Resource Centre (the Proposal). The Proposal is located at 510 Summerhill Road, Wollert, VIC.

The Proposal will be designed to thermally treat 380,000 tonnes per annum (tpa) of waste feedstock that would otherwise be sent to landfill, primarily consisting of residual Municipal Solid Waste (MSW) and residual Commercial and Industrial (C&I) waste. The Proposal will also incorporate maturation and processing of bottom ash to recover recyclable metals, with the intent to utilise the remaining ash as an aggregate in construction and on-site stabilisation of air pollution control residue (APCr) prior to disposal off-site at an appropriately licenced landfill.

The purpose of the report is to address Victorian requirements for the proposed facility in regard to community health. These requirements include 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 the proposed facility as outlined in *Ministerial guidelines for assessment of environmental effects under the Environment Effects Act 1978* (Victorian Government 2006).

The human health risk assessment has addressed potential impacts to community health from air emissions using a quantitative approach while potential impacts to community health from water, noise or chemical hazards have been assessed using a qualitative approach. This is because potential impacts to community health from emissions to air are the primary issue for a waste-to-energy facility.

Assessment of emissions to air

Types of exposure

The human health risk assessment has evaluated exposure to the air emissions from the facility in a quantitative fashion. People may be exposed to contaminants in air via:

- Inhalation of air containing the emissions
- Deposition of particles onto soil and including:
    - direct contact with that soil
    - uptake into and consumption of home grown fruit and vegetables
    - uptake into and consumption of home grown eggs
    - uptake into and consumption of home grown meat
    - uptake into and consumption of home grown milk
- Deposition of particles onto a roof, collection into household rainwater tanks and consumption of tank water for domestic purposes.

It is noted that the pathways related to consumption of fruit, vegetables, eggs, meat or milk refer to home grown produce. This means the calculations are designed to assess consumption of fruit, vegetables, eggs, meat or milk grown on a farm or in a backyard by those living on that farm or in that house as this will be the pathway with highest exposure potential (i.e. worst-case).

Exposure scenarios evaluated

An exposure scenario is a description of how a person might be exposed and includes the assumptions made about how they will be exposed. For example, how long each day they will be present at a location etc.

The exposure scenarios chosen for this assessment were those relevant for each type of land use in the community surrounding the proposed facility – i.e. places where people live, where they work or places where children go to school or childcare. For each of these land use types, the worst case location was chosen for the assessment. This is usually the closest location to the proposed facility with that land use. All other locations with the same land use will have lower risks – if the worst case location shows that risks are acceptable (based on national health authority guidance), then all locations with that land use will have risks that are acceptable.

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 (this is the worst case anywhere around the facility)
- 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 (as 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 other places (includes schools, hospitals, aged care facilities, child care facilities and places of worship) location (location that is currently used for one of these “other uses” that has the maximum ground level concentrations for any site used for these other purposes) – exposure via inhalation and direct contact with soil
- Maximum other places location (as residential) – assumes this location changes land use to residential – assumes this location may change land use to residential at some time in the future
- Maximum on-site location – exposure via inhalation for visitors to the Visitor and Education Centre.

Modelling

Emissions from the facility were modelled by Katestone in line with EPA Victoria requirements for air quality modelling (EPA Victoria 2013). The facility has been designed to meet emission concentration limits set out in the "Industrial Emissions Directive (EU 2010), for short-term (30 minute) emission limits, and the BREF 2019 (EU 2019), for daily emission limits or emission limits over other relevant averaging period." These documents are the most recent update on best practice technologies for such facilities developed in Europe.

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. The potential for impacts on human health due to long term exposure scenarios were assessed using annual average ground level concentrations for the cumulative case. The cumulative case considered existing air quality in Wollert, estimated emissions from the facility as well as emissions to air from the Austral Bricks facility to the south (the Brickworks).

The air quality modelling and the risk calculations have assumed the following mitigation measures will be in place during operation of the 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 the tipping hall
- transport of waste to the site using enclosed trucks or other relevant techniques
- 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 that are always present in the atmosphere particularly in urban areas. They need to be managed well to maintain acceptable air quality. These pollutants include sulfur dioxide, nitrogen dioxide, carbon monoxide and particles (PM_{10}, $PM_{2.5}$). They are emitted by WtE facilities but there are many other sources of these types of air pollutants including all combustion sources – fires, bushfires, cooking, vehicles, wood fired heaters, open fireplaces, ship engines and power stations – and other sources like windblown dust and salt spray. This is why this sub group of potential air pollutants is covered by the national guidance – the Ambient Air Quality NEPM (NEPC 2021b).

The guidelines provided in the Ambient Air Quality NEPM have been developed by government health and environment protection authorities They are based on protection of human health for communities in Australia.

Air quality modelling was undertaken by Katestone to estimate the ground level concentrations at all relevant locations around the proposed facility. They estimated the concentration at ground level for all relevant pollutants across a grid covering a 10 km x 10 km area using the model specified by EPA Victoria. These ground level concentrations were estimated every 15 m across this grid (north to south and east to west). In addition, a range of specific locations (the discrete sensitive receptors) were also assessed – i.e. houses or commercial areas or childcare centres/schools or hospitals.

The ground level concentrations used in this assessment were:

- maximum found anywhere (i.e. around the boundary of the site)
- maximum location where a house is currently located
- maximum location where a commercial/industrial facility is located
- maximum location where a school/childcare centre/aged care facility/hospital is located
- maximum location on the site in relation to visitors to the education centre.

The results for criteria pollutants at the maximum residential location have been listed in **Table ES-1**.

Other locations around the facility (commercial, schools, childcare centres, hospitals, worship locations, hospitals) will have similar or lower concentrations than those listed here.

Table ES-1: Criteria pollutants (maximum residential location) (µg/m^3)

Scenario	Sulfur dioxide	Nitrogen dioxide	Carbon monoxide	$PM_{2.5}$	PM_{10}
Guideline (NEPC 2021a)	52	28	10,000	8	25^
Averaging period	24 hour average	Annual average	8 hour average	Annual average	Annual average
Contribution from project	2.3	0.2	7.9	0.03	0.03
% contribution from project (compared to NEPM guideline value)	6.9%	0.7%	0.08%	0.4%	0.1%
Cumulative case	44	20	1,369	8.9	19

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

- For nitrogen dioxide, the cumulative concentration (i.e. including background and the Brickworks) is below the relevant national guidelines (i.e. in compliance) and the increment of the guideline sourced from this facility alone is less than 1%.
- For carbon monoxide, the cumulative concentration (i.e. including background and the Brickworks) is below the relevant national guidelines (i.e. in compliance) and the increment of the guideline sourced from this facility alone is less than 0.1%.
- For PM_{10}, the cumulative concentration (i.e. including background and the Brickworks) is below the relevant national guidelines (i.e. in compliance) and the increment of the guideline sourced from this facility alone is less than 1%.
- For sulfur dioxide, the cumulative concentration (i.e. including background and the Brickworks) is below the relevant national guidelines (i.e. in compliance) and the increment of the guideline sourced from this facility alone is less than 10%.
- For particles, particularly those that are very small ($PM_{2.5}$), levels contributed by this facility are low (incremental contribution from this facility is less than 1% of the guideline), but the overall cumulative concentrations are similar to the national guidelines. This results in the same situation as is currently present at this location (i.e. no change to the existing), given the small contribution from the Proposal. It is important to note that there are many sources of $PM_{2.5}$ in Melbourne (and any other city). Any combustion process will emit these types of particles. This includes wood heaters, gas fired BBQs, gas cooking, candles, all types of vehicles, bushfires and power stations. Other sources of particles in the atmosphere include windblown dust and degradation/breakdown of vegetation.

Other pollutants

Short term exposures

The assessment of short term exposure for air pollutants other than those listed above (i.e. including gases like hydrogen chloride (HCl), hydrogen fluoride (HF) and ammonia; volatile heavy metals such as cadmium, mercury; and volatile organic compounds such as benzene) 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 Proposal). This location varies depending on the chemical (gases versus attached to particles). However, it will be located just outside the Proposal area (i.e. fence line) 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)).

The public health based guidelines are taken from government sources – both in Australian guidance and in guidance from international sources like the US Environmental Protection Agency or the World Health Organisation. For short-term exposures, the public health based guidelines were 1 hour average concentrations (i.e. average concentration to which a person may be exposed for 1 hour) taken primarily from EPA Victoria guidance (EPA Victoria 2022). Such guidelines are based on ensuring no effects to community health would be expected.

For this assessment, the individual risk quotients were between five and 30,000 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. 0.26). This indicates that the estimated ground level concentrations are well below the public health based guidelines.

These findings indicate that risks to community health in relation to short term exposures at the most affected location are low and acceptable based on guidance from Australian health authorities.

Long term exposures

The assessment of long term exposures to emissions has been assessed using the same exposure scenarios as discussed above. The assessment used the annual average ground level concentrations for the cumulative case (i.e. with consideration of ambient background and the emissions from the Brickworks).

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 (people are exposed by ingestion and dermal 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 (people are exposed by consuming the produce)
- deposit onto the roofs and mix into rainwater tanks (people are exposed by ingestion or dermal contact with the water).

The multi-pathway assessment includes consideration of these additional types of exposure.

Risks (using risk quotients and risk indices) have been calculated for each exposure scenario and each type of exposure. These risk calculations consider consequences using national/international guidance on the hazards posed by the relevant pollutants and consider likelihood in the design of the exposure scenarios used in the calculations. It is assumed in these calculations that exposure as specified in the exposure scenario will occur – i.e. likelihood is "certain".

The column labelled Risk (deposition) includes the sum of exposure pathways related to particle deposition relevant at that location (i.e. for maximum off-site will be just exposure to soil where particles may have deposited; for maximum residential (as farm) will be sum of exposure to soil and exposure via consumption for all types of produce).

An overall summary of the findings for the risk calculations is provided in **Table ES-2**.

Table ES-2: Calculated risks (cumulative case)

Scenario	Risk (inhalation)	Risk (deposition)	Risk (rainwater tank)	Risk (total)
Maximum off-site	0.5	0.01	NA	0.5
Maximum residential	0.2	0.001	0.00004	0.2
Maximum residential (as farm)	0.2	0.01	0.00004	0.2
Maximum commercial	0.07	0.005	0.0002	0.07
Maximum other places	0.02	0.0003	0.000006	0.02
Maximum on-site	0.007	NA	NA	0.007
Guideline	≤1	≤1	≤1	≤1

Risks calculated for the various scenarios evaluated for this facility are at least two times below the relevant guideline using the cumulative case (this facility + background + Brickworks) at the locations where ground level concentrations are expected to be highest anywhere around the proposed facility and between five and 50 times lower than the relevant guideline for all the other worst-case locations (residential, commercial/industrial or other places location with the highest ground level concentrations). These overall risk estimates include those for dioxin-like compounds and heavy metals.

There were a number of chemicals that was assessed for their potential to cause effects via non-threshold mechanisms – benzene and polycyclic aromatic hydrocarbons (PAHs as BaP) – the PAHs are assumed to be present as benzo[a]pyrene – the chemical in the group that poses the highest hazard.

In regard to benzene – the assessment has assumed all total volatile organic carbon (TVOC) emitted by the facility is present as benzene (or formaldehyde – included in the risks listed in **Table ES-2**). To assume all the TVOC is benzene 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 some of the most sensitive guideline values. Exposure via inhalation is the only relevant pathway for these types of chemicals and the risk estimates were 10 to 1,250 fold lower than the relevant guidelines (i.e. well in compliance).

For PAHs, risk estimates range from 10 to 1,000 fold lower than the relevant guidelines (i.e. well in compliance).

These findings indicate that risks to community health in relation to long term exposures at the most affected location are low and acceptable based on guidance from Australian health authorities.

Assessment of other matters

To assess the potential for impacts to community health for matters such as noise, water or chemical hazards, information from other technical studies prepared for this assessment has been used.

The approach taken for these assessments was to determine if those technical studies have included consideration of potential impacts to human health and whether those matters (noise, water or chemical hazards) have the potential to cause any changes to the current situation.

For matters related to noise, the Proposal will comply with relevant Victorian guidelines and guidelines from WHO which are more targeted at limiting impacts to community health.

For matters related to water, the Proposal will cause little change to the current situation.

For matters related to chemical hazards, the Proposal must comply with all relevant Victorian guidance in relation to storage and handling of such materials.

Overall Conclusions

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

- Air quality

Based on the operation at (or below) the specified emission limits, modelled changes in air quality in the local area (Katestone 2023), and consideration of the adopted human health guidance, risks to community health in relation to emissions to air from this facility will be low and acceptable in line with guidance from Australian health authorities.

In particular, this assessment has shown:

- No unacceptable risks for criteria pollutants (NO_x, SO_x, CO, $PM_{2.5}$, PM_{10})
- No unacceptable risks for short term exposures (via inhalation) 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 other places location (including farms)
  - Maximum on-site location
- No unacceptable risks for relevant exposure scenarios for rainwater tanks.

It is noted that:

- risks via inhalation are based on the modelled ground level concentrations at the worst-case locations and assume 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 which may deposit onto the soil around the facility have been assessed assuming such deposition occurs for 70 years (more than the lifetime of the facility) (i.e. the soil concentrations used in the calculations are those at the end of the 70 years of deposition).
- This assessment is, therefore, inherently conservative.

- Noise

Based on consideration of the EPA Victoria requirements for noise assessment and ensuring that the facility is designed to meet the relevant levels and that all identified noise mitigation measures are implemented, risks to community health in relation to nose from this facility will be low and acceptable in line with guidance from Australian health authorities.

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

Based on the evaluations provided, risks to community health in relation to issues related to water or chemical hazards from this facility will be low and acceptable in line with guidance from Australian health authorities.

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 a waste-to-energy facility in Melbourne, Victoria.

Cleanaway, an Australian waste management, recycling, and industrial services company, is developing a waste-to-energy (WtE) facility in Victoria known as the Melbourne Energy and Resource Centre (the Proposal). The Proposal is located at 510 Summerhill Road, Wollert, VIC.

The Proposal will be designed to thermally treat 380,000 tonnes per annum (tpa) of waste feedstock that would otherwise be sent to landfill, primarily consisting of residual Municipal Solid Waste (MSW) and residual Commercial and Industrial (C&I) waste. The Proposal will also incorporate maturation and processing of bottom ash to recover recyclable metals, with the intent to utilise the remaining ash as an aggregate in construction and on-site stabilisation of air pollution control residue (APCr) prior to disposal off-site at an appropriately licenced landfill.

1.2 Objectives

This HHRA is being undertaken to evaluate the potential for the Proposal 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.

This is in line with Victorian 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 as described in *Ministerial guidelines for assessment of environmental effects under the Environment Effects Act 1978* (Victorian Government 2006). Other aspects listed in this guidance are displacement of residences or associated transport issues. These are of less relevance to this assessment than those listed above (i.e. air, water, noise or chemical hazards).

The HHRA has been based on the proposed waste to be used as fuel and the proposed thermal treatment (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.

1.3 Assessment limitations

The assessment has only evaluated risks to community health. This includes potential for issues related to construction or operation to impact on people living, working or recreating in areas around the proposed facility.

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 the terrestrial or aquatic environments are considered in other assessments related to the Proposal and are not normally covered in a Human Health Risk Assessment.

1.4 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 2012b)
- enHealth 2012. Australian Exposure Factors Guide (enHealth 2012a).

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)
- EPA Victoria guidelines on energy from waste (EPA Victoria 2017)
- EPA Victoria guideline for assessing and minimising air pollution in Victoria (EPA Victoria 2022)

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

- *Planning and Environment Act 1987*
- *Public Health and Wellbeing Act 2008*
- *Public Health and Wellbeing Regulations 2019*
- *Environment Protection Act 2017* including the Environment Reference Standard
- Noise limit and assessment protocol for the control of noise from commercial, industrial and trade premises and entertainment venues (EPA Victoria 2021)
- Victorian Public Health and Wellbeing Plan 2019–2023 (DHHS 2019).

To address the above, the HHRA has presents 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 – water, soil contamination, chemicals management, groundwater (**Section 7**)
- Conclusions (**Section 8**).

1.5 Definitions

For the conduct of the HHRA the following definitions are relevant 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 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.

1.6 Available information

In relation to the Proposal, the HHRA has been undertaken on the basis of existing information which is available in the following reports:

- Project Description Information from Arup (Arup 2023)
- Katestone (2023), Melbourne Energy and Resource Centre, Air quality assessment. Report dated 23 February 2023.
- Air Quality Modelling Output Spreadsheets provided by Katestone (received 17/3/23)
- Arup (2023a), Melbourne Energy and Resource Centre, Noise & Vibration Technical Report. DRAFT Report dated 8 February 2023.
- Arup (2023b). Hydrology and flood risk technical report, Melbourne Energy and Resource Centre (MERC). Dated 10 February 2023.
- Douglas Partners (2022). Report on Soil Contamination and Baseline Groundwater Investigation, Melbourne Energy and Resource Centre, 510 Summerhill Road, Wollert. Dated January 2023.
- Arup (2023c). Hazardous substances and industrial hazards technical report, Melbourne Energy and Resource Centre (MERC). Dated 3 February 2023.
- Arup (2023d). Surface water, Chapter 15, Development License Application. Dated 17 March 2023.

1.7 Legislative background

1.7.1 General

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

1.7.2 Australian Government

Table 1.1 summarises the legislation, policy and guideline documents relating to the conduct of human health assessments in Australia, with some providing more specific guidance in health impacts associated with 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
National Environment Protection Council Act 1994	The *National Environment Protection Council Act 1994* 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
	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. 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 2012b)	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 2012a)	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 for the National Environment Protection Measure under section 20(1) of the *National Environment Protection Act 1994*, and the *National Environment Protection Council (Victoria) Act 1995* (and other State Acts) in relation to guidelines relevant to the assessment of site contamination, provided in Schedules B1 to B9. This includes the derivation of health-based guidelines for the assessment of contaminated land (NEPC 1999 amended 2013e, 1999 amended 2013d).
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 *National Environment Protection Council Act 1994*. 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 PM_{10} 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 *National Environment Protection Council Act 1994*. 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 (NEPC 1999 amended 2013b)	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.

1.7.3 Victorian Government

Table 1.2 summarises the legislation and guidance that broadly relate to the protection of health and wellbeing.

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

Legislation, policy, or guideline	Overview and implications for this Project
Planning and Environment Act 1987 Ministerial Direction 19 (2018)	The *Planning and Environment Act 1987* (incorporating amendments as at 24 March 2021) and the *Planning and Environment Regulations 2015* provide no specific reference to the assessment or protection of community health. There are some references to the requirement to consider any relevant State Environment Protection Policy (SEPP). However, under sections 12(1)(f) and 12(2)(a) of the *Planning and Environment Act 1987*, a Ministerial Direction was issued on 10 October 2018, and came into effect on 18 October 2019, on the planning and content, and requirements of information for authorisation or preparation, of amendments that may significantly impact the environment, amenity and human health. Direction 19 of the Ministerial Direction requires planning authorities to *'seek the views of the Environment Protection Authority (EPA) in the preparation of planning scheme review and amendments that could result in use of development of land that may result in significant impacts on the environment, amenity and human health due to pollution and waste.'* This requirement relates to the development of potentially contaminated land as well as developments that result in water, noise, air or land pollution impacts on the environment, amenity, or human health. This references the SEPPs. Although the SEPPs no longer have statutory effect since the commencement of the *Environment Protection Act 2017*, they are still considered relevant to the state of knowledge about potential environmental effects of pollution. Ministerial Direction 19 (2018) requires the assessment of human health by planning, under direction from EPA Victoria, in relation to developments. It is expected this would apply to the Project. While not stated in the direction, an assessment of human health impacts could be in the form of a health risk assessment, or health impact assessment.
Public Health and Wellbeing Act 2008	Under Part 5, Division 3 of the *Public Health and Wellbeing Act 2008*, the Minister may require the conduct of a health impact assessment of the public health and wellbeing impact of a matter. The timing for completion of the assessment may be determined in such a direction. Part 6, Division 1 of the Act relates to nuisances which are liable to be dangerous to health or offensive. The Act also outlines and references the precautionary principal and the principle of primacy of prevention (that is, preventing disease, illness, injury, disability, or premature death, and building health promotion activities).

Legislation, policy, or guideline	Overview and implications for this Project
Public Health and Wellbeing Regulations 2019	The Public Health and Wellbeing Regulations came into effect on 14 December 2019 and replaced the 2009 Regulations. The 2019 Regulations are made under the Public Health and Wellbeing Act 2008 and addresses a broad range of specific issues that directly or indirectly affect or influence health and includes vector-borne disease (mosquito control), certain business premises (aquatic facilities, premises other that aquatic facilities such as tattooing, skin piercing), cooling towers, legionella, pest control, notifiable conditions/diseases, immunisation, tissue donation, sex workers and clients. The Regulations do not include any specific reference to or provide guidance on the conduct of a human health assessment.
Environment Protection Act 2017	The Environment Protection Act 2017 (2017 Act) provides the legal framework for the protection of the environment in Victoria from pollution and waste. The 2017 Act and the subordinate documents (including the Environment Reference Standard) discuss that health and wellbeing, local amenity and aesthetic considerations are important beneficial uses and values of the environment. The 2017 Act also states that the objective of EPA Victoria is to protect human health and the environment by reducing the harmful effects of pollution and waste. The 2017 Act outlines penalties for pollution of air, water, or land. It also addresses objectionable noise and powers to address noise complaints. The 2017 Act does not specify any details about the completion of a health impact/risk assessment. The 2017 Act introduces a General Environmental Duty (GED) which is focused on preventing harm. This requires people and businesses (or companies) to eliminate, or otherwise reduce risks of harm to human health and the environment from pollution and waste so far as reasonably practicable. The definition of human health within the Act includes psychological health. The GED is a new concept for Victoria and requires the following: ■ The duty holder needs to understand the risks that pollution or waste from their activities might present to human health or the environment ■ The ways those risks can be controlled need to be identified and understood ■ Duty holders are required to put in place reasonably practicable measures to eliminate or reduce the risk of the harm arising ■ The considerations for reasonably practicable include likelihood, consequence, state of knowledge, availability and suitability, cost elimination or reduction. The Act provides reference to the National Environment Protection Council (Victoria) Act 1995. The Act does not provide any specific requirements to undertake a human health assessment, although to determine if individuals, businesses, and operations have the potential to result in risks to human health or the environment, a risk assessment is required. There is no specific guidance in the Act as to the conduct of a risk assessment. The inclusion of the requirement to assess controls and implement measures to reduce the likelihood of harm is consistent with the conduct of a human health assessment. Section 7 of the Act includes a definition of harm. In relation to human health or the environment, harm means an adverse effect on human health or the environment (of whatever degree of duration) and includes amenity. Harm may also arise from a cumulative effect. Material harm is defined as harm caused by pollution or waste that results in actual adverse health effects on human health or the environment that are not negligible (and further details). Harm may become material harm regardless of the time period, so it includes a single event/occurrence, multiple occurrences, and cumulative effects. The above definitions of harm and the requirements to understand and characterise risks of harm are consistent with HIA guidance.

Legislation, policy, or guideline	Overview and implications for this Project
Environment Reference Standards (ERSs)	The Environment Reference Standards (ERSs, replacing the State Environment Protection Policies [SEPPs]) will be key to the assessment of health impacts in Victoria. The ERSs support the protection of human health and the environment from pollution and waste by providing a benchmark to assess and report on environmental conditions in the whole or any part of Victoria. The ERSs identify environmental values that specify the environmental condition to be achieved or maintained in the whole or any part of Victoria; and specify indicators and objectives to be used to measure, determine or assess whether those environmental values are being achieved, maintained or threatened. Environmental values for the ambient air environment include life, health and well-being of humans and other forms of life, local amenity and aesthetic enjoyment, visibility, the useful life and appearance of buildings and climate systems. The indicators for these values are similar to the air quality SEPPs and also include a qualitative indicator for odour. Environmental values for noise include consideration of health determinants such as peoples' sleep, domestic and recreational activities, ability to sustain normal conversation, an environment that supports child learning and development and human tranquillity and enjoyment outdoors in natural areas. Indicators for noise depend on land use category and include a qualitative indicator for acoustic quality conducive to human tranquillity and enjoyment. Environmental values of the land environment include human health (the land quality is suitable for the specific land use and safe for the human use of that land) and apply to all land use categories. That is: parks and reserves, agricultural land, sensitive land uses, recreation/open space, commercial and industrial land use. The ERSs for all water environments refer to various health determinants in the environmental values and indicators. In summary, the emphasis on human health and well-being as an 'environmental value' in the air, noise, land, and water environments in Victoria is significantly broadened in the new environment protection regime.
Guidelines for assessing and minimising air pollution in Victoria (EPA Victoria 2022)	This guideline provides a framework to assess and control risks associated with air pollution and has been developed to address requirements under the Environment Protection Act 2017. In relation to the assessment of health impacts the draft guidance allows for different levels of assessment, consistent with the impacts identified and quantified. This includes quantification of risks to human health from air toxics, criteria pollutants and conducting multi-pathway assessments.
Noise limit and assessment protocol for the control of noise from commercial, industrial and trade premises and entertainment venues	A protocol for determining noise limits for new and existing commercial, industrial and trade premises and entertainment venues. It also sets the methodology for assessing the effective noise level to determine unreasonable noise under the proposed Environment Protection Regulations.
Victorian Public Health and Wellbeing Plan 2019–2023 (DHHS 2019)	It is a requirement of Victoria's Public Health and Wellbeing Act 2008 to produce a Victorian Public Health and Wellbeing Plan every four years. The Plan sets out a comprehensive approach to deliver improved public health and wellbeing outcomes for all Victorians. The Plan considers wider determinants of health, consistent with the WHO definition, noting that a whole of government approach is required to deliver public health and wellbeing improvements. The Victorian Public Health and Wellbeing Plan 2019–2023 continues the Victorian Government's vision for the public health and wellbeing of Victorians: *A Victoria free of the avoidable burden of disease and injury so that all Victorians can enjoy the highest attainable standards of health, wellbeing, and participation at every age.* The Plan provides continuity for the priorities of the previous plan, while recognising two leading threats to health and wellbeing globally: the health impacts of climate change and antimicrobial resistance (the ability to effectively treat infections in our community). The Plan provides a range of priorities with four identified as focus areas. One focus area is increasing active living, which is of relevance to the Project.

1.7.4 General Environmental Duty

The cornerstone of the new environmental protection legislation (detailed above) is the General Environmental Duty (GED). The GED requires that anyone conducting an activity that poses risks of harm to human health and the environment from pollution or waste must minimise those risks, so far as reasonably practicable.

'Reasonably practicable' measures, mean putting in controls to eliminate the risk of harm to human health and the environment so far as is reasonably practicable. If eliminating the risk of harm is not reasonably practicable, then the risk of harm must be reduced so far as reasonably practicable.

A number of matters must be considered in deciding what is reasonably practicable in the circumstances:

- The likelihood of those risks eventuating
- The degree of harm that would result if those risks eventuated
- What the person concerned knows, or ought reasonably to know, about the harm or risks of harm and any ways of eliminating or reducing those risks
- The availability and suitability of ways to eliminate or reduce those risks
- The cost of eliminating or reducing those risks.

EPA Victoria (EPA Victoria 2020a) explains that when dealing with a common risk or harm, demonstrating the person or business undertaking the activity has done what is reasonably practicable can be achieved if:

- Well-established effective practices or controls have been adopted to eliminate or manage risk
- Where well-established practices or controls do not exist, it can be shown that effective controls have been assessed and adopted.

Section 2. Project description

2.1 Overview

Cleanaway is an Australian waste management, recycling, and industrial services company. Cleanaway is developing a waste-to-energy (WtE) facility in Victoria known as the Melbourne Energy and Resource Centre (MERC) (the Proposal).

The MERC has been designed to thermally treat a design capacity of 380,000 tonnes per annum (tpa) of waste feedstock, consisting of residual Municipal Solid Waste (MSW) and residual commercial waste, which is waste that would otherwise be sent to landfill. Waste feedstock processed by the MERC will be subject to a Waste Acceptance Protocol to determine eligibility and suitability for processing both prior to arrival and upon arrival on-site. The Proposal will also incorporate maturation and processing of bottom ash to recover recyclable metals, with the intent to utilise the remaining ash as an aggregate in construction.

Residual waste is waste that is left over from recycling and resource recovery operations and waste from source separated collections. Source separation involves separating waste into common material streams or categories for separate collection. Waste processed at the site will be subject to a Waste Acceptance Protocol to ensure only appropriate waste is used as feedstock.

The WtE process would generate approximately 46.3 MW gross of electricity, 4.7 MW of which would be used to power the facility itself and the associated on-site by-product and residue handling processes, with 41.6 MW (328,700 MWh/year) exported to the grid as base load electricity. In addition to supplying electricity to the grid, there is also potential to supply energy in the form of heat and/or process steam to local industrial users.

Some residual materials are produced because of the WtE process, including Incinerator Bottom Ash (IBA), boiler ash and flue gas treatment residue. The boiler ash and flue gas treatment residue are typically combined and together are referred to as Air Pollution Control residue (APCr). Overall, the WtE process typically leads to about 90% reduction in the volume, or 80% reduction in mass (tonnes), of waste that would otherwise go to landfill. If IBA is reused as an alternative construction product to virgin materials, this percentage increases further to approximately 95% reduction in volume and mass of waste that would otherwise go to landfill. The final volume of waste diverted from landfill is dependent on the classification and market for the residues and by-products generated by the WtE facility.

The Proposal includes the construction and operation of an IBA maturation and processing facility on site. The purpose of this facility is to store the IBA to mature (stabilise) it, before mechanically processing IBA from the WtE facility into an aggregate for reuse. As part of this process, both ferrous and non-ferrous metals will be recovered from the IBA for recycling and sale to market.

The Proposal also includes a stabilisation facility for APCr, a necessary treatment step to immobilise leachable components of the APCr prior to removal from site by vehicle and disposal at an appropriately licenced landfill.

The Proposal will use best available techniques and technologies in the engineering design, operation, mainte nance and monitoring activities associated with the MERC. Moving grate technology has been chosen as the means to thermally treat incoming waste to recover energy and other resources. Current international best-practice techniques, including automated combustion controls and advanced flue gas treatment technology will be applied so that air emissions meet

stringent emission standards. The moving grate combustion system is a common form of thermal WtE technology in which the waste is fed through the combustion chamber on a travelling grate. This enables efficient and complete combustion of the waste, with primary combustion air introduced from below the grate and secondary combustion air introduced directly into the combustion zone above the grate. Moving grate technology has been used globally for over 100 years, and in that time the technology has been subject to continual improvement responding to regulatory, industry and public demands. There are approximately 500 similar operational examples across Europe alone, the majority of which use the moving grate-type technology being proposed for the MERC.

The Proposal involves the building of all on-site infrastructure required to support the WtE facility, including site utilities, internal roads, weighbridges, parking and hardstand areas, stormwater infrastructure, fencing and landscaping. The Proposal will also include a visitor and education centre to help educate and inform the community on the circular economy, recycling, resource recovery, the benefits of landfill diversion and the WtE process. The intent behind this education is to drive a shift in community thinking and actions around waste management.

The Victorian Waste to Energy Framework (2021) recognises the role of WtE to divert waste from landfills, helping Victoria transition to a circular economy. Recycling Victoria recognises a role for WtE investment and supports WtE facilities where they meet best-practice environment protection requirements. This includes reducing waste to landfill, supporting waste avoidance, reusing and recycling, and demonstrating social license with affected communities. The Victorian Environment Protection Authority (EPA) Energy from Waste Guideline (Publication 1559, 1 July 2017) also notes that efficient recovery of energy from the thermal processing of waste is considered a resource recovery as opposed to a waste disposal option.

The EPA VIC Guideline: Energy from Waste stipulates that *'Proponents of EfW proposals…will be expected to demonstrate that the siting, design, construction and operation of EfW facilities will incorporate best practice measures for the protection of the land, water and air environments as well as for energy efficiency and greenhouse gas emissions management. Facilities should be able to provide evidence of how they minimise and manage emissions (including pollutants, odour, dust, litter, noise and residual waste) in accordance with relevant statutory requirements.'*

The WtE facility has been designed to meet the European Industrial Emissions Directive (IED) (2010) and the associated Best Available Techniques Reference (BREF) Document for Waste Incineration published in December 2019 which sets the European Union environmental standards for waste incineration. The facility will also comply with the technical criteria set out in the EPA Victoria Guideline: Energy from Waste publication 1559.1.

The purpose of this specialist assessment is to demonstrate compliance with the various authority requirements in regard to human health.

2.2 Site description

The Proposal is located at 510 Summerhill Road, Wollert, VIC. **Figures 2.1** and **2.2** provide the regional context and the proposed layout for the Proposal.

The Proposal area will incorporate the following key elements:

- Two-line WtE facility comprising: a combustor, boiler, and flue gas treatment system. The processing capacity of each line will be approximately 190,000 tpa. The two lines will supply

steam to a single steam turbine while flue gas will be discharged through a single stack containing two internal flues. Moving grate combustion will be employed as the preferred thermal treatment technology.

- Incinerator Bottom Ash (IBA) treatment area incorporating: a fully enclosed sorting facility, storage areas for sorted and matured IBA, conveyor for delivery of IBA from WtE facility to the IBA treatment building, open-air IBA maturation piles (1-2 months) with dust control using water sprays, a bund and water capture system around the IBA facility.
- Air Pollution Control residues (APCr) stabilisation facility.
- Site infrastructure including: roads, weighbridges, inspection bays, dangerous goods tanks, firewater and process water tanks, electrical substation, truck movement areas, visitor centre, offices, and parking spaces.

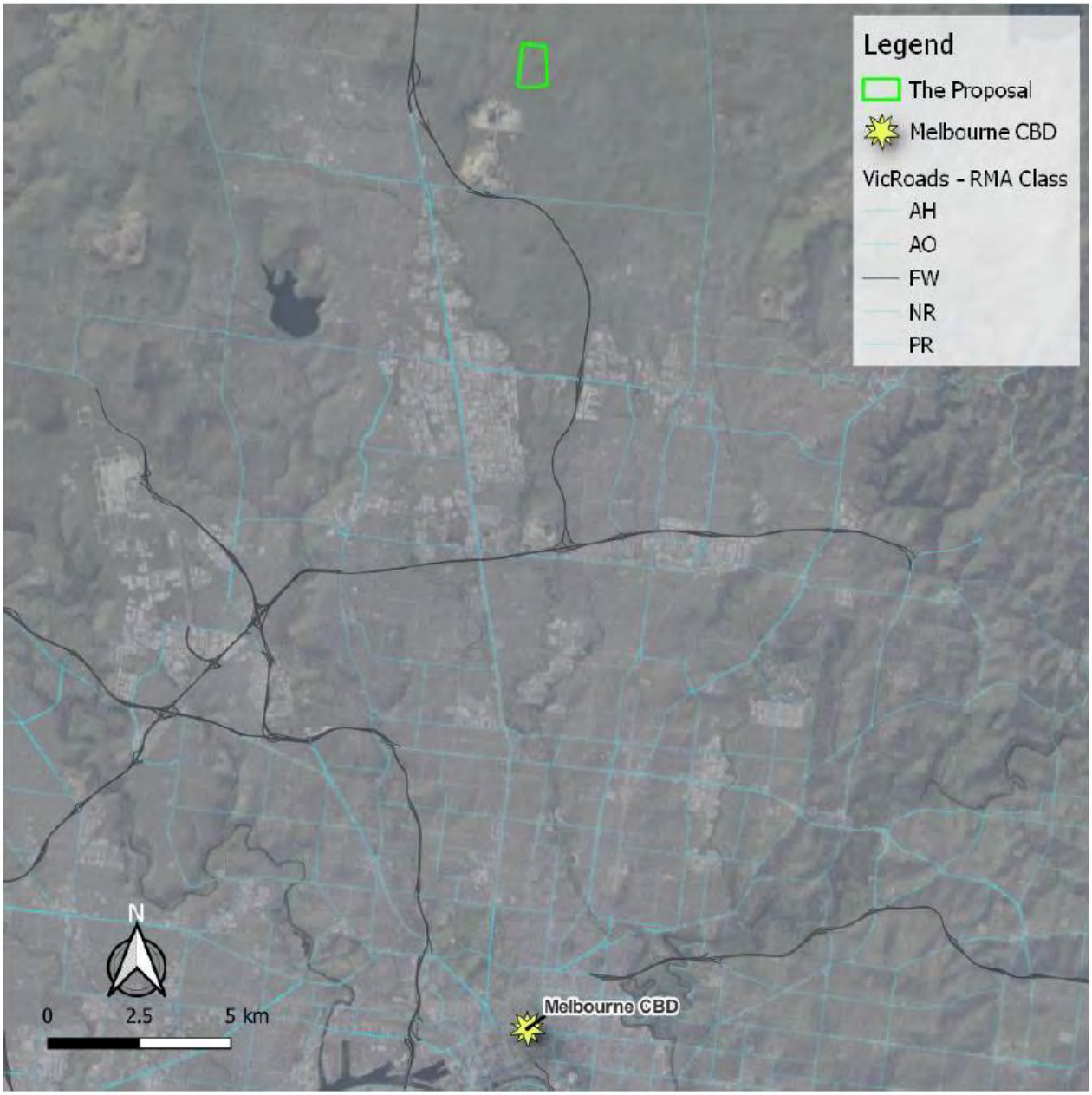

Figure 2.1: Regional context

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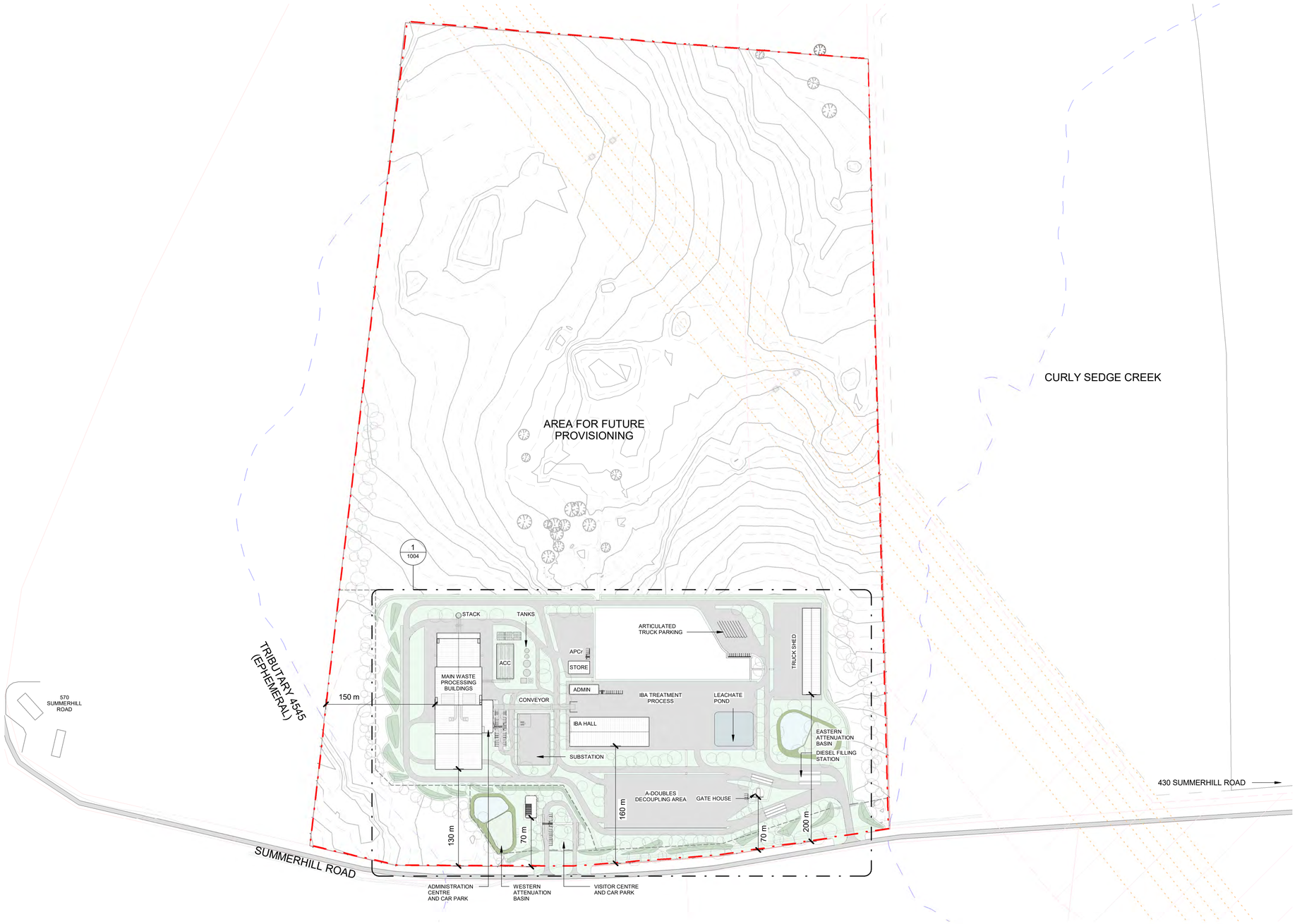

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N

0 100 m 200m

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Do not scale

Figure 2.2

Issue	Date	By	Chkd	Appd
Final Draft	20/03/23	ZR	GF	BP
Final Draft	10/02/23	ZR	GF	BP
DLA Draft	07/12/22	ZR	GF	BP

Client: CLEANAWAY — Making a sustainable future possible

Architectural Certification
Name:
Signature: Date:

Job Title: MELBOURNE ENERGY AND RESOURCE CENTRE
50 SUMMERHILL RD
WOLLERT
VICTORIA 3750

Scale at A1: 1 : 3000

Discipline: ARCHITECTURE

ARUP

Wurundjeri Woiwurrung Country
Sky Park, One Melbourne Quarter
699 Collins Street Docklands
VIC 3008 Australia

CONSULT AUSTRALIA
Member Firm
Arup Pty Ltd
ABN 18 000 966 165

Drawing Title
GENERAL ARRANGEMENT
SITE
INCLUDING AREA OF FUTURE PROVISIONING

Drawing Status
S0 - FOR INFORMATION ONLY

Job No	Drawing No	Issue
287710-00	MERC-ARU-MEL-ARAR-DRG-1003	C

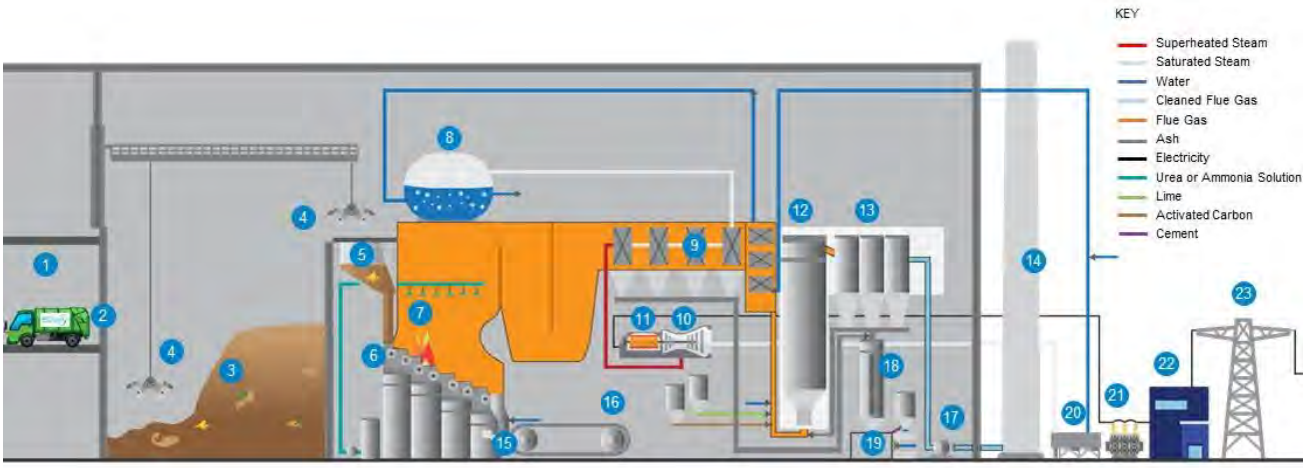
2.3 Construction

Construction will have the potential to generate dust emissions from land clearing, the handling of materials, windblown dust from exposed areas, vehicle movements and exhaust emissions from diesel generators.

2.4 Operation

2.4.1 General

The following section describes the day-to-day operational characteristics of the WtE facility. **Figure 2.3** shows the typical process through a WtE facility.



LEGEND

1	Waste receiving hall	7	Boiler with SNCR (de-NOx)	13	Bag filter	19	Treated APCr to stabilization area for curing prior to removal off-site for disposal
2	Tipping bay	8	Steam drum	14	Stack		
3	Waste bunker	9	Heat exchangers	15	Incinerator bottom ash (IBA) quenching	20	Air cooled condenser
4	Waste crane	10	Steam turbine	16	IBA conveyor to treatment area for maturation and on-site metals recovery	21	Transformer
5	Feed hopper (chute)	11	Generator			22	Substation
6	Moving grate	12	Semi dry reactor	17	ID Fan	23	Local electricity grid or 'behind the meter' connection points
				18	Air Pollution Control residues (APCr) and boiler fly ash silo		

Figure 2.3: Schematic of the WtE operational process

Residual waste (after source separation by the householder, business or institution) is placed into the combustion chamber where it is burnt to convert the waste materials into heat energy. The heat generated is used to heat water sufficiently to generate steam. The steam is used to turn a turbine which generates electricity (this is the same process that occurs in coal or gas fired power stations). The steam can also be used for heating or as a source of steam for industrial purposes at nearby locations.

The handling of waste at the facility occurs within the tipping hall and waste bunker which are held under negative air pressure (air moves into the building rather than out from the building when doors open). Trucks containing the waste will be driven into the building before they are emptied. Air within the tipping hall/waste bunker is taken into the combustion chamber to be used as combustion air.

The air from inside the combustion chamber (that from the tipping hall/waste bunker and from within the waste and fresh air brought from outside to maintain appropriate internal air pressure) is cleaned through a range of pollution control equipment to ensure it is of a suitable quality to discharge through the stack.

The ash generated in the combustion chamber when solid waste materials are burnt is known as IBA (incinerator bottom ash). Subject to relevant approvals and commercial arrangements, this material can be processed and reused. There are also residues collected in the baghouse (one of the pollution control devices) and boiler ash collected in hoppers below the convection section of the boiler. These are known as air pollution control residues (APCr) and these are treated on-site then disposed to landfill.

2.4.2 Flue gas treatment

Combustion gases created through the combustion of waste must be cleaned before being released through the stack.

The flue gas treatment system will include:

- Selective Non-Catalytic Reduction (SNCR) – Ammonia or urea solution is injected into the flue gas path prior to the boiler (when temperatures exceed 900°C). Either of these chemicals will react with NOx 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 SO_2 to knock out 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 – 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 filters within the baghouse. These 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 flue gas before the flue gas is 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 2019).

2.4.3 Ash management

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

The Proposal 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 considered non-hazardous. It is often recycled into aggregate used in

construction in Europe and this approach will be pursued. If recycling is not possible the material will be disposed to an inert landfill.

- Air pollution control residues (APCr)
    - Boiler Ash – some of the ash from the combustion process becomes entrained in the flue gas and makes its way up into the main boiler section where water is heated to form steam. It then falls out (gets deposited) within the boiler sections before any flue gas treatment reagents are injected into the process. This material will be mixed with the APCr from the baghouse. This ash is considered hazardous particularly due to its elevated concentrations of heavy metals.
    - 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 and the leftover entrained ash within the flue gas that did not fall out of the air as it moved through the boiler section. APCr are collected from the flue gas as it moves through the baghouse section of the flue gas treatment system. This material is considered hazardous and must be treated to minimise leaching of hazardous chemicals. Treatment will involve mixing the material with cement to form a solid concrete 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 City of Whittlesea Local Government Area (LGA). Local government areas Hume City Council, Nillumbik Shire Council and City of Darebin adjoin the City of Whittlesea to the west, east and south respectively.

The closest boundary to the City of Hume LGA is around 1 km to the west of the site, for Nillumbik Shire Council LGA, the closest boundary is around 14 km to the east of the site and the boundary for City of Darebin LGA is around 13 km to the south of the site.

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 statistical area 2 groupings (i.e. SA2s). This includes Wollert, Whittlesea, Craigieburn Central, Craigieburn-North, Mernda-North and Mernda-South. The profile has been specifically developed using information for Wollert, Craigieburn Central, Craigieburn-North SA2s and City of Whittlesea and Hume City Council LGAs.

The key focus of this health risk assessment is the local community surrounding the site.

3.2 Land uses

The existing site includes buildings and dams associated with farm use.

The site is bounded to the east by an ephemeral creek – Curly Sedge Creek. The property directly adjoining the site to the east is permitted for use and development as a future quarry and is known as the Phillips Quarry. To the south is Summerhill Road with the Austral Bricks site (quarry and brick making) located approximately 500 m further to the south. To the west and north are areas used for livestock grazing. A concrete quarry is located to the north of the site (around 1 km north). Another quarry and landfill (operated by Hanson) is located around 5 km to the east of the site.

The Hume Freeway is located around 2.8 km to the west and 2.9 km to the south of the site.

There is a high voltage transmission line that crosses the site (northwest to southeast). There is an easement for this infrastructure.

An existing industrial area is located along the Hume Freeway/Highway approximately 3.5 km to the south. Existing residential areas are located around 2.8 km west of the site (Craigieburn), 4.3 km to the southeast of the site (Wollert) and 3.1 km to the northwest of the site (Kalkallo).

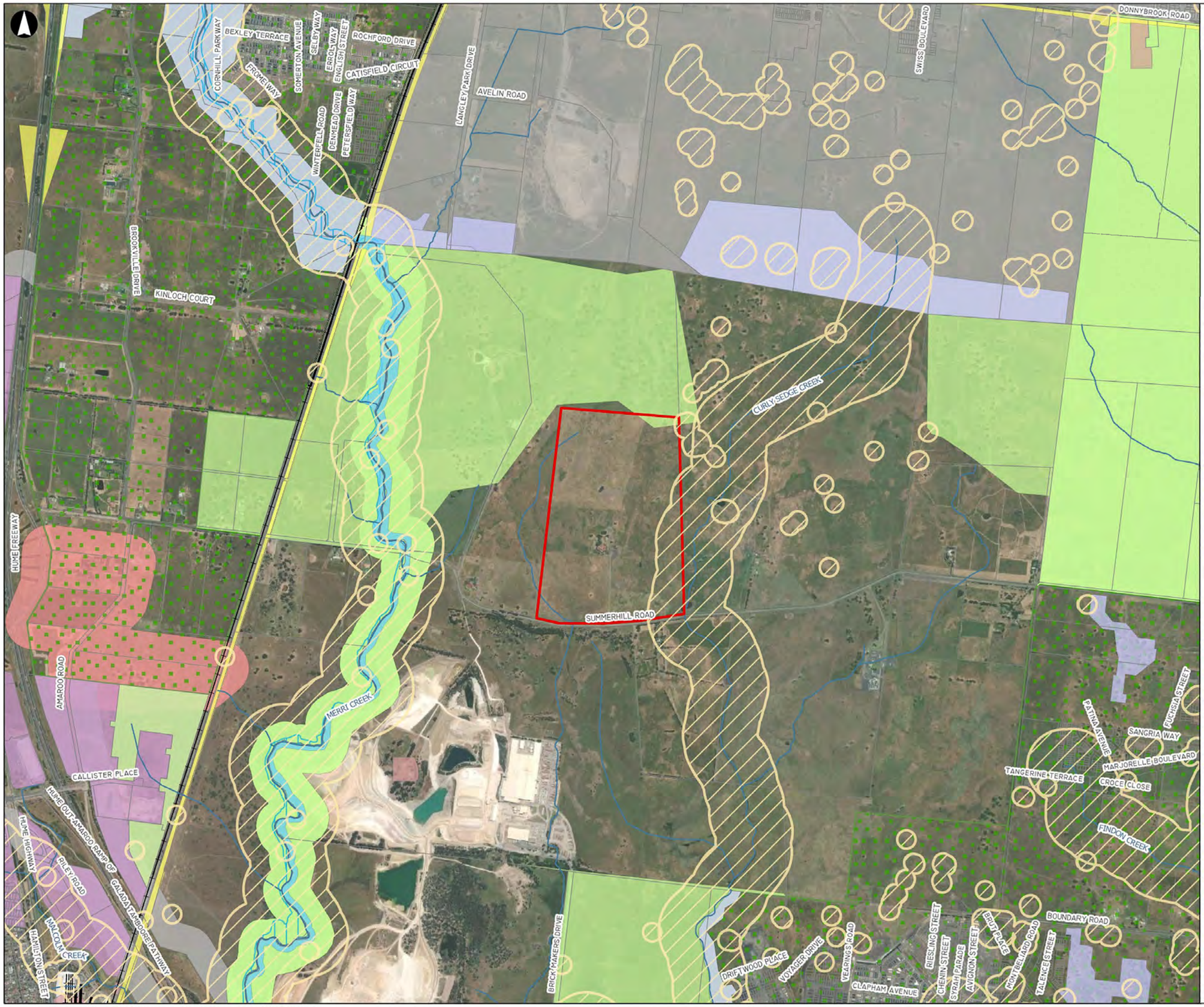
Planning overlays and planning zones are provided in **Figures 3.1** and **3.2**.

There are a range of proposed developments currently being considered in the area surrounding this site. These are detailed in Precinct Structure Plans (PSPs). These are plans that form part of the Victorian Planning Authorities process for coordinating planning between state and local government in the urban growth corridors identified in 2005. The PSPs identified to be of interest for this development include:

- Shenstone Park PSP – 800 m to the north of the site. This area is planned for residential and industrial use. The residential areas will be at least 1.3 km from the facility. There is an existing quarry located in this area (Woody Hill) and a recycled water treatment facility is proposed just south of the area specified in this PSP.

- English Street PSP – 1.6 km northwest of the site. This area is proposed for residential land use with a commercial precinct and community facilities at around 2.3 km northwest of the site. A conservation area is to be included in this PSP around Merri Creek which is located to the west of the site.
- Wollert PSP – 1.4 km east of the site. The plans for this area include residential properties, 5 schools (closest at 2.5 km southeast of the site), conservation areas, sporting fields and an emergency services precinct. The Wollert Landfill and Quarry are located within this area (5.1 km east of the site). Another quarry is also proposed for this area which will be 5.9 km to the southeast of the site of interest for this assessment.
- Craigieburn North Employment PSP – 1.3 km west of the site. The plans for this area are for a range of commercial (north of area) and industrial sites (south of area) with a conservation area adjacent to Merri Creek.
- Northern Quarries PSP – this area is being proposed for implementation in 5-10 years time. The site of interest for this assessment is located within the area specified in this PSP. Land to the east of this site is approved for use as a basalt quarry (Phillips Quarry).

Figure 3.3 shows that Victorian Planning Authority map for PSPs as at September 2022.



A3

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- Proposal area
- Cadastre
- Rail
- Watercourses

Planning overlays

- Bushfire Management Overlay (BMO)
- Development Contributions Plan Overlay (DCPO)
- Design And Development Overlay (DDO)
- Development Plan Overlay (DPO)
- Environmental Significance Overlay (ESO)
- Heritage Overlay (HO)
- Infrastructure Contributions Overlay (ICO)
- Incorporated Plan Overlay (IPO)
- Land Subject To Inundation Overlay (LSIO)
- Public Acquisition Overlay (PAO)
- Rural Floodway Overlay (RFO)
- Specific Controls Overlay (SCO)
- Aboriginal cultural heritage sensitivity

Coordinate System: GDA2020 MGA Zone 55

Maxar VICMAP (2022)

Metres

0 250 500 1,000

Rev	Date	By	Chkd	Appd	Authd
A	2/7/2023	CT	JB	–	–

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Client
Cleanaway Operations Pty Ltd

Project Name
Melbourne Energy and Resource Centre (MERC)

Drawing Title
FIGURE 3.1 - Planning overlays

Scale at A3
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Role
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Suitability
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Project Number
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Drawing Number
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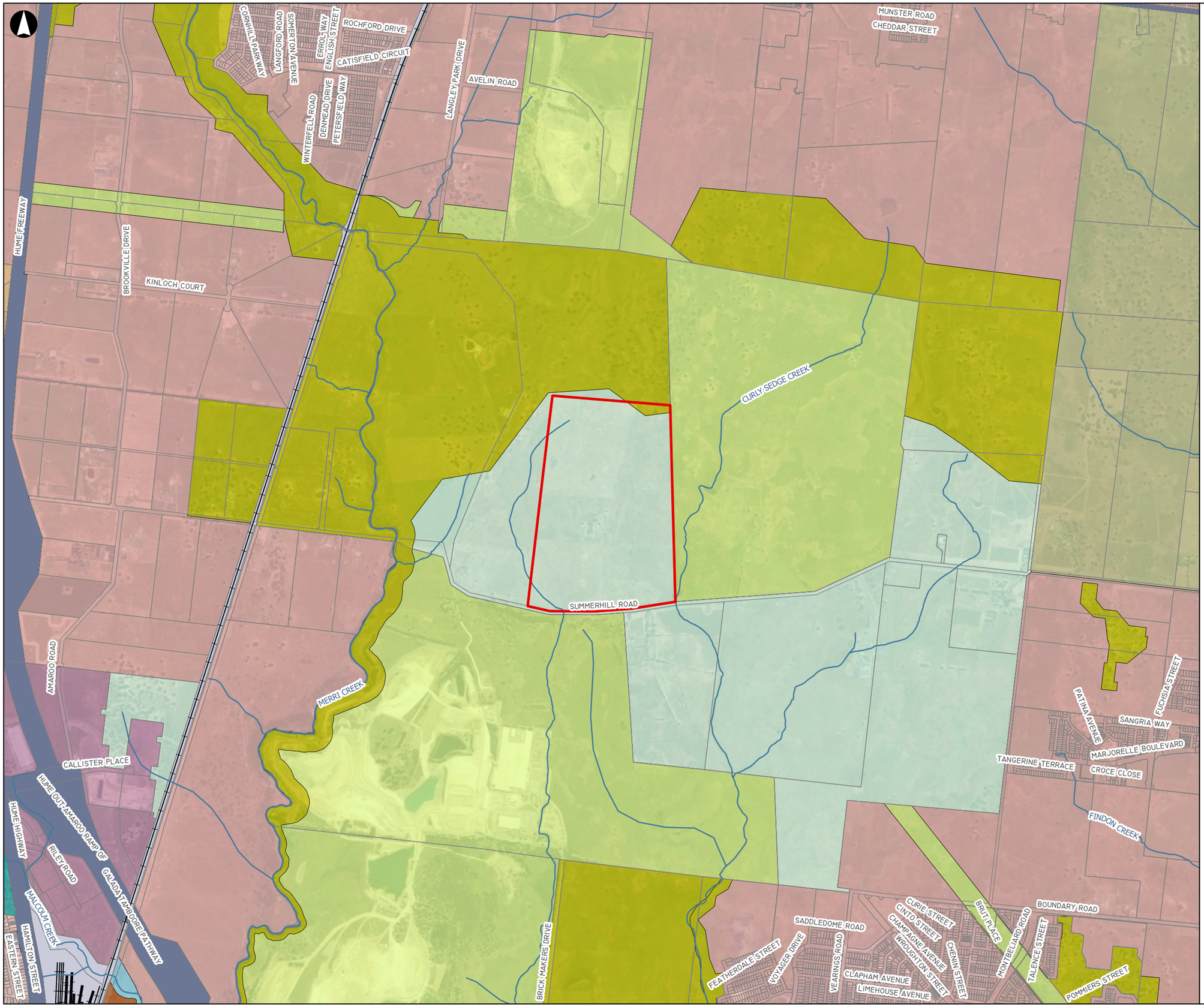

A3

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- Proposal area
- Cadastre
- Rail
- Watercourses

Planning zones

- Commercial 1 Zone (C1Z)
- Commercial 2 Zone (C2Z)
- Comprehensive Development Zone - Schedule 1 (CDZ1)
- Farming Zone (FZ)
- General Residential Zone - Schedule 1 (GRZ1)
- Green Wedge Zone (GWZ)
- Industrial 3 Zone (IN3Z)
- Rural Conservation Zone (RCZ)
- Rural Living Zone (RLZ)
- Special Use Zone (SUZ)
- State Transport Infrastructure (TRZ1)
- Principal Road Network (TRZ2)
- Urban Floodway Zone (UFZ)
- Urban Growth Zone (UGZ)

Coordinate System: GDA2020 MGA Zone 55

Earthstar Geographics VICMAP (2022)

Metres

0 250 500 1,000

Rev	Date	By	Chkd	Appd	Authd
A	4/11/2022	JB	JB	--	--

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Client

Cleanaway Operations Pty Ltd

Project Name

Melbourne Energy and Resource Centre (MERC)

Drawing Title

FIGURE 3.2 - Planning zones

Scale at A3

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Role

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Suitability

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287710	**A**

Drawing Number

01

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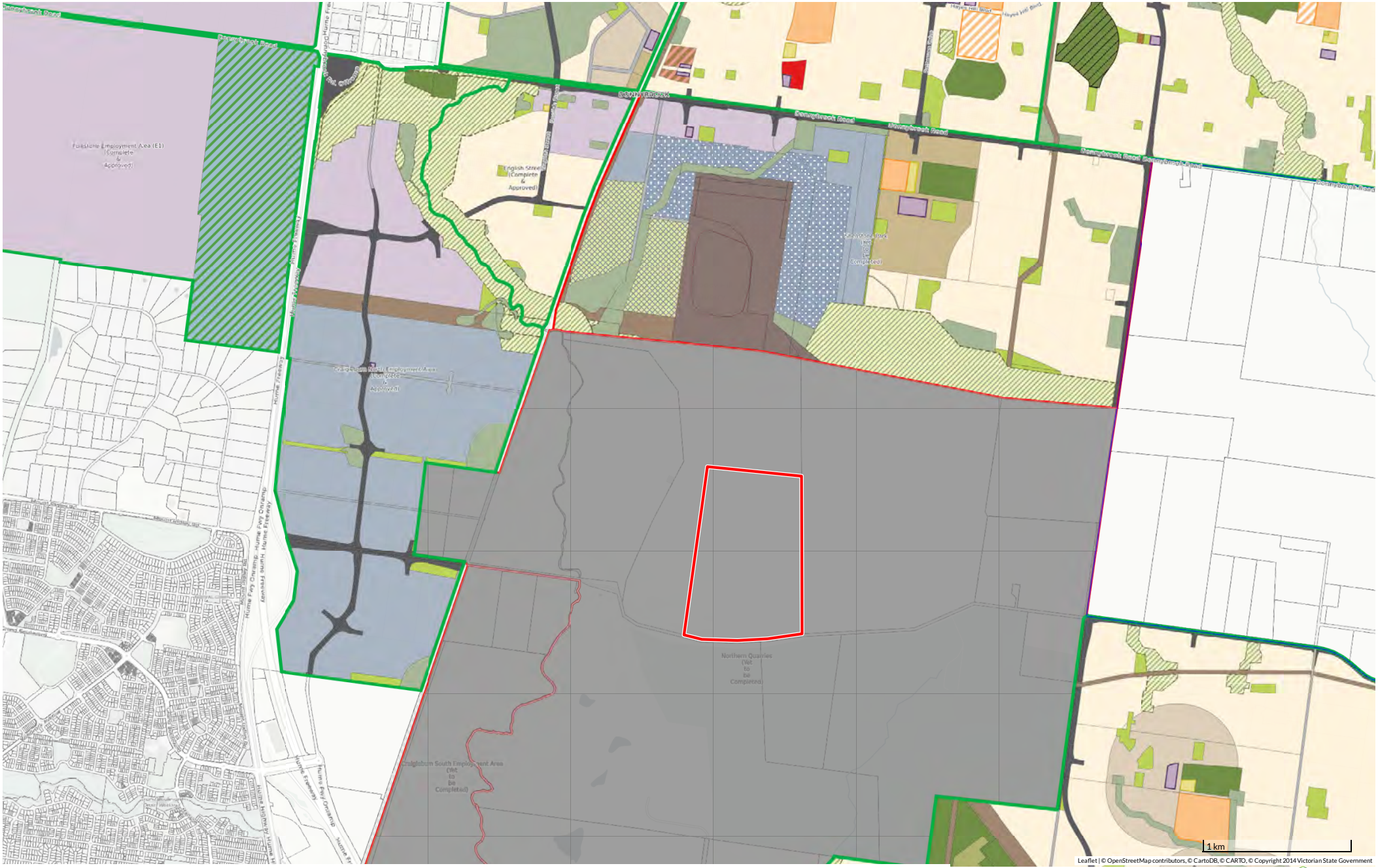
Donnybrook Road
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Fairstone Employment Area (E1)
(Complete
&
Approved)
English Street
(Complete
&
Approved)
Fairstone Park
(Not
to
be
Completed)
Craigieburn West Employment Area
(Complete
&
Approved)
Hume Freeway
Hume Freeway
Hume Fwy Onramp
Hume Fwy Onramp
Hume Freeway
Hume Freeway
Hume Fwy Onramp
Northern Quarries
(Not
to
be
Completed)
Craigieburn South Employment Area
(Not
to
be
Completed)
1 km
Leaflet | © OpenStreetMap contributors, © CartoDB, © CARTO, © Copyright 2014 Victorian State Government
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Figure 3.3

3.3 Population

Table 3.1 presents a summary of the populations in the relevant LGAs. Population data have been sourced from 2021 Census QuickStats[1] and 2021 Socio-Economic Indexes[2] from the Australian Bureau of Statistics. Data are compared to data for Victoria 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			Victoria	Australia
	City of Whittlesea LGA	Hume City Council LGA	Wollert	Craigieburn – north	Craigieburn - central		
Total population	229,396	243,901	24,407	12,309	8,099	6,503,491	25,422,789
Population 0 - 4 years	7.2% (16,508)	7.7% (18,851)	11.7% (2,867)	7.4% (916)	6.8% (551)	5.8% (375,900)	5.8% (1,463,817)
Population 5 - 19 years	20% (45,323)	21% (51,128)	22% (5,373)	25% (3,130)	20.7% (1,679)	18% (1,156,757)	18% (4,632,001)
Population 20 - 64 years	60% (138,039	60% (147,284)	62% (15,017)	61.2% (7,534)	61.4% (4,958)	60% (3,877,997)	59% (14,948,880)
Population 65 years and over	13% (29,522)	11% (26,623)	4.6% (1,154)	6% (737)	11% (894)	17% (1,092,843)	17% (4,378,088)
Median age	35	33	30	32	34	38	38
Household size	2.9	3.1	3.3	3.5	3.3	2.5	2.5
Unemployment	5.9%	7.5%	6.2%	7.6%	7.9%	5%	5.1%
Tertiary education	22.4%	21.2%	20.5%	20.6%	21.6%	24.5%	23.3%
SEIFA IRSAD	982	947	1,019	994	925	--	--
SEIFA IRSAD quintile	4	3	3	3	1	--	--
SEIFA IRSD	991	947	1,022	997	942	--	--
SEIFA IRSD quintile	4	2	3	3	2	--	--
Indigenous	1%	0.8%	0.6%	0.7%	1%	1%	3.2%
Born overseas	41.8%	44.9%	53.6%	49.5%	42.4%	35%	33.1%

Notes:

SEIFA IRSAD = index of socioeconomic advantage and disadvantage, Quintile is related to rank within Australia and ranges from 1 = most disadvantaged to 5 = most advantaged

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 VIC

lower than

greater than

Based on the population data available and presented in **Table 3.1**, these communities have:

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

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

These communities range from somewhat disadvantaged (Craigieburn-central) to around the average socioeconomic advantage/disadvantage compared to Victoria overall. The City of Whittlesea LGA is less disadvantaged compared to Victoria 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.

This Proposal will be implemented in proximity to a community that has indicated both potential for increased susceptibility (e.g. higher unemployment, lower levels of education) and decreased susceptibility (e.g. younger overall and less older people) to impacts from this facility based on the existing data.

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 population adjacent to the proposed site is relatively small and health data are not available that specifically relate to this population. However, it is assumed that the health of the local community is consistent with that reported in the larger City of Whittlesea LGA.

The Victorian Public Health Survey health statistics are provided at https://www.health.vic.gov.au/population-health-systems/victorian-population-health-survey . These statistics are generally updated on a yearly basis but not all factors are updated yearly. For this assessment, the Victorian Population Health Surveys from 2017 and 2020 have been used as these provide the greatest coverage of factors.

In relation to assessing health and wellbeing, the following additional baseline indicators are relevant (additional to and complementary to those presented in **Table 3.1** which addressed smaller population areas) and are presented in **Table 3.2**:

- Health indicators (health related behaviours and baseline health)
    - Compliance with fruit and vegetable consumption guidelines
    - Risk of alcohol related harm
    - Proportion overweight and obese
    - Physical activity levels which is relevant to understanding baseline levels of recreational activity that is an important factor in managing wellbeing
    - Proportion of the population that are smokers
    - Mortality in the population (all causes, cardiovascular and respiratory)
    - Morbidity/hospitalisations for cardiovascular disease and respiratory disease
    - Prevalence of asthma
- Wellbeing indicators
    - Whether the population feels safe walking outdoors within the community during the day or night time periods
    - Proportion of adults who experienced high or very high levels of psychological distress
    - Proportion of adults with self-reported health status as very good or excellent

- Proportion of adults with self-reported health status as fair/poor
- Proportion of adults with very high levels of satisfaction with life
- Proportion of adults with low or medium levels of satisfaction with life
- Proportion of the population wo reported a mental health condition in the most recent Census (ABS 2021)
- Wellbeing score, as determined by Vic Health, based on survey data on subjective wellbeing. The score is a value out of 100, where the following is noted (Weinberg & Tomyn 2015):
  - A score of 70 and higher reflects a normally functional homeostatic system, in the community
  - A score equal to or below 50 represents homeostatic failure
  - A score between 51 and 69 cannot be equivocally interpreted

- Indicators of social aspects of the community, as the proportion of adults who:
  - had feelings of trust (could trust most people)
  - had feelings of being valued by society
  - resided in the neighbourhood for 10 years or longer
  - had close friends or family to talk to regularly
  - talked to friends every day or a few times per week
  - talked to family every day or a few times per week
  - felt people in their neighbourhood would help out
  - felt they live in a close-knit neighbourhood
- Resilience score for the community, which is a score ranging from 0 to 8, with higher values representative of more resilient communities.

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

Parameter/measure	Whittlesea LGA	Victoria
Health behaviours		
Met vegetable consumption guidelines – adults[4]	2.5%	5.4%
Met fruit consumption guidelines – adults[4]	44.2%	43.2%
Increased risk of alcohol-related harm – adults yearly[4]	18.9%	18.2%
Adult population defined as overweight but not obese[3]	28.5%	30.1%
Adult population defined as obese[3]	24.6%	20.9%
Adults - current smokers[3]	21.4%	16.4%
Sufficient levels of physical activity – adults[4]	48.8%	50.9%
Baseline health (as age standardised rate per 100,000)		
Premature mortality - all causes (2016-2020)[6]	267.1	220.0
Premature mortality – cardiovascular (2016-2020)[6]	43.3	40.7
Premature mortality – respiratory disease (2016-2020)[6]	16.6	13.9
Hospitalisations – cardiovascular disease (2019/20)[6]	1888.1	1450.7
Hospitalisations – respiratory disease (2019/20)[6]	1440.7	1264.9
Adults ever diagnosed with asthma[4]	18.6%	20.0%
Children reported to have asthma[7]	7.7%	9.6%
Children with asthma and have an action plan at school[7]	61.7%	62.3%
Wellbeing[3]		
Feelings of safety during the day[5]	88.2%	92.5%
Feelings of safety at night[5]	47%	55.1%
Proportion of adults who experienced high or very high levels of psychological distress	30.6%	23.5%
Proportion of adults with self-reported health status as very good or excellent	30.7%	40.5%
Proportion of adults with self-reported health status as fair/poor	24.2%	21.4%
Proportion of adults with very high levels of satisfaction with life	28.3%	26.9%

Parameter/measure	Whittlesea LGA	Victoria
Proportion of adults with low or medium levels of satisfaction with life	26%	22.3%
Wellbeing score (out of 100)[5]	76.9	77.3
Social capital, as proportion of adults who[3]:		
had feelings of trust (could trust most people)[5]	60.1%	71.9%
had feelings of being valued by society	46.5%	51.6%
resided in the neighbourhood for 10 years or longer	34.2%	41.5%
had close friends or family to talk to regularly	94.0%	95.2%
talked to friends every day or a few times per week	69.6%	79.5%
talked to family every day or a few times per week	78.1%	75.7%
felt people in their neighbourhood would help out[5]	68.9%	74.1%
felt they live in a close-knit neighbourhood[5]	51.7%	61.0%
Resilience		
Resilience score (score out of 8)[5]	6.1	6.4

Notes:

* = Estimate has been determined (by Vic health) to be statistically different to the estimate for Victoria, with values in red being statistically higher, and values in blue being statistically lower (noting the statistical significance is not available for all indicators listed)

3 = Data reported from Victorian Population Health Survey – 2020 Results

4 = Data reported from Victorian Population Health Survey 2017 (noting data for individual LGAs not provided in surveys reported between 2018 and 2020)

5 = Data reported by Vic Health and presented in LGA Profiles, based on data from 2015 survey (noting no more recent data is available for these indicators), https://www.vichealth.vic.gov.au/media-and-resources/publications/vichealth-indicators-lga-profiles-2015

6 = Data for Victoria from Social Health Atlas of Australia (accessed in 2022), https://phidu.torrens.edu.au/social-health-atlases/data#social-health-atlases-of-australia-local-government-areas

7 = Data from school entrant health questionnaire 2021, as reported in the Outcomes for Victorian Children at School Entry, https://www.vic.gov.au/school-entrant-health-questionnaire

Review of the data presented in **Table 3.2** indicates that the population in the Whittlesea LGA, assumed to be representative of the population potentially impacted by the Proposal, is generally similar to the larger Victorian population, however, this LGA has a higher proportion of individuals who are obese and higher rates of mortality (all causes) and hospitalisations for cardiovascular and respiratory disease than Victoria overall.

The population in the LGA has a lower proportion with a self-reported health status as very good or excellent but a statistically higher proportion with a very high satisfaction with life. Many of the other wellbeing indicators are similar to the Victorian population, however, Whittlesea LGA has a lower resilience score.

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 (2023), Melbourne Energy and Resource Centre, Air quality assessment. Report dated 23 February 2023.
- Air Quality Modelling Output Spreadsheets provided by Katestone (received 17/3/23)

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

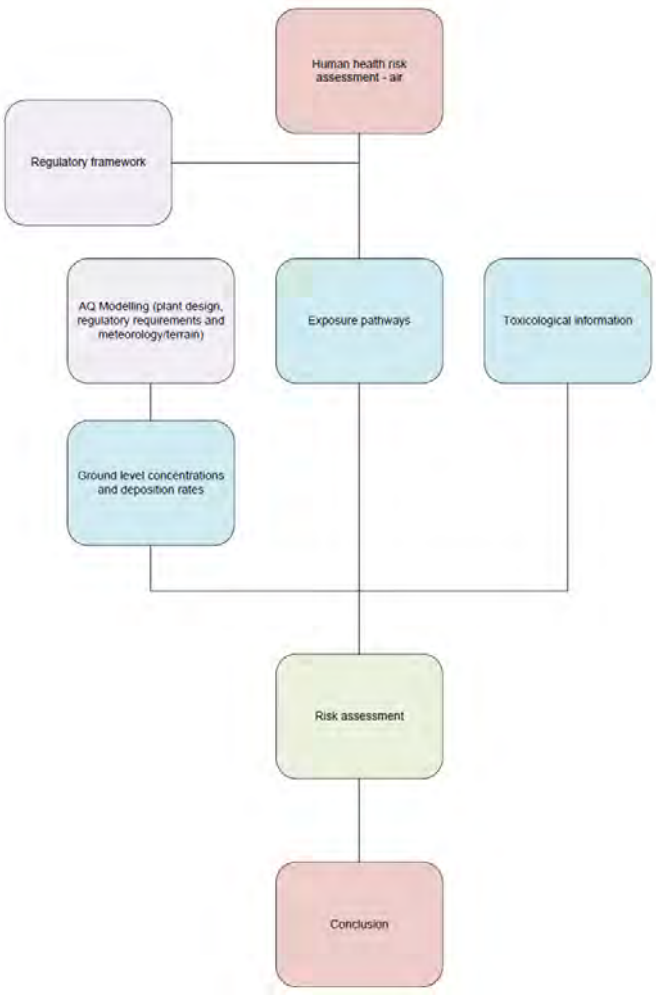


Chart 4.1: Approach to quantitative risk assessment for air emissions

4.2 Site History

The information in the contamination report[3] provides the following understanding of the site conditions (including historical uses) relevant for this assessment:

- The study area is underlain by the Quaternary Age 'Newer Volcanics' comprising basalt with groundwater expected to be within the basalt at depths of between 5 m and 20 m below surface levels.
- Surface levels vary between approximately 224 m AHD (Australian Height Datum) and 208 m AHD and slope downward in a south-westerly, south and south-easterly direction.
- Surface water features include two ephemeral streams and three farm dams.
- The Study Area has been used for agricultural purposes since at least 1963. The site was vacant until between 1991 and 2002 when the current house and associated buildings were constructed.
- Major overhead powerlines were constructed in an easement through the north-eastern portion of the Study Area sometime between 1963 and 1972. The lines were duplicated prior to 1984.
- Evidence of the historic placement of fill was noted in the south-eastern corner of the area in the 2009 aerial photograph.
- Surrounding land is typically used for agricultural purposes, with the exception of the Austral Bricks facility located on the southern side of Summerhill Road where quarrying activities occur for brick making. A quarry is also present to the north of the Study Area.
- One EPA Licensed Site (i.e. Austral Bricks) is within 2 km of the site, however, this is considered to be located down-hydraulic gradient of the site.
- A former solid inert waste landfill (closed in 1998) was identified within the area. This site is considered to represent a low risk of contamination to the Study Area.

4.3 Conceptual site model

Understanding how a community member may come into contact with pollutants released in air emissions from the proposed WtE 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 the following **Figures 4.1** and **4.2**.

[3] Douglas Partners (2022) Draft report on Soil Contamination and Baseline Groundwater Investigation, Melbourne Energy and Resource Centre, 510 Summerhill Road, Wollert, VIC, Dated November 2022.

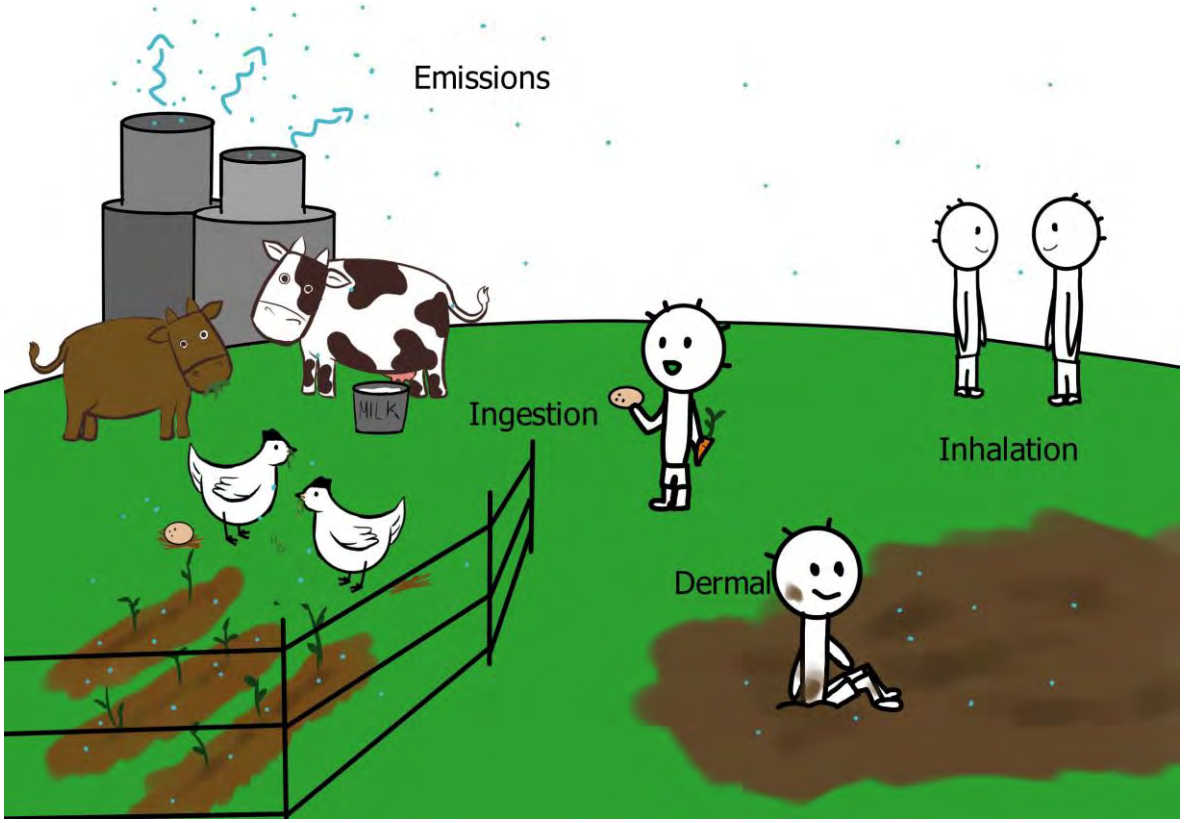


Figure 4.1: Conceptual site model (illustrative only)

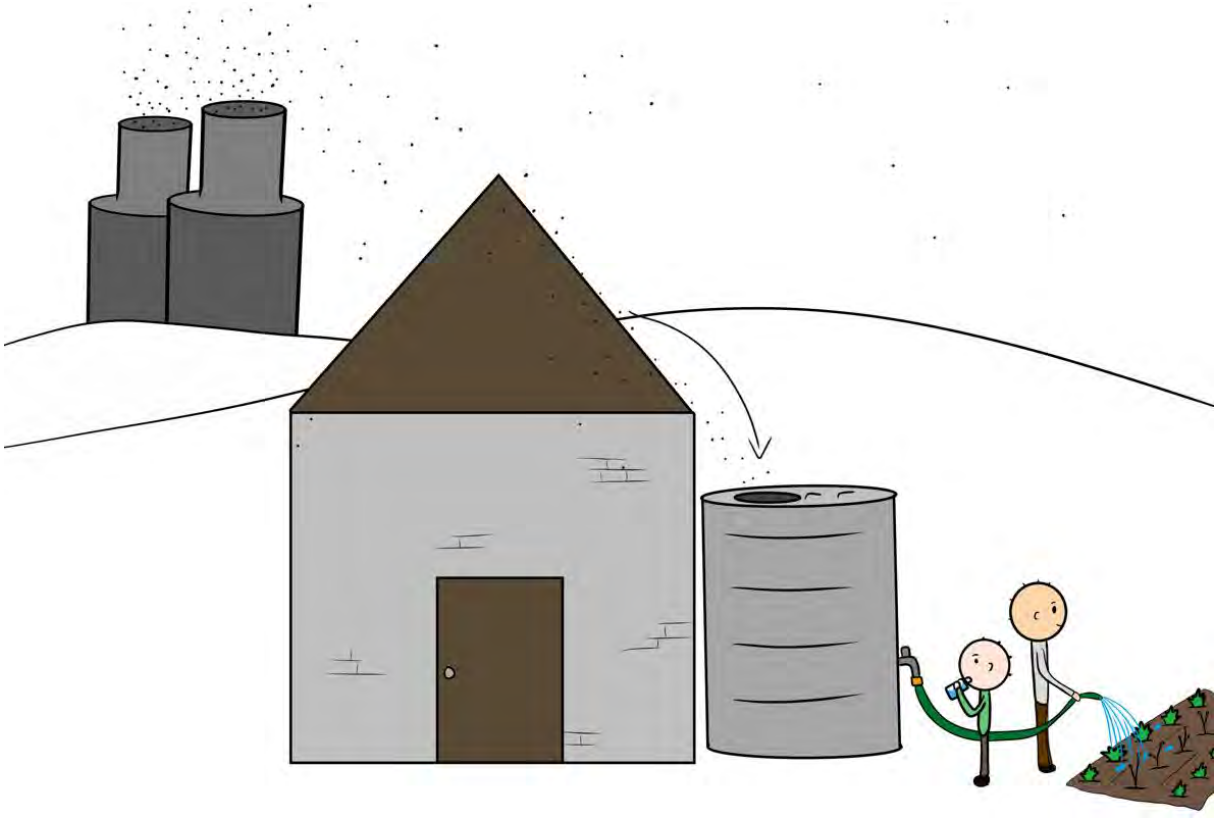


Figure 4.2: Conceptual site model (illustrative only) – Rainwater tanks

There are three main ways a person may be exposed to a chemical substance emitted from the proposed WtE 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 4.3, 4.4** and **4.5**) illustrate how the emissions get mixed into the atmosphere.

Figure 4.3: Steam rising as it is discharged from an industrial facility

Gases (and fine particles) are emitted at around 131^{o}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 hot gases and gases are travelling at a faster speed are less dense and rise above the air surrounding the stack. This can be seen in **Figure 4.3**.

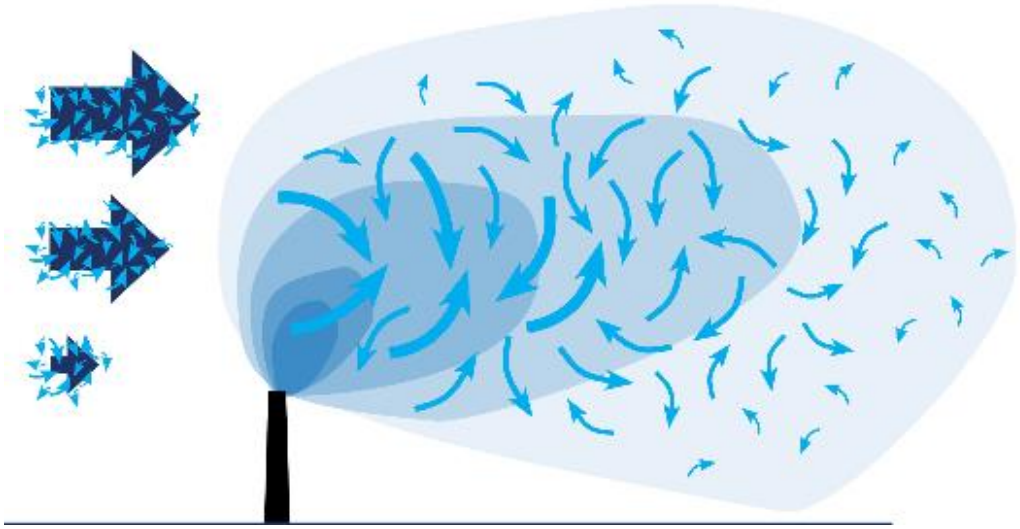


Figure 4.4: Turbulence in the air, how it mixes and dilutes pollutants (NSW Chief Scientist 2018)

As the gases (and fine particles) cool and slow down they begin to interact with the wind above the stack (60 m high). This mixes the gases (and fine particles) into the atmosphere decreasing the actual concentration present in any particular location. **Figure 4.4** 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).

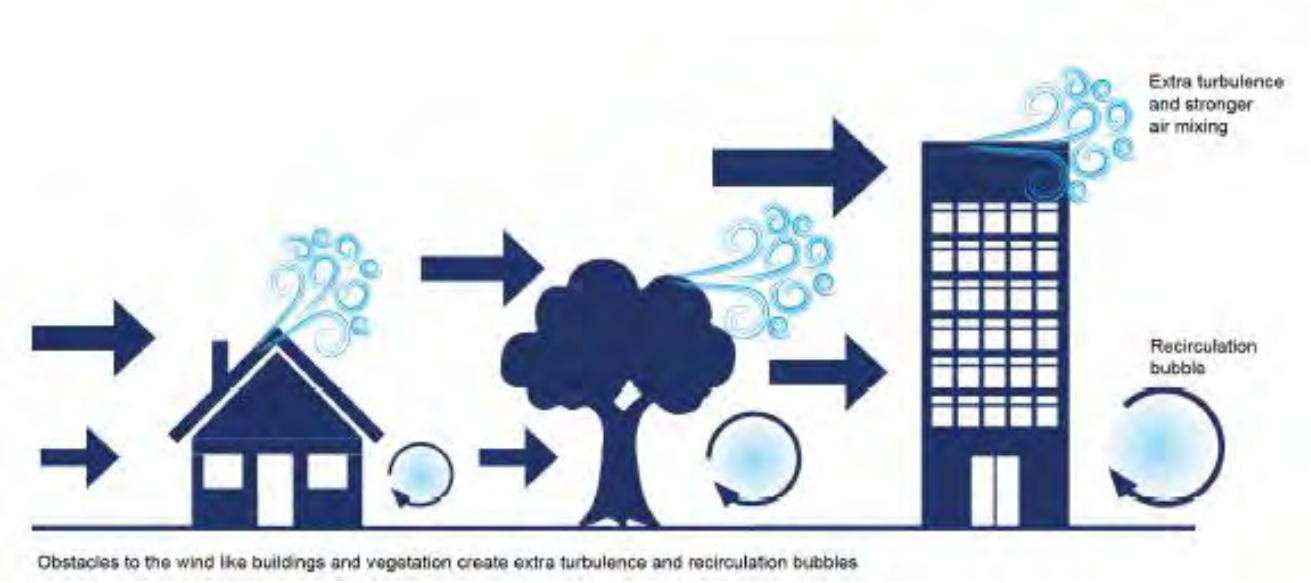


Figure 4.5: Turbulence in the air and how it is affected by buildings and vegetation (NSW Chief Scientist 2018)

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 4.5**). 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 emissions from the proposed WtE 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 NO_2, SO_2, HCl, HF and CO can be considered in this category.

Other chemicals are more likely to be attached to particles. Fine particulate matter is normally classified as particles less than 10 micrometres (i.e. PM_{10}) and particles less than 2.5 micrometres (i.e. $PM_{2.5}$). For combustion sources like the proposed facility, it is conservatively assumed that most of the particles that could be released would be classified as $PM_{2.5}$. Particles 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 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 WtE 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 NO_2, SO_2, 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 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 wastes).

Dioxin-like compounds are also commonly formed in all combustion processes (including natural combustion processes such as bushfires, grass fires or controlled burns, which have no emission controls other than extinguishing them) 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 organic compounds (TOC)	
Ammonia	
PM_{10}	**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.
$PM_{2.5}$	
Antimony	**Inhalation** of these pollutants adhered to fine particulates **Ingestion and dermal contact** with these pollutants deposited to soil or water **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.
Arsenic	
Beryllium	
Cadmium	
Chromium	
Cobalt	
Copper	
Lead	
Manganese	
Mercury	
Nickel	
Selenium	
Thallium	
Tin	
Vanadium	
Dioxins & furans	

The full set of exposure pathways considered in this assessment is shown in **Figures 4.1** and **4.2**.

The assessment has relied on the Katestone Air Quality Assessment (AQA) (Katestone 2022). No independent review of the AQA has been undertaken as part of this assessment but is being undertaken as part of the development of all the planning documents. 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 2012b).

4.4 Existing Air Quality

Katestone (2022) provides a detailed assessment of local air quality relevant for the site.

The closest air quality monitoring station (operated by EPA Victoria) is Alphington, which is located approximately 23 km to the south of the site (**Figure 4.6**). Information about the air quality monitoring stations is available at the EPA Victoria website[4].

The station collects data for NOx, SO_2, particulate matter (PM_{10}, $PM_{2.5}$) and CO. The station also collects meteorological data.

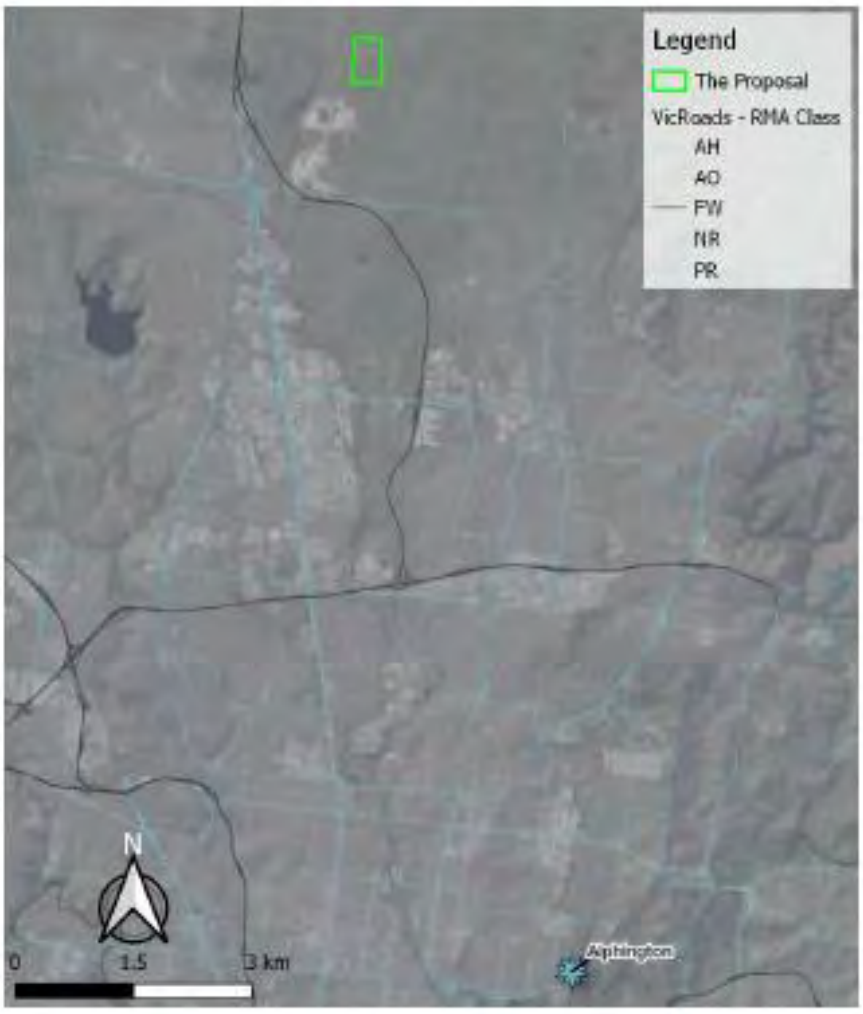


Figure 4.6: Monitoring station location

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

[4] https://www.epa.vic.gov.au/for-community/airwatch?location-search-field=Alphington+3078&siteId=c69ed768-34d2-4d72-86f3-088c250758a8&location=%5B-37.7805624%2C145.0322776%5D.

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.

4.5 Modelling

4.5.1 General

Modelling was undertaken using a combination of the AERMET/AERMOD Modelling System and The Air Pollution Model (TAPM). TAPM is a prognostic air model used to simulate data for the upper atmosphere for input into AERMET/AERMOD. AERMET/AERMOD is endorsed for this purpose by the EPA Victoria (Katestone 2022).

The modelling was undertaken to predict the concentration of emissions from the proposed WtE facility (Katestone 2022).

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 2020):

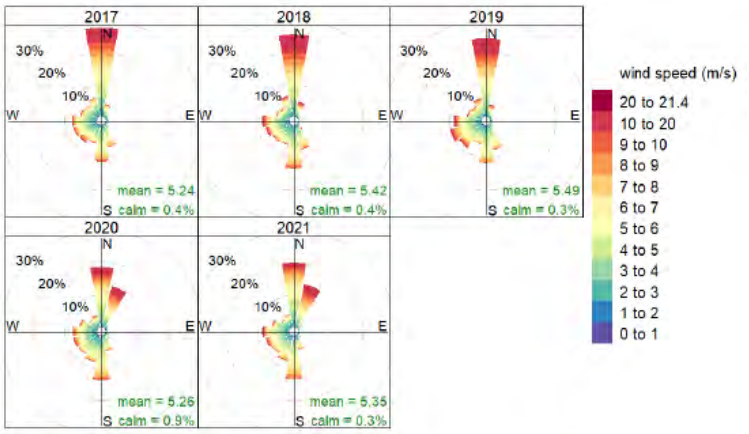
- local terrain (topography and buildings)
- meteorological data
- plant design (for example, stack location and height)
- air emissions estimates (stack concentrations).

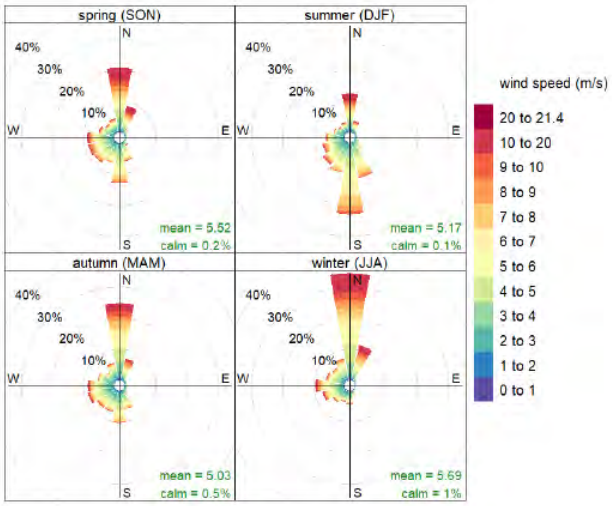
Modelling evaluated potential concentrations for relevant chemicals at ground level across a grid of approximately 3 km x 3 km centred on the facility. The grid used a 25 m resolution.

4.5.2 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 4.7**.

The annual pattern indicates that winter exhibits dominant northerly winds, while in autumn and spring the dominant winds are from the north but there is an increase in winds from the southwest. Winds from the south are most prevalent for summer months. These roses also show that the strongest winds occur between midday to 6 pm, with an average wind speed reaching 6.3 m/s. Wind directions are predominantly from the north between midnight to midday, shifting to southerly from midday to 6pm before northerly winds dominate in the evening.





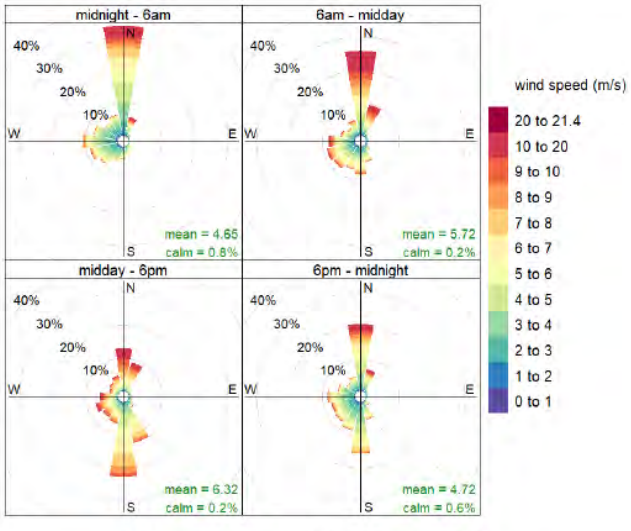


Figure 4.7: Annual and seasonal windroses

4.5.3 Stack Parameters

The stack parameters (i.e. plant design parameters) considered in the dispersion modelling scenarios for the various different load points are summarised below in **Table 4.3.** The concept of load points is discussed in **Section 2.4.4**.

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 should be engineered to optimise mixing, however, the height of the stack is also influenced by community feedback and preferences.

The stack parameters used in the modelling – assumed relevant for LP1 (i.e. average/optimum conditions) (as per **Section 2.4.4**) are shown in **Table 4.3**.

Table 4.3: Stack parameters assumed for modelling emissions when the plant is operating at LP1

Parameter	Value	Units
Single line		
Stack height	60	m
Exhaust diameter	1.85	m
Exhaust temperature	131	°C
Exhaust velocity	20	m/s
Moisture content	16.7	% by volume
Oxygen content	6.3	% by volume (wet)
Flow rate	190,136	m^3 per hour (wet, actual)
Flow rate (dry, NTP, 11% O_2 with 10% additional flow rate)	158,705	Nm^3 per hour (@ 11% O_2, dry)
Two lines (single stack)		
Exhaust diameter (effective)	2.6	m
Exit velocity	20	m/s
Flow rate (dry, NTP, 11% O_2 with 10% additional flow rate)	317,409	Nm^3 per hour (@ 11% O_2, dry)

The plant will have two lines and each will have a stack. Each stack or flue has a diameter of 1.85 m and a height of approximately 60 m. The 2 stacks will be encased together so that there is 1 visible stack for the facility.

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.

It is important to note, that the systems are designed to ensure the combustion chamber is fuelled at an optimal rate, and there are limits to how long the plant can operate at higher or lower load point rates, without affecting the life of the equipment. 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 (2022)). This is the basis of the load points discussed next.

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. The moving grate combustion system is designed to handle a wide calorific value range for the waste feedstock, as this will vary continuously depending on the mix of waste being burnt. Premixing of waste in the bunker helps to smooth out these inevitable variations. The combustion system has both thermal and mechanical limitations which are defined by the combustion capacity (or firing) diagram. When the calorific value of the waste is low, more waste can be fed into the combustion system to achieve the desired level of thermal input, unless the value is so low that its not physically possible to input enough waste (i.e. a mechanical limit on throughput is reached) to reach the appropriate temperatures. Conversely, if the calorific value of the waste is high, the combustion system will reach its thermal limit more quickly and the waste feed rate will need to be reduced.

The plant will be designed to reduce throughput or suspend combustion of waste or utilise auxiliary firing, 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 while the boiler line is being shutdown. Emergency generators 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 will typically never be more than modelled in this assessment as the pollution control equipment will continue to operate in most circumstances.

In regard to the start-up phase, no waste will be permitted to enter the combustion chamber until the chamber has reached the correct operating temperature.

4.5.4 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 Victoria include:

- Environment Protection Act (2021)
- EPA Victoria Guideline: Energy from Waste (EPA Victoria 2017)

- 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 relevant legislation or policy – i.e. the most recent European values. This is a conservative approach as, when a plant operates, it's actual stack concentrations will be lower than these limit values so emissions from the facility into the surrounding environment will be lower.

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.

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 (EU 2010, 2019).

Table 4.4: Modelled stack emission concentrations (EU 2019)

Pollutant	Units @ 11% O_2	Design/modelled concentrations – LP1	
		Daily average	Short term (0.5 hr)
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_2	mg/Nm^3	30	200
NO_x (calculated as NO_2)	mg/Nm^3	120	400
NH_3	mg/Nm^3	10	ND
Hg	mg/Nm^3	0.02	ND
Cd+Tl	mg/Nm^3	0.02	ND
Other metals	mg/Nm^3	0.3	ND
Dioxin-like compounds (WHO TEQ) (i.e. dioxins, furans and dioxin like PCBs)	ng/Nm^3	0.06	0.1
Polycyclic aromatic hydrocarbons (PAHS (as BaP)	$\mu g/Nm^3$	0.2	ND

Notes:
ND not defined

The emissions considered in the AQA include the following pollutants:

- oxides of nitrogen (NOx)
- sulfur dioxide (SO_2)
- carbon monoxide (CO)
- particulates as PM_{10} and $PM_{2.5}$
- hydrogen fluoride (HF)
- hydrogen chloride (HCl)
- ammonia (NH_3)

- metals (cadmium, thallium, mercury, antimony, arsenic, lead, chromium (conservatively assumed to be 100% chromium VI as this is the most toxic form), cobalt, copper, manganese, nickel, vanadium)
- volatile organic compounds (assumed to be present as 100% benzene or to be present as 100% formaldehyde – both options considered in the assessment)
- dioxins, furans and dioxin-like PCBs (i.e. dioxin-like compounds)
- polycyclic aromatic hydrocarbons (as benzo[a]pyrene).

In regard to oxides of nitrogen, the most important form that needs to be considered in this risk assessment is NO_2. Katestone (2023) notes that the modelling assumed 30% conversion of NOx to NO_2.

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 waste-to-energy facilities in Europe. The wastes used as fuels for these facilities are similar to what is to be used for this facility. 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 have 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 4.5**.

Table 4.5: Metal distribution provided by Ramboll

Metal	Clean gas		Short term upset condition scenario	
	Cd and Tl	Other metals	Cd and Tl	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 (Tl)	41%		41%	
Vanadium (V)		1%		0.005%

4.5.5 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 15 m) across a 100 km^2 area (i.e. approximate 10 km x 10 km) and at each discrete receptor location as shown on **Figure 4.8**:

- Meteorological data for the area
- Grid across the relevant area
- Stack parameters (stack design and loading information)
- Stack concentrations

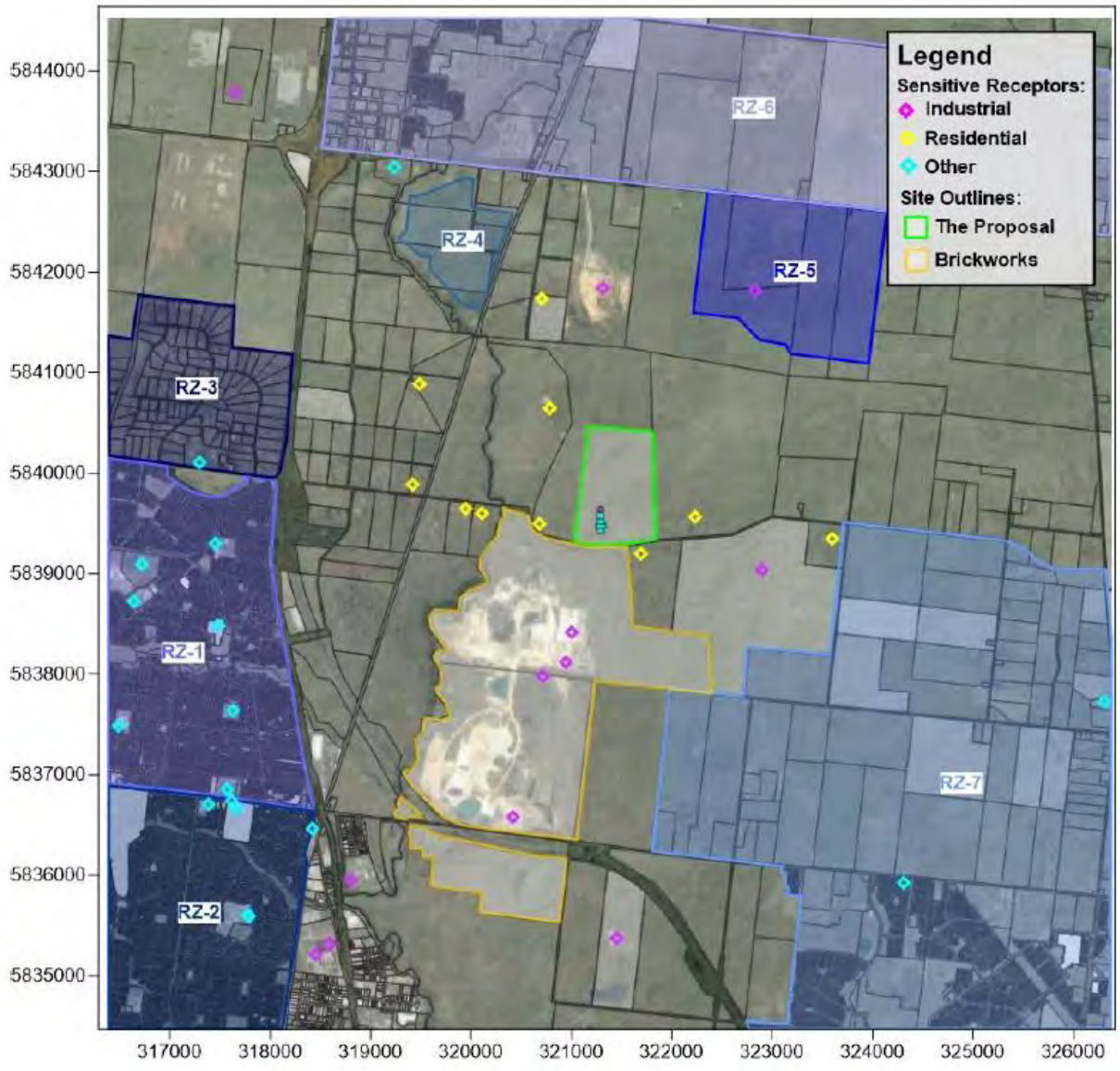


Figure 4.8: Sensitive receptors and receptor zones evaluated in the air quality modelling

The assumed concentrations in the stack for each pollutant, as discussed in **Section 4.5.4,** were combined with the engineering information about the design of the stack (size, exit velocity) 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.

The modelling results were then refined into 2 different scenarios for use in the air quality assessment and in this risk assessment. 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* stack concentration limits
    - Assumes both lines are operating at these stack concentrations
    - Relevant meteorological conditions across the year
    - Stack parameters as discussed in **Section 4.5.3.**

  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
    - *short term* 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 4.5.3.**

  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.

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 have been assessed for all pathways in **Section 5**.

Understanding the potential ground level concentrations from this facility in the context of what is already likely to be present in a location is important. This concept of cumulative exposure has been considered in this assessment. For the criteria pollutants (NOx, SOx, CO, PM), government air quality monitoring stations provide data on regional air quality – these ambient background levels have been incorporated into the modelling. In addition, the Brickworks facility, located to the south of the site, also has emissions which contribute to ground level concentrations for key pollutants. The Brickworks is already in operation so emissions from the Brickworks are already present in air in the region.

The cumulative case has been developed as shown in **Table 4.6**.

Table 4.6: Basis of cumulative case

Pollutant	Proposed facility	Brickworks	Ambient background monitoring
NO_2	✓	✓	✓
CO	✓	✓	✓
SO_2	✓	✓	✓
$PM_{2.5}$	✓	✓	✓
PM_{10}	✓	✓	✓
HCl	✓	✓	×
HF	✓	✓	×
NH_3	✓	×	×
TVOC (as 100% benzene)	✓	×	×
TVOC (as 100% formaldehyde)	✓	×	×
Dioxin-like compounds	✓	✓	×
Cadmium	✓	✓	×
Thallium	✓	×	×
Arsenic	✓	✓	×
Cobalt	✓	✓	×
Chromium	✓	✓	×
Copper	✓	✓	×
Mercury	✓	✓	×
Manganese	✓	✓	×
Nickel	✓	✓	×
Lead	✓	×	×
Antimony	✓	×	×
Vanadium	✓	×	×
Polycyclic aromatic hydrocarbons (PAHs as BaP)	✓	×	×

This assessment has also been based on the ground level concentrations for the cumulative case – i.e. including the Brickworks and background data where appropriate.

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 at any residential location
- maximum at any commercial/retail/industrial location

- maximum at any of the “other places” (i.e. churches, schools, childcare, hospitals, clubs)
- 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 and so demonstrating that the risks for these “maximum” locations are acceptable confirms that the risks at all locations in each category are acceptable.

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.

5.2 Construction

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

Katestone (2023) undertook a review of the potential for such emissions of dust during construction.

The assessment noted that there were only a small number of receptors present within the area where dust from the works might reach. The closest receptors are two residential locations at around 350 m from the site.

The sensitivity of the area to dust was judged to be low, however, a range of mitigation measures are still proposed to be included in the Construction Environmental Management Plan (CEMP) to ensure dust is well managed during construction. These may include:

- Preparing a Dust Management Plan (DMP) (a normal requirement for such sites).
- Planning site layout so that machinery and dust causing activities are located away from receptors, as far as reasonably practicable.
- Erect solid screens or barriers around dusty activities or the site boundary that are at least as high as any stockpiles on site.
- Fully enclose site or specific operations where there is a high potential for dust production and the site is active for an extended period.
- Avoid site runoff of water or mud.
- Keep site fencing, barriers and scaffolding clean using wet methods.
- 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.
- Ensure all vehicles switch off engines when stationary - no idling vehicles.
- Avoid the use of diesel or petrol powered generators 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 (if long haul routes are required these speeds may be increased with suitable additional control measures provided, subject to the approval of the nominated undertaker and with the agreement of the local authority, where appropriate).
- Covering or watering exposed stockpiles when not in use.
- Revegetating disturbed areas as soon as is feasible.
- Covering truck cargo when entering or leaving the site.
- Hosing down truck tyres before exiting the site.
- Avoiding dry sweeping of large areas, instead using hoses, sprays, or water trucks.

- 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.
- Use dust suppression techniques when grinding, cutting, or sawing (water sprays, filter extractions).
- Ensure adequate supply of water for mitigation use.
- 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 the local authority when asked.
- Carry out regular site inspections to monitor compliance with the DMP, record inspection results, and make an inspection log available to the local authority when asked.
- 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 and during prolonged dry or windy conditions.
- 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 to the local authority when asked.
- Record any exceptional incidents that cause dust and/or air emissions, either on- or off-site, and the action taken to resolve the situation in the log book.

These are mitigation measures which are common for large construction projects and will ensure that dust 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 bunker as the wastes are prepared for introduction into the combustion chamber. This means the exposures relevant for these workers are those similar to workers at a landfill or at other waste processing facilities.

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 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 VIC 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. Altona, Benalla, Bairnsdale, Alexandra, Dandenong)
- Cleanaway Resource Recovery Centres (e.g. Clayton South)
- Cleanaway Daniels Medical Waste Services (e.g. Dandenong South)

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 5.5.3.6**). 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 PM_{10}, $PM_{2.5}$, NO_2, SO_2, CO. 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 April 2021. This version includes an update to the goals for NO_2 and SO_2 as well as the updated guidelines for particulates that come into effect in 2025 (NEPC 2021b).

The ground level concentrations of these criteria pollutants have been estimated in the air quality modelling (Katestone 2023).

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 and the emissions from the Brickworks nearby.

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 NEPM criteria are the guidelines that have been adopted in the Environmental Reference Standard (ERS) for Victoria (Victorian Government 2021).

The ERS/NEPM criteria relate to total exposures to these criteria pollutants (PM_{10}, $PM_{2.5}$, NO_2, SO_2, CO) which means background or existing levels plus any additional impact from the proposed facility. The cumulative case including the Brickworks facility has also been included.

The results for the criteria pollutants have been assessed for:

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

5.4.2 Sulfur oxides (SOx)

Sulfur oxides are formed during combustion when chemicals present in fuels (such as coal, gas, petrol) containing sulfur 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. Volcanos, wildfires and other types of fires are also sources to the atmosphere of sulfur oxides (USEPA 2018a).

Sulfur dioxide (SO_2) 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 2018a).

Guidelines are available from EPA Victoria (EPA Victoria 2022), ERS (Victorian Government 2021) and NEPC (NEPC 2021b) which indicate concentrations of sulfur dioxide considered to be acceptable by national health authorities. EPA Victoria guidelines for sulfur dioxide are the same as the guidelines listed in the ambient air NEPM (NEPC 2021b).

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.

Table 5.1 shows the comparison of modelled SO_2 levels and the relevant NEPM guidelines for the facility alone and, for the cumulative case, including this project + existing background + Brickworks.

Table 5.1: SO_2 impacts from the project

Parameter	SO_2 (µg/m^3)		
	1-hour average	24-hour average	Annual average
Guideline (NEPM 2021a)	262 (100 ppb)	52 (20 ppb)	Guideline removed
Maximum off-site location			
Contribution from project (increment)	14	6.4	1.2
% contribution of project to NEPM/ERS	5.3%	12%	--
Cumulative case (project + background + Brickworks)	197	64	9.3
Maximum residential location			
Contribution from project (increment)	8.6	2.3	0.2
% contribution of project to NEPM/ERS	3.3%	4.4%	--
Cumulative case (project + background + Brickworks)	132	44	5.5
Maximum commercial location			
Contribution from project (increment)	6.4	3.5	0.7
% contribution of project to NEPM/ERS	2.4%	6.7%	--
Cumulative case (project + background + Brickworks)	82	21	4.1
Maximum other places location			
Contribution from project (increment)	3.2	0.3	0.02
% contribution of project to NEPM/ERS	1.2%	0.6%	--
Cumulative case (project + background + Brickworks)	178	18	2.1
Maximum on-site location			
Contribution from project (increment)	8.1	NA	NA
% contribution of project to NEPM/ERS	3.1%	--	--

Parameter	SO_2 (µg/m^3)		
	1-hour average	24-hour average	Annual average
Cumulative case (project + background + Brickworks)	130	NA	NA

On this basis, the operation of the facility is not expected to significantly contribute to, or change ambient levels of SO_2 and the cumulative ground level concentrations are in compliance with the most recent national guidelines.

It is noted that these national guidelines are actually not 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 Melbourne as a whole, for example) (NEPC 2021a).

The results for the maximum off-site location shows that cumulative sulfur dioxide levels are around the NEPM guideline. The primary source for sulfur dioxide in this situation is the Brickworks. The incremental emissions from this facility are around 12% of the guideline and the ambient background levels are around 20% of the guideline. The rest of the estimated daily average is from the Brickworks. This proposed facility will not change the emissions sulfur dioxide to any measurable extent.

5.4.3 Nitrogen Oxides (NOx)

Nitrogen oxides (NOx) 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 2013b). Asthmatics, the elderly and people with existing cardiovascular and respiratory disease are particularly susceptible to the effects of elevated nitrogen dioxide (Morgan et al. 2013; NEPC 2010). The health effects associated with exposure to nitrogen dioxide depend on the duration of exposure as well as the concentration.

Guidelines are available from EPA Victoria (EPA Victoria 2022), ERS (Victorian Government 2021) and NEPC (NEPC 2021b) which indicate concentrations of nitrogen dioxide considered to be acceptable by national health authorities. The EPA Victoria guidelines for nitrogen dioxide are the same as the guidelines listed in the ambient air NEPM.

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.

Table 5.2 shows the comparison of modelled NO_2 levels and the relevant NEPM guidelines for the facility alone and, for the cumulative case, including this project + existing background + Brickworks.

Table 5.2: NO_2 impacts from the project

Parameter	NO_2 (µg/m^3)	
	1-hour average	Annual average
Guideline (NEPM 2021a)	150 (0.08 ppm)	28 (0.015 ppm)
Maximum off-site location		
Contribution from project	17	1.4
% contribution of project to NEPM/ERS	11%	5%
Cumulative case (project + background + Brickworks)	117	22
Maximum residential location		
Contribution from project	10.4	0.2
% contribution of project to NEPM/ERS	6.9%	0.7%
Cumulative case (project + background + Brickworks)	117	20
Maximum commercial location		
Contribution from project	7.7	0.8
% contribution of project to NEPM/ERS	5.1%	2.9%
Cumulative case (project + background + Brickworks)	117	20
Maximum other places location		
Contribution from project	3.9	0.02
% contribution of project to NEPM/ERS	2.6%	0.07%
Cumulative case (project + background + Brickworks)	117	20
Maximum on-site location		
Contribution from project	9.7	NA
% contribution of project to NEPM/ERS	6.5%	--
Cumulative case (project + background + Brickworks)	117	NA

On this basis, the operation of the facility is not expected to significantly contribute to, or change ambient levels of NO_2 and the cumulative ground level concentrations are in compliance with the most recent national guidelines.

It is noted that these national guidelines are actually not 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 Melbourne as a whole, for example) (NEPC 2021a).

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 EPA Victoria (EPA Victoria 2022), ERS (Victorian Government 2021) and NEPC (NEPC 2021b) which indicate concentrations of carbon monoxide considered to be

acceptable by national health authorities. The EPA Victoria guidelines for carbon monoxide are the same as the guidelines listed in the ambient air NEPM (NEPC 2021b).

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 + Brickworks.

Table 5.3: CO impacts from the project

Parameter	CO (µg/m^3)
	8-hour average
Guideline (NEPM 2021a)	10,000
Maximum off-site location	
Contribution from project	12
% contribution of project to NEPM/ERS	0.1%
Cumulative case (project + background + Brickworks)	1,402
Maximum residential location	
Contribution from project	7.9
% contribution of project to NEPM/ERS	0.08%
Cumulative case (project + background + Brickworks)	1,369
Maximum commercial location	
Contribution from project	7.2
% contribution of project to NEPM/ERS	0.07%
Cumulative case (project + background + Brickworks)	1,350
Maximum other places location	
Contribution from project	1.3
% contribution of project to NEPM/ERS	0.01%
Cumulative case (project + background + Brickworks)	1,346

On this basis, the operation of the facility is not expected to significantly contribute to, or change ambient levels of CO, and there are no health risk issues of concern in relation to emissions of CO from this facility.

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 ($PM_{2.5}$).

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 $PM_{2.5}$ – 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 ($PM_{2.5}$).

Numerous epidemiological studies[5] have reported significant positive associations between particulate air pollution measured as PM_{10} 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, 2018b; 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 PM_{10} and $PM_{2.5}$ were considered in the derivation of the NEPM air guidelines for particles.

Guidelines are available from EPA Victoria (EPA Victoria 2022), ERS (Victorian Government 2021) and NEPC (NEPC 2021b) which indicate concentrations of particles considered to be acceptable by national health authorities. The EPA Victoria guidelines for particles are the same as the guidelines listed in the ambient air NEPM.

Review of the guidelines by NEPC identified additional supporting studies[6] for the evaluation of potential adverse health effects (Golder 2013; NEPC 2010). The review recommended an amendment to the guidelines for particles which comes into effect in 2025. The change is that the 24 hour average for $PM_{2.5}$ drops from 25 to 20 $\mu g/m^3$ and the annual average drops from 8 to 7 $\mu g/m^3$. The change is incorporated into the most recent version of the ambient air NEPM but the change does not take effect until 2025 (NEPC 2021b).

Tables 5.4 ($PM_{2.5}$) and **5.5 (PM_{10})** 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 + Brickworks.

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 PM_{10} separately for every hour of the year – matching the meteorological conditions for each hour. This allows an

5 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.

6 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.

evaluation of whether the proposed facility makes any difference to the total number of days per year that $PM_{2.5}$ and PM_{10} might exceed guideline values in the region.

Table 5.4: $PM_{2.5}$ impacts from the project

Parameter	$PM_{2.5}$ (µg/m³)	
	24-hour average	Annual average
Guideline (NEPM 2016)	25 (current) 20 (2025)	8 (current) 7 (2025)
Maximum off-site location		
Contribution from project	1.1	0.2
% contribution of project to NEPM/ERS	4.4% (current) 5.5% (post 2025)	2.5% (current) 2.9% (post 2025)
Cumulative case (project + background + Brickworks)	42	9.2
Maximum residential location		
Contribution from project	0.4	0.03
% contribution of project to NEPM/ERS	1.6% (current) 2% (post 2025)	0.4% (current) 0.4% (post 2025)
Cumulative case (project + background + Brickworks)	42	8.9
Maximum commercial location		
Contribution from project	0.5	0.1
% contribution of project to NEPM/ERS	2% (current) 2.5% (post 2025)	1.3% (current) 1.4% (post 2025)
Cumulative case (project + background + Brickworks)	42	8.9
Maximum other places location		
Contribution from project	0.06	0.003
% contribution of project to NEPM/ERS	0.2% (current) 0.3% (post 2025)	0.04% (current) 0.04% (post 2025)
Cumulative case (project + background + Brickworks)	42	8.8

Table 5.5: PM_{10} impacts from the project

Parameter	PM_{10} (µg/m³)	
	24-hour average	Annual average
Guideline (NEPM 2016)	50	25
Maximum off-site location		
Contribution from project	1.1	0.2
% contribution of project to NEPM/ERS	2.2%	0.8%
Cumulative case (project + background + Brickworks)	226	19.5
Maximum residential location		
Contribution from project	0.4	0.03
% contribution of project to NEPM/ERS	0.8%	0.1%
Cumulative case (project + background + Brickworks)	226	19
Maximum commercial location		
Contribution from project	0.6	0.1
% contribution of project to NEPM/ERS	1.2%	0.2%
Cumulative case (project + background + Brickworks)	226	18
Maximum other places location		
Contribution from project	0.06	0.003
% contribution of project to NEPM/ERS	0.1%	0.01%
Cumulative case (project + background + Brickworks)	226	18

These results indicate that the proposed facility will make little change to the local concentrations of particles in ambient air and only contribute a small proportion of the NEPM goal (i.e. <6% of the NEPM goal for the daily average and <3% of the NEPM goal for the annual average.

It is noted that, while the contribution from this plant is small, the background air quality is above the NEPM guideline value (i.e. cumulative case). Further assessment of the potential for health impacts from particles has, therefore, been undertaken.

To refine this assessment exposure response relationships developed by the WHO to estimate the incremental individual risk associated with the change in $PM_{2.5}$ from the facility have 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 PM_{10} is not as robust, hence the focus is on exposures to $PM_{2.5}$. Focusing on $PM_{2.5}$ is also protective for PM_{10} as the exposure-response relationships based on PM_{10} provide a similar or lower risk estimate (i.e. even low risks).

The air quality modelling for this assessment has conservatively assumed that all PM_{10} is in the $PM_{2.5}$ size fraction. This means the results used here are also conservative as PM_{10} has been assessed as if it were all $PM_{2.5}$ – the fraction having higher risks.

Using this health indicator also covers a wide range of other health effects associated with $PM_{2.5}$ – 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 considers the baseline mortality rate for Whittlesea LGA. This is the mortality rate that currently exists in these areas without this facility. The rate in Whittlesea LGA is 267.1 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 5×10^{-7} (i.e. 5 in 10 million or 0.5 in 1 million). Values for any location further from the facility than this location will have lower risks than this value.

This risk level (5×10^{-7}) is considered to be very low and is lower than the mortality risk criteria considered to indicate negligible risk outlined by NEPM and in other literature (Capon & Wright 2019; NEPC 2011b). On this basis, changes in $PM_{2.5}$ (and, therefore, PM_{10}) derived for this proposed facility are considered to have a negligible impact on the health of the community.

5.5 Other Pollutants

5.5.1 Approach

For all 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 worst case conditions) as well as chronic exposures (based on long term normal operating conditions).

The AQA prepared by Katestone included modelling emissions over a grid of 10 km x 10 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 10 x 10 km grid) as well as at the maximum

locations for residential, commercial/industrial and other relevant land uses (schools, preschools, places of worship, farms) were provided by Katestone for use in this assessment.

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, 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. This means the calculations 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 calculations undertaken for this assessment 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.

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

and has considered international guidance from the World Health Organisation and the US Environmental Protection Agency 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	✓	×	×	×	×	×	×	×	×	×
	Particles	✓	✓	✓	×	×	×	×	×	×	×
Maximum Residential	Gases	✓	×	×	×	×	×	×	×	×	×
	Particles	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Maximum Commercial/ Industrial	Gases	✓	×	×	×	×	×	×	×	×	×
	Particles	✓	✓	✓	×	×	×	×	×	×	×
Maximum Other Places	Gases	✓	×	×	×	×	×	×	×	×	×
	Particles	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
Maximum On-site	Gases	✓	×	×	×	×	×	×	×	×	×
	Particles	✓	×	×	×	×	×	×	×	×	×

Notes:
✓ Exposure pathway complete
× Incomplete exposure pathway

The specific aspects that have been assessed for the exposure scenarios outlined in **Section 4.6.5** 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, meat, milk and eggs and use of a rainwater tank.
- maximum commercial/industrial location – exposure via inhalation and direct contact with soil.
- 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, meat, milk and eggs and use of a rainwater tank.
- maximum other places location – exposure via inhalation and direct contact with soil.

- maximum other places location should these sites change land use to residential – 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 on-site location – exposure via inhalation for visitors to the education centre.

Short term exposures via inhalation only have been assessed using data from modelling of Scenario 2.

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

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		Commercial (including café/gallery staff on-site)	Education Centre Visitors
		Young children	Adults	Young children	Adults	Adult	Pre school child
BW	Body weight	15	70	15	70	70	15
		((enHealth 2012a) and ASC NEPM (NEPC 1999 amended 2013d))					
EF	Exposure frequency (days/year)	365	365	365	365	240	12
		All year				Working year	Assume a child (or a teacher) may visit the facility once per month
ED	Exposure duration (years)	6	29	6	29	30	10
		As per (enHealth 2012a) and ASC NEPM (NEPC 1999 amended 2013d)					Professional judgement
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)	Not applicable
		Ingestion rate of outdoor soil and dust (tracked or deposited indoors) as per enHealth (enHealth 2012a)					
	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	73% from aboveground crops 27% from root crops (Total fruit and vegetable intakes per day as per ASC NEPM (NEPC	85% from aboveground crops 16% from root crops (Total fruit and vegetable intakes per day as per ASC NEPM (NEPC 1999	73% from aboveground crops 27% from root crops (Total fruit and vegetable intakes per day as per ASC NEPM (NEPC 1999		

Parameter		Residential		Farm		Commercial (including café/gallery staff on-site)	Education Centre Visitors
		Young children	Adults	Young children	Adults	Adult	Pre school child
		(NEPC 1999 amended 2013d))	1999 amended 2013d))	amended 2013d))	amended 2013d))		
	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)					
	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%	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 2013d))		Standard conservative assumption for rural residential production			
	Eggs	100%	100%	100%	100%	Not applicable	
		Assume all eggs consumed are home-produced					
	Meat	NR		100%	100%	Not applicable	

Parameter		Residential		Farm		Commercial (including café/gallery staff on-site)	Education Centre Visitors
		Young children	Adults	Young children	Adults	Adult	Pre school child
				Assume all 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%	100%
		Conservative assumption – maximum possible					
SA	Surface area of body exposed to soil per day (cm^2/day)	2700	6300	2700	6300	3800	2700
		Exposed skin surface area relevant to adults/children and workplaces as per ASC NEPM (NEPC 1999 amended 2013d)					
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	0.5
		Default (conservative) value from ASC NEPM (NEPC 1999 amended 2013d)					
ABSd	Dermal absorption fraction (unitless)	Chemical specific Refer to **Tables B1 and B2**					
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

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.

5.5.3.1 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 than those indicated in **Table 5.8**.

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 4.6.5**.

Information about the relevant short term health based criteria adopted for each chemical from reputable sources is provided in **Appendix B**. Information specific to this assessment in particular includes:

- Criteria are provided in EPA Victoria (2022) as well as other sources such as USEPA, WHO (EPA Victoria 2022). There are a number of chemicals for which criteria are not provided within the EPA Victoria guidance – either for short term exposure only or for any type of exposure. These chemicals include mercury, antimony, cobalt, and vanadium.
- This assessment has been undertaken using just the EPA Victoria recommended guidelines as well as using those guidelines plus other international guidelines for which there is detailed information about the derivation and the guidelines are well targeted for the protection of human health.
- There are a number of chemicals for which short term exposure guidelines are not available from any relevant sources. This is because short term exposures to these chemicals are not particularly relevant to health impacts. These chemicals (lead, thallium and dioxins/furans) have not been included in this part of the assessment.

The ratio of the maximum predicted concentration to the acute guideline 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.

Off-site

Table 5.8 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 present in **Appendix D**. All of these will pose lower risks than those listed in **Table 5.8**.

Table 5.8: Review of acute exposures and risks (maximum off-site – 1 hour average) – Scenario 2

Pollutants	Acute air guideline (1-hour average) (mg/m^3)	Modelled Air Concentration		Calculated RQ
		($\mu g/m^3$)	(mg/m^3)	Maximum
Hydrogen chloride (HCl)	0.66[1]	15.83	0.01583285	0.02
Hydrogen fluoride (HF)	0.06[1]	1.197	0.00119699	0.02
Ammonia	0.59[1]	4.8	0.00480000	0.008
Cadmium	0.018[1]	0.00566	0.00000566	0.0003
Mercury	0.0006[2]	0.0958	0.00009580	0.2
Antimony	0.001[3]	0.01012	0.00001012	0.01
Arsenic	0.0099[1]	0.00318	0.00000318	0.0003
Chromium (VI)	0.0013[1]	0.0120	0.00001202	0.009
Cobalt	0.00069[1]	0.00359	0.00000359	0.005
Copper	0.1[2]	0.01496	0.00001496	0.0002
Manganese	0.0091[1]	0.020190	0.00002019	0.002
Nickel	0.0011[1]	0.00968	0.00000968	0.009
Vanadium	0.03[2]	0.0018	0.00000180	0.00006
Benzene[4]	0.58[1]	7.21	0.00721000	0.01
Formaldehyde[4]	0.1[5]	8.27	0.00827000	0.08
			Total RI (other pollutants)	**0.33**
			Target (acceptable RI)	**≤ 1**

References for health-based acute air guidelines (1-hour average):
1 = Guideline available from the Texas Commission on Environmental Quality (TCEQ), https://www.tceq.texas.gov/toxicology/dsd/final
2 = 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
3 = Guideline available from ATSDR
4 = Total volatile organic compounds have been modelled as 100% benzene or as 100% formaldehyde. Risks based on both of these results have been assessed. However, only the highest risk estimate between the 2 has been included in the risk index (sum).
5 = Guideline from WHO (WHO 2010a)

Based on the assessment presented in **Table 5.8**, all the individual RQs as well as the total RI are less than 1. In this case, the individual risk quotients are between 5 times and 30,000 times lower than the relevant guideline based on short term exposure in air.

On this basis, the risks to community health for short term inhalation exposures to emissions to air from the facility are low and acceptable (based on guidance from national health authorities).

5.5.3.2 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.

The assessment has also considered emissions from the Brickworks to the south to provide a cumulative assessment for the facility. There are a number of pollutants that are also emitted by the

Brickworks and so it is important to check that the combination of the 2 facilities is acceptable. The pollutants of most importance for this combination are the gases – hydrogen chloride and hydrogen fluoride. The other pollutants are either not emitted by the Brickworks at measurable levels or only make a small difference to the levels of these chemicals that might be present in air or attached to particles.

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.

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 C**.

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

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

Where:

Exposure concentration = concentration in air relevant to the exposure period – annual average (mg/m^3)
Note: this includes the contribution from the Brickworks

Health based criterion or tolerable concentration (TC) = health-based threshold protective of all health effects for the community (mg/m^3)

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.

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

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

$$"Carcinogenic\ Risk"\ ("inhalation")" = Adjusted\ exposure\ concentration\ * Inhalation\ Unit\ Risk$$

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

Sections 5.4.3.3 to **5.4.3.5** 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.

5.5.3.3 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 ground level concentration at:

- maximum off-site location
- maximum residential location (current)
- maximum location for the other places assessed (i.e. schools, preschool, places of worship) (potential future location of a residence)
- maximum commercial/industrial location (potential future location of a residence)

These modelled concentrations (facility plus Brickworks) have been assessed for the emissions assumed for normal operations – i.e. Scenario 1. The assessment is shown in **Tables 5.9/5.10**.

The maximum off-site location will be a location close to the boundary of the facility – i.e. on the road to the south or along the boundary fence. It is not likely to be a location where houses might be constructed.

Table 5.9: Calculated chronic risks for inhalation exposures – Residential

Pollutants	Calculated RQ – Maximum off-site	Calculated RQ – Maximum residential	Calculated RQ – Maximum other places	Calculated RQ – Maximum commercial /ind
Hydrogen chloride (HCl)	0.27	0.15	0.01	0.08
Hydrogen fluoride (HF)	0.01	0.008	0.0006	0.004
Ammonia	0.001	0.0002	0.00002	0.0007
Cadmium	0.04	0.008	0.0009	0.03
Thallium	0.0002	0.00003	0.000006	0.0001
Mercury	0.001	0.0002	0.00002	0.0008
Antimony	0.001	0.0002	0.00001	0.0006
Arsenic	0.002	0.0004	0.00006	0.001
Lead	0.004	0.0006	0.00008	0.002
Chromium (Cr VI assumed)	0.1	0.03	0.003	0.06
Cobalt	0.001	0.0002	0.00004	0.0006
Copper	0.000002	0.0000003	0.00000004	0.000001
Manganese	0.006	0.002	0.0002	0.003
Nickel	0.01	0.002	0.0002	0.006
Vanadium	0.0006	0.00008	0.0004	0.0003
Dioxin-like compounds	0.0002	0.00004	0.000004	0.0001
Benzene[1]	0.02	0.0009	0.0001	0.003
Formaldehyde[1]	0.02	0.003	0.0003	0.01
Total RI (other pollutants)	**0.5**	**0.2**	**0.02**	**0.2**
Negligible risk	**≤1**			

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

1 = Total volatile organic compounds have been modelled as 100% benzene or as 100% formaldehyde. Risks based on both of these results have been assessed. However, only the highest risk estimate between the 2 has been included in the risk index (sum).

At the maximum off-site location, hydrogen chloride and chromium have the highest individual risk quotients for normal operations. The maximum concentrations for these chemicals are 3-10 times lower than the guideline issued by health authorities for continuous exposure (i.e. 24 hours a day 365 days a year). The other pollutants range from 1,000 fold to 10 million fold lower than the

relevant guidelines. The sum of all risk quotients for the various chemicals indicates that the overall risk index is at least 2 times lower than the maximum acceptable value at the maximum impacted site assuming someone could live in that location 24 hours a day, 7 days a week. These results include the contribution from the Brickworks – i.e. cumulative case.

This assessment has assumed all total volatile organic carbon (TVOC) emitted by the facility is present as benzene and as formaldehyde. Both options are a conservative assessment as these emissions (measured as TVOC) will actually be a mix of chemicals. Benzene and/or formaldehyde has been used to assess this mix as they have the most sensitive guideline values.

Table 5.10: Non-threshold chronic risks for inhalation exposures – Residential

Pollutants	Calculated Risk – Maximum off-site	Calculated Risk – Maximum residential	Calculated Risk – Maximum other places	Calculated RQ – Maximum commercial/ind
Benzene	$1x10^{-6}$	$7x10^{-8}$	$8x10^{-9}$	$3x10^{-7}$
PAHs	$9x10^{-10}$	$1x10^{-10}$	$1x10^{-10}$	$5x10^{-10}$
Negligible risk	**≤$1x10^{-5}$**			

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

This non-threshold assessment has assumed all total volatile organic carbon (TVOC) emitted by the facility is 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 values so assuming all TOC is benzene results in a conservative assessment.

At the maximum off-site location, the maximum concentration of benzene anywhere in the off-site area is approximately 10 fold lower than the guideline issued by health authorities for continuous exposure (i.e. 24 hours a day 365 days a year). The risks at the other locations are even lower.

At the maximum off-site location, the maximum concentration of PAHs anywhere in the off-site area is approximately 10,000 fold lower than the guideline issued by health authorities for continuous exposure (i.e. 24 hours a day 365 days a year). The risks at the other locations are even lower.

Incremental

To show how much this facility alone contributes to these risks, especially for hydrogen chloride and chromium, the maximum off-site location has also been assessed using the facility only data. The spreadsheets showing this assessment are also included in **Appendix D** and results are shown in **Table 5.11**.

Table 5.11: Calculated chronic risks for inhalation exposures – Residential – this facility only

Pollutants	Calculated RQ – Maximum off-site – contributions from this facility only
Hydrogen chloride (HCl)	0.009
Hydrogen fluoride (HF)	0.001
Ammonia	0.001
Cadmium	0.04
Thallium	0.0002
Mercury	0.001
Antimony	0.001
Arsenic	0.002
Lead	0.004
Chromium (Cr VI assumed)	0.1
Cobalt	0.001
Copper	0.000002

Pollutants	Calculated RQ – Maximum off-site – contributions from this facility only
Manganese	0.006
Nickel	0.01
Vanadium	0.0006
Dioxin-like compounds	0.0002
Benzene	0.02
Formaldehyde	0.02
Total RI (other pollutants)	**0.2**
Negligible risk	**≤1**

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

These results show that hydrogen chloride and hydrogen fluoride are primarily discharged from the Brickworks. The risk estimated for hydrogen chloride for this facility alone is around 30 times lower than for the 2 facilities combined. This proposed facility will not change the emissions of these gases to any measurable extent. Other pollutants are similar for both the facility only and the cumulative scenario.

The non-threshold estimates of risk have not been repeated for the facility only as the assessment discussed above using the non-threshold approach is already based on the increment from the facility alone – as is normal for that type of assessment.

5.5.3.4 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 5.12/5.13**.

Table 5.12: Calculated chronic risks for inhalation exposures – commercial/industrial

Pollutants	Calculated RQ – Maximum off-site	Calculated RQ – Maximum commercial/industrial
Hydrogen chloride (HCl)	0.07	0.02
Hydrogen fluoride (HF)	0.004	0.001
Ammonia	0.0003	0.0002
Cadmium	0.01	0.02
Thallium	0.00005	0.00008
Mercury	0.0004	0.0006
Antimony	0.0003	0.0002
Arsenic	0.0006	0.0009
Lead	0.001	0.001
Chromium (Cr VI assumed)	0.04	0.02
Cobalt	0.0003	0.0002
Copper	0.0000005	0.0000003
Manganese	0.002	0.0009
Nickel	0.003	0.002
Vanadium	0.0002	0.00008
Dioxin-like compounds	0.00007	0.00004
Benzene[1]	0.01	0.0009
Formaldehyde[1]	0.02	0.003
Total RI (other pollutants)	**0.15**	**0.07**
Negligible risk	**≤1**	

Notes:
Refer to **Appendices B, C** and **D** for detail on health based criteria and risk calculations
1 = Total volatile organic compounds have been modelled as 100% benzene or as 100% formaldehyde. Risks based on both of these results have been assessed. However, only the highest risk estimate between the 2 has been included in the risk index (sum).

At the maximum off-site location, hydrogen chloride, cadmium, chromium and benzene have the highest individual risk quotients. These maximum concentrations, however, are still 15-100 times lower than the guideline issued by 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 250 to 2 million times lower than the relevant guidelines for each chemical individually.

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 10 times lower than the maximum acceptable risk index recommended by national health authorities in Australia.

This assessment has assumed all total volatile organic carbon (TVOC) emitted by the facility is present as benzene and as formaldehyde. Both options are a conservative assessment as these emissions (measured as TVOC) will actually be a mix of chemicals. Benzene and/or formaldehyde has been used to assess this mix as they have the most sensitive guideline values.

Table 5.13: Non-threshold chronic risks for inhalation exposures – commercial/industrial

Pollutants	Calculated Risk – Maximum off-site	Calculated Risk – Maximum commercial/industrial
Benzene	$8x10^{-7}$	$2x10^{-7}$
PAHs	$6x10^{-10}$	$1x10^{-10}$
Negligible risk	**≤$1x10^{-5}$**	

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 (TOC) emitted by the facility will be present as benzene. This is a conservative assessment as these emissions (measured as TOC) 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).

At the maximum off-site location, the concentration of benzene anywhere in the off-site area is approximately 12 times lower than the guideline issued by health authorities for the protection of the general public adjusted for worker exposure (i.e. 10 hours a day 240 days a year).

At the maximum off-site location, the maximum concentration of PAHs anywhere in the off-site area is approximately 15,000 fold lower than the guideline issued by health authorities for the protection of the general public adjusted for worker exposure (i.e. 10 hours a day 240 days a year). The risks at the other locations are even lower.

5.5.3.5 Chronic exposures – other locations

The receptor locations identified as “other” in this assessment are the locations for schools, childcare centres, churches, hospitals, aged care facilities and other similar sensitive locations other than homes.

In regards to assessing risks due to inhalation at these locations, it has been assumed that a person may spend 24 hours a day, 365 days per year at one of these sites – i.e. the same exposure scenario as used for assessing residential locations has been used. The results for this assessment are shown in **Section 5.5.3.3**.

No additional assessment is required in relation to long term exposure via inhalation as the worst case has been assessed above.

5.5.3.6 Chronic exposures – on-site visitors

The potential for risks to visitors to the education centre at the site has been assessed using the maximum on-site ground level concentrations and deposition rate. This scenario has been assessed based on assuming a person could visit the site for 4 hours per day on 12 days per year and that they would be in the worst case location on each occasion.

This scenario has been developed to cover the situation where a teacher may regularly bring students to the site over a year – presumably different students each time. It has been assumed the person would only breathe the air on each occasion they visit. Even though it is not expected that the same children will visit the site every time a teacher might, the scenario has been assessed based on children being exposed. This assessment is shown in **Table 5.14**.

Table 5.14: Calculated chronic risks for inhalation exposures – on-site

Pollutants	Calculated RQ – Visitor
Hydrogen chloride (HCl)	0.0003
Hydrogen fluoride (HF)	0.00005
Ammonia	0.00000005
Cadmium	0.001
Thallium	0.000007
Mercury	0.00008
Antimony	0.00001
Arsenic	0.0002
Lead	0.0001
Chromium (Cr VI assumed)	0.004
Cobalt	0.00004
Copper	0.00000007
Manganese	0.0002
Nickel	0.0004
Vanadium	0.00002
Dioxin-like compounds	0.000009
Benzene[1]	0.0002
Formaldehyde[1]	0.0001
Total RI (other pollutants)	**0.007**
Negligible risk	**≤1**

Notes:

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

1 = Total volatile organic compounds have been modelled as 100% benzene or as 100% formaldehyde. Risks based on both of these results have been assessed. However, only the highest risk estimate between the 2 has been included in the risk index (sum).

At the maximum on-site location, chromium and cadmium have the highest individual risk quotients. These maximum concentrations, however, are still 250 times lower than the guideline issued by health authorities for the protection of the general public. The other pollutants range from 2,000 to 30 million times lower than the relevant guidelines for each chemical individually.

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 140 times lower than the maximum acceptable risk index recommended by national health authorities in Australia.

This assessment has assumed all total volatile organic carbon (TVOC) emitted by the facility is present as benzene and as formaldehyde. Both options are a conservative assessment as these

emissions (measured as TVOC) will actually be a mix of chemicals. Benzene and/or formaldehyde has been used to assess this mix as they have the most sensitive guideline values.

Table 5.15: Non-threshold chronic risks for inhalation exposures – on-site

Pollutants	Calculated Risk – Visitor
Benzene	$1x10^{-8}$
PAHs	$4x10^{-12}$
Negligible risk	**$\leq 1x10^{-5}$**

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

The concentration of benzene at the maximum on-site location is approximately 1,000 fold lower than the guideline issued by health authorities relevant for people visiting the education centre (i.e. 4 hours a day 12 days a year).

At the maximum on-site location, the maximum concentration of PAHs anywhere is approximately 2.5 million fold lower than the guideline issued by health authorities relevant for people visiting the education centre (i.e. 4 hours a day 12 days a year).

5.5.3.7 Chronic exposures – summary

Risks associated with chronic exposures by inhalation are considered to be acceptable/negligible where the individual and total RI's are less than or equal to 1 for all exposure and operational scenarios.

Based on the assessment presented in these tables (and in the spreadsheet images in **Appendix D**), all the individual RQs and the total RI's for the maximum inhalation exposures that may occur in residential locations or in commercial/industrial areas or in other locations or on the site where visitors may be exposed are all less than 1.

The risks to community health for long term inhalation exposures to emissions to air from the facility are low and acceptable (based on guidance from national health authorities).

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 is also 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 are not assessed for these other pathways. Benzene is present as a gas not attached to a particle. In addition, formaldehyde, hydrogen chloride, hydrogen fluoride and ammonia are not relevant for this part of the assessment as they are always present as gases.

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.

This assessment is based on the cumulative case for the combination of this facility and the Brickworks to the south of the site.

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. As a result, only PAHs have been assessed for non-threshold risks in **Sections 5.5.3.2** to **5.5.3.6.**

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). Home grown produce is not included in this part of the assessment.

This assessment is shown in **Table 5.16** and **5.17**.

Table 5.16: Chronic risks for multiple pathway exposures – Maximum off-site

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	0.5	0.5
Soil ingestion (SI)	0.0009	0.009
Soil dermal contact (SD)	0.0003	0.0005
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.5	0.5
Negligible risk	**≤1**	**≤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 approximately 2 fold lower than health authorities indicate is acceptable/negligible.

Table 5.17: Non-threshold chronic risks for multiple pathway exposures – Maximum off-site

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	$1x10^{-6}$	$1x10^{-6}$
Soil ingestion (SI)	$9x10^{-11}$	$2x10^{-10}$
Soil dermal contact (SD)	$4x10^{-10}$	$1x10^{-10}$
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	$1x10^{-6}$	$1x10^{-6}$
Negligible risk	**≤$1x10^{-5}$**	**≤$1x10^{-5}$**

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 approximately 10 fold lower than health authorities indicate is acceptable/negligible.

5.5.4.3 Residential

The residential exposure scenario has been assessed for three locations:

- Maximum location which is currently used for residential purposes (**Table 5.18/5.19**)
- Maximum location which is currently used for commercial/industrial purposes (**Table 5.20/5.21**)
- Maximum location currently used for other purposes (e.g. schools, preschools, places of worship) (**Table 5.22/5.23**)

At each of these 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, growing and consuming home grown produce (fruit, vegetables, eggs).

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

Table 5.18: Chronic risks for multiple pathway exposures – current maximum residential

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	0.2	0.2
Soil ingestion (SI)	0.0002	0.001
Soil dermal contact (SD)	0.00003	0.00006
Ingestion of homegrown fruit and vegetables (F&V)	0.0001	0.0003
Ingestion of homegrown eggs (E)	0.00004	0.00007
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.2	0.2
I + SI + SD + F&V	0.2	0.2
I + SI + SD + E	0.2	0.2
All pathways combined	0.2	0.2
Negligible risk	**≤1**	**≤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 total risk is approximately 5 fold lower than health authorities indicates is acceptable/negligible when considering all relevant pathways.

Table 5.19: Non-threshold chronic risks for multiple pathway exposures – current maximum residential

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	$7x10^{-8}$	$7x10^{-8}$
Soil ingestion (SI)	$1x10^{-11}$	$3x10^{-11}$
Soil dermal contact (SD)	$5x10^{-11}$	$2x10^{-11}$
Ingestion of homegrown fruit and vegetables (F&V)	$1x10^{-10}$	$8x10^{-11}$
Ingestion of homegrown eggs (E)	$2x10^{-14}$	$1x10^{-14}$
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	$7x10^{-8}$	$7x10^{-8}$
I + SI + SD + F&V	$7x10^{-8}$	$7x10^{-8}$
I + SI + SD + E	$7x10^{-8}$	$7x10^{-8}$
All pathways combined	$7x10^{-8}$	$7x10^{-8}$
Negligible risk	**$\leq 1x10^{-5}$**	**$\leq 1x10^{-5}$**

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 for all relevant exposure pathways is approximately 1,000 fold lower than health authorities indicate is acceptable/negligible when considering all relevant pathways.

Table 5.20: Chronic 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)	0.2	0.2
Soil ingestion (SI)	0.0005	0.005
Soil dermal contact (SD)	0.0001	0.0003
Ingestion of homegrown fruit and vegetables (F&V)	0.0004	0.001
Ingestion of homegrown eggs (E)	0.0002	0.0003
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.2	0.2
I + SI + SD + F&V	0.2	0.2
I + SI + SD + E	0.2	0.2
All pathways combined	0.2	0.2
Negligible risk	**≤1**	**≤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 site should change to residential land use, the total risk is approximately 5 fold lower than health authorities indicate is acceptable /negligible when considering all relevant pathways.

Table 5.21: Non-threshold chronic 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)	$3x10^{-7}$	$3x10^{-7}$
Soil ingestion (SI)	$5x10^{-11}$	$1x10^{-10}$
Soil dermal contact (SD)	$2x10^{-10}$	$8x10^{-11}$
Ingestion of homegrown fruit and vegetables (F&V)	$4x10^{-10}$	$3x10^{-10}$
Ingestion of homegrown eggs (E)	$1x10^{-13}$	$4x10^{-14}$
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	$3x10^{-7}$	$3x10^{-7}$
I + SI + SD + F&V	$3x10^{-7}$	$3x10^{-7}$
I + SI + SD + E	$3x10^{-7}$	$3x10^{-7}$
All pathways combined	$3x10^{-7}$	$3x10^{-7}$
Negligible risk	**$\leq 1x10^{-5}$**	**$\leq 1x10^{-5}$**

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 site should change to residential land use, the total risk for all relevant exposure pathways is approximately 30 fold lower than health authorities indicate is acceptable/negligible when considering all relevant pathways.

Table 5.22: Chronic risks for multiple pathway exposures – current maximum for other places location assuming residential could occur at that location

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	0.02	0.02
Soil ingestion (SI)	0.00001	0.0002
Soil dermal contact (SD)	0.000005	0.00001
Ingestion of homegrown fruit and vegetables (F&V)	0.00001	0.00003
Ingestion of homegrown eggs (E)	0.000005	0.00001
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**	**≤1**

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

At the current maximum "other" location if this site should change to residential land use, the total risk is approximately 30 fold lower than health authorities indicate is acceptable/negligible when considering all relevant pathways.

Table 5.23: Non-threshold chronic risks for multiple pathway exposures– current maximum for other places location assuming residential could occur at that location

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	$1x10^{-8}$	$1x10^{-8}$
Soil ingestion (SI)	$1x10^{-11}$	$3x10^{-11}$
Soil dermal contact (SD)	$5x10^{-11}$	$2x10^{-11}$
Ingestion of homegrown fruit and vegetables (F&V)	$1x10^{-10}$	$8x10^{-11}$
Ingestion of homegrown eggs (E)	$3x10^{-14}$	$1x10^{-14}$
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	$1x10^{-8}$	$1x10^{-8}$
I + SI + SD + F&V	$1x10^{-8}$	$1x10^{-8}$
I + SI + SD + E	$1x10^{-8}$	$1x10^{-8}$
All pathways combined	$1x10^{-8}$	$1x10^{-8}$
Negligible risk	**$\leq 1x10^{-5}$**	**$\leq 1x10^{-5}$**

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

At the current maximum "other" location if this site should change to residential land use, the total risk for all relevant exposure pathways is approximately 1,000 fold lower than health authorities indicate is acceptable/negligible when considering all relevant pathways.

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 and having direct contact with soil 240 days per year for up to 30 years as an adult. 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.

This assessment is shown in **Table 5.24** and **5.25**.

Table 5.24: Chronic risks for multiple pathway exposures – maximum commercial

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	0.07	0.07
Soil ingestion (SI)	0.0005	0.005
Soil dermal contact (SD)	0.0001	0.0003
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.07	0.08
Negligible risk	**≤1**	**≤1**

* 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 12 fold lower than health authorities indicate is acceptable/negligible when considering all relevant pathways.

Table 5.25: Non-threshold chronic risks for multiple pathway exposures– maximum commercial

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	$6x10^{-8}$	$6x10^{-8}$
Soil ingestion (SI)	$5x10^{-11}$	$1x10^{-10}$
Soil dermal contact (SD)	$2x10^{-10}$	$8x10^{-11}$
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	$6x10^{-8}$	$6x10^{-8}$
Negligible risk	**$\leq 1x10^{-5}$**	**$\leq 1x10^{-5}$**

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 all relevant exposure pathways is approximately 150 fold lower than health authorities indicate is acceptable/negligible when considering all relevant pathways.

5.5.4.5 Farming

Given that locations around the proposed facility are currently used for grazing, an exposure scenario for a farm has been assessed for the maximum residential location.

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.

This assessment is shown in **Table 5.26** and **5.27**.

Table 5.26: Chronic risks for multiple pathway exposures – maximum residential location assuming farming occurs

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	0.2	0.2
Soil ingestion (SI)	0.0002	0.001
Soil dermal contact (SD)	0.00003	0.00006
Ingestion of homegrown fruit and vegetables (F&V)	0.0001	0.0003
Ingestion of homegrown eggs (E)	0.00004	0.00007
Ingestion of homegrown meat (B)	0.0001	0.0003
Ingestion of homegrown milk (M)	0.002	0.008
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.2	0.2
I + SI + SD + F&V	0.2	0.2
I + SI + SD + E	0.2	0.2
I + SI + SD + F&V + E	0.2	0.2
I + SI + SD + B	0.2	0.2
I + SI + SD + M	0.2	0.2
All pathways combined	0.2	0.2
Negligible risk	**≤1**	**≤1**

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

At the maximum farm location in the area surrounding the proposed facility, the total risk is at least 5 fold lower than the guidance issued by health authorities indicates is acceptable/negligible when considering all relevant pathways.

Table 5.27: Non-threshold chronic risks for multiple pathway exposures– maximum residential location assuming farming occurs

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Inhalation (I)	$7x10^{-8}$	$7x10^{-8}$
Soil ingestion (SI)	$1x10^{-11}$	$3x10^{-11}$
Soil dermal contact (SD)	$5x10^{-11}$	$2x10^{-11}$
Ingestion of homegrown fruit and vegetables (F&V)	$1x10^{-10}$	$8x10^{-11}$
Ingestion of homegrown eggs (E)	$2x10^{-14}$	$1x10^{-14}$
Ingestion of homegrown meat (B)	$8x10^{-11}$	$4x10^{-11}$
Ingestion of homegrown milk (M)	$1x10^{-9}$	$1x10^{-9}$
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	$7x10^{-8}$	$7x10^{-8}$
I + SI + SD + F&V	$7x10^{-8}$	$7x10^{-8}$
I + SI + SD + E	$7x10^{-8}$	$7x10^{-8}$
I + SI + SD + F&V + E	$7x10^{-8}$	$7x10^{-8}$
I + SI + SD + B	$7x10^{-8}$	$7x10^{-8}$
I + SI + SD + M	$7x10^{-8}$	$7x10^{-8}$
All pathways combined	$7x10^{-8}$	$7x10^{-8}$
Negligible risk	**$\leq 1x10^{-5}$**	**$\leq 1x10^{-5}$**

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

At the maximum farm location in the area surrounding the proposed facility, the total risk is at least 140 fold lower than the guidance issued by health authorities indicates is acceptable/negligible when considering all relevant pathways.

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.

The Department of Health has issued guidance about the use of rainwater tanks in urban areas for drinking (https://www.health.vic.gov.au/water/rainwater). They note that 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.

Noting this advice, the estimated concentrations in a rainwater tank have been assessed 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 2022). This assessment has been undertaken for both operational scenarios.

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 5.28/5.29**)
- Maximum location which is currently used for commercial/industrial purposes (**Table 5.30/5.31**)

Table 5.28: Summary of risks for rainwater tanks – maximum residential location

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Water ingestion	0.00004	0.00004
Water dermal contact	0.0000005	0.000001
Total	0.00004	0.00004
Negligible risk	**≤1**	**≤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 risk related to using water from a rainwater tank is at least 25,000 fold lower than health authorities indicate as acceptable/negligible.

Table 5.29: Non-threshold chronic risks for rainwater tanks – maximum residential location

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Water ingestion	$5x10^{-12}$	$9x10^{-13}$
Water dermal contact	$2x10^{-11}$	$1x10^{-11}$
Total	$3x10^{-11}$	$1x10^{-11}$
Negligible risk	**$\leq 1x10^{-5}$**	**$\leq 1x10^{-5}$**

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 at least 300,000 fold lower than health authorities indicate as acceptable/ negligible.

Table 5.30: Summary of risks for rainwater tanks – maximum commercial location

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Water ingestion	0.0002	0.0002
Water dermal contact	0.000002	0.000005
Total	0.0002	0.0002
Negligible risk	**≤1**	**≤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 risk related to using water from a rainwater tank is at least 5,000 fold lower than health authorities indicate as acceptable/negligible.

Table 5.31: Non-threshold chronic risks for rainwater tanks – maximum commercial location

Exposure pathway	Calculated HI	
	Adults	Children
Individual exposure pathways		
Water ingestion	$2x10^{-11}$	$3x10^{-12}$
Water dermal contact	$8x10^{-11}$	$4x10^{-11}$
Total	$1x10^{-10}$	$4x10^{-11}$
Negligible risk	**$\leq 1x10^{-5}$**	**$\leq 1x10^{-5}$**

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 at least 100,000 fold lower than health authorities indicate as acceptable/ negligible.

5.6 Summary

This assessment has shown:

- No unacceptable risks for criteria pollutants (NO_x, SO_x, CO, $PM_{2.5}$, 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) (including farms)
- Maximum commercial/industrial location
- Maximum other places location
- Maximum on-site location

- No unacceptable risks for relevant exposure scenarios for rainwater tanks.

5.7 Additional Considerations – PFAS

Another 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 2020).

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
- Cosmetics
- Sunscreens
- Cleaning products
- Paper products
- Surfactants used in chrome plating or electronics manufacture (HEPA 2020)

It is possible 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 2020). PFAS have been reported to be present in leachate from some landfills in Australia (Gallen et al. 2017).

Concerns regarding this group of chemicals were raised internationally around 2000. 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. As a result, 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.

Methods for the analysis of these chemicals in air are not routinely available (HEPA 2020). There is no requirement for analysis of these chemicals in emissions from similar plants in Europe due to the difficulty in undertaking such analysis and the expected low levels. As a result, there are no monitoring data available, and it is not currently possible to undertake a detailed quantitative assessment.

It is noted, however, that this facility has the capacity to manage small amounts of such chemicals appropriately if they were to be present in residual waste. The flue gas treatment technology proposed for this facility can address the presence of these chemicals using the following:

- Combustion chamber – PFAS are usually 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^{o}C and these temperatures along with sufficient oxygen will allow for effective combustion of these chemicals.
- Acid gas treatment (injection of lime) – the flue gas treatment 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 – chemicals attached to particles (including activated carbon particles) are captured within the baghouse. This will include PFAS.

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 expected to be low to negligible.

5.8 Uncertainties

The characterisation of potential health risks related to exposures to emissions to air from the proposed WtE 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 (2023) 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 Victorian 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.

For the purposes of this assessment, 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 2 fold less than the maximum acceptable value when considering the emissions from both this facility and the Brickworks to the south of the site and 5 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 and other places locations assuming people could one day live at these locations. This covers the situation if land use changes at a site closer to the facility at some time in the future.

Industrial workers:

For workers at commercial/industrial sites around the proposed facility, it has been assumed that they are present for 10 hours a day for 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 conservatively assumed 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 still be some deposition during rain events.

The modelling for this assessment has assumed all the particles are in the PM_{10} fraction and that they do deposit to the ground under wet and dry conditions. This results in a conservative assessment of deposition and exposure via deposition related pathways. It is also noted that if most of the particles are in the PM_{10} fraction, then the risk due to inhalation of such particles has been overestimated, given the assumption for that pathway that the particles are primarily $PM_{2.5}$.

The assessment has also assumed that the particles have deposited for 70 years prior to determining the concentration of a chemical that might be present and available for uptake into produce.

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.

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 (2023a), Melbourne Energy and Resource Centre, Noise & Vibration Technical Report. DRAFT Report dated 8 February 2023.

This aspect (noise) has been assessed in line with Victorian 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 (Victorian Government 2006).

6.2 Health impacts associated with noise

Environmental noise has been identified (I-INCE 2011; WHO 2011a) 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 1999b, 2011a):

- 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**.

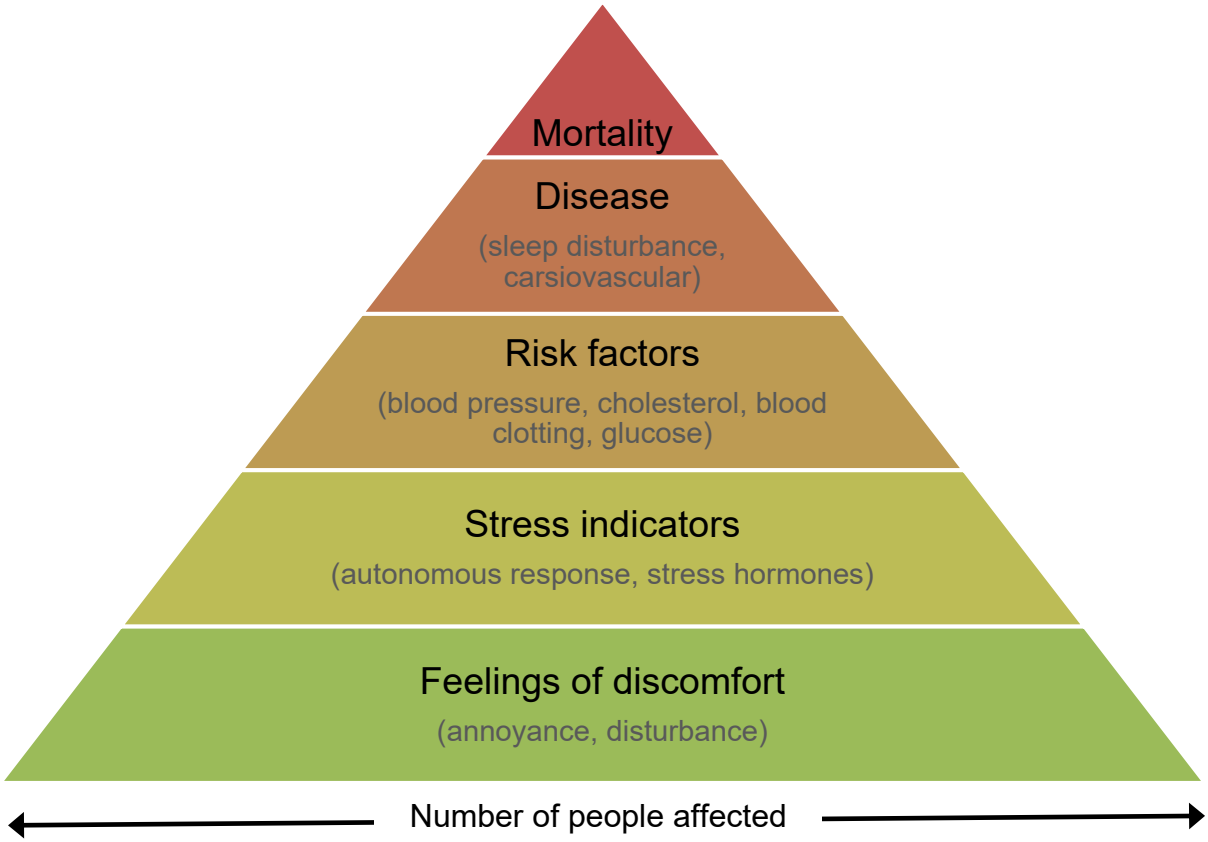


Figure 6.1: Schematic of severity of health effects of exposure to noise and the number of people affected (WHO 2011a)

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 2011a, 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 2011a, 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 2011a, 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) provided 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 1999b) and night noise guidelines (WHO 2009).

6.3 Summary of noise assessment

6.3.1 Background noise levels

The NV assessment (Arup 2023a) has evaluated background noise levels at 4 attended monitoring locations around the site (at rural residential locations in the main) and 2 unattended monitoring locations within the boundary of the site. These are shown on **Figure 6.2**. The nearest residential locations are listed in **Table 6.1**.

Table 6.1: Nearest noise sensitive receivers (Arup 2022a)

ID	Noise sensitive receiver address	Distance to subject site boundary (m)
R1	620 Summerhill Road	400
R2	585 Summerhill Road	430
R3	570 Summerhill Road	350
R4	475 Summerhill Road	110
R5	430 Summerhill Road	350

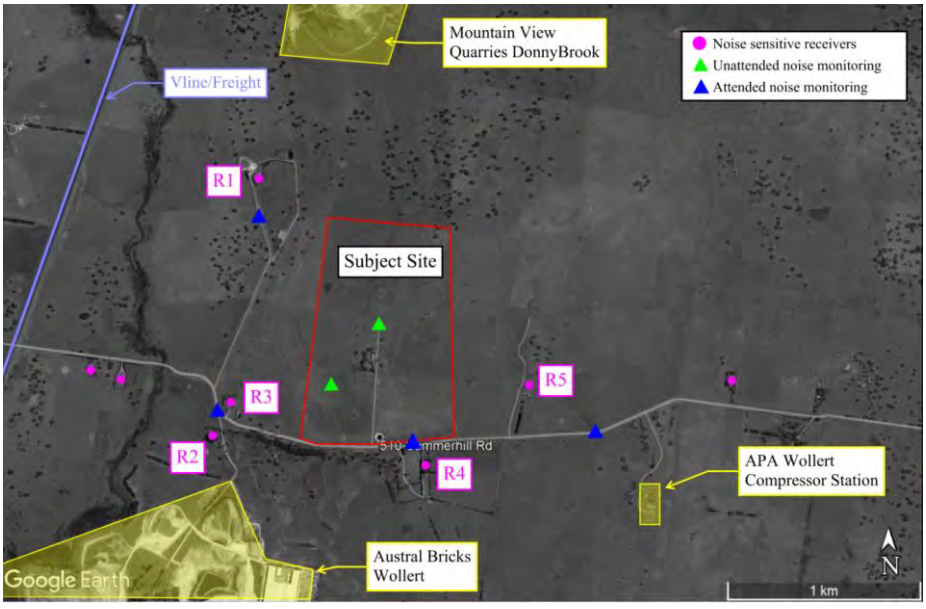


Figure 6.2: Receiver and monitoring locations (Arup 2023a)

Attended noise monitoring was undertaken at night. Background noise levels ranged from 38 to 42 dB $L_{A90\ (10\ min)}$. These measurements were taken between 2 and 4 am on 11 October 2022 (ARUP 2023a).

Unattended monitoring was undertaken at the site in early September 2022. The Leq values ranged from 35 to 65 dB(A) (for the data indicated as valid) (Arup 2023a).

The background noise levels determined for the area are shown in **Table 6.2**.

Table 6.2: Background (existing) noise levels (Arup 2023a)

Time of day	Background noise level (dBL_{A90})
Normal working hours (day)	37
Outside normal working hours (evening)	37
Night R1-3	38
Night R4	37
Night R5	41

These results indicate that the assessment has assumed existing noise levels on and around the site are essentially the same all day and all night which is unusual but, given the limited activities currently undertaken in the area, makes sense.

These levels are also quite low indicating the area is quiet. It is not clear how the background noise levels were determined for normal working hours, given that the unattended monitoring indicated noise levels of up to 65 dB(A) were measured at times.

The attended monitoring provided some additional information about the types of noise that were occurring during the monitoring periods. The noise sources identified by the people undertaking the monitoring were:

- M31 traffic hum
- Wildlife – insects, birds, crickets, frogs and cicadas
- Truck or plane movements nearby
- Steady state factory noise (at R5 only – potentially due to the compressor station used for a high pressure gas line located 1.7 km to the south east of the site) (Arup 2023a).

Noise criteria adopted for assessing noise impacts in Arup (2023a) utilise these background noise levels.

6.3.2 Construction noise guidelines

Management noise limits were determined to manage impacts from noise during construction using the NSW EPA Draft Construction Noise Guidelines (NSW DECC 2009).

This guideline provides noise criteria (as $L_{Aeq,15\text{-}min}$) for works during standard construction hours, and for works outside standard hours. The noise criteria are the background noise levels plus 10 dB(A) (standard hours) or +5 dB(A) (outside standard hours), with a criteria for determining highly noise affected premises of 75 dB(A).

Using the background noise levels and this guidance gives the limits listed in **Table 6.3**. These are the limits to be applied during construction for this facility. Management actions will be put in place, where required, to ensure these limits are met at the relevant receptor locations.

Table 6.3: Construction noise limits (Arup 2023a)

Time of day	Management level ($dBL_{Aeq, 15\ min}$)
Normal working hours (day)	47
	75 (highly noise affected)
Outside normal working hours (evening)	42
	65 (highly noise affected)
Night (R1-5)	30 (i.e. inaudible)

The Civil Construction, Building and Demolition guide (EPA Victoria 2020b), provides general information on the assessment of noise from construction activities. This includes measures to reduce noise and vibration impacts and general narrative requirements about noise levels.

The NSW EPA guidance has been adopted as this guidance provides more details on setting noise levels during construction than Victorian guidance. The narrative requirements in the EPA Victoria guidance document are supported by the limits identified in **Table 6.3** based on NSW EPA guidance.

6.3.3 Operational noise guidelines

Potential noise from the proposed facility has been assessed on the basis of the EPA Victoria guidance on noise limits for commercial, industrial and trade premises (EPA Victoria 2021). This guidance provides noise limits for sensitive receptors, relevant to various land use zonings, where a zoning level is determined. An influencing factor is determined for each zone, with residential areas having an influencing factor of 0, and commercial, business and light industry, general industry and roads requiring an impact factor to be calculated. The influencing factor allows for the development of noise criteria (in conjunction with the background noise levels) for each relevant area, for day, evening and night periods.

The Environmental Reference Standard (ERS) is a new tool under the EP Act that incorporates environmental values and objectives into the assessment of noise. This includes protecting environments that support night time sleep, recreational and domestic activities, normal conversations indoors without the need for raised voices, supports cognitive learning in children and enjoyment of natural landscapes. Objectives are set under land use categories, where the guidelines for low density populations includes farming areas.

Based on the above, the following noise guidelines have been adopted in the noise and vibration assessment (Arup 2023a) for the sensitive receptors/residential properties located closest to the facility:

- Day: Noise limits for normal operations: 48 to 57 dB(A) as $L_{eq,30min}$
- Evening: Noise limits for normal operations: 44 to 52 dBA as $L_{eq,30min}$
- Day and evening ERS objective: 40 dBA as $L_{Aeq,16\ hr}$ (6am to 10pm)
- Night: Noise limits for normal operations: 41 to 52 dBA as $L_{eq,30min}$
- Night: ERS objective: 35 dBA as $L_{Aeq,8\ hr}$ (10pm to 6am).

The ERS objectives are the average noise over either 8 hours or 16 hours whereas the site specific limits to be adopted at the facility are based on 30 minute averages which is why these limits are higher than the ERS objectives.

6.4 Review and assessment of health impacts from noise

6.4.1 Review of proposed noise limits

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 somnifacient drugs and sedatives.

The night-time noise guidelines adopted for the project are set at 41-52 dB(A), as $LA_{eq,30\text{-}min}$, outside a building. However, the predicted noise levels for the night period considering the relevant mitigation measures to be included in the facility range from 36 to 45 dB(A), as $LA_{eq,30\text{-}min}$.

For converting a noise level as $L_{Aeq,period}$ to a short-term level such as $L_{Aeq,15\text{-}min\ or\ 30\text{-}min}$, guidance provides for adding 3 dB to the $L_{Aeq,period}$ value. Hence where a day time noise criteria of 45 to 50 dBA as $L_{Aeq,day}$ is adopted this could be converted to noise levels around 48 to 53 dBA as $L_{Aeq,\ 30\text{-}min}$ which would be considered protective of health.

Using the predicted levels at night and this conversion from short term to night period levels, the expected noise at night is likely to be in the range 33 to 42 dB $L_{night,\ outside}$. These values are protective of health effects.

During the day, noise guidelines that are protective of moderate levels of annoyance in outdoor living areas are 50 dBA as LA_{eq} (outdoors) and 35 dBA indoors to protect conversations and learning for children (for day or evening periods) (WHO 1999b). The conversion of noise levels outdoors (as modelled) to indoors by reducing the level by 10 dBA is considered conservative. Therefore, adopting an outdoor noise guideline of 45 dBA as $LA_{eq(day\ and/or\ evening)}$ would be protective of these values.

The noise limits adopted for the day and evening period range from 44 to 57 dBA as $LA_{eq,\ 30\text{-}min}$. However, the predicted noise levels for the day/evening period considering the relevant mitigation measures to be included in the facility range from 37 to 53 dB(A), as $LA_{eq,30\text{-}min}$.

Using the predicted levels during the day and evening and this conversion from short term to day/evening period levels, the expected noise during the day/evening is likely to be in the range 34 to 50 dB $L_{day,evening,\ outside}$. These values are protective of health effects.

It will be important that the noise mitigation measures included in the noise modelling (and any other aspects of the modelling) are incorporated into this facility and its operations.

6.5 Outcomes

With consideration of the noise limits adopted and where the project is designed to meet the noise specifications identified (i.e. predicted levels), and the identified noise mitigation measures are implemented, the risks to community health in relation to noise from the proposed facility are expected to be low and acceptable, based on this information and guidance from Australian health authorities.

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:

- Arup (2023b). Hydrology and flood risk technical report, Melbourne Energy and Resource Centre (MERC). Dated 10 February 2023.
- Douglas Partners (2022). Report on Soil Contamination and Baseline Groundwater Investigation, Melbourne Energy and Resource Centre, 510 Summerhill Road, Wollert. Dated January 2023.
- Arup (2023c). Hazardous substances and industrial hazards technical report, Melbourne Energy and Resource Centre (MERC). Dated 3 February 2023.
- Arup (2023d). Surface water, Chapter 15, Development License Application. Dated 17 March 2023.

These aspects have been assessed in line with Victorian 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 (Victorian Government 2006).

7.2 Overview and assessment of issues

7.2.1 Water management

Operation of the Proposal does not generate a wastewater stream requiring discharge.

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 2023b).

Potable water 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. An on-site wastewater management system will be installed to manage wastewater from the various buildings on-site (i.e. from kitchens and bathrooms) (Arup 2023d).

Some potable water and/or captured rainwater will be used for process water after being treated on-site through a water treatment plant. The treatment plant technologies are likely to be reverse osmosis or electro deionisation. This water will be used for filling the boilers and other processes. The waste-to-energy facility has been designed to reuse all process water during normal operation, so there is not expected to be discharge of wastewater during normal operations (Arup 2023d).

Other issues regarding water management at the site are the management of stormwater and the potential for flooding.

The Proposal area is open farmland at this time. As such, there is no formal stormwater management infrastructure present currently. Also, given Summerhill Road is gravel, there is no formal stormwater management infrastructure located within the roadway. Stormwater under the existing situation runs off the site based on the topography/slope and reaches Curly Sedge Creek or Merri Creek depending on the drainage lines (Arup 2023b).

Modelling 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 (Arup 2023b).

Management of stormwater has been based on relevant guidance from state and local government as well as Melbourne Water. Currently the site is not covered by impervious surfaces (i.e. hard stand) to any significant extent (Arup 2023b).

Managing stormwater appropriately 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 2023b).

Most the parts of the facility that could impact on the quality of stormwater will be contained within structures (i.e. inside buildings). The stormwater system will be designed to minimise potential impacts on the quality of stormwater and will include on-site detention in line with government requirements. Such detention facilities also allow for some level of treatment should this be required (Arup 2023b).

Most of the site will remain undeveloped, so stormwater quality from these areas will not change. Runoff from sensitive areas (i.e. areas where quality could be impacted) will be bunded to prevent runoff of potentially impacted water. In particular, the IBA treatment area will have a dedicated stormwater system with a retention basin and treatment as required (Arup 2023b).

Managing potential for flooding requires an understanding of the potential for flooding of the site currently, and then designing the facility to ensure relevant parts of the facility are high enough above predicted flood levels to not be impacted. All industrial facilities must be designed to ensure they are appropriately flood proofed based on government guidance. The same applies for this site. The flood modelling has indicated that significant changes in flood levels during peak events are not expected, given the use of detention basins to manage runoff from the site (Arup 2023b).

Neither of these aspects (flooding or stormwater management) have significant potential to impact on human health or to change any existing impacts on human health.

Water on the site is expected to be managed through soil and water management plans (during construction and during operation). A range of management measures are proposed including:

- reuse stormwater in detention basins for dust suppression.
- monitor water quality in detention basins to indicate when/if treatment is needed.
- locate facilities and access tracks away from areas that could be impacted during floods.
- ensure design of earthworks does not impact on Curly Sedge Creek.
- use water sensitive urban design elements where appropriate (including rainwater harvesting and reuse, gross pollutant traps)
- buildings constructed with a minimum floor level of 210 m AHD (Arup 2023b).

As a result, risks to community health in relation to the management of water on the site are expected to be low and acceptable based on this information and guidance from Australian health authorities.

7.2.2 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 CEMP will be developed prior to the commencement of works in line with government guidance. This plan will include procedures to ensure all vehicles transporting such materials to the site are in compliance with all legislative requirements and that bunded storage is provided where required and that separation distances between different categories of dangerous goods are maintained as required in legislation (Arup 2023c).

A detailed review has been undertaken of potential hazards resulting from dangerous goods transport and storage as well as the activities undertaken at the facility. The review identified that up to 3 types of materials that require specific storage and transport may be used at the site at levels triggering these specific requirements. These include:

- 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)
- Air pollution control residue
- Diesel (Arup 2023c).

Issues that may arise during transport and storage of these items are well understood and requirements of the Dangerous Goods Code provide appropriate management. Victorian legislation requires that written advice be requested from Fire Rescue Victoria to ensure all procedures and equipment are appropriate and in line with legislative requirements (and that they are modified to be so, if the advice indicates that modifications are required). An Emergency Management Plan (EMP)/ Incident/Emergency Response Plan (ERP) is also required for the site to ensure everyone knows what to do should an issue arise on-site in relation to these materials (Arup 2023c).

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

- Issues around managing the waste used as fuel for the facility (potential for fires or receipt of inappropriate materials)
- Issues related to spills/leaks or other types of incidents for dangerous goods stored at the site (Arup 2023c).

Management of these matters will be included in the design of the facility (e.g. nitrogen blanket for activated carbon storage or designing the furnace to ensure it is not damaged should inappropriate materials manage to reach the furnace (i.e. if inappropriate waste types are received) or in the sensors included in the facility (e.g. infrared sensors to be placed in the waste bunker and monitored by plant operators to ensure overheating of materials is managed prior to a fire occurring) (Arup 2023c).

Emergency response measures, including fire systems, infrared sensors, appropriate facility design and an Incident/Emergency Response Plan (ERP) are included in this facility and would manage potential hazards in line with government requirements.

As a result, risks to community health in relation to the management of hazardous substances on the site are expected to be low and acceptable based on this information and guidance from Australian health authorities.

7.2.3 Contamination

Soil conditions at the site typically comprise a 0.2 m to 2.9 m thick layer (average thickness of 1.0 m) of very stiff and hard, high plasticity, silty clay overlying basalt rock. In the south east corner of the site, imported fill is present. Also, fill is located in the vicinity of the existing buildings which appears to be reworked soil from other parts of the site placed during construction (Douglas Partners 2023).

Assessment of the potential for soil contamination has found that all contaminants assessed were not present at the site or were present at levels below relevant national guidelines for soil that are protective for human health (Douglas Partners 2023).

Based on the above, the Proposal 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.

As a result, risks to community health in relation to the management of soil on the site are expected to be low and acceptable based on this information and guidance from Australian health authorities.

7.2.4 Groundwater

The Proposal area is situated within the New Volcanics Basalt geology. Groundwater is found at 0.3 to 4 m below ground level in this aquifer (Douglas Partners 2023).

Water quality within the existing groundwater has been assessed. Only copper, zinc and total nitrogen were reported at concentrations above guidelines including ones protective of human health such as drinking water guidelines (Douglas Partners 2023).

Copper and zinc are metals that are commonly found at levels slightly above relevant guideline concentrations based on the protection of ecosystems. Ecosystem protection guidelines are significantly lower than drinking water guidelines (human health protective) for these chemicals. These metals can be present in groundwater due to natural sources (i.e. weathering of basalt) or due to human sources such as road runoff (Douglas Partners 2023).

Total nitrogen is an indicator of the nutrients present in groundwater which reach groundwater due to the use of fertiliser or presence of manure due to grazing activities (Douglas Partners 2023).

The construction and operation of the Proposal is not expected to impact on or change groundwater conditions on or off the site (Douglas Partners 2023).

Based on the above, the Proposal is not expected to have any impact on existing groundwater quality and hence no changes to existing conditions relevant to the access and use of groundwater in the community or potential for impacts on human health in the off-site community are expected.

7.3 Outcomes

Based on the evaluations provided, in relation to water, soil contamination, groundwater or dangerous goods/chemical hazards are expected to be low and acceptable for this proposed facility based on this information and guidance from Australian health authorities.

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 Wollert, Victoria.

Based on the assessment undertaken in relation to the Proposal 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 from the project, where the project is designed to meet the specifications identified, there are no health issues of concern in relation to air quality changes in relation to the Proposal. In particular, this assessment has shown:

  - No unacceptable risks for criteria pollutants (NO_x, SO_x, CO, $PM_{2.5}$, PM_{10})
  - No unacceptable risks for short term exposures (via inhalation) 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 other places location (including farms)
    - Maximum on-site location
  - No unacceptable risks for relevant exposure scenarios for rainwater tanks.

It is noted that:

- risks via exposure to chemicals attached to particles which may deposit onto the soil around the facility have been assessed after 70 years of deposition to the soil (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.

- Noise

With consideration of the noise limits adopted, the assessment of noise impacts from the project, and where the project is designed to meet the noise specifications identified (i.e. predicted levels), and the identified noise mitigation measures are implemented, there are no issues of concern for the health of the off-site community in relation to noise in relation to the Project.

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

Based on the evaluations provided, there are no issues of concern for the health of the off-site community in relation to water, soil contamination, groundwater or dangerous goods/chemical hazards in relation to the Proposal.

Section 9. References

Site Specific References

Project Description Information from Arup

Project Description Information from Arup (Arup 2023)
Katestone (2023), Melbourne Energy and Resource Centre, Air quality assessment. Report dated 23 February 2023.

Air Quality Modelling Output Spreadsheets provided by Katestone (received 17/3/23)
Arup (2023a), Melbourne Energy and Resource Centre, Noise & Vibration Technical Report. DRAFT Report dated 8 February 2023.

Arup (2023b). Hydrology and flood risk technical report, Melbourne Energy and Resource Centre (MERC). Dated 10 February 2023.

Douglas Partners (2022). Report on Soil Contamination and Baseline Groundwater Investigation, Melbourne Energy and Resource Centre, 510 Summerhill Road, Wollert. Dated January 2023.

Arup (2023c). Hazardous substances and industrial hazards technical report, Melbourne Energy and Resource Centre (MERC). Dated 3 February 2023.

Arup (2023d). Surface water, Chapter 15, Development License Application. Dated 17 March 2023.

Other References

APVMA 2005, *The Reconsideration of Registrations of Arsenic Timber Treatment Products (CCA and Arsenic Trioxide) and their Associated Labels, Report of Review Findings and Regulatory Outcomes, Summary Report, Review Series 3*, Australian Pesticides &Veterinary Medicines Authority, Canberra, Australia.

Armstrong, R, Anderson, L, Synnot, A, Burford, B, Waters, E, BaoLe, L, Weightman, A, Morgan, H, Turley, R & Steele, E 2014, *Evaluation of evidence related to exposure to lead*, National Health and Medical Research Council, Canberra.

ATSDR 1998, *Toxicological Profile for Sulfur Trioxide and Sulfuric Acid*, Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services.

ATSDR 1999, *Toxicological Profile for Mercury*, Agency for Toxic Substances and Disease Registry. <http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=115&tid=24>.

ATSDR 2004a, *Interaction Profile for Benzene, Toluene, Ethylbenzene and Xylenes (BTEX)*, US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. <http://www.atsdr.cdc.gov/interactionprofiles/ip05.html >.

ATSDR 2004b, *Toxicological Profile for Ammonia*, Agency for Toxic Substances and Disease Registry.

ATSDR 2005a, *Toxicological Profile for Nickel*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Atlanta, Georgia, USA. <http://www.atsdr.cdc.gov/ToxProfiles/tp15.pdf>.

ATSDR 2005b, *Toxicological Profile for Tin and Tin Compounds*, U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

ATSDR 2007a, *Toxicological Profile for Benzene*, US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. <http://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=40&tid=14>.

ATSDR 2007b, *Toxicological Profile for Arsenic*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Atlanta, Georgia, USA. <http://www.atsdr.cdc.gov/ToxProfiles/tp132.pdf>.

ATSDR 2010, *Toxicological Profile for Chlorine*, Agency for Toxic Substances and Disease Registry. <https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=1079&tid=36>.

ATSDR 2012a, *Toxicological Profile for Chromium* Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Atlanta, Georgia, USA. <http://www.atsdr.cdc.gov/ToxProfiles/tp7.pdf>.

ATSDR 2012b, *Toxicological Profile for Vanadium*, Agency for Toxic Substances and Disease Registry.

ATSDR 2012c, *Toxicological Profile for Manganese*, US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry. <https://www.atsdr.cdc.gov/ToxProfiles/tp151.pdf>.

ATSDR 2012d, *Toxicological Profile for Chromium*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Atlanta, Georgia, USA. <http://www.atsdr.cdc.gov/ToxProfiles/tp7.pdf>.

ATSDR 2012e, *Toxicological Profile for Cadmium*, U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry. <https://www.atsdr.cdc.gov/ToxProfiles/tp5.pdf>.

ATSDR 2019a, *Toxicological Profile for Lead - Draft*, Agency for Toxic Substances and Disease Registry, the Public Health Service, or the U.S. Department of Health and Human Services.

ATSDR 2019b, *Toxicological Profile for Antimony and Compounds*, Agency for Toxic Substances and Disease Registry, U.S. Department of Health and Human Services. <https://www.atsdr.cdc.gov/ToxProfiles/tp23.pdf>.

ATSDR 2022, *Toxicological Profile for Copper, Draft for Public Comment*, Agency for Toxic Substances and Disease Registry, the Public Health Service, or the U.S. Department of Health and Human Services.

Baars, AJ, Theelen, RMC, Janssen, PJCM, Hesse, JM, Apeldorn, MEv, Meijerink, MCM, Verdam, L & Zeilmaker, MJ 2001, *Re-evaluation of human-toxicological maximum permissible risk levels*, RIVM.

Boyce, CP, Lewis, AS, Sax, SN, Eldan, M, Cohen, SM & Beck, BD 2008, 'Probabilistic Analysis of Human Health Risks Associated with Background Concentrations of Inorganic Arsenic: Use of a Margin of Exposure Approach', *Human and Ecological Risk Assessment: An International Journal,* vol. 14, no. 6, 2008/11/21, pp. 1159-1201.

Brown, KG 2007, 'How Creditable are Cancer Risk Estimates from the S.W. Taiwan Database for Arsenic in Drinking Water?', *Human and Ecological Risk Assessment: An International Journal,* vol. 13, no. 1, 2007/03/01, pp. 180-190.

Capon, A & Wright, J 2019, 'An Australian incremental guideline for particulate matter (PM_{2.5}) to assist in development and planning decisions', *Public Health Research & Practice*.

CCME 1999, *Canadian Soil Quality Guidelines for the Protection of Environmental and Human Health, Mercury (Inorganic)*, Canadian Council of Ministers of the Environment.

Chu, HA & Crawford-Brown, DJ 2006, 'Inorganic arsenic in drinking water and bladder cancer: a meta-analysis for dose-response assessment', *Int J Environ Res Public Health,* vol. 3, no. 4, Dec, pp. 316-322.

Clewell, HJ, Thomas, RS, Gentry, PR, Crump, KS, Kenyon, EM, El-Masri, HA & Yager, JW 2007, 'Research toward the development of a biologically based dose response assessment for inorganic arsenic carcinogenicity: a progress report', *Toxicology and applied pharmacology,* vol. 222, no. 3, Aug 1, pp. 388-398.

CRC CARE 2011, *Health screening levels for petroleum hydrocarbons in soil and groundwater. Part 1: Technical development document*, CRC for Contamination Assessment and Remediation of the Environment, CRC CARE Technical Report no. 10, Adelaide. <http://www.crccare.com/products-and-services/health-screening-levels>.

De Flora, S, Camoirano, A, Bagnasco, M, Bennicelli, C, Corbett, GE & Kerger, BD 1997, 'Estimates of the chromium(VI) reducing capacity in human body compartments as a mechanism for attenuating its potential toxicity and carcinogenicity', *Carcinogenesis,* vol. 18, no. 3, Mar, pp. 531-537.

DEFRA 2008, *Consultation on Addendum to Guidelines for halogen and hydrogen halides in ambient air for protecting human health against acute irritancy effects*, Department for Environment, Food and Rural Affairs, London. <https://uk-air.defra.gov.uk/assets/documents/reports/cat11/0805151602_Halogen_and_Hydrogen_Halides_Addendum.pdf>.

DEH 2004, *National Dioxin Program: Dioxins in Australia: A summary of the findings of studies conducted form 2001 to 2004*, Department of the Environment and Heritage, Australian Government.

DEH 2005, *National Dioxins Program, Technical Report No. 12, Human Health Risk Assessment of Dioxins in Australia*, Office of Chemical Safety, Australian Government Department of the Environment and Heritage.

DHHS 2019, *Victorian public health and wellbeing plan 2019–2023*, Victorian Government, Department of Health & Human Services, Prevention and Population Health. <https://www2.health.vic.gov.au/about/health-strategies/public-health-wellbeing-plan>.

EA 2002, *Contaminants in Soil: Collation of Toxicological Data and Intake Values for Humans. Mercury*, Environment Agency.

EA 2009, *Supplementary information for the derivation of SGV for mercury*, Environment Agency, Bristol, UK.

EC 2006, *Integrated Pollution Prevention and Control - Reference Document on Best Available Techniques for Waste Incineration*, European Commission.
<http://eippcb.jrc.ec.europa.eu/reference/BREF/wi_bref_0806.pdf>.

EFSA 2010, *Scientific Opinion on Arsenic in Food*, EFSA Panel on Contaminants in the Food Chain (CONTAM), European Food Safety Authority, Parma, Italy.
<http://www.efsa.europa.eu/en/efsajournal/pub/1351.htm>.

enHealth 2012a, *Australian Exposure Factors Guide*, Commonwealth of Australia, Canberra.
<http://www.health.gov.au/internet/main/publishing.nsf/Content/health-pubhlth-publicat-environ.htm>.

enHealth 2012b, *Environmental Health Risk Assessment, Guidelines for assessing human health risks from environmental hazards*, Commonwealth of Australia, Canberra.
<https://www1.health.gov.au/internet/main/publishing.nsf/Content/A12B57E41EC9F326CA257BF0001F9E7D/$File/Environmental-health-Risk-Assessment.pdf>.

EPA Victoria 2013, *Guidance notes for using the regulatory air pollution model AERMOD in Victoria*, EPA Victoria. <https://www.epa.vic.gov.au/~/media/Publications/1551.pdf>.

EPA Victoria 2017, *Energy from Waste - Guideline, Publication 1559.1*, EPA Victoria, Melbourne, Victoria.
<https://www.epa.vic.gov.au/about-epa/publications/1559-1>.

EPA Victoria 2020a, *Reasonably Practicable. Publication 1856*, State of Victoria (Environment Protection Authority Victoria). <https://www.epa.vic.gov.au/about-epa/publications/1856>.

EPA Victoria 2020b, *Civil construction, building and demolition guide, Publication 1834*, State of Victoria (Environment Protection Authority Victoria). <https://www.epa.vic.gov.au/about-epa/publications/1834>.

EPA Victoria 2021, *Noise limit and assessment protocol for the control of noise from commercial, industrial and trade premises and entertainment venues. EPA Publication 1826.4*, EPA Victoria.
<https://www.epa.vic.gov.au/about-epa/publications/1826-4>.

EPA Victoria 2022, *Guideline for assessing and minimising air pollution in Victoria (for air pollution managers and specialists), Publication Number 1961*, EPA Victoria. <https://www.epa.vic.gov.au/about-epa/publications/1961>.

EPHC 2005, *National Dioxins Program - National Action Plan for Addressing Dioxins in Australia*, Environment Protection and Heritage Council. <http://www.nepc.gov.au/system/files/resources/74b7657d-04ce-b214-d5d7-51dcbce2a231/files/cmgt-rev-national-dioxins-program-national-action-plan-addressing-dioxins-australia-200510.pdf>.

EPHC 2010, *Expansion of the multi-city mortality and morbidity study, Final Report*, Environment Protection and Heritage Council.

EU 2002, *Ambient Air Pollution by Mercury (Hg), Position Paper*, Office for Official Publications of the European Communities, Working Group on Mercury, Luxembourg.
<https://ec.europa.eu/environment/air/pdf/pp_mercury.pdf>.

EU 2010, *Directive 2010/75/EU of the European Parliament and of the Council on Industrial Emissions (Integrated Pollution Prevention and Control)*. <https://eur-lex.europa.eu/legal-content/en/TXT/?uri=CELEX:32010L0075>.

EU 2019, 'Commission Implementing Decision (EU) 2019/2010 of 12 November 2019 establishing the best available techniques (BAT) conclusions, under Directive 2010/75/EU of the European Parliament and of the Council, for waste incineration (notified under document C(2019) 7987) (Text with EEA relevance)', *Official Journal of the European Union,* vol. 312, pp. 55-91.

FAO/WHO 2018, *Joint FAO/WHO Food Standards Programme. Codex Committee on Contaminants in Foods. 12th Session, Utrecht, 12–16 March 2018. Proposed draft revision of the Code of Practice for the Prevention and Reduction of Dioxins and Dioxin-like PCBs in Food and Feed*, Food and Agriculture Organzation and World Health Organization. <http://www.fao.org/fao-who-codexalimentarius/sh-proxy/en/?lnk=1&url=https%253A%252F%252Fworkspace.fao.org%252Fsites%252Fcodex%252FMeetings%252FCX-735-12%252FWD%252Fcf12_08e.pdf>.

Friebel, E & Nadebaum, P 2011, *Health screening levels for petroleum hydrocarbons in soil and groundwater. Part 1: Technical development document*, CRC for Contamination Assessment and Remediation of the Environment, CRC CARE Technical Report no. 10, Adelaide. <http://www.crccare.com/products-and-services/health-screening-levels>.

FSANZ 2003, *The 20th Australian Total Diet Survey*, Food Standards Australia and New Zealand. <http://www.foodstandards.gov.au/Pages/default.aspx>.

FSANZ 2004, *Dioxins in Food, Dietary Exposure Assessment and Risk Characterisation, Technical Report Series No 27*, Food Standards Australia New Zealand.

FSANZ 2008, *The 22nd Australian Total Diet Study*, Food Standards Australia and New Zealand. <http://www.foodstandards.gov.au/Pages/default.aspx>.

FSANZ 2011, *The 23rd Australian Total Diet Study*, Food Standards Australia and New Zealand. <http://www.foodstandards.gov.au/Pages/default.aspx>.

FSANZ 2017, *Supporting Document 2 Assessment of potential dietary exposure to perfluorooctane sulfonate (PFOS), perfluorooctanoic acid (PFOA) and perfluorohexane sulfonate (PFHxS) occurring in foods sampled from contaminated sites*, Food Standards Australia and New Zealand, Commonwealth Department of Health. <http://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-pfas-hbgv.htm>.

FSANZ 2019, *25th Australian Total Diet Study*, Food Standards Australia New Zealand (FSANZ).

Gallen, C, Drage, D, Eaglesham, G, Grant, S, Bowman, M & Mueller, JF 2017, 'Australia-wide assessment of perfluoroalkyl substances (PFASs) in landfill leachates', *Journal of Hazardous Materials,* vol. 331, 2017/06/05/, pp. 132-141.

Golder 2013, *Exposure Assessment and Risk Characterisation to Inform Recommendations for Updating Ambient Air Quality Standards for PM2.5, PMN10, O3, NO2, SO2*, Golder Associates for National Environment Protection Council Service Corporation. <https://www.environment.gov.au/system/files/pages/dfe7ed5d-1eaf-4ff2-bfe7-dbb7ebaf21a9/files/exposure-assessment-risk-characterisation.pdf>.

Health Canada 1993, *Priority Substances List Assessment Report, Arsenic and its Compounds*, Health Canada, <http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/psl1-lsp1/arsenic_comp/index-eng.php>.

Health Canada 1994, *Nickel and its compounds. Priority Substances List Assessment Report*. <http://www.hc-sc.gc.ca/ewh-semt/pubs/contaminants/psl1-lsp1/compounds_nickel_composes/index-eng.php>.

Health Canada 2004, *Contaminated Sites Program, Federal Contaminated Site Risk Assessment in Canada, Part I: Guidance of Human Health Preliminary Quantitative Risk Assessment (PQRA)*. <http://www.hc-sc.gc.ca/ewh-semt/pubs/contamsite/part-partie_i/index-eng.php>.

HEPA 2020, *PFAS National Environmental Management Plan Version 2.0*, The National Chemicals Working Group (NCWG) of the Heads of EPAs Australia and New Zealand (HEPA). <http://www.environment.gov.au/protection/chemicals-management/pfas>.

I-INCE 2011, *Guidelines for Community Noise Impact Assessment and Mitigation, I-INCE Publication Number: 11-1*, International Institute of Noise Control Engineering (I-INCE) Technical Study Group on Community Noise: Environmental Noise Impact Assessment and Mitigation.

IARC 2012a, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 100F: Chemical Agents and Related Occupations*, International Agency for Research on Cancer. <http://monographs.iarc.fr/ENG/Monographs/vol100F/>.

IARC 2012b, *Arsenic, Metals, Fibres, and Dusts, IARC Monographs on the Evaluation of Carcinogenic Risks to Humans Volume 100C*, Arsenic, Metals, Fibres, and Dusts

IARC Monographs on the Evaluation of Carcinogenic Risks to Humans Volume 100C.

IARC 2012c, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 100C, Arsenic, Metals, Fibres and Dusts*. <http://monographs.iarc.fr/ENG/Monographs/vol100C/index.php>.

Imray, P & Neville, G 1996, *Setting a Health-Based Investigation Threshold for Mercury in Soil*, Adelaide.

ITER 1998, *ITER Peer Review on Hexavalent Chromium Meeting Summary, April 16, 1998*. <http://www.tera.org/Peer/HexavalentChromium1998MeetingReport.pdf>.

Jalaudin, B & Cowie, C 2012, *Health Risk Assessment - Preliminary Work to Identify Concentration-Response Functions for Selected Ambient Air Pollutants*, Woolcock Institute of Medical Research. <http://www.nepc.gov.au/system/files/pages/18ae5913-2e17-4746-a5d6-ffa972cf4fdb/files/health-report.pdf>.

Janssen, PJCM, van Apeldoorn, ME, van Engelen, JGM, Schielen, PCJI & Wouters, MFA 1998, *Maximum Permissible Risk Levels for Human Intake of Soil Contaminants: Fourth Series of Compounds*, RIVM, National Institute of Public Health and the Environment, Bilthoven, The Netherlands.

JECFA 2002, *Polychlorinated Dibenzodioxins (PCDDs), Polychlorinated Dibenzofurans (PCDFs), and Coplanar Polychlorinated Biphenyls (PCBs)*, Joint FAO/WHO Expert Committee on Food Additives. <https://apps.who.int/food-additives-contaminants-jecfa-database/chemical.aspx?chemID=2753>.

JECFA 2011, *WHO Technical Report Series 959, Evaluation of certain contaminants in food: seventy-second report of the Joint FAO/WHO Expert Committee on Food Additives*, Joint FAO/WHO Expert Committee on Food Additives.

Jones, RE 1990, 'Hexavalent chrome: threshold concept for carcinogenicity', *Biomed Environ Sci,* vol. 3, no. 1, Mar, pp. 20-34.

JRC 2018, *Working Draft - Best Available Techniques (BAT) Reference Document on Waste Incineration*, Joint Research Centre, European Commission. <http://eippcb.jrc.ec.europa.eu/reference/BREF/WT/WT_Final_Draft1017.pdf>.

Klein, CB, Leszczynska, J, Hickey, C & Rossman, TG 2007, 'Further evidence against a direct genotoxic mode of action for arsenic-induced cancer', *Toxicology and applied pharmacology,* vol. 222, no. 3, 8/1/, pp. 289-297.

Krewski, D, Jerrett, M, Burnett, RT, Ma, R, Hughes, E, Shi, Y, Turner, MC, Pope, CA, 3rd, Thurston, G, Calle, EE, Thun, MJ, Beckerman, B, DeLuca, P, Finkelstein, N, Ito, K, Moore, DK, Newbold, KB, Ramsay, T, Ross, Z, Shin, H & Tempalski, B 2009, 'Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality', *Research report*, no. 140, May, pp. 5-114; discussion 115-136.

Lamm, SH & Kruse, MB 2005, 'Arsenic Ingestion and Bladder Cancer Mortality—What Do the Dose-Response Relationships Suggest About Mechanism?', *Human and Ecological Risk Assessment: An International Journal,* vol. 11, no. 2, 2005/04/01, pp. 433-450.

Langley, AJ 1991, *Response Levels for Arsenic, in The Health Risk Assessment and Management of Contaminated Sites – Proceedings of a National Workshop*, National Workshop on the Health Risk Assessment and Management of Contaminated Sites, El Saadi, O & Langley, A (Eds), South Australian Health Commission, Adelaide, Australia.

Leeman, WR, Van Den Berg, KJ & Houben, GF 2007, 'Transfer of chemicals from feed to animal products: The use of transfer factors in risk assessment', *Food Additives & Contaminants,* vol. 24, no. 1, 2007/01/01, pp. 1-13.

Lizárraga-Mendiola, L, Vázquez-Rodríguez, G, Blanco-Piñón, A, Rangel-Martínez, Y & González-Sandoval, M 2015, 'Estimating the Rainwater Potential per Household in an Urban Area: Case Study in Central Mexico', *Water,* vol. 7, no. 9, pp. 4622-4637.

McKenzie, RD, Byerrum, RU, Decker, CF, Hoppert, CA & Langham, RF 1958, 'Chronic toxicity studies: Hexavalent and trivalent chromium administered by drinking water to rats', *American Medical Association Archives of Industrial Health,* vol. 18, pp. 232-234.

MfE 2002, *Ambient Air Quality Guidelines, 2002 Update*, New Zealand Ministry for the Environment. <https://www.mfe.govt.nz/publications/air/ambient-air-quality-guidelines-2002-update/2-health-based-guideline-values>.

MfE 2011a, *Toxicological intake values for priority contaminants in soil*, Wellington.

MfE 2011b, *Toxicological intake values for priority contaminants in soil*, New Zealand Ministry for the Environment, Wellington. <http://mfe.govt.nz/publications/hazards/toxicological-intake-values-priority-contaminants-soil>.

Morgan, G, Broom, R & Jalaludin, B 2013, *Summary for Policy Makers of the Health Risk Assessment on Air Pollution in Australia*, Prepared for National Environment Protection Council by the University Centre for Rural Health, North Coast, Education Research Workforce, A collaboration between The University of Sydney, Southern Cross University, The University of Western Sydney, The University of Wollongong, Canberra.

NEPC 1999 amended 2013a, *Schedule B1, Guideline on Investigation Levels For Soil and Groundwater, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 1999 amended 2013b, *Schedule B8 Guideline on Community Engagement and Risk Communication, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 1999 amended 2013c, *Schedule B7, Guideline on Health-Based Investigation Levels, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 1999 amended 2013d, *Schedule B7, Guideline on Derivation of Health-Based Investigation Levels, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 1999 amended 2013e, *Schedule B4, Guideline on Site-Specific Health Risk Assessment Methodology, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 2004, *National Environment Protection (Air Toxics) Measure*, National Environment Protection Council. <http://scew.gov.au/nepms/air-toxics>.

NEPC 2010, *Review of the National Environment Protection (Ambient Air Quality) Measure, Discussion Paper, Air Quality Standards*, National Environmental Protection Council.

NEPC 2011a, *National Environment Protection (Air Toxics) Measure*, National Environment Protection Council <http://www.nepc.gov.au/nepms/air-toxics>.

NEPC 2011b, *Methodology for setting air quality standards in Australia Part A*, National Environment Protection Council, Adelaide.

NEPC 2013, *National Environment Protection (Assessment of Site Contamination) Amendment Measure 2013 (No. 1)*, National Environment Protection Council.

NEPC 2016, *National Environment Protection (Ambient Air Quality) Measure*, Federal Register of Legislative Instruments F2016C00215.

NEPC 2021a, *Key Changes to the Ambient Air Quality Measure agreed by Ministers April 2021 - Fact Sheet*, National Environment Protection Council. <http://www.nepc.gov.au/nepms/ambient-air-quality/variation-ambient-air-quality-nepm-ozone-nitrogen-dioxide-and-sulfur>.

NEPC 2021b, *National Environment Protection (Ambient Air Quality) Measure*, Australian Government. <https://www.legislation.gov.au/Details/F2021C00475>.

NHMRC 1999a, *Dental Amalgam and Mercury in Dentistry, Report of an NHMRC working party*, National Health & Medical Research Council.

NHMRC 1999b, *Toxicity Assessment for Carcinogenic Soil Contaminants*, National Health and Medical Research Council.

NHMRC 2002, *Dioxins: Recommendation for a Tolerable Monthly Intake for Australians*, National Health and Medical Research Council and Therapeutic Goods Administration.

NHMRC 2006, *Nutrient Reference Values for Australia and New Zealand, Including Recommended Dietary Intakes*, National Health and Medical Research Council and New Zealand Ministry of Health,

NHMRC 2011 updated 2022, *Australian Drinking Water Guidelines 6, Version 3.8 Updated September 2022, National Water Quality Management Strategy*, National Health and Medical Research Council, National Resource Management Ministerial Council, Canberra.

NICNAS 2001, *Benzene, Priority Existing Chemicals Assessment Report No. 21*, National Industrial Chemicals Notification and Assessment Scheme.

Nielsen, GD, Soderberg, U, Jorgensen, PJ, Templeton, DM, Rasmussen, SN, Andersen, KE & Grandjean, P 1999, 'Absorption and retention of nickel from drinking water in relation to food intake and nickel sensitivity', *Toxicology and applied pharmacology,* vol. 154, no. 1, Jan 1, pp. 67-75.

NRC 2001, *Arsenic in Drinking Water: 2001 Update.*, National Research Council, National Academy Press. <http://www.nap.edu/catalog/10194/arsenic-in-drinking-water-2001-update>.

NSW Chief Scientist 2018, *Advisory Committee on Tunnel Air Quality - Technical Paper 5: Road Tunnel Stack Emissions*, Advisory Committee on Tunnel Air Quality, NSW Chief Scientist and Engineer. <https://chiefscientist.nsw.gov.au/reports/advisory-committee-on-tunnel-air-quality>.

NSW DEC 2003, *Ambient Air Quality Research Project (1996-2001), Internal working paper no. 4, Ambient concentrations of heavy metals in NSW*, Department of Environment and Conservation (NSW).

NSW DECC 2009, *Interim Construction Noise Guideline*, NSW Department of Environment and Climate Change. <www.environment.nsw.gov.au/resources/stormwater/0801soilsconststorm2a.pdf>.

NSW EPA 2004, *Ambient Air Quality Research Project (1996-2001). Internal working paper no. 2, Ambient concentrations of toxic organic compounds in NSW*, Department of Environment and Conservation.

NSW EPA 2013, *Air Emissions in My Community web tool, Substance information*, NSW Environment Protection Authority. <http://www.epa.nsw.gov.au/resources/air/130841AEsubstance.pdf >.

NSW Health 2004, *Comparison of personal exposures to air pollutants by commuting mode in Sydney, BTEX & NO_2*, NSW Department of Health, Sydney.

NTP 2008, *NTP technical report on the toxicology and carcinogenesis studies of sodium dichromate dihydrate (CAS No. 7789-12-0) in F344/N rats and B6C3F1 mice (drinking water studies)*. <http://ntp.niehs.nih.gov/files/546_web_FINAL.pdf>.

OEHHA, *OEHHA Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary*, Office of Expoure and health Hazard Assessment. <https://oehha.ca.gov/air/general-info/oehha-acute-8-hour-and-chronic-reference-exposure-level-rel-summary>.

OEHHA 2003, *The Air Toxics Hot Spots Program Guidance Manual for Preparation of Health Risk Assessments*, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency.

OEHHA 2009, *Chronic Toxicity Summary, Nickel, Evaluation from OEHHA, current to December 2009*. <http://www.oehha.org/air/hot_spots/2008/AppendixD1_final.pdf#page=502>.

OEHHA 2011, *Public Health Goals for Chemicals in Drinking Water, Hexavalent Chromium (Cr VI)*. <http://oehha.ca.gov/water/phg/pdf/Cr6PHG072911.pdf>.

OEHHA 2012, *Air Toxics Hot Spots Program, Risk Assessment Guidelines, Technical Support Document, Exposure Assessment and Stochastic Analysis*, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency.

OEHHA 2015, *Air Toxics Hot Spots Program, Risk Assessment Guidelines, Guidance Manual for Preparation of Health Risk Assessments*, Air, Community, and Environmental Research Branch, Office of Environmental Health Hazard Assessment, California Environmental Protection Agency.

Ostro, B, Broadwin, R, Green, S, Feng, WY & Lipsett, M 2006, 'Fine particulate air pollution and mortality in nine California counties: results from CALFINE', *Environmental health perspectives,* vol. 114, no. 1, Jan, pp. 29-33.

Pearson, AJ & Ashmore, E 2020, 'Risk assessment of antimony, barium, beryllium, boron, bromine, lithium, nickel, strontium, thallium and uranium concentrations in the New Zealand diet', *Food Additives & Contaminants: Part A,* vol. 37, no. 3, 2020/03/03, pp. 451-464.

PHE 2016, *Hydrogen Cyanide, Toxicological Overview*, Public Health England. <https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/500821/Hydrogen_Cyanide_PHE_TO_120216.pdf>.

Pope, IC, Burnett, RT, Thun, MJ & et al. 2002, 'Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution', *JAMA,* vol. 287, no. 9, pp. 1132-1141.

RAIS *The Risk Assessment Information System*, Department of Energy's (DOE's) Oak Ridge Operations Office (ORO).

RIVM 2001, *Risk assessment of historical soil contamination with cyanides; origin, poential human exposure and evaluation of Intervention Values*.

SAB 2005, *Arsenic-Contaminated Soils, Questions and Discussion Materials*, Science Advisory Board, US Environmental Protection Agency. <http://water.epa.gov/scitech/swguidance/standards/criteria/aqlife/arsenic/sab_index.cfm>.

SAHC 1998, *The Health Risk Assessment and Management of Contaminated Sites, Proceedings of the Fourth National Workshop on the Assessment of Site Contamination*.

Sams, R, 2nd, Wolf, DC, Ramasamy, S, Ohanian, E, Chen, J & Lowit, A 2007, 'Workshop overview: arsenic research and risk assessment', *Toxicology and applied pharmacology,* vol. 222, no. 3, Aug 1, pp. 245-251.

Schoen, A, Beck, B, Sharma, R & Dubé, E 2004, 'Arsenic toxicity at low doses: epidemiological and mode of action considerations', *Toxicology and applied pharmacology,* vol. 198, no. 3, 8/1/, pp. 253-267.

Soares, ME, Vieira, E & Bastos Mde, L 2010, 'Chromium speciation analysis in bread samples', *J Agric Food Chem,* vol. 58, no. 2, Jan 27, pp. 1366-1370.

Stevens, B 1991, '2,3,7,8-Tetrachlorobenzo-p-Dioxin in the Agricultural Food Chain: Potential Impact of MSW Incineration on Human Health', in HA Hattemer-Frey & T Curtis (eds), *Health Effects of Municipal Waste Incineration*, CRC Press.

TCEQ 2012, *Arsenic and Inorganic Arsenic Compounds, Development Support Document*, Texas Commission on Environmental Quality.

TCEQ 2013a, *Development Support Document, Benzene*, Texas Commission on Environmental Quality.

TCEQ 2013b, *Development Support Document, Formaldehyde*, Texas Commission on Environmental Quality.

TCEQ 2014a, *Development Support Document, Ammonia*, Texas Commission on Environmental Quality.

TCEQ 2014b, *Hexavalent Chromium (Particulate Compounds), Development Support Document*, Texas Commission on Environmental Quality.

TCEQ 2015a, *Development Support Document, Benzene*, Texas Commission on Environmental Quality. <https://www.tceq.texas.gov/toxicology/dsd/final.html>.

TCEQ 2015b, *Hydrogen Fluoride and Other Soluble Inorganic Fluorides*, Texas Commission on Environmental Quality.

TCEQ 2015c, *Hydrogen Chloride, Development Support Document*, Texas Commission on Environmental Quality.

TCEQ 2016a, *Cadmium and Cadmium Compounds, Development Support Document*, Texas Comission on Environmental Quality.

TCEQ 2016b, *Effects Screening Levels*, <https://www.tceq.texas.gov/toxicology/esl>.

TCEQ 2017a, *Manganese and Inorganic Manganese Compounds, CAS Registry Number: 7439-96-5 (except inorganic manganese compounds in the (VII) oxidation state such as permanganates), Development Support Document*, Texas Commission on Environmental Quality.

TCEQ 2017b, *Cobalt and Cobalt Compounds, Development Support Document*, Texas Commission on Environmental Quality.

TCEQ 2017c, *Nickel and Inorganic Nickel Compounds, Development Support Document*, Texas Commission on Environmental Quality.

TCEQ 2017d, *Chlorine, Development Support Document*, Texas Commission on Environmental Quality. <https://www.tceq.texas.gov/downloads/toxicology/dsd/final/chlorine.pdf>.

TERA 1999, *Toxicological Review of Soluble Nickel Salts prepared for Metal Finishing Association of Southern California, Inc, US Environmental Protection Agency and Health Canada*.

Tiesjema, B & Baars, AJ 2009, *Re-evaluation of some human toxicological Maximum Permissible Risk levels earlier evaluated in the period 1991-2001*, RIVM, National Institute for Public Health and the Environment.

UK Air Quality Standards 2010, *The Air Quality Standards Regulations 2010*, UK Government. <http://www.legislation.gov.uk/uksi/2010/1001/contents/made>.

UK DEFRA & EA 2002, *Contaminants in Soil: Collation of Toxicological and Intake Data for Humans: Chromium*. <http://webarchive.nationalarchives.gov.uk/20140328084622/http://www.environment-agency.gov.uk/static/documents/Research/chromium_old_approach_2028660.pdf>.

UK DEFRA & EA 2014, *SP1010 – Development of Category 4 Screening Levels for Assessment of Land Affected by Contamination*, Contaminated Land: Applications in Real Environments (CL:AIRE), UK DEFRA. <http://randd.defra.gov.uk/Default.aspx?Module=More&Location=None&ProjectID=18341>.

UK EA 2009a, *Contaminants of soil: updated collation of toxicological data and intake values for humans, Nickel*. <https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/291234/scho0409bpvz-e-e.pdf>.

UK EA 2009b, *Supplementary information for the derivation of SGV for mercury*, Environment Agency, Bristol, UK.

UK EA 2009c, *Soil Guideline Values for inorganic arsenic in Soil*, Environment Agency, UK. <https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/313869/scho0409bpvy-e-e.pdf>.

UK EA 2009d, *Supplementary information for the derivation of SGV for arsenic, Science Report SC050021*, UK Environment Agency.

UK EA 2009e, *Contaminants in soil: updated collation of toxicological data and intake values for humans, Benzene. Science Report: SC050021*, UK Environment Agency. <https://www.gov.uk/government/publications/contaminants-in-soil-updated-collation-of-toxicological-data-and-intake-values-for-humans>.

UK EA 2009f, *Soil Guideline Values for cadmium in soil, Science Report SC050021 / Cadmium SGV*, UK Environment Agency

Protocol to the 1979 Convention of Long-Range Transboundary Air Pollution on heavy Metals

USEPA *Integrated Risk Information System (IRIS)*, United States Environmental Protection Agency, 2012.

USEPA 1989, *Risk Assessment Guidance for Superfund, Volume I, Human Health Evaluation Manual (Part A)*, Office of Emergency and Remedial Response, United States Environmental Protection Agency, Washington.

USEPA 1995, *Technical Guidance Manual, Assessing Dermal Exposure from Soil*,

USEPA 1997a, *Mercury Study Report to Congress, Volume V: Health Effects of Mercury and Mercury Compounds*, United States Environmental Protection Agency, Office of Air Quality Planning & Standards and Office of Research and Development.

USEPA 1997b, *Mercury Study Report to Congress, Volume III: Fate and Transport of Mercury in the Environment*, United States Environmental Protection Agency, Office of Air Quality Planning & Standards and Office of Research and Development.

USEPA 1998a, *Carcinogenic Effects of Benzene: an Update*, National Center for Environmental Assessment–Washington Office, Office of Research and Development, U.S. Environmental Protection Agency, Washinton.

USEPA 1998b, *Toxicological Review of Hexavalent Chromium*. <http://www.epa.gov/iris/toxreviews/0144tr.pdf>.

USEPA 1998c, *Toxicological Review of Beryllium and Compounds*, U.S. Environmental Protection Agency, EPA/635/R-98/008.

USEPA 1999, *Extrapolation of the Benzene Inhalation Unit Risk Estimate to the Oral Route of Exposure*, U.S. Environmental Protection Agency, Washington.

USEPA 2000, *Exposure and Human Health Reassessment of 2,3,7,8-Tetrachlorodibenzo-p-Dioxin (TCDD) and Related Compounds*, United States Environmental Protection Agency.

USEPA 2001, *Inorganic Arsenic - Report of the Hazard Identification Assessment Review Committee*, USEPA Health Effects Division, United States Environmental Protection Agency, Washington, DC. <http://www.epa.gov/scipoly/sap/meetings/2001/october/inorganicarsenic.pdf>.

USEPA 2002, *Toxicological Review of Benzene (Noncancer Effects) (CAS NO. 1330-20-7), In Support of Summary Information on the Integrated Risk Information System (IRIS)*, U.S. Environmental Protection Agency.

USEPA 2004, *Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual, (Part E, Supplemental Guidance for Dermal Risk Assessment)*, United States Environmental Protection Agency, Washington, D.C.

USEPA 2005a, *Toxicological Review of Barium and Compounds, In Support of Summary Information on the Integrated Risk Information System (IRIS)*, U.S. Environmental Protection Agency, EPA/635/R-05/001.

USEPA 2005b, *Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities*, Office of Solid Waste and Emergency Response, US Environmental Protection Agency. <https://archive.epa.gov/epawaste/hazard/tsd/td/web/html/risk.html>.

USEPA 2005c, *Supplemental Guidance for Assessing Susceptibility from Early-Life Exposure to Carcinogens*, Risk Assessment Forum, United States Environmental Protection Agency, Washington.

USEPA 2005d, *Guidelines for Carcinogen Risk Assessment*, Risk Assessment Forum, United States Environmental Protection Agency, Washington.

USEPA 2008, *Provisional Peer Reviewed Toxicity Values for Cobalt*, Superfund Health Risk Technical Support Center, National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency.

USEPA 2009, *Risk Assessment Guidance for Superfund, Volume I: Human Health Evaluation Manual, (Part F, Supplemental Guidance for Inhalation Risk Assessment)*, United States Environmental Protection Agency, Washington, D.C.

USEPA 2012, *Provisional Assessment of Recent Studies on Health Effects of Particulate Matter Exposure*, National Center for Environmental Assessment RTP Division, Office of Research and Development, U.S. Environmental Protection Agency.

USEPA 2017, *Toxicological Review of Benzo[a]pyrene*, Integrated Risk Information System, National Center for Environmental Assessment, Office of Research and Development, U.S. Environmental Protection Agency. <https://cfpub.epa.gov/ncea/iris/iris_documents/documents/toxreviews/0136tr.pdf>.

USEPA 2018a, *Risk and Exposure Assessment for the Review of thePrimary National Ambient Air Quality Standard for Sulfur Oxides*, U.S. Environmental Protection Agency Office of Air Quality Planning and Standards Health and Environmental Impacts Division Research Triangle Park, NC. <https://www.epa.gov/sites/production/files/2018-05/documents/primary_so2_naaqs_-_final_rea_-_may_2018.pdf>.

USEPA 2018b, *Integrated Science Assessment for Particulate Matter (External Review Draft), EPA/600/R-18/179*, National Center for Environmental Assessment—RTP Division, Office of Research and Development, U.S. Environmental Protection Agency.

USEPA IRIS *Integrated Risk Information System (IRIS)*, United States Environmental Protection Agency.

Van den Berg, M, Birnbaum, LS, Denison, M, De Vito, M, Farland, W, Feeley, M, Fiedler, H, Hakansson, H, Hanberg, A, Haws, L, Rose, M, Safe, S, Schrenk, D, Tohyama, C, Tritscher, A, Tuomisto, J, Tysklind, M, Walker, N & Peterson, RE 2006, 'The 2005 World Health Organization Reevaluation of Human and Mammalian Toxic Equivalency Factors for Dioxins and Dioxin-Like Compounds', *Toxicological Sciences,* vol. 93, no. 2, October 1, 2006, pp. 223-241.

van Vlaardingen, PLA, Posthumus, R & Posthuma-Doodeman, CJAM 2005, *Environmental Risk Limits for Nine Trace Elements*, National Institute for Public Health and the Environment, RIVM.

Victorian Government 2006, *Ministerial guidelines for assessment of environmental effects under the Environment Effects Act 1978*, Victorian Government Department of Sustainability and Environment.

Victorian Government 2021, *Environmental Reference Standard*, Victoria Government Gazette, No. S 245, 26 May 2021.

Weinberg, M & Tomyn, A 2015, *Community survey of young Victorians' resilience and mental wellbeing Full Report: part A and part B*, Vic Health, Victorian Health Promotion Foundation. <https://www.vichealth.vic.gov.au/-/media/ResourceCentre/PublicationsandResources/Research/Youth-resilience_mental-wellbeing_Full-Report.pdf?la=en&hash=5D6C001F0B6BF33C246A967A9D13A6E5D5E22566>.

WHO 1989, *Arsenic, WHO Food Additives Series 24*, Joint FAO/WHO Expert Committee on Food Additives, World Health Organization. <http://www.inchem.org/documents/jecfa/jecmono/v024je08.htm>.

WHO 1991a, *Environmental Health Criteria 118, Inorganic Mercury*, World Health Organization.

WHO 1991b, *Environmental Health Criteria 108. Nickel.* <http://inchem.org/documents/ehc/ehc/ehc108.htm>.

WHO 1993, *Benzene, Environmental Health Criteria 150*, International Programme on Chemical Safety, World Health Organisation. <http://inchem.org/documents/ehc/ehc/ehc150.htm>.

WHO 1998, *Environmental Health Criteria 202 , Selected Non-Heterocyclic Polycyclic Aromatic Hydrocarbons*, World Health Organization, Geneva.

WHO 1999a, *Manganese and its Compounds. Concise International Chemicals Assessment Document 12*, United Nations Environment Programme, the International Labour Organisation, and the World Health Organization. <http://www.inchem.org/documents/cicads/cicads/cicad12.htm>.

WHO 1999b, *Guidelines for Community Noise*, World Health Organisation, Geneva.

WHO 2000a, *WHO air quality guidelines for Europe, 2nd edition, 2000 (CD ROM version)*, World Health Organisation.

WHO 2000b, *Air Quality Guidelines for Europe, Second Edition*, Copenhagen.

WHO 2000c, *Safety evaluation of certain food additives and contaminants, Fifty-third meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA)*, World Health Organization, Geneva. <http://www.inchem.org/documents/jecfa/jecmono/v44jec13.htm>.

WHO 2000d, *Guidelines for Air Quality*, World Health Organisation, Geneva.

WHO 2000e, *Diphenylmethane Diisocyanate (MDI), Concise International Chemical Assessment Document 27*, United Nations Environment Programme, the International Labour Organization, and the World Health Organization. <http://www.inchem.org/documents/cicads/cicads/cicad27.htm>.

WHO 2000f, *Assessment of the health risk of dioxins: Re-evaluation of the tolerable daily intake (TDI). WHO Consultation, Geneva, 25–29 May 1998. Geneva*, World Health Organization. <http://www.who.int/ipcs/publications/en/exe-sum-final.pdf>.

WHO 2000g, *Air Quality Guidelines for Europe, Second Edition*, Copenhagen. <http://www.euro.who.int/en/publications/abstracts/air-quality-guidelines-for-europe>.

WHO 2001a, *Barium and Barium Compounds, Concise International Chemical Assessment Document 33*, World Health Organization, Published under the joint sponsorship of the United Nations Environment Programme, theInternational Labour Organization, and the World Health Organization. <https://apps.who.int/iris/bitstream/handle/10665/42398/9241530332.pdf;jsessionid=26D79046AD06F3939A4A91E6233A0FFE?sequence=1>.

WHO 2001b, *Beryllium and Beryllium Compounds, Concise International Chemical Assessment Document 32*, World Health Organization. <http://www.inchem.org/documents/cicads/cicads/cicad32.htm#1.0>.

WHO 2001c, *Environmental Health Criteria No. 224, Arsenic and arsenic compounds*, World Health Organisation. <http://www.who.int/ipcs/publications/ehc/ehc_224/en/>.

WHO 2003, *Elemental Mercury and Inorganic Mercury Compounds: Human Health Aspects*, World Health Organization, Geneva.

WHO 2004a, *Safety evaluation of certain food additives and contaminants: methylmercury (addendum)*, World Health Organization, Geneva. <http://www.inchem.org/documents/jecfa/jecmono/v52je23.htm>.

WHO 2004b, *WHO Food Additives Series: 52, Cadmium (addendum)*, World Health Organisation. <http://www.inchem.org/documents/jecfa/jecmono/v52je22.htm>.

WHO 2006a, *Health risks or particulate matter from long-range transboundary air pollution*, World Health Organisation Regional Office for Europe.

WHO 2006b, *Cobalt and Inorganic Cobalt Compounds. Concise International Chemical Assessment Document No. 69*. <http://www.inchem.org/documents/cicads/cicads/cicad69.htm>.

WHO 2009, *Night Noise Guidelines for Europe*, World Health Organisation Regional Office for Europe.

WHO 2010a, *WHO Guidelines for Indoor Air Quality, Selected Pollutants*, WHO Regional Office for Europe.

WHO 2010b, *JECFA 73rd Meeting, Summary and Conclusions*. <http://www.who.int/foodsafety/publications/chem/summary73.pdf>.

WHO 2011a, *Burden of disease from environmental noise, Quantification of healthy life years lost in Europe*, World Health Organisation and JRC European Commission.

WHO 2011b, *Guidelines for Drinking-water Quality, Fourth Edition*, International Program on Chemical Safety, World Health Organisation. <http://www.who.int/water_sanitation_health/publications/2011/dwq_guidelines/en/>.

WHO 2011c, *Evaluation of certain contaminants in food: seventy-second meeting report*, Joint FAO/WHO Expert Committee on Food Additives. WHO technical report series; no. 959. World Health Organisation. <http://whqlibdoc.who.int/trs/who_trs_959_eng.pdf>.

WHO 2013a, *Inorganic chromium(VI) compounds, Concise international chemical assessment document 78*, World Health Organization.

WHO 2013b, *Review of evidence on health aspects of air pollution - REVIHAAP Project, Technical Report*, World Health Organization, Regional Office for Europe.

WHO 2013c, *Health Effects of Particulate Matter, Policy implications for countries in eastern Europe, Caucasus and central Asia*, WHO Regional Office for Europe.

WHO 2017, *Guidelines for Drinking Water Quality, Fourth Edition incorporating the First Addendum*, World Health Organisation. <http://www.who.int/water_sanitation_health/publications/drinking-water-quality-guidelines-4-including-1st-addendum/en/>.

WHO 2018, *Environmental Noise Guidelines for the European Region*, World Health Organization Regional Office for Europe. <http://www.euro.who.int/en/publications/abstracts/environmental-noise-guidelines-for-the-european-region-2018>.

WHO 2019, *Exposure to Dioxins and Dioxin-like Substances: A Major Public Health Concern*, World Health Organization.

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 PM_{10} 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 PM_{10} being assessed. Given that this assessment, has assumed that all of the PM_{10} 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 PM_{10}.

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 linear[7]. The calculation of a relative risk based

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7 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

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 $RR = \exp[\beta(X-X0)]$

Where:

R = relative risk

X-X0 = the change in particulate matter concentration to which the population is exposed (µg/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 µg/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 (µg/m^3)

10 = increase in particulate matter concentration associated with the RR (where the RR is associated with a 10 µg/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)[8] where the exposure-response relationships identified have been directly considered on the basis of the approach outlined below.

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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.

8 For regional guidance, such as that provided for Europe by the WHO (WHO 2006a) 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 $\text{Risk} = \beta \times \Delta X \times B$

Where:

β = slope coefficient relevant to the per cent change in response to a 1 µg/m^3 change in exposure

ΔX = change (increment) in exposure concentration in µg/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 $PM_{2.5}$ 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 0.03 µg/m^3.
- Public health discussions in the literature have noted that a change in particles concentration of 0.02 µg/m^3 or less is generally considered negligible. This assessment has still be undertaken (Capon & Wright 2019).
- The calculation has considered the baseline mortality rate for Whittlesea LGA. The rate in Whittlesea LGA is 267.1 per 100,000 for 2016/20 (all ages and all causes) (refer to **Table 3.2**).
- The baseline incidence is, therefore, 0.002671 for Whittlesea 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 $PM_{2.5}$ (Pope et al. 2002).
- The study found a relative risk (RR) of all-cause mortality of 1.06 per 10 µg/m^3 change in $PM_{2.5}$, and that this risk relationship was in the form of an exponential function. Based on a RR of 1.06 per 10 µg/m^3 change in $PM_{2.5}$, 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:

$$\text{Risk} = \beta \times \Delta X \times B$$

$$= 0.0058 \times 0.03 \times 0.002671$$

$$= 5 \times 10^{-7}$$

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 NSW and Victoria 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 1999b).

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 2012b; NEPC 1999 amended 2013d, 2004) by the WHO (WHO 2000d, 2000g, 2010a). 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 sources including ATSDR.

Some pollutants are not considered to be acute toxicants, which means that they have a very low acute toxicity and, 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, dioxins and furans and thallium.

Based on the above, the acute TRVs listed in **Table B1** have been adopted in this assessment.

In addition to the guidelines provided using the process described above, EPA Victoria has recently published new guidelines for such assessments. These guidelines for short term exposures are listed in **Table B2**. These guidelines are listed separately as it has not been straightforward to assess the basis for these guidelines so the APACs do not always line up with the most relevant guideline available internationally that is known to be based on protection of health.

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 (TCEQ 2015c)	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. Acute air guidelines is protective of all acute effects, with respiratory effects in individuals with asthma being the most sensitive effect (TCEQ 2015c).
Hydrogen fluoride (HF)	0.06 (TCEQ 2015b)	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. Acute air guideline based on increased airway inflammation in human studies (TCEQ 2015b).
Ammonia	0.59 (TCEQ 2014a)	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. Acute air guideline based on the most sensitive effects, namely mild, transient effects in respiratory system and CNS effects in human studies (TCEQ 2014a).
VOCs as 100% benzene	0.58 (TCEQ 2015a)	The group of volatile organic compounds (VOCs) has been assessed on the assumption that100% of the VOCs present comprise benzene, the most toxic VOC compound 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) (TCEQ 2015a) as well as CSN excitation and depression and neurological effects. The acute air guideline is based on decreased lymphocytes in an animal study (TCEQ 2015a). The study used by TCEQ is the same adopted by ATSDR (ATSDR 2007a) 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
Inorganics and organics bound to particulates (where acute effects are relevant)		
Antimony	0.001 (ATSDR 2019b)	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 2019b). Acute air guideline adopted is based on respiratory effects (epithelium effects at base of epiglottis) in an animal study (ATSDR 2019b).
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 2016a)	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). Acute air guideline is based on immunological effects in animals (most sensitive effect identified).
Chromium (Cr VI assumed)	0.0013 (TCEQ 2014b)	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 2014b). Acute air guideline is based on respiratory effects (increased lung weight) in animals.
Cobalt	0.00069 (TCEQ 2017b)	The key health effect associated with inhalation exposures to cobalt in humans and animals are respiratory effects (TCEQ 2017b). 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 toxicity. Acute inhalation value is based on occupational exposures to copper fume (unlikely to be representative of copper bound to particulates). In the absence of any other acute guidelines, this value has been conservatively adopted in this assessment.
Manganese	0.0091 (TCEQ 2017a)	Manganese is an essential element and hence health effects occur as a result of deficiency as well as toxicity. 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 of inorganic mercury attached to particulates. Acute exposure to high concentrations of mercury vapour has been associated with chest pains, haemoptysis, breathlessness, cough and

Chemicals evaluated	Acute air guideline (1-hour average) (mg/m^3)	Key health effects
		impaired lung function with the lung identified as the main target following acute exposure (ATSDR 1999). 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. Acute air guideline is based on protection of CNS effects in an animal study.
Nickel	0.0011 (TCEQ 2017c)	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 (UK EA 2009a). 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.

References

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

Table B2: Acute TRVs adopted in this assessment – EPA Victoria APACxs

Chemicals evaluated	Acute air guideline (1-hour average) (mg/m^3)
Hydrogen chloride (HCl)	2.1
Hydrogen fluoride (HF)	0.06
Ammonia	3.2
VOCs as 100% benzene	0.58
Antimony	No value listed for 1 hour average
Arsenic	0.0099
Cadmium	0.018
Chromium (Cr VI assumed)	0.0013
Cobalt	Not listed
Copper	0.1
Manganese	0.0091
Mercury (as inorganic and elemental)	No value listed for 1 hour average
Nickel	0.0002
Thallium	Not listed
Vanadium	Not listed

B3 Values adopted for the assessment of chronic exposures

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 2013e) and enHealth (enHealth 2012b). The identification of the most appropriate and robust TRVs has followed guidance from Australia (enHealth 2012b), as noted above.

For carcinogens, this guidance requires consideration of the mechanism of action for the development of cancer. Some cancers are caused by a threshold mechanism, where there needs to be sufficient exposures to trigger the damage that results in or promotes the development of cancer. Other carcinogens are genotoxic/mutagenic and act in a way such that and any level of exposure is assumed to result in damage that may increase the lifetime risk of cancer. Not all carcinogenic (and not all mutagenic) pollutants cause cancer in the same way and hence the mechanism of action has been considered in the identification of appropriate TRVs for use in this assessment.

All chronic TRVs adopted for the assessment of chronic exposures are protective of all 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 as well as dioxins, 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 determined if the health effects identified can occur.

Tables B3 and B4 present the TRVs adopted for the assessment of chronic health effects associated with exposure to the other pollutants considered in this assessment. **Table B3** presents the threshold TRVs, while **Table B4** presents the non-threshold TRVs.

Table B2: Summary of hazards – chronic exposures

Pollutant evaluated	Summary of chronic health effects
Gases	
Hydrogen chloride (HCl)	The key hazards associated with HF, 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 determined HCl not classifiable in relation to carcinogenicity. The available data does 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 2015c). 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 2015b). Chronic air guideline adopted is based on the most sensitive effect, namely skeletal fluorosis, based on an occupational study (TCEQ 2015b). Ambient or background levels of HCl in air are expected to be negligible (DEFRA 2008).
Chlorine	The key hazards associated with chlorine, relate to acute effects, where the respiratory system is the most sensitive health effect (refer to **Table B1**). Chlorine gas has been used in industrial processes for many years and several occupational studies have been published, however most of these do not show adverse health effects for workers exposed to low concentrations (around 1 ppm). Several long-term controlled inhalation exposure studies have been conducted in animals, where the tissues inside the nose were mainly affected following chronic inhalation exposures. There is no evidence that chlorine is carcinogenic (ATSDR 2010; TCEQ 2017d). The chronic air guideline adopted is based on the most sensitive effect identified, namely eye irritation and nasal and tracheal lesions in animals (TCEQ 2017d). Ambient or background levels of chlorine in air are expected to be negligible as chlorine is rapidly degraded in air (ATSDR 2010).
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 2004b; TCEQ 2014a). 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 and occupational study (TCEQ 2014a). 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).
Cyanide (as HCN)	The key hazards associated with ammonia, relate to acute effects, where CNS effects are the most sensitive health effects (refer to **Table B1**). In relation to chronic exposures, the available studies indicate chronic low level exposures in occupational environments can cause neurological, respiratory, cardiovascular and thyroid effects (OEHHA). IARC has not classified hydrogen cyanide as carcinogenic, and the USEPA has classified cyanide s Group D: not classifiable. Hydrogen cyanide does not have genotoxic potential (PHE 2016).

Pollutant evaluated	Summary of chronic health effects
	The chronic air guideline adopted from the USEPA is based on the most current evaluation and is based on the most sensitive effects, namely CNS effects in a rat study. The value adopted is the same as the evaluation available from OEHHA which is based on CNS effects, thyroid enlargement and haematological disorders in an occupation study, and more conservative than that available from RIVM (RIVM 2001). WHO (WHO 2017) notes that even healthy individuals have a small amount of cyanide in their bodies (mainly associated with the breakdown of cyanogenic foods, vitamin B12 and heavy smoking). Ambient or background concentrations in air (for non-smokers) are considered to be negligible.
Sulfuric acid mist (as H_2SO_4)	The key hazards associated with sulfuric acid mists, relate to acute effects, where respiratory irritation is the most sensitive health effect (refer to **Table B1**). In relation to chronic exposures, the corrosive and irritant aspect of the acid gas is also relevant. Irritation to the respiratory system, and etching and erosion of teeth are the key effects (ATSDR 1998; OEHHA). There is no data to indicate that sulfuric acid by itself is carcinogenic, however occupational studies have shown increased cancers of the larynx (potentially attributable to smoking and other chemicals present). IARC has classified occupational exposures to strong inorganic acid mists containing sulfuric acid as carcinogenic (current to 2022). This classification does not relate to sulfuric acid mists along or for non-occupational environments (ATSDR 1998). The chronic air guideline is based on respiratory effects in animals (OEHHA). Ambient or background levels of sulfuric acid mists in air are expected to be negligible.
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 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 2012a; USEPA 2005d, 2005c). Benzene is carcinogenic via oral and inhalation routes of exposure (ATSDR 2007a; IARC 2012a; UK EA 2009e; 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 (WHO 2000d), 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 2015a). 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.
Inorganics and organics bound to particulates	
Antimony	Antimony in 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 EPA 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).

Pollutant evaluated	Summary of chronic health effects
	The chronic air guideline adopted in this assessment is based on respiratory effects (lung inflammation) in animals from ATSDR (ATSDR 2019b), noting no other chronic inhalation guidelines are available. Oral (and dermal) exposures have been assessed on the basis of the tolerable daily intake adopted by the NHMRC and WHO in deriving drinking water guidelines (NHMRC 2011 updated 2022; WHO 2017). Background intakes have been considered (where relevant).
Arsenic	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 2012c). The mechanism of action in relation to carcinogenicity is not clear and remains debated (IARC 2012c; 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 (NEPC 1999 amended 2013d), 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 is 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 relates to exposures within and near copper smelters, however there is insufficient data to indicate that these effects are not of importance in other 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 of have been considered (where relevant).
Barium	At low doses, barium acts as a muscle stimulant and at higher doses affects the nervous system eventually leading to paralysis. Acute and subchronic oral doses of barium cause vomiting and diarrhea, followed by decreased heart rate and elevated blood pressure. Higher doses result in cardiac irregularities, weakness, tremors, anxiety, and dyspnea. Subchronic and chronic oral or inhalation exposure primarily affects the cardiovascular system resulting in elevated blood pressure. Subchronic and chronic inhalation exposure of human populations to barium-containing dust can result in a benign pneumoconiosis called "baritosis." This condition is often accompanied by an elevated blood pressure but does not result in a change in pulmonary function. The International Agency for Research on Cancer (IARC) has not classified barium as to its carcinogenicity. The USEPA (USEPA 2005a) concluded that barium is considered not likely to be carcinogenic to humans via oral intake. Other agencies have concluded that there is no evidence that barium is carcinogenic (WHO 2001a). In addition, the weight of evidence supports that barium is not genotoxic. Oral (and dermal) exposures have been assessed on the basis of the tolerable daily intake adopted by the NHMRC and WHO in deriving drinking water guidelines (NHMRC 2011 updated 2022; WHO 2017). Inhalation exposures have been assessed on the basis of the only quantitative value from RIVM (Baars et al. 2001). Background or ambient intakes are considered negligible.
Beryllium	Occupational exposure to beryllium has been associated with acute and chronic lung diseases. Chronic disease is associated with long-term inhalation exposures to dust particles containing beryllium, has an immunological component and a latent period which varies depending on the beryllium species. The inhalation data led the International Agency for Research on Cancer to conclude that beryllium and beryllium compounds are carcinogenic to humans (Group 1, sufficient evidence of carcinogenicity in humans and sufficient evidence in animals) (IARC 1993). The USEPA has classified beryllium as B1 – probable human carcinogen. The WHO (WHO 2001b) also classified beryllium as carcinogenic based on occupational inhalation studies. Further review of genotoxicity by IARC (IARC 2012b) indicates that the evidence for mutagenic activity was weak or negative, however review of the available studies indicates that the underlying mechanism for carcinogenesis is complex and likely to involve several possible interactive mechanisms. Hence the evidence for a genotoxic mode of action is not clear, however there may be some mechanisms that relate to genotoxicity that affect carcinogenicity.

Pollutant evaluated	Summary of chronic health effects
	Based on the available data carcinogenic effects of inhaled beryllium in non-occupational environments are not genotoxic and a threshold can be adopted. There is, however, no clear evidence that the compounds are carcinogenic when administered orally. Beryllium was not mutagenic in tests with different strains of bacteria but caused chromosomal aberrations and gene mutations in cultured mammalian cells. Hence a threshold is adopted for the assessment of oral exposures. Oral (and dermal) exposures have been assessed on the basis of the tolerable daily intake adopted by the NHMRC and WHO in deriving drinking water guidelines (NHMRC 2011 updated 2022; WHO 2017). Inhalation exposures have been assessed on the basis of the value from the WHO and USEPA (USEPA 1998c; WHO 2001b). Background or ambient intakes are considered to be negligible.
Boron	The primary health effects associated with inhalation exposure of humans to boron are acute respiratory irritation. Health effects related to chronic exposures include reproductive and developmental effects, haematological effects. The International Agency for Research on Cancer (IARC) has not evaluated boron due to inadequate data. And the limited studies available do not indicate the compound is genotoxic. Limited evaluations are available for boron, hence the oral (and dermal) exposures have been assessed on the basis of the tolerable daily intake adopted by the NHMRC and WHO in deriving drinking water guidelines (NHMRC 2011 updated 2022; WHO 2017), which is consistent with the USEPA evaluated. There are no chronic inhalation guidelines available, hence the oral value has been used to assess all pathways of exposure. Background or ambient intakes have also been considered.
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 is 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 2010b) which is consistent with the approach and value adopted by the NHMRC (NHMRC 2011 updated 2022). Sufficient data is available to conclude cadmium is carcinogenic via inhalation exposures. The inhalation air guideline adopted WHO 2000) 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. 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 2013a). 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 2013a). 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 2013a). IARC (IARC 2012c) has classified Cr VI compounds as Group 1 carcinogens: carcinogenic to humans. Chromium is classified by the US EPA 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 1998b). 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 2012a) is most appropriate. Inhalation exposures need to be assessed on the basis of data that is protective of noncarcinogenic and carcinogenic effects, with a non-threshold approach relevant for the

Pollutant evaluated	Summary of chronic health effects
	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, 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 sulphate 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 suggests a non-genotoxic mechanism for carcinogenicity. Oral and dermal exposures have been assessed on the basis of a threshold value from the 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 2011b), 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 2019a). 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 2011b). 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 of 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 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.

Pollutant evaluated	Summary of chronic health 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 of inorganic mercury attached to particulates. 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. 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 of 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 noncarcinogenic 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.
Selenium	Selenium is an essential element for many species, including humans, hence health effects may occur as a result of deficiency as well as toxicity. Exposure to elevated levels of selenium can result in brittle hair and deformed nails, CNS effects, gastrointestinal disturbances, dermatitis and dizziness. Selenium is not considered to be carcinogenic. Assessment of exposure to selenium has been undertaken on the basis of a threshold that is based on an upper tolerable limit from the diet, accounting for background intakes (predominantly via the diet).
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 is 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 by RIVM (Janssen et al. 1998) and recommended following more recent review (Pearson & Ashmore 2020) adopted, with background intakes also considered.
Tin	There is limited information available in relation to tin, however inorganic tin is considered to be of low toxicity. The main route of exposure to tin is via food, in particular canned food. health effects may include gastrointestinal effects, anaemia and effects on the liver and kidney (ATSDR 2005b). Inorganic tin compounds are not considered carcinogenic (ATSDR 2005b). Exposure to tin has been assessed on the basis of a threshold toxicity value from RIVM (Tiesjema & Baars 2009) that is lower than the JECFA guideline for safe levels of tin in food. Background intakes are 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 relates to vanadium pentoxide which is considered to have

Pollutant evaluated	Summary of chronic health effects
	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.
Zinc	Zinc is an essential element for all living things, including man. Hence adverse effects are associated with deficiency and toxicity associated with excess intake. There are a number of significant health effects associated with zinc deficiency. Toxicity in relation to dietary intakes has not been reported, however data is available from occupational exposures or pharmacological interventions where high levels of exposure have resulted in health effects. These effects include respiratory effects, effects on the immune system, gastrointestinal effects and changes in cholesterol levels (WHO 2017). Zinc is not considered to be carcinogenic. All exposures to zinc have been assessed on the basis of the upper limit recommended by NHMRC in the diet (NHMRC 2006), noting that 80% of the upper limit is considered to be background intakes (predominantly from the diet).
Dioxins and furans	Dioxins and furans are widely present in the environment, some occurring naturally but most as unwanted by-products of combustion. 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, 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. Developmental effects in males are the most sensitive reproductive health end-point, making children, particularly breastfed infants, a population at elevated risk. 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. 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 the 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 comprise 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/m^3)	Oral/dermal TRV (mg/kg/day)	GI absorption factor*	Dermal absorption*	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)			10%	10%
Antimony	0.0003 A	0.00086 NH	15%	0	0%	0%
Arsenic	0.000067 T	0.002 N	100%	0.03	50%	0%
Cadmium	0.000005 W	0.0008 W	2.5%	0.001	60%	20%
Chromium (Cr VI assumed)	0.0000043 T	0.0009 A	2.5%	0	10%	0%
Cobalt	0.0001 W	0.0014 D	100%	0	20%	0%
Copper	0.49 R	0.14 W	100%	0	60%	60%
Lead	0.0005 N	0.0006 X	100%	0	50%	0%
Manganese	0.00015 W	0.14 A	100%	0	50%	20%
Mercury (as inorganic and elemental)	0.0002 W	0.0006 W	7%	0	60%	0%
Nickel	0.000059 T	0.012 W	4%	0	60%	10%
Thallium	0.0007R	0.0002 D	100%	0	10%	10%
Vanadium	0.0001 A	0.002 D	2.6%	0	0%	0%
Dioxins and furans	8.05E-09 R	2.3E-09 NH	100%	0.03	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*	Background intakes
VOCs as 100% benzene	0.006 U	NA (Gaseous pollutant)			NA for non-threshold risk calculations

Notes for Table B3 and B4:

* GI factor and dermal absorption values adopted from RAIS (accessed in 2022) (RAIS), except for BaP where 6% has been adopted (MfE 2011b)

** 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 2013d) 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

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 2012b, 2012c, 2012d)

D = TRV available from RIVM (Baars et al. 2001; Janssen et al. 1998; van Vlaardingen et al. 2005)

N = Inhalation guideline adopted for lead from the NEPM (NEPC 2016), arsenic oral/dermal value and BaP oral/dermal value as adopted in ASC-NEPM (NEPC 1999 amended 2013d)

NH = Dioxin value (and background intakes, which includes natural soil) adopted from NHMRC (NHMRC 2002) and Environment Australia (DEH 2005; EPHC 2005), and antimony, barium, boron, selenium and lead value consistent with that adopted by NHMRC to assess intakes in drinking water (NHMRC 2011 updated 2022) and the value adopted for zinc based on the tolerable upper limit in the diet from NHMRC (NHMRC 2006)

T = TRV available from TCEQ, relevant to chronic inhalation exposures (and HI=1) (TCEQ 2013b, 2014a, 2015c, 2015b, 2017c, 2017d)

U = TRV available from the USEPA IRIS (current database) (USEPA IRIS)

W = TRV available from the WHO, relevant to chronic inhalation exposures (WHO 1999a, 2000g, 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 Introduction

This appendix presents the methodology and assumptions adopted in the calculation of risks related to the assessment of acute and chronic risks via inhalation and other pathways assessed for chronic exposure that may occur following deposition of particles with relevant persistent chemicals attached.

B5 Acute toxicity reference values

Acute toxicity reference values are values protective for exposure over short time periods. It is not possible for such concentrations to be present in air at ground level around this facility for extended periods as equipment at the facility would fail if operated under these conditions.

There are a number of reputable sources including the following:

- **Texas Commission on Environmental Quality (2015)**. Short term criteria have been obtained from the TCEQ Environmental Screening Levels (TCEQ 2016b). 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 2012b; NEPC 1999 amended 2013c, 2004) by the WHO (WHO 2000d, 2000g, 2010a). 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. It is noted that TCEQ also list values termed as ESLs or effect screening levels which are based on a target HI of 0.3 instead of 1. TCEQ have adopted a policy that initial screening occurs against a guideline using this lower HI. This policy has not been adopted in Australia so the original ReV value which is based on the toxicity data directly has been adopted for this assessment. TCEQ also have an older spreadsheet database but the derivation of values in that database is not clearly outlined so these values have not been used.
- **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.
- **USEPA Protective Action Criteria (PAC)** values, which are all based on a target HI of 1. PACs provide threshold values with varying levels of protection, as a result of elevated exposure (PAC-1, PAC-2 and PAC-3). For this assessment, the most conservative PAC value has been adopted, PAC-1> The PAC-1 guidelines have been adjusted by 100 fold (i.e. reduced by 100) to adjust from an occupational relevant value to a public health relevant value. This is then the concentration above which the public, including sensitive individuals, for 1 hour may experience discomfort, irritation or other non-sensory effects that are not disabling and transient (i.e. reversible upon cessation of exposure). Exposures below these thresholds are considered protective for these effects. These values have only been adopted where no acute guidelines are available from the above (or any other reliable source).

Based on the above the following acute TRVs have been adopted in this assessment:

Table B1: Short term toxicity reference values

Chemical	Acute health based criteria (inhalation only)		
	Acute air guideline (mg/m^3)	Averaging time	Source of guideline
Hydrogen chloride (HCl)	0.66	1 hour	(TCEQ 2015c)
Hydrogen fluoride (HF)	0.06	1 hour	(TCEQ 2015b)
Ammonia	0.59	1 hour	(TCEQ 2014a)
Cadmium (Cd)	0.018	1 hour	(TCEQ 2016a)
Thallium (Tl)	0.0006	1 hour	USEPA PAC-1#
Beryllium (Be)	0.000023	1 hour	USEPA PAC-1#
Mercury (Hg)	0.0006	1 hour	OEHHA
Antimony (Sb)	0.015	1 hour	USEPA PAC-1#
Arsenic (As)	0.0099	1 hour	(TCEQ 2012)
Lead (Pb)	0.15	1 hour	USEPA PAC-1
Chromium (Cr VI assumed)	0.0013	1 hour	(TCEQ 2014b)
Cobalt (Co)	0.00069	1 hour	(TCEQ 2017b)
Copper (Cu)	0.1	1 hour	OEHHA
Manganese (Mn)	0.0091	1 hour	(TCEQ 2017a)
Nickel (Ni)	0.0011	1 hour	(TCEQ 2017c)
Selenium (Se)	0.006	1 hour	USEPA PAC-1#
Vanadium (V)	0.03	1 hour	OEHHA
Tin (Sn)	0.06	1 hour	USEPA PAC-1#
Dioxin-like compoundss and furans	0.0000013	1 hour	USEPA PAC-1#
Benzene	0.58	1 hour	(TCEQ 2015a)

adjusted from occupational relevant value to public health relevant value

B6 Chronic toxicity reference values

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.

Threshold

A threshold chemical has a concentration below which health effects are not considered to occur. IT is assumed that for a non-threshold chemical substance it is theoretically possible to 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 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 1999b).

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 2013d). Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.
- Beryllium - review of genotoxicity by IARC (IARC 2012b) indicates that the evidence for mutagenic activity was weak or negative (i.e. non-genotoxic), and review by NHMRC and NEPC (NEPC 1999 amended 2013d; NHMRC 2011 updated 2022) 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.
- Cadmium – the available data suggests only weak evidence of genotoxicity and review by NEPC (NEPC 1999 amended 2013d) 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 2013d) 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.
- 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 2013d). Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.

- Dioxins and furans, 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. Thus, the quantification of risk for $PM_{2.5}$ follows a non-threshold approach as described in **Appendix A**.

Values adopted

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 2013e) and enHealth (enHealth 2012b). The identification of the most appropriate and robust TRVs has followed guidance from Australia (enHealth 2012b).

For the gaseous pollutants considered in this assessment, only inhalation TRVs are relevant and have been adopted. For inorganics (metals) as well as dioxin-like compounds, 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 presents the TRVs adopted for the assessment of chronic health effects associated with exposure to the other pollutants considered in this assessment.

Table B2: Summary of chronic TRVs adopted for pollutants – threshold effects

Pollutant	Inhalation TRV (mg/m^3)	Oral/dermal TRV (mg/kg/day)	GI absorption factor*	Dermal absorption*	Background intakes (as percentage of TRV)
Hydrogen chloride (HCl)	0.026 T	NA (gaseous pollutant)			0%
Hydrogen fluoride (HF)	0.029 T	NA (gaseous pollutant)			0%
Ammonia	0.32 T	NA (gaseous pollutant)			0%
Cadmium	0.000005 W	0.0008 W	100%	0	60%
Thallium	0.0028 R	0.0008 U	3%	0	0%
Beryllium	0.00002 W, U	0.002 W, U	100%	0.001	10%
Mercury (as inorganic and elemental)	0.0002 W	0.0006 W	7%	0.001	40%
Antimony	0.0002 U	0.00086 NH	15%	0	0%
Arsenic	0.001 D	0.002 N	100%	0.005	50%
Lead	0.0005 N	0.0035 NH	100%	0	50%
Chromium (Cr VI assumed)	0.0001 U	0.001 A	100%	0	10%
Cobalt	0.0001 W	0.0014 D	100%	0.001	20%
Copper	0.49 R	0.14 W	100%	0	60%
Manganese	0.00015 W	0.14 A	100%	0	50%
Nickel	0.00002 E	0.012 W	100%	0.005	60%
Vanadium	0.0001 A	0.002 D	100%	0	0%
Tin	0.7 R	0.2 D	100%	0	50%
Dioxin-like compounds	8.05E-09 R	2.3E-09 NH	100%	0.03	54%
Benzene	0.03 U	NA (gaseous pollutant)			10%

Notes for Table B2:

* GI factor and dermal absorption values adopted from RAIS (accessed in 2018) (RAIS)

** 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 2013d) 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

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 2012b, 2012c, 2012d)

D = TRV available from RIVM (Baars et al. 2001; van Vlaardingen et al. 2005)

E = TRV available from the UK Environment Agency (UK EA 2009a)

N = Inhalation guideline adopted for lead from the NEPM (NEPC 2016), and arsenic oral/dermal value as adopted in ASC NEPM (NEPC 1999 amended 2013d).

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 value 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 2014a, 2015c, 2015b)

U = TRV available from the USEPA IRIS (current database) (USEPA IRIS)

W = TRV available from the WHO, relevant to chronic inhalation exposures (WHO 1999a, 2000g, 2000e, 2006b, 2017), noting inhalation value adopted for mercury is for elemental mercury (worst case) (WHO 2003, 2011b)

B7 Toxicological profiles

Arsenic

Background

Several comprehensive reviews of arsenic in the environment and toxicity to humans are available (ATSDR 2007b; NRC 2001; UK EA 2009c, 2009d; WHO 2001c).

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 2007b). Under reducing conditions arsenite (AsIII) is the dominant form and in well oxygenated environments, arsenate (AsV) predominates (WHO 2001c). Arsenic is the 20th most commonly occurring element in the earth's crust occurring at an average concentration of 3.4 ppm (ATSDR 2007b).

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 2011c). 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 2012c).

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 2012c) 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 2012c) 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 2005d) 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; and
- 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 associate 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 (ATSDR 2007b; IARC 2012c) 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 2011b) 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 µg/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 2011c) following further review (refer to discussion below).
- The previous ADWG (NHMRC 2004) derived a guideline of 7 µg/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 µg/kg/day. The current ADWG (NHMRC 2011 updated 2022) has adopted a guideline of 10 µg/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 µg/L. It is noted that this level is equivalent to an adult (70 kg) intake of 0.28 µg/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 µg/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 µg/kg/day (rounded to 3 µg/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 (WHO 2011c) 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 ($BMDL_{0.5}$) of lung cancer

(most sensitive endpoint) of 3 µg/kg/day (ranging from 2-7 µg/kg/day). Uncertainties associated with the assumptions related to total exposure, extrapolation of the $BMDL_{0.5}$ and influences of the existing health status of the population were identified. Given the uncertainties and that the $BMDL_{0.5}$ 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 µg/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 µg/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 2011b) 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 µg/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 2011c, 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 2011c, 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 or 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 2011c, 2017) is not considered appropriate for the Australian population.

Based on the above considerations a TRV of 2 µg/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 2011c);
- The value is within the range of no observable adverse effect levels (NOAELs) identified by RIVM (Baars et al. 2001), US EPA (USEPA IRIS) and ATSDR (ATSDR 2007b) that are associated with non-carcinogenic effects (and derived from drinking water studies in Taiwan and Bangladesh) of 0.8 to 8 µg/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 2013d).

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 µg/m^3 (based on the review (ATSDR 2007b)). Applying an uncertainty factor of 10 to address variability in human susceptibility, a tolerable concentration (TC) in air of 1 µg/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 2001c) 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 2001c)) demonstrate a statistically significant excess risk of lung cancer at cumulative exposure levels of approximately ≥750 μg/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 μg/m^3 is lower than the cumulative exposure value identified by WHO (WHO 2001c) of 750 μg/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 μg/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 mg/m^3 relevant to assessing 1 hour average exposures was determined based on maternal toxicity in rats exposed via an inhalation study ($NOEAL_{HEC}$ of 3.89 mg/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 (μg/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 μg/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 mg/kg/day for oral and dermal intakes
- Inhalation TRV = 0.000067 mg/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 2007a).

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 2007a).

Exposure, absorption, health effects

There is no clinical disease which is unique to benzene toxicity. However, the effects on the haemotopoietic 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 2007a).

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 2007a).

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 2012a; USEPA 2005d, 2005c).

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 2007a; IARC 2012a; UK EA 2009e; 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 2004a). 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.

Source	Value	Basis/Comments
New Zealand (MfE 2002)	Air GV = 0.03 mg/m^3	Air guideline value (as an annual average) base ion the WHO upper value inhalation unit risk (as below) and an acceptable carcinogenic risk between 1 in10,000 and 1 in 100,000.
WHO	**SF = 0.035 (mg/kg/day)$^{-1}$** **UR =6x10^{-6} (ug/m^3)$^{-1}$**	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 (WHO 2000d) 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 2009e)	Derived index doses	Oral index dose derived on the basis of US EPA 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.
Texas (TCEQ 2013a)	Chronic ESL (non-threshold) = 0.0045 mg/m^3, UR = 2.2 x 10^{-6} (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 2007a)	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 1998a, 1999) (non-threshold)	SF = 0.015 to 0.055 (mg/kg/day)$^{-1}$ UR =2.2 to 7.8x10^{-6} (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 US EPA. 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. Data collected in Sydney (NSW EPA 2004) for the period 1996 to 2001 reported a range of average concentrations that included 0.0074 mg/m^3 in 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. Concentrations of benzene in other cities are noted to contribute 2.5% (in Perth) and 6% (Melbourne) of the TRV. It is noted that the CRC CARE (Friebel & Nadebaum 2011) derivation of HSLs adopted the maximum background level of 20% from the DEC (2004) study for Sydney CBD.

The data reported by DEC (2004) is dated and is not considered to reflect more current benzene emissions. Specifically, since 2006 the national cleaner fuel standards required that refineries reduce benzene levels in petrol from around four per cent to less than one per cent (Fuel Standard (Petrol) Determination 2001). 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/m^3 (NSW EPA 2013) (consistent with the lowering of benzene content in fuel). These levels are more relevant to current levels of benzene in urban air and comprise up to 5% of the TRV. To be conservative a background intake of 10% of the TRV has been considered where the threshold TRVs are adopted. It is noted that this is lower than the default adopted in the development of the HSLs (Friebel & Nadebaum 2011) however it is based on more current data that reflects lower standards for benzene in fuel.

These background intakes are only of significance for the assessment of chronic exposures, and where the data is from one source only. Where the data is from measured air concentrations that include all significant air sources then background intakes from water or food are negligible.

Background intakes are only relevant where the threshold TRVs are utilised. They are not applicable to the assessment of non-threshold effects.

Cadmium

General

Several comprehensive reviews of cadmium in the environment and toxicity to humans are available (ATSDR 2012e; UK EA 2009f; WHO 2004b).

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 2011b).

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 2004b). 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 2011b), 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/m^3, ranging from 0.3 to 1 ng/m^3. 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 2012c). 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 2011b):

- 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 is 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 2011b)	TDI = 0.0008 mg/kg/day	Adopted the toxicity value from the WHO review (as below).
JECFA (WHO 2010b)	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

Source	Value	Basis/Comments
		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.
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 2012c). 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 2000g). 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 $\mu g/m^3$ and the UK EA (2009) derived an inhalation TDI of 0.0014 µg/kg/day (which can be converted to a guideline value of 0.005 $\mu g/m^3$ – the same as the WHO value).

The review by TCEQ (TCEQ 2016a) 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 µg/m^3) was lower than that derived for carcinogenic effects (0.02 µg/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 2010b)
- Dermal absorption (DAF) = negligible (0%)
- Inhalation TRV (TRV_I) = 0.000005 mg/m^3 (WHO 2000g)
- 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 2012a; 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 2012a).

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 2012a).

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 2012a).

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 2012a). 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 2012a).

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 2014b).

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 2013a).

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 2013a).

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 2013a).

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 et al. 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 old's, 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 is 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/m^3 (Baars et al. 2001) to 3 ng/m^3 (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 2012c) 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 US EPA 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 1998b).

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 2011b)	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 2012a)	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 1998b)	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 2012a) 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 US EPA (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 US EPA 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 1998b) 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 2013a) has derived a tolerable concentration of 0.03 µg/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 µg/m^3 (based on lifetime exposures and 1 in 100,000 risk). This is based on the WHO (WHO 2000b, 2013a) inhalation unit risk of 0.04 (µg/m^3)$^{-1}$ derived from the mean of a number of occupational studies.
- The USEPA (USEPA 1998b) derived an inhalation RfC of 0.0001 mg/m^3 or 0.1 µg/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 mg/m^3 or 0.3 µg/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 µg/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 (WHO 2000b) has derived a range of air guideline values based on an inhalation unit risk of 0.04 (µg/m^3)$^{-1}$ derived from the mean of a number of occupational studies.
- USEPA (USEPA 1998b) also derived a unit risk of 0.012 (µg/m^3)$^{-1}$ derived from one occupational study (also considered by WHO).
- TCEQ (TCEQ 2014b) has derived a noncarcinogenic air guideline of 0.22 µg/m^3 based on changes in lung weight in rats, and a carcinogenic air guideline of 0.0043 µg/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 µg/kg/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 µg/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 0.0015-0.0025 µg/m^3).
- ATSDR (ATSDR 2012a) 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 2014b) 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 mg/kg/day (ATSDR 2012a)
- Inhalation TRV (TRV_i) = 0.0043 µg/m^3 (TCEQ 2014b)
- 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 WHO_{05} TEQ.

The National Dioxins Program (NDP) has focused on the 29 most toxic of these compounds which are recognised internationally as being 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 manufactured or used 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

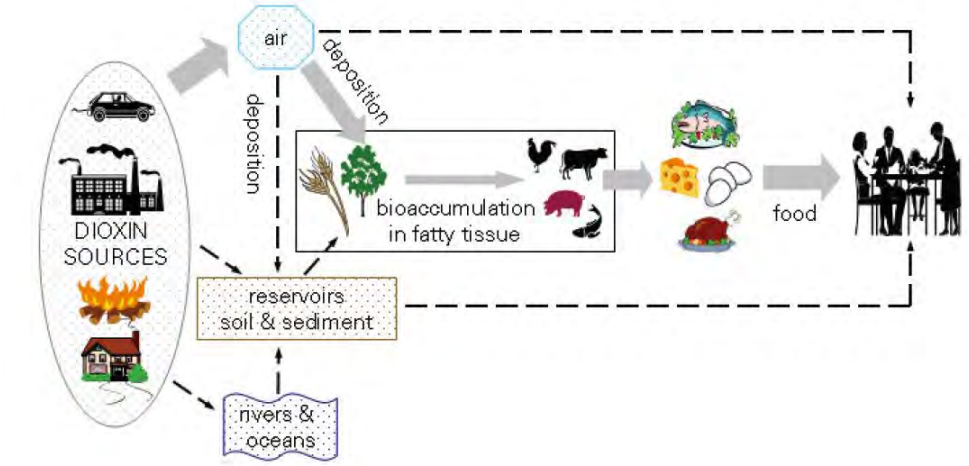
- Dioxin-like compounds are mainly unintended by–products of combustion processes. It has been estimated that 96 per cent of dioxin-like compounds in the environment are from emissions to air.
- The new inventory estimates that total emissions to air in Australia are between 160–1,788 g TEQ/year with 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.
- Dioxins from motor vehicles account for less than 2 per cent of total dioxins emissions to air.
- Disposal and landfilling is 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 2011b) 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 that has been shown by 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 2011b) 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 has 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 US EPA 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 dioxins 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%.

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 1999; CCME 1999; JECFA 2011; UK EA 2009b; 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 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 (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 presented by USEPA (USEPA 1995).

ATSDR (ATSDR 1999) also noted no information was identified for absorption of methylmercury via dermal absorption. The UK (UK EA 2009b) 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 or 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 1999).

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 1999).

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 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 (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.

<u>Exposure to methylmercury:</u>

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 2009b). 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 2009b)
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 (UK EA 2009b) 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 (NHMRC 1999a) of intakes associated with amalgam fillings in Australian children and adults (based on average number of fillings of 0.5 and 8 respectively) provides an 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 2009b), 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/m^3 (rural) to 10 to 20 ng/m^3 (urban areas) with no indication on speciation between elemental an inorganic. Where elemental mercury is measured levels are low approximately 1 to 3 ng/m^3 (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 2004a) 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 2004a). 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 (UK EA 2009b) 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 2011b)	TDI = 0.002 mg/kg/day	MfE adopted the TDI from the RIVM evaluation (noted below), which is also consistent with eh TDI adopted in the derivation of the WHO drinking water guideline.
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.

Source	Value	Basis/Comments
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 rates 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 2009b)	TDI = 0.002 mg/kg/day TC = 0.0002 mg/m^3	TDI referenced from the WHO (WHO 2000b) 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 1999)	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/m^3**	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 1999)	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 occupation al 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 2009b)	TC = 0.0002 mg/m^3	TDI referenced from the WHO (WHO 2000b) 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/m3 for CNS effects from a number of occupation 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 2009b) 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 2004a). 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 2004a) 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 2004a)	**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 (WHO 2000c) 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 2009b)	PTWI = 0.0016 mg/kg/week (PTDI = 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.
ATSDR (ATSDR 1999)	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 2004a) 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/m^3 (WHO 2003) – note this is for elemental mercury
- Background intakes from other sources (as % of TRV):
    - BI_O = 60% for oral and dermal intakes
    - BIi = 0% for inhalation
- Uptake into edible produce may occur and should be evaluated where relevant.

Elemental mercury:

- Inhalation TRV (TRV_I) = 0.0002 mg/m^3 (WHO 2003)
- Background intakes from other sources (as % of TRV): negligible

Methyl mercury:

- Oral TRV (TRV_O) = 0.00023 mg/kg/day (WHO 2004a) for all routes of exposure
- Dermal absorption factor (DAF) = 0.001 (0r 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 2005a; UK EA 2009a; 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 2005a). 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 2005a) and UK (UK EA 2009a) 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 2005a) 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/m^3 (average of 3.5 ng/m^3). Typical background concentrations in air have been reported by (UK EA 2009a) to be from 0.3 to 4.5 ng/m^3, 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 2009a):

> *"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 2012c) 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 (UK EA 2009a)noted that while not all expert groups (WHO, US EPA, 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 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

Source	Value	Basis/Comments
		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 2009a))	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 2005a)	No oral MRL derived	
US EPA (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 (UK EA 2009a) 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 (WHO 2000b), nickel sulfate (TCEQ 2017c) 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 2009a) 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 2009a), 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 μg/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 2017c)	Acute ReV = 1.1 μg/m^3 Chronic ReV = 0.23 μg/m^3 Carcinogenic ReV = 0.059 μg/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 (μg/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 μg/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 μg/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 μg/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 2009a)	TC = 0.02 μg/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 μg/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.
TERA (TERA 1999)	RfC = 0.2 μg/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 2005a)	Inhalation MRL = 0.09 μg/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.
US EPA IRIS (USEPA)	GV = 0.04 μg/m^3	Review by the US EPA (last reviewed in 1991) established a range of air guideline values for nickel based on a non-threshold approach with a unit

Source	Value	Basis/Comments
		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 2017c) 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 mg/kg/day (WHO 2017) for oral and dermal routes of exposure
- Inhalation TRV (TRVi) = 0.000059 mg/m^3 (TCEQ 2017c)
- 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 has been assessed on the basis of the inhalation guidance available from the USEPA and recommended for use in the ASC NEPM and enHealth (enHealth 2012b; NEPC 1999 amended 2013d; 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:

$$\text{Inhalation Exposure Concentration} = C_{air} \times \frac{ET \times FI \times RF \times EF \times ED}{AT} \quad (mg/m^3)$$

Table C1: Inhalation exposure assumptions

Parameter		Value adopted	Basis
Ca	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 2013d)
ET	Exposure time (dependant 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 Assume workers exposed all day, every workday
EF	Exposure frequency (days/year)	Residents = 365 days Workers = 240 days	
ED	Exposure duration (years)	Residents = 35 years Workers = 30 years	Duration of residency and at a workplace as per enHealth (enHealth 2012a)
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 2012b) guidance

C3 Multiple pathway exposures

C3.1 Ingestion and dermal absorption

Chemical substances that are deposited on the ground have the potential to be ingested either directly through accidental consumption of dirt 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 2012b; USEPA 1989). This approach is presented in the following equation, and parameters adopted in this assessment are presented in **Table C2**:

$$\text{Daily Chemical Intake}_{\text{Ingestion}} = C_M \bullet \frac{IR_M \bullet FI \bullet B \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

Chemical substances 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**:

$$\text{Daily Chemical Intake}_{\text{Dermal}} = C_M \bullet \frac{SA \bullet AF \bullet ABSd \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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 particulates to soil (refer to **Section 4.2**), adopting the maximum from all residential receptors		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 mg/day	50 mg/day	Ingestion rate of outdoor soil and dust (tracked or deposited indoors) as per enHealth (enHealth 2012a) and ASC NEPM
	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	Total fruit and vegetable intakes per day as per ASC NEPM (NEPC 1999 amended 2013d)
	Eggs (kg/day)	0.006 kg/day	0.014 kg/day	Ingestion rate of eggs per day as per enHealth (enHealth 2012a), also consistent with P90 intakes from FSANZ (FSANZ 2017)
	Beef (kg/day)	0.085	0.16 kg/day	Ingestion rate for adults aged 19 years and older (enHealth 2012a), 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)

Parameter		Value adopted		Basis
		Young children	Adults	
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	200%	200%	Assume higher intake of home-produced eggs in rural areas (SAHC 1998)
	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)	2700	6300	Exposed skin surface area relevant to adults as per ASC NEPM (NEPC 1999 amended 2013d)
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 2013d)
ABSd	Dermal absorption fraction (unitless)	Chemical specific		Refer to **Tables B1 and B2**
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 enHealth (enHealth 2012a) and ASC NEPM (NEPC 1999 amended 2013d)
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 2012a) and split between young children and adults as per ASC NEPM (NEPC 1999 amended 2013d)
AT	Averaging time (days)	Threshold = ED x 365 days/year Non-threshold = 70 years x 365 days/year		As per enHealth (enHealth 2012b) 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**.

$$C_s = \frac{DR \bullet [1-e^{-k \bullet t}]}{d \bullet \rho \bullet k} \bullet 1000 \qquad \text{(mg/kg)}$$

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 maximum deposition rate for residential receptors		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**:

$$C_p = \frac{DR \bullet F \bullet [1-e^{-k \bullet t}]}{Y \bullet k} \qquad \text{(mg/kg plant – wet weight)}$$

The potential uptake of persistent chemical substances into edible crops via the roots can be estimated using the following equation (OEHHA 2015; USEPA 2005b), with the parameters and assumptions adopted outlined in **Table B6:**

$$C_{rp}=C_s \bullet RUF \qquad \text{(mg/kg plant – wet weight)}$$

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)
Cs	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 et al. 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**.

C4 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 2012b; NEPC 1999 amended 2013d; 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 of the assumptions adopted in this assessment:

$$\text{Exposure Concentration} = C_a \bullet \frac{ET \bullet EF \bullet ED}{AT} \quad (mg/m^3)$$

Table C1: Inhalation exposure assumptions

Parameter		Value adopted	Basis
Ca	Concentration of chemical substance in air (mg/m^3)	Modelled from facility, adopting the maximum predicted anywhere (i.e. grid maximum) and the maximum from all discrete receptors	Calculations undertaken on the basis of the maximum predicted impacts
ET	Exposure time (dependent on activity) (hours/day)	Industrial workers: 8 hours/day Residents: 24 hours/day	Assume someone is exposed at the maximum location all day, every day of the year, and workers are exposed every work day
EF	Exposure frequency (days/year)	Industrial workers: 240 days/year Residents: 365 days/year	
ED	Exposure duration (years)	Industrial workers: 30 years Residents: 35 years	Duration of work and residency as per enHealth (enHealth 2012a)
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 2012b) guidance

Threshold Risk

The quantification of potential exposure and risks to human health associated with the presence of key chemicals in air (or other media) involves comparing the estimated exposure concentration with the threshold concentration adopted from relevant sources of toxicity reference values as listed in **Table B2**. The calculated ratio is termed a Hazard or Risk Index (HI/RI), which is the sum of all ratios (termed Hazard or Risk Quotients [HQ/RQ]) over all relevant pathways of exposure.

These are calculated using the following equations for inhalation exposures:

$$\text{Hazard or Risk Quotient (HQ or RQ)(inhalation)} = \frac{\text{Exposure Concentration in air (adjusted for site-specific assumptions)}}{\text{(TRV-background)}}$$

$$\text{Hazard or Risk Index (HI or RI)} = \sum_{\text{all chemicals and pathways}} \text{H(R)Qs}$$

The interpretation of an acceptable HI/RI needs to recognise an inherent degree of conservatism that is built into the establishment of appropriate guideline (threshold) values (using many uncertainty factors) and in the way exposures are calculated.

Hence, in reviewing and interpreting the calculated HI/RI, the following is noted:

- A HI/RI less than or equal to a value of 1 (where intake or exposure is less than or equal to the threshold) represents no cause for concern (as per risk assessment industry practice, supported by protocols outlined in enHealth and the ASC NEPM (enHealth 2012b; NEPC 1999 amended 2013a, 1999 amended 2013e); and
- A HI/RI greater than 1 requires further consideration within the context of the assessment undertaken, particularly with respect to the level of conservatism in the assumptions adopted for the quantification of exposure and the level of uncertainty within the toxicity (threshold) values adopted (enHealth 2012b; NEPC 1999 amended 2013a, 1999 amended 2013e).

Non-Threshold Risk

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

Carcinogenic Risk (inhalation)=Exposure concentration (adjusted for site-specific assumptions)*Inhalation Unit Risk

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

enHealth and ASC NEPM define an acceptable non-threshold carcinogenic risk (as a sum over all non-threshold chemicals and exposure pathways) as equal to or less than $1x10^{-5}$. On this basis, a total Target Risk value of $>1 \times 10^{-5}$ has been adopted as indicating conditions that would warrant further assessment. Risk values $\leq 1 \times 10^{-5}$ are considered to be representative of acceptable risks (enHealth 2012b; NEPC 1999 amended 2013a, 1999 amended 2013e).

The risk quotients and index are provided in the spreadsheet pages in **Appendix D** and they are summarised in **Section 5.4.3**.

C5 Multiple pathway exposures

C5.1 Ingestion and dermal absorption

Emissions from the stack contain gases and particles. Chemicals like the metals are usually attached to the particles. The particles can settle out of the air onto the ground over time or due to rain. Such particles mix with the soil and so these chemicals that are deposited on the ground via the particles have the potential to be ingested by people either directly (through accidental/incidental consumption of dirt) or indirectly (through eating food grown or raised (fruits, vegetables, eggs, milk, meat) at a property that might be impacted by the deposited dust).

The assessment of the potential ingestion of chemical substances has been undertaken using the approach presented by enHealth and the USEPA (enHealth 2012b; USEPA 1989). This approach is presented in the following equation, and parameters adopted in this assessment are presented in **Table C2**:

$$\text{Daily Chemical Intake}_{\text{Ingestion}} = C_M \bullet \frac{IR_M \bullet FI \bullet B \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

where:

TRV	= Toxicity reference value relevant for the chemical (mg/kg/day)
BI_O	= Background intake (% of TRV)
IRs	= Ingestion rate of soil (mg/day)
B	= Bioavailability or absorption of chemical via ingestion (unitless) (assumed to be 100%)

CF = Conversion factor of $1x10^{-6}$ to convert mg to kg
EF = Exposure frequency (days/year)
ED = Exposure duration (years)
BW = Body weight (kg)
AT = Averaging time for threshold exposures, (=ED x 365 days)

Chemicals attached to particles that are deposited on the ground also have the potential to be absorbed through the skin when skin comes in contact with soil or dust (into which these particles have mixed).

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**:

$$\text{Daily Chemical Intake}_{Dermal} = C_M \bullet \frac{SA \bullet AF \bullet ABSd \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

where:
TRV = Toxicity reference value relevant for the chemical (mg/kg/day)
BI_O = Background intake (% of TRV)
SA = Surface area of skin exposed (cm^2)
AF = adherence factor (mg soil/cm^2 skin)
ABSd = chemical specific factor for absorption through skin
B = Bioavailability or absorption of chemical via ingestion (unitless) (assumed to be 100%)
CF = Conversion factor of $1x10^{-6}$ to convert mg to kg
EF = Exposure frequency (days/year)
ED = Exposure duration (years)
BW = Body weight (kg)
AT = Averaging time for threshold exposures, (=ED x 365 days)

Table C2: Exposure parameter assumptions

Parameter		Value adopted		Basis
		Young children	Adults	
C_M	Concentration of chemical substance in media or relevance (soil, fruit and vegetables or eggs) (mg/kg)	Modelled based on deposition of particulates to soil, adopting the maximum from all residential receptors		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 mg/day	50 mg/day	Ingestion rate of outdoor soil and dust (tracked or deposited indoors) as per enHealth (enHealth 2012a)
	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	Total fruit and vegetable intakes per day as per ASC NEPM (NEPC 1999 amended 2013d)
	Eggs (kg/day)	0.013 kg/day	0.023 kg/day	Ingestion rate of eggs per day – mean consumption for consumers 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

Parameter		Value adopted		Basis
		Young children	Adults	
	Fruit and vegetables	35%	35%	Rate assumed for rural area (higher than the default of 10% for urban areas)
	Eggs	200%	200%	Assume higher intake of home-produced eggs in rural areas (SAHC 1998)
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)	2700	6300	Exposed skin surface area relevant to adults as per ASC NEPM (NEPC 1999 amended 2013d)
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 2013d)
ABSd	Dermal absorption fraction (unitless)	Chemical specific		Refer to **Table B2**
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	15	70	As per enHealth (enHealth 2012a) and ASC NEPM (NEPC 1999 amended 2013d)
EF	Exposure frequency (days/year)	365	365	Assume residents exposed every day
ED	Exposure duration (years)	6 years	29	Duration of residency as per enHealth (enHealth 2012a) and split between young children and adults as per ASC NEPM (NEPC 1999 amended 2013d)
AT	Averaging time (days)	Threshold = ED x 365 days/year Non-threshold = 70 years x 365 days/year		As per enHealth (enHealth 2012b) guidance

C5.2 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**.

$$C_s = \frac{DR \bullet [1-e^{-k \bullet t}]}{d \bullet \rho \bullet k} \bullet 1000 \qquad \text{(mg/kg)}$$

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)	Adopted maximum deposition rate for discrete receptors		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)	1600000	1600000	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**:

$$C_p = \frac{DR \bullet F \bullet [1-e^{-k \bullet t}]}{Y \bullet k} \qquad \text{(mg/kg plant – wet weight)}$$

The potential uptake of persistent chemical substances into edible crops via the roots can be estimated using the following equation (OEHHA 2015; USEPA 2005b), with the parameters and assumptions adopted outlined in **Table C4:**

$$C_{rp} = C_s \bullet RUF \qquad \text{(mg/kg plant – wet weight)}$$

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)	Adopted maximum deposition rate for discrete receptors	Relevant to areas where multi-pathway exposures may occur
F	Fraction for the surface area of plant (unitless)	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

Parameter		Value adopted	Basis
			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	0.0024 kg/day	Based on data from OEHHA 2015 (2% total produce intakes from soil)
	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)
Cs	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		

Parameter		Value adopted	Basis
	Eggs	Chemical specific	Transfer factors adopted from OEHHA (2015)
	Beef	Chemical specific	Transfer factors adopted from OEHHA (2015) and RAIS
	Milk	Chemical specific	Transfer factors adopted from OEHHA (2015) and RAIS

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**:

$$C_W = \frac{DM}{VR \times Kd \times \rho}$$

$$VR = \frac{R \times Area \times Rc}{1000}$$

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 for accidental release (mg/m^2/year)	Modelled in the Air Quality Assessment for each receptor	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)	663.2	Average rainfall at Mudgee Airport for all years of records (1994 – 2019). No first flush devise 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)
ρ	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

Normal operations – assuming emissions at the regulatory limits

Acute (1 Hour Average)

Predicted ground level concentrations and screening assessment - acute exposures - EPA VIC APACs

		Air Concentration (mg/m^3)											Calculated RI/HI									
COPC	Acute air guideline (mg/m3)	Grid Maximum Off-site	Maximum for residential locations	Maximum for commercial/ industrial locations	Maximum for other locations	Zone 1	Zone 2	Zone 3	Zone 4	Zone 5	Zone 6	Zone 7	Grid Maximum Off-site	Maximum from residential receptor	Maximum from commercial/ industrial receptors	Maximum for other locations	Zone 1	Zone 2	Zone 3	Zone 4	Zone 5	Zone 6	Zone 7
Hydrogen chloride (HCl)	2.1	0.01583285	0.00950434	0.00708532	0.00356349	0.00210236	0.00179424	0.00264332	0.00577677	0.00604511	0.00450672	0.00661493	7.5E-03	4.5E-03	3.4E-03	1.7E-03	1.0E-03	8.5E-04	1.3E-03	2.8E-03	2.9E-03	2.1E-03	3.1E-03
Hydrogen fluoride (HF)	0.06	0.00119699	0.00071854	0.00053566	0.00026940	0.00015894	0.00013565	0.00019984	0.00043673	0.00045702	0.00034071	0.00050010	2.0E-02	1.2E-02	8.9E-03	4.5E-03	2.6E-03	2.3E-03	3.3E-03	7.3E-03	7.6E-03	5.7E-03	8.3E-03
Ammonia	3.2	0.00480000	0.00283000	0.00215000	0.00108000	0.00064000	0.00054000	0.00080000	0.00175000	0.00183000	0.00137000	0.00200000	1.5E-03	8.8E-04	6.7E-04	3.4E-04	2.0E-04	1.7E-04	2.5E-04	5.5E-04	5.7E-04	4.3E-04	6.3E-04
Cadmium (Cd)	0.018	0.00000566	0.00000340	0.00000253	0.00000127	0.00000075	0.00000064	0.00000094	0.00000206	0.00000216	0.00000161	0.00000236	3.1E-04	1.9E-04	1.4E-04	7.1E-05	4.2E-05	3.6E-05	5.2E-05	1.1E-04	1.2E-04	8.9E-05	1.3E-04
Mercury (Hg)	No APAC	0.00009580	0.00000575	0.00000429	0.00000216	0.00000130	0.00000109	0.00000148	0.00000349	0.00000366	0.00000273	0.00000400											
Antimony (Sb)	No APAC	0.00001012	0.00000608	0.00000453	0.00000228	0.00000134	0.00000115	0.00000169	0.00000369	0.00000386	0.00000288	0.00000423											
Arsenic (As)	0.0099	0.00000318	0.00000191	0.00000142	0.00000072	0.00000042	0.00000036	0.00000053	0.00000116	0.00000121	0.00000090	0.00000133	3.2E-04	1.9E-04	1.4E-04	7.3E-05	4.2E-05	3.6E-05	5.4E-05	1.2E-04	1.2E-04	9.1E-05	1.3E-04
Chromium (Cr VI assumed)	0.0013	0.00001202	0.00000722	0.00000538	0.00000271	0.00000160	0.00000136	0.00000201	0.00000439	0.00000459	0.00000342	0.00000502	9.2E-03	5.6E-03	4.1E-03	2.1E-03	1.2E-03	1.0E-03	1.5E-03	3.4E-03	3.5E-03	2.6E-03	3.9E-03
Cobalt (Co)	No APAC	0.00000359	0.00000212	0.00000161	0.00000081	0.00000048	0.00000041	0.00000060	0.00000131	0.00000137	0.00000102	0.00000150											
Copper (Cu)	0.1	0.00001496	0.00000898	0.00000670	0.00000337	0.00000199	0.00000170	0.00000250	0.00000546	0.00000571	0.00000426	0.00000625	1.5E-04	9.0E-05	6.7E-05	3.4E-05	2.0E-05	1.7E-05	2.5E-05	5.5E-05	5.7E-05	4.3E-05	6.2E-05
Manganese (Mn)	0.0091	0.00002019	0.00001212	0.00000903	0.00000454	0.00000268	0.00000229	0.00000337	0.00000736	0.00000771	0.00000575	0.00000843	2.2E-03	1.3E-03	9.9E-04	5.0E-04	2.9E-04	2.5E-04	3.7E-04	8.1E-04	8.5E-04	6.3E-04	9.3E-04
Nickel (Ni)	0.0002	0.00000968	0.00000581	0.00000433	0.00000218	0.00000129	0.00000110	0.00000162	0.00000353	0.00000370	0.00000276	0.00000405	4.8E-02	2.9E-02	2.2E-02	1.1E-02	6.4E-03	5.5E-03	8.1E-03	1.8E-02	1.8E-02	1.4E-02	2.0E-02
Vanadium (V)	No APAC	0.00000180	0.00000108	0.00000080	0.00000040	0.00000024	0.00000020	0.00000030	0.00000066	0.00000069	0.00000051	0.00000075											
Benzene	0.58	0.00721000	0.00432000	0.00322000	0.00162000	0.00096000	0.00082000	0.00120000	0.00263000	0.00275000	0.00205000	0.00301000	1.2E-02	7.4E-03	5.6E-03	2.8E-03	1.7E-03	1.4E-03	2.1E-03	4.5E-03	4.7E-03	3.5E-03	5.2E-03
Formaldehyde	0.1	0.00826859	0.00496358	0.00370026	0.00186101	0.00146392	0.00124937	0.00184061	0.00402251	0.00420936	0.00313815	0.00460614	8.3E-02	5.0E-02	3.7E-02	1.9E-02	1.5E-02	1.2E-02	1.8E-02	4.0E-02	4.2E-02	3.1E-02	4.6E-02
													1.7E-01	**1.0E-01**	**7.7E-02**	**3.9E-02**	**2.7E-02**	**2.3E-02**	**3.3E-02**	**7.3E-02**	**7.6E-02**	**5.7E-02**	**8.4E-02**

Predicted ground level concentrations and screening assessment - acute exposures - preferred guidelines

		Air Concentration (mg/m^3)											Calculated RI/HI									
COPC	Acute air guideline (mg/m3)	Grid Maximum Off-site	Maximum for residential locations	Maximum for commercial/ industrial locations	Maximum for other locations	Zone 1	Zone 2	Zone 3	Zone 4	Zone 5	Zone 6	Zone 7	Grid Maximum Off-site	Maximum from residential receptor	Maximum from commercial/ industrial receptors	Maximum for other locations	Zone 1	Zone 2	Zone 3	Zone 4	Zone 5	Zone 6	Zone 7
Hydrogen chloride (HCl)	0.66	0.01583285	0.00950434	0.00708532	0.00356349	0.00210236	0.00179424	0.00264332	0.00577677	0.00604511	0.00450672	0.00661493	2.4E-02	1.4E-02	1.1E-02	5.4E-03	3.2E-03	2.7E-03	4.0E-03	8.8E-03	9.2E-03	6.8E-03	1.0E-02
Hydrogen fluoride (HF)	0.06	0.00119699	0.00071854	0.00053566	0.00026940	0.00015894	0.00013565	0.00019984	0.00043673	0.00045702	0.00034071	0.00050010	2.0E-02	1.2E-02	8.9E-03	4.5E-03	2.6E-03	2.3E-03	3.3E-03	7.3E-03	7.6E-03	5.7E-03	8.3E-03
Ammonia	0.59	0.00480000	0.00283000	0.00215000	0.00108000	0.00064000	0.00054000	0.00080000	0.00175000	0.00183000	0.00137000	0.00200000	8.1E-03	4.8E-03	3.6E-03	1.8E-03	1.1E-03	9.2E-04	1.4E-03	3.0E-03	3.1E-03	2.3E-03	3.4E-03
Cadmium (Cd)	0.018	0.00000566	0.00000340	0.00000253	0.00000127	0.00000075	0.00000064	0.00000094	0.00000206	0.00000216	0.00000161	0.00000236	3.1E-04	1.9E-04	1.4E-04	7.1E-05	4.2E-05	3.6E-05	5.2E-05	1.1E-04	1.2E-04	8.9E-05	1.3E-04
Mercury (Hg)	0.0006	0.00009580	0.00000575	0.00000429	0.00000216	0.00000130	0.00000109	0.00000148	0.00000349	0.00000366	0.00000273	0.00000400	1.6E-01	9.6E-03	7.2E-03	3.6E-03	2.2E-03	1.8E-03	2.5E-03	5.8E-03	6.1E-03	4.6E-03	6.7E-03
Antimony (Sb)	0.001	0.00001012	0.00000608	0.00000453	0.00000228	0.00000134	0.00000115	0.00000169	0.00000369	0.00000386	0.00000288	0.00000423	1.0E-02	6.1E-03	4.5E-03	2.3E-03	1.3E-03	1.2E-03	1.7E-03	3.7E-03	3.9E-03	2.9E-03	4.2E-03
Arsenic (As)	0.0099	0.00000318	0.00000191	0.00000142	0.00000072	0.00000042	0.00000036	0.00000053	0.00000116	0.00000121	0.00000090	0.00000133	3.2E-04	1.9E-04	1.4E-04	7.3E-05	4.2E-05	3.6E-05	5.4E-05	1.2E-04	1.2E-04	9.1E-05	1.3E-04
Chromium (Cr VI assumed)	0.0013	0.00001202	0.00000722	0.00000538	0.00000271	0.00000160	0.00000136	0.00000201	0.00000439	0.00000459	0.00000342	0.00000502	9.2E-03	5.6E-03	4.1E-03	2.1E-03	1.2E-03	1.0E-03	1.5E-03	3.4E-03	3.5E-03	2.6E-03	3.9E-03
Cobalt (Co)	0.00069	0.00000359	0.00000212	0.00000161	0.00000081	0.00000048	0.00000041	0.00000060	0.00000131	0.00000137	0.00000102	0.00000150	5.2E-03	3.1E-03	2.3E-03	1.2E-03	7.0E-04	5.9E-04	8.7E-04	1.9E-03	2.0E-03	1.5E-03	2.2E-03
Copper (Cu)	0.1	0.00001496	0.00000898	0.00000670	0.00000337	0.00000199	0.00000170	0.00000250	0.00000546	0.00000571	0.00000426	0.00000625	1.5E-04	9.0E-05	6.7E-05	3.4E-05	2.0E-05	1.7E-05	2.5E-05	5.5E-05	5.7E-05	4.3E-05	6.2E-05
Manganese (Mn)	0.0091	0.00002019	0.00001212	0.00000903	0.00000454	0.00000268	0.00000229	0.00000337	0.00000736	0.00000771	0.00000575	0.00000843	2.2E-03	1.3E-03	9.9E-04	5.0E-04	2.9E-04	2.5E-04	3.7E-04	8.1E-04	8.5E-04	6.3E-04	9.3E-04
Nickel (Ni)	0.0011	0.00000968	0.00000581	0.00000433	0.00000218	0.00000129	0.00000110	0.00000162	0.00000353	0.00000370	0.00000276	0.00000405	8.8E-03	5.3E-03	3.9E-03	2.0E-03	1.2E-03	1.0E-03	1.5E-03	3.2E-03	3.4E-03	2.5E-03	3.7E-03
Vanadium (V)	0.03	0.00000180	0.00000108	0.00000080	0.00000040	0.00000024	0.00000020	0.00000030	0.00000066	0.00000069	0.00000051	0.00000075	6.0E-05	3.6E-05	2.7E-05	1.3E-05	8.0E-06	6.7E-06	1.0E-05	2.2E-05	2.3E-05	1.7E-05	2.5E-05
Benzene	0.58	0.00721000	0.00432000	0.00322000	0.00162000	0.00096000	0.00082000	0.00120000	0.00263000	0.00275000	0.00205000	0.00301000	1.2E-02	7.4E-03	5.6E-03	2.8E-03	1.7E-03	1.4E-03	2.1E-03	4.5E-03	4.7E-03	3.5E-03	5.2E-03
Formaldehyde	0.1	0.00826859	0.00496358	0.00370026	0.00186101	0.00146392	0.00124937	0.00184061	0.00402251	0.00420936	0.00313815	0.00460614	8.3E-02	5.0E-02	3.7E-02	1.9E-02	1.5E-02	1.2E-02	1.8E-02	4.0E-02	4.2E-02	3.1E-02	4.6E-02
													3.3E-01	**1.1E-01**	**8.4E-02**	**4.2E-02**	**2.9E-02**	**2.4E-02**	**3.6E-02**	**7.8E-02**	**8.2E-02**	**6.1E-02**	**9.0E-02**

Chronic Exposures

Maximum off-site

Predicted ground level concentrations - chronic exposures - cumulative case

	Air Concentration - annual average ($\mu g/m^3$)	Air Concentration - annual average (mg/m^3)	Exposure pathways							
Key Chemicals	**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)	6.899	0.006899	✓	×	×	×	×	×	×	×
Hydrogen fluoride (HF)	0.388	0.000388	✓	×	×	×	×	×	×	×
Ammonia	0.39141	0.00039141	✓	×	×	×	×	×	×	×
Cadmium (Cd)	0.00046	0.000000460	✓	✓	✓	×	×	✓	×	×
Thallium (Tl)	0.00032	0.000000320	✓	✓	✓	×	×	✓	×	×
Mercury (Hg)	0.00078	0.000000780	✓	✓	✓	×	×	✓	×	×
Antimony (Sb)	0.00083	0.000000830	✓	✓	✓	×	×	✓	×	×
Arsenic (As)	0.00036	0.000000360	✓	✓	✓	×	×	✓	×	×
Lead (Pb)	0.0047	0.000004700	✓	✓	✓	×	×	✓	×	×
Chromium (Cr VI assumed)	0.00157	0.00000157	✓	✓	✓	×	×	✓	×	×
Cobalt (Co)	0.00029	0.000000290	✓	✓	✓	×	×	✓	×	×
Copper (Cu)	0.00103	0.00000103	✓	✓	✓	×	×	✓	×	×
Manganese (Mn)	0.00177	0.00000177	✓	✓	✓	×	×	✓	×	×
Nickel (Ni)	0.00142	0.00000142	✓	✓	✓	×	×	✓	×	×
Vanadium (V)	0.00015	0.000000150	✓	✓	✓	×	×	✓	×	×
Dioxins and furans	2.35E-09	2.35E-12	✓	✓	✓	×	×	✓	×	×
Benzene	1.17379	0.00117379	✓	×	×	×	×	×	×	×
Formaldehyde	0.39141	0.00039141	✓	×	×	×	×	×	×	×
Polycyclic aromatic hydrocarbons (PAHs)	0.000008	0.000000008	✓	✓	✓	×	×	✓	×	×

	Deposition Rate - annual average ($ng/m^2/year$)	Deposition Rate - annual average ($mg/m^2/year$)
Key Chemicals	**Maximum Off-site**	**Maximum Off-site**
Hydrogen chloride (HCl)	NR	NR
Hydrogen fluoride (HF)	NR	NR
Ammonia	NR	NR
Cadmium (Cd)	4729	0.004729
Thallium (Tl)	3274	0.003274
Mercury (Hg)	8003	0.008003
Antimony (Sb)	8458	0.008458
Arsenic (As)	3701	0.003701
Lead (Pb)	48202	0.048202
Chromium (Cr VI assumed)	12096	0.012096
Cobalt (Co)	3001	0.003001
Copper (Cu)	10595	0.010595
Manganese (Mn)	18099	0.018099
Nickel (Ni)	14551	0.014551
Vanadium (V)	1501	0.001501
Dioxins and furans	NR	1.362E-07
Polycyclic aromatic hydrocarbons (PAHs)	NR	1.261E-02

Inhalation - gases and particulates
Cumulative maximum off-site for this facility plus background plus brickworks

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * EF * ED}{AT}$$ (mg/m^3) for gases

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * DRF * EF * ED}{AT}$$ (mg/m^3) 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 (NEPM 1999 amended 2013) - NA for gasses
Exposure Frequency - normal conditions (EF, days/yr)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	35	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	306600	US EPA 2009

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	6.9E-03	3.4E-03	6.9E-03	--		0.265	52%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	3.9E-04	1.9E-04	3.9E-04	--		0.0134	3%
Ammonia		3.2E-01	0%	3.2E-01	3.9E-04	2.0E-04	3.9E-04	--		0.00122	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	4.6E-07	8.6E-08	1.7E-07	--		0.0431	9%
Thallium (Tl)		7.0E-04	10%	6.3E-04	3.2E-07	6.0E-08	1.2E-07	--		0.000190	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	7.8E-07	1.5E-07	2.9E-07	--		0.00146	0%
Antimony (Sb)		3.0E-04	0%	3.0E-04	8.3E-07	1.6E-07	3.1E-07	--		0.00104	0%
Arsenic (As)		6.7E-05	0%	6.7E-05	3.6E-07	6.8E-08	1.4E-07	--		0.00201	0%
Lead (Pb)		5.0E-04	0%	5.0E-04	4.7E-06	8.8E-07	1.8E-06	--		0.00353	1%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	1.6E-06	2.9E-07	5.9E-07	--		0.137	27%
Cobalt (Co)		1.0E-04	0%	1.0E-04	2.9E-07	5.4E-08	1.1E-07	--		0.00109	0%
Copper (Cu)		4.9E-01	60%	2.0E-01	1.0E-06	1.9E-07	3.9E-07	--		0.00000197	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	1.8E-06	3.3E-07	6.6E-07	--		0.00553	1%
Nickel (Ni)		5.9E-05	10%	5.3E-05	1.4E-06	2.7E-07	5.3E-07	--		0.0100	2%
Vanadium (V)		1.0E-04	0%	1.0E-04	1.5E-07	2.8E-08	5.6E-08	--		0.000563	0%
Dioxins and furans		8.1E-09	54%	3.7E-09	2.4E-12	4.4E-13	8.8E-13	--		0.000238	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	1.2E-03	2.2E-04	4.4E-04	1.3E-6	100%	0.0163	3%
Formaldehyde		7.0E-03	0%	7.0E-03	3.9E-04	7.3E-05	1.5E-04	--		0.0210	4%
PAHs	6.0E-01				8.0E-09	1.5E-09	3.0E-09	9.0E-10	0%	--	
							TOTAL	**1.3E-06**		**0.507**	

Inhalation - gases and particulates
Grid maximum for this facility plus background plus brickworks

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * EF * ED}{AT}$$ (mg/m^3) for gases

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * DRF * EF * ED}{AT}$$ (mg/m^3) 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 (NEPM 1999 amended 2013) - NA for gasses
Exposure Frequency - normal conditions (EF, days/yr)	240	Days at work (NEPM)
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	262800	US EPA 2009

	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
Key Chemical	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	6.9E-03	8.1E-04	1.9E-03	--		0.0727	49%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	3.9E-04	4.6E-05	1.1E-04	--		0.00367	2%
Ammonia		3.2E-01	0%	3.2E-01	3.9E-04	4.6E-05	1.1E-04	--		0.000335	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	4.6E-07	2.0E-08	4.7E-08	--		0.0118	8%
Thallium (Tl)		7.0E-04	10%	6.3E-04	3.2E-07	1.4E-08	3.3E-08	--		0.0000522	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	7.8E-07	3.4E-08	8.0E-08	--		0.000401	0%
Antimony (Sb)		3.0E-04	0%	3.0E-04	8.3E-07	3.7E-08	8.5E-08	--		0.000284	0%
Arsenic (As)		6.7E-05	0%	6.7E-05	3.6E-07	1.6E-08	3.7E-08	--		0.000552	0%
Lead (Pb)		5.0E-04	0%	5.0E-04	4.7E-06	2.1E-07	4.8E-07	--		0.000966	1%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	1.6E-06	6.9E-08	1.6E-07	--		0.0375	25%
Cobalt (Co)		1.0E-04	0%	1.0E-04	2.9E-07	1.3E-08	3.0E-08	--		0.000298	0%
Copper (Cu)		4.9E-01	60%	2.0E-01	1.0E-06	4.5E-08	1.1E-07	--		0.000000540	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	1.8E-06	7.8E-08	1.8E-07	--		0.00152	1%
Nickel (Ni)		5.9E-05	10%	5.3E-05	1.4E-06	6.3E-08	1.5E-07	--		0.00275	2%
Vanadium (V)		1.0E-04	0%	1.0E-04	1.5E-07	6.6E-09	1.5E-08	--		0.000154	0%
Dioxins and furans		8.1E-09	54%	3.7E-09	2.4E-12	1.0E-13	2.4E-13	--		0.0000652	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	1.2E-03	1.4E-04	3.2E-04	8.3E-7	100%	0.0119	8%
Formaldehyde		7.0E-03	0%	7.0E-03	3.9E-04	4.6E-05	1.1E-04	--		0.0153	10%
PAHs	6.0E-01				8.0E-09	9.4E-10	2.2E-09	5.6E-10	0%	--	
							TOTAL	8.3E-07		0.148	

Predicted ground level concentrations - chronic exposures - incremental case

	Air Concentration - annual average ($\mu g/m^3$)	Air Concentration - annual average (mg/m^3)	Exposure pathways							
Key Chemicals	**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)	0.23476	0.00023476	✓	×	×	×	×	×	×	×
Hydrogen fluoride (HF)	0.03914	0.00003914	✓	×	×	×	×	×	×	×
Ammonia	0.39141	0.00039141	✓	×	×	×	×	×	×	×
Cadmium (Cd)	0.00046	0.000000460	✓	×	×	×	×	×	×	×
Thallium (Tl)	0.00032	0.000000320	✓	×	×	×	×	×	×	×
Mercury (Hg)	0.00078	0.000000780	✓	×	×	×	×	×	×	×
Antimony (Sb)	0.00083	0.000000830	✓	×	×	×	×	×	×	×
Arsenic (As)	0.00036	0.000000360	✓	×	×	×	×	×	×	×
Lead (Pb)	0.0047	0.000004700	✓	×	×	×	×	×	×	×
Chromium (Cr VI assumed)	0.00118	0.00000118	✓	×	×	×	×	×	×	×
Cobalt (Co)	0.00029	0.000000290	✓	×	×	×	×	×	×	×
Copper (Cu)	0.00103	0.00000103	✓	×	×	×	×	×	×	×
Manganese (Mn)	0.00177	0.00000177	✓	×	×	×	×	×	×	×
Nickel (Ni)	0.00142	0.00000142	✓	×	×	×	×	×	×	×
Vanadium (V)	0.00015	0.000000150	✓	×	×	×	×	×	×	×
Dioxins and furans	2.35E-09	2.35E-12	✓	×	×	×	×	×	×	×
Benzene	1.17379	0.00117379	✓	×	×	×	×	×	×	×
Formaldehyde	0.39141	0.00039141	✓	×	×	×	×	×	×	×
Polycyclic aromatic hydrocarbons (PAHs)	0.000008	0.000000008	✓	×	×	×	×	×	×	×

	Deposition Rate - annual average ($ng/m^2/year$)	Deposition Rate - annual average ($mg/m^2/year$)
Key Chemicals	**Maximum Off-site**	**Maximum Off-site**
Hydrogen chloride (HCl)	NR	NR
Hydrogen fluoride (HF)	NR	NR
Ammonia	NR	NR
Cadmium (Cd)	4729	0.004729
Thallium (Tl)	3274	0.003274
Mercury (Hg)	8003	0.008003
Antimony (Sb)	8458	0.008458
Arsenic (As)	3701	0.003701
Lead (Pb)	48202	0.048202
Chromium (Cr VI assumed)	12096	0.012096
Cobalt (Co)	3001	0.003001
Copper (Cu)	10595	0.010595
Manganese (Mn)	18099	0.018099
Nickel (Ni)	14551	0.014551
Vanadium (V)	1501	0.001501
Dioxins and furans	NR	1.362E-07
Polycyclic aromatic hydrocarbons (PAHs)	NR	1.261E-02

Inhalation - gases and particulates
Incremental maximum off-site

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * EF * ED}{AT}$$ (mg/m^3) for gases

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * DRF * EF * ED}{AT}$$ (mg/m^3) 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 (NEPM 1999 amended 2013) - NA for gasses
Exposure Frequency - normal conditions (EF, days/yr)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	35	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	306600	US EPA 2009

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	2.3E-04	1.2E-04	2.3E-04	--		0.00903	4%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	3.9E-05	2.0E-05	3.9E-05	--		0.00135	1%
Ammonia		3.2E-01	0%	3.2E-01	3.9E-04	2.0E-04	3.9E-04	--		0.00122	1%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	4.6E-07	8.6E-08	1.7E-07	--		0.0431	21%
Thallium (Tl)		7.0E-04	10%	6.3E-04	3.2E-07	6.0E-08	1.2E-07	--		0.000190	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	7.8E-07	1.5E-07	2.9E-07	--		0.00146	1%
Antimony (Sb)		3.0E-04	0%	3.0E-04	8.3E-07	1.6E-07	3.1E-07	--		0.00104	1%
Arsenic (As)		6.7E-05	0%	6.7E-05	3.6E-07	6.8E-08	1.4E-07	--		0.00201	1%
Lead (Pb)		5.0E-04	0%	5.0E-04	4.7E-06	8.8E-07	1.8E-06	--		0.00353	2%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	1.2E-06	2.2E-07	4.4E-07	--		0.103	50%
Cobalt (Co)		1.0E-04	0%	1.0E-04	2.9E-07	5.4E-08	1.1E-07	--		0.00109	1%
Copper (Cu)		4.9E-01	60%	2.0E-01	1.0E-06	1.9E-07	3.9E-07	--		0.00000197	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	1.8E-06	3.3E-07	6.6E-07	--		0.00553	3%
Nickel (Ni)		5.9E-05	10%	5.3E-05	1.4E-06	2.7E-07	5.3E-07	--		0.0100	5%
Vanadium (V)		1.0E-04	0%	1.0E-04	1.5E-07	2.8E-08	5.6E-08	--		0.000563	0%
Dioxins and furans		8.1E-09	54%	3.7E-09	2.4E-12	4.4E-13	8.8E-13	--		0.000238	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	1.2E-03	2.2E-04	4.4E-04	1.3E-6		0.01630	8%
Formaldehyde		7.0E-03		7.0E-03	3.9E-04	7.3E-05	1.5E-04	--		0.02097	10%
PAHs	6.0E-01				8.0E-09	1.5E-09	3.0E-09	9.0E-10		--	
							TOTAL	**1.3E-06**		**0.204**	

Inhalation - gases and particulates
Grid maximum

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * EF * ED}{AT}$$ (mg/m^3) for gases

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * DRF * EF * ED}{AT}$$ (mg/m^3) 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 (NEPM 1999 amended 2013) - NA for gasses
Exposure Frequency - normal conditions (EF, days/yr)	240	Days at work (NEPM)
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	262800	US EPA 2009

	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
Key Chemical	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	2.3E-04	2.8E-05	6.4E-05	--		0.00247	4%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	3.9E-05	4.6E-06	1.1E-05	--		0.000370	1%
Ammonia		3.2E-01	0%	3.2E-01	3.9E-04	4.6E-05	1.1E-04	--		0.000335	1%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	4.6E-07	2.0E-08	4.7E-08	--		0.0118	18%
Thallium (Tl)		7.0E-04	10%	6.3E-04	3.2E-07	1.4E-08	3.3E-08	--		0.0000522	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	7.8E-07	3.4E-08	8.0E-08	--		0.000401	1%
Antimony (Sb)		3.0E-04	0%	3.0E-04	8.3E-07	3.7E-08	8.5E-08	--		0.000284	0%
Arsenic (As)		6.7E-05	0%	6.7E-05	3.6E-07	1.6E-08	3.7E-08	--		0.000552	1%
Lead (Pb)		5.0E-04	0%	5.0E-04	4.7E-06	2.1E-07	4.8E-07	--		0.000966	1%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	1.2E-06	5.2E-08	1.2E-07	--		0.0282	43%
Cobalt (Co)		1.0E-04	0%	1.0E-04	2.9E-07	1.3E-08	3.0E-08	--		0.000298	0%
Copper (Cu)		4.9E-01	60%	2.0E-01	1.0E-06	4.5E-08	1.1E-07	--		0.000000540	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	1.8E-06	7.8E-08	1.8E-07	--		0.00152	2%
Nickel (Ni)		5.9E-05	10%	5.3E-05	1.4E-06	6.3E-08	1.5E-07	--		0.00275	4%
Vanadium (V)		1.0E-04	0%	1.0E-04	1.5E-07	6.6E-09	1.5E-08	--		0.000154	0%
Dioxins and furans		8.1E-09	54%	3.7E-09	2.4E-12	1.0E-13	2.4E-13	--		0.0000652	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	1.2E-03	1.4E-04	3.2E-04	8.3E-7		0.0119	18%
Formaldehyde		7.0E-03		7.0E-03	3.9E-04	4.6E-05	1.1E-04	--		0.0153	23%
PAHs	6.0E-01				8.0E-09	9.4E-10	2.2E-09	5.6E-10		--	
							TOTAL	**8.3E-07**		**0.0655**	

Calculation of Concentrations in Soil

$$C_s = \frac{DR \bullet \left[1 - e^{-k \bullet t}\right]}{d \bullet \rho \bullet k} \bullet 1000 \quad \textbf{(mg/kg)}$$ ref: Stevens B. (1991)

where:

DR=	Particle deposition rate (mg/m^2/year)
K =	Chemical-specific soil-loss constant (1/year) = ln(2)/T0.5
T0.5 =	Chemical half-life in soil (years)
t =	Accumulation time (years)
d =	Soil mixing depth (m)
ρ =	Soil bulk-density (g/m^3)
1000 =	Conversion from g to kg

General Parameters		Surface (for direct contact)	Depth (for agricultural pathways)	
Soil bulk density (p)	g/m^3	1600000	1600000	Default for fill materials
General mixing depth (d)	m	0.01	0.15	As per OEHHA (2015) guidance
Duration of deposition (T)	years	70	70	As per OEHHA (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	4.7E-03	2.1E-02	1.4E-03
Thallium (Tl)	273973	2.5E-06	3.3E-03	1.4E-02	9.5E-04
Mercury (Hg)	273973	2.5E-06	8.0E-03	3.5E-02	2.3E-03
Antimony (Sb)	273973	2.5E-06	8.5E-03	3.7E-02	2.5E-03
Arsenic (As)	273973	2.5E-06	3.7E-03	1.6E-02	1.1E-03
Lead (Pb)	273973	2.5E-06	4.8E-02	2.1E-01	1.4E-02
Chromium (Cr VI assumed)	273973	2.5E-06	1.2E-02	5.3E-02	3.5E-03
Cobalt (Co)	273973	2.5E-06	3.0E-03	1.3E-02	8.8E-04
Copper (Cu)	273973	2.5E-06	1.1E-02	4.6E-02	3.1E-03
Manganese (Mn)	273973	2.5E-06	1.8E-02	7.9E-02	5.3E-03
Nickel (Ni)	273973	2.5E-06	1.5E-02	6.4E-02	4.2E-03
Vanadium (V)	273973	2.5E-06	1.5E-03	6.6E-03	4.4E-04
Dioxins and furans	15.00	4.6E-02	1.4E-07	1.8E-07	1.2E-08
PAHs	1.18	0.588	1.3E-02	1.3E-03	8.9E-05

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: OEHHA 2015

Exposure to Chemicals via Incidental Ingestion of Soil - Grid Maximum Off-site

$$Daily\ Chemical\ Intake_{IS} = C_S \bullet \frac{IR_S \bullet FI \bullet CF \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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	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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.1E-02	6.1E-09	1.5E-08	--		0.0000462	5%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	1.4E-02	4.2E-09	1.0E-08	--		0.0000568	6%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	3.5E-02	1.0E-08	2.5E-08	--		0.000104	11%
Antimony (Sb)		8.6E-04		8.6E-04	100%	3.7E-02	1.1E-08	2.6E-08	--		0.0000307	3%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.6E-02	4.8E-09	1.2E-08	--		0.0000116	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	2.1E-01	6.2E-08	1.5E-07	--		0.000502	53%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	5.3E-02	1.6E-08	3.8E-08	--		0.0000467	5%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.3E-02	3.9E-09	9.4E-09	--		0.00000837	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	4.6E-02	1.4E-08	3.3E-08	--		0.000000591	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	7.9E-02	2.3E-08	5.7E-08	--		0.000000808	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	6.4E-02	1.9E-08	4.5E-08	--		0.00000947	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	6.6E-03	1.9E-09	4.7E-09	--		0.00000235	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.8E-07	5.2E-14	1.3E-13	--		0.000119	13%
PAHs	2.3E-01				100%	1.3E-03	4.0E-10	9.6E-10	9.2E-11		--	
								TOTAL	**9.2E-11**		**0.000939**	

Exposure to Chemicals via Incidental Ingestion of Soil - Grid Maximum Off-site

$$Daily\ Chemical\ Intake_{IS} = C_S \bullet \frac{IR_S \bullet FI \bullet CF \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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	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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.1E-02	1.2E-08	1.4E-07	--		0.000431	5%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	1.4E-02	8.2E-09	9.5E-08	--		0.000530	6%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	3.5E-02	2.0E-08	2.3E-07	--		0.000973	11%
Antimony (Sb)		8.6E-04		8.6E-04	100%	3.7E-02	2.1E-08	2.5E-07	--		0.000287	3%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.6E-02	9.3E-09	1.1E-07	--		0.000108	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	2.1E-01	1.2E-07	1.4E-06	--		0.00469	53%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	5.3E-02	3.0E-08	3.5E-07	--		0.000436	5%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.3E-02	7.5E-09	8.8E-08	--		0.0000781	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	4.6E-02	2.6E-08	3.1E-07	--		0.00000552	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	7.9E-02	4.5E-08	5.3E-07	--		0.00000754	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	6.4E-02	3.6E-08	4.2E-07	--		0.0000884	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	6.6E-03	3.8E-09	4.4E-08	--		0.0000219	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.8E-07	1.0E-13	1.2E-12	--		0.00112	13%
PAHs	2.3E-01				100%	1.3E-03	7.7E-10	8.9E-09	1.8E-10		--	
								TOTAL	**1.8E-10**		**0.00877**	

Dermal Exposure to Chemicals via Contact with Soil - Grid Maximum Off-site

$$Daily\ Chemical\ Intake_{DS} = C_S \bullet \frac{SA_S \bullet AF \bullet FE \bullet ABS \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	2.1E-02	3.9E-10	9.3E-10	--		0.00000291	1%
Thallium (Tl)		2.0E-04	10%	1.8E-04		1.4E-02			--		--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		3.5E-02			--		--	
Antimony (Sb)		8.6E-04		8.6E-04		3.7E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.03	1.6E-02	9.1E-09	2.2E-08	--		0.0000219	9%
Lead (Pb)		6.0E-04	50%	3.0E-04		2.1E-01			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		5.3E-02			--		--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		1.3E-02			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		4.6E-02			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		7.9E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		6.4E-02			--		--	
Vanadium (V)		2.0E-03		2.0E-03		6.6E-03			--		--	
Dioxins and furans		2.3E-09	54%	1.1E-09	0.03	1.8E-07	9.9E-14	2.4E-13	--		0.000226	90%
PAHs	2.3E-01				0.06	1.3E-03	1.5E-09	3.6E-09	3.5E-10		--	
								TOTAL	**3.5E-10**		**0.000251**	

Dermal Exposure to Chemicals via Contact with Soil - Grid Maximum Off-site

$$Daily\ Chemical\ Intake_{DS} = C_S \bullet \frac{SA_S \bullet AF \bullet FE \bullet ABS \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	2.1E-02	1.6E-10	1.9E-09	--		0.00000582	1%
Thallium (Tl)		2.0E-04	10%	1.8E-04		1.4E-02			--		--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		3.5E-02			--		--	
Antimony (Sb)		8.6E-04		8.6E-04		3.7E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.03	1.6E-02	3.7E-09	4.4E-08	--		0.0000437	9%
Lead (Pb)		6.0E-04	50%	3.0E-04		2.1E-01			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		5.3E-02			--		--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		1.3E-02			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		4.6E-02			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		7.9E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		6.4E-02			--		--	
Vanadium (V)		2.0E-03		2.0E-03		6.6E-03			--		--	
Dioxins and furans		2.3E-09	54%	1.1E-09	0.03	1.8E-07	4.1E-14	4.8E-13	--		0.000452	90%
PAHs	2.3E-01				0.06	1.3E-03	6.2E-10	7.2E-09	1.4E-10		--	
TOTAL									**1.4E-10**		**0.000501**	

Current Maximum Residential Location

Predicted ground level concentrations - chronic exposures - cumulative case

	Air Concentration - annual average ($\mu g/m^3$)	Air Concentration - annual average (mg/m^3)	Exposure pathways							
Key Chemicals	**Maximum residential**	**Maximum residential**	**inhalation**	**soil ingestion**	**soil - dermal**	**egg ingestion**	**fruit and vegetable ingestion**	**rainwater tank**	**meat ingestion**	**milk ingestion**
Hydrogen chloride (HCl)	3.985	0.003985	✓	×	×	×	×	×	×	×
Hydrogen fluoride (HF)	0.221	0.000221	✓	×	×	×	×	×	×	×
Ammonia	0.0577	0.0000577	✓	×	×	×	×	×	×	×
Cadmium (Cd)	0.00008	0.000000080	✓	✓	✓	✓	✓	✓	✓	✓
Thallium (Tl)	0.00005	0.000000050	✓	✓	✓	✓	✓	✓	✓	✓
Mercury (Hg)	0.00012	0.000000120	✓	✓	✓	✓	✓	✓	✓	✓
Antimony (Sb)	0.00012	0.000000120	✓	✓	✓	✓	✓	✓	✓	✓
Arsenic (As)	0.00008	0.000000080	✓	✓	✓	✓	✓	✓	✓	✓
Lead (Pb)	0.00084	0.000000840	✓	✓	✓	✓	✓	✓	✓	✓
Chromium (Cr VI assumed)	0.00037	0.00000037	✓	✓	✓	✓	✓	✓	✓	✓
Cobalt (Co)	0.00004	0.000000040	✓	✓	✓	✓	✓	✓	✓	✓
Copper (Cu)	0.00015	0.00000015	✓	✓	✓	✓	✓	✓	✓	✓
Manganese (Mn)	0.00058	0.00000058	✓	✓	✓	✓	✓	✓	✓	✓
Nickel (Ni)	0.00025	0.00000025	✓	✓	✓	✓	✓	✓	✓	✓
Vanadium (V)	0.00002	0.000000020	✓	✓	✓	✓	✓	✓	✓	✓
Dioxins and furans	3.51E-10	3.51E-13	✓	✓	✓	✓	✓	✓	✓	✓
Benzene	0.0628	0.0000628	✓	×	×	×	×	×	×	×
Formaldehyde	0.05766	0.00005766	✓	×	×	×	×	×	×	×
Polycyclic aromatic hydrocarbons (PAHs)	0.0000012	1.2E-09	✓	✓	✓	✓	✓	✓	✓	✓

	Deposition Rate - annual average ($ng/m^2/year$)	Deposition Rate - annual average ($mg/m^2/year$)
Key Chemicals	**Maximum residential**	**Maximum residential**
Hydrogen chloride (HCl)	NR	NR
Hydrogen fluoride (HF)	NR	NR
Ammonia	NR	NR
Cadmium (Cd)	982	0.000982
Thallium (Tl)	99.7	0.0000997
Mercury (Hg)	977	0.000977
Antimony (Sb)	919	0.000919
Arsenic (As)	98.3	0.0000983
Lead (Pb)	9747	0.009747
Chromium (Cr VI assumed)	1852	0.001852
Cobalt (Co)	100	0.0001
Copper (Cu)	970	0.00097
Manganese (Mn)	996	0.000996
Nickel (Ni)	970.8	0.0009708
Vanadium (V)	89.9	0.0000899
Dioxins and furans	NR	1.780E-08
Polycyclic aromatic hydrocarbons (PAHs)	NR	1.892E-03

Inhalation - gases and particulates
Cumulative maximum residential for this facility plus background plus brickworks

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * EF * ED}{AT}$$ (mg/m^3) for gases

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * DRF * EF * ED}{AT}$$ (mg/m^3) 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 (NEPM 1999 amended 2013) - NA for gasses
Exposure Frequency - normal conditions (EF, days/yr)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	35	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	306600	US EPA 2009

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	4.0E-03	2.0E-03	4.0E-03	--		0.153	73%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	2.2E-04	1.1E-04	2.2E-04	--		0.00762	4%
Ammonia		3.2E-01	0%	3.2E-01	5.8E-05	2.9E-05	5.8E-05	--		0.000180	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	8.0E-08	1.5E-08	3.0E-08	--		0.00750	4%
Thallium (Tl)		7.0E-04	10%	6.3E-04	5.0E-08	9.4E-09	1.9E-08	--		0.0000298	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	1.2E-07	2.3E-08	4.5E-08	--		0.000225	0%
Antimony (Sb)		3.0E-04	0%	3.0E-04	1.2E-07	2.3E-08	4.5E-08	--		0.000150	0%
Arsenic (As)		6.7E-05	0%	6.7E-05	8.0E-08	1.5E-08	3.0E-08	--		0.000448	0%
Lead (Pb)		5.0E-04	0%	5.0E-04	8.4E-07	1.6E-07	3.2E-07	--		0.000630	0%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	3.7E-07	6.9E-08	1.4E-07	--		0.0323	15%
Cobalt (Co)		1.0E-04	0%	1.0E-04	4.0E-08	7.5E-09	1.5E-08	--		0.000150	0%
Copper (Cu)		4.9E-01	60%	2.0E-01	1.5E-07	2.8E-08	5.6E-08	--		0.000000287	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	5.8E-07	1.1E-07	2.2E-07	--		0.00181	1%
Nickel (Ni)		5.9E-05	10%	5.3E-05	2.5E-07	4.7E-08	9.4E-08	--		0.00177	1%
Vanadium (V)		1.0E-04	0%	1.0E-04	2.0E-08	3.8E-09	7.5E-09	--		0.0000750	0%
Dioxins and furans		8.1E-09	54%	3.7E-09	3.5E-13	6.6E-14	1.3E-13	--		0.0000355	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	6.3E-05	1.2E-05	2.4E-05	7.1E-8	100%	0.000872	0%
Formaldehyde		7.0E-03	0%	7.0E-03	5.8E-05	1.1E-05	2.2E-05	--		0.00309	1%
PAHs	6.0E-01				1.2E-09	2.3E-10	4.5E-10	1.4E-10	0%	--	
TOTAL								**7.1E-08**		**0.209**	

Calculation of Concentrations in Soil

$$C_s = \frac{DR \bullet \left[1 - e^{-k \bullet t}\right]}{d \bullet \rho \bullet k} \bullet 1000 \quad \textbf{(mg/kg)}$$ ref: Stevens B. (1991)

where:

DR=	Particle deposition rate (mg/m^2/year)
K =	Chemical-specific soil-loss constant (1/year) = ln(2)/T0.5
T0.5 =	Chemical half-life in soil (years)
t =	Accumulation time (years)
d =	Soil mixing depth (m)
ρ =	Soil bulk-density (g/m^3)
1000 =	Conversion from g to kg

General Parameters		Surface (for direct contact)	Depth (for agricultural pathways)	
Soil bulk density (p)	g/m^3	1600000	1600000	Default for fill materials
General mixing depth (d)	m	0.01	0.15	As per OEHHA (2015) guidance
Duration of deposition (T)	years	70	70	As per OEHHA (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	9.8E-04	4.3E-03	2.9E-04
Thallium (Tl)	273973	2.5E-06	1.0E-04	4.4E-04	2.9E-05
Mercury (Hg)	273973	2.5E-06	9.8E-04	4.3E-03	2.8E-04
Antimony (Sb)	273973	2.5E-06	9.2E-04	4.0E-03	2.7E-04
Arsenic (As)	273973	2.5E-06	9.8E-05	4.3E-04	2.9E-05
Lead (Pb)	273973	2.5E-06	9.7E-03	4.3E-02	2.8E-03
Chromium (Cr VI assumed)	273973	2.5E-06	1.9E-03	8.1E-03	5.4E-04
Cobalt (Co)	273973	2.5E-06	1.0E-04	4.4E-04	2.9E-05
Copper (Cu)	273973	2.5E-06	9.7E-04	4.2E-03	2.8E-04
Manganese (Mn)	273973	2.5E-06	1.0E-03	4.4E-03	2.9E-04
Nickel (Ni)	273973	2.5E-06	9.7E-04	4.2E-03	2.8E-04
Vanadium (V)	273973	2.5E-06	9.0E-05	3.9E-04	2.6E-05
Dioxins and furans	15.00	4.6E-02	1.8E-08	2.3E-08	1.5E-09
PAHs	1.18	0.588	1.9E-03	2.0E-04	1.3E-05

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: OEHHA 2015

Exposure to Chemicals via Incidental Ingestion of Soil - Max Residential

$$Daily\ Chemical\ Intake_{IS} = C_S \bullet \frac{IR_S \bullet FI \bullet CF \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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	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)		(mg/kg)	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-03	1.3E-09	3.1E-09	--		0.00000959	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	4.4E-04	1.3E-10	3.1E-10	--		0.00000173	1%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	4.3E-03	1.3E-09	3.1E-09	--		0.0000127	8%
Antimony (Sb)		8.6E-04		8.6E-04	100%	4.0E-03	1.2E-09	2.9E-09	--		0.00000334	2%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	4.3E-04	1.3E-10	3.1E-10	--		0.00000031	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	4.3E-02	1.3E-08	3.0E-08	--		0.000102	66%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	8.1E-03	2.4E-09	5.8E-09	--		0.00000714	5%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	4.4E-04	1.3E-10	3.1E-10	--		0.000000279	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	4.2E-03	1.3E-09	3.0E-09	--		0.0000000541	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	4.4E-03	1.3E-09	3.1E-09	--		0.0000000445	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.2E-03	1.3E-09	3.0E-09	--		0.000000632	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	3.9E-04	1.2E-10	2.8E-10	--		0.000000140	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	2.3E-08	6.8E-15	1.7E-14	--		0.0000156	10%
PAHs	2.3E-01				100%	2.0E-04	5.9E-11	1.4E-10	1.4E-11		--	
								TOTAL	**1.4E-11**		**0.000153**	

Exposure to Chemicals via Incidental Ingestion of Soil - Max Residential

$$Daily\ Chemical\ Intake_{IS} = C_S \bullet \frac{IR_S \bullet FI \bullet CF \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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	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)		(mg/kg)	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-03	2.5E-09	2.9E-08	--		0.0000895	6%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	4.4E-04	2.5E-10	2.9E-09	--		0.0000162	1%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	4.3E-03	2.4E-09	2.8E-08	--		0.000119	8%
Antimony (Sb)		8.6E-04		8.6E-04	100%	4.0E-03	2.3E-09	2.7E-08	--		0.0000312	2%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	4.3E-04	2.5E-10	2.9E-09	--		0.0000029	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	4.3E-02	2.4E-08	2.8E-07	--		0.000948	66%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	8.1E-03	4.6E-09	5.4E-08	--		0.00006668	5%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	4.4E-04	2.5E-10	2.9E-09	--		0.00000260	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	4.2E-03	2.4E-09	2.8E-08	--		0.00000051	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	4.4E-03	2.5E-09	2.9E-08	--		0.00000041	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.2E-03	2.4E-09	2.8E-08	--		0.00000590	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	3.9E-04	2.2E-10	2.6E-09	--		0.00000131	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	2.3E-08	1.3E-14	1.5E-13	--		0.000146	10%
PAHs	2.3E-01				100%	2.0E-04	1.1E-10	1.3E-09	2.7E-11		--	
								TOTAL	**2.7E-11**		**0.00143**	

Dermal Exposure to Chemicals via Contact with Soil - Max Residential

$$Daily\ Chemical\ Intake_{DS} = C_S \bullet \frac{SA_S \bullet AF \bullet FE \bullet ABS \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	4.3E-03	8.0E-11	1.9E-10	--		0.000000604	2%
Thallium (Tl)		2.0E-04	10%	1.8E-04		4.4E-04			--		--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		4.3E-03			--		--	
Antimony (Sb)		8.6E-04		8.6E-04		4.0E-03			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.03	4.3E-04	2.4E-10	5.8E-10	--		0.0000005805	2%
Lead (Pb)		6.0E-04	50%	3.0E-04		4.3E-02			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		8.1E-03			--		--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		4.4E-04			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		4.2E-03			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		4.4E-03			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		4.2E-03			--		--	
Vanadium (V)		2.0E-03		2.0E-03		3.9E-04			--		--	
Dioxins and furans		2.3E-09	54%	1.1E-09	0.03	2.3E-08	1.3E-14	3.1E-14	--		0.0000295	96%
PAHs	2.3E-01				0.06	2.0E-04	2.2E-10	5.4E-10	5.2E-11		--	
								TOTAL	**5.2E-11**		**0.0000307**	

Dermal Exposure to Chemicals via Contact with Soil - Max Residential

$$Daily\ Chemical\ Intake_{DS} = C_S \bullet \frac{SA_S \bullet AF \bullet FE \bullet ABS \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	4.3E-03	3.3E-11	3.9E-10	--		0.00000121	2%
Thallium (Tl)		2.0E-04	10%	1.8E-04		4.4E-04			--		--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		4.3E-03			--		--	
Antimony (Sb)		8.6E-04		8.6E-04		4.0E-03			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.03	4.3E-04	1.0E-10	1.2E-09	--		0.000001161	2%
Lead (Pb)		6.0E-04	50%	3.0E-04		4.3E-02			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		8.1E-03			--		--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		4.4E-04			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		4.2E-03			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		4.4E-03			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		4.2E-03			--		--	
Vanadium (V)		2.0E-03		2.0E-03		3.9E-04			--		--	
Dioxins and furans		2.3E-09	54%	1.1E-09	0.03	2.3E-08	5.4E-15	6.2E-14	--		0.0000590	96%
PAHs	2.3E-01				0.06	2.0E-04	9.3E-11	1.1E-09	2.2E-11		--	
TOTAL									**2.2E-11**		**0.0000614**	

Calculation of Concentrations in Plants

ref: Stevens B. (1991)

Uptake Due to Deposition in Aboveground Crops

$$C_p = \frac{DR \bullet F \bullet \left[1 - e^{-k \bullet t}\right]}{Y \bullet k}$$ (mg/kg plant – wet weight)

where:

DR= Particle deposition rate for accidental release (mg/m^2/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/m^2)

Uptake via Roots from Soil

$$C_{rp} = C_s \bullet RUF$$ (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

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	2.7E-06	1.3E-06	0.125	2.9E-04	3.6E-05
Thallium (Tl)	14	0.05	2.7E-07	1.4E-07	0.001	2.9E-05	2.9E-08
Mercury (Hg)	14	0.05	2.7E-06	1.3E-06	0.225	2.8E-04	6.4E-05
Antimony (Sb)	14	0.05	2.5E-06	1.3E-06	0.05	2.7E-04	1.3E-05
Arsenic (As)	14	0.05	2.7E-07	1.3E-07	0.01	2.9E-05	2.9E-07
Lead (Pb)	14	0.05	2.7E-05	1.3E-05	0.0113	2.8E-03	3.2E-05
Chromium (Cr VI assumed)	14	0.05	5.1E-06	2.5E-06	0.00188	5.4E-04	1.0E-06
Cobalt (Co)	14	0.05	2.7E-07	1.4E-07	0.005	2.9E-05	1.5E-07
Copper (Cu)	14	0.05	2.7E-06	1.3E-06	0.1	2.8E-04	2.8E-05
Manganese (Mn)	14	0.05	2.7E-06	1.4E-06	0.0625	2.9E-04	1.8E-05
Nickel (Ni)	14	0.05	2.7E-06	1.3E-06	0.015	2.8E-04	4.2E-06
Vanadium (V)	14	0.05	2.5E-07	1.2E-07	0.00138	2.6E-05	3.6E-08
Dioxins and furans	14	0.05	4.9E-11	2.4E-11	0.000876	1.5E-09	1.4E-12
PAHs	14	0.05	5.2E-06	2.6E-06	0.00214	1.3E-05	2.9E-08

$ 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 - Max Residential

$$i \quad e \; i \quad i \quad ke = \quad x \; \frac{R \; x \quad x \quad x \quad x \quad x}{B \; x} \qquad R \; x \; \frac{R_p \; x \; R \; x \quad x \quad x \quad x}{B \; x} \qquad \text{(mg/kg/day)}$$

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 NEPM (2013)
Proportion of total intake from aboveground crops (%A	73%	Proportions as per NEPM (2013)
Proportion of total intake from root crops (%R)	27%	Proportions as per NEPM (2013)
Fraction ingested that is homegrown (%)	10%	Relevant to urban areas as per NEPM (2013)
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 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				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.3E-06	3.6E-05	2.5E-09	6.1E-09	--		0.0000190	17%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	1.4E-07	2.9E-08	2.5E-11	6.1E-11	--		0.000000341	0%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.3E-06	6.4E-05	4.3E-09	1.0E-08	--		0.0000435	39%
Antimony (Sb)		8.6E-04		8.6E-04	100%	1.3E-06	1.3E-05	1.1E-09	2.6E-09	--		0.00000301	3%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.3E-07	2.9E-07	4.2E-11	1.0E-10	--		0.000000100	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.3E-05	3.2E-05	4.4E-09	1.1E-08	--		0.0000350	31%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.5E-06	1.0E-06	5.0E-10	1.2E-09	--		0.00000150	1%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.4E-07	1.5E-07	3.3E-11	8.0E-11	--		0.0000000710	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.3E-06	2.8E-05	2.0E-09	4.9E-09	--		0.0000000878	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.4E-06	1.8E-05	1.4E-09	3.4E-09	--		0.0000000481	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.3E-06	4.2E-06	5.0E-10	1.2E-09	--		0.000000252	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	1.2E-07	3.6E-08	2.4E-11	5.7E-11	--		0.0000000284	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	2.4E-11	1.4E-12	4.3E-15	1.0E-14	--		0.00000979	9%
PAHs	2.3E-01				100%	2.6E-06	2.9E-08	4.5E-10	1.1E-09	1.0E-10		--	
							TOTAL			**1.0E-10**		**0.000113**	

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables - Max Residential

$$i \quad e \; i \quad i \quad ke = \quad x \; \frac{R \quad x \quad x \quad x \quad x \quad x}{B \quad x} \qquad R \; x \; \frac{R_p \; x \; R \quad x \quad x \quad x \quad x}{B \quad x} \qquad \text{(mg/kg/day)}$$

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 NEPM (2013)
Proportion of total intake from aboveground crops (%A	84%	Proportions as per NEPM (2013)
Proportion of total intake from root crops (%R)	16%	Proportions as per NEPM (2013)
Fraction ingested that is homegrown (%)	10%	Relevant to urban areas as per NEPM (2013)
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 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				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.3E-06	3.6E-05	1.1E-09	1.3E-08	--		0.0000400	14%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	1.4E-07	2.9E-08	1.9E-11	2.2E-10	--		0.00000124	0%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.3E-06	6.4E-05	1.8E-09	2.1E-08	--		0.0000885	31%
Antimony (Sb)		8.6E-04		8.6E-04	100%	1.3E-06	1.3E-05	5.1E-10	6.0E-09	--		0.00000694	2%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.3E-07	2.9E-07	2.5E-11	3.0E-10	--		0.000000296	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.3E-05	3.2E-05	2.6E-09	3.0E-08	--		0.000102	36%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.5E-06	1.0E-06	3.7E-10	4.3E-09	--		0.00000528	2%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.4E-07	1.5E-07	2.2E-11	2.6E-10	--		0.000000230	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.3E-06	2.8E-05	9.0E-10	1.1E-08	--		0.000000188	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.4E-06	1.8E-05	6.5E-10	7.6E-09	--		0.000000108	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.3E-06	4.2E-06	2.9E-10	3.3E-09	--		0.000000698	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	1.2E-07	3.6E-08	1.7E-11	2.0E-10	--		0.000000102	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	2.4E-11	1.4E-12	3.3E-15	3.9E-14			0.0000364	13%
PAHs	2.3E-01				100%	2.6E-06	2.9E-08	3.5E-10	4.1E-09	8.1E-11		--	
								TOTAL		**8.1E-11**		**0.000282**	

Calculation of Concentrations in Eggs

Uptake in to chicken eggs

$$= (\quad x \; R \quad x \quad R_S \; x \quad _S \; x \; B) \; x \qquad \text{(mg/kg egg – wet weight)}$$

where:
FI = Fraction of pasture/crop ingested by chickens each day (unitless)
IRc = Ingestion rate of pasture/crop by chicken each day (kg/day)
C = Concentration of chemical in grain/crop eaten by chicken (mg/kg)
IRs = Ingestion rate of soil by chickens each day (kg/day)
Cs = 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	Assume 100% of crops consumed by chickens is grown in the same soil
IRc (ingestion rate of crops)	kg/day	0.12	Assumed ingestion rate from OEHHA 2015 (assume concentration the same as predicted for aboveground crops)
IRs (ingestion rate of soil)	kg/day	0.01	USEPA (2005) (Ag Victoria recommendation)
B (bioavailability)	%	100%	

Chemical-specific Inputs and calculations - Grid Maximum Off-site

Chemical	Concentration in crops ingested by chickens mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to eggs day/kg	Egg Concentration mg/kg ww	
Cadmium (Cd)	1.3E-06	2.9E-04	1.0E-02	3.0E-08	OEHHA (2015)
Thallium (Tl)	1.4E-07	2.9E-05	1.7E-02	5.2E-09	95% from Leeman et al (2007)
Mercury (Hg)	1.3E-06	2.8E-04	9.0E-02	2.7E-07	OEHHA (2015)
Antimony (Sb)	1.3E-06	2.7E-04	1.7E-01	4.8E-07	95% from Leeman et al (2007)
Arsenic (As)	1.3E-07	2.9E-05	7.0E-02	2.1E-08	OEHHA (2015)
Lead (Pb)	1.3E-05	2.8E-03	4.0E-02	1.2E-06	OEHHA (2015)
Chromium (Cr VI assumed)	2.5E-06	5.4E-04	9.2E-03	5.2E-08	OEHHA (2015)
Cobalt (Co)	1.4E-07	2.9E-05	3.3E-03	1.0E-09	MacLachlan (2011)
Copper (Cu)	1.3E-06	2.8E-04	1.7E-01	5.1E-07	95% from Leeman et al (2007)
Manganese (Mn)	1.4E-06	2.9E-04	1.7E-01	5.2E-07	95% from Leeman et al (2007)
Nickel (Ni)	1.3E-06	2.8E-04	2.0E-02	6.0E-08	OEHHA (2015)
Vanadium (V)	1.2E-07	2.6E-05	1.7E-01	4.7E-08	95% from Leeman et al (2007)
Dioxins and furans	2.4E-11	1.5E-09	1.0E+01	1.8E-10	OEHHA (2015)
PAHs	2.6E-06	1.3E-05	3.0E-03	1.3E-09	OEHHA (2015)

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Eggs - Max Residential

$$i\ e\ i\ i\ ke = {}_{E}x \frac{R_E\ x\ x\ x\ x}{B\ x} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Eggs (IRE) (kg/day)	0.014	Ingestion rate of eggs relevant for adults as per enHealth (2012)
Fraction ingested that is homegrown (%)	100%	Assume all eggs consumed in urban area 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 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				Bioavailability (%)	Egg concentration	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)		(mg/kg wet weight)	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-08	2.5E-12	6.0E-12	--		0.0000000189	0%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	5.2E-09	4.3E-13	1.0E-12	--		0.00000000573	0%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	2.7E-07	2.2E-11	5.4E-11	--		0.000000226	1%
Antimony (Sb)		8.6E-04		8.6E-04	100%	4.8E-07	4.0E-11	9.6E-11	--		0.000000112	0%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	2.1E-08	1.8E-12	4.2E-12	--		0.00000000424	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.2E-06	1.0E-10	2.4E-10	--		0.000000801	2%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	5.2E-08	4.3E-12	1.0E-11	--		0.0000000130	0%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.0E-09	8.4E-14	2.0E-13	--		0.000000000182	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	5.1E-07	4.2E-11	1.0E-10	--		0.00000000181	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	5.2E-07	4.3E-11	1.0E-10	--		0.00000000149	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	6.0E-08	5.0E-12	1.2E-11	--		0.00000000249	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	4.7E-08	3.9E-12	9.4E-12	--		0.00000000471	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.8E-10	1.5E-14	3.7E-14	--		0.0000347	97%
PAHs	2.3E-01				100%	1.3E-09	1.1E-13	2.7E-13	2.6E-14		--	
								TOTAL	**2.6E-14**		**0.0000359**	

Exposure to Chemicals via Ingestion of Eggs - Max Residential

$$i\ e\ i\ i\ ke = {}_{E}x\ \frac{R_E\ x\ x\ x\ x}{B\ x} \qquad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Young children		
Ingestion Rate of Eggs (IRE) (kg/day)	0.006	Ingestion rate of eggs relevant for young children as per enHealth (2012)
Fraction ingested that is homegrown (%)	100%	Assume all eggs consumed in urban area 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 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				Bioavailability (%)	Egg concentration	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)		(mg/kg wet weight)	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-08	1.0E-12	1.2E-11	--		0.0000000378	0%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	5.2E-09	1.8E-13	2.1E-12	--		0.0000000115	0%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	2.7E-07	9.3E-12	1.1E-10	--		0.000000451	1%
Antimony (Sb)		8.6E-04		8.6E-04	100%	4.8E-07	1.7E-11	1.9E-10	--		0.00000022	0%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	2.1E-08	7.3E-13	8.5E-12	--		0.00000000848	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.2E-06	4.1E-11	4.8E-10	--		0.00000160	2%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	5.2E-08	1.8E-12	2.1E-11	--		0.0000000259	0%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.0E-09	3.5E-14	4.1E-13	--		0.000000000363	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	5.1E-07	1.7E-11	2.0E-10	--		0.00000000363	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	5.2E-07	1.8E-11	2.1E-10	--		0.00000000298	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	6.0E-08	2.1E-12	2.4E-11	--		0.00000000498	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	4.7E-08	1.6E-12	1.9E-11	--		0.00000000942	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.8E-10	6.3E-15	7.3E-14	--		0.0000693	97%
PAHs	2.3E-01				100%	1.3E-09	4.6E-14	5.3E-13	1.1E-14		--	
								TOTAL	**1.1E-14**		**0.0000717**	

Calculation of Concentrations in Homegrown Beef

Uptake in to beef meat

$$= (\quad \times R \times \quad R_S \times \quad _S \times B) \times \quad _B \qquad \text{(mg/kg beef – wet weight)}$$

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	Assume 100% of pasture consumed by cattle is grown in the same soil
IRc (ingestion rate of crops)	kg dw/day	9	Assumed ingestion rate from OEHHA 2015 (assume concentration the same as predicted for aboveground crops)
IRs (ingestion rate of soil)	kg/day	0.45	OEHHA (2015) Table 5.4 (soil ingestion = 5% of pasture ingestion (as dry weight))
B (bioavailability)	%	100%	

Chemical-specific Inputs and calculations

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)	1.3E-06	2.9E-04	5.5E-04	7.8E-08
Thallium (Tl)	1.4E-07	2.9E-05	4.0E-02	5.7E-07
Mercury (Hg)	1.3E-06	2.8E-04	3.0E-04	4.2E-08
Antimony (Sb)	1.3E-06	2.7E-04	1.0E-03	1.3E-07
Arsenic (As)	1.3E-07	2.9E-05	2.0E-03	2.8E-08
Lead (Pb)	1.3E-05	2.8E-03	4.0E-04	5.6E-07
Chromium (Cr VI assumed)	2.5E-06	5.4E-04	5.5E-03	1.5E-06
Cobalt (Co)	1.4E-07	2.9E-05	2.0E-02	2.9E-07
Copper (Cu)	1.3E-06	2.8E-04	1.0E-02	1.4E-06
Manganese (Mn)	1.4E-06	2.9E-04	4.0E-04	5.7E-08
Nickel (Ni)	1.3E-06	2.8E-04	3.0E-04	4.2E-08
Vanadium (V)	1.2E-07	2.6E-05	2.5E-03	3.2E-08
Dioxins and furans	2.4E-11	1.5E-09	1.6E-01	1.5E-10
PAHs	2.6E-06	1.3E-05	3.4E-02	1.0E-06

Transfer factors from RAIS (accessed in 2023)

Exposure to Chemicals via Ingestion of Beef - Max Residential

i chemical i ke= $_B$ x $\frac{R_B \text{ x } \quad \text{x} \quad \text{x} \quad \text{x}}{\text{B} \quad \text{x}}$ (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 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				Bioavailability (%)	Beef concentration	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)		(mg/kg wet weight)	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%	7.8E-08	2.6E-11	6.2E-11	--		0.000000194	0%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	5.7E-07	1.9E-10	4.6E-10	--		0.00000254	2%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	4.2E-08	1.4E-11	3.4E-11	--		0.000000140	0%
Antimony (Sb)		8.6E-04		8.6E-04	100%	1.3E-07	4.4E-11	1.1E-10	--		0.000000123	0%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	2.8E-08	9.4E-12	2.3E-11	--		0.0000000226	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	5.6E-07	1.9E-10	4.5E-10	--		0.00000149	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	1.5E-06	4.8E-10	1.2E-09	--		0.00000144	1%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	2.9E-07	9.5E-11	2.3E-10	--		0.000000205	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.4E-06	4.6E-10	1.1E-09	--		0.0000000199	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	5.7E-08	1.9E-11	4.6E-11	--		0.000000000654	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.2E-08	1.4E-11	3.3E-11	--		0.00000000697	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	3.2E-08	1.1E-11	2.6E-11	--		0.0000000129	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.5E-10	4.8E-14	1.2E-13	--		0.000110	95%
PAHs	2.3E-01				100%	1.0E-06	3.3E-10	8.0E-10	7.7E-11		--	
								TOTAL	7.7E-11		0.000117	

Exposure to Chemicals via Ingestion of Beef - Max Residential

$$i\ \text{chemical}\ i \quad ke = {}_{B} x \frac{R_B x \quad x \quad x \quad x}{B \quad x} \qquad \text{(mg/kg/day)}$$

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 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				Bioavailability (%)	Beef concentration	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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	7.8E-08	1.3E-11	1.5E-10	--		0.000000481	0%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	5.7E-07	9.7E-11	1.1E-09	--		0.00000631	2%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	4.2E-08	7.2E-12	8.3E-11	--		0.000000348	0%
Antimony (Sb)		8.6E-04		8.6E-04	100%	1.3E-07	2.2E-11	2.6E-10	--		0.000000304	0%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	2.8E-08	4.8E-12	5.6E-11	--		0.0000000560	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	5.6E-07	9.5E-11	1.1E-09	--		0.00000370	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	1.5E-06	2.5E-10	2.9E-09	--		0.00000358	1%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	2.9E-07	4.9E-11	5.7E-10	--		0.000000508	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.4E-06	2.4E-10	2.8E-09	--		0.0000000493	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	5.7E-08	9.7E-12	1.1E-10	--		0.00000000162	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.2E-08	7.1E-12	8.3E-11	--		0.0000000173	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	3.2E-08	5.5E-12	6.4E-11	--		0.0000000320	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.5E-10	2.5E-14	2.9E-13	--		0.000274	95%
PAHs	2.3E-01				100%	1.0E-06	1.7E-10	2.0E-09	3.9E-11		--	
								TOTAL	**3.9E-11**		**0.000289**	

Calculation of Concentrations in Dairy Milk

Uptake in to milk (dairy cows)

=(x R x R_S x $_S$ x B) x $_B$ (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 milk (day/kg)

General Parameters	Units	Value	
FI (fraction of crops ingested from property)		1	Assume 100% of pasture consumed by cattle is grown in the same soil
IRc (ingestion rate of crops)	kg (dw)/day	22	Assumed ingestion rate from OEHHA 2015 for lactating cattle (assume concentration the same as predicted for aboveground crops)
IRs (ingestion rate of soil)	kg/day	1.1	OEHHA (2015) Table 5.4 (soil ingestion = 5% of pasture ingestion (as dry weight))
B (bioavailability)	%	100%	

Chemical-specific Inputs and calculations - Grid Maximum Off-site

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)	1.3E-06	2.9E-04	1.0E-03	3.4E-07
Thallium (Tl)	1.4E-07	2.9E-05	2.0E-03	7.0E-08
Mercury (Hg)	1.3E-06	2.8E-04	9.0E-07	3.1E-10
Antimony (Sb)	1.3E-06	2.7E-04	1.0E-04	3.2E-08
Arsenic (As)	1.3E-07	2.9E-05	5.0E-05	1.7E-09
Lead (Pb)	1.3E-05	2.8E-03	2.5E-04	8.6E-07
Chromium (Cr VI assumed)	2.5E-06	5.4E-04	1.5E-03	9.7E-07
Cobalt (Co)	1.4E-07	2.9E-05	2.0E-03	7.0E-08
Copper (Cu)	1.3E-06	2.8E-04	1.5E-03	5.1E-07
Manganese (Mn)	1.4E-06	2.9E-04	3.5E-04	1.2E-07
Nickel (Ni)	1.3E-06	2.8E-04	1.0E-03	3.4E-07
Vanadium (V)	1.2E-07	2.6E-05	2.0E-05	6.3E-10
Dioxins and furans	2.4E-11	1.5E-09	5.0E-02	1.1E-10
PAHs	2.6E-06	1.3E-05	1.1E-02	7.7E-07

Transfer factors from RAIS (accessed in 2023)

Exposure to Chemicals via Ingestion of Milk - Max Residential

$$ke_i = C_{M\,i} \times \frac{R_M \times \quad \times \quad \times \quad \times}{B \quad \times} \qquad \text{(mg/kg/day)}$$

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 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				Bioavailability (%)	Milk concentration	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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.4E-07	2.6E-09	6.4E-09	--		0.0000199	1%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	7.0E-08	5.4E-10	1.3E-09	--		0.00000719	0%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	3.1E-10	2.4E-12	5.7E-12	--		0.0000000238	0%
Antimony (Sb)		8.6E-04		8.6E-04	100%	3.2E-08	2.5E-10	6.0E-10	--		0.000000694	0%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.7E-09	1.3E-11	3.2E-11	--		0.0000000319	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	8.6E-07	6.6E-09	1.6E-08	--		0.0000527	3%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	9.7E-07	7.5E-09	1.8E-08	--		0.0000223	1%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	7.0E-08	5.4E-10	1.3E-09	--		0.00000116	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	5.1E-07	3.9E-09	9.4E-09	--		0.000000169	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.2E-07	9.4E-10	2.3E-09	--		0.0000000323	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.4E-07	2.6E-09	6.3E-09	--		0.00000131	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	6.3E-10	4.8E-12	1.2E-11	--		0.00000000584	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.1E-10	8.5E-13	2.1E-12	--		0.001943	95%
PAHs	2.3E-01				100%	7.7E-07	5.9E-09	1.4E-08	1.4E-9		--	
								TOTAL	**1.4E-9**		**0.00205**	

Exposure to Chemicals via Ingestion of Milk - Max Residential

$$i\ \text{chemical}\ i \quad ke = {}_{M} x \frac{R_M x \ x \ x \ x}{B \ x} \qquad \text{(mg/kg/day)}$$

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 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				Bioavailability (%)	Milk concentration	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)		(mg/kg wet weight)	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%	3.4E-07	2.2E-09	2.5E-08	--		0.0000787	1%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	7.0E-08	4.4E-10	5.1E-09	--		0.0000284	0%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	3.1E-10	1.9E-12	2.3E-11	--		0.0000000940	0%
Antimony (Sb)		8.6E-04		8.6E-04	100%	3.2E-08	2.0E-10	2.4E-09	--		0.00000274	0%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.7E-09	1.1E-11	1.3E-10	--		0.000000126	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	8.6E-07	5.4E-09	6.3E-08	--		0.000208	3%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	9.7E-07	6.1E-09	7.1E-08	--		0.0000880	1%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	7.0E-08	4.4E-10	5.1E-09	--		0.00000458	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	5.1E-07	3.2E-09	3.7E-08	--		0.000000667	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.2E-07	7.7E-10	8.9E-09	--		0.000000128	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.4E-07	2.1E-09	2.5E-08	--		0.00000519	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	6.3E-10	4.0E-12	4.6E-11	--		0.0000000231	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.1E-10	7.0E-13	8.1E-12	--		0.00768	95%
PAHs	2.3E-01				100%	7.7E-07	4.8E-09	5.6E-08	1.1E-9		--	
								TOTAL	**1.1E-9**		**0.00810**	

Maximum Commercial/Industrial Location

Predicted ground level concentrations - chronic exposures - cumulative case

	Air Concentration - annual average ($\mu g/m^3$)	Air Concentration - annual average (mg/m^3)	Exposure pathways							
Key Chemicals	**Max commercial**	**Max commercial**	**inhalation**	**soil ingestion**	**soil - dermal**	**egg ingestion**	**fruit and vegetable ingestion**	**rainwater tank**	**meat ingestion**	**milk ingestion**
Hydrogen chloride (HCl)	2.104	0.002104	✓	×	×	×	×	×	×	×
Hydrogen fluoride (HF)	0.119	0.000119	✓	×	×	×	×	×	×	×
Ammonia	0.22515	0.00022515	✓	×	×	×	×	×	×	×
Cadmium (Cd)	0.00027	0.000000270	✓	✓	✓	×	×	✓	×	×
Thallium (Tl)	0.00018	0.000000180	✓	✓	✓	×	×	✓	×	×
Mercury (Hg)	0.00045	0.000000450	✓	✓	✓	×	×	✓	×	×
Antimony (Sb)	0.00047	0.000000470	✓	✓	✓	×	×	✓	×	×
Arsenic (As)	0.00021	0.000000210	✓	✓	✓	×	×	✓	×	×
Lead (Pb)	0.00271	0.000002710	✓	✓	✓	×	×	✓	×	×
Chromium (Cr VI assumed)	0.00068	0.00000068	✓	✓	✓	×	×	✓	×	×
Cobalt (Co)	0.00017	0.000000170	✓	✓	✓	×	×	✓	×	×
Copper (Cu)	0.00059	0.00000059	✓	✓	✓	×	×	✓	×	×
Manganese (Mn)	0.00102	0.00000102	✓	✓	✓	×	×	✓	×	×
Nickel (Ni)	0.00082	0.00000082	✓	✓	✓	×	×	✓	×	×
Vanadium (V)	0.00008	0.000000080	✓	✓	✓	×	×	✓	×	×
Dioxins and furans	1.35E-09	1.35E-12	✓	✓	✓	×	×	✓	×	×
Benzene	0.22515	0.00022515	✓	×	×	×	×	×	×	×
Formaldehyde	0.22515	0.00022515	✓	×	×	×	×	×	×	×
Polycyclic aromatic hydrocarbons (PAHs)	0.0000045	4.5E-09	✓	✓	✓	×	×	✓	×	×

	Deposition Rate - annual average ($ng/m^2/year$)	Deposition Rate - annual average ($mg/m^2/year$)
Key Chemicals	**Max commercial**	**Max commercial**
Cadmium (Cd)	2544	0.002544
Thallium (Tl)	1761	0.001761
Mercury (Hg)	4305	0.004305
Antimony (Sb)	4549	0.004549
Arsenic (As)	1991	0.001991
Lead (Pb)	25926	0.025926
Chromium (Cr VI assumed)	6506	0.006506
Cobalt (Co)	1614	0.001614
Copper (Cu)	5699	0.005699
Manganese (Mn)	9734	0.009734
Nickel (Ni)	7827	0.007827
Vanadium (V)	807	0.000807
Dioxins and furans	NR	7.326E-08
Polycyclic aromatic hydrocarbons (PAHs)	NR	7.096E-03

Inhalation - gases and particulates

Maximum commercial/industrial location (i.e. maximum concentrations at commercial or industrial sites around the facility)

$$InhalationExposureConc_V = C_a \bullet \frac{ET \bullet FI \bullet EF \bullet ED}{AT} \quad (mg/m^3)$$

Parameters Relevant to Quantification of Community Exposures - Workers		
Exposure Time at Home (ET, hr/day)	10	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 (NEPM 1999 amended 2013) - NA for gasses
Exposure Frequency - normal conditions (EF, days/yr)	240	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
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	2.1E-03	2.5E-04	5.8E-04	--		0.02217	33%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	1.2E-04	1.4E-05	3.3E-05	--		0.001124	2%
Ammonia		3.2E-01	0%	3.2E-01	2.3E-04	2.6E-05	6.2E-05	--		0.000193	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	2.7E-07	3.2E-08	7.4E-08	--		0.01849	27%
Thallium (Tl)		7.0E-04	10%	6.3E-04	1.8E-07	2.1E-08	4.9E-08	--		0.0000783	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	4.5E-07	5.3E-08	1.2E-07	--		0.000616	1%
Antimony (Sb)		3.0E-04	0%	3.0E-04	4.7E-07	2.1E-08	4.8E-08	--		0.000161	0%
Arsenic (As)		6.7E-05	0%	6.7E-05	2.1E-07	2.5E-08	5.8E-08	--		0.000859	1%
Lead (Pb)		5.0E-04	0%	5.0E-04	2.7E-06	3.2E-07	7.4E-07	--		0.00148	2%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	6.8E-07	3.0E-08	7.0E-08	--		0.01625	24%
Cobalt (Co)		1.0E-04	0%	1.0E-04	1.7E-07	7.5E-09	1.7E-08	--		0.000175	0%
Copper (Cu)		4.9E-01	60%	2.0E-01	5.9E-07	2.6E-08	6.1E-08	--		0.000000309	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	1.0E-06	4.5E-08	1.0E-07	--		0.000873	1%
Nickel (Ni)		5.9E-05	10%	5.3E-05	8.2E-07	3.6E-08	8.4E-08	--		0.00159	2%
Vanadium (V)		1.0E-04	0%	1.0E-04	8.0E-08	3.5E-09	8.2E-09	--		0.0000822	0%
Dioxins and furans		8.1E-09	54%	3.7E-09	1.4E-12	5.9E-14	1.4E-13	--		0.0000375	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	2.3E-04	9.9E-06	2.3E-05	5.9E-8		0.000857	1%
Formaldehyde		7.0E-03	0%	7.0E-03	2.3E-04	9.9E-06	2.3E-05	--		0.003305	5%
PAHs	6.0E-01				4.5E-09	2.0E-10	4.6E-10	1.2E-10		--	
							TOTAL	**6.0E-08**		**0.0675**	

Calculation of Concentrations in Soil

$$C_s = \frac{DR \bullet \left[1 - e^{-k \bullet t}\right]}{d \bullet \rho \bullet k} \bullet 1000 \quad \textbf{(mg/kg)}$$ ref: Stevens B. (1991)

where:

DR=	Particle deposition rate (mg/m^2/year)
K =	Chemical-specific soil-loss constant (1/year) = ln(2)/T0.5
T0.5 =	Chemical half-life in soil (years)
t =	Accumulation time (years)
d =	Soil mixing depth (m)
ρ =	Soil bulk-density (g/m^3)
1000 =	Conversion from g to kg

General Parameters		Surface (for direct contact)	Depth (for agricultural pathways)	
Soil bulk density (p)	g/m^3	1600000	1600000	Default for fill materials
General mixing depth (d)	m	0.01	0.15	As per OEHHA (2015) guidance
Duration of deposition (T)	years	70	70	As per OEHHA (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	2.5E-03	1.1E-02	7.4E-04
Thallium (Tl)	273973	2.5E-06	1.8E-03	7.7E-03	5.1E-04
Mercury (Hg)	273973	2.5E-06	4.3E-03	1.9E-02	1.3E-03
Antimony (Sb)	273973	2.5E-06	4.5E-03	2.0E-02	1.3E-03
Arsenic (As)	273973	2.5E-06	2.0E-03	8.7E-03	5.8E-04
Lead (Pb)	273973	2.5E-06	2.6E-02	1.1E-01	7.6E-03
Chromium (Cr VI assumed)	273973	2.5E-06	6.5E-03	2.8E-02	1.9E-03
Cobalt (Co)	273973	2.5E-06	1.6E-03	7.1E-03	4.7E-04
Copper (Cu)	273973	2.5E-06	5.7E-03	2.5E-02	1.7E-03
Manganese (Mn)	273973	2.5E-06	9.7E-03	4.3E-02	2.8E-03
Nickel (Ni)	273973	2.5E-06	7.8E-03	3.4E-02	2.3E-03
Vanadium (V)	273973	2.5E-06	8.1E-04	3.5E-03	2.4E-04
Dioxins and furans	15.00	4.6E-02	7.3E-08	9.5E-08	6.3E-09
PAHs	1.18	0.588	7.1E-03	7.5E-04	5.0E-05

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: OEHHA 2015

Exposure to Chemicals via Incidental Ingestion of Soil - Max Commercial

$$Daily\ Chemical\ Intake_{IS} = C_S \bullet \frac{IR_S \bullet FI \bullet CF \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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)	240	Days at work
Exposure Duration (ED, years)	30	Time at one workplace
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 2009
Averaging Time - Threshold (Atn, days)	10950	USEPA 2009

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	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)		(mg/kg)	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%	1.1E-02	2.2E-09	5.2E-09	--		0.00001633	5%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	7.7E-03	1.6E-09	3.6E-09	--		0.00002010	6%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.9E-02	3.8E-09	8.8E-09	--		0.0000369	11%
Antimony (Sb)		8.6E-04		8.6E-04	100%	2.0E-02	4.0E-09	9.3E-09	--		0.00001087	3%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	8.7E-03	1.8E-09	4.1E-09	--		0.00000409	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.1E-01	2.3E-08	5.3E-08	--		0.000178	53%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.8E-02	5.7E-09	1.3E-08	--		0.00001650	5%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	7.1E-03	1.4E-09	3.3E-09	--		0.000002961	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.5E-02	5.0E-09	1.2E-08	--		0.0000002091	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	4.3E-02	8.6E-09	2.0E-08	--		0.0000002857	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.4E-02	6.9E-09	1.6E-08	--		0.000003350	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	3.5E-03	7.1E-10	1.7E-09	--		0.000000829	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	9.5E-08	1.9E-14	4.5E-14	--		0.0000423	13%
PAHs	2.3E-01				100%	7.5E-04	1.5E-10	3.5E-10	3.5E-11		--	
								TOTAL	**3.5E-11**		**0.000332**	

Exposure to Chemicals via Incidental Ingestion of Soil - Max Commercial

$$Daily\ Chemical\ Intake_{IS} = C_S \bullet \frac{IR_S \bullet FI \bullet CF \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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)	240	Days at work
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 2009
Averaging Time - Threshold (Atn, days)	2190	USEPA 2009

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	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)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.1E-02	4.2E-09	4.9E-08	--		0.0001525	5%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	7.7E-03	2.9E-09	3.4E-08	--		0.0001876	6%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.9E-02	7.1E-09	8.3E-08	--		0.000344	11%
Antimony (Sb)		8.6E-04		8.6E-04	100%	2.0E-02	7.5E-09	8.7E-08	--		0.0001014	3%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	8.7E-03	3.3E-09	3.8E-08	--		0.0000382	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.1E-01	4.3E-08	5.0E-07	--		0.001657	53%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.8E-02	1.1E-08	1.2E-07	--		0.00015403	5%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	7.1E-03	2.7E-09	3.1E-08	--		0.00002763	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.5E-02	9.4E-09	1.1E-07	--		0.00000195	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	4.3E-02	1.6E-08	1.9E-07	--		0.00000267	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.4E-02	1.3E-08	1.5E-07	--		0.00003127	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	3.5E-03	1.3E-09	1.5E-08	--		0.00000774	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	9.5E-08	3.6E-14	4.2E-13	--		0.000394	13%
PAHs	2.3E-01				100%	7.5E-04	2.8E-10	3.3E-09	6.6E-11		--	
								TOTAL	**6.6E-11**		**0.00310**	

Dermal Exposure to Chemicals via Contact with Soil - Max Commercial

$$Daily\ Chemical\ Intake_{DS} = C_S \bullet \frac{SA_S \bullet AF \bullet FE \bullet ABS \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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)	240	Days at work
Exposure Duration (ED, years)	30	Time at one workplace
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 2009
Averaging Time - Threshold (Atn, days)	10950	USEPA 2009

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	1.1E-02	1.4E-10	3.3E-10	--		0.000001029	1%
Thallium (Tl)		2.0E-04	10%	1.8E-04		7.7E-03			--		--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		1.9E-02			--		--	
Antimony (Sb)		8.6E-04		8.6E-04		2.0E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.03	8.7E-03	3.3E-09	7.7E-09	--		0.000007731	9%
Lead (Pb)		6.0E-04	50%	3.0E-04		1.1E-01			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		2.8E-02			--		--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		7.1E-03			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		2.5E-02			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		4.3E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		3.4E-02			--		--	
Vanadium (V)		2.0E-03		2.0E-03		3.5E-03			--		--	
Dioxins and furans		2.3E-09	54%	1.1E-09	0.03	9.5E-08	3.6E-14	8.4E-14	--		0.0000799	90%
PAHs	2.3E-01				0.06	7.5E-04	5.7E-10	1.3E-09	1.3E-10		--	
								TOTAL	**1.3E-10**		**0.0000886**	

Dermal Exposure to Chemicals via Contact with Soil - Max Commercial

$$Daily\ Chemical\ Intake_{DS} = C_S \bullet \frac{SA_S \bullet AF \bullet FE \bullet ABS \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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)	240	Days at work
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 2009
Averaging Time - Threshold (Atn, days)	2190	USEPA 2009

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	1.1E-02	5.6E-11	6.6E-10	--		0.00000206	1%
Thallium (Tl)		2.0E-04	10%	1.8E-04		7.7E-03			--		--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		1.9E-02			--		--	
Antimony (Sb)		8.6E-04		8.6E-04		2.0E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.03	8.7E-03	1.3E-09	1.5E-08	--		0.00001546	9%
Lead (Pb)		6.0E-04	50%	3.0E-04		1.1E-01			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		2.8E-02			--		--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		7.1E-03			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		2.5E-02			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		4.3E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		3.4E-02			--		--	
Vanadium (V)		2.0E-03		2.0E-03		3.5E-03			--		--	
Dioxins and furans		2.3E-09	54%	1.1E-09	0.03	9.5E-08	1.4E-14	1.7E-13	--		0.0001597	90%
PAHs	2.3E-01				0.06	7.5E-04	2.3E-10	2.7E-09	5.3E-11		--	
								TOTAL	**5.3E-11**		**0.0001772**	

Assuming this location could be changed to residential

Inhalation - gases and particulates
Cumulative maximum commerical assuming this could be used for residential
Facility plus background plus brickworks

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * EF * ED}{AT}$$ (mg/m^3) for gases

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * DRF * EF * ED}{AT}$$ (mg/m^3) 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 (NEPM 1999 amended 2013) - NA for gasses
Exposure Frequency - normal conditions (EF, days/yr)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	35	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	306600	US EPA 2009

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	2.1E-03	1.1E-03	2.1E-03	--		0.0809	41%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	1.2E-04	6.0E-05	1.2E-04	--		0.00410	2%
Ammonia		3.2E-01	0%	3.2E-01	2.3E-04	1.1E-04	2.3E-04	--		0.000704	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	2.7E-07	5.1E-08	1.0E-07	--		0.02531	13%
Thallium (Tl)		7.0E-04	10%	6.3E-04	1.8E-07	3.4E-08	6.8E-08	--		0.0001071	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	4.5E-07	8.4E-08	1.7E-07	--		0.000844	0%
Antimony (Sb)		3.0E-04	0%	3.0E-04	4.7E-07	8.8E-08	1.8E-07	--		0.000588	0%
Arsenic (As)		6.7E-05	0%	6.7E-05	2.1E-07	3.9E-08	7.9E-08	--		0.001175	1%
Lead (Pb)		5.0E-04	0%	5.0E-04	2.7E-06	5.1E-07	1.0E-06	--		0.002033	1%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	6.8E-07	1.3E-07	2.6E-07	--		0.0593	30%
Cobalt (Co)		1.0E-04	0%	1.0E-04	1.7E-07	3.2E-08	6.4E-08	--		0.000638	0%
Copper (Cu)		4.9E-01	60%	2.0E-01	5.9E-07	1.1E-07	2.2E-07	--		0.000001129	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	1.0E-06	1.9E-07	3.8E-07	--		0.00319	2%
Nickel (Ni)		5.9E-05	10%	5.3E-05	8.2E-07	1.5E-07	3.1E-07	--		0.00579	3%
Vanadium (V)		1.0E-04	0%	1.0E-04	8.0E-08	1.5E-08	3.0E-08	--		0.0003000	0%
Dioxins and furans		8.1E-09	54%	3.7E-09	1.4E-12	2.5E-13	5.1E-13	--		0.0001367	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	2.3E-04	4.2E-05	8.4E-05	2.5E-7	100%	0.003127	2%
Formaldehyde		7.0E-03	0%	7.0E-03	2.3E-04	4.2E-05	8.4E-05	--		0.01206	6%
PAHs	6.0E-01				4.5E-09	8.4E-10	1.7E-09	5.1E-10	0%	--	
							TOTAL	**2.5E-07**		**0.197**	

Calculation of Concentrations in Soil

$$C_s = \frac{DR \bullet \left[1 - e^{-k \bullet t}\right]}{d \bullet \rho \bullet k} \bullet 1000 \quad \textbf{(mg/kg)}$$ ref: Stevens B. (1991)

where:

DR=	Particle deposition rate (mg/m^2/year)
K =	Chemical-specific soil-loss constant (1/year) = ln(2)/T0.5
T0.5 =	Chemical half-life in soil (years)
t =	Accumulation time (years)
d =	Soil mixing depth (m)
ρ =	Soil bulk-density (g/m^3)
1000 =	Conversion from g to kg

General Parameters		Surface (for direct contact)	Depth (for agricultural pathways)	
Soil bulk density (p)	g/m^3	1600000	1600000	Default for fill materials
General mixing depth (d)	m	0.01	0.15	As per OEHHA (2015) guidance
Duration of deposition (T)	years	70	70	As per OEHHA (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	2.5E-03	1.1E-02	7.4E-04
Thallium (Tl)	273973	2.5E-06	1.8E-03	7.7E-03	5.1E-04
Mercury (Hg)	273973	2.5E-06	4.3E-03	1.9E-02	1.3E-03
Antimony (Sb)	273973	2.5E-06	4.5E-03	2.0E-02	1.3E-03
Arsenic (As)	273973	2.5E-06	2.0E-03	8.7E-03	5.8E-04
Lead (Pb)	273973	2.5E-06	2.6E-02	1.1E-01	7.6E-03
Chromium (Cr VI assumed)	273973	2.5E-06	6.5E-03	2.8E-02	1.9E-03
Cobalt (Co)	273973	2.5E-06	1.6E-03	7.1E-03	4.7E-04
Copper (Cu)	273973	2.5E-06	5.7E-03	2.5E-02	1.7E-03
Manganese (Mn)	273973	2.5E-06	9.7E-03	4.3E-02	2.8E-03
Nickel (Ni)	273973	2.5E-06	7.8E-03	3.4E-02	2.3E-03
Vanadium (V)	273973	2.5E-06	8.1E-04	3.5E-03	2.4E-04
Dioxins and furans	15.00	4.6E-02	7.3E-08	9.5E-08	6.3E-09
PAHs	1.18	0.588	7.1E-03	7.5E-04	5.0E-05

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: OEHHA 2015

Exposure to Chemicals via Incidental Ingestion of Soil - Max Commercial

$$Daily\ Chemical\ Intake_{IS} = C_S \bullet \frac{IR_S \bullet FI \bullet CF \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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
Exposure Duration (ED, years)	29	Time living at one residence
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 2009
Averaging Time - Threshold (Atn, days)	10585	USEPA 2009

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.1E-02	3.3E-09	7.9E-09	--		0.00002484	5%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	7.7E-03	2.3E-09	5.5E-09	--		0.00003057	6%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.9E-02	5.6E-09	1.3E-08	--		0.0000560	11%
Antimony (Sb)		8.6E-04		8.6E-04	100%	2.0E-02	5.9E-09	1.4E-08	--		0.00001653	3%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	8.7E-03	2.6E-09	6.2E-09	--		0.00000622	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.1E-01	3.4E-08	8.1E-08	--		0.000270	53%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.8E-02	8.4E-09	2.0E-08	--		0.00002510	5%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	7.1E-03	2.1E-09	5.0E-09	--		0.000004503	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.5E-02	7.4E-09	1.8E-08	--		0.0000003180	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	4.3E-02	1.3E-08	3.0E-08	--		0.0000004345	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.4E-02	1.0E-08	2.4E-08	--		0.000005095	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	3.5E-03	1.0E-09	2.5E-09	--		0.000001261	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	9.5E-08	2.8E-14	6.8E-14	--		0.0000643	13%
PAHs	2.3E-01				100%	7.5E-04	2.2E-10	5.4E-10	5.2E-11		--	
								TOTAL	**5.2E-11**		**0.000505**	

Exposure to Chemicals via Incidental Ingestion of Soil - Max Commercial

$$Daily\ Chemical\ Intake_{IS} = C_S \bullet \frac{IR_S \bullet FI \bullet CF \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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
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 2009
Averaging Time - Threshold (Atn, days)	2190	USEPA 2009

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	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)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.1E-02	6.4E-09	7.4E-08	--		0.0002319	5%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	7.7E-03	4.4E-09	5.1E-08	--		0.0002853	6%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.9E-02	1.1E-08	1.3E-07	--		0.000523	11%
Antimony (Sb)		8.6E-04		8.6E-04	100%	2.0E-02	1.1E-08	1.3E-07	--		0.0001543	3%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	8.7E-03	5.0E-09	5.8E-08	--		0.0000581	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.1E-01	6.5E-08	7.6E-07	--		0.002520	53%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.8E-02	1.6E-08	1.9E-07	--		0.00023425	5%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	7.1E-03	4.0E-09	4.7E-08	--		0.00004203	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.5E-02	1.4E-08	1.7E-07	--		0.00000297	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	4.3E-02	2.4E-08	2.8E-07	--		0.00000406	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.4E-02	2.0E-08	2.3E-07	--		0.00004756	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	3.5E-03	2.0E-09	2.4E-08	--		0.00001177	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	9.5E-08	5.4E-14	6.3E-13	--		0.000600	13%
PAHs	2.3E-01				100%	7.5E-04	4.3E-10	5.0E-09	1.0E-10		--	
								TOTAL	**1.0E-10**		**0.00472**	

Dermal Exposure to Chemicals via Contact with Soil - Max Commercial

$$Daily\ Chemical\ Intake_{DS} = C_S \bullet \frac{SA_S \bullet AF \bullet FE \bullet ABS \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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
Exposure Duration (ED, years)	29	Time living at one residence
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 2009
Averaging Time - Threshold (Atn, days)	10585	USEPA 2009

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	1.1E-02	2.1E-10	5.0E-10	--		0.000001565	1%
Thallium (Tl)		2.0E-04	10%	1.8E-04		7.7E-03			--		--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		1.9E-02			--		--	
Antimony (Sb)		8.6E-04		8.6E-04		2.0E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.03	8.7E-03	4.9E-09	1.2E-08	--		0.000011758	9%
Lead (Pb)		6.0E-04	50%	3.0E-04		1.1E-01			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		2.8E-02			--		--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		7.1E-03			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		2.5E-02			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		4.3E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		3.4E-02			--		--	
Vanadium (V)		2.0E-03		2.0E-03		3.5E-03			--		--	
Dioxins and furans		2.3E-09	54%	1.1E-09	0.03	9.5E-08	5.3E-14	1.3E-13	--		0.0001215	90%
PAHs	2.3E-01				0.06	7.5E-04	8.4E-10	2.0E-09	2.0E-10		--	
								TOTAL	**2.0E-10**		**0.0001348**	

Dermal Exposure to Chemicals via Contact with Soil - Max Commercial

$$Daily\ Chemical\ Intake_{DS} = C_S \bullet \frac{SA_S \bullet AF \bullet FE \bullet ABS \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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)	240	Days at home
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 2009
Averaging Time - Threshold (Atn, days)	2190	USEPA 2009

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	1.1E-02	5.6E-11	6.6E-10	--		0.00000206	1%
Thallium (Tl)		2.0E-04	10%	1.8E-04		7.7E-03			--		--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		1.9E-02			--		--	
Antimony (Sb)		8.6E-04		8.6E-04		2.0E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.03	8.7E-03	1.3E-09	1.5E-08	--		0.00001546	9%
Lead (Pb)		6.0E-04	50%	3.0E-04		1.1E-01			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		2.8E-02			--		--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		7.1E-03			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		2.5E-02			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		4.3E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		3.4E-02			--		--	
Vanadium (V)		2.0E-03		2.0E-03		3.5E-03			--		--	
Dioxins and furans		2.3E-09	54%	1.1E-09	0.03	9.5E-08	1.4E-14	1.7E-13	--		0.0001597	90%
PAHs	2.3E-01				0.06	7.5E-04	2.3E-10	2.7E-09	5.3E-11		--	
								TOTAL	**5.3E-11**		**0.0001772**	

Calculation of Concentrations in Plants

ref: Stevens B. (1991)

Uptake Due to Deposition in Aboveground Crops

$$C_p = \frac{DR \bullet F \bullet \left[1 - e^{-k \bullet t}\right]}{Y \bullet k}$$ (mg/kg plant – wet weight)

where:

DR= Particle deposition rate for accidental release (mg/m^2/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/m^2)

Uptake via Roots from Soil

$$C_{rp} = C_s \bullet RUF$$ (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

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	7.0E-06	3.5E-06	0.125	7.4E-04	9.3E-05
Thallium (Tl)	14	0.05	4.8E-06	2.4E-06	0.001	5.1E-04	5.1E-07
Mercury (Hg)	14	0.05	1.2E-05	5.9E-06	0.225	1.3E-03	2.8E-04
Antimony (Sb)	14	0.05	1.2E-05	6.2E-06	0.05	1.3E-03	6.6E-05
Arsenic (As)	14	0.05	5.5E-06	2.7E-06	0.01	5.8E-04	5.8E-06
Lead (Pb)	14	0.05	7.1E-05	3.5E-05	0.0113	7.6E-03	8.5E-05
Chromium (Cr VI assumed)	14	0.05	1.8E-05	8.9E-06	0.00188	1.9E-03	3.6E-06
Cobalt (Co)	14	0.05	4.4E-06	2.2E-06	0.005	4.7E-04	2.4E-06
Copper (Cu)	14	0.05	1.6E-05	7.8E-06	0.1	1.7E-03	1.7E-04
Manganese (Mn)	14	0.05	2.7E-05	1.3E-05	0.0625	2.8E-03	1.8E-04
Nickel (Ni)	14	0.05	2.1E-05	1.1E-05	0.015	2.3E-03	3.4E-05
Vanadium (V)	14	0.05	2.2E-06	1.1E-06	0.00138	2.4E-04	3.2E-07
Dioxins and furans	14	0.05	2.0E-10	1.0E-10	0.000876	6.3E-09	5.6E-12
PAHs	14	0.05	1.9E-05	9.7E-06	0.00214	5.0E-05	1.1E-07

$ 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 - Max Commercial as residential

$$i \quad e \; i \quad i \quad ke = \quad x \; \frac{R \quad x \quad x \quad x \quad x \quad x}{B \quad x} \qquad R \; x \; \frac{R_p \; x \; R \quad x \quad x \quad x \quad x}{B \quad x} \qquad \text{(mg/kg/day)}$$

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 NEPM (2013)
Proportion of total intake from aboveground crops (%A	73%	Proportions as per NEPM (2013)
Proportion of total intake from root crops (%R)	27%	Proportions as per NEPM (2013)
Fraction ingested that is homegrown (%)	10%	Relevant to urban areas as per NEPM (2013)
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 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	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.5E-06	9.3E-05	6.5E-09	1.6E-08	--		0.0000492	12%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	2.4E-06	5.1E-07	4.5E-10	1.1E-09	--		0.000006019	1%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	5.9E-06	2.8E-04	1.9E-08	4.6E-08	--		0.0001918	47%
Antimony (Sb)		8.6E-04		8.6E-04	100%	6.2E-06	6.6E-05	5.3E-09	1.3E-08	--		0.00001492	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	2.7E-06	5.8E-06	8.4E-10	2.0E-09	--		0.000002031	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	3.5E-05	8.5E-05	1.2E-08	2.8E-08	--		0.0000932	23%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	8.9E-06	3.6E-06	1.8E-09	4.3E-09	--		0.00000526	1%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	2.2E-06	2.4E-06	5.3E-10	1.3E-09	--		0.0000011459	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	7.8E-06	1.7E-04	1.2E-08	2.9E-08	--		0.0000005159	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.3E-05	1.8E-04	1.4E-08	3.3E-08	--		0.0000004704	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.1E-05	3.4E-05	4.0E-09	9.7E-09	--		0.000002030	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	1.1E-06	3.2E-07	2.1E-10	5.1E-10	--		0.0000002551	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.0E-10	5.6E-12	1.8E-14	4.3E-14	--		0.00004030	10%
PAHs	2.3E-01				100%	9.7E-06	1.1E-07	1.7E-09	4.1E-09	3.9E-10		--	
							TOTAL			**3.9E-10**		**0.000407**	

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables - Max Commercial as residential

$$i \quad e \; i \quad i \quad ke = \quad x \; \frac{R \quad x \quad x \quad x \quad x \quad x}{B \quad x} \qquad R \; x \; \frac{R_p \; x \; R \quad x \quad x \quad x \quad x}{B \quad x} \qquad \text{(mg/kg/day)}$$

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 NEPM (2013)
Proportion of total intake from aboveground crops (%A	84%	Proportions as per NEPM (2013)
Proportion of total intake from root crops (%R)	16%	Proportions as per NEPM (2013)
Fraction ingested that is homegrown (%)	10%	Relevant to urban areas as per NEPM (2013)
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 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	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.5E-06	9.3E-05	2.8E-09	3.3E-08	--		0.0001036	10%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	2.4E-06	5.1E-07	3.4E-10	3.9E-09	--		0.00002182	2%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	5.9E-06	2.8E-04	8.0E-09	9.4E-08	--		0.0003900	39%
Antimony (Sb)		8.6E-04		8.6E-04	100%	6.2E-06	6.6E-05	2.5E-09	3.0E-08	--		0.00003437	3%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	2.7E-06	5.8E-06	5.1E-10	6.0E-09	--		0.000006002	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	3.5E-05	8.5E-05	7.0E-09	8.1E-08	--		0.000270	27%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	8.9E-06	3.6E-06	1.3E-09	1.5E-08	--		0.00001853	2%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	2.2E-06	2.4E-06	3.6E-10	4.2E-09	--		0.000003716	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	7.8E-06	1.7E-04	5.3E-09	6.2E-08	--		0.000001105	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.3E-05	1.8E-04	6.3E-09	7.4E-08	--		0.000001055	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.1E-05	3.4E-05	2.3E-09	2.7E-08	--		0.000005626	1%
Vanadium (V)		2.0E-03		2.0E-03	100%	1.1E-06	3.2E-07	1.6E-10	1.8E-09	--		0.000000913	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.0E-10	5.6E-12	1.4E-14	1.6E-13			0.0001500	15%
PAHs	2.3E-01				100%	9.7E-06	1.1E-07	1.3E-09	1.5E-08	3.0E-10		--	
									TOTAL	**3.0E-10**		**0.001007**	

Calculation of Concentrations in Eggs

Uptake in to chicken eggs

=(x R x R_S x $_S$ x B) x (mg/kg egg – wet weight)

where:
FI = Fraction of pasture/crop ingested by chickens each day (unitless)
IRc = Ingestion rate of pasture/crop by chicken each day (kg/day)
C = Concentration of chemical in grain/crop eaten by chicken (mg/kg)
IRs = Ingestion rate of soil by chickens each day (kg/day)
Cs = 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	Assume 100% of crops consumed by chickens is grown in the same soil
IRc (ingestion rate of crops)	kg/day	0.12	Assumed ingestion rate from OEHHA 2015 (assume concentration the same as predicted for aboveground crops)
IRs (ingestion rate of soil)	kg/day	0.01	USEPA (2005) (Ag Victoria recommendation)
B (bioavailability)	%	100%	

Chemical-specific Inputs and calculations - Grid Maximum Off-site

Chemical	Concentration in crops ingested by chickens mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to eggs day/kg	Egg Concentration mg/kg ww	
Cadmium (Cd)	3.5E-06	7.4E-04	1.0E-02	7.8E-08	OEHHA (2015)
Thallium (Tl)	2.4E-06	5.1E-04	1.7E-02	9.1E-08	95% from Leeman et al (2007)
Mercury (Hg)	5.9E-06	1.3E-03	9.0E-02	1.2E-06	OEHHA (2015)
Antimony (Sb)	6.2E-06	1.3E-03	1.7E-01	2.4E-06	95% from Leeman et al (2007)
Arsenic (As)	2.7E-06	5.8E-04	7.0E-02	4.3E-07	OEHHA (2015)
Lead (Pb)	3.5E-05	7.6E-03	4.0E-02	3.2E-06	OEHHA (2015)
Chromium (Cr VI assumed)	8.9E-06	1.9E-03	9.2E-03	1.8E-07	OEHHA (2015)
Cobalt (Co)	2.2E-06	4.7E-04	3.3E-03	1.6E-08	MacLachlan (2011)
Copper (Cu)	7.8E-06	1.7E-03	1.7E-01	3.0E-06	95% from Leeman et al (2007)
Manganese (Mn)	1.3E-05	2.8E-03	1.7E-01	5.1E-06	95% from Leeman et al (2007)
Nickel (Ni)	1.1E-05	2.3E-03	2.0E-02	4.8E-07	OEHHA (2015)
Vanadium (V)	1.1E-06	2.4E-04	1.7E-01	4.2E-07	95% from Leeman et al (2007)
Dioxins and furans	1.0E-10	6.3E-09	1.0E+01	7.5E-10	OEHHA (2015)
PAHs	9.7E-06	5.0E-05	3.0E-03	5.0E-09	OEHHA (2015)

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Eggs - Max Commercial as residential

$$i\ e\ i\ i\ ke = {}_{E}x\ \frac{R_E\ x\ x\ x\ x}{B\ x} \qquad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Eggs (IRE) (kg/day)	0.014	Ingestion rate of eggs relevant for adults as per enHealth (2012)
Fraction ingested that is homegrown (%)	100%	Assume all eggs consumed in urban area 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 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	ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	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)		(mg/kg wet weight)	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%	7.8E-08	6.5E-12	1.6E-11	--		0.0000000490	0%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	9.1E-08	7.6E-12	1.8E-11	--		0.00000010126	0%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.2E-06	9.9E-11	2.4E-10	--		0.000000995	1%
Antimony (Sb)		8.6E-04		8.6E-04	100%	2.4E-06	2.0E-10	4.8E-10	--		0.000000554	0%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	4.3E-07	3.6E-11	8.6E-11	--		0.00000008586	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	3.2E-06	2.6E-10	6.4E-10	--		0.000002130	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	1.8E-07	1.5E-11	3.7E-11	--		0.0000000455	0%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.6E-08	1.4E-12	3.3E-12	--		0.000000002930	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	3.0E-06	2.5E-10	6.0E-10	--		0.00000001066	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	5.1E-06	4.2E-10	1.0E-09	--		0.00000001456	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.8E-07	4.0E-11	9.6E-11	--		0.00000002009	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	4.2E-07	3.5E-11	8.5E-11	--		0.00000004226	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	7.5E-10	6.3E-14	1.5E-13	--		0.0001427	97%
PAHs	2.3E-01				100%	5.0E-09	4.1E-13	1.0E-12	9.7E-14		--	
								TOTAL	**9.7E-14**		**0.0001467**	

Exposure to Chemicals via Ingestion of Eggs - Max Commercial as residential

$$i\ e\ i\ i\ ke = {}_{E}x\ \frac{R_E\ x\ x\ x\ x}{B\ x} \qquad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Young children		
Ingestion Rate of Eggs (IRE) (kg/day)	0.006	Ingestion rate of eggs relevant for young children as per enHealth (2012)
Fraction ingested that is homegrown (%)	100%	Assume all eggs consumed in urban area 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 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	ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	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)		(mg/kg wet weight)	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%	7.8E-08	2.7E-12	3.1E-11	--		0.0000000980	0%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	9.1E-08	3.1E-12	3.6E-11	--		0.0000002025	0%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.2E-06	4.1E-11	4.8E-10	--		0.000001989	1%
Antimony (Sb)		8.6E-04		8.6E-04	100%	2.4E-06	8.2E-11	9.5E-10	--		0.00000111	0%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	4.3E-07	1.5E-11	1.7E-10	--		0.00000017173	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	3.2E-06	1.1E-10	1.3E-09	--		0.00000426	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	1.8E-07	6.3E-12	7.4E-11	--		0.0000000911	0%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.6E-08	5.6E-13	6.6E-12	--		0.000000005860	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	3.0E-06	1.0E-10	1.2E-09	--		0.00000002132	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	5.1E-06	1.7E-10	2.0E-09	--		0.00000002913	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.8E-07	1.7E-11	1.9E-10	--		0.00000004018	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	4.2E-07	1.4E-11	1.7E-10	--		0.00000008452	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	7.5E-10	2.6E-14	3.0E-13	--		0.0002854	97%
PAHs	2.3E-01				100%	5.0E-09	1.7E-13	2.0E-12	4.0E-14		--	
								TOTAL	**4.0E-14**		**0.0002935**	

Maximum Other Location

Predicted ground level concentrations - chronic exposures - cumulative case

	Air Concentration - annual average ($\mu g/m^3$)	Air Concentration - annual average (mg/m^3)	Exposure pathways							
Key Chemicals	**Max other**	**Max other**	**inhalation**	**soil ingestion**	**soil - dermal**	**egg ingestion**	**fruit and vegetable ingestion**	**rainwater tank**	**meat ingestion**	**milk ingestion**
Hydrogen chloride (HCl)	0.312	0.000312	✓	×	×	×	×	×	×	×
Hydrogen fluoride (HF)	0.0176	0.0000176	✓	×	×	×	×	×	×	×
Ammonia	0.00618	0.00000618	✓	×	×	×	×	×	×	×
Cadmium (Cd)	0.00001	0.000000010	✓	✓	✓	✓	✓	×	×	×
Thallium (Tl)	0.00001	0.000000010	✓	✓	✓	✓	✓	×	×	×
Mercury (Hg)	0.00001	0.000000010	✓	✓	✓	✓	✓	×	×	×
Antimony (Sb)	0.00001	0.000000010	✓	✓	✓	✓	✓	×	×	×
Arsenic (As)	0.00001	0.000000010	✓	✓	✓	✓	✓	×	×	×
Lead (Pb)	0.0001	0.000000100	✓	✓	✓	✓	✓	×	×	×
Chromium (Cr VI assumed)	0.00004	0.00000004	✓	✓	✓	✓	✓	×	×	×
Cobalt (Co)	0.00001	0.000000010	✓	✓	✓	✓	✓	×	×	×
Copper (Cu)	0.00002	0.00000002	✓	✓	✓	✓	✓	×	×	×
Manganese (Mn)	0.00006	0.00000006	✓	✓	✓	✓	✓	×	×	×
Nickel (Ni)	0.00003	0.00000003	✓	✓	✓	✓	✓	×	×	×
Vanadium (V)	0.00001	0.000000010	✓	✓	✓	✓	✓	×	×	×
Dioxins and furans	3.80E-11	3.8E-14	✓	✓	✓	✓	✓	×	×	×
Benzene	0.00723	0.00000723	✓	×	×	×	×	×	×	×
Formaldehyde	0.00618	0.00000618	✓	×	×	×	×	×	×	×
Polycyclic aromatic hydrocarbons (PAHs)	0.0000012	1.2E-09	✓	✓	✓	✓	✓	×	×	×

	Deposition Rate - annual average ($ng/m^2/year$)	Deposition Rate - annual average ($mg/m^2/year$)
Key Chemicals	**Max other**	**Max other**
Cadmium (Cd)	2544	0.002544
Thallium (Tl)	1761	0.001761
Mercury (Hg)	4305	0.004305
Antimony (Sb)	4549	0.004549
Arsenic (As)	1991	0.001991
Lead (Pb)	25926	0.025926
Chromium (Cr VI assumed)	6506	0.006506
Cobalt (Co)	1614	0.001614
Copper (Cu)	5699	0.005699
Manganese (Mn)	9734	0.009734
Nickel (Ni)	7827	0.007827
Vanadium (V)	807	0.000807
Dioxins and furans	NR	7.326E-08
Polycyclic aromatic hydrocarbons (PAHs)	NR	1.892E-03

Inhalation - gases and particulates

Maximum other location (i.e. maximum concentrations at school, childcare, hospital, worship, aged care sites around the facility) (as residential)

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * EF * ED}{AT}$$ (mg/m^3) for gases

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * DRF * EF * ED}{AT}$$ (mg/m^3) 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 (NEPM 1999 amended 2013) - NA for gasses
Exposure Frequency - normal conditions (EF, days/yr)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	35	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	306600	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	3.1E-04	1.6E-04	3.1E-04	–		0.0120	67%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	1.8E-05	8.8E-06	1.8E-05	–		0.000607	3%
Ammonia		3.2E-01	0%	3.2E-01	6.2E-06	3.1E-06	6.2E-06	–		0.0000193	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	1.0E-08	1.9E-09	3.8E-09	–		0.000938	5%
Thallium (Tl)		7.0E-04	10%	6.3E-04	1.0E-08	1.9E-09	3.8E-09	–		0.00000595	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	1.0E-08	1.9E-09	3.8E-09	–		0.0000188	0%
Antimony (Sb)		3.0E-04	0%	3.0E-04	1.0E-08	1.9E-09	3.8E-09	–		0.0000125	0%
Arsenic (As)		6.7E-05	0%	6.7E-05	1.0E-08	1.9E-09	3.8E-09	–		0.0000560	0%
Lead (Pb)		5.0E-04	0%	5.0E-04	1.0E-07	1.9E-08	3.8E-08	–		0.0000750	0%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	4.0E-08	7.5E-09	1.5E-08	–		0.00349	19%
Cobalt (Co)		1.0E-04	0%	1.0E-04	1.0E-08	1.9E-09	3.8E-09	–		0.0000375	0%
Copper (Cu)		4.9E-01	60%	2.0E-01	2.0E-08	3.8E-09	7.5E-09	–		0.0000000383	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	6.0E-08	1.1E-08	2.3E-08	–		0.000188	1%
Nickel (Ni)		5.9E-05	10%	5.3E-05	3.0E-08	5.6E-09	1.1E-08	–		0.000212	1%
Vanadium (V)		1.0E-04	0%	1.0E-04	1.0E-08	1.9E-09	3.8E-09	–		0.0000375	0%
Dioxins and furans		8.1E-09	54%	3.7E-09	3.8E-14	7.1E-15	1.4E-14	–		0.00000385	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	7.2E-06	1.4E-06	2.7E-06	8.1E-9	98%	0.000100	1%
Formaldehyde		7.0E-03	0%	7.0E-03	6.2E-06	1.2E-06	2.3E-06	–		0.000331	2%
PAHs	6.0E-01				1.2E-09	2.3E-10	4.5E-10	1.4E-10	2%	–	
TOTAL								**8.3E-09**		**0.0180**	

Inhalation - gases and particulates

Maximum other location (i.e. maximum concentrations at school, childcare, hospital, worship, aged care sites around the facility) assuming a workplace

$$InhalationExposureConc_V = C_a \bullet \frac{ET \bullet FI \bullet EF \bullet ED}{AT} \quad (mg/m^3)$$

Parameters Relevant to Quantification of Community Exposures - Workers		
Exposure Time at Home (ET, hr/day)	8	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 (NEPM 1999 amended 2013) - NA for gasses
Exposure Frequency - normal conditions (EF, days/yr)	240	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
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	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	3.1E-04	1.1E-05	2.6E-05	--		0.00098630	43%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	1.8E-05	6.2E-07	1.4E-06	--		0.00004988	2%
Ammonia		3.2E-01	0%	3.2E-01	6.2E-06	2.2E-07	5.1E-07	--		0.00000159	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	1.0E-08	3.5E-10	8.2E-10	--		0.00020548	9%
Thallium (Tl)		7.0E-04	10%	6.3E-04	1.0E-08	3.5E-10	8.2E-10	--		0.00000130	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	1.0E-08	3.5E-10	8.2E-10	--		0.00000411	0%
Antimony (Sb)		3.0E-04	0%	3.0E-04	1.0E-08	3.5E-10	8.2E-10	--		0.00000274	0%
Arsenic (As)		6.7E-05	0%	6.7E-05	1.0E-08	9.4E-10	2.2E-09	--		0.00003271	1%
Lead (Pb)		5.0E-04	0%	5.0E-04	1.0E-07	9.4E-09	2.2E-08	--		0.00004384	2%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	4.0E-08	1.4E-09	3.3E-09	--		0.00076457	33%
Cobalt (Co)		1.0E-04	0%	1.0E-04	1.0E-08	3.5E-10	8.2E-10	--		0.00000822	0%
Copper (Cu)		4.9E-01	60%	2.0E-01	2.0E-08	7.0E-10	1.6E-09	--		0.00000000839	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	6.0E-08	2.1E-09	4.9E-09	--		0.00004110	2%
Nickel (Ni)		5.9E-05	10%	5.3E-05	3.0E-08	1.1E-09	2.5E-09	--		0.00004644	2%
Vanadium (V)		1.0E-04	0%	1.0E-04	1.0E-08	3.5E-10	8.2E-10	--		0.00008219	0%
Dioxins and furans		8.1E-09	54%	3.7E-09	3.8E-14	1.3E-15	3.1E-15	--		0.000000843	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	7.2E-06	2.5E-07	5.9E-07	1.5E-9	98%	0.000022009	1%
Formaldehyde		7.0E-03	0%	7.0E-03	6.2E-06	2.2E-07	5.1E-07	--		0.000072564	3%
PAHs	6.0E-01				1.2E-09	4.2E-11	9.9E-11	2.5E-11	2%	--	
							TOTAL	**1.6E-09**		**0.00229**	

Calculation of Concentrations in Soil

$$C_s = \frac{DR \bullet \left[1 - e^{-k \bullet t}\right]}{d \bullet \rho \bullet k} \bullet 1000 \quad \textbf{(mg/kg)}$$ ref: Stevens B. (1991)

where:

DR=	Particle deposition rate (mg/m^2/year)
K =	Chemical-specific soil-loss constant (1/year) = ln(2)/T0.5
T0.5 =	Chemical half-life in soil (years)
t =	Accumulation time (years)
d =	Soil mixing depth (m)
ρ =	Soil bulk-density (g/m^3)
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 OEHHA (2015) guidance
Duration of deposition (T)	years	70	70	As per OEHHA (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	9.0E-05	3.9E-04	2.6E-05
Thallium (Tl)	273973	2.5E-06	7.0E-05	3.0E-04	2.0E-05
Mercury (Hg)	273973	2.5E-06	9.3E-05	4.1E-04	2.7E-05
Antimony (Sb)	273973	2.5E-06	9.4E-05	4.1E-04	2.7E-05
Arsenic (As)	273973	2.5E-06	9.9E-05	4.3E-04	2.9E-05
Lead (Pb)	273973	2.5E-06	8.7E-04	3.8E-03	2.5E-04
Chromium (Cr VI assumed)	273973	2.5E-06	2.0E-04	8.7E-04	5.8E-05
Cobalt (Co)	273973	2.5E-06	6.4E-05	2.8E-04	1.9E-05
Copper (Cu)	273973	2.5E-06	9.7E-05	4.2E-04	2.8E-05
Manganese (Mn)	273973	2.5E-06	7.0E-04	3.1E-03	2.0E-04
Nickel (Ni)	273973	2.5E-06	1.0E-04	4.4E-04	2.9E-05
Vanadium (V)	273973	2.5E-06	9.9E-06	4.3E-05	2.9E-06
Dioxins and furans	15.00	4.6E-02	2.5E-09	3.2E-09	2.2E-10
PAHs	1.18	0.588	1.9E-03	2.0E-04	1.3E-05

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: OEHHA 2015

Exposure to Chemicals via Incidental Ingestion of Soil (Maximum other location (i.e. maximum concentrations at school, childcare, hospital, worship, aged care sites around the facility)) as residential

$$Daily\ Chemical\ Intake_{IS} = C_S \bullet \frac{IR_S \bullet FI \bullet CF \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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	As per ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	As per ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	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)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.9E-04	1.2E-10	2.8E-10	--		0.0000008739	5%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	3.0E-04	9.0E-11	2.2E-10	--		0.0000012065	7%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	4.1E-04	1.2E-10	2.9E-10	--		0.0000012160	7%
Antimony (Sb)		8.6E-04		8.6E-04	100%	4.1E-04	1.2E-10	2.9E-10	--		0.0000003415	2%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	4.3E-04	1.3E-10	3.1E-10	--		0.0000003106	2%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	3.8E-03	1.1E-09	2.7E-09	--		0.0000090721	56%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	8.7E-04	2.6E-10	6.2E-10	--		0.0000007646	5%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	2.8E-04	8.2E-11	2.0E-10	--		0.0000001777	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	4.2E-04	1.3E-10	3.0E-10	--		0.0000000054	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.1E-03	9.1E-10	2.2E-09	--		0.0000000313	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.4E-04	1.3E-10	3.1E-10	--		0.0000000648	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	4.3E-05	1.3E-11	3.1E-11	--		0.0000000155	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	3.2E-09	9.6E-16	2.3E-15	--		0.0000021815	13%
PAHs	2.3E-01				100%	2.0E-04	5.9E-11	1.4E-10	1.4E-11		--	
								TOTAL	**1.4E-11**		**0.0000163**	

Exposure to Chemicals via Incidental Ingestion of Soil (Maximum other location (i.e. maximum concentrations at school, childcare, hospital, worship, aged care sites around the facility)) as residential

$$Daily\ Chemical\ Intake_{IS} = C_S \bullet \frac{IR_S \bullet FI \bullet CF \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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	As per ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	As per ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.9E-04	2.2E-10	2.6E-09	--		0.00000816	5%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	3.0E-04	1.7E-10	2.0E-09	--		0.00001126	7%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	4.1E-04	2.3E-10	2.7E-09	--		0.00001135	7%
Antimony (Sb)		8.6E-04		8.6E-04	100%	4.1E-04	2.3E-10	2.7E-09	--		0.00000319	2%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	4.3E-04	2.5E-10	2.9E-09	--		0.00000290	2%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	3.8E-03	2.2E-09	2.5E-08	--		0.00008467	56%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	8.7E-04	5.0E-10	5.8E-09	--		0.00000714	5%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	2.8E-04	1.6E-10	1.9E-09	--		0.00000166	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	4.2E-04	2.4E-10	2.8E-09	--		0.00000005	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.1E-03	1.8E-09	2.0E-08	--		0.00000029	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.4E-04	2.5E-10	2.9E-09	--		0.00000060	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	4.3E-05	2.5E-11	2.9E-10	--		0.00000014	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	3.2E-09	1.8E-15	2.2E-14	--		0.00002036	13%
PAHs	2.3E-01				100%	2.0E-04	1.1E-10	1.3E-09	2.7E-11		--	
								TOTAL	**2.7E-11**		**0.000152**	

Dermal Exposure to Chemicals via Contact with Soil (Maximum other location (i.e. maximum concentrations at school, childcare, hospital, worship, aged care sites around the facility)) as residential

$$Daily\ Chemical\ Intake_{DS} = C_S \bullet \frac{SA_S \bullet AF \bullet FE \bullet ABS \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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	As per ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	As per ASC NEPM

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	3.9E-04	7.3E-12	1.8E-11	--		0.0000000551	1%
Thallium (Tl)		2.0E-04	10%	1.8E-04		3.0E-04			--		--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		4.1E-04			--		--	
Antimony (Sb)		8.6E-04		8.6E-04		4.1E-04			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.03	4.3E-04	2.4E-10	5.9E-10	--		0.000000587	12%
Lead (Pb)		6.0E-04	50%	3.0E-04		3.8E-03			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		8.7E-04			--		--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		2.8E-04			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		4.2E-04			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		3.1E-03			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		4.4E-04			--		--	
Vanadium (V)		2.0E-03		2.0E-03		4.3E-05			--		--	
Dioxins and furans		2.3E-09	54%	1.1E-09	0.03	3.2E-09	1.8E-15	4.4E-15	--		0.00000412	87%
PAHs	2.3E-01				0.06	2.0E-04	2.2E-10	5.4E-10	5.2E-11		--	
								TOTAL	**5.2E-11**		**0.00000477**	

Dermal Exposure to Chemicals via Contact with Soil (Maximum other location (i.e. maximum concentrations at school, childcare, hospital, worship, aged care sites around the facility)) as residential

$$Daily\ Chemical\ Intake_{DS} = C_S \bullet \frac{SA_S \bullet AF \bullet FE \bullet ABS \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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	As per ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	As per ASC NEPM

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	0.001	3.9E-04	3.0E-12	3.5E-11	--		0.000000110	1%
Thallium (Tl)		2.0E-04	10%	1.8E-04		3.0E-04			--		--	
Mercury (Hg)		6.0E-04	60%	2.4E-04		4.1E-04			--		--	
Antimony (Sb)		8.6E-04		8.6E-04		4.1E-04			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.03	4.3E-04	1.0E-10	1.2E-09	--		0.00000117	12%
Lead (Pb)		6.0E-04	50%	3.0E-04		3.8E-03			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		8.7E-04			--		--	
Cobalt (Co)		1.4E-03	20%	1.1E-03		2.8E-04			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		4.2E-04			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		3.1E-03			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03		4.4E-04			--		--	
Vanadium (V)		2.0E-03		2.0E-03		4.3E-05			--		--	
Dioxins and furans		2.3E-09	54%	1.1E-09	0.03	3.2E-09	7.5E-16	8.7E-15	--		0.00000825	87%
PAHs	2.3E-01				0.06	2.0E-04	9.3E-11	1.1E-09	2.2E-11		--	
								TOTAL	**2.2E-11**		**#########**	

Calculation of Concentrations in Plants

ref: Stevens B. (1991)

Uptake Due to Deposition in Aboveground Crops

$$C_p = \frac{DR \bullet F \bullet \left[1 - e^{-k \bullet t}\right]}{Y \bullet k}$$ (mg/kg plant – wet weight)

where:
DR= Particle deposition rate for accidental release (mg/m^2/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/m^2)

Uptake via Roots from Soil

$$C_{rp} = C_s \bullet RUF$$ (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

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	2.5E-07	1.2E-07	0.125	2.6E-05	3.3E-06
Thallium (Tl)	14	0.05	1.9E-07	9.5E-08	0.001	2.0E-05	2.0E-08
Mercury (Hg)	14	0.05	2.6E-07	1.3E-07	0.225	2.7E-05	6.1E-06
Antimony (Sb)	14	0.05	2.6E-07	1.3E-07	0.05	2.7E-05	1.4E-06
Arsenic (As)	14	0.05	2.7E-07	1.4E-07	0.01	2.9E-05	2.9E-07
Lead (Pb)	14	0.05	2.4E-06	1.2E-06	0.0113	2.5E-04	2.9E-06
Chromium (Cr VI assumed)	14	0.05	5.4E-07	2.7E-07	0.00188	5.8E-05	1.1E-07
Cobalt (Co)	14	0.05	1.7E-07	8.7E-08	0.005	1.9E-05	9.3E-08
Copper (Cu)	14	0.05	2.7E-07	1.3E-07	0.1	2.8E-05	2.8E-06
Manganese (Mn)	14	0.05	1.9E-06	9.6E-07	0.0625	2.0E-04	1.3E-05
Nickel (Ni)	14	0.05	2.7E-07	1.4E-07	0.015	2.9E-05	4.4E-07
Vanadium (V)	14	0.05	2.7E-08	1.4E-08	0.00138	2.9E-06	4.0E-09
Dioxins and furans	14	0.05	6.8E-12	3.4E-12	0.000876	2.2E-10	1.9E-13
PAHs	14	0.05	5.2E-06	2.6E-06	0.00214	1.3E-05	2.9E-08

$ 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 other location (i.e. maximum concentrations at school, childcare, hospital, worship, aged care sites around the facility)) as residential

$$i \quad e \; i \quad i \quad ke = \quad x \; \frac{R \; x \quad x \quad x \quad x \quad x}{B \; x} \qquad R \; x \; \frac{R_p \; x \; R \; x \quad x \quad x \quad x}{B \; x} \qquad \text{(mg/kg/day)}$$

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 NEPM (2013)
Proportion of total intake from aboveground crops (%A	73%	Proportions as per NEPM (2013)
Proportion of total intake from root crops (%R)	27%	Proportions as per NEPM (2013)
Fraction ingested that is homegrown (%)	10%	Relevant to urban areas as per NEPM (2013)
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 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	As per ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	As per ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.2E-07	3.3E-06	2.3E-10	5.5E-10	--		0.0000017326	15%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	9.5E-08	2.0E-08	1.8E-11	4.3E-11	--		0.0000002375	2%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.3E-07	6.1E-06	4.1E-10	1.0E-09	--		0.0000041619	37%
Antimony (Sb)		8.6E-04		8.6E-04	100%	1.3E-07	1.4E-06	1.1E-10	2.7E-10	--		0.0000003082	3%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.4E-07	2.9E-07	4.2E-11	1.0E-10	--		0.0000001014	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.2E-06	2.9E-06	3.9E-10	9.4E-10	--		0.0000031318	28%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.7E-07	1.1E-07	5.4E-11	1.3E-10	--		0.0000001602	1%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	8.7E-08	9.3E-08	2.1E-11	5.1E-11	--		0.0000000452	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.3E-07	2.8E-06	2.0E-10	4.9E-10	--		0.0000000088	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	9.6E-07	1.3E-05	9.8E-10	2.4E-09	--		0.0000000339	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.4E-07	4.4E-07	5.1E-11	1.2E-10	--		0.0000000258	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	1.4E-08	4.0E-09	2.6E-12	6.3E-12	--		0.0000000031	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	3.4E-12	1.9E-13	6.0E-16	1.4E-15	--		0.0000013679	12%
PAHs	2.3E-01				100%	2.6E-06	2.9E-08	4.5E-10	1.1E-09	1.0E-10		--	
									TOTAL	**1.0E-10**		**0.0000113**	

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables (Maximum other location (i.e. maximum concentrations at school, childcare, hospital, worship, aged care sites around the facility)) as residential

$$i \quad e \; i \quad i \quad ke = \quad x \; \frac{R \; x \quad x \quad x \quad x \quad x}{B \; x} \qquad R \; x \; \frac{R_p \; x \; R \; x \quad x \quad x \quad x}{B \; x} \qquad \text{(mg/kg/day)}$$

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 NEPM (2013)
Proportion of total intake from aboveground crops (%A	84%	Proportions as per NEPM (2013)
Proportion of total intake from root crops (%R)	16%	Proportions as per NEPM (2013)
Fraction ingested that is homegrown (%)	10%	Relevant to urban areas as per NEPM (2013)
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 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	As per ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	As per ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.2E-07	3.3E-06	1.0E-10	1.2E-09	--		0.000003645	13%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	9.5E-08	2.0E-08	1.3E-11	1.6E-10	--		0.000000861	3%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	1.3E-07	6.1E-06	1.7E-10	2.0E-09	--		0.000008461	29%
Antimony (Sb)		8.6E-04		8.6E-04	100%	1.3E-07	1.4E-06	5.2E-11	6.1E-10	--		0.000000710	2%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.4E-07	2.9E-07	2.6E-11	3.0E-10	--		0.000000300	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.2E-06	2.9E-06	2.3E-10	2.7E-09	--		0.000009081	31%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.7E-07	1.1E-07	3.9E-11	4.6E-10	--		0.000000565	2%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	8.7E-08	9.3E-08	1.4E-11	1.6E-10	--		0.000000147	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.3E-07	2.8E-06	9.0E-11	1.1E-09	--		0.000000019	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	9.6E-07	1.3E-05	4.6E-10	5.3E-09	--		0.000000076	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.4E-07	4.4E-07	2.9E-11	3.4E-10	--		0.000000072	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	1.4E-08	4.0E-09	1.9E-12	2.2E-11	--		0.000000011	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	3.4E-12	1.9E-13	4.6E-16	5.4E-15			0.000005092	18%
PAHs	2.3E-01				100%	2.6E-06	2.9E-08	3.5E-10	4.1E-09	8.1E-11		--	
									TOTAL	**8.1E-11**		**0.0000290**	

Calculation of Concentrations in Eggs

Uptake in to chicken eggs

=(x R x R_S x $_S$ x B) x (mg/kg egg – wet weight)

where:
FI = Fraction of pasture/crop ingested by chickens each day (unitless)
IRc = Ingestion rate of pasture/crop by chicken each day (kg/day)
C = Concentration of chemical in grain/crop eaten by chicken (mg/kg)
IRs = Ingestion rate of soil by chickens each day (kg/day)
Cs = 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	Assume 100% of crops consumed by chickens is grown in the same soil
IRc (ingestion rate of crops)	kg/day	0.12	Assumed ingestion rate from OEHHA 2015 (assume concentration the same as predicted for aboveground crops)
IRs (ingestion rate of soil)	kg/day	0.01	USEPA (2005) (Ag Victoria recommendation)
B (bioavailability)	%	100%	

Chemical-specific Inputs and calculations - Grid Maximum Off-site

Chemical	Concentration in crops ingested by chickens mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to eggs day/kg	Egg Concentration mg/kg ww	
Cadmium (Cd)	1.2E-07	2.6E-05	1.0E-02	2.8E-09	OEHHA (2015)
Thallium (Tl)	9.5E-08	2.0E-05	1.7E-02	3.6E-09	95% from Leeman et al (2007)
Mercury (Hg)	1.3E-07	2.7E-05	9.0E-02	2.6E-08	OEHHA (2015)
Antimony (Sb)	1.3E-07	2.7E-05	1.7E-01	4.9E-08	95% from Leeman et al (2007)
Arsenic (As)	1.4E-07	2.9E-05	7.0E-02	2.1E-08	OEHHA (2015)
Lead (Pb)	1.2E-06	2.5E-04	4.0E-02	1.1E-07	OEHHA (2015)
Chromium (Cr VI assumed)	2.7E-07	5.8E-05	9.2E-03	5.6E-09	OEHHA (2015)
Cobalt (Co)	8.7E-08	1.9E-05	3.3E-03	6.5E-10	MacLachlan (2011)
Copper (Cu)	1.3E-07	2.8E-05	1.7E-01	5.1E-08	95% from Leeman et al (2007)
Manganese (Mn)	9.6E-07	2.0E-04	1.7E-01	3.7E-07	95% from Leeman et al (2007)
Nickel (Ni)	1.4E-07	2.9E-05	2.0E-02	6.1E-09	OEHHA (2015)
Vanadium (V)	1.4E-08	2.9E-06	1.7E-01	5.2E-09	95% from Leeman et al (2007)
Dioxins and furans	3.4E-12	2.2E-10	1.0E+01	2.6E-11	OEHHA (2015)
PAHs	2.6E-06	1.3E-05	3.0E-03	1.3E-09	OEHHA (2015)

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Eggs (Maximum other location (i.e. maximum concentrations at school, childcare, hospital, worship, aged care sites around the facility)) as residential

$$I_{ke} = E_i \times I_i \times \frac{IRE_i \times f_i \times C_{ke}}{B_i \times x}$$

(mg/kg/day)

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Eggs (IRE) (kg/day)	0.014	Ingestion rate of eggs relevant for adults as per enHealth (2012)
Fraction ingested that is homegrown (%)	100%	Assume all eggs consumed in urban area 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 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	As per ASC NEPM
Averaging Time - Threshold (Atn, days)	10585	As per ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	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)		(mg/kg wet weight)	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%	2.8E-09	2.3E-13	5.5E-13	--		0.00000000172	0%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	3.6E-09	3.0E-13	7.2E-13	--		0.00000000400	0%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	2.6E-08	2.1E-12	5.2E-12	--		0.00000002158	0%
Antimony (Sb)		8.6E-04		8.6E-04	100%	4.9E-08	4.1E-12	9.8E-12	--		0.00000001145	0%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	2.1E-08	1.8E-12	4.3E-12	--		0.00000000429	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.1E-07	8.9E-12	2.1E-11	--		0.00000007155	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	5.6E-09	4.7E-13	1.1E-12	--		0.00000000139	0%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	6.5E-10	5.4E-14	1.3E-13	--		0.00000000012	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	5.1E-08	4.2E-12	1.0E-11	--		0.00000000018	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.7E-07	3.0E-11	7.4E-11	--		0.00000000105	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	6.1E-09	5.1E-13	1.2E-12	--		0.00000000026	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	5.2E-09	4.3E-13	1.0E-12	--		0.00000000052	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	2.6E-11	2.1E-15	5.1E-15	--		0.00000484340	98%
PAHs	2.3E-01				100%	1.3E-09	1.1E-13	2.7E-13	2.6E-14		--	
								TOTAL	**2.6E-14**		**0.00000496**	

Exposure to Chemicals via Ingestion of Eggs (Maximum other location (i.e. maximum concentrations at school, childcare, hospital, worship, aged care sites around the facility)) as residential

$$i\ e\ i\ i\ ke = {}_{E}x \frac{R_E\ x\ x\ x\ x}{B\ x} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Young children		
Ingestion Rate of Eggs (IRE) (kg/day)	0.006	Ingestion rate of eggs relevant for young children as per enHealth (2012)
Fraction ingested that is homegrown (%)	100%	Assume all eggs consumed in urban area 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 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	As per ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	As per ASC NEPM

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	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)		(mg/kg wet weight)	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%	2.8E-09	9.5E-14	1.1E-12	--		0.00000000345	0%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	3.6E-09	1.2E-13	1.4E-12	--		0.00000000799	0%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	2.6E-08	8.9E-13	1.0E-11	--		0.00000004316	0%
Antimony (Sb)		8.6E-04		8.6E-04	100%	4.9E-08	1.7E-12	2.0E-11	--		0.00000002290	0%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	2.1E-08	7.3E-13	8.6E-12	--		0.00000000857	0%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.1E-07	3.7E-12	4.3E-11	--		0.00000014310	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	5.6E-09	1.9E-13	2.2E-12	--		0.00000000277	0%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	6.5E-10	2.2E-14	2.6E-13	--		0.00000000023	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	5.1E-08	1.7E-12	2.0E-11	--		0.00000000036	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.7E-07	1.3E-11	1.5E-10	--		0.00000000210	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	6.1E-09	2.1E-13	2.5E-12	--		0.00000000051	0%
Vanadium (V)		2.0E-03		2.0E-03	100%	5.2E-09	1.8E-13	2.1E-12	--		0.00000000104	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	2.6E-11	8.8E-16	1.0E-14	--		0.00000968681	98%
PAHs	2.3E-01				100%	1.3E-09	4.6E-14	5.3E-13	1.1E-14		--	
								TOTAL	**1.1E-14**		**0.00000992**	

On-site Visitors

Predicted ground level concentrations - chronic exposures - cumulative case

	Air Concentration - annual average ($\mu g/m^3$)	Air Concentration - annual average (mg/m^3)	Exposure pathways							
Key Chemicals	**Maximum On-site**	**Maximum On-site**	**inhalation**	**soil ingestion**	**soil - dermal**	**egg ingestion**	**fruit and vegetable ingestion**	**rainwater tank**	**meat ingestion**	**milk ingestion**
Hydrogen chloride (HCl)	1.61E+00	0.00161218	✓	×	×	×	×	×	×	×
Hydrogen fluoride (HF)	2.69E-01	0.0002688	✓	×	×	×	×	×	×	×
Ammonia	3.17E-03	0.00000317	✓	×	×	×	×	×	×	×
Cadmium (Cd)	2.48E-03	0.000002480	✓	×	×	×	×	×	×	×
Thallium (Tl)	2.19E-03	0.000002190	✓	×	×	×	×	×	×	×
Mercury (Hg)	8.11E-03	0.000008110	✓	×	×	×	×	×	×	×
Antimony (Sb)	2.01E-03	0.000002010	✓	×	×	×	×	×	×	×
Arsenic (As)	7.10E-03	0.000007100	✓	×	×	×	×	×	×	×
Lead (Pb)	3.2E-02	0.000032300	✓	×	×	×	×	×	×	×
Chromium (Cr VI assumed)	8.1E-03	0.00000811	✓	×	×	×	×	×	×	×
Cobalt (Co)	2.0E-03	0.000002010	✓	×	×	×	×	×	×	×
Copper (Cu)	7.1E-03	0.0000071	✓	×	×	×	×	×	×	×
Manganese (Mn)	1.2E-02	0.00001213	✓	×	×	×	×	×	×	×
Nickel (Ni)	9.8E-03	0.00000976	✓	×	×	×	×	×	×	×
Vanadium (V)	1.0E-03	0.000001010	✓	×	×	×	×	×	×	×
Dioxins and furans	1.6E-08	1.61E-11	✓	×	×	×	×	×	×	×
Benzene	2.7E+00	0.00268871	✓	×	×	×	×	×	×	×
Formaldehyde	0.39141	0.00039141	✓	×	×	×	×	×	×	×
Polycyclic aromatic hydrocarbons (PAHs)	0.000008	0.000000008	✓	×	×	×	×	×	×	×

Inhalation - gases and particulates
Maximum on-site for this facility plus background plus brickworks

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * EF * ED}{AT}$$ (mg/m^3) for gases

$$Inhalation\ Exposure\ Concentration = Ca * \frac{ET * FI * DRF * EF * ED}{AT}$$ (mg/m^3) for chemicals attached to particles

Parameters Relevant to Quantification of Community Exposures - Visitors (teachers and school students)		
Exposure Time at Home (ET, hr/day)	4	Assume visit may take this time on each occasion
Fraction Inhaled from Source (FI, unitless)	1	Assume present at the site
Dust lung retention factor (unitless)	0.375	Percentage of respirable dust that is small enough to reach and be retained in the lungs (NEPM 1999 amended 2013) - NA for gasses
Exposure Frequency - normal conditions (EF, days/yr)	12	Days at the site each year
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
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	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-03	3.8E-06	8.8E-06	--		0.000340	5%
Hydrogen fluoride (HF)		2.9E-02	0%	2.9E-02	2.7E-04	6.3E-07	1.5E-06	--		0.0000508	1%
Ammonia		3.2E-01	0%	3.2E-01	3.2E-06	7.4E-09	1.7E-08	--		0.0000000543	0%
Cadmium (Cd)		5.0E-06	20%	4.0E-06	2.5E-06	2.2E-09	5.1E-09	--		0.00127	19%
Thallium (Tl)		7.0E-04	10%	6.3E-04	2.2E-06	1.9E-09	4.5E-09	--		0.00000714	0%
Mercury (Hg)		2.0E-04	0%	2.0E-04	8.1E-06	7.1E-09	1.7E-08	--		0.0000833	1%
Antimony (Sb)		3.0E-04	0%	3.0E-04	2.0E-06	1.8E-09	4.1E-09	--		0.0000138	0%
Arsenic (As)		6.7E-05	0%	6.7E-05	7.1E-06	6.3E-09	1.5E-08	--		0.000218	3%
Lead (Pb)		5.0E-04	0%	5.0E-04	3.2E-05	2.8E-08	6.6E-08	--		0.000133	2%
Chromium (Cr VI assumed)		4.3E-06	0%	4.3E-06	8.1E-06	7.1E-09	1.7E-08	--		0.00388	57%
Cobalt (Co)		1.0E-04	0%	1.0E-04	2.0E-06	1.8E-09	4.1E-09	--		0.0000413	1%
Copper (Cu)		4.9E-01	60%	2.0E-01	7.1E-06	6.3E-09	1.5E-08	--		0.0000000744	0%
Manganese (Mn)		1.5E-04	20%	1.2E-04	1.2E-05	1.1E-08	2.5E-08	--		0.000208	3%
Nickel (Ni)		5.9E-05	10%	5.3E-05	9.8E-06	8.6E-09	2.0E-08	--		0.000378	6%
Vanadium (V)		1.0E-04	0%	1.0E-04	1.0E-06	8.9E-10	2.1E-09	--		0.0000208	0%
Dioxins and furans		8.1E-09	54%	3.7E-09	1.6E-11	1.4E-14	3.3E-14	--		0.00000893	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	2.7E-03	2.4E-06	5.5E-06	1.4E-8	100%	0.000205	3%
Formaldehyde		7.0E-03	0%	7.0E-03	3.9E-04	3.4E-07	8.0E-07	--		0.000115	2%
PAHs	6.0E-01				8.0E-09	7.0E-12	1.6E-11	4.2E-12	0%	--	
							TOTAL	**1.4E-08**		**0.00686**	

Rainwater Tanks

Maximum Off-Site

Calculation of Concentrations in Rainwater tank

CW = DM/(VR*Kd*ρ) **(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/m^3)
Rc =	Runoff coefficient (unitless)
Kd =	Soil-water partition coefficient (cm3/g)
1000 =	Conversion from mm to m

General Parameters			
Average rainfaill	mm/year	537.5	mean for all years (1970 - 2022) for Melbourne Airport (086282)
Roof area	m2	200	4 bedroom australian home
Runoff coefficient	-	0.7	assumes 30% water loss in capture into tank
Volume of rainwater	m3/year	75.25	calculated
Volume of rainwater	L/year	75250	
Bulk density of deposited dust	g/cm3	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 Deposition Rate (DR)	PM10 Mass deposited each year (DM)	Kd	Particulate Concentration in water	Dissolved Concentration in water
	$mg/m^2/year$	mg	(cm3/g)	mg/L	mg/L
Cadmium (Cd)	4.73E-03	0.9	75	1.3E-05	3.4E-07
Thallium (Tl)	3.27E-03	0.7	29	8.7E-06	6.0E-07
Mercury (Hg)	8.00E-03	1.6	790	2.1E-05	5.4E-08
Antimony (Sb)	8.46E-03	1.7	45	2.2E-05	1.0E-06
Arsenic (As)	3.70E-03	0.7	29	9.8E-06	6.8E-07
Lead (Pb)	4.82E-02	9.6	900	1.3E-04	2.8E-07
Chromium (Cr VI assumed)	1.21E-02	2.4	19	3.2E-05	3.4E-06
Cobalt (Co)	3.00E-03	0.6	45	8.0E-06	3.5E-07
Copper (Cu)	1.06E-02	2.1	35	2.8E-05	1.6E-06
Manganese (Mn)	1.81E-02	3.6	65	4.8E-05	1.5E-06
Nickel (Ni)	1.46E-02	2.9	65	3.9E-05	1.2E-06
Vanadium (V)	1.50E-03	0.3	1000	4.0E-06	8.0E-09
Dioxins and furans	1.36E-07	0.00003	63096	3.6E-10	1.1E-14
PAHs	1.26E-02	2.5	5874	3.4E-05	1.1E-08

Kd for dioxins and furans based on Log Koc of 6.8 and 1% organic carbon (0.01 Foc), Kd = Koc x Foc
Kd for BaP based on Koc of 587400 (from USEPA RSLs, May 2022) and 1% organic carbon

	Log Koc (a	Koc	Assume 1% OC Kd	
Dioxin	6.8	6309573	63095.7344	Pubchem
BaP		587400	5874	USEPA RSLs 2022

Exposure to Chemicals via Incidental Ingestion of Water - Grid Maximum Off-site

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

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	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	As per ASC NEPM
Averaging Time - Threshold (Atn, days)	10950	As per 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/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.4E-07	4.1E-09	9.6E-09	--		0.0000299	9%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	6.0E-07	7.3E-09	1.7E-08	--		0.0000953	27%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	5.4E-08	6.6E-10	1.5E-09	--		0.00000641	2%
Antimony (Sb)		8.6E-04	0%	8.6E-04	100%	1.0E-06	1.2E-08	2.9E-08	--		0.0000332	10%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	6.8E-07	8.3E-09	1.9E-08	--		0.0000194	6%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	2.8E-07	3.5E-09	8.1E-09	--		0.0000271	8%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	3.4E-06	4.1E-08	9.7E-08	--		0.000119	34%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	3.5E-07	4.3E-09	1.0E-08	--		0.00000904	3%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.6E-06	2.0E-08	4.6E-08	--		0.000000821	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.5E-06	1.8E-08	4.2E-08	--		0.000000604	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.2E-06	1.5E-08	3.4E-08	--		0.00000708	2%
Vanadium (V)		2.0E-03	0%	2.0E-03	100%	8.0E-09	9.8E-11	2.3E-10	--		0.000000114	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.1E-14	1.4E-16	3.3E-16	--		3.1E-07	0%
PAHs	2.3E-01				100%	1.1E-08	1.4E-10	3.3E-10	3.3E-11		--	
								TOTAL	**3.3E-11**		**0.000349**	

Exposure to Chemicals via Incidental Ingestion of Water - Grid Maximum Off-site

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

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 NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	As per ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	As per 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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.4E-07	7.7E-10	8.9E-09	--		0.0000279	9%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	6.0E-07	1.4E-09	1.6E-08	--		0.0000889	27%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	5.4E-08	1.2E-10	1.4E-09	--		0.00000598	2%
Antimony (Sb)		8.6E-04	0%	8.6E-04	100%	1.0E-06	2.3E-09	2.7E-08	--		0.0000310	10%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	6.8E-07	1.6E-09	1.8E-08	--		0.0000181	6%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	2.8E-07	6.5E-10	7.6E-09	--		0.0000253	8%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	50%	3.4E-06	7.7E-09	9.0E-08	--		0.000111	34%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	3.5E-07	8.1E-10	9.5E-09	--		0.00000844	3%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.6E-06	3.7E-09	4.3E-08	--		0.000000766	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.5E-06	3.4E-09	3.9E-08	--		0.000000564	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.2E-06	2.7E-09	3.2E-08	--		0.00000661	2%
Vanadium (V)		2.0E-03	0%	2.0E-03	100%	8.0E-09	1.8E-11	2.1E-10	--		0.000000106	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.1E-14	2.6E-17	3.1E-16	--		2.9E-07	0%
PAHs	2.3E-01				100%	1.1E-08	2.6E-11	3.0E-10	6.1E-12		--	
								TOTAL	**6.1E-12**		**0.000325**	

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 (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	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	As per ASC NEPM
Averaging Time - Threshold (Atn, days)	10950	As per 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/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	3.4E-07	1.94E-13	2.4E-11	5.6E-11	--		0.000000174	4%
Thallium (Tl)		2.0E-04	10%	1.8E-04	1.00E-3	6.0E-07	3.48E-13	4.3E-11	9.9E-11	--		0.000000552	14%
Mercury (Hg)		6.0E-04	60%	2.4E-04	1.00E-3	5.4E-08	3.12E-14	3.8E-12	8.9E-12	--		0.0000000372	1%
Antimony (Sb)		8.6E-04		8.6E-04	1.00E-3	1.0E-06	5.79E-13	7.1E-11	1.7E-10	--		0.000000193	5%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	6.8E-07	3.93E-13	4.8E-11	1.1E-10	--		0.000000112	3%
Lead (Pb)		6.0E-04	50%	3.0E-04	1.00E-4	2.8E-07	1.65E-14	2.0E-12	4.7E-12	--		0.0000000157	0%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	2.00E-3	3.4E-06	3.93E-12	4.8E-10	1.1E-09	--		0.00000138	35%
Cobalt (Co)		1.4E-03	20%	1.1E-03	4.00E-4	3.5E-07	8.22E-14	1.0E-11	2.3E-11	--		0.0000000210	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	1.6E-06	9.33E-13	1.1E-10	2.7E-10	--		0.00000000476	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	1.5E-06	8.58E-13	1.1E-10	2.5E-10	--		0.00000000350	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	1.2E-06	1.38E-13	1.7E-11	3.9E-11	--		0.00000000822	0%
Vanadium (V)		2.0E-03		2.0E-03	1.00E-3	8.0E-09	4.63E-15	5.7E-13	1.3E-12	--		0.00000000066	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	8.00E-1	1.1E-14	5.32E-18	6.5E-16	1.5E-15	--		0.00000144	36%
PAHs	2.3E-01				7.13E-1	1.1E-08	4.72E-12	5.8E-10	1.3E-09	1.3E-10		--	
										1.3E-10		**0.00000394**	

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 (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)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	As per ASC NEPM
Averaging Time - Threshold (Atn, days)	2190	As per 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/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	3.4E-07	3.35E-13	1.2E-11	1.4E-10	--		0.000000426	4%
Thallium (Tl)		2.0E-04	10%	1.8E-04	1.00E-3	6.0E-07	6.00E-13	2.1E-11	2.4E-10	--		0.00000136	14%
Mercury (Hg)		6.0E-04	60%	2.4E-04	1.00E-3	5.4E-08	5.38E-14	1.9E-12	2.2E-11	--		0.0000000912	1%
Antimony (Sb)		8.6E-04		8.6E-04	1.00E-3	1.0E-06	9.99E-13	3.5E-11	4.1E-10	--		0.000000472	5%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	6.8E-07	6.78E-13	2.4E-11	2.8E-10	--		0.000000276	3%
Lead (Pb)		6.0E-04	50%	3.0E-04	1.00E-4	2.8E-07	2.85E-14	9.9E-13	1.2E-11	--		0.0000000386	0%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	2.00E-3	3.4E-06	6.77E-12	2.4E-10	2.8E-09	--		0.00000340	35%
Cobalt (Co)		1.4E-03	20%	1.1E-03	4.00E-4	3.5E-07	1.42E-13	4.9E-12	5.8E-11	--		0.0000000515	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	1.6E-06	1.61E-12	5.6E-11	6.5E-10	--		0.0000000117	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	1.5E-06	1.48E-12	5.2E-11	6.0E-10	--		0.00000000860	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	1.2E-06	2.38E-13	8.3E-12	9.7E-11	--		0.0000000202	0%
Vanadium (V)		2.0E-03		2.0E-03	1.00E-3	8.0E-09	7.98E-15	2.8E-13	3.2E-12	--		0.00000000162	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	8.00E-1	1.1E-14	9.18E-18	3.2E-16	3.7E-15	--		0.00000353	36%
PAHs	2.3E-01				7.13E-1	1.1E-08	8.14E-12	2.8E-10	3.3E-09	6.6E-11		--	
										6.6E-11		**0.00000968**	

Maximum Residential

Calculation of Concentrations in Rainwater tank

CW = DM/(VR*Kd*ρ) **(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/m^3)
Rc =	Runoff coefficient (unitless)
Kd =	Soil-water partition coefficient (cm3/g)
1000 =	Conversion from mm to m

General Parameters			
Average rainfaill	mm/year	537.5	mean for all years (1970 - 2022) for Melbourne Airport (086282)
Roof area	m2	200	4 bedroom australian home
Runoff coefficient	-	0.7	assumes 30% water loss in capture into tank
Volume of rainwater	m3/year	75.25	calculated
Volume of rainwater	L/year	75250	
Bulk density of deposited dust	g/cm3	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 Deposition Rate (DR) mg/m^2/year	PM10 Mass deposited each year (DM) mg	Kd (cm3/g)	Particulate Concentration in water mg/L	Dissolved Concentration in water mg/L
Cadmium (Cd)	9.82E-04	0.2	75	2.6E-06	7.0E-08
Thallium (Tl)	9.97E-05	0.0	29	2.6E-07	1.8E-08
Antimony (Sb)	9.77E-04	0.2	790	2.6E-06	6.6E-09
Arsenic (As)	9.19E-04	0.2	45	2.4E-06	1.1E-07
Lead (Pb)	9.83E-05	0.0	29	2.6E-07	1.8E-08
Chromium (Cr VI assumed)	9.75E-03	1.9	900	2.6E-05	5.8E-08
Cobalt (Co)	1.85E-03	0.4	19	4.9E-06	5.2E-07
Copper (Cu)	1.00E-04	0.0	45	2.7E-07	1.2E-08
Manganese (Mn)	9.70E-04	0.2	35	2.6E-06	1.5E-07
Nickel (Ni)	9.96E-04	0.2	65	2.6E-06	8.1E-08
Vanadium (V)	9.71E-04	0.2	65	2.6E-06	7.9E-08
Benzene	8.99E-05	0.0	1000	2.4E-07	4.8E-10
PAHs	1.78E-08	0.00000	63096	4.7E-11	1.5E-15
PAHs	1.89E-03	0.4	5874	5.0E-06	1.7E-09

Kd for dioxins and furans based on Log Koc of 6.8 and 1% organic carbon (0.01 Foc), Kd = Koc x Foc
Kd for BaP based on Koc of 587400 (from USEPA RSLs, May 2022) and 1% organic carbon

	Log Koc (a	Koc	Assume 1% OC Kd	
Dioxin	6.8	6309573	63095.7344	Pubchem
BaP		587400	5874	USEPA RSLs 2022

Exposure to Chemicals via Incidental Ingestion of Water - Max Residential

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

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	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10950	US EPA 1989 and CSMS 1996

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)		(mg/L)	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%	7.0E-08	8.5E-10	2.0E-09	--		0.00000621	16%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	1.8E-08	2.2E-10	5.2E-10	--		0.00000290	7%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	6.6E-09	8.0E-11	1.9E-10	--		0.000000783	2%
Antimony (Sb)		8.6E-04	0%	8.6E-04	100%	1.1E-07	1.3E-09	3.1E-09	--		0.000003607	9%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.8E-08	2.2E-10	5.1E-10	--		0.000000515	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	5.8E-08	7.0E-10	1.6E-09	--		0.000005483	14%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	5.2E-07	6.3E-09	1.5E-08	--		0.0000183	47%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.2E-08	1.4E-10	3.4E-10	--		0.0000003013	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.5E-07	1.8E-09	4.2E-09	--		0.0000000752	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	8.1E-08	1.0E-09	2.3E-09	--		0.0000000332	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	7.9E-08	9.7E-10	2.3E-09	--		0.000000473	1%
Vanadium (V)		2.0E-03	0%	2.0E-03	100%	4.8E-10	5.9E-12	1.4E-11	--		0.00000000683	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.5E-15	1.8E-17	4.3E-17	--		0.0000000405	0%
PAHs	2.3E-01				100%	1.7E-09	2.1E-11	4.9E-11	4.9E-12		--	
								TOTAL	**4.9E-12**		**0.0000387**	

Exposure to Chemicals via Incidental Ingestion of Water - Max Residential

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

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 NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	US EPA 1989 and CSMS 1996

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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	7.0E-08	1.6E-10	1.9E-09	--		0.00000580	16%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	1.8E-08	4.2E-11	4.9E-10	--		0.00000271	7%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	6.6E-09	1.5E-11	1.8E-10	--		0.000000730	2%
Antimony (Sb)		8.6E-04	0%	8.6E-04	100%	1.1E-07	2.5E-10	2.9E-09	--		0.000003366	9%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	1.8E-08	4.1E-11	4.8E-10	--		0.000000480	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	5.8E-08	1.3E-10	1.5E-09	--		0.000005117	14%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	50%	5.2E-07	1.2E-09	1.4E-08	--		0.0000171	47%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.2E-08	2.7E-11	3.1E-10	--		0.0000002812	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.5E-07	3.4E-10	3.9E-09	--		0.0000000702	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	8.1E-08	1.9E-10	2.2E-09	--		0.0000000310	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	7.9E-08	1.8E-10	2.1E-09	--		0.000000441	1%
Vanadium (V)		2.0E-03	0%	2.0E-03	100%	4.8E-10	1.1E-12	1.3E-11	--		0.00000000637	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.5E-15	3.4E-18	4.0E-17	--		0.0000000378	0%
PAHs	2.3E-01				100%	1.7E-09	3.9E-12	4.6E-11	9.1E-13		--	
								TOTAL	**9.1E-13**		**0.0000361**	

Dermal Exposure to Chemicals via Contact with Water - Max 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 (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	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10950	US EPA 1989 and CSMS 1996

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/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	7.0E-08	4.04E-14	4.9E-12	1.2E-11	–		0.0000000360	7%
Thallium (Tl)		2.0E-04	10%	1.8E-04	1.00E-3	1.8E-08	1.06E-14	1.3E-12	3.0E-12	–		0.0000000168	3%
Mercury (Hg)		6.0E-04	60%	2.4E-04	1.00E-3	6.6E-09	3.81E-15	4.7E-13	1.1E-12	–		0.00000000454	1%
Antimony (Sb)		8.6E-04		8.6E-04	1.00E-3	1.1E-07	6.30E-14	7.7E-12	1.8E-11	–		0.0000000209	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	1.8E-08	1.05E-14	1.3E-12	3.0E-12	–		0.00000000299	1%
Lead (Pb)		6.0E-04	50%	3.0E-04	1.00E-4	5.8E-08	3.34E-15	4.1E-13	9.5E-13	–		0.00000000318	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	2.00E-3	5.2E-07	6.01E-13	7.4E-11	1.7E-10	–		0.0000002120	44%
Cobalt (Co)		1.4E-03	20%	1.1E-03	4.00E-4	1.2E-08	2.74E-15	3.4E-13	7.8E-13	–		0.000000000699	0%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	1.5E-07	8.54E-14	1.0E-11	2.4E-11	–		0.000000000436	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	8.1E-08	4.72E-14	5.8E-12	1.3E-11	–		0.000000000193	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	7.9E-08	9.21E-15	1.1E-12	2.6E-12	–		0.000000000548	0%
Vanadium (V)		2.0E-03		2.0E-03	1.00E-3	4.8E-10	2.77E-16	3.4E-14	7.9E-14	–		0.0000000000396	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	8.00E-1	1.5E-15	6.96E-19	8.5E-17	2.0E-16	–		0.0000001879	39%
PAHs	2.3E-01				7.13E-1	1.7E-09	7.08E-13	8.7E-11	2.0E-10	2.0E-11		–	
										2.0E-11		**0.000000486**	

Dermal Exposure to Chemicals via Contact with Water - Max 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 (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)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	US EPA 1989 and CSMS 1996

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/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	7.0E-08	6.96E-14	2.4E-12	2.8E-11	--		0.0000000884	7%
Thallium (Tl)		2.0E-04	10%	1.8E-04	1.00E-3	1.8E-08	1.83E-14	6.4E-13	7.4E-12	--		0.0000000413	3%
Antimony (Sb)		6.0E-04	60%	2.4E-04	1.00E-3	6.6E-09	6.57E-15	2.3E-13	2.7E-12	--		0.0000000111	1%
Arsenic (As)		8.6E-04		8.6E-04	1.00E-3	1.1E-07	1.09E-13	3.8E-12	4.4E-11	--		0.00000005133	4%
Lead (Pb)		2.0E-03	50%	1.0E-03	1.00E-3	1.8E-08	1.80E-14	6.3E-13	7.3E-12	--		0.00000000733	1%
Chromium (Cr VI assumed)		6.0E-04	50%	3.0E-04	1.00E-4	5.8E-08	5.76E-15	2.0E-13	2.3E-12	--		0.00000000780	1%
Cobalt (Co)		9.0E-04	10%	8.1E-04	2.00E-3	5.2E-07	1.04E-12	3.6E-11	4.2E-10	--		0.000000520	44%
Copper (Cu)		1.4E-03	20%	1.1E-03	4.00E-4	1.2E-08	4.72E-15	1.6E-13	1.9E-12	--		0.00000000172	0%
Manganese (Mn)		1.4E-01	60%	5.6E-02	1.00E-3	1.5E-07	1.47E-13	5.1E-12	6.0E-11	--		0.00000000107	0%
Nickel (Ni)		1.4E-01	50%	7.0E-02	1.00E-3	8.1E-08	8.15E-14	2.8E-12	3.3E-11	--		0.000000000473	0%
Vanadium (V)		1.2E-02	60%	4.8E-03	2.00E-4	7.9E-08	1.59E-14	5.5E-13	6.5E-12	--		0.000000001345	0%
Benzene		2.0E-03		2.0E-03	1.00E-3	4.8E-10	4.78E-16	1.7E-14	1.9E-13	--		0.0000000000972	0%
PAHs		2.3E-09	54%	1.1E-09	8.00E-1	1.5E-15	1.20E-18	4.2E-17	4.9E-16	--		0.000000461	39%
PAHs	2.3E-01				7.13E-1	1.7E-09	1.22E-12	4.3E-11	5.0E-10	9.9E-12		--	
										9.9E-12		**0.00000119**	

Maximum Commercial/Industrial

Calculation of Concentrations in Rainwater tank

CW = DM/(VR*Kd*ρ) **(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/m^3)
Rc =	Runoff coefficient (unitless)
Kd =	Soil-water partition coefficient (cm3/g)
1000 =	Conversion from mm to m

General Parameters			
Average rainfaill	mm/year	537.5	mean for all years (1970 - 2022) for Melbourne Airport (086282)
Roof area	m2	200	4 bedroom australian home
Runoff coefficient	-	0.7	assumes 30% water loss in capture into tank
Volume of rainwater	m3/year	75.25	calculated
Volume of rainwater	L/year	75250	
Bulk density of deposited dust	g/cm3	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 Deposition Rate (DR) mg/m^2/year	PM10 Mass deposited each year (DM) mg	Kd (cm3/g)	Particulate Concentration in water mg/L	Dissolved Concentration in water mg/L
Cadmium (Cd)	2.54E-03	0.5	75	6.8E-06	1.8E-07
Thallium (Tl)	1.76E-03	0.4	29	4.7E-06	3.2E-07
Antimony (Sb)	4.31E-03	0.9	790	1.1E-05	2.9E-08
Arsenic (As)	4.55E-03	0.9	45	1.2E-05	5.4E-07
Lead (Pb)	1.99E-03	0.4	29	5.3E-06	3.6E-07
Chromium (Cr VI assumed)	2.59E-02	5.2	900	6.9E-05	1.5E-07
Cobalt (Co)	6.51E-03	1.3	19	1.7E-05	1.8E-06
Copper (Cu)	1.61E-03	0.3	45	4.3E-06	1.9E-07
Manganese (Mn)	5.70E-03	1.1	35	1.5E-05	8.7E-07
Nickel (Ni)	9.73E-03	1.9	65	2.6E-05	8.0E-07
Vanadium (V)	7.83E-03	1.6	65	2.1E-05	6.4E-07
Benzene	8.07E-04	0.2	1000	2.1E-06	4.3E-09
PAHs	7.33E-08	0.00001	63096	1.9E-10	6.2E-15
PAHs	7.10E-03	1.4	5874	1.9E-05	6.4E-09

Kd for dioxins and furans based on Log Koc of 6.8 and 1% organic carbon (0.01 Foc), Kd = Koc x Foc
Kd for BaP based on Koc of 587400 (from USEPA RSLs, May 2022) and 1% organic carbon

	Log Koc (a	Koc	Assume 1% OC Kd	
Dioxin	6.8	6309573	63095.7344	Pubchem
BaP		587400	5874	USEPA RSLs 2022

Exposure to Chemicals via Incidental Ingestion of Water - Max Commercial

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

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	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.8E-07	2.2E-09	5.2E-09	--		0.00001610	9%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	3.2E-07	4.0E-09	9.2E-09	--		0.00005124	27%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	2.9E-08	3.5E-10	8.3E-10	--		0.000003448	2%
Antimony (Sb)		8.6E-04	0%	8.6E-04	100%	5.4E-07	6.6E-09	1.5E-08	--		0.000017852	10%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	3.6E-07	4.5E-09	1.0E-08	--		0.000010427	6%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.5E-07	1.9E-09	4.4E-09	--		0.000014583	8%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	1.8E-06	2.2E-08	5.2E-08	--		0.0000642	34%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.9E-07	2.3E-09	5.4E-09	--		0.0000048636	3%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	8.7E-07	1.1E-08	2.5E-08	--		0.0000004416	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	8.0E-07	9.7E-09	2.3E-08	--		0.0000003249	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	6.4E-07	7.8E-09	1.8E-08	--		0.000003810	2%
Vanadium (V)		2.0E-03	0%	2.0E-03	100%	4.3E-09	5.3E-11	1.2E-10	--		0.00000006128	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	6.2E-15	7.6E-17	1.8E-16	--		0.0000001667	0%
PAHs	2.3E-01				100%	6.4E-09	7.9E-11	1.8E-10	1.8E-11		--	
								TOTAL	**1.8E-11**		**0.0001875**	

Exposure to Chemicals via Incidental Ingestion of Water - Max Commercial

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

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 NEPM (2013)
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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.8E-07	4.1E-10	4.8E-09	--		0.00001503	9%
Thallium (Tl)		2.0E-04	10%	1.8E-04	100%	3.2E-07	7.4E-10	8.6E-09	--		0.00004782	27%
Mercury (Hg)		6.0E-04	60%	2.4E-04	100%	2.9E-08	6.6E-11	7.7E-10	--		0.000003219	2%
Antimony (Sb)		8.6E-04	0%	8.6E-04	100%	5.4E-07	1.2E-09	1.4E-08	--		0.000016662	10%
Arsenic (As)		2.0E-03	50%	1.0E-03	100%	3.6E-07	8.3E-10	9.7E-09	--		0.000009732	6%
Lead (Pb)		6.0E-04	50%	3.0E-04	100%	1.5E-07	3.5E-10	4.1E-09	--		0.000013611	8%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	50%	1.8E-06	4.2E-09	4.9E-08	--		0.0000599	34%
Cobalt (Co)		1.4E-03	20%	1.1E-03	100%	1.9E-07	4.4E-10	5.1E-09	--		0.0000045394	3%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	8.7E-07	2.0E-09	2.3E-08	--		0.0000004122	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	8.0E-07	1.8E-09	2.1E-08	--		0.0000003033	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	6.4E-07	1.5E-09	1.7E-08	--		0.000003556	2%
Vanadium (V)		2.0E-03	0%	2.0E-03	100%	4.3E-09	9.8E-12	1.1E-10	--		0.00000005720	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	6.2E-15	1.4E-17	1.6E-16	--		0.0000001556	0%
PAHs	2.3E-01				100%	6.4E-09	1.5E-11	1.7E-10	3.4E-12		--	
								TOTAL	**3.4E-12**		**0.0001750**	

Dermal Exposure to Chemicals via Contact with Water - Max 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	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
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 HI
	(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	1.00E-3	1.8E-07	1.05E-13	1.3E-11	3.0E-11	--		0.0000000934	4%
Thallium (Tl)		2.0E-04	10%	1.8E-04	1.00E-3	3.2E-07	1.87E-13	2.3E-11	5.3E-11	--		0.0000002972	14%
Mercury (Hg)		6.0E-04	60%	2.4E-04	1.00E-3	2.9E-08	1.68E-14	2.1E-12	4.8E-12	--		0.00000002000	1%
Antimony (Sb)		8.6E-04		8.6E-04	1.00E-3	5.4E-07	3.12E-13	3.8E-11	8.9E-11	--		0.0000001035	5%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	3.6E-07	2.12E-13	2.6E-11	6.0E-11	--		0.00000006048	3%
Lead (Pb)		6.0E-04	50%	3.0E-04	1.00E-4	1.5E-07	8.88E-15	1.1E-12	2.5E-12	--		0.00000000846	0%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	2.00E-3	1.8E-06	2.11E-12	2.6E-10	6.0E-10	--		0.0000007448	35%
Cobalt (Co)		1.4E-03	20%	1.1E-03	4.00E-4	1.9E-07	4.42E-14	5.4E-12	1.3E-11	--		0.000000011284	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	8.7E-07	5.02E-13	6.1E-11	1.4E-10	--		0.000000002561	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	8.0E-07	4.62E-13	5.7E-11	1.3E-10	--		0.000000001884	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	6.4E-07	7.42E-14	9.1E-12	2.1E-11	--		0.000000004420	0%
Vanadium (V)		2.0E-03		2.0E-03	1.00E-3	4.3E-09	2.49E-15	3.0E-13	7.1E-13	--		0.0000000003554	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	8.00E-1	6.2E-15	2.86E-18	3.5E-16	8.2E-16	--		0.0000007734	36%
PAHs	2.3E-01				7.13E-1	6.4E-09	2.66E-12	3.3E-10	7.6E-10	7.6E-11		--	
										7.6E-11		**0.000002122**	

Dermal Exposure to Chemicals via Contact with Water - Max 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, 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 (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)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
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/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	1.8E-07	1.80E-13	6.3E-12	7.3E-11	--		0.0000002291	4%
Thallium (Tl)		2.0E-04	10%	1.8E-04	1.00E-3	3.2E-07	3.23E-13	1.1E-11	1.3E-10	--		0.0000007293	14%
Antimony (Sb)		6.0E-04	60%	2.4E-04	1.00E-3	2.9E-08	2.90E-14	1.0E-12	1.2E-11	--		0.0000000491	1%
Arsenic (As)		8.6E-04		8.6E-04	1.00E-3	5.4E-07	5.37E-13	1.9E-11	2.2E-10	--		0.00000025410	5%
Lead (Pb)		2.0E-03	50%	1.0E-03	1.00E-3	3.6E-07	3.65E-13	1.3E-11	1.5E-10	--		0.00000014841	3%
Chromium (Cr VI assumed)		6.0E-04	50%	3.0E-04	1.00E-4	1.5E-07	1.53E-14	5.3E-13	6.2E-12	--		0.00000002076	0%
Cobalt (Co)		9.0E-04	10%	8.1E-04	2.00E-3	1.8E-06	3.64E-12	1.3E-10	1.5E-09	--		0.000001828	35%
Copper (Cu)		1.4E-03	20%	1.1E-03	4.00E-4	1.9E-07	7.63E-14	2.7E-12	3.1E-11	--		0.00000002769	1%
Manganese (Mn)		1.4E-01	60%	5.6E-02	1.00E-3	8.7E-07	8.66E-13	3.0E-11	3.5E-10	--		0.00000000629	0%
Nickel (Ni)		1.4E-01	50%	7.0E-02	1.00E-3	8.0E-07	7.96E-13	2.8E-11	3.2E-10	--		0.000000004625	0%
Vanadium (V)		1.2E-02	60%	4.8E-03	2.00E-4	6.4E-07	1.28E-13	4.5E-12	5.2E-11	--		0.000000010846	0%
Benzene		2.0E-03		2.0E-03	1.00E-3	4.3E-09	4.29E-15	1.5E-13	1.7E-12	--		0.0000000008722	0%
PAHs		2.3E-09	54%	1.1E-09	8.00E-1	6.2E-15	4.94E-18	1.7E-16	2.0E-15	--		0.000001898	36%
PAHs	2.3E-01				7.13E-1	6.4E-09	4.58E-12	1.6E-10	1.9E-09	3.7E-11		--	
										3.7E-11		**0.00000521**	