Appendix 7

Human Health Risk Assessment - Updated

prepared by

Environmental Risk Sciences Pty Ltd

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BOWDENS SILVER

Part 7

Human Health Risk Assessment – Updated

State Significant Development No. 5765

Prepared by:

Environment Risk Sciences Pty Ltd (enRiskS)

June 2021

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BOWDENS SILVER

ABN: 37 009 250 051

Human Health Risk Assessment – Updated

Prepared for: R.W. Corkery & Co. Pty Limited
1st Floor, 12 Dangar Road
PO Box 239
BROOKLYN NSW 2083

Tel: (02) 9985 8511
Email: brooklyn@rwcorkery.com

On behalf of: Bowdens Silver Pty Limited
ABN: 37 009 250 051

Sydney Office
Level 11, 52 Phillip Street
SYDNEY NSW 2000

Tel: (02) 8316 3997
Fax: (02) 8316 3999

Operations Office
68 Maloneys Road
LUE NSW 2850
P.O. Box 1115
MUDGEE NSW 2850

Tel: (02) 6373 6420

Email: information@bowdenssilver.com.au

A Silver Mines Limited company

Prepared by: Environmental Risk Sciences Pty Ltd
PO Box 2537
CARLINGFORD COURT NSW 2118

Tel: (02) 9614 0297
Email: jackie@enrisks.com.au

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EnRiskS

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TABLES

COMMONLY USED ACRONYMS

ABS	Australian Bureau of Statistics
AEP	Annual exceedance probability
ANZECC	Australia and New Zealand Environment and Conservation Council.
ANZG	Australian and New Zealand Guidelines
CNMP	Construction Noise Management Plan
CNS	central nervous systems
DECCW	Department of Environment, Climate Change and Water.
DPE	Department of Planning and Environment
EIS	Environmental Impact Statement
enRiskS	Environmental Risk Sciences Pty Ltd
ENM	Environmental Noise Model
EPA	Environment Protection Authority.
EPL	environment protection licence
ESC	erosion and sediment control
FSANZ	Food Standards Australia New Zealand
HCN	hydrogen cyanide
HHRA	Human Health Risk Assessment.
ICNG	Interim Construction Noise Guideline
I-INCE	International Institute of Noise Control Engineers
IoPC	issues of potential concern
LGA	Local Government Area.
NAF	non-acid forming
NEPC	National Environment Protection Council.
NEPM	National Environment Protection Measure.
NHMRC	National Health and Medical Research Council.
NOAEL	No-observed-adverse-effect-level - The highest tested dose of a substance that has been reported to have no harmful (adverse) health effects on people or animals.

NPfI	Noise Policy for Industry
OEHHA	Office of Environmental Health Hazard Assessment, California Environment Protection Agency (Cal EPA).
PAC	Protective Action Criteria
PAF	Potentially acid forming
PM	Particulate matter.
PM_{10}	Particulate matter of aerodynamic diameter 10 µm and less
$PM_{2.5}$	Particulate matter of aerodynamic diameter 2.5 µm and less
PMP	Probable maximum precipitation
RI	Risk Index
RQ	Risk Quotients
RME	Reasonable Maximum Exposure
SBRC	Solubility Bioaccessibility Research Consortium
SEARs	Secretary's Environmental Assessment Requirements
SLR	SLR Consulting Australia Pty Ltd
SSC	state suburbs
TECQ	Texas Commission on Environmental Quality
TDI	Tolerable daily intake
TRV	Toxicity Reference Values
TSF	tailings storage facility
TSP	Total suspended particulate
USEPA	United States Environmental Protection Agency
VLAMP	Voluntary Land Acquisition Mitigation Policy
WAD	weakly acid dissociable
WHO	World Health Organization
WRE	waste rock emplacement
WRM	WRM Water and Environment Pty Ltd

COMMONLY USED TERMS

A weighted decibels (dB(A))	The A weighting is a frequency filter applied to measured noise levels to represent how the human ear hears sounds. The A-weighting filter emphasises frequencies in the speech range (between 1 kHz and 4 kHz) to which the human ear is most sensitive. When an overall sound level is A-weighted it is expressed in units of dB(A).
Acute or short-term exposure	Contact with a substance that occurs only once or for a short period of time, typically an hour or less, but may be 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.
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.
Chronic or long-term exposure	Contact with a substance that occurs over a long time (more than 1 year) [compare with acute exposure and intermediate duration exposure].
Co-exposure	Exposure to more than one pollutant or stressor (such as noise) by a population.
Conservative	A term used throughout the HHRA to describe where modelling, a parameter or assumption is a worst-case or reasonable worst-case. Using such information and data would result in an overestimation of potential impacts and risks to human health. As a result, the conclusions of the HHRA include an additional level of protection as all the conservative approaches and assumptions have been compounded throughout the assessment.
Cumulative	Total exposure, used in the health impact assessment to refer to exposures that include the background plus project, or to multiple different sources from the project.

Decibel (dB)	The measurement unit of sound. A 3 decibel increase or decrease is typically considered the smallest change in sound level that a listener can detect. A change of 5 dB is clearly noticeable. A 10 decibel increase is typically considered to sound twice as loud.
Dermal Contact	Contact with (touching) the skin (see route of exposure).
Detection limit	The lowest concentration of a chemical 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 milligrams (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 gets into the body through the eyes, skin, stomach, intestines, or lungs.
Exposure	Contact with a substance by swallowing, breathing, or touching the skin or eyes. Exposure may be short-term [acute exposure], of intermediate duration, or long-term [chronic exposure]. Exposure is the first part in the process where humans come into contact with, and subsequently absorb, chemicals in their environment.
Exposure assessment	The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.
Exposure pathway	The route a substance takes from its source (where it began) to its end point (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 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 receiver population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.
Guideline value	A 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 Organisation (WHO)). The guideline value 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, or the environment. Dependent on the source, guidelines will have different names, such as investigation level, trigger value, ambient guideline etc.

Health protection	The adoption of approaches and/or health-based guidelines that are protective of the health of all members of the community, including infants, the elderly and sensitive individuals.
Inhalation	The act of breathing. A hazardous substance can enter the body this way [see route of exposure].
Intermediate exposure duration	Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].
Intake	The ingestion or inhalation of a chemical following exposure.
L_{10}	The sound pressure level exceeded for 10% of the measurement period. The A-weighted form is denoted 'LA10'.
$LA_{10(18h)}$	The LA10(18-hour) noise level refers to the noise level exceeded for 10 per cent of the time during an 18-hour period (from 6am to midnight). This noise descriptor is calculated using the arithmetic average of the LA10 noise levels for each hour from 6am to midnight.
L_{den}	The average noise level over the day, evening and night (i.e. a 24-hour period).
L_{eq}	Equivalent continuous sound level. The constant sound level which, when occurring over the same period of time, would result in the receptor experiencing the same amount of sound energy. The A-weighted form is denoted 'L_{Aeq}'.
L_{night}	The average noise level over the night-time period, typically between 11:00pm or midnight and 6:00am.
L_{OAEL}	Lowest-observed-adverse-effect-level – The lowest tested dose of a substance that has been reported to cause harmful (adverse) health effects in people or animals.
Metabolism	The conversion or breakdown of a substance from one form to another by a living organism.
Morbidity	A diseased condition or state or the incidence or prevalence of disease in a population.
Mortality	Death, which may occur as a result of a range of reasons or diseases.
Not measurable	The term "no measurable" or "not measurable" is used in this health impact assessment when referring to changes in air quality, noise or health outcomes in a population. For air quality and noise, a change that would not be measurable is one where the estimated change in the concentration of the pollutant in ambient air, or noise, is so small that it could not be measured - i.e. within the error of the analytical method/measurement equipment. For health outcomes, it refers to exposures that are below a threshold so there are no health effects, or to changes in the number of people that may be affected (i.e. increase or decrease in deaths or hospitalisations) that is within the error/variability of the statistical measures (i.e. is not measurable).

Point of exposure	The place where someone comes 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).
Receiver population	People who could come into contact with hazardous substances [see exposure pathway].
Risk	The probability that something will cause injury or harm.
Route of exposure	The way people come into contact with a hazardous substance. The three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].
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 for relevant exposure pathway (inhalation, oral or dermal), with special emphasis on dose-response characteristics. The data is based on available toxicity studies relevant to humans and/or animals and relevant safety factors.
Toxicological profile	An assessment that examines, summarizes, 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.
Uncertainty factor	Mathematical adjustments for reasons of safety when knowledge is incomplete. For example, factors used in the calculation of doses that are not harmful (adverse) to people. These factors are applied to the lowest-observed-adverse-effect-level (LOAEL) or the no-observed-adverse-effect-level (NOAEL) to derive a minimal risk level (MRL). Uncertainty factors are used to account for variations in people's sensitivity, for differences between animals and humans, and for differences between a LOAEL and a NOAEL. Scientists use uncertainty factors when they have some, but not all, the information from animal or human studies to decide whether an exposure may cause harm to people [also sometimes called a safety factor].

EXECUTIVE SUMMARY

Introduction

Bowdens Silver Pty Ltd (Bowdens Silver) plans to apply for a development consent under Part 4 of the *Environmental Planning and Assessment Act 1979* to develop and operate an open cut silver mine near Lue, NSW (the Project). The Project is classified as State Significant Development.

Located approximately 26km east of Mudgee and approximately 2km to 3km northeast of Lue, the proposed Mine Site is a greenfield site, however, a range of exploration activities have been undertaken. It is proposed that the Mine would have a capacity to extract and process up to approximately 2 Mtpa of ore and would have a mine life in the order of 16.5 years.

Environmental Risk Sciences Pty Ltd (enRiskS) has been commissioned to undertake a human health risk assessment (HHRA) to evaluate impacts of the proposed mine on human health. More specifically, the HHRA has addressed health impacts in the community surrounding the Mine Site related to air emissions, noise and changes in the quantity and quality of water (groundwater and surface water).

The assessment of health impacts related to the Project has relied on impact assessment completed for air quality (Ramboll, 2021), noise (SLR, 2020), surface water (WRM, 2020), and groundwater (Jacobs, 2020).

Community

The population surrounding the Mine Site is small and comprises a number of rural-residential properties along with residential properties and other key premises such as Lue Public School within Lue. The population demographics and health-related behaviours in the areas surrounding the Mine Site is generally similar to the population in the larger Mid-West Region and rural NSW areas. There are some smaller areas with higher rates of unemployment.

In relation to the health of the population in the local area, data from NSW Health indicates this area has a higher rate of smoking and a higher prevalence of obesity than the overall population of NSW. In addition, the area has a higher rate of cardiovascular and respiratory disease than the overall population of NSW, which may have some influence on the susceptibility of the population to environmental stressors.

Some members of the local community have expressed a number of concerns in relation to the Project, with most concerns relating to dust from the mining activities and the impact of this dust, which includes lead, on the health of all members of the community including sensitive groups such as children and those with existing health conditions. The assessment undertaken has addressed potential health impacts of lead in dust emissions, along with and range of other metals and pollutants.

Health Impacts of Air Emissions

The assessment of health impacts has focused on dust emissions from the Project, and more specifically the presence of lead and other metals on dust emissions from the Project.

As metals are ubiquitous in the environment and all members of the population are exposed to these metals in dust, soil, water and dietary sources, it is important that the assessment of impacts of dust emissions from the Project addresses existing exposures as well as exposures that may occur as a result of the Project (construction and operation). The HHRA addressed the following:

- Acute (or maximum short-term) inhalation exposures to metals present on dust emissions
- Chronic (or long-term) inhalation exposures to fine particulates (including silica) as well as lead and other metals bound to these dust particles
- Chronic (or long-term) exposures to lead and other metals as a result of dust generated from the Project, and then:
  - depositing onto the roof of homes/buildings and accumulating and affecting water quality in rainwater tanks used as drinking/household water (where water is ingested)
  - depositing onto soil and dust or other surfaces where the community may come into direct contact and incidentally ingest some of the dust
  - accumulation of lead and other metals into home-grown produce (fruit and vegetables, eggs, meat and milk) and consumption of this produce by the community

All these exposures have been evaluated at all privately-owned residences within 4km to 6km of the Mine Site as well as other key locations such as Lue Public School. Exposures to metals by all members of the public are dominated by existing exposures to metals in the environment. Dust emissions from the Project would make a negligible contribution to these exposures and there would be no Project-related exposures that are considered to result in any health impacts for any member of the community.

An Air Quality Management Plan would be developed prior to commencement of operations at the Project that would outline the measures to manage air emissions (consistent with those considered and outlined in the Air Quality Impact Assessment (Ramboll, 2021).

Health Impacts of Water

The assessment has not identified any impacts to groundwater or surface water that would have the potential to adversely affect community health. The quantity and quality of water that may be accessed by the community is not expected to be significantly affected by Project activities.

Health Impacts of Noise

The assessment of noise impacts from the Project has not identified noise levels within the community that exceed health-based WHO guidelines for the protection of health, during the day, evening or night.

Regardless of the above, a Construction Noise Management Plan, Blast Management Plan and Operational Noise Management Plan would be developed prior to commencement of the Project for managing and minimising noise and blasting impacts from the Project.

1. INTRODUCTION

1.1 BACKGROUND

Bowdens Silver Pty Ltd (Bowdens Silver) plans to apply for a development consent under Part 4 of the *Environmental Planning and Assessment Act 1979* to develop and operate an open cut silver mine near Lue, NSW (the Project). The Project is classified as State Significant Development.

The Mine Site is located approximately 26km east of Mudgee and approximately 2km to 3km northeast of Lue (refer to **Figures 1.1** and **1.2**).

The proposed Mine Site is a greenfield site, however, a range of exploration activities have been undertaken in the area of the proposed open cut pits. It is proposed that the Mine would have a capacity to extract and process up to approximately 2 Mtpa of ore (containing silver and small percentages of zinc and lead) and would have a mine life in the order of 16.5 years i.e. from the commencement of the site establishment and construction stage to the completion of concentrate production. It is envisaged final rehabilitation activities would be completed over a period of approximately 7 years resulting in a total Project life of approximately 23 years.

The proposed operations would involve a conventional open cut mine including an out-of-pit waste rock emplacement (WRE), tailings storage facility (TSF), processing plant (with concentrate storage), one or more water storage dams and other ancillary infrastructure. As part of the Project, a new road would be constructed to the Mine Site from a location to the west of Lue and a 500kV power line that traverses the proposed open cut area would be realigned. A pipeline is proposed to transport excess mine water from the Ulan Coal Mine and/or Moolarben Coal Mine to the proposed Mine Site to be used in ore processing.

As the Project is a State Significant development, an Environmental Impact Statement (EIS) is required to be prepared in accordance with the *Environmental Planning and Assessment Regulation 2000*. The Secretary of the Department of Planning and Environment (the Department) prescribed environmental impact assessment requirements (SEARs) dated June 2019. The SEARs are developed in consultation with a wide range of public authorities, including the Mid-Western Regional Council.

The SEARs require that an assessment of impacts to human health be evaluated within the EIS.

Environmental Risk Sciences Pty Ltd (enRiskS) has been commissioned to undertake a HHRA to evaluate impacts of the Project on human health.

It is noted that some members of the local community have expressed a particular concern in relation to the impact of the proposed mine on community exposures to lead. This report has included an assessment of community exposures to lead, as well as a range of other pollutants and issues that have the potential to impact on health.

Figure 1.1 Locality Plan

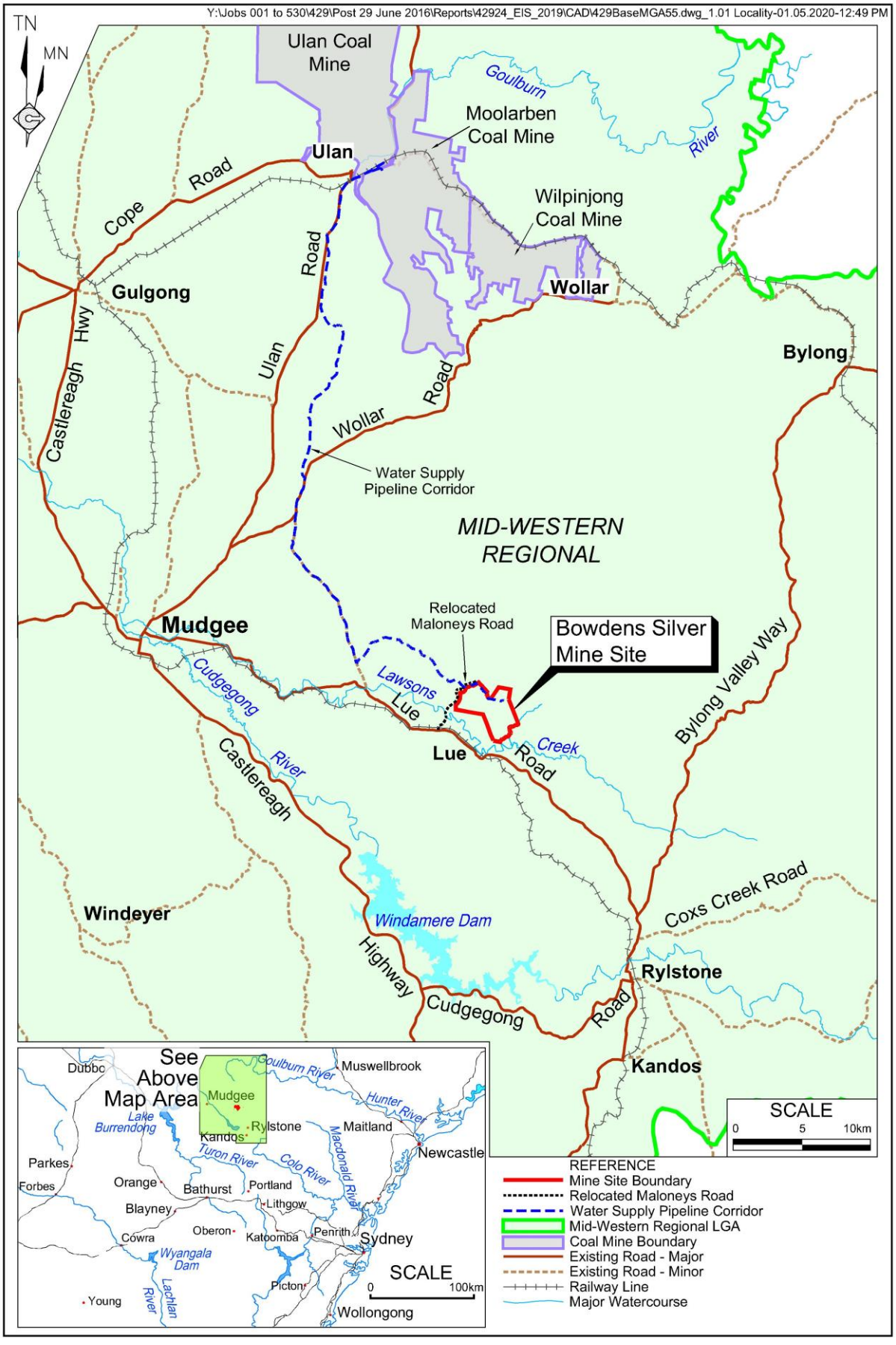
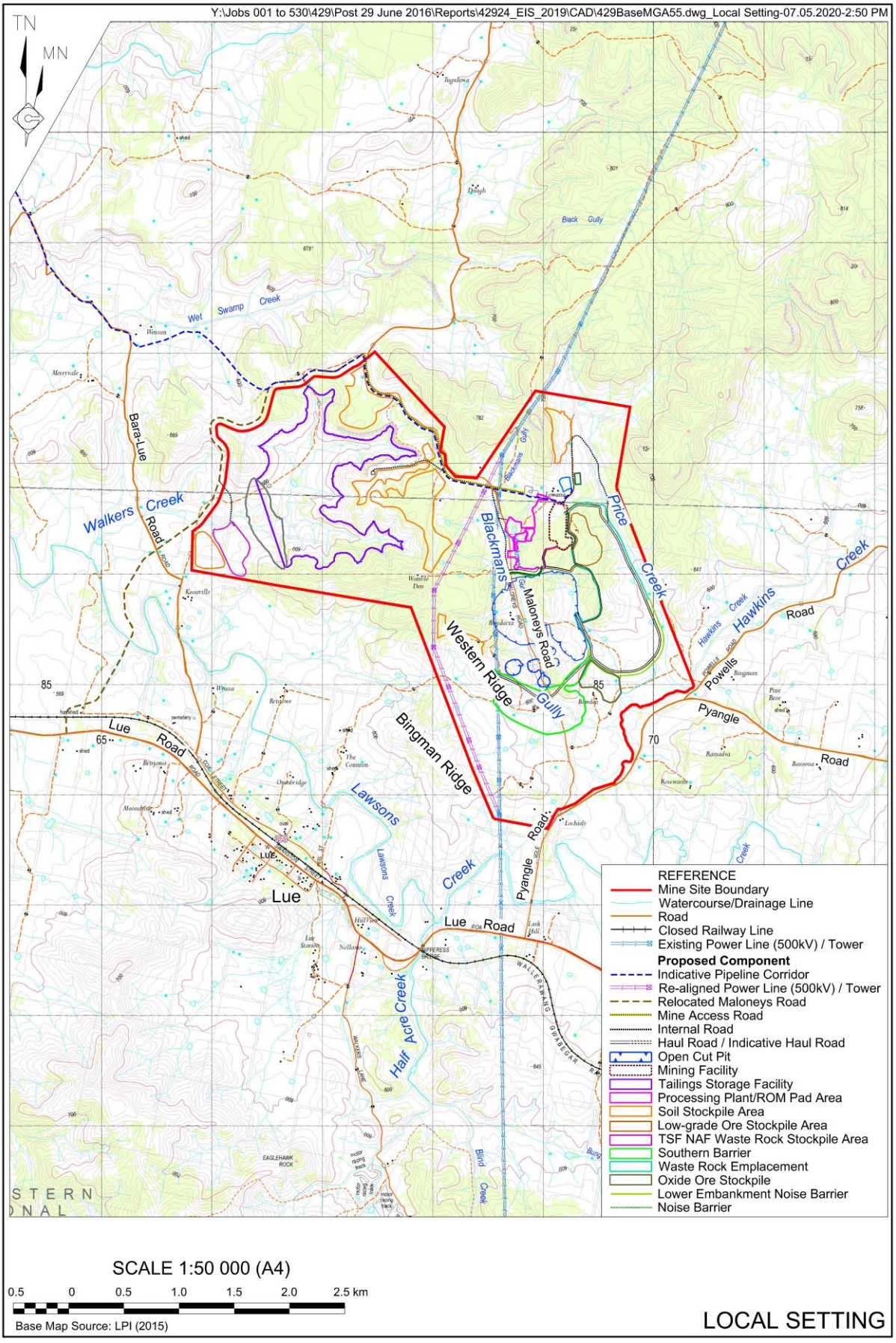


Figure 1.2 Local Setting Plan



1.2 OBJECTIVES

The overall objective of the HHRA presented in this report is to provide an assessment of the impacts of the Project on the health of the community. More specifically, the HHRA has been undertaken to address impacts to human health as outlined in the SEARs, which are:

- air quality (particulates from crustal materials and heavy metals, in particular);
- noise and vibration (including blasting); and
- drinking water quality (surface and/or groundwater).

Additional detail in relation to specific aspects of the above that address the SEARs, agency requirements and concerns raised by some members of the community are detailed in Section 1.6 and presented in this report.

1.3 APPROACH TO HUMAN HEALTH RISK ASSESSMENT

1.3.1 What is a Risk Assessment?

Risk

Risk assessment is used extensively in Australia and overseas to assist in decision making on the acceptability of the risks associated with the presence of contaminants in the environment and evaluation of projects with potential risks to the public. Risk is commonly defined as the chance of injury, damage, or loss. Therefore, to put oneself or the environment "at risk" means to participate either voluntarily or involuntarily in an activity or activities that could lead to injury, damage, or loss.

Voluntary risks are those associated with activities that people decide to undertake such as driving a car, riding a motorcycle and smoking cigarettes.

Involuntary risks are those associated with activities that may happen to us without our prior consent or forewarning. Acts of nature such as being struck by lightning, fires, floods, tornados, etc., and exposures to environmental contaminants are examples of involuntary risks.

Defining Risk

Risks to the public and the environment are determined by direct observation or by applying mathematical models and a series of assumptions to infer risk. No matter how risks are defined or quantified, they are usually expressed as a probability of adverse effects associated with a particular activity. Risk is typically expressed as a likelihood of occurrence and/or consequence (such as negligible, low or significant) or quantified as a fraction of, or relative to, an acceptable risk number.

Risks from specific projects, such as a mine, are usually assessed through qualitative or quantitative risk assessment techniques. In general, risk assessments seek to identify all relevant hazards; assess or quantify their likelihood of occurrence and consequences; and estimate risk levels for people who could be exposed, such as those beyond the perimeter boundary of the Mine Site.

1.4 METHODOLOGY AND SCOPE

The HHRA has been undertaken as a desk-top assessment. The term desk-top assessment is used to describe that the assessment has not involved the collection of any additional data over and above that provided by project-specific EIS technical studies, community consultation and statistics on the existing population. The assessment has been conducted using existing information with additional detail obtained via literature review.

The HHRA has been undertaken in accordance with the following national guidelines:

- enHealth Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012b);
- Health Impact Assessment: A practical guide (Harris 2007)
- Health Impact Assessment Guidelines, Commonwealth Department of Health and Aged Care (enHealth 2017)
- SEPP No. 33 - Hazardous and Offensive Development (NSW Government 2014)
- NEPC National Environment Protection (Ambient Air Quality) Measure (NEPC 2016)
- National Environmental Protection Measure – Assessment of Site Contamination including:
  - Schedule B1 Investigation Levels for Soil and Groundwater (NEPC 1999 amended 2013b);
  - Schedule B4 Guideline on Health Risk Assessment Methodology (NEPC 1999 amended 2013c);
  - Schedule B6 Guideline on Risk Based Assessment of Groundwater Contamination (NEPC 1999 amended 2013d);
  - Schedule B7 Guideline on Health-Based Investigation Levels (NEPC 1999 amended 2013e); and
  - Schedule B8 Guideline on Community Consultation and Risk Communication (NEPC 1999 amended 2013a).
- NSW Approved Methods for the Modelling and Assessment of Air Pollutants (NSW EPA 2016)
- NSW Noise Policy for Industry (NSW EPA 2017)
- NHMRC Australian Drinking Water Guidelines (NHMRC 2011 updated 2018)
- Australian and New Zealand Guidelines for Fresh and Marine Water Quality (ANZG 2018).

Where required, 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 Organisation (WHO), consistent with current industry best practice. These documents are referenced, where relevant, throughout this report.

The HHRA has been undertaken to address the following:

- Identify and outline the demographics, health, key health indicators and existing environment in which the local community resides. This provides information on the general health of the community, whether the community is particularly vulnerable to changes that may result in health impacts, and the nature and level of exposures that occur within the existing community. Many of the impacts evaluated in the HHRA relate to compounds or pollutants that are already present in the environment, and the community is exposed to on a daily basis from many sources.
- Identify and assess changes in exposures that may occur as a result of the proposed Project. Specifically, the HHRA has addressed changes in air quality, noise and vibration and water quality and how these affect the health of the community. Specific details relevant to the assessment of health risks associated with impacts of the Project on air quality, noise and water quality are addressed in the relevant sections.

The HHRA has not addressed occupational exposures during the construction or operation of the Project. Occupational health and safety aspects of the Project would be managed separately under current occupational health and safety regulations and guidelines as outlined and enforced by SafeWork NSW.

1.5 FRAMEWORK FOR HUMAN HEALTH RISK ASSESSMENT

The assessment of risks to human health is undertaken using the methodology and framework outlined by enHealth (enHealth 2012b) (see **Figure 1.3**), where the following four key tasks are undertaken:

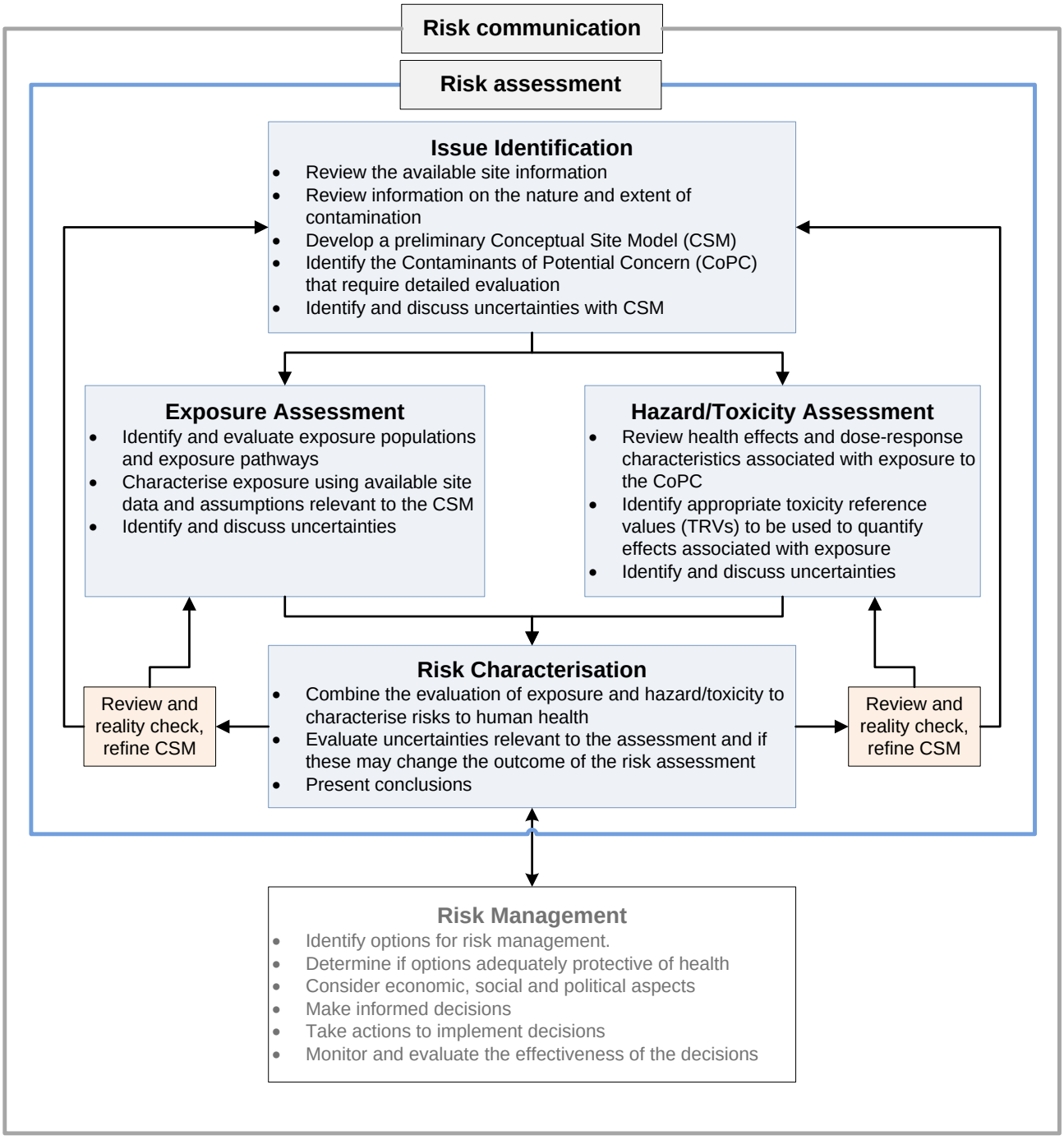
Task 1 – Data Review, Evaluation and Issue Identification (Problem Identification)

This task involves a review of the existing information on the community, existing environmental exposures, and all information available on the potential impact of the Project on air quality, noise and water quality. The review is undertaken to understand who is or may be exposed to Project-related impacts, and whether these impacts are significant enough to warrant a more detailed assessment of health impacts. The review has focused in on issues of potential concern (IoPC) (chemicals or stressors such as noise) that require detailed evaluation in the HHRA.

Task 2 – Toxicity/Hazard Assessment

This task involves understanding and identifying how community exposures to the IoPC can result in adverse health effects (i.e. how toxic are the chemicals or stressors), what these health effects are and how they can be quantified. The quantification of potential hazards or toxicity is undertaken using quantitative guidelines or toxicity reference values. In some cases this aspect is undertaken on a qualitative basis.

Figure 1.3 Health Risk Assessment Process



Task 3 – Exposure Assessment

Exposure to the identified IoPC is quantified based on who may be exposed (receptors) and how they may be exposed (pathways). The quantification of exposure considers how the community may be exposed via a wide range of pathways including inhalation, the ingestion and dermal contact with dust deposited on surface soil or as indoor dust, the accumulation of chemicals deposited onto surface soil into edible produce, the presence of chemicals (from the Project) in water used for recreational use (i.e. ingestion and dermal absorption during swimming), irrigation (using surface water or groundwater) or drinking water (including tank water).

Where noise sources are considered, the exposure aspect is used to identify changes in noise (and vibration) levels during the day time and night time at key areas such as privately-owned residences.

Task 4 – Risk Characterisation

The findings of Tasks 1 to 3 are then used to provide a quantitative assessment of human health risk. The health risk results are expressed as hazard quotients for key chemicals that are not genotoxic carcinogens, or where there are thresholds relevant to the assessment of impacts (such as noise guidelines). No genotoxic carcinogens are associated with this Project, hence there has been no assessment of potential risks to human health associated with exposure to these chemicals.

The characterisation of risk considers uncertainties identified in Tasks 1 to 3 when presenting conclusions and any recommendations.

In relation to the HHRA conducted for this project, the following provides an outline of the structure of the report and how the above tasks fit into the assessment of health impacts associated with air quality, noise and vibration and water quality:

1.6 SEARS, AGENCY AND COMMUNITY REQUIREMENTS AND CONCERNS

The HHRA has addressed the SEARs and other government agency requirements for the EIS that are relevant to human health. **Table 1.1** provides a summary of the relevant SEARs and agency requirements and where these are addressed in this report.

Table 1.1
SEARs and Agency Requirements Relevant to Human Health

Page 1 of 3

Relevant Requirement(s)	Coverage in Report
Secretary's Environmental Assessment Requirements	
The EIS must include an assessment of: • Human Health Risk, addressing how the development's environmental impacts in relation to air quality (including heavy metals) and noise may impact on the health of the local community; and	Air quality impacts addressed in Section 5 Noise impacts addressed in Section 7
• monitoring and management measures to reduce risk to human health.	Sections 5.6, 6.5 and 7.6, and summarised in Section 8

Table 1.1 (Cont'd)
SEARs and Agency Requirements Relevant to Human Health

Relevant Requirement(s)		Coverage in Report
Relevant Requirements Nominated by Other Government Agencies		
Health Western NSW – Local Health District Undated	The Proponent must assess the potential health impacts of the project, in accordance with current guidelines. The guidelines include, but are not limited to: • Environmental Health Risk Assessment, Guidelines for assessing human health risks from environmental hazards, Commonwealth of Australia (enHealth,2012) • Health Impact Assessment Guidelines, Commonwealth Department of Health and Aged Care (enHealth, 2001)	As noted in Section 1.4 (adopting the most current guidance) and incorporated throughout the HHRA report
	The assessment must: • assess health risks associated with exposure to environmental hazards;	Air quality impacts addressed in Section 5 Water quality impacts addressed in Section 6 Noise impacts addressed in Section 7
	• provide appropriate and proven management and monitoring measures to reduce any identified risk,	As referenced in Sections 5.6, 6.5 and 7.6 and as detailed in the Air Quality Assessment (Ramboll, 2021), Surface Water Assessment (WRM, 2020), Groundwater Assessment (Jacobs, 2020) and Noise and Vibration Assessment (SLR, 2020)
	• assess opportunities for health improvement; and	Refer to EIS Section 4.8.8
	discuss how, in the broader social and economic context of the project, the project will minimise negative health impacts while maximising the health benefits.	Refer to EIS Section 4.8.8
Greater Western Area Health Service 24/01/13	Lead is an issue as they will be processing it along with all other extracted material. Lead has already been found in the environment without mining contributing to levels. Dust control on site and during processing of materials should be of a level to prevent further contamination.	Section 5 and the Air Quality Assessment (Ramboll, 2021)
	Provide information on what may/will be used for dust suppression. If substances other than water are used they should be in the project plan with controls identified.	Section 6 and the Surface Water Assessment (WRM, 2020)

Table 1.1 (Cont'd)
SEARs and Agency Requirements Relevant to Human Health

Relevant Requirement(s)		Coverage in Report
Relevant Requirements Nominated by Other Government Agencies (Cont'd)		
Mid-Western Regional Council 14/02/13	Council requires the applicant to undertake a full assessment of the impacts on air quality from dust and particulate matter as a result of the Project including monitoring of background lead levels to ensure there are no adverse impacts on the Lue community and the surrounding area. Council requests that consideration be given to the findings in Port Augusta*[sic]* (Pirie) where unexpected high lead levels were found locally and at sites remote from the Mine Site *[sic]* (smelter).	Section 5 as relevant to the Project
Department of Education and Communities 13/02/13	Assess the potential impact of dust and dust toxicity on the school.	Section 5
Department of Education 03/08/17	The impacts on the quality of school's rooftop rainwater supply from mine pollutants and traffic fumes during construction and operation of the mine.	Section 5.2.6.4

1.7 LINKAGES TO OTHER TECHNICAL REPORTS

The HHRA has relied on assessments completed on other key aspects of the project. The assessment relied on the technical reports outlined in **Table 1.2**.

Table 1.2
Other Technical Reports Relied on for the HHRA

Assessment Aspect	Technical Report
Air quality	Ramboll (2021), Air Quality Assessment
Noise and vibration	SLR (2020), Noise and Vibration Assessment
Groundwater	Jacobs Group (Australia) Pty Ltd (2020). Bowdens Silver Project, Groundwater Impact Assessment
Surface water	WRM Water & Environment Pty Ltd (WRM) (2020). Bowdens Silver Project, Surface Water Assessment

1.8 CONSIDERATIONS WHEN EVALUATING HEALTH RISKS

There are certain features of a HHRA that are important to acknowledge. These relate to the limitations of the methodology and the constraints applied within the HHRA to ensure a focus on aspects that can be influenced as part of the Project. These are summarised below (also refer to Section 9 for discussion of uncertainties):

- The risk assessment does not present an evaluation of the health status of any specific individuals in the community. Rather, it is a logical process of calculating the potential daily exposure to chemicals and noise within a community associated the Project. This estimate is then compared to regulatory and published estimates of daily exposures or noise levels that a person may be exposed to over a lifetime without unacceptable risks to their health.

