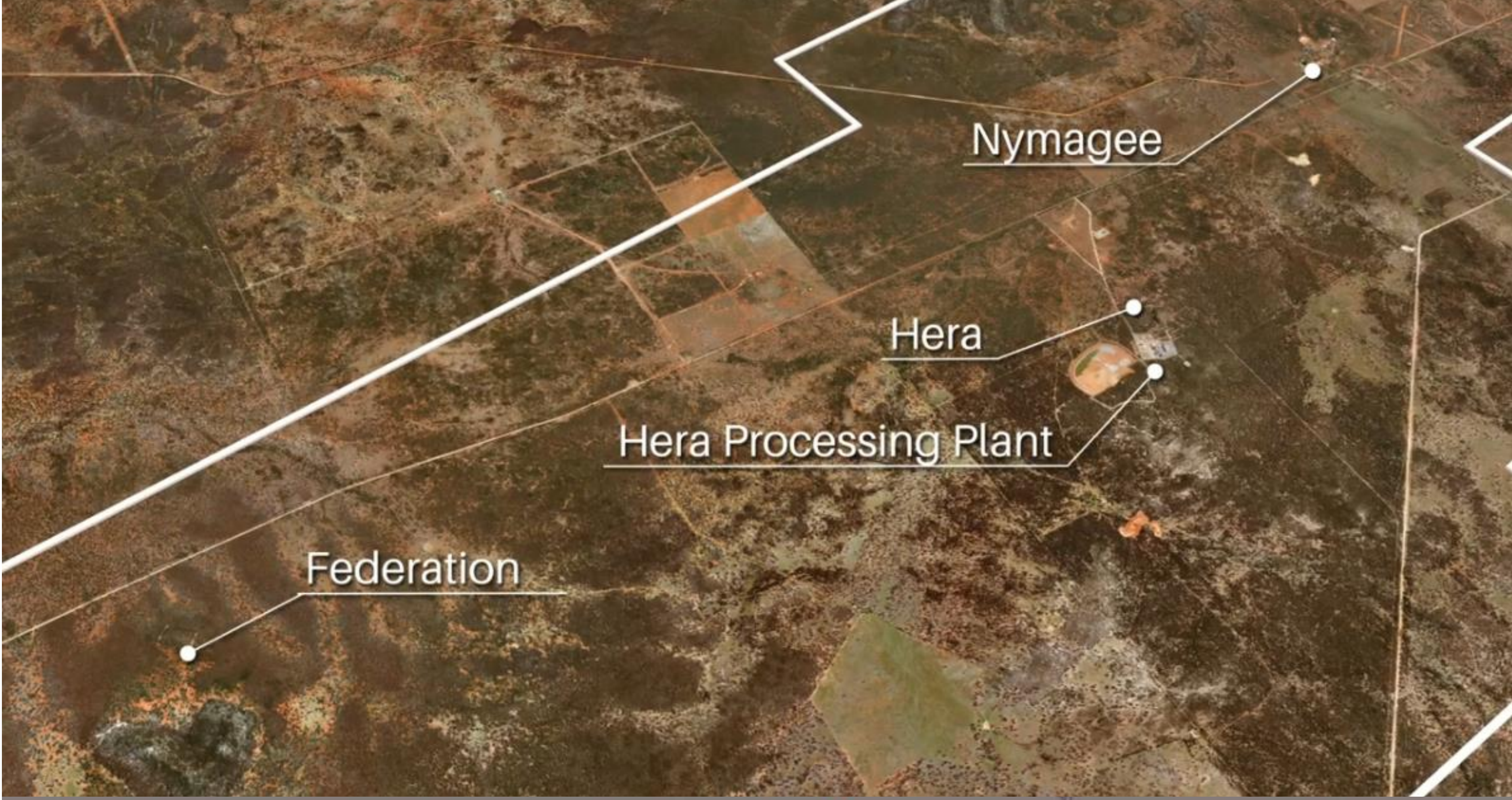

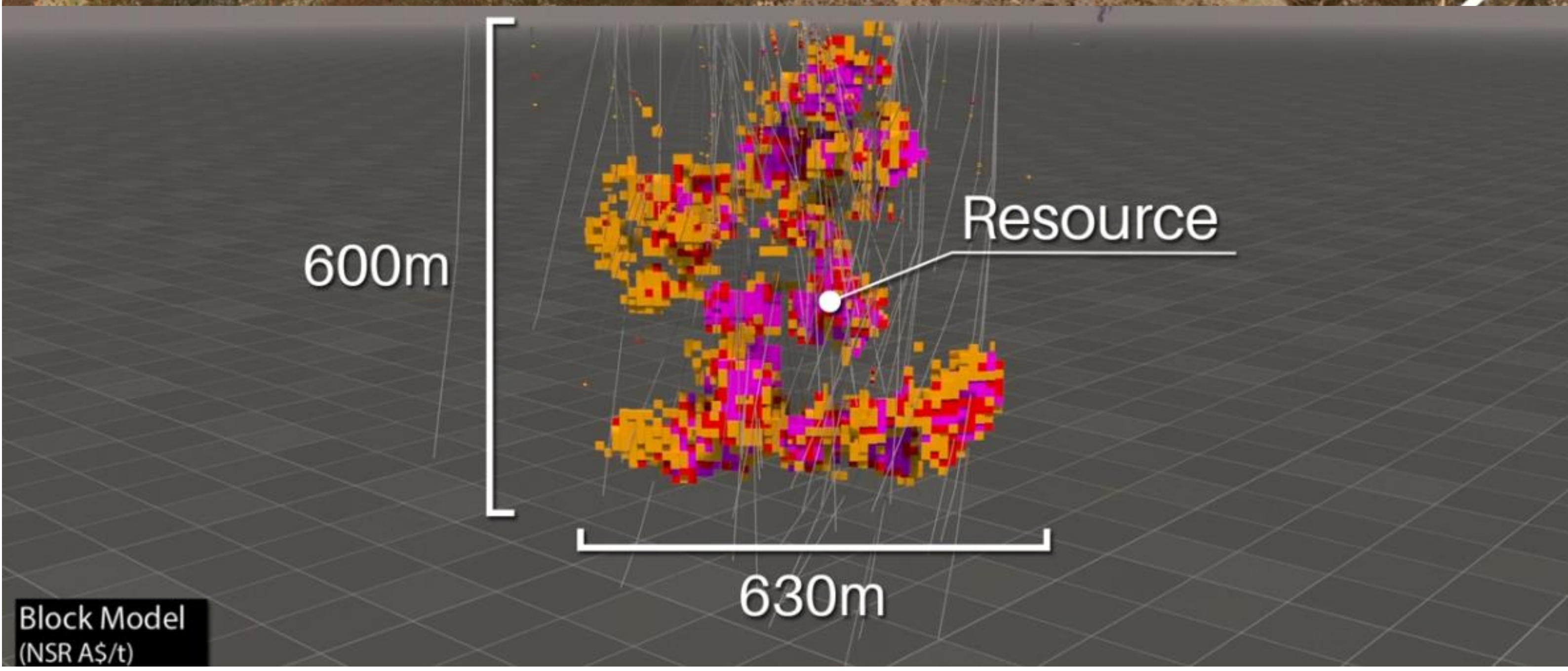

Federation Project - Human Health Assessment

Prepared for: SLR Consulting Australia Pty Ltd and Hera Resources Pty Ltd

9 November 2021

EnRiskS

Document History and Status

Report Reference	AS/21/FPR001
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Limitations

Environmental Risk Sciences has prepared this report for the use of SLR Consulting Aurelia Pty Ltd and Hera Resources Pty Ltd in accordance with the usual care and thoroughness of the consulting profession. It is based on generally accepted practices and standards at the time it was prepared. No other warranty, expressed or implied, is made as to the professional advice included in this report.

It is prepared in accordance with the scope of work and for the purpose outlined in the Section 1 of this report.

The methodology adopted, and sources of information used are outlined in this report. Environmental Risk Sciences has made no independent verification of this information beyond the agreed scope of works and assumes no responsibility for any inaccuracies or omissions. No indications were found that information contained in the reports provided for use in this assessment was false.

This report was prepared in October and November 2021 and is based on the information provided and reviewed at that time. Environmental Risk Sciences disclaims responsibility for any changes that may have occurred after this time.

This report should be read in full. No responsibility is accepted for use of any part of this report in any other context or for any other purpose or by third parties. This report does not purport to give legal advice. Legal advice can only be given by qualified legal practitioners.

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Appendices

Glossary of Terms and Abbreviations

Term	Definition
AAQ	Ambient air quality.
ABS	Australian Bureau of Statistics.
Acute exposure	Contact with a substance that occurs once or for only a short time (up to 14 days).
Absorption	The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.
Adverse health effect	A change in body function or cell structure that might lead to disease or health problems.
Aerodynamic diameter	Airborne particles have irregular shapes, their aerodynamic behaviour is expressed in terms of the diameter of an idealised spherical particle.
AIHW	Australian Institute of Health and Welfare.
ANZECC	Australia and New Zealand Environment and Conservation Council.
AQGGA	Air Quality and Greenhouse Gas Assessment.
ATSDR	Agency for Toxic Substances and Disease Register.
Background level	An average or expected amount of a substance or material in a specific environment, or typical amounts of substances that occur naturally in an environment.
Biodegradation	Decomposition or breakdown of a substance through the action of micro-organisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).
Body burden	The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.
Carcinogen	A substance that causes cancer.
CCME	Canadian Council of Ministers of the Environment.
Chronic exposure	Contact with a substance or stressor that occurs over a long time (more than one year) [compare with acute exposure and intermediate duration exposure].
COMEAP	Committee on the Medical Effects of Air Pollutants.
dBA	Decibels (A-weighted).
DEC	NSW Department of Environment and Conservation.
DECC	NSW Department of Environment and Climate Change.
DECCW	NSW Department of Environment, Climate Change and Water.
DEFRA	Department for Environment, Food & Rural Affairs.
DEH	Australian Department of Environment and Heritage.
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.
EIS	Environmental Impact Statement.
EL	Exploration Licence.
ENM	Environmental Noise Model.
EPHC	Environment Protection and Heritage Council.
EU	European Union.
Exposure	Contact with a substance by swallowing, breathing, or touching the skin or eyes. Also includes contact with a stressor such as noise or vibration. Exposure may be short term [acute exposure], of intermediate duration, or long term [chronic exposure].
Exposure assessment	The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

Term	Definition
Exposure pathway	The route a substance takes from its source (where it began) to its endpoint (where it ends), and how people can come into contact with (or get exposed) to it. An exposure pathway has five parts: a source of contamination (such as chemical substance leakage into the subsurface); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.
Genotoxic carcinogen	These are carcinogens that have the potential to result in genetic (DNA) damage (gene mutation, gene amplification, chromosomal rearrangement). Where this occurs, the damage may be sufficient to result in the initiation of cancer at some time during a lifetime.
Guideline value	Guideline value is a concentration in soil, sediment, water, biota or air (established by relevant regulatory authorities such as the NSW Department of Environment and Conservation (DEC) or institutions such as the National Health and Medical Research Council (NHMRC), Australia and New Zealand Environment and Conservation Council (ANZECC) and World Health Organization (WHO)), that is used to identify conditions below which no adverse effects, nuisance or indirect health effects are expected. The derivation of a guideline value utilises relevant studies on animals or humans and relevant factors to account for inter and intra-species variations and uncertainty factors. Separate guidelines may be identified for protection of human health and the environment. Dependent on the source, guidelines would have different names, such as investigation level, trigger value and ambient guideline.
HHRA	Human health risk assessment.
HI	Hazard Index.
IARC	International Agency for Research on Cancer.
ICNG	Interim Construction Noise Guideline.
I-INCE	International Institute of Noise Control Engineering.
Inhalation	The act of breathing.
Intermediate exposure	Contact with a substance that occurs for more than 14 days and less than a year [compared with acute exposure and chronic exposure].
LGA	Local Government Area.
LOAEL	Lowest-observed-adverse-effect level.
LOR	Limit of Reporting.
Metabolism	The conversion or breakdown of a substance from one form to another by a living organism.
ML	Mining Lease.
Morbidity	This is the condition of being ill, diseased or unhealthy. This can include acute illness (which has a sudden onset and may improve or worsen over a short period of time) as well as chronic illness (which can present and progress slowly over a long period of time).
Mortality	This is the condition of being dead. It may be presented as the number of deaths in a population over time, either in general or due to a specific cause.
NCAs	Noise catchment areas.
NCG	Noise Criteria Guideline (various, as referenced in the report).
NEPC	National Environment Protection Council.
NEPM	National Environment Protection Measure.
NHMRC	National Health and Medical Research Council.
NO_2	Nitrogen dioxide.
NOx	Nitrogen oxides.
NSW	New South Wales.
NSW EPA	NSW Environment Protection Authority.

Term	Definition
OEH	NSW Office of Environment and Heritage.
OEHHA	Office of Environmental Health Hazard Assessment, California Environment Protection Agency (Cal EPA).
PM	Particulate matter.
$PM_{2.5}$	Particulate matter of aerodynamic diameter 2.5 micrometres (µm) and less.
PM_{10}	Particulate matter of aerodynamic diameter 10 micrometres (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).
RBL	Rating Background Level.
Receptor	An assessed location for potential air, noise or blasting impacts. Typically, receptors are residences, however can include commercial and industrial premises, places of worship, schools, etc. Also known as receivers.
Receptor population	People who could come into contact with hazardous substances [see exposure pathway].
Risk	The probability that something would cause injury or harm.
ROM	Run-of-mine.
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].
SEARs	Secretary's Environmental Assessment Requirements.
SEIFA	Socio-Economic Index for Areas.
TCEQ	Texas Commission on Environmental Quality.
Toxicity	The degree of danger posed by a substance to human, animal or plant life.
Toxicity data	Characterisation or quantitative value estimated (by recognised authorities) for each individual chemical substance for relevant exposure pathway (inhalation, oral or dermal), with special emphasis on dose-response characteristics. The data are based on based on available toxicity studies relevant to humans and/or animals and relevant safety factors.
Toxicological profile	An assessment that examines, summarises, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.
Toxicology	The study of the harmful effects of substances on humans or animals.
TSP	Total suspended particulates.
UK	United Kingdom.
US	United States of America.
USEPA	United States Environmental Protection Agency.
WHO	World Health Organization.
mg/m^3	Milligrams per cubic metre.
mg	Milligram
$\mu g/m^3$	Micrograms per cubic metre.
µm	Micrometre.

Section 1. Introduction

1.1 Background

Environmental Risk Sciences Pty Ltd (enRiskS) has been commissioned by SLR Consulting Pty Ltd (SLR) and Hera Resources Pty Limited (Hera Resources) to undertake a human health risk assessment (HHRA) in relation to the Federation project, which is a State Significant Development (SSD) in New South Wales (NSW).

Hera Resources is a wholly owned subsidiary of Aurelia Metals Limited (Aurelia). Hera Resources currently own and operate the Hera Mine located approximately 80km south-east of Cobar and approximately 5km south of the township of Nymagee in western NSW. Aurelia owns and operates the Peak Gold Mine (PGM) near Cobar in western NSW.

Hera Resources is evaluating the development of the Federation Project (the Project), a proposed underground metalliferous mine development. The Project comprises underground mining activities and surface infrastructure at the Federation Site, amendments at Hera Mine to facilitate processing of ore from the Federation Site, and a Services Corridor connecting the Federation Site with Hera Mine. The Federation Site is located approximately 15km south of the Nymagee township and 10km south of the Hera Mine, refer to **Figure 1.1.**

The Project is a proposed underground mine development which will establish and operate underground gold, silver and metalliferous mining activities with a projected 7 million tonnes (Mt) of ore extracted over a period of 12 to 14 years.

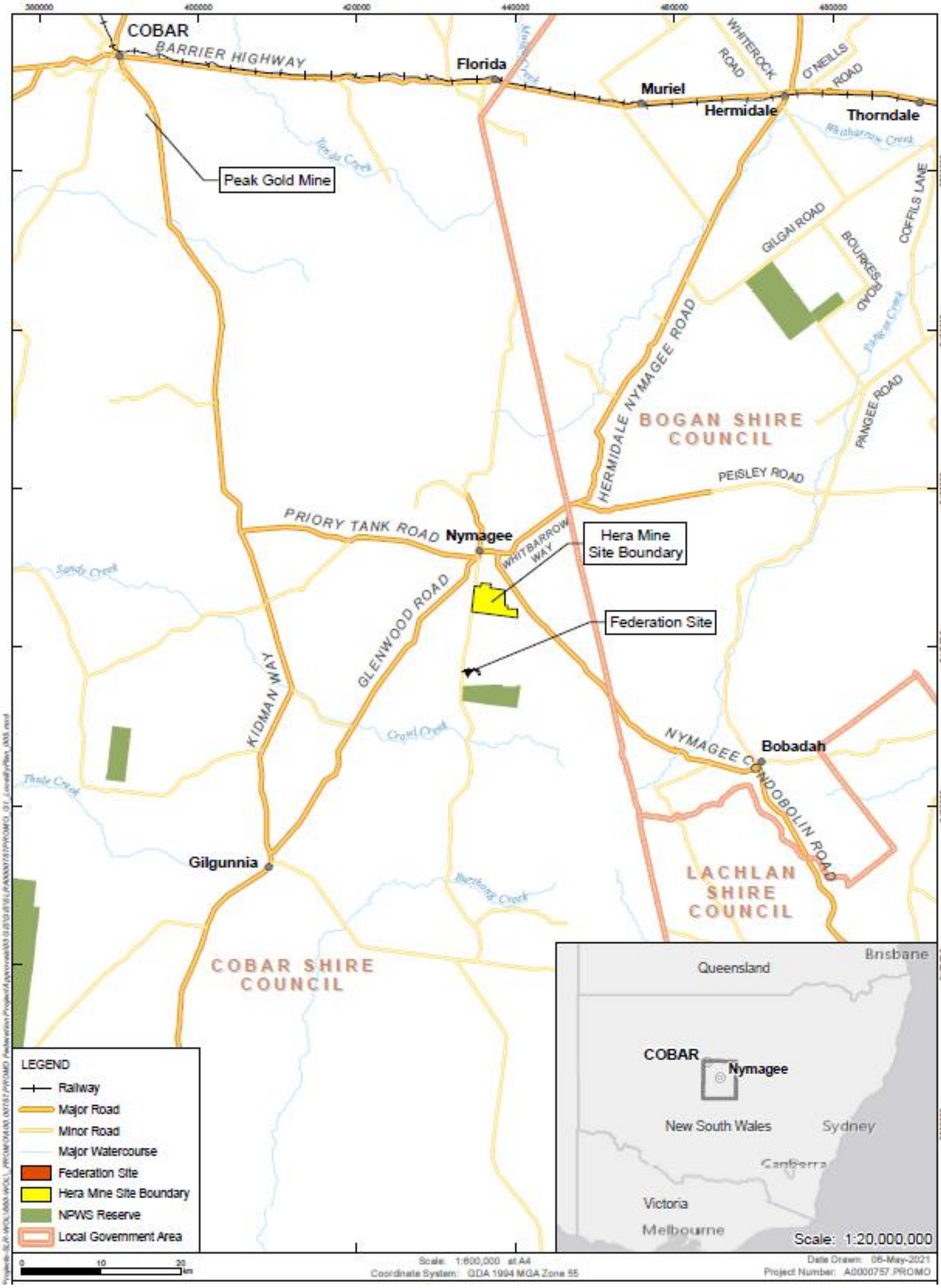


Figure 1.1: Location of Federation and Hera Mine Sites

1.2 Secretary's Environmental Assessment Requirements

The Secretary's Environmental Assessment Requirements (SEARs) for this SSD does not include a specific requirement for the conduct of a health risk assessment for the Environmental Impact Statement (EIS). However as impacts of the proposed Project on health is of concern to the community surrounding the Project, a HHRA has been prepared to assess potential impacts relevant to the Project on the community, including air quality and noise.

1.3 Objectives

The overall objective of the HHRA will be to provide an assessment of impacts to human health in relation to the proposed Project, specifically in relation to the impact of changes in air quality and noise on the health of the local community.

No assessment of impacts to on-site workers is presented. Workplace health and safety is expected to be managed separately through application of the NSW *Work Health and Safety Act 2011* and NSW *Work Health and Safety (Mines and Petroleum Sites) Act 2013*, and associated regulations.

1.4 Methodology

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

- enHealth Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a) (as required in the SEARs).
- State Environmental Planning Policy No. 33 - Hazardous and Offensive Development (NSW Government 2014).
- National Environment Protection Council (NEPC) National Environment Protection (Ambient Air Quality) Measure (NEPM) (NEPC 2021).
- National Environmental Protection Measure – Assessment of Site Contamination including:
    - Schedule B1 Investigation Levels for Soil and Groundwater (NEPC 1999 amended 2013a).
    - Schedule B4 Guideline on Health Risk Assessment Methodology (NEPC 1999 amended 2013b).
    - Schedule B6 Guideline on Risk Based Assessment of Groundwater Contamination (NEPC 1999 amended 2013c).
    - Schedule B7 Guideline on Health-Based Investigation Levels (NEPC 1999 amended 2013d).
    - Schedule B8 Guideline on Community Consultation and Risk Communication (NEPC 1999 amended 2013e).
- NSW Environment Protection Authority (EPA) Approved Methods for the Modelling and Assessment of Air Pollutants in New South Wales (NSW EPA 2017a).
- NSW Noise Policy for Industry (NSW EPA 2017b).
- National Health and Medical Research Council (NHMRC) Australian Drinking Water Guidelines (NHMRC 2011 updated 2018).

Where relevant, additional guidance has been obtained from relevant Australian and International guidance, such as that available from the United States Environmental Protection Agency (USEPA) and the World Health Organization (WHO), consistent with current industry best practice.

1.5 Available information

The HHRA has been prepared on the basis of information available for the Project, including information and data provided by other technical specialists, as detailed below:

- ERM 2021, Federation Project, Air Quality and Greenhouse Gas Assessment (AQGGA).
- Muller Acoustic Consulting (MAC) 2021, Noise and Vibration Impact Assessment, Federation Project, Nymagee, NSW (NVIA).
- SLR 2021, Community and Stakeholder Participation Strategy.

Section 2. Project description

2.1 Background

Hera Resources Pty Limited (Hera Resources) is a wholly owned subsidiary of Aurelia Metals Limited (Aurelia). Hera Resources currently own and operate the Hera Mine located approximately 80km south-east of Cobar and approximately 5km south of the township of Nymagee in western NSW. Aurelia owns and operates the Peak Gold Mine (PGM) near Cobar in western NSW.

Hera Mine has been operational since 2012 and produces gold and silver doré (unrefined bars) and a zinc/lead concentrate. Waste rock and metalliferous ore are extracted using underground open stope mining methods and underground load and haul operations. Hera Mine is approved to process up to 505,000 tonnes of ore on site annually, with lead/zinc concentrate transported to the Hermidale rail siding located approximately 75km to the north-east. The Hera Mine Project Approval was modified in June 2021 to allow for operations up to 31 December 2025.

In April 2019 high grade lead, zinc and gold mineralisation was discovered at the Federation deposit. Subsequent surface drilling programs have delineated a substantial gold-lead-zinc-copper-silver mineral deposit. Hera Resources is evaluating the development of a satellite underground mine at the Federation Site that leverages established infrastructure at the Hera Mine to minimise environmental impacts and allow for the continuation of mining operations in the Nymagee area. Mining of the Federation deposit will allow for a transition of mining operations from Hera Mine to Federation, as ore from the Federation deposit replaces ore from the Hera Mine.

2.2 Overview of the Project

The Project is a proposed underground mine development located in central-western NSW, approximately 15 km south of Nymagee and 10 km south of Hera Mine. The Hera Mine is also owned and operated by Hera Resources.

The Project comprises:

- The establishment and operation of underground gold and metalliferous mining activities, with supporting surface infrastructure, mining approximately 6.95 million tonnes (Mt) of ore over a period of 12 to 14 years, referred to as the Federation Site.
- Amendments at the Hera Mine to facilitate mining and processing of Federation deposit ore, including new process plant and disposal of tailings in the Hera Mine tailings storage facility (TSF).
- Services Corridor between the Federation Site and Hera Mine, including powerline, water pipeline, and access track and potentially a tailings pipeline.

The majority of ore produced will be sent to Hera Mine for processing. However up to 200 ktpa will be transported to PGM during the initial four years of processing (total of 750 kt over this period), whilst the new processing plant at Hera Mine is being commissioned and ramped up.

Access to the underground mine will be via a portal developed through the base of a box cut. The main decline will be developed to gain access to all production levels, where stopes will be excavated. The loosened ore from the stopes will be brought to the surface via underground truck and placed on the Federation Site Run of Mine (ROM) ore stockpile near the boxcut. Ore will then be transported by surface trucks via Burthong Road to the Hera Mine ROM stockpile at the Hera Mine process plant.

Figure 1.1 shows the local area, the location of the existing Hera Mine and the proposed location of the Federation Site.

2.3 Federation Site

The Federation Site will utilise a surface box cut and decline to address the underground workings, with the mining method comprising a mix of longhole stoping and transverse longhole stoping.

Ore from the Federation Site is proposed to be transported approximately 10 km along Burthong Road and will be processed at Hera Mine. Ore proposed to be processed at Peak Mine will be transported in trucks of an approximate 50 t payload along Burthong Road, Priory tank Road and Kidman Way.

Figure 2.1 shows the Federation Site plan and on-site features.

2.4 Hera Mine

Hera Mine infrastructure is proposed to be modified to facilitate the Federation Project. The following activities at Hera Mine have been proposed:

- Amendments to facilitate mining and processing of Federation ore, including new process plant and disposal of tailings in the approved Hera Mine tailings storage facility (TSF).
- The new process plant will recover gold in doré and produce separate copper, lead and zinc concentrates. Up to 750 kt of ore will be processed annually at the new plant. Up to 200 kt of ore will be processed annually at Peak Mine.
- Sixty percent of the total tailings from Hera Mine process plant will be used for paste backfilling of the stope voids at Federation Site. The remaining tailings will be disposed within the approved TSF at Hera Mine.
- Approximately 100-150 ktpa of concentrate from Hera Mine will be transported by road trains to Hermidale Siding, approximately 100 km north of Hera Mine, via Hermidale Nymagee Road.

Figure 2.2 presents the Hera Mine site plan.

2.5 Transitional Period

It is anticipated that approval for the Project will be obtained in early 2023. Prior to the construction and operation of the Federation Project, an Exploration Decline Program will be undertaken. This activity will be undertaken under a separate approval to that being sought for the Project. The main objectives of the Exploration Decline Program are to further define the mineral resources associated with the Federation deposit, including permitting drilling of exploration drill holes from underground.

Key components of the Exploration Decline Program include:

- Establishment of a surface infrastructure area required to support the exploration decline.
- Development of a box cut, portal, exploration decline, two ventilation rises and one escapeway.

- Transportation to and storage of waste rock within the surface infrastructure area, with subsequent transport of waste rock to Hera Mine.
- Establishment and use of an approximately 14.8 km surface pipeline to transfer water from the exploration decline to Hera Mine.
- Exploration drilling from the exploration decline.
- Extraction of one or more bulk samples together totalling no more than 20,000 t and transportation of that material to Hera Mine processing plant via Burthong Road.

It is anticipated that the Exploration Decline Program will commence in November 2021 with the surface infrastructure area established and waste rock being generated from the decline. It is anticipated that ore from the bulk sample will be extracted and processed between the third quarter of 2022 and first quarter of 2023. Based on the current schedule for the Project, there will be a transitional period between Exploration Decline Program activities, mining operations at Hera Mine, and Project construction and operations. Following approval of the Project:

- Construction of Project infrastructure (including the new process plant) will commence in the first half of 2023.
- Exploration Decline Program activities will transition into mining operations at the Federation Site.
- Hera Mine operations may continue over a period of 6 to 12 months.

From early 2024, it is anticipated that all activities will be related to the Project operations. The operational workforce numbers will be transitioned from Hera Mine operations to Project operations.

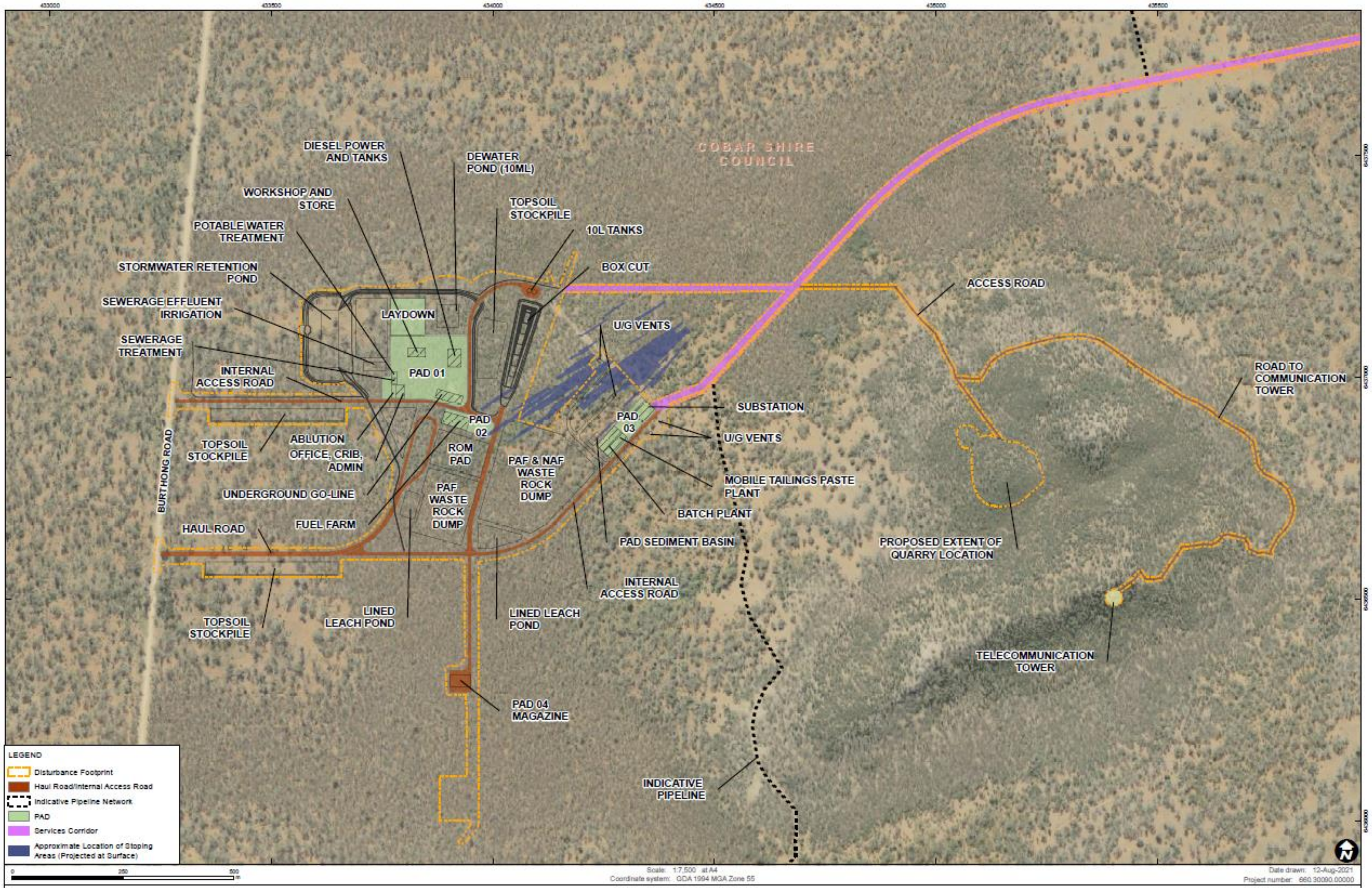


Figure 2.1: Federation Site Plan

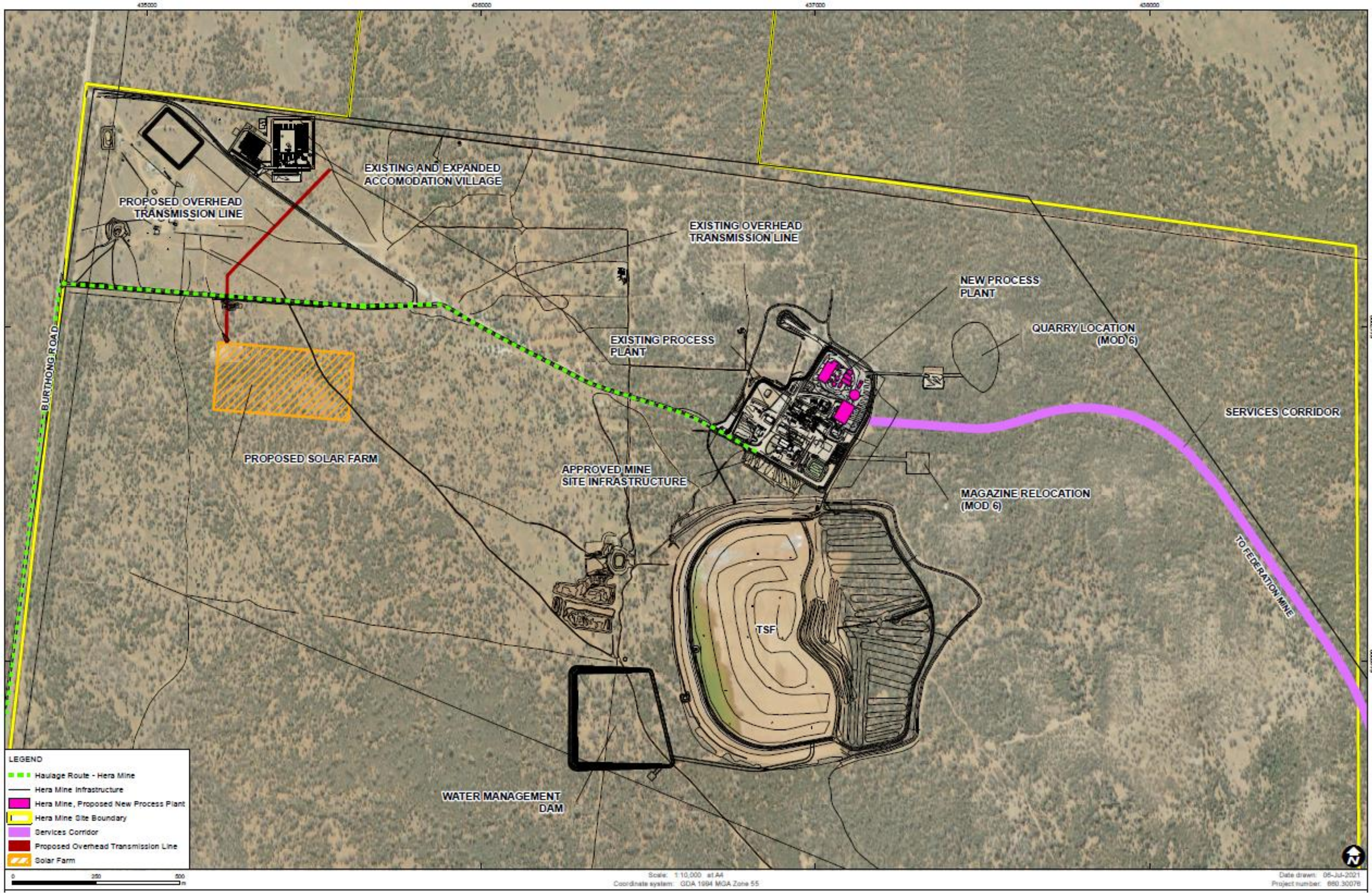


Figure 2.2: Hera Mine Site Plan

2.6 Local setting

The Project site is located in central New South Wales, to the south of the small town of Nymagee, which is approximately 100 km to the southeast of Cobar. The Project sits within Cobar Shire Council and Cobar local government area (LGA).

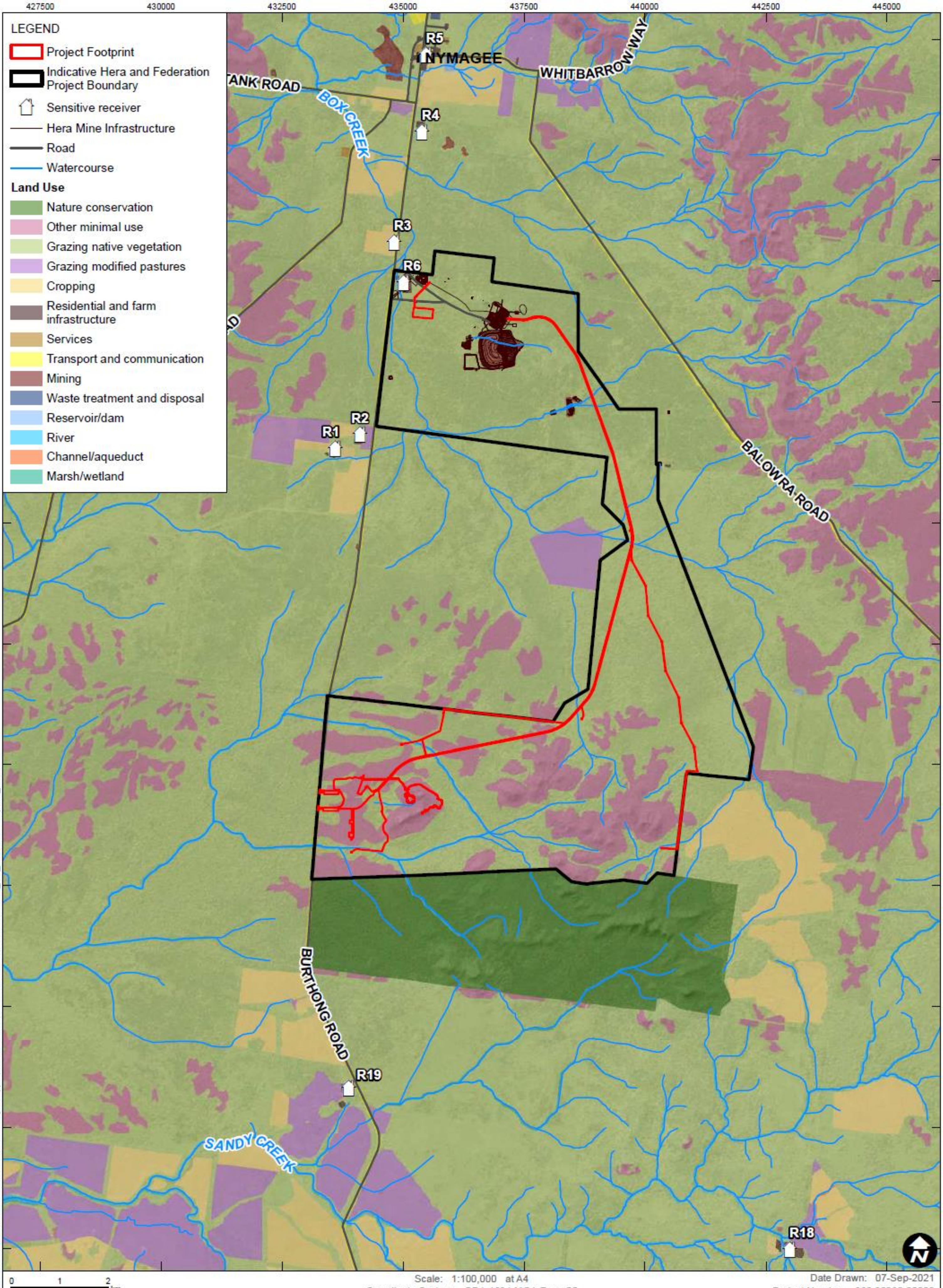
The area has a history of mining and agricultural uses. The land surrounding the Project site is used for a range of agricultural use including cropping and grazing. Some of the land surrounding the Project is designated as nature conservation areas.

Cobar has a semi-arid climate with hot summers and cool to mild winters. Winter nights can be quite cold. On average, rainfall tends to be uniformly distributed throughout the year, with a mean annual rainfall for Cobar MO of 390 mm. The rainfall is however extremely variable, and this is particularly so in late summer and early spring when the highest observed falls have been in excess of 200 mm in any one month (BoM).

The assessment of potential air and noise impacts from Project related activities on the surrounding community has focused on the closest sensitive receptors to the Project, including receptors located adjacent to haul routes between Federation Site and Hera Mine. The receptors evaluated are shown on **Figure 2.3** (along with the various land uses in the area surrounding the Project), and include the following (noting that R6 is the Hera Mine accommodation village)

Table 2.1: Sensitive receptors surrounding the Project site

Receptor number/ID	Address	Land use/description
R1	1245 Burthong Road	Rural residential
R2	688 Burthong Road	Rural residential
R3	224 Burthong Road	Rural residential
R4	39 Burthong Road	Rural residential
R5	Nymagee Village	Residential
R18	2781 Balowra Road	Rural residential
R19	2120 Burthong Road	Rural residential



Scale: 1:100,000 at A4
Coordinate System: GDA 1994 MGA Zone 55

Date Drawn: 07-Sep-2021
Project Number: 660.30090.00000

Data Source: Basedata NSW SS, 2019
Aerial imagery supplied by © Department of Customer Service 2020
Land Use © State Government of NSW and Department of Planning, Industry and Environment 2020

Figure 2.3: Location of sensitive receptors surrounding the Project

Section 3. Community profile

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

The Project is situated in an area that includes existing agricultural and rural residential properties as well as residential properties located in the township of Nymagee.

The boundary of the community evaluated in this assessment has been determined based on modelling completed to evaluate key potential health impacts, specifically air quality and noise, with the area evaluated encompassing the residential receptors presented in **Table 2.1** and shown on **Figure 2.3**.

The Project and all relevant community receptors are located within the Cobar Local government area (LGA).

Table 3.1 presents a summary of the population within the Cobar LGA and the state suburb of Nymagee (based on 2016 Census and 2016 Socio-Economic data from the Australian Bureau of Statistics [ABS]) with comparison to NSW and Australia.

Table 3.1: Summary of populations surrounding the Project

Indicator	State suburb Nymagee	LGA Cobar	NSW	Australia
Total population	100	4,647	7,480,228	23,401,892
Population 0 - 4 years	4%	7.9%	6.2%	6.3%
Population 5 - 19 years	15%	19.7%	18.3%	18.5%
Population 20 - 64 years	55%	58.7%	59.2%	59.6%
Population 65 years and over	22%	13.8%	16.3%	15.7%
Median age	48	36	38	38
Average household size	2.3	2.4	2.6	2.6
Unemployment (in June 2021)	NA	2.7%	6.0%	6.2%
Tertiary or technical institution	NA**	9.8%	22.4%	22%
SEIFA IRSD	922	968	--	--
SEIFA IRSD rank	1	3	--	--
Indigenous	16%	13.7%	2.9%	2.8%
Born overseas	0%	7%	34.5%	26.3%

Most data presented in the table derived from the ABS 2016 Census (ABS 2016).
* Data presented for unemployment is based on available data (Australian Government 2018) to June 2021: https://lmip.gov.au/default.aspx?LMIP/Downloads/SmallAreaLabourMarketsSALM
** No data available as population size too small (as data confidentiality may be affected in small population areas)

SEIFA IRSD = index of socioeconomic disadvantage, rank relates to rank in Australia that ranges from
1 = most disadvantaged to 5 = least disadvantaged. Ranks lower than 3 are more disadvantaged than Australia on average.
Shading relates to comparison against NSW:

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.

statistics/data materially different to that of NSW and Australia, however this indicator is not a clear determinant of higher or lower vulnerability to health stressors.

Based on the population data available and presented in **Table 3.1**, the community in Nymagee and Cobar generally have a similar age distribution as NSW and Australia, noting that Nymagee has a slightly older population. Nymagee and the Cobar LGA also has a higher proportion of the population that is indigenous with the smaller population of Nymagee considered to be socioeconomically disadvantaged. The area of Cobar has a lower rate of unemployment when compared with NSW and Australia.

The indicators outlined in **Table 3.1** reflect the vulnerability of the population and its ability to adapt to environmental stresses. In general, the Nymagee population has aspects that may indicate some parts of the population may be more vulnerable relative to the rest of NSW.

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

The population adjacent to the Project site, as well as Cobar LGA is small and health data is not available that specifically relates to this population.

The Project is located within the Western NSW Local Health District (WNSWLHD). WNSWLHD is one of the largest Local Health Districts in New South Wales covering an area of 246,676 km^2 (similar to the size of Britain). The district incorporates a resident population of approximately 276,000 people. Aboriginal and Torres Strait Islander people living in the Local Health District represent 11.1% of the total population. This is significantly higher than the NSW average of 2.1%. In general, the district has some of the most vulnerable population in NSW and even Australia who generally have a lower socio-economic status, shorter life expectancy, and poorer health than other people living in NSW. More people also have at least one of the risk factors that contribute to poorer health including smoking, harmful use of alcohol, obesity and lack of physical activity[1].

Table 3.2 presents a summary of the general population health relevant to the area, based on currently available data. The table presents available information on health-related behaviours (i.e. key lifestyle and behaviours factors known to be important to health) and indicators for the burden of disease within the WNSWLHD with data for Cobar LGA where available. These data are compared with data for NSW.

[1] https://wnswlhd.health.nsw.gov.au/our-organisation/our-story/welcome

Table 3.2: Summary of health indicators/data

Health indicator/data[1]	Western NSW LHD	NSW
Health behaviours (95% confidence interval)		
Adults - compliance with fruit consumption guidelines (2020)	43.9% (34.3% - 53.2%)	40.3% (38.6% – 42%)
Adults - compliance with vegetable consumption guidelines (2020)	4.8% (2.1% - 7.6%)	5.9% (5.2% – 6.7%)
Children - compliance with fruit consumption guidelines (2019-2020)	72.6% (64.8% - 80.4%)	64.2% (61.7% - 66.7%)
Children - compliance with vegetable consumption guidelines (2019-2020)	3.5% (0.7% - 6.3%)	5.2% (4.1% - 6.3%)
Adults - increased lifetime risk of alcohol related harm (2020)	35.8% (26.3% - 45.2%)	32.5% (30.8% - 34.2%)
Adults - body weight (overweight) (2020)	41.3% (31.4% - 51.2%)	34.3% (32.7% - 35.9%)
Adults - body weight (obese) (2020)	29.9% (22.2% - 37.6%)	22.5% (21.1% - 24.0%)
Adults – sufficient physical activity (2020)	60.3% (51% - 69.7%)	61.7% (60.0% - 63.4%)
Children – adequate physical activity (2019-2020)	17.3% (10.7% - 23.8%)	18.1% (15.7% - 20.4%)
Current smoker, adult (2020)	12.5% (7.0% - 18.0%)	13.3% (12.1% - 14.5%)
Burden of disease (95% confidence interval) as rate per 100,000 unless indicated otherwise		
Morbidity - cardiovascular disease hospitalisations (all ages, 2019-2020)	1915.2 (1869.6 – 1961.6)	1583.8 (1475.9 – 1591.8)
Morbidity – respiratory disease hospitalisations (all ages, 2018-2019)	2157.5 (2105.6 – 2210.3)	1675.2 (1666.4 – 1684.0)
Mortality – all causes, all ages (2019)	652.2 (626.8 – 678.4)	513.8 (509.5 – 518.2)
Mortality – all causes (2017-2018)	Cobar LGA = 594.6 (455.8 – 762.1)	520.9 (517.7 – 524.0)
Mortality – respiratory (all ages) (2016-2018)	75.1 (70.3 – 80.2)	49.6 (48.5 – 50.1)
Adults – prevalence of high blood pressure (2018)	31.1% (25.7% - 36.4%)	24.8% (23.7% - 25.9%)
Adult asthma – prevalence (2019)	19.3% (12.7% - 25.9%)	11.5% (10.5% – 12.5%)
Children (2 to15 years) – prevalence of current asthma (2017 – 2019)	14.3% (9.6% - 19.1%)	13.1% (11.8% - 14.4%)

* Rate per 100,000 population.

1 Data from NSW Health Statistics: http://www.healthstats.nsw.gov.au/ and https://beta.healthstats.nsw.gov.au/#/topics

Shading relates to comparison against NSW:

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.

As described above, the Western NSW Local Health District covers a large area.

The data relevant to the population in this area indicates that in general most health-related behaviours are similar to NSW, with the rate of overweight and obesity noted to be higher than for NSW. The data relevant to the burden of disease, however, indicates that the population in the WNSWLHD has higher rates (when compared with NSW) of mortality (including higher rates in Cobar LGA), hospitalisations for cardiovascular and respiratory disease, prevalence of high blood pressure and asthma (particularly in adults). This data indicates that the population in the area is consider more vulnerable to stressors such as Project-related impacts. The underlying reasons for this increased vulnerability are expected to be complex, and may include a broad range of lifestyle, behaviour and environmental factors.

Section 4. Community engagement

Community consultation has been undertaken for the Project during the preparation of the EIS (SLR 2021). The consultation included establishing and holding meetings with a Community Consultative committee (CCC) in December 2020 and June 2021 and running community information sessions in March 2021 and September 2021. A community hotline has been established and engagement has been undertaken with directly affected landholders (i.e. landholders within the nominated Project boundary). Aboriginal parties have been consulted through the Aboriginal cultural heritage assessments and an Aboriginal Focus Group Meeting (AFGM). Activities have also included meetings with key stakeholders including Crown Lands, EPA, NSW Heritage, Transport for NSW, Department of Planning, Industry and Environment (DPIE), Biodiversity, Conservation and Science, WaterNSW, MEG CPDP, Cobar City Council and Bogan Shire Council.

In terms of community health, the consultation outcomes to date relate to the following:

- Concerns relating to occupational health and safety about the Hera Mine
- Concerns relating to dust emissions
- Concerns relating to noise
- Road safety and traffic

Impacts of the Project on physical health, specifically in relation to changes in air quality and noise are addressed in this report. Impacts of the other aspects of the project such as road safety and traffic are addressed in further detail in the Traffic Impact Assessment.

Section 5. Health impact assessment: Air emissions

5.1 Approach

This section presents a review of impacts on health associated with predicted air emissions, relevant to the operation of the Project. The assessment presented has relied on the following:

- ERM 2021, Federation Project, Air Quality and Greenhouse Gas Assessment. This report is referred to as the AQGGA.

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

5.2 Background on particulate matter

The focus of the AQIA and this assessment of potential health impacts is the emissions to air of dust or particulate matter. The assessment has considered both the size of the particulate matter as well as the composition of metals that may be present on the particulates.

Dust or Particulate Matter (PM) is a widespread air pollutant (that has and will always be present in air) with a mixture of physical and chemical characteristics that vary by location (and source). Unlike many other pollutants, particulates comprise a broad class of diverse materials and substances, with varying morphological, chemical, physical and thermodynamic properties, with sizes that vary from <0.005 micrometres (µm) to >100 µm. Particulates can be derived from natural sources such as crustal dust (soil), pollen and moulds, and other sources that include combustion and industrial processes. Secondary particulate matter is formed via atmospheric reactions of primary gaseous emissions. The gases that are the most significant contributors to formation of secondary particulates include: nitrogen oxides, ammonia, sulfur oxides, and certain organic gases (derived from vehicle exhaust; combustion sources; and agricultural, industrial and biogenic emissions).

The potential for particulate matter to result in adverse health effects is dependent on the size and composition of the particulate matter.

The size of particulates is important as it determines how far from an emission source the particulates may be present in air (with larger particulates settling out close to the source and smaller particles remaining airborne for greater distances) and also the potential for adverse effects to occur as a result of exposure (how far the particles can infiltrate into the human respiratory system).

The common measures of particulate matter that are considered in the assessment of air quality and health risks are:

- **Total Suspended Particulates (TSP):** This refers to all particulates with an equivalent aerodynamic particle[2] size below 50 µm in diameter[3]. It is a gross indicator of the presence of dust with a wide range of sizes. The larger particles included in TSP (termed "inspirable", comprise particles around 10 µm and larger) are more of a nuisance as they will deposit out of the air (measured as deposited dust) close to the source and, if inhaled, are mostly trapped in the upper respiratory tract[4] and do not reach the lungs, hence, there is no potential for adverse health effects. Finer particles included in TSP (smaller than 10 µm, termed "respirable", as described below) tend to be transported further from the source and are of more concern with respect to human health as these particles can penetrate into the lungs. Not all of the dust characterised as TSP is relevant for the assessment of health impacts, and hence TSP as a measure of dust impact in the community, is difficult to directly include in this assessment. TSP can be used as a measure of dust that may give rise to nuisance impacts close to the source, where the heavier particles readily deposit out of the air causing dust to deposit onto surfaces (including vegetation and within homes). The deposition of dust is more often directly measured using dust deposition gauges, however, these data relate to an assessment of nuisance effects only. The assessment of potential health impacts relates to particles of a size where significant associations have been identified between exposure and adverse health effects.
- **PM_{10}, particulate matter below 10 µm in diameter, $PM_{2.5}$, particulate matter below 2.5 µm in diameter, PM_1, particulate matter below 1 µm in diameter and $PM_{0.1}$, particulate matter below 0.1 µm in diameter (PM_1 and $PM_{0.1}$ are termed ultrafine particles):** These particles are small and have the potential to penetrate beyond the body's natural filter mechanisms of cilia and mucous in the nose and upper respiratory system, with the smaller particles able to further penetrate into the lower respiratory tract[5] and lungs. Once in the lungs, adverse health effects may occur that include mortality and morbidity, which may be associated with a range of adverse cardiovascular and respiratory effects (OEHHA 2002)[6].

Figure 5.1 provides a general illustration to provide some context in relation to the size of different particles (discussed above) and relevance/importance for the assessment of inhalation exposures.

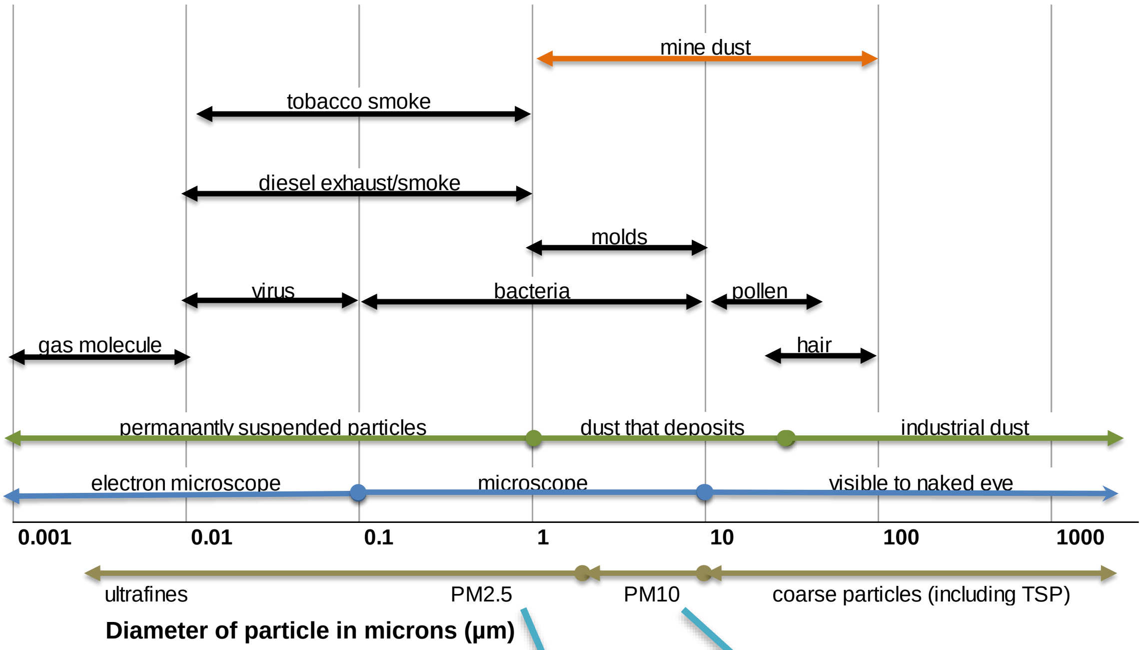
[2] The term equivalent aerodynamic particle is used to reference the particle to a particle of spherical shape and density 1 gram per cubic centimetre (g/cm^3).

[3] The size, diameter, of dust particles is measured in micrometres.

[4] The upper respiratory tract comprises the mouth, nose, throat and trachea. Larger particles are mostly trapped by the cilia and mucosa and swept to the back of the throat and swallowed.

[5] The lower respiratory tract comprises the smaller bronchioles and alveoli, the area of the lungs where gaseous exchange takes place. The alveoli have a very large surface area and absorption of gases occurs rapidly with subsequent transport to the blood and the rest of the body. Small particles can reach these areas, be dissolved by fluids and absorbed.

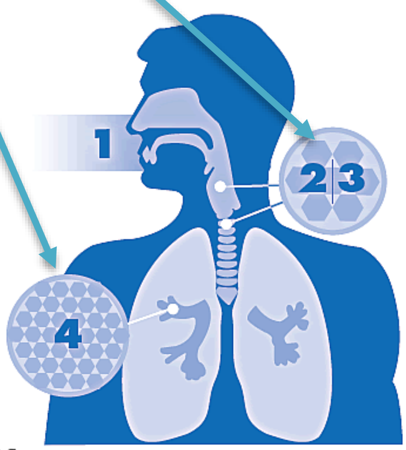
[6] OEHHA – Office of Environmental Health Hazard Assessment.



1 Particulate matter enters our respiratory (lung) system through the nose and throat.

2|3 The larger particulate matter (PM_{10}) is eliminated from the respiratory system through coughing, sneezing and swallowing.

4 $PM_{2.5}$ can penetrate deep into the lungs. It can travel all the way to the alveoli, causing lung and heart problems, and delivering harmful chemicals (where present) to the blood system.



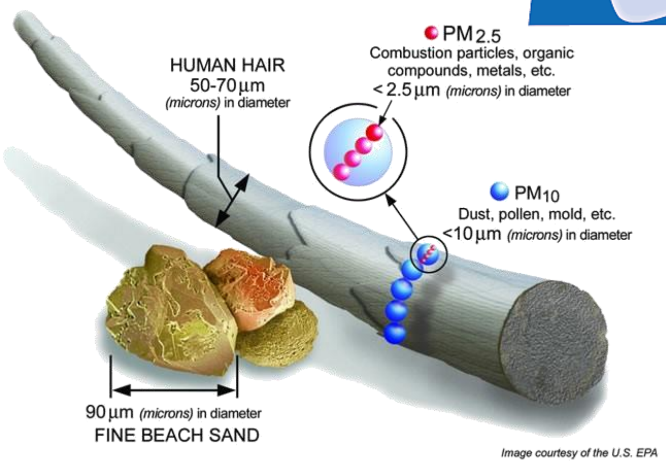


Figure 5.1: Illustrative comparison of relative particle sizes and importance for health

It is well accepted nationally and internationally that monitoring for PM_{10} is a good method of determining the community's exposure to potentially harmful dust (regardless of the source) and is most commonly measured in local and regional air quality monitoring programs. Reliable methods for the monitoring of PM_{10} concentrations have been available for a long time and hence these data are most widely available in urban and rural areas.

Smaller particles such as $PM_{2.5}$, however, are seen as more significant with respect to evaluating health effects, as a higher proportion of these particles penetrate into the lungs. Very fine particles, specifically ultrafine particles (PM_1 or $PM_{0.1}$), are also considered to be of importance for the assessment of health effects as these particles penetrate the deepest into the respiratory system.

5.3 Summary of air modelling

5.3.1 Existing air quality

The main sources of particulate matter in the area surrounding the Project include active mining from the Hera Mine, agriculture, and emissions from local anthropogenic activities such as motor vehicle exhaust, domestic wood heaters, and bushfire activity.

Data evaluated in the AQGGA in relation to the existing air quality is based on data from existing air quality monitors within the mining lease and in close proximity to haul roads for the Hera Mine (which record TSP, PM_{10} and dust deposition), and data on PM_{10} and $PM_{2.5}$ from DPIE monitoring stations at Tamworth, Albury, Bathurst and Wagga Wagga.

Data from these monitoring stations indicate the following in relation to dust (refer to AQGGA for additional detail):

- Dust deposition levels reported at the Hera Mine site range from 1.3 to 4.5 g/m^2/month. This data is not representative of dust deposition in off-site areas, where levels of 2 g/m^2/month are considered more typical of semi-arid rural areas.
- TSP monitoring conducted at the Hera Mine site, which includes all the large particulates which do not reach the lungs when inhaled, reports levels in the range 28.2 $\mu g/m^3$ to 74.3 $\mu g/m^3$ (annual average).This data, however is not relevant to the off-site areas and in the absence of TSP data from DPIE monitoring stations background levels of TSP have been determined on the assumption that approximately 40% of TSP comprises PM10.
- PM_{10} data from the DPIE monitoring stations typically report concentrations (annual average) that are below the relevant guidelines. The exception is data from 2018 and 2019 (and part 2020) which is affected by a higher prevalence of dust storms and bushfires. In relation to the short-term average data (24-hour average), there were some exceedances of the relevant guideline due to drought conditions (including dust storms) and bushfires. Background levels adopted in the AQGGA for PM_{10} were based on the average from all four monitoring stations in 2017 (considered representative for the area), utilised on the basis of a daily average (for each individual day in 2017) and an annual average.
- $PM_{2.5}$ data from the DPIE monitoring stations typically show that 24-hour average and annual average concentrations are below the relevant guidelines, with the exception of days and years affected by a higher prevalence of dust storms and bushfires (relevant to 2018, 2019 and part 2020). Consistent with the approach adopted for PM10, background concentrations of $PM_{2.5}$ adopted in the AQGGA are from 2017, as an average over all

stations utilised on the basis of a daily average (for each individual day in 2017) and an annual average.

5.3.2 Modelling impacts from the Project

Modelling of air quality impacts requires consideration of the local area, specifically the local terrain and meteorological conditions, as well as emissions to air from the various activities relevant to the Project. The assessment of air quality impacts is presented in the AQGGA (EMM 2021). Key features of the air modelling and impact assessment are summarised below.

The local meteorological conditions have been evaluated on the basis of data collected from the Bureau of Meteorology (BoM) monitoring station at Cobar data has been utilised within the Weather Research and Forecasting model (WRF) to generate gridded meteorological data for the Project and surrounding area. The influence of the local terrain of the Project areas and surrounding environments on meteorological conditions have also been taken into account. The data was processed using AERMET for use in the air model AERMOD.

Dust emissions from the Project have been estimated on the basis of emission factors for all the relevant activities, volumes to be handled and equipment proposed to be used. This includes:

- Transportation of material from the Federation Site to the Hera Mine along the sealed Burthong Road
- Processing of Federation deposit ore at the processing plant at Hera Mine
- Disposal of approximately 40% of the tailings at TSF at Hera Mine
- Transportation of approximately 60% of the tailings to the Federation Site for paste backfill of underground stopes
- Transportation of concentrate to Hermidale Siding via Hermidale Nymagee Road.

Emission rates of TSP, PM_{10} and $PM_{2.5}$ have been calculated using emission factors developed both within NSW and by the US EPA. Modelling of TSP, PM_{10} and $PM_{2.5}$ was undertaken using the particle size specific inventories and was assumed to emit any deposit from the plume in accordance with the deposition rate appropriate for particles with an aerodynamic diameter equal to the geometric mass of the particle size range.

Dust control measures to be employed for the Project and assumed as part of this assessment include the following:

- Use of additional water application, if required, on active unsealed haul roads (50% control applied)
- Use of additional water sprays, if required, on activities such as loading, unloading, front end loader operations, stockpiles and pads, tailings storage facility (50% control applied)
- Use of sealed road, Burthong Road (90% control applied).

Modelling was undertaken using AERMOD to predict impacts associated with Project activities over two grids: an area of 14.5 km x 29.5 km at 500 m spacing; and a smaller grid focusing on Burthong Road of 4 km x 18 km at 200 m spacing. The modelling also predicted impacts at the individual residential receptor locations (refer to **Figure 2.3**).

The focus of the assessment of air impacts related to financial year 28 (FY28). This is the year which has the highest amount/mass of ore mined (tailings and concentrate).

The assessment of potential health impacts of exposure to changes in air quality relates to maximum emissions that occur in FY28.

5.4 Assessment of health impacts – particulate size

5.4.1 Health effects

Evaluation of size alone as a single factor in determining the potential for particulate toxicity is difficult since the potential health effects are not independent of chemical composition. There are certain particle size fractions that tend to contain certain chemical components, such as metals or other organic compounds.

There is strong evidence to conclude (USEPA 2012; WHO 2003a, 2013) that fine particles (<2.5 µm, $PM_{2.5}$) are more hazardous than larger ones (coarse particles), primarily on the basis of studies conducted in urban air environments where there is a higher proportion (as a percentage of all particulates) of fine particles and other gaseous pollutants present from fuel combustion sources, as compared to particles derived from crustal origins. It should be noted that recent detailed review of the available studies in relation to the health effects of particulates (Hime, Marks & Cowie 2018) concluded that while there is some evidence that particulate matter from traffic and coal-fired power station emissions may elicit greater health effects compared to particulate matter from other sources (diesel exhaust, domestic wood combustion heaters and crustal materials), overall the evidence to date does not indicate a clear 'hierarchy' of harmfulness for particulate matter from different emission sources. Hime et al (Hime, Marks & Cowie 2018) identified that making such conclusions is limited by studies, many of which are not comparable. For this assessment, the health effects of exposure to particulate matter have been evaluated as being the same from all sources.

When undertaking any quantitative assessment of health impacts, it is important that the assessment considers health effects where there is sufficient evidence to demonstrate a causal link between exposure to particulates and the health outcome identified. There are numerous studies where statistical associations have been identified. Association does not mean causation; hence it is important that robust reviews are considered where the strength of the available data is fully evaluated and only health effects where there is strong causal evidence is evaluated. Such robust reviews are undertaken by key organisations such as the USEPA, WHO and Australian authorities (as noted below). Assessing health impacts based on associations only (not causation) would be misleading and inappropriate.

A significant amount of research, primarily from large epidemiology studies, has been conducted on the health effects of particulates with causal effects relationships identified for exposure to $PM_{2.5}$ (acting alone or in conjunction with other pollutants) (USEPA 2012, 2019). A more limited body of evidence suggests an association between exposure to larger particles, PM_{10} and adverse health effects (USEPA 2009a, 2019; WHO 2003a).

Adverse health effects associated with exposure to particulate matter have been well studied and reviewed by Australian 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, where there have been clear associations determined between health effects and exposure to $PM_{2.5}$ and, to a lesser extent, PM_{10}. These studies are complemented by findings from other key investigations conducted in relation to the characteristics of inhaled particles; deposition and clearance of particles in the respiratory tract; animal and cellular toxicity studies; and studies on inhalation toxicity by human volunteers (NEPC 2010).

Particulate matter has been strongly linked to adverse health effects after both short term exposure (days to weeks) and long term exposure (months to years). The health effects 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 pre-existing (underlying) respiratory or cardiovascular disease. For long term exposures in a population, this relates to mortality rates over a lifetime (i.e. shortening the lifespan), 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 2009a, 2019):

- 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).
- Changes to lung tissues and structure.
- Altered respiratory defence mechanisms.

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 and those with underlying health conditions (USEPA 2009a, 2019). The exposure-response relationships adopted incorporate (and are expected to be dominated by) data from these sensitive groups. This is important to note given the population in the off-site community may have some increased vulnerability to Project related particulate exposures. The approach adopted for assessing risk is considered to address this increased vulnerability.

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 2012). Similar relationships have also been determined for PM_{10}, however, the supporting studies do not show causal relationships as clear as those shown with $PM_{2.5}$ (USEPA 2012).

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 2018). A causal relationship has not been established for these health effects. 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 2018).

5.4.2 Assessment of cumulative exposures to particulates

The assessment of cumulative exposures to $PM_{2.5}$ and PM_{10} is based on a comparison of the predicted cumulative concentrations to the current air quality standards and goals presented in the NEPM (NEPC 2021).

In relation to the current NEPM PM_{10} standard, the following is noted (NEPC 1998b, 2010, 2014, 2021):

- The standard was derived through a review of appropriate health studies by a technical review panel of the NEPC where short term exposure-response relationships for PM_{10} and mortality and morbidity health endpoints were considered.
- Mortality health impacts were identified as the most significant and were the primary basis for the development of the standard.
- On the basis of the available data for key air sheds in Australia, the criterion of 50 micrograms per cubic metre (µg/m^3) was based on analysis of the number of premature deaths that would be avoided and associated cost savings to the health system (using data from the US). The development of the standard is not based on any acceptable level of risk and hence simply meeting the standard does not cover all aspects that need to be considered in terms of health impacts.
- The assessment undertaken considered exposures and issues relevant to urban air environments that are expected to also be managed through the PM_{10} standard. These issues included emissions from vehicles and wood heaters.

A similar approach has been adopted by NEPC (Burgers & Walsh 2002; NEPC 2002, 2014) in relation to the derivation of the $PM_{2.5}$ air quality standards, with specific studies related to $PM_{2.5}$ and mortality and morbidity indicators considered. Goals for lower $PM_{2.5}$ standards to be met by 2025 are also outlined by NEPC (NEPC 2021).

Table 5.1 presents a comparison of the current NEPC standards and goals with those established by the WHO (WHO 2021), the European Union (EU) (2015) and the USEPA (2012). The WHO (2021) update provided air quality goals along with interim targets for the reduction in concentrations over time. Review of the USEPA air quality standards in 2020 (USEPA 2020) recommended retaining the 2012 standards without revision for PM_{10} and $PM_{2.5}$.

The 2025 goals established by the NEPM for $PM_{2.5}$ (and adopted in this assessment) are similar to, but slightly more conservative (health protective) than, those provided by the EU and the USEPA. The 2025 goals are generally similar to the interim target 4 criteria established by the WHO (2021) but are higher than the WHO (2021) goals.

The NEPM PM_{10} guidelines are also similar to those established by the EU, however the 24-hour average guideline is significantly lower than the 24-hour average guideline of the USEPA. The NPM guidelines are generally similar to the interim target 4 criteria established by the WHO (2021) but are higher than the WHO (2021) goals.

Table 5.1: Comparison of particulate matter air quality goals

Pollutant	Averaging period	Criteria/guidelines/goals			
		NEPC (2021)	**WHO (2021)**	**EU #**	**USEPA (2012)**
PM_{10}	24-hour	50 µg/m^3	Goal = 45 µg/m^3 Interim target 4 = 50 µg/m^3	50 µg/m^3 as limit value to be met, with 35 exceedances permitted each year	150 µg/m^3 (not to be exceeded more than once per year on average over 3 years)
	Annual	25 µg/m^3	Goal = 15 µg/m^3 Interim target 4 = 20 µg/m^3	40 µg/m^3 as limit value to be met	NA
$PM_{2.5}$	24-hour	25 µg/m^3 20 µg/m^3 (goal for 2025)	Goal = 15 µg/m^3 Interim target 4 = 25 µg/m^3	NA	35 µg/m^3 (98th percentile, averaged over 3 years)
	Annual	8 µg/m^3 7 µg/m^3 (goal for 2025)	Goal = 5 µg/m^3 Interim target 4 = 10 µg/m^3	25 µg/m^3 as target value to be met from 2010 and limit value to be met from 2015 20 µg/m^3 as a 3-year average (average exposure indicator) from 2015 with requirements for ongoing percentage reduction and target of 18 µg/m^3 as 3-year average to be attained by 2020	12 µg/m^3 (annual mean averaged over 3 years)

Current EU Air Quality Standards (EU 2015) available from **http://ec.europa.eu/environment/air/quality/standards.htm**

The air quality standards and goals for $PM_{2.5}$ and PM_{10} relate to total concentrations in the air (from all sources including the Project). This has been modelled and evaluated in detail within the AQGGA.

In relation to impacts at the sensitive receptors surrounding the site, the AQGGA determined the following in relation to cumulative exposures to dust (background + Project) for FY28:

- There are no predicted exceedances of the annual average TSP criterion of 90 µg/m^3
- There are no predicted exceedances of the annual average PM_{10} criterion of 25 µg/m^3
- For 24-hour average PM_{10}, there are predicted exceedances of the maximum 24-hour average criterion for PM_{10} of 50 µg/m^3 at all receptors. The exceedance is due to a high background concentration of 53.8 µg/m^3. There are no additional exceedances caused by the Project.
- For annual average $PM_{2.5}$, there are no predicted exceedances of the criterion of 8 µg/m^3, however there are exceedances of the pending NEPM AAQ standard of 7 µg/m^3. It should be noted that the exceedance would be due to the background concentration which is already exceeding 7 µg/m^3, and not due to the Project.
- For 24-hour average $PM_{2.5}$, there are no predicted exceedances of the maximum 24-hour average assessment criteria of 25 µg/m^3 or the pending NEPM AAQ standards of 20 µg/m^3.

When considering the Project contribution, these concentrations are low and range between 0.1% and 2.7% of the cumulative concentration.

5.4.3 Assessing incremental impacts associated with particulates

In relation to the assessment of exposures to particulate matter, there is sufficient evidence to demonstrate that there is causal link between exposure to $PM_{2.5}$ (and to a lesser extent PM_{10}) and particular health effects. These health effects relate to exposures to $PM_{2.5}$ (or PM_{10}) alone (i.e. without co-exposures).

Where a causal link has been established in relation to exposure to changes in $PM_{2.5}$ or PM_{10} exposure and health effects, risks can be quantified using 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. 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.

The available evidence does not suggest that there is a threshold below which health effects do not occur. Hence 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. Guidelines are currently available for the assessment of $PM_{2.5}$ and PM_{10} in Australia (NEPC 1998 amended 2016, 2002, 2021). These guidelines are not based on any acceptable level of risk, rather they are based on levels that are desirable in the community to balance background/urban sources with lowering impacts on health and cost savings in the health system.

Risk calculations relevant to exposures to $PM_{2.5}$ and PM_{10} by the community have been undertaken utilising concentration-response functions relevant to the most significant health effect associated with exposure for all members of the community, 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[7]. 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):

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

Where:
X-X0 = the change in particulate matter concentration to which the population is exposed (µg/m³)

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

β = regression/slope coefficient, or the slope of the exposure-response function which can also be expressed as the per cent change in response per 1 μg/m³ 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:

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

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; WHO 2006a) 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:

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

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 (eg 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 residential receptors adopted in this calculation. For this assessment the change in PM relates to the change in annual average air concentrations, where the following has been adopted (as modelled from AQGGA) for FY28:
  - Maximum incremental increase in PM_{10} (all receptors) = 1.3 μg/m³
  - Maximum incremental increase in $PM_{2.5}$ (all receptors) = 0.2 μg/m³
- Baseline incidence of the key health endpoints that are relevant to the population exposed – the assessment undertaken has considered the baseline mortality data (all cause, all ages) relevant to the Cobar LGA, with the most recent data indicating a rate of 594.6 per 100,000 (or 0.005946) as an age standardised rate (refer to **Table 2.3**) which has been adopted in this assessment.

- 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. The concentration response functions used in this assessment is based on the following:
- PM_{10}: The exposure response function adopted for the assessment of risks related to exposure to PM_{10} is based on analysis of data from European studies from 33 cities and includes panel studies of symptomatic children (asthmatics, chronic respiratory conditions) (Anderson et al. 2004). The study found a relative risk (RR) of all-cause mortality of 1.006 per 10µg/m^3 change in PM_{10}. Based on a RR of 1.006 per 10µg/m^3 change in PM_{10}, this results in a $\beta = 0.0006$. It is noted that this relationship is not as strong as for $PM_{2.5}$ and relates to short-term changes in PM_{10}. The calculation of risk using this relationship based on a change in annual average concentration provides the same outcomes as calculating the daily risk and summing for the year.
- $PM_{2.5}$: the exposure response function adopted is that recommended in a NEPC published report (Jalaudin & Cowie 2012). It was derived from a study in the United States which examined the health outcomes of hundreds of thousands of people living in cities all over the United States. These people were exposed to all different concentrations of $PM_{2.5}$ (Pope et al. 2002). The study found a relative risk (RR) of all-cause mortality of 1.06 per 10µg/m^3 change in $PM_{2.5}$, and that this risk relationship was in the form of an exponential function. Based on a RR of 1.06 per 10µg/m^3 change in $PM_{2.5}$, this results in a $\beta = 0.0058$. It is noted that the exposure response relationship established in this study was re-affirmed in a follow-up study (that included approximately 500,000 participants in the US) (Krewski et al. 2009) and is consistent with findings from California (Ostro et al. 2006). The relationship is also more conservative than a study undertaken in Australia and New Zealand (EPHC 2010).

The above approach is consistent with that presented in Australia (Burgers & Walsh 2002), US (OEHHA 2002; USEPA 2005a, 2010) and Europe.

Based on the above assumptions the Project's incremental risk associated with exposure to PM_{10} and $PM_{2.5}$ for FY28 is calculated as follows (rounded to 1 significant figure):

- PM_{10}: $Risk=\beta \times \Delta X \times B = 0.0006 \times 1.3 \times 0.005946 = 5 \times 10^{-6}$
- $PM_{2.5}$: $Risk=\beta \times \Delta X \times B = 0.0058 \times 0.2 \times 0.005946 = 7 \times 10^{-6}$

These risk levels are considered to be negligible or acceptable, as per guidance from enHealth and NEPC (enHealth 2012a; NEPC 2011) and NSW EPA (NSW EPA 2017a).

The calculated risks (above) relate to the maximum impacted offsite residential receptor location. Risks are lower at all other residential receptors assessed.

On the basis of the above, incremental changes in PM_{10} and $PM_{2.5}$ derived from the Project are considered to have a negligible impact on the health of the off-site community.

5.5 Assessment of health impacts – particulate composition

5.5.1 Approach

The proposed mining operations involve the handling of ore that is mineralised. In particular, the ore comprises silver, arsenic, cadmium, cobalt, copper, mercury, lead and zinc. As a result, particulate matter released to air from Project emissions will comprise metals. The composition of metals in particulate emissions has been determined on the basis of geochemical analysis of 20 samples of fresh and weathered ore at depths ranging from 10 m to 681 m, reported by Terrenus Earth Sciences. The maximum percentage of each metal from these samples has been assumed to be present in all dust released to air from the Project activities, and is as follows:

- Silver = 0.0001%
- Arsenic = 0.03%
- Cadmium = 0.01%
- Cobalt = 0.0025 %
- Copper = 0.04%
- Mercury = 0.000002%
- Lead = 0.14%
- Zinc = 0.21%

The modelling undertaken and presented in the AQIA for TSP, PM_{10} and $PM_{2.5}$ has been used along with the composition of metals to provide predicted metal impacts at each residential receptor location and across the whole modelling grid. For the assessment of potential exposure to metals in dust the following model outputs have been utilised:

- Metal concentrations present in air as PM_{10} which is the dominant size fraction relevant to the emissions from the Project, that may be inhaled, and which may penetrate into the lungs where it is assumed to be 100% available to be absorbed into the body following exposure. The assessment of exposure has addressed:
  - Peak short-term or acute exposures, based on the maximum modelled 1 hour average concentration
  - Long-term exposures based on the maximum annual average concentration.
- Metal concentration present on TSP that is deposited to the ground (as dust deposition) where the metals may accumulate and influence soil concentration, be taken up into homegrown or agricultural produce or deposited onto residential roof areas and washed into rainwater tanks, potentially affecting drinking water quality.

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

For this assessment the focus relates to rural residential and residential uses surrounding the Project. However, this assessment has also addressed potential inhalation exposures that may occur by any member of the community at all locations outside of the Project boundary.

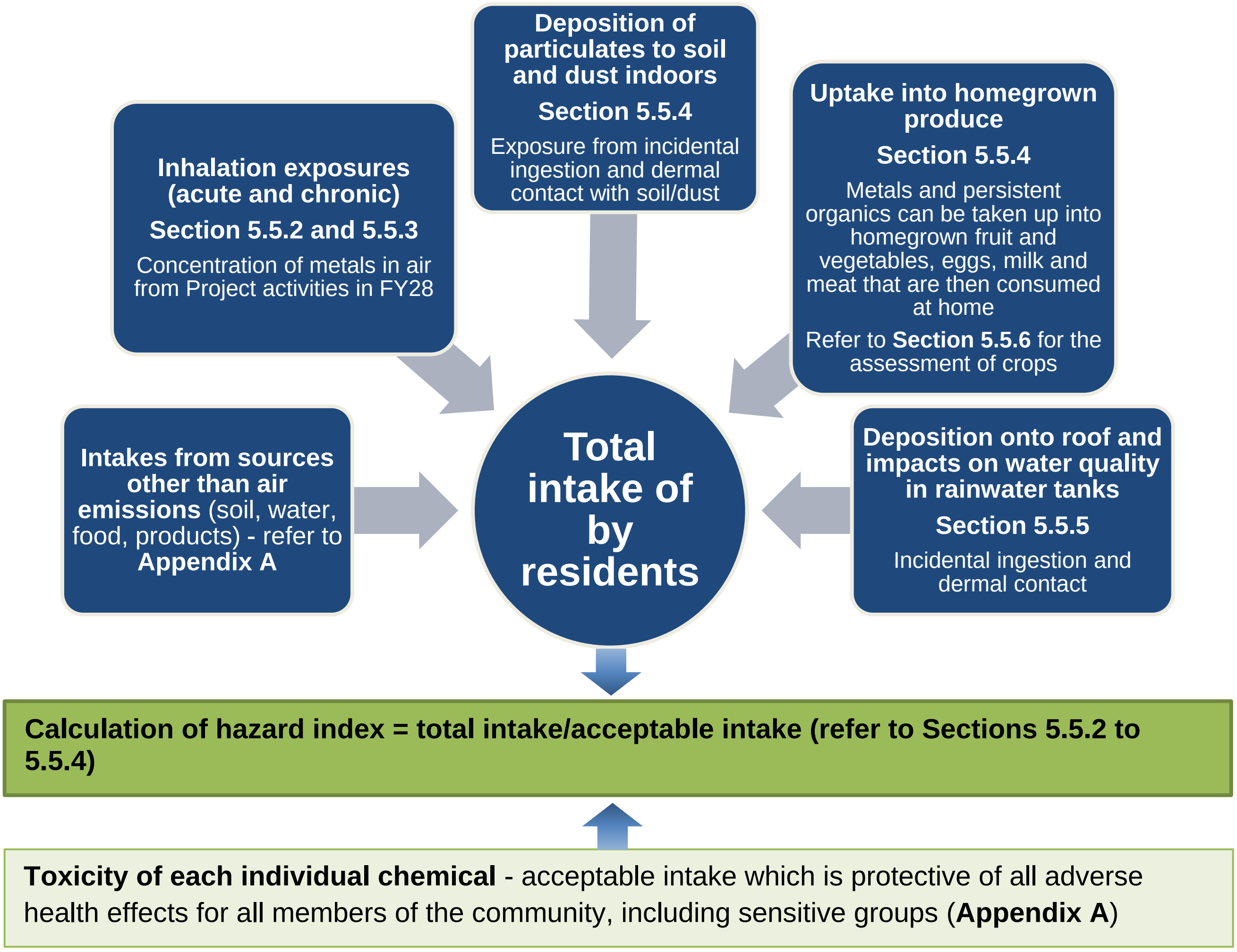
There are two main ways a community member may be exposed to a chemical substance emitted from the plant:

- Inhalation of particulate matter in air, which may occur anywhere outside the Project boundary including the surrounding residential receptor locations.
- Direct contact, which may include ingestion and/or dermal absorption of chemicals present in dust that may deposit onto surfaces and then be present in soil, be take up into homegrown produce or accumulated in water collected in rainwater tanks. These exposures are relevant to the receptor locations surrounding the site.

The assessment of risk, relevant to the presence of metals follows the principles outlined in the enHealth document Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a). 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, as noted above)
- the hazards posed by (or toxicity of) the chemicals present in the emissions (i.e. hazard or toxicity assessment)
- calculation of potential risks to health or risk characterisation.

The following diagram presents an overview of the assessment approach detailed in the following sections.



Deposition of particulates to soil and dust indoors
Section 5.5.4
Exposure from incidental ingestion and dermal contact with soil/dust
Inhalation exposures (acute and chronic)
Section 5.5.2 and 5.5.3
Concentration of metals in air from Project activities in FY28
Uptake into homegrown produce
Section 5.5.4
Metals and persistent organics can be taken up into homegrown fruit and vegetables, eggs, milk and meat that are then consumed at home
Refer to Section 5.5.6 for the assessment of crops
Intakes from sources other than air emissions (soil, water, food, products) - refer to Appendix A
Total intake of by residents
Deposition onto roof and impacts on water quality in rainwater tanks
Section 5.5.5
Incidental ingestion and dermal contact
Calculation of hazard index = total intake/acceptable intake (refer to Sections 5.5.2 to 5.5.4)
Toxicity of each individual chemical - acceptable intake which is protective of all adverse health effects for all members of the community, including sensitive groups (Appendix A)

5.5.2 Acute inhalation exposures

The assessment of acute exposures is based on comparing the maximum predicted 1-hour average exposure concentration with health-based criteria relevant to an 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 of pollutant in air as PM_{10} (mg/m^3)
Acute TRV = health based toxicity reference value (TRV) or guideline that is protective of short-duration exposures for all members of the community including sensitive individuals, as per **Appendix A** (mg/m^3)

Consistent with guidance provided by enHealth (enHealth 2012a), 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 A**. It is noted that for the assessment of exposure to lead and zinc there are no health-based guidelines available as the key issues related to these chemicals relates to chronic exposures or long-term body burdens. The acute assessment has therefore focused on the chemicals where acute health effects are relevant.

Table 5.2 presents a summary of the relevant health-based guideline, the predicted maximum 1-hour average concentrations at the maximum impacted receptor and the maximum impacted location anywhere outside the Project boundary and the calculated HI for each metal evaluated.

Table 5.2: Review of acute exposures and risks

Metal	Acute air guideline - health (mg/m^3)	Air Concentration (PM_{10}) - Maximum 1 hour average (mg/m^3)		Calculated HI	
		Maximum anywhere outside Project	Maximum – residential receptors	Maximum anywhere outside Project	Maximum – residential receptors
Silver	0.0025 M	1.2E-06	9.4E-08	0.00049	0.000037
Arsenic	0.0099 T	3.5E-04	2.7E-05	0.036	0.0027
Cadmium	0.00055 T	8.2E-06	6.3E-07	0.015	0.0011
Cobalt	0.00069 T	2.6E-05	2.0E-06	0.038	0.0029
Copper	0.1 O	4.3E-04	3.3E-05	0.0016	0.00033
Mercury	0.0006 O	2.0E-08	1.5E-09	0.000033	0.0000026
			Total HI	**0.091**	**0.0072**
			Acceptable HI	**≤ 1**	**≤ 1**

References for health-based acute air guidelines (1-hour average):
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
M = Guideline available from Ontario Ministry of the Environment and Climate Change (OMECC), as Ambient Air Quality Criteria that are protective of health. The value adopted relates to a 24-hour average which has been converted to a 1-hour average (as described in **Appendix A**) https://www.ontario.ca/page/ontarios-ambient-air-quality-criteria

Review of **Table 5.2** indicates all maximum predicted concentrations of chemicals in air are 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 Project.

5.5.3 Chronic inhalation exposures

For the assessment of chronic exposures, all the chemicals evaluated have a threshold guideline value that enables the predicted annual average concentration to be compared with a health based, or acceptable, guideline. For the assessment of chronic effects, the assessment has also considered potential intakes of these chemical substances from other sources, i.e. background intakes. As a result, the individual HI is calculated as follows (enHealth 2012a):

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

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

Where:
Exposure concentration = concentration in air relevant to the exposure period – annual average, modelled air concentration present on dust as PM_{10} (mg/m^3)
TRV = health-based toxicity reference value based on a threshold that is protective of all health effects for all members of the community (mg/m^3) (refer to **Appendix A**)
Background = proportion of the TRV that may be derived from other sources/exposures such as water, soil or products (%) (refer to **Appendix A**)

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.

Inhalation exposures have been quantified for the following:

- Maximum impacted residential receptor, where it is assumed that a resident is at home 24 hours per day, every day for the duration of the Project (assumed to be 14 years)
- Maximum impacted location outside the Project boundary – this is not a location where anyone lives as the maximum impact relates to a location on or very close to the Project boundary, however inhalation exposures may occur where farm work may be undertaken in the area, or visitors access areas close to the Project. Exposures in this area are assumed to occur for up to 8 hours per day, 240 days per year for the duration of the Project (assumed to be 14 years).

Appendix A presents the relevant health-based values 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 C** presents the calculations undertaken to evaluate inhalation exposures.

Table 5.3 presents the calculated individual HI and the incremental lifetime cancer risk relevant to the assessment of chronic inhalation exposures for the maximum impacted residential receptor.

Table 5.3: Review of chronic inhalation risks

Metal	Air Concentration (PM_{10}) - Maximum annual average (mg/m^3)		Calculated HI	
	Maximum anywhere outside Project	Maximum – residential receptors	Maximum anywhere outside Project (worker exposures)	Maximum – residential receptors (residential exposures)
Silver	1.8E-08	1.6E-09	0.00000019	0.000000078
Arsenic	5.1E-06	4.5E-07	0.033	0.013
Cadmium	1.2E-07	1.0E-08	0.013	0.0052
Cobalt	3.8E-07	3.3E-08	0.0010	0.00042
Copper	6.2E-06	5.5E-07	0.0000069	0.0000028
Mercury	2.9E-10	2.6E-11	0.00000053	0.00000021
Lead	2.1E-05	1.8E-06	0.013	0.00053
Zinc	3.1E-05	2.8E-06	0.000020	0.0000079
		Total HI	**0.049**	**0.020**
		Acceptable HI	**≤ 1**	**≤ 1**

Review of **Table 5.3** indicates all individual and the total HI relevant to chronic inhalation exposures are less than 1.

On the basis of the above assessment there are no chronic risk issues of concern in relation to inhalation exposures to emissions from the Project.

5.5.4 Multiple pathway exposures

General

Where pollutants may be bound to particulates (as TSP), 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 depositing to the environment where a range of other exposures may then occur. These include:

- Deposition to water (refer to **Section 5.5.5**), specifically 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 is also present in the soil where the plants are grown, and where chemicals are taken up into these plants

- Ingestion of eggs where chemicals may deposit onto pasture and be present in soil (which the 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 grain or fodder crops such as wheat and barley. There is the potential for metals to be taken up into these products. These products, however would not be consumed at home but would be sold into the market. For some metals, there are residue limits that would need to be complied with for the sale of these products. These have been considered further in **Section 5.5.6**.

The above exposures are chronic or long-term exposures.

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 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 on dust as TSP (i.e. including the coarser fractions that deposit to the ground as well as the fine fractions) relevant. These have been used in this assessment.

Appendix B 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 C**.

It is noted that assessment of potential risks related to exposure to water in rainwater tanks is presented separately in **Section 5.5.5**. In addition, assessment of risks relevant to the growing of grain crops are presented separately in **Section 5.5.6**.

Calculated risks

Table 5.4 presents the calculated risks associated with the most multiple pathway exposures relevant to both adults and children. These risks have been calculated on the basis of the maximum predicted deposition rate for all of the sensitive residential receptors in the surrounding community. Calculated risks for all other receptors would be lower than presented in this table.

The table presents the total 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 5.3**), as these exposures are additive to the other exposure pathways for residential properties.

Depending on the use a property, the types 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 may occur. The sum of risks associated with these multiple exposures is presented in **Table 5.4**.

Table 5.4: Summary of risks for multiple pathway exposures (maximum residential receptor)

Exposure pathway	Calculated HI	
	Young children	Adults
Individual exposure pathways		
Inhalation (I)	0.020	0.020
Soil ingestion (SI)	0.0062	0.00066
Soil dermal contact (SD)	0.0018	0.00088
Ingestion of homegrown fruit and vegetables (F&V)	0.0031	0.0011
Ingestion of homegrown eggs (E)	0.000056	0.000028
Ingestion of home produced milk (M)	0.0011	0.00027
Ingestion of home produced beef (B)	0.00020	0.000081
Ingestion of home produced lamb (L)	0.00011	0.000055
Multiple pathways (i.e. combined exposure pathways)		
I + SI + SD	0.028	0.021
I + SI + SD + F&V	0.031	0.022
I + SI + SD + E	0.028	0.021
I + SI + SD + F&V + E	0.031	0.022
I + SI + SD + M	0.029	0.021
I + SI + SD + B	0.028	0.021
I + SI + SD + L	0.028	0.021
I + SI + SD + F&V + E + M	0.032	0.023
I + SI + SD + F&V + E + B	0.031	0.022
I + SI + SD + F&V + E + L	0.031	0.022
I + SI + SD + F&V + E + M + B + L	0.032	0.023
Acceptable risk	**≤1**	**≤1**
Negligible risk	**≤1**	**≤1**

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

Review of **Table 5.4** 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/acceptable risks. The calculated HI is dominated by inhalation exposures, with the multi-pathway exposures contributing less to the total HI.

The MOS relevant to the calculated multi-pathway risks range from 31 to 43 for the most conservative scenario where a rural resident produces and consumes fruit and vegetables, eggs, mild, beef and lamb from the same property at the maximum impacted receptor location.

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.

5.5.5 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. For many of the residential and rural properties surrounding the Project, drinking water may be sourced from rainwater tanks. Hence it is important to evaluate potential impacts of the Project 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 Cobar MO (from the Bureau of Meteorology), a roof that is consistent with a 4 bedroom Australian home and the use of a first-flush device (noting that outcomes do not change if this devise is not included). Using this approach concentrations of chemicals in the water as suspended sediment and dissolved has been calculated. Rainwater tanks are designed such that suspended sediment deposits or settles and is not consumed. For the purpose of this assessment, it is assumed that both suspended sediment and dissolve phase concentrations may be present in the water used every day.

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 also consume water from rainwater tanks.

Table 5.5 presents the maximum predicted concentrations in rainwater tanks with comparison against drinking water guidelines. The table also presents 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 but is not considered realistic in relation to long-term exposures.

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

Table 5.5: Summary and review of exposures to chemicals in drinking water (maximum residential receptor)

Metal	Calculated maximum concentration in rainwater tanks (mg/L)		Drinking water guideline A (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)		
Silver	2.3E-07	1.2E-06	0.1	0.0000023
Arsenic	1.9E-05	2.9E-04	0.01	0.0019
Cadmium	1.7E-07	6.5E-06	0.002	0.000085
Cobalt	9.0E-07	2.1E-05	0.006	0.00015
Copper	1.2E-06	2.1E-05	2	0.00000058
Mercury	7.8E-07	2.1E-05	0.001	0.00078
Lead	4.5E-08	2.0E-05	0.01	0.0000045
Zinc	6.6E-07	2.1E-05	6	0.00000011
			Total HI	**0.0029**
			Acceptable/negligible HI	**≤1**

Refer to **Appendix C and D** for the calculation of water concentrations
A = Australian Drinking Water Guidelines (NHMRC 2011 updated 2021)

Review of **Table 5.5** indicates that the predicted water concentrations in rainwater tanks are all well below drinking water 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 disturbed during cleaning).

The calculations also demonstrate that the contribution of Project emissions to water quality in rainwater tanks are negligible and would not have any measurable change to the existing water quality in rainwater tanks. Hence these intakes and exposures (from using water from rainwater tanks) have not be calculated in detail and added to intakes from soil and produce.

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 ion **Table 5.4**) the following is noted:

- Young children (based on maximum HI calculated for all exposure pathways), HI = 0.032 (**Table 5.4**) + 0.0029 (**Table 5.5**) = 0.035
- Adults (based on maximum HI calculated for all exposure pathways), HI = 0.023 (**Table 5.4**) + 0.0029 (**Table 5.5**) = 0.026

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

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.

5.5.6 Assessment of risk issues relevant to crops

Where rural properties in the surrounding areas are used for the growing of crops such as grain crops (e.g., wheat, barley), these crops would not be home consumed. The crops, however would 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 residue limits (MRLs) in the Food Standards Code (FSANZ 2017a).

To enable this evaluation to be undertaken the maximum impacted rural residential receptor location has been considered, with the maximum predicted concentration in soil used to estimate concentrations in grain or similar crops (such as canola) using relevant uptake factors (refer to **Appendix B** for methodology and assumptions and **Appendix C** for calculations). The predicted concentration in grain crops have then been directly compared with the MRL. This is presented in **Table 5.6**. It is noted that the predicted concentrations are considered worst case as these relate to the deposition of pollutants from the Project and other sources to ground continuously for 14 years.

It is noted that there are MRLs for only 3 metals. Hence to determine if deposition from the Project has the potential to be of significance to crops produced in the area, the maximum predicted concentrations in crops have been compared with the range of concentrations reported by Food Standards in cereal products (breads, cereals and oats). These have been included for pollutants where there is no MRL. These are also included in **Table 5.6**.

Table 5.6: Review of concentrations in grain (and similar) crops – maximum sensitive receptor

Pollutant	Estimated maximum concentration in grain (mg/kg)	Food Standards Code – MRL 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)
Silver	0.000015	--	No data available
Arsenic	0.0011	1	--
Cadmium	0.00036	0.1	--
Cobalt	0.000012	--	0.0054 to 0.071 (F1)
Copper	0.00081	--	0.67 to 4.1 (F1)
Mercury	0.00028	--	0.005 (F2)
Lead	0.000015	0.2	--
Zinc	0.00032	--	4.5 to 38 (F1)

F = Food Standards Australian Total Diet Surveys
https://www.foodstandards.gov.au/science/surveillance/Pages/australiantotaldiets1914.aspx
F1 = 23rd Diet Survey (2011), F2= 25th Diet Survey (2019)

Review of **Table 5.6** indicates that the maximum predicted concentrations of arsenic, cadmium and lead are well below the MRLs relevant to these pollutants. The maximum predicted concentrations of other pollutants are below the range of mean concentrations reported in food products comprising these products. Hence emissions from the Project are considered to be negligible in terms of their contribution to existing background levels in grain (or similar) crop products consumed in the market.

5.6 Uncertainties

It is considered that the assessment of health impacts in relation to changes in air quality, associated with the Project, is conservative. This is due to the incorporation of a number of conservative assumptions in the modelling of air quality impacts.

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 Project.

In addition, the following should be noted:

- The assessment of potential health impacts has assumed that the off-site community remains at home (or on their property) all day, every day for the duration of the Project. This approach overestimates actual exposures where residents spend time away from the home (shopping, holidays, working at other premises).
- The changes in air quality evaluated in this assessment relate to FY28. It has been assumed that these impacts occur throughout all years of operation, which will not be the case as FY28 is considered the worst-case year in terms of maximum volume of ore handled and emissions to air.
- The calculated soil concentrations assume that deposition occurs throughout the whole Project life at the same rate predicted for FY28 with all impacts accumulating in surface soil

and indoor dust. No cleaning of indoor dust or use of any other topsoil/mulch/soil conditioner or fertiliser is used that would reduce concentrations in surface soil or indoor dust.
- Concentrations calculated on aboveground 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.

As a result of the above, the risk calculations presented are considered to be conservative.

5.7 Outcomes: Health impacts from air quality

A detailed assessment of risks to human health has considered potential exposure to dust/particulate matter emissions from the Project, for the worst-case emissions year FY28. The assessment has considered potential community exposures to particulate matter based on the size of the particulates, as well as the composition of the particulates, specifically the presence of metals.

In relation to the assessment of exposure to metals bound to particulates, this assessment has evaluated acute and chronic inhalation exposures as well as multi-pathway exposures associated with the deposition of metals 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 would have the potential to adversely affect water quality in rainwater tanks and grain (or similar) crops grown in the area.

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. More specifically the following has been concluded:
    - No acute inhalation risk issues of concern
    - No chronic risk issues of concern
    - Exposure to PM_{10} and $PM_{2.5}$ derived from the Project within the community are considered negligible.
- Multi-pathway exposures
  - All chronic risks to human health are considered negligible. 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 Project would have a negligible impact on water quality in rainwater tanks used for drinking water
  - Emissions from the Project would have a negligible impact on grains and other similar crops grown in the area.

Section 6. Health impact assessment: Noise

6.1 Background

This section presents a review and further assessment of impacts on health associated with noise, relevant to the Project. The assessment presented has relied on the information provided in the following report:

- Muller Acoustic Consulting (MAC) 2021, Noise and Vibration Impact Assessment, Federation Project, Nymagee, NSW (NVIA).

The noise impact assessment has considered impacts that may occur in the off-site community, addressing impacts relevant to the residential receptor locations identified in the areas surrounding the Project, as shown in **Figure 2.3**. It is noted that these receptors are consistent with those evaluated in the AQGGA (refer to **Section 5**).

6.2 Health impacts associated with noise

Environmental noise has been identified (I-INCE 2011; WHO 2011c, 2018)[8] as a growing concern because it has negative effects on quality of life and wellbeing and has the potential for causing harmful physiological health effects. With increasingly urbanised or developed societies, impacts of noise on communities have the potential to increase over time.

Sound is a natural phenomenon that only becomes noise when it has some undesirable effect on people or animals. Unlike chemical pollution, noise energy does not accumulate either in the body or in the environment, but it can have both short-term and long-term adverse effects on people. These health effects include (WHO 1999, 2011c, 2018):

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

Other effects for which evidence of health impacts exists, and are considered to be important, but for which the evidence is weaker, include:

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

[8] I-INCE – International Institute of Noise Control Engineering.

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

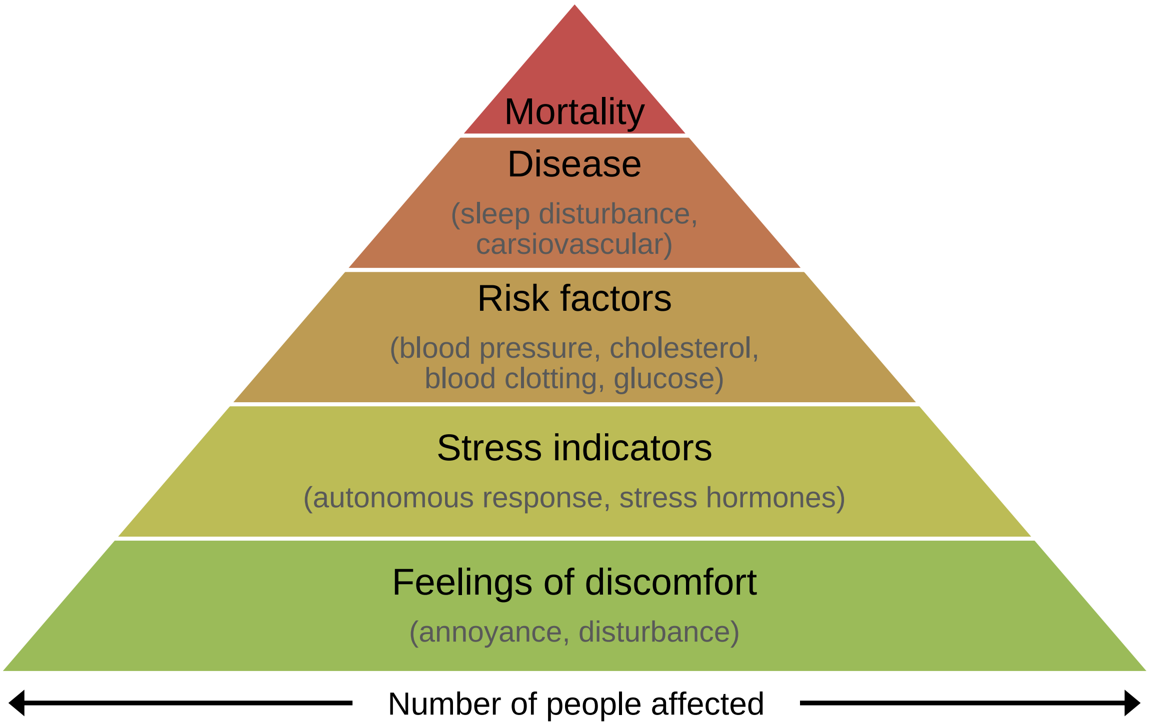


Figure 6.1: Schematic of severity of health effects of exposure to noise and the number of people affected (WHO 2011c)

Often, annoyance is the major consideration because it reflects the community's dislike of noise and their concerns about the full range of potential negative effects, and it affects the greatest number of people in the population (I-INCE 2011; WHO 2011c, 2018).

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

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

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

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

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

6.3 Review of the noise guidelines adopted

6.3.1 Noise and blasting criteria

For the assessment of potential Project noise impacts, rating background levels (RBLs, as LA_{90}) have been established in the NVIA. For all rural residential receptors, the RBLs are 35 dBA during the day and 30 dBA during the evening and night.

During construction, noise criteria outlined in the Interim Construction Noise Guideline (ICNG) (NSW DECC 2009). This guideline provides for the identification of Noise Management Levels (NML) during normal work hours and out of hours works. The NML is determined by adding 10dB (standard hours) or 5dB for Out of Hours (OOH) to the Rating Background Level (RBL) for each specific assessment period. The NMLs for construction are therefore 45 dBA as $L_{Aeq,15min}$ during the day and 35 dBA as $L_{Aeq,15min}$ during the evening and night. This guideline also identifies highly noise affected locations as those where the noise level exceeds 75 dBA $LA_{eq,15\ min}$.

For the Project noise criteria for the community have been established in the NVIA in accordance with the NSW Industrial Noise Policy. These guidelines incorporate background noise levels and criteria that protect against noise intrusion and noise amenity. The criteria adopted, as Project Noise Trigger Levels (PNTL) for the residential receptors evaluated in the NVIA are as follows:

- Day: 40 dBA as $L_{Aeq,15min}$
- Evening: 35 dBA as $L_{Aeq,15min}$
- Night: 35 dBA as $L_{Aeq,15min}$ with a maximum noise trigger levels established to protect against sleep disturbance issue set as 40 dBA as $L_{Aeq,15min}$ and 52 dBA as L_{Amax}

These noise criteria do not apply to noise-affected land subject to acquisition in accordance with the Voluntary Land Acquisition and Mitigation Policy (VLAMP). This relates to properties where the Project Noise Level (PNL) exceeds the PNTLs by more than 5 dB, or the PNL exceed the relevant criteria on more than 25% of a privately-owned land parcel.

Road traffic noise has been evaluated on the basis of the noise criteria as outlined in the Road Noise Policy (NSW DECCW 2011). For the principal haulage route proposed the noise criteria are 60 dBA as $L_{Aeq,15min}$ during the day and evening and 55 dBA as $L_{Aeq,15min}$ during the night.

Blasting has been assessed on the basis of criteria for the minimisation of human annoyance, which apply to impacts at privately-owned and other sensitive receptors. These criteria are:

- maximum overpressure due to blasting should not exceed 115 dB for more than 5% of blasts in any year, and should not exceed 120 dB for any blast
- maximum peak particle ground velocity should not exceed 5 millimetres per second (mm/s) for more than 5% of blasts in any year, and should not exceed 10 mm/s for any blast.

Criteria have also been adopted to address cosmetic and structural damage to buildings and structures.

6.3.2 Review of criteria

Noise criteria adopted in the Noise Impact Assessment are consistent with those outlined in the NPfI (NSW EPA 2017b), which indicate that intrusive noise from a specific industrial source should not exceed the RBL by more than 5 dBA. In addition, consideration has also been made to noise amenity, with the Project noise trigger levels adopted based on the lower noise criteria relevant to intrusiveness and amenity.

The noise criteria adopted (**Section 6.3.1**) are sufficiently low to be protective of health, based on available guidance from the WHO (WHO 1999, 2011c). The NPfI provides guidance on the interpretation of noise impacts in relation to these trigger levels, particularly in relation to predicted/estimated changes in noise levels.

The maximum noise criteria are set to protect residence from sleep disturbance and for this Project, an L_{AFmax} of 52 dBA is relevant to the night-time period. This maximum noise level is sufficiently low to be protective of health, based on available guidance from the WHO (WHO 1999).

Road traffic noise was assessed on the basis of the NSW Road Noise Policy (NSW DECCW 2011)[9], as it applies to existing residences on sub-arterial roads affected by additional traffic. This provides a guideline of 60 dBA as $L_{Aeq,15\ hour}$ (day and evening) and 55 dBA as $L_{Aeq,9\ hour}$ (night). Residences experiencing increases in total traffic noise above the relative increase criteria of Existing traffic LAeq(15hr) + 12dB should be considered for mitigation. In addition, the NSW Road Noise policy also indicates that "*an increase of up to 2 dB represents a minor impact that is considered barely perceptible to the average person*" and "*For existing residences and other sensitive land uses affected by additional traffic on existing roads generated by land use developments, any increase in the total traffic noise level should be limited to 2 dB above the corresponding 'no build option'*". These guidelines are higher than the health based goals relevant to road noise traffic from the WHO (WHO 2018) but consistent with the upper end of noise criteria established in previous WHO guidelines for outdoor noise predictions (WHO 1999, 2009). Further discussion on predicted noise levels as a result of the Project, in the context of WHO (2018) is presented is **Section 6.4.3**.

[9] DECCW – NSW Department of Environment, Climate Change and Water.

Blasting impacts have been evaluated in accordance with criteria established to protect human annoyance and structural damage (NSW DEC 2006)[10]. Provided the human comfort criteria are met, there would be no concern in relation to health impacts.

6.4 Review and assessment of health impacts from noise

6.4.1 Approach

The NVIA has considered noise impacts from the Project operations as well as road noise impacts. The noise assessment has utilised the model DGMR (iNoise, Version 2021.1).

Construction noise modelling has been undertaken in the NVIA to address the following scenarios, representative of realistic worst-case conditions based on the number and location of noise sources:

- Scenario 1 (Federation Site) – Site establishment
- Scenario 2 – Construction of Services Corridor between the Federation Site and Hera Mine
- Scenario 3 (Hera Mine) – Construction of solar farm and construction and installation of new processing plant

Construction works are expected to be undertaken during daylight hours over a period of 6 to 12 months.

During Project operations activities that are proposed to be undertaken during the Project, at the Federation and Hera Mine sites including the time and location of operations, sound power levels generated by the equipment/activities and modifying factors to address annoying noise characteristics, have been considered in the noise model. The NVIA has also assessed noise from a quarry located to the east of mine infrastructure (which utilises conventional drilling and blasting methods). The mine activities have been assumed to occur 24 hours per day with quarrying and the transportation of ore from Federation Site to Hera Mine, and tailings from Hera Mine to Federation Site, occurring during daylight hours (7am to 7pm) only. It is understood that a new processing plant is to be installed at Hera Mine. The noise modelling has assumed that both the old and new plants are operating at the same time (to address the transition period). These assumptions are expected to result in worst-case assessment of noise from the Project.

The noise modelling has also considered meteorological conditions. Some meteorological conditions have the potential to enhance received noise levels as a result of winds and the presence of temperature inversions. Conditions representative of noise enhanced meteorology during the day (calm conditions), evening (calm conditions) and night (inversion) were considered in the NVIA.

6.4.2 Noise impacts during construction

Based on the assessment presented in the NVIA in relation to construction activities, the following was determined:

[10] DEC – NSW Department of Environment and Conservation.

- Predicted noise levels at all residential receptors as a result of activities during each of the 3 scenarios evaluated were below the construction noise guidelines relevant to works that may be conducted during both standard hours and out of hours periods.

On this basis, there are no health issues of concern in relation to noise generated during construction activities associated with the Project.

6.4.3 Noise impacts during operations

Based on the assessment presented in the NVIA in relation to operations, the following was determined:

- Predicted noise levels as a result of operations at Federation Site, Hera Mine and both (cumulative) at all residential receptors were predicted to be below the PNTLs during the day, evening and night. Maximum noise levels predicted during the night were below the maximum noise trigger levels at all receptor locations.
- No residential receptor locations or land parcels exceeded the criteria relevant to the VLAMP. Hence there are no properties that would require consideration for acquisition.
- The total change in noise levels for the closest residential receptors to the haul roads is less than the +12 dB relative increase criteria as well as being below the criteria adopted for assessing noise impacts from road traffic. It is noted that the predicted noise levels at residential receptors are well below the adopted noise criteria, and for most receptors, the predicted noise levels are below 53 dBA, the noise level established by the WHO (2018) as a criterion to be protective of health effects from road noise. One receptor is predicted to have noise levels in excess of 53 dBA, however this receptor has existing road traffic noise in excess of this level, with Project related traffic adding only 1 dBA to the existing noise levels. A change of 1 dBA would not be discernible and is therefore not considered to be of concern.
- Overpressure levels from blasting would comply with the relevant criteria.

While all noise criteria would be met, the Project may implement a noise management plan (NMP) to outlines the most feasible and reasonable best management practices to minimise noise from the Project. The NMP may also include requirements for noise monitoring, should a noise complaint be received.

On the basis of the above, there are no health issues of concern in relation to noise generated during operation of the Project.

6.5 Uncertainties

The assessment presented in relation to potential noise impacts, and the potential for impacts on community health as a result of changes in noise as a result of the Project, is considered to be conservative. There are a number of areas within the noise impact assessment where conservative assumptions and approaches have been adopted. This includes the consideration of the worst-case meteorological conditions and assuming these occur at all times and the assumption that various Project activities occur at the same time.

On the basis of the above, conclusions in relation to potential impacts on community health are expected to be conservative.

6.6 Outcomes of health impacts from noise

With consideration of the noise guidelines adopted and the assessment of noise impacts from Project construction and operations, the potential for adverse health impacts from noise during the day, evening and night at off-site receptors is considered to be negligible.

Section 7. Conclusions

The HHRA presented in this report has considered potential impacts of the proposed Project on community health in relation to air quality and noise.

Based on the available information, and with consideration of the uncertainties identified, no health risk issues of concern have been identified for the off-site community. More specifically, the following is concluded:

Air quality:

- Inhalation exposures
  - All risks to human health are considered negligible. More specifically the following has been concluded:
    - No acute inhalation risk issues of concern
    - No chronic inhalation risk issues of concern
    - Exposure to PM_{10} and $PM_{2.5}$ derived from the Project within the community are considered negligible.
- Multi-pathway exposures (where metals present in dust are deposited to the ground or onto roof areas and accumulated in rainwater tanks)
  - All chronic risks to human health are considered negligible. This includes inhalation exposures, direct contact with soil and dust, ingestion of produce grown where dust may be deposited including homegrown fruit and vegetables, eggs, milk, beef and lamb.
  - Emissions from the Project would have a negligible impact on water quality in rainwater tanks used for drinking water
  - Emissions from the Project would have a negligible impact on grains and other similar crops grown in the area.

Noise

Noise generated during construction and operation – potential for noise levels that would result in adverse health impacts during the day, evening or night is considered to be negligible.

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Appendix A Toxicity of key chemicals evaluated

A1 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 1999a).

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_{10} and $PM_{2.5}$ follows a non-threshold approach as described in the main report.

A2 Values adopted for the assessment of acute exposures

The assessment of potential acute exposures relates to inhalation exposures only. The assessment is based on the maximum predicted 1-hour average air concentration. Hence the selection of relevant and appropriate acute toxicity reference values (TRVs) has focused on guidelines that relate to a peak 1-hour exposure. There are other guidelines available that can be termed acute or short-term, however these relate to exposure periods longer than 1-hour, e.g. an 8-hour average or averaging periods up to 14 days (as is adopted by ATSDR). Guidelines for averaging periods longer than 1-hour are not preferred as the assessment would not then be comparing exposure concentrations and guidelines on the same basis.

The acute TRVs are protective of all adverse health effects for all members of the community including sensitive groups, such as children and the elderly.

For this assessment the acute TRVs have been selected on the basis of the following approach:

- Acute guidelines relevant to a 1-hour average exposure period are preferred
- The TRVs have been selected on the basis of the following hierarchy:
  1. Texas Commission on Environmental Quality (TCEQ) Acute Reference Value (Acute ReV), which is based on a target HI of 1, consistent with the target HI adopted in the derivation of guidelines in Australia (enHealth 2012a; NEPC 1999 amended 2013d, 2004) by the WHO (WHO 2000a, 2000d, 2010a). These are used as the primary source of acute guidelines as they specifically relate to and consider studies relevant to a 1-hour exposure and they have undergone the most recent detailed review process.
  2. California Office of Environmental Health Hazard Assessment (OEHHA) acute Reference Exposure Level (REL), which are all based on a target HI of 1 with RELs relevant to 1-hour average exposures adopted.
  3. Ontario Ministry of the Environment and Climate Change, with 24-hour average guideline adopted as a 1-hour average guideline.

As part of their air dispersion modelling guideline, the Ontario Ministry for the Environment reviewed the use of the power relationship to convert between averaging times (Ontario MfE 2004).

The equation used to convert between different averaging times is:

$$\text{Concentration (averaging time A)} = \text{concentration (averaging time B)} \times \left(\frac{\text{Averaging time B}}{\text{Averaging time A}}\right)^{n}$$

Where

n = stability dependent exponent based on the stability classes commonly used in air dispersion models.

These stability classes are as follows:

Stability class	n value
A&B	0.5
C	0.33
D	0.2
E&F	0.167

The literature around air dispersion modelling includes a wide range of values for n. The Ontario MfE reviewed these values. They have historically used a value of 0.28 which relates to the C & D stabilities. During consultation for this guidance in Ontario, comments were received that an average power exponent would be more relevant given that a number of the air dispersion models commonly used do not actually use stability classes. The average of the n values for the stability classes A-F is also approximately 0.28. Consequently, this value has been adopted for this review (Ontario MfE 2004).

This approach is also consistent with guidance provided by the Californian Office of Environmental Health Hazard Assessment (OEHHA 2015).

The conversion factors to be used in this review are listed in the following table.

Averaging time A	Averaging time B	Adjustment factor
Annual average	1 hour average	Multiply by 12.5
24 hour average	1 hour average	Multiply by 2.5
8 hour average	1 hour average	Multiply by 1.7
3 minute average	1 hour average	Multiply by 0.43

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

Table A1: Acute TRVs adopted in this assessment

Metal	Acute air guideline - health (mg/m^3)
Silver	0.0025 [M]
Arsenic	0.0099 [T]
Cadmium	0.00055 [T]
Cobalt	0.00069 [T]
Copper	0.1 [O]
Mercury	0.0006 [O]

References for health-based acute air guidelines (1-hour average):
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
M = Guideline available from Ontario Ministry of the Environment and Climate Change (OMECC), as Ambient Air Quality Criteria that are protective of health. The value adopted relates to a 24-hour average, which has been converted to a 1 hour average as described above https://www.ontario.ca/page/ontarios-ambient-air-quality-criteria

A3 Values adopted for the assessment of chronic exposures

A3.1 General

Chronic toxicity reference values (TRVs) associated with inhalation, ingestion and dermal exposures have been adopted from credible peer-reviewed sources as detailed in the NEPM (NEPC 1999 amended 2013b) and enHealth (enHealth 2012a). The identification of the most appropriate and robust TRVs has followed guidance from Australia (enHealth 2012a), as noted above.

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

For the metals evaluated in this assessment, TRVs relevant to all exposure pathways have been adopted as detailed in the toxicity summaries provided in the following sections. The TRVs and assumptions adopted are summarised in **Table A2**.

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.

Table A2: Summary of chronic TRVs adopted for chemicals – threshold effects

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**
Silver	0.02 R	0.0057 NH	4%	0	0%	0%
Arsenic	0.000067 T	0.002 N	100%	0.03	50%	50%
Cadmium	0.000005 W	0.0008 W	100%	0.001	60%	60%
Cobalt	0.0001 W	0.0014 D	100%	0	20%	20%
Copper	0.49 R	0.14 W	100%	0	60%	60%
Mercury (as inorganic and elemental)	0.0002 W	0.0006 W	7%	0	40%	40%
Lead	0.007 R	0.002 X	100%	0	50%	50%
Zinc	1.75 R	0.5 NH	100%	0	80%	80%

* GI factor and dermal absorption values adopted from RAIS (accessed in 2021) (RAIS)

** Background intakes relate to intakes from inhalation, drinking water and food products. The values adopted based on information provided in the ASC-NEPM (NEPC 1999 amended 2013d) and relevant sources as noted for the TRVs. Background intakes (as % of TRV) have been adopted to be the same vial all pathways.

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 2009b)

X = TRV relevant to ensuring lead exposures remain less than 5 µg/dL, above which is considered to be associated with higher than background levels of exposure as pe NHMRC (NHMRC 2015a). Note that a value relevant to 10 is protective of adverse health effects, hence this approach is conservative.

N = Arsenic values consistent with the ASC-NEPM evaluation (NEPC 1999 amended 2013d)

NH = Values consistent with that adopted by NHMRC to assess intakes in drinking water (NHMRC 2011 updated 2021)

T = TRV available from TCEQ, relevant to chronic inhalation exposures (and HI=1) (TCEQ 2012)

W = TRV available from the WHO, relevant to chronic inhalation exposures (WHO 2000d, 2006b, 2017), noting inhalation value adopted for mercury is for elemental mercury (WHO 2003b)

A3.2 Silver

The toxicity of silver has been considered in the development of the Australian Drinking Water Guideline value of 0.1 mg/L(NHMRC 2011 updated 2021). In addition, silver has also been considered by the ATSDR (ATSDR 1990). The following information is based on the information provided in these evaluations.

Silver is one of the basic elements that make up our planet. Silver is rare but occurs naturally in the environment as a soft, "silver" coloured metal. Because silver is an element, there are no manmade sources of silver. People make jewellery, silverware, electronic equipment, and dental fillings with silver in its metallic form. It also occurs in powdery white (silver nitrate and silver chloride) or dark-gray to black compounds (silver sulfide and silver oxide). Silver could be found at hazardous waste sites. It would usually be present as one or more of these salts if present at such sites and mixed with soil and/or water. Therefore, these silver compounds will be the main topic of this profile. Throughout the profile, the various silver compounds will at times be referred to simply as silver.

Photographers use silver compounds to make photographs. Photographic materials are the major source of the silver that is released into the environment. Another source is mines that produce silver and other metals.

The natural wearing down of silver-bearing rocks and soil by the wind and rain also releases large amounts of silver into the environment.

Most people are exposed daily to very low levels of silver mainly in food and drinking water, and less in air. The silver in these sources is at least partially due to naturally occurring silver in water and soil.

Although silver can be found in many biological substances, it is not considered an essential trace element for mammals. It has been estimated that less than 10% of dietary silver is absorbed by the gastrointestinal tract (RAIS indicates absorption is 4%).

Silver is stored mainly in the liver and skin and is capable of binding to amino acids and proteins. The best-known clinical condition of silver intoxication is argyria, which results in a (permanent) bluish-grey metallic discolouration of the skin, hair, mucous membranes, mouth and eye. Most cases have been associated with self-administration of silver preparations, or occupational exposure to silver and silver compounds.

Experiments with laboratory rats and mice have reported similar results. Very high concentrations of silver in drinking water (over 600 mg/L) for a lifetime caused discolouration in the thyroid and adrenal glands, the choroids of the eyes, the choroid plexus of the brain, and the liver and kidney. Some hypoactive behaviour was also reported.

No data are available on the carcinogenicity of silver. Silver salts are not mutagenic in tests with bacteria, but can induce damage in mammalian DNA.

The oral TRV for silver is 0.4 mg/day based on a human lifetime no effect level of 10 grams. The no effect level is from a human study and hence no uncertainty factor is applied. To get a TRV for use in risk assessment this value has been derived by the lifetime body weight of 70 kg, to get 0.0057 mg/kg/day (NHMRC 2011 updated 2021).

No inhalation values are available for silver, hence the oral value is adopted and extrapolated for inhalation exposures as per USEPA (USEPA 2009b).

Intakes from sources such as water and food are considered negligible, compared with the no effect level identified.

Recommendation

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

- Oral TRV (TRV_O) = 0.0057 mg/kg/day (NHMRC 2011 updated 2021) for all routes of exposure
- Background intakes from other sources (as % of TRV) = negligible.

A3.3 Arsenic

Background

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

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

- The most recent Australian Total Diet Survey (ATDS) that addresses arsenic in food was published by FSANZ in 2011 (FSANZ 2011). Based on data presented in this report, dietary intake of arsenic for children aged 2-5 years ranges from a mean of 1.2 µg/kg/day to a 90th percentile of 2.8 µg/kg/day. These intakes are based on total arsenic in produce, rather than inorganic arsenic.
- Review of background intakes from food, water, air, soil and contact with play equipment based on available Australian data presented by (APVMA 2005) suggests background intakes of inorganic arsenic by young children may be on average 0.62 µg/kg/day. Further review of inorganic arsenic intakes by the Joint FAO/WHO Expert Committee on Food Additives indicated that for populations (not located in areas of arsenic contaminated groundwater) intakes by young children ranged from 0.14 to 1.39 µg/kg/day (WHO 2011b). 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 assumed to be 50% of the TRV.

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 2012).

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 2012) 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 2012) 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.

Revision to the WHO guidelines on drinking water (WHO 2011a) adopted a practical value based on the analytical limit of reporting rather than based on a dose-response approach. The oral slope factor derived by the USEPA has not been used to derive a guideline as the slope factor is noted by the WHO as likely to be an overestimate.

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

- the choice of the cancer endpoint;
- the choice of the mathematical model used to estimate risk (shape of the dose-response curve at low doses) as there is no clear biological basis for extrapolation; and
- the assumptions used to estimate exposure from studies (primarily epidemiological studies) (Boyce et al. 2008; Brown 2007; Chu & Crawford-Brown 2006; Lamm & Kruse 2005; SAB 2005).

Review of recent studies presented by (Boyce et al. 2008) has indicated that for carcinogenic effects associated with arsenic exposure a linear (or non-threshold) dose-response is not supported (also note discussion by (Clewell et al. 2007). This is based on the following:

- Epidemiological studies (worldwide) that have repeatedly demonstrated that cancers associated with inorganic arsenic ingestion are observed only in populations exposed to arsenic concentrations in drinking water that are greater than 150 µg/L. In the US, exposures to concentrations in drinking water have only been associated with carcinogenic effects where mean concentrations are greater than 190 µg/L (Schoen et al. 2004).
- Mechanistic information on how arsenic affects the cellular processes associate with carcinogenicity. This includes consideration that arsenic and its metabolites may modify DNA function through more indirect mechanisms such as inhibition of DNA repair, induction of dysfunctional cell division, perturbation of DNA methylation patterns, modulation of signal transduction pathways (leading to changes in transcriptional controls and the over-stimulation of growth factors), and generation of oxidative stress (ATSDR 2007b; IARC 2012) and that evidence for the indirect mechanisms for genotoxicity identified in in vitro studies have nearly all been at concentrations that are cytotoxic (Klein et al. 2007).

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

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

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

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

Assessment of End-Points – Oral Exposures

Existing Oral Dose-Response Approaches - Australia

Oral intakes of arsenic were considered in Australia in (Langley 1991) and the Australian Drinking Water Guidelines (ADWG) (NHMRC 2011 Updated 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 2011b) 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 2021) 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 2011b) 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 2010b). 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 2010b) 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 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 2010b; WHO 2011b) 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 2010b; WHO 2011b). 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 2010b)) 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 2010b; WHO 2011b) 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 2010b; WHO 2011b);
- The value is within the range of no observable adverse effect levels (NOAELs) identified by RIVM (Baars et al. 2001), US EPA (USEPA IRIS) and ATSDR (ATSDR 2007b) that are associated with non-carcinogenic effects (and derived from drinking water studies in Taiwan and Bangladesh) of 0.8 to 8 µg/kg/day. Consistent with the approach discussed above in relation to the range of TRVs relevant to a cancer endpoint, it is not considered appropriate that the most conservative end of this range is adopted for the Australian population.

Due to the level of uncertainty in relation to determining a single TRV for the assessment of arsenic exposures, the oral TRV utilised is not considered to be a definitive value but is relevant for the current assessment. The approach adopted is based on developing science that should be reviewed in line with further developments in both science and policy.

The dermal absorption factor adopted for nickel in the ASC NEPM 2013 is 0.005 (NEPC 1999 amended 2013d).

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 2007b)). Applying an uncertainty factor of 10 to address variability in human susceptibility, a tolerable concentration (TC) in air of 1 μg/m^3 was derived.

Given the above, there is some basis for the assessment of inhalation exposures to arsenic to adopt an appropriate threshold value but the available epidemiological studies associated with exposures in copper smelters suggest a linear or non-threshold approach may be relevant. The WHO (2000) review of arsenic by WHO (WHO 2000e) 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 $\geq$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 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. 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 that may be derived from soil, including carcinogenicity.

Recommendation

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

- Oral TRV = 0.002 mg/kg/day for oral, dermal and inhalation intakes
- Oral Bioavailability of 100% assumed
- Dermal absorption = 0.03
- Background Intakes from other sources (as % of TRV) = 50%

A3.4 Cadmium

General

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

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 2012).

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 2012).

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

Background

The WHO review of cadmium included food intakes provided by FSANZ of 0.1 µg/kg/day (FSANZ 2003; WHO 2004). 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). 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 in Australia. 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.

For this assessment, intakes from all other sources have been assumed to be 60% of the 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 2012).

Review of Available Values/Information

The following has been summarised from the review of cadmium presented by MfE:

- 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 2012). 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 only 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.

<u>Oral (and Dermal) Intakes</u>

Insufficient data are available to assess carcinogenicity via oral intakes and, therefore, the oral TRV has been based on a threshold approach with renal tubular dysfunction considered to be the most sensitive endpoint. The following are available for oral intakes from Level 1 Australian and International sources.

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 updated 2021)	TDI = 0.0007 mg/kg/day	The threshold oral value available from the ADWG 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.
International		

Source	Value	Basis/Comments
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 2012)	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 2021).

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 2012). 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 2000d). 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).

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)
- Dermal absorption (DAF) = 0.001
- Inhalation TRV (TRV_I) = 0.000005 mg/m^3 (WHO 2000d)
- Background intakes = 60%.

A3.5 Cobalt

General

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

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 2006b).

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 2004b; WHO 2006b).

Cobalt is a dietary essential element as it is a key component of Vitamin B12 (ATSDR 2004b). 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 2004b). Cobalt in the body partakes in reactions which generate oxidants and free radicals capable of deoxyribonucleic acid (DNA) damage and other deleterious effects (ATSDR 2004b).

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 2006b). 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 2006b) (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.

For this assessment, intakes from all other sources have been assumed to comprise 20% of the 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).

It is noted that the USEPA has not evaluated cobalt with respect to classification of carcinogenicity.

Review of Available Values/Information

While data are limited, based on the weight of evidence, cobalt is not (or weakly) genotoxic (ATSDR 2004b; Baars et al. 2001). However, it is noted that some information suggests that some metallic cobalt species may be genotoxic, and this may need to be considered in occupational environments. On this basis, it is recommended that a threshold approach be adopted for the assessment of cobalt.

Few quantitative evaluations are available for cobalt, however the following are available from Level 1 Australian and International sources:

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 updated 2021)	No evaluation available	
International		
WHO DWG (WHO 2011a)	No evaluation available	
WHO (WHO 2006b)	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.
ATSDR (ATSDR 2004b)	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).
USEPA (IRIS) (USEPA IRIS)	No evaluation available	

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.

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
- Inhalation TRV (TRV_i) = 0.0001 mg/m^3 (WHO 2006b)
- Background intakes from other sources (as % of TRV) = 20% for oral intakes.

A3.6 Copper

General

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

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.

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 2021) 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 2011a) were higher, with intakes by a young child estimated to be 0.013 mg/kg/day.
- 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 concentrations are negligible compared with intakes from food.
- (Baars et al. 2001) reviewed background intakes which were considered to be 30 µg/kg/day for adults. Based on data from Australia and 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.

For this assessment, intakes from all other sources have been assumed to comprise 60% of the 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.

Review of Available Values/Information

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

The following threshold values are available from Level 1 Australian and International sources:

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 updated 2021)	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).
International		
WHO DWG (WHO 2011a)	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.
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 2004a)	No chronic MRLs available	
USEPA IRIS (USEPA IRIS)	No evaluation available	

Based on the available data an oral TRV of 0.14 mg/kg/day 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 2011a) for all routes of exposure
- Background intakes for the general population = 0.08 mg/kg/day = 60% of the oral TRV.

A3.7 Mercury

General

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 methylmercury.

This assessment has only considered mercury as inorganic mercury and elemental mercury.

Mercury occurs naturally as a mineral and 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 (ATSDR 1999; HSDB database).

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).

Properties

Elemental mercury is a dense, silvery white metal which is liquid at room temperature, readily volatilises and is considered to be the predominant form of mercury in the atmosphere. Mercury compounds differ greatly in general properties and solubility. Due to the wide range in properties associated with the forms of mercury, key properties have not been listed here, however, they are available in a number of published reviews (ATSDR 1999; WHO 2003b).

Exposure

Exposure of the general population to mercury may occur via inhalation, oral or dermal contact. Exposure to elemental mercury may occur in the workplace or home if mercury is spilled. Inorganic mercury compounds are found in some batteries, pharmaceuticals, ointments and herbal medicines. Exposure to inorganic mercury can occur via inhalation or ingestion. Methylmercury is most commonly found in fish, especially larger fish at the top of the food chain with exposure typically associated with ingestion.

Current literature indicates that mercury (Hg) 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, in groundwater, is dependent on: biogeochemical processes; partitioning between solids, groundwater, and vapour; and complexation with dissolved organic and inorganic ligands. Redox, pH conditions, and groundwater composition are, consequently, all important components of determining the likely form, and, therefore, potential fate of mercury in the environment.

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. The UN Governing Council decided that national, regional and global actions should be initiated as soon as possible and urged all countries to adopt goals and take actions, as appropriate, to identify populations at risk and to reduce human-generated releases.

Background Exposure/Intake

Background intakes from food, water and air were listed in the documentation associated with the derivation of the current health investigation level (HIL) for soil (Imray & Neville 1996), with the total intake of mercury (derived from inorganic or elemental sources, both of which add to the body burden of mercury) estimated for a 2 year old child was 2.1 µg/day (50% of the adopted tolerable intake of 5 µg/day which was based on methylmercury rather than inorganic mercury). The most significant exposures were derived from dietary intakes and dental amalgams.

Review of current information from Australia indicates the following:

- Mercury levels are reported in the 20th Australian Total Diet Survey (FSANZ 2003). Dietary intakes of total mercury (which includes organic mercury in seafood) ranged from 0.01 to 0.2 µg/kg/day for toddlers (aged 2 years). This is consistent with intakes reported in the more recent survey (FSANZ 2011).
- Typical concentrations of mercury reported in drinking water in the ADWG (NHMRC 2011 updated 2021) 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.
- Review (NHMRC 1999b) 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 the use of mercury dental amalgams has declined.
- Based on the above, background intakes by young children may be up to 0.23 µg/kg/day from oral intakes (dietary, dental and water). This is slightly higher than estimated intakes of 0.1 µg/kg/day from the Netherlands (Baars et al. 2001) and 0.037 µg/kg/day from the UK (UK EA 2009b) for a 20kg child. These intakes comprise approximately 40% of the recommended oral TRV.
- Levels of inorganic mercury in air are not available for Australia with estimates from the WHO (2003) for mercury in air ranging from 10 to 20 ng/m^3 from the US (no indication of speciation between elemental and inorganic). These concentrations comprise up to 10% of the recommended inhalation TRV.

For this assessment, intakes from all other sources have been assumed to be 40% of the TRV.

Health Effects

The following information is available from UK (UK EA 2002, 2009b) and ATSDR (1999).

Elemental Mercury (Hg^0)

General

Limited data are 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 and the vapour inhalation pathway is of most importance.

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.

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 a salt. Approximately 80% is excreted as salt via faeces and urine. Half-life elimination is approximately 58 days.

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.

Carcinogenicity and Genotoxicity

Both USEPA and IARC indicate that elemental mercury is not classifiable as to its human carcinogenicity. No adequate animal studies are available for elemental mercury and occupational studies have indicated conflicting results.

Inorganic Mercury Compounds

General

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.

Carcinogenicity and Genotoxicity

IARC have considered inorganic mercury compounds not classifiable as to human carcinogenicity. The USEPA has classified mercuric chloride as a possible human carcinogen (Class C) based on increased incidence of squamous cell papillomas of the forestomach and marginally increased incidence of thyroid follicular cell adenomas and carcinomas from a long term oral studies in rats.

Carcinogenicity studies in experimental animals are available on mercuric chloride only where no carcinogenic effect was observed in mice or female rats, while 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*, where 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 2011a). The current US evaluation (USEPA IRIS) of mercuric chloride indicates that a linear low-dose extrapolation is not appropriate as kidney tumours seen in mice occurred at doses that were also nephrotoxic (i.e. at elevated doses). On this basis, in accordance with Australian (enHealth 2012a) guidance it is not considered appropriate that a non-threshold dose-response approach is adopted for the assessment of mercuric chloride.

Quantitative Toxicity Values

Review of toxicological studies and risk assessments by several countries and international organisations have established levels of daily or weekly intakes of mercury that are estimated to be "safe" (refer to the WHO (UNEP 2008) review). That is, there is a threshold or reference level below which exposures/intakes are not associated with adverse effects. The WHO makes it clear in their assessment that these reference levels are not a clear dividing line between safe and unsafe. This is because they have incorporated a number of safety/uncertainty factors into their calculation of the reference level for mercury which means a slight exceedance of this value does not immediately result in adverse effects.

On the basis of the available information in relation to elemental and inorganic mercury, a threshold approach is consider appropriate based on the most sensitive effect associated with mercury exposure. The following threshold values are available from relevant Australian and International sources.

Toxicity Reference Values for Inorganic and Elemental Mercury

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 updated 2021)	NA	Guideline established on the basis of methylmercury only
FSANZ (FSANZ 2011)	NA	Value for total mercury referenced from JECFA 1989, based on methylmercury
International		
WHO DWG (WHO 2011a)	TDI = 0.002 mg/kg/day	The current WHO DWG (2011, consistent with the previous evaluation conducted in 2003) has derived a guideline of 0.006 mg/L based on a TDI of 0.002 mg/kg/day derived from a NOAEL of 0.23 mg/day associated with kidney effects in a 26-week study in rats and an uncertainty factor of 100. A similar TDI was derived on the basis of a LOAEL of 1.9 mg/kg/day associated with renal effects in a 2-year rat study and an uncertainty factor of 1000.
JECFA (JECFA 2011)	**PTWI = 0.004 mg/kg (equivalent to PTDI = 0.0006 mg/kg/day)**	Review of mercury by JECFA indicated that the predominant form of mercury indoors, other than fish and shellfish, is inorganic mercury and while data on speciation is limited the toxicological database on mercury (II) chloride was relevant for establishing a PTWI for foodborne inorganic mercury. A PTWI was established on the bases of a benchmark dose approach, where the $BMDL_{10}$ of 0.06 mg/kg/day for relative kidney weight

Source	Value	Basis/Comments
		increases in male rates was considered as the point of departure. A 100 fold uncertainty factor was applied.
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 "since cationic inorganic mercury is retained only half as much as the vapour, the guideline also protects against mild renal effects caused by cationic inorganic mercury". "Present knowledge suggests, however, that effects of the immune system at lower exposures cannot be excluded".
WHO (WHO 2003b)[1]	TDI = 0.002 mg/kg/day **TC = 0.0002 mg/m^3**	TDI derived for inorganic mercury 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 a LOAEL associated with CNS effects in workers exposed to elemental mercury. The evaluation provides a revision on the limited TC presented in the WHO (2000).
UK (UK EA 2009b)	TDI = 0.002 mg/kg/day TC = 0.0002 mg/m^3	TDI referenced from the WHO (2003) and WHO DWG (2011). Inhalation value (covered to a does by the UK) based on the WHO (2003) value assumed to be relevant to inorganic mercury in air.
RIVM (Baars et al. 2001)	TDI = 0.002 mg/kg/day TC = 0.0002 mg/m^3	TDI for mercuric chloride derived on the same basis as WHO. TC derived on the same basis as ATSDR and WHO (2003).
ATSDR (ATSDR 1999)	Inh. MRL = 0.0002 mg/m^3	No chronic duration MRLs have been derived for inorganic mercury. An intermediate duration (or sub-chronic) oral MRL of 0.002 mg/kg/day was derived. The chronic inhalation MRL for elemental mercury based on a LOAEL (HEC) of 0.0062 mg/m^3 associated with CNS effects in workers and an uncertainty factor of 30.
USEPA (IRIS)	RfD = 0.0003 mg/kg/day RfC = 0.0003 mg/m^3	RfD (last reviewed in 1995) for inorganic mercury 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. RfC (last reviewed in 1995) for elemental mercury based on a LOAEL (HEC) of 0.009 mg/m^3 associated with CNS effects in workers and an uncertainty factor of 30. A subchronic RfC is also available from HEAST (1995), which is equal to the chronic RfC.

Notes:

1 This document is an update of a former evaluation of inorganic mercury presented in the WHO EHC 118 (WHO 1991). In this evaluation the WHO states that following review of a number of animal studies in relation to inorganic mercury, no "no-observed-adverse-effect-level" (NOAEL) could be determined. This is a reflection of the limitations in the available animal studies rather than because there is no safe dose. These studies typically only consider perhaps 3-4 different doses and depending on the spacing of the quantitative magnitude of these doses it may or may not be possible to ascertain a dose which could be a NOAEL as the lowest dose use in the study may have been too high resulting in some effects being observed at all the dose levels. Hence this is not a definitive statement in relation to the determination of whether or not there is a safe level of mercury exposure and certainly does not imply that the WHO evaluation has stated that the safe dose for mercury is zero. It is important to note that since the 1991 WHO evaluation there have been numerous more robust studies undertaken that have enabled a safe dose to be more reliably determined as outlined in this table.

The PTWI derived for inorganic mercury available from JECFA (2011) is considered to provide the most current review of the available studies in relation to exposure to inorganic mercury and has been adopted for the assessment of exposure to inorganic mercury, via all pathways of exposure.

Inhalation values for elemental mercury are derived from occupational studies associated with elemental mercury vapour. The more current review provided by WHO (2003), consistent with that adopted by UK (UK EA 2009b), RIVM (Baars et al. 2001) and ATSDR (1999), has been adopted for the assessment of inhalation exposures to elemental mercury. Limited subchronic evaluations are available and hence the chronic TRV has been adopted for the assessment of sub-chronic exposures.

Limited subchronic evaluations are available and hence the chronic TRV has been adopted for the assessment of sub-chronic exposures.

Recommendation

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

- Oral TRV (TRV_O) = 0.0006 mg/kg/day (JECFA 2011) for ingestion and dermal
- Inhalation TRV (TRV_i) = 0.0002 mg/m^3 (WHO 2003b)
- Background intakes for the general population are 40%.

A3.8 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 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 2006) 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 have been reported from (FSANZ 2003, 2011). Intakes reported in this study range from 0.02-0.4 µg/kg/day for adults to 0.01-1.2 µg/kg/day for infants. These data are the most current from FSANZ.
- The ADWG (NHMRC 2011 updated 2021) notes that lead concentrations in drinking water range up to 0.01 mg/L with typical concentrations less than 0.005 mg/L. Data available from South Australia (based on 5 years of data) suggest concentrations of lead in drinking water are on average 0.0007 mg/L, with a maximum of 0.014 mg/L. Intakes derived for a young child (consuming 1 L/day and a body weight of 15.5 kg) are approximately 0.04 µg/kg/day.
- 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 estimated to be 0.41 µg/kg/day for adults and 1.24 for children based on intakes from dietary and water sources.
- 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). For adults this results in an intake of 0.06 µg/kg/day and for young children this is 0.5 µg/kg/day.

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 2014b). 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 2014b).

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 2000c). 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 2011b):

- 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.
    - 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 2011b) 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 2013d) – this reflects the gastrointestinal absorption, with 100% bioaccessibility from soil assumed.

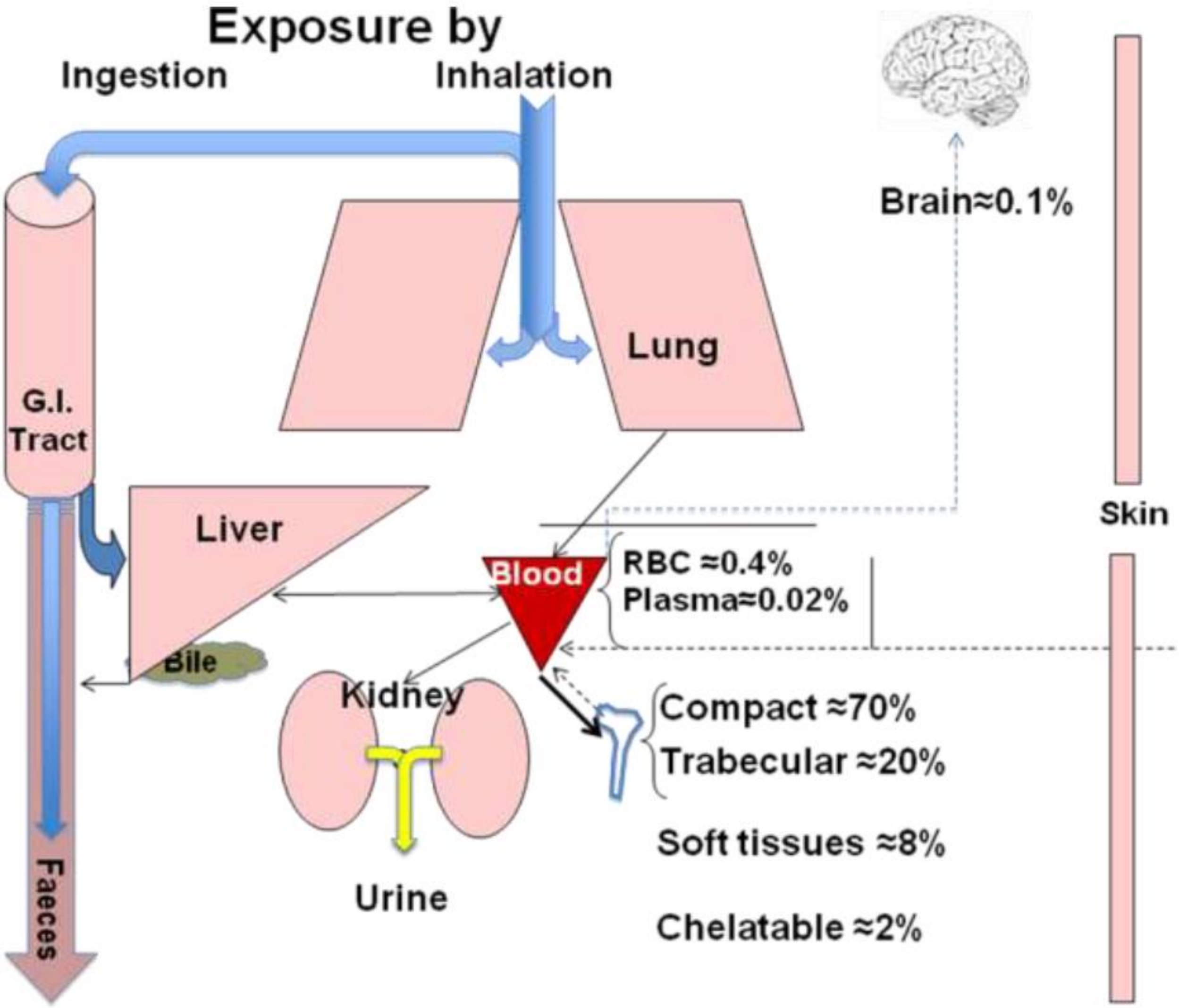
Where site specific bioaccessibility is available the bioavailability is adjusted to be 50% absorption x bioaccessible fraction.

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 2006). This is consistent with approaches adopted in New Zealand (MfE 2011b) and the UK (UK DEFRA & EA 2002). 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 2010a).



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

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 2014b). 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 2014b).

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 2010a; 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 2019).

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

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

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

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

The International Agency for Research on Cancer (IARC 2006) has classified inorganic lead as Group 2A: probably carcinogenic to humans. Organic lead was classified as Group 3: not classifiable (IARC 2006). 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 2011a). 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 2002; 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).

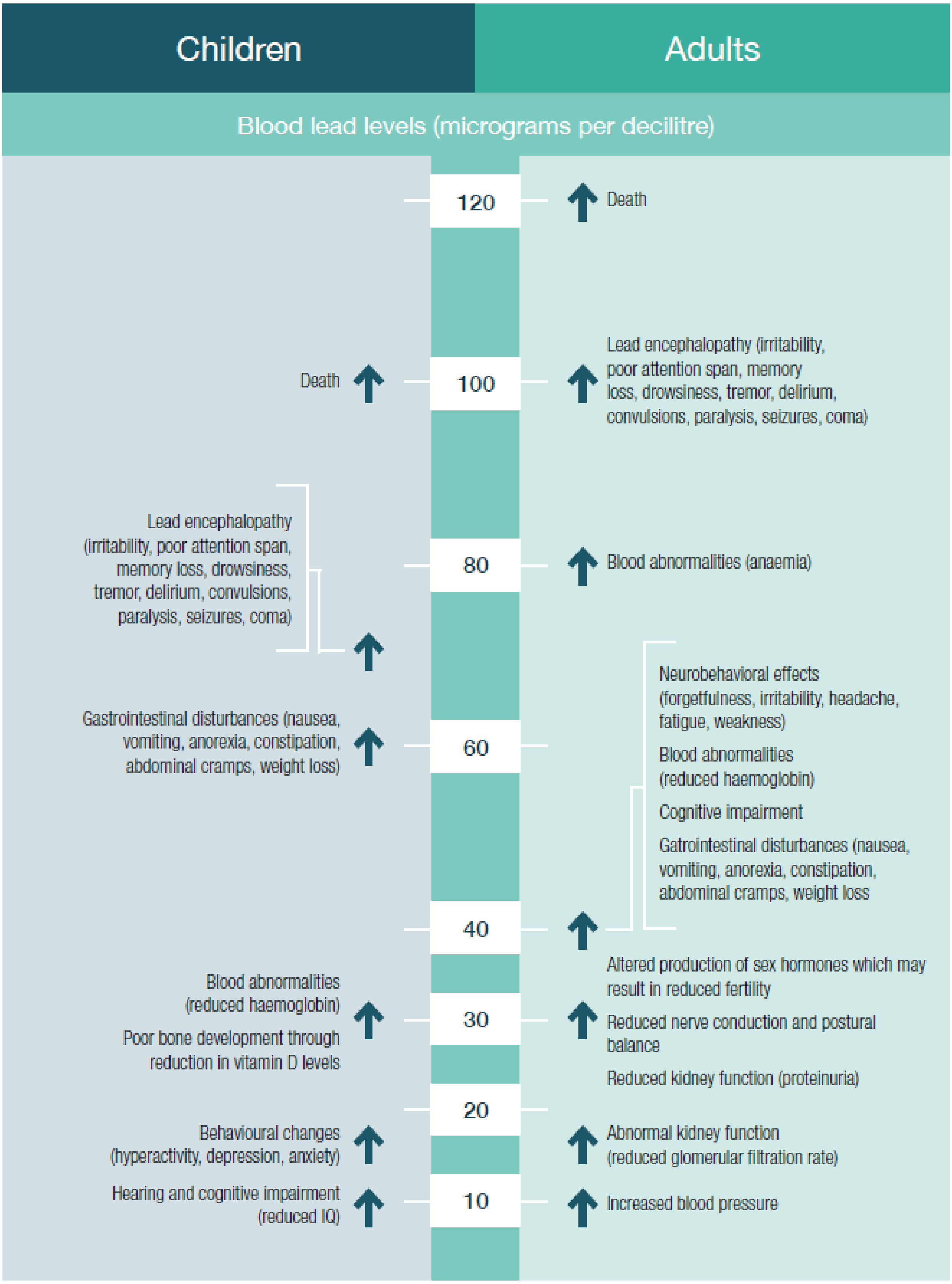


Figure: Summary of health effects of lead exposure above 10 µg/dL

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 2013d) 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.

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 updated 2021)	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).
FSANZ (FSANZ 2003)	PTDI = 0.0035 mg/kg/day	As for ADWG above.
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 1998a, 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 2014a)	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.
International		
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 2000e)	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 2010a)	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 2010a) 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.

The DEFRA (UK DEFRA & EA 2014) review has considered a range of health effects, with neurobehavioral effects in children, cardiovascular and kidney effects in adults being identified as the critical health endpoints. Review of the available studies relevant to these critical health endpoints has been undertaken by NHMRC (Armstrong et al. 2014) determined that the studies related to the health effects reported (and where there is an association) 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 was the cause of the effects reported. Health effects at blood lead level of 10 µg/dL and higher are considered to be causal. While DEFRA (UK DEFRA & EA 2014) has not critically reviewed the studies in the same way, the NHMRC (Armstrong et al. 2014) review forms the policy approach to the assessment of lead exposures in Australia.

The DEFRA review, however has undertaken blood lead modelling to establish intakes of lead (from all sources) that may result in specific blood lead levels. The DEFRA (UK DEFRA & EA 2014) review considered a range of blood lead levels, as well as biokinetic modelling specific to children and adults. The blood lead models utilised by DEFRA include the IEUBK model and Adult Lead Model (ALM) adopted in the NEPM (NEPC 1999 amended 2013d), along with the Carlisle and Wade (Carlisle & Wade 1992) model for adults. The biokinetic modelling includes consideration of the absorption of lead, however it does not account for site-specific bioaccessibility.

For the blood lead goals established by NHMRC the intakes of lead (from all sources) required to achieve those levels are as follows:

- Blood lead level considered representative of background exposures ≤5 µg/dL
    - Intakes by adults = 1.8 µg/kg/day (based on the Carlisle & Wade) model
    - Intakes by children = 2.1 µg/kg/day

  The above result in a value of 2 µg/kg/day that may be adopted for both adults and children.

- Blood lead level where health effects are of concern ≥ 10 µg/dL
    - Intakes by adults = 3.6 µg/kg/day (via extrapolation of modelling)
    - Intake by children = 4 µg/kg/day (via extrapolation of modelling).

  The above intakes are consistent with the TRV adopted by NHMRC in the derivation of the Australian Drinking Water guideline (NHMRC 2011 updated 2021).

  For the purpose of this assessment a **TRV of 2 µg/kg/day** relevant to background blood lead levels has been adopted. This is a conservative approach.

A3.9 Zinc

General

Several comprehensive reviews of zinc in the environment and toxicity to humans are available (ATSDR 2005; WHO 2001a).

Zinc is ubiquitous in the environment and occurs in the earth's crust at an average concentration of about 70 mg/kg. Zinc is not found in elemental form in nature and occurs in the +2 oxidation state primarily as various minerals such as sphalerite (zinc sulfide), smithsonite (zinc carbonate), and zincite (zinc oxide). Fifty-five zinc containing minerals are known to exist. In its pure elemental (or metallic) form, zinc is a bluish white, shiny metal (WHO 2001a).

Most rocks and many minerals contain zinc in varying amounts. Commercially, sphalerite (ZnS) is the most important ore mineral and the principal source of the metal for the zinc industry (WHO 2001a).

Inorganic zinc salts have numerous commercial uses. Zinc oxide is used in the rubber industry as a vulcanisation activator and accelerator and to slow down oxidation, and also as a reinforcing agent, heat conductor, pigment, UV stabilizer, supplement in animal feeds and fertilisers, catalyst, chemical intermediate, and mildew inhibitor. Zinc sulfate is used in rayon manufacture, agriculture, zinc plating, and as a chemical intermediate and mordant. Zinc chloride is used in smoke bombs, in cements for metals, in wood preservatives, in flux for soldering; in the manufacture of parchment paper, artificial silk, and glues; as a mordant in printing and dye textiles, and as a deodorant, antiseptic and astringent. Zinc chromate is used as a pigment in paints, varnishes, and oil colours. In addition, zinc phosphide is used as a rodenticide while zinc cyanide is used in electroplating (WHO 2001a).

Zinc is an essential element for all living things, including people. Zinc-containing proteins and enzymes are involved in every aspect of metabolism, including the replication and translation of genetic material. Hence adverse effects are associated with deficiency as well as toxicity associated with excess intake. Zinc deficiency has been reported to affect children of many countries while other groups identified at particular risk are women of child-bearing age and elderly. The main cause of human zinc deficiency is consumption of diets that contain little highly bioavailable zinc (NEHF 1997).

Background

Review of current information from Australia indicates the following:

- Zinc in dietary intakes has been assessed most recently in the 20th and 23rd Total Diet Survey where mean dietary exposures ranged from 0.627 mg/kg/day for infants and 0.5 mg/kg/day for toddlers aged 2-3 years to 0.128 mg/kg/day for adult females (FSANZ 2003, 2011). These intakes were higher than the recommended daily intakes (RDI) established by NHMRC (as noted by FSANZ 2003) for adult males, boys, toddlers and infants and lower than the RDI for adult females and girls. The RDI for zinc ranges from 3 mg/day for breastfed infants, 3-6 mg/day for formula fed infants to 4-5 mg/day for children aged 7 months to 3 years, 6 mg/day for 4-7 year olds, 9 mg/day for 8-11 year olds and 12 mg/day for 12-18 year olds (NHMRC 2006). The mean intake by infants was considered to comprise up to 63% of the tolerable limit of 1 mg/kg/day established by the WHO.

- Typical concentrations of zinc reported in the ADWG are up to a maximum 0.26 mg/L with typical concentrations less than 0.05 mg/L. Based on typical and maximum concentrations these result in intakes (1 L/day and body weight of 15.5 kg) by toddlers of 3 to 20 µg/kg/day (NHMRC 2011 updated 2021).
- Zinc was reported in ambient air data collected in NSW where concentrations (24-hour averages) in urban, regional and industrial areas assessed ranged from 11 to 71 ng/m^3 (average of 33 ng/m^3) (NSW DEC 2003). These concentrations are consistent with those reported in New Zealand and Canada (HSDB) but lower than those reported in the US and Germany (from older data) (WHO 2001a) and the UK (HSDB database). Based on the mean concentration reported in Australian air, intakes by young children is approximately 25 ng/kg/day, significantly less than intakes from food and water.
- Based on the above, background intakes by young children (2 years) are estimated to be approximately 0.4 mg/kg/day (dominated by dietary intakes), which is above the RDI of 0.32 mg/kg/day and approximately 80% of the recommended TDI. Intakes estimated by the WHO for infants and children aged 2 months to 19 years range from 5.6 to 13 mg/day (from dietary intakes) (WHO 2001a). For a 2 year old child these intakes range from 0.4 to 0.9 mg/kg/day (80% to greater than 100% of the recommended TD). Based on mean intakes from Australian data, background intakes can be assumed to comprise up to 80% of the recommended oral TRV.

Classification

The International Agency for Research on Cancer (IARC) has not evaluated zinc with respect to human carcinogenicity.

It is noted that the USEPA has evaluated zinc in their 2005 review (USEPA 2005b). The evaluation notes "*there is inadequate information to assess carcinogenic potential of zinc*" because studies of humans occupationally-exposed to zinc are inadequate or inconclusive, adequate animal bioassays of the possible carcinogenicity of zinc are not available, and results of genotoxic tests of zinc have been equivocal.

Review of Available Values/Information

Insufficient information is available to adequately assess zinc for carcinogenicity. The WHO (2001) notes that the weight of evidence supports the conclusion that zinc is not genotoxic or teratogenic. At high concentrations zinc can be cytotoxic (i.e. kills cells). Other reviews of genotoxicity studies for zinc by EU and USEPA are equivocal (EU 2003; USEPA 2005b). The EU (2003) review concluded that: *In vitro* tests indicated that zinc has a genotoxic potential, while the *in vivo* studies as presented are inconclusive with sometimes contradictory results. However, there are indications of some weak clastogenic, and possibly aneugenic effects following zinc exposure. The relevance of these findings needs to be clarified.

On the basis of the available information, consideration of a threshold approach for the quantification of risks due to zinc exposure is considered reasonable. It is noted that since zinc is an essential element, a number of the threshold values available are associated with recommended dietary intakes (RDIs) or adequate intake (AI) and associated upper limits (ULs) based on available studies. It is noted that in reviewing the available information threshold values such as TDIs or RfDs should lie between the RDI or AI and the UL established for zinc intakes. TDIs or RfDs that are lower than the RDI or AI are considered overly conservative and may lead to deficiency. The following quantitative values are available from Level 1 Australian and International sources.

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 updated 2021)	No health based guideline established	The ADWG has not derived a health based guideline for zinc with the current guideline based on aesthetic considerations (taste).
FSANZ (FSANZ 2003)	TDI = 1 mg/kg/day	TDI noted to be derived from the WHO (refer to comments provided below from JECFA).
NHMRC (NHMRC 2006)	Infants: AI = 2-3 mg/day UL = 4-5 mg/day 1-3 years: RDI = 3 mg/day UL = 7 mg/day Children 4-18 yrs: RDI = 4-13 mg/day UL= 12-35 mg/day Adults: RDI = 8-14 mg/day UL = 35-40 mg/day including during pregnancy and lactation	The upper limit (UL) applies to total zinc intake from food, water and supplements (including fortified food). The UL for infants is based on a NOAEL at a level of 5.8 mg zinc/L of infant formula fed for 6 months, equal to a NOAEL of 4.5 mg/day at 0.78 L milk per day. An UF of 1 was applied, given the length and quality of the study and the fact that there is no evidence of harm from intakes of formula at 5.8 mg zinc/L. Rounding down; a UL of 4 mg was therefore set for infants of 0–6 months. As there were no data for older children and adolescents, this figure was adjusted on a body weight basis, for older infants, children and adolescents and values rounded down. The adverse effect of excess zinc on copper metabolism has been identified as the critical effect on which to base the adult UL. This is based on the consistency of findings from a number of studies where the sensitivity of the marker used (erythrocyte copper-zinc superoxide dismutase) and the quality and completeness of the database for this endpoint. A LOAEL of 60 mg/day was adopted (and is supported by other studies). An UF of 1.5 is applied to account for inter-individual variability in sensitivity and for extrapolation from a LOAEL to NOAEL. As reduced copper status is rare in humans, a higher UF was unjustified. The adult UL was therefore set at 40 mg/day.
International		
WHO DWG (WHO 2017)	No health based guideline established	The current WHO DWG (2011) derived a guideline of 3 mg/L based on aesthetic issues. The review notes that in 1982, JECFA proposed a daily dietary requirement of zinc of 0.3 mg/kg of body weight and a provisional maximum tolerable daily intake (PMTDI) of 1.0 mg/kg of body weight. The daily requirement for adult humans is 15–22 mg/day. Hence it was concluded that the derivation of a health-based guideline value is not required.
JECFA (WHO 1982)	TDI = 1 mg/kg/day	Provisional maximum tolerable daily intake estimated to be 1 mg/kg/day based on the evaluation that there is a wide margin between nutritionally required amounts of zinc and toxic levels. Clinical studies in which up to 600 mg of zinc sulfate (equivalent to 200 mg elemental zinc) has been administered daily in divided doses for a period of several months, provides a basis for the evaluation.
RIVM (Baars et al. 2001)	TDI = 0.5 mg/kg/day	TDI derived on the basis of a LOAEL (adjusted) of 1 mg/kg/day associated with haematological effects in a 1989 human study (from supplements) and an UF of 2.
ATSDR (ATSDR 2005)	MRL = 0.3 mg/kg/day	Chronic oral MRL derived based on a NOAEL of 0.83 mg/kg/day from the same study considered by RIVM (however interpretation of the study differed) and an UF of 3.

Source	Value	Basis/Comments
USEPA (USEPA 2005b)	RfD = 0.3 mg/kg/day	RfD (last reviewed in 2005) based on a LOAEL of 0.91 of 0.015 mg/kg/day, identified as the point of departure associated with haematological effects from a number of oral human studies published from 1984 to 2000 (including the study considered by ATSDR and RIVM) and an UF of 3.

It would be relevant and consistent to consider potential exposures to zinc in soil on the same basis as considered by FSANZ (also noted in WHO DWG (WHO 2017)) where dietary intakes are addressed). However, it is noted that the upper limit of zinc intakes identified for children by NHMRC (NHMRC 2006) is lower than that considered in the Australian Total Diet Survey (FSANZ 2003), where an upper limit of 7 mg/day for children aged 1-3 years, equivalent to 0.5 mg/kg/day (based on a 15.5 kg child) is identified. This is the same as derived by RIVM (Baars et al. 2001) and is lower than the upper limit recommended for adults of 40 mg/day, equivalent to 0.57 mg/kg/day (based on 70 kg adult). It is recommended that the lower value for children of 0.5 mg/kg/day recommended by NHMRC (2006) be adopted.

There are no dermal or inhalation specific values available for zinc, therefore, the TDI adopted is considered relevant for all intakes.

Recommendation

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

- Oral TRV (TRV_O) = 0.5 mg/kg/day for all routes of exposure (NHMRC 2006)
- Background intakes from other sources (as % of TRV) = 80%.

Appendix B Methodology and assumptions

B1 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.

B2 Quantification of inhalation exposure

Intakes via inhalation has been assessed on the basis of the inhalation guidance available from the USEPA and recommended for use in the ASC NEPM and enHealth (enHealth 2012a; NEPC 1999 amended 2013d; USEPA 2009b).

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 B1** 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)	Particulate bound chemicals = 1	For particulates, these assessed on the basis of the concentration bound to PM_{10} or $PM_{2.5}$, 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, every day of the year. Workers: Working 8 hours per day, 5 days per week for 48 weeks of the year (enHealth 2012a)
EF	Exposure frequency (days/year)	Residents = 365 days Workers = 240 days	
ED	Exposure duration (years)	14 years	Duration of Project, inhalation exposures can occur as a result of Project activities
AT	Averaging time (hours)	Threshold = ED x 365 days/year x 24 hours/day Non-threshold = 70 years x 365 days/year x 24 hours/day	As per enHealth (enHealth 2012a) guidance

B3 Multiple pathway exposures

B3.1 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 enHealth and the USEPA (enHealth 2012a; USEPA 1989). This approach is presented in the following equation, and parameters adopted in this assessment are presented in **Table B2**:

$$\text{Daily Chemical Intake}_{\text{Ingestion}} = C_M \bullet \frac{IR_M \bullet FI \bullet B \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

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 B2**:

$$\text{Daily Chemical Intake}_{\text{Dermal}} = C_M \bullet \frac{SA \bullet AF \bullet ABSd \bullet CF \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

Table B2: 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)	100 mg/day	50 mg/day	Ingestion rate of outdoor soil and dust (tracked or deposited indoors) as per enHealth (enHealth 2012b)
	Fruit and vegetables (kg/day)	0.28 kg/day 85% from aboveground crops 16% from root crops	0.4 kg/day 73% from aboveground crops 27% from root crops	Total fruit and vegetable intakes per day as per ASC NEPM (NEPC 1999 amended 2013d)
	Eggs (kg/day)	0.006 kg/day	0.014 kg/day	Ingestion rate of eggs per day as per enHealth (enHealth 2012b)

Parameter		Value adopted		Basis
		Young children	Adults	
	Milk (L/day)	1.097	1.295	Ingestion rate consistent with P90 intakes from FSANZ (FSANZ 2017b)
	Beef (kg/day)	0.085	0.16	Ingestion rate consistent with P90 intakes from FSANZ (FSANZ 2017b)
	Lamb (kg/day)	0.036	0.085	Ingestion rate consistent with P90 intakes from FSANZ (FSANZ 2017b)
FI	Fraction of media ingested derived from impacted media, or fraction of produce consumed each day derived from the property			
	Soil	100%	100%	Assume all soil contact occurs on the one property
	Fruit and vegetables	35%	35%	Default of 35% for rural areas (NEPC 1999 amended 2013d)
	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 (not 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)	2700	6300	Exposed skin surface area relevant to adults as per ASC NEPM (NEPC 1999 amended 2013d)
AF	Adherence factor, amount of soil that adheres to the skin per unit area which depends on soil properties and area of body (mg/cm^2 per event)	0.5	0.5	Default (conservative) value from ASC NEPM (NEPC 1999 amended 2013d)
ABSd	Dermal absorption fraction (unitless)	Chemical specific		Refer to **Appendix A**
CF	Conversion factor			
	Soil	$1x10^{-6}$ to convert mg to kg		Conversion of units relevant to soil ingestion and dermal contact
	Produce	1		No units conversion required for these calculations
BW	Body weight	15	70	As per enHealth (enHealth 2012b) and ASC NEPM (NEPC 1999 amended 2013d)
EF	Exposure frequency (days/year)	365	365	Assume residents exposed every day
ED	Exposure duration (years)	6	29	Duration of residency as per enHealth (enHealth 2012b) and split between young children and adults as per ASC NEPM (NEPC 1999 amended 2013d). The longer exposure time than the Project operation is assumed as once metals are deposited to ground they are assumed to remain unchanged
AT	Averaging time (days)	Threshold = ED x 365 days/year Non-threshold = 70 years x 365 days/year		As per enHealth (enHealth 2012a) guidance

B3.2 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 B3**.

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

Table B3: 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)
t	Accumulation time (years)	14 years	14 years	Project duration
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 B4**:

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

The potential uptake of persistent chemical substances into edible crops via the roots can be estimated using the following equation (OEHHA 2015; USEPA 2005c), with the parameters and assumptions adopted outlined in **Table B4:**

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

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 B4: 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 B3**
RUF for root crops	Root uptake factor (unitless)	Chemical specific value adopted	Root uptake factors from 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 silver).

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 B5**:

$$C_P = (FI \times IR_C \times C + IR_S \times C_s \times B) \times TF_P$$

Table B5: 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 B4**
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 B3**
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) where available. Other values are the 95% value for the transfer of heavy metals into eggs (Leeman, Van Den Berg & Houben 2007).
	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).

Parameter		Value adopted	Basis
	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 C**.

Rainwater tanks

The concentration in rainwater tanks depends on the deposition rate of dust, the size of the roof, the volume of rainfall each year and how much of the rain that falls onto the roof is captured in the tank. 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.

In addition, health authorities[11] recommends the use of first flush devices 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 NSW Health and enHealth guidance (enHealth 2010), 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.

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 B6**:

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

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

[11] https://www.health.nsw.gov.au/environment/water/Documents/rainwater_tanks.pdf

Table B6: 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	
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)	200	Based on the average roof size for a 4 bedroom house in Australia (refer to Footnote 1)
VR	Volume of water collected from the roof each year (L)	calculated	Equation as above
R	Rainfall each year (mm)	390	Average rainfall at from Cobar MO for all years (1962 to 2021) (BoM 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).
ρ	Soil bulk density (g/cm^3)	0.5	Assumed for loose deposited dust on roof (upper end measured for powders)

1 - https://www.nedlands.wa.gov.au/sites/default/files/Rainwater%20tank%20factsheet.pdf

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

Appendix C Risk calculations

Inhalation - gases and particulates

$$InhalationExposureConc_V = C_a \bullet \frac{ET \bullet FI \bullet EF \bullet ED}{AT}$$ (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 (enHealth 2012)
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 (NEPM 1999 amended 2013) - assumed dust is PM10 and 100% reaches the lungs
Exposure Frequency - normal conditions (EF, days/yr)	240	Number of workdays per year as per enHealth (2012)
Exposure Duration (ED, years)	14	Duration of Project
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	122640	US EPA 2009

Maximum anywhere

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk (mg/m^3)$^{-1}$	Chronic TC Air (mg/m^3)	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background) (mg/m^3)	Estimated Concentration in Air - Maximum anywhere (Ca) (mg/m^3)	Inhalation Exposure Concentration - NonThreshold (mg/m^3)	Inhalation Exposure Concentration - Threshold (mg/m^3)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Silver	0.0E+00	2.0E-02	0%	2.0E-02	1.8E-08	7.7E-10	3.9E-09	--		1.9E-07	0%
Arsenic	0.0E+00	6.7E-05	50%	3.4E-05	5.1E-06	2.2E-07	1.1E-06	--		3.3E-02	68%
Cadmium	0.0E+00	5.0E-06	60%	2.0E-06	1.2E-07	5.2E-09	2.6E-08	--		1.3E-02	27%
Cobalt	0.0E+00	1.0E-04	20%	8.0E-05	3.8E-07	1.7E-08	8.3E-08	--		1.0E-03	2%
Copper	0.0E+00	4.9E-01	60%	2.0E-01	6.2E-06	2.7E-07	1.4E-06	--		6.9E-06	0%
Mercury	0.0E+00	2.0E-04	40%	1.2E-04	2.9E-10	1.3E-11	6.4E-11	--		5.3E-07	0%
Lead	0.0E+00	7.0E-03	50%	3.5E-03	2.1E-05	9.2E-07	4.6E-06	--		1.3E-03	3%
Zinc	0.0E+00	1.8E+00	80%	3.5E-01	3.1E-05	1.4E-06	6.9E-06	--		2.0E-05	0%
							TOTAL	0.0E+00		4.9E-02	

Inhalation - gases and particulates

$$InhalationExposureConc_V = C_a \bullet \frac{ET \bullet FI \bullet EF \bullet ED}{AT}$$ (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 (NEPM 1999 amended 2013) - assumed dust is PM10 and 100% reaches the lungs
Exposure Frequency - normal conditions (EF, days/yr)	365	Days at home, as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	14	Duration of Project
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	122640	US EPA 2009

Maximum for residential receptors

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk (mg/m^3)$^{-1}$	Chronic TC Air (mg/m^3)	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background) (mg/m^3)	Estimated Concentration in Air - Maximum sensitive receptors (Ca) (mg/m^3)	Inhalation Exposure Concentration - NonThreshold (mg/m^3)	Inhalation Exposure Concentration - Threshold (mg/m^3)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Silver	0.0E+00	2.0E-02	0%	2.0E-02	1.6E-09	3.1E-10	1.6E-09	--		7.8E-08	0%
Arsenic	0.0E+00	6.7E-05	50%	3.4E-05	4.5E-07	9.0E-08	4.5E-07	--		1.3E-02	68%
Cadmium	0.0E+00	5.0E-06	60%	2.0E-06	1.0E-08	2.1E-09	1.0E-08	--		5.2E-03	27%
Cobalt	0.0E+00	1.0E-04	20%	8.0E-05	3.3E-08	6.7E-09	3.3E-08	--		4.2E-04	2%
Copper	0.0E+00	4.9E-01	60%	2.0E-01	5.5E-07	1.1E-07	5.5E-07	--		2.8E-06	0%
Mercury	0.0E+00	2.0E-04	40%	1.2E-04	2.6E-11	5.1E-12	2.6E-11	--		2.1E-07	0%
Lead	0.0E+00	7.0E-03	50%	3.5E-03	1.8E-06	3.7E-07	1.8E-06	--		5.3E-04	3%
Zinc	0.0E+00	1.8E+00	80%	3.5E-01	2.8E-06	5.5E-07	2.8E-06	--		7.9E-06	0%
							TOTAL	0.0E+00		2.0E-02	

Calculation of Concentrations in Soil

$$C_s = \frac{DR \bullet \left[1 - e^{-k \bullet t}\right]}{d \bullet \rho \bullet k} \bullet 1000$$ **(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	14	14	Duration of operation

Chemical-specific Inputs and calculations - maximum residential 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
Silver	273973	2.5E-06	2.6E-03	2.3E-03	1.5E-04
Arsenic	273973	2.5E-06	7.5E-01	6.5E-01	4.4E-02
Cadmium	273973	2.5E-06	1.7E-02	1.5E-02	1.0E-03
Cobalt	273973	2.5E-06	5.5E-02	4.9E-02	3.2E-03
Copper	273973	2.5E-06	5.5E-02	4.9E-02	3.2E-03
Mercury	273973	2.5E-06	5.5E-02	4.9E-02	3.2E-03
Lead	273973	2.5E-06	5.5E-02	4.9E-02	3.2E-03
Zinc	273973	2.5E-06	5.5E-02	4.9E-02	3.2E-03

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)	50	As per NEPM 2013
Fraction Ingested from Source (FI, unitless)	100%	All of daily soil intake occurs from site
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

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
Silver		5.7E-03		5.7E-03	100%	2.3E-03	6.7E-10	1.6E-09	--		2.8E-07	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	6.5E-01	1.9E-07	4.7E-07	--		4.7E-04	70%
Cadmium		8.0E-04	60%	3.2E-04	100%	1.5E-02	4.5E-09	1.1E-08	--		3.4E-05	5%
Cobalt		1.4E-03	20%	1.1E-03	100%	4.9E-02	1.4E-08	3.5E-08	--		3.1E-05	5%
Copper		1.4E-01	60%	5.6E-02	100%	4.9E-02	1.4E-08	3.5E-08	--		6.2E-07	0%
Mercury		6.0E-04	40%	3.6E-04	100%	4.9E-02	1.4E-08	3.5E-08	--		9.6E-05	15%
Lead		2.0E-03	50%	1.0E-03	100%	4.9E-02	1.4E-08	3.5E-08	--		3.5E-05	5%
Zinc		5.0E-01	80%	1.0E-01	100%	4.9E-02	1.4E-08	3.5E-08	--		3.5E-07	0%
								TOTAL			**6.6E-4**	

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)	100	Assumed daily soil ingestion rate for young children, enHealth (2012)
Fraction Ingested from Source (FI, unitless)	100%	All of daily soil intake occurs from site
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

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
Silver		5.7E-03		5.7E-03	100%	2.3E-03	1.3E-09	1.5E-08	--		2.6E-06	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	6.5E-01	3.7E-07	4.4E-06	--		4.4E-03	70%
Cadmium		8.0E-04	60%	3.2E-04	100%	1.5E-02	8.7E-09	1.0E-07	--		3.2E-04	5%
Cobalt		1.4E-03	20%	1.1E-03	100%	4.9E-02	2.8E-08	3.2E-07	--		2.9E-04	5%
Copper		1.4E-01	60%	5.6E-02	100%	4.9E-02	2.8E-08	3.2E-07	--		5.8E-06	0%
Mercury		6.0E-04	40%	3.6E-04	100%	4.9E-02	2.8E-08	3.2E-07	--		9.0E-04	15%
Lead		2.0E-03	50%	1.0E-03	100%	4.9E-02	2.8E-08	3.2E-07	--		3.2E-04	5%
Zinc		5.0E-01	80%	1.0E-01	100%	4.9E-02	2.8E-08	3.2E-07	--		3.2E-06	0%
								TOTAL			**6.2E-3**	

Dermal Exposure to Chemicals via Contact with Soil

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

Parameters Relevant to Quantification of Exposure by Adults		
Surface Area (SAs, cm^2)	6300	Exposed skin surface area for adults as per NEPM (2013)
Adherence Factor (AF, mg/cm^2)	0.5	Default as per NEPM (2013)
Fraction of Day Exposed	1	Assume skin is washed after 24 hours
Conversion Factor (CF)	1.E-06	Conversion of units
Dermal absorption (ABS, unitless)	Chemical-specific (as below)	
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum from sensitive receptors

Key Chemical	Toxicity Data					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)	Dermal Absorption (ABS)	(mg/kg)	Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Silver		5.7E-03		5.7E-03		2.3E-03			--		--	
Arsenic		2.0E-03	50%	1.0E-03	0.03	6.5E-01	3.7E-07	8.8E-07	--		8.8E-4	
Cadmium		8.0E-04	60%	3.2E-04	0.001	1.5E-02	2.8E-10	6.8E-10	--		2.1E-06	
Cobalt		1.4E-03	20%	1.1E-03		4.9E-02			--		--	
Copper		1.4E-01	60%	5.6E-02		4.9E-02			--		--	
Mercury		6.0E-04	40%	3.6E-04		4.9E-02			--		--	
Lead		2.0E-03	50%	1.0E-03		4.9E-02			--		--	
Zinc		5.0E-01	80%	1.0E-01		4.9E-02			--		--	

TOTAL		**8.8E-04**

Dermal Exposure to Chemicals via Contact with Soil

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

Parameters Relevant to Quantification of Exposure by Young Children		
Surface Area (SAs, cm^2)	2700	Exposed skin surface area for young children as per NEPM (2013)
Adherence Factor (AF, mg/cm^2)	0.5	Default as per NEPM (2013)
Fraction of Day Exposed	1	Assume skin is washed after 24 hours
Conversion Factor (CF)	1.E-06	Conversion of units
Dermal absorption (ABS, unitless)	Chemical-specific (as below)	
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum from sensitive receptors

Key Chemical	Toxicity Data					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)	Dermal Absorption (ABS)	(mg/kg)	Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Silver		5.7E-03		5.7E-03		2.3E-03			--		--	
Arsenic		2.0E-03	50%	1.0E-03	0.03	6.5E-01	1.5E-07	1.8E-06	--		1.8E-3	
Cadmium		8.0E-04	60%	3.2E-04	0.001	1.5E-02	1.2E-10	1.4E-09	--		4.3E-06	
Cobalt		1.4E-03	20%	1.1E-03		4.9E-02			--		--	
Copper		1.4E-01	60%	5.6E-02		4.9E-02			--		--	
Mercury		6.0E-04	40%	3.6E-04		4.9E-02			--		--	
Lead		2.0E-03	50%	1.0E-03		4.9E-02			--		--	
Zinc		5.0E-01	80%	1.0E-01		4.9E-02			--		--	

TOTAL		**1.8E-03**

Calculation of Concentrations in Plants

ref: Stevens B. (1991)

Uptake Due to Deposition in Aboveground Crops	Uptake via Roots from Soil
$C_p = \frac{DR \bullet F \bullet [1 - e^{-k \bullet t}]}{Y \bullet k}$ (mg/kg plant – wet weight)	$C_{rp} = C_s \bullet RUF$ (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)	**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
Silver	14	0.05	7.1E-06	3.5E-06	0.1	1.5E-04	1.5E-05	0.1	1.5E-05
Arsenic	14	0.05	2.0E-03	1.0E-03	0.01	4.4E-02	4.4E-04	0.026	1.1E-03
Cadmium	14	0.05	4.8E-05	2.4E-05	0.125	1.0E-03	1.3E-04	0.36	3.6E-04
Cobalt	14	0.05	1.5E-04	7.6E-05	0.005	3.2E-03	1.6E-05	0.0037	1.2E-05
Copper	14	0.05	1.5E-04	7.6E-05	0.1	3.2E-03	3.2E-04	0.25	8.1E-04
Mercury	14	0.05	1.5E-04	7.6E-05	0.225	3.2E-03	7.3E-04	0.0854	2.8E-04
Lead	14	0.05	1.5E-04	7.6E-05	0.011	3.2E-03	3.6E-05	0.0047	1.5E-05
Zinc	14	0.05	1.5E-04	7.6E-05	0.0113	3.2E-03	3.7E-05	0.1	3.2E-04

(A) Root uptake factors from RAIS (soil to wet weight of plant)
(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 silver)

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)	0.4	Total fruit and vegetable consumption rate for adults as per NEPM (2013)
Proportion of total intake from aboveground crops (%A	73%	Proportions as per NEPM (2013)
Proportion of total intake from root crops (%R)	27%	Proportions as per NEPM (2013)
Fraction ingested that is homegrown (%)	10%	Relevant to urban areas as per NEPM (2013)
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	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)	(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Silver		5.7E-03		5.7E-03	100%	3.5E-06	1.5E-05	1.6E-09	3.8E-09	--		6.7E-07	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	1.0E-03	4.4E-04	2.0E-07	4.9E-07	--		4.9E-04	47%
Cadmium		8.0E-04	60%	3.2E-04	100%	2.4E-05	1.3E-04	1.2E-08	2.9E-08	--		9.2E-05	9%
Cobalt		1.4E-03	20%	1.1E-03	100%	7.6E-05	1.6E-05	1.4E-08	3.4E-08	--		3.0E-05	3%
Copper		1.4E-01	60%	5.6E-02	100%	7.6E-05	3.2E-04	3.4E-08	8.2E-08	--		1.5E-06	0%
Mercury		6.0E-04	40%	3.6E-04	100%	7.6E-05	7.3E-04	6.0E-08	1.4E-07	--		4.0E-04	38%
Lead		2.0E-03	50%	1.0E-03	100%	7.6E-05	3.6E-05	1.5E-08	3.7E-08	--		3.7E-05	4%
Zinc		5.0E-01	80%	1.0E-01	100%	7.6E-05	3.7E-05	1.5E-08	3.7E-08	--		3.7E-07	0%
									TOTAL			1.1E-03	

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables

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

Scenario 2

Parameters Relevant to Quantification of Exposure by Young children		
Ingestion Rate of Produce (IRp) (kg/day)	0.28	Total fruit and vegetable consumption rate for children as per NEPM (2013)
Proportion of total intake from aboveground crops (%A	84%	Proportions as per NEPM (2013)
Proportion of total intake from root crops (%R)	16%	Proportions as per NEPM (2013)
Fraction ingested that is homegrown (%)	10%	Relevant to urban areas as per NEPM (2013)
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration (mg/kg wet weight)	Root crops concentrations (mg/kg wet weight)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)				NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Silver		5.7E-03		5.7E-03	100%	3.5E-06	1.5E-05	8.6E-10	1.0E-08	--		1.8E-06	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	1.0E-03	4.4E-04	1.5E-07	1.7E-06	--		1.7E-03	55%
Cadmium		8.0E-04	60%	3.2E-04	100%	2.4E-05	1.3E-04	6.4E-09	7.5E-08	--		2.3E-04	7%
Cobalt		1.4E-03	20%	1.1E-03	100%	7.6E-05	1.6E-05	1.1E-08	1.2E-07	--		1.1E-04	4%
Copper		1.4E-01	60%	5.6E-02	100%	7.6E-05	3.2E-04	1.8E-08	2.2E-07	--		3.8E-06	0%
Mercury		6.0E-04	40%	3.6E-04	100%	7.6E-05	7.3E-04	2.9E-08	3.4E-07	--		9.3E-04	30%
Lead		2.0E-03	50%	1.0E-03	100%	7.6E-05	3.6E-05	1.1E-08	1.3E-07	--		1.3E-04	4%
Zinc		5.0E-01	80%	1.0E-01	100%	7.6E-05	3.7E-05	1.1E-08	1.3E-07	--		1.3E-06	0%
									TOTAL			3.1E-03	

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	
Silver	3.5E-06	1.5E-04	1.7E-01	3.3E-07	95% from Leeman et al (2007)
Arsenic	1.0E-03	4.4E-02	7.0E-02	3.9E-05	
Cadmium	2.4E-05	1.0E-03	1.0E-02	1.3E-07	
Cobalt	7.6E-05	3.2E-03	1.7E-01	7.0E-06	95% from Leeman et al (2007)
Copper	7.6E-05	3.2E-03	1.7E-01	7.0E-06	95% from Leeman et al (2007)
Mercury	7.6E-05	3.2E-03	8.0E-01	3.3E-05	
Lead	7.6E-05	3.2E-03	4.0E-02	1.7E-06	
Zinc	7.6E-05	3.2E-03	1.7E-01	7.0E-06	95% from Leeman et al (2007)

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)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

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
Silver		5.7E-03		5.7E-03	100%	3.3E-07	2.7E-11	6.6E-11	--		1.2E-08	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	3.9E-05	3.2E-09	7.8E-09	--		7.8E-06	28%
Cadmium		8.0E-04	60%	3.2E-04	100%	1.3E-07	1.1E-11	2.6E-11	--		8.1E-08	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	7.0E-06	5.8E-10	1.4E-09	--		1.3E-06	5%
Copper		1.4E-01	60%	5.6E-02	100%	7.0E-06	5.8E-10	1.4E-09	--		2.5E-08	0%
Mercury		6.0E-04	40%	3.6E-04	100%	3.3E-05	2.7E-09	6.6E-09	--		1.8E-05	66%
Lead		2.0E-03	50%	1.0E-03	100%	1.7E-06	1.4E-10	3.3E-10	--		3.3E-07	1%
Zinc		5.0E-01	80%	1.0E-01	100%	7.0E-06	5.8E-10	1.4E-09	--		1.4E-08	0%
								TOTAL			2.8E-05	

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)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

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
Silver		5.7E-03		5.7E-03	100%	3.3E-07	1.1E-11	1.3E-10	--		2.3E-08	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	3.9E-05	1.3E-09	1.6E-08	--		1.6E-05	28%
Cadmium		8.0E-04	60%	3.2E-04	100%	1.3E-07	4.5E-12	5.2E-11	--		1.6E-07	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	7.0E-06	2.4E-10	2.8E-09	--		2.5E-06	5%
Copper		1.4E-01	60%	5.6E-02	100%	7.0E-06	2.4E-10	2.8E-09	--		5.0E-08	0%
Mercury		6.0E-04	40%	3.6E-04	100%	3.3E-05	1.1E-09	1.3E-08	--		3.7E-05	66%
Lead		2.0E-03	50%	1.0E-03	100%	1.7E-06	5.7E-11	6.6E-10	--		6.6E-07	1%
Zinc		5.0E-01	80%	1.0E-01	100%	7.0E-06	2.4E-10	2.8E-09	--		2.8E-08	0%
									TOTAL		**5.6E-05**	

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	
Silver	3.5E-06	1.5E-04	3.0E-03	3.0E-07	RAIS
Arsenic	1.0E-03	4.4E-02	2.0E-03	5.8E-05	
Cadmium	2.4E-05	1.0E-03	2.0E-04	1.3E-07	
Cobalt	7.6E-05	3.2E-03	2.0E-02	4.3E-05	RAIS
Copper	7.6E-05	3.2E-03	1.0E-02	2.1E-05	RAIS
Mercury	7.6E-05	3.2E-03	4.0E-04	8.6E-07	
Lead	7.6E-05	3.2E-03	3.0E-04	6.4E-07	
Zinc	7.6E-05	3.2E-03	1.0E-01	2.1E-04	RAIS

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)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

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
Silver		5.7E-03		5.7E-03	100%	3.0E-07	9.9E-11	2.4E-10	--		4.2E-08	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	5.8E-05	1.9E-08	4.6E-08	--		4.6E-05	57%
Cadmium		8.0E-04	60%	3.2E-04	100%	1.3E-07	4.4E-11	1.1E-10	--		3.3E-07	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	4.3E-05	1.4E-08	3.4E-08	--		3.1E-05	38%
Copper		1.4E-01	60%	5.6E-02	100%	2.1E-05	7.1E-09	1.7E-08	--		3.1E-07	0%
Mercury		6.0E-04	40%	3.6E-04	100%	8.6E-07	2.8E-10	6.8E-10	--		1.9E-06	2%
Lead		2.0E-03	50%	1.0E-03	100%	6.4E-07	2.1E-10	5.1E-10	--		5.1E-07	1%
Zinc		5.0E-01	80%	1.0E-01	100%	2.1E-04	7.1E-08	1.7E-07	--		1.7E-06	2%
								TOTAL			**8.1E-05**	

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)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

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
Silver		5.7E-03		5.7E-03	100%	3.0E-07	5.1E-11	5.9E-10	--		1.0E-07	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	5.8E-05	9.8E-09	1.1E-07	--		1.1E-04	57%
Cadmium		8.0E-04	60%	3.2E-04	100%	1.3E-07	2.3E-11	2.7E-10	--		8.3E-07	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	4.3E-05	7.3E-09	8.5E-08	--		7.6E-05	38%
Copper		1.4E-01	60%	5.6E-02	100%	2.1E-05	3.6E-09	4.2E-08	--		7.6E-07	0%
Mercury		6.0E-04	40%	3.6E-04	100%	8.6E-07	1.5E-10	1.7E-09	--		4.7E-06	2%
Lead		2.0E-03	50%	1.0E-03	100%	6.4E-07	1.1E-10	1.3E-09	--		1.3E-06	1%
Zinc		5.0E-01	80%	1.0E-01	100%	2.1E-04	3.6E-08	4.2E-07	--		4.2E-06	2%
								TOTAL			2.0E-04	

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 ww	
Silver	3.5E-06	1.5E-04	5.0E-03	1.2E-06	Median transfer factor for metals (Leeman et al 2007)
Arsenic	1.0E-03	4.4E-02	5.0E-05	3.5E-06	
Cadmium	2.4E-05	1.0E-03	5.0E-06	8.2E-09	
Cobalt	7.6E-05	3.2E-03	2.0E-03	1.0E-05	RAIS
Copper	7.6E-05	3.2E-03	1.5E-03	7.8E-06	RAIS
Mercury	7.6E-05	3.2E-03	7.0E-05	3.7E-07	
Lead	7.6E-05	3.2E-03	6.0E-05	3.1E-07	
Zinc	7.6E-05	3.2E-03	2.7E-09	1.4E-11	RIAS

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Milk

$$\text{Daily chemical intake} = C_M \times \frac{IR_M \times FI \times ME \times EF \times ED}{BW \times AT} \qquad \text{(mg/kg/day)}$$

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)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration (mg/L)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor $(mg/kg\text{-}day)^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)			NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Silver		5.7E-03		5.7E-03	100%	1.2E-06	9.3E-09	2.3E-08	--		3.9E-06	1%
Arsenic		2.0E-03	50%	1.0E-03	100%	3.5E-06	2.7E-08	6.5E-08	--		6.5E-05	24%
Cadmium		8.0E-04	60%	3.2E-04	100%	8.2E-09	6.3E-11	1.5E-10	--		4.7E-07	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	1.0E-05	8.0E-08	1.9E-07	--		1.7E-04	64%
Copper		1.4E-01	60%	5.6E-02	100%	7.8E-06	6.0E-08	1.5E-07	--		2.6E-06	1%
Mercury		6.0E-04	40%	3.6E-04	100%	3.7E-07	2.8E-09	6.8E-09	--		1.9E-05	7%
Lead		2.0E-03	50%	1.0E-03	100%	3.1E-07	2.4E-09	5.8E-09	--		5.8E-06	2%
Zinc		5.0E-01	80%	1.0E-01	100%	1.4E-11	1.1E-13	2.6E-13	--		2.6E-12	0%
								TOTAL			**2.7E-04**	

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)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

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
Silver		5.7E-03		5.7E-03	100%	1.2E-06	7.6E-09	8.9E-08	--		1.6E-05	1%
Arsenic		2.0E-03	50%	1.0E-03	100%	3.5E-06	2.2E-08	2.6E-07	--		2.6E-04	24%
Cadmium		8.0E-04	60%	3.2E-04	100%	8.2E-09	5.1E-11	6.0E-10	--		1.9E-06	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	1.0E-05	6.6E-08	7.6E-07	--		6.8E-04	64%
Copper		1.4E-01	60%	5.6E-02	100%	7.8E-06	4.9E-08	5.7E-07	--		1.0E-05	1%
Mercury		6.0E-04	40%	3.6E-04	100%	3.7E-07	2.3E-09	2.7E-08	--		7.4E-05	7%
Lead		2.0E-03	50%	1.0E-03	100%	3.1E-07	2.0E-09	2.3E-08	--		2.3E-05	2%
Zinc		5.0E-01	80%	1.0E-01	100%	1.4E-11	8.8E-14	1.0E-12	--		1.0E-11	0%
								TOTAL			1.1E-03	

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	
Silver	3.5E-06	1.5E-04	3.1E-02	3.8E-07	MW adjustment
Arsenic	1.0E-03	4.4E-02	2.1E-02	7.4E-05	MW adjustment
Cadmium	2.4E-05	1.0E-03	2.1E-03	1.7E-07	MW adjustment
Cobalt	7.6E-05	3.2E-03	2.1E-01	5.5E-05	MW adjustment
Copper	7.6E-05	3.2E-03	1.0E-01	2.7E-05	MW adjustment
Mercury	7.6E-05	3.2E-03	4.2E-03	1.1E-06	MW adjustment
Lead	7.6E-05	3.2E-03	3.1E-03	8.2E-07	MW adjustment
Zinc	7.6E-05	3.2E-03	1.0E+00	2.7E-04	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)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

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
Silver		5.7E-03		5.7E-03	100%	3.8E-07	6.7E-11	1.6E-10	--		2.9E-08	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	7.4E-05	1.3E-08	3.1E-08	--		3.1E-05	57%
Cadmium		8.0E-04	60%	3.2E-04	100%	1.7E-07	3.0E-11	7.3E-11	--		2.3E-07	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	5.5E-05	9.7E-09	2.3E-08	--		2.1E-05	38%
Copper		1.4E-01	60%	5.6E-02	100%	2.7E-05	4.8E-09	1.2E-08	--		2.1E-07	0%
Mercury		6.0E-04	40%	3.6E-04	100%	1.1E-06	1.9E-10	4.7E-10	--		1.3E-06	2%
Lead		2.0E-03	50%	1.0E-03	100%	8.2E-07	1.4E-10	3.5E-10	--		3.5E-07	1%
Zinc		5.0E-01	80%	1.0E-01	100%	2.7E-04	4.8E-08	1.2E-07	--		1.2E-06	2%
								TOTAL			**5.5E-05**	

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)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

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
Silver		5.7E-03		5.7E-03	100%	3.8E-07	2.8E-11	3.2E-10	--		5.6E-08	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	7.4E-05	5.3E-09	6.2E-08	--		6.2E-05	57%
Cadmium		8.0E-04	60%	3.2E-04	100%	1.7E-07	1.2E-11	1.4E-10	--		4.5E-07	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	5.5E-05	3.9E-09	4.6E-08	--		4.1E-05	38%
Copper		1.4E-01	60%	5.6E-02	100%	2.7E-05	2.0E-09	2.3E-08	--		4.1E-07	0%
Mercury		6.0E-04	40%	3.6E-04	100%	1.1E-06	7.9E-11	9.2E-10	--		2.6E-06	2%
Lead		2.0E-03	50%	1.0E-03	100%	8.2E-07	5.9E-11	6.9E-10	--		6.9E-07	1%
Zinc		5.0E-01	80%	1.0E-01	100%	2.7E-04	2.0E-08	2.3E-07	--		2.3E-06	2%
								TOTAL			1.1E-04	

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

General Parameters			
Average rainfall (R)	mm	390	mean for all years (1962 to 2021) for Cobar MO (BoM data)
Roof area (Area)	m^2	200	4 bedroom australian home
Runoff coefficient (Rc)	-	0.7	assumes 30% loss in capture into tank
Volume of rainwater (VR)	L	54600	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 residences

Chemical	Deposited dust entering tank: Deposition Rate TSP (DR)	Deposited dust entering tank: Mass deposited each year into tank (DM)	Kd	Particulate Concentration in water	Dissolved Concentration in water	Total (particulate and dissolved) - worst-case
	mg/m^2/year	mg	(cm^3/g)	mg/L	mg/L	mg/L
Silver	2.6E-03	5.16E-02	8.3	9.5E-07	2.3E-07	1.2E-06
Arsenic	7.5E-01	1.49E+01	29	2.7E-04	1.9E-05	2.9E-04
Cadmium	1.7E-02	3.47E-01	75	6.4E-06	1.7E-07	6.5E-06
Cobalt	5.5E-02	1.11E+00	45	2.0E-05	9.0E-07	2.1E-05
Copper	5.5E-02	1.11E+00	35	2.0E-05	1.2E-06	2.1E-05
Mercury	5.5E-02	1.11E+00	52	2.0E-05	7.8E-07	2.1E-05
Lead	5.5E-02	1.11E+00	900	2.0E-05	4.5E-08	2.0E-05
Zinc	5.5E-02	1.11E+00	62	2.0E-05	6.6E-07	2.1E-05