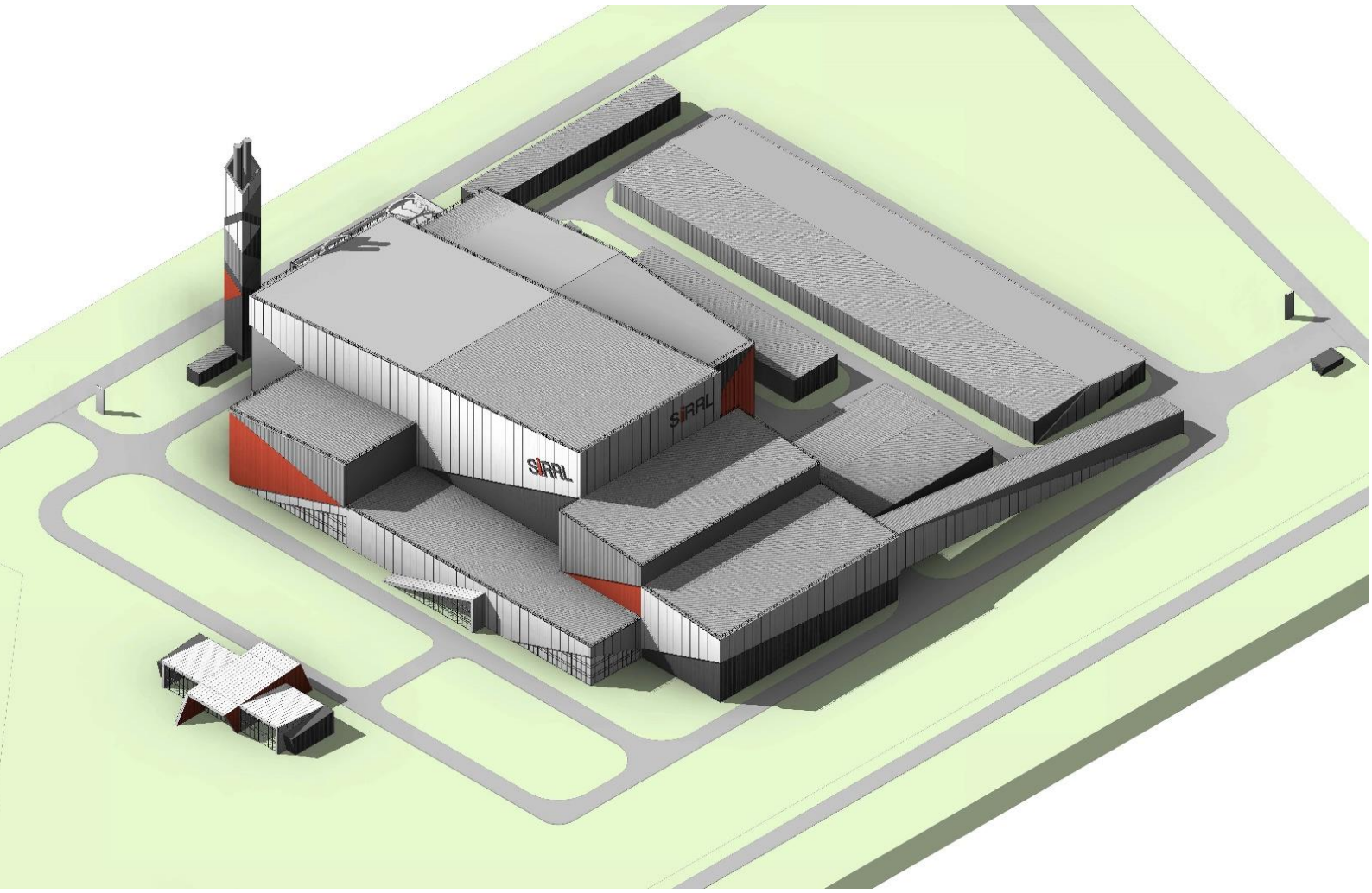

Project Kea: Human Health Risk Assessment

Prepared for: Babbage Consultants Limited and South Island Resource Recovery Ltd

17 November 2022

Document History and Status

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Limitations

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Appendices

Glossary of terms and abbreviations

Term	Definition
AAQ	Ambient air quality
Acute exposure	Contact with a substance that occurs once or for only a short time, typically an hour but may be up to 14 days [compare with chronic exposure and intermediate duration exposure].
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
Background level	A pre-existing average or expected amount of a substance or material in a specific environment, or typical amounts of substances that occur naturally in an environment.
BAT	Best available techniques
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.
C&I	Commercial and industrial
Carcinogen	A substance that causes cancer.
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
Detection limit	The lowest concentration of a substance that can reliably be distinguished from a zero concentration.
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.
enHealth	Environmental Health Standing Committee (Department of Health)
EfW	Energy from waste
EPA	Environment Protection Authority
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 [intermediate exposure], 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.
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.

Term	Definition
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 New Zealand Ministry for the Environment (MfE), National Health and Medical Research Council (NHMRC) 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.
HHRA	Human health risk assessment
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	Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].
LFG	Landfill gas
LOR	Limit of Reporting
Metabolism	The conversion or breakdown of a substance from one form to another by a living organism.
MfE	Ministry for the Environment
MJ/kg	Megajoules per kilogram
MOH	Ministry of Health
MSW	Municipal solid waste
Mtpa	Million tonnes per annum
MW	Mega watt
MWth	Mega watt thermal
NHMRC	National Health and Medical Research Council
NO_2	Nitrogen dioxide
NOx	Nitrogen oxides
OEHHA	Office of Environmental Health Hazard Assessment, California Environment Protection Agency (Cal EPA)
PFAS	Per- and polyfluoroalkyl substances
PM	Particulate matter
$PM_{2.5}$	Particulate matter of aerodynamic diameter 2.5 µm and less
PM_{10}	Particulate matter of aerodynamic diameter 10 µm and less
Point of exposure	The place where someone can come into contact with a substance present in the environment [see exposure pathway].
Population	A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).
Receptor population	People who could come into contact with hazardous substances [see exposure pathway].
Risk	The probability that something would cause injury or harm.
Route of exposure	The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].
SO_2	Sulfur dioxide
TCEQ	Texas Commission on Environmental Quality
TEQ	Toxicity equivalent

Term	Definition
Toxicity	The degree of danger posed by a substance to human, animal or plant life.
Toxicity data	Characterisation or quantitative value estimated (by recognised authorities) for each individual chemical substance for relevant exposure pathway (inhalation, oral or dermal), with special emphasis on dose-response characteristics. The data are based on available toxicity studies relevant to humans and/or animals and relevant safety factors.
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.
tpa	tonnes per annum
TSP	Total suspended particulates
UK	United Kingdom
US	United States
USEPA	United States Environmental Protection Agency
VOC	Volatile organic compound
WHO	World Health Organization
$\mu g/m^3$	Micrograms per cubic metre

Executive Summary

Introduction

An energy from waste (EfW) plant is proposed to be located in Waimate (the “proposed Plant”) and referred to as Project Kea.

Project Kea will have the capacity to process 365,000 tonnes of solid waste (SW) per year. The SW will consist of municipal solid waste (MSW) and construction waste (CW). The energy released from the processing will be converted to steam and electricity (via a steam turbine and generator).

SW will initially be delivered to the site by trucks, however, once the proposed Plant is operating, transport by rail will be used. The SW will consist of non-recyclable materials (i.e., material going to landfill), and would include organic waste and non-recyclable fossil fuel derived products. The proposed Plant will not take hazardous materials or tyres.

The steam generated by the proposed Plant will be available as a heat source for local industries. The electricity would be fed into the local network.

This human health risk assessment (HHRA) has been developed for the proposed Project by identifying and estimating the health impacts of the proposed project, as a result of emissions to air, on the health of the surrounding (local and regional) community.

Assessment approach

The HHRA has been conducted as a desktop assessment in accordance with guidelines relevant to the assessment of human health risks in New Zealand, Australia and the United States. The focus of the assessment has been the assessment of exposures that may occur as a result of emissions to air from the proposed Plant. As a result, the HHRA has relied on the air modelling presented in the Air Quality Emissions Assessment (PDP 2022).

The area surrounding the proposed Project site largely comprises rural land with uses in the areas surrounding the proposed Plant including cropping and livestock, with dairying being a significant use on many properties. The HHRA has considered the various land uses and activities that may occur in these areas.

The HHRA has been undertaken to address the following:

<u>Emissions evaluated</u>

The HHRA has focused on modelled impacts from emissions to air from the proposed Plant.

This assessment has considered impacts in the off-site community based on the maximum or guaranteed emission rates for the operation of the proposed Plant. These emission rates are consistent with the limits detailed in the Best Available Techniques (BAT) (EU 2019) as defined by the Industrial Emissions Directive 2010/75/EU (IED) (EU 2010).

It is expected that emissions to air from the proposed Plant would be lower than these emission rates.

Chemicals evaluated

The chemicals evaluated include $PM_{2.5}$, nitrogen dioxide, sulfur dioxide, carbon monoxide, gases (hydrogen chloride, hydrogen fluoride, ammonia and volatile organic compounds as benzene, toluene, xylenes and trimethylbenzenes), metals and dioxins and furans. The chemicals evaluated are consistent with the key chemicals identified in the EU Directives and relevant to EfW facilities processing MSW and CW.

Toxicity of chemicals evaluated in emissions

The assessment of toxicity for all the chemicals evaluated has adopted values that are protective of exposures by all members of the community including sensitive groups such as children and the elderly.

Location of exposure

The HHRA has evaluated potential exposures by workers on the boundary of the site, as well as within the surrounding community, based on the maximum predicted impacts at rural homes in the area surrounding the proposed Plant.

Time period of exposure

For the assessment of inhalation exposures, the HHRA has considered health impacts associated with both acute and chronic exposures. Acute exposures are assessed assuming anyone may be exposed to the maximum 1-hour average concentration for each chemical in air. Chronic exposures are assessed based on the maximum annual average air concentration anywhere, in commercial/industrial areas and in rural residential or residential areas.

For the assessment of other exposure pathways following deposition of dust, this has focused on chronic exposures as these pathways relate to the accumulation of chemicals in deposited dust over time.

Adopted conservative assumptions for assessing chronic inhalation exposures

When assessing chronic inhalation exposures, the following has been assumed:

- At the location of maximum concentrations on the site boundary - it is assumed that workers spend 8 hours per day, every workday (230 days per year) for 20 years at this location.
- At the location of maximum concentrations in air in rural residential and other residential areas - it is assumed that residents spend 24 hours per day, 350 days per year for 30 years.

Consideration of other pathways of exposure

In addition to assessing inhalation exposures, metals and persistent organic pollutants bound to dust may deposit to the ground or settle on roof areas where the following exposure may occur:

- incidental ingestion and dermal contact with chemicals deposited to soil and indoor dust
- uptake of these chemicals into home grown and consumed produce including fruit and vegetables, eggs, milk and meat (beef and lamb)
- accumulation in rainwater tanks used for drinking water.

The above exposures are assessed using worst-case assumptions that include:

- the concentration in soil and indoor dust is a cumulative concentration following emissions and continual deposition for 70 years with no cleaning indoors, no addition of fertiliser or other soil to gardens, no washing of produce prior to consuming
- rainwater tanks are used as potable water, there is no first flush device used on the tank and all the deposition that occurs onto the roof over a year accumulates into the tank
- residents are at the location of maximum deposition (after 70 years of operation) at all times that they may live in the area, i.e. 24 hours per day, 350 days of the year for 30 years.

The HHRA has also considered impacts on groundwater quality and the sale of crops and produce into the market (including impacts on organic produce).

Outcomes of the HHRA

Based on the available data and conservative assumptions adopted in this assessment, the following has been concluded:

- Inhalation exposures
    - All risks to human health are considered negligible for the duration of the proposed Plant. More specifically the following has been concluded:
        - no acute inhalation risk issues of concern
        - no chronic risk issues of concern
        - exposure to particulates derived from the proposed Plant within the community are considered negligible.
- Multi-pathway exposures
    - All chronic risks to human health are considered negligible for the duration of the proposed Plant. More specifically the following has been concluded:
        - all calculated risks for individual exposure pathways are negligible and essentially representative of zero risk
        - all calculated risks for combined multiple pathway exposures are negligible and essentially representative of zero risk.
    - Emissions from the proposed Plant would have a negligible impact on water quality in rainwater tanks used for drinking water
    - Emissions from the proposed Plant would have a negligible impact on crops and produce grown in the area.

Section 1. Introduction

1.1 Background

Environmental Risk Sciences Pty Ltd (enRiskS) has been engaged by Babbage Consultants Limited to undertake a Human Health Risk Assessment (HHRA) for an Energy from Waste (EfW) Plant proposed, referred to as Project Kea, to be located in Waimate.

The EfW proposed Plant will be owned and operated by South Island Resource Recovery Limited (SIRRL). The HHRA has been prepared to support the resource consent application to establish and operate the EfW Plant.

Project Kea will have the capacity to process 365,000 tonnes of solid waste (SW) per year. The SW will consist of municipal solid waste (MSW) and construction waste (CW). The energy released from the processing will be converted to steam and electricity (via a steam turbine and generator).

SW will initially be delivered to the site by trucks, however, once the proposed Plant is operating, transport by rail will be used. The SW will consist of non-recyclable materials (i.e., material going to landfill), and would include organic waste and non-recyclable fossil fuel derived products. The proposed Plant will not take hazardous materials or tyres.

The steam generated by the proposed Plant will be available as a heat source for local industries. The electricity would be fed into the local network. Enabling the proposed Plant to support local industries first and reduce the current demand for burning fossil fuel (coal). Overall, the production of steam and electricity will:

- strengthen the electricity supply to the local network
- provide energy to enable local business expansion
- eliminate the current annual one-week shut down period suffered by local businesses due to Transpower maintenance.

1.2 Project location and setting

The proposed Plant site is located in rural South Canterbury on the corner of Morven Glenavy Road as shown in **Figure 1.1**. The proposed Plant site is zoned Rural in the Waimate District Plan.

Figure 1.1: Location of proposed Plant and surrounding areas (image from Google Earth)

Areas surrounding the proposed Plant comprise of the following:

- rural properties used for a range of farming activities that include cropping and livestock, with dairying being a significant use on many properties
- commercial dairy (Oceania Dairy Factory)
- town of Glenavy (approximately 2 km south southwest)
- town of Waimate (approximately 18 km to the north northwest)
- Waitaki River (approximately 3 km to the south)
- Pacific Ocean (approximately 3.8 km to the east).

1.3 Project overview

The project involves construction and operation of the following key components:

- in / out truck weighbridges
- a common waste receival area
- a common 7,000 tonne storage bunker for MSW

  The waste hall and waste bunker will be held under negative pressure to prevent odours escaping to ambient air

- two incineration and steam generation lines
- two flue gas treatment lines
- two 75 m stacks (enclosed in a single housing)
- one grate ash handling and export system
- one fly ash plasma treatment and export system
- one water treatment plant
- one process wastewater treatment system
- one domestic wastewater treatment system.

In addition to the above the proposed Plant will also include three 2 MW diesel generators for initial start-up and following significant maintenance shutdowns (as the facility would not provide sufficient electricity during these periods).

Figure 1.2 shows the layout of the proposed Plant.

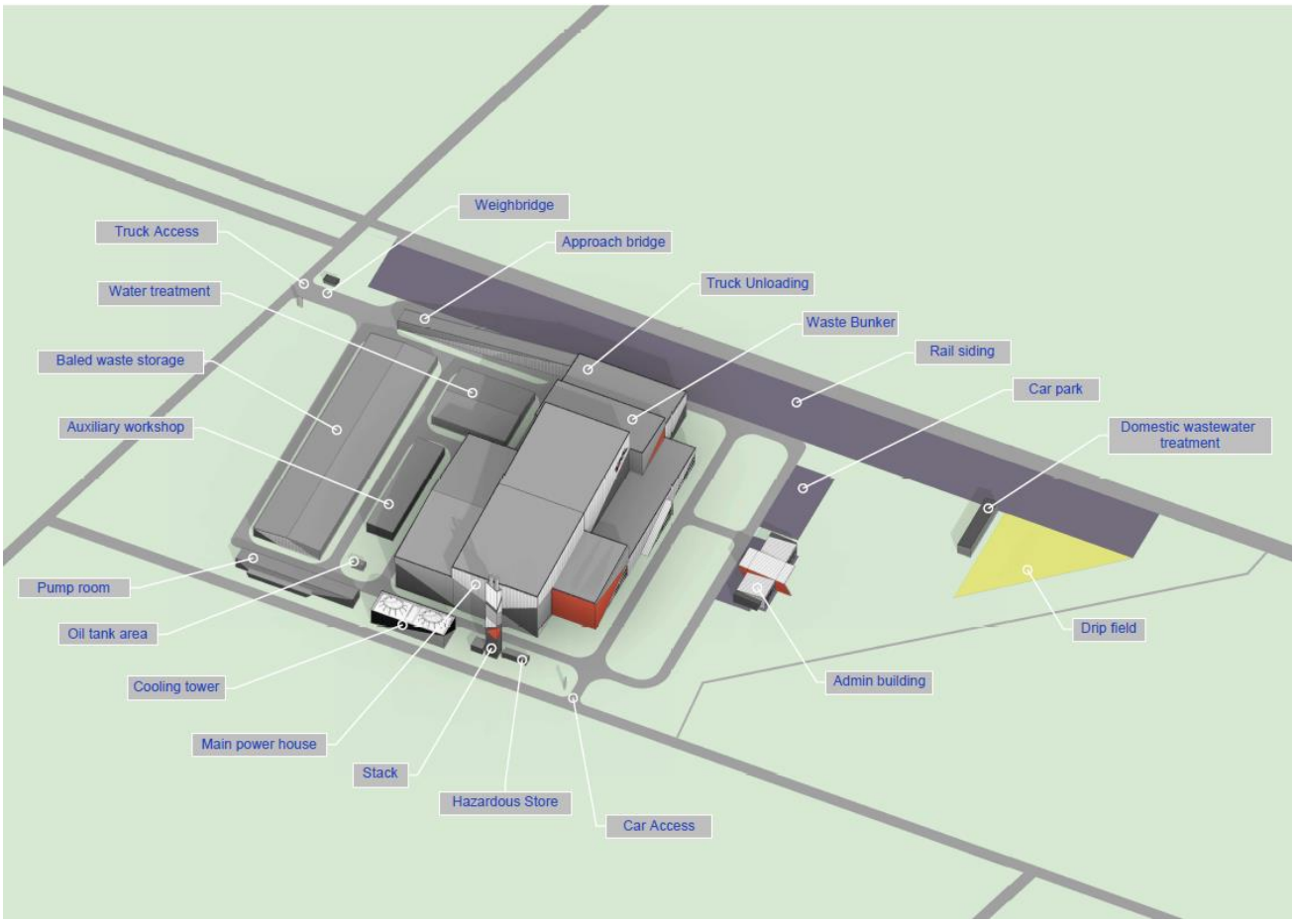

Figure 1.2: Proposed site layout

The proposed Plant has been assessed on incinerating a 50:50 split of MSW and CW. The proposed Plant would operate 24 hours per day, 7 days per week with the exception of a staged yearly shutdown for annual maintenance.

In relation to the management of the various waste streams from the proposed Plant, the following is noted:

- flue gas emissions from the combustion of waste: this will be treated to reduce contaminants levels in a multi-step process that includes:
    - selective non-catalytic reduction (SNCR) which involves the injection of ammonia to remove oxides of nitrogen (NOx)
    - semi-dry deacidification using lime to remove acid gases
    - dry spraying using sodium bicarbonate to remove acid gases
    - activated carbon absorption to remove dioxins (and other organics) and metals
    - filtration to remove particulates
    - wet scrubber to remove sodium hydroxide acid components
    - SCR using ammonia to further remove NOx and decompose dioxins.

- flue gas emissions from the plasma furnace will also be treated using:
    - quenching to reduce the temperature and wet scrubbing to remove acid gases, salts, metals and particulates
    - alkali washing to remove acid gases
    - wet electrostatic precipitator to remove particulates and fog droplets.
- industrial wastewater: this will be treated to remove contaminants and then 100% recycled back into the process – there will be no discharge to the environment
- domestic wastewater: this will be biologically treated and disposed of to land via drip field
- grate ash: this will be sorted to recover metal for recycling and then either used as an aggregate or disposed of to landfill
- fly ash: this will be treated via a plasma process to recover iron and create an inert solid which will then be combined with the Grate ash for use as an aggregate or otherwise disposed of to landfill.

1.4 Objectives

The overall objective of this report is to undertake a human health risk assessment (HHRA) in relation to potential impacts on the community from the operation of the proposed Plant.

The focus of the HHRA is on impacts on community health associated with changes in air quality and has not addressed any other impacts related to the proposed Plant.

The HHRA has focused on impacts on community health for populations located outside of the site boundaries of the proposed Plant. The HHRA has not addressed risks to workers involved in construction or operation of the proposed Plant. Workers involved in construction and operation of the proposed Plant would be managed under the *Health and Safety at Work Act 2015* and associated regulations and instruments.

1.5 Approach and scope of works

The overriding purpose of The *Resource Management Act 1991* (RMA) is to promote the sustainable management of natural and physical resources. The purpose of the RMA is set out in section 5:

"(1) The purpose of this Act is to promote the sustainable management of natural and physical resources.

(2) In this Act, sustainable management means managing the use, development, and protection of natural and physical resources in a way, or at a rate, which enables people and communities to provide for their social, economic, and cultural well-being and for their health and safety while—

***(a)** sustaining the potential of natural and physical resources (excluding minerals) to meet the reasonably foreseeable needs of future generations; and*

***(b)** safeguarding the life-supporting capacity of air, water, soil, and ecosystems; and*

***(c)** avoiding, remedying, or mitigating any adverse effects of activities on the environment."*

Noting the importance of the concept of "health and safety" in the purpose of the RMA, Ministry of Health provided guidance in 1995, stating that a balanced assessment of effects on the

environment should include potential effects of the proposal on health of persons and communities, in conjunction with other effects that are normally addressed (such as ecological, cultural, economic). The purpose of this guidance was to set out a number of principles and a systematic process for health impact assessment and risk analysis within the context of the RMA at a project level. This guidance was updated in 2005 (PHAC 2005). Instead of at a project level, the updated guidance focuses on the policy making level.

In general, New Zealand has limited detailed guidance in relation to the assessment of risks to community health in relation to environmental exposures from the operation of industrial facilities such as Project Kea.

Hence the HHRA presented in this report has been undertaken in accordance with the following guidance from New Zealand, Australia and the United States (and associated references as relevant):

- Environment Canterbury 2014, Methodologies to assess the environmental risks associated with non-natural rural waste (Environment Canterbury 2014)
- PHAC 2005, A guide to health impact assessment (PHAC 2005)
- enHealth 2012, Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a), and associated Australian Exposure Factor Guidance (enHealth 2012b), consistent with guidelines to be used in the conduct of the HHRA as detailed in the SEARs.
- Guidance and guidelines available from the National Environment Protection Council in relation to ambient air quality (NEPC 2016, 2021) and contaminated land (NEPC 1999 amended 2013a).
- USEPA Risk Assessment Guidance (USEPA 1989, 1991a, 1991b, 2001b, 2004, 2009d)
- California Office of Environmental Health Hazard Assessment (OEHHA) guidance on health risk assessment (OEHHA 2012, 2015).

The framework and key steps for undertaking a HHRA comprise of:

- Issue identification: which relates to identifying the vulnerability of the population to environmental stressors (**Section 2**), the source (**Section 3**) issues and key chemicals (**Section 4**) that need to be evaluated in the assessment.
- Hazard identification: which relates to the toxicity or hazards posed by exposure to the key chemicals evaluated, with quantitative dose-response values identified for each chemical evaluated (**Section 4.3** and **Appendix B**)
- Exposure assessment: which relates to who may be exposed to the key chemicals and how (via inhalation, ingestion and/or dermal absorption), with quantitative values adopted to characterise exposure (**Section 4.2** and **Appendix C**)
- Risk characterisation: where the above combined to provide a quantitative assessment of potential risks to human health (**Section 4**).

1.6 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 specific 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. This is in line with the methods and levels of evidence currently available to assess risk.

1.7 Available information

In relation to the proposed Plant, and potential for impacts on air quality within the local community, this HHRA has been developed on the basis of information provided within the following report:

- PDP 2022, Air Quality Emissions Assessment – Project Kea. Report dated 2022.

Section 2. Community profile

2.1 Introduction

This section provides an overview of the community potentially impacted by the proposed Plant. It is noted that the focus of this assessment is the community surrounding the site.

2.2 Community location and receptors

The proposed Plant is located in rural South Canterbury on the corner of Morven Glenavy Road and Carrolls Road (refer to **Section 1.2** and **Figure 1.1**). Key features surrounding the Project site include:

- rural properties and businesses surrounding the site
- small township of Glenavy (approximately 2 km south southwest)
- larger townships of Waimate (approximately 18 km to the north northwest), Oamaru (approximately 20 km to the southwest) and Duntroon (approximately 34 km to the west).

Rural uses in the areas surrounding the proposed Plant include cropping and livestock, with dairying being a significant use on many properties.

The focus of the HHRA relates to the community surrounding the proposed Plant. As a result, the assessment has considered all land uses surrounding the proposed Plant, with specific focus on key receptor locations modelled in the Air Quality Emissions Assessment (PDP 2022). The modelling has focused on a range of receptors as shown on **Figure 2.1**. These include 14 receptor locations surrounding the site, and three receptors at each of the larger towns of Oamaru, Waimate and Duntroon. The receptor locations identified relate to rural residential or residential homes and schools surrounding the proposed Plant.

Table 2.1 presents a summary of the number of receptors evaluated and the distance to the proposed Plant.

In addition to these individual receptor locations, PDP (2022) has also modelled potential impacts at two nested grids. These are centred on the proposed Plant and extend 2,000 m and 3,500 m with a receptor spacing of 50 m and 100 m respectively. The grid is used to ensure that impacts at all locations outside of the Plant boundary are evaluated.

Table 2.1: Summary of receptor locations (PDP 2022)

Receptor ID	Address	Closest Distance to Plant (m)	Direction Relative to Plant
R1	77 Mairos Road, Morven	1,250	N
R2	190 Mairos Road, Morven	1,300	NE
R3	197 Mairos Road, Morven	1,500	NE
R4	362 Archibalds Road, Morven	3,400	NE
R5	540 Archibalds Road, Morven	4,300	NE
R6	425 Carrolls Road, Glenavy	2,350	E
R7	91 Andrews Road, Glenavy	1,800	SE
R8	70 Andrews Road, Glenavy	1,600	SE
R9	319 Andrews Road, Glenavy	3,400	SE
R10	42 Parker Street, Glenavy	2,000	S
R11	Glenavy School	2,300	S
R12	26 Te Maiharoa Road, Glenavy	1,750	SW
R13	192 Glenavy Tawai Road, Glenavy	2,800	SW
R14	4636 Waimate Highway, Morven	1,800	W
R15	212 Waihao Back Road, Waimate	16,000	NW
R16	387 McEneany Road, Pukeuri	15,500	S
R17	Duntroon School	33,000	W

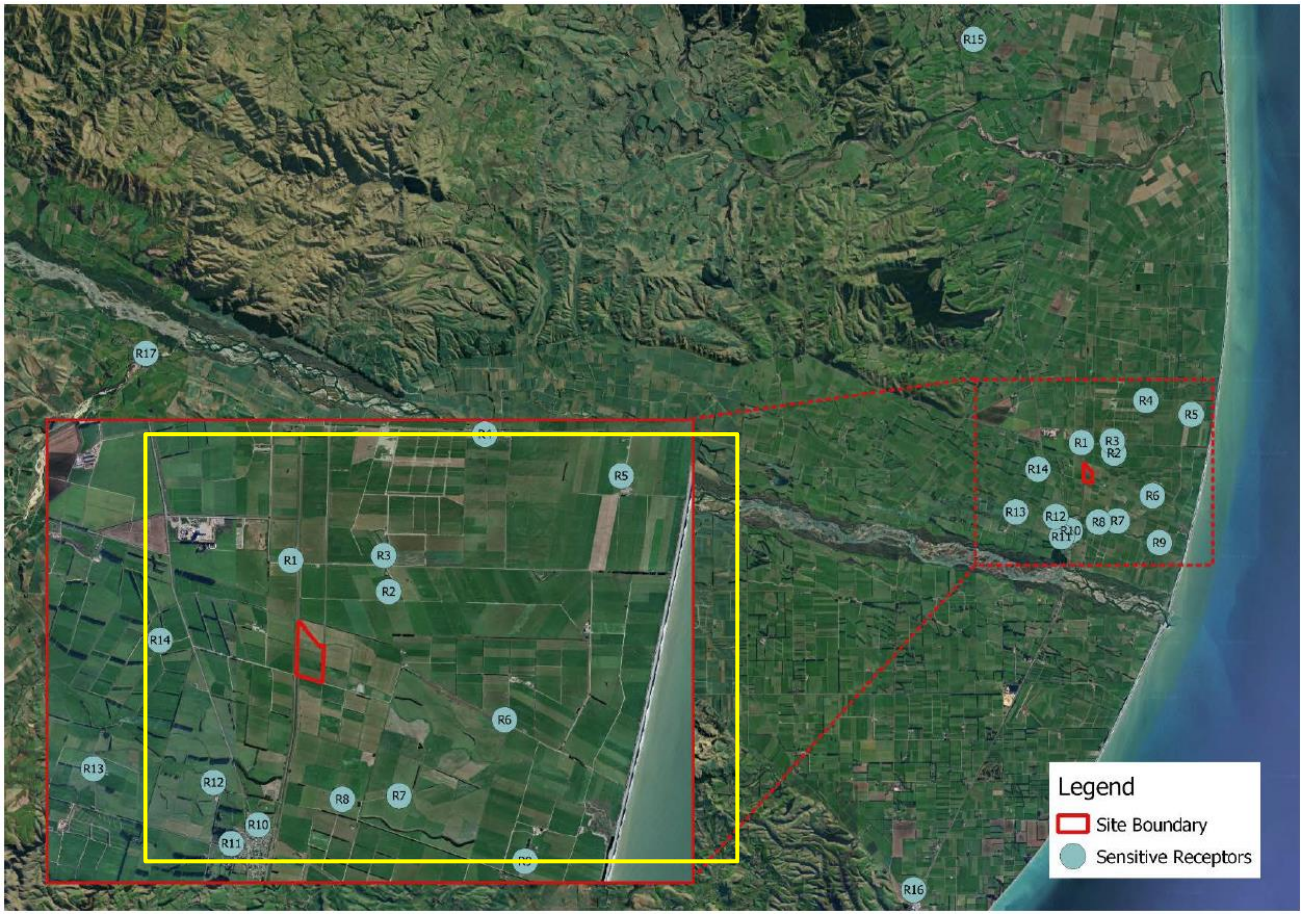

Figure 2.1: Location of sensitive receptors surrounding the proposed Plant

2.3 Demographics

The proposed Plant and most of the receptors evaluated are located within the Waimate District, with the town of Oamaru located within the Waitaki District. For the purpose of this assessment, the demographics and health of the population within the Waimate District has been considered representative of the community closest to and surrounding the proposed Plant.

Table 2.2 presents a summary of the population demographics for the community surrounding the proposed Plant. These data are based on data available from the 2018 Census data available Stats NZ[1]. The data presented include the NZ Index of Deprivation for 2018 from Environmental Health Intelligence New Zealand[2]. In general, people in more deprived areas are more susceptible to environmental risks. They may have less capacity to cope with the effects of environmental risks and fewer resources to protect themselves from environmental risks.

Table 2.2 also provides some review of the demographics data relevant to the population surrounding the proposed Plant to indicate where the population may be more or less vulnerable, compared with the larger populations in the Canterbury Region and New Zealand.

The vulnerability of the population is considered to potentially reflect the ability of the population to adapt to environmental change and stressors. Communities with higher rates of unemployment, ranked more socioeconomically deprived, with higher proportions of young children or the elderly are considered to be potentially more vulnerable to the environmental stressors considered in this assessment.

Table 2.2: Summary of populations surrounding the proposed Plant

Indicator	Waimate District	Canterbury Region	New Zealand
Total population	7,815	599,694	4,699,755
Population 0 - 4 years	5.3%	5.8%	6.3%
Population 5 - 19 years	17.0%	18.6%	19.8%
Population 20 - 64 years	55.2%	59.7%	58.7%
Population 65 years and over	22.7%	16.0%	15.2%
Median age	46.4	38.7%	37.4
Unemployment – all people	2.7%	3.2%	4%
Level 1 to 6 certificate or diploma	55.4%	53.2%	51.1%
Tertiary education	12%	22.5%	24.8%
NZ Index of Deprivation (socioeconomic)	5 for Morven-Glenavy-Ikawa 5 for Makikihi-Willowbridge		
Top 4 countries of birth (if not NZ)	United Kingdom and Ireland Asia Australia Europe (excluding UK and Ireland)	Asia United Kingdom and Ireland Europe (excluding UK and Ireland) Australia	Asia United Kingdom and Ireland Pacific Islands Middle East and Africa

Index of Deprivation (socioeconomic), with 1 = least deprived and 10 = most deprived
Shading relates to comparison against Canterbury and NZ (potential): more vulnerable; less vulnerable

[1] https://www.stats.govt.nz/

[2] https://www.ehinz.ac.nz/indicators/population-vulnerability/socioeconomic-deprivation-profile/

Based on the population data available and presented in **Table 2.2**, the community in the area surrounding the proposed Plant are generally similar to the larger population in Canterbury and New Zealand overall with the exception of the following:

- the population surrounding the proposed Plant are small which would result in more variable statistics for these areas compared with larger population areas
- the population in Waimate has a lower proportion of young children (aged 0 to 4 years) but a higher proportion of older people (aged 65 years and older) which is reflected in the higher median age.

Overall, the demographics data do not indicate any aspects that suggest the population would have any increased vulnerability to project related impacts in the communities surrounding the proposed Plant.

2.4 Existing community health

The health of the community is influenced by a complex range of interactive factors including age, socio-economic status, social capital, behaviours, beliefs and lifestyle, life experiences, country of origin, genetic predisposition and access to health and social care. The health indicators available and reviewed in this report (**Table 2.3**) generally reflect a wide range of these factors.

The population in the area surrounding the proposed Plant is relatively small and health data specifically relating to this population are not available. However, it is assumed that the health of the local community is consistent with that reported in the Canterbury District and Canterbury District Health Board.

Table 2.3 presents a summary of the general population health considered relevant to the area. The table presents available information on health-related behaviours (i.e., key factors related to lifestyle and behaviours known to be of importance to health) and indicators for the burden of disease within the community compared to New Zealand.

Table 2.3: Summary of health indicators/data

Health indicator/data[1]	Waimate District	Canterbury Region	New Zealand
Health behaviours (age-standardised rates)			
Children - compliance with fruit consumption guidelines (2017-2020)	--	72.9%	73.1%
Children - compliance with vegetable consumption guidelines (2017-2020)	--	57.2%	48.1%
Children – body weight as overweight or obese (2017-2020)	--	18.9%	20.1%
Adults – compliance with fruit consumption guidelines (2014-2017)	--	51.7%	51.5%
Adults – compliance with vegetable consumption guidelines (2017-2020)	--	56.6%	52.4%
Adults (15 years and older) – hazardous drinking (2016-2017)	--	20.8%	21.1%
Adults – body weight (overweight) (2017-2020)	--	34.7%	33.5%
Adults – body weight (obese) (2017-2020)	--	25.5%	30.6%
Adults – physically active (2017-2020)	--	55.7%	52.9%
Health behaviours from 2018 Census data			
Adults – activity limitations (i.e. a lot of difficulty or cannot do at all)	7.7%	6.3%	6.5%
Children (under 15 years) – activity limitations (i.e. a lot of difficulty or cannot do at all)	2.2%	2.8%	3%
Current smoker, adult	16.2%	12.3%	13.2%
Burden of disease (95% confidence interval) as age-adjusted rate per 100,000 unless indicated otherwise			
Cardiovascular disease hospitalisations (all ages, 2013-2015)[3]	--	911.8 (898.8-924.9) (also refer to Note 1)	955.8 (951.2-960.3)
Asthma hospitalisations (0-14 years, 2013-2015)[3]	--	354.4 (332.2-377.7) (also refer to Note 2)	325.3 (318.4-332.3)
Asthma hospitalisations (15 years and older, 2013-2015)[3]	--	71.2 (66.3-76.4)	71.1 (69.4-72.8)
Mortality – all causes, all ages (2019)[2]	--	--	373.6
Mortality – avoidable, 0-74 years (2011-2013)[3]	--	131.4 (126.1-136.9)	144.3 (142.4-146.2)
Mortality – respiratory (all ages) (2019)[2]	--	--	30.4
Mortality – cardiovascular (all ages) Data for 2011-2013[3] Data for 2019[2]	 -- --	 121.0 (117.1-125.1) --	 118.6 (117.2-120.0) 101.7
Adult asthma – current (2017 - 2020)[1]	--	13.0%	11.8%
Children with asthma (to 15 years) – current (2017 - 2020)[1]	--	10.9%	13.9%

1 Data from the Ministry of Health – New Zealand Health Survey: https://minhealthnz.shinyapps.io/nz-health-survey-2017-20-regional-update/_w_5ea00edc/#!/home

2 Data from the Ministry of Health, Mortality Web Tool: https://www.health.govt.nz/publication/mortality-web-tool

3 Data from the Ministry of Health – Environmental Health Intelligence New Zealand (ehinz) for Canterbury District Health Board (DHB): https://www.ehinz.ac.nz/projects/health-profiles/

Shading relates to comparison against New Zealand:

statistic/data suggestive of a potential higher vulnerability within the population to health stressors.

statistic/data suggestive of a potential lower vulnerability within the population to health stressors.

Note 1: In addition to the statistics presented in relation to cardiovascular disease, the following is of note for all statistics in NZ: males have statistically significantly higher rates than females; Māori and Pacific people have statistically significantly higher rates than people of other ethnicity; Asian people have statistically significantly lower rates than people of other ethnicity. In relation to the Canterbury DHB, it is noted that people in this area (overall and for Māori people) have a statistically significantly lower rates compared with New Zealand overall.

Note 2: In addition to the statistics presented on asthma hospitalisations for children, the following is noted for all statistics in New Zealand: males have statistically significantly higher rates than females; Māori, Pacific people and Asians have statistically significantly higher rates than people of other ethnicity. In relation to the Canterbury DHB, it is noted that people in this area (overall, for males and for Pacific people) have a statistically significantly higher rates compared with New Zealand overall.

The key indicators of health for the population in areas surrounding the proposed Plant indicate the following are different, when compared with the data for New Zealand:

- There is limited data available for the smaller population in the Waimate District. The data that is available indicates a higher proportion of adults with activity limitations and are smokers, and a lower proportion of children with activity limitations.
- The population in the Canterbury District has a higher proportion of the child population who consume the recommended intake of vegetables, and there is a lower proportion of the adult population (and to a lesser degree children) determined to be obese. Adults in the area may be less active and have higher rates of smoking, however children may be more active in this area.
- The population for the Canterbury District Health Board area has a higher rate of mortality for cardiovascular disease, however the rate of hospitalisations for cardiovascular disease is lower. The rate of asthma hospitalisations for asthma in children is higher, however the rate of asthma in children is lower (potentially suggesting asthma in children is less well managed in this area). The rate of avoidable mortality is lower for this area.

The above indicates that, based on existing health related behaviours and health statistics, the data is variable and there are no significant indications that the population could considered vulnerable to project related stressors. Further, as with all population health data, the statistics presented above relate to a large population. No data are available for the smaller population in the areas immediately surrounding the proposed Plant.

Section 3. Modelled air emissions

3.1 Outline of emission sources relevant to the proposed Plant

The key component of the proposed Plant relates to emissions to air from the combustion of waste and the combustion of diesel (i.e., the operation of the diesel generators).

The proposed Plant would utilise proven Best Available Techniques (BAT) (EU 2019) as defined by the Industrial Emissions Directive 2010/75/EU (IED) (EU 2010).

The Air Quality Report Air Quality Report (PDP 2022) has evaluated emissions to air from the proposed Plant based on guaranteed emission rates provided for the operation of the proposed Plant. These emission rates are detailed in **Table 3.1**, along with a comment as to the basis for the emission rate adopted in the assessment.

Table 3.1: Guarantee emission rates for proposed Plant

Pollutant	Daily average value (mg/Nm^3)	Emission rate (g/s)	Comments
Particulates	5	0.76	Based on BAT upper limit, and lower than the IED emission limit
Hydrogen chloride (HCl)	6	0.91	Based on BAT upper limit, and lower than the IED emission limit
Hydrogen fluoride (HF)	1	0.15	Based on BAT and IED emission limit
Sulfur dioxide (SO_2)	30	4.57	Based on BAT upper limit, and lower than the IED emission limit
Oxides of nitrogen (NO_2)	120	18.26	Based on BAT upper limit, and lower than the IED emission limit
Carbon monoxide (CO)	50	7.61	Based on BAT and IED emission limit
Total volatile organic compounds (TVOCs)	10	1.52	Based on BAT and IED emission limit. The modelling of TVOCs has focused on individual VOCs, namely benzene, toluene, xylenes and trimethylbenzenes (noting PDP adopted the same emission rate for each individual VOC, which assumes that each individual VOC is released at the same rate, being equal to the total VOC emission rate)
Ammonia (NH_3)	10	1.52	Based on BAT upper limit
Mercury (Hg)	20	0.003	Based on BAT upper limit, and lower than the IED emission limit
Total metals (excluding mercury)	0.3	--	Based on BAT upper limit, and lower than the IED emission limit. The total metal emission rate is 0.076 g/s which is 1% of total particulates. Where the composition of individual metals (as proportion of particulates) is known or can be estimated it has been used to determine individual metal concentrations. Where not known it is assumed 100% of the total metals emissions comprises the individual metal. This approach will significantly overestimate total metals. Metals evaluated that are relevant to the assessment of health are antimony, arsenic, beryllium, cadmium, chromium, cobalt, copper, lead, manganese, nickel, selenium, thallium, tin and vanadium
Dioxins (as toxicity equivalents, TEQ)	0.06 ng/Nm^3	9.13×10^{-9}	Lower than IED emission limit

It is expected that actual emissions to air from the proposed Plant would be less than assumed in **Table 3.1**.

3.2 General concepts relevant to air modelling

To be able to determine the concentration of pollutants that may be in the air, off-site within the community, from a proposed project (i.e., one that has not yet been built), an air dispersion model has to be used. The model uses a range of information such as:

- the concentration (or emission rate) of pollutant in the stack before discharge
- information about the stack itself such as height and width at the top, the discharge velocity and temperature as well as the presence of any tall buildings close to the stack
- information about the meteorological conditions
- information about the terrain in the surrounding areas.

All this information is used to estimate how the pollutants are mixed and transported in the air and the concentration that may be present at ground level at different locations.

Figures 3.1 and 3.2 illustrate the processes which govern how the emissions get mixed into the atmosphere.

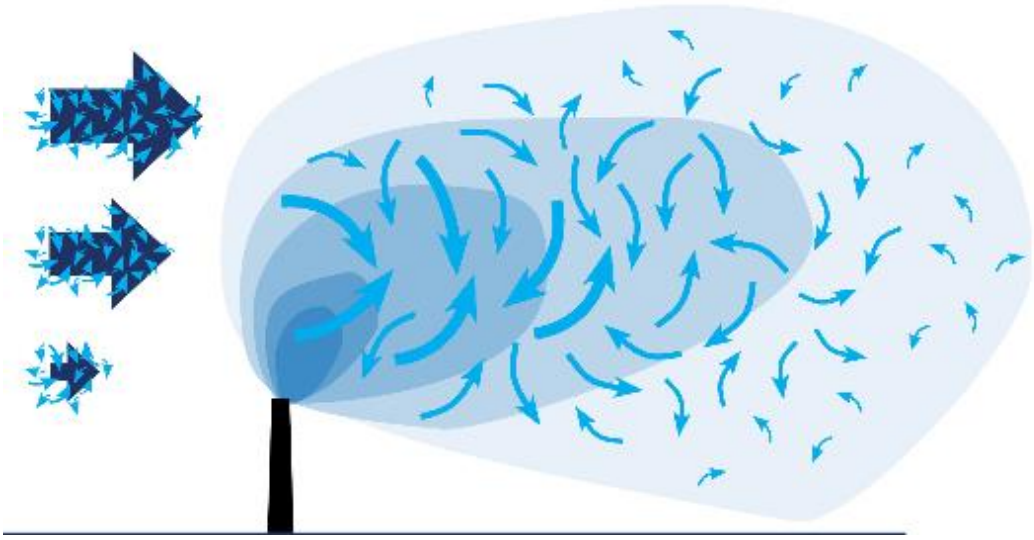

Figure 3.1: Turbulence in the air, how it mixes and dilutes pollutants emitted from a stack (NSW Chief Scientist 2018)

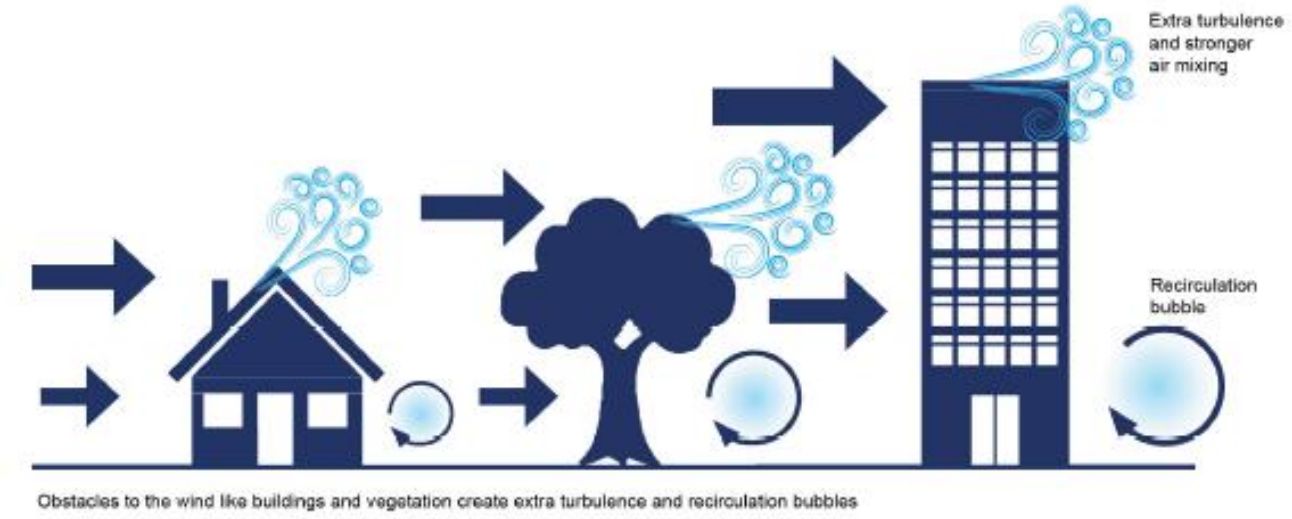

Figure 3.2: Turbulence in the air and how it is affected by buildings and vegetation (NSW Chief Scientist 2018)

Gases (and any fine particles such as PM_{10} and $PM_{2.5}$ that remain after flue gas treatment) are emitted at around 413^{o}C and are pushed out of the stack using fans (i.e. at some speed) so these gases (and fine particles) rise or are pushed up significant distances above the top of the stack – because hot gases rise and because these gases are travelling at a faster speed than the air surrounding the stack. This can be seen in the figures above.

As the gases (and fine particles) cool and slow down they begin to interact with the wind above the stack (i.e., well above the 75 m stack height). This mixes the gases (and fine particles) into the atmosphere decreasing the actual concentration present in any one particular place.

Figure 3.1 shows that most of the pollutants remain up in the atmosphere away from where people would be exposed. However, small amounts do eventually reach ground level. The air dispersion modelling determines what proportion of the amount in the stack could reach ground level at different locations. Such modelling looks at worst case weather characteristics (that can actually occur – based on real meteorological data) to ensure that the amount that could reach ground level in areas where people live or work neighbouring the proposed facility are not underestimated. It is these ground level concentrations that are then used to assess potential for health impacts.

Data from the modelling can also be used to estimate the rate at which particles in the emissions could fall out of the atmosphere (due to gravity) or get washed out of the atmosphere (due to rain). It is this deposition rate that is then used to estimate how much of chemicals attached to particles could get into soil or water around the facility.

3.3 Overview of Project air modelling

To predict the concentration of emissions from the proposed Plant, a study area was defined and shown in **Section 2.2** and predicted emissions from the stacks, and scrubber stack, were modelled by PDP (2022) using the CALPUFF air dispersion model.

The CALPUFF air dispersion model is a regulatory air pollution model accepted in New Zealand (MfE 2004a) that was selected based on the need to evaluate complex terrain and heterogeneous land use (relevant to the area evaluated). This model uses air emission estimates for the proposed Plant, plant design (for example, stack height and building sizes), local terrain and meteorological data to predict the ground level concentrations of emissions within the defined study area. Meteorological data for the study area was generated by PDP using CALMET from data from the Waimate Meteorological site.

Background air quality is influenced by a range of sources in the areas around the proposed Plant. This has been evaluated by PDP (where relevant) based on background air quality relevant to the whole country developed by New Zealand Transport Agency Waka Kotahi, for the Waihao area. These data are considered more relevant to rural areas than the closest air monitoring station at Waimate as the station at Waimate is significantly influenced by urban sources.

It is important to note that there are always a range of chemicals present in the air we breathe. The issue that is important for a new facility is whether the facility will change these levels significantly.

Full details on the air model are presented in the Air Quality Report (PDP 2022). This model is used to provide predicted air concentrations and particulate deposition rates over the study area and at all the individual receptor locations (as detailed in **Section 2.2** and **Figure 2.2**), with the results averaged over different time periods.

Section 4. Detailed assessment of potential health impacts from air emissions

4.1 General

This section presents a detailed assessment of potential risks to human health as a result of emissions to air from the proposed Plant. The assessment of risk has relied on air modelling presented in the Air Quality Report (PDP 2022) and follows the risk assessment principles detailed in guidance referenced in **Section 1.5**. This approach requires assessment of:

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

Figure 4.1 presents an overview of the assessment approach detailed in the following sections.

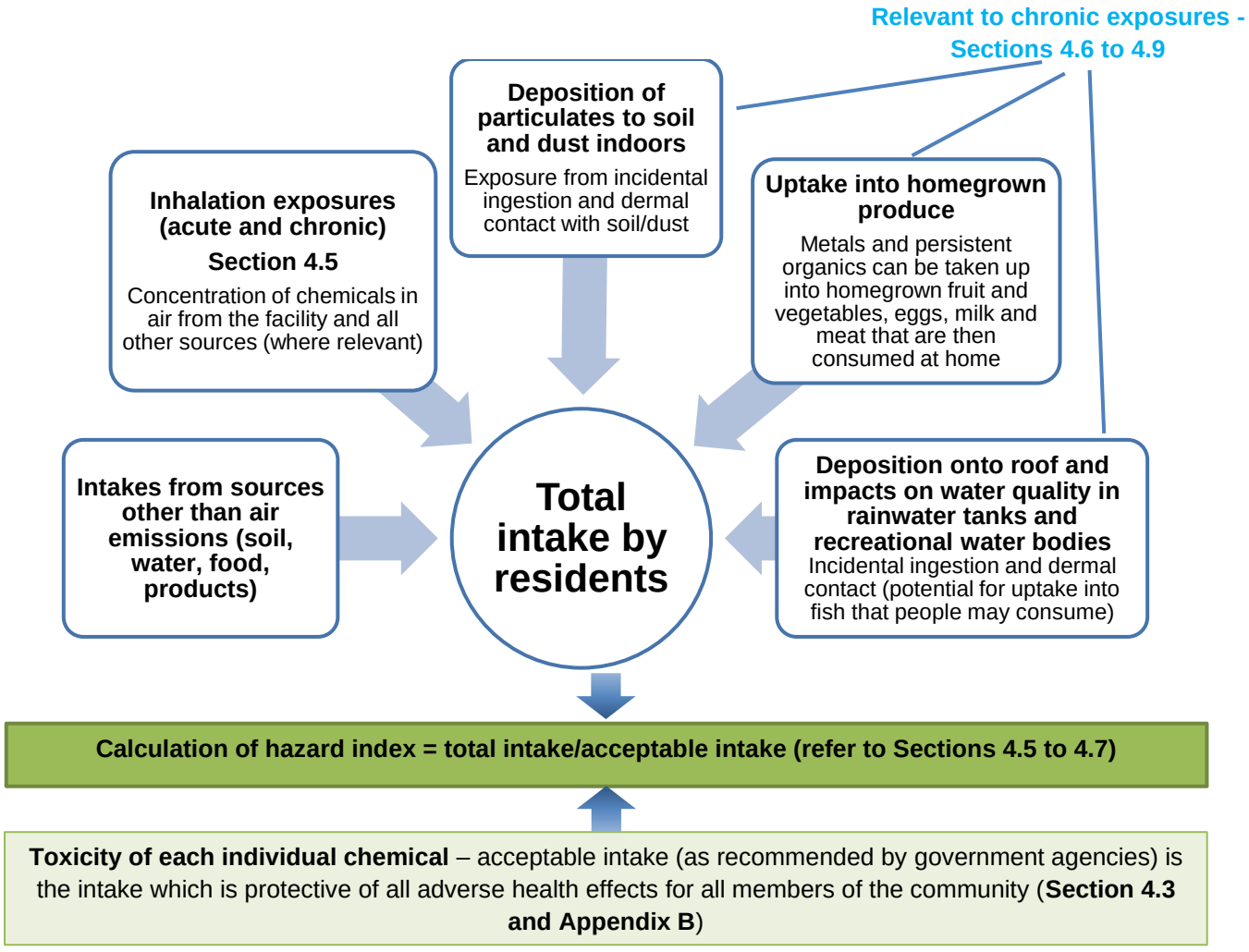

Figure 4.1: Overview of health risk assessment

4.2 Exposure assessment – conceptual site model

Understanding how a community member may come into contact with pollutants released in air emissions from the proposed Plant 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.

There are three main ways a community member may be exposed to a chemical substance emitted from the plant:

- inhalation of gases, vapour or fine particulate matter in air
- direct contact, which may include ingestion and/or dermal absorption of chemicals present in dust that may deposit onto surfaces or accumulate in water collected in rainwater tanks or water in recreational areas
- ingestion of persistent and bioaccumulative chemicals that may be deposited to soil and then taken up into homegrown produce that may be consumed.

For some of the emissions from the proposed Plant, inhalation is considered the only route of exposure. This is due to the substance's chemical properties, which make the other pathways inconsequential. This includes gases such as nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), hydrogen chloride (HCl), ammonia (NH_3), hydrogen fluoride (HF) and VOCs (assessed as benzene, toluene, xylenes and trimethylbenzenes) as well as fine particulate matter as particulates less than 2.5 micrometres ($PM_{2.5}$) that are so small they remain suspended in air (i.e. inhalation only exposure pathway).

Other chemicals in the emissions may be inhaled, but they may also be deposited on the ground/surfaces with the deposition of dust. These emissions can then be ingested either directly through accidental/incidental consumption of soil or indirectly through food/produce grown or raised in the soil (fruit, vegetables, eggs, meat or milk), or in drinking water where dust is deposited onto a roof where it may be washed into and affect water quality in rainwater tanks. Skin contact with the soil and water in rainwater tanks is also possible. Therefore, it is important with these emissions that all exposure pathways are considered. In this instance, metals and dioxin-like chemicals that are bound to the heavier particulate matter that may fall out and deposit onto the ground could be considered for these exposure pathways.

Table 4.1 lists the pollutants or chemicals evaluated in the Air Quality Report from the proposed Plant (from all emission sources evaluated) and the exposure pathway/s of potential concern.

It is noted that the list of individual metals evaluated in this assessment comprises key metals[3] detected in flue gas particulates (as per PDP 2022) excluding inorganics and metals of very low toxicity to human health, and other metals specifically listed in the Best Available Techniques (BAT) (EU 2019) as defined by the Industrial Emissions Directive 2010/75/EU (IED) (EU 2010) or expected to be present in such emissions and sufficiently toxic to require assessment. **Figure 4.2** provides a

[3] Key metals are those that are sufficiently toxic to have the potential be of concern to human health and where robust toxicity reference values are available to enable quantification of effects to be undertaken

diagrammatical representation of the community exposures to emissions from the facility (conceptual site model).

Table 4.1: Substances and routes of exposure

Substance	Route of exposure
Nitrogen dioxide	**Inhalation only** as these are gases.
Sulfur dioxide	
Hydrogen chloride	
Hydrogen fluoride[1]	
Carbon monoxide	
Ammonia	
Volatile organic compounds (VOCs) as benzene, toluene, xylenes and trimethylbenzenes	
PM_{10}	**Inhalation relevant for particulates based on particle size** as these particulates are very small and will remain suspended in air. It is noted that other exposure pathways have also been assessed for the individual chemical substances bound to these particles that may be deposited to the ground. These other pathways relate to the individual chemical substances, rather than the physical size of the particulates, however, they do relate to the more coarse fractions of dust in PM_{10} (rather than $PM_{2.5}$) as some PM_{10} will deposit to the ground.
$PM_{2.5}$	
Antimony	**Inhalation** of these pollutants adhered to fine particulates. **Ingestion and dermal contact** with these pollutants deposited to soil, deposited to a roof where they wash into and impact on water quality in rainwater tanks, It is recognised that the surrounding rural and residential areas include rainwater tanks that are used for drinking water/potable water. **Ingestion** of produce grown in soil potentially impacted by these pollutants. For this assessment, the surrounding rural residential areas may include homegrown fruit and vegetables, eggs, home consumed beef and lamb as well as crops such as oats, barley and canola. Metals, dioxins/furans, dioxin-like PCBs and PAHs can be taken up/bioaccumulated into plants and animal products that may be consumed.
Arsenic	
Beryllium	
Cadmium	
Chromium	
Copper	
Cobalt	
Lead	
Manganese	
Mercury	
Nickel	
Selenium	
Thallium	
Tin	
Vanadium	
Dioxins-like chemicals	

For some of the pollutants evaluated, a conservative approach has been adopted for an individual pollutant or group of chemicals where the composition is less well known. The following conservative assumptions have been adopted in this assessment:

- Particulates are assumed to be present as 100% TSP, 100% PM_{10} and 100% $PM_{2.5}$
- Dioxins have been assessed assuming this comprises all dioxin-like chemicals and the group is characterised by the toxicity of the most potent compound, 2,3,7,8-TCDD, assuming that the emissions limits evaluated relate to a WHO toxicity equivalent concentration (as WHO-TEQ from WHO in 2005)
- Chromium exposures have been assessed assuming all chromium present is present as chromium VI, the most toxic form of chromium
- Inorganic mercury exposures have been assessed assuming that it is present in air as elemental mercury (the more toxic form), and when deposited to the ground forms inorganic mercury

- The more general chemical group of volatile organic compounds (VOCs) includes a large number of individual volatile chemicals with varying toxicities. For this group, it has been conservatively assumed that this group is represented by benzene, one of the more toxic (and likely) components of VOCs expected to be present.

4.3 Hazard assessment

This assessment has addressed potential exposures to chemicals present in emissions to air from the proposed Plant. The chemicals evaluated, as listed in **Table 4.1**, include gases and particulates as well as metals and organics that are bound to the particulates. This assessment has addressed acute inhalation exposures, along with chronic inhalation and multi-pathway exposures. To quantify the potential for the chemicals of concern in relation to health risks, the hazards associated with these chemicals have been quantified for acute and chronic inhalation, and chronic oral and dermal exposures, using current and robust toxicity reference values (TRVs).

Appendix B presents further discussion and detail relating to the TRVs adopted for the quantification of hazards for the chemicals evaluated in this assessment. Some additional discussion on hazards and the TRVs or health-based guidelines adopted is also presented in **Section 4.5**, with information specific to assessing particulate size, nitrogen dioxide, sulfur dioxide and carbon monoxide presented in relevant subsections.

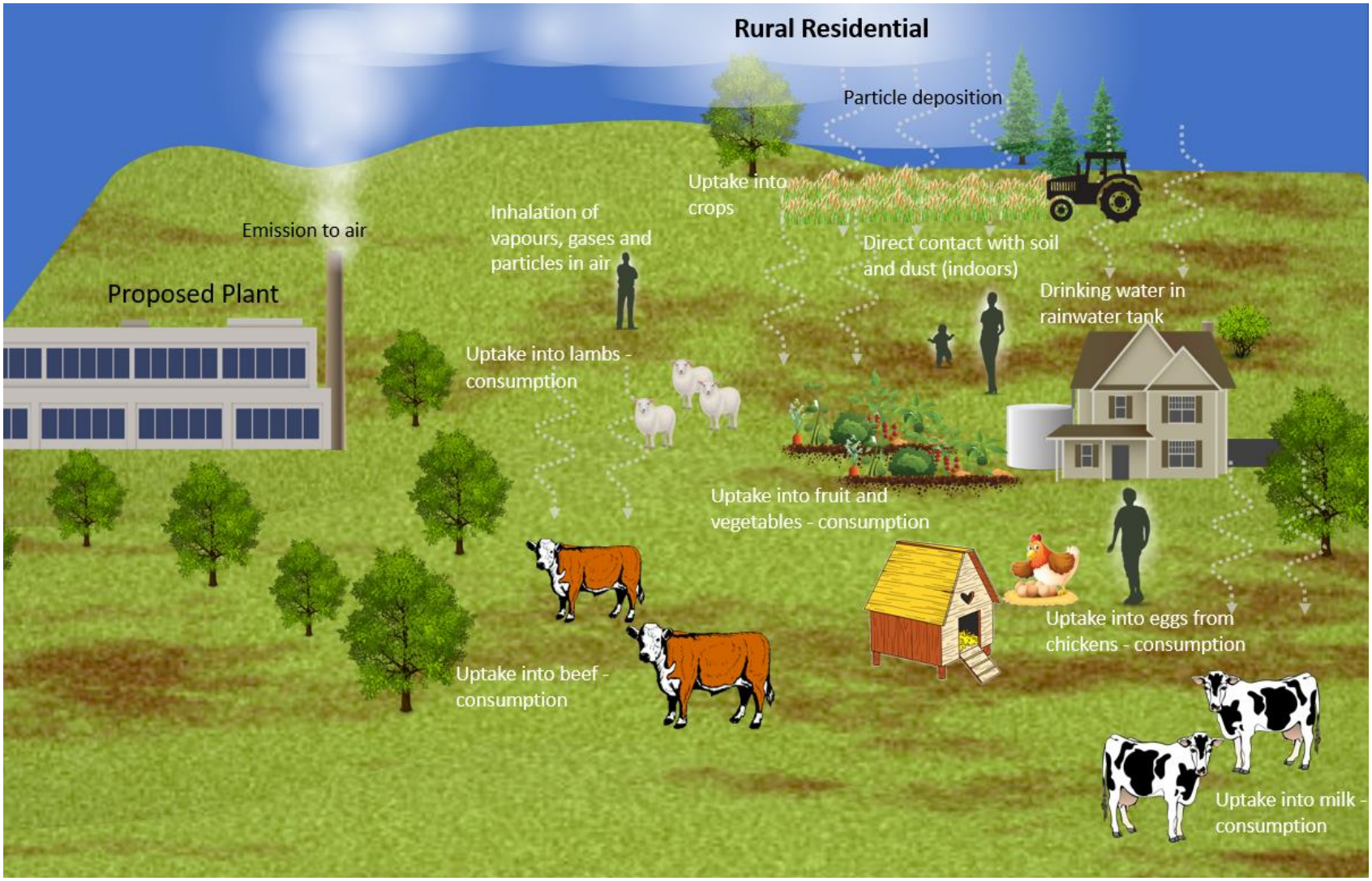

Figure 4.2: Conceptual site model (illustrative only, not to scale)

4.4 Use of air modelling data in HHRA

This assessment has evaluated exposures and risks relevant to emissions to air from the proposed Plant where operating at the guaranteed emission limits detailed in **Table 3.1**. These are expected to provide a conservative assessment of upper bound emissions from the facility.

Ground level concentrations for gases and particulate bound metals and organics (assuming 100% are present as $PM_{2.5}$), and deposition rates for particle bound metals and organics (bound to dust, assumed to be as TSP where 100% of the particulates are assumed to be in this fraction) have been predicted by PDP and provided for use in this assessment.

The focus of this assessment relates to the evaluation of health impacts that may occur as a result of acute or chronic exposures to emissions from the facility. This requires the use of 1-hour average (for the assessment of acute exposures to most pollutants except particulates), 24-hour average data (for short-term exposures to particulates) and annual average (for the assessment of chronic exposures) data. All data required for use in this assessment have been provided by PDP (2022) and are from the same model as presented in the Air Quality Report.

The modelling undertaken has provided the following for use in this assessment:

- estimated ground level concentrations for gases and vapours as well as chemicals bound to particulates, assuming 100% of the particulates are present as $PM_{2.5}$ (which is of most relevance to the assessment of health effects)
- estimated deposition rates for chemicals bound to particulates, based on the deposition of dust which is assumed to be total suspended particulates (TSP) which include the larger sized particulates and PM_{10} and $PM_{2.5}$.

In relation to the assessment of individual metals, where the proportion of each individual metal as a % of particulates has been determined by PDP (2022) based on the measured proportion or estimated proportion, this has been used to further speciate the concentrations of the individual metals in air or deposited dust from the modelled total metals data. Where the speciation is not known 100% of the total metals concentration or dust deposition rate has been adopted (which is conservative).

The modelling provides data relevant to emissions from proposed Plant. Where relevant, background air concentrations have also been included. Where background has not been included, the quantification of risk (for chronic risks) has included consideration of intakes from other sources, where such intakes are significant.

Risk calculations, relevant to long-term or chronic exposures have been presented for the following locations within the community:

- **Maximum impacted location** which includes all modelled locations regardless of location and use – this is a location on or close to the boundary, where inhalation exposures by workers and visitors may occur on occasion. Inhalation exposures have been assumed to occur for 8 hours per day, 230 days per year for 20 years at this location to provide a conservative maximum.

- **Maximum impacted sensitive receptor** – this is the maximum impacted receptor from the individual residential, rural residential or schools shown on **Figure 2.2**. Exposures (inhalation and multi-pathway) are assumed to occur for 24 hours per day, 350 days per year for 30 years at this location.

Acute inhalation exposures have also been calculated on the basis of the maximum 1 hour average air concentration assuming anyone may be present at the location of maximum impact.

4.5 Inhalation exposures

4.5.1 General

For all the pollutants released to air from the proposed facility, whether present as a gas or as particulates, there is the potential for the community to be exposed via inhalation. Assessment of potential health impacts relevant to inhalation exposures for these pollutants is discussed below.

4.5.2 Particulates (size)

Health effects

Adverse health effects associated with exposure to particulate matter have been well studied and reviewed by Australian, New Zealand and International agencies. Most of the studies and reviews have focused on population-based epidemiological studies in large urban areas in North America, Europe and Australia/New Zealand, where there have been clear associations determined between health effects and exposure to $PM_{2.5}$ and to a lesser extent, PM_{10}. The robustness/quality of these studies and the weight of evidence established by these studies is an important aspect for determining wither specific adverse health effects are important (USEPA 2019, 2022a; WHO 2021). Detailed reviews into whether the findings of the epidemiological studies can support causal links with exposure to particulate matter also consider whether the findings can be supported by animal and cellular toxicity studies and studies on inhalation toxicity by human volunteers (NEPC 2010; USEPA 2019, 2022a). Not all detailed reviews have considered the supporting mechanistic, toxicological and clinical evidence (Kuschel et al. 2022b; WHO 2021)

Particulate matter has been linked to adverse health effects after both short-term exposure (days to weeks) and long-term exposure (months to years). The health effects associated with exposure to particulate matter vary widely (with the respiratory and cardiovascular systems most affected) and include mortality and morbidity effects.

In relation to mortality, for short-term exposures in a population this relates to the increase in the number of deaths due to existing (underlying) respiratory or cardiovascular disease; for short-term exposures as well as long-term exposures in a population this relates to mortality rates over a lifetime, where long-term exposure is considered to accelerate the progression of disease or even initiate disease.

In relation to morbidity effects, this refers to a wide range of health indicators used to define illness that have been associated with (or caused by) exposure to particulate matter. In relation to exposure to particulate matter, effects are primarily related to the respiratory and cardiovascular system and include (Morawska, Moore & Ristovski 2004; USEPA 2009c, 2018b, 2022a):

- aggravation of existing respiratory and cardiovascular disease (as indicated by increased hospital admissions and emergency room visits)
- changes in cardiovascular risk factors such as blood pressure
- changes in lung function and increased respiratory symptoms (including asthma, as indicated by increased hospital admissions and emergency room visits)
- changes to lung tissues and structure
- altered respiratory defence mechanisms.

The most recent detailed review of the available studies (USEPA 2019, 2022a; WHO 2021) have also indicated that effects on the nervous system and carcinogenic effects are likely to have a causal relationship with long-term exposures to $PM_{2.5}$. IARC (2013) has classified particulate matter as carcinogenic to humans based on data relevant to lung cancer.

There are a number of studies that have been undertaken where other health effects have been evaluated. These studies have a large degree of uncertainty or a limited examination of the relationship and are generally only considered to be suggestive or inadequate (in some cases) of an association with exposure to $PM_{2.5}$ (USEPA 2019). This includes long term exposures and metabolic effects, male and female reproduction and fertility, pregnancy and birth outcomes; and short term exposures and nervous system effects (USEPA 2019).

These effects are commonly used as measures of population exposure to particulate matter in community epidemiological studies (from which most of the available data in relation to health effects is derived) and are more often grouped (through the use of hospital codes) into the general categories of cardiovascular morbidity/effects and respiratory morbidity/effects. The available studies provide evidence for increased susceptibility for various populations, particularly older populations, children with suggestive evidence suggesting those with underlying health conditions , genetic factors, socioeconomic status and smoking are relevant (USEPA 2019).

There is consensus in the available studies and detailed reviews that exposure to fine particulates, $PM_{2.5}$, is associated with (and causal to) cardiovascular and respiratory effects and mortality (all causes) (USEPA 2012b). While similar relationships have also been determined for PM_{10}, the supporting studies do not show relationships as clear as shown with $PM_{2.5}$ (USEPA 2012b, 2019). The focus of the more recent review of particulate matter impacts in New Zealand focused on $PM_{2.5}$ with supporting information and evaluations in relation to PM_{10} (Kuschel et al. 2022a, 2022b).

Review of the available studies by the WHO (WHO 2021) and in New Zealand (Kuschel et al. 2022a, 2022b) identified critical or primary health outcomes, and exposure-response relationships that are most relevant to evaluate these impacts within populations. These critical or primary health outcomes, relevant to long-term exposures to $PM_{2.5}$. are as follows:

- Premature morality (all cause), particular for adults aged 30 years and older
- Premature mortality for specific cases (as noted by the WHO), namely cardiovascular disease, respiratory disease and lung cancer
- Hospital admissions in relation to cardiovascular disease (including stroke) for all ages
- Hospital admissions in relation to respiratory disease for all ages
- Restricted activity days for all ages.

The available evidence does not suggest a threshold below which health effects do not occur. Accordingly, there are likely to be health effects associated with background levels of $PM_{2.5}$ and PM_{10}, even where the concentrations are below the current guidelines.

These exposure-response relationships are used by key organisations to establish ambient air quality goals/criteria and assess the impact (health costs/savings) of various air policy decisions. Such assessments also consider background levels.

The exposure-response relationships adopted to assess these effects are based on large epidemiological studies that relate to changes in population health with changes in $PM_{2.5}$. As a result, they are used to determine and review air quality goals and standards that relate to population average or regional exposures from all sources. They do not relate to exposures in localised 'hot-spot' areas such as locations near industry, busy roads or mining. Hence it is generally not appropriate to apply the exposure-response relationships identified to assess localised impacts from a specific project, as the populations impacted are very small (or individuals) and not consistent with the populations evaluated in the epidemiological studies and the existing health status is not known.

Assessment of exposure

The principal approach to the assessment of potential impacts on community health to changes in particulate matter concentrations as a result of the Project is to assess compliance with ambient air quality guidelines established for PM_{10} and $PM_{2.5}$. These guidelines are those established following evaluation of the available evidence and exposure-response relationships for adverse health effects in New Zealand populations.

Assessment of potential health impacts associated with exposure to particulate matter has been undertaken and presented within the Air Quality Report (PDP 2022). The assessment of particulates has assumed that 100% of the particulate emissions may be present as PM_{10} and 100% of the emissions may be present as $PM_{2.5}$, which is a highly conservative approach.

New Zealand currently provides a National Environmental Standards for Air Quality (NESAQ) for PM_{10} as 50 $\mu g/m^3$ over a 24-hour averaging period (MfE 2004b) as well as an ambient air quality guideline of 20 $\mu g/m^3$ for an annual average (MfE 2002). The ambient air quality guidelines also recognise the importance of $PM_{2.5}$ in relation to health effects and included a monitoring value of 25 $\mu g/m^3$ as a 24-hour average (MfE 2002) with a guideline of 10 $\mu g/m^3$ as an annual average proposed in more recent reviews (MfE 2020). Further works is recommended in the NAPINZ 3.0 review to better characterise, assess and manage $PM_{2.5}$ (Kuschel et al. 2022a, 2022b).

These guidelines were considered by the Air Quality Report (PDP 2022) in the assessment of impacts from the proposed Plant.

In addition to the recommendations provided for the assessment of $PM_{2.5}$ in New Zealand, the WHO (WHO 2021) has recently revised air quality guidelines for $PM_{2.5}$, which include interim guidelines or incremental steps to be considered to progressively reduce population exposures to $PM_{2.5}$. These guidelines are based on the most current information on health effects and robust exposure-response relationships and are summarised in **Table 4.1**.

Table 4.1: $PM_{2.5}$ air guidelines

Level/target	WHO guideline (with New Zealand guideline noted) (µg/m³)	
	24-hour average	Annual average
Interim target 1	75	35
Interim target 2	50	25
Interim target 3	37.5	15
Interim target 4	25 NZ monitoring value (MfE 2002)	10 Proposed NZ guideline (MfE 2020)
Air Quality Guideline (AQG) level (WHO goal)	15	5

In relation to the assessment of health impacts, assessment of $PM_{2.5}$ impacts is or most relevance. The air criteria relate to total exposures to $PM_{2.5}$, that is background or existing levels as well as the additional impact from the proposed Plant.

In relation to $PM_{2.5}$, the modelling completed in the Air Quality Report (PDP 2022) indicated the following:

- Maximum 24-hour average concentration of $PM_{2.5}$ anywhere off-site = 4.7 µg/m³ (from the project + background) which is well below the NZ guideline of 25 µg/m³ as well as the most stringent WHO AQG (goal) of 15 µg/m³
- Maximum annual average concentration of $PM_{2.5}$ anywhere off-site = 2.3 µg/m³ (from the project + background) which is well below the guideline of 10 µg/m³ as well as the most stringent WHO AQG (goal) of 5 µg/m³
- The contribution from the proposed Plant (1.0 µg/m³ for a 24-hour average and 0.03 µg/m³ for an annual average) is very low and would be considered negligible.

In addition to the analysis presented above, it is possible to also estimate the incremental individual risk associated with the change in $PM_{2.5}$ from the facility. As noted above such a calculation is challenging as it utilises population exposure-response relationships which are not directly applicable to small populations or individual risks. However, to further demonstrate that the incremental impact of $PM_{2.5}$ emissions from the Plant would not be of concern to health an indicative calculation is included. 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 health indicator also captures a wide range of other health effects associated with $PM_{2.5}$. The calculation has considered the baseline mortality rate for New Zealand (all ages and all causes – refer to **Table 2.3**), along with the exposure-response relationship relevant to assessing all-cause mortality. Further details and calculations are presented in **Appendix A**. These calculations assume that someone is present at the location of maximum increase in $PM_{2.5}$ from the proposed Plant for 24 hours a day, every day of the year.

For an annual average increase of $PM_{2.5}$ of 0.03 µg/m³, the maximum incremental increase in $PM_{2.5}$ from the proposed Project anywhere in the off-site community, this results in an incremental risk of 1×10^{-6}. This risk level is considered to be negligible, consistent with guidance use to develop drinking water guidelines in New Zealand (Ministry of Health 2018) as well as international guidance (enHealth 2012a; NEPC 2011; OEHHA 2015; USEPA 1989).

On the basis of the above, changes in $PM_{2.5}$ derived from the proposed Plant are considered to have a negligible impact on the health of the off-site community.

Assessment of community exposures to metals and organics bound to particulates is presented in **Sections 4.5.6** and **Section 4.6**.

4.5.3 Sulfur dioxide

Sulfur oxides are formed during combustion when chemicals present in fuels (such as coal, gas, petrol etc) 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. Sulfur oxides are also released from volcanos. Wildfires and other types of fires are also sources to the atmosphere of these chemicals (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).

New Zealand currently provides a National Environmental Standards for Air Quality (NESAQ) for sulfur dioxide of 570 µg/m^3 (with no exceedances) and 350 µg/m^3 (with 9 exceedances in 12 months) as a 1-hour average (MfE 2004b) and ambient air quality guidelines of 350 µg/m^3 for a 1-hour average and 120 µg/m^3 for a 24-hour average (MfE 2002). These standards and guidelines are based on the protection of health for all members of the population including sensitive populations like asthmatics, children and the elderly, noting that short-term health effects assessed on the basis of a 1-hour and 24-hour average are the most important for sulfur dioxide.

The standards and guidelines for sulfur dioxide relate to total exposures, which is the impact from the proposed Plant and background.

In relation to sulfur dioxide, the modelling completed by PDP (2022) indicated the following:

- Maximum 1-hour average concentration of sulfur dioxide anywhere off-site = 42.7 µg/m^3 (from the project + background) which is well below the guideline of 350 µg/m^3
- Maximum 24-hour average concentration of sulfur dioxide anywhere off-site = 14.1 µg/m^3 (from the project + background) which is well below the guideline of 120 µg/m^3

On the basis of the above, there are no risk issues of concern for community health in relation to sulfur dioxide emissions from the proposed Plant.

4.5.4 Nitrogen dioxide

Health effects

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 residual waste is used as fuel. Motor vehicles, along with industrial, commercial and residential (e.g., gas heating 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 has been found to cause inflammation of the respiratory system and increase susceptibility to respiratory infection, resulting in increased mortality, hospital admissions and emergency room visits (WHO 2021). Asthmatics, the elderly and people with existing cardiovascular and respiratory disease are particularly susceptible to the effects of elevated nitrogen dioxide (Morgan, Broom & Jalaludin 2013; NEPC 2010; USEPA 2016b; WHO 2021). Other adverse health effects identified, where causality is suggested include: cardiovascular effects and diabetes, reproductive and developmental effects (specifically birth outcomes), mortality and cancer (USEPA 2016b). The health effects associated with exposure to nitrogen dioxide depend on the duration of exposure as well as the concentration.

As with the assessment of particulate matter (size, particularly PM2.5), the identification of health effects and assessment of health impacts of nitrogen dioxide has relied on data from large epidemiological studies. These studies are reviewed in detail by key agencies (NEPC 2021; USEPA 2016b; WHO 2021) with the most recent New Zealand review also focusing on the assessment of exposure and health effects of nitrogen dioxide (Kuschel et al. 2022a, 2022b). These reviews have identified exposure-response relationships that can be used to quantify health impacts of short- and long-term exposures by large populations to nitrogen dioxide. The New Zealand review established and used exposure-response relationships from a New Zealand population or cohort study. The primary health effects identified in the New Zealand study, for which exposure-response relationships were established for long-term exposures to nitrogen dioxide are (Kuschel et al. 2022b):

- Premature mortality for adults aged 30 years and older
- Hospital admissions for cardiovascular disease (including stroke) and respiratory disease for all ages
- Childhood asthma for children aged 0 to 18 years, as asthma and wheeze hospitalisations and prevalence of childhood asthma.

These exposure-response relationships are used by key organisations to establish ambient air quality goals/criteria and assess the impact (health costs/savings) of various air policy decisions. Such assessments also consider background levels.

The exposure-response relationships adopted to assess these effects are based on large epidemiological studies that relate to changes in population health with changes in nitrogen dioxide. As a result, they are used to determine and review air quality goals and standards that relate to population average or regional exposures from all sources. They do not relate to exposures in localised ‘hot-spot’ areas such as locations near industry or busy roads. Hence it is generally not appropriate to apply the exposure-response relationships identified to assess localised impacts from a specific project, as the populations impacted are very small (or individuals) and not consistent with the populations evaluated in the epidemiological studies and the existing health status is not known.

Assessment of exposure

The principal approach to the assessment of potential impacts on community health to changes in nitrogen dioxide concentrations as a result of the Project is to assess compliance with ambient air quality guidelines established for nitrogen dioxide. These guidelines are those established following

evaluation of the available evidence and exposure-response relationships for adverse health effects in New Zealand populations.

New Zealand currently provides a National Environmental Standards for Air Quality (NESAQ) for nitrogen dioxide of 200 µg/m^3 (with 9 exceedances in 12 months) as a 1-hour average (MfE 2004b) and ambient air quality guidelines of 200 µg/m^3 for a 1-hour average and 100 µg/m^3 for a 24-hour average (MfE 2002). These standards and guidelines are based on the protection of health for all members of the population including sensitive populations like asthmatics, children and the elderly. These standards, however, are noted to be dated.

Hence, in addition to the recommendations provided for the assessment of nitrogen dioxide in New Zealand, the WHO (WHO 2021) has recently revised air quality guidelines for nitrogen dioxide, which include interim guidelines or incremental steps to be considered to progressively reduce population exposures. These guidelines are based on the most current information on health effects and robust exposure-response relationships, with the 24-hour average guidelines summarised in **Table 4.2**. The WHO also recommends annual average air guidelines for nitrogen dioxide, which is not an averaging time currently considered in New Zealand policy.

Table 4.1: Nitrogen dioxide air guidelines

Level/target	WHO guideline (with New Zealand guideline noted) (µg/m^3)	
	24-hour average	Annual average
Interim target 1	120 100 = NZ guideline (MfE 2002)	40
Interim target 2	50	30
Interim target 3	--	20
Air Quality Guideline (AQG) level (WHO goal)	25	10

The standards and guidelines for nitrogen dioxide relate to total exposures, which is the impact from the proposed Plant and background. The modelling undertaken by PDP (2022) has assumed that 100% of NOx comprises nitrogen dioxide, which is a highly conservative assumption, which will overestimate the concentration of nitrogen dioxide in ambient air.

In relation to nitrogen dioxide, the modelling completed by PDP (2022) indicated the following:

- Maximum 1-hour average concentration of nitrogen dioxide anywhere off-site = 69 µg/m^3 (from the project + background) which is well below the guideline of 200 µg/m^3
- Maximum 24-hour average concentration of nitrogen dioxide anywhere off-site = 42.6 µg/m^3 (from the project + background) which is well below the guideline of 100 µg/m^3
- The maximum 24-hour average concentration of nitrogen dioxide anywhere off-site is also below the WHO (WHO 2021) interim target 1 and interim target 2 guidelines, but exceeds the AQG goal of 25 µg/m^3. The WHO AQG is very stringent and essentially equal to existing background air quality in the area, with much of New Zealand not able to meet the goal[4]. The approach adopted for quantifying nitrogen dioxide in air from the Project, assuming

[4] https://environment.govt.nz/publications/our-air-2021/

100% of NOx comprises nitrogen dioxide will have overestimated impacts and hence it is not possible to determine if the WHO goal would be met.

- The maximum annual average concentration of nitrogen dioxide is 3.7 µg/m^3 (PDP 2022), which is well below all the WHO (WHO 2021) interim targets and the most stringent AQG of 10 µg/m^3.

On the basis of the above, there are no risk issues of concern for community health in relation to nitrogen dioxide emissions from the proposed Plant.

4.5.5 Carbon monoxide

Motor vehicles are the dominant source of carbon monoxide in air. Carbon monoxide is produced during combustion when there is a limited supply of oxygen. This facility is designed to provide oxygen greater than the stoichiometric mixture (i.e., it achieves excess oxygen in the combustion gasses) and as such the production of carbon monoxide will be very low.

The sorts of effects that can be expected due to exposure to carbon monoxide are those linked with carboxyhaemoglobin (COHb) in blood – i.e., where carbon monoxide 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).

New Zealand currently provides a NESAQ for carbon monoxide of 10 mg/m^3 (or 10,000 µg/m^3) (with 1 exceedance in 12 months) as an 8-hour average (MfE 2004b) and ambient air quality guidelines of 30 mg/m^3 (or 30,000 µg/m^3) for a 1-hour average and 10 mg/m^3 (or 10,000 µg/m^3) for an 8-hour average (MfE 2002). These standards and guidelines are based on the protection of health for all members of the population including sensitive populations like asthmatics, children and the elderly.

The standards and guidelines for carbon monoxide relate to total exposures, which is the impact from the proposed Plant and background.

In relation to carbon monoxide, the modelling completed by PDP (2022) indicated the following:

- Maximum 1-hour average concentration of carbon monoxide anywhere off-site = 5,038 µg/m^3 (from the project + background) which is well below the guideline of 30,000 µg/m^3
- Maximum 8-hour average concentration of carbon monoxide anywhere off-site = 3,013 µg/m^3 (from the project + background) which is well below the guideline of 10,000 µg/m^3

On the basis of the above, there are no risk issues of concern for community health in relation to carbon monoxide emissions from the proposed Plant.

4.5.6 All other pollutants

For all other pollutants, inhalation exposures have considered both short-term/acute exposures as well as chronic exposures. This assessment relates to all other gases as well as metals and organics that are bound to the particulates. The approach adopted for the modelling of emissions to air for metals and VOCs is highly conservative (refer to **Table 3.1**) and will overestimate actual emissions.

Acute exposures

The assessment of acute exposures is based on comparing the maximum predicted 1-hour average exposure concentration with health-based criteria relevant to acute or short-term exposure, also based on a 1-hour average exposure time. The ratio of the maximum predicted concentration to the acute guideline is termed a hazard index (HI) and is calculated as follows:

$$HI = \frac{\text{Exposure concentration (maximum modelled 1-hour average)}}{\text{(Acute TRV)}}$$

$$\text{Total HI} = \sum \text{HI (individual pollutants)}$$

Where:
Exposure concentration = maximum modelled concentration as 1-hour average of pollutant in air for the proposed Plant as a gas/vapour or present bound to $PM_{2.5}$ (mg/m^3)
Acute TRV = health based toxicity reference value (TRV) or guideline that is protective of acute/short-duration exposures for all members of the community including sensitive individuals, as per **Appendix B** (mg/m^3)

Risks associated with acute exposures are considered to be acceptable where the individual and total HI's are less than or equal to 1.

The acute health-based guidelines, or acute toxicity reference values (TRVs), adopted in this assessment have been selected on the basis of the approach detailed in **Appendix B**. It is noted that for the assessment of exposure to dioxin-like chemicals as well as some metals, there are no relevant health-based guidelines available as the key issues associated with these chemicals relate to chronic exposures or long-term body burdens. The acute assessment has, therefore, focused on the chemicals where acute health effects are relevant.

Table 4.3 presents a summary of the relevant health-based guideline, the predicted maximum 1-hour average concentrations for the maximum impacted location and the maximum impacted sensitive receptor, and the calculated HI for each chemical. Exposures at all other locations, including the other sensitive receptors will be lower than presented in **Table 4.3**.

Table 4.3: Review of acute inhalation exposures and risks – proposed Plant

		Air Concentration - Maximum 1 hour average (mg/m^3)		Calculated HI	
Key chemical	**Acute inhalation TRV - health (mg/m^3)**	**Maximum anywhere**	**Maximum - residential, rural, school receptors**	**Maximum anywhere**	**Maximum - residential, rural, school receptors**
Hydrogen chloride (HCl)	0.66 [T]	0.0045	0.0029	0.0069	0.0044
Hydrogen fluoride (HF)	0.06 [T]	0.00075	0.00050	0.013	0.0083
Ammonia	0.59 [T]	0.0076	0.0048	0.013	0.0081
Benzene	0.58 [T]	0.0076	0.0048	0.013	0.0083
Antimony	0.001 [A]	0.000036	0.000022	0.036	0.022
Arsenic	0.0099 [T]	0.000007	0.0000044	0.0007	0.00044
Cadmium	0.018 [T]	0.0000014	0.00000088	0.00008	0.000049
Chromium (Cr VI assumed)	0.0013 [T]	0.0000036	0.0000022	0.0028	0.0017
Copper	0.1 [O]	0.0000036	0.0000022	0.00004	0.000022
Manganese	0.0091 [T]	0.0000036	0.0000022	0.0004	0.00024
Mercury	0.0006 [O]	0.000014	0.0000087	0.024	0.015
Nickel	0.0011 [T]	0.00000036	0.00000022	0.00033	0.00020
Vanadium	0.03 [O]	0.000036	0.000022	0.0012	0.00073
Toluene	15 [T]	0.0076	0.0048	0.00050	0.00032
Xylenes	7.4 [T]	0.0076	0.0048	0.0010	0.00065
Trimethylbenzenes	15 [T]	0.0076	0.0048	0.00050	0.00032
			Total HI	**0.11**	**0.057**
			Acceptable HI	**≤ 1**	**≤ 1**

References for health-based acute air guidelines (1-hour average) (also refer to Appendix B):

T = Guideline available from the Texas Commission on Environmental Quality (TCEQ), https://www.tceq.texas.gov/toxicology/dsd/final.html

O = 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

A = 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

Review of **Table 4.3** indicates all maximum predicted concentrations of chemicals in from the operation of the proposed Plant are well below the health-based criteria protective of acute effects.

On the basis of the above assessment, there are no acute risk issues of concern in relation to inhalation exposures to emissions from the proposed Plant.

Chronic exposures

For the assessment of chronic exposures, all the chemicals have been evaluated using a threshold approach, that enables the predicted annual average concentration to be compared with a health based, or acceptable, guideline. The health based guidelines or toxicity reference values (TRVs) adopted may be based on the protection of genotoxic carcinogenic effects (where a non-threshold TRV is used to establish a guideline based on a 1 in 100,000 incremental lifetime risk) or a threshold that is protective of all health effects consistent with MfE guidance (MfE 2011c).

For the assessment of chronic effects where the TRV relates to threshold effects, the assessment has also considered potential intakes of these chemical substances from other sources, i.e., background intakes.

The individual HI is calculated as follows:

$$\text{Exposure concentration} = C_{air} \times \frac{ET \times EF \times ED}{AT}$$

$$HI = \frac{\text{Exposure concentration}}{TRV \times (100\% - \text{Background})}$$

$$\text{Total HI} = \sum \text{HI (individual pollutants)}$$

Where:

C_{air} = concentration in air based on an annual average, modelled air concentration from the proposed Plant for gas/vapour and others present on dust as $PM_{2.5}$ (mg/m^3)

ET = exposure time where inhalation exposures occur (hours per day) (refer to **Appendix C**)

EF = exposure frequency, days per year exposed (days per year) (refer to **Appendix C**)

ED = number of years of exposure (years) (refer to **Appendix C**)

AT = averaging time (in hours) which is relevant to the TRV approach adopted. For threshold TRVs, the AT = ED x 365 days x 24 hours/day; for non-threshold TRVs the AT = 75 years x 365 days x 4 hours/day (refer to **Appendix C**)

TRV = health-based toxicity reference value based on a threshold or non-threshold (where an incremental risk of 1 in 100,000 is adopted) that is protective of all health effects for all members of the community (mg/m^3) (refer to **Appendix B**)

Background = proportion of the threshold TRV that may be derived from other ambient or background sources/exposures such as water, soil or consumer products (%) (refer to **Appendix B**). Where a non-threshold TRV is adopted background intakes are not included in the calculation.

Risks associated with chronic exposures are considered to be negligible (or acceptable) where the individual and total HI's are less than or equal to 1.

When quantifying inhalation exposures, the following has been assumed:

- The maximum concentration reported occurs on the site boundary which is an industrial area, where inhalation exposures are assumed to occur at this maximum impacted location for 8 hours per day, 240 days of the year for 30 years.
- The maximum concentrations at sensitive receptors, namely residential, rural residential, and schools, are all assumed to be a residential location where a resident spends 24 hours per day at home, 350 days per year for 30 years.

Appendix B presents the relevant health-based TRVs adopted in these calculations, along with assumptions adopted for the assessment of background intakes and the quantification of inhalation exposures for the calculation of the HI and incremental lifetime risk. **Appendix D** presents the calculations undertaken to evaluate inhalation exposures for the proposed Plant.

Table 4.4 presents the calculated individual HI and the incremental lifetime cancer risk relevant to the assessment of chronic inhalation exposures for workers and residents based on maximum concentrations relevant to these receptors.

Table 4.4: Calculated chronic inhalation risks

Pollutant	Chronic inhalation TRV - health (mg/m^3)	Calculated HI	
		Maximum on site boundary (worker exposures)	Maximum at all off-site receptor locations (residential exposures)
Hydrogen chloride (HCl)	0.026 T	0.00028	0.0010
Hydrogen fluoride (HF)	0.029 T	0.000042	0.00015
Ammonia	0.32 T	0.000038	0.00014
Benzene*	0.0036 NZ	0.00090	0.0053
Antimony	0.0003 A	0.00020	0.00074
Arsenic*	0.0000055 NZ	0.00059	0.0034
Beryllium	0.00002 W	0.0030	0.011
Cadmium	0.000005 W	0.00061	0.0022
Chromium (Cr VI assumed)*	0.0000011 NZ	0.0015	0.0086
Copper	0.49 R	0.000000019	0.000000067
Cobalt	0.0001 W	0.00061	0.0022
Lead	0.0002 NZ	0.000061	0.00022
Manganese	0.00015 W	0.000051	0.00018
Mercury	0.0002 W	0.00012	0.00043
Nickel	0.00002 E	0.000038	0.00014
Thallium	0.0007 R	0.0000039	0.000014
Vanadium	0.0001 A	0.00061	0.0022
Selenium	0.02 O	0.0000041	0.000015
Tin	0.7 R	0.00000017	0.0000063
Dioxins and furans (WHO-TEQ)	3.5E-09 R	0.000030	0.00011
Toluene	5 U	0.0000024	0.0000090
Xylenes	0.2 A	0.000061	0.00022
Trimethylbenzenes	0.06 U	0.00023	0.00083
Total Risk/HI		**0.0090**	**0.039**
Negligible risk		**≤ 1**	

Notes

Refer to **Appendix B** for additional information on TRVs adopted, and assumptions adopted for the calculation of the HI

* = Chemical evaluated on the basis of a non-threshold TRV where calculation of the HI is based on the use of this value as per MfE (MfE 2011c)

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 2009d)

NZ = New Zealand ambient air guideline (MfE 2002) for annual average exposures, adopted where this is more conservative than the most current health based guideline relevant to the assessment of chronic health effects; or NZ toxicological value used in the derivation of soil guideline values (MfE 2011a). For benzene, arsenic and chromium the TRVs adopted are based on protection of carcinogenic effects based on a non-threshold (linear) approach and adoption of 1 in 100,000 risk level. For these chemicals and calculations, it is not relevant to include background intakes as the calculation relates to an incremental lifetime risk

T = TRV available from TCEQ, relevant to chronic inhalation exposures (and HI=1) (TCEQ 2012, 2013c, 2014a, 2015d, 2015b)

A = TRV available from ATSDR, relevant to chronic intakes (ATSDR 2007c, 2012a, 2012c, 2012b, 2019a)

E = TRV available from the UK Environment Agency (UK EA 2009d) for nickel, noting this value is protective of all adverse effects including carcinogenicity

O = TRV from OEHHA, as chronic reference exposure level (REL) (OEHHA)

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

W = TRV available from the WHO, relevant to chronic inhalation exposures (WHO 1999, 2000c, 2006a, 2017), noting inhalation value adopted for mercury is for elemental mercury (WHO 2003) which is lower than the NZ ambient air quality guideline (MfE 2002)

Review of **Table 4.4** indicates the calculated HI for all chemicals individually and as a sum are less than 1 which is representative of negligible/acceptable exposures on the site boundary (for workers) and in the off-site community. It is noted that the margin of safety (ratio between the guideline and exposure concentration) ranges from 90 to >1,000,000 for individual chemicals and is 25 for the total HI. This margin of safety is more than sufficient to account for any future changes in air guidelines.

On this basis, there are no chronic risk issues of concern in relation to inhalation exposures in the community surrounding the proposed Plant.

In relation to lead, the chronic TRV adopted in the calculations above is the NZ ambient air guideline of 0.0002 mg/m^3 (MfE 2002). While this air guideline has been adopted in the calculations above assuming it relates to an annual average, the air guideline applies to a 3-month average concentration in air.

The maximum concentration of lead in air (anywhere off-site), as a 3-month average, is 0.00000037 mg/m^3, significantly lower than the air guideline of 0.0002 mg/m^3. Hence, in addition to the calculations presented in **Table 4.4**, there are no risk issues of concern in relation to lead exposures where the NZ ambient air guidelines are further considered.

4.6 Multiple pathway exposures

4.6.1 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 particulates (as TSP) depositing to the environment where a range of other exposures may then occur. These include:

- Deposition to water (refer to **Section 4.7**):
  - rainwater tanks, where water may be used as potable/drinking water where ingestion and dermal contact is relevant
- Deposition to soil:
  - incidental ingestion and dermal contact with soil (and dust indoors that is derived from outdoor soil or deposited particulates)
  - ingestion of homegrown fruit and vegetables where chemicals may deposit onto the plants and onto the soil where the plants are grown resulting in such chemicals being taken up into these plants
  - ingestion of eggs where chemicals may deposit onto pasture and be present in soil (which is the same soil present where backyard chickens are kept and ingested during feeding), and the chemicals are taken up into the eggs
  - ingestion of other produce at a rural residential property, that may include milk (from dairy cows), beef from cattle and lamb.

It is also noted that some rural properties also grow crops and produce such as meat and milk where there is the potential for metals and organics to be taken up into these products, and these products may be sold into the market. The uptake of these metals and organics into produce that may be sold, has been further evaluated in **Section 4.8**.

The above exposures are chronic or long-term exposures.

4.6.2 Assessment approach

In relation to these exposures, such exposures will only occur on rural residential or residential properties where people live and where rainwater tanks are used, and/or homegrown produce is grown and consumed. This assessment has assessed multi-pathway exposures for the maximum predicted impacts in all sensitive receptor locations, specifically rural and residential areas. Exposures in all other residential areas will be lower than the maximum presented in this assessment.

The calculation of risks posed by multiple pathway exposures only relates to pollutants that are bound to the particulates. The air modelling has provided deposition rates for metals and organics on dust relevant to each pollutant, relevant to emissions to air from the proposed Plant. These have been used in this assessment.

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**.

It is noted that assessment of potential risks related to exposure to water in rainwater tanks is presented separately in **Section 4.7**. In addition, assessment of risks relevant to the growing of crops or uptake into meat, milk or eggs are presented separately in **Section 4.8**.

4.6.3 Calculated risks

Table 4.3 presents the calculated risks associated with the multiple pathway exposures relevant to both adults and children. These risks have been calculated on the basis of the maximum predicted dry deposition rate for all of the sensitive receptors in the surrounding community. Calculated risks for all other receptors would be lower than presented in this table.

The table presents the total non-threshold risk and HI for each exposure pathway, calculated as the sum over all the pollutants evaluated. The table also includes the calculated HI associated with inhalation exposures (as per **Table 4.4**), as these exposures are additive to the other exposure pathways for residential properties.

Depending on the use of a property, the mix or combination of exposures that may occur are likely to vary. For this assessment, a number of scenarios have been considered where a range of different exposures or combination of exposures may occur. The sum of risks associated with these multiple exposures is presented in **Table 4.5**.

Table 4.5: Summary of risks for multiple pathway exposures

Exposure pathway	Calculated HI	
	Young children	Adults
Individual exposure pathways		
Inhalation (I)	0.039	0.039
Soil ingestion (SI)	0.010	0.0015
Soil dermal contact (SD)	0.000078	0.000015
Ingestion of homegrown fruit and vegetables (F&V)	0.0070	0.0058
Ingestion of homegrown eggs (E)	0.0014	0.00063
Ingestion of home produced milk (M)	0.065	0.014
Ingestion of home produced beef (B)	0.023	0.0080
Ingestion of home produced lamb (L)	0.013	0.0056
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.049	0.041
I + SI + SD + F&V	0.056	0.047
I + SI + SD + E	0.051	0.041
I + SI + SD + F&V + E	0.058	0.047
I + SI + SD + M	0.11	0.055
I + SI + SD + B	0.072	0.049
I + SI + SD + L	0.062	0.046
I + SI + SD + F&V + E + M	0.12	0.062
I + SI + SD + F&V + E + B	0.080	0.055
I + SI + SD + F&V + E + L	0.070	0.053
I + SI + SD + F&V + E + M + B + L	0.16	0.075
Negligible risk	**≤1**	**≤1**

Refer to **Appendix D** for detailed risk calculations for each exposure pathway

Review of **Table 4.5** indicates that all calculated risks associated with each individual exposure pathway as well as a combination of multiple exposure pathways, remain below the target risk levels considered representative of negligible risks.

On the basis of the assessment undertaken, there are no chronic risk issues of concern in relation to multiple pathway exposures that may be relevant to the off-site community.

4.7 Residential drinking water exposures

Where there may be deposition of persistent chemicals in areas where rainwater tanks are used for collecting and storing water used for drinking/potable water, there is the potential for these chemicals to accumulate and impact on water quality. Particles can deposit onto a roof and then be washed off the roof into a rainwater tank when it rains. For many of the residential and rural properties surrounding the proposed Plant drinking water is sourced from rainwater tanks and/or groundwater. Hence it is important to evaluate potential impacts of the proposed Plant on the quality of water in rainwater tanks.

The deposition of chemicals to a roof, and accumulation in rainwater has been estimated for the maximum impacted receptor location assuming the average rainfall for Oamaru, a roof that is consistent with typical house size in Waimate and no use of a first flush device. Using this approach, concentrations of chemicals in the water as suspended sediment and as dissolved chemicals have been calculated assuming 100% of the dust that deposits on the roof washed into the tank. Rainwater tanks are designed such that suspended sediment deposits or settles and is not consumed. For the purpose of this assessment, dissolved phase concentrations are assumed to be representative of concentrations that would be consumed on a daily basis.

Predicted concentrations in rainwater tanks have then been compared with drinking water guidelines, which are protective of all exposures relevant to potable water use including ingestion, dermal contact, bathing and irrigation of produce that may be consumed. These guidelines are also protective of the health of pets who may consume water from rainwater tanks.

Table 4.6 presents the maximum predicted concentrations in rainwater tanks with comparison against current drinking water guidelines, applicable to drinking water quality in all areas of New Zealand. The tables also present a calculated HI, which is the ratio of the exposure concentration to the drinking water guideline. For the assessment of exposure, it is only appropriate to consider the dissolved phase concentration as this is representative of concentrations present in the tank that may be accessed and used on a daily basis. The total (dissolved + particulate) concentration is only presented for comparison and as a worst-case concentration (which may reflect concentrations in a drought where water levels are low) but is not considered realistic in relation to long-term drinking water exposures.

Appendix C presents detail on the modelling undertaken and assumptions adopted, and **Appendix D** presents the calculated water concentrations.

Table 4.6: Summary and review of exposures to chemicals in drinking water

Persistent and bioaccumulative chemical	**Calculated maximum concentration in rainwater tanks (mg/L)**		**Drinking water standard/ guideline (mg/L)**	**HI** (ratio of dissolved concentration to drinking water guideline)
	Dissolved – relevant to exposure	**Total (particulate and dissolved) –** highly conservative (assumes sediment is stirred up in tank)		
Antimony	1.5E-06	3.4E-05	0.02 [N]	7.3E-05
Arsenic	4.5E-07	7.0E-06	0.01 [N]	4.5E-05
Beryllium	8.3E-08	3.3E-05	0.012 [W]	6.9E-06
Cadmium	3.5E-08	1.3E-06	0.004 [N]	8.7E-06
Chromium (Cr VI assumed)	3.6E-12	3.3E-06	0.05 [N]	7.3E-11
Copper	1.9E-07	3.5E-06	2 [N]	9.4E-08
Cobalt	1.5E-06	3.4E-05	0.006 [U]	2.4E-04
Lead	1.5E-08	6.6E-06	0.01 [N]	1.5E-06
Manganese	1.0E-07	3.4E-06	0.4 [N]	2.5E-07
Mercury	4.9E-07	1.3E-05	0.007 [N]	7.0E-05
Nickel	1.0E-08	3.4E-07	0.08 [N]	1.3E-07
Thallium	3.7E-08	1.3E-06	0.0002 [U]	1.8E-04
Vanadium	6.5E-08	3.3E-05	0.086 [U]	7.6E-07
Selenium	1.3E-05	4.6E-05	0.04 [N]	3.3E-04
Tin	2.6E-07	3.3E-05	12 [U]	2.2E-08
Dioxins and furans (WHO-TEQ)	1.2E-19	3.9E-11	0.000000012 [U]	1.0E-11
			Total HI	**0.001**
			Acceptable/negligible HI	**≤1**

Refer to **Appendix C and D** for the methodology, assumptions and calculation of water concentrations

N = *Water Services (Drinking Water Standards for New Zealand) Regulations 2022* (New Zealand 2022) as Maximum Acceptable Values

W = WHO Guidelines for Drinking Water Quality (WHO 2017)

U = Residential tap water guideline from USEPA Regional Screening Levels (USEPA 2022b), adopted where no guidelines available from New Zealand or the WHO

Review of **Table 4.6** indicates that the predicted water concentrations in rainwater tanks are all well below drinking water standards/guidelines. This is particularly relevant to the maximum dissolved phase concentration which is representative of concentrations that would be accessed and used from the rainwater tank. The total concentration only reflects a peak, where sediment is disturbed (unlikely to occur unless sediment is disturbed during cleaning or drought conditions where low levels of water may be present in the tank).

Where water samples are collected from a rainwater tank (or other water source) for the purpose of analysis, an analytical limit of reporting (LOR)[5] applies to the results, as follows:

- For metals, the LOR is commonly around 0.001 mg/L, with trace analysis reporting a LOR in the range of 0.0001 to 0.0005 mg/L with cadmium reported to a LOR of 0.00005 mg/L. All concentrations of metals calculated in rainwater tanks are below these analytical LORs, and hence these chemicals would not be detected where water sampling occurred.
- For dioxins and furans (including dioxin-like PCBs) the LOR can be variable between laboratories, however, it is typically around 4 to 5 pg/L (or 4 to 5 x 10^{-9} mg/L) as an upper limit (i.e. using the LOR for all individual congeners) WHO_{05}TEQ. The concentration of dioxins and furans, and dioxin-like PCBs calculated in rainwater tanks are well below the analytical LOR, and hence these chemicals would not be detected where water sampling occurred.

Based on the above, emissions to air from the proposed Plant would not have a measurable change in water quality in rainwater tanks at the most affected relevant location, hence impacts on drinking water quality are considered to be negligible. Intakes and exposures (from using water from rainwater tanks) have not been calculated in detail and they have not been added to intakes from soil and produce, as the contribution to total exposure is considered negligible.

Note that the total HI calculated for the rainwater tank concentrations conservatively applies to both adults and young children. Where this is added to the total HI calculated for all other multi-pathway exposures presented in **Table 4.5** the following is noted:

- Young children (based on maximum HI calculated for all exposure pathways), HI = 0.16 (**Table 4.5**) + 0.001 (**Table 4.6**) = 0.16
- Adults (based on maximum HI calculated for all exposure pathways occurring all the time for 70 years), HI = 0.075 (**Table 4.5**) + 0.001 (**Table 4.6**) = 0.076

These conservative maximum combined HI's are unchanged and remain representative of acceptable/negligible risks.

[5] Limit of reporting (LOR) for chemical parameters is the minimum concentration of a substance in a sample that can be reliably detected by a laboratory. This will depend on the type of sample analysed and the methodology used by the laboratory. Where reported as not detected, this means that the concentration in the sample analysed is lower than the LOR that can be achieved by the laboratory.

Based on the assessment undertaken, there are no risk issues of concern in relation to potential exposures of persistent and bioaccumulative chemicals that may be present in rainwater tanks surrounding the site.

Groundwater sources of water

It is noted that drinking water in the local area may also sourced from groundwater. The potential for emissions to air to deposit onto the ground and change water quality in groundwater extracted and used for drinking water is considered to be negligible. This is due to the following:

- The organic pollutants considered, namely dioxins-like chemicals, have very low water solubility. Hence when deposited to the ground these chemicals will not wash out from the soil and move into groundwater.
- In relation to metals, the concentration that may be present in soil as a result of deposition is very low (refer to **Section 4.9.2**) and would not be discernible from background soil. Hence the impacts would not result in any change to regional groundwater which would reflect background/existing geology of the area.

4.8 Assessment of risk issues relevant to produce

4.8.1 Crops

Chemicals may be present attached to particles that are emitted from the proposed facility. Once emitted to the atmosphere the particles may fall out of the air and deposit onto the surface of plants, buildings, roads and soil. If attached chemicals are persistent and the particles mix into the soil or are present on the leaves of a plant, they may be taken up by plants into the parts people may consume – i.e., accumulation. This pathway can be assessed using relevant modelling calculations as shown in **Appendix D**. Chemicals that are relevant for this pathway are the metals and persistent organics like dioxin-like compounds.

Where rural properties in the surrounding areas are used for the growing of crops such as grain that may be sold to the market for use in a range of products.

Hence it is not appropriate to assess exposures associated with grain production and consumption for the rural properties where the grain is grown. However, it is relevant to evaluate if the grain produced would remain in compliance with the maximum limits (MLs) in the Australia New Zealand Food Standards Code (relevant to the presence of selected metals in produce)[6].

To enable evaluation of this pathway to be undertaken, the deposition rate for each relevant chemical at the maximum impacted rural residential receptor location has been considered. Using that rate, the maximum predicted concentration in soil has been estimated and that soil concentration has then been used to estimate concentrations in grain or similar crops (such as canola) using relevant uptake factors (refer to **Appendix C** for methodology and assumptions and **Appendix D** for calculations).

[6] https://gazette.govt.nz/notice/id/2015-gs1944

It is noted that the predicted concentrations are considered worst case as these relate to the deposition of pollutants from the proposed Plant to ground continuously for 35 years.

The predicted concentration in grain crops have then been directly compared with the MLs or other relevant information as described below.

It is noted that there are MLs for only 3 of the relevant pollutants (arsenic, cadmium and lead).

To determine if deposition from the project has the potential to be of significance to crops produced in the area for other relevant pollutants, the maximum predicted concentrations in crops have been compared with the range of concentrations reported in New Zealand in cereal products (breads, cereals and oats).

All of these comparisons are included in **Table 4.7**.

Table 4.7: Review of concentrations in grain (and similar) crops

Pollutant	Estimated maximum concentration in grain (mg/kg)	Food Standards Code – ML for cereals, grains, wheat etc or equivalent (mg/kg)	Range of mean concentrations reported in cereal products evaluated in dietary surveys in Australia (mg/kg)
Antimony	0.0006	--	0.003 (F5)
Arsenic	0.0001	1	--
Beryllium	0.00004	--	NA
Cadmium	0.0003	0.1	--
Chromium (Cr VI assumed)	0.000008	--	0.015 to 0.13 (F3)
Copper	0.0005	--	0.67 to 4.1 (F3)
Cobalt	0.00007	--	0.0054 to 0.071 (F3)
Lead	0.00002	0.2	--
Manganese	0.0006	--	6.7 to 35 (F3)
Mercury	0.0006	--	<0.002 (N1)
Nickel	0.000002	--	0.212 to 0.41 (F4)
Thallium	0.000003	--	NA
Vanadium	0.00003	--	NA
Selenium	0.00004	--	0.014 – 0.166 (N1)
Tin	0.0001	--	<0.05 (N1)
Dioxins and furans (WHO-TEQ)	1×10^{-11}	--	1×10^{-8} to 4×10^{-8} (F1)

N = New Zealand Total Diet Studies
https://www.mpi.govt.nz/food-business/food-monitoring-surveillance/new-zealand-total-diet-study/
F = Food Standards Australian Total Diet Surveys (adopted where no data available from NZ)
https://www.foodstandards.gov.au/science/surveillance/Pages/australiantotaldiets1914.aspx
N1 = 2016 New Zealand Total Diet Survey
F1 = 26th Diet Survey (2020)
F2= 25th Diet Survey (2019)
F3 = 23rd Diet Survey (2011)
F4 = 22nd Diet Survey (2008)
F5 = 20th Diet Survey (2003)

Review of **Table 4.7** indicates that:

- the maximum predicted concentrations for arsenic, cadmium and lead are well below the MLs relevant to these pollutants
- maximum predicted concentrations for other pollutants are well below the range of mean concentrations reported in existing/typical food products.

The LOR for the analysis of food products varies depending on the chemical and the type of food product being evaluated. For most foods analysed, the LOR is as follows:

- Metals have a LOR typically around 0.005 to 0.01 mg/kg. Concentrations of most metals predicted in crops are lower than these LOR and hence would not be measurable. In relation to zinc, the predicted concentrations in grain crops are just above the analytical LOR, and while these levels may be measurable, they are only a very small proportion of the concentrations typically reported in grain products grown in New Zealand and would not result in any discernible change in the quality of produce derived from the local area.
- Dioxins and furans have a LOR typically around 1 to 2 x 10^{-7} mg/kg. All concentrations of dioxin-like compounds predicted in crops are well below than these LOR and hence would not be measurable.

On this basis, emissions from the proposed Plant are considered to be negligible in terms of their contribution to existing background levels in cereal products consumed in the market.

The predicted concentrations in cereal crops, as a result of emissions from the proposed Plant, would not be detectable or discernible in any analysis.

In addition, deposition of particles from emissions from the facility would not result in any measurable change in soil quality in the area (refer to **Section 4.9.2**). Hence the proposed Plant would not change existing conditions or result in impacts on crops grown on farms with organic farming status.

4.8.2 Other produce

The assessment of potential multi-pathway exposures presented in **Section 4.6** included an assessment of risks to human health where metals and persistent organic compounds may accumulate into eggs, milk and meat. For some of these products, maximum limits (MLs) are detailed in the Australia New Zealand Food Standards Code. Where these produce are sold to the market, compliance with these maximum limits is a legal requirement. It is relevant to ensure that the maximum calculated concentrations estimated using deposition of particles from the emissions of the facility are below the MLs relevant to these products. There are limited MLs available for some metals, as follows (refer to **Appendices D and E** for calculations):

- For cadmium, the ML for meat is 0.05 mg/kg. The maximum concentrations of cadmium from the proposed Plant calculated in beef (0.00000081 mg/kg) and lamb (0.0000010 mg/kg) are well below the ML. The predicted concentrations are also noted to be well below the LOR for analysis of meat.
- For lead, the ML for meat is 0.1 mg/kg. The maximum concentrations of lead from the proposed Plant calculated in beef (0.00000060 mg/kg) and lamb (0.00000078 mg/kg) are

well below the ML. The predicted concentrations are also noted to be well below the LOR for analysis of meat.

There are no MLs for dioxins and furans (i.e., dioxin-like compounds) in the Australia New Zealand Food Standards Code. In the absence of local MLs, general Code provisions apply including that food must be safe and suitable. This requirement has been demonstrated in the risk calculations presented in **Section 4.6**.

Another source of food guidelines for dioxin-like compounds is the European Union (EU). The EU[7] has established regulatory limits for the sum of all dioxin-like compounds (including dioxin-like PCBs) on a TEQ basis for meat, eggs and milk. The EU values are listed below along with the predicted concentrations from this assessment as provided in **Appendix D**).

- Beef and lamb meat
  - limit of 4 pg/g fat (or 0.000004 mg/kg (i.e., 4 x 10^{-6} mg/kg fat))
  - conversion to wet weight assuming meat contains 10-20% fat gives a limit of 0.4-0.8 pg/g wet weight (ww) or 4 x 10^{-7} to 8 x 10^{-7} mg/kg ww
  - the concentrations based on wet weight are relevant for use in this assessment
  - these concentrations are significantly higher than the maximum predicted concentrations relevant to the proposed Plant (as per calculations shown in **Appendix D**) in beef (6.6 x 10^{-9} mg/kg ww) and lamb (8.5 x 10^{-9} mg/kg ww).
- Eggs
  - limit of 5 pg/g fat (or 0.000005 mg/kg (i.e., 5 x 10^{-6} mg/kg fat))
  - conversion to wet weight assuming egg contains 11% fat gives a limit of 0.55 pg/g wet weight or 5.5 x 10^{-7} mg/kg ww
  - the concentration based on wet weight is relevant for use in this assessment
  - this is significantly higher than the maximum predicted concentration relevant to the proposed Plant (as per calculations shown in **Appendix D**) in eggs (1.9 x 10^{-9} mg/kg ww).
- Milk
  - limit of 5.5 pg/g fat (or 0.0000055 mg/kg (i.e., 5.5 x 10^{-6} mg/kg fat))
  - conversion to wet weight assuming milk contains around 4% fat gives a limit of 0.22 pg/g wet weight or 2.2 x 10^{-7} mg/kg ww
  - this is significantly higher than the maximum predicted concentration relevant to the proposed Plant (as per calculations shown in **Appendix D**) in milk (4.6 x 10^{-10}).
- These comparisons indicate that the worst-case concentrations in produce predicted for this facility using conservative assumptions are at least 100 times (approximately) lower than the limits put in place for food in the EU.

The predicted concentrations of dioxins/furans + dioxin-like PCBs in various produce noted above are also well below the analytical LOR and also the range of background concentrations reported in food products (FSANZ 2020).

[7] Most recent assessment by EFSA of dioxin-like compounds in food (2018) – https://efsa.onlinelibrary.wiley.com/doi/epdf/10.2903/j.efsa.2018.5333

Based on the above, emissions from the proposed Plant would not result in any measurable impact on produce grown in the local area. Concentrations of metals derived from these emissions are predicted to result in produce levels below the regulatory MLs, and concentrations of dioxin-like compounds are predicted to result in produce levels below EU regulatory levels. Hence the proposed Plant would not impact on the quality of produce sold from farms in the area.

The predicted concentrations in produce, as a result of emissions from the proposed Plant, would also not be detectable in any analysis. In addition, emissions from the facility would not have any measurable change in soil quality in the area (refer to **Section 4.9.2**). Therefore, the proposed Plant would not change existing conditions or result in impacts on crops grown on farms with organic farming status.

4.9 Uncertainties and additional considerations

4.9.1 General

The quantification of human health risks has relied on the modelling of emissions to air and prediction of worst-case or maximum impacts in the off-site community. Hazards associated with potential exposure to the chemicals evaluated is based on current toxicological information relevant to the chemicals evaluated. Quantification of risk has utilised a number of assumptions that are expected to overestimate actual exposure to chemicals derived from the proposed Plant.

Some key assumptions adopted on how individual chemicals have been assessed are detailed in **Section 4.2**. These assumptions would result in overestimation of risk relevant to these individual chemicals.

In addition, the following should be noted:

- The calculated soil concentrations assume that deposition occurs continuously throughout a 35-year period, which is an overly conservative assumption. Further all impacts derived from the facility accumulate in surface soil and indoor dust for the whole 35 years. No cleaning of indoor dust or use of any other topsoil/mulch/soil conditioner or fertiliser is assumed to occur which would reduce concentrations in surface soil or indoor dust.
- Concentrations predicted in produce is based on the maximum accumulated concentration in soil over the whole 35-year period.
- Concentrations calculated for above ground plants that may be consumed (and also consumed by livestock) assumes that all dust settled on these parts of the plant are ingested, and that the produce is not washed prior to consumption.
- Rural residents live and work on their property as a child and adult 350 days per year for 30 years.

Further review of some aspects of the HHRA has been undertaken as detailed below.

4.9.2 Soil concentrations

The focus of the assessment of deposition and multi-pathway exposures has been for the closest sensitive receptor.

It is also relevant to understand the contribution of the proposed Plant to existing soil concentrations in the area.

To address these considerations, the maximum predicted surface soil concentrations (refer to **Appendices C and D**), have been compared against soil guidelines protective of both low-density rural residential land use (which is protective of ingestion, dermal contact, dust inhalation and ingestion of homegrown produce) and recreational use (protective of ingestion, dermal contact and dust inhalation). Background levels of metals and dioxin-like compounds in soil are also presented. Where possible, background levels in soil have been sourced from data relevant to the area, however, where no data from the area are available, data from New Zealand have been used. This comparison is presented in **Table 4.8**.

Table 4.8: Review of maximum predicted surface soil concentrations against background and low-density residential and recreational soil guidelines

Persistent chemical	Maximum calculated concentration from proposed Plant (mg/kg)#		Background levels in soil** (mg/kg)	Health based guideline (mg/kg)	
	Surface soil	Agricultural soil		Low density rural residential	Recreational
Antimony	0.28	0.019	NA	31 U	31 U
Arsenic	0.057	0.0038	3.4 – 11.5	17 NZ	80 NZ
Beryllium	0.28	0.019	NA	160 U	160 U
Cadmium	0.011	0.00075	0.06 – 0.18	0.8 NZ	400 NZ
Chromium (Cr VI assumed)	0.028	0.0019	11 – 20.8	290 NZ	2,700 NZ
Copper	0.028	0.0019	7.1 – 18.8	NL NZ	NL NZ
Cobalt	0.28	0.019	NA	23 U	23 U
Lead	0.057	0.0038	18.7 – 37.4	160 NZ	880 NZ
Manganese	0.028	0.0019	5 – 9	1,800 U	1,800 U
Mercury (inorganic)	0.11	0.0074	0.04 – 0.1	200 NZ	1,800 NZ
Nickel	0.0028	0.00019	8.7 – 19	1,500 U	1,500 U
Thallium	0.011	0.00075	NA	0.78 U	0.78 U
Vanadium	0.28	0.0019	NA	390 U	390 U
Selenium	0.28	0.0019	NA	390 U	390 U
Tin	0.28	0.0019	NA	47,000 U	47,000 U
Dioxins and furans (WHO-TEQ)	2.5×10^{-7}	1.7×10^{-8}	NA	1.2×10^{-4} NZ	6×10^{-4} NZ

\# Calculated concentration in soil assumes maximum deposition rate from all off-site receptors occurs continuously and cumulatively for 35 years and accumulates in surface soil, or soil mixed in the top 15 cm (agricultural soil)

** Based on data for regional soil (yellow brown stony, yellow grey earth, yellow brown earth and recent – relevant to study area) from "Background concentrations of selected trace elements in Canterbury soils, Addendum 1: Additional samples and Timaru Specific background levels" (Environment Canterbury 2007)

NZ = New Zealand soil contaminant standards (MfE 2011c)

U = USEPA Regional Screening Levels (RSLs) for residential soil, adopted for the assessment of residential and recreational exposures (USEPA 2022b)

Review of **Table 4.8** indicates the following:

- the maximum predicted concentrations in soil derived from the proposed Plant are well below soil guidelines protective of residential and recreational exposures
- the contribution of emissions derived from the proposed Plant are similar to or below soil concentrations considered consistent with background in the study area

- the analytical LOR for metals in soil is typically around 1 to 5 mg/kg and the maximum concentrations predicted in soil from the proposed Plant are generally below these LOR.

Based on the above, the worst-case cumulative emissions derived from the proposed Plant would not be detectable or discernible in soil and would not make any measurable change to existing soil concentrations in areas surrounding the facility. Hence impacts to soil from the proposed Plant are considered to be negligible.

4.9.3 PFAS

Another group of chemicals that has been of concern to communities is 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 constitute 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
- cleaning products
- surfactants used in chrome plating or electronics manufacture (HEPA 2020).

It is possible that low levels may be present in the proposed residual waste fuel due to the low levels of PFAS that have been used in various consumer products and packaging (especially fast-food packaging) that would be present in domestic MSW.

Concerns regarding this group of chemicals were raised internationally around the year 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 New Zealand 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 waste fuel would be expected to continue to decrease and to already be much lower than the levels currently discussed in the scientific literature relating to waste materials.

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. In addition, the EU BREF emission limits do not include consideration of PFAS emissions.

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

- Combustion chamber – PFAS are usually present in materials that could be in the residual waste as mixtures. Within those mixtures some chemicals are readily degradable at temperatures easily reached in the chamber. Some of the chemicals do require higher temperatures to breakdown. It is noted that much of the chamber will have temperatures in excess of 850^{o}C and these temperatures along with sufficient oxygen will allow for effective combustion (at least 90%) of these chemicals.
- Acid gas treatment (injection of hydrated 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.
- PTFE filters – chemicals attached to particles (including activated carbon particles) are captured within the filters – this will include PFAS bound to these particulates.
- Wet scrubber – PFAS are highly soluble and hence this technology will be effective in removing any remaining PFAS.

Risks due to the presence of the expected very low to negligible levels of these chemicals within the fuel to be combusted at this facility are expected to be low to negligible.

4.9.4 Community studies

The assessment presented in this report provides a quantitative evaluation of risks to community health following appropriate and robust assessment guidelines. These guidelines are consistent with the approaches to assessing health risks for such facilities from international jurisdictions.

The scientific literature also provides a number of other studies, specifically epidemiological studies that have focused on emissions to air from EfW facilities and potential health effects within communities surrounding the particular facility. Many of the published studies relate to older facilities that do not comply with more recent EU directives (IED emission limits and BREF limits). Only studies that relate to more recent facilities complying with these emissions standards and guidance are relevant for any comparison with the proposed Plant. Many of the EfW facilities evaluated in the epidemiological studies are facilities combusting domestic waste (along with other non-putrescible waste). This is consistent with the proposed Plant.

Reports or studies that have reviewed published information and studies on EfW facilities designed to meet EU IED or equivalent emissions limits, have not identified evidence of adverse impacts on community health. Most studies also acknowledge that the number of available studies is limited in

relation to these newer facilities, however, in the available studies relevant to modern facilities that meet these standards, no adverse health effects have been identified.

These studies include:

- Literature review undertaken for EPA Victoria (EPA Victoria 2018) and by other Australian researchers (Cole-Hunter et al. 2020; Morgan et al. 2019; Tait et al. 2020) as well as the review completed by the NSW Chief Scientist (NSW Chief Scientist & Engineer 2020)
- Review of research into health effects of EfW facilities focusing on facilities operating in the UK (Broomfield 2012; Marner, Richardson & Laxen 2020), with a series of more recent epidemiological studies (Freni-Sterrantino et al. 2019; Ghosh et al. 2019; Parkes et al. 2019) specifically addressing foetal growth, stillbirth, congenital abnormalities, infant mortality and sex ratio and other birth outcomes finding no evidence of adverse effects in the community. These studies also indicate that the results should be generalisable to other facilities operating to similar standards.

It should be noted that studies related to older facilities[8], where emissions did not or do not meet the EU IED or equivalent emission limits, have shown measurable impacts and links with adverse health effects (Tait et al. 2020). Further, the former operation of these older waste incinerators has resulted in the accumulation of dioxin-like compounds in soil and produce (specifically eggs and vegetables) in areas surrounding the facilities (for example a facility operating in France from 1974 to 2002 and a facility operating from 1958 to 1982 in Lausanne Switzerland[9] (Petrlik et al. 2022; Pirard et al. 2004)). Investigations conducted in the 1990s, in relation to these older facilities, identified the need to reduce emissions from waste incineration facilities and ongoing technology reviews. These changes have resulted in significant measured improvements in emissions. For example, emissions of dioxin-like compounds from waste incineration in France and Japan have reduced more than 99% from the 1990's to around 2010 (Coudon et al. 2019; Li et al. 2019; Nzihou et al. 2012). This means impacts on air quality from these types of facilities are significantly smaller now than they were previously.

Studies related to these older facilities are not relevant to the assessment of potential health impacts from new energy from waste facilities that comply with the more stringent emissions limits from the EU IED and BREF limits.

There are few studies available that measure concentrations of pollutants in soil and produce in rural areas surrounding operational modern energy from waste facilities (that meet IED emission limits or equivalent).

[8] Older facilities are those that were constructed and operated prior to the introduction and enforcement of emission limits in the European Union Waste Incineration Directive (EU-WID) (2000/76/EC), which was incorporated into and further revised in the EU Industrial Emissions Directive (IED) (2010/75/EU) where emission limits for some pollutants were reduced. IED 2010/75/EU is incorporated into the Best Available Technologies (BAT) Reference Document for Waste Incineration (BREF)(2019) where the emission limits for some pollutants were further reduced, and emission limits were recommended for ammonia and total volatile organic compounds (TVOCs) which were not included in the IED.

[9] https://www.euronews.com/green/2021/10/17/lausanne-discovers-soil-has-been-polluted-with-dangerous-chemicals-for-more-than-years ; https://www.vd.ch/themes/environnement/sols/pollution-des-sols-aux-dioxines/

The study by van Dijk et al (van Dijk, van Doorn & van Alfen 2015) involved testing for levels of cadmium, mercury and PAHs in crops (spinach and kale) and dioxin-like compounds (i.e. dioxins, furans and dioxin-like PCBs) in milk from dairy farms and fluoride in pasture grass around three waste incinerators (combusting municipal solid waste) operating in the Netherlands between 2004 and 2013. The facilities were operating using best available technology applicable at the time of operation. The study showed that emissions from these facilities did not affect the quality of crops and milk in the surrounding areas. Concentrations reported were similar to background levels and did not exceed maximum allowable standards applicable to food products in the Netherlands.

Monitoring of dioxins and furans has also been undertaken in areas surrounding other EfW facilities in Europe (CEWEP 2022) where the following is noted in relation to soil and produce:

- Dioxins and furans were measured in vegetation surrounding an Austrian EfW facility – no significant difference was seen between areas close to the facility and distant
- Dioxins and furans were measured in blood of people living near and distant from an EfW facility in Turin over a period of 3 years – there was no increase in dioxin levels in blood [i.e. no evidence of bioaccumulation] and no difference in levels between those close to the facility and those distant from the facility (background)
- Dioxins and furans were measured in cow milk in areas surrounding a Dutch EfW facility between 2009 and 2020 – levels in milk near the plant were no different from background
- Dioxins and furans were measured in soil samples collected in the area surrounding an EfW facility in Mallorca (Spain) from 1997 to 2020 –the levels reported were variable (with no clear trend of accumulation), but all samples were well below the maximum limit value relevant for soil

Sampling of dioxins and furans was also undertaken in the area of Harlingen (Netherlands). Levels in grass and eggs were reported to be higher within 2 km of a waste incinerator (noting the area also includes a range of other industries) and some concentrations in eggs exceeded the EU guidelines (Arkenbout 2014; Arkenbout & Esbensen 2017). The facility is an industrial waste incinerator (not a municipal waste incinerator) that was commissioned in 2011 and has a low emissions limit for dioxins and furans. However, the facility has had a number of reported operational issues that resulted in elevated dioxin and furan emissions at times (including levels that exceeded their emissions limit). These elevated emissions are reflected in the egg data reported for 2014/2015 (Arkenbout & Esbensen 2017), however it should be noted that more than one source of dioxin-like compounds was identified for this area (Arkenbout 2014). Another study also reported elevated levels of dioxins and furans in chicken eggs in other areas in Europe. These findings were found to be related to keeping chickens in industrial areas or areas affected by backyard burning of waste (Hoogenboom et al. 2016).

Consistent with the approach outlined by the NSW Chief Scientist (NSW Chief Scientist & Engineer 2020), the potential for accumulation of persistent and bioaccumulative chemicals into produce, including chicken eggs, meat, milk and other produce has been evaluated for this facility using robust risk assessment methods. This is presented in this assessment for the proposed Plant and is relevant to the proposed operation of the EfW facility.

Section 5. Conclusions

The assessment has evaluated potential risks to community health in relation to emissions to air from proposed Project KeaThe assessment of human health risks has relied on air modelling undertaken and presented in the Air Quality Emissions Assessment (PDP 2022).

The area surrounding the proposed Project comprises a number of rural residential and residential properties. The area also includes the smaller town of Glenavy and larger townships of Waimate, Oamaru and Duntroon.

A detailed assessment of risks to human health has considered acute and chronic inhalation exposures as well as multi-pathway exposures associated with the deposition of metals and persistent organic pollutants (specifically dioxins-like compounds) to the ground and the potential for direct contact with soil and dust (indoors) and uptake of these chemicals into homegrown produce (fruit and vegetables, eggs, milk, and meat [beef and lamb]) and consumption of this produce.

The assessment has also considered whether the deposition of metals and dioxins/furans would have the potential to adversely affect water quality in rainwater tanks, recreational water in the nearby lakes and the quality of produce such as grain and vineyard crops, as well as meat, milk and eggs grown in the area.

This assessment has considered impacts in the off-site community for the worst-case emissions scenario relevant to the operation of the proposed Plant. In addition, a number of conservative assumptions were adopted in relation to the emissions to air of individual metals and volatile organic compounds.

Based on the available data and conservative assumptions adopted in this assessment, the following has been concluded:

- Inhalation exposures
  - All risks to human health are considered negligible for the duration of the proposed Plant. More specifically the following has been concluded:
    - no acute inhalation risk issues of concern
    - no chronic risk issues of concern
    - exposure to particulates derived from the proposed Plant within the community are considered negligible.
- Multi-pathway exposures
  - All chronic risks to human health are considered negligible for the duration of the proposed Plant. More specifically the following has been concluded:
    - all calculated risks for individual exposure pathways are negligible and essentially representative of zero risk
    - all calculated risks for combined multiple pathway exposures are negligible and essentially representative of zero risk.
  - Emissions from the proposed Plant would have a negligible impact on water quality in rainwater tanks used for drinking water
  - Emissions from the proposed Plant would have a negligible impact on crops and produce grown in the area.

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Appendix A Calculation of risks from $PM_{2.5}$

Calculation of risk: $PM_{2.5}$

A quantitative assessment of risk for these endpoints 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 and is relevant to the range of health effects (or endpoints) identified as relevant (to the nature of the emissions assessed) and robust (as identified in the main document). An exposure-response relationship can have a threshold, 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. Non-threshold exposure-response relationships have been identified for the health endpoints considered in this assessment.

Risk calculations relevant to exposures to $PM_{2.5}$ by the community 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).

The assessment of potential risks associated with exposure to particulate matter 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[10]. The calculation of a relative risk based on the change in relative risk exposure concentration from baseline/existing (ie based on incremental impacts from the project) can be calculated on the basis of the following equation (Ostro 2004):

Equation 1 $\quad RR = \exp[\beta(X-X0)]$

Where:
X-X0 = the change in particulate matter concentration to which the population is exposed ($\mu g/m^3$)
β = regression/slope coefficient, or the slope of the exposure-response function which can also be expressed as the per cent change in response per 1 $\mu g/m^3$ 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:

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10 Some reviews have identified that a log-linear exposure-response function may be more relevant for some of the health endpoints considered in this assessment. Review of outcomes where a log-linear exposure-response function has been adopted (Ostro 2004) for $PM_{2.5}$ identified that the log-linear relationship calculated slightly higher relative risks compared with the linear relationship within the range 10–30 micrograms per cubic metre, (relevant for evaluating potential impacts associated with air quality goals or guidelines) but lower relative risks below and above this range. For this assessment (where impacts from a particular project are being evaluated) the impacts assessed relate to concentrations of $PM_{2.5}$ that are well below 10 micrograms per cubic metre and hence use of the linear relationship is expected to provide a more conservative estimate of relative risk.

Equation 2

$$\beta = \frac{\ln(RR)}{10}$$

Where:
RR = relative risk for the relevant health endpoint as published (µg/m³)
10 = increase in particulate matter concentration associated with the RR (where the RR is associated with a 10 µg/m³ increase in concentration).

The assessment of health impacts for a particular population associated with exposure to particulate matter has been undertaken utilising the methodology presented by the WHO (Ostro 2004)[11] where the exposure-response relationships identified have been directly considered on the basis of the approach outlined below.

An additional risk can be calculated as:

Equation 3 **Risk=β x ΔX x B**

Where:
β = slope coefficient relevant to the per cent change in response to a 1 µg/m³ change in exposure
ΔX = change (increment) in exposure concentration in µg/m³ 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 the 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 Project, with the maximum change from all community receptors (where regional air quality is of most relevance) adopted in this calculation. For this assessment the change in $PM_{2.5}$ relates to the change in annual average air concentrations and the value considered in this assessment is 0.03 µg/m³ as a maximum change.
- Baseline incidence of the key health endpoints that are relevant to the population exposed – the assessment undertaken has considered the baseline mortality rate of 373.6 per 100,000 as an age standardised rate for New Zealand in the calculation. This rate relates to all ages, with the calculation presented using an exposure-response relationship for adults aged 30

11 For regional guidance, such as that provided for Europe by the WHO WHO 2006b, Health risks or particulate matter from long-range transboundary air pollution 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).

years and over. Baseline data for ages 30 years and over is not readily available for the population. Use of the baseline health data for all ages is considered adequately representative of the population (noting that HAPINZ calculations identified the same number of deaths for all populations and people aged 30 years and over for the Warmate district).

- Exposure-response relationships expressed as a percentage change in health endpoint per microgram per cubic metre change in particulate matter exposure, where a relative risk (RR) is determined (refer to Equation 1). The concentration response function used in this report is that recommended in the most recent HAPINZ review (Kuschel et al. 2022b). This provides a RR of 1.105 per 10 µg/m^3 change in $PM_{2.5}$, and is noted to relate to adults aged 30 years and over.

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 calculations undertaken the calculated incremental individual risk (rounded to 1 significant figure):

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

$$= 0.03 \times 0.003736 \times 0.00998$$

$$= 1 \times 10^{-6}$$

Appendix B Toxicity of key chemicals evaluated

B1 Approach to the identification of toxicity reference values

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 the chemicals evaluated in this assessment there are no health based acute ambient air quality guidelines established in New Zealand.

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 approach adopted by the WHO (WHO 2000d, 2000c, 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. ATSDR acute air guidelines (noting these are applicable to exposures ranging from 1-hour to 14 days)
  3. 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.

For this assessment, all air concentrations have been provided by PDP (2022), for the correct averaging periods that need to be evaluated. Hence there has been no need to convert any of the data received to different averaging periods.

Based on the above Table B1 presents the acute TRVs that have been adopted in this assessment along with a summary of the hazards/adverse health effects relevant to acute exposures. It is noted that no acute TRVs are available for a number of chemicals, specifically beryllium, cobalt[12], lead, thallium, selenium, tin and dioxins-like chemicals as these chemicals are either not acute toxicants

[12] In relation to cobalt, an acute TRV is available from TCEQ, however this value is based on data from occupational exposures to cobalt metal (hard metal) particulates from the metal industry which is not relevant to the presence of inorganic cobalt compounds bound to particulates following combustion (which would not include metal particles). There are no suitable acute TRVs for cobalt that can be used in this assessment.

or no suitable acute inhalation TRVs are available. All these chemicals have been assessed in relation to chronic exposures.

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 2015d)	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 2015d).
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).
Benzene	0.58 (TCEQ 2015c)	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 2015c) as well as CSN excitation and depression and neurological effects. The acute air guideline is based on decreased lymphocytes in an animal study (TCEQ 2015c). The study used by TCEQ is the same adopted by ATSDR (ATSDR 2007b) in establishing their acute air guideline(noting the ATSDR review is more dated).
Toluene	15 (TCEQ 2013b)	The available studies indicate that acute inhalation exposures to toluene may result in CNS or neurotoxicity effects (including changes in reaction time, coordination, visual performance, dizziness, intoxication) as well as

Chemicals evaluated	Acute air guideline (1-hour average) (mg/m^3)	Key health effects
		irritation to the eyes and respiratory tract (ATSDR 2000). The CNS is the most sensitive effect, with the acute air guideline based on the most recent review, with the most sensitive effects being CNS and irritation effects in human volunteers (TCEQ 2013b).
Xylenes	7.4 (TCEQ 2013b)	The available studies indicate that acute inhalation exposures to xylenes may result in CNS/neurological and respiratory effects. Irritation of the eyes, nose and throat may also occur. Neurological effects include fatigue, headache, dizziness, and a feeling of intoxication. The acute air guideline is based on the most recent review with the sensitive effects being mild respiratory and subjective neurological effects in human volunteers (TCEQ 2013b).
Inorganics and organics bound to particulates (where acute effects are relevant)		
Antimony	0.001 (ATSDR 2019a)	The most sensitive effects related to acute inhalation exposures to antimony have been identified as respiratory effects, with effects on the cardiovascular system less sensitive (ATSDR 2019a). Acute air guideline adopted is based on respiratory effects (epithelium effects at base of epiglottis) in an animal study (ATSDR 2019a).
Arsenic	0.0099 (TCEQ 2012)	Short-term exposures to arsenic have been reported to result in severe irritation to both the upper and lower parts of the respiratory system, followed by symptoms of cough, dyspnea, and chest pain. In addition, exposure to arsenic dust has been reported to cause laryngitis, bronchitis, and/or rhinitis. Further, exposure to arsenic via inhalation and/or ingestion can also cause gastrointestinal symptoms such as garlic-like breath, vomiting, and diarrhea. The available occupational and epidemiological studies have not identified developmental or reproductive effects; however these effects have been observed in animals but only at doses exceeding maternal toxicity. Acute air guideline adopted is based on the most sensitive effect, namely maternal effects in a reproductive study in animals.
Cadmium	0.018 (TCEQ 2016)	The toxicity of cadmium in air is dependent on the form of cadmium. The toxicity is higher with the more soluble cadmium compounds. Acute inhalation exposure to cadmium 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.
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 2017b)	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,

Chemicals evaluated	Acute air guideline (1-hour average) (mg/m^3)	Key health effects
		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 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 2017a)	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 2009d). 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 for health-based acute air guidelines (1-hour average):
TCEQ = Acute reference exposure value (Acute ReV) available from the Texas Commission on Environmental Quality as referenced, also available from: https://www.tceq.texas.gov/toxicology/dsd/final.html
OEHHA = Guideline available from California Office of Environmental Health Hazard Assessment (OEHHA) https://oehha.ca.gov/air/general-info/oehha-acute-8-hour-and-chronic-reference-exposure-level-rel-summary
ATSDR = Guideline available from the Agency for Toxic Substances and Disease Registry (ATSDR), as an acute air guideline (relevant to exposures from 1 hour to 14 days) https://www.atsdr.cdc.gov/mrls/index.html

B3 Values adopted for the assessment of chronic exposures

Chronic toxicity reference values (TRVs) associated with inhalation, ingestion and dermal exposures have been adopted from credible peer-reviewed sources as by enHealth (enHealth 2012a).

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.

For the gaseous chemicals 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 chemicals 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.

Table B3 presents the TRVs adopted for the assessment of chronic health effects associated with exposure to the other chemicals considered in this assessment. Where available, the TRVs adopted are based on values adopted in New Zealand guidance.

Section B4 presents more detailed toxicity reviews of the metals and dioxin-like chemicals that provide additional information that supports the values adopted in this assessment.

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 2015d). 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).
Ammonia	The key hazards associated with ammonia, relate to acute effects, where the respiratory and CNS systems are the most sensitive health effects (refer to **Table B1**). In relation to chronic exposures, there are few studies addressing long-term inhalation exposures to low concentrations. The key health effects identified in occupational studies relate to respiratory irritation, including cough, chest tightness, stuffy/runny nose, sneezing, phlegm, wheezing, dyspnea, chronic bronchitis, and asthma. Studies have shown acclimation of effects (ATSDR 2004a; TCEQ 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).

Pollutant evaluated	Summary of chronic health effects
Benzene	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 2012c; USEPA 2005b, 2005a). Benzene is carcinogenic via oral and inhalation routes of exposure (ATSDR 2007b; IARC 2012c; UK EA 2009c; WHO 1993) indicates that the overall results of available studies show that it is appropriate to consider benzene (and/or its metabolites) as genotoxic (though the genotoxic profile is considered unusual (Baars et al. 2001)). The assessment of benzene toxicity needs to consider carcinogenic effects where a non-threshold dose-response approach is appropriate. New Zealand (MfE 2002) has established a chronic air guideline value (based on an annual average) for benzene of 10 µg/m^3, with a lower value of 3.6 µg/m^3 to be achieved by 2010. For this assessment the lower value of 3.6 µg/m^3 of 0.0036 mg/m^3 has been adopted. This guideline value is based on precautionary guideline values from Europe and the UK and are protective of carcinogenic effects. This air guideline is consistent with air guidelines derived on the basis of a non-threshold approach to assess carcinogenicity from TCEQ and the WHO (TCEQ 2015c; WHO 2000d). As the guideline is based on a non-threshold approach background intakes do not need to be accounted for.
Toluene	The key health effects associated with inhalation exposures to toluene relate to the CNS (headaches, dizziness, and impaired neurobehavioral performance), kidneys, liver, respiratory system and reproduction. Toluene is classified by IARC and the US EPA as not classifiable as to human carcinogenicity due to inadequate evidence of carcinogenicity. Review of available data (Baars et al. 2001; UK EA 2009e; USEPA 2005c; USEPA IRIS; WHO 2011b) suggest that toluene has not been demonstrated to be genotoxic. On the basis of the available information, it is considered appropriate that a threshold dose-response approach be adopted for toluene. Toluene exposures have been assessed on the basis of the chronic inhalation air guideline from the USEPA (USEPA 2005c) which is similar to the more recent evaluation from TCEQ (TCEQ 2013b). Background or ambient concentrations of toluene are negligible compared with the chronic air guideline adopted.
Xylenes	Health effects of mixed xylenes, o-xylene, m-xylene and p-xylene, appear to be similar, although the individual isomers are not necessarily equal in potency with respect to a particular effect. Studies indicate that the central nervous system (CNS) is a major and sensitive target of xylene toxicity via inhalation and oral routes. The primary target organs following chronic oral and inhalation exposures are likely to be the CNS and development. Some studies indicate enlargement of the liver and kidneys following oral exposure to mixed xylene. Other target organs identified following inhalation exposure include the respiratory system, altered haematological parameters, nose and throat irritation. Xylene is classified by IARC and the US EPA as not classifiable as to human carcinogenicity due to inadequate evidence of carcinogenicity. The available studies suggest that xylenes are not considered genotoxic (UK EA 2009h; USEPA IRIS; WHO 1997). On the basis of the available information, it is considered appropriate that a threshold dose-response approach be adopted for xylenes. Xylenes exposures have been assessed on the basis of the chronic inhalation guideline from ATSDR (ATSDR 2007c), which is consistent with evaluations provided by the UK, TCEQ and USEPA (TCEQ 2013a; UK EA 2009h; USEPA 2003). Background or ambient concentrations of xylenes are negligible compared with the chronic air guideline adopted.
Trimethylbenzene	Trimethylbenzenes comprise 1,2,4-trimethylbenzene and 1,3,5-trimethylbenzene, both of which are aromatic petroleum hydrocarbons, which have health effects consistent with other aromatic hydrocarbons noted above (toluene and xylenes). The key adverse health effects associated with inhalation exposures are CNS and respiratory effects. Other effects include liver effects and anaemia. Neither the US EPA nor IARC has classified trimethylbenzene with respect to carcinogenicity and the limited data available on genotoxicity shows negative results. On the basis of the

Pollutant evaluated	Summary of chronic health effects
	available information, it is considered appropriate that a threshold dose-response approach be adopted for trimethylbenzenes. Trimethylbenzene exposures have been assessed on the basis of the inhalation guideline established by the USEPA (USEPA 2016a), which applies to the sum of trimethylbenzenes, and is the most recent detailed review available which also provides a more conservative approach than TCEQ (TCEQ 2015a). The available data suggests background or ambient concentrations contribute around 10% of the adopted toxicity reference values.
Inorganics and organics bound to particulates (refer to Section B4 for additional detail)	
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). The chronic air guideline adopted in this assessment is based on respiratory effects (lung inflammation) in animals from ATSDR (ATSDR 2019a), noting no other chronic inhalation guidelines are available. 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 of antimony are assumed to be 20% for oral and dermal exposures and negligible for inhalation exposures.
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 2012b). The mechanism of action in relation to carcinogenicity is not clear and remains debated (IARC 2012b; 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. However due to uncertainties relating to the mechanism of action New Zealand has adopted a non-threshold approach to the assessment of all exposures to arsenic. On this basis the recommended TRV values from MfE (MfE 2002, 2011a), derived to be protective of the most sensitive effect, carcinogenicity using a non-threshold approach and 1 in 100,000 risk have been adopted in this assessment. Background intakes are not relevant to include where a non-threshold approach is adopted.
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 2001c) also classified beryllium as carcinogenic based on occupational inhalation studies. Further review of genotoxicity by IARC (IARC 2012a) 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. 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.

Pollutant evaluated	Summary of chronic health effects
	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 1998b; WHO 2001c). Background or ambient intakes are considered to be negligible.
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 2013). 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 2013). 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 2013). IARC (IARC 2012b) 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 1998a). Assessment of oral and dermal exposures is undertaken on the basis of a threshold (noting limited data to support carcinogenicity), where the current value from ASTDR (ATSDR 2012d) is most appropriate, and more conservative than the value identified in the older review from MfE (MfE 2011a) 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 assessment of carcinogenic effects. The ambient air guideline from MfE (MfE 2002), which is similar to the more recent review from TCEQ has been adopted. This guideline 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.

Pollutant evaluated	Summary of chronic health effects
	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 2006a) which is considered protective of all adverse health effects. Background or ambient intakes have also been considered.
Copper	Copper is an essential element and as such adverse effects may occur as a result of deficiency as well as excess intakes resulting from contamination. Liver and gastrointestinal effects are the most sensitive health effects from exposure to high levels of copper (ATSDR 2022; MfE 2011a), particularly in sensitive subpopulations. Copper is not considered to be carcinogenic. Exposure to copper has been evaluated on the basis of a toxicity reference value derived from a tolerable upper limit, with background intakes determined on the basis of information on dietary intakes (the key source of copper exposure).
Lead	The key health effects associated with exposure to lead are chronic. There is a large amount of information available about the health effects of lead, with information and data from epidemiological studies being the major lines of evidence. The health effects of lead are the same regardless of the route of exposure (ATSDR 2019b). Health effects associated with exposure to inorganic lead and compounds include, but are not limited to: neurological, renal, cardiovascular, haematological, immunological, reproductive, and developmental effects. Neurological effects of Pb are of greatest concern because effects are observed in infants and children and may result in life-long decrements in neurological function. The most sensitive targets for lead toxicity are the developing nervous system in children; and effects on the haematological and cardiovascular systems, and the kidney in adults. However, due to the multi-modes of action of lead in biological systems, lead could potentially affect any system or organs in the body. The effects of lead exposure have often been related to the blood lead content, which is generally considered to be the most accurate means of assessing exposure (MfE 2011a). Children and pregnant women are particularly sensitive to lead exposure, and low lead exposure studies have focused on a range of health outcomes including on neurological (such as cognitive and behavioural functioning), cardiovascular and reproductive and developmental health endpoints (Armstrong et al. 2014). The International Agency for Research on Cancer (IARC 2006) has classified inorganic lead as Group 2A: probably carcinogenic to humans. While it is appropriate to utilise a blood lead model to evaluate exposure to lead, toxicity reference values have been developed using blood lead models that are protective of adverse health effects with changes in IQ identified as the most sensitive effect by MfE (MfE 2011a). The threshold value adopted from MfE is consistent with intakes determined to be protective of IQ effects in children based on blood lead modelling and are considered appropriate. Inhalation exposures have been assessed on the basis of the air guideline from MfE (MfE 2002). 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. By the oral route, manganese is regarded as one of the least toxic elements, however there is some concern that the neurological effects observed from inhalation exposures also occur with oral exposures. Manganese is not considered to be carcinogenic. The chronic inhalation guideline is based on based on protection of neurological effects. The oral value is based on a tolerable upper intake for the element, with background intakes considered (principally from the diet).
Mercury (as inorganic and elemental)	This assessment has assumed that mercury in air comprises 100% elemental mercury vapour, which will result in a conservative assessment of inhalation exposures 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 including New Zealand MfE (MfE 2002).

Pollutant evaluated	Summary of chronic health effects
	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 a tolerable daily intake recommended by MfE, WHO and ATSDR, 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 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.
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.

Pollutant evaluated	Summary of chronic health effects
	The assessment of exposure, from all pathways, has been undertaken on the basis of a threshold toxicity value established by the Ministry of Health (MfE 2011a), which is more conservative than the value adopted by the NHMRC (NHMRC 2002) and WHO (FAO/WHO 2018; WHO 2019). Background intakes relevant to New Zealand have been considered.

Table B3: Summary of chronic TRVs adopted for chemicals

Chemical	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 chemical)			NA	0%
Hydrogen fluoride (HF)	0.029 T	NA (gaseous chemical)			NA	0%
Ammonia	0.32 T	NA (gaseous chemical)			NA	0%
Benzene	0.0036 NZ	NA (gaseous chemical)			NA	0%
Toluene	5 U	NA (gaseous chemical)			NA	0%
Xylenes	0.2 A	NA (gaseous chemical)			NA	0%
Trimethylbenzene	0.06 U	NA (gaseous chemical)			NA	10%
Antimony	0.0003 A	0.00086 W	15%	0	20%	0%
Arsenic	0.0000055 NZ	0.0000086 NZ	100%	0.005	0%	0%
Beryllium	0.00002 W	0.002 W	0.7%	0	0%	0%
Cadmium	0.000005 W	0.0008 W, NZ	100%	0	50%	20%
Chromium (Cr VI assumed)	0.0000011 NZ	0.003 NZ	100%	0	0%	0%
Copper	0.49 R	0.14 W, NZ	100%	0	33%	33%
Cobalt	0.0001 W	0.0014 D	100%	0	20%	0%
Lead***	0.0002 NZ	0.0019 NZ	100%	0	50%	0%
Manganese	0.00015 W	0.16 A	4%	0	50%	20%
Mercury (as inorganic and elemental)	0.0002 W	0.002 NZ	7%	0.001	5%	5%
Nickel	0.00002 E	0.012 W	100%	0.005	60%	20%
Thallium	0.0007 R	0.0002 D1	100%	0	80%	80%
Vanadium	0.0001 A	0.002 D	2.6%	0	0%	0%
Selenium	0.02 O	0.006 N1	100%	0	0%	0%
Tin	7 R	2 W	100%	0	0%	0%
Dioxin-like chemicals assumed to be WHO_{05} TEQs	3.5E-09 R	1E-09 NZ	100%	0.03	33%	33%

Notes

* GI factor and dermal absorption values adopted from RAIS (accessed in 2022) (RAIS)

** Background intakes relate to intakes from inhalation, drinking water and food products. The values adopted are based on information available for New Zealand, where available or international data. Gaseous chemical background intakes are not known and hence for this assessment they have been assumed to be negligible

*** Inhalation exposures to lead have been evaluated on the basis of the ambient air guideline of 0.0002 mg/m^3 for a 3-month average (MfE 2002), which has been assumed to also apply as an annual average (refer to main report for discussion)

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 2009d)

NZ = New Zealand ambient air guideline (MfE 2002) for annual average exposures, adopted where this is more conservative than the most current health based guideline relevant to the assessment of chronic health effects; or NZ toxicological value used in the derivation of soil guideline values (MfE 2011a). For benzene, arsenic and chromium the TRVs adopted are based on protection of carcinogenic effects based on a non-threshold (linear) approach and adoption of

1 in 100,000 risk level. For these chemicals and calculations, it is not relevant to include background intakes as the calculation relates to an incremental lifetime risk

T = TRV available from TCEQ, relevant to chronic inhalation exposures (and HI=1) (TCEQ 2012, 2013c, 2014a, 2015d, 2015b)

A = TRV available from ATSDR, relevant to chronic intakes (ATSDR 2007c, 2012a, 2012c, 2012b, 2019a)

D = TRV available from RIVM (Baars et al. 2001; van Vlaardingen, Posthumus & Posthuma-Doodeman 2005), D1 relates to the values adopted for thallium which are consistent with those recommended, and based on diet surveys in New Zealand (Pearson & Ashmore 2020)

E = TRV available from the UK Environment Agency (UK EA 2009d) for nickel, noting this value is protective of all adverse effects including carcinogenicity

O = TRV from OEHHA, as chronic reference exposure level (REL) (OEHHA)

N1 = TRV for selenium based on the upper intake limit for selenium in food and supplements as determined by NHMRC and MoH (NHMRC 2006)

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

W = TRV available from the WHO, relevant to chronic inhalation exposures (WHO 1999, 2000c, 2006a, 2017), noting inhalation value adopted for mercury is for elemental mercury (WHO 2003) which is lower than the NZ ambient air quality guideline (MfE 2002)

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.

B4 Detailed toxicity summaries for metals and dioxin-like compounds

B4.1 Antimony

Several comprehensive reviews of the potential health effects of antimony are available (ATSDR 1992a, 2019a; IARC 1989; USEPA IRIS).

Antimony is a silvery white metal of medium hardness that breaks easily. Small amounts of antimony are found in the earth's crust. Antimony ores are mined and then either changed into antimony metal or combined with oxygen to form antimony oxide (ATSDR 1992a, 2019a).

Antimony oxide is a white powder that does not evaporate. Only a small amount of it will dissolve in water. Most antimony oxide produced is added to textiles and plastics to prevent their catching on fire (ATSDR 1992a, 2019a).

Antimony metal is too easily broken to be used much by itself. To make it stronger, a little antimony is usually mixed with other metals such as lead and zinc to form mixtures of metals called alloys. These alloys are used in lead storage batteries, solder, sheet and pipe metal, bearings, castings, type metal, ammunition, and pewter (ATSDR 1992a, 2019a).

Antimony enters the environment during the mining and processing of its ores and in the production of antimony metal, alloys, antimony oxide, and combinations of antimony with other substances. Little or no antimony is mined in the United States, Antimony ore and impure metals are brought into this country from other countries for processing. Small amounts of antimony are also released into the environment by incinerators and coal-burning power plants. The antimony that comes out of the smoke stacks of these plants is attached to very small particles that settle to the ground or are washed out of the air by rain. It usually takes many days for antimony to be removed from the air. Antimony attached to very small particles may stay in the air for more than a month. Antimony

cannot be destroyed in the environment. It can only change its form or become attached to or separated from particles. Most antimony will end up in the soil or sediment, where it attaches strongly to particles that contain iron, manganese, or aluminium (ATSDR 1992a, 2019a).

Antimony and its compounds are among the oldest known remedies in the practice of medicine, and they have been used to treat a variety of illnesses over the last 600 years. Currently, antimony compounds are used to treat the parasitic disease leishmaniasis. Toxic side effects in humans following intraperitoneal, intravenous, or intramuscular injection of an antimony-containing drug have been reported, including altered electrocardiograms (EKGs), vomiting, diarrhea, and joint and/or muscle pain. These side effects are more frequently observed following administration of trivalent antimony compounds (ATSDR 2019a). Adverse health effects have also been observed in humans and animals following inhalation, oral, or dermal exposure to antimony and antimony compounds.

The most sensitive targets appear to be the respiratory tract, heart, gastrointestinal tract, serum glucose, and developing animal. A systematic review of these endpoints by ATSDR (2019) resulted in the following hazard identification conclusions:

- Respiratory effects following inhalation exposure are a presumed health effect for humans
- Myocardial effects and EKG alterations are a suspected health effect for humans
- Gastrointestinal effects are a presumed health effect for humans
- Developmental effects are a suspected health effect for humans
- Alterations in blood glucose levels are a suspected health effect for humans.

Background

Review of current information from Australia with respect to antimony indicates the following:

- Intakes of antimony were addressed by FSANZ (FSANZ 2003). Estimated dietary intakes for infants and 2-3 year olds ranged from 0.01 to 0.25 µg/kg bw/day which ranges from 3 to 61% of the adopted tolerable intake – 0.4 µg/kg bw/day – taken from USEPA IRIS summary for antimony (USEPA IRIS). The average intake of antimony is estimated to be 0.13 µg/kg/day for 2-3 year olds, approximately 20% of the TDI from the ADWG ((NHMRC 2011 updated 2018)) – the recommended oral TRV.
- Antimony was reported in ambient air data collected in (NSW DEC 2003) where concentrations in urban, regional and industrial areas assessed ranged from 0.04 to 4.6 ng/m^3. Intakes associated with these are concentrations are negligible compared with intakes from food.

Classification

IARC (IARC 1989) classified antimony trioxide as Group 2B: possibly carcinogenic to humans and antimony trisulfide as Group 3: not classifiable as to its carcinogenicity to humans.

Review of Available Values/Information

The following are available for the assessment of toxicity:

Table B4: Toxicity reference values - Antimony

Source	Value	Basis/Comments
ADWG (NHMRC 2011 updated 2022)	**TDI = 0.00086 mg/kg/day**	The ADWG derived a guideline of 0.003 mg/L based on a lowest effect level of 0.43 mg/kg/d from a lifetime study in rats showing decreased lifespan and altered blood levels of glucose and cholesterol and a safety factor of 500 (10 for interspecies, 10 for intraspecies and 5 as result was a lowest observed effect level rather than a no effect level.
WHO DWG (WHO 2017)	TDI = 0.00086 mg/kg/day	The WHO DWG derived a guideline of 0.005 mg/L using the same study as the ADWG but including rounding.
ATSDR (ATSDR 2019a)	Acute MRL = 0.001 mg/m^3 **Chronic MRL = 0.0003 mg/m^3**	Acute inhalation MRL based on respiratory effects (epithelium effects at base of epiglottis) in mice and a 30 fold uncertainty factor Chronic inhalation MRL based on respiratory effects (lung inflammation) in rats, and a 30 fold uncertainty factor.
USEPA IRIS (USEPA IRIS)	RfD = 0.0004 mg/kg/d	The USEPA IRIS entry (last reviewed in 1991) derived an oral RfD of 0.0004 mg/kg/day based on a LOAEL of 0.35 mg/kg/day from the same study in rats used in the ADWG with an uncertainty factor of 1000. The confidence level in the study, database and RfD is noted to be low.

It is recommended that the oral TDI from the Australian Drinking Water Guidelines be adopted for oral and dermal exposures as this is consistent with the value adopted by the WHO and similar to the USEPA evaluation, with the ATSDR chronic MRL adopted for inhalation exposures.

Recommendation

The following toxicity reference values (TRVs) have been adopted for antimony:

- Oral TRV (TRV_O) = 0.0009 mg/kg/day (NHMRC 2011 updated 2018)
- Inhalation TRV = 0.0003 mg/m^3 (ATSDR 2019a)
- Background intakes from other sources (as % of TRV) = 20% for oral/dermal intakes and negligible for inhalation exposures

B4.2 Arsenic

Background

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

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

Review of current information from Australia with respect to arsenic indicates the following:

- The most recent Australian Total Diet Survey (ATDS) that addresses arsenic in food was published by FSANZ in 2011 (FSANZ 2011). Based on data presented in this report, dietary intake of arsenic for children aged 2-5 years ranges from a mean of 1.2 µg/kg/day to a 90th

percentile of 2.8 µg/kg/day. These intakes are based on total arsenic in produce, rather than inorganic arsenic.

- Review of background intakes from food, water, air, soil and contact with play equipment based on available Australian data presented by (APVMA 2005) suggests background intakes of inorganic arsenic by young children may be on average 0.62 µg/kg/day. Further review of inorganic arsenic intakes by the Joint FAO/WHO Expert Committee on Food Additives indicated that for populations (not located in areas of arsenic contaminated groundwater) intakes by young children ranged from 0.14 to 1.39 µg/kg/day (WHO 2011a). On the basis of the range of intake estimations available, a reasonable estimation of 50% of the oral toxicity reference value (TRV) from sources other than soil has been assumed.
- Intakes from inhalation exposures are low (around 0.0017 µg/kg/day (APVMA 2005)), comprising <1% of the inhalation TRV adopted.

For this assessment, intakes from all other sources have been calculated separately based on available information on the existing environment.

With respect to arsenic toxicity and the identification of appropriate toxicity reference values a number of issues need to be considered. These include: the relevance of non-threshold carcinogenic values for the assessment of oral exposures; identification of an appropriate oral toxicity value; and identification of an appropriate approach and value for inhalation exposures. These are discussed in the following:

Classification

The International Agency for Research on Cancer (IARC) has classified arsenic and inorganic arsenic compounds as Group 1 ‘carcinogenic to humans’ (IARC 2012b).

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 2012b) 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 2012b) 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 2001a). The USEPA approach adopted follows a review by the (NRC 2001) which concluded that "... *internal cancers are more appropriate as endpoints for risk assessment than non-melanoma skin cancers*". Slope factors relevant for the assessment of these end points range from 0.4 to 23 $(mg/kg/day)^{-1}$. The use of a non-threshold approach (slope factor), however, is more by default through following the USEPA Carcinogenic Guidelines (USEPA 2005b) as there remains uncertainty on the carcinogenic MOA for arsenic (Sams et al. 2007). Further research is required to define and review the MOA prior to the USA revising the dose-response approach currently adopted. Inherent in the current US approach (where a non-threshold slope factor is derived) are some key uncertainties that likely result in an overestimate of risk, which include:

- the choice of the cancer endpoint;
- the choice of the mathematical model used to estimate risk (shape of the dose-response curve at low doses) as there is no clear biological basis for extrapolation; 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 2007d; IARC 2012b) and that evidence for the indirect mechanisms for genotoxicity identified in in vitro studies have nearly all been at concentrations that are cytotoxic (Klein et al. 2007).

Hence the default approach adopted by the USEPA in adopting a non-threshold approach to the assessment of the carcinogenic effects associated with arsenic exposure is not well supported by the available data. This is consistent with the most recent Australian review available (APVMA 2005). The review conducted considered current information on arsenic carcinogenicity and genotoxicity which noted the following:

> *"Although exposure to high concentrations of inorganic arsenic results in tumour formation and chromosomal damage (clastogenic effect), the mechanism by which these tumours develop does not appear to involve mutagenesis. Arsenic appears to act on the chromosomes and acts as a tumour promoter rather than as an initiator ...". "Furthermore, the epidemiological evidence from occupational exposure studies indicates that arsenic acts at a later stage in the development of cancer, as noted with the increased risk of lung cancer mortality with increasing age of initial exposure, independent of time after exposure...". "Hence arsenic appears to behave like a carcinogen which exhibits a threshold effect. This would also be conceptually consistent with the notion that humans have ingested food and water containing arsenic over millennia and so the presence of a threshold seems likely. Nevertheless the mechanism by which tumour formation develops following arsenic exposure has been and still continues to be a source of intensive scientific investigation."*

On the basis of the above the use of a threshold dose-response approach for the assessment of carcinogenic effects associated with arsenic exposure is considered.

The review of arsenic by the New Zealand Ministry for the Environment (MfE 2011a) noted that while there is general consensus that arsenic is likely to act indirectly on DNA in a sub-linear or threshold manner, it is considered that there is insufficient data available to determine a "well-defined non-linear dose-response". For this reason, the derivation of the New Zealand soil guideline values has adopted a non-threshold (linear) approach for arsenic (i.e. adopting a default non-threshold approach similar to that adopted by default by the USEPA). This differs from the approach adopted in Australia.

Assessment of End-Points – Oral Exposures

Existing Oral Dose-Response Approaches - Australia

Oral intakes of arsenic were considered in Australia in (Langley 1991) and the Australian Drinking Water Guidelines (ADWG) (NHMRC 2011 Updated 2016). 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 2011a) 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 2011a) 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 2010a). 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 2010a) 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 2011a) acknowledges the debate relating to the mechanism of action in relation to carcinogenicity. However, they have adopted a linear or non-threshold approach to the assessment of carcinogenic effects, as they consider there is insufficient data to define a threshold. The approach adopted for the quantification of the most sensitive effect, carcinogenicity, is to adopt a risk-specific dose of 0.0086 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 2010a; WHO 2011a, 2017) are based on drinking water studies. No studies considered are derived from other sources including soil. There are uncertainties inherent in the epidemiological studies considered by the WHO and EFSA (EFSA 2010a; WHO 2011a, 2017). These uncertainties include limitations or absence of information on levels of individual exposure or arsenic intake (from drinking water), limited quantification of arsenic intakes from other sources including food, size 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 2010a)) 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 2010a; WHO 2011a, 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 2010a; WHO 2011a);
- 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 2007d) 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 nickel in the ASC NEPM 2013 is 0.005 (NEPC 1999 amended 2013b).

Inhalation

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). 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 2007d)). 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 (2000) review of arsenic by WHO (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 review by WHO (WHO 2001b) 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 2001b)) 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 2001b) 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 has been linked to increased risk of lung cancer. The mechanisms of action for carcinogenicity has not been clearly identified, however as noted above, there is 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)$^{-1}$ determined
- 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.

Adopted TRVs

On the basis of the discussion above the following toxicity reference values (TRVs) have been adopted for the assessment of arsenic exposures in New Zealand, noting the values adopted are based on a non-threshold approach to the assessment of carcinogenicity, adopting a 1 in 100,000 risk, consistent with the approach detailed by MfE (as referenced):

- Oral TRV = 0.0086 µg/kg/day for oral and dermal intakes (MfE 2011a)
- Inhalation TRV = 0.0055 µg/m^3 (MfE 2002)
- Oral Bioavailability of 100% assumed
- Background Intakes from other sources (as % of TRV) = NA as the TRVs adopted are based on a non-threshold approach.

B4.3 Beryllium

General

Potential exposures to beryllium and the toxicity of beryllium have been evaluated and summarised in a number of reviews available from the WHO (WHO 2001c, 2017) and the US (ATSDR 2002; USEPA 1998b). The following provides a summary of information available from these reviews.

Beryllium is present in the earth's crust, in emissions from coal combustion, in surface water and soil, and in house dust, food, drinking water, and cigarette smoke. Beryllium is a very light metal, which is stronger than steel. It has a high melting point of 1287℃, conducts heat well and is resistant to corrosion. Its properties have made it useful for applications across many industries. Beryllium ores are used to make specialty ceramics for electrical and high-technology applications. Beryllium alloys are used in a wide range of applications including automobiles, aircraft engine parts and disc brakes, computers and calculators, televisions, sports equipment (such as golf clubs and bicycle frames), and dental bridges.

Occupational exposure to beryllium has been associated with acute and chronic lung diseases. Acute disease is normally associated with inhalation exposures to high levels of soluble beryllium salts (e.g. sulphate, chloride) and beryllium oxide (BeO) and may lead to chronic disease. 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. Dermatological effects may also occur on skin contact (Di Marco & Buckett 1996).

Exposure

Ingestion of soil and dust is considered the most significant pathway of exposure for inorganics. The consideration of bioavailability and inclusion of other exposure pathways has been further reviewed as noted below:

Dermal absorption:

In humans and animals sensitised to beryllium, contact with beryllium and its soluble and insoluble compounds can cause dermatitis and skin granulomas. In general, the more soluble the compound the greater the sensitising potential. Dermal effects usually occur on abraded skin. Dermal absorption of beryllium is assumed to be poor and would not likely cause further systemic effects.

It is noted that the US (RAIS) has recommended the use of a gastrointestinal absorption factor (GAF) of 0.7% based on consideration of the rat study (with water) used in the derivation of the oral TRV. The GAF is used to modify the oral toxicity reference value to a dermal value in accordance with the US EPA (2004) guidance provided.

Inhalation:

Beryllium is not volatile and inhalation exposures will be associated with particulates outdoors and indoors.

Plant Uptake:

Limited data are available on the potential for the uptake of beryllium into plants, in particular edible fruit and vegetable crops. Review by ATSDR (ATSDR 2002) notes that in plants the uptake of beryllium appears to be restricted to the root system with no significant translocation of beryllium to aboveground parts of the plant. Soluble forms of beryllium must be present for plant uptake to occur. In solution in the pH range of 6-8 beryllium is most commonly transformed to beryllium hydroxide which has a very low solubility. Hence the potential for plant uptake to be significant is considered to be low.

Based on the above the uptake of beryllium into root crops only has been considered. Limited plant uptake data are available, hence the value presented by RAIS of 0.0025 mg/kg fresh produce per mg/kg soil produce can be adopted.

Intakes from Other Sources – Background:

Limited data are available from Australia with respect to levels of beryllium in drinking water or food. Beryllium is not routinely monitored in Australian Drinking Water (NHMRC 2011 updated 2018). ATSDR (2002) report concentrations of beryllium in Australian rainwater tanks between 0.05-0.08 µg/L. Beryllium was not detected in any air sample collected in NSW (NSW DEC 2003). Hence intakes that may be derived from ambient air are considered negligible.

WHO (WHO 2009), which is consistent with IARC (IARC 2012a), estimated that intakes of beryllium were around 0.423 µg per day based on data from the US and Australia. These intakes (0.0000282 mg/kg/day for a 15 kg child) are negligible compared with the TRV adopted for the assessment of oral and dermal exposures.

Health effects

There are no human studies addressing the toxicokinetics of beryllium or beryllium compounds; however, beryllium has been found in the lungs and urine of non-occupationally exposed individuals. Beryllium and beryllium compounds are not metabolised. Animal studies have demonstrated that inhaled beryllium particles (insoluble) are cleared from the lungs slowly, so beryllium may remain in the lungs for many years after exposure. Pulmonary clearance of the soluble and sparingly soluble beryllium compounds via inhalation or intratracheal instillation appears to be biphasic, with a rapid first phase of a few days/weeks and a slower second phase, which may vary from a few weeks/months for the soluble compounds to months/years for the sparingly soluble compounds (WHO 2001c).

Soluble beryllium compounds are absorbed to a greater degree than sparingly soluble compounds following inhalation. Ingested beryllium is poorly absorbed (<1%) from the gastrointestinal tract. Absorbed beryllium is distributed primarily to the skeleton, where it accumulates where it has a biological half-life of more than 1 year. Elimination is very slow and occurs primarily in the urine. Unabsorbed beryllium is eliminated via the faeces shortly after exposure via inhalation (WHO 2001c).

There are no reliable data on the oral toxicity of beryllium in humans. Acute oral exposures to single doses of soluble beryllium compounds are moderately toxic; however, in the case of sparingly

soluble beryllium compounds, no oral single-dose studies are available. Short-, medium-, and long-term studies in animals showed that the gastrointestinal and skeletal systems are target organs for beryllium following oral exposure (WHO 2001c).

The lung is the primary target of inhalation exposure to beryllium in animals and humans. With respect to repeated or continuous exposures, the most marked effects (pneumonitis, fibrosis, proliferative lesions, metaplasia, and hyperplasia) were observed in the lungs of various animal species exposed to both soluble and sparingly soluble beryllium compounds. In humans, there is little information on the toxic effects of beryllium or its compounds following a single exposure via inhalation, although chemical pneumonitis (acute beryllium disease, or ABD) has been observed following single massive exposures. Short-term or repeated exposures of humans to beryllium or its compounds can result in an acute or chronic form of lung disease, depending upon the exposure concentration. ABD is generally associated with exposure levels above 100 µg beryllium/m^3, which may be fatal in 10% of cases. Chronic beryllium disease (CBD) is characterised by the formation of granulomas (a type of lung tumour), resulting from an immune reaction to beryllium particles in the lung. There is an extensive body of evidence documenting beryllium sensitization and CBD as the sensitive effects of inhalation exposure to beryllium (WHO 2001c).

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 2001c) also classified beryllium as carcinogenic based on occupational inhalation studies. It is noted that the evidence is limited because of relatively small increases in lung cancer risks, poorly defined estimates of beryllium exposure, incomplete smoking data, and lack of control for potential exposure to other carcinogens, including co-exposure to sulfuric or hydrofluoric acid mists during employment in the beryllium industry (WHO 2001c).

Genotoxicity data for beryllium are mixed and compound dependant (WHO 2001c). Although the bacterial assays have been largely negative, the mammalian test systems exposed to beryllium compounds have shown evidence of mutations, chromosomal aberrations, and cell transformations. ATSDR (2002) has considered beryllium compounds to be weakly genotoxic.

The mode of action for beryllium carcinogenicity is not well understood and the relevance of a non-threshold approach to the quantification of inhalation exposures is not clear. The following is noted by Di Marco and Buckett (1996) and is considered to remain relevant for the assessment of inhalation exposures:

> "*Whilst lung cancer is the most important endpoint, it is unlikely to be a concern for beryllium in soil. Acute beryllium lung disease appears to occur prior to the development of lung cancer and may play a role in its induction. In addition, this disease has only been reported after exposure to high levels of specific beryllium compounds in the workplace; conditions which are unlikely to be achieved on exposures to dust generated from beryllium contaminated soil*."

This is supported by a more recent review by Hollins et al. (Hollins et al. 2009) where it was concluded that "*the increase in potential risk of lung cancer was observed among those exposed to*

very high levels of beryllium and that beryllium's carcinogenic potential in humans at exposure levels that exist in modern industrial settings should be considered either inadequate or marginally suggestive".

Further review of genotoxicity by IARC (IARC 2012a) 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.

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 (NHMRC 2011 updated 2022).

Toxicity reference values

The following are available for beryllium:

Table B5: Toxicity reference values for beryllium

Source	Value	Basis/Comments
ADWG (NHMRC 2011 updated 2022)	TDI = 0.002 mg/kg/day	The ADWG derived a guideline of 0.06 mg/L for beryllium in drinking water based on a BMD of 0.46 mg/kg/day for gastrointestinal effects in a chronic dog study and application of a 300 fold uncertainty factor (10 for interspecies variation, 10 for intraspecies variation and 3 for database deficiencies).
WHO DWG (WHO 2017)	TDI = 0.002 mg/kg/day	The WHO DWG did not present a drinking water guideline, however they note a health based value of 12 mg/L may be derived using the same study as the ADWG but allowing for a different proportion of intakes from drinking water.
WHO (WHO 2001c)	TC = 0.00002 mg/m^3	TC based on CDB, characterised by the formation of granulomas. The TC is derived from a duration adjusted LOAEL or occupationally exposed workers and application of a 10 fold uncertainty factor. The WHO has also derived a non-threshold value for inhalation exposures, unit risk = 0.0024 (mg/m^3)$^{-1}$. This value has not been utilised in this assessment as it was derived on the basis of data relevant to a specific occupational exposure and there is insufficient evidence to indicate that beryllium in non-occupational environments is genotoxic and a non-threshold approach is applicable. The value also includes a significant level of uncertainty, particularly in relation to the estimation of beryllium exposures in the workplace.
ATSDR (ATSDR 2002)	MRL = 0.002 mg/kg/day	The MRL is based on the same study and approach adopted in the ADWG.
USEPA IRIS (USEPA 1998b)	RfD = 0.002 mg/kg/d RfC = 0.00002 mg/m^3	The oral RfD based on the same study and approach as outlined in the ADWG. RfC is based on CBD effects in humans and application of an uncertainty factor of 10. This is the same study and approach adopted by WHO

Based on the above table there is consensus across a wide number of evaluations that an oral TDI of 0.002 mg/kg/day as adopted in the ADWG and WHO (NHMRC 2011 updated 2022; WHO 2017). In addition, there is consensus that the appropriate threshold inhalation TRV is 0.00002 mg/m^3.

Recommendation

On the basis of the discussion above the following toxicity reference values (TRVs) have been adopted for beryllium:

- Oral TRV = 0.002 mg/kg/day for oral and dermal intakes, where derived from background/other sources are negligible
- Gastrointestinal absorption factor = 0.7%
- Inhalation TRV = 0.00002 mg/m^3, where background intakes are negligible

B4.4 Cadmium

General

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

Pure cadmium is a silver-white, lustrous and malleable metal, is a solid at room temperature, is insoluble in water, and has a relatively low melting point and vapour pressure. The most common oxidation state of cadmium is 2+. Naturally occurring cadmium is commonly found in the earth's crust associated with zinc, lead, and copper ores. Whereas pure cadmium and cadmium oxides are insoluble in water, some cadmium salts including cadmium chloride, cadmium nitrate, cadmium sulfate and cadmium sulfide are soluble in water (ATSDR 2012e).

Cadmium is found naturally in mineral forms (primarily sulfide minerals) in association with zinc ores, zinc-bearing lead ores, and complex copper-lead-zinc ores. Due to its corrosion-resistant properties, a wide range of commercial and industrial applications have been developed involving cadmium-containing compounds and alloys that are used in a wide range of materials and products including batteries, pigments, metal coatings and platings, stabilisers for plastics, nonferrous alloys and solar cell devices (ATSDR 2012e).

Cadmium is toxic to a wide range of organs and tissues, and a variety of toxicological endpoints (reproductive toxicity, neurotoxicity, carcinogenicity) have been observed in experimental animals and subsequently investigated in human populations (MfE 2011a).

The toxicity of cadmium in air is dependent on the form of cadmium. The toxicity is higher with the more soluble cadmium compounds. Acute inhalation exposure to cadmium at concentrations may cause destruction of lung epithelial cells, resulting in decreased lung function, pulmonary oedema, tracheobronchitis, and pneumonitis in both humans and animals. Other effects identified in animal studies include decreased immune response, erosion of the stomach, decreased body weight gain and tremors (ATSDR 2012e).

Numerous studies examining the toxicity of cadmium in workers have identified the respiratory tract, the kidney and bone as sensitive targets of toxicity. Other effects identified include developmental and reproductive effects, hepatic effects, haematological effects and immunological effects (ATSDR 2012e).

Background

The WHO review of cadmium included food intakes provided by FSANZ of 0.1 µg/kg/day (FSANZ 2003; WHO 2004a). Intakes for a young child aged 2-5 years from the 23rd Australian Food Survey ranged from a mean of 0.32 µg/kg/day to a 90th percentile of 0.44 µg/kg/day (FSANZ 2011). These intakes are similar to those estimated in New Zealand (MfE 2011a), which are 0.41 µg/kg/day for children and 0.26 µg/kg/day for adults. While the WHO (2004) review notes that intakes of cadmium from food can exceed the adopted toxicity reference value, data from FSANZ (2011) does not suggest this is the case. Based on the available data from FSANZ (2011), intakes from food comprise up to 60% of the recommended oral TRV.

Cadmium was detected in air samples collected from urban and rural areas in NSW (NSW DEC 2003). The average concentration reported was 0.17 ng/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 2012b). The USEPA has classified cadmium as a probable human carcinogen via inhalation.

Review of Available Values/Information

The following has been summarised from the review of cadmium presented by MfE (MfE 2011a):

- Cadmium is primarily toxic to the kidney, especially to the proximal tubular cells where it accumulates over time and may cause renal dysfunction. Loss of calcium from the bone and increased urinary excretion of calcium are also associated with chronic cadmium exposure. Recent studies have reported the potential for endocrine disruption in humans as a result of exposure to cadmium. Notably, depending on the dosage, cadmium exposure may either enhance or inhibit the biosynthesis of progesterone, a hormone linked to both normal ovarian cyclicity and maintenance of pregnancy. Exposure to cadmium during human pregnancy has also been linked to decreased birth weight and premature birth.
- While cadmium has been classified as known human carcinogen (based on inhalation data from occupational inhalation data), there is no evidence of carcinogenicity via the oral route of exposure.
- There 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:

Table B6: Toxicity reference values for cadmium - Oral

Source	Value	Basis/Comments
ADWG (NHMRC 2011 updated 2022)	TDI = 0.0007 mg/kg/day	The threshold oral value available from the ADWG (NHMRC 2011 updated 2022) of 0.0007 mg/kg/day is derived from a WHO/JECFA evaluation in 2000. The JECFA summary provided in 2004 noted that a PTWI of 0.007 mg/kg was established in 1988. This differs from that referenced (not cited) and considered in the ADWG. It is noted however that the WHO may have rounded the TDI adapted as both values are similar.
MfE (MfE 2011a)	TDI = 0.0008 mg/kg/day	Adopted the toxicity value from the WHO review (as below).
JECFA (WHO 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 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 2012b). 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 µg/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 µg/m^3 – the same as the WHO value).

The review by TCEQ (TCEQ 2016) indicated that multiple mechanisms (e.g., aberrant gene expression, inhibition of DNA damage repair, induction of oxidative stress/reactive oxygen species and genomic instability, inhibition of apoptosis) appear to be involved in cadmium-induced carcinogenesis. The approach adopted for the derivation of a chronic air guideline was to consider noncarcinogenic effects (kidney effects most sensitive) and carcinogenic effects using a linear (non-threshold) approach. The air guideline derived based on protection of kidney effects 0.011 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.

Recommendation

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), with 60% background intakes
- Inhalation TRV (TRV_I) = 0.000005 mg/m^3 (WHO 2000g), with 20% background intakes

B4.5 Chromium VI

Several comprehensive reviews of chromium VI (Cr VI) in the environment and toxicity to humans are available (APVMA 2005; ATSDR 2012d; UK DEFRA & EA 2002a).

Cr VI is less stable than the commonly occurring trivalent chromium but can be found naturally in the rare mineral crocoite. Cr VI typically exists as strongly oxidizing species such as CrO_3 and CrO_4^{2-}. Some Cr VI compounds, such as chromic acid and the ammonium and alkali metal salts (e.g., sodium and potassium) of chromic acid are readily soluble in water. The Cr VI compounds are reduced to the trivalent form in the presence of oxidisable organic matter. However, in natural waters where there is a low concentration of reducing materials, Cr VI compounds are more stable (ATSDR 2012d).

Chromium is of fundamental use in a wide range of industries including the metallurgical (to produce stainless steels, alloy cast irons and nonferrous alloys), refractory (to produce linings used for high temperature industrial furnaces) and chemical industries. In the chemical industry, Cr VI is used in pigments, metal finishing and in wood preservatives (ATSDR 2012d).

The soil chemistry and toxicity of chromium is complex and hence the form of chromium in soil is of importance. In general soil chromium is present as Cr III, however the distribution of Cr III and Cr VI depends of factors such as redox potential, pH, presence of oxidising or reducing compounds and formation of Cr complexes and salts (ATSDR 2012d).

Cr VI can readily pass through cell membranes and be absorbed by the body. Inside the body, Cr VI is rapidly reduced to Cr III. This reduction reaction can act as a detoxification process when it occurs at a distance from the target site for toxic or genotoxic effect. Similarly if Cr VI is reduced to Cr III extracellularly, this from of the metal is not readily transported into cells and so toxicity is not observed (ATSDR 2012d). However, if Cr VI is transported into cells, and close to the target site for toxic effect, under physiological conditions it can be reduced. This reduction reaction produces reactive intermediates, which can attack DNA, proteins, and membrane lipids, thereby disrupting cellular integrity and functions (ATSDR 2012d).

The toxicity is higher for soluble forms of Cr VI than insoluble forms. The respiratory system is the most sensitive health effect for both forms (TCEQ 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 2013).

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 2013).

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 2013).

Background

Review of current information with respect to chromium intakes indicates the following:

- Intakes of total chromium were addressed in the FSANZ 22nd Australian Total Diet Survey (FSANZ 2008). Estimated dietary intakes of chromium (total) for infants and 2-3 year old's ranged from 14 µg/day to 26 µg/day, and for adults ranged from 14 µg/day to 53 µg/day for males 19-30 years. The average values reported are consistent with intakes reported from Germany and US by APVMA (APVMA 2005). Dietary intakes of total chromium may comprise a significant portion of the TDI for Cr VI. However, it is noted that the most common form of chromium in fresh produce is Cr III. If Cr VI comprised 10% of the total Cr intake from the diet (based on data from bread analyses, (Soares, Vieira & Bastos Mde 2010) then background intakes may comprise 0.09 to 0.17 µg/kg/day for young children aged 2-3 years. It is considered reasonable that an average intake be adopted given additional intakes from plant uptake are included in addition to these intakes, resulting in some doubling up of intakes from food sources. The average intake of Cr VI is estimated to be 0.13 µg/kg/day for 2-3 year 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 2002a). 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 2012b) 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 1998a).

Review of Available Values/Information

Oral

There is 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:

Table B7: Toxicity reference values for Cr VI – Oral

Source	Value	Basis/Comments
ADWG (NHMRC 2011 updated 2022)	No evaluation available	The ADWG does not specifically derive a guideline; however it references the WHO DWG assessment, where the basis for derivation is not clear. No quantitative toxicity values can be obtained from these sources.
New Zealand (MfE 2011a)	0.003 mg/kg/day	Adopted the RfD from the USEPA evaluation.
WHO DWG (WHO 2017)	No evaluation available	Current guideline based on limit of detection as no adequate toxicity studies were available to provide the basis for a NOAEL. It is noted that chromium is included in the plan of work of rolling revisions to the WHO DWG (2011).
UK DEFRA & EA (UK DEFRA & EA 2002a)	TDI = 0.003 mg/kg/day	Adopted oral RfD from the USEPA.
RIVM (Baars et al. 2001)	TDI = 0.005 mg/kg/day	RIVM has adopted a provisional threshold TDI of 0.005 mg/kg/day based on a 1-year drinking water study in rats as used in the derivation of the former and current USEPA RfD (with a small difference in the application of uncertainty factors).
ATSDR (ATSDR 2012d)	MRL = 0.0009 mg/kg/day	The chronic oral MRL is based on a $BMDL_{10}$ of 0.09 mg/kg/day for non-neoplastic lesions of the duodenum in a 2-year drinking water study in rats and mice (NTP 2008) and an uncertainty factor of 90. The study considered by ATSDR was not available when the other organisations (USEPA etc) reviewed Cr VI.
USEPA IRIS (USEPA 1998a)	RfD = 0.003 mg/kg/day	The USEPA IRIS entry (last reviewed in 1998) derived an oral RfD of 0.003 mg/kg/day based on a NOAEL of 2.5 mg/kg/day from a 1-year drinking water study in rats and an uncertainty factor of 300 and modifying factor of 3 to address uncertainties in the study. The confidence level in the study, database and RfD is noted to be low.

It is recommended that the lower value derived by (ATSDR 2012d) be adopted for the assessment of oral exposures to Cr VI as the assessment provides the most current comprehensive assessment of the available studies, including a more recent key study (NTP 2008) not available at the time of review by other organisations. The values adopted by New Zealand 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 2002a), there is some suggestion that chromium-induced cancer of the respiratory tract may be exclusively a high-dose phenomenon with a threshold approach relevant to low-dose exposures but quantitative data is lacking.

Chromium is not volatile and hence inhalation exposures are only relevant to dust intakes. These are not likely to be significant for soil contamination and hence the consideration of carcinogenic effects using a non-threshold approach may not be appropriate. It is appropriate to consider intakes on the basis of a threshold approach associated with the most significant end-point. In addition, inhalation exposures relating to soil contamination (dust) are expected to differ from the occupation studies from which the non-threshold criteria are derived (where inhalation of fine dust and chromic acid mists occurs). These issues were considered by ITER (ITER 1998) in the derivation of an RfC that is relevant for environmental exposures only, not to occupational exposures associated with mists and aerosols, and USEPA (USEPA 1998a) in the derivation of an RfC.

The following are available for inhalation exposures for Cr VI particulates or dust:

- No Australian guideline values are available for Cr VI.
- The WHO (WHO 2013) 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, 2013) inhalation unit risk of 0.04 (µg/m^3)$^{-1}$ derived from the mean of a number of occupational studies.
- The USEPA (USEPA 1998a) 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 1998a) 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 2002a) 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 2002a) 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 2012d) has derived a chronic inhalation MRL for Cr VI aerosols and mists but this is not considered relevant to the derivation of toxicity reference values for Cr VI bound to particulates.

Based on the above there are a range of values available, with mixed guidance as to the most appropriate approach to adopt for assessing inhalation exposures to Cr VI bound to particulates. To be sufficiently conservative the air guideline adopted in New Zealand (MfE 2002) which is similar to the more recent value from TCEQ (TCEQ 2014b) has been adopted in this assessment.

Recommendation

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 2012d)
- Inhalation TRV (TRV_i) = 0.0011 µg/m^3 (MfE 2002)
- Background intakes from other sources (as % of TRV) = 10% for oral/dermal intakes and 0% for inhalation (noting inhalation exposures are assessed on the basis of a non-threshold approach where background is not relevant).

B4.6 Copper

Several comprehensive reviews of copper in the environment and toxicity to humans are available (ATSDR 2004b, 2022; NEHF 1997; WHO 1998).

Copper (Cu) can occur naturally in its elemental form. Copper may also occur in the environment in various mineral forms including cuprite (Cu_2O), malachite ($CuCO_3 \cdot Cu(OH)_2$), azurite ($2CuCO_3 \cdot Cu(OH)_2$), chalcopyrite ($CuFeS_2$), chalcocite (Cu_2S), and bornite (Cu_5FeS_4). Metallic copper is a malleable and ductile solid that has strong electrical and thermal conducting properties and low corrosiveness. Copper is a transition metal and may occur as either the monovalent or divalent cation]. Copper may exist in four oxidation states Cu(0), Cu(I), Cu(II) and Cu(III) (ATSDR 2004b; WHO 1998).

Copper is a naturally occurring trace element of significant societal importance. It is not only an essential nutrient in virtually all forms of life; it is also an important constituent in numerous consumer and industrial materials, both as the free metal and as a component in metal alloys. Common copper metal alloys include brass, bronze and gun metal. Copper and copper alloys are used in plumbing, telecommunications, power utilities, air conditioning, automotives, business electronics and industrial valves. Copper sulfate and other copper compounds are important

constituents in products having agricultural (namely fungicides), and other applications including metal finishing, wood preservatives and water treatment (ATSDR 2004b).

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.

Because copper is an essential metal, cells, tissues and organisms have mechanisms to maintain copper levels within defined limits and for maintaining its availability while limiting its toxicity (homoeostasis). However, there are several disorders of homoeostatic mechanisms – such as Wilson's disease, Indian childhood cirrhosis and idiopathic copper toxicosis, which can result in deficiency or toxicity from exposure to copper at levels that are tolerated by the general population (MfE 2011a). High levels of exposure, however can overwhelm the homeostatic mechanisms and lead to toxicity.

Thus, toxic effects arising from copper tend to be observed only in people who have disorders in copper metabolism, and/or whose copper intake levels are excessive. Health effects include liver damage (e.g., hepatitis, jaundice, hepatic necrosis) which is key health effect insusceptible sub-populations, gastrointestinal effects (e.g., nausea, vomiting, diarrhoea) and contact dermatitis in susceptible individuals. Liver and gastrointestinal effects are the most sensitive (ATSDR 2022; MfE 2011a).

Background

Review of current information from Australia with respect to copper indicates the following:

- Intakes of copper were reported in the 20th Total Diet Survey (FSANZ 2003) where intakes by infants were identified as highest, at 0.065 mg/kg/day. Intakes by toddlers (2 years) were up to 0.04 mg/kg/day. Intakes of copper in the 23rd Australian Food Survey (FSANZ 2011) indicated intakes by young children aged 2-3 years ranged from a mean of 0.068 mg/kg/day to a 90th percentile of 0.094 mg/kg/day.
- Typical concentrations of copper reported in the ADWG (NHMRC 2011 updated 2022) are 0.05 mg/L, resulting in an intake (1 L/day and body weight of 15.5 kg) by toddlers of 0.004 mg/kg/day. It is noted that intakes of copper in drinking water supplies in New Zealand (MfE 2011b) were higher, with intakes by a young child estimated to be 0.013 mg/kg/day. It should also be noted that intakes of copper as reported in the Total Diet Surveys include intakes of water as part of the diet.
- Copper was reported in ambient air data collected in (NSW DEC 2003) where concentrations in urban, regional and industrial areas assessed ranged from 2.4 to 28 ng/m^3. Intakes associated with these are concentrations are negligible compared with intakes from food.

RIVM (Baars et al. 2001) reviewed background intakes which were considered to be 30 µg/kg/day for adults. ATSDR (ATSDR 2022) indicates the average daily dietary intake from food is around 2 mg/day.

New Zealand has adopted a background dietary intake (from National Nutrition Surveys in 1997 and 2002) of 0.056 mg/kg/day for children and 0.02 mg/kg/day for adults.

Based on data from Australia (which is conservative for New Zealand) for infants and young children, background intakes may comprise approximately 0.08 mg/kg/day, which is 60% of the recommended oral TRV.

Classification

The International Agency for Research on Cancer (IARC) has not classified copper and copper compounds, however copper 8-hydroxyquinoline has been classified (IARC 1977) as Group 3: not classifiable. It is noted that the US EPA has assessed copper as Group D: not classified. These classifications remain current.

Review of Available Values/Information

Copper is not considered to be carcinogenic and therefore the consideration of a threshold dose-response approach is considered appropriate.

The following threshold values are available for the assessment of copper toxicity.

Table B8: Toxicity reference values for copper

Source	Value	Basis/Comments
ADWG (NHMRC 2011 updated 2022)	TDI = 0.5 mg/kg/day	The Australian Drinking Water Guidelines derived a health based guideline of 2 mg/L based on the provisional TDI of 0.5 mg/kg/day derived from the WHO (1982). The evaluation from 1982, which has not been updated, identified a range of provisional maximum tolerable daily intakes (PMTDI) of 0.05-0.5 mg/kg/day. The ADWG have adopted the upper end of the range provided.
OCS (OCS 2014)	ADI = 0.2 mg/kg/day	The ADI of 0.2 mg/kg/day is also listed on the current ADI list where it is noted to have been set in June 2005, based on the upper safe limit for adults set by FSANZ.
FSANZ (FSANZ 2003)	TL = 0.2 mg/kg/day	FSANZ have adopted a tolerable limit of 0.2 mg/kg/day for copper referenced from the WHO (“Trace Elements in Human Nutrition”, 1996).
WHO DWG (WHO 2017)	TDI = 0.14 mg/kg/day	The current drinking water guidelines have also derived a guideline of 2 mg/L, however they also note that intakes derived from consuming 2-3 L water per day are not expected to exceed a tolerable upper intake level of 10 mg/day (IOM 2001). This upper intake would be equal to a TDI of 0.14 mg/kg/day for a 70 kg adult. Copper is noted to be in the current WHO list for rolling revisions to the drinking water guidelines.
NZ MfE (MfE 2011a)	TDI = 0.15 mg/kg/day	Consistent with the WHO, MfE has adopted the IOM (2001) upper intake of 10 mg/day, and used a body weight of 70kg for an adult. The value is rounded up to 0.15 mg/kg/day.
RIVM (Baars et al. 2001)	TDI = 0.14 mg/kg/day TC = 0.001 mg/m^3	RIVM identified an oral TDI of 0.14 mg/kg/day based on a LOAEL from a chronic oral study in mice. This study was not available at the time the WHO conducted their evaluation. The TDI derived is noted to be above the minimum dietary requirements for copper. Despite a poor database, RIVM also derived an inhalation TC of 0.001 mg/m^3 based on a NOAEC of 0.1 mg/kg/day (adjusted) associated with lung and immune system effects from a subacute study with rabbits and an uncertainty factor of 100. It is not recommended that the inhalation TC be considered due to the limited data available with respect to chronic inhalation exposures to copper.
ATSDR (ATSDR 2004b, 2022)	No chronic MRLs available	ATSDR (ATSDR 2022) provides acute and intermediate duration oral MRLs for copper, but no inhalation or chronic values due to a lack of suitable studies.
US EPA IRIS (USEPA)	No evaluation available	

Based on the available data an oral TRV of 0.14 mg/kg/day from the WHO (WHO 2017) evaluation is recommended to be adopted. The value is based on a tolerable upper limit (IOM 2001) and is

similar to the TDI currently adopted by (Baars et al. 2001; FSANZ 2003; OCS 2014) (where the value may be rounded). The recommended TRV is considered relevant for the assessment of copper intakes from oral, dermal and inhalation routes of exposure.

Recommendation

On the basis of the discussion above the following toxicity reference values (TRVs) have been adopted for copper:

- Oral TRV (TRV_O) = 0.14 mg/kg/day (Baars et al. 2001; WHO 2011b) for all routes of exposure
- Background intakes for the general population = 0.08 mg/kg/day = 60% of the oral TRV

B4.7 Cobalt

Several comprehensive reviews of cobalt in the environment and toxicity to humans are available (ATSDR 2004c; WHO 2006a).

Cobalt (Co) is a silvery grey solid at room temperature. Naturally occurring cobalt is most commonly found in association with nickel, silver, lead, copper, and iron ores. Common cobalt minerals include linnaeite (Co_3S_4), carrolite ($CuCo_2S_4$), safflorite ($CoAs_2$), skutterudite ($CoAs_3$) and glaucodot (CoAsS). In the natural environment, cobalt may be found in two oxidation states, Co^{2+} and Co^{3+} dependent upon redox potential and pH of the environment (WHO 2006a).

Cobalt comprises approximately 0.0025% of the weight of the earth's crust, making it the 33rd most abundant element. Cobalt is a key constituent in several alloys including alnico, an alloy with powerful permanent magnetic properties which is used for high-speed, heavy-duty, high temperature cutting tools. Cobalt has also been used as a colorant in glass, ceramics, and paints; is of catalytic use to the petrochemical and plastic industries and is applied to soils as a fertiliser to increase plant yields or to increase the cobalt concentration in forage crops and prevent the symptoms of cobalt deficiency in livestock (ATSDR 2004c; WHO 2006a).

Cobalt is a dietary essential element as it is a key component of Vitamin B12 (ATSDR 2004c). As such adverse effects can occur as a result of deficiency as well as contamination. Without sufficient levels of dietary cobalt, red blood cell production may be severely inhibited leading to anaemia, heart disease, reduced growth and the breakdown of both the nervous and the immune systems in humans (IARC 1991). Excess amounts of cobalt may also have harmful effects in humans. Inhaled cobalt primarily targets the respiratory tract. From the respiratory tract, cobalt particles may be absorbed into the blood via dissolution or transported to the gastrointestinal tract with mucous when swallowing. Gastrointestinal cobalt absorption rates are reported to vary greatly in humans, with some studies associating iron deficiencies with increased cobalt absorption rates (ATSDR 2004c). Cobalt in the body partakes in reactions which generate oxidants and free radicals capable of deoxyribonucleic acid (DNA) damage and other deleterious effects (ATSDR 2004c).

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).

Background

Review of current information from Australia with respect to cobalt indicates the following:

- The most significant source of intake of cobalt from sources other than contamination is dietary intake (WHO 2006a). Cobalt intakes were considered in the 23rd Australian Food Survey (FSANZ 2011) where intakes for a child aged 2-3 years ranged from a mean of 1 µg/kg/day to a 90th percentile of 1.3 µg/kg/day. RIVM (Baars et al. 2001) reviewed background intakes of cobalt which were considered to be 0.3 µg/kg/day, consistent with intakes from food noted by the WHO (WHO 2006a) (where a body weight of 70 kg was assumed). These intakes are between 20% and 70% of the recommended oral TRV. Given the lack of data in support of oral TRVs for cobalt, and that the only available value from RIVM has been adopted, the lower value of 20% (based on the review by RIVM) has been used.
- Cobalt was reported in ambient air data collected in (NSW DEC 2003) where concentrations in urban, regional and industrial areas assessed ranged from 0.1 to 0.39 ng/m^3. Intakes associated with these are concentrations are negligible compared with intakes from food and the recommended inhalation TRV.

Classification

The International Agency for Research on Cancer (IARC 1991) has classified cobalt metal, cobalt sulphate and other soluble cobalt (II) salts as Group 2B: possible human carcinogen. IARC provided further review in 2006 classifying cobalt sulphate and other soluble cobalt (II) salts as Group 2B, cobalt metal without tungsten carbide as Group 2B and cobalt metal with tungsten carbide as Group 2A (probable human carcinogen).

Under the 2005 Guidelines for Carcinogen Risk Assessment, cobalt sulfate (soluble) is described as “likely to be carcinogenic to humans by the inhalation route,” based on both the limited evidence of carcinogenicity in humans and sufficient evidence of carcinogenicity in animals (USEPA 2008).

Review of Available Values/Information

While data are limited, based on the weight of evidence cobalt is not (or weakly) genotoxic (ATSDR 2004c; Baars et al. 2001). However, it is noted that some information suggests that some metallic cobalt species may be genotoxic (WHO 2006a), particularly in relation to the assessment of inhalation exposures. Review of the available data by TCEQ concludes that while the evidence is mixed, the weight of evidence for genotoxicity is weak and carcinogenicity is likely to occur via a non-genotoxic mechanism. While the USEPA and TCEQ have derived inhalation values on the basis of a default linear/non-threshold approach, these are not considered appropriate for cobalt.

Few quantitative evaluations are available for cobalt, however the following are available:

Table B9: Toxicity reference values for cobalt

Source	Value	Basis/Comments
WHO DWG (WHO 2017)	No evaluation available	
WHO (WHO 2006a)	TC = 0.0001 mg/m^3	The WHO (2006) derived a TC in air of 0.0001 mg/m^3 based on a NOAEC from an occupational inhalation study with conversions to address exposures by the general population. The WHO did not derive an oral threshold value due to the lack of suitable data.
RIVM (Baars et al. 2001)	TDI = 0.0014 mg/kg/day TC = 0.0005 mg/m^3	RIVM (2001) derived a TDI of 0.0014 mg/kg/day based on a LOAEL of 0.04 mg/kg/day associated with cardiomyopathy from oral exposures in workers and an uncertainty factor of 30. TC based on a LOAEC of 0.005 mg/m^3 for interstitial lung disease in workers and an uncertainty factor of 100.
TCEQ (TCEQ 2017c)	Acute ReV = 0.69 µg/m^3 Chronic ReV = 0.063 µg/m^3	Acute ReV based on respiratory irritation effects in a human study and application of a 100 fold uncertainty factor. Chronic ReV is based on respiratory irritation effects in an occupational study and application of a 30 fold uncertainty factor. This value is similar to that derived by the WHO (2006).
ATSDR (ATSDR 2004c)	Inhalation MRL = 0.0001 mg/m^3	Chronic inhalation MRL of 0.0001 mg/m^3 based on a NOAEL of 0.0013 mg/m^3 (adjusted) for decreased respiratory function in workers and an uncertainty factor of 10. No chronic oral MRL is available from ATSDR (2004).
US EPA (IRIS) (USEPA)	No evaluation available	
USEPA PPRTV (USEPA 2008)	p-RfD = 0.0003 mg/kg/day p-RfC = 0.006 µg/m^3	Provisional RfD based on decreased iodine uptake in humans and application of a 3000 fold uncertainty factor. Provisional RfC based on studies related to respiratory effects in humans and animals, with the tox value based on a NOAEL from an occupational study and application of a 300 fold uncertainty factor. These are provisional values only and are not considered appropriate to adopt in this assessment. Provisional inhalation unit risk of 9 (mg/m^3)$^{-1}$ based on a liner/non-threshold approach for carcinogenic effects. For a 1 in 100,000 risk, this result in an air guideline of 0.0011 µg/m^3, similar to the noncarcinogenic pRfC.

Only one oral value is available from RIVM, which is recommended to be adopted. The available inhalation values are fairly consistent with the most recent detailed evaluations provided by WHO and ATSDR.

Inhalation exposures have been assessed on the basis of the WHO evaluation, which is similar to the more recent review from TCEQ.

Recommendation

On the basis of the discussion above the following toxicity reference values (TRVs) have been adopted for cobalt in this assessment:

- Oral TRV (TRV_O) = 0.0014 mg/kg/day (Baars et al. 2001) for oral and dermal routes of exposure
- Background intakes from other sources (as % of TRV) = 20% for oral intakes
- Inhalation exposures, TC = 0.0001 mg/m^3 (WHO 2006a), where background intakes are considered negligible.

B4.8 Lead

General

Lead (Pb) is a naturally occurring element found in the earth's crust at an average concentration of approximately 15 to 20 mg/kg. It is most commonly found in ores such as galena (PbS), anglesite ($PbSO_4$) and cerussite ($PbCO_3$). Lead is a bluish-grey, soft, dense, malleable, corrosion resistant metal that is solid at room temperature and has a low melting point. It exists in three oxidation states, Pb(0) (metallic lead) Pb(II) and Pb(IV). The most common oxidation state of lead is Pb(II) (ATSDR 2007a).

Lead is of primary use in a wide range of materials including batteries, metal alloys, x-ray shielding materials, ammunition, chemical resistant linings and pigments. Lead has been widely used historically as an additive in petrol and also in many paints (ATSDR 2007a).

Exposure

Most people in Australia and New Zealand live in places where there are very small amounts of lead in food, drinking water, air, dust, soil, and consumer products. Most of this lead is left over from when lead was widely used in the manufacture of industrial and household goods. Lead added to paint and petrol was previously the main source of lead exposure in the community. Prior to initiatives that limited the use of lead in manufacturing, most Australians handled, breathed and swallowed small amounts of lead every day (NHMRC 2015b).

Inhalation

Lead is not volatile, so inhalation of lead may occur when lead is actively placed into the air. This may occur during dust generation from lead contaminated soil or uncontrolled emissions from lead smelting. The NHMRC note that when old houses and buildings are renovated, lead paint is often stripped or sanded which creates very fine particles of lead in dust that may be inhaled or consumed by people living or working inside or nearby the property (NHMRC 2015b).

Dermal absorption

Dermal exposure to lead may occur during contact with lead contaminated soil or lead products. Dermal absorption of inorganic lead is considered to be negligible, while organic lead is considered far more permeable to the skin and can have a role in lead exposure (ATSDR 2007a).

Ingestion

Lead occurs in the environment as a wide variety of compounds and remains permanently in dust and soil until it is physically removed. In some communities with a history of high traffic flow, soil may still contain lead deposited from traffic fumes prior to the removal of lead from petrol (NHMRC 2015b). Ingestion of soil and dust is considered a significant pathway of exposure where soil has raised lead concentrations.

Ingestion of plants grown in contaminated soil is also considered a small but possible pathway. IARC (IARC 2006a) has noted that plant uptake of lead from soil is low due to the low bioavailability of lead in soil and its poor translocation from the root to the shoot. Of all the toxic heavy metals,

lead is considered the least phytoavailable. While soil properties affect the potential for uptake and translocation, water soluble and exchangeable lead that is readily available for uptake by plants constitutes only 0.1% of the total lead in most soils. Hence a chelate (such as EDTA) is used to increase lead uptake and translocation where phytoremediation is required. In most instances intake of lead from home grown produce is accounted for through background dietary exposures, except in the case where the form of lead in soil is more soluble and available for plant uptake.

Background Intake (Exposure)

Information available from Australian in relation to background intakes of lead includes the following:

- Dietary intakes of lead in Australia have been reported in 2003 and 2011 with the most recent data from 2019 (FSANZ 2003, 2011, 2019). Mean intakes in 2019 for adults are in the range 0.018 to 0.16 µg/kg/day (with P90 intakes in the range 0.036 to 0.24 µg/kg/day), 0.048 to 0.38 µg/kg/day for children aged 2-5 years (with P90 intakes in the range 0.1 to 0.56 µg/kg/day), 0.029 to 0.24 µg/kg/day for children aged 6-12 years (with P90 intakes of 0.027 to 0.39 µg/kg/day) and 0.02 to 0.18 µg/kg/day for all individuals aged 2 years and above (with P90 intakes in the range 0.04 to 0.28 µg/kg/day).
- The ADWG (NHMRC 2011 updated 2022) notes that lead concentrations in drinking water range up to 0.01 mg/L with typical concentrations less than 0.005 mg/L. enHealth (enHealth 2021) has issues a guidance statement relating to minimising lead in drinking water. Intakes of lead in drinking water are included in the assessment of dietary intakes conducted by FSANZ.
- Concentrations of lead in air have been derived from Australian data on lead levels in urban, suburban and rural areas. (NSW DEC 2003) report concentrations of lead in air that range from 2.4 to 99 ng/m^3 with an average of 30 ng/m^3. Intakes derived from urban air are considered negligible in comparison with that derived from dietary and water sources.
- Total intakes from sources other than soil are dominated by dietary intakes, where mean intakes for relevant age groups may be considered. These intakes have been taken to be 0.2 µg/kg/day for adults, 0.4 µg/kg/day for children aged 2-5 years, 0.2 µg/kg/day for children aged 6-12 years and for all individuals aged 2 and above.
- Background levels of lead in soil (in non-contaminated areas) can be highly variable. For NSW, the mean lead level in urban soil is 83.8 mg/kg (Olszowy, Torr & Imray 1995). This results in an intake of 0.06 µg/kg/day for adults, 0.5 µg/kg/day for children aged 2-5 years and 0.2 µg/kg/day for children aged 6-12 years.

Data from New Zealand (MfE 2011a) identified background intakes were 0.97 µg/kg/day for young children and 0.41 µg/kg/day for adults, higher than identified in Australia.

Where site-specific or area-specific information is available on background intakes of lead, these should be used in preference to the information above, which is generic.

Absorption, Distribution, Metabolism and Excretion

The absorption of lead will depend on the route of exposure, but oral or inhalation intake provide a far more efficient route of absorption than the dermal route. The absorption and distribution of lead varies depending on duration and intensity of the exposure, particle size, age, and various physiological variables (e.g. nutritional status and pregnancy) (ATSDR 2007a).

Absorption - Inhalation

For inhalation, absorption of inorganic lead will be influenced by particle size, solubility and age-related factors that determine breathing patterns. Larger particles (>2.5 µm) that are deposited in the ciliated airways (nasopharyngeal and tracheobronchial regions) can be transferred by mucociliary transport into the esophagus and swallowed. Smaller particles (<1 µm), which can be deposited in the alveolar region, can be absorbed after extracellular dissolution or ingestion by phagocytic cells (ATSDR 2007a). Several studies have shown lead particles deposited in the alveoli of the lung are absorbed relatively quickly and completely. Most of the lead deposited in the alveoli is absorbed into the systemic circulation and little is brought up by cilliary action and swallowed (Safe Work Australia 2014a). This is in contrast to the larger particles (>2.5 µm) that are transferred within hours by mucociliary transport into the oesophagus and mainly swallowed, meaning the digestive tract can also be an important avenue of lead absorption following inhalation (Safe Work Australia 2014a).

A review of studies by the ATSDR found that approximately 25% of inhaled inorganic lead particles were deposited in the lung, of which 95% were absorbed. For organic lead particles 37% of inhaled organic lead particles were deposited in the lung, of which 80% were absorbed (ATSDR 2007a).

Absorption - Oral

The extent and rate of gastrointestinal absorption of ingested inorganic lead are influenced by physiological states of the exposed individual (e.g., age, fasting, nutritional calcium and iron status, pregnancy) and physicochemical characteristics of the medium ingested (e.g., particle size, mineralogy, solubility, and lead species). Lead absorption may also vary with the amount of lead ingested (ATSDR 2007a). The WHO indicate that absorption of lead can range from 3% to 80% with typical absorption rates in adults and infants considered to be 10 and 50% respectively (WHO 2000e). The gastrointestinal absorption of lead appears higher for children than adults, while the presence of food in the gastrointestinal tract decreases lead absorption. Deficiencies in dietary iron and calcium is believed to be related to higher lead absorption, as is pregnancy. The intake of lead via the oral route is considered a capacity limiting process, where the percentage of absorption may decrease with increased intake. Smaller lead particles are believed to be absorbed more readily, while lead in soil is absorbed less than dissolved lead (ATSDR 2007a).

The oral bioavailability of lead in soil (availability of lead to be dissolved from the soil particle and absorbed in the gastrointestinal tract) is of particular concern for international agencies where a number have considered bioavailability in the derivation of soil guideline values. For soil the bioavailability includes the movement of lead from soil into solution (bioaccessibility) and absorption into body. The available approaches include (MfE 2011a):

- RIVM (Baars et al. 2001) use a relative bioavailability (the bioavailability from a soil matrix with respect to the bioavailability from the matrix in toxicity studies used to assess tolerable intakes) for lead of 0.6 (60%) in the derivation of serious (human health) risk concentrations.
- UK and US agencies have developed models based on the relationship between exposure and blood lead concentrations to derive soil guideline values, where the following is noted:
    - The IEUBK model was developed in the US to describe the exposure of children to lead from multiple sources, and incorporates data on the toxicokinetics of lead – five

exposure pathways are considered (air, water, diet, soil and dust). Using the various generic default parameters, including absorption factors of 0.3 for soil and dust, and 0.5 for food and water, a soil guideline value of 400 mg/kg is derived, and is considered appropriate for use in a residential scenario.

- In contrast, the UK model considers the background exposure to lead from sources other than soil and dust, and the slope or response of the blood lead concentration versus soil and dust lead relationship.

The review by MfE (MfE 2011a) identified issues in the range of lead bioavailability/ bioaccessibility values, no agreed (in New Zealand, at that time) laboratory methods available, and uncertainties with the dose-response used for blood lead. Hence the MfE considered 100% bioavailability in the derivation of a soil guideline value.

Review of bioavailability by IARC (2006) identified a range of values and factors that have the potential to affect absorption. Based on the range of bioavailability values presented by IARC, an oral bioavailability of 50% (from soil/dust, food and water) is considered to be sufficiently conservative. Adopting a bioavailability of 50% is consistent with adopting a soil bioaccessibility value of 100% (i.e. assumes 10% of the lead in soil can move into solution and be available for absorption) and 50% absorption (the value from WHO relevant to children – noting a lower value is relevant for adults). Therefore a default 50% oral bioavailability value for children is used in the current derivation of the Australian HIL for lead (NEPC 1999 amended 2013b) – this reflects the gastrointestinal absorption, with 100% bioaccessibility from soil assumed.

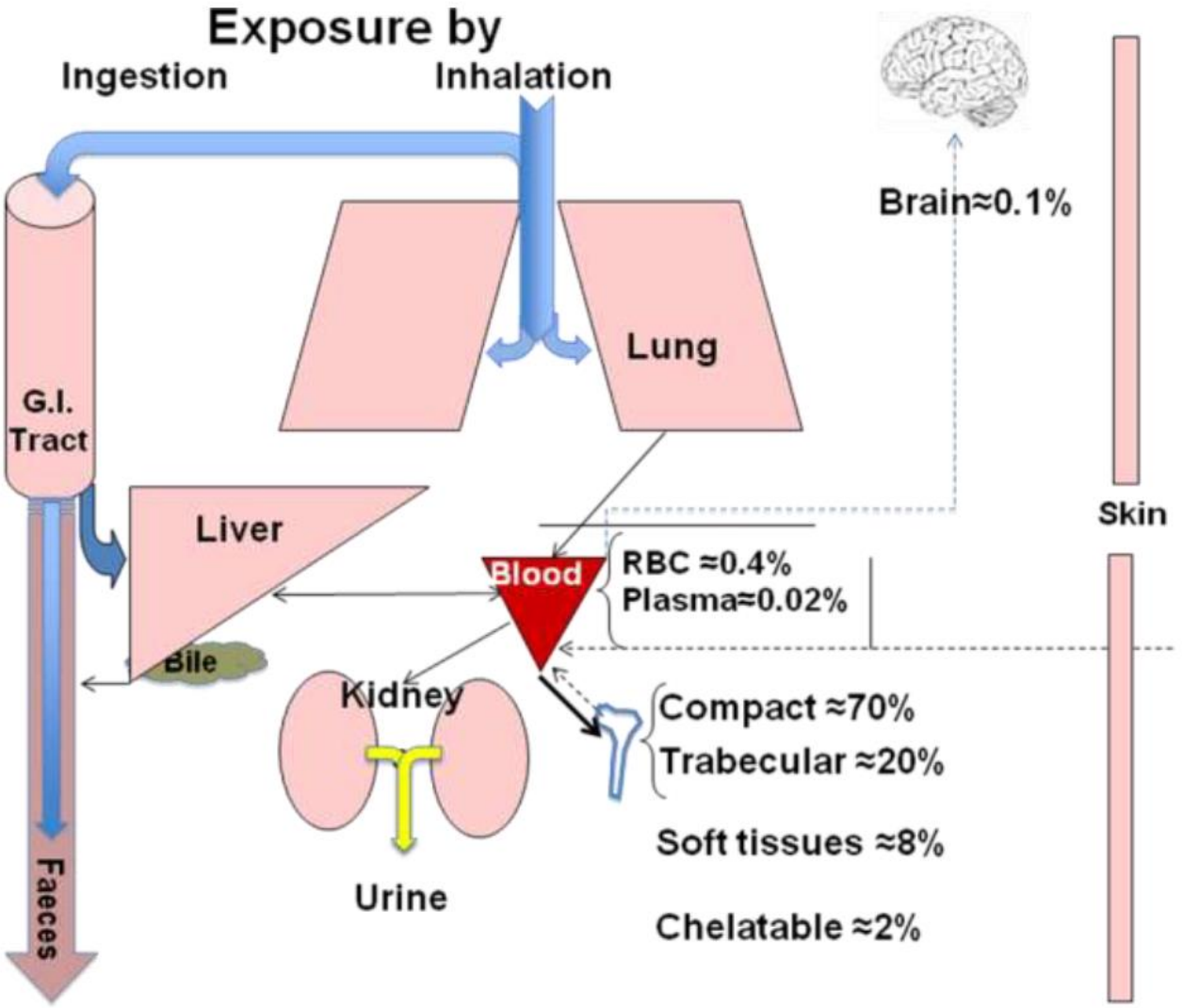
Where site specific bioaccessibility is available the bioavailability is adjusted to be 50% absorption x bioaccessible fraction.

Absorption - Dermal

Dermal absorption of inorganic lead is considered to be negligible. A review by the IARC of dermal absorption of inorganic lead studies concluded dermal absorption of inorganic lead is negligible, although slightly enhanced by high perspiration rates (IARC 2006a). This is consistent with approaches adopted in New Zealand (MfE 2011a) and the UK (UK DEFRA & EA 2002b). Organic lead is considered far more permeable to the skin and can have a role in lead exposure (ATSDR 2007a).

Distribution

Once adsorbed, lead moves between blood, soft tissues and bone within the body. However, the majority of lead in the body is found in bone. For adults 90% of lead can be found in bone, while for children it is less, at approximately 70%. Only about 1% of lead is found in the blood which is primarily (≈99%) bound to red blood cells (USEPA 2013). The following presents a schematic diagram of the distribution of lead in the body (EFSA 2010b).



Schematic: Distribution of lead in the body (EFSA 2010b)

Lead is not evenly distributed in bone. Rather it will accumulate in regions of the bone undergoing the most active calcification at the time of exposure, suggesting that lead accumulation will occur predominantly in trabecular bone during childhood, and in both cortical and trabecular bone in adulthood (ATSDR 2007a).

Some lead diffuses into deeper bone regions, where it is relatively inert, particularly in adults. These bone compartments are much more labile in infants and children than in adults as reflected by half-times for movement of lead from bone into plasma (e.g. cortical half-time = 0.23 years at birth, 3.7 years at 15 years of age, and 23 years at > 25 years; trabecular half-time = 0.23 years at birth, 2 years at 15 years of age, and 3.8 years at > 25 years) (USEPA 2013).

However, lead is not fixed to the bone and may be remobilised into blood especially during pregnancy, from health conditions such as osteoporosis, menopause, hyperparathyroidism or from severe weight loss (USEPA 2013).

Concentrations of lead in blood vary considerably with age physiological state (e.g. pregnancy, lactation, menopause) and numerous factors that affect exposure to lead (ATSDR 2007a). The excretory half-life of lead in blood, in adult humans, is approximately 30 days. Lead in blood is primarily in the red blood cells with most of the lead bound to proteins within the cell rather than the erythrocyte membrane. The primary protein the lead binds to in the cell is δ-aminolevulinic acid dehydratase (ALAD). While close to 99% bind to the red blood cells, less than 1% bind to blood plasma of which 40-75% is bound to proteins (primarily albumin) (Safe Work Australia 2014a). Thus only a small fraction of PbB (<1%) is the biologically labile and toxicologically active fraction of the circulating lead (USEPA 2013).

Bone lead has a half-life of several decades, however the labile phase, exhibited shortly after a change in exposure occurs, has a half-life of approximately 20 to 30 days.

Lead in soft tissue is predominately in the liver and kidneys, where it is assumed it predominately bound to protein. The liver and kidneys rapidly accumulate systemic lead, and in contrast to lead in bone, concentrations in soft tissues are relatively constant in adults reflecting a faster turnover of lead in soft tissue relative to bone (USEPA 2013).

Information on the distribution of organic lead in humans is extremely limited, but has been found predominately in the liver and kidneys, with the remaining distributed widely throughout the body (ATSDR 2007a).

The concentration of lead in blood reflects mainly the exposure history of the previous few months and does not necessarily reflect the larger burden and much slower elimination kinetics of lead in bone (ATSDR 2007a).

Maternal-to-foetal transfer of lead in humans, measured as the ratio of cord PbB to maternal PbB, has been found to range from 0.7 to 1.0 at the time of delivery for maternal PbB ranging from 1.7-8.6 µg/dL (US EPA 2013). The transfer appears to be partly related to the mobilisation of lead from the maternal skeleton during pregnancy. Koyashiki et al. (Koyashiki, Paoliello & Tchounwou 2010) reviewed published epidemiologic studies containing information on the excretion of lead in breast milk. They found the milk to maternal PbB ratios from 11 studies varied between 0.01 and 0.48, and concluded the available information does not indicate a health risk from breast milk exposure. One of the most recent reviews on the health effects of lead exposure (US EPA 2013) does not make a conclusion regarding exposure and health risk to children from ingesting breast milk (Safe Work Australia 2014a).

Metabolism

Metabolism of inorganic lead consists of formation of complexes with a variety of protein and nonprotein ligands. Major extracellular ligands include albumen and nonprotein sulfhydryls. The major intracellular ligand in red blood cells is ALAD. Lead also forms complexes with proteins in the cell nucleus and cytosol. Organic lead is metabolised in the liver by oxidative dealkylation catalysed by cytochrome P-450 (ATSDR 2007a).

Elimination

Lead is primarily eliminated through urine and faeces with sweat, saliva, hair, nails, and breast milk being minor routes of excretion (USEPA 2013). The half-life of lead in blood and bone is approximately 30 - 40 days and 10-30 years respectively (EFSA 2010b; USEPA 2013). Because of the relatively rapid elimination for lead from blood compared with bone, blood lead levels will mainly reflect exposures in the previous few months and not necessarily the larger body burden of lead in bone.

Mechanisms of secretory and absorptive transfer of lead in the kidney and the mechanisms by which inorganic lead is excreted in urine have not been fully characterised. Measurement of the renal clearance of ultrafilterable lead in plasma indicates that, in dogs and humans, lead undergoes glomerular filtration and net tubular reabsorption. Studies conducted in preparations of mammalian small intestine support the existence of saturable and nonsaturable pathways of lead transfer and suggest that lead can interact with transport mechanisms for calcium and iron (ATSDR 2007a).

In humans, absorbed inorganic lead is excreted in faeces. The mechanisms for faecal excretion of absorbed lead have not been elucidated; however, pathways of excretion may include secretion into the bile, gastric fluid and saliva (ATSDR 2007a).

Health Effects

There is a large amount of information available about the health effects of lead, with information and data from epidemiological studies being the major lines of evidence. The health effects of lead are the same regardless of the route of exposure (ATSDR 2019b).

Health effects associated with exposure to inorganic lead and compounds include, but are not limited to: neurological, renal, cardiovascular, haematological, immunological, reproductive, and developmental effects. Neurological effects of Pb are of greatest concern because effects are observed in infants and children and may result in life-long decrements in neurological function.

The most sensitive targets for lead toxicity are the developing nervous system in children; and effects on the haematological and cardiovascular systems, and the kidney in adults.

However, due to the multi-modes of action of lead in biological systems, lead could potentially affect any system or organs in the body. The effects of lead exposure have often been related to the blood lead content, which is generally considered to be the most accurate means of assessing exposure (MfE 2011a).

Children and pregnant women are particularly sensitive to lead exposure, and low lead exposure studies have focused on a range of health outcomes including on neurological (such as cognitive and behavioural functioning), cardiovascular and reproductive and developmental health endpoints (Armstrong et al. 2014).

The International Agency for Research on Cancer (IARC 2006) has classified inorganic lead as Group 2A: probably carcinogenic to humans. Organic lead was classified as Group 3: not classifiable (IARC 2006a). It is noted that the US EPA has classified lead and compounds as Class B2: probable human carcinogen (USEPA IRIS). While there is some evidence of carcinogenic

effects associated with exposure to lead (in experimental animals, with inadequate evidence in humans), there is evidence from human studies that adverse effects other than cancer may occur at lower lead levels (WHO 2011b). Hence the adoption of a guideline that addresses the most sensitive non-carcinogenic effects is considered to also be adequately protective of carcinogenic effects.

Blood lead levels have been found to be a good indicator of exposure to lead. A blood lead level reflects lead's dynamic equilibrium between adsorption, excretion and deposition in soft and hard tissues. Epidemiological studies (and expert groups) do not provide definitive evidence of a threshold in relation to blood lead levels and neurotoxic effects (ATSDR 2007a; Baars et al. 2001; UK DEFRA & EA 2002b; USEPA IRIS), however, blood lead goals and associated intakes have been identified by various agencies for the assessment of lead exposures by the general public. The NHMRC has noted that there are no benefits of human exposure to lead and that all demonstrated effects of exposure are adverse.

For the assessment of lead exposures in Australia, the current advice/statement from NHMRC on the evidence of health effects from lead, released in 2015 has been considered. This statement identified that the average Australian blood lead level was less than 5 micrograms per decilitre (µg/dL). Therefore, if an Australian had a blood lead level of 5 µg/dL or greater, and were not in a lead endemic area, this is a positive indicator of a non-background exposure to lead. Given that lead is not beneficial to human health, the NHMRC recommended that the non-background source be investigated and reduced (NHMRC 2015a). This recommendation follows a well-worn policy approach of reducing non-beneficial exposures to environmental pollutants, where possible, irrespective of their health impacts.

The NHMRC have acknowledged that health effects from blood lead levels greater than 10 µg/dL are well established. These effects include increased blood pressure, abnormally low haemoglobin, abnormal kidney function, long-term kidney damage and abnormal brain function. These health effects are summarised in the following figure (NHMRC 2015a).

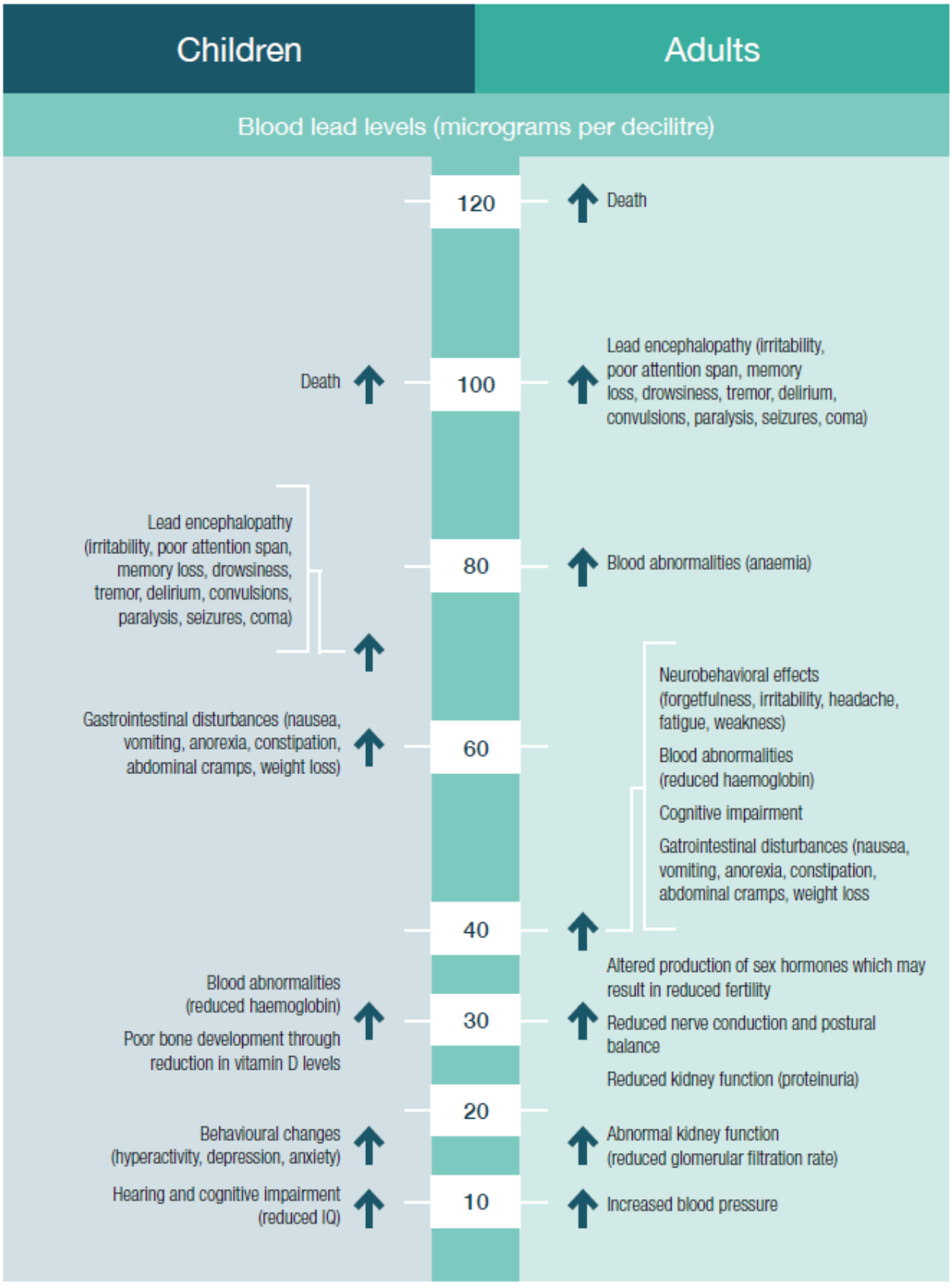


Figure: Summary of health effects of lead exposure above 10 µg/dL

However, for blood lead levels less than 10 µg/dL the evidence is less clear and must be treated with caution (Armstrong et al. 2014). This is because those studies that found a relationship (association) between blood lead levels below 10 µg/dL and health effects (such as reduced Intelligence Quotient) failed to account for other factors that may be responsible for the health effects (Armstrong et al. 2014). Further, for blood lead levels less than 10 µg/dL and cardiovascular effects it was concluded that *the clinical significance of the finding regarding increased blood pressure and increased risk of hypertension among adults and pregnant women may be minimal* (Armstrong et al. 2014). As a result, with regard to blood lead levels less than 10 µg/dL the NHMRC concluded that there is insufficient evidence that blood lead at this level caused any of the health effects observed (NHMRC 2015a).

With regard to contaminated sites, enHealth considered the NHMRC statement and confirmed the current approach for lead in the NEPM is still valid and did not requiring changing at this point in time. However, it is noted that the lack of certainty regarding possible health effects from blood lead levels below 10 µg/dL along with a lack of beneficial effects of lead is the basis for the NHMRC recommendation to reduce unnecessary exposure to lead, irrespective of its concentration.

For the purpose of any lead assessment, all unnecessary exposures to lead should be minimised, in line with NHMRC (2015). An upper concentration limit of lead, based on the protection of adverse health effect can be estimated using the IEUBK lead model as undertaken in the Contaminated sites NEPM (NEPC 1999 amended 2013b) and the blood lead criteria of 10 µg/dL, however this should not preclude the consideration of taking reasonable and feasible approaches to reduce exposures (where possible).

Approaches for the characterisation of hazards/toxicity

The assessment of the toxicity of lead may be undertaken on the basis of a threshold dose or the use of a blood lead goal, or both. The following table presents a summary of the approaches available from Australia and International agencies.

Table B10: Toxicity reference values (TRVs) and goals for lead

Source	Value	Basis/Comments
ADWG (NHMRC 2011 updated 2022)	PTDI = 0.0035 mg/kg/day	PTDI considered in the ADWG is based on the evaluation provided by JECFA and WHO DWG associated with a Provisional Tolerable Weekly Intake (PTWI) of 0.025 mg/kg/week (see comments below).
NHMRC (NHMRC 2015a)	PbB investigation level > 5 µg/dL PbB health based level > 10 µg/dL	The NHMRC evaluation in 2015 noted that it is well established that blood lead levels greater than 10 µg/dL can have harmful effects on many organs and functions. The evidence for health effects occurring as a result of blood lead levels less than 10 µg/dL is less clear. An association has been found between levels below 10 µg/dL and effects on Intelligence Quotient and academic achievement in children, behavioural problems in children, increased blood pressure in adults and a delay in sexual maturation in adolescent boys and girls. However, the evidence is insufficient to conclude lead at these levels is causal for any of these effects. Hence the revised guidance reflects that 5 µg/dL is considered representative of background and a level greater than 5 µg/dL warrants further evaluation, i.e. investigation. This advice replaces the previous blood lead goal of 10 µg/dL (NHMRC 2009). It is noted that the current NEPM HIL for lead in soil is based on the old blood lead goal of 10 µg/dL.

Source	Value	Basis/Comments
NEPM (NEPC 1998, 2016)	Air Quality Goal = 0.5 µg/m^3	Air guideline (based on an annual average) set by NEPM. Basis or the value is not stated; however, it is the same as that set by the WHO Air Quality Guidelines.
Safe Work Australia (Safe Work Australia 2014b)	Target PbB goals of 20 µg/dL Blood lead removal level 30 µg/dL	Relevant for nearly all workers, including females of non-reproductive capacity and males. For females of reproductive capacity, a lower blood lead goal is recommended, namely 10 µg/dL.
New Zealand (MfE 2002, 2011a)	PTDI = 0.0019 mg/kg/day Air GV = 0.2 µg/m^3 as 3-month average	Based on dose-response modelling by FAO/WHO (2010) that indicated this level of exposure may give rise to decreased IQ at a population level, but effects were considered insignificant at an individual level. Ambient air quality guideline is more precautionary than the value established by the WHO and consistent with the UK long term objective of 0.25 µg/m^3.
JECFA (WHO 2010b)	PTWI = 0.025 mg/kg	In 1972 the JECFA set a PTWI of 0.05 mg/kg. The current PTWI was established in 1986 for infants and children based on metabolic studies showing a mean daily intake of 3-4 µg/kg was not associated with an increase in blood lead levels or in the body burden of lead. An intake of 5 µg/kg was associated with an increase in lead retention. The PTWI was reconfirmed in 1993 and extended to all age groups. The PTWI was estimated to be responsible for a blood lead concentration of 5.6 µg/dL for a 10 kg child, which is thought to be below that associated with effects on intellectual performance. This PTWI was withdrawn by JECFA in 2010 as the committee could no longer consider the value to be health protective. The committee estimated that the previous PTWI was associated with a decrease of at least 3 intelligence quotient (IQ) points in children and an increase in systolic blood pressure of approximately 3 mmHg in adults. Both these effects were considered important within a population. The committee did not provide any indication of a suitable threshold for the key adverse effects of lead and no alternate PTWI was established.
RIVM (Baars et al. 2001)	PTWI = 0.025 mg/kg	Adopted the JECFA evaluation.
WHO DWG (WHO 2017)	No value provided	WHO has adopted a provisional guideline of 0.01 mg/L based on treatment performance and analytical achievability. The WHO evaluation notes the withdrawal of the JECFA PTWI and that no new value is available. The review notes that there does not appear to be a threshold for the key effects of lead.
WHO (WHO 2000b)	TC = 0.5 µg/m^3	Air guideline (based on an annual average) established for lead based on an objective of 98% of the general population having a blood lead concentration of < 10 µg/dL, where the median blood lead levels would be no more than 5.4 µg/dL.
EFSA (EFSA 2010b)	PbB levels relevant for critical health effects Developmental effects in children: 1.2 µg/dL Renal effects in adults: 1.5 µg/dL Cardiovascular effects in adults: 3.6 µg/dL	Based on benchmark dose response levels for 1% change in IQ or blood pressure (BMDL01) and a 10% change in prevalence of CKD (considered significant for population health effects) (BMD10). EFSA also converted the blood lead goals to an intake using blood lead modelling.
UK DEFRA (DEFRA 2014)	PbB goals of 1.6 to 5 µg/dL	Conversion of blood lead criteria to intake dose levels of lead based on the IEUBK model for children and two different adult lead models for adults, refer to further discussion below.
CDC (CDC 2012)	PbB goal of 5 µg/dL	Recommends that the PbB goal be used to identify children aged 1-5 years may have elevated blood lead levels. The level is intended to trigger education, investigation and monitoring.

The more recent reviews of lead completed by EFSA (EFSA 2010b) and the UK DEFRA (UK DEFRA & EA 2014) have focused on the critical health endpoints for adults and children, using benchmark dose (BMD) modelling methods to identify blood lead levels associated with points of departure considered to represent significant health outcomes, and the use of blood lead modelling to determine the intake (external intake of lead) that corresponds to the blood lead levels. The most detailed review of this process is presented by DEFRA (UK DEFRA & EA 2014), which is noted to be consistent with the EFSA evaluation, where the following has been further considered.

Neurobehavioral effects in children

While the NHMRC review (Armstrong et al. 2014) determined that the studies related to neurobehavioral effects in children at blood lead levels less than 10 µg/dL are subject to a number of confounders that make it difficult to clearly determine that exposure to lead caused the changes in IQ reported, the DEFRA review has considered these studies. The study by Lanphear et al (Lanphear et al. 2005) is identified as the key study, using pooled data from 7 studies on blood lead levels and IQ.

The modelling undertaken was based on a 1% response level (BMD01), which relates to a decrease of 1 IQ point would have an impact on the socioeconomic status of the population and its productivity. Evaluation of the different BMD models (logarithmic, piecewise linear and a linear model) with blood lead levels predicted in the range 1.2 to 5.6 µg/dL, which suggests some variability, with the median value of 3.7 µg/dL (rounded by DEFRA to 3.5 µg/dL) from piecewise linear and linear modelling. For this assessment it is appropriate to adopt the value of 3.5 µg/dL.

An intake of lead that corresponds to the blood lead levels outlined above were modelled by DEFRA on the basis of the IEUBK model, which is suitable for children and consistent with the blood lead modelling utilised in Australia (NEPC 1999 amended 2013b). Based on this modelling, for a blood lead level of 3.5 µg/dL an intake of **1.4 µg/kg/day** is derived for children. This is the intake adopted in this assessment for the evaluation potential health effects in children, exposed to lead.

This intake is consistent with the PTDI adopted in New Zealand, and hence the PTDI from MfE (MfE 2011a) has been adopted in this assessment.

In relation to inhalation exposures the air guideline value established in New Zealand has also been adopted and applied as an annual average as well as a 3-month average.

In relation to background intakes, data available for New Zealand indicates that for young children (the most sensitive group), background intakes are around 0.00097 mg/kg/day, which comprise 50% of the adopted toxicity reference value for oral and dermal intakes. Concentrations of lead in air are assumed to be negligible.

B4.9 Manganese

General

Several comprehensive reviews of manganese in the environment and toxicity to humans are available (ATSDR 2012c; Health Canada 2010; WHO 1999, 2004b).

Manganese (Mn) is the 12th most abundant element and comprises approximately 0.01% of the earth's crust. Manganese does not occur naturally in its elemental state and is most commonly found in mineral form as oxides, carbonate and silicates. Elemental manganese is a steel-gray coloured solid at room temperature. Manganese can exist in a relatively wide range of oxidation states from -3 to +7. The most common oxidation state of manganese is Mn(IV), the form associated with manganese dioxide (MnO_2) (ATSDR 2012c).

Manganese is used to increase stiffness, hardness and strength in a range of alloys including carbon steel, stainless steel, high temperature steel, cast iron and super-alloys. Manganese is additionally used in the manufacture of dry cell batteries, matches, fireworks, porcelain, brick colorant, glass, animal feed, and plant fertilizers. Strongly oxidising forms of manganese, such as potassium permanganate are used as a disinfectant, an anti-algal agent, a water purifying agent, for metal cleaning, tanning and as bleach (ATSDR 2012c).

Manganese is a dietary essential element that is required in several important processes including bone mineralization, energy metabolism, metabolic regulation, and the formation of glycosaminoglycans (ATSDR 2012c). As it is an essential element, adverse effects can occur as a result of deficiency as well as toxicity associated with excess intake from contamination.

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. 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 (WHO 2017).

Background

Review of current information indicates the following:

- Review of manganese by FSANZ indicates that for young children aged 2-3 years, intakes range from a mean of 0.19 mg/kg/day to a 90th percentile of 0.26 mg/kg/day. Dietary intakes of manganese reported by the WHO are approximately 0.06 mg/kg/day for young children. Estimates provided by ATSDR suggest that adult intakes of food are 3.8 mg/day (or 0.05 mg/kg/day) (ATSDR 2012c; FSANZ 2011; Lindon & Sabordo 1996).
- Typical concentrations of manganese reported in the ADWG are less than 0.01 mg/L, resulting in an intake (1 L/day and body weight of 15.5 kg) by toddlers of 0.00076 mg/kg/day(NHMRC 2011 updated 2022).

- Based on the above background intakes for young children, it has been assumed that background oral intakes comprise 50% of the recommended oral TRV.
- Manganese was reported in ambient air data collected in NSW where concentrations (24-hour averages) in urban, regional and industrial areas assessed ranged from 3.7 to 119 ng/m^3 (average of 18 ng/m^3) (NSW DEC 2003). Typical concentrations in air have been reported by ATSDR to be 23 ng/m^3, consistent with that reported by NSW DEC (2003) (ATSDR 2012c). These background concentrations comprise (based on average concentrations) approximately 15% of the recommended inhalation TRV. A conservative background of 20% of the inhalation TRV could be assumed for intakes from air.

Classification

The International Agency for Research on Cancer (IARC) has not classified manganese. The USEPA has classified manganese as Group D: no classifiable.

Review of Available Values/Information

Insufficient data are available to assess whether manganese is carcinogenic to humans. Some *in vitro* and *in vivo* assays are available for manganese, with studies providing conflicting results. Overall review of the data shows that some chemical forms of manganese have mutagenic potential, however, most results are inconsistent and hence no overall conclusion as to the genotoxic potential associated with exposure to manganese can be determined (ATSDR 2012c). On this basis, a threshold approach is considered appropriate based on the most sensitive effect associated with manganese exposure (CNS effects).

The following threshold values are available for manganese:

Table B11: Toxicity reference values for manganese

Source	Value	Basis/Comments
ADWG (NHMRC 2011 updated 2022)	Safe level of 10 mg/day	The ADWG (NHMRC 2011) derived a health based guideline of 0.5 mg/L based on a level of 10 mg/day which is the amount of manganese that can be safely consumed from all sources, referenced from WHO 1973 evaluation.
WHO DWG (WHO 2017)	TDI = 0.05 mg/kg/day	The current WHO DWG (2017) has not established a guideline for drinking water as the compound is not considered to be of health concern at the levels found in drinking water. The review notes that a health-based guideline of 0.4 mg/L can be derived based on the upper range value of manganese intake of 11 mg/day from dietary studies (IOM 2001) and an uncertainty factor of 3 (to allow for the increased bioavailability of manganese from water), which results in a TDI of 0.05 mg/kg/day for 70kg adult. The guidance also notes that the presence of manganese in drinking water will be objectionable (water discolouration) above 0.05 mg/L.
WHO (WHO 1999)	TC = 0.00015 mg/m^3	Tolerable concentration or guideline value derived by WHO on the basis of the same study considered by the USEPA (IRIS 2012) and ATSDR (2012), with the guideline value derived on the basis of a NOAEL of 0.03 mg/m^3 for neurotoxicological effects from a benchmark dose (BMD) analysis, adjustment for continuous exposure (5/7 x 8/24) and an uncertainty factor of 50. The value derived is similar to that from ATSDR (2012) with the main difference being the application of the BMD model. No oral guideline value was provided.
EFSA (EFSA Panel on Dietetic	Adequate intakes ranging from 0.5 to 3 mg/day	Adequate intake based on observed mean intakes, noting insufficient data is available to enable these levels to be established on the basis of health protection.

Source	Value	Basis/Comments
Products & Allergies 2013)		
TCEQ (TCEQ 2017b)	Acute ReV = 0.0091 mg/m^3 Chronic ReV = 0.00084 mg/m^3	Acute value is based on inflammatory airway changes in monkeys, and use of a 360 fold uncertainty factor Chronic value is based on neurological effects in an occupational study and use of a 60 fold uncertainty factor. The value derived is similar to the WHO (WHO 1999) TC.
Health Canada (Health Canada 2010)	RfC = 0.00005 mg/m^3	RfC derived based on most sensitive benchmark dose analysis associated with neurotoxicological effects in an occupational inhalation study. A range of RfCs were derived that varied from 0.00005 to 0.00014 mg/m^3. The range derived is consistent with values derived from ATSDR and WHO.
ATSDR (ATSDR 2012c)	Interim oral value of 0.16 mg/kd/day Inhalation MRL = 0.0003 mg/m^3	No oral MRLs have been derived by ATSDR; however, they provide an interim guidance value of 0.16 mg/kg/day based on a tolerable upper intake level of 11 mg/day. Chronic inhalation MRL derived on the basis of a benchmark concentration (at the lower 95% confidence limit for the level of manganese exposure expected to result in 10% response rate) $BMCL_{10}$ (adjusted for continuous exposure) of 0.03 mg/m^3 associated with neurobehavioural effects in an occupational study and an uncertainty factor of 100.
USEPA (USEPA IRIS)	RfD = 0.14 mg/kg/day RfC = 0.00005 mg/m^3	RfD (last reviewed in 1993) based on a NOAEL of 0.14 mg/kg/day associated with CNS effects in a number of dietary human studies and an uncertainty factor of 1. The USEPA also note that individual requirements for and effects associated with manganese exposure may be highly variable and that some individuals may consume more than 10 mg/day of manganese without any cause for concern. RfC (last reviewed in 1993) based on the same study considered by ATSDR (2012) however the USEPA considered the LOAEL (HEC) of 0.05 mg/m^3 and applied an uncertainty factor of 1000.

As manganese toxicity via inhalation has been shown to be more significant than via oral intakes, it is reasonable that quantitative values for inhalation exposures are significantly lower than for oral exposures. Based on the available data an oral threshold value of 0.16 mg/kg/day as derived by ATSDR (2012) in the most recent detailed review of manganese toxicity. It is noted that the basis for the value is consistent with the upper range of manganese intake considered by the USEPA, NHMRC and WHO (NHMRC 2011 updated 2022; USEPA IRIS; WHO 2017) (especially if the additional uncertainty factor of 3 used in the WHO drinking water guidelines is not included for exposures from soil (based on increased bioavailability from water)).

The quantitative values available for the assessment of inhalation exposures are all essentially based on the same critical study (with the exception of Health Canada) with the main difference being the approach used to quantify a threshold value from the study data (using different benchmark dose models, not using a benchmark dose model), and consideration of uncertainty factors. The air guideline value derived by the WHO (1999) is recommended based on the use of a benchmark dose analysis which is also within the range of threshold values derived by Health Canada (2010) using a number of benchmark dose approaches using a different study. The value is also similar to that derived by ATSDR (2012).

Recommendation

On the basis of the discussion above the following toxicity reference values (TRVs) have been adopted for manganese:

- Oral TRV (TRV_O) = 0.16 mg/kg/day (ATSDR 2012c), where background intakes are 50% of the TRV
- Inhalation TRV (TRV_I) = 0.00015 mg/m^3 (WHO 1999), where background intakes are 20% of the TRV.

B4.10 Mercury (elemental and inorganic)

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 1999c; JECFA 2011; UK EA 2009f; USEPA 1997a; WHO 1991b, 2000f, 2003). The following provides a summary of the key aspects of mercury.

Mercury is a heavy metal which exists in three oxidation states: 0 (elemental), +1 (mercurous) and +2 (mercuric). As well as the common mercurous and mercuric inorganic salts, mercury can also bind covalently to at least one carbon atom. Thus the most commonly encountered exposures associated with mercury are with elemental mercury, inorganic mercuric compounds and methyl mercury.

Mercury occurs naturally as a mineral is widely distributed by natural and anthropogenic processes. The most significant natural source of atmospheric mercury is the degassing of the Earth's crust and oceans and emissions from volcanoes. Man-made sources such as mining, fossil fuel combustion and industrial emissions generally contribute less on a global scale, but more on a local scale. Wet and dry deposition to land and surface water result in mercury sorption to soil and sediments.

Uses of mercury include use in the electrical and chlor-alkali industry (lamps, batteries and as cathodes in the electrolysis of sodium chloride to produce caustic soda and chloride), industrial and domestic instruments, laboratory and medical instruments and dental amalgam (mixed in proportion of 1:1 with a silver-tin alloy).

Mercury in the environment, including groundwater, exhibits complex behaviour that affects both its mobility and potential toxicity. Mercury has a low solubility in water; however, it also has the potential to form multiple species in the environment, which can lead to increased total mercury concentrations in aqueous systems. The relative toxicity of mercury is also dependent on the form in which it occurs, which, is dependent on: biogeochemical processes, partitioning between solids, and complexation with dissolved organic and inorganic ligands.

On the basis of the potential for long-range transport, persistence in water, soil and sediment, bioaccumulation, toxicity and ecotoxicity, mercury is considered persistent and is addressed in the 1998 UN-ECE Convention on Long-Range Transboundary Air Pollution on Heavy Metals (UNECE 1998). The United Nations Environment Programme (UNEP) Governing Council concluded, at its 22nd session in February 2003, after considering the key findings of the Global Mercury

Assessment report, that there is sufficient evidence of significant global adverse impacts from mercury to warrant further international action to reduce the risks to humans and wildlife from the release of mercury to the environment.

Potential for exposure

Ingestion of soil and dust is considered the most significant pathway of exposure for inorganics in soil. The consideration of bioavailability and other exposure pathways has been further reviewed as noted below:

Oral Bioavailability

The bioavailability of different forms of mercury, by different routes of exposure, are expected to vary considerably (Imray & Neville 1996) with oral bioavailabilities reported in the range 2% – 15% for inorganic mercury and 80% to 100% for methyl mercury. Insufficient data are available to adequately define the bioavailability of the different forms of mercury from soil. On this basis a default approach of assuming 100% oral (and inhalation) bioavailability has been adopted. It is noted that site-specific assessment of bioavailability can be considered where required.

Dermal absorption:

Review of dermal absorption by MfE (MfE 2011a) has noted that "*Mercury reacts with skin proteins, and as a result penetration does not increase commensurably with increasing exposure concentration but rather approaches a plateau value. Mercury has a permeability coefficient in the order of 10^{-5} cm/h (Guy et al., 1999), which compares to permeability coefficients in the order of 10^{-4} cm/h for lead*."

ATSDR (ATSDR 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 2009f) 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 2011b) has noted that "*Mercury reacts with skin proteins, and as a result penetration does not increase commensurably with increasing exposure concentration but rather approaches a plateau value. Mercury has a permeability coefficient in the order of 10^{-5} cm/h (Guy et al., 1999), which compares to permeability coefficients in the order of 10^{-4} cm/h for lead*." ATSDR (1999) note that absorption of mercurous salts in animals can occur through

the skin, however no quantitative data are available, hence a default value of 0.1% has been adopted based on the lower end of the range for metals (USEPA 1995).

The USEPA (USEPA 2004) has recommended the use of a gastrointestinal absorption factor (GAF) of 7% for inorganic mercury based on mercuric chloride and other soluble mercury salt studies used in the derivation of the oral RfD. The GAF is used to modify the oral toxicity reference value to a dermal value in accordance with the USEPA (2004) guidance provided.

Inorganic mercury compounds are rapidly distributed to all tissues following absorption. The fraction that crosses the blood-brain and foetal barriers is less than for elemental mercury due to poor lipid solubility. The major site of systemic deposition of inorganic mercury is the kidney. Most inorganic mercury is excreted in the urine or faeces.

Acute exposure to high concentrations of ingestion of inorganic mercury has been associated with gastrointestinal damage, cardiovascular damage, acute renal failure and shock.

The kidney is the critical organ associated with chronic exposure to inorganic mercury compounds. The mechanism for the end toxic effect on the kidney, namely autoimmune glomerulonephritis, is the same for inorganic mercury compounds and elemental mercury and results in a condition sometimes known as nephrotic syndrome.

There is some evidence that inorganic mercury may cause neurological effects, particularly associated with studies of mercuric chloride. Reproductive and developmental effects have been observed in rats given mercuric chloride.

Plant Uptake:

A detailed review of the plant uptake of mercury (primarily inorganic mercury) is presented by The UK (UK EA 2009f). 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 2009f)
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 2009f) 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 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 2009f), 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.

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:

Table B12: Toxicity reference values for inorganic and elemental mercury

Source	Value	Basis/Comments
Inorganic mercury		
ADWG (NHMRC 2011 updated 2022)	Guideline established on the basis of methyl mercury	
FSANZ (FSANZ 2003)	PTWI = 0.003 mg/kg/week	Value for total mercury referenced from JECFA 1989, based on methyl mercury.
New Zealand (MfE 2011a)	**TDI = 0.002 mg/kg/day**	MfE adopted the TDI from the RIVM evaluation (noted below), which is also consistent with 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.
JECFA (JECFA 2011)	PTWI = 0.004 mg/kg	Review of mercury by JECFA indicated that the predominant form of mercury indoors, other than fish and shellfish, is inorganic mercury and

Source	Value	Basis/Comments
	(equivalent to PTDI = 0.0006 mg/kg/day)	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 2009f)	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 2009f)	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.

In relation to oral exposures most agencies/reviews, including the current WHO Guidelines for Drinking Water Quality WHO 2017) have adopted a TDI of around 0.002 mg/kg/day. This is the value adopted by MfE (MfE 2011a) and has been adopted in this assessment.

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.

For the assessment of exposure to elemental mercury the value from WHO, ATSDR and USEPA (ATSDR 1999; WHO 2003) which is similar to the value available from MfE (MfE 2002) has been adopted.

TRVs adopted

On the basis of the discussion above the following toxicity reference values (TRVs) have been adopted for inorganic and elemental mercury:

- Oral TRV (TRV_O) = 0.002 mg/kg/day (MfE 2011a) for oral and dermal routes of exposure, where background intakes are assumed to be 5% of the TRV (based on data from New Zealand)
- 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) where background intakes are considered negligible

B4.11 Nickel

Several comprehensive reviews of nickel in the environment and toxicity to humans are available (ATSDR 2005a; UK EA 2009d; WHO 1991a).

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 1991a).

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.

Health effects

The following is noted in relation to the toxicity of nickel (UK EA 2009d):

> *"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."

Background

The available information in relation to background intakes of nickel are as follows:

- 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 2009d) 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 2009d) 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.

Classification

IARC (IARC 2012b) 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 US EPA 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 the (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 2009d)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:

Table B13: Toxicity reference values for nickel – Oral

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 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 2009d))	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	

Source	Value	Basis/Comments
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 2009d) 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 2017a) and nickel refinery dusts (USEPA).
- The WHO (WHO 1991a) 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 2009d) 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 2009d), 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:

Table B14: Toxicity reference values for nickel – Inhalation

Source	Value	Basis/Comments
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 2017a)	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 2009d)	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 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.

Identified TRVs

With respect to oral exposures, the more recent review by the 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 the UK (UK EA 2009d) be adopted (which is protective of adverse health effects including carcinogenicity at an excess lifetime cancer risk level of 1 in 100 000) and is similar to the more recent review from TCEQ (TCEQ 2017a).

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 2017a)
- Background intakes from other sources (as % of TRV) = 60% for oral and dermal intakes and 10% for inhalation intakes.

B4.12 Thallium

Information relevant to the assessment of thallium is available from some evaluations (ATSDR 1992b; CCME 1999a; Janssen et al. 1998; USEPA 2009a; WHO 1996).

Metallic thallium (Tl) is bluish white or grey, very soft, malleable, and insoluble in water. Many of the thallium salts are soluble in water with the exception of thallium (III) oxide, which is insoluble (USEPA 2009a).

Thallium is a Group IIIA metal, one whose salts do not hydrolyze at pH ≥ 7 to form insoluble hydroxides. In addition, monovalent thallium is similar to potassium (K_+) in ionic radius and electrical charge. Both these properties contribute to the toxicity of this element (USEPA 2009a).

Thallium is a naturally occurring trace element that is widely distributed in the earth's crust, with a crustal abundance of approximately 1 mg/kg. In soil, thallium concentrations are on the order of 0.1 to 1 mg/kg; higher concentrations occur in the vicinity of metallic ore deposits (USEPA 2009a). Thallium and thallium salts are currently used in the semiconductor and electronic industries, as additives in fireworks, and in the manufacturing of imitation gems, optic lenses, thermometers, and machinery parts operating at sub-zero temperatures.

Due to its ability to remove hair, thallium (I) sulfate was used in the past as a depilatory agent. Thallium (I) sulfate was once used in medicine to treat infections, such as venereal diseases, ringworm of the scalp, typhus, tuberculosis, and malaria. It was also used in the past as a pesticide for various rodents and insects but has been banned for this use in the U.S. since 1972 (USEPA 2009a).

Thallium compounds are readily absorbed through various routes of exposure, and rapidly distributed throughout the body. The highest concentrations are typically in the kidney and thallium can cross the placenta (USEPA 2009a).

Data relating to existing intakes or exposures to thallium are limited. RIVM estimated background intakes around 0.03 µg/kg/day (Janssen et al. 1998). ATSDR indicates intakes for the general population are expected to be dominated by dietary intakes with intakes estimated to be 0.071 µg/kg/day. The UK diet survey included thallium where mean intakes were estimated to be 0.01 for adults µg/kg/day as referenced by (Pearson & Ashmore 2020)). In New Zealand a dietary survey (Pearson & Ashmore 2020) only detected the presence of thallium in 16% of the foods sampled, with intakes estimated to range from 0.02 to 0.06 µg/kg/day for adults similar to that reported by the UK and RIVM. These intakes are around 10% of the adopted toxicity reference value.

Thallium does not have a known biological function in humans and is considered one of the most toxic heavy metals. The mechanism for toxicity remains poorly understood as thallium interacts with cells at different levels. Thallium can mimic the potassium ion vital for biological pathways because it has the same ionic radius and electrical charge and there is an inability of the cell membrane to differentiate between the two cations. The thallium ion has a ten-fold higher affinity for Na^+/K^+ ATPase than the potassium ion. Other possible mechanisms include: the ability of thallium to react with thiol groups and inhibit a range of enzyme reactions, interfering with key metabolic processes, disrupting cell equilibrium resulting in poisoning; the ability of thallium to bind to membrane phospholipids and change membrane characteristics; triggering oxidative stress; and disturbing the mitochondrial function (Cvjetko, Cvjetko & Pavlica 2010; USEPA 2009a).

Exposure to thallium salts is known to cause a wide spectrum of adverse effects in humans and animals, and thallium is considered a cumulative poison (USEPA 2009a). Alopecia is an effect that is characteristic of thallium exposure. Alopecia generally occurs within 2 weeks of exposure and is reversible when thallium exposure is removed. Acute thallium poisoning is usually accompanied by gastrointestinal symptoms, while neurological findings (sensory and motor changes) predominate and are the primary target in chronic exposure. Other symptoms include polyneuritis, encephalopathy, tachycardia and degenerative changes of the heart, liver and kidneys (Cvjetko, Cvjetko & Pavlica 2010). Low birth weight is a likely adverse effect of thallium exposure (USEPA 2009a).

The USEPA has indicated there is inadequate information to assess the carcinogenic potential for thallium and thallium compounds and IARC has not evaluated these compounds. The few studies available have not identified carcinogenicity and the data on genotoxicity is inconsistent, but cannot be excluded (Janssen et al. 1998; USEPA 2009a).

On the basis of the above a threshold approach is considered appropriate for the assessment of potential health effects in relation to thallium. There are few quantitative evaluations available for tin, which are summarised in the following table.

Table B15: Toxicity reference values for thallium

Source	Value	Basis/Comment
WHO (WHO 1996)	No TDI established	Guidance provided on occupational exposures with thallium in urine considered a reliable indicator of exposure. Adverse effects unlikely where urinary concentrations are less than 5 µg /L (from epidemiological studies in Germany). Golder (Golder 2012) utilised this value to derive a tolerable intake of 0.16 µg/kg/day.
RIVM (Janssen et al. 1998)	pTDI = 0.2 µg/kg/day	Provisional TDI derived on the basis of a NOAEL from a rat study (same study used by the USEPA) and 1000 fold uncertainty factor
Canada (CCME 1999a)	Provisional RfD = 0.07 µg/kg/day	Noted to be a previous RfD established by the USEPA based on the lowest NOAEL in the available literature (Stoltz et al (1996)) and applying an uncertainty factor of 3000.
USEPA (as referenced by (ATSDR 1992b))	Former RfDs range from 0.07 to 0.09 µg/kg/day	Range of former RfD's from the USEPA for different thallium compounds. Derivation not provided and these values have been withdrawn.
USEPA (USEPA 2009a)	Potential RfD = 0.003 µg/kg/day	Potential or candidate RfD based on clinical observations for soluble thallium salts and inclusion of 3000 fold uncertainty factor. Another candidate RfD was established at 0.01 µg/kg/day based on hair follicle atrophy. The database is noted to be poor and hence no final RfD is determined. The USEPA considers the available data inadequate to establish a RfD for thallium III oxide or thallium I selenite
USEPA (USEPA 2012a)	PPRTVs: Screening pRfD = 0.01 µg/kg/day for soluble thallium (also thallium I acetate, thallium I chloride and thallium I nitrate) Screening pRfD = 0.02 µg/kg/day for thallium I sulfate (also thallium I carbonate)	No PPRTVs recommended, however screening level values have been determined to assist in reviewing potential risks associated with thallium. The screening values are based on limited data and utilise the same studies as all the above evaluations, applying a 3000 fold uncertainty factor.

All the oral toxicity reference values listed above are based on the same source studies (originally reported in Stoltz et al., 1986 and updated as MRI, 1988) which provide a NOAEL of 0.2 mg/day. The differences in the values listed above principally relates to the uncertainty factors adopted. The RIVM value of 0.2 µg/kg/day is consistent with the tolerable intake derived from a urinary concentration established to be protective of adverse health effects (from an epidemiological study). On this basis the RIVM pTDI has been adopted as the toxicity reference value for the assessment of all exposures. This is the same value recommended in the review completed by Pearson et al (Pearson & Ashmore 2020).

Based on the available data background intakes of thallium may be up to 10% of the TRV adopted.

Recommendation

On the basis of the discussion above the following toxicity reference values (TRVs) have been adopted for thallium:

- Oral TRV (TRV_O) = 0.2 µg/kg/day (Janssen et al. 1998) for all exposures
- Background intakes from other sources (as % of TRV) = 10%.

B4.13 Vanadium

Information relevant to the assessment of vanadium is available from a range of evaluations (ATSDR 2012a; CCME 1999b; OEHHA; WHO 2001a).

Vanadium (CAS No. 7440-62-2) is a soft silvery grey metal that can exist in a number of different oxidation states. The most common commercial form is vanadium pentoxide. Vanadium is an abundant element with a very wide distribution and is mined in South Africa, Russia, and China (WHO 2001a). The environmental chemistry of vanadium is complex as the oxidation state may vary.

Following inhalation vanadium is absorbed, however absorption many be low following ingestion (depending on the form present). Once absorbed, vanadium is distributed throughout the body and distributed to the body tissues, with the principal organs of vanadium retention being the kidneys, liver, testicles, spleen and bones (WHO 2000c). Vanadium can cross the blood-placenta barrier.

Inhalation exposures may result in effects on the respiratory system (which is the most significant health effect). Oral exposures may result in gastrointestinal effects, haematological effects and reproductive effects. Other effects include systemic effects on the liver, kidneys, gonads and the nervous, haematological and cardiovascular systems (ATSDR 2012a; WHO 2000c).

Vanadium is a potent inhibitor of many enzymes, while it stimulates adenylate cyclase. It has been shown to inhibit cholesterol biosynthesis and lower plasma cholesterol levels. Vanadium can also directly influence glucose metabolism *in vitro* and may play a role in its regulation.

IARS has classified vanadium pentoxide as Group 2B, possibly carcinogenic to humans based on sufficient evidence in animals but inadequate evidence in humans. The IARC review identified that vanadium pentoxide was mutagenic *in vitro* and possibly *in vivo* in mice (IARC 2006b). Review by RIVM (Tiesjema & Baars 2009) suggests the presence of a threshold for the observed DNA damaging activity. Vanadium (other than vanadium pentoxide) has not been classified in relation to carcinogenicity. The relevance of the carcinogenic assessments for vanadium pentoxide for other vanadium compounds is not clear.

On the basis of the above a threshold approach is considered appropriate for the assessment of potential health effects in relation to vanadium. The following table provides a summary of the toxicity reference values available.

Table B16: Toxicity reference values for vanadium

Source	Value	Basis/Comment
WHO (WHO 2000c)	Air guideline = 0.001 mg/m^3 as 24 hour average	Air guideline based on a LOAEL of 0.02 mg/m^3 for chronic upper respiratory effects and a protection factor of 20 to address a sensitive subpopulation.
RIVM (Tiesjema & Baars 2009)	pTDI = 0.002 mg/kg/day pTC = 0.001 mg/m^3	Provisional oral TDI based on a reproduction study with rats (applicable to vanadium compounds) and uncertainty factor of 1000. Provisional tolerable concentration in air based on vanadium pentoxide studies in rats and mice and a 1000 fold uncertainty factor.
ATSDR (ATSDR 2012a)	Acute inhalation MRL = 0.0008 mg/m^3 Chronic inhalation MRL = 0.0001 mg/m^3	Acute inhalation MRL based on protection of respiratory effects in an occupational study with vanadium pentoxide. The acute value has relates to exposures up to 14 days. Chronic inhalation MRL based on protection of respiratory effects from two year rat and mouse studies with vanadium pentoxide dust.

Source	Value	Basis/Comment
OEHHA (OEHHA)	Acute REL = 0.03 mg/m^3	Acute REL based on protection of respiratory effects in healthy human volunteers for vanadium pentoxide
USEPA (USEPA IRIS)	RfD = 0.009 mg/kg/day	Oral RfD (last evaluated in 1998) is for vanadium pentoxide and based on changes in cystine level in the hair of rate exposed via the diet and a 100 fold uncertainty factor. Confidence in the value is considered low.
USEPA (USEPA 2009b)	pRfD = 0.00007 mg/kg/day	Provisional peer-reviewed toxicity value for soluble vanadium compounds (excluding vanadium pentoxide), calculated based on a LOAEL for kidney toxicity in a 6-month drinking water study with rats and a 3000 fold uncertainty factor. Confidence in the value is considered low.

There are limited quantitative toxicity reference values available for vanadium, with most of the data available for vanadium pentoxide. Based on the limited data available the oral TRV from RIVM (Tiesjema & Baars 2009) has been adopted, as this similar to the current USEPA evaluation and relevant to vanadium compounds. For the assessment of chronic inhalation exposures the lower air guideline value from ATSDR (ATSDR 2012a) has been adopted.

Background intakes of vanadium are less well known. RIVM indicates background intakes may be around 10% of the pTDI, however the data for this value is not available. FSANZ does not provide any data specific to the Australian dietary intakes. The WHO indicates dietary intakes for the general population of around 0.011 to 0.03 mg/day (0.00016 to 0.0004 mg/kg/day), which is negligible when compared with the TRV adopted.

Recommendation

On the basis of the discussion above the following toxicity reference values (TRVs) have been adopted for vanadium:

- Oral TRV (TRV_O) = 0.002 mg/kg/day (Tiesjema & Baars 2009) for oral and dermal exposures
- Inhalation TRV = 0.0001 mg/m^3 (ATSDR 2012a)
- Background intakes from other sources (as % of TRV) = negligible.

B4.14 Selenium

Information relevant to the assessment of selenium is available from a range of evaluations (ATSDR 2003; CCME 2009; NHMRC 2011 updated 2022; WHO 2017).

Selenium is a naturally occurring, solid substance that is widely but unevenly distributed in the earth's crust. It is also commonly found in rocks and soil. Selenium, in its pure form of metallic grey to black crystals, is often referred to as elemental selenium or selenium dust. Elemental selenium is commercially produced, primarily as a by-product of copper refining. Selenium is not often found in the environment in its elemental form, but is usually combined with other substances (ATSDR 2003).

Selenium and its compounds are used in some photographic devices, gun bluing (a liquid solution used to clean the metal parts of a gun), plastics, paints, anti-dandruff shampoos, vitamin and mineral supplements, fungicides, and certain types of glass (ATSDR 2003).

In Australia and New Zealand, the main dietary sources are seafood, poultry and eggs and, to a lesser extent, other muscle meats. The contribution of cereal products depends on the source (NHMRC 2006). Average daily intakes for Australian adults are between 0.06 mg and 0.13 mg (NHMRC 2011 updated 2022). Data available from FSANZ (FSANZ 2011) indicates intakes of selenium in Australian diets range from 0.038 mg/day for infants to 0.086 mg/day for children aged 2-3 years and 0.13 mg/day for adults. In New Zealand (Ministry for Primary Industries 2018) the estimated intake of selenium in the diet was similar, at 0.042 mg/day for children and 0.068 mg/day for adults. The total diet surveys in Australian and New Zealand include consumption of water.

Selenium is an essential element for many species, including humans, hence health effects may occur as a result of deficiency as well as toxicity. Signs of selenium deficiency in humans are not well established but may include a chronic disorder of the heart muscle, other heart diseases and cancer (NHMRC 2011 updated 2022).

Most water-soluble selenium compounds are effectively absorbed by the gastrointestinal tract. Selenium is then distributed to most organs, with highest concentrations found in the kidney, liver and spleen (NHMRC 2011 updated 2022).

The toxicity of selenium varies considerably among the different selenium compounds. Selenite and selenate are much more toxic than selenium sulfide (NHMRC 2011 updated 2022).

Adverse health effects associated with exposure to elevated levels of selenium have been identified from occupational or accidental poisoning as well as animal studies. Inhalation exposures (at high levels) in occupational settings have resulted in dizziness, fatigue, and irritation of mucous membranes.

Chronic intakes of elevated levels of selenium can result in brittle hair and deformed nails. In extreme cases, people may lose feeling and control in arms and legs (CNS effects). These health effects, called selenosis, were seen in several villages in China where people were exposed to foods high in selenium for months to years (ATSDR 2003).

Other features of excess selenium intake include nonspecific symptoms such as gastrointestinal disturbances, dermatitis, dizziness, lassitude and a garlic odour to the breath (NHMRC 2011 updated 2022). Fertility was found to be reduced in animal studies but only at levels high enough to be toxic.

The International Agency for Research on Cancer has concluded that selenium is not classifiable as to its carcinogenicity in humans (Group 3, inadequate evidence in humans and in animals). The USEPA also concluded selenium was not classifiable in relation to carcinogenicity.

On the basis of the above a threshold approach is considered appropriate for the assessment of potential health effects in relation to selenium. The following table provides a summary of the toxicity reference values available.

Table B17: Toxicity reference values for selenium

Source	Value	Basis/Comment
ADWG (NHMRC 2011 updated 2022)	ADI = 0.24 mg/day = 0.0034 mg/kg/day	ADI based on an upper level for selenium in the diet and use of a 70 km body weight.
NHMRC (NHMRC 2006)	Upper level of intake = 0.4 mg/day for adults = 0.006 mg/kg/day	Upper level based on the protection of adverse effects from selenium intakes. For young children the upper limit results in a value of 0.007 mg/kg/day, which is slightly higher than the value calculated for adults (adopting a 70 kg body weight)
WHO (WHO 2017)	TDI = 0.4 mg/day = 0.006 mg/kg/day	Value is an upper tolerable intake established by FAO/WHO.
RIVM (Janssen et al. 1998)	TDI = 0.005 mg/kg/day	Value based on the same study and approach as USEPA and ATSDR.
ATSDR (ATSDR 2003)	Chronic MRL = 0.005 mg/kg/day	MRL based on a NOAEL of 0.015 mg/kg/day for the disappearance of symptoms of selenosis in recovering individuals and an uncertainty factor of 3.
OEHHA (OEHHA)	Chronic REL = 0.02 mg/m^3 Oral REL = 0.005 mg/kg/day	Chronic values are based on protection of liver, cardiovascular and nervous system effects. The inhalation value is consistent with utilising the oral value and route extrapolation.
USEPA (USEPA IRIS)	RfD = 0.005 mg/kg/day	Value based on the approach as ATSDR

Based on the above the upper level of intake of 0.4 mg/day, which is equivalent to 0.006 mg/kg/day, has been adopted for the assessment of all exposures to selenium. It is noted that the one inhalation value (from OEHHA) is the same as using the oral value and route extrapolation (oral to inhalation).

Background intakes from dietary sources (which include water) may contribute up to 25% of the TRV adopted.

Recommendation

On the basis of the discussion above the following toxicity reference values (TRVs) have been adopted for selenium:

- Oral TRV (TRV_O) = 0.005 mg/kg/day (NHMRC 2006) for all routes of exposure
- Background intakes from other sources (as % of TRV) = 25%.

B4.15 Tin

Information relevant to the assessment of tin is available from some evaluations (ATSDR 2005b; IPCS; Tiesjema & Baars 2009).

Tin is a soft, white, silvery metal that is insoluble in water. Tin metal is used to line cans for food, beverages, and aerosols. It is present in brass, bronze, pewter, and some soldering materials. Approximately 50% of the world production of tin is used for plating. Tin is a metal that can combine with other chemicals to form various compounds. When tin is combined with chlorine, sulfur, or oxygen, it is called an inorganic tin compound. Inorganic tin compounds are found in small amounts in the earth's crust. They are also present in toothpaste, perfumes, soaps, colouring agents, food additives, and dyes. Tin also can combine with carbon to form organotin

compounds. These compounds are used in making plastics, food packages, plastic pipes, pesticides, paints, wood preservatives, and rodent (rats and mice) repellents (ATSDR 2005b).

Tin is present in the environment and is also present in the tissues of the body. There is no evidence that tin is an essential element.

The main route of exposure to tin is via food, in particular canned food. Once ingested, the gastrointestinal absorption of tin is low (with around 5% absorbed). Once absorbed tin is widely distributed in the body depositing in bone, kidney and liver (Tiesjema & Baars 2009).

There is limited data on the acute effects of tin exposure however gastrointestinal effects have been reported following ingestion of canned foods with a high tin content.

Chronic 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 organotin compounds has the potential to result in skin and eye irritation, respiratory irritation, gastrointestinal effects, and neurological problems (ATSDR 2005b).

On the basis of the above a threshold approach is considered appropriate for the assessment of potential health effects in relation to tin. There are few quantitative evaluations available for tin, which are summarised in the following table.

Table B18: Toxicity reference values for tin

Source	Value	Basis/Comment
RIVM (Tiesjema & Baars 2009)	TDI = 0.2 mg/kg/day	TDI based on a NOAEL of 20 mg/kg/day for a rat study and application of a 100 fold uncertainty factor, which results in a value that is 10 times lower than the JECFA PTWI of 14 mg/kg/week, established in 1988. The RIVM value is further strengthened by other studies.
JECFA (IPCS)	PTWI = 14 mg/kg PTDI = 2 mg/kg/day	PTWI established for food, which remains current.
ATSDR (ATSDR 2005b)	No chronic MRL established	An intermediate duration guideline of 0.3 mg/kg/day was derived for inorganic tin.

There are limited quantitative toxicity reference values available for tin, with the value available from RIVM adopted for the assessment of all exposures.

Background intakes of tin may be up to 50% of the TRV (Tiesjema & Baars 2009) adopted as the compound is present in food.

Recommendation

On the basis of the discussion above the following toxicity reference values (TRVs) have been adopted for tin:

- Oral TRV (TRV_O) = 0.2 mg/kg/day (Tiesjema & Baars 2009) for all exposures
- Background intakes from other sources (as % of TRV) = 50%.

B4.16 Dioxin-like chemicals

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 2000h) (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.[6] 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

- Dioxins are mainly unintended by–products of combustion processes. It has been estimated that 96 per cent of dioxins 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.

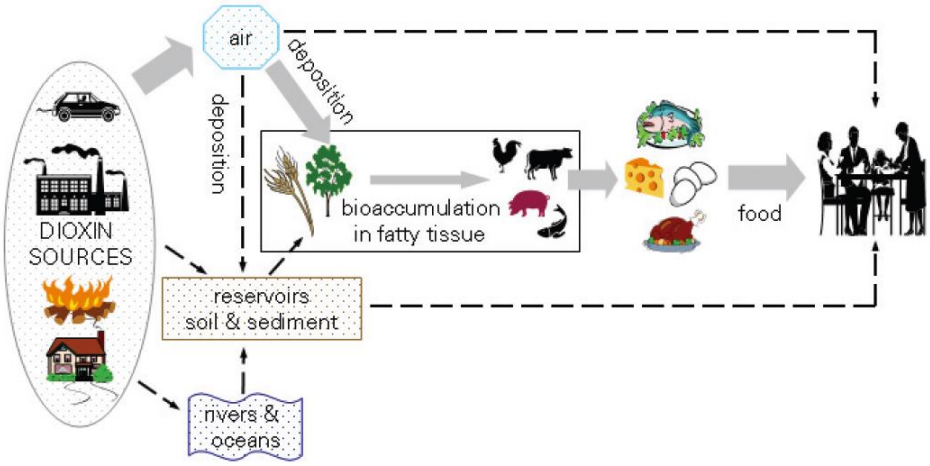
Body burden

- Blood serum levels of dioxins 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 dioxins.
- Dioxins were also detected in breastmilk with a mean of 9 pg TEQ/g lipid. While breast milk contains low levels of dioxins because of its fat content, all babies are exposed to dioxins whether breastfed or not. This is because other foods such as infant formula also contain dioxins 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 levels in air, soil, water and our diet. This was used to determine the range of likely background intakes of dioxins for Australians. For the general population, over 95 per cent of exposure to dioxins is through the diet, with foods of animal origin such as meat, dairy products and fish being the main sources. These intakes of dioxins into the human body are illustrated below.
- Based on the dietary study of dioxins, the intake of dioxins 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 dioxins 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 56% of the adopted tolerable intake.
- Intakes are lower in females than males for the same age, and decline with age in both sexes, the most rapid decline occurring after puberty. Infants and toddlers had a higher intake.



Pathway for dioxins entering our bodies (DEH 2004)

Background intakes for New Zealand populations have been estimated (MfE 2011a) to be 10 pg/kg/month (i.e., 33% of the tolerable monthly intake adopted in New Zealand) based on the dietary intake of adult males, assumed to be also relevant to children.

Health effects

These compounds are persistent in the environment and tend to accumulate in biological systems. One of the most extensively studied PCDD congeners, 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD), exhibits a broad range of toxic effects in laboratory animals, some at very low doses.

Human exposure to dioxins and dioxin-like substances has been associated with a range of toxic effects, including chloracne; reproductive, developmental and neurodevelopmental effects; immunotoxicity; and effects on thyroid hormones, liver and tooth development. Dioxins are also carcinogenic. Developmental effects in males are the most sensitive reproductive health end-point, making children – particularly breastfed infants – a population at elevated risk.

In 1997, IARC classified TCDD as Group 1: carcinogenic to humans, based on evidence from occupationally exposed workers and animal studies. The overall evaluation concluded:

- 2,3,7,8-Tetrachlorodibenzo-*p*-dioxin (TCDD) is carcinogenic to humans (Group 1).
- Other polychlorinated dibenzo-*p*-dioxins are not classifiable as to their carcinogenicity to humans (Group 3).
- Dibenzo-*p*-dioxin is not classifiable as to its carcinogenicity to humans (Group 3).
- Polychlorinated dibenzofurans are not classifiable as to their carcinogenicity to humans (Group 3).

The USEPA has not classified TCDD in relation to carcinogenicity

It can be concluded that TCDD is not a genotoxic carcinogen, but a multi-site carcinogen in experimental animals 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 2011a) also concluded that TCDD is not a genotoxic carcinogen, with developmental effects identified as the most sensitive health endpoint, which is also protective of carcinogenicity. The review acknowledges there is general agreement between the various expert committees that a threshold, or tolerable intakes are appropriate for assessing dioxin like compounds, where the monthly intake value of 70 pg/kg/month is appropriate. Given the long half-lives of dioxins, and thus the likely lack of effect of small excursions of a daily or even weekly intake, it is recommended that a monthly intake toxic-equivalent dose (TEQ) is used.

The Ministry of Health, however, established a maximum monthly intake of 30 pg/kg/month (or 1 pg/kg/day where long term exposures are evaluated on the basis of a daily intake), based on the lower end of the range of tolerable intakes determined by the older WHO (1998) review. The MfE has retained use of this lower tolerable monthly intake, which has been adopted for assessments completed in New Zealand.

Other assessments

The DEH Risk Assessment (DEH 2005) on dioxins provides a review of the other international assessments available at the time of the publication. These support the approach outlined above.

The review completed by RIVM (Tiesjema & Baars 2009) identified a provisional TDI of 2 pg/kg/day based on the JECFA evaluation.

The USEPA 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.

Appendix C Methodology and assumptions

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 for residential and commercial/industrial areas (USEPA 2009d).

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:

$$\text{Exposure Concentration} = C_a \bullet \frac{ET \bullet EF \bullet ED}{AT} \quad (mg/m^3)$$

Table C1: Inhalation exposure assumptions

Parameter		Value adopted	Basis
Ca	Concentration of chemical substance in air (mg/m^3)	Maximum from receptors modelled	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)	Gasses = 1 Particulate bound chemicals = 1	100% of gases reach the lungs. For particulates, these assessed on the basis of the concentration bound to $PM_{2.5}$, which is assumed to all reach the lungs and behave similar to gases
ET	Exposure time (dependant on activity) (hours/day)	Residents = 24 hours/day Workers = 8 hours/day	Residents: Assume someone is exposed at the maximum location all day for 350 days of the year (MfE 2011c). Workers: Working 8 hours per day, for 230 days per year (MfE 2011c)
EF	Exposure frequency (days/year)	Residents = 350 days Workers = 230 days	
ED	Exposure duration (years)	Residents = 30 years Workers = 20 years	For residents in the surrounding areas (rural areas) and for workplaces (MfE 2011c)
AT	Averaging time (hours)	Threshold = ED x 365 days/year x 24 hours/day Non-threshold = 75 years x 365 days/year x 24 hours/day	As per NZ guidance (MfE 2011c)

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, lamb and milk) that is subsequently consumed.

The assessment of the potential ingestion of chemical substances has been undertaken using the approach presented by MfE and the USEPA (MfE 2011c; 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, milk or meat) (mg/kg or mg/L)	Modelled based on deposition of particulates to soil, adopting the maximum from all sensitive 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)	50 mg/day	25 mg/day	Ingestion rate of soil as per MfE (MfE 2011c)
	Fruit and vegetables (kg/day)	0.077 kg/day 46% from aboveground crops 54% from root crops	0.253 kg/day 57% from aboveground crops 43% from root crops	Produce intake defaults based on wet weight (as consumed) (MfE 2011c)
	Eggs (kg/day)	0.006 kg/day	0.014 kg/day	Ingestion rate of eggs per day as per enHealth (enHealth 2012b)
	Milk (L/day)	1.097	1.295	Ingestion rate consistent with P90 intakes from FSANZ (FSANZ 2017)

Parameter		Value adopted		Basis
		Young children	Adults	
	Beef (kg/day)	0.085	0.16	Ingestion rate consistent with P90 intakes from FSANZ (FSANZ 2017)
	Lamb (kg/day)	0.036	0.085	Ingestion rate consistent with P90 intakes from FSANZ (FSANZ 2017)
FI	Fraction of media ingested derived from impacted media, or fraction of produce consumed each day derived from the property			
	Soil	100%	100%	Assume all soil contact occurs on the one property
	Fruit and vegetables	50%	50%	Previous default adopted, noting current guidance suggests this is around 25% (MfE 2011c)
	Eggs and milk	100%	100%	Assume all eggs and milk are from the property
	Beef and lamb	35%	35%	Assume 35% all meat consumed is from the property (note conclusions remain unchanged if this was assumed to be 100%)
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)	1900	4850	Exposed skin surface area relevant to adults and children as per MfE (MfE 2011c)
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)	0.04	0.01	Default values from MfE (MfE 2011c)
ABSd	Dermal absorption fraction (unitless)	Chemical specific		Refer to **Tables B2 and B3**
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	13	70	As per MfE (MfE 2011c)
EF	Exposure frequency (days/year)	350	350	Relevant to adult and child residents as per MfE (MfE 2011c)
ED	Exposure duration (years)	6	24	Duration of residency as per MfE (MfE 2011c)
AT	Averaging time (days)	Threshold = ED x 365 days/year Non-threshold = 75 years x 365 days/year		As per MfE (MfE 2011c) 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 from Stevens (1991), with assumptions adopted in this assessment presented in **Table C3**.

$$C_s = \frac{DR \cdot [1-e^{-k \cdot t}]}{d \cdot \rho \cdot k} \cdot 1000 \qquad \text{(mg/kg)}$$

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 particulates emitted from the facility based on the deposition of TSP		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) with the value for dioxins from Lowe (Lowe, Dietrich & Alberts 1991)
t	Accumulation time (years)	35 years	35 years	Assumed operation time for the proposed Plant
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 intakes utilise soil concentrations calculated for agricultural intakes (OEHHA 2015)

Homegrown fruit and vegetables

Plants may become contaminated with persistent chemical substances via deposition directly onto the plant outer surface and following uptake via the root system. Both mechanisms have been assessed.

The potential concentration of persistent chemical substances that may be present within the plant following atmospheric deposition can be estimated using the following equation (Stevens 1991), with the parameters and assumptions adopted outlined in **Table C4**:

$$C_p = \frac{DR \cdot F \cdot [1-e^{-k \cdot t}]}{Y \cdot k} \qquad \text{(mg/kg plant – wet weight)}$$

The potential uptake of persistent chemical substances into edible crops via the roots can be estimated using the following equation (OEHHA 2015; USEPA 2005d), with the parameters and assumptions adopted outlined in **Table C4:**

$$C_{rp}=C_s \bullet RUF \qquad \text{(mg/kg plant – wet weight)}$$

For the assessment of concentrations in grain crops (or similar crops), only the uptake from roots and translocation to grain or upper parts of the plant has been considered. Any deposition on the surface of the plant would be minor and would also be removed during processing of the grain (or other crop). The RUF adopted for this calculation is then specific to the movement of the chemical from soil to grain of upper part of the plant. This differs from the RUF from soil to the root.

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 particulates emitted from the facility based on the deposition of TSP	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 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 C3**
RUF for root crops	Root uptake factor (unitless)	Chemical specific value adopted	Root uptake factors from MfE (MfE 2011c) and RAIS (RAIS) (soil to wet weight of plant)
RUF for grains and upper parts of plant	Root uptake factor (unitless)	Chemical specific value adopted	Uptake factors adopted for grain based bioconcentration factors for grains and cereals (geometric mean value) from USEPA (USEPA 1996) and Staven (Staven et al. 2003). Where no value is available the root uptake factor has been assumed to be relevant to the uptake into grains (relevant to vanadium, tin and dioxin-like compounds).

Eggs, milk, beef and lamb

The concentration of bioaccumulative chemicals in animal products is calculated on the basis of the intakes of these chemicals by the animal (chicken or cow) and the transfer of these chemicals to the edible produce. The approach adopted in this assessment has involved calculation of intakes from soil and pasture, where grown.

The concentration (C_P) calculated in eggs, milk, beef and lamb meat 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 pasture is grown on the property
IR_C	Ingestion rate of pasture/crops by each animal considered (kg/day)		
	Chickens	0.12	As per OEHHA (2015)
	Beef cattle	9	Ingestion rate from OEHHA (2015)
	Lactating cattle	22	Ingestion rate for lactating cattle from OEHHA (2015)
	Lambs	1.1	Based on assumption of consuming 4.2% body weight per day dry matter (and assuming 20% moisture in feed)
C	Concentration of chemical in crops consumed by animals (mg/kg)	Assume equal to that calculated in aboveground produce	Calculated as described above with assumptions in **Table C4**
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)
	Lambs	0.055	Assumed to be 5% crop intake
Cs	Concentration of chemical in soil (mg/kg)	Calculated value for agricultural soil	Calculated as described above and assumptions in **Table C3**
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 and cobalt where the uptake value from an Australian database has been used (MacLachlan 2011). Other values are the 95% value for the transfer of heavy metals into eggs (Leeman, Van Den Berg & Houben 2007).

Parameter		Value adopted	Basis
	Beef	Chemical specific	Transfer factors adopted from OEHHA (OEHHA 2003, 2015) and RAIS (RAIS).
	Milk	Chemical specific	Transfer factors adopted from OEHHA (2015) and RAIS (RAIS).
	Lamb	Chemical specific	Transfer factors calculated using a metabolic weight adjustment factor of 10.4 from beef as per OEHHA (2012 and 2015 guidance).

All calculations relevant to the estimation of chemical concentrations in soil, fruit and vegetables as well as animal products are presented in **Appendix D**.

Rainwater tanks

The Waimate District Council operates six rural water schemes (Otaio-Makikihi, Cannington-Motukaika, Hook-Waituna, Waihaorunga, Waikakahi & Lower Waihao) and in addition, incorporated societies run Hakataramea and Cattle Creek (Upper Waihao), with Downlands being supplied and administered by Timaru District Council, with a shareholding by Waimate District Council[13]. Hence potable water in rural areas of Waimate is expected to be sourced from mains supply. Where mains water is not available, water from a rainwater tank may be used as potable water, however it is noted that it is the owner's responsibility to make sure that the water is safe to drink[14].

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. When dust is deposited onto a roof, some will be remobilised into air (wind) and blown off the roof before it can be washed into the tank. This has not been considered in this assessment.

It is recommended that first flush devices are used with rainwater tanks to minimise the movement of accumulated dust, bird droppings and organic matter into the tank which can affect water quality (contamination and bacterial load). The use of a first flush device has not been considered in this assessment as it is unknown how many existing tanks use this device. For rainwater tanks used for drinking water purposes, it is expected that these would be maintained appropriately, in line with New Zealand guidance (MoH 2021), which includes the regular cleaning of tanks to remove accumulated sediments, maintaining roof materials, gutters and tank inlet, use of first flush devices and disinfection. The proper maintenance of rainwater tanks (specifically the cleaning out of sediments) would further reduce concentrations below those estimated in this assessment.

Based on mass balance modelling undertaken on rainwater tanks with first flush devices (Martinson & Thomas 2009) and measurements conducted in Australia (Kus et al. 2010), first flush devices can reduce concentrations in rainwater tanks by 90% or more. As noted above the use of a first flush devise has not been considered in this assessment.

[13] https://www.waimatedc.govt.nz/environment-waste/water

[14] https://www.ecan.govt.nz/your-region/your-environment/water/our-drinking-water/

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**:

$$C_W = \frac{DM}{VR \times Kd \times \rho}$$

$$VR = \frac{R \times Area \times Rc \times 1000}{1000}$$

Table C6: Assumptions adopted to estimate concentration in rainwater tanks

Parameter		Value adopted	Basis
DM	Mass of dust deposited on the roof each year that would enter the tank (mg)	DR x Area x 1 year	Conservative assumption that 100% of the dust deposited on the roof for a full year, washes into the rainwater tank (i.e., there is no first flush device and no dust is blown of the roof before being washed into the tank)
DR	Particle deposition rate ($mg/m^2/year$)	Relevant to the maximum sensitive receptor (for deposition of chemicals attached to TSP)	Relevant to areas where multi-pathway exposures may occur
Area	Area of the roof (m^2)	150	Based on the average house size for the Canterbury Region in 2022 (refer to Note 1)
VR	Volume of water collected from the roof each year (L)	calculated	Equation as above
R	Rainfall each year (mm)	564.1	Historical average rainfall for Oamaru (from MetService data)
Rc	Runoff coefficient	0.7	Assumes 30% loss in capture of water into the tank (Lizárraga-Mendiola et al. 2015)
1000	Conversion from m^3 to L Conversion from mm to m		
Kd	Soil-water partition coefficient (cm^3/g)	Chemical-specific	All values for metals from RAIS (RAIS). For organics Kd has been calculated as Kd = Koc x Foc. Koc values obtained from RAIS or PubChem (for dioxins). FoC (fraction of organic carbon) assumed to be 1%.
ρ	Soil bulk density (g/cm^3)	0.5	Assumed for loose deposited dust on roof (upper end measured for powders)

Note 1 - https://www.canstar.co.nz/home-loans/how-much-to-build-a-new-house-in-nz/

All calculations relevant to the estimation of pollutant concentrations in water are presented in **Appendix D**.

Appendix D Risk calculations

Inhalation exposures

Predicted ground level concentrations and screening assessment - acute exposures

		Air Concentration - Maximum 1 hour		Calculated HI	
COPC	**Acute air guideline - health (mg/m^3)**	**Maximum anywhere**	**Maximum - residential, rural, school receptors**	**Maximum anywhere**	**Maximum - residential, rural, school receptors**
Hydrogen chloride (HCl)	0.66	0.0045	0.0029	0.0069	0.0044
Hydrogen fluoride (HF)	0.06	0.00075	0.00050	0.013	0.0083
Ammonia	0.59	0.0076	0.0048	0.013	0.0081
Benzene	0.58	0.0076	0.0048	0.013	0.0083
Antimony	0.001	0.000036	0.000022	0.036	0.022
Arsenic	0.0099	0.000007	0.0000044	0.0007	0.00044
Cadmium	0.018	0.0000014	0.00000088	0.00008	0.000049
Chromium (Cr VI assumed)	0.0013	0.0000036	0.0000022	0.0028	0.0017
Copper	0.1	0.0000036	0.0000022	0.00004	0.000022
Manganese	0.0091	0.0000036	0.0000022	0.0004	0.00024
Mercury	0.0006	0.000014	0.0000087	0.024	0.015
Nickel	0.0011	0.00000036	0.00000022	0.00033	0.00020
Vanadium	0.03	0.000036	0.000022	0.0012	0.00073
Toluene	15	0.0076	0.0048	0.00050	0.00032
Xylenes	7.4	0.0076	0.0048	0.0010	0.00065
Trimethylbenzenes	15	0.0076	0.0048	0.00050	0.00032
				0.11	0.057

Chronic exposures

Inhalation - gases and particulates

$$InhalationExposureConc_V = C_a \bullet \frac{ET \bullet FI \bullet EF \bullet ED}{AT} \quad (mg/m^3)$$

Parameters Relevant to Quantification of Community Exposures - Commercial/industrial workers		
Exposure Time (ET, hr/day)	8	Assume exposure for 8 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Assume worker is at the same location all the time
Dust lung retention factor (unitless)	1	Percentage of respirable dust that is small enough to reach and be retained in the lungs - assumed dust is PM2.5 for inhalation
Exposure Frequency - normal conditions (EF, days/yr)	230	Number of workdays per year as per MfE (2011)
Exposure Duration (ED, years)	20	Duration of work at any one location as per MfE (2011)
Averaging Time - NonThreshold (Atc, hours)	657000	MfE (2011)
Averaging Time - Threshold (Atn, hours)	175200	MfE (2011)

Maximum anywhere (boundary and off-site)

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	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)	
Hydrogen chloride (HCl)	0.0E+00	2.6E-02	0%	2.6E-02	3.5E-05	2.0E-06	7.4E-06	0.00028	3%
Hydrogen fluoride (HF)	0.0E+00	2.9E-02	0%	2.9E-02	5.8E-06	3.2E-07	1.2E-06	0.000042	0%
Ammonia	0.0E+00	3.2E-01	0%	3.2E-01	5.8E-05	3.2E-06	1.2E-05	0.000038	0%
Benzene	0.0E+00	3.6E-03	0%	3.6E-03	5.8E-05	3.2E-06	1.2E-05	0.00090	10%
Antimony	0.0E+00	3.0E-04	0%	3.0E-04	2.9E-07	1.6E-08	6.1E-08	0.00020	2%
Arsenic	0.0E+00	5.5E-06	0%	5.5E-06	5.8E-08	3.2E-09	1.2E-08	0.00059	7%
Beryllium	0.0E+00	2.0E-05	0%	2.0E-05	2.9E-07	1.6E-08	6.1E-08	0.0030	34%
Cadmium	0.0E+00	5.0E-06	20%	4.0E-06	1.2E-08	6.5E-10	2.4E-09	0.00061	7%
Chromium (Cr VI assumed)	0.0E+00	1.1E-06	0%	1.1E-06	2.9E-08	1.6E-09	6.1E-09	0.0015	16%
Copper	0.0E+00	4.9E-01	33%	3.3E-01	2.9E-08	1.6E-09	6.1E-09	0.000000019	0%
Cobalt	0.0E+00	1.0E-04	0%	1.0E-04	2.9E-07	1.6E-08	6.1E-08	0.00061	7%
Lead	0.0E+00	2.0E-04	0%	2.0E-04	5.8E-08	3.2E-09	1.2E-08	0.000061	1%
Manganese	0.0E+00	1.5E-04	20%	1.2E-04	2.9E-08	1.6E-09	6.1E-09	0.000051	1%
Mercury	0.0E+00	2.0E-04	0%	2.0E-04	1.1E-07	6.2E-09	2.3E-08	0.00012	1%
Nickel	0.0E+00	2.0E-05	20%	1.6E-05	2.9E-09	1.6E-10	6.1E-10	0.000038	0%
Thallium	0.0E+00	7.0E-04	10%	6.3E-04	1.2E-08	6.5E-10	2.4E-09	0.0000039	0%
Vanadium	0.0E+00	1.0E-04	0%	1.0E-04	2.9E-07	1.6E-08	6.1E-08	0.00061	7%
Selenium	0.0E+00	2.0E-02	25%	1.5E-02	2.9E-07	1.6E-08	6.1E-08	0.0000041	0%
Tin	0.0E+00	7.0E-01	50%	3.5E-01	2.9E-07	1.6E-08	6.1E-08	0.00000017	0%
Dioxins and furans (WHO-TEQ)	0.0E+00	3.5E-09	33%	2.3E-09	3.4E-13	1.9E-14	7.1E-14	0.000030	0%
Toluene	0.0E+00	5.0E+00	0%	5.0E+00	5.8E-05	3.2E-06	1.2E-05	0.0000024	0%
Xylenes	0.0E+00	2.0E-01	0%	2.0E-01	5.8E-05	3.2E-06	1.2E-05	0.000061	1%
Trimethylbenzenes	0.0E+00	6.0E-02	10%	5.4E-02	5.8E-05	3.2E-06	1.2E-05	0.00023	3%
							TOTAL	**0.0090**	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Inhalation - gases and particulates

$$InhalationExposureConc_V = C_a \bullet \frac{ET \bullet FI \bullet EF \bullet ED}{AT} \quad (mg/m^3)$$

Parameters Relevant to Quantification of Community Exposures - 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)	1	Percentage of respirable dust that is small enough to reach and be retained in the lungs - assumed dust is PM2.5 for inhalation
Exposure Frequency - normal conditions (EF, days/yr)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	30	Duration at one residence - assumed for area as per MfE (2011)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	262800	US EPA 2009

Maximum for sensitive receptors

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 sensitive receptors (Ca)	Inhalation Exposure Concentration - NonThreshold	Inhalation Exposure Concentration - Threshold	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)	
Hydrogen chloride (HCl)	0.0E+00	2.6E-02	0%	2.6E-02	2.8E-05	1.2E-05	2.7E-05	0.0010	3%
Hydrogen fluoride (HF)	0.0E+00	2.9E-02	0%	2.9E-02	4.7E-06	1.9E-06	4.5E-06	0.00015	0%
Ammonia	0.0E+00	3.2E-01	0%	3.2E-01	4.7E-05	1.9E-05	4.5E-05	0.00014	0%
Benzene	0.0E+00	3.6E-03	0%	3.6E-03	4.7E-05	1.9E-05	4.5E-05	0.0053	14%
Antimony	0.0E+00	3.0E-04	0%	3.0E-04	2.3E-07	9.5E-08	2.2E-07	0.00074	2%
Arsenic	0.0E+00	5.5E-06	0%	5.5E-06	4.6E-08	1.9E-08	4.4E-08	0.0034	9%
Beryllium	0.0E+00	2.0E-05	0%	2.0E-05	2.3E-07	9.5E-08	2.2E-07	0.011	28%
Cadmium	0.0E+00	5.0E-06	20%	4.0E-06	9.2E-09	3.8E-09	8.8E-09	0.0022	6%
Chromium (Cr VI assumed)	0.0E+00	1.1E-06	0%	1.1E-06	2.3E-08	9.5E-09	2.2E-08	0.0086	22%
Copper	0.0E+00	4.9E-01	33%	3.3E-01	2.3E-08	9.5E-09	2.2E-08	0.000000067	0%
Cobalt	0.0E+00	1.0E-04	0%	1.0E-04	2.3E-07	9.5E-08	2.2E-07	0.0022	6%
Lead	0.0E+00	2.0E-04	0%	2.0E-04	4.6E-08	1.9E-08	4.4E-08	0.00022	1%
Manganese	0.0E+00	1.5E-04	20%	1.2E-04	2.3E-08	9.5E-09	2.2E-08	0.00018	0%
Mercury	0.0E+00	2.0E-04	0%	2.0E-04	9.0E-08	3.7E-08	8.6E-08	0.00043	1%
Nickel	0.0E+00	2.0E-05	20%	1.6E-05	2.3E-09	9.5E-10	2.2E-09	0.00014	0%
Thallium	0.0E+00	7.0E-04	10%	6.3E-04	9.2E-09	3.8E-09	8.8E-09	0.000014	0%
Vanadium	0.0E+00	1.0E-04	0%	1.0E-04	2.3E-07	9.5E-08	2.2E-07	0.0022	6%
Selenium	0.0E+00	2.0E-02	25%	1.5E-02	2.3E-07	9.5E-08	2.2E-07	0.000015	0%
Tin	0.0E+00	7.0E-01	50%	3.5E-01	2.3E-07	9.5E-08	2.2E-07	0.00000063	0%
Dioxins and furans (WHO-TEQ)	0.0E+00	3.5E-09	33%	2.3E-09	2.7E-13	1.1E-13	2.6E-13	0.00011	0%
Toluene	0.0E+00	5.0E+00	0%	5.0E+00	4.7E-05	1.9E-05	4.5E-05	0.0000090	0%
Xylenes	0.0E+00	2.0E-01	0%	2.0E-01	4.7E-05	1.9E-05	4.5E-05	0.00022	1%
Trimethylbenzenes	0.0E+00	6.0E-02	10%	5.4E-02	4.7E-05	1.9E-05	4.5E-05	0.00083	2%
							TOTAL	**0.039**	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Multi-pathway exposures for maximum sensitive receptor

Soil exposures

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	35	35	Duration of operation (conservative assumption)

Chemical-specific Inputs and calculations - maximum sensitive receptors

Chemical	Half-life in soil years	Loss constant (K) per year	Deposition Rate (DR) mg/m^2/year	Surface Concentration in Soil mg/kg	Agricultural Concentration in Soil mg/kg
Antimony	273973	2.5E-06	1.3E-01	2.8E-01	1.9E-02
Arsenic	273973	2.5E-06	2.6E-02	5.7E-02	3.8E-03
Beryllium	273973	2.5E-06	1.3E-01	2.8E-01	1.9E-02
Cadmium	273973	2.5E-06	5.2E-03	1.1E-02	7.5E-04
Chromium (Cr VI assumed)	273973	2.5E-06	1.3E-02	2.8E-02	1.9E-03
Copper	273973	2.5E-06	1.3E-02	2.8E-02	1.9E-03
Cobalt	273973	2.5E-06	1.3E-01	2.8E-01	1.9E-02
Lead	273973	2.5E-06	2.6E-02	5.7E-02	3.8E-03
Manganese	273973	2.5E-06	1.3E-02	2.8E-02	1.9E-03
Mercury	273973	2.5E-06	5.0E-02	1.1E-01	7.4E-03
Nickel	273973	2.5E-06	1.3E-03	2.8E-03	1.9E-04
Thallium	273973	2.5E-06	5.2E-03	1.1E-02	7.5E-04
Vanadium	273973	2.5E-06	1.3E-01	2.8E-01	1.9E-02
Selenium	273973	2.5E-06	1.3E-01	2.8E-01	1.9E-02
Tin	273973	2.5E-06	1.3E-01	2.8E-01	1.9E-02
Dioxins and furans (WHO-TEQ)	41	0.017	1.5E-07	2.5E-07	1.7E-08

Half-life in soil: dioxin value from Lowe et al (1991) and metals, PAHs from OEHHA (2015)

Exposure to Chemicals via Incidental Ingestion of Soil

$$Daily\ Chemical\ Intake_{IS} = C_S \bullet \frac{IR_S \bullet FI \bullet CF \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate (IRs, mg/day)	25	As per MfE (2011)
Fraction Ingested from Source (FI, unitless)	100%	All of daily soil intake occurs from site
Exposure Frequency (EF, days/year)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	24	Time at one residence as adult as per MfE (2011)
Body Weight (BW, kg)	70	For male and females combined as per MfE (2011)
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	8760	MFfE (2011)

Maximum from sensitive receptors

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)	
Antimony		8.6E-04	20%	6.9E-04	100%	2.8E-01	3.1E-08	9.7E-08	--		1.4E-04	10%
Arsenic		8.6E-06		8.6E-06	100%	5.7E-02	6.2E-09	1.9E-08	--		7.2E-04	50%
Beryllium		2.0E-03		2.0E-03	100%	2.8E-01	3.1E-08	9.7E-08	--		4.8E-05	3%
Cadmium		8.0E-04	50%	4.0E-04	100%	1.1E-02	1.2E-09	3.9E-09	--		9.7E-06	1%
Chromium (Cr VI assumed)		9.0E-04		9.0E-04	100%	2.8E-02	3.1E-09	9.7E-09	--		1.1E-05	1%
Copper		1.4E-01	33%	9.4E-02	100%	2.8E-02	3.1E-09	9.7E-09	--		1.0E-07	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	2.8E-01	3.1E-08	9.7E-08	--		8.6E-05	6%
Lead		2.0E-04	50%	1.0E-04	100%	5.7E-02	6.2E-09	1.9E-08	--		1.9E-04	13%
Manganese		1.6E-01	50%	8.0E-02	100%	2.8E-02	3.1E-09	9.7E-09	--		1.2E-07	0%
Mercury		2.0E-03	5%	1.9E-03	100%	1.1E-01	1.2E-08	3.8E-08	--		2.0E-05	1%
Nickel		1.2E-02	60%	4.8E-03	100%	2.8E-03	3.1E-10	9.7E-10	--		2.0E-07	0%
Thallium		2.0E-04	10%	1.8E-04	100%	1.1E-02	1.2E-09	3.9E-09	--		2.2E-05	1%
Vanadium		2.0E-03		2.0E-03	100%	2.8E-01	3.1E-08	9.7E-08	--		4.8E-05	3%
Selenium		6.0E-03	25%	4.5E-03	100%	2.8E-01	3.1E-08	9.7E-08	--		2.2E-05	1%
Tin		2.0E-01	50%	1.0E-01	100%	2.8E-01	3.1E-08	9.7E-08	--		9.7E-07	0%
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	100%	2.5E-07	2.8E-14	8.6E-14	--		1.3E-04	9%
								TOTAL	**--**		**1.5E-3**	

Chemical	Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Exposure to Chemicals via Incidental Ingestion of Soil

$$Daily\ Chemical\ Intake_{IS} = C_S \bullet \frac{IR_S \bullet FI \bullet CF \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Young Children		
Ingestion Rate (IRs, mg/day)	50	Assumed daily soil ingestion rate for young children, MfE (2011)
Fraction Ingested from Source (FI, unitless)	100%	All of daily soil intake occurs from site
Exposure Frequency (EF, days/year)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	13	Representative weight as per MfE (2011)
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	2190	MfE (2011)

Maximum from sensitive receptors

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
Antimony		8.6E-04	20%	6.9E-04	100%	2.8E-01	8.3E-08	1.0E-06	--		1.5E-03	15%
Arsenic		8.6E-06		8.6E-06	100%	5.7E-02	1.7E-08	2.1E-07	--		1.9E-03	20%
Beryllium		2.0E-03		2.0E-03	100%	2.8E-01	8.3E-08	1.0E-06	--		5.2E-04	5%
Cadmium		8.0E-04	50%	4.0E-04	100%	1.1E-02	3.3E-09	4.2E-08	--		1.0E-04	1%
Chromium (Cr VI assumed)		9.0E-04		9.0E-04	100%	2.8E-02	8.3E-09	1.0E-07	--		1.2E-04	1%
Copper		1.4E-01	33%	9.4E-02	100%	2.8E-02	8.3E-09	1.0E-07	--		1.1E-06	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	2.8E-01	8.3E-08	1.0E-06	--		9.3E-04	9%
Lead		2.0E-04	50%	1.0E-04	100%	5.7E-02	1.7E-08	2.1E-07	--		2.1E-03	21%
Manganese		1.6E-01	50%	8.0E-02	100%	2.8E-02	8.3E-09	1.0E-07	--		1.3E-06	0%
Mercury		2.0E-03	5%	1.9E-03	100%	1.1E-01	3.3E-08	4.1E-07	--		2.1E-04	2%
Nickel		1.2E-02	60%	4.8E-03	100%	2.8E-03	8.3E-10	1.0E-08	--		2.2E-06	0%
Thallium		2.0E-04	10%	1.8E-04	100%	1.1E-02	3.3E-09	4.2E-08	--		2.3E-04	2%
Vanadium		2.0E-03		2.0E-03	100%	2.8E-01	8.3E-08	1.0E-06	--		5.2E-04	5%
Selenium		6.0E-03	25%	4.5E-03	100%	2.8E-01	8.3E-08	1.0E-06	--		2.3E-04	2%
Tin		2.0E-01	50%	1.0E-01	100%	2.8E-01	8.3E-08	1.0E-06	--		1.0E-05	0%
Dioxins and furans (WHO-TE		1.0E-09	33%	6.7E-10	100%	2.5E-07	7.4E-14	9.3E-13	--		1.4E-03	14%
								TOTAL	--		9.8E-3	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Dermal Exposure to Chemicals via Contact with Soil

$$Daily\ Chemical\ Intake_{DS} = C_S \bullet \frac{SA_S \bullet AF \bullet FE \bullet ABS \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Surface Area (SAs, cm^2)	4850	Exposed skin surface area for adults as per MfE (2011)
Adherence Factor (AF, mg/cm^2)	0.01	Default as per MfE (2011)
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)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	24	Time at one residence as adult as per MfE (2011)
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	8760	MfE (2011)

Maximum from sensitive receptors

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)	
Antimony		8.6E-04	20%	6.9E-04		2.8E-01			--		--	
Arsenic		8.6E-06		8.6E-06	0.005	5.7E-02	6.0E-11	1.9E-10	--		7.0E-6	
Beryllium		2.0E-03		2.0E-03		2.8E-01			--		--	
Cadmium		8.0E-04	50%	4.0E-04		1.1E-02			--		--	
Chromium (Cr VI assumed)		9.0E-04		9.0E-04		2.8E-02			--		--	
Copper		1.4E-01	33%	9.4E-02		2.8E-02			--		--	
Cobalt		1.4E-03	20%	1.1E-03		2.8E-01			--		--	
Lead		2.0E-04	50%	1.0E-04		5.7E-02			--		--	
Manganese		1.6E-01	50%	8.0E-02		2.8E-02			--		--	
Mercury		2.0E-03	5%	1.9E-03	0.001	1.1E-01	2.3E-11	7.3E-11	--		3.9E-08	
Nickel		1.2E-02	60%	4.8E-03	0.005	2.8E-03	3.0E-12	9.4E-12	--		2.0E-09	
Thallium		2.0E-04	10%	1.8E-04		1.1E-02			--		--	
Vanadium		2.0E-03		2.0E-03		2.8E-01			--		--	
Selenium		6.0E-03	25%	4.5E-03		2.8E-01			--		--	
Tin		2.0E-01	50%	1.0E-01		2.8E-01			--		--	
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	0.03	2.5E-07	1.6E-15	5.0E-15	--		7.5E-06	
								TOTAL	**--**		**1.5E-05**	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Dermal Exposure to Chemicals via Contact with Soil

$$Daily\ Chemical\ Intake_{DS} = C_S \bullet \frac{SA_S \bullet AF \bullet FE \bullet ABS \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Young Children		
Surface Area (SAs, cm^2)	1900	Exposed skin surface area for young children as per MfE (2011)
Adherence Factor (AF, mg/cm^2)	0.04	Default as per MfE (2011)
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)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	13	Representative weight as per MfE (2011)
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	2190	MfE (2011)

Maximum from sensitive receptors

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)	
Antimony		8.6E-04	20%	6.9E-04		2.8E-01			--		--	
Arsenic		8.6E-06		8.6E-06	0.005	5.7E-02	1.3E-10	1.6E-09	--		1.5E-5	
Beryllium		2.0E-03		2.0E-03		2.8E-01			--		--	
Cadmium		8.0E-04	50%	4.0E-04		1.1E-02			--		--	
Chromium (Cr VI assumed)		9.0E-04		9.0E-04		2.8E-02			--		--	
Copper		1.4E-01	33%	9.4E-02		2.8E-02			--		--	
Cobalt		1.4E-03	20%	1.1E-03		2.8E-01			--		--	
Lead		2.0E-04	50%	1.0E-04		5.7E-02			--		--	
Manganese		1.6E-01	50%	8.0E-02		2.8E-02			--		--	
Mercury		2.0E-03	5%	1.9E-03	0.001	1.1E-01	4.9E-11	6.2E-10	--		3.3E-07	
Nickel		1.2E-02	60%	4.8E-03	0.005	2.8E-03	6.3E-12	7.9E-11	--		1.7E-08	
Thallium		2.0E-04	10%	1.8E-04		1.1E-02			--		--	
Vanadium		2.0E-03		2.0E-03		2.8E-01			--		--	
Selenium		6.0E-03	25%	4.5E-03		2.8E-01			--		--	
Tin		2.0E-01	50%	1.0E-01		2.8E-01			--		--	
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	0.03	2.5E-07	3.4E-15	4.2E-14	--		6.3E-05	
								TOTAL	--		7.8E-05	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Homegrown fruit and vegetables and crops

Calculation of Concentrations in Plants

ref: Stevens B. (1991) Reference case

Uptake Due to Deposition in Aboveground Crops	Uptake via Roots from Soil
$C_p = \frac{DR \bullet F \bullet \left[1 - e^{-k \bullet t}\right]}{Y \bullet k}$ (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)	$C_{rp} = C_s \bullet RUF$ (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 - Maximum sensitive receptors									
Chemical	**Half-life in plant ($T_{0.5}$)**	**Loss constant (k)**	**Deposition Rate (DR)**	**Aboveground Produce Concentration via Deposition**	**Root Uptake Factor (RUF) (A)**	**Soil Concentration (Cs)**	**Below Ground Produce Concentration**	**Uptake factor into grain crops (from soil) (B)**	**Concetration in grain crops**
	days	**per day**	**mg/m^2/day**	**mg/kg ww**	**unitless**	**mg/kg**	**mg/kg ww**	**unitless**	**mg/kg ww**
Antimony	14	0.05	3.5E-04	1.8E-04	0.05	1.9E-02	9.4E-04	0.03	6E-04
Arsenic	14	0.05	7.1E-05	3.5E-05	0.011	3.8E-03	4.1E-05	0.026	1E-04
Beryllium	14	0.05	3.5E-04	1.8E-04	0.0025	1.9E-02	4.7E-05	0.002	4E-05
Cadmium	14	0.05	1.4E-05	7.1E-06	0.125	7.5E-04	9.4E-05	0.36	3E-04
Chromium (Cr VI assumed)	14	0.05	3.5E-05	1.8E-05	0.0324	1.9E-03	6.1E-05	0.0045	8E-06
Copper	14	0.05	3.5E-05	1.8E-05	0.1	1.9E-03	1.9E-04	0.25	5E-04
Cobalt	14	0.05	3.5E-04	1.8E-04	0.005	1.9E-02	9.4E-05	0.0037	7E-05
Lead	14	0.05	7.1E-05	3.5E-05	0.015	3.8E-03	5.7E-05	0.0047	2E-05
Manganese	14	0.05	3.5E-05	1.8E-05	0.0625	1.9E-03	1.2E-04	0.3	6E-04
Mercury	14	0.05	1.4E-04	6.9E-05	0.07	7.4E-03	5.2E-04	0.0854	6E-04
Nickel	14	0.05	3.5E-06	1.8E-06	0.015	1.9E-04	2.8E-06	0.01	2E-06
Thallium	14	0.05	1.4E-05	7.1E-06	0.001	7.5E-04	7.5E-07	0.004	3E-06
Vanadium	14	0.05	3.5E-04	1.8E-04	0.00138	1.9E-02	2.6E-05	0.00138	3E-05
Selenium	14	0.05	3.5E-04	1.8E-04	0.00625	1.9E-02	1.2E-04	0.002	4E-05
Tin	14	0.05	3.5E-04	1.8E-04	0.0075	1.9E-02	1.4E-04	0.0075	1E-04
Dioxins and furans (WHO-TEQ)	14	0.05	4.2E-10	2.1E-10	0.000876	1.7E-08	1.5E-11	0.000876	1E-11

(A) Root uptake factors from MfE (2011) and RAIS (soil to wet weight of plant)
Note uptake into plants from soil considered insignificant as dioxins are very poorly soluble (OEHHA 2015 and USEPA 1994)
(B) Uptake factors adopted for grain based bioconcentration factors for grains and cereals (geometric mean value) from USEPA (1996) and Staven (2003)
Where no value is available the root uptake factor has been assumed to be relevant to the uptake into grains (relevant to vanadium, tin and dioxins)

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables

$$\text{Daily chemical intake} = C_A \times \frac{IR_P \times \%A \times FI \times ME \times EF \times ED}{BW \times AT} + C_R \times \frac{IR_p \times \%R \times FI \times ME \times ED \times ED}{BW \times AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Produce (IRp) (kg/day)	0.253	Total produce consumption rate for adults as per MfE (2011)
Proportion of total intake from aboveground crops (%A	57%	Proportions as per MfE (2011)
Proportion of total intake from root crops (%R)	43%	Proportions as per MfE (2011)
Fraction ingested that is homegrown (%)	50%	Relevant to rural areas as per MfE (2011)
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	24	Time at one residence as adult as per MfE (2011)
Body Weight (BW, kg)	70	For male and females combined as per MfE (2011)
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	8760	MfE (2011)

Maximum from sensitive receptors

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)	
Antimony		8.6E-04	20%	6.9E-04	100%	1.8E-04	9.4E-04	2.8E-07	8.8E-07	--		1.3E-03	22%
Arsenic		8.6E-06		8.6E-06	100%	3.5E-05	4.1E-05	2.1E-08	6.6E-08	--		2.4E-03	42%
Beryllium		2.0E-03		2.0E-03	100%	1.8E-04	4.7E-05	6.7E-08	2.1E-07	--		1.0E-04	2%
Cadmium		8.0E-04	50%	4.0E-04	100%	7.1E-06	9.4E-05	2.5E-08	7.7E-08	--		1.9E-04	3%
Chromium (Cr VI assumed)		9.0E-04		9.0E-04	100%	1.8E-05	6.1E-05	2.0E-08	6.3E-08	--		7.0E-05	1%
Copper		1.4E-01	33%	9.4E-02	100%	1.8E-05	1.9E-04	5.1E-08	1.6E-07	--		1.7E-06	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	1.8E-04	9.4E-05	7.8E-08	2.4E-07	--		2.2E-04	4%
Lead		2.0E-04	50%	1.0E-04	100%	3.5E-05	5.7E-05	2.5E-08	7.7E-08	--		7.7E-04	13%
Manganese		1.6E-01	50%	8.0E-02	100%	1.8E-05	1.2E-04	3.4E-08	1.1E-07	--		1.3E-06	0%
Mercury		2.0E-03	5%	1.9E-03	100%	6.9E-05	5.2E-04	1.4E-07	4.5E-07	--		2.4E-04	4%
Nickel		1.2E-02	60%	4.8E-03	100%	1.8E-06	2.8E-06	1.2E-09	3.9E-09	--		8.0E-07	0%
Thallium		2.0E-04	10%	1.8E-04	100%	7.1E-06	7.5E-07	2.4E-09	7.5E-09	--		4.2E-05	1%
Vanadium		2.0E-03		2.0E-03	100%	1.8E-04	2.6E-05	6.2E-08	1.9E-07	--		9.7E-05	2%
Selenium		6.0E-03	25%	4.5E-03	100%	1.8E-04	1.2E-04	8.4E-08	2.6E-07	--		5.8E-05	1%
Tin		2.0E-01	50%	1.0E-01	100%	1.8E-04	1.4E-04	9.0E-08	2.8E-07	--		2.8E-06	0%
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	100%	2.1E-10	1.5E-11	6.9E-14	2.2E-13	--		3.2E-04	6%
									TOTAL	--		5.8E-03	

Chemical	Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables

$$\text{Daily chemical intake} = C_A \times \frac{IR_P \times \%A \times FI \times ME \times EF \times ED}{BW \times AT} + C_R \times \frac{IR_P \times \%R \times FI \times ME \times ED \times ED}{BW \times AT} \quad \text{(mg/kg/day)}$$

Scenario 2

Parameters Relevant to Quantification of Exposure by Young children		
Ingestion Rate of Produce (IRp) (kg/day)	0.077	Total produce consumption rate for children as per MfE (2011)
Proportion of total intake from aboveground crops (%A	46%	Proportions as per MfE (2011)
Proportion of total intake from root crops (%R)	54%	Proportions as per MfE (2011)
Fraction ingested that is homegrown (%)	50%	Relevant to rural areas as per MfE (2011)
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	13	Representative weight as per MfE (2011)
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	2190	MfE (2011)

Maximum from sensitive receptors

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)	
Antimony		8.6E-04	20%	6.9E-04	100%	1.8E-04	9.4E-04	1.3E-07	1.7E-06	--		2.4E-03	35%
Arsenic		8.6E-06		8.6E-06	100%	3.5E-05	4.1E-05	8.8E-09	1.1E-07	--		1.0E-03	15%
Beryllium		2.0E-03		2.0E-03	100%	1.8E-04	4.7E-05	2.4E-08	3.0E-07	--		1.5E-04	2%
Cadmium		8.0E-04	50%	4.0E-04	100%	7.1E-06	9.4E-05	1.2E-08	1.5E-07	--		3.8E-04	6%
Chromium (Cr VI assumed)		9.0E-04		9.0E-04	100%	1.8E-05	6.1E-05	9.3E-09	1.2E-07	--		1.3E-04	2%
Copper		1.4E-01	33%	9.4E-02	100%	1.8E-05	1.9E-04	2.5E-08	3.1E-07	--		3.3E-06	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	1.8E-04	9.4E-05	3.0E-08	3.8E-07	--		3.4E-04	5%
Lead		2.0E-04	50%	1.0E-04	100%	3.5E-05	5.7E-05	1.1E-08	1.3E-07	--		1.3E-03	19%
Manganese		1.6E-01	50%	8.0E-02	100%	1.8E-05	1.2E-04	1.6E-08	2.0E-07	--		2.5E-06	0%
Mercury		2.0E-03	5%	1.9E-03	100%	6.9E-05	5.2E-04	7.0E-08	8.8E-07	--		4.6E-04	7%
Nickel		1.2E-02	60%	4.8E-03	100%	1.8E-06	2.8E-06	5.3E-10	6.6E-09	--		1.4E-06	0%
Thallium		2.0E-04	10%	1.8E-04	100%	7.1E-06	7.5E-07	8.3E-10	1.0E-08	--		5.8E-05	1%
Vanadium		2.0E-03		2.0E-03	100%	1.8E-04	2.6E-05	2.2E-08	2.7E-07	--		1.4E-04	2%
Selenium		6.0E-03	25%	4.5E-03	100%	1.8E-04	1.2E-04	3.3E-08	4.1E-07	--		9.1E-05	1%
Tin		2.0E-01	50%	1.0E-01	100%	1.8E-04	1.4E-04	3.6E-08	4.5E-07	--		4.5E-06	0%
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	100%	2.1E-10	1.5E-11	2.4E-14	2.9E-13	--		4.4E-04	6%
									TOTAL	--		7.0E-03	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Ingestion of eggs

Calculation of Concentrations in Eggs

Uptake in to chicken eggs

$$C_E = (FI \times IR_C \times C + IR_S \times C_S \times B) \times TF_E$$ (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 pasture is grown on the site
IRc (ingestion rate of crops)	kg/day	0.12	As per OEHHA (2015)
IRs (ingestion rate of soil)	kg/day	0.01	As per OEHHA (2015) and advice from AgVIC
B (bioavailability)	%	100%	

Chemical-specific Inputs and calculations - Maximum sensitive receptors

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	
Antimony	1.8E-04	1.9E-02	1.7E-01	3.6E-05	95% from Leeman et al (2007)
Arsenic	3.5E-05	3.8E-03	7.0E-02	2.9E-06	
Beryllium	1.8E-04	1.9E-02	9.0E-02	1.9E-05	
Cadmium	7.1E-06	7.5E-04	1.0E-02	8.4E-08	
Chromium (Cr VI assumed)	1.8E-05	1.9E-03	9.2E-03	1.9E-07	OEHHA (2003)
Copper	1.8E-05	1.9E-03	1.7E-01	3.6E-06	95% from Leeman et al (2007)
Cobalt	1.8E-04	1.9E-02	3.3E-03	6.9E-07	MacLachlan (2011)
Lead	3.5E-05	3.8E-03	4.0E-02	1.7E-06	
Manganese	1.8E-05	1.9E-03	1.7E-01	3.6E-06	
Mercury	6.9E-05	7.4E-03	8.0E-01	6.5E-05	
Nickel	1.8E-06	1.9E-04	2.0E-02	4.2E-08	
Thallium	7.1E-06	7.5E-04	1.7E-01	1.4E-06	95% from Leeman et al (2007)
Vanadium	1.8E-04	1.9E-02	1.7E-01	3.6E-05	95% from Leeman et al (2007)
Selenium	1.8E-04	1.9E-02	3.0E+00	6.3E-04	
Tin	1.8E-04	1.9E-02	1.7E-01	3.6E-05	95% from Leeman et al (2007)
Dioxins and furans (WHO-TEQ)	2.1E-10	1.7E-08	1.0E+01	1.9E-09	

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Eggs

$$\text{Daily chemical intake} = C_E \times \frac{IR_E \times FI \times ME \times EF \times ED}{BW \times AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Eggs (IRE) (kg/day)	0.014	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)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	24	Time at one residence as adult as per MfE (2011)
Body Weight (BW, kg)	70	For male and females combined as per MfE (2011)
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	8760	MfE (2011)

Maximum from sensitive receptors

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
Antimony		8.6E-04	20%	6.9E-04	100%	3.6E-05	2.2E-09	6.8E-09	--		9.9E-06	2%
Arsenic		8.6E-06		8.6E-06	100%	2.9E-06	1.8E-10	5.6E-10	--		2.1E-05	3%
Beryllium		2.0E-03		2.0E-03	100%	1.9E-05	1.2E-09	3.6E-09	--		1.8E-06	0%
Cadmium		8.0E-04	50%	4.0E-04	100%	8.4E-08	5.1E-12	1.6E-11	--		4.0E-08	0%
Chromium (Cr VI assumed)		9.0E-04		9.0E-04	100%	1.9E-07	1.2E-11	3.7E-11	--		4.1E-08	0%
Copper		1.4E-01	33%	9.4E-02	100%	3.6E-06	2.2E-10	6.8E-10	--		7.3E-09	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	6.9E-07	4.2E-11	1.3E-10	--		1.2E-07	0%
Lead		2.0E-04	50%	1.0E-04	100%	1.7E-06	1.0E-10	3.2E-10	--		3.2E-06	1%
Manganese		1.6E-01	50%	8.0E-02	100%	3.6E-06	2.2E-10	6.8E-10	--		8.5E-09	0%
Mercury		2.0E-03	5%	1.9E-03	100%	6.5E-05	4.0E-09	1.3E-08	--		6.6E-06	1%
Nickel		1.2E-02	60%	4.8E-03	100%	4.2E-08	2.6E-12	8.0E-12	--		1.7E-09	0%
Thallium		2.0E-04	10%	1.8E-04	100%	1.4E-06	8.8E-11	2.7E-10	--		1.5E-06	0%
Vanadium		2.0E-03		2.0E-03	100%	3.6E-05	2.2E-09	6.8E-09	--		3.4E-06	1%
Selenium		6.0E-03	25%	4.5E-03	100%	6.3E-04	3.9E-08	1.2E-07	--		2.7E-05	4%
Tin		2.0E-01	50%	1.0E-01	100%	3.6E-05	2.2E-09	6.8E-09	--		6.8E-08	
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	100%	1.9E-09	1.2E-13	3.7E-13	--		5.5E-04	88%
								TOTAL	--		6.3E-04	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Exposure to Chemicals via Ingestion of Eggs

$$\text{Daily chemical intake} = C_E \times \frac{IR_E \times FI \times ME \times EF \times ED}{BW \times AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Young children		
Ingestion Rate of Eggs (IRE) (kg/day)	0.006	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)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	13	Representative weight as per MfE (2011)
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	2190	MfE (2011)

Maximum from sensitive receptors

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
Antimony		8.6E-04	20%	6.9E-04	100%	3.6E-05	1.3E-09	1.6E-08	--		2.3E-05	2%
Arsenic		8.6E-06		8.6E-06	100%	2.9E-06	1.0E-10	1.3E-09	--		1.2E-05	1%
Beryllium		2.0E-03		2.0E-03	100%	1.9E-05	6.7E-10	8.4E-09	--		4.2E-06	0%
Cadmium		8.0E-04	50%	4.0E-04	100%	8.4E-08	3.0E-12	3.7E-11	--		9.3E-08	0%
Chromium (Cr VI assumed)		9.0E-04		9.0E-04	100%	1.9E-07	6.8E-12	8.5E-11	--		9.5E-08	0%
Copper		1.4E-01	33%	9.4E-02	100%	3.6E-06	1.3E-10	1.6E-09	--		1.7E-08	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	6.9E-07	2.5E-11	3.1E-10	--		2.7E-07	0%
Lead		2.0E-04	50%	1.0E-04	100%	1.7E-06	5.9E-11	7.4E-10	--		7.4E-06	1%
Manganese		1.6E-01	50%	8.0E-02	100%	3.6E-06	1.3E-10	1.6E-09	--		2.0E-08	0%
Mercury		2.0E-03	5%	1.9E-03	100%	6.5E-05	2.3E-09	2.9E-08	--		1.5E-05	1%
Nickel		1.2E-02	60%	4.8E-03	100%	4.2E-08	1.5E-12	1.9E-11	--		3.9E-09	0%
Thallium		2.0E-04	10%	1.8E-04	100%	1.4E-06	5.1E-11	6.3E-10	--		3.5E-06	0%
Vanadium		2.0E-03		2.0E-03	100%	3.6E-05	1.3E-09	1.6E-08	--		7.9E-06	1%
Selenium		6.0E-03	25%	4.5E-03	100%	6.3E-04	2.2E-08	2.8E-07	--		6.2E-05	4%
Tin		2.0E-01	50%	1.0E-01	100%	3.6E-05	1.3E-09	1.6E-08	--		1.6E-07	
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	100%	1.9E-09	6.8E-14	8.5E-13	--		1.3E-03	90%
								TOTAL	**--**		**1.4E-03**	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Ingestion of beef

Calculation of Concentrations in Homegrown Beef

Uptake in to beef meat

$$C_E=(FI \times IR_C \times C+IR_S \times C_S \times B) \times TF_B$$ (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 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/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	Based on data from OEHHA 2015 (5% total produce intakes from soil from pasture)
B (bioavailability)	%	100%	

Chemical-specific Inputs and calculations - maximum sensitive receptors

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	
Antimony	1.8E-04	1.9E-02	1.0E-03	1.0E-05	RAIS
Arsenic	3.5E-05	3.8E-03	2.0E-03	4.0E-06	
Beryllium	1.8E-04	1.9E-02	3.0E-04	3.0E-06	
Cadmium	7.1E-06	7.5E-04	2.0E-03	8.1E-07	
Chromium (Cr VI assumed)	1.8E-05	1.9E-03	5.5E-03	5.5E-06	RAIS
Copper	1.8E-05	1.9E-03	1.0E-02	1.0E-05	RAIS
Cobalt	1.8E-04	1.9E-02	2.0E-02	2.0E-04	RAIS
Lead	3.5E-05	3.8E-03	3.0E-04	6.0E-07	
Manganese	1.8E-05	1.9E-03	4.0E-04	4.0E-07	RAIS
Mercury	6.9E-05	7.4E-03	4.0E-02	1.6E-04	
Nickel	1.8E-06	1.9E-04	3.0E-04	3.0E-08	
Thallium	7.1E-06	7.5E-04	4.0E-02	1.6E-05	RAIS
Vanadium	1.8E-04	1.9E-02	2.5E-03	2.5E-05	RAIS
Selenium	1.8E-04	1.9E-02	4.0E-02	4.0E-04	
Tin	1.8E-04	1.9E-02	1.0E-03	1.0E-05	RAIS
Dioxins and furans (WHO-TEQ)	2.1E-10	1.7E-08	7.0E-01	6.6E-09	

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Beef

$$\text{Daily chemical intake} = C_B \times \frac{IR_B \times FI \times ME \times EF \times ED}{BW \times AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Beef (IRB) (kg/day)	0.16	Ingestion rate of beef for adults >19 years (enHealth 2012, noted to be the same as P90 from FSANZ 2017)
Fraction ingested that is homegrown (%)	35%	Assume 35% beef intakes from home-sourced meat
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	24	Time at one residence as adult as per MfE (2011)
Body Weight (BW, kg)	70	For male and females combined as per MfE (2011)
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	8760	MfE (2011)

Maximum from sensitive receptors

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
Antimony		8.6E-04	20%	6.9E-04	100%	1.0E-05	2.5E-09	7.7E-09	--		1.1E-05	0%
Arsenic		8.6E-06		8.6E-06	100%	4.0E-06	9.9E-10	3.1E-09	--		1.2E-04	1%
Beryllium		2.0E-03		2.0E-03	100%	3.0E-06	7.4E-10	2.3E-09	--		1.2E-06	0%
Cadmium		8.0E-04	50%	4.0E-04	100%	8.1E-07	2.0E-10	6.2E-10	--		1.5E-06	0%
Chromium (Cr VI assumed)		9.0E-04		9.0E-04	100%	5.5E-06	1.4E-09	4.3E-09	--		4.7E-06	0%
Copper		1.4E-01	33%	9.4E-02	100%	1.0E-05	2.5E-09	7.7E-09	--		8.2E-08	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	2.0E-04	4.9E-08	1.5E-07	--		1.4E-04	2%
Lead		2.0E-04	50%	1.0E-04	100%	6.0E-07	1.5E-10	4.6E-10	--		4.6E-06	0%
Manganese		1.6E-01	50%	8.0E-02	100%	4.0E-07	9.9E-11	3.1E-10	--		3.9E-09	0%
Mercury		2.0E-03	5%	1.9E-03	100%	1.6E-04	3.9E-08	1.2E-07	--		6.4E-05	1%
Nickel		1.2E-02	60%	4.8E-03	100%	3.0E-08	7.4E-12	2.3E-11	--		4.8E-09	0%
Thallium		2.0E-04	10%	1.8E-04	100%	1.6E-05	4.0E-09	1.2E-08	--		6.9E-05	1%
Vanadium		2.0E-03		2.0E-03	100%	2.5E-05	6.2E-09	1.9E-08	--		9.7E-06	0%
Selenium		6.0E-03	25%	4.5E-03	100%	4.0E-04	9.9E-08	3.1E-07	--		6.9E-05	1%
Tin		2.0E-01	50%	1.0E-01	100%	1.0E-05	2.5E-09	7.7E-09	--		7.7E-08	0%
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	100%	6.6E-09	1.6E-12	5.1E-12	--		7.5E-03	94%
								TOTAL			**8.0E-03**	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Exposure to Chemicals via Ingestion of Beef

$$\text{Daily chemical intake} = C_B \times \frac{IR_B \times FI \times ME \times EF \times ED}{BW \times AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Children		
Ingestion Rate of Beef (IRB) (kg/day)	0.085	Ingestion rate of beef by children aged 2-6 years (P90 value) FSANZ (2017)
Fraction ingested that is homegrown (%)	35%	Assume 35% beef intakes from home-sourced meat
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	13	Representative weight as per MfE (2011)
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	2190	MfE (2011)

Maximum from sensitive receptors

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
Antimony		8.6E-04	20%	6.9E-04	100%	1.0E-05	1.8E-09	2.2E-08	--		3.2E-05	0%
Arsenic		8.6E-06		8.6E-06	100%	4.0E-06	7.1E-10	8.8E-09	--		8.2E-05	0%
Beryllium		2.0E-03		2.0E-03	100%	3.0E-06	5.3E-10	6.6E-09	--		3.3E-06	0%
Cadmium		8.0E-04	50%	4.0E-04	100%	8.1E-07	1.4E-10	1.8E-09	--		4.4E-06	0%
Chromium (Cr VI assumed)		9.0E-04		9.0E-04	100%	5.5E-06	9.7E-10	1.2E-08	--		1.4E-05	0%
Copper		1.4E-01	33%	9.4E-02	100%	1.0E-05	1.8E-09	2.2E-08	--		2.4E-07	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	2.0E-04	3.5E-08	4.4E-07	--		3.9E-04	2%
Lead		2.0E-04	50%	1.0E-04	100%	6.0E-07	1.1E-10	1.3E-09	--		1.3E-05	0%
Manganese		1.6E-01	50%	8.0E-02	100%	4.0E-07	7.1E-11	8.8E-10	--		1.1E-08	0%
Mercury		2.0E-03	5%	1.9E-03	100%	1.6E-04	2.8E-08	3.5E-07	--		1.8E-04	1%
Nickel		1.2E-02	60%	4.8E-03	100%	3.0E-08	5.3E-12	6.6E-11	--		1.4E-08	0%
Thallium		2.0E-04	10%	1.8E-04	100%	1.6E-05	2.8E-09	3.5E-08	--		2.0E-04	1%
Vanadium		2.0E-03		2.0E-03	100%	2.5E-05	4.4E-09	5.5E-08	--		2.8E-05	0%
Selenium		6.0E-03	25%	4.5E-03	100%	4.0E-04	7.1E-08	8.8E-07	--		2.0E-04	1%
Tin		2.0E-01	50%	1.0E-01	100%	1.0E-05	1.8E-09	2.2E-08	--		2.2E-07	0%
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	100%	6.6E-09	1.2E-12	1.4E-11	--		2.2E-02	95%
								TOTAL			**2.3E-02**	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Milk ingestion

Calculation of Concentrations in Dairy Milk

Uptake in to milk (dairy cows)

$$C_E=(FI \times IR_C \times C+IR_S \times C_S \times B) \times TF_B$$ (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/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	Based on data from OEHHA 2015 (5% total produce intakes from soil from pasture)
B (bioavailability)	%	100%	

Chemical-specific Inputs and calculations - maximum sensitive receptors

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 or mg/L	
Antimony	1.8E-04	1.9E-02	1.0E-04	2.5E-06	RAIS
Arsenic	3.5E-05	3.8E-03	5.0E-05	2.5E-07	
Beryllium	1.8E-04	1.9E-02	9.0E-07	2.2E-08	
Cadmium	7.1E-06	7.5E-04	2.0E-03	2.0E-06	
Chromium (Cr VI assumed)	1.8E-05	1.9E-03	9.0E-06	2.2E-08	
Copper	1.8E-05	1.9E-03	1.5E-03	3.7E-06	RAIS
Cobalt	1.8E-04	1.9E-02	2.0E-03	4.9E-05	RAIS
Lead	3.5E-05	3.8E-03	6.0E-05	3.0E-07	
Manganese	1.8E-05	1.9E-03	3.5E-04	8.6E-07	RAIS
Mercury	6.9E-05	7.4E-03	7.0E-05	6.7E-07	
Nickel	1.8E-06	1.9E-04	3.0E-05	7.4E-09	
Thallium	7.1E-06	7.5E-04	2.0E-03	2.0E-06	RAIS
Vanadium	1.8E-04	1.9E-02	2.0E-05	4.9E-07	RAIS
Selenium	1.8E-04	1.9E-02	9.0E-03	2.2E-04	
Tin	1.8E-04	1.9E-02	1.0E-03	2.5E-05	RAIS
Dioxins and furans (WHO-TEQ)	2.1E-10	1.7E-08	2.0E-02	4.6E-10	

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Milk

$$\text{Daily chemical intake} = C_M \times \frac{IR_M \times FI \times ME \times EF \times ED}{BW \times AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Milk (IRM) (L/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)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	24	Time at one residence as adult as per MfE (2011)
Body Weight (BW, kg)	70	For male and females combined as per MfE (2011)
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	8760	MfE (2011)

Maximum from sensitive receptors

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-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony		8.6E-04	20%	6.9E-04	100%	2.5E-06	1.4E-08	4.4E-08	--		6.4E-05	0%
Arsenic		8.6E-06		8.6E-06	100%	2.5E-07	1.4E-09	4.4E-09	--		1.6E-04	1%
Beryllium		2.0E-03		2.0E-03	100%	2.2E-08	1.3E-10	3.9E-10	--		2.0E-07	0%
Cadmium		8.0E-04	50%	4.0E-04	100%	2.0E-06	1.1E-08	3.5E-08	--		8.7E-05	1%
Chromium (Cr VI assumed)		9.0E-04		9.0E-04	100%	2.2E-08	1.3E-10	3.9E-10	--		4.4E-07	0%
Copper		1.4E-01	33%	9.4E-02	100%	3.7E-06	2.1E-08	6.6E-08	--		7.0E-07	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	4.9E-05	2.8E-07	8.7E-07	--		7.8E-04	5%
Lead		2.0E-04	50%	1.0E-04	100%	3.0E-07	1.7E-09	5.2E-09	--		5.2E-05	0%
Manganese		1.6E-01	50%	8.0E-02	100%	8.6E-07	4.9E-09	1.5E-08	--		1.9E-07	0%
Mercury		2.0E-03	5%	1.9E-03	100%	6.7E-07	3.8E-09	1.2E-08	--		6.3E-06	0%
Nickel		1.2E-02	60%	4.8E-03	100%	7.4E-09	4.2E-11	1.3E-10	--		2.7E-08	0%
Thallium		2.0E-04	10%	1.8E-04	100%	2.0E-06	1.1E-08	3.5E-08	--		1.9E-04	1%
Vanadium		2.0E-03		2.0E-03	100%	4.9E-07	2.8E-09	8.7E-09	--		4.4E-06	0%
Selenium		6.0E-03	25%	4.5E-03	100%	2.2E-04	1.3E-06	3.9E-06	--		8.7E-04	6%
Tin		2.0E-01	50%	1.0E-01	100%	2.5E-05	1.4E-07	4.4E-07	--		4.4E-06	0%
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	100%	4.6E-10	2.6E-12	8.2E-12	--		1.2E-02	85%
								TOTAL	**--**		**1.4E-02**	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Exposure to Chemicals via Ingestion of Milk

$$\text{Daily chemical intake} = C_M \times \frac{IR_M \times FI \times ME \times EF \times ED}{BW \times AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Children		
Ingestion Rate of Milk (IRM) (L/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)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	13	Representative weight as per MfE (2011)
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	2190	MfE (2011)

Maximum from sensitive receptors

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/L)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony		8.6E-04	20%	6.9E-04	100%	2.5E-06	1.6E-08	2.0E-07	--		2.9E-04	0%
Arsenic		8.6E-06		8.6E-06	100%	2.5E-07	1.6E-09	2.0E-08	--		1.9E-04	0%
Beryllium		2.0E-03		2.0E-03	100%	2.2E-08	1.4E-10	1.8E-09	--		9.0E-07	0%
Cadmium		8.0E-04	50%	4.0E-04	100%	2.0E-06	1.3E-08	1.6E-07	--		4.0E-04	1%
Chromium (Cr VI assumed)		9.0E-04		9.0E-04	100%	2.2E-08	1.4E-10	1.8E-09	--		2.0E-06	0%
Copper		1.4E-01	33%	9.4E-02	100%	3.7E-06	2.4E-08	3.0E-07	--		3.2E-06	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	4.9E-05	3.2E-07	4.0E-06	--		3.6E-03	5%
Lead		2.0E-04	50%	1.0E-04	100%	3.0E-07	1.9E-09	2.4E-08	--		2.4E-04	0%
Manganese		1.6E-01	50%	8.0E-02	100%	8.6E-07	5.6E-09	7.0E-08	--		8.7E-07	0%
Mercury		2.0E-03	5%	1.9E-03	100%	6.7E-07	4.4E-09	5.4E-08	--		2.9E-05	0%
Nickel		1.2E-02	60%	4.8E-03	100%	7.4E-09	4.8E-11	6.0E-10	--		1.2E-07	0%
Thallium		2.0E-04	10%	1.8E-04	100%	2.0E-06	1.3E-08	1.6E-07	--		8.9E-04	1%
Vanadium		2.0E-03		2.0E-03	100%	4.9E-07	3.2E-09	4.0E-08	--		2.0E-05	0%
Selenium		6.0E-03	25%	4.5E-03	100%	2.2E-04	1.4E-06	1.8E-05	--		4.0E-03	6%
Tin		2.0E-01	50%	1.0E-01	100%	2.5E-05	1.6E-07	2.0E-06	--		2.0E-05	0%
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	100%	4.6E-10	3.0E-12	3.7E-11	--		5.6E-02	85%
								TOTAL	**--**		**6.5E-02**	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Lamb ingestion

Calculation of Concentrations in Homegrown Lamb

Uptake in to lamb meat

$$C_E=(FI \times IR_C \times C+IR_S \times C_S \times B) \times TF_B$$ (mg/kg meat – wet weight)

where:
FI = Fraction of grain/crop ingested by lambs each day (unitless)
IRc = Ingestion rate of grain/crop by lambs each day (kg/day)
C = Concentration of chemical in grain/crop eaten by lamb (mg/kg)
IRs = Ingestion rate of soil by lambs each day (kg/day)
Cs = Concentration in soil the lambs ingest (mg/kg)
B = Bioavailability of soil ingested by lambs (%)
TFE = Transfer factor from ingestion to lamb (day/kg)

General Parameters	Units	Value	
FI (fraction of crops ingested from property)		1	Assume 100% of pasture consumed by lambs is grown in the same soil
IRc (ingestion rate of crops)	kg/day	1.1088	4.2% body weight per day dry weight, then correcting for 20% moisture (assuming 22 kg weight)**
IRs (ingestion rate of soil)	kg/day	0.05544	Assumes 5% total produce intakes from soil from pasture, consistent with cattle
B (bioavailability)	%	100%	

** https://www.mla.com.au/contentassets/34ac9a74c56e4dbf8273d2a9bb2900c5/l.lsm.0022_-_production_feeding_for_lamb_growth__.pdf

Chemical-specific Inputs and calculations - maximum sensitive receptors					
Chemical	**Concentration in crops ingested by lambs mg/kg ww**	**Soil Concentration - Agriculture (Cs) mg/kg**	**Transfer factor to lambs day/kg**	**Lamb Concentration mg/kg ww**	
Antimony	1.8E-04	1.9E-02	1.0E-02	1.3E-05	MW adjustment
Arsenic	3.5E-05	3.8E-03	2.1E-02	5.2E-06	MW adjustment
Beryllium	1.8E-04	1.9E-02	3.1E-03	3.9E-06	MW adjustment
Cadmium	7.1E-06	7.5E-04	2.1E-02	1.0E-06	MW adjustment
Chromium (Cr VI assumed)	1.8E-05	1.9E-03	5.7E-02	7.1E-06	MW adjustment
Copper	1.8E-05	1.9E-03	1.0E-01	1.3E-05	MW adjustment
Cobalt	1.8E-04	1.9E-02	2.1E-01	2.6E-04	MW adjustment
Lead	3.5E-05	3.8E-03	3.1E-03	7.8E-07	MW adjustment
Manganese	1.8E-05	1.9E-03	4.2E-03	5.2E-07	MW adjustment
Mercury	6.9E-05	7.4E-03	4.2E-01	2.0E-04	MW adjustment
Nickel	1.8E-06	1.9E-04	3.1E-03	3.9E-08	MW adjustment
Thallium	7.1E-06	7.5E-04	4.2E-01	2.1E-05	MW adjustment
Vanadium	1.8E-04	1.9E-02	2.6E-02	3.2E-05	MW adjustment
Selenium	1.8E-04	1.9E-02	4.2E-01	5.2E-04	MW adjustment
Tin	1.8E-04	1.9E-02	1.0E-02	1.3E-05	MW adjustment
Dioxins and furans (WHO-TEQ)	2.1E-10	1.7E-08	7.3E+00	8.5E-09	MW adjustment

Transfer factors from OEHHA 2015 unless otherwise noted
MW weight adjustment = metabolic weight adjustmenta approach, modifying the TF for beef meet to pigs to acount for differences in tissue trasnfer due to different weights.
Approach adopted for pigs as per OEHHA (2012) to calculate transfer factors Tco as below. Approach also adopted for lambs (cattle = 500 kg and lambs = 22 kg (average for Australian lambs))

$$\text{Pig } Tco_i = (W^{0.75}_{cow}) / (W^{0.75}_{pig}) \times \text{cow } Tco_i$$

Transfer factor adjustment for lambs = 10.4

Exposure to Chemicals via Ingestion of Lamb

$$\text{Daily chemical intake} = C_B \times \frac{IR_B \times FI \times ME \times EF \times ED}{BW \times AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Beef (IRB) (kg/day)	0.085	Ingestion rate of sheep meat for adults, 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)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	24	Time at one residence as adult as per MfE (2011)
Body Weight (BW, kg)	70	For male and females combined as per MfE (2011)
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	8760	MfE (2011)

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Lamb 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)	
Antimony		8.6E-04	20%	6.9E-04	100%	1.3E-05	1.7E-09	5.3E-09	--		7.7E-06	0%
Arsenic		8.6E-06		8.6E-06	100%	5.2E-06	6.7E-10	2.1E-09	--		7.8E-05	1%
Beryllium		2.0E-03		2.0E-03	100%	3.9E-06	5.1E-10	1.6E-09	--		7.9E-07	0%
Cadmium		8.0E-04	50%	4.0E-04	100%	1.0E-06	1.3E-10	4.2E-10	--		1.1E-06	0%
Chromium (Cr VI assumed)		9.0E-04		9.0E-04	100%	7.1E-06	9.3E-10	2.9E-09	--		3.2E-06	0%
Copper		1.4E-01	33%	9.4E-02	100%	1.3E-05	1.7E-09	5.3E-09	--		5.6E-08	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	2.6E-04	3.4E-08	1.1E-07	--		9.4E-05	2%
Lead		2.0E-04	50%	1.0E-04	100%	7.8E-07	1.0E-10	3.2E-10	--		3.2E-06	0%
Manganese		1.6E-01	50%	8.0E-02	100%	5.2E-07	6.7E-11	2.1E-10	--		2.6E-09	0%
Mercury		2.0E-03	5%	1.9E-03	100%	2.0E-04	2.6E-08	8.2E-08	--		4.3E-05	1%
Nickel		1.2E-02	60%	4.8E-03	100%	3.9E-08	5.1E-12	1.6E-11	--		3.3E-09	0%
Thallium		2.0E-04	80%	4.0E-05	100%	2.1E-05	2.7E-09	8.4E-09	--		2.1E-04	4%
Vanadium		2.0E-03		2.0E-03	100%	3.2E-05	4.2E-09	1.3E-08	--		6.6E-06	0%
Selenium		6.0E-03		6.0E-03	100%	5.2E-04	6.7E-08	2.1E-07	--		3.5E-05	1%
Tin		2.0E-01		2.0E-01	100%	1.3E-05	1.7E-09	5.3E-09	--		2.6E-08	0%
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	100%	8.5E-09	1.1E-12	3.4E-12	--		5.1E-03	91%
								TOTAL			**5.6E-03**	

Chemical	Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Exposure to Chemicals via Ingestion of Lamb

$$\text{Daily chemical intake} = C_B \times \frac{IR_B \times FI \times ME \times EF \times ED}{BW \times AT} \quad \text{(mg/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Children		
Ingestion Rate of Beef (IRB) (kg/day)	0.036	Ingestion rate of sheep meat 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)	350	Days at home (normal conditions), as per MfE (2011)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	13	Representative weight as per MfE (2011)
Averaging Time - NonThreshold (Atc, days)	27375	MfE (2011)
Averaging Time - Threshold (Atn, days)	2190	MfE (2011)

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Lamb 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
Antimony		8.6E-04	20%	6.9E-04	100%	1.3E-05	9.6E-10	1.2E-08	--		1.7E-05	0%
Arsenic		8.6E-06		8.6E-06	100%	5.2E-06	3.8E-10	4.8E-09	--		4.5E-05	0%
Beryllium		2.0E-03		2.0E-03	100%	3.9E-06	2.9E-10	3.6E-09	--		1.8E-06	0%
Cadmium		8.0E-04	50%	4.0E-04	100%	1.0E-06	7.7E-11	9.6E-10	--		2.4E-06	0%
Chromium (Cr VI assumed)		9.0E-04		9.0E-04	100%	7.1E-06	5.3E-10	6.6E-09	--		7.3E-06	0%
Copper		1.4E-01	33%	9.4E-02	100%	1.3E-05	9.6E-10	1.2E-08	--		1.3E-07	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	2.6E-04	1.9E-08	2.4E-07	--		2.1E-04	2%
Lead		2.0E-04	50%	1.0E-04	100%	7.8E-07	5.8E-11	7.2E-10	--		7.2E-06	0%
Manganese		1.6E-01	50%	8.0E-02	100%	5.2E-07	3.8E-11	4.8E-10	--		6.0E-09	0%
Mercury		2.0E-03	5%	1.9E-03	100%	2.0E-04	1.5E-08	1.9E-07	--		9.9E-05	1%
Nickel		1.2E-02	60%	4.8E-03	100%	3.9E-08	2.9E-12	3.6E-11	--		7.5E-09	0%
Thallium		2.0E-04	80%	4.0E-05	100%	2.1E-05	1.5E-09	1.9E-08	--		4.8E-04	4%
Vanadium		2.0E-03		2.0E-03	100%	3.2E-05	2.4E-09	3.0E-08	--		1.5E-05	0%
Selenium		6.0E-03		6.0E-03	100%	5.2E-04	3.8E-08	4.8E-07	--		8.0E-05	1%
Tin		2.0E-01		2.0E-01	100%	1.3E-05	9.6E-10	1.2E-08	--		6.0E-08	0%
Dioxins and furans (WHO-TEQ)		1.0E-09	33%	6.7E-10	100%	8.5E-09	6.3E-13	7.9E-12	--		1.2E-02	92%
								TOTAL			**1.3E-02**	

Chemical — Chemical evaluated on the basis of a non-threshold value (hence non-threshold intake used)

Rainwater tanks

Calculation of Concentrations in Rainwater tank

CW = DM/(VR*Kd*ρ) **(mg/L)**

where:

DM =	Mass of dust deposited on roof each year that enters tank (mg) = DR x Area x 0.1 x 1 year
DR =	Deposition rate from model for TSP (mg/m^2/year)
Area =	Area of roof (m^2)
VR =	Volume of water collected from roof over year (L) = (R x Area x Rc x 1000)/1000
R =	Rainfall each year (mm)
ρ =	Soil bulk-density (g/cm^3)
Rc =	Runoff coefficient (unitless)
Kd =	Soil-water partition coefficient (cm^3/g)
1000 =	Conversion from mm to m; and conversion from m^3 to L

General Parameters			
Average rainfall (R)	mm	564.1	average historical yearly rainfall for Oamaru
Roof area (Area)	m^2	150	average house size for Waimate area in 2022
Runoff coefficient (Rc)	-	0.7	assumes 30% loss in capture into tank
Volume of rainwater (VR)	L	59230.5	calculated
Bulk density of deposited dust	g/cm^3	0.5	assumed for loose deposited dust on roof (similar to upper end measured for powders)

Chemical-specific Inputs and calculations - maximum sensitive receptors						
Chemical	Deposited dust entering tank: Deposition Rate TSP (DR) mg/m^2/year	Deposited dust entering tank: Mass deposited each year into tank (DM) mg	Kd (cm^3/g)	Particulate Concentration in water mg/L	Dissolved Concentration in water mg/L	Total (particulate and dissolved) - worst-case mg/L
Antimony	1.3E-01	1.94E+00	45	3.3E-05	1.5E-06	3.4E-05
Arsenic	2.6E-02	3.88E-01	29	6.5E-06	4.5E-07	7.0E-06
Beryllium	1.3E-01	1.94E+00	790	3.3E-05	8.3E-08	3.3E-05
Cadmium	5.2E-03	7.76E-02	75	1.3E-06	3.5E-08	1.3E-06
Chromium (Cr VI assumed)	1.3E-02	1.94E-01	1800000	3.3E-06	3.6E-12	3.3E-06
Copper	1.3E-02	1.94E-01	35	3.3E-06	1.9E-07	3.5E-06
Cobalt	1.3E-01	1.94E+00	45	3.3E-05	1.5E-06	3.4E-05
Lead	2.6E-02	3.88E-01	900	6.5E-06	1.5E-08	6.6E-06
Manganese	1.3E-02	1.94E-01	65	3.3E-06	1.0E-07	3.4E-06
Mercury	5.0E-02	7.57E-01	52	1.3E-05	4.9E-07	1.3E-05
Nickel	1.3E-03	1.94E-02	65	3.3E-07	1.0E-08	3.4E-07
Thallium	5.2E-03	7.76E-02	71	1.3E-06	3.7E-08	1.3E-06
Vanadium	1.3E-01	1.94E+00	1000	3.3E-05	6.5E-08	3.3E-05
Selenium	1.3E-01	1.94E+00	5	3.3E-05	1.3E-05	4.6E-05
Tin	1.3E-01	1.94E+00	250	3.3E-05	2.6E-07	3.3E-05
Dioxins and furans (WHO-TEQ)	1.5E-07	2.28E-06	630957344	3.9E-11	1.2E-19	3.9E-11