- A HHRA is a systematic tool used to review key aspects of a specific Project that may affect the health of the local community. The assessment includes both qualitative and quantitative assessment methods.
- Where quantitative assessment methods are presented, a HHRA is typically based on a conservative estimate of impacts in the local community and thus is expected to overestimate the risks for all members of the community (including sensitive individuals).
- A HHRA involves a number of aspects where a qualitative assessment is required to be undertaken. Where this is undertaken, it provides a general indication of potential impacts only.
- A HHRA relies on data provided from other studies prepared for the EIS (as listed for this project in **Table 1.2**). The conclusions of this HHRA, therefore, depend on the assumptions and calculations undertaken to generate the data from these other studies utilised in this assessment.
- Conclusions can only be drawn with respect to impacts related to a Project as outlined in the EIS. Other health issues, not related to the Project, that may be of significance to the local community are not addressed in the HHRA or EIS.
- The health impact assessment reflects the current state of knowledge regarding the potential health effects of identified chemicals and pollutants for this Project. This knowledge base may change as more insight into biological processes is gained, further studies are undertaken, and more detailed and critical review of information is conducted.

2. PROJECT DESCRIPTION

Bowdens Silver is proposing to develop and operate the Bowdens Silver Project to recover mineralised rock (ore) containing silver and small percentages of zinc and lead from an open cut pit. The Bowdens Silver Project (the Project) comprises seven principal components, namely:

i) a main open cut pit and two satellite open cut pits, collectively covering approximately 52ha;

ii) a processing plant and related infrastructure covering approximately 22ha;

iii) a WRE covering approximately 77ha;

iv) a low grade ore stockpile covering approximately 14ha (9ha above WRE);

v) an oxide ore stockpile covering approximately 8ha;

vi) a TSF covering approximately 117ha; and

vii) the southern barrier to stockpile NAF waste rock for later use in rehabilitation activities and provide visual and acoustic protection to properties south of the Mine Site covering approximately 32ha.

The above components would be supported by a range of on-site and off-site infrastructure. The on-site infrastructure comprises haul roads, water management structures, power/water reticulation, workshops, stores, compounds and offices/amenities. The off-site infrastructure comprises a relocated section of Maloneys Road (including a new railway crossing and new crossing of Lawsons Creek), a 132kV power line and a water supply pipeline for the delivery of water from the Ulan Coal Mine and/or Moolarben Coal Mine.

Figure 2.1 shows the indicative locations of the principal mine components.

The Project would incorporate a conventional open cut pit operation with one main open cut pit and two satellite pits, where overburden/waste rock is removed from above and around the silver-zinc-lead ore and either used for on-site construction activities or placed in the out-of-pit WRE or the southern barrier. The mined ore would be transported by haul trucks to the on-site processing plant where it would be crushed, milled and processed to liberate the silver, zinc and lead minerals. These minerals would be collected by conventional froth flotation to produce two concentrates that would be dewatered and transported off site by truck. The residual materials from processing (tailings) would be pumped in the form of a slurry to a TSF located to the west of the open cut pit.

The Project would require a site establishment and construction period of approximately 18 months during which the processing plant and all related infrastructure and the initial embankment of the TSF would be constructed. Once operational, Bowdens Silver anticipates the mine would produce concentrates for approximately 15 years. In total, it is proposed the mine life would be approximately 16.5 years, i.e. from the commencement of the site establishment and construction stage to the completion of concentrate production. It is envisaged rehabilitation activities would be completed over a period of approximately 7 years, i.e. from Year 16 to Year 23. **Figure 2.2** displays the duration of each of the main components throughout the mine life and Project life.

Figure 2.1 Mine Site Layout

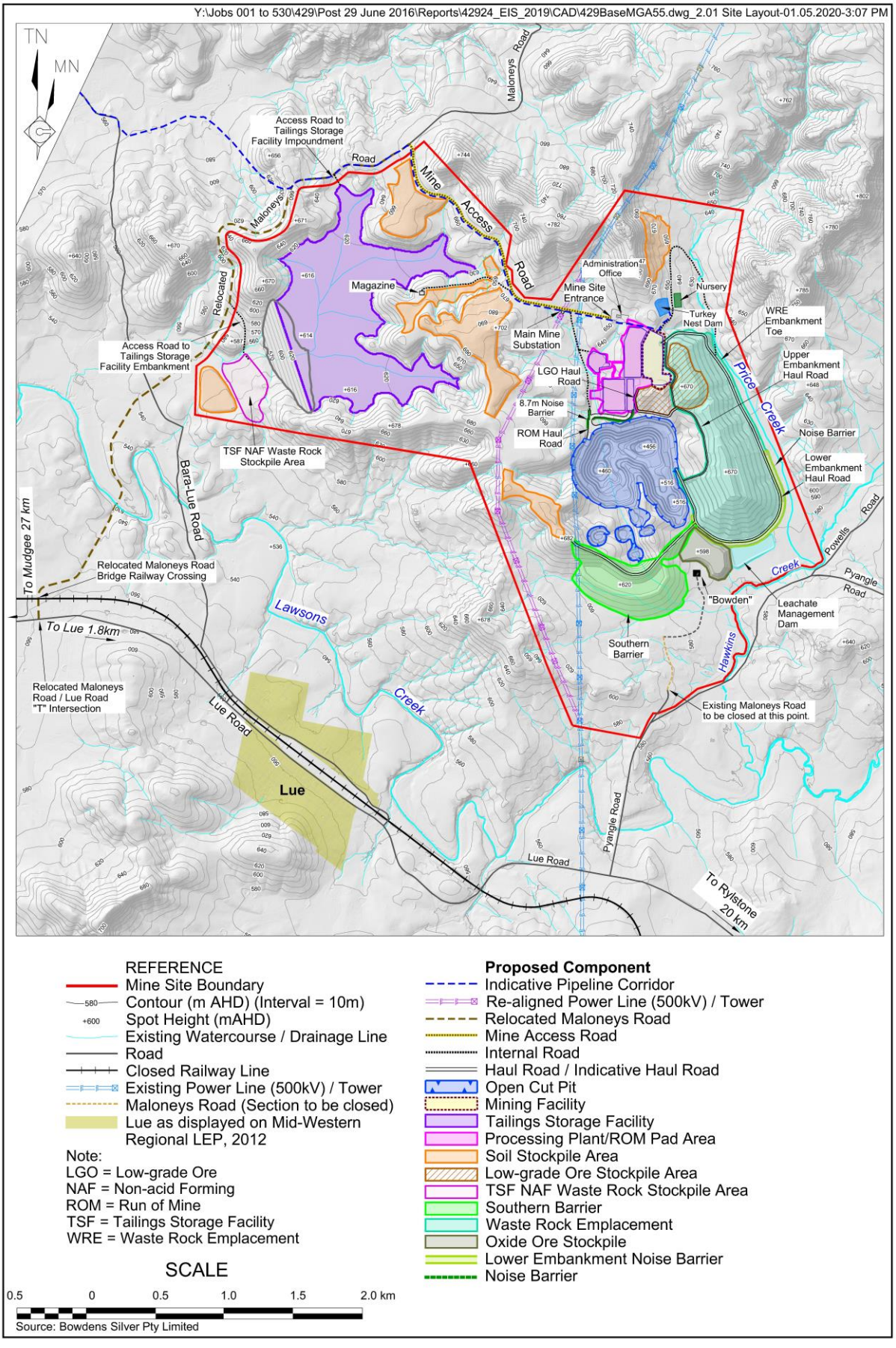
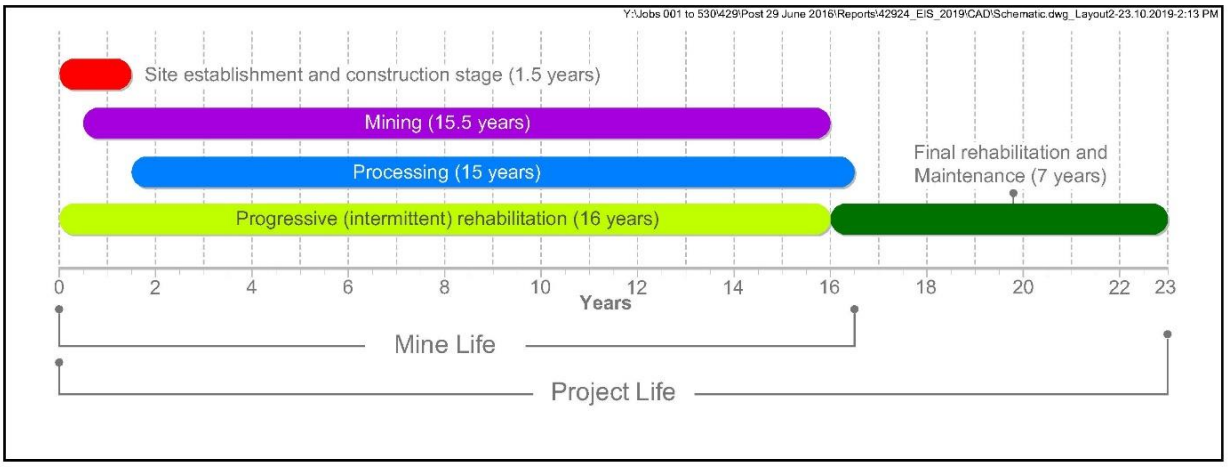


Figure 2.2 Mine Life and Project Life



The estimated annual ore and waste rock production is shown in **Table 2.1**, with the operational year scenarios chosen for assessment of impacts shaded grey and highlighted in bold.

Table 2.1
Estimated Annual Waste Rock and Ore Production

Operational Year	Ore (t)			Waste rock (t)	
	Ore	Low Grade Ore	Oxide Ore	Non-Acid Forming	Potentially-Acid Forming
SE&CS	113 722	27 212	94 467	3 886 107	1 201 545
1	1 744 717	260 511	293 439	927 755	2 773 578
2	1 908 260	228 710	237 645	2 433 037	1 192 348
3	1 702 839	411 050	338 161	2 057 928	1 490 023
4	1 955 782	575 512	96 984	1 712 068	1 659 655
5	2 010 709	505 487	-	1 601 690	1 882 114
6	2 070 259	504 965	1 463	1 109 668	1 313 645
7	2 048 673	435 549	144 594	909 633	1 408 766
8	1 477 833	368 361	255 872	1 720 556	1 177 379
9	498 246	203 257	263 882	2 381 835	1 652 780
10	1 313 773	338 695	56 406	807 046	2 484 080
11	1 377 297	474 018	-	200 188	2 948 498
12	1 679 457	568 307	-	49 706	2 702 531
13	1 661 617	427 979	-	19 573	1 413 339
14	1 501 122	498 878	-	588	1 061 239
15	769 451	230 549	-	-	221 093

SE&CS = Site Establishment and Construction Stage

3. COMMUNITY PROFILE

3.1 GENERAL

This section summarises the demographics and existing health of the community potentially impacted by the Project. The area surrounding the Mine Site comprises rural-residential and residential land uses.

The larger townships of Mudgee, Rylstone and Kandos are located approximately 24.5 km (to the northwest), 20.5 km (to the southeast) and 28km (to the southeast) respectively from Lue.

When considering potential health impacts within any community, the HHRA considers the whole population as well as specific sensitive or vulnerable groups within the population. These communities and their related sensitive or vulnerable groups are:

- Community groups:
  - Residents (including rural, rural-residential and residential within towns and villages)
  - Recreational users (including bushwalkers, recreational swimming in local creeks/rivers)
  - Commercial and industrial (i.e. workplaces).
- Sensitive and vulnerable groups within the community groups:
  - Young children
  - Older populations (>65 years of age)
  - Disabled and those with pre-existing medical conditions
  - Disadvantaged (socio-economically disadvantaged).

These receptors may reside or access any areas within the community.

3.2 SURROUNDING AREA AND POPULATION

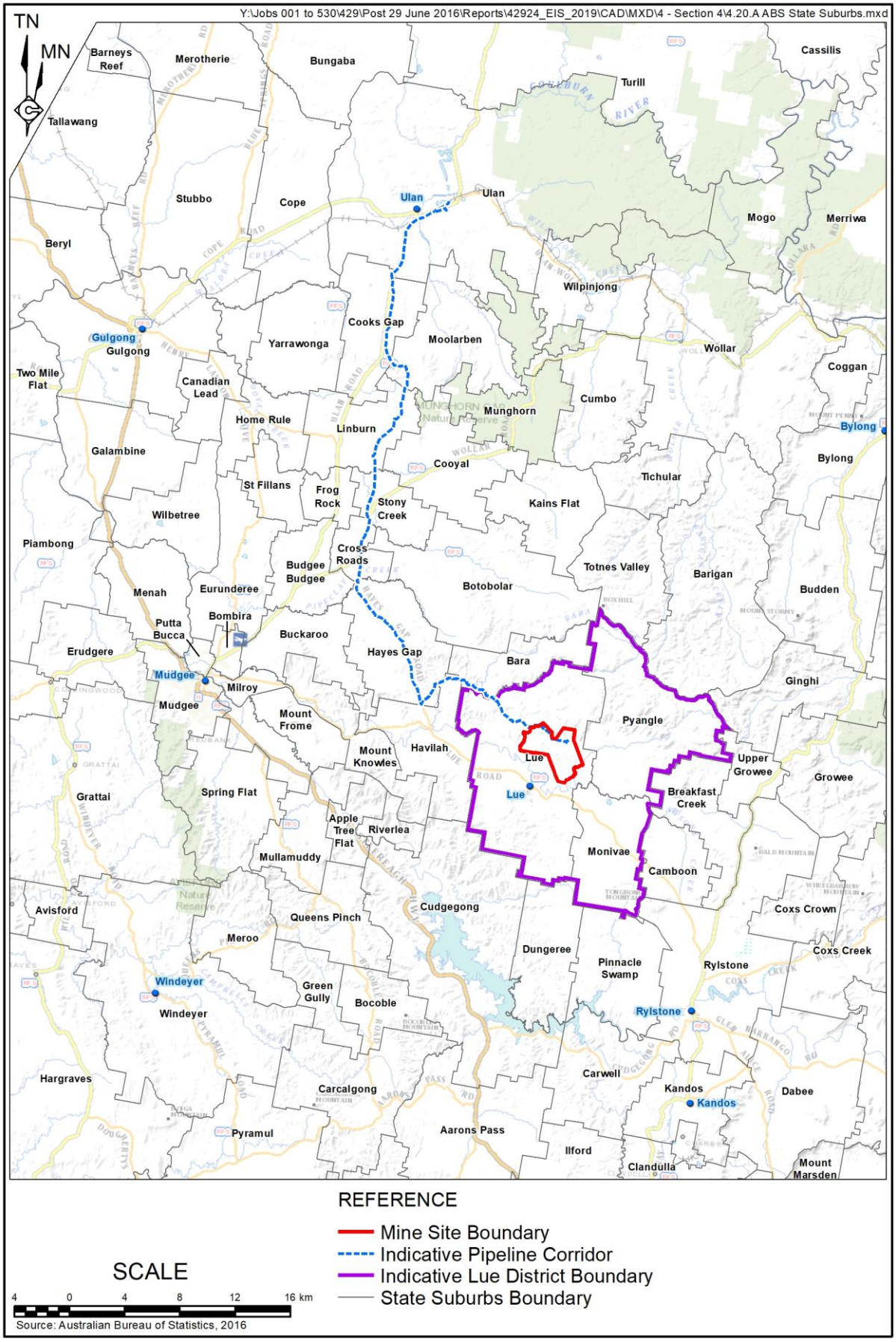
The Mine Site and surrounding areas (including Lue, Mudgee, Rylstone and Kandos) are located within the Mid-Western Regional, Local Government Area (LGA).

The LGA covers a large area, approximately 8737 km^2, with the eastern edge of the LGA incorporating the Wollemi National Park. The LGA sits within the Western NSW Health Area, which covers a very large area extending west (well west of Bourke and Cobar) and north to the NSW border.

Statistics relevant to the populations who may reside in the areas adjacent to and surrounding the Mine Site have been obtained from the Australian Bureau of Statistics (ABS), for state suburbs (SSC, as defined by the ABS, refer to **Figure 3.1**):

- Lue
- Rylstone
- Kandos
- Gulgong
- Mudgee

Figure 3.1 ABS State Suburbs – Lue Surrounds



It should be noted that there are a number of small state suburbs surrounding the Lue State Suburb (including Bara, Havilah, Pyangle and Monivae), as well as a number of smaller communities that are intersected by the proposed Pipeline from the Ulan Coal Mine and/or Moolarben Coal Mine that are also considered relevant to the Project. However, due to low populations within some of these communities, the majority of data captured in the ABS census and other sources is withheld or randomly adjusted in order to safeguard residents' anonymity. Hence the characteristics of populations in these smaller suburbs are assumed to be consistent with the characteristics of the key state suburbs listed above.

Data for these areas has been compared with statistics for the larger LGA of Mid-Western Regional (A) and (B) and for NSW (rural areas).

Population statistics for these state suburbs and larger areas are available from the ABS for the census year 2016 and are summarised in **Tables 3.1** and **3.2**. The composition of the populations located adjacent to the Mine Site is expected to be generally consistent with population statistics for the individual state suburb areas.

Table 3.1
Summary of Population statistics

Location	Total Population		% Population by Key Age Groups			
	Male	Female	0–4	5–19	20–64	65+
State suburbs (SSC)						
Lue	101	92	3%	26%	50%	21%
Rylstone	453	467	5%	16%	51%	28%
Kandos	671	647	5%	15%	52%	28%
Gulgong	1 246	1 272	8%	20%	52%	20%
Mudgee	5 330	5 594	9%	19%	55%	17%
Larger Local Statistical Areas (SA3 – includes all State Suburbs)						
Mid-Western Regional (A)	12 099	11 975	7%	19%	55%	20%
State						
NSW (Rural)	285 013	269 300	5%	20%	57%	18%

Ref: Australian Bureau of Statistics, Census Data 2016

SSC are statistical areas based on state suburbs

SA3 are larger statistical areas that are aggregates of SA2 areas with populations between 30 000 and 130 000

Table 3.2 summarises a selected range of demographic measures relevant to the population of interest with comparison against the larger population areas. This includes the Index of Relative Socio-economic Disadvantage, which is an index that summarises a range of information about the economic and social conditions of people and households in an area. The index uses 5 quintiles (ranging from 1 to 5, with each quintile representing 20% of the index range), with a low score indicating a relatively greater disadvantage (for example, many households with low income, many people with no qualifications or low in skills) and a high score indicating a general relative lack of disadvantage.

Table 3.2
Selected Demographics of Population of Interest

Location	Median Age	Median Household Income ($/week)	Median Mortgage Repayment ($/month)	Median Rent ($/week)	Average Household Size	Unemployment Rate (%)	Index of Relative Socio-Economic Disadvantage (Quintile)*
Suburb (SA2)							
Lue	46	504	1322	250	2.6	5%	3
Rylstone	50	856	1495	220	2.2	8.4%	2
Kandos	52	698	867	190	2	16.5%	1
Gulgong	41	1 086	1 517	250	2.4	8.6%	1
Mudgee	37	1 256	1 733	300	2.4	5.8%	2
Larger local statistical areas (SA3 – includes SA2 areas)							
Mid-Western Regional (A)	42	547	1 690	270	2.4	6.5%	3
State							
NSW (Rural)	45	626	1 733	200	2.7	4.4%	NA

Source: Australian Bureau of Statistics, Census Data 2016

-- insufficient population for these measures to be determined

SA2 are statistical areas based on state suburbs

SA3 are larger statistical areas that are aggregates of SA2 areas with populations between 30,000 and 130,000

* Quintile within NSW ranges from 1 which is most disadvantaged to 5 which is the least disadvantaged

Review of **Tables 3.1** and **3.2** indicates that the population of Lue and surrounding populations in Rylstone, Kandos, Gulgong and Mudgee have relatively similar population distributions to the larger Mid-Western Regional area and rural NSW. The key differences relate to the higher proportion of people aged 65 years and older, and lower proportion of people aged 5 to 65 years in Rylstone and Kandos. Household sizes are lower in Rylstone and Kandos, and these state suburbs along with Gulgong have higher levels of unemployment when compared with Mid-Western LGA. The index of relative socio-economic disadvantage indicates that most of the state suburbs in the area sit in the lower to mid quintiles (i.e. more disadvantaged to average in NSW in terms of disadvantage), with Kandos and Gulgong considered to be most socio-economically disadvantaged. This indicates that there are some areas of the community surrounding the Mine Site that may be more vulnerable to impacts, but also may benefit more should the project improves employment opportunities in these areas.

Lue Public School is located within Lue. Enrolments at the school between 2015 and 2018 ranged from 18 to 22 children from Kindergarten to Year 6. A total of 21 children are attending the school in 2019 and 24 are currently enrolled in 2020.

3.3 EXISTING HEALTH OF THE POPULATION

3.3.1 General

When considering the health of a local community there are a large number of factors to consider. The health of the Lue and district community is influenced by a complex range of interacting 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. While it is possible to review existing health statistics for the areas surrounding the Mine Site and compare them with larger areas such as the LGA or NSW, it is not possible or appropriate to be able to identify a causal source, particularly individual or localised sources.

Information relevant to the health of populations in NSW is available from various State and Australian government agencies including NSW Health, the Australian Institute of Health and Welfare and the Australian Commission on Safety and Quality in Health Care. This data relates to populations grouped by local government area or health district. These data sets are not available for individual suburbs. In addition, not all the health data that may need to be considered in the completion of a HHRA is available for all these areas. The data that can be considered in a HHRA depends on the availability of data relevant to the populations in the areas to be evaluated.

3.3.2 Health-related Behaviours

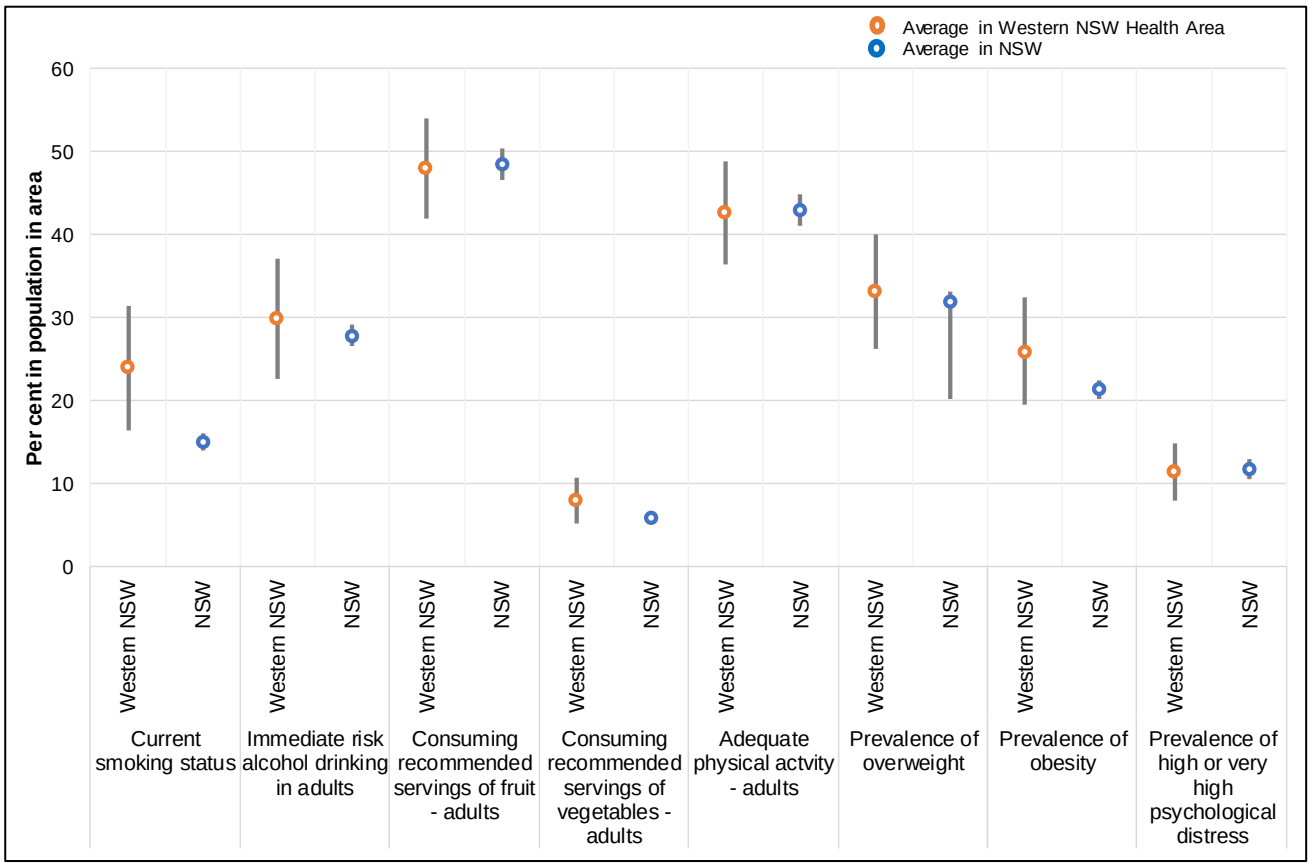
Information in relation to health-related behaviours linked to poorer health status and chronic disease including cardiovascular and respiratory diseases, cancer and other conditions that account for much of the burden of morbidity and mortality in later life is available for larger populations within LGAs. This data is regularly collected by NSW Health. This provides information on rates of smoking and alcohol consumption, physical activity, fruit and vegetable consumption, prevalence of overweight and obesity in the population and prevalence of high or very high levels of psychological distress.

Figure 3.2 shows a comparison of the available data from 2015 for the Western NSW Health Area and NSW in relation to these factors. These statistics are expected to remain representative of 2019 and the operation of the project.

The health-related behaviours presented are those with potential to adversely affect the health of the population.

Review of the data relevant to Western NSW indicates that this area has a higher rate of smoking and a higher prevalence of obesity than NSW, however for most other indicators Western NSW is similar to (on average) NSW.

Figure 3.2 Summary of Health-related Behaviours for Western NSW (average and 95% confidence interval)



3.3.3 Health Indicators

NSW Health provides data relevant to selected chronic diseases within the NSW population that relate to understanding the burden of disease (from a wide range of causes). Chronic diseases[1] considered generally relevant to the assessment of health impacts related to coarse particulates and noise from mining activities include hypertension, heart disease, stroke and respiratory disease (including asthma). In addition, data relevant to asthma are also relevant.

Figure 3.3 provides a summary of the prevalence data relevant to hypertension and asthma in the communities evaluated in this assessment, with comparison against NSW.

Figure 3.4 presents data relevant to hospitalisations and **Figure 3.5** presents data relevant to mortality associated with cardiovascular and cardiovascular (circulatory system) diseases, in the communities evaluated in this assessment, with comparison against NSW.

[1] Many different illness and health conditions can be classified under the broad heading of chronic disease. Typically, chronic diseases are long-lasting, and have persistent effects. Chronic diseases can range from mild conditions, such as short-sightedness, dental decay and minor hearing loss, to debilitating arthritis and low back pain, and to life-threatening heart disease and cancers. These conditions may never be cured completely, so there is generally a need for long term management. Once present, chronic diseases often persist throughout life, although they are not always the cause of death (refer to the Australian Government Department of Health for further details on chronic diseases).

Figure 3.3 Summary of Prevalence of Hypertension and Asthma (average and 95% confidence interval)

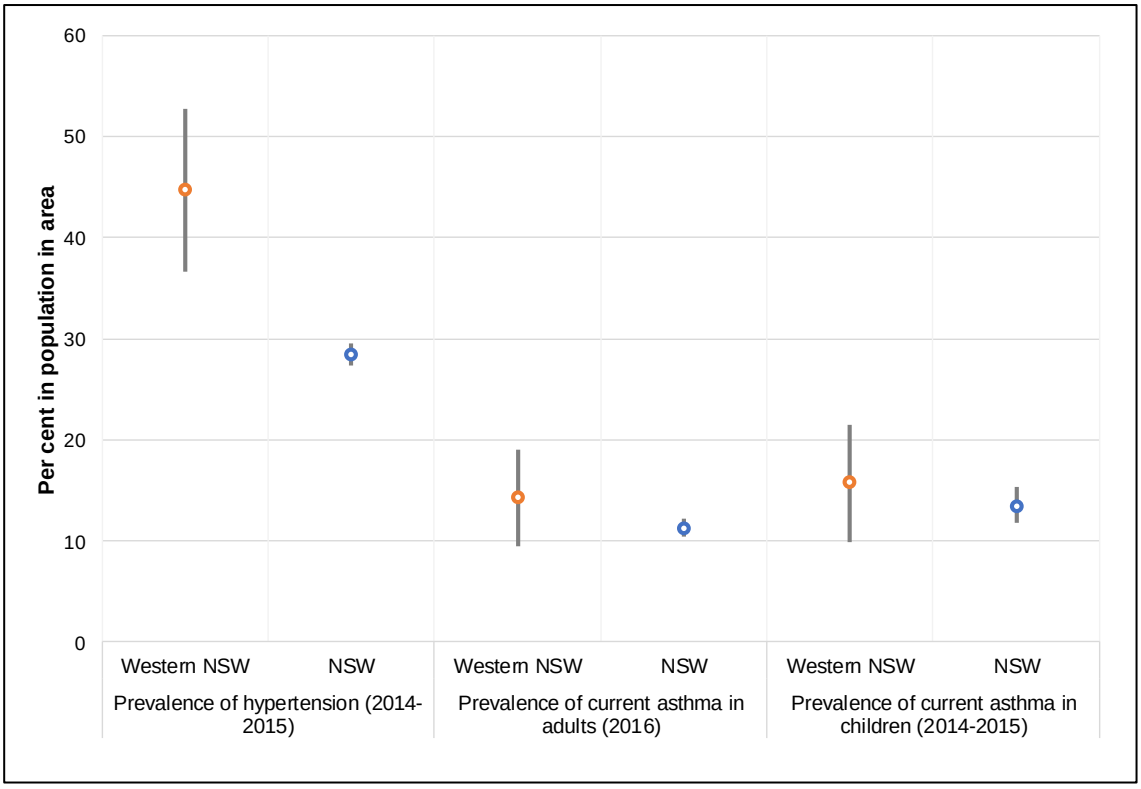


Figure 3.4 Summary of Hospitalisation Data for Hypertension, Respiratory and Cardiovascular Disease (average and 95% confidence interval)

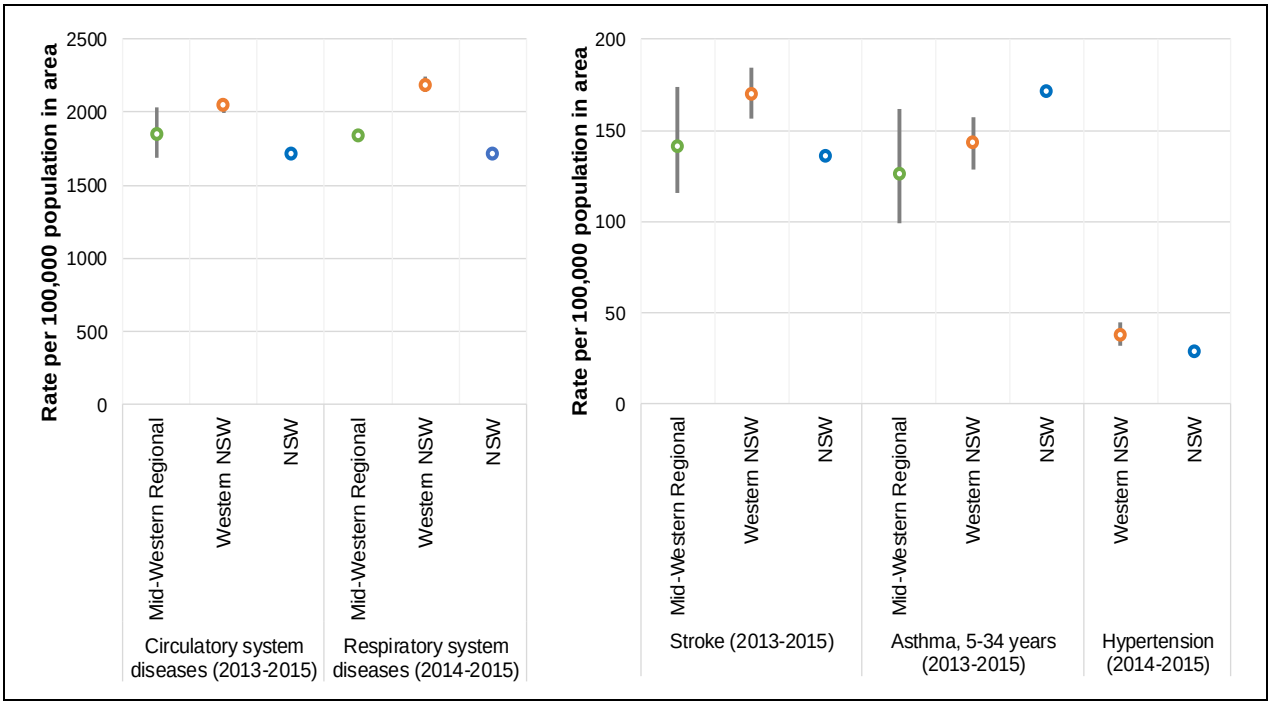
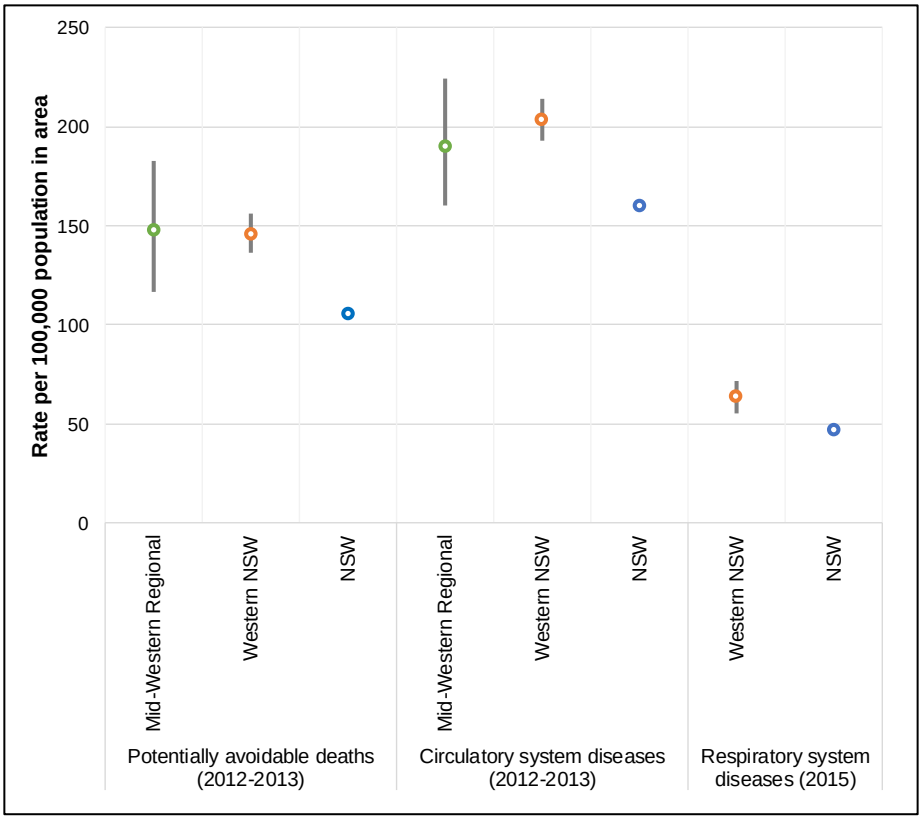


Figure 3.5 Summary of Mortality Data, including Respiratory and Cardiovascular Disease (average and 95% Confidence Interval)



Review of the available data in relation to mortality, the prevalence of disease and hospitalisation rates for the Mid-Western Regional LGA (where available) and the larger Western NSW Health Area, indicates the following:

- The prevalence of hypertension in Western NSW is significantly higher than the NSW average. Hospitalisations for hypertension are a little higher than for NSW.
- The prevalence of asthma in Western NSW higher than the NSW average. The hospitalisation rate for asthma, however, is lower than the NSW average in the Mid-Western Regional LGA and Western NSW. This may mean that asthma is better managed in this area, preventing hospitalisations.
- In relation to cardiovascular disease, the rate of hospitalisations for stroke and circulatory system diseases, and the mortality rate for circulatory system diseases, are higher in the Mid-Western Regional LGA and Western NSW, when compared with NSW. The hospitalisation and mortality rates are lower in the Mid-Western Regional LGA than the larger Western NSW Health Area, suggesting the cardiovascular health of the population in the LGA is better than that reflected in the data for the larger health area.
- In relation to respiratory disease, limited data is available for the Mid-Western Regional LGA, however data from the larger Western NSW health area suggests that hospitalisations and mortality associated with respiratory disease are higher, compared with NSW. The rate of hospitalisations for respiratory system disease in the Mid-Western Regional LGA is higher than NSW, but lower than for the larger Western NSW health area. This suggests that respiratory health may be better in the LGA than reflected in the data for the larger health area.

- The rates of potentially avoidable mortality in the Mid-Western Regional LGA and the larger Western NSW health area are higher than for NSW.

The above indicates that the population in the areas surrounding the proposed mine is expected to have higher than average rates of respiratory and cardiovascular disease, which may mean they may have some potential to be more susceptible to changes in exposures related to the Project. This has been considered in the HHRA.

3.4 COMMUNITY CONCERNS AND PERCEPTIONS

Community consultation activities have been undertaken throughout the EIS process. This process has identified a range of community concerns that relate to human health, that are summarised in **Table 3.3**, along with reference to where these concerns have been addressed in this report.

Table 3.3
Issues raised by Lue and District Community relevant to Human Health

Issue(s)	Coverage in Report
Potential impacts from dust and any associated metals on drinking water supplies, livestock and aquatic environments.	Section 5.2
Modelling of dust dispersion from the mine and processing activities.	Section 5.2 and the Air Quality Assessment (Ramboll, 2021)
Potential impacts from dust and any associated metals on human health.	Section 5.2
Potential impacts on stress and anxiety levels of local residents, local property values, likely change in demographics of local population of Lue, health implications, impact upon future viability of Lue Public School, etc.	Refer to EIS Section 4.8.8
What contaminants are likely to be dispersed by air and water as a result of mining operations?	Sections 5 and 6
Is the buffer zone around the mine adequate? Why isn't it 8km like coal mines?	The HHRA has not identified any requirement for a buffer zone
What are the potential health impacts of the potential contaminants/minerals comprising the dust?	Section 5.2
Which metals / contaminants will be assessed in the EIS?	Section 5.2
Will it still be safe to drink water from rainwater tanks?	Section 5.2
What are the impacts of dust on human health?	Section 5.2
What other health effects will people be exposed to from the mine?	Sections 5, 6 and 7 which address health impacts of emissions to air, water (surface water and groundwater) and noise

Table 3.3 (Cont'd)
Issues raised by Lue and District Community relevant to Human Health

Issue(s)	Coverage in Report
Will the mine result in more people on dialysis?	The assessment relates to the assessment of potential health impacts for all members of the Lue and district community including sensitive individuals. The health effects considered include those related to the kidney (refer to Section 5 and **Annexure B**)
Does silica have the capacity to cause health impacts (e.g. silicosis)?	Section 5.3
How will stress and anxiety within the community be managed?	Refer to EIS Section 4.8.8
What are the health impacts of noise fatigue?	Section 7
What is a safe distance to be living from the mine?	The HHRA has not identified any requirement for a buffer zone
Will a more detailed health and environmental risk assessment be conducted?	Health risks are addressed in detail in this report
What level of lead would we be exposed to in Lue?	Sections 4 and 5
How will the lead from the mine enter bodies?	Refer to **Annexure B**
How will the exposures to lead in Lue compare to those in Mt Isa?	Section 5
Concerned about the health, and psychological impacts / "Will be very traumatic for everyone" (having the project approved and in operation)	Health impacts addressed in this report.

3.5 OVERVIEW OF EXISTING COMMUNITY AND HEALTH

Overall, the population surrounding the Mine Site is small, but generally similar to the population in the larger Mid-West Regional LGA and rural NSW areas. There are some smaller areas with higher rates of unemployment.

In relation to the health of the population in the local area, this area has a higher rate of smoking and a higher prevalence of obesity than NSW. In addition, the area has a higher rate of cardiovascular and respiratory disease than NSW, which may have some influence on the susceptibility of the population to environmental stressors.

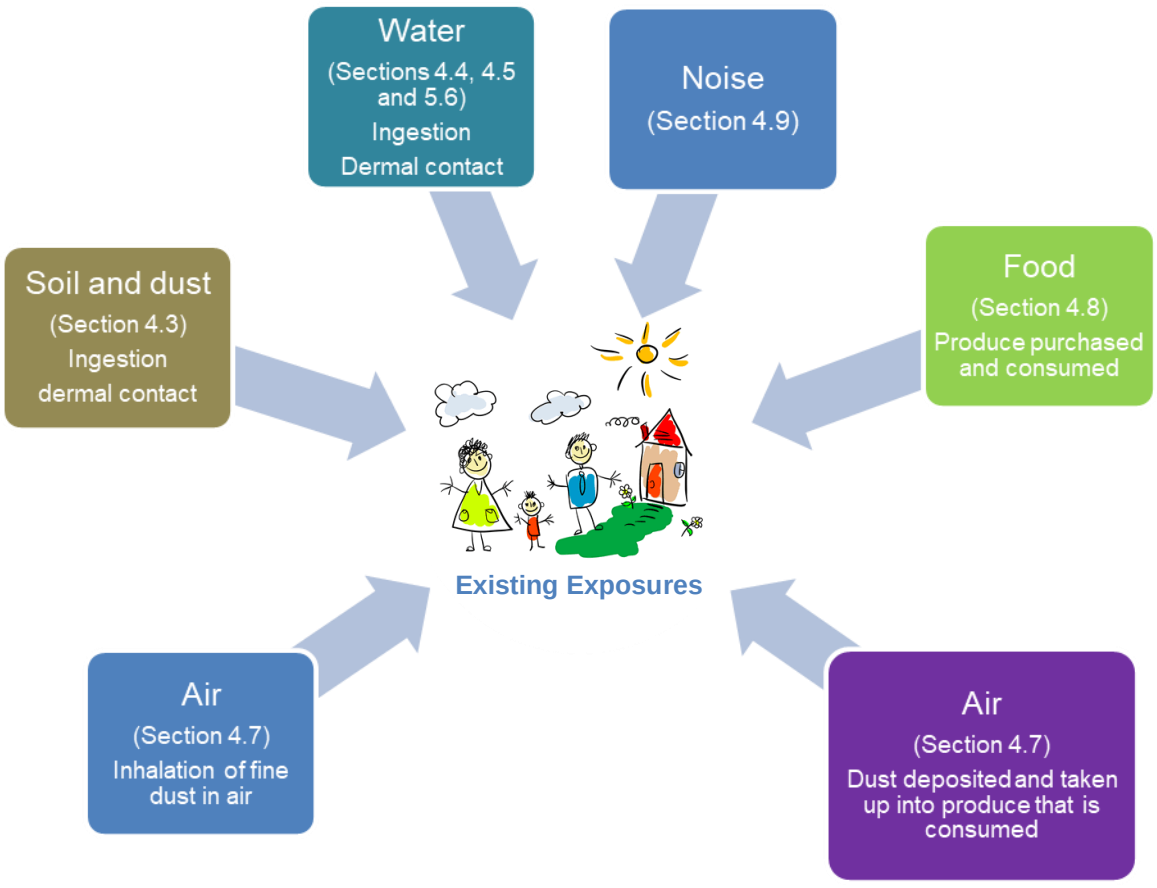
Some members of the local community have expressed a number of concerns in relation to the Project, with most concerns relating to dust from the mining activities and the impact of this dust, which includes lead, on the health of all members of the community including sensitive groups such as children and those with existing health conditions.

4. EXISTING ENVIRONMENT

4.1 GENERAL

The assessment of health impacts, associated with the Project, involves assessment of potential community exposures to a range of contaminants, pollutants and stressors, most of which are already present in the environment. As such it is important to understand the existing environment, and what data is available to define the existing exposures that may occur within the community. This section discusses the available information relevant to the assessment of metals in the existing environment, that contribute to existing levels of exposure within the community, as illustrated in **Figure 4.1**. This relates to levels present in soil, indoor dust, water (groundwater, bore water, surface water and rainwater tanks), air (as particulates in the air and dust deposited onto the ground) and in foods consumed by the community.

Figure 4.1 Existing Exposures to Metals in the Environment: Sources and Pathways of Exposure



4.2 GENERAL DESCRIPTION OF THE AREA

The Mine Site is located approximately 2km to 3km northeast of Lue and 26km east of Mudgee in the Central Tablelands region of NSW. The land use of the area is a mixture of cleared and semi - cleared agricultural land and scattered forests.

The Project and surrounding region is defined by undulating topography, with elevated terrain adjacent to the north and south of a dominant west-northwest to east-southeast aligned valley. An area of elevated terrain separates Lue and the Mine Site.

The region surrounding the Mine Site is sparsely populated with rural and rural-residential properties located at varying distances from the proposed on-site activities. The spatial distribution and current status of residences surrounding the Mine Site is illustrated in **Figure 4.2** with **Figure 4.3** showing the properties and places if interest within Lue. It is noted that some of the residences closest to the Mine Site are owned by Bowdens Silver and are referred to as 'project-related'. The non-project-related ("privately-owned") properties are the receivers considered in the assessment of air and noise impacts for the Project.

The Mine Site is located inland with moderate levels of rainfall. The average annual rainfall in Mudgee is 663 mm, while that recorded at Lue is 635 mm, with the wetter months being December and January and drier months being April and May.

4.3 GEOLOGY AND SOIL

The geology of the study area comprises Ordovician bedrock and Early Permian Rylstone Volcanics overlain by shallow marine sandstones, conglomerates and shales, in an area that includes a number of faults and fractures. The upper soil profile comprises alluvium and colluvium particularly around surface water/drainage features, which are described as silty sandy gravel and clays.

A mineralised area, the target of the proposed open cut pits, occurs as a thick zone extending from the surface (or near surface) to vertical depths of around 200 m. The ore body dips at up to 30 degrees and is not uniformly mineralised. The mineralised materials comprise silver (Ag), zinc (Zn) and lead (Pb). The presence of the mineralised area is expected to have already had some influence on the nature of natural soil materials in the area, as well as sediments and water quality.

Soil samples have been collected from a number of areas, principally within the Mine Site, as well as other prospect areas within Bowdens Silver's exploration licence areas, to understand the composition of these materials. Data is available from the following sampling programs.

- A regional soil sampling program undertaken in 2012 to cover the exploration licence areas (where accessible), where samples were collected from the soil zone below the root zone, with laboratory analysis for a wide range of metals including trace metals.
- A select number of soil and dust samples were collected from the Lue Public School, and other buildings in the local area as part of early sampling works, conducted in 2012. These samples were also analysed for selected other metals. These data reflect concentrations of these metals in soil, as well as levels that are present in dust indoors (as a bulk dust sample or as surface sample).
- Collection of soil samples from a number of test pits across the Mine Site in 2017.

In relation to the soil samples collected, **Table 4.1** presents a summary of the soil and dust data for metals (excluding trace metals). The data relevant to lead is presented in **Table 4.2**.

Figure 4.2 Location and Land Ownership of Properties Surrounding the Mine Site

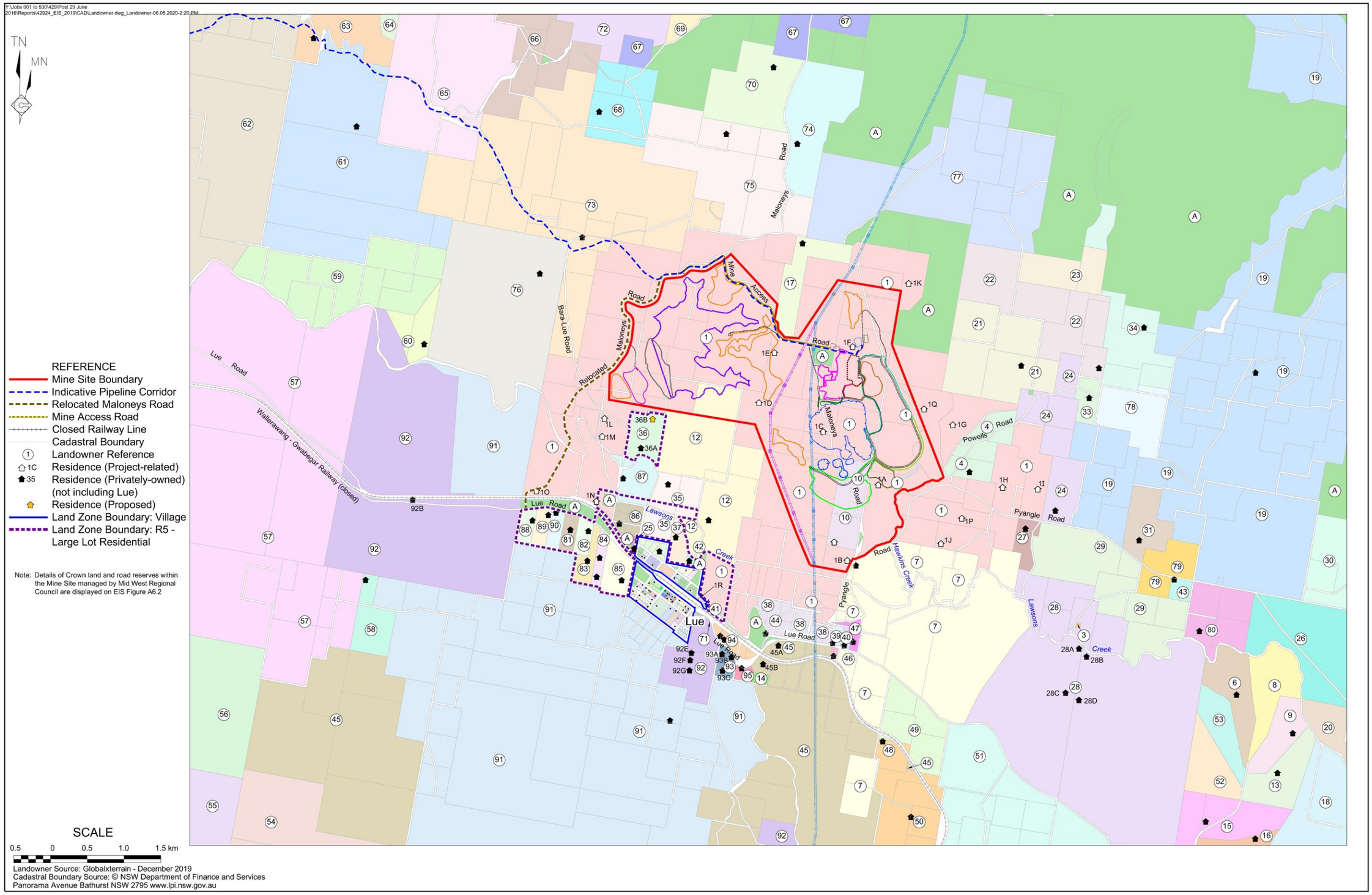


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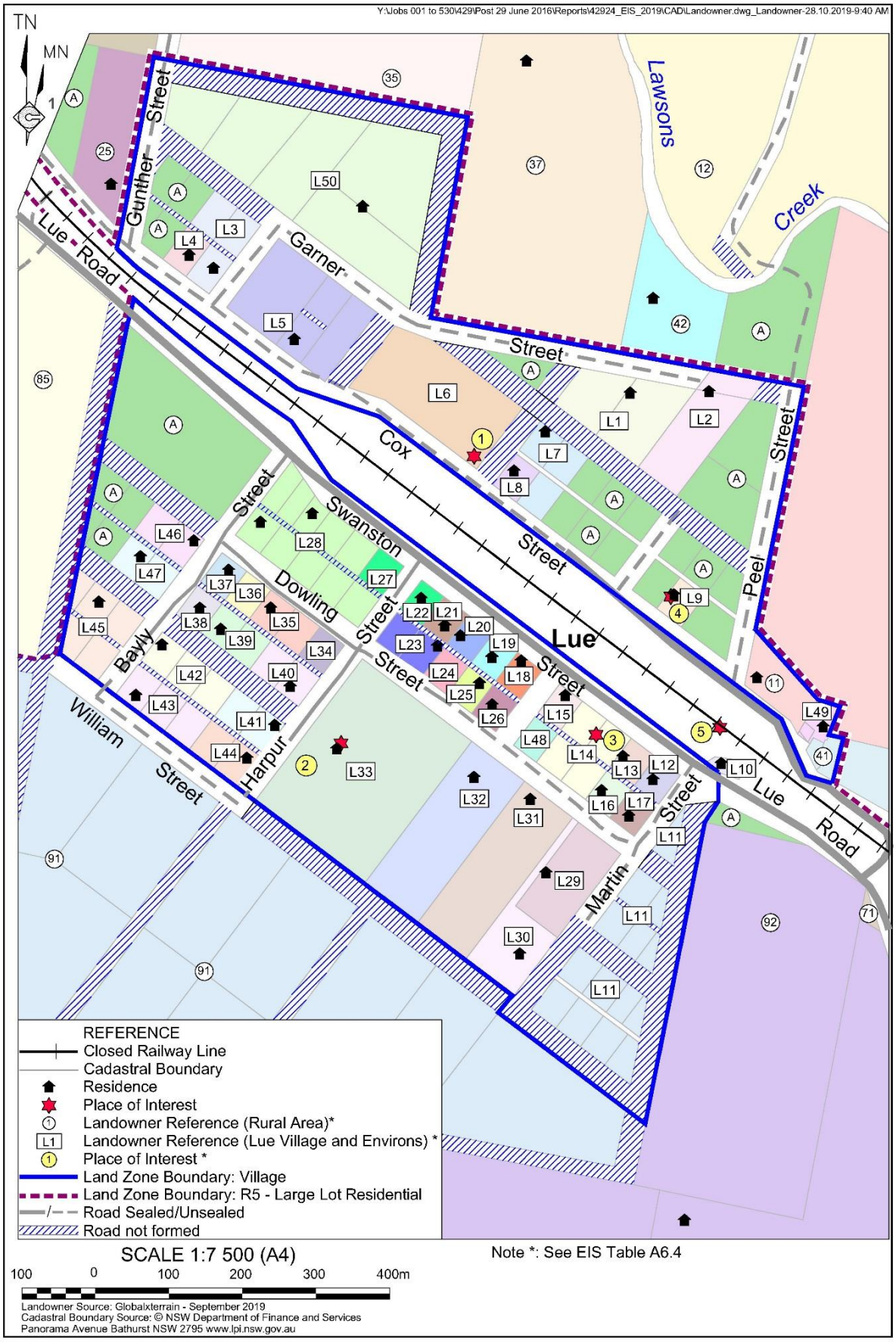
Figure 4.3 Location of Properties and Places of Interest in Lue



The soil data have been compared against health-based guidelines relevant to low-density residential land-use available from Australia (NEPC 1999 amended 2013b) and the US (USEPA 2016). These criteria include exposures via ingestion of soil and dust (indoors), dermal absorption from soil and dust, inhalation of dust and ingestion from homegrown produce (fruit and vegetables). The guidelines may not be adequately protective of all exposures that may occur on a rural property in relation to produce, however the guidelines do assist in understanding the significance, or otherwise, of existing levels or metals in soil.

Concentrations that exceed the adopted soil guidelines are highlighted in **Tables 4.1** and **4.2**, in blue text. The exceedances relate to the maximum concentrations of arsenic, manganese and zinc in soil. None of the average or mean concentrations exceed the available health-based guidelines.

For the assessment of exposure to existing soil concentrations in the community this assessment has used the mean concentrations reported on the mine site from **Table 4.1**. For the assessment of lead, soil samples collected from the mine site away from the proposed main open cut pit reported lead concentrations less than 50 mg/kg (refer to **Table 4.2**). To be conservative, the HHRA has assumed an existing lead concentrations in soil of 50 mg/kg.

In relation to the metals reported at the public school and in other properties near the site, in general the concentrations reported were similar to those reported in soil, with the exception of zinc, where high levels were reported in roof dust, likely reflecting zinc from building materials (zinc roofing materials which is common in the area). The level of mercury reported indoors may also reflect older building materials.

In relation to lead levels in the environment, the indoor data reflects the presence (known or otherwise) of lead paint and lead materials in roofing materials. In particular, lead paint was identified at the Lue Public School which has resulted in the presence of elevated levels of lead in dust indoors, and higher than average levels of lead in soil close to the buildings. Exposure to lead paint at Lue School (and associated elevated levels of lead in dust indoors[2] and in soil close to the building) should be addressed and managed by the school, and Department of Education.

Soil concentrations are generally lower than the health-based guideline, with the exception of levels reported in the proposed main open cut pit (which is not where people live).

4.4 GROUNDWATER

The occurrence and quality of groundwater beneath and surrounding the Mine Site is described in the Groundwater Assessment for the Project by Jacobs (2020).

Within the Study Area, five key aquifer types have potential to exist or have been identified in the vicinity of the Mine Site by Jacobs (2020), these being:

- Alluvial / Colluvial Aquifers – Unconsolidated sedimentary / detrital aquifers
- Porous Rock Aquifers – Consolidated sedimentary / detrital rock with connected primary porosity

[2] Elevated levels of lead were identified in indoor dust, in excess of the current guidelines from NSW EPA and NSW Planning (2003), which includes a guideline of 5.4 mg/m^2 for interior window sills and ledges. It is noted that the USEPA has proposed revisions to lead dust criteria for indoor surfaces, with the revised criteria ranging from 0.1 mg/m^2 for floors and 1 mg/m^2 for window sills (https://www.federalregister.gov/documents/2019/07/09/2019-14024/review-of-the-dust-lead-hazard-standards-and-the-definition-of-lead-based-paint). These more stringent guidelines may need to be considered in any further assessment and management of lead exposures from lead paint at the school.

Table 4.1
Soil and Dust Samples for Metals: Existing Environment

Metal	Concentration Reported in Soil – Mine Site (and other Licence Areas) (mg/kg)				Concentration Reported in Soil and Dust, Range (mg/kg)			Soil Guidelines – Low-density Residential Land Use (mg/kg)
	Mean	Median	Minimum	Maximum	Lue Public School – Soil	Lue Public School - Dust	Other Local Areas - Dust	
Silver (Ag)*	0.50	0.045	0.007	49.8				390 U
Aluminium (Al)	1.4	1.2	0.16	4.35				77000 U
Arsenic (As)*	15	6.5	0.26	558	4	4 - 12	6 - 120	100 N
Boron (B)	0.051	0	0	10				4500 N
Barium (Ba)	151	129	12.8	667				15000 U
Beryllium (Be)	0.78	0.68	0.04	5.39				160 U
Cadmium (Cd)*	0.13	0.036	0	5.72	<0.5	<0.5 - 12	<0.5 – 11	20 N
Cobalt (Co)*	9.5	6.2	0.598	55.394				100 N
Chromium (Cr)*	21	15.54	2.4	204.3	29 - 40	31 - 110	10 - 190	100 N as Cr VI
Copper (Cu)*	21	8.32	1.37	140.5	24 - 30	32 - 33	22 – 180	6000 N
Iron (Fe)	2.4	1.75	0.53	8.14				55000 U
Mercury (Hg)*	0.029	0.022	0	0.31	<0.1	1.3 – 2.4	<0.1 – 7.1	40 N
Lithium (Li)*	5.7	4.45	0.3	27.6				160 U
Manganese (Mn)*	1113	594	37	14350				3800 N
Molybdenum (Mo)	0.97	0.81	0.18	8.69				390 U
Nickel (Ni)*	12	6	0.9	157.5	9 - 13	6 - 11	5 - 270	400 N
Antimony (Sb)	0.25	0.042	0	10.05				31 U
Selenium (Se)	0.76	0.4	0.03	23.5				200 N
Tin (Sn)	2.1	0.9	0.25	13.895				47000U
Strontium (Sr)	21	9.2	0.1	241				47000 U
Titanium (Ti)	0.014	0.007	0	0.087				140000 U
Vanadium (V)	14	6	0.009	139				390U
Tungsten (W)	0.27	0.14	0	2.959				63 U
Zinc (Zn)*	24	5.9	0.005	863	43 - 410	4900 - 89000	680 - 24000	7400 N

Notes:
Data for the Mine Site is from 388 soil samples collected from the base of the organic root zone (lower B and upper C horizons in the presented soil horizon profile).
Blue text = exceeds the residential soil guideline
N = NEPM Health Investigation Levels for Low-Density Residential HIL-A which is applicable to soil and indoor dust (NEPC 1999 amended 2013b)
U = USEPA Regional Screening Level for Residential Soil (USEPA 2019)
* = Metals further considered in the modelling of dust emissions from the Project

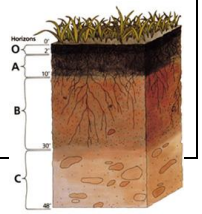


Table 4.2
Soil and Dust Samples for Lead: Existing Environment

Media/Measure	Lead Level (Range or Maximum)	Guideline
Dust Indoors		
Dust wipes from indoor surfaces - Lue	0.002 to 9.92 mg/m^2	1.076 mg/m^2 for interior window sills [U] 0.108 mg/m^2 for floors [U] 10.76 mg/m^2 for celling spaces [U1]
Dust wipes from indoor surface - Lue Public School where lead paint is present*	70 mg/m^2 in ceiling space	
Accumulated dust in ceilings and indoor surfaces - Lue	20 to 5600 mg/kg	300 mg/kg [N] for indoor surfaces No criteria for ceiling spaces
Accumulated dust in ceiling and indoor surfaces – Lue Public School where lead paint is present*	48,000 mg/kg in ceiling space	
Soil		
Soil on Mine Site (exploration licence areas) (388 samples)	< 50 mg/kg away from proposed main open cut pit (with 50 mg/kg adopted as representative of existing lead concentrations in soil) 1.5 to 1380 mg/kg in main open cut pit area	300 mg/kg [N]
Soil adjacent to building at Lue Public School where lead paint is present*	280 mg/kg adjacent to building 190 mg/kg, 1m away 36 mg/kg, 2m away 35 mg/kg, 3m away 42 mg/kg, 4 m away 12 mg/kg in another location	

Notes:
Data provided in the following reports: JBS 2013c, JBS 2013b, JBS 2013a, JBS 2012. Data for soil at the Mine Site is from 388 samples collected from the base of the organic root zone (refer to Table 4.1). Data for soil and dust at Lue Public School is from 6 surface soil samples and 2 dust samples. Data for dust at Lue is from 14 samples
* Lead paint was confirmed to be present, with analysis of paint chips indicating lead content of 3% to 8.1%
N = NEPM Health Investigation Levels for Low-Density Residential HIL-A which is applicable to soil and indoor dust (NEPC 1999 amended 2013b)
U = USEPA 2020, Review of the Dust-Lead Hazard Standards and the Definition of Lead-Based Paint, 84 FR 32632, US Environmental Protection Agency (USEPA 2020)
1 = Any exposures to dust from the ceiling space would be expected to be very limited and much lower than for window sills. Where this limited exposure is considered an additional 10 fold adjustment factor can be applied to the standard for window sills. This assumes that children are in contact with dust from the ceiling space 10 times less often than on window sills which remains conservative for dust in ceiling spaces

- Fractured Rock Aquifers – Consolidated rock with secondary fracture controlled permeability
- Shear / Fault Controlled Aquifer – Typically linear/planar fractured aquifer of defined width and extent
- Regolith Transition Zone Aquifers – In situ weathered rock with permeability enhanced by chemical weathering processes.

Within each of these aquifer types, there are potentially very broad variations in hydraulic properties.

Alluvial aquifers are poorly developed in the vicinity of the proposed open cut pits, however, more substantial alluvial deposits are associated with Hawkins and Lawsons Creeks and have the potential to be within the area of groundwater drawdown resulting from the development of the open cut pit.

Groundwater occurs in all of the rock formations underlying the Mine Site, these being the Rylstone Volcanics, the overlying Sydney Basin sedimentary rocks, and the underlying Ordovician basement lithologies. Within these rock formations, most of the groundwater would be found within the cracks and fractures in the rock.

Approximately 106 groundwater bores are registered within 10 km of the centre of the main open cut pit (refer to **Figure 4.4**). Twenty four of those are monitoring bores currently utilised by Bowdens Silver. The majority of private bores are used for stock, domestic and irrigation purposes.

Groundwater in the local area has been sampled, to determine existing concentrations of metals. These data are summarised in **Table 4.3**.

4.5 SURFACE WATER

The surface water catchments and water quality within and surrounding the Mine Site are described in the Surface Water Assessment for the Project by WRM Water and Environment (2020).

The Project is located within the Lawsons Creek catchment, in the eastern headwaters of the Macquarie River basin. Lawsons Creek flows in a northwesterly direction and drains to the Cudgegong River near Mudgee. The Cudgegong River flows in a northwesterly direction from Mudgee, before turning to the southwest and eventually draining to Lake Burrendong. Hawkins Creek, a tributary of Lawsons Creek, flows in a southwesterly direction along the southeastern boundary of the Project (WRM, 2020).

The bulk of the original vegetation of the Lawsons Creek catchment has been cleared to support agricultural activities. Historically, Lawsons Creek was likely to have been an intermittent to perennially discharging watercourse, however, subsequent land use changes and the construction of dams and storage structures to support agriculture have altered the hydrologic regime such that Lawsons Creek may now be described as an intermittent to ephemeral watercourse.

Ephemeral first and second order drainages (streams not fed by a perennial stream) traverse the Mine Site grading generally in a southerly direction to Hawkins Creek. Hawkins Creek is also an intermittent to ephemeral watercourse and joins Lawsons Creek just south of the Mine Site.

Figure 4.4 Registered Groundwater Bores and Potential Terrestrial Groundwater Dependent Ecosystems within 10km of Mine Site (Jacobs, 2020)

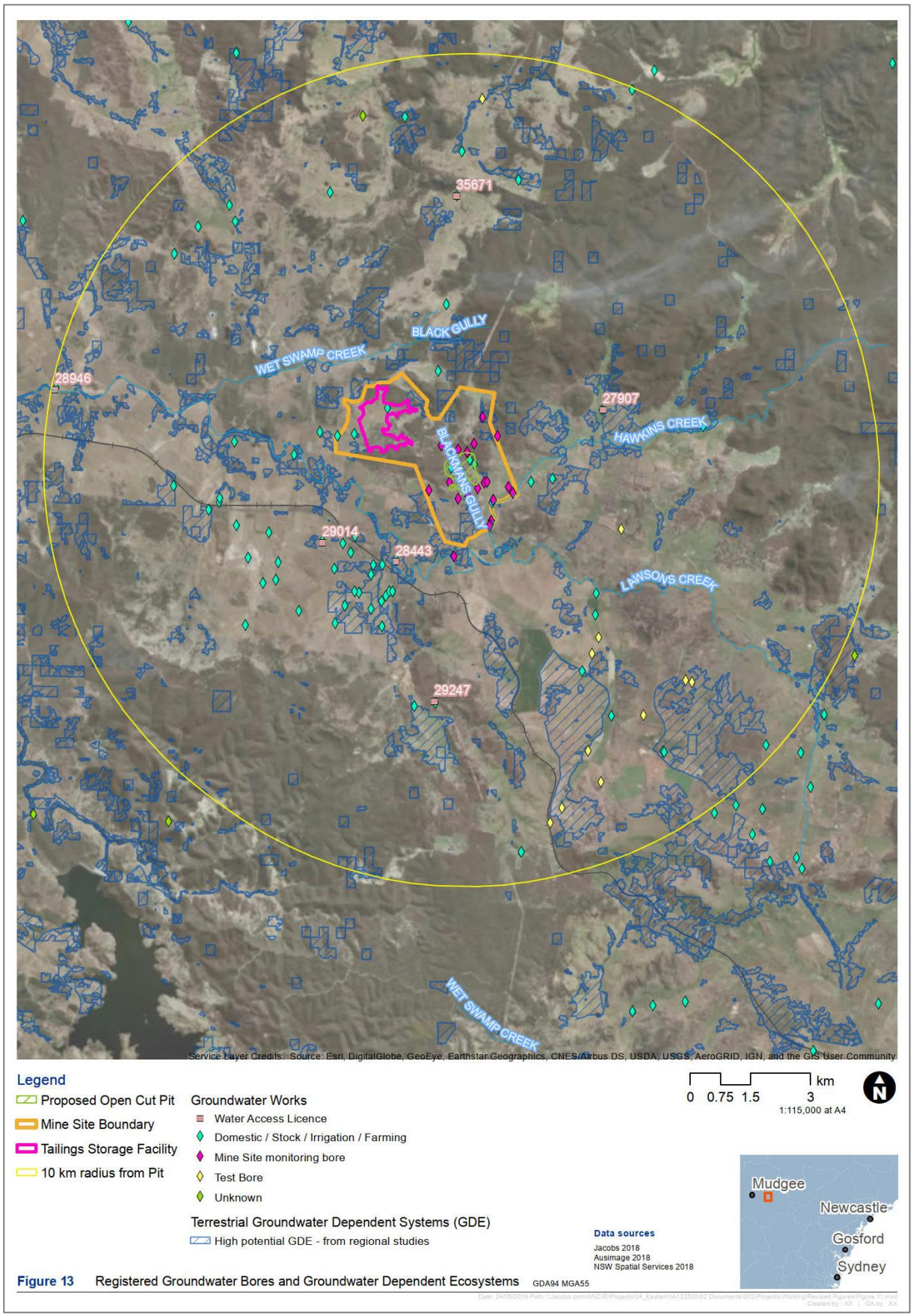


Table 4.3
Summary of Existing Surface Water and Groundwater Concentrations

Metal or Indicator	Concentration – Range of Averages (mg/L)						Water Quality Guidelines (mg/L)	
	Alluvium	Site (fractured rock aquifers)	Regional (fractured rock aquifers)	Springs	Domestic Bores^	Surface Water	Drinking water	Recreational water
Electrical conductivity (µS/cm)	131 - 2320	294 – 4364	708 – 3095	107 – 174	35 - 3180	71.7 – 1449.6	Converted to and evaluated as TDS as below	
Palatability as TDS (mg/L)	83.7 - 1485	191 - 1519	411 - 3032	68 - 112	22 - 2035	46 - 928	0 – 600 = good 600 – 900 = fair 900 – 1200 = poor >1200 = unacceptable No health criteria	
pH	5.98 – 7.23	5.4 – 7.94	6.78 – 8.13	4.68 – 7.54	3.9 – 9.0	4.0 – 7.8	6.5 to 8.5 for aesthetics[A] No health guideline	
Ammonia	0.02 – 2.4	0.024 – 0.475	0.02 – 0.33	0.027 – 0.24	-	<0.01-0.06	0.5 aesthetics[A] No health guideline	
Arsenic	0.002 – 0.02	0.001 – 0.290	0.001 – 0.07	0.028 – 0.235	<0.001 – 0.002	0.001 – 0.0025	0.01[A]	0.1
Cadmium	0.0001 – 0.0008	0.0001 – 0.0003	0.0001 – 0.0042	0.001 - 0.007	-	<0.0001 – 0.0002	0.002[A]	0.02
Chromium	0.001	0.001 – 0.002	0.0001 – 0.003	-	-	-	0.05[A]	0.5
Cobalt	0.002 – 0.0069	0.001 – 0.15	0.001 – 0.004	0.001 – 0.011	-	0.001 – 0.004	0.006[U]	0.06
Copper	0.001 – 0.015	0.001 – 0.013	0.002 – 0.068	--	0.006 – 0.9	0.001 – 0.002	2[A]	20
Iron	0.08 – 14.4	0.087 – 143.2	0.085 – 3.2	0.19 – 1.2	<0.05 – 1.1	0.06 – 0.535	0.3 for taste[A]	
Lead	0.002 – 0.007	0.001 – 0.016	0.001 – 0.068	0.001 – 0.007	<0.001 – 0.03	<0.001 – 0.007	0.01[A]	0.1
Lithium	0.002 – 0.704	0.002 – 0.656	0.001 – 0.287	0.001 - 0.008	-	0.001 – 0.012	0.04 [U]	0.4
Manganese	0.006 – 1.916	0.004 – 29.495	0.004 – 1.354	0.001	0.006 – 1.1	0.0315 – 0.293	0.5[A]	5
Mercury	-	-	-	-	<0.0001 – 0.0001	-	0.001[A]	0.01
Molybdenum	0.002	0.002 – 0.013	0.002 – 0.013	-	<0.001 – 0.007	0.003 – 0.004	0.05[A]	0.5
Nickel	0.001 – 0.006	0.001 – 0.25	0.001 – 0.038	0.032 – 0.423	-	0.002 – 0.004	0.02[A]	0.2
Strontium	0.028 – 0.73	0.073 – 3.77	0.26 – 4.3	0.017 – 0.069	-	0.027 – 0.56	12 [U]	120
Zinc	0.006 – 0.039	0.007 – 1.112	0.017 – 0.285	0.013 – 0.054	0.008 – 2.9	0.006 – 0.014	3 for taste[A] 6 for health[U]	60
Nitrate	0.04 – 3.407	0.033 – 2.708	0.105 – 10.878	0.02 – 0.72		0.03 – 0.175	50[A]	500
Nitrite	0.02 – 0.42	0.01 – 0.27	0.01 – 0.064	-		<0.01 – 0.045	3[A]	30

Blue text = exceeds the drinking water guideline; Purple text – exceeds the recreational water guideline
A = Australian Drinking Water Guidelines (NHMRC 2011 updated 2018), U = USEPA Regional Screening Levels for Tap Water (USEPA 2019)

Source: WRM (2020) and Jacobs (2020). Data is for 9 alluvium wells, 22 site and 19 regional fractured rock wells, 6 springs, 9 domestic bores and 33 surface water locations. Data for domestic bores is a selection from within the surrounding area where water quality data is available.

The main drainage catchments on the proposed Mine Site are ephemeral in nature and as such they depend on rainfall, with negligible baseflow from groundwater.

Hawkins Creek is expected to be sustained by groundwater baseflow but is best described as ephemeral to semi-perennial. Approximately 50% of this catchment has also been altered to support agricultural activities.

Other watercourses in the area include:

- Blackman's Gully (which flows intermittently into Lawsons Creek). This gully is expected to be intersected by the open cut pits and hence the upper catchment would be diverted. Blackmans Gully is characterised as a watercourse in a confined valley setting with occasional floodplain pockets.
- Price Creek which flows intermittently through the eastern side of the proposed Mine Site. It is understood that runoff from the upper catchment of this watercourse would be captured and stored for mine use. Price Creek is characterised as a watercourse in a confined valley setting with occasional floodplain pockets.
- Walkers Creek flows intermittently through the western side of the proposed Mine Site. Runoff from the upper catchment would be captured and stored for mine use. Walkers Creek is the site of the proposed TSF. The southern headwaters of the Walkers Creek system are characterised as being watercourses in a confined valley setting with occasional floodplain pockets, whilst the northern headwaters transition from a confined valley setting to a partially confined, low sinuosity, planform controlled system. Below the confluence of the headwaters, the Walkers Creek system transitions again into a low sinuosity, gravel bed watercourse in an alluvial valley setting.

Lawsons Creek has a catchment area of approximately 503 km^2 to the Cudgegong River confluence (near Mudgee). Where the creek flows into Mudgee, it is understood to be used for irrigation purposes.

Surface water quality has been evaluated through the collection of samples from 33 sampling locations (WRM, 2020). Monitoring has been undertaken since 2013.

In general, the data collected on existing water quality indicates the following.

- The water runoff from the upper catchment of the Hawkins and Lawsons Creeks both show elevated levels of nitrogen, phosphorus and electrical conductivity. This may be due to majority of the land in the area and upper catchment being used for agricultural purposes, which is noted to have altered the Lawson Creek catchment.
- The downstream water quality from the Mine Site has been shown to have elevated nitrogen, phosphorus and electrical conductivity.

Data relevant to the concentrations of metals in surface water are summarised in **Table 4.3**. Some groundwater (and potentially spring water) is reportedly used to supplement drinking water stored in rainwater tanks. Hence, the concentrations reported in groundwater (particularly the alluvial aquifer, springs and domestic bores) have been compared against drinking water guidelines from Australia (NHMRC 2011 updated 2018), WHO (WHO 2017) and US (USEPA 2019). It is more likely that the community may have more incidental contact with groundwater

and surface water during use for irrigation or stock watering, or recreational use of creeks. Hence, the concentrations reported have also been compared against recreational water guidelines, which are set to be 10 times higher than drinking water guidelines as outlined in NHMRC (NHMRC 2008) guidance for recreational water quality. Recreational water guidelines are only provided for metals where there are health-based criteria. It is not relevant to adjust criteria based on aesthetics considerations (such as taste or impacts on infrastructure).

Concentrations that exceed either the health-based drinking water or recreational water guidelines are highlighted in **Table 4.3**, in blue (drinking water) and purple (recreational water). In general, for the alluvium, springs and domestic bores which are more likely to be used for drinking water, there are exceedances of drinking water guidelines for cadmium, cobalt, lead, lithium, nickel, and manganese.

In relation to recreational water quality there are exceedances in groundwater (all aquifers) recorded within bores located within the Mine Site for arsenic, cobalt, lithium, manganese and nickel (particularly in relation to groundwater in the deeper fractured rock aquifers). There are no exceedances of recreational water guidelines for any of the metals reported within the regional groundwater monitoring bores.

4.6 TANK WATER

The occupants in residences, including within Lue, surrounding the Mine Site utilise rainwater tanks as the primary source of potable water, for drinking and other household uses. It is understood that water from rainwater tanks is supplemented with groundwater or water trucked in from Mudgee, when necessary.

A rainwater tank sampling program was undertaken in 2012 by JBS on behalf of Bowdens Kingsgate Pty Ltd to evaluate concentrations of metals that may be present in the tanks, dissolved in the water or in sediments within the tank. Metals may be present in the tanks as a result of dust deposition onto the roof, with this dust then washed into the tank with rainwater. Some of the metals may dissolve and others may remain bound to the dust particles and remain as sediment within the tanks.

The type and condition of roof materials can also influence rainwater quality. The materials observed in the properties evaluated in the local area were galvanised iron, colorbond, zincalume, ceramic tiles and slate. The products more likely to affect water quality are galvanised iron, colorbond and zincalume, with zinc (and cadmium and aluminium, also present in zinc products) commonly present in rainwater tanks from such materials. Most roof materials observed in the properties evaluated in the rainwater tank study were galvanised iron, colorbond or zincalume. Other materials on roofs, such as flashing (zinc, bitumen, aluminium and lead – no longer used), other metals and nylons in bolts and washers, and guttering (where older systems included lead solder) may enter rainwater.

The type of rainwater tank can also affect water quality. Tanks constructed of sheet metal can result in metals being introduced into the water. Other tanks, constructed of poly, fibreglass and concrete do not result in metals being introduced into the rainwater. It is noted that concrete tanks, however, can result in higher pH levels.

Organic matter that may enter rainwater tanks (from adjacent vegetation) may absorb some of the dissolved metals, however the pH may also be lowered (from organic acids) which would mobilise (dissolve) more metals.

The rainwater tank sampling program involved 84 tanks that were between 0 km and 5.85 km from the proposed Mine Site (refer to **Figure 4.5**). The sampling conducted involved the collection of:

- a preliminary water sample (prior to cleaning and sediment sampling) from the tank or outlet;
- cleaning of the tank to enable sediments to be sampled; and
- sampling of tank water post cleaning (at some locations).

Rainwater quality in the area is characterised by low levels of salts with electrical conductivity, EC, around 27 µS/cm) and a slightly low pH (average of 6.52). Where bore water or Mudgee water is used to supplement rainwater the EC is higher (higher than 200 µS/cm).

Table 4.4 presents a summary of metals reported in rainwater tanks, as dissolved concentrations in water. The data is presented for the different types of tanks included in this study. All but one of the properties evaluated had metal roof materials. One property (with a poly tank) had a tiled roof. **Table 4.5** presents a summary of the concentrations reported in sediments from these tanks.

Table 4.4
Metals in Rainwater Tanks (Water)

Metal or Indicator	Concentration in rainwater tanks, by tank type (mg/L)					Drinking Water Guideline (mg/L)
	Galvanised Iron	Concrete	PVC or poly	Fibreglass	Average	
pH Value	4.5 - 8.1	5.7 – 8.2	3.9 – 7.7	4.6 – 7.5	--	6.5 – 8.5 for aesthetics (corrosion and taste).*
Arsenic	<0.001 - 0.053	<0.001 – 0.005	<0.001	<0.001	0.0033	0.01
Cadmium	<0.0001 - 0.0017	<0.0001 – 0.005	<0.0001 – 0.0019	<0.0001 – 0.0058	0.00065	0.002
Chromium	<0.001 - 0.013	<0.001 – 0.006	<0.001 – 0.006	<0.001	0.0015	0.05
Copper	<0.001 - 0.3	<0.001 – 0.593	<0.001 – 0.624	<0.001 – 0.436	0.065	2
Iron	<0.05 - 0.66	<0.05 – 1.08	<0.05 – 0.86	<0.05 – 0.06	0.23	0.3 for taste*
Lead	<0.001 - 0.015	<0.001 – 0.037	<0.001 – 0.035	<0.001 – 0.004	0.0059	0.01
Manganese	0.001 - 0.064	<0.001 – 0.061	<0.001 – 0.08	0.003 – 0.075	0.013	0.5
Mercury	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	0.001
Nickel	<0.001 - 0.029	<0.001 – 0.039	<0.001 – 0.05	<0.001	0.014	0.02
Zinc	0.038 – 6.52	0.027 – 2.89	0.053 – 1.51	0.034 – 4.19	0.89	3 for taste*

Notes:

Blue text – exceedance of health based drinking water guideline (cadmium = 4 exceedances, iron = 2 exceedances, lead = 2 exceedances and nickel = 3 exceedances)

* No health guideline available

The data in this table is for 42 samples collected from rainwater tanks prior to cleaning

Figure 4.5 Rainwater Tank Sample Locations

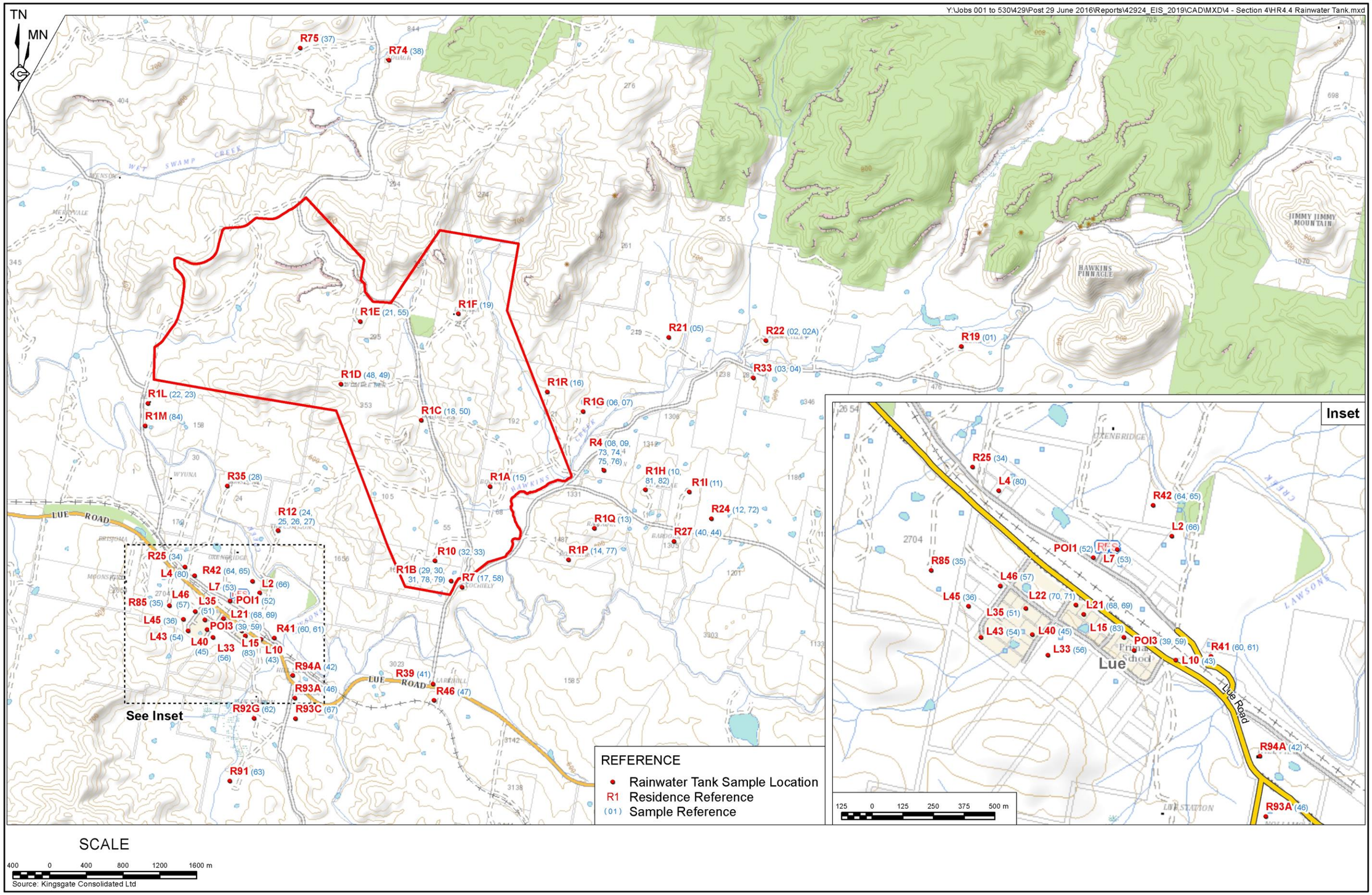


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Table 4.5
Metals in Sediments from Rainwater Tanks

Metal or Indicator	Concentration in Sediments in Tanks (mg/kg)			
	Galvanised Iron	Concrete	PVC or poly	Fibreglass
Arsenic	5 – 21 2450 at one property	5 – 23	6 – 156	65 – 57
Cadmium	<1 - 127	<1 – 93	<1 – 33	<1 – 43
Chromium	15 – 415	13 – 278	18 – 638	20 – 96
Copper	28 – 652 4140 at one property	8 – 368	48 – 740	34 – 251
Iron	16700 – 91200	3860 – 71900	8600 – 339000	19600 – 83100
Lead	59 – 2310	14 – 1810	52 – 4490	208 – 1890
Manganese	78 – 2530 17200 at one property	198 – 2390	91 – 2980	118 – 1830
Mercury	0.2 – 0.8	0.3 – 0.9	0.1 – 0.7 6.3 at one property	0.2 – 0.5
Nickel	10 – 64	3 – 35	6 – 86	10 – 15
Zinc	2430 - 33100	852 - 77600	344 - 23400	718 - 65300

As rainwater tanks are used for drinking water, the concentrations reported have been compared with current drinking water guidelines. Concentrations of arsenic, cadmium, lead and nickel exceed the drinking water guidelines in some tanks (as highlighted with blue text in **Table 4.4**).

There are no guidelines for sediments in rainwater tanks relevant to human health, hence no guidelines have been included. It is noted that these data indicate that there are a range of metals present in sediments where concentrations are quite elevated. In four tanks (at three separate properties) there are concentrations of arsenic, copper, manganese or mercury that are significantly higher than the range reported in other tanks. These anomalous data likely reflect specific building materials, and condition of these materials, on the roof or guttering at the specific property.

4.7 AIR QUALITY

The existing air quality within and surrounding the Mine Site has been described in the Air Quality Assessment (Ramboll, 2021).

The local area is a rural area, with local air quality considered to be good. Dust and particulates are present in air, from a wide range of sources including:

- Traffic on unsealed roads;
- Agricultural activities;
- Motor cycle activity (at the Louee Enduro and Motorcross complex); and
- Small-scale quarrying activity.

The area is well vegetated and there is generally little dust arising from the vegetated land surface.

When bushfires, or controlled burning occurs, these activities also add to particulates in air.

Existing air quality in the local area is currently monitored using an air quality monitoring network, as follows (and as shown on **Figure 4.6**):

- Measurement of fine particulates as PM_{10} and $PM_{2.5}$ from two locations:
  - BAM1 – located in the south-eastern corner of the Mine Site, measuring continuous PM_{10} only (since 2012);
  - BAM2 – located in Lue, measuring continuous PM_{10} and $PM_{2.5}$ (since 2013).

This data is of most relevance to the assessment of health as these particulates are small enough to penetrate into the lungs (refer to Section 5 for further discussion)

- Measurement of total suspended particulates (TSP), and the proportion of lead in the TSP from two locations operated on a one-in-six-day routine (excluding the period from November 2014 to October 2016):
  - BHV1 – located in the south-eastern corner of the Mine Site;
  - BHV2 – located in Lue.

The TSP data includes both coarse particles (that cannot penetrate into the lungs) and the fine particles and as such is not used in the assessment of health impacts. However, the data has been analysed for lead content and can be used as an indication of the likely content of lead in the smaller/fine particles.

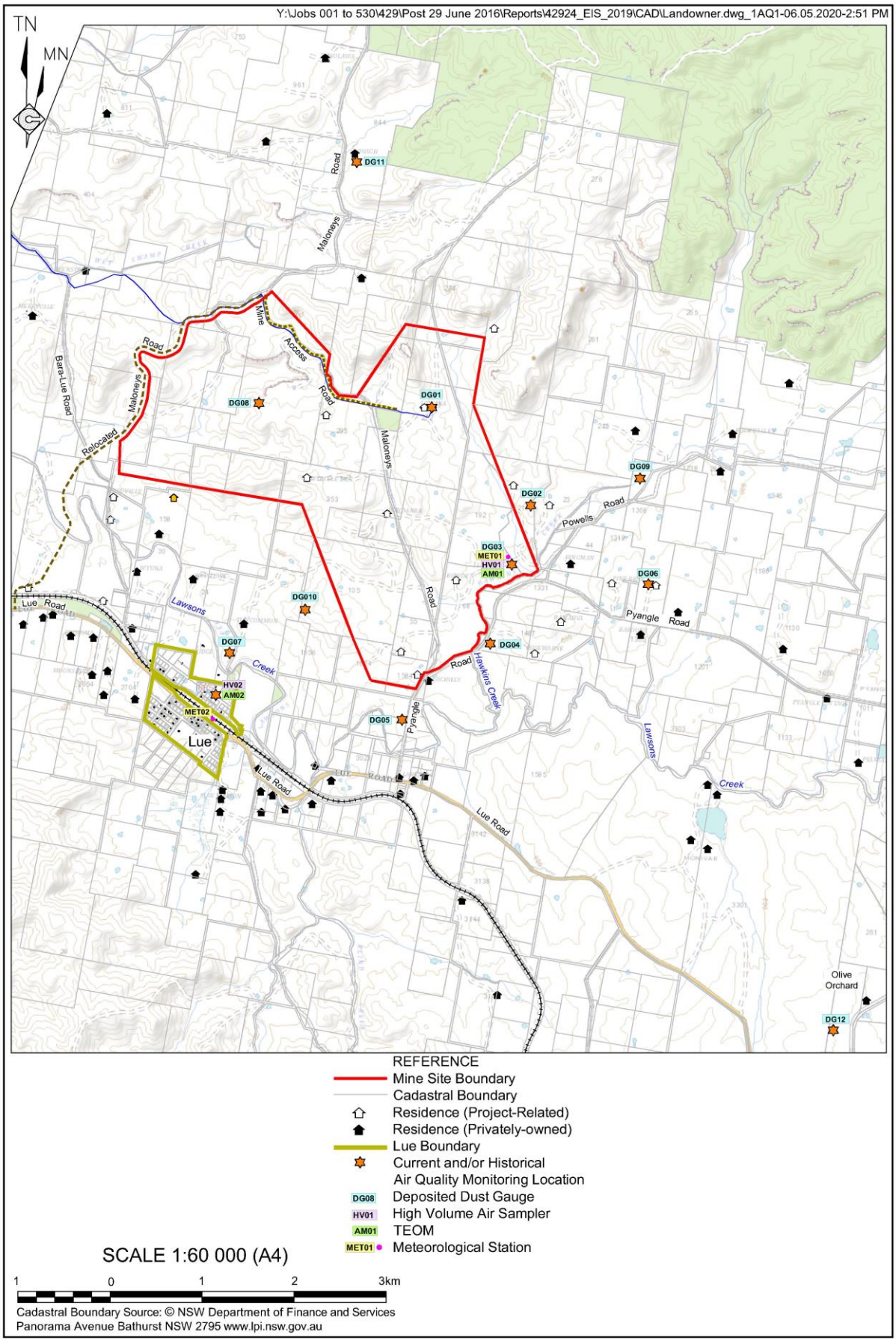
- Measurement of dust deposition on a monthly basis from 12 locations, with analysis of metals (namely arsenic, lead and zinc) content since 2012:
  - BDG1 to BDG12 – located at various locations surrounding the Mine Site. It is noted that sampling at BDG12 was discontinued in 2015.

Dust deposition measures the large dust particles that readily settle out of the air. These are too large to be of concern for inhalation exposures, however it reflects the amount and nature of dust that may deposit onto soil and other surfaces in the local area, where people may be exposed through direct contact or consumption of produce in these areas.

- Metrological data is collected from two weather stations:
  - BME1 – located in the southeastern corner of the Mine Site;
  - BME2 – located in Lue. The station was relocated in 2014 from a more sheltered site to a site approximately 350m to the south which provides better wind exposure.

Figures 4.7a and 4.7b presents a summary of the PM_{10} and $PM_{2.5}$ concentrations reported on a 24-hour average basis for 2017, the most complete monitoring period available. The 24-hour average levels vary throughout the year. With the exception of a dust storm event affecting 2 days of 2017, the levels reported were in compliance with the relevant regulatory guideline value (NEPC 2016).

Figure 4.6 Project Air Quality Monitoring Network



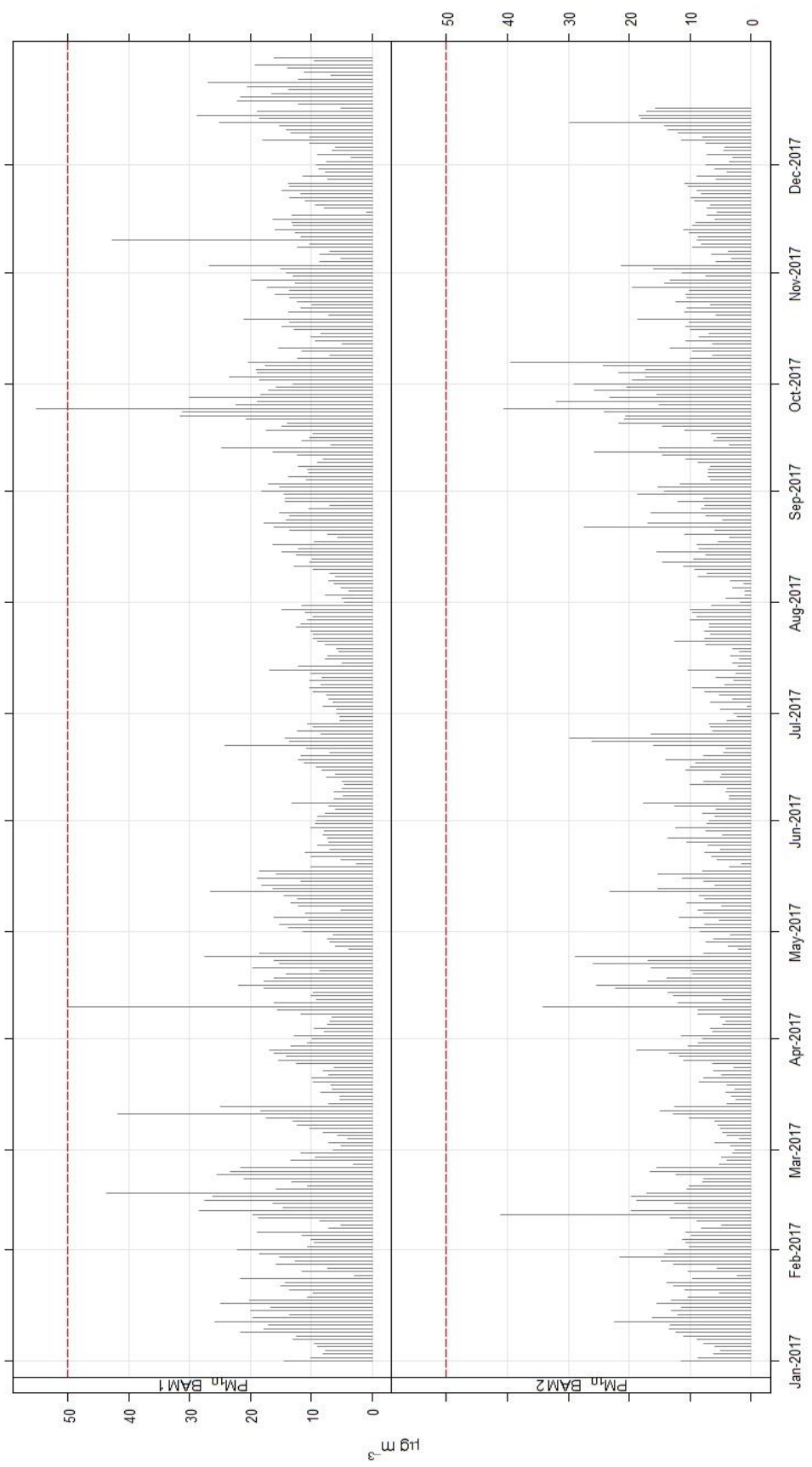


Figure 4.7a 24-hour average PM_{10} Concentrations for 2017 (from Ramboll, 2021)

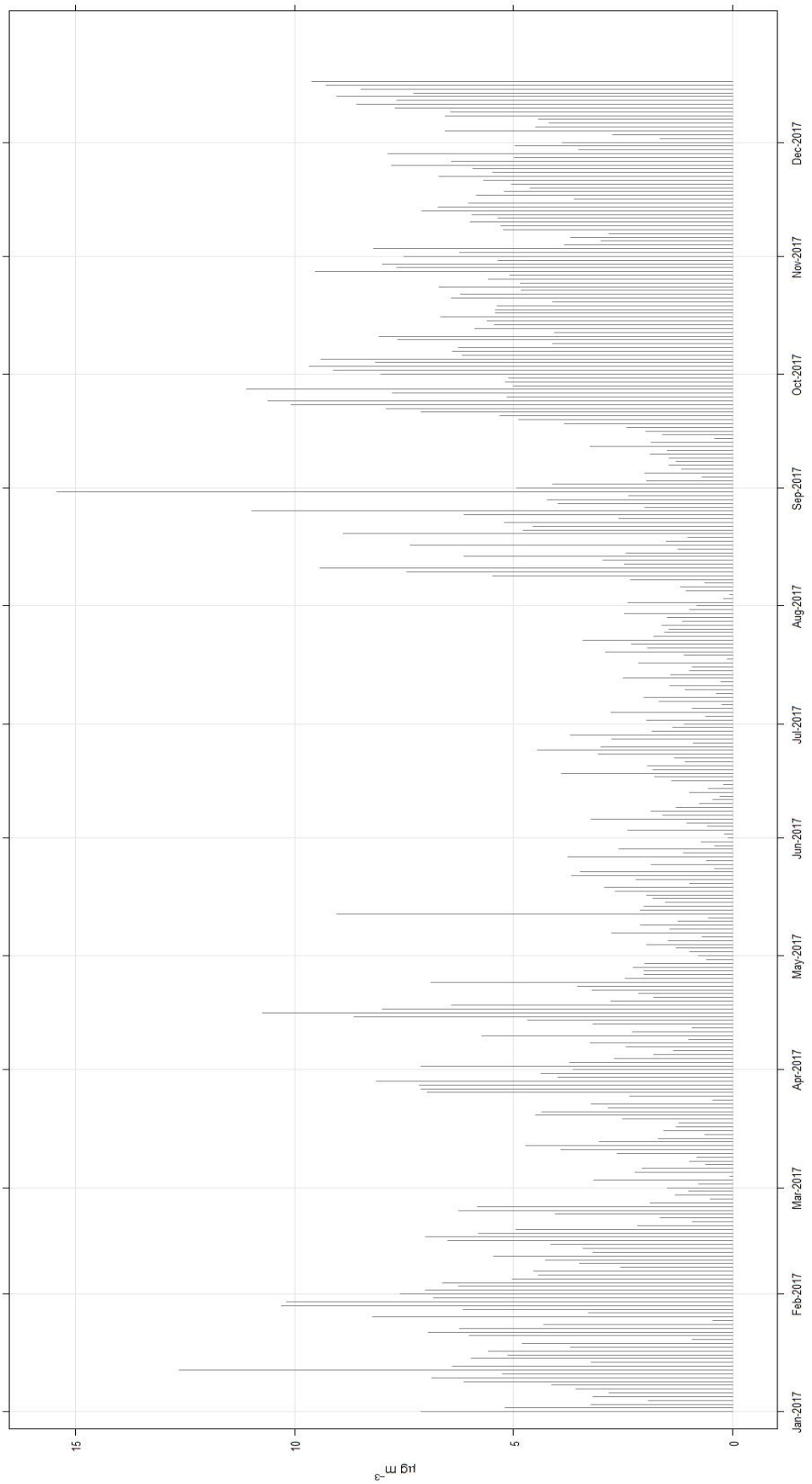
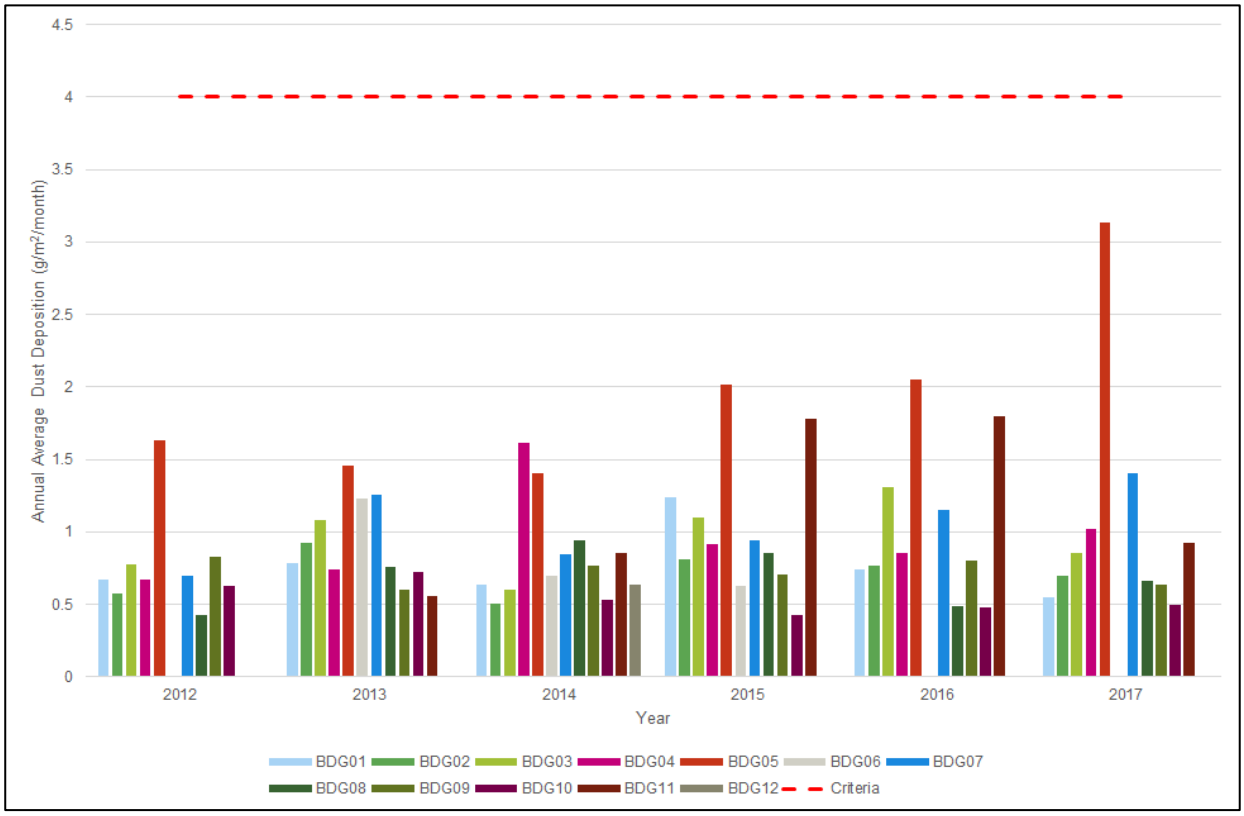


Figure 4.7b 24-hour average $PM_{2.5}$ Concentrations for 2017 (BAM2) (from Ramboll, 2021)

Figure 4.8 presents a summary of dust deposition levels at the various sampling locations from 2012 to 2017. These data indicate that existing levels of dust deposition are low, well below the relevant guideline. In terms of metal composition within the deposited dust, only lead, arsenic and zinc levels have been determined.

Figure 4.8 Annual Average Dust Deposition Monitoring – 2012-2017 (from Ramboll, 2021)



(Criteria adopted in figure as dashed line is the NSW EPA impact assessment criteria for TSP of 90 µg/m^3, refer to Ramboll (2021) for further detail)

In terms of the composition of dust reported as TSP and deposited dust shows that, on average, the deposition rate for arsenic, lead and zinc is 0.002 g/m^2/month, 0.001 g/m^2/month and 0.002 g/m^2/month respectively.

Further speciation of metals present in PM_{10} and $PM_{2.5}$ was undertaken from July 2017. Based on the analysis undertaken, the composition of metals in PM_{10} and $PM_{2.5}$ was determined (see **Table 4.6**).

Table 4.6
Composition of Metals in Fine Particulates

	Proportion of PM_{10} or $PM_{2.5}$	
Metal Reported	**PM_{10} (%)**	**$PM_{2.5}$ (%)**
Lead	0.01	0.026
Arsenic	0.013	0.06
Cadmium	0.0026	0.012
Copper	0.07	0.15
Zinc	0.084	0.18
Chromium	0.01	0.04
Source: Data provided by Bowdens on analysis of metals in PM_{10} and $PM_{2.5}$		

4.8 OTHER SOURCES OF EXPOSURE TO METALS

Community exposures to metals also occurs through the intake of produce. As metals are naturally occurring in the environment, produce purchased and consumed would have some level of metals present, reflecting the environment of origin for the produce. Food Standards Australia New Zealand (FSANZ) provides data on the levels of metals (and other chemicals such as pesticides) within food products consumed by the public. These intakes are from drinking water and other beverages (including alcohol, milk, formula etc) and commercially purchased foods (including cereals and grain based products, condiments, dairy products, fats and oils, fruit and nuts, meats, poultry and eggs, seafood, sugars and confectionery and vegetables).

In relation to the key metals expected to be evaluated in relation to impacts from the Project, the following present a summary of data available from FSANZ (FSANZ 2011) in relation to intakes from food/diet. **Table 4.7** presents the median intakes reported for children aged 2-5 years and adults 17 years and older, with the intakes presented as mg ingested/kg body weight/day, adopting the body weights referenced by FSANZ. It is noted that the dietary intakes evaluated by FSANZ also include drinking water. The table also includes these mean intakes as a percentage of the tolerable daily intake (TDI, where available) for metals adopted by FSANZ (FSANZ 2011)[3].

Table 4.7
Median Intakes of Metals from all Dietary Sources

	Intakes from all dietary sources (mg/kg/day) [% of TDI]	
Metal	**Children**	**Adults**
Lead	0.00027 [--]	0.00013 [--]
Arsenic	0.0014 [--]	0.00055 [--]
Cadmium	0.00032 [40%]	0.00011 [14%]
Copper	0.054 [96%*]	0.021 [15%*]
Manganese	0.15 [--]	0.063 [--]
Zinc	0.40 [100%*]	0.15 [30%*]
Cobalt	0.00078 [--]	0.00038 [--]
Mercury	0.00013 [3%]	0.000047 [1%]
Nickel	0.0046 [--]	0.0016 [--]

Source: FSANZ (2011)

-- No TDI adopted by FSANZ

* Based on an upper limit for nutrient intake as defined by FSANZ. No TDI has been determined by FSANZ for these metals.

[3] It is noted that the TDI or nutrient upper limits adopted by FSANZ may differ from the toxicity reference values adopted in this assessment for the characterisation of health effects relevant to exposure from all sources (refer to Section 5.2.2 for the further details on the approach adopted in this assessment).

4.9 EXISTING NOISE ENVIRONMENT

The existing noise environment within and surrounding the Mine Site has been described in the Noise and Vibration Assessment (SLR, 2020). This involved the monitoring of noise between 2011 and 2013 in Lue and the rural areas.

Background noise sources in the vicinity of the Mine Site are typical of a relatively undeveloped rural environment, with negligible industrial noise contributions, and a single moderately active road corridor, with noise sources that include:

- Traffic on Lue Road;
- Occasional light aircraft;
- Domestic and rural noise such as lawn mowers, tractors etc;
- Rural fauna noise such as stock, insects and birds; and
- Rural natural noise such as wind in the trees.

Measured background noise levels in the area from all sources as a $L_{Aeq(period)}$ range from 44 to 55 dB(a) during the day-time[4], 36 to 57 dB(A) during the evening[5], and 35 to 51 during the night-time[6].

Based on the monitoring of existing noise levels in the area, background noise levels (termed Rating Background Noise Levels or RBLs, which relates to noise over a 15-minute period) have been determined by SLR (2020) in accordance with the Noise Policy for Industry (NSW EPA 2017) to be 35 dB(A) during the day-time and 30 dB(A) during the evening and night-time periods (the minimum RBLs). These RBLs for the basis for determining project noise trigger levels for the assessment of noise impacts from the Project (refer to Section 7 for further detail on noise impacts from the Project).

[4] Day-time is Monday to Saturday 7am to 6pm and for Sunday and public holidays it is 8 am to 6 pm

[5] Evening is Monday to Sunday 6pm to 10pm

[6] Night-time is Monday to Saturday 10pm to 7am and for Sunday and public holidays it is 10 pm to 8 am

5. ASSESSMENT OF HEALTH: AIR QUALITY

5.1 INTRODUCTION

The assessment of potential health impacts associated with air emissions from the Project draws on the Air Quality Assessment prepared by Ramboll (2021). The Air Quality Assessment has considered all Project-related activities, including construction and operation, and provided modelled air concentrations and dust deposition throughout the surrounding community.

The Air Quality Assessment has addressed emissions to air that may occur during the following years, or scenarios:

- Scenario 1 - representative of the site establishment and construction stage where total waste rock is highest and the Stage 1 TSF embankment construction is undertaken;
- Scenario 2 - mining operations in operational Year 3, representing the year where total extracted material (ore and waste rock) is highest and the Stage 2 TSF embankment raise is undertaken;
- Scenario 3 - mining operations in operational Year 8, representing the year with the maximum extent of the southern barrier construction and the final (Stage 3) TSF embankment raise is undertaken; and
- Scenario 4 - representative of mining operations in operational Year 9, with the second highest year of waste rock extraction and when NAF waste rock transport to the TSF has ceased.

The assessment of air quality impacts relates to the activities relevant to the Project. These activities include:

- Dust emissions from all activities during construction and operations. The assessment of these emissions has considered TSP, PM_{10} and $PM_{2.5}$, as well as the composition of metals on these particulates based on elemental analysis of the waste materials, ore and soil (presented in Section 5.2);
- Emissions of fine particulates as PM_{10} and $PM_{2.5}$ from diesel combustion in mining equipment (included in the assessment presented in Section 5.2);
- Emissions of silica within $PM_{2.5}$, based on the silica (quartz) content of the Bowdens deposit (presented in Section 5.3); and
- Emissions of hydrogen cyanide from the volatilisation from the processing area and the active surface of the TSF (presented in Section 5.4).

Potential health impacts related to all these emissions have been addressed in this section.

The assessment of potential health impacts associated with exposure to these emissions from the Project has been undertaken on a quantitative basis. This has involved understanding how the community may be exposed to air emissions from the Project, as well as within the existing environment, and how toxic the various pollutants (at the predicted concentrations) are to humans.

It is noted that Project activities involve site establishment and construction works, open cut mining (including blasting), ore handling and processing and product transport. The processing operations include a jaw crusher, two mills and sequential floatation processes to produce silver/lead and zinc concentrates. Emissions to air that occur during all these processes are evaluated in the Air Quality Assessment (Ramboll, 2021).

Emissions to air from the Project differ significantly from those in other well-known lead mining operations such as Port Pirie, Broken Hill and Mt Isa. The key differences are described below.

- Broken Hill is a large mine site and Mt Isa is a large mine and smelting operation, both of which have been operating a long time. These have community (towns) located directly adjacent to the mine sites and smelting operations. The scale of the mines at Mt Isa and Broken Hill (significantly larger) and the proximity of the community to the operations is very different to the Project. The further the community is located away from the mining operations, the lower the exposure to dust generated from the activities undertaken. Communities such as Broken Hill and Mt Isa are situated close to the mine, with the town located on soil and rock that include naturally elevated levels of lead (similar to the ore body being mined). Historical operations at these sites have meant that there has been a long time where dust management measures and pollution control technology was not available or used. This means here has been a long history of dust deposition within the towns, and the communities are exposed to both naturally elevated levels of lead and historical deposition. Best practice dust management measures, consistent with current technology/methods and expectations would be used on the Bowdens Project, and the community is not located adjacent to the mine or in areas with naturally elevated levels of lead in soil.
- The inclusion of lead smelting at Port Pirie (which is only smelting operations) and Mt Isa – NOT present at the Bowdens Silver site. Port Pirie is one of world's largest lead smelting facilities, that has been operating for 130 years. The smelter at Mt Isa has been operating for 90 years. Emissions of lead from these smelters is the most significant source of lead exposures for the surrounding communities as smelting results in emissions of fine lead particulates from the smelter stack, which can then disperse throughout the community. These emissions, licenced through the South Australia EPA (Port Pirie) and the Queensland Department of Environment and Science (Mt Isa), are much more significant than those from the related open cut mining and crushing activities.

The major differences between the operations at Port Pirie, Broken Hill and Mount Isa, and the Project, detailed above, mean that the Project has no similarity to Port Pirie, Broken Hill or Mt Isa operations

The assessment of the Project has assessed all emissions that would be generated by all activities proposed within the Mine Site. This is specific to the Project and is very different to the operations at Port Pirie, Broken Hill or Mt Isa.

5.2 ASSESSMENT OF IMPACTS FROM DUST EMISSIONS

5.2.1 Dust Exposures

This Project is an open cut mine, where the most significant emissions to air relate to dust generated from activities that disturb soil and rock, and the pollutants that may be present on the dust.

In terms of community exposures to these emissions, the assessment addresses the inhalation of dust particles that are small enough to reach the lungs, namely PM_{10} and $PM_{2.5}$ (refer to Section 5.2.2). This assessment has considered potential health effects that are related to this particle size range only, as well as health effects related to the inhalation of various metals (present in the soil and rock) bound to these particles.

For this assessment, the metals evaluated are those modelled in the Air Quality Assessment based on elemental analysis of the ore to be mined, which are:

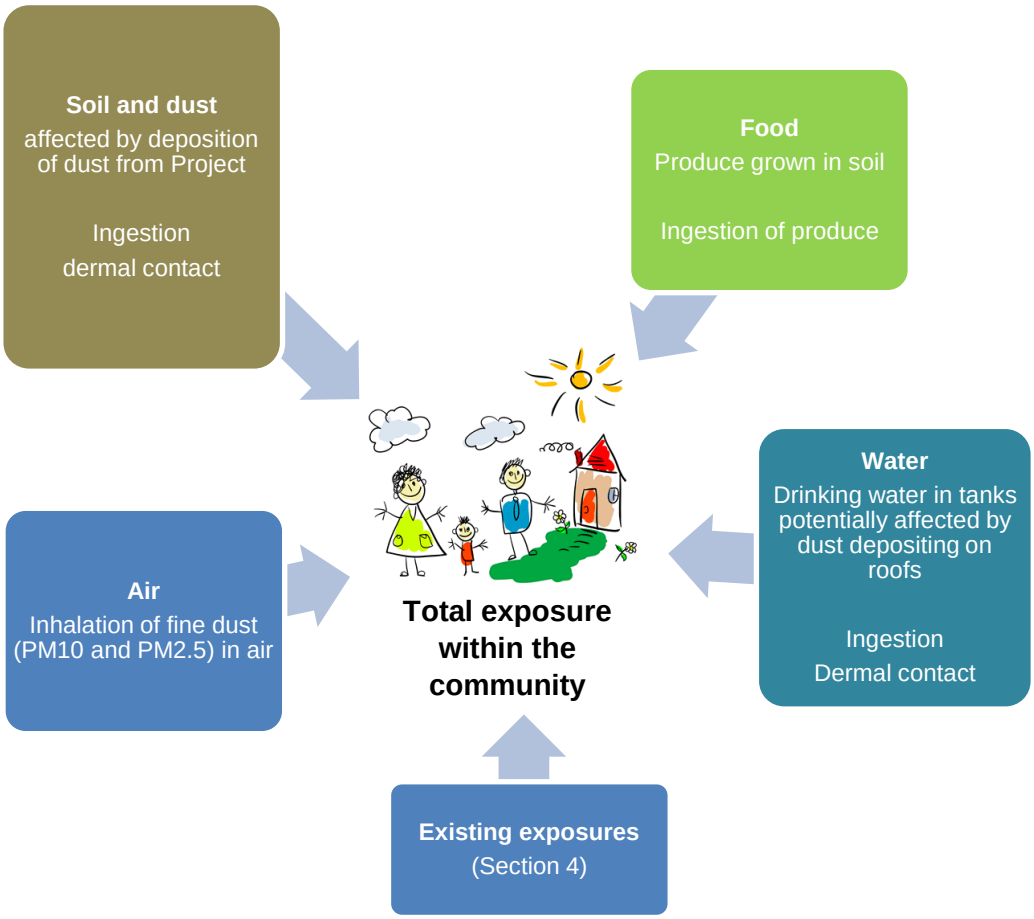
- Lead (Pb);
- Silver (Ag);
- Arsenic (As);
- Cadmium (Cd);
- Copper (Cu);
- Manganese (Mn);
- Zinc (Zn);
- Cobalt (Co);
- Chromium (Cr);
- Mercury (Hg);
- Lithium (Li); and
- Nickel (Ni).

In addition, the dust generated by the proposed activities may deposit onto the ground, where metals present in the dust may accumulate in topsoil, in household dust or be deposited onto a roof where it may then be washed into rainwater tanks. The community may then be exposed to these metals through direct contact with soil and dust on a property, and/or drinking rainwater. Once deposited to soil, any produce grown in the soil that is edible, such as homegrown fruit and vegetables, eggs from chickens, milk and meat, may accumulate these metals. The community may be exposed to these metals through the ingestion of this produce, with ingestion of homegrown produce of most significance. These pathways are collectively referred to as multi-pathway exposures.

Given the rural/agricultural nature of the areas surrounding the Project, inhalation and multi-pathway exposures have been evaluated in this assessment.

Figure 5.1 presents an overview of the exposures addressed in the assessment of dust emissions. This includes consideration of exposures to metals that occur in the existing environment, and then adding on additional exposures that may occur as a result of dust emissions from the Project.

Figure 5.1 Media and Pathways Evaluated for Assessing Community Exposures to Dust Emissions



5.2.2 Health Effects of Particulates

5.2.2.1 General

Particulate matter is a widespread air pollutant 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 less than 0.005 microns to greater than 100 microns. 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 secondary particulates include nitrogen oxides, ammonia, sulphur oxides, and certain organic gases (derived from vehicle exhaust, combustion sources, agricultural, industrial and biogenic emissions).

Numerous epidemiological studies[7] have reported significant positive associations between particulate air pollution and adverse health outcomes, particularly mortality as well as a range of adverse cardiovascular and respiratory effects.

The potential for particulate matter to result in adverse health effects is dependent on the size and composition of the particulate matter. 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[8] size below approximately 50 microns in diameter[9]. It is a fairly gross indicator of the presence of dust with a wide range of sizes. Larger particles (termed 'inspirable', comprising particles around 10 microns and larger) are more of a nuisance than a health hazard as they would deposit out of the air (measured as deposited dust) close to the source and, if inhaled, are mostly trapped in the upper respiratory system[10] and do not reach the lungs. Finer particles (smaller than 10 microns, termed 'respirable') 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 (see following point). Not all of the dust characterised as total suspended particulates is thus relevant for the assessment of health impacts, and TSP has not been further evaluated in this assessment. The assessment has only focused on particulates of a size where significant associations have been identified between exposure and adverse health effects.
- Fine particulates as PM_{10} (particulate matter below 10 microns in diameter, µm) and $PM_{2.5}$ (particulate matter below 2.5 µm in diameter) and ultrafines (particulate matter below 0.1 µm in diameter), as illustrated in **Figure 5.2**. These particles are small and have the potential to penetrate beyond the body's natural clearance mechanisms of cilia and mucous in the nose and upper respiratory system, with smaller particles able to further penetrate into the lower respiratory tract[11] and lungs. Once in the lungs, adverse health effects may result (OEHHA 2002). In relation to dust emissions from mining activities, these are predominantly from crustal materials and comprise PM_{10}, with a smaller fraction of $PM_{2.5}$ present.

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

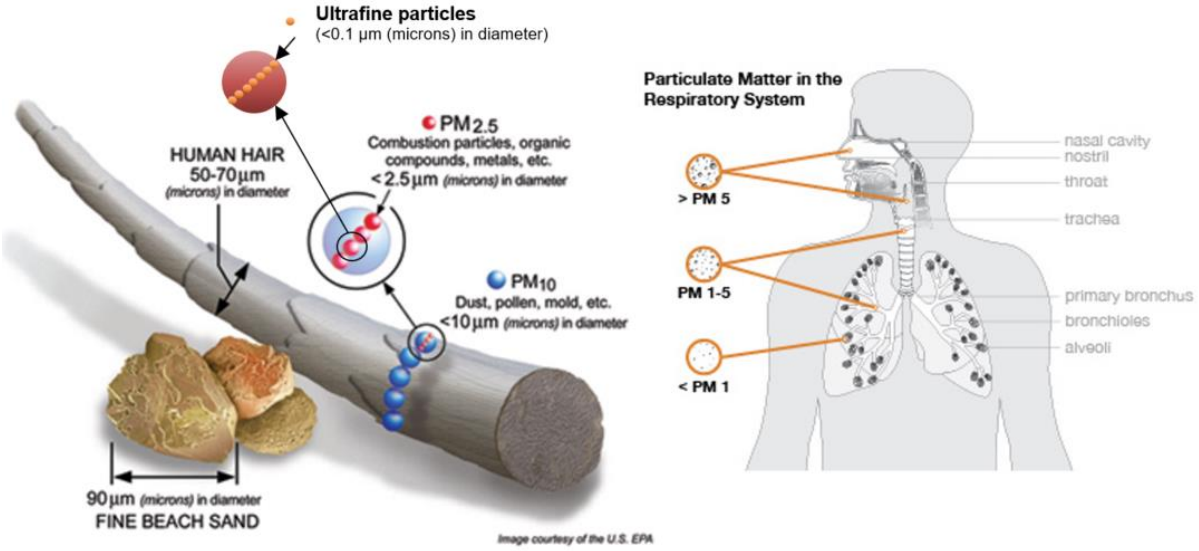
[8] The term equivalent aerodynamic particle is used to reference the particle to a particle of spherical shape and particle of density one gram per cubic metre.

[9] The size, diameter, of dust particles is measured in micrometers (microns).

[10] 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.

[11] 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.

Figure 5.2 Illustrative Representation of Particle Sizes and Penetration into the Lungs



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 particulate size fractions that tend to contain certain chemical components. Metals are commonly found attached to fine particulates (less than $PM_{2.5}$) while crustal materials (like soil) are usually larger and are present as PM_{10} or larger. In addition, different sources of particulates have the potential to result in the presence of other pollutants in addition to particulate matter. For example, combustion sources, result in the emission of particulate matter (more dominated by $PM_{2.5}$) as well as gaseous pollutants (such as nitrogen dioxide and carbon monoxide). This results in what is referred to as co-exposure and is an issue that has to be accounted for when evaluating studies that come from studying health effects in large populations exposed to pollution from many sources (as is the case in urban air).

Where co-exposure is accounted for the available science supports that exposure to fine particulate matter (less than 2.5 µm, $PM_{2.5}$) is associated (and shown to be causal in some cases) with health impacts in the community (USEPA 2012). A more limited body of evidence suggests an association between exposure to larger particles, PM_{10} and adverse health effects (USEPA 2009a, 2018; WHO 2003a).

5.2.2.2 Health Effects of Particle Size Only

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 or the shape of the particulate. 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.

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). A more limited body of evidence suggests an association between exposure to larger particles, PM_{10} and adverse health effects (USEPA 2009a; 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 linked to adverse health effects after both short term exposure (days to weeks) and long term exposure (months to years). The health effects associated with exposure to particulate matter vary widely (with the respiratory and cardiovascular systems most affected) and include mortality and morbidity effects. For particulates, no threshold has been established, hence for any change in exposure to $PM_{2.5}$, there is a change in health risk.

Annexure A presents further detail in relation to the health effects of particle size and the approach adopted for the characterisation of health effects relevant to these inhalation exposures. For this assessment, cumulative (i.e. exposures from all sources – existing and the Project) have been compared against the NEPM ambient air guidelines (NEPC 2016). The Air Quality Assessment (Ramboll, 2021) has presented an assessment of the Project on cumulative $PM_{2.5}$ and PM_{10} concentrations, with comparison against the NEPM air guidelines. Based on the assessment presented the following was determined:

PM_{10}:

There are no privately-owned residences where the cumulative concentrations of PM_{10} exceed the NEPM air guideline for an annual average, noting the maximum predicted is 16.9 µg/m³ which is well below the NEPM guideline of 25 µg/m³.

There are no privately-owned residences where the cumulative concentrations of PM_{10} exceed the NEPM air guideline for a 24-hour average, noting the maximum predicted is 48.1 µg/m³ which is below the NEPM guideline of 50 µg/m³.

In relation to potential impacts on health, the more important assessment relates to the sub-fraction of PM_{10}, which is $PM_{2.5}$ (refer to **Annexure A**), which are further evaluated below.

$PM_{2.5}$:

There are no privately-owned residences where the cumulative concentrations of $PM_{2.5}$ exceed the NEPM air guideline for an annual average, noting the maximum predicted concentration is 4.7 µg/m³ which is well below the NEPM guideline of 8 µg/m³ and the NEPM goal for 2025 of 7 µg/m³.

There are no privately-owned residences where the cumulative concentrations of $PM_{2.5}$ exceed the NEPM air guideline for a 24-hour average, noting the maximum predicted concentration is 16.2 µg/m³ which is well below the NEPM guideline of 25 µg/m³ and the NEPM goal for 2025 of 20 µg/m³.

Incremental risks

In addition, a calculation of incremental changes in $PM_{2.5}$ exposures from the Project alone has been undertaken, focusing on the key health endpoint, mortality (all causes). This health endpoint captures all other health effects found to be causally related to $PM_{2.5}$ exposure and is the most significant in terms of calculating risks related to changes in $PM_{2.5}$ exposures. **Annexure A** includes discussion on the methodology and calculations undertaken to determine an incremental risk. The maximum incremental risk for exposure to changes in $PM_{2.5}$ at the privately-owned residences is calculated to be 3×10^{-5}, which is lower than the risk level outlined in the NSW EPA Approved Methods (NSW EPA 2016) as unacceptable. Hence health impacts related to exposure to $PM_{2.5}$, based on the particle size alone are considered to be acceptable.

5.2.2.3 Health Effects of Metals on Particles

The assessment of exposures to metals that are bound to particulates has been undertaken on the basis of the toxicity of these metals, relevant to the exposures evaluated. Exposure to metals has the potential to result in a range of health effects, where exposures are sufficiently elevated.

For this assessment inhalation exposures have been evaluated on the basis of peak short-term or acute exposures as well as chronic or long-term exposures. Hence inhalation guidelines relevant to assessing acute exposures as 1-hour average, and chronic exposures as an annual average are relevant. In addition to inhalation exposures, multi-pathway exposures where ingestion and dermal contact with soil, produce and/or water may occur requires consideration of health effects related to ingestion and dermal absorption (where this is significant).

Annexure B presents detailed toxicity summaries for lead (a key metal of concern for some members of the Lue and district community), and **Annexure C** presents toxicity summaries for the other metals evaluated in this assessment.

Table 5.1 presents a summary of the acute inhalation guidelines adopted in this assessment. **Table 5.2** presents a summary of the chronic guidelines or toxicity reference values adopted for this assessment. These are guidelines that are considered to be protective of adverse health effects from exposure to these pollutants within the general population, including sensitive individuals.

For this assessment, the assessment of potential health effects or the toxicity of all the metals evaluated has been undertaken on the basis of threshold values. This means that for all the metals evaluated there is a threshold above which there is the potential for adverse health effects to occur. Where exposures are below these thresholds, no adverse health effects would occur.

Table 5.1
Summary of Acute Inhalation Guidelines

Metal	Acute Inhalation Guideline (mg/m^3)	Averaging Time	Source
Lead	0.15	1-hour	USEPA[1]
Silver	0.3	1-hour	USEPA[1]
Arsenic	0.003	1-hour	TCEQ[2]
Cadmium	0.0054	1-hour	TCEQ[2]
Copper	0.1	1-hour	OEHHA
Manganese	0.0091	1-hour	TCEQ[2]
Zinc	6	1-hour	USEPA[1]
Cobalt	0.00069	1-hour	TCEQ[2]
Chromium	0.0013	1-hour	TCEQ[2]
Mercury	0.0006	1-hour	OEHHA
Lithium	3.3	1-hour	USEPA[1]
Nickel	0.0011	1-hour	TCEQ[2]

Notes:
1 USEPA Protective Action Criteria (PAC), with level 1 protection defined as the concentration in air above which the general population, including susceptible individuals, could experience mild transient effects such as discomfort, irritation, or certain asymptomatic, non-sensory effects. These effects are reversible at the cessation of exposure, available from https://www.energy.gov/ehss/protective-action-criteria-pac-aegls-erpgs-teels-rev-29-chemicals-concern-may-2016
2 Acute inhalation Reference Exposure Values available from the Texas Commission on Environmental Quality, that provides detailed Development Support Documents for establishing air guidelines that are protective of community health. For metals these relate to arsenic (TCEQ 2012), cadmium (TCEQ 2016), manganese (TCEQ 2017b), cobalt (TCEQ 2017a), chromium as Cr VI (TCEQ 2014) and nickel (TCEQ 2011)
3 Acute Reference Exposure Levels from OEHHA https://oehha.ca.gov/air/general-info/oehha-acute-8-hour-and-chronic-reference-exposure-level-rel-summary

Table 5.2
Summary of Chronic Guidelines, Toxicity Reference Values (TRV) (Annual Average) and Dermal Absorption Parameters

Metal	Inhalation TRV (mg/m^3)	Ingestion TRV (mg/kg/day)	Dermal TRV[4] (mg/kg/day)	Dermal Absorption[3] – for contact with Soil (unites)	Dermal Permeability[3] - for contact with Water (cm/hr)
Lead[1]	Children = 0.002 Adults = 0.002	Children = 0.0014 Adults = 0.0006	Children = 0.0007 Adults = 0.0003	Negligible	0.0001
Silver[2]	0.02	0.0057	0.00023	Negligible	0.0006
Arsenic[2]	0.001	0.002	0.002	0.005	0.001
Cadmium[2]	0.000005	0.0008	0.0008	Negligible	0.001
Copper[2]	0.49	0.14	0.14	Negligible	0.001
Manganese[2]	0.00015	0.14	0.14	Negligible	0.001
Zinc[2]	1.75	0.5	0.5	0.001	0.0006
Cobalt[2]	0.0001	0.0014	0.0014	0.001	0.0004
Chromium[2]	0.0001	0.001	0.001	Negligible	0.002
Mercury[2]	0.0002	0.0006	0.00004	0.001	0.001
Lithium[2]	0.007	0.002	0.002	Negligible	0.001
Nickel[2]	0.00002	0.012	0.012	0.005	0.0002

Notes:
1 Refer to **Annexure B** for details in relation to the toxicity reference values adopted for the assessment of lead
2 Refer to **Annexure C** for details in relation to the toxicity reference values adopted for all other metals
3 Dermal parameters available from the Risk Assessment Information System https://rais.ornl.gov/
4 Dermal toxicity reference value adjusted by the gastrointestinal absorption, which is 50% for lead (refer to **Annexure B**), 4% for silver (refer to **Annexure C**) and 7% for inorganic mercury (refer to **Annexure C**)

5.2.2.4 Bioavailability of Lead

In relation to potential exposures to lead, the total bioavailability relates to the amount / proportion of lead that can move from the media being ingested into solution in either in the stomach or intestine and then how much lead in solution within the body can then get into the system circulation (i.e. be absorbed by the body such that the lead can get into the blood and then move into other systems in the body). For lead, this is of particular importance as the data used to develop a toxicity reference value is based on studies related to blood lead levels, not exposures from various media.

Total bioavailability = Bioaccessibility x Absorption

Bioaccessibility

Bioaccessibility is the proportion of lead present in the media that is ingested that can move into the gastrointestinal fluids. For most media ingested, such as water and food products the bioaccessibility is 100%. However, for soil, the bioaccessibility varies significantly between different sources of lead (including mineralologies) and soil types. Where no site-specific data is available, the default bioaccessibility value for soil is 100%. Lead in soil or rock from mine sites is considered to be less bioaccessible and hence site-specific bioaccessibility testing has been undertaken for the Mine Site.

Lead bioaccessibility testing for the Project has been undertaken by the University of South Australia using the Solubility Bioaccessibility Research Consortium (SBRC) method/assay, which is considered to be a suitable and reliable method (NEPC 1999 amended 2013c) on 14 soil samples. The samples selected for analysis are from the Mine Site and relate to different soil types within these areas, which are representative of materials to be disturbed during Project works. **Annexure D** presents a summary of the soil samples selected for bioaccessibility analysis (which is noted to cover a range of different materials in the Mine Site), a figure showing the location of these samples and the report issued by the University of South Australia, which presents the results of bioaccessibility testing.

Lead bioaccessibility reported in the samples analysed ranged from 14.6% to 53.8%, with an average of 33%. For this assessment, where oral exposures to lead in soil relate to emissions of dust to air from Project activities (where all different soil types and materials would be disturbed and contribute to these dust emissions) and the deposition of dust to soil and other surfaces, the average bioaccessible fraction of 33% has been adopted. This bioaccessibility value only relates the ingestion of soil or dust, not the ingestion of lead from any other media such as water or food products.

Absorption

Absorption relates to how much lead that is in solution in the gastrointestinal fluids is absorbed into the blood and circulated throughout the body. Sufficient data is available to support that absorption is 50% for children and 20% for adults (CRC CARE 2010; NEPC 1999 amended 2013e). For this assessment, 50% absorption has been adopted for the ingestion of lead vial all pathways for both children and adults. This is conservative.

5.2.3 Characterising Exposure

This task involves the quantification of the potential exposure pathways relevant to the surrounding community.

The exposure assessment is undertaken to be representative of a particular population, and does not calculate the exposure for a given individual. Populations are grouped so as to reflect common activities undertaken by that group (such as adults or children) or by the location of the population in relation to the contaminant distribution. For this reason, it is important that the exposure assessment be undertaken in such a way that the most sensitive individuals within the potentially exposed population are adequately protected.

When quantifying chemical intake or exposure to environmental contaminants, the risk assessment has primarily focused on exposure occurring over a prolonged period of years, and, possibly, a lifetime, i.e. a chronic exposure. Whilst an activity might occur infrequently (i.e., several days a year), it might occur regularly over a long period, and, therefore, have the potential to increase long-term or chronic exposures to the chemical. This assessment has also addressed acute inhalation exposures.

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

The exposure assessment involves the following.

- Identification of the population(s) that might be exposed – for this assessment, residents (adults and children) in the surrounding community areas have been addressed. **Figures 4.2** and **4.3** present the location of properties and the receptors evaluated within the community surrounding the Mine Site. The assessment of children in these areas also adequately assesses the children attending the Lue Public School;
- Identification of the activities by which exposure might take place for each population – for this assessment, the community comprises rural-residential areas where exposures may occur via:
  - Inhalation
  - Incidental ingestion and dermal contact with soil and dust
  - Ingestion and dermal contact with water from rainwater tanks
  - Ingestion of home-grown produce such a fruit and vegetables, eggs from chickens, meat and milk from livestock.
- Identification of parameters which define these activity exposure parameters (such as time spent at home) and physiological exposure parameters (such as body weight, inhalation rate and ingestion rates); and
- Identification of the chemical concentrations in air, soil, water and produce. This may include the identification and use of models to estimate chemical concentrations for receptors and exposure pathways that cannot be measured directly.

For this assessment, existing exposures to metals in the environment and exposures to metals in dust emissions from the Project have been evaluated as follows.

Existing Exposures

- Evaluated metal intakes in existing environment - exposures for adults and children assumed to be the same for all members of the community (regardless of where they live)
- Data on concentrations in air, soil, rainwater tanks and in the diet as summarised in Section 4

Exposure to Project Emissions

- Concentrations of metals in dust as $PM_{2.5}$ modelled in the Air Quality Assessment (Ramboll 2021) - used to calculate inhalation exposures
- Deposition of metals in dust modelled by Ramboll (2021) and used to assess exposures to metals in rainwater tanks, soil and in home-grown produce
- Exposures assessed for all privately-owned properties in the rural areas surrounding the Project, including Lue (**Figures 4.2** and **4.3**)

Annexure E presents the equations used to quantify exposures via inhalation, incidental soil ingestion and dermal contact, ingestion and dermal contact with water (from rainwater tanks) and the ingestion of home-grown produce. **Annexure E** also includes the assumptions adopted for characterising exposures for adults and children, and the methodology used to estimate concentrations in soil, rainwater tanks and produce.

5.2.4 Characterising Risks to Human Health

Risk characterisation is the final step in a quantitative risk assessment. It involves the incorporation of the exposure and toxicity assessment to provide a quantitative evaluation of risk.

Risks can be defined to be “acceptable” or tolerable if the exposed public could be expected to bear them without undue concern. Risks may be considered to be unacceptable if they exceed a specified regulatory limit, or if the circumstances are such that the risks cannot be accepted. Negligible risks are those that are so small that there is no cause for concern about them, or so unlikely that there is no reason to take action to reduce them.

Perceptions of risk are also important in determining whether risks from contamination in particular locations can be considered tolerable. The risks that tend to be of greatest concern are those that are involuntary (such as groundwater contamination), man-made and perceived as potentially catastrophic in their consequences.

While risk assessments can help to quantify levels of risk, and consider acceptable levels of risk outlined in the NEPM (NEPC 1999 amended 2013c), risk is usually an emotive issue and the level of perceived risk acceptable to the community may differ depending on the knowledge and lifestyle expectations of the community involved.

The process of risk assessment aims to assist risk managers in addressing the potential impact of a proposed development or an existing or possibly foreseeable future situation on the surrounding community and the communication of the potential risks.

The quantification of potential exposure and risks to human health associated with the emissions from the Project has been undertaken by comparing the estimated intake from existing exposures and exposures related to the Project (or exposure concentrations) with the threshold values adopted that represent a tolerable intake (or concentration). The calculated ratio is termed a Risk Index (RI), which is the sum of all ratios (termed Risk Quotients (RQ)) over all relevant pathways of exposure. These are calculated using the following equations:

Inhalation Exposures

$$\text{Risk Quotient(RQ)(existing)} = \frac{\text{Exposure Concentration (existing)}}{\text{(Inhalation toxicity reference value)}}$$

$$\text{Risk Quotient(RQ)(Project)} = \frac{\text{Exposure Concentration (Project emissions)}}{\text{(Inhalation toxicity reference value)}}$$

Oral and dermal exposures (calculated for exposures to soil, water and the ingestion of fruit and vegetables, eggs, meat and milk)

$$\text{Risk Quotient(RQ)(existing)} = \frac{\text{Daily Chemical Intake (existing)}}{\text{(Oral or dermal toxicity reference value)}}$$

$$\text{Risk Quotient(RQ)(Project)} = \frac{\text{Daily Chemical Intake (Project)}}{\text{(Oral or dermal toxicity reference value)}}$$

Total Risk

$$\text{Risk Index(RI)} = \sum_{\substack{\text{All pathways for existing} \\ \text{and Project exposures}}} \text{RQ}$$

The interpretation of an acceptable RI should recognise an inherent degree of conservatism that is built into the establishment of appropriate toxicity reference values adopted (using many uncertainty factors) and the exposure assessment. Hence, in reviewing and interpreting the calculated RI, the following is noted:

- A RI less than or equal to a value of 1 (where intake or exposure is less than or equal to the threshold) represents no cause for concern as outlined in NEPM guidance (NEPC 1999 amended 2013c);
- A RI greater than 1 requires further consideration within the context of the assessment undertaken, particularly with respect to the level of conservatism in the assumptions adopted for the quantification of exposure and the level of uncertainty within the toxicity (threshold) values adopted.

Annexure F presents all the calculations undertaken to evaluate existing exposures to metals in the Lue and district community.

Annexure G presents all the calculations undertaken to evaluate exposures to Project emissions for Scenarios 1 to 4.

5.2.5 Acute inhalation exposures

The calculated RI for acute inhalation exposures to the maximum 1-hour average concentration of metals attached to $PM_{2.5}$ predicted at all the modelled receptors and the privately-owned residences for each of the Project scenarios, are presented in **Table 5.3**. The table presents the total RI for exposure to all metals (as a sum) as well as the individual RI calculated for exposures to lead. The calculated RI relate to exposures by all members of the community, of all ages. The detailed calculations are presented in **Annexure G**.

Table 5.3
Calculated Risk Indices – Acute Inhalation Exposures to Metals in Air ($PM_{2.5}$) from the Project

Project Scenario	Calculated RI – Lead		Calculated RI – Total for all metals	
	Maximum of all Receptors (Project Related and Privately-owned Residences)	Maximum of all Privately-owned Residences	Maximum of all Receptors (Project Related and Privately-owned Residences)	Maximum of all Privately-owned Residences
Scenario 1 (SE&CS)	0.0014	0.00017	0.079	0.015
Scenario 2 (Year 3)	0.00051	0.00012	0.030	0.0084
Scenario 3 (Year 8)	0.00044	0.00013	0.027	0.0090
Scenario 4 (Year 9)	0.00047	0.00013	0.027	0.010
Acceptable RI	≤ 1	≤ 1	≤ 1	≤ 1
SE&CS = Site Establishment and Construction Stage				

Review of **Table 5.3** indicates that all calculated RI, related to acute exposures to all metals in dust (including lead, which comprises around 2% of the total RI), at all locations, including the Project-related properties, are below 1 and hence there are no acute inhalation exposure risks of concern for the Project.

It is noted that inhalation exposures at all other locations in the community, including Lue Public School are lower than those presented in **Table 5.3**, and are therefore also not considered to be of concern.

5.2.6 Chronic Exposures

5.2.6.1 Existing Exposures

The community may be exposed to existing metals within the community as a result of the inhalation of dust in air, ingestion of drinking water and dermal contact with water from rainwater tanks, ingestion and dermal contact with soil, and the ingestion of food products. The assessment of these existing exposures has been undertaken based on the available data presented in Section 4. These are exposures that occur regardless of the operation of the

Project. When assessing the potential impact of the Project, these existing exposures are an important consideration as the assessment of potential risks relates to all exposures (existing plus the Project) to these metals.

As described in Section 4, this "existing exposures" assessment has considered the following sources of metals.

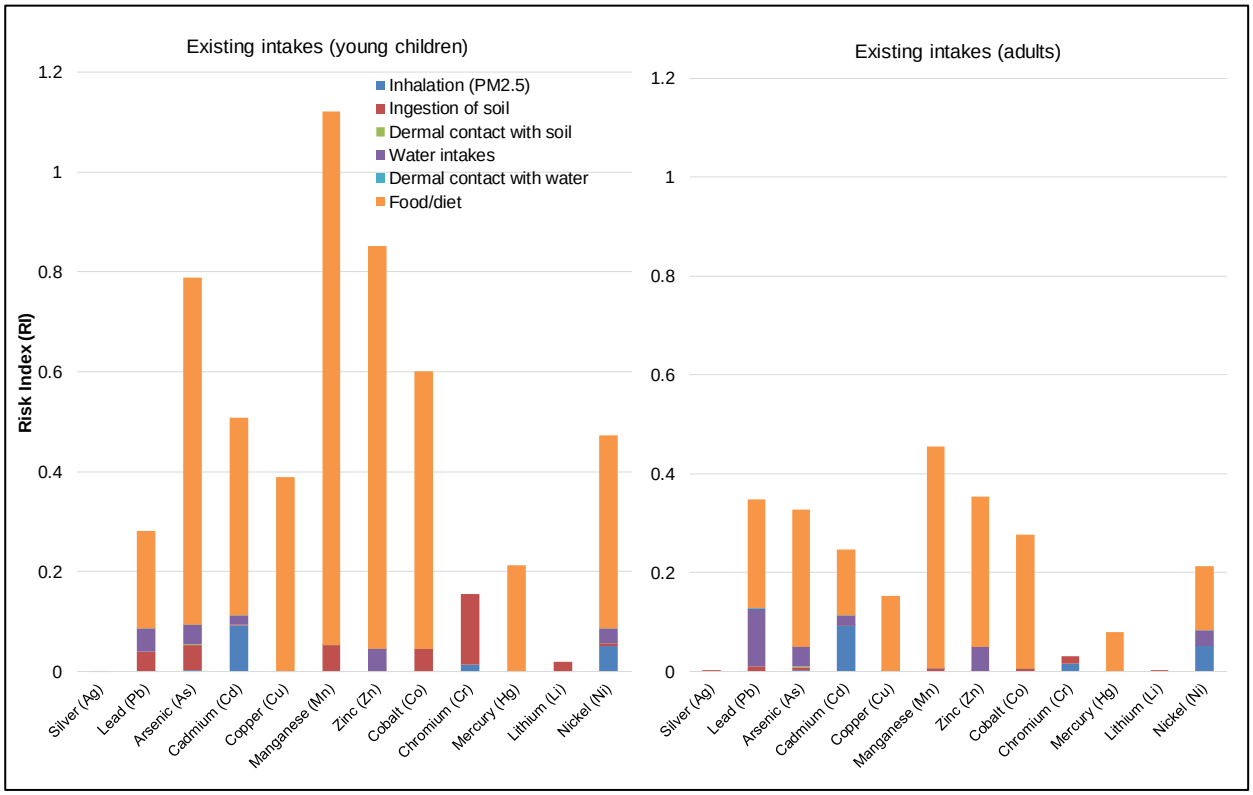
- Food (using the FSANZ food surveys which include exposure via consumption of drinking water as one of the types of food people consume).
- Rainwater tanks (corrected where needed for the exposure already included in the FSANZ food surveys from consumption of drinking water across Australia).
- Inhalation of dust.
- Direct contact with soil.

The calculated RI associated with existing exposures are presented in **Annexure F**. **Table 5.4** and **Figure 5.3** presents a summary of the calculated RI's for each metal for young children and adults. **Figure 5.3** also illustrates the contribution of each exposure pathway to the total RI calculated.

Table 5.4
RI for Existing Exposures to Metals in the Environment (i.e. no Project)

Metal	Calculated RI	
	Young Children	**Adults**
Lead	0.28	0.35
Silver	0.00058	0.000063
Arsenic	0.79	0.33
Cadmium	0.51	0.25
Copper	0.39	0.15
Manganese	1.1	0.45
Zinc	0.85	0.35
Cobalt	0.6	0.28
Chromium	0.15	0.03
Mercury	0.21	0.079
Lithium	0.019	0.0020
Nickel	0.47	0.21
Acceptable RI	≤ 1	≤ 1

Figure 5.3 Calculated RI for Existing Exposures to Metals in the Environment



Review of **Table 5.4** and **Figure 5.3** indicates the following.

- Existing exposures to metals such as silver and lithium are low, mainly due to the lack of available data on the presence of these metals in air and dietary sources.
- Existing exposures to the more abundant metals in the environment account for between 15% and 45% of the acceptable/tolerable daily intake (RI between 0.15 and 0.45) for adults.
- Existing exposures to metals in the environment for children are similar and generally higher than for adults, mainly as a result of a greater intake (per unit body weight) of these metals from dietary/food sources.

This is of particular relevance to manganese, where dietary intakes are significant and account for all of the tolerable daily intake. It is noted that intakes of metals from dietary sources are based on the available data from surveys completed by FSANZ, most of which are more than 10 years old. For manganese, the key sources in the diet are cereal products including breads, and vegetables which comprise a significant part of the diet for young children. The data is only representative of potential dietary intakes and it is noted that while an acceptable/tolerable daily intake has been adopted in this assessment for manganese, FSANZ has indicated that no upper limit for manganese intakes has been determined. Hence the assessment of manganese in this assessment is expected to be conservative.

5.2.6.2 Inhalation Exposures from Project Emissions

The maximum calculated RI for chronic inhalation exposures of metals attached to $PM_{2.5}$ predicted at all the modelled receptors and the privately-owned residences for each of the Project scenarios, are presented in **Table 5.5**. The table presents the total RI for exposure to all

metals (as a sum) as well as the individual RI calculated for exposures to lead. The calculated RI relates to exposures by all members of the community, of all ages. The detailed calculations are presented in **Annexure G**.

Table 5.5
Calculated Risk Indices – Chronic Inhalation Exposures to Metals in Air ($PM_{2.5}$) from the Project

Project Scenario	Calculated RI – Lead		Calculated RI – Total for all metals	
	Maximum of all Receptors (Project related and Privately-owned Residences)	Maximum of all Privately-owned Residences	Maximum of all Receptors (Project related and Privately-owned Residences)	Maximum of all Privately-owned Residences
Scenario 1 (SE&CS)	0.0036	0.00013	0.16	0.0086
Scenario 2 (Year 3)	0.0036	0.00017	0.25	0.015
Scenario 3 (Year 8)	0.0030	0.00010	0.14	0.0085
Scenario 4 (Year 9)	0.0029	0.00010	0.14	0.011
Acceptable RI	≤ 1	≤ 1	≤ 1	≤ 1
SE&CS = Site Establishment and Construction Stage				

Review of **Table 5.5** indicates that all calculated RI, related to chronic inhalation exposures to all metals (including lead which comprises around 2% of the total RI) in dust emitted to air from the Project, at all locations including the Project-owned properties, are below 1. The largest contributor to the calculated total RI is manganese (at least 90% of the total RI). This indicates that the incremental increase in exposure to metals from the inhalation of dust generated from the mine is very low and would be considered negligible.

It is noted that inhalation exposures at all other locations in the community, including Lue Public School are lower than presented in **Table 5.5** and are also considered to be negligible. For example, for Lue Public School the calculated maximum total RI is 0.005 from all years of the Project (refer to the detailed calculations in **Annexure G**), which is at least 10 fold lower than presented in **Table 5.5**.

It is noted that the NEPM (NEPC 2016) also sets a standard for lead in air, measured as TSP. The maximum modelled concentration of lead in air as TSP (at all receptors over all years modelled) is 0.059 $\mu g/m^3$ which is approximately 10 times lower than the NEPM guideline of 0.5 $\mu g/m^3$.

Further discussion on total exposures, from all pathways of exposure from the Project and existing exposures is presented below.

5.2.6.3 Multi-pathway Exposures from Project Emissions

The calculated RI for exposures to metals derived from the Project that may deposit onto soil and surfaces and result in exposure to soil, water in rainwater tanks, and produce that is homegrown at the maximum impacted privately-owned residence for each of the Project scenarios, are presented in **Tables 5.6** and **5.7** for young children and adults respectively. The table presents the total RI for exposure to all metals (as a sum) as well as the individual RI calculated for exposures to lead, relevant to exposures to young children and adults. The detailed calculations are presented in **Annexure G**.

Table 5.6
Calculated Risk Indices for Multi-pathway Exposures to Metal Deposited from the Project - Young Children

Project Scenario	RI Calculated for each Exposure Pathway – maximum Impacted Privately-owned Residence					
	Ingestion and Dermal Contact with Soil	Ingestion and Dermal Contact with Water in Rainwater Tanks	Ingestion of Homegrown Produce			
			Fruit and Vegetables	Eggs	Meat	Milk
Exposure to Lead in Dust Emissions						
Scenario 1 (SE&CS)	0.0026	0.016	0.0030	0.000003	0.000009	0.00032
Scenario 2 (Year 3)	0.0025	0.015	0.0028	0.000003	0.000009	0.00015
Scenario 3 (Year 8)	0.0029	0.017	0.0033	0.000004	0.000010	0.00018
Scenario 4 (Year 9)	0.0030	0.018	0.0034	0.000004	0.000010	0.00019
Exposure to All Metals in Dust Emissions						
Scenario 1 (SE&CS)	0.0071	0.025	0.0052	0.000012	0.00025	0.0038
Scenario 2 (Year 3)	0.0069	0.023	0.0049	0.000011	0.00023	0.0034
Scenario 3 (Year 8)	0.0081	0.027	0.0058	0.000013	0.00027	0.0041
Scenario 4 (Year 9)	0.0092	0.030	0.0064	0.000016	0.00031	0.0051
Acceptable RI	≤ 1	≤ 1	≤ 1	≤ 1	≤ 1	≤ 1
SE&CS = Site Establishment and Construction Stage						

Review of **Tables 5.6** and **5.7** indicates that all calculated RI, related to chronic exposures to all metals (including lead) that may be deposited to soil or other surfaces at privately-owned residences from dust emissions from the Project are all well below 1. This indicates that the incremental increase in exposure to metals via these multi-pathway exposures from dust generated from the Mine Site is very low and would be considered negligible. In relation to emissions that occur over the different scenarios, or years of operation, there is only a small difference between the calculate RIs for the scenarios, with Scenario 4 (Year 9) indicating a slightly higher level of exposure.

Table 5.7
Calculated Risk Indices for Multi-pathway Exposures to Metal Deposited from the Project – Adults

Project Scenario	RI Calculated for each Exposure Pathway - maximum Impacted Privately-owned Residence					
	Ingestion and Dermal Contact with Soil	Ingestion and Dermal Contact with Water in Rainwater Tanks	Ingestion of Homegrown Produce			
			Fruit and Vegetables	Eggs	Meat	Milk
Exposure to Lead in Dust Emissions						
Scenario 1 (SE&CS)	0.00065	0.039	0.0024	0.000004	0.000008	0.00010
Scenario 2 (Year 3)	0.00061	0.037	0.0022	0.000004	0.000008	0.00009
Scenario 3 (Year 8)	0.00072	0.043	0.0026	0.000004	0.000009	0.00011
Scenario 4 (Year 9)	0.00075	0.045	0.0027	0.000005	0.000010	0.00011
Exposure to All Metals in Dust Emissions						
Scenario 1 (SE&CS)	0.0012	0.049	0.0033	0.000008	0.00010	0.00098
Scenario 2 (Year 3)	0.0011	0.046	0.0032	0.000008	0.00010	0.00092
Scenario 3 (Year 8)	0.0013	0.054	0.0037	0.000009	0.00012	0.0011
Scenario 4 (Year 9)	0.0015	0.058	0.0041	0.000010	0.00013	0.0013
Acceptable RI	≤ 1	≤ 1	≤ 1	≤ 1	≤ 1	≤ 1

SE&CS = Site Establishment and Construction Stage

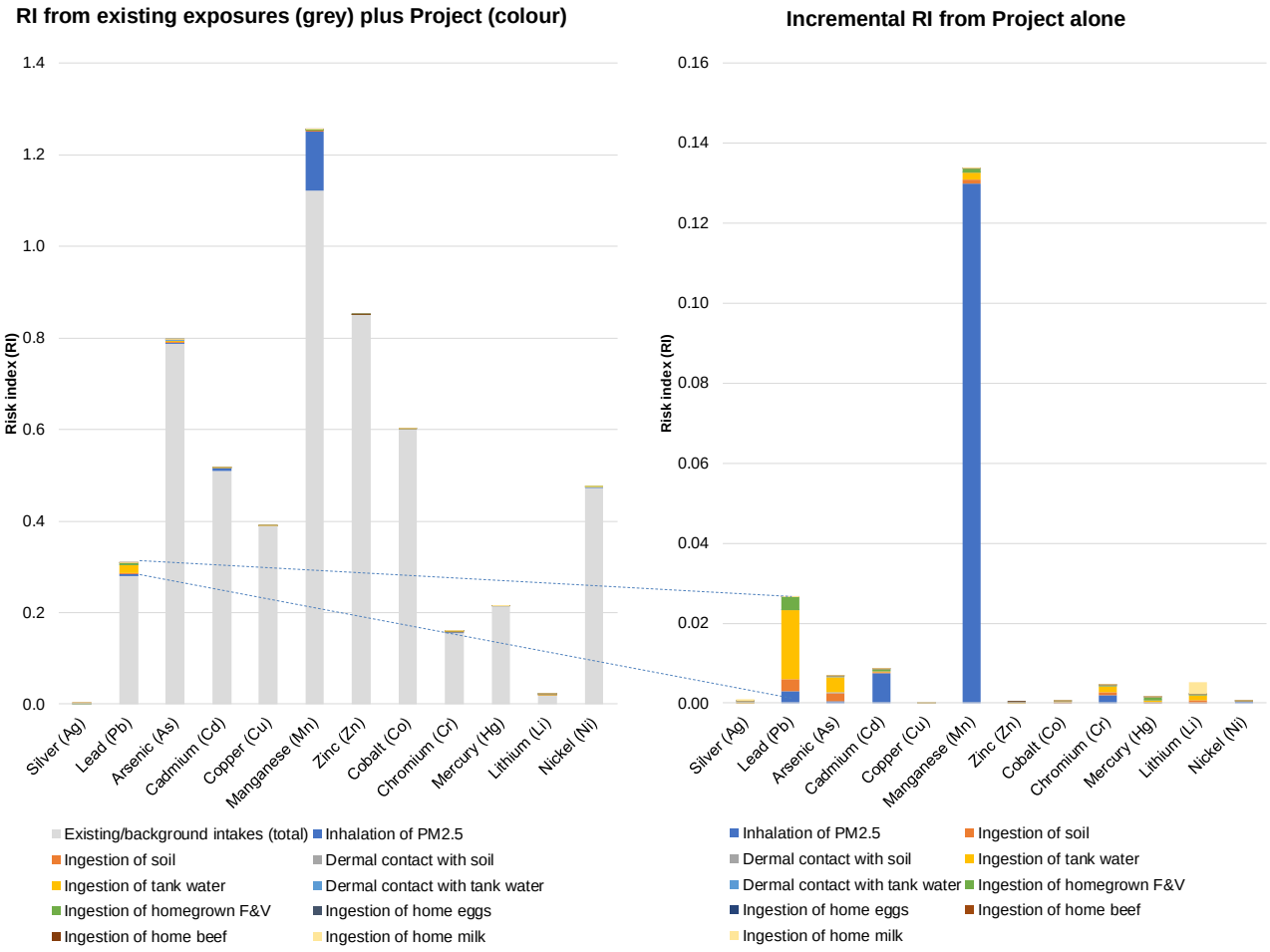
Further discussion on total exposures, from all pathways of exposure from the project and existing exposures is presented below.

5.2.6.4 Exposures from all sources including the Project

The assessment presented above has indicated that the calculated RI associated with chronic inhalation and multi-pathway exposures to metals emitted from the Project are very low and are considered negligible. When evaluating the risks related to exposures to metals, all sources of these metals needs to be considered. Where all sources are considered, exposures are dominated by existing exposures of metals, with Project-related emissions making a negligible change in the total calculated RI.

For the maximum impacted privately-owned residence, **Figures 5.4** and **5.5** present the calculated RI for each metal for young children and adults for Scenario 3 (Year 8). Emissions during Year 8 are similar to those in other years and therefore Year 8 is suitable to illustrate the contribution of the Project to the total RI. The figures show the total RI calculated, existing exposures plus all Project-related exposure pathways (assuming these all occur on the property, as well the incremental RI from the Project (for each pathway).

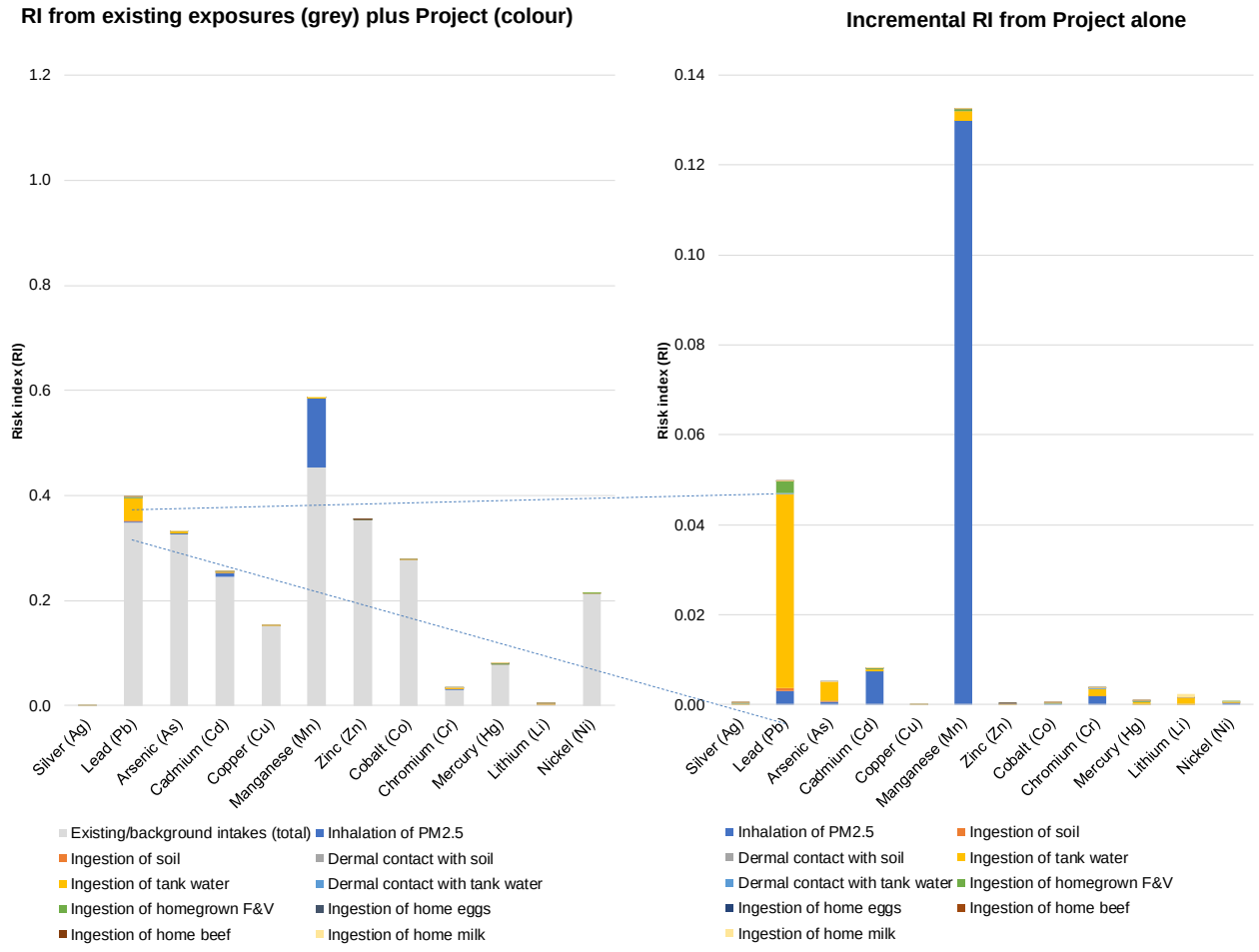
Figure 5.4 Calculated RI for Existing and Project Exposures (Scenario 3 – Year 8) – Young Children



Review of the calculations undertaken, as illustrated in **Figures 5.4** and **5.5**, indicate the following:

- For young children:
  - For manganese exposures where existing exposures are already elevated (due to dietary intakes), any incremental exposure from the Project (dominated by inhalation exposures) is low
  - For lead exposures, the Project contributes a small amount to the total RI. The total RI associated with existing and Project related exposures is below the target RI of 1
  - For exposure to all other metals, the Project contribution to the total RI is negligible and no total RI (existing plus Project) results in the RI exceeding the target of 1.

Figure 5.5 Calculated RI for Existing and Project Exposures (Scenario 3 – Year 8) – Adults



- For adults:
  - For lead exposures, the Project contributes a small amount to the total RI. The total RI associated with existing and Project related exposures is below the target RI of 1
  - For exposure to all other metals, the Project contribution to the total RI is small or negligible and no total RI (existing plus Project) results in the RI exceeding the target of 1.

Based on the above, which has focused on the maximum impacted private residential property, there are no health risk issues of concern in the community in relation to emissions of metals in dust from the Project.

The above provides the worst-case exposures for the properties located within the Lue and district community. All other exposures to Project-related emissions of dust are lower than presented above.

Figures 5.6 and **5.7** present the calculated RI for lead (the metal with the highest contribution from Project related emissions) for young children and adults for all privately-owned residences (refer to **Figures 4.2** and **4.3** for these locations). The figure presents the RI from existing exposures plus exposures from lead in dust from the Project for Scenario 3 - Year 8 (the representative year selected to present this data). The RI from exposure to emission from the Project is the sum of all exposure pathways evaluated. This is a worst-case as it assumes residents consume home-grown fruit and vegetables, eggs, beef and milk from the same property all of the time.

These figures show that the maximum impacted privately-owned residence from Project emissions is R4. The second highest impacts are at R21. The calculated RI from the Project at all other residences are much lower. All calculated RI for all locations are well below 1 and hence the contribution from the Project is negligible.

For Lue Public School (receptor 3 in **Figures 5.6** and **5.7**), the calculated RI for exposures by young children to lead from the Project via the inhalation of dust, ingestion and dermal contact with lead deposited to soil and dust on surfaces and lead that accumulates in rainwater tanks (assuming these are used to supply water at the school) is 0.002, which is significantly lower than presented for the maximum impacted location, 140 times lower than risks from existing exposures and 500 times lower than the acceptable RI of 1. These Project related impacts of lead in dust are considered negligible for the school.

The above further supports the conclusion that there are no risk issues of concern in relation to exposures in the community from Project related emissions of lead, and other metals in dust.

5.3 ASSESSMENT OF SILICA EXPOSURES

Respirable crystalline silica (or quartz) was one of the earliest recognised occupational hazards, particularly in quarries and mines. Studies have been carried out in a number of occupational groups, and have amassed ample evidence of respiratory effects of exposure. Specific health effects are related to repeated and prolonged workplace exposure (typically over many years) to concentrations of respirable crystalline silica. **Annexure C** presents a toxicity summary for respirable crystalline silica.

The Air Quality Assessment (Ramboll, 2021) evaluated potential emissions of crystalline silica as a result of Project operations where crustal materials are disturbed, and where these materials have the potential to comprise quartz. The percentage of quartz in ore and waste rock has been considered in the modelling of dust generated from the Project. The assessment presented has considered respirable crystalline silica as $PM_{2.5}$, with the maximum predicted concentration of silica (as $PM_{2.5}$) at all locations (Project related and privately-owned) predicted to be 0.76 µg/m³ and at all privately-owned residences predicted to be 0.21 µg/m³ (as an annual average).

In relation to non-occupational exposures, there are limited guidelines available. **Annexure C** indicates that EPA Victoria (EPA Victoria 2007) has established a guideline of 3 µg/m³ for non-occupational exposures to respirable crystalline silica (as $PM_{2.5}$, over an annual average). This is consistent to the public health guideline established by the California EPA Office for Environmental Health Hazard Assessment (OEHHA 2005), and slightly higher than the TCEQ long-term guideline of 2 µg/m³ (TCEQ 2009).

Figure 5.6 Calculated RI for Exposure to Lead at each Private Receptor / Residence – Young Children

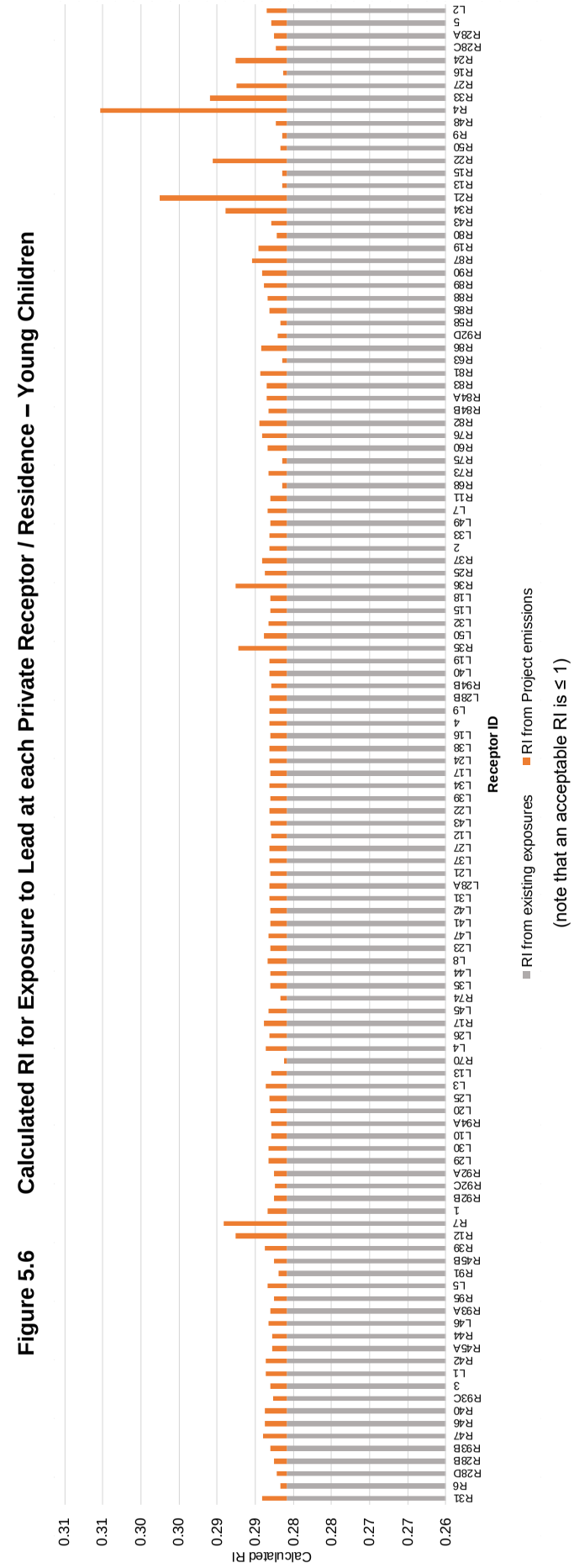
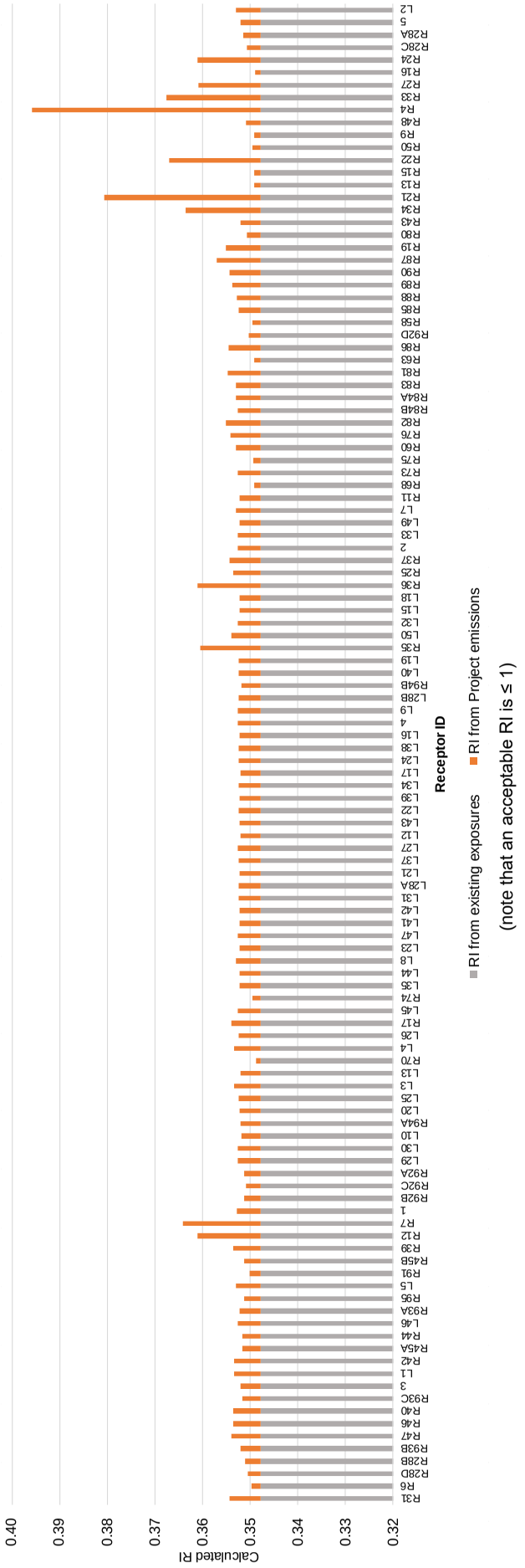


Figure 5.7 Calculated RI for Exposure to Lead at each Private Receptor/Residence – Adults



The maximum concentrations of crystalline silica derived from the Project, over all years, is lower than the available health-based guidelines. The maximum concentrations predicted in at privately-owned residences are also below the health-based guidelines. Exposures at other properties would be lower than these maximum concentrations.

On this basis, there are no health risk issues of concern in relation to community exposures to crystalline silica derived from Project operations.

5.4 ASSESSMENT OF HYDROGEN CYANIDE EXPOSURES

Sodium cyanide (NaCN) is proposed to be used as a zinc depressant in the processing plant. Once dissolved in water, the cyanide component takes a number of chemical forms, including hydrogen cyanide (HCN). A small proportion of this would volatilise (become gaseous) during the processing operation (estimates based on the National Pollutant Inventory suggest ~1% of the total cyanide). The remainder of cyanide that is lost from the process is contained within the tailings pumped to the TSF. Cyanide would be present in a number of forms including: strongly complexed forms (e.g. bound with iron); weakly complexed forms; and free cyanide (in the form of HCN or the free cyanide ion CN^-). The weakly complexed forms and free cyanide are often measured as weakly acid dissociable (WAD) cyanide.

The fate of cyanide within the TSF may follow several routes including volatilisation as HCN gas which is subsequently broken down through UV light (photolysis) or biological oxidation. Cyanide that is not volatised may also be broken down over time by biological processes (ultimately producing methane, ammonia and carbon dioxide) or form a stable complex which precipitates within the TSF sediments.

The Air Quality Assessment (Ramboll, 2021) evaluated potential emissions to air of gaseous hydrogen cyanide from Project operations. The Air Quality Assessment has predicted the maximum 1-hour average concentration of hydrogen cyanide in air as 5.9 µg/m³ at all properties (mine related and privately-owned) and 4.1 µg/m³ at all privately-owned residences.

Annexure C presents a toxicity summary for hydrogen cyanide. This review has identified that the most appropriate criteria for the assessment of acute or short duration exposures (as 1 hour average) is 340 µg/m³ established by Office of Environmental Health Hazard Assessment (OEHHA) (OEHHA 2008). The maximum concentrations of hydrogen cyanide predicted to be in air as a 1-hour average, are well below this health-based criteria.

On the basis of the above, there are no health risk issues of concern in relation to community exposures to hydrogen cyanide derived from Project operations.

5.5 UNCERTAINTIES

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

- Data;
- Receptor exposure assessment; and
- Toxicological assessment.

The risk assessment process following enHealth and NEPM guidance provides a systematic means for organising, analysing and presenting information on the nature and magnitude of risks to public health posed by chemical exposures. Despite the advanced state of the current risk assessment methodology, uncertainties and limitations are inherent in the risk assessment process. This section discusses the uncertainties and limitations associated with this risk assessment as well as the sensitivity of the calculated risk to variation in assumptions and inputs and the relative confidence and importance of potential variations.

Data

Existing rainwater data

The assessment presented in this report has relied on the available data on existing concentrations of dust and metals in the local area. Specifically, this data relates to the analysis of soil, water from rainwater tanks and air quality. This data has shown some variability in the concentrations reported, particularly in relation to the concentrations in rainwater tanks as these are influenced by the nature of roofing materials and tank construction. For this assessment, it is assumed that the average concentration in rainwater tanks is sufficiently representative of current exposures.

Rainwater tank samples have also been collected from 25 residences in Lue by Macquarie University in 2012. This data has not been published; however a summary of this data has been provided by Macquarie University for consideration as part of the Lue Action Group (LAG) submission. The concentrations considered in this assessment are within the range reported by Macquarie University in 2012. Hence this data does not significantly change the concentration adopted and the calculated existing intake of lead, and other metals from rainwater tanks in the community. The data provided by Macquarie University does include water concentrations of silver in rainwater tanks. This would increase the existing intakes of silver in the community from those considered in the assessment. Where the average silver concentration in existing rainwater tanks of 0.9 μg/L is adopted, the RI from existing exposures remains negligible at 0.004 for adults and children. The RI from Project emissions plus existing exposures remains negligible.

Existing soil data

Limited soil data is available for areas other than the mine site and surrounds due to access constraints. Soil samples collected from the mine site away from the proposed main open cut pit reported lead concentrations less than 50 mg/kg (refer to **Table 4.2**). Given this, and to be conservative, the HHRA has assumed an existing lead concentrations in soil of 50 mg/kg.

Additional data on lead in soil in the Lue Village and surrounding rural areas has been collected by Macquarie University in 2012. This data has not been published; however a summary of this data has been provided by Macquarie University for consideration as part of the LAG submission. The concentrations considered in this assessment are within the range reported by Macquarie University in 2012. Hence this data does not significantly change the concentration adopted and the calculated existing intake of lead, and other metals from soil in the community.

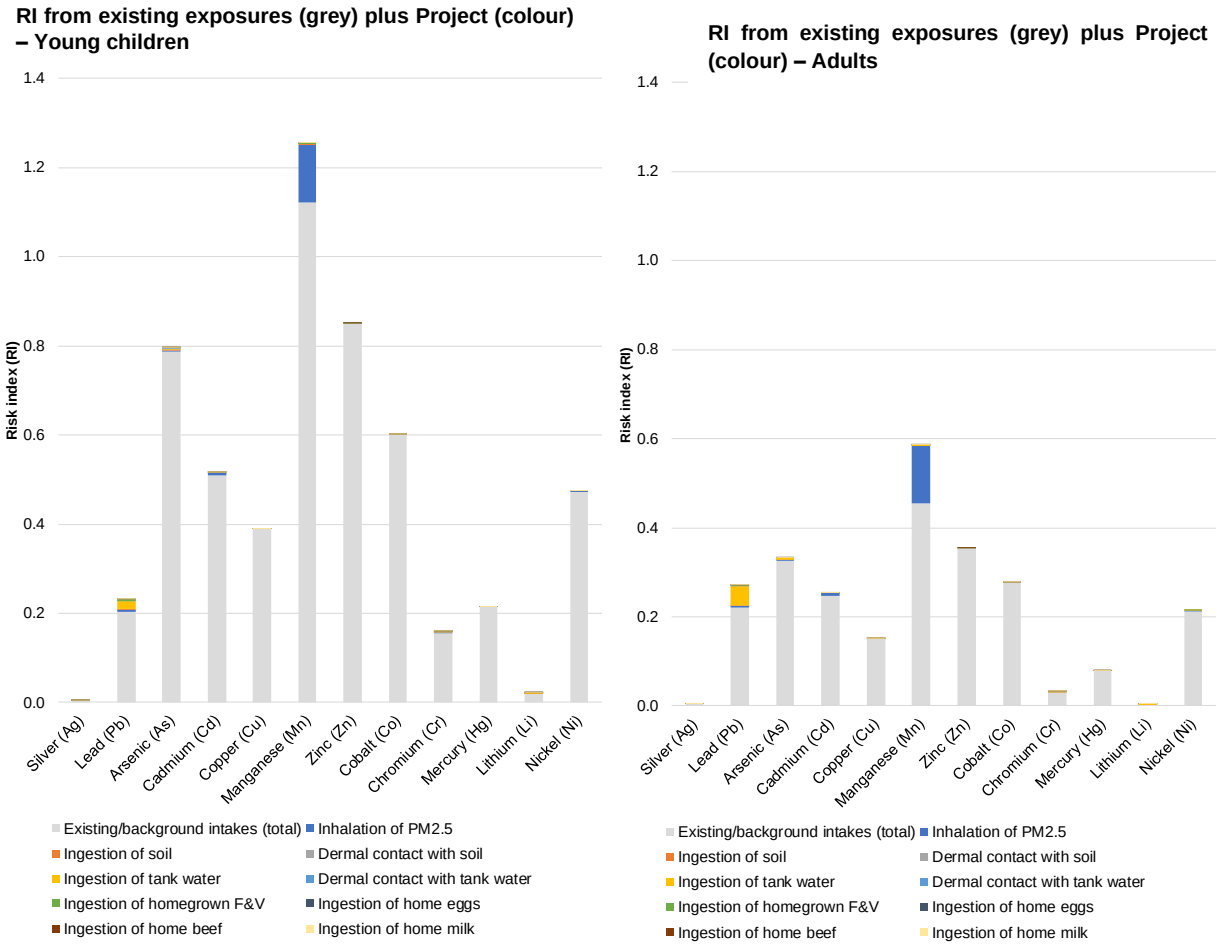
In relation to lead, a sensitivity calculation has been undertaken to illustrate the effect of adopting a lower background/existing soil concentration in the calculation of risk and the conclusions in relation to the contribution of Project emissions in the community. For the purpose of the sensitivity calculation, it has been assumed that the existing concentration of lead in soil in the

community is lower, at 13.3 mg/kg. This is the geometric mean of 34 soil samples collected in Lue and close rural areas and is not significantly different to the concentration reported in soil approximately 4 to 6 km away (range reported from 6 samples was 5.9 to 9.9 mg/kg). For this sensitivity calculation a lower concentration of lead in rainwater tanks of 0.0009 mg/L, which is the geometric mean of lead in rainwater tanks as measured by Macquarie University in 2012 (25 samples). These changes do not significantly change the total RI from existing exposures as these are dominated by dietary intakes (food), with the total RI for lead changing from 0.28 for children and 0.35 for adults to 0.20 for children and 0.22 for adults.

Figure 5.8 presents a graphical illustration of the revised total calculated RI for all metals (including silver, where a rainwater tank concentrations is included as discussed above) for young children and adults showing risks from existing exposures (using this revised data) and the contribution from the Project. These figures can be compared with the calculated RI presented in this report, in **Figures 5.4** and **5.5**.

Review of **Figure 5.8** indicates that use of alternate data for the assessment of existing exposures does not change the outcomes of the risk assessment and the contribution of the Project to total risks remains negligible.

Figure 5.8 Calculated RI for Existing and Project Exposures (Scenario 3 – Year 8) – Revised existing concentrations for lead in soil and rainwater tanks, and inclusion of silver in rainwater tanks



It is noted that the data from Lue Public School indicates the presence of lead paint. Exposures to lead from lead paint at the school have not been specifically considered in this assessment as the nature of these exposures is difficult to quantify. Exposures relate to the presence of lead paint at the school should be addressed by the school (and Department of Education) separately.

Air modelling

In relation to the assessment of impacts from the Project, this assessment has relied on the modelling of emissions as presented in the Air Quality Assessment (Ramboll, 2021). The Air Quality Assessment has also relied on data relevant to the characteristics of metals in soil and rock materials to be disturbed during operations, along with assumptions about the emissions during different activities. The modelling has incorporated a range of dust management measures (preventative measures). In addition, it is expected that further management measures (reactive and corrective measures) would be employed which would result in lower levels of dust emissions than evaluated. The modelling has also not accounted for rainfall, which would wash out some dust from the atmosphere and mitigate dust emissions. Review of this aspect in the Air Quality Assessment (Ramboll, 2021) indicates that during rainfall PM_{10} exposure concentrations are reduced by 20% to 50%. As a result, the predicted dust concentrations are expected to be an overestimate.

Exposure Assessment

Risk assessments require the adoption of several assumptions in order to assess potential human exposure. This risk assessment includes assumptions about general characteristics and patterns of human exposure relevant to the community. These assumptions are conservative and are developed to provide an estimate of maximum possible exposures rather than the actual exposures. This approach is expected to overestimate the risks.

Where possible, data that specifically relate to exposure have been used in this assessment. However, in some cases models have also been used to assist in the quantification of exposures for a number of exposure pathways where data are not available. This includes the modelling of metal concentrations in soil (from deposition) and the uptake of metals into home-grown produce (fruit and vegetables, eggs, meat and milk), and concentrations in rainwater tanks (washing off dust from roofs). The models used in this assessment are based on established multi-pathway exposure methods as detailed in **Annexure E**. These models have included conservative assumptions and are expected to overestimate actual concentrations. For the estimation of metal concentrations in rainwater tanks, which is a dominant exposure pathway, the model has not considered the use of any first-flush devices (which divert the first flush, or so, of rainwater from the roof such that it does not end up in the tank) which are commonly used to minimise the collection of dust and other materials (including bird droppings) into the rainwater tank. As a result, the concentrations predicted are conservative.

The assessment has only modelled the update of metals into beef. This has been undertaken as a representative meat product with soil and pasture intakes per unit body weight considered at the higher end of most stock likely to be present. These calculations are therefore considered representative and sufficiently protective of other meat products.

The assessment of consumption of home-grown produce has assumed as significant proportion of the diet for residents in the area comprises fruit and vegetables, eggs, meat and milk sourced from the one property. Inclusion of these intakes would result in some double counting of the intakes of metals from dietary sources as the assessment of existing intakes also include

produce where metals have been reported. It is difficult to adjust the dietary intake data from FSANZ, hence intake from fruit and vegetables, eggs, meat and milk would have been double counted, and resulted in a conservative assessment of total intakes. In relation to water intakes, the concentration of metals in drinking water, as assessed by FSANZ has been subtracted from the concentration reported in existing rainwater tanks (for the assessment of existing intakes only) to ensure intakes of metals from drinking water sources is not double counted.

It is noted that risks to human health associated with the predicted impacts from the Project are very low (considered negligible) and exposures (including concentrations) would need to increase by many orders of magnitude for risks to be considered significant. Hence there is no basis for undertaking any specific sensitivity analysis on the individual parameters chosen in these models as the variability in such an assessment would be very low.

Toxicity Assessment

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

This may introduce two types of uncertainties into the risk assessment, as follows:

- Those related to extrapolating from one species to another; and
- Those related to extrapolating from high exposure doses, usually used in experimental animal studies, to lower doses usually estimated for human exposure situations.

The majority of the toxicological knowledge of chemicals comes from experiments with laboratory animals, although there may be interspecies differences in chemical absorption, metabolism, excretion and toxic response. There may also be uncertainties concerning the relevance of animal studies using exposure routes that differ from human exposure routes. In addition, the frequent necessity to extrapolate results of short-term or subchronic animal studies to humans exposed over a lifetime has inherent uncertainty.

In order to adjust for these uncertainties, ADIs and RfDs incorporate safety factors that may vary from 10 to 1000.

The assessment undertaken, and the toxicity reference values adopted are considered current and sufficiently protective of adverse health effects for all members of the community including sensitive individuals.

5.6 OUTCOMES OF HEALTH RISK ASSESSMENT

Table 5.8 presents a summary of the outcomes of the assessment undertaken in relation to the impacts of changes in air quality, associated with the Project, on community health.

Table 5.8
Summary of Health Risks – Air Quality

Air Emissions	
Impacts	Based on the available data and information in relation to emissions to air from the Project, which include dust which comprises lead and a range of other metals, potential impacts on the health of the community have been assessed. The impact assessment has concluded that impacts derived from the Project make a negligible contribution to overall exposures to these metals and there are no health risk issues of concern relevant to the Project (including construction and operational phases). These conclusions apply to all members of the community, adults and children as well as sensitive individuals.
Mitigation	An Air Quality Management Plan is expected to be developed prior to commencement of operations at the Project that would outline the measures to manage air emissions (consistent with those considered and outlined in the Air Quality Impact Assessment, Ramboll 2021).

6. ASSESSMENT OF HEALTH: WATER QUALITY

6.1 APPROACH

Health impacts associated with potential impacts of the Project on water access and quality relevant to the local community have been evaluated on the basis of information provided in the following reports:

- Jacobs Group (Australia) Pty Ltd, 2020. Bowdens Silver Project, Groundwater Impact Assessment
- WRM Water & Environment Pty Ltd (WRM), 2020. Bowdens Silver Project, Surface Water Assessment.

The assessment undertaken in relation to water, has involved a qualitative review of the available information to determine if there is the potential for the Project to result in changes to surface water of groundwater quality or quantity, and where such changes may occur, if these may adversely affect the health of the community who may access and use these water resources.

6.2 PROJECT MANAGEMENT AND USE OF WATER

The water management system for the Project has been developed to manage potential impacts on surface water in the receiving environment within and around the Mine Site (WRM, 2020). The proposed system comprises distinct three distinct water management zones, the containment zone, erosion and sediment control (ESC) zone and clean water zone, as described below.

Containment Zone

Groundwater seepage and surface runoff from the open cut pit areas, the TSF, processing plant area, oxide ore stockpile and WRE are likely to have elevated dissolved metals levels. This water would be managed within a closed water management system.

Potentially-acid forming (PAF) waste rock would be placed within the WRE, and non-acid forming (NAF) waste rock would be placed over this material as part of a store-and-release capping layer with topsoil on the upper surface. The emplacement would be progressively revegetated/rehabilitated. Runoff from exposed rock within the WRE, as well as WRE leachate would be conveyed to a dedicated leachate management dam via a buried pipeline.

To minimise water accumulating within the leachate management dam, TSF and mine pit, water captured within the containment system would be the first priority water source for use in the processing plant, including dust suppression (WRM, 2020).

Erosion and Sediment Control (ESC) Zone

Runoff from disturbed areas outside the containment zone, including the southern barrier, which would be constructed using NAF waste rock, would be directed to sediment dams. This would include surface runoff from out-of-pit areas upslope of the southern barrier, which would be directed beneath the barrier itself.

The sediment dams would be sized and operated in accordance with requirements for Type D sediment basins. It is, therefore, anticipated that after the settlement of suspended sediment in these dams, the water would be suitable for release in accordance with the site EPL discharge conditions. A program of water quality monitoring would be required to ensure water collected in the sediment dams is suitable for release (WRM, 2020).

Clean Water Zone

A clean water diversion channel is proposed to divert the upper catchment of Blackmans Gully into Price Creek to reduce the potential volume of water flowing towards the open cut pits. The channel would largely follow the natural contours of the hill slopes and have a gentle gradient.

Clean water diversion channels are also proposed to divert Blackmans Gully and its associated tributary catchments away from the open cut pits both during operations and after mine closure (WRM, 2020).

6.3 REVIEW OF PROJECT IMPACTS ON SURFACE WATER AND GROUNDWATER

6.3.1 Surface Water

A daily timestep water balance model was used to assess the site water balance over the Project life under the range of historical rainfall and evaporation conditions. The results showed under historical conditions, water captured in the containment zone can be contained without discharge or significant interruption to mining operations throughout the operation of the mine (WRM, 2020).

The potential for impact on downstream water flows has been considered. There are two mechanisms by which impacts could occur.

Changes in water flows due to interception of stormwater runoff within the Mine Site area

Water runoff impacted by mining activities would be captured in the water management system. This water would be contained on site and reused in processing operations or in the case of the ECS zone, released, provided it meets the relevant discharge licence limits. This would result in some loss of flow in the catchment area of the surrounding creeks. The diversion of runoff from the undisturbed area of Blackmans Gully into Price Creek would, however, slightly increase the flows within Price Creek and the section of Hawkins Creek between its confluence with Price Creek and its with Blackmans Gully. Overall, the diversion covers only a small area so the change in flows is not considered significant (WRM, 2020).

Changes in water flows due to loss of baseflow recharge into local groundwater

Some changes to groundwater recharge from rainfall are expected during the Project.

As a result of the above two mechanisms, during operations, the maximum impact of the Project on downstream flow is a decrease in flows of:

- a 3.5 km section of Hawkins Creek extending upstream from the Lawsons Creek confluence by up to 4.4%;

- Lawsons Creek, between the Hawkins Creek confluence and upstream of the Walker Creek confluence by up to 1.2%; and
- Lawsons Creek downstream of the Walker Creek confluence by up to 2.2%.

After mining, the maximum impact of the Project on downstream flow is to decrease flows in:

- a 3.5 km section of Hawkins Creek extending upstream from the Lawsons Creek confluence by up to 1.4%;
- Lawsons Creek, between the Hawkins Creek confluence and upstream of the Walker Creek confluence by up to 0.4%;
- Lawsons Creek downstream of the Walker Creek confluence by up to 0.4%.

The relative impact on Lawsons Creek would reduce significantly with increasing distance downstream due to the contribution of other tributaries to total streamflow in Lawsons Creek.

Impact on Availability of Water to Downstream Users

The principal mechanism by which the Project would affect the quantity of water supplies available to other surface water users in the Lawsons Creek Water Source would be by reducing flows such that the frequency and duration of low flow periods would be increased. This could affect water users with cease-to-pump flow conditions specified in their licence conditions.

The impact of the loss on the availability of water to downstream water users has been determined (WRM, 2020) to be negligible.

Impact on Downstream Water Quality

Impacts on water quality are not expected due to the proposed mechanisms for storing and encasing tailings and PAF waste rock. These mechanisms have been designed to prevent seepage and runoff. Appropriate procedures to manage seepage and runoff are proposed for use during operations as well as after closure and decommissioning.

Geochemical assessment of the NAF waste rock suggests they would have little impact on water quality. During operations, runoff from the areas where these rocks would be placed such as the TSF outer embankment, WRE and southern barrier would be captured and treated in sediment dams sized in accordance with Blue Book requirements for Type F basins (DECCW, 2008) before release from the Mine Site in accordance with the requirements of the NSW EPA under the environment protection licence (EPL). However, based on the testing of leachate from kinetic testing off NAF waste rock samples, there is a possibility that runoff and seepage from NAF waste rock would contain dissolved metals particularly manganese.

Therefore, if the ongoing program of geochemical testing and characterisation of runoff determines that runoff must be contained on site to ensure the water source is not contaminated, sufficient storage capacity would be provided to minimise the likelihood of discharge by returning captured runoff to the Containment Zone. The proposed design storage capacity would be sufficient to contain runoff resulting from the 1 in 20 AEP 72 hour design storm (with a design volumetric runoff coefficient of 0.75) (equivalent to 1.2 ML/ha). In addition, sediment storage equivalent to 50% of the water storage capacity would be provided with each dam. Pumping infrastructure would be provided to enable the water to be transferred into the containment system within 5 days.

The southern barrier would be decommissioned after closure, leaving the outer embankment of the TSF, and the store-and-release cover of the WRE as potential sources of runoff from NAF waste rock. Sediment dams would remain in place until vegetative cover is sufficiently established to control erosion from these embankments.

With the implementation of these measures the water quality within the waterways would not be expected to be observably different from existing conditions, and hence there are no health impacts identified for the community accessing as using these waterways for recreational purposes.

A site water quality monitoring plan would be implemented during operations to verify that the captured water quality is suitable for off-site release, and to monitor receiving water conditions. Existing background monitoring points would continue to be monitored and on-site sediment dams would also be monitored at the discharge point.

Potential Impacts on Flooding

A detailed flood impact assessment was carried out for the Project.

Key points with regards to predicted peak flood levels and depths across the study area are summarised below.

- The proposed dams are designed to hold sufficient volumes of water such that water release would not be required.
- The Project disturbance area is located outside of the Lawsons Creek flood extent for all events up to the probable maximum precipitation (PMP) design event.
- The area along the southeastern Mine Site boundary would be affected by flooding from Hawkins Creek. However, the proposed open cut pits and WRE would be located outside of the predicted flood extent for Hawkins Creek for all events up to the PMP design event.
- Flooding along the Hawkins and Lawsons Creeks tributaries is characterised by shallow overland flows. Flows in these tributaries are generally confined within the narrow floodplains, with no breakouts occurring except near the confluences of these tributaries with Hawkins and Lawsons Creeks. Due to the narrow floodplains, the difference in predicted flood extents along these tributaries between the 1% (1 in 100) AEP (annual exceedance probability) and PMP design events are not significant.
- Predicted peak flood depths along the overbank areas of the Hawkins and Lawsons Creeks tributaries are generally below 1 m for events up to and including 0.2% (1 in 500) AEP. Peak flood depths of up to 1.5 m for the PMP design event are predicted in some sections along these tributaries.

These impacts are not expected to result in increased flood hazards for the off-site community.

6.3.2 Groundwater

The groundwater impact assessment has undertaken modelling to assess potential impacts on groundwater due to the Project (Jacobs, 2020). In addition, extensive monitoring of existing groundwater levels and quality has been undertaken. These data have been used to inform the development of the model.

Once mining has extended below the surface sufficiently to reach the groundwater table, dewatering of the main open cut pit would be required to allow mining to continue. The dewatering would result in drawdown of groundwater levels in the rocks immediately surrounding the pit. Over time, the drawdown would extend outwards resulting in a drop of up to 1 m in the groundwater level ranging from 1.5 to 2.2 km from the main open pit.

The impacts of potential changes in groundwater levels have been assessed in accordance with the NSW Aquifer Interference Policy. Using the conservative assumptions required within the Policy, the impacts on groundwater users and groundwater dependent ecosystems are expected to be negligible. There is one bore to the north of the Mine Site (GW061475) where some level of impact is predicted on groundwater levels using the conservative approach required but the modelled drawdown is not expected to actually occur as this bore is located on ground that is higher than the open cut pits. This bore is screened at around 15 m bgl and draws water from the Illawarra Coal Measures rather than alluvium. Another well that was assessed and found to have some potential for drawdown was GW802888 located to the east of the Mine Site. This well is screened at 51 m bgl and so a small predicted change in water level would not be expected to change the operation or yield of the well. Other bores where drawdown was predicted to occur were wells present on the Mine Site itself.

The baseflow contribution of groundwater to flow in both Hawkins and Lawsons Creeks has been estimated using the numerical groundwater model. It identified that the groundwater contribution is relatively low with baseflow from groundwater in Hawkins Creek (approximately 72 m^3/day) being less than half that of Lawsons Creek at approximately 184 m^3/day. During mining, the baseflow from groundwater to both Hawkins and Lawsons Creeks would reduce as drawdown occurs due to dewatering of the open cut pits. A maximum baseflow reduction of approximately 30 m^3/day is predicted for Hawkins Creek and 24 m^3/day for Lawsons Creek within 100 years of commencement of mining.

Oxidation of rocks that have the potential to generate acid when exposed at the surface has the potential to mobilise metals moving them from the rocks into waters within the open cut pits. These waters would be captured and processed to ensure any metals that are dissolved cannot percolate into the groundwater.

It is considered that there would be negligible impact on groundwater quality in the alluvium (the aquifer most likely to be extracted and used, potentially for stock watering, irrigation or domestic purposes) and so no health risk issues of concern related to impacts from the Project are expected, regardless of the likely use of groundwater in the local area.

A Groundwater Management Plan would be developed and implemented for the Project, and would define a groundwater monitoring strategy, groundwater level triggers and a trigger action response plan. In addition, the groundwater model that has been used in this assessment would be reviewed and updated within 2 years of operation of the mine to ensure that actual circumstances are reflected in the modelling.

6.4 UNCERTAINTIES

The assessment presented in relation to potential surface water and groundwater impacts, and the potential for impacts on community health as a result of surface water and groundwater impacts as a result of the Project are considered to be conservative. There are a number of

areas within the surface water and groundwater assessments where conservative assumptions and approaches have been adopted. The conclusions of these assessments have also been informed by sensitivity and uncertainty analysis.

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

6.5 OUTCOMES OF HEALTH RISK ASSESSMENT: WATER

Table 6.1 presents a summary of the outcomes of the assessment undertaken in relation to the impacts of changes in surface water and groundwater, associated with the Project, on community health.

Table 6.1
Summary of Health Risks – Water

Water	
Impacts	Based on the assessments undertaken, the potential for adverse health impacts within the off-site community associated with impacts to surface water and groundwater as a result of the Project is considered to be negligible.
Mitigation	Implementation of the water management system.

7. ASSESSMENT OF HEALTH: NOISE

7.1 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:

- SLR 2020, Noise and Vibration Assessment.

The noise assessment has considered impacts at each residential property surrounding the Project (refer to **Figures 4.2** and **4.3** for the location of these properties). These are the same properties as have been evaluated within the air quality assessment. These receivers include privately-owned and Project-related properties/premises surrounding the Project including Lue Public School.

7.2 HEALTH IMPACTS ASSOCIATED WITH NOISE

Environmental noise has been identified (I-INCE 2011; WHO 2011c, 2018)[12] 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 1999b, 2011c, 2018):

- Sleep disturbance (sleep fragmentation that results in fatigue and affects 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).

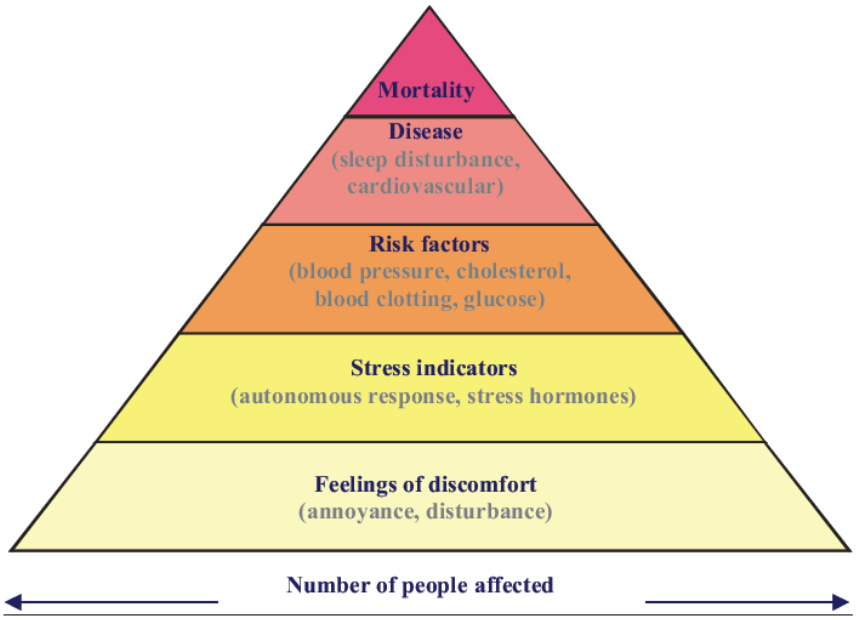
Other effects for which evidence of health impacts exists, and are considered to be important, but for which the evidence is weaker, include:

- 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).

[12] 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 7.1**.

Figure 7.1 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).

Based on the relevant WHO guidelines for noise, **Table 7.1** presents thresholds that have been determined to be protective of health effects. These noise levels relate to levels outside a home/building as the modelling of noise impacts presented by SLR (2020) are outside of a home (not inside). The guidelines for outside assume windows are left open, which may be the case during at least some of the year in the Lue area.

Table 7.1
Health Protective Noise Thresholds from WHO (Noise Levels Outside) (WHO 1999b, 2009)

Environment and Exposure Time (T)	**Critical (Most Sensitive Health Effect)**	**$LA_{eq,T}$ (dB(A))**	**LA_{max} (dB)**
Residents			
Day and evening – 16 hours	Annoyance, cardiovascular effects and disturbance of conversation	50	NA
Night – 8 hours	Sleep disturbance	42	60
Schools			
Day – during class (6 hours)	Speech intelligibility, communication	50	NA

7.3 REVIEW OF THE NOISE GUIDELINES ADOPTED

Noise guidelines adopted in the Noise and Vibration Assessment are those outlined in the Noise Policy for Industry (NPfI) (NSW EPA 2017), which indicate that intrusive noise from a specific industrial source should not exceed the RBLs by more than 5 dB(A). In addition, consideration has also been given to noise amenity, with the project noise trigger levels adopted based on the lower noise criteria relevant to intrusiveness and amenity. The noise trigger levels adopted were $LA_{eq,15\text{-}minute}$ of 40 dB(A) during the day and 35 dB(A) during the evening and night for residences and 43 dB(A) for Lue Public School (when in use). While these noise trigger levels are sufficiently low to be protective of health, they are more conservative than the thresholds for health effects established by the WHO (WHO 1999b, 2011c). This is because the NPfI utilises a short-duration time for averaging noise levels, 15-minutes, whereas the WHO guidelines relate to exposures over the day and evening combined (16 hours) or night-time (8 hours). For assessing health effects of potential exposure to noise, the thresholds for health effects established by the WHO (and summarised in **Table 7.1**) are relied upon in this assessment.

Maximum noise levels were also established based on the NPfI guidance (NSW EPA 2017). The maximum noise criteria are set to protect residents from sleep disturbance and for this Project, an LA_{Fmax} of 52 dB(A) is relevant to the night-time period. This maximum noise level is lower than the maximum noise level of 60 dB(A) outside established by the WHO for the protection of health sufficiently low to be protective of health (WHO 1999b).

The NPfI and the Voluntary Land Acquisition Mitigation Policy (VLAMP) (NSW DPE 2018) provides guidance on the interpretation of noise impacts in relation to these trigger levels for noise impact assessments, particularly in relation to predicted/estimated changes in noise levels.

Blasting impacts have been evaluated in accordance with criteria established to protect human annoyance and structural damage (Australian Standard [AS] 2187: Part 2-2006 Explosives – Storage and Use. Provided the human comfort criteria are met, there would be no concern in relation to health impacts.

Road traffic noise was assessed on the basis of the NSW Road Noise Policy (NSW DECCW 2011)[13], as it applies to existing properties affected by additional traffic. This provides a guideline of 55 to 60 dB(A) as $LA_{eq,15\ hour}$ (day and evening) and 55 dB(A) as $LA_{eq,9\ hour}$ (night) for residential properties and 50 dB(A) for Lue Public School (relevant to school hours). 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 1999b, 2009).

Construction noise criteria have been adopted from the Interim Construction Noise Guideline (ICNG) (NSW DECC 2009)[14] which provide management levels relevant to the assessment of noise impacts above the RBL during standard hours (guideline is RBL + 10 dB(A) = 45 dB(A) for residences and 55 dB(A) for Lue Public School) with noise levels (total noise from all sources) above 75 dB(A) at residences during standard hours considered to be highly noise affected. While these criteria may result in some construction noise being noticeable, the noise criteria adopted for the Project are protective of health, including annoyance and sleep disturbance[15], where they relate to outside noise levels (WHO 1999b, 2009).

7.4 REVIEW AND ASSESSMENT OF HEALTH IMPACTS FROM NOISE

7.4.1 Construction

Assessment of noise impacts during construction involved consideration of the relevant construction activities (equipment used, hours of use and location of use).

In relation to the assessment of noise generated during a range of construction activities, these have been assessed separately from the operational noise impacts (discussed below). The assessment of construction noise was undertaken using the Environmental Noise Model (ENM), which provides noise predictions at each individual receptor – as an outdoor noise level.

Assessment of construction noise impacts identified some exceedances of the ICNG at privately-owned residences. Five exceedances were predicted during the construction of the new intersection between Lue Road and the proposed relocated Maloneys Road. For one property the exceedance of the guideline was determined to be negligible to marginal (1 to 5 dB(A) above the guideline). For four properties, the exceedance of the guideline was determined to be moderate (>5 dB(A) above the guideline). These exceedances are expected to occur over a 1-2 month period. No properties were considered to be highly noise affected.

[13] DECCW – NSW Department of Environment, Climate Change and Water.
[14] DECC – NSW Department of Environment and Climate Change.
[15] No night-time construction activities are proposed.

Some noise impacts were also predicted at a number of properties during the power transmission line re-alignment works, during the operational Year 3 works, which were determined to be negligible (12 properties), marginal to moderate (5 properties) and significant (5 properties), also associated with the most intensive works related to the power transmission line re-alignment works which are expected to occur over a 1-2 month period.

Some noise impacts were also predicted during the construction of the water supply pipeline. SLR (2020) calculate that the highly noise affected level of 75 dB(A) would be satisfied at an offset distance of approximately 50m from the water supply pipeline. There are seven residences located less than this distance. However, whilst noise exceedances would be noticeable at these residences, the duration of these noise levels would likely occur for 1 to 2 days at each residence. As such the impact from these exceedances would be minimal, particularly with discussion of the planned activities with the occupants of each residence prior to the commencement of construction to ensure impacts are minimised or avoided.

The noise impacts identified would be managed by Bowdens Silver in accordance with an approved Construction Noise Management Plan (CNMP). The CNMP would also address noise impacts also identified at Project-related properties.

Where noise impacts are appropriately managed during the noise intensive works, potential impacts on health would be minimised. It is noted that, while the potential for health impacts would be minimised, noise may be noticeable at some properties at times.

7.4.2 Blasting

The assessment conducted by SLR (2020) determined that the human comfort criteria relevant to blasting activities would be met at all locations except at three properties. These impacts would be managed through the implementation of a Blast Management Plan (BMP) to ensure impacts are minimised. It is not expected that the impacts, where managed, would result in health impacts.

7.4.3 Operational Noise

Approach

The assessment of noise (SLR, 2020) has considered noise impacts from the Project during operations. The noise assessment has utilised the ENM (Environmental Noise Model) that provides predictions of noise impacts at each modelled receptor as an outdoor noise level. The assessment of noise impacts has addressed four noise scenarios:

- Scenario 1 (Year 0) when only day-time operations occur that involve site development and construction;
- Scenario 2 (Year 3) when there are day, evening and night-time operations and construction of the second raise of the TSF embankment;
- Scenario 3 (Year 8) when there are day, evening and night-time operations and construction of the third raise of the TSF embankment; and
- Scenario 4 (Year 10) when there are day, evening and night-time operations without any construction works for the TSF embankment.

Activities that are proposed to be undertaken during these Project years, including the time and location of operation, and sound power levels generated by these equipment/activities, have been considered in the noise model, along with terrain and meteorological conditions (i.e. wind enhancing as well as temperature inversions and drainage flows), with standard meteorological conditions as well as worst-case meteorological conditions evaluated (SLR, 2020).

The noise modelling undertaken has been conducted in an iterative manner, incorporating and evaluating various combinations or noise management and mitigation measures. As a result, a range of specific mitigation measures have been identified in the noise impact assessment to reduce noise emissions from the Project. The assessment has also considered the use of a range of mitigation measures, with modelling being conducted with and without these measures.

The noise assessment predicted noise levels as $LA_{eq,15\text{-}min}$ values. These values are relevant for the evaluation of noise impacts on the basis of the NPfI (NSW EPA 2017), however for the assessment of health impacts of noise, the noise levels evaluated need to relate to the assessment period of a day, evening or night. Guidance in the NPfI indicates that $LA_{eq,period}$ = $LA_{eq,15min}$ – 3dB. This conversion has been used to predict $LA_{eq,day}$, $LA_{eq,evening}$ and $LA_{eq,night}$ levels.

Noise Impacts

The Noise and Vibration Assessment (SLR, 2020) identified a number of residences in the rural area where noise levels were predicted to exceed the adopted project noise trigger levels. These exceedances were evaluated to be significant for one privately-owned property, marginal to moderate at a further four privately-owned properties, and negligible for a further six privately-owned residences. All properties in Lue, including places of interest such as Lue Public School, met the adopted guidelines. On the basis of the assessment presented by SLR (2020), an Operational Noise Management Plan would be implemented by Bowdens Silver to minimise operational noise impacts. Based on guidance provided by the VLAMP, the Noise and Vibration Assessment (SLR, 2020) concluded that no privately-owned land is predicted to be impacted by the Project.

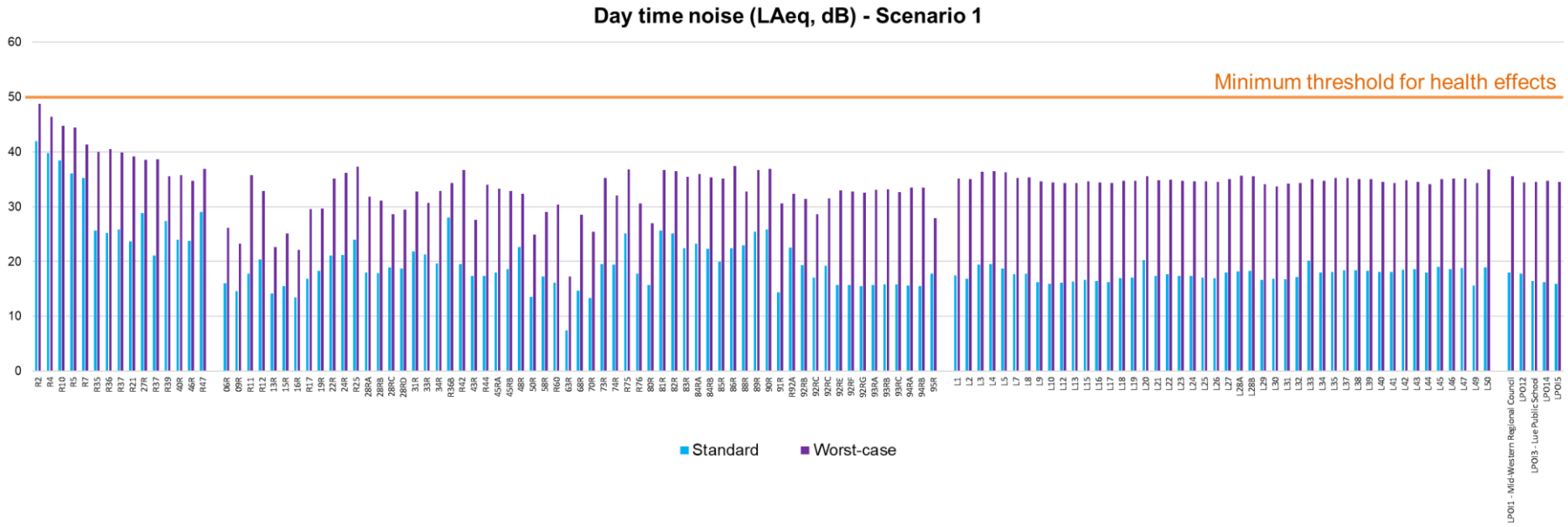
Assessment of Health Impacts

Predicted maximum noise levels at each privately-owned property as $L_{Aeq,15\text{-}minute}$ during the day, evening and night-time periods, for the modelled scenarios for standard and worst-case meteorological conditions were provided by SLR (2020) for use in this assessment. As discussed above, to be able to compare these noise predictions with thresholds for health effects, presented in **Table 7.1**, $L_{Aeq,period}$ was calculated for the day, evening and night-time periods.

The following figures show the predicted noise levels as $LA_{eq,day}$, $LA_{eq,evening}$ and $LA_{eq,night}$ for the modelled scenarios with the highest impact with comparison against the health-based thresholds in **Table 7.1**. Figures for all scenarios are included in **Annexure H**.

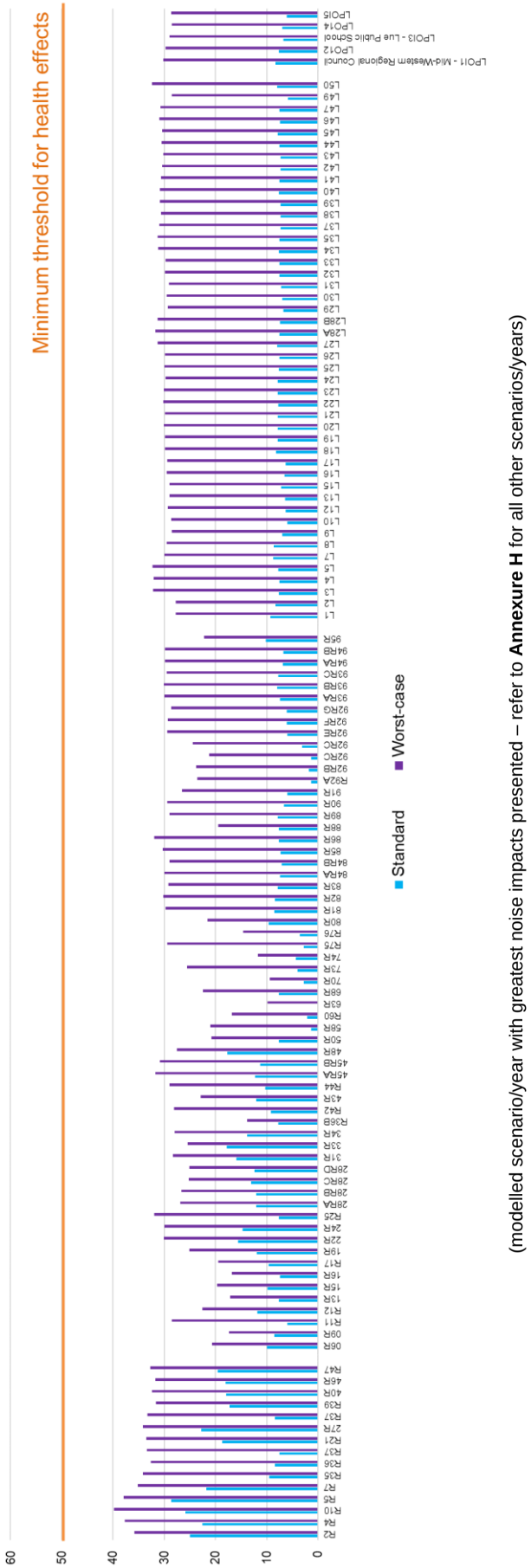
Review of **Figures 7.2**, **7.3** and **7.4** indicates that all modelled noise levels during the day, evening and night at all privately-owned properties, including places of interest such as Lue Public School are below the health-based threshold.

Figure 7.2 Predicted Day-time Noise Levels at all Privately-owned Residences as LA_{eq}, day (dB(A)) with Comparison against Day-time threshold for Health Effects



(modelled scenario/year with greatest noise impacts presented – refer to **Annexure H** for all other scenarios/years)

Figure 7.3 Predicted Evening Noise Levels at all Privately-owned Residences as LA_{eq}, evening (dB(A)) with Comparison against Day-time threshold for Health Effects



(modelled scenario/year with greatest noise impacts presented – refer to **Annexure H** for all other scenarios/years)

Figure 7.4 Predicted Night-time Noise Levels at all Privately-owned Residences as LA_{eq}, night (dB(A)) with Comparison against Day-time threshold for Health Effects

(modelled scenario/year with greatest noise impacts presented – refer to **Annexure H** for all other scenarios/years)

On this basis, there are no health impacts of concern in relation to noise impacts from the Project. It is noted, that given the existing noise environment of the area, it is likely that at times noise from the Project may be distinguishable above background. While these noises may be distinguishable, they would remain too low to impact on community health.

Road Noise

Assessment of road noise impacts considered expected road traffic volumes relevant to the Project, on Lue Road, Pyangle Road and the relocated Maloneys Road. The assessment determined that noise at all receptors along these roads would comply with the relevant noise guidelines with the exception of Lue Public School. While the predicted total noise level at the school exceeds the adopted guideline for road traffic noise, this exceedance mainly relates to existing traffic on Lue Road with the Project impacts contributing a 0.8 dB(A) increase in noise. Such noise impacts are considered to be minor and would not be perceptible.

All noise impacts predicted at all privately-owned roadside residences as a result of the Project meet the relevant noise criteria. As these noise criteria are protective of health, there would be no health impacts of concern in relation to road noise.

Overall

Based on the available information, the potential for noise impacts to result in adverse health impacts within the community is considered to be negligible.

7.5 UNCERTAINTIES

The assessment of potential noise impacts, and the potential for impacts on community health as a result of changes in noise as a result of the Project are 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 consideration of the worst-case meteorological conditions and assuming these occur on a regular basis, and use of the maximum impacted noise from a 15-minute period to interpret an average noise level over an 8 hour time period of the day, evening or night. The noise levels predicted during these time periods would be a worst case as they relate to the worst-case conditions evaluated for each scenario, and are not representative of noise levels on every day of the year, or the duration of the Project.

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

7.6 OUTCOMES OF HEALTH RISK ASSESSMENT: NOISE

Table 7.2 presents a summary of the outcomes of the assessment undertaken in relation to the impacts of changes in noise, associated with the Project, on community health.

Table 7.2
Summary of Health Risks - Noise

Noise Emissions	
Impacts	Based on the predicted noise levels and potential mitigation measures, the potential for adverse health impacts within the off-site community associated with noise generated during construction and operations is considered to be negligible
Mitigation	Development of a Construction Noise Management Plan, Blast Management Plan and Operational Noise Management Plan prior to commencement of the Project has been identified as an important aspect of managing and minimising noise and blasting impacts from the Project.

8. CONCLUSIONS

The HHRA presented in this report has considered potential impacts on community health in relation to the proposed Project from changes in air quality, water (both surface water and groundwater) and noise.

The assessment undertaken has considered the rural-residential nature of the existing community, as well as Lue where Lue Public School is located. The assessment of air quality has focused on dust emissions from the Project as this is of key concern to some members of the Lue and district community. The presence of lead and other metals that may be present on these dust emissions has been evaluated in detail. Metals are ubiquitous in the environment, and all members of the community are already exposed to some levels of metals in the existing environment (air, soil, water and dietary intakes). Hence, the HHRA has considered both the existing exposures and exposures that may occur as a result of the Project. The assessment has addressed all exposures that may occur in the area, such as the inhalation of dust, the deposition of dust onto roofs and the washing of these dusts into rainwater tanks where water may be used for drinking/household, the deposition of dust to soil and other surfaces where people may come into direct contact, and/or the accumulation of these metals into home-grown produce that may be consumed.

The HHRA has also addressed other emissions to air, along with health impacts related to changes in water quantity or quality, and noise generated from the Project.

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, **Table 8.1** presents a summary of the health impact assessment and mitigation measures relevant to ensuring impacts are minimised or mitigated. The HHRA has not identified any additional management measures, over and above those identified within the air quality, noise and water assessments.

Table 8.1
Summary of Health Risks

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Air Emissions	
Impacts	Based on the available data and information in relation to emissions to air from the Project, including dust which comprises lead and a range of other metals, potential impacts on the health of the community have been assessed. The impact assessment has concluded that impacts attributed to the Project would make a negligible contribution to overall exposures to these metals and there would be no health risk issues of concern relevant to the Project (including construction and operational phases). These conclusions apply to all members of the community, adults and children as well as sensitive individuals.
Mitigation	An Air Quality Management Plan is expected to be developed prior to commencement of operations at the Project that would outline the measures to manage air emissions (consistent with those considered and outlined in the Air Quality Impact Assessment, Ramboll 2021).
Water	
Impacts	Based on the assessments undertaken, the potential for adverse health impacts within the off-site community associated with impacts to surface water and groundwater as a result of the Project is considered to be negligible.
Mitigation	Implementation of the water management system.

Table 8.1 (Cont'd)
Summary of Health Risks

Noise Emissions	
Impacts	Based on the predicted noise levels and potential mitigation measures, the potential for adverse health impacts within the off-site community associated with noise generated during construction and operations is considered to be negligible.
Mitigation	Development of a Construction Noise Management Plan, Blast Management Plan and Operational Noise Management Plan prior to commencement of the Project has been identified as an important aspect of managing and minimising noise and blasting impacts from the project.

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Annexures

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Annexure A

Assessment of Impacts of Fine Particulates

(Total No. of pages including blank pages = 10)

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A1 HEALTH EFFECTS OF EXPOSURE TO PARTICULATES

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

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

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

- 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.

The most recent review of the available studies (USEPA 2018) have also indicated that effects on the nervous system and carcinogenic effects are likely to have a causal relationship with long-term exposures to $PM_{2.5}$. IARC (2013) has classified particulate matter as carcinogenic to humans based on data relevant to lung cancer.

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).

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 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 are suggestive (but do not show effects as clearly as the effects noted above) of an association between exposure to $PM_{2.5}$ and reproductive and developmental effects as well as cancer, mutagenicity and genotoxicity (USEPA 2012). IARC (IARC 2013b, 2013a) has classified particulate matter as carcinogenic to humans based on data relevant to lung cancer.

There are a number of studies that have been undertaken where other health effects have been evaluated. These studies have a large degree of uncertainty or a limited examination of the relationship and are generally only considered to be suggestive or inadequate (in some cases) of an association with exposure to $PM_{2.5}$ (USEPA 2018). 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).

In relation to the key health endpoints relevant to evaluating exposures to $PM_{2.5}$, there are some associated health measures or endpoints where the exposure-response relationships are not as strong or robust as those for the key health endpoints and are considered to be a subset of the key health endpoints. This includes mortality (for different age groups), chronic bronchitis, medication use by adults and children with asthma, respiratory symptoms (including cough), restricted work days, work days lost, school absence and restricted activity days (Anderson et al. 2004; EC 2011; Ostro 2004; WHO 2006b).

A2 APPROACH TO THE ASSESSMENT OF PARTICULATE EXPOSURES

In relation to the assessment of exposures to particulate matter there is sufficient evidence to demonstrate that there is an association between exposure to $PM_{2.5}$ (and to a lesser extent PM_{10}) and effects on health that are causal.

The available evidence does not suggest a threshold below which health effects do not occur. Accordingly, there are likely to be health effects associated with background levels of $PM_{2.5}$ and PM_{10}, even where the concentrations are below the current guidelines. Standards and goals are currently available for the assessment of $PM_{2.5}$ and PM_{10} in Australia (NEPC 2016). These standards and goals are not based on a defined level of risk that has been determined to be acceptable, rather they are based on balancing the potential risks due to background and urban sources to lower impacts on health in a practical way.

The air quality standards and goals relate to average or regional exposures by populations from all sources, not to localised ‘hot-spot’ areas such as locations near industry, busy roads or mining. They are intended to be compared against ambient air monitoring data collected from appropriately sited regional monitoring stations. In some cases, there may be local sources (including busy roadways and industry) that result in background levels of PM_{10} and $PM_{2.5}$ that are close to, equal to, or in exceedance of, the air quality standards and goals. Where impacts

are being evaluated from a local source it is important to not only consider cumulative impacts associated with the project (undertaken using the current air quality goals) but also evaluate the impact of changes in air quality within the local community.

This assessment has therefore been undertaken to consider both cumulative exposure impacts (refer to Section A3) and incremental exposure impacts associated with changes in $PM_{2.5}$ and PM_{10} concentrations that are associated with the Project (refer to Section A4). Incremental changes are those due to the project alone while cumulative changes are those where background air quality in addition to those due to the project alone are considered.

A3 ASSESSMENT OF CUMULATIVE EXPOSURES

The assessment of cumulative exposures to $PM_{2.5}$ and PM_{10} is based on a comparison of the cumulative concentrations predicted with the current air quality standards and goals presented in the National Environment Protection Council (NEPC) (Ambient Air Quality) Measure (NEPM) (NEPC 2016). These standards and goals are total concentrations in ambient air, within the community, that are based on the most current science in relation to health effects. The most current standards and goals, based on the protection of community health presented by the NEPC, have been further considered in this health impact assessment report.

In relation to the current NEPM PM_{10} standard, the following is noted (NEPC 1998b, 2010, 2014, 2016):

- 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 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.
- 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 2016).

Table A1 presents a comparison of the current NEPC standards and goals with those established by the WHO (WHO 2005), the EU and the USEPA (2012). 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 WHO, EU and the USEPA. The NEPM PM_{10} guidelines are also similar to those established by the WHO and EU, however the guidelines are significantly lower than the 24-hour average guideline available from the USEPA.

Table A1
Comparison of Particulate Matter Air Quality Goals

Pollutant	Averaging Period	Criteria / Guidelines / Goals			
		NEPC	WHO (2005)	EU #	USEPA (2012)
PM_{10}	24-hour	50 µg/m³	50 µg/m³	50 µg/m³ as limit value with 35 exceedances permitted each year	150 µg/m³ (not to be exceeded more than once per year on average over 3 years)
	Annual	25 µg/m³	20* µg/m³	40 µg/m³ as limit value	NA
$PM_{2.5}$	24-hour	25 µg/m³ 20 µg/m³ (goal for 2025)	25 µg/m³	NA	35 µg/m³ (98th percentile, averaged over 3 years)
	Annual	8 µg/m³ 7 µg/m³ (goal for 2025)	10* µg/m³	25 µg/m³ as target value from 2010 and limit value from 2015. 20 µg/m³ as a 3 year average (average exposure indicator) from 2015 with requirements for ongoing percentage reduction and target of 18 µg/m³ as 3 year average by 2020	12 µg/m³ (annual mean averaged over 3 years)

\# Current EU Air Quality Standards available from http://ec.europa.eu/environment/air/quality/standards.htm

* The WHO Air Quality guidelines are based on the lowest levels at which total, cardiopulmonary and lung cancer mortality have been shown to increase with more than 95 per cent confidence in response to $PM_{2.5}$ in the ACS study (Pope, CA, 3rd et al. 2002). The use of a $PM_{2.5}$ guideline is preferred by the WHO (WHO 2005).

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).

A3 ASSESSMENT OF INCREMENTAL EXPOSURES

A quantitative assessment of risk for these endpoints uses a mathematical relationship between an exposure concentration (i.e. concentration in air) and a response (namely a health effect). This relationship is termed an exposure-response relationship and is relevant to the range of health effects (or endpoints) identified as relevant (to the nature of the emissions assessed) and robust (as identified in the main document). An exposure-response relationship can have a threshold, where there is a safe level of exposure, below which there are no adverse effects; or the relationship can have no threshold (and is regarded as linear) where there is some potential for adverse effects at any level of exposure.

In relation to the health effects associated with exposure to particulate matter, no threshold has been identified. Non-threshold exposure-response relationships have been identified for the health endpoints considered in this assessment.

Risk calculations relevant to exposures to $PM_{2.5}$ by the community have been undertaken utilising concentration-response functions relevant to the most significant health effect associated with exposure to $PM_{2.5}$, namely mortality (all cause).

The assessment of potential risks associated with exposure to particulate matter involves the calculation of a relative risk (RR). For the purpose of this assessment the shape of the exposure-response function used to calculate the relative risk is assumed to be linear[16]. The calculation of a relative risk based on the change in relative risk exposure concentration from baseline/existing (i.e. based on incremental impacts from the project) can be calculated on the basis of the following equation (Ostro 2004):

Equation 1 RR = exp[β(X-X0)]

Where:

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

β = regression/slope coefficient, or the slope of the exposure-response function which can also be expressed as the per cent change in response per 1 µg/m^3 increase in particulate matter exposure.

Based on this equation, where the published studies have derived relative risk values that are associated with a 10 micrograms per cubic metre increase in exposure, the β coefficient can be calculated using the following equation:

Equation 2 $$\beta = \frac{\ln(RR)}{10}$$

Where:

RR = relative risk for the relevant health endpoint as published (µg/m^3)

10 = increase in particulate matter concentration associated with the RR (where the RR is associated with a 10 µg/m^3 increase in concentration).

The assessment of health impacts for a particular population associated with exposure to particulate matter has been undertaken utilising the methodology presented by the WHO (Ostro 2004)[17] where the exposure-response relationships identified have been directly considered on the basis of the approach outlined below.

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

17 For regional guidance, such as that provided for Europe by the WHO WHO 2006b, Health risks or particulate matter from long-range transboundary air pollution regional background incidence data for relevant health endpoints are combined with exposure-response functions to present an impact function, which is expressed as the number/change in incidence/new cases per 100,000 population exposed per microgram per cubic metre change in particulate matter exposure. These impact functions are simpler to use than the approach adopted in this assessment, however in utilising this approach it is assumed that the baseline incidence of the health effects is consistent throughout the whole population (as used in the studies) and is specifically applicable to the sub-population group being evaluated. For the assessment of exposures in the areas evaluated surrounding the project it is more relevant

An additional risk can be calculated as:

Equation 3 $Risk=\beta \times \Delta X \times B$

Where:

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

ΔX = change (increment) in exposure concentration in µg/m^3 relevant to the project at the point of exposure

B = baseline incidence of a given health effect per person (e.g. annual mortality rate)

The calculation of the incremental individual risk for relevant health endpoints associated with exposure to particulate matter as outlined by 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 proposed Project, with the maximum change for all privately-owned residences. For this assessment, the change in $PM_{2.5}$ relates to the change in annual average air concentrations and the value considered in this assessment is 0.8 µg/m^3 (at receptor R7).
- Baseline incidence of the key health endpoints that are relevant to the population exposed – the assessment undertaken has considered the baseline mortality data relevant to the Mid-Western Regional LGA (data from NSW health for mortality all causes and all ages for 2016-17) of 665.9 as the rate per 100,000.
- Exposure-response relationships expressed as a percentage change in health endpoint per microgram per cubic metre change in particulate matter exposure, where a relative risk (RR) is determined (refer to Equation 1). The concentration response function used in this report is that recommended in 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, IC et al. 2002). The study found a relative risk of all-cause mortality of 1.06 per 10 µg/m^3 change in $PM_{2.5}$, and that this risk relationship was in the form of an exponential function. 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). Using a RR of 1.06, results in a $\beta = 0.006$.

The above approach (while presented slightly differently) is consistent with that presented in Australia (Burgers & Walsh 2002), US (OEHHA 2002; USEPA 2005b, 2010) and Europe (Martuzzi et al. 2002; Sjoberg et al. 2009).

to utilise local data in relation to baseline incidence rather than assume that the population is similar to that in Europe (where these relationships are derived).

Based on the above:

$$\text{Risk} = 0.006 \times 0.8 \times 665.9/100000 = 3 \times 10^{-5}$$

Population risks are calculated to be 5×10^{-6} (based on an average concentration at all community receptors of 0.13 µg/m^3) which is lower than this maximum risk.

For the assessment of population or community risks from changes in particulate matter exposures an acceptable risk is = ≤ 1×10^{-5} as established as a policy consideration in NEPM guidance for establishing air guidelines (NEPC 2011). The population risk for this area is lower than this criteria and considered acceptable.

For the assessment of localised impacts (i.e. maximum risks) there is limited guidance available. The assessment of community/population risk provides an evaluation of potential health impacts within a larger population based on the average (or population weighted average) change in exposure that occurs within that population or region. For the assessment of particulate matter such calculations are appropriate as they draw on exposure-response relationships that are derived from population wide epidemiological studies (where regional or average air quality is evaluated against changes in population health).

Within any such region or larger population there will be areas where exposures and risks will be higher, as some individuals are located closer to localised sources, and some areas where exposures and risks will be lower, as some individuals will be well away from localised sources. This will also be the case, but not evaluated, with the populations considered in the underlying epidemiological studies from which the exposure-response relationships are derived. For the assessment of a local source, it is important to provide an upper limit for the localised exposures and risks to minimise health impacts associated with these sources. Such a limit will be higher than that adopted for the assessment of community/population risks as noted above. However, it should not be so large that risks are in the range that is considered to be unacceptable.

A level of 10^{-4} for increased risk (one chance in 10,000) has been generally adopted by health authorities as a point where risk is considered to be unacceptable in the development of drinking water guidelines (that impact on whole populations) (for exposure to carcinogens as well as for annual risks of disease (Fewtrell & Bartram 2001)), from the USEPA and in the evaluation of exposures from air pollutants from specific sources (NSW DEC 2005; NSW EPA 2016). Hence it is relevant to consider an upper limit for a localised risk that is no greater than 10^{-4} (above which risk would be considered unacceptable).

This upper level of risk for the assessment of localised impacts, 10^{-4}, is 10 times higher than the level adopted for the assessment of community or population risks from changes in nitrogen dioxide, particulate matter and noise. Adopting an upper limit for the assessment of localised impacts that is 10 times higher than that adopted for population exposures is consistent with the difference in acceptable risks adopted for population exposures to carcinogens (10^{-6} as outlined by the NHMRC (NHMRC 2011 updated 2018)) and the assessment of localised carcinogenic risks from contaminated land (10^{-5} as outlined by NEPC (NEPC 1999 amended 2013c)).

The maximum risk calculated in this assessment is less than 10^{-4}.

The calculation is also considered conservative as the air modelling has adopted conservative assumptions, in particular rainfall, which would reduce dust emissions on wet days and increase wet deposition.

Annexure B

Toxicity Summary for Lead

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B1 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 2007b).

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 2007b).

B2 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 2015a).

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 2015a).

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 2007b). When lead salts are dissolved in water, low levels of lead can be absorbed through the skin – this is termed dermal permeability (RAIS).

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 2015a). Incidental 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 to be the least phytoavailable. Soil properties affect the potential for uptake and translocation, however, lead that is water soluble and/or exchangeable

is readily available for uptake by plants and 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 Australia 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 2018) 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 with 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 0.44 µg/kg/day for adults 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. Where these intakes are considered in addition to dietary and water intakes, they contribute <10% of the adopted threshold value.

Where site-specific or area-specific information is available on background intakes of lead (as is the case for this assessment), these should be used in preference to the information above, which is generic.

B3 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 2007b).

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 oesophagus 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 2007b). 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 ciliary action and swallowed (Safe Work Australia 2014a). This is in contrast to the larger particles (>2.5 µm) that are transferred within hours by mucociliary transport into the oesophagus and mainly swallowed, meaning the digestive tract can also be an important avenue of lead absorption following inhalation (Safe Work Australia 2014a).

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

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 2007b). 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 2000a). 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 are 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 2007b).

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) followed by absorption into the 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 for the USA, 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 relationship 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 100% of the lead in soil can move into solution and be available for absorption) but only 50% of that is actually absorbed (i.e. the value from WHO relevant to children – noting a lower value is relevant for adults so using this value for both adults and children is conservative). Therefore, a default 50% oral bioavailability value for children (with the same assumed for adults where relevant) is used in the current derivation of the Australian HIL for lead (NEPC 1999 amended 2013e) – this reflects the gastrointestinal absorption, with 100% bioaccessibility from soil assumed.

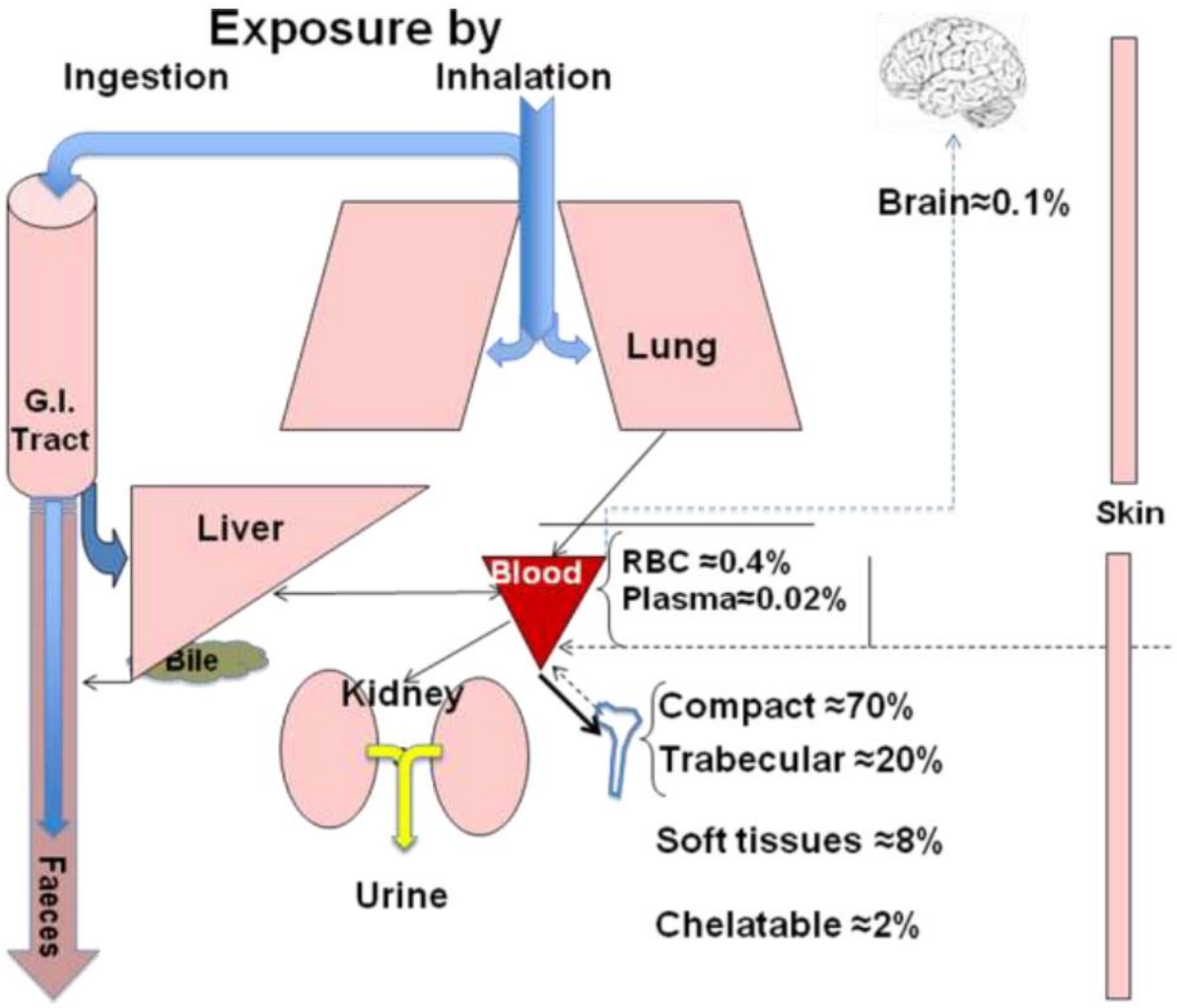
Where site specific bioaccessibility is available, the bioavailability is adjusted to be 50% absorption x bioaccessible fraction. This adjustment can be made for this assessment as bioaccessibility has been measured for the site.

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 2002b). Organic lead is considered far more permeable to the skin and can have a role in lead exposure (ATSDR 2007b). When lead salts are dissolved in water, low levels of lead can be absorbed through the skin – this is termed dermal permeability (RAIS).

Distribution

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



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

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

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, or 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 2007b). 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% binds to the red blood cells, less than 1% binds to blood plasma of which 40-75% is bound to proteins (primarily albumin) (Safe Work Australia 2014a). Thus only a small fraction of PbB (<1%) is the biologically labile and toxicologically active fraction of the circulating lead (USEPA 2013).

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

Lead in soft tissue is predominately in the liver and kidneys, where it is assumed it is 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 2007b).

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 2007b).

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 (USEPA 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 epidemiological 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 (USEPA 2013) does not make a conclusion regarding exposure and health risk to children from ingesting breast milk (Safe Work Australia 2014a).

Metabolism

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

Elimination

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

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

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 2007b).

B4 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 (i.e. studies in people). The health effects of lead are the same regardless of the route of exposure (ATSDR 2019b).

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

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

However, due to the multi-modes of action of lead in biological systems, lead could potentially affect any system or organs in the body. The effects of lead exposure have often been related to the blood lead content, which is generally considered to be the most accurate means of assessing exposure (MfE 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 2007b; Baars et al. 2001; UK DEFRA & EA 2002b; USEPA IRIS), however, blood lead goals and associated intakes have been identified by various agencies for the assessment of lead exposures by the general public. The NHMRC has noted that there are no benefits of human exposure to lead and that all demonstrated effects of exposure are adverse.

For the assessment of lead exposures in Australia, the current advice/statement from NHMRC on the evidence of health effects from lead, released in 2015 has been considered. This statement identified that the average Australian blood lead level was less than 5 micrograms per decilitre (µg/dL). Therefore, if an Australian had a blood lead level of 5 µg/dL or greater, and they did not live 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 2015b). 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 has 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 2015b).

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 has concluded that there is insufficient evidence that blood lead at this level caused any of the health effects observed (NHMRC 2015b).

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 (2015a). An upper concentration limit of lead, based on the protection of adverse health effects can be estimated using the IEUBK lead model as undertaken in the Contaminated sites NEPM (NEPC 1999 amended 2013e) 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).

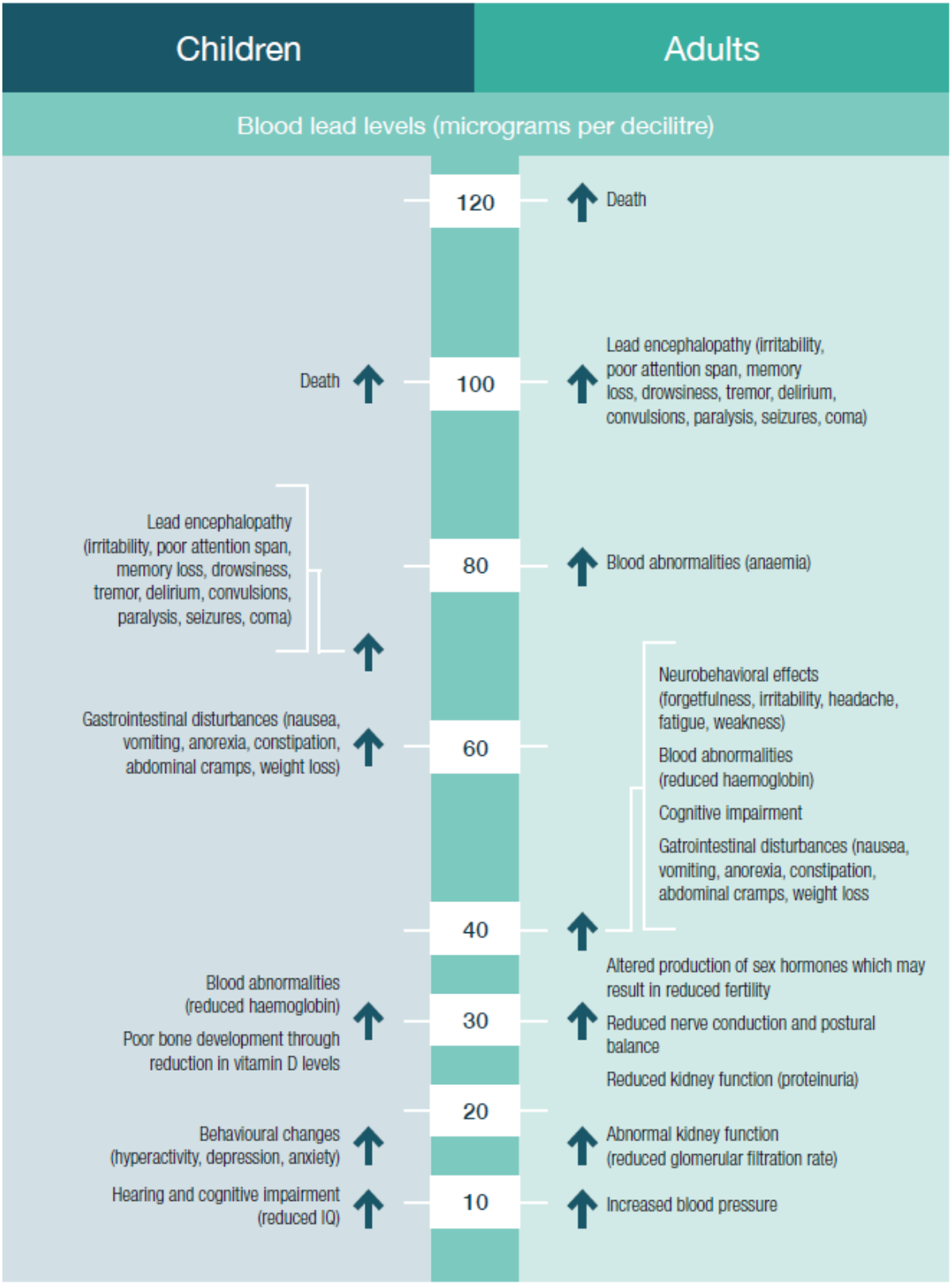


Figure B1 Summary of health effects of lead exposure above 10 µg/dL

B5 APPROACHES FOR THE CHARACTERISATION OF HAZARDS/TOXICITY

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

Table B1
Toxicity reference values (TRVs) and goals for lead

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 Updated 2016)	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 2015b)	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.
NEPM (NEPC 1998a, 2016)	Air Quality Goal = 0.5 µg/m^3	Air guideline (based on an annual average) set by NEPM (ambient air quality) as applied to TSP. The limited documentation available indicates that this value was set to ensure blood lead levels are below 10 µg/dL. The derivation of the guideline (including studies considered) is not provided and hence this value, while relevant from a regulatory perspective, is not suitable for use in risk assessment. It is noted that the value is the same value as that set by the WHO Air Quality Guidelines.
Safe Work Australia (Safe Work Australia 2014b)	Target PbB goals of 20 µg/dL Blood lead removal level 30 µg/dL	Relevant for nearly all workers, including females of non-reproductive capacity and males. For females of reproductive capacity, a lower blood lead goal is recommended, namely 10 µg/dL.

Table B1 (Cont'd)
Toxicity reference values (TRVs) and goals for lead (Cont'd)

Source	Value	Basis/Comments
International		
JECFA (WHO 2010)	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 in drinking water systems and analytical achievability. The WHO evaluation notes the withdrawal of the JECFA PTWI and that no new value is available. The review notes that there does not appear to be a threshold for the key effects of lead.
WHO (WHO 2000b)	TC = 0.5 µg/m^3	Air guideline (based on an annual average) established for lead based on an objective of 98% of the general population having a blood lead concentration of < 10 µg/dL, where the median blood lead levels would be no more than 5.4 µg/dL.
EFSA (EFSA 2010b)	PbB levels relevant for critical health effects Developmental effects in children: 1.2 µg/dL Renal effects in adults: 1.5 µg/dL Cardiovascular effects in adults: 3.6 µg/dL	Based on benchmark dose response levels for 1% change in IQ or blood pressure (BMDL01) and a 10% change in prevalence of chronic kidney disease (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 that may have elevated blood lead levels. The level is intended to trigger education, investigation and monitoring.

The more recent reviews of lead completed by EFSA (EFSA 2010b) and the UK DEFRA (UK DEFRA & EA 2014) have focused on the critical health endpoints for adults and children as identified in other reviews but then used benchmark dose (BMD) modelling methods to identify blood lead levels associated with points of departure considered to represent significant health outcomes. These blood lead goals are more conservative than those identified and adopted by

the NHMRC (NHMRC 2015b). These benchmark doses are then related to blood lead concentrations using 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 can be summarised for the critical health endpoints identified.

Neurobehavioural effects in children

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

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

This blood lead concentration was then related to a daily intake of lead using the IEUBK model, which is suitable for children and consistent with the blood lead modelling utilised in Australia (NEPC 1999 amended 2013e). Based on this modelling, an intake of **1.4 µg/kg/day** results in a blood lead level of 3.5 µg/dL for children. This is the intake adopted in this assessment for the evaluation of potential health effects in children, exposed to lead, via all exposure pathways.

For the assessment of inhalation exposures and inhalation value of **2 µg/m^3** has been derived assuming children (aged 2-3 years) have a body weight of 15 kg and inhale an average of 9.5 m^3 air/day (enHealth 2012a).

Cardiovascular effects (hypertension) in adults

This type of approach can also be applied to data for adults.

The evaluation by DEFRA considered 4 human studies that related blood lead levels with increases in systolic blood pressure (Glenn, Barbara S. et al. 2006; Glenn, B. S. et al. 2003; Nash et al. 2003; Vupputuri et al. 2003).

The modelling undertaken was based on a 1% response level (BMD01) for a 1% increase in systolic blood pressure (SBP) (which is an increase of 1.2 mmHg above a baseline of 120 mmHg). This change in blood pressure was determined to be a significant health effect as it is within the range of observable effects and can have significant consequences for human health at a population level. There is still some debate as to whether a 1% increase is significant for an individual. Evaluation of the BMD modelling from the 4 studies identified blood lead levels predicted in the range 1.6 to 13.3 µg/dL could be related to this 1% response level. The range indicates some variability from the studies. The average was 3.6 to 6.1 µg/dL. The value of 3.6 µg/dL (rounded to 3.5 µg/dL by DEFRA) was identified as a point of departure for the assessment of these effects. This is the same blood lead concentration as discussed above to be protective for children.

The intake of lead that corresponds to these blood lead levels outlined above were modelled by DEFRA on the basis of the USEPA Adult Lead Model (ALM) and the Carlisle and Wade (Carlisle & Wade 1992) model. The Carlisle and Wade model was adopted by EFSA (EFSA 2010b) and the ALM is consistent with the modelling undertaken in Australia for adult lead exposures (NEPC 1999 amended 2013e).

Based on this modelling, for a blood lead level of 3.5 µg/dL an intake of **1.3 µg/kg/day** is derived using the Carlisle and Wade model. A more conservative value of 0.6 µg/kg/day was derived on the basis of the ALM.

Renal effects in adults

There are other effects that are also relevant for adults.

One study involving 14,778 adults was adopted for the evaluation of effects on the kidney. The effects on kidney function included reduced estimated glomerular filtration rate (eGFR) and were found to be related to blood lead levels (Navas-Acien et al. 2009).

The modelling undertaken was based on a 10% response level (BMD10) – i.e. a GFR below 60 mL/1.73 m^2 body surface/min. This is a level that is considered to have significant consequences on human health on a population basis. In addition, chronic exposures to lead that lead to chronic GFR levels below this level could be harmful to an individual. Evaluation of the BMD modelling (using a large number of different models) identified blood lead levels predicted in the range 1.5 to 2.7 µg/dL to reach this 10% response level. It is acknowledged that the nature of the GFR endpoint is complex and low levels of exposure are not yet confirmed to be causative. A pragmatic low value of 1.6 µg/dL may be considered as a point of departure for the assessment of these effects. The DEFRA review also considered a BMD20 level of 3.5 µg/dL in the consideration of the uncertainties associated with the studies relating to renal effects.

The intake of lead that corresponds to the blood lead levels outlined above were modelled by DEFRA on the basis of the USEPA Adult Lead Model (ALM) and the Carlisle and Wade (Carlisle & Wade 1992) model. The Carlisle and Wade model was adopted by EFSA (EFSA 2010b) and the ALM is consistent with the modelling undertaken in Australia for adult lead exposures (NEPC 1999 amended 2013e).

Based on this modelling, for a blood lead level of 1.6 µg/dL an intake of **0.6 µg/kg/day** is derived, and for a blood lead level of 3.5 µg/dL an intake of 1.3 µg/kg/day using the Carlisle and Wade model. More conservative values of 0.3 to 0.6 µg/kg/day were derived on the basis of the ALM.

For the purpose of this assessment a lead intake of **0.6 µg/kg/day** has been adopted as protective of renal effects in adults. This value will also be protective for cardiovascular effects in adults. This intake has been adopted for all pathways of exposure. For the assessment of inhalation exposures and inhalation value of **2 µg/m^3** has been derived assuming an adult body weight of 70 kg and inhale an average of 20 m^3 air/day (enHealth 2012a; USEPA 2009b).

Summary of TRVs adopted:

Based on the discussion above, the following TRVs have been adopted for the assessment of intakes of lead, from all sources:

- Children: 1.4 µg/kg/day for oral and dermal intakes and 2 µg/m3 for inhalation

- Adults: 0.6 µg/kg/day for oral and dermal intakes and 2 µg/m3 for inhalation

It is noted that, based on the above review, the TRV adopted for adults is lower (more conservative) than the TRV adopted for children. While this may be a little counter-intuitive, given that children are more sensitive than adults, the following should be noted:

- The TRVs adopted for adults and young children only differ by a factor of approximately 2, which is considered to be small in terms of the uncertainty and variability inherent in the derivation of TRVs from the available studies and using the available models.
- The TRVs have been derived
  - using different key health endpoints for children (neurobehavioural) and adults (kidney)
  - utilising data from studies specific to these effects in these age groups
  - incorporating different statistical models to identify relevant points of departure as a blood lead level and
  - utilising different biokinetic models (different for young children and adults) to estimate what intakes correspond to the adopted point of departures for each of the key health effects.

  As a result of these calculations, the TRVs derived for adults and children would be expected to differ somewhat.
- With consideration of the variability noted in the above point, review of the range of points of departure derived (as a blood lead level) and the relevant intakes (from the biokinetic models) indicates that there is some crossover with the range of TRVs that may be derived for adults and children.
- The TRVs adopted in this assessment have been based on a pragmatic but reasonable review of the range of TRVs that are derived from the above approach. In addition, given community concern in relation to the impact of lead emissions from the Project on individuals with kidney disease (refer to Table 3.3 in the main report), the use of a conservative approach in the identification of TRVs that are protective of these health effects in adults is appropriate.
- The approach adopted for this assessment leads to more conservative TRVs than would normally be used in environmental assessments using the nationally recommended guidance.

To reiterate, the approach adopted here is more conservative than the approach adopted in the development of the health investigation levels in the NEPM and more conservative than the approach recommended by the NHMRC (NEPC 1999 amended 2013e; NHMRC 2015a, 2015b). All of these calculate guidelines based on either 5 or 10 µg/dL for the blood lead concentrations whereas the assessment described here uses an endpoint of either 1.6 or 3.5 µg/dL.

Annexure C

Toxicity Summaries for Other Metals, Respirable Crystalline Silica and Hydrogen Cyanide

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C1 INTRODUCTION

This appendix presents toxicity summaries relevant to the metals evaluated in this HHRA. A detailed toxicity summary specific to lead is included in **Appendix B**.

The objective of the toxicity assessment is to identify toxicity values for CoPCs that can be used to quantify potential risks to human health associated with calculated intake. Toxicity can be defined as *"the quality or degree of being poisonous or harmful to plant, animal or human life"* (NEPC 1999 amended 2013c).

The objective of the toxicity review is to identify appropriate quantitative toxicity values for each chemical and pathway of exposure (oral, dermal or inhalation) that can be used to quantify risk. This has involved the following key steps:

1. Identify the relevant health end-points, and, where carcinogenicity is identified, the mechanism of action. This has enabled the identification of whether a threshold or non-threshold dose-response approach is appropriate; and

2. Identify the most appropriate quantitative value for the assessment of threshold or non-threshold effects. This includes consideration of susceptible populations, where relevant.

Step 1: Identify Health End-Points and Dose-Response

The quantitative assessment of potential risks to human health for any chemical requires the consideration of the relevant (and most sensitive) health end-points, and, where carcinogenicity is identified, the mechanism of action needs to be reviewed and considered.

For chemicals 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 (using toxicity reference values (TRVs) such as an acceptable/tolerable daily intake (ADI/TDI) or a 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 TRVs are considered "safe", or not associated with an adverse health risk (NHMRC 1999b).

Where the chemical has the potential for carcinogenic effects, the mechanism of action needs to be understood as this defines the most appropriate dose-response approach to be considered. 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 1999b). Hence, no safe-dose or threshold is assumed (hence any exposure is associated with some level of incremental lifetime risk), and assessment of exposure is based on a linear or non-threshold approach using TRVs termed as 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 chemicals. For these chemicals, carcinogenic effects are associated with indirect mechanisms (that do not directly interact with genetic material) where a threshold is believed to exist, and are characterised using threshold TRVs such as an ADI/TDI or a TC.

The United States Environmental Protection Agency (USEPA 2005b) requires the mode of action for carcinogenicity to be clearly understood before accepting a threshold approach for assessing exposures to non-genotoxic carcinogens. Where data are lacking and the mechanism is poorly understood, the default is to adopt a non-threshold approach. Current industry practice in Australia is to not simply default to a non-threshold approach where understanding (or data) is lacking (as in the US); rather, the approach is to provide an adequate review of available information to enable a decision to be made based on the weight of evidence (enHealth 2012b; NEPC 1999 amended 2013c).

Step 2: Identify Appropriate Quantitative Toxicity Reference Values

Once the most appropriate dose-response approach has been reviewed, quantitative TRVs can be selected for use in a risk assessment in accordance with the current Australian guidance (enHealth 2012b; NEPC 1999 amended 2013c).

C2 ARSENIC

Several comprehensive reviews of arsenic in the environment and toxicity to humans are available (ATSDR 2007a; NRC 2001; UK EA 2009a, 2009d; 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 2007a). Under reducing conditions arsenite (AsIII) is the dominant form and in well oxygenated environments, arsenate (As V) 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 2007a).

Background

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 calculated separately based on available information on the existing environment.

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

Classification

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

Identification of Toxicity Reference Values

Oral

Arsenic is a known human carcinogen, based on human epidemiological studies that show skin and internal cancers (in particular bladder, liver and lung) associated with chronic exposures to arsenic in drinking water. The research available on arsenic carcinogenicity is dominated by epidemiological studies (which have limitations) rather than animal studies which differs from carcinogenic assessments undertaken on many other chemicals. The principal reason for the lack of animal studies is because arsenic has not been shown to cause cancer in rodents (most common species used in animal tests) due to interspecies differences between rodents and humans.

Review of arsenic by (IARC 2012b) has concluded the following:

- For inorganic arsenic and its metabolites, the evidence points to weak or non-existent direct mutagenesis (genotoxicity), which is seen only at highly cytotoxic concentrations.
- Long-term, low-dose exposures to inorganic arsenic (more relevant to human exposure) is likely to cause increased mutagenesis as a secondary effect of genomic instability. While the mechanism of action (MOA) is not fully understood it is suggested by (IARC 2012b) that it may be mediated by increased levels of reactive oxygen species, as well as co-mutagenesis with other agents. The major underlying mechanisms observed at low concentrations include the rapid induction of oxidative DNA damage and DNA-repair inhibition, and slower changes in DNA-methylation patterns, aneuploidy, and gene amplification.
- Inhibition of DNA repair leads to co-carcinogenicity.

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 2005b) as there remains uncertainty on the carcinogenic MOA for arsenic (Sams et al. 2007). Further research is required to define and review the MOA prior to the USA revising the dose-response approach currently adopted. Inherent in the current US approach (where a non-threshold slope factor is derived) are some key uncertainties that likely result in an overestimate of risk, which include:

- the choice of the cancer endpoint;
- the choice of the mathematical model used to estimate risk (shape of the dose-response curve at low doses) as there is no clear biological basis for extrapolation; and

- the assumptions used to estimate exposure from studies (primarily epidemiological studies) (Boyce et al. 2008; Brown 2007; Chu & Crawford-Brown 2006; Lamm & Kruse 2005; SAB 2005).

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

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

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

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

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

The review of arsenic by the New Zealand Ministry for the Environment (MfE 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 are 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 2018). 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 2018) 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 2010a). The review concluded that the PTWI was "no longer appropriate as data are available that shows inorganic arsenic causes cancer of the lung and bladder in addition to skin, and that the range of adverse effects had been reported at exposures lower than those reviewed by the JECFA" in establishing the PTWI. Modelling conducted by EFSA considered the available epidemiological studies and selected a benchmark response (lower limits) of 1% extra risk ($BMBL_{01}$). $BMBL_{01}$ range from 0.3 to 8 µg/kg/day for cancers of the lung, bladder and skin. The CONTAM Panel (EFSA 2010a) concluded that the overall range of $BMDL_{01}$ values of 0.3 to 8 µg/kg/day should be used for the risk characterisation of inorganic arsenic rather than a single reference point, primarily due to the number of uncertainties associated with the possible dose-response relationships considered. On this basis, it would not be appropriate to consider just one value in the range presented.

The determination of an appropriate TRV requires a single value that can be used in a quantitative assessment, rather than a wide range of values. The value chosen needs to be considered adequately protective of the population potentially exposed. The determination of an appropriate TRV for arsenic in soil in Australia has, therefore, considered the following:

- The studies considered in the derivation of the different ranges of BMDL values (EFSA 2010a; WHO 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 2010a; 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 2010a)) have shown that populations with poor nutrition (and health status) are more susceptible to the prevalence and severity of arsenic-related health effects.
- The largest of the studies conducted was within rural Asian populations which differ from Australian populations with respect to generic lifestyle factors.

In view of the above, consideration of the lower end of the range of BMDL values available from WHO and EFSA (EFSA 2010a; WHO 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 2010a; 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 2007a) 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 2013e).

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

Given the above, there is some basis for the assessment of inhalation exposures to arsenic to adopt an appropriate threshold value but the available epidemiological studies associated with exposures in copper smelters suggest a linear or non-threshold approach may be relevant. The WHO (2000) review of arsenic by WHO (WHO 2000b) also suggested the use of a linear (non-threshold) approach to the assessment of inhalation exposures to arsenic. The assessment presented is limited and essentially adopts the US approach with no discussion or consideration of the relevance of the linear model adopted. The review by WHO (WHO 2001b) with respect to inhalation exposures and lung cancer provides a more comprehensive review and assessment. The review presented identified that a linear dose–response relationship is supported by the occupational and epidemiological studies. The three key studies associated with copper smelters in Tacoma, Washington (USA), Anaconda, Montana (USA) and Ronnskar (Sweden) (as summarised in (WHO 2001b)) demonstrate a statistically significant excess risk of lung cancer at cumulative exposure levels of approximately ≥750 µg/m^3 per year.

The relevance of inhalation values derived from studies near smelters to the assessment of contaminated arsenic in soil in areas away from smelters 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; and
- Background Intakes from other sources (as % of TRV) = 50% for oral and dermal.

C3 CADMIUM

General

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

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 2012c).

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 2012c).

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 2004c). 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 calculated separately based on available information on the existing environment.

Classification

IARC has classified cadmium and cadmium compounds as a Group 1 agent (i.e., carcinogenic to humans) based on additional evidence of carcinogenicity in humans and animals. It is noted that there is limited evidence of carcinogenicity in experimental animals following exposure to cadmium metal (IARC 2012b).

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 2012c). 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.

Oral (and Dermal) Intakes

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

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 updated 2018)	TDI = 0.0007 mg/kg/day	The threshold oral value available from the ADWG (NHMRC 2011) 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.

Source	Value	Basis/Comments
International		
JECFA (WHO 2010)	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 2012c)	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) (NHMRC 2011 updated 2018).

Inhalation Exposures

Inhalation of cadmium has been associated with carcinogenic effects (as well as others). Sufficient evidence is available (IARC 1993) to conclude that cadmium can produce lung cancers via inhalation (IARC 2012b). While cadmium is thought to be potentially genotoxic, the weight of evidence is not clear. In addition, epidemiology studies associated with lung cancer have confounding issues that limit useful interpretation (WHO 2000c). 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 2010);
- Dermal absorption (DAF) = negligible (0%); and
- Inhalation TRV (TRV_I) = 0.000005 mg/m^3 (WHO 2000c).

C4 CHROMIUM

For this assessment, all chromium present is assumed to be chromium VI, the most toxic form of chromium.

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

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

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

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

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

Background

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

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

resulting in some doubling up of intakes from food sources. The average intake of Cr VI is estimated to be 0.13 µg/kg/day for 2-3 year olds, approximately 10% of the recommended oral TRV.

- No data on Cr VI in air is available for Australia. Intakes of Cr VI from air may comprise up to 30% of total chromium (Baars et al. 2001), which has been reported up to 1.5 ng/m^3 by (Baars et al. 2001) and up to 3 ng/m^3 by (UK DEFRA & EA 2002a). It is noted that concentrations of Cr VI in Europe and the UK are expected to be higher than in Australia due to the potential for long-range atmospheric transport from a greater proportion of industry in these general regions. Based on the recommended TRV for particulate phase Cr VI, these conservative air concentrations comprise less than 1% of the TC and are assumed negligible.

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

Classification

IARC (IARC 2012b) has classified Cr VI compounds as Group 1 carcinogens: carcinogenic to humans based on: sufficient evidence in humans for the carcinogenicity of Cr VI compounds as encountered in the chromate production, chromate pigment production and chromium plating industries.

Chromium is classified by the US EPA as a Group A: known human carcinogen by the inhalation route, with carcinogenicity by the oral route of exposure noted to be Group D: not classified (USEPA 1998).

Review of Available Values/Information

Oral

There are limited data available regarding the carcinogenic potential of ingested Cr VI. Cr VI compounds appear to be genotoxic and some reviews (Baars et al. 2001) suggest that a non-threshold approach is relevant to all routes of exposure. Some drinking water studies (NTP 2008) are available that show a statistically significant increase in tumours in rats and mice. However, there are currently no peer-reviewed data available to determine a quantitative non-threshold value for ingestion of Cr VI compounds (note a value has been recently published by (OEHHA 2011) using a non-threshold approach). There is also some suggestion (De Flora et al. 1997; Jones 1990) that there may be a threshold for the carcinogenicity of Cr VI based on a hypothesis that it is a high dose phenomenon where the dose must exceed the extracellular capacity to reduce Cr VI to Cr III.

The following are available for oral intakes from Level 1 Australian and International sources

Toxicity reference values for Cr VI – Oral

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 Updated 2016)	No evaluation available	The ADWG does not specifically derive a guideline; however it references the WHO DWG assessment, where the basis for derivation is not clear. No quantitative toxicity values can be obtained from these sources.

Toxicity reference values for Cr VI – Oral (Cont'd)

Source	Value	Basis/Comments
International		
WHO DWG (WHO 2011a)	No evaluation available	Current guideline based on limit of detection as no adequate toxicity studies were available to provide the basis for a NOAEL. It is noted that chromium is included in the plan of work of rolling revisions to the WHO DWG (2011).
UK DEFRA & EA (UK DEFRA & EA 2002a)	TDI = 0.003 mg/kg/day	Adopted oral RfD from the USEPA.
RIVM (Baars et al. 2001)	TDI = 0.005 mg/kg/day	RIVM has adopted a provisional threshold TDI of 0.005 mg/kg/day based on a 1-year drinking water study in rats as used in the derivation of the former and current USEPA RfD (with a small difference in the application of uncertainty factors).
ATSDR (ATSDR 2012b)	MRL = 0.001 mg/kg/day	The chronic oral MRL is based on a $BMDL_{10}$ of 0.09 mg/kg/day for non-neoplastic lesions of the duodenum in a 2-year drinking water study in rats and mice (NTP 2008) and an uncertainty factor of 90. The study considered by ATSDR was not available when the other organisations (USEPA etc) reviewed Cr VI.
USEPA IRIS (USEPA 1998)	RfD = 0.003 mg/kg/day	The USEPA IRIS entry (last reviewed in 1998) derived an oral RfD of 0.003 mg/kg/day based on a NOAEL of 2.5 mg/kg/day from a 1-year drinking water study in rats and an uncertainty factor of 300 and modifying factor of 3 to address uncertainties in the study. The confidence level in the study, database and RfD is noted to be low.

It is recommended that the lower value derived by (ATSDR 2012b) be adopted for the assessment of oral exposures to Cr VI as the assessment provides the most current comprehensive assessment of the available studies, including a more recent key study (NTP 2008) not available at the time of review by other organisations. The values adopted by RIVM and the UK are essentially the same, using the study considered by the USEPA (McKenzie et al. 1958) in the derivation of the RfD. It is noted that review by Health Canada (Health Canada 2004) considered the study used by the USEPA to be of poor quality however it was utilised due to the lack of additional, better quality data.

Inhalation

Epidemiological studies have shown an association between exposure to Cr VI and lung cancer. These studies have involved chromate production, chromate pigment production and use, chromium plating, stainless steel welding, ferrochromium alloy production and leather tanning. Various Cr VI compounds have also been shown to be carcinogenic via inhalation in experimental animals. Cr VI has also been shown to be genotoxic. As noted by UK DEFRA & EA (UK DEFRA & EA 2002a), there is some suggestion that chromium-induced cancer of the respiratory tract may be exclusively a high-dose phenomenon with a threshold approach relevant to low-dose exposures but quantitative data is lacking.

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

The following are available for inhalation exposures for Cr VI particulates or dust from Level 1 Australian and International sources:

- No Australian guideline values are available for Cr VI.
- The USEPA (USEPA 1998) derived an inhalation RfC of 0.0001 mg/m^3 for Cr VI particulates based on lower respiratory effects in a subchronic rat study. The USEPA review of particulate exposures indicated chromium inhalation induced pneumocyte toxicity and suggested that inflammation is essential for the induction of most chromium inhalation effects and may influence the carcinogenicity of Cr VI compounds. The USEPA has also derived a separate RfC (lower) for exposure to chromic acid mists and dissolved Cr VI aerosols, which would be relevant for the assessment of an occupational environment.
- ITER (ITER 1998) derived an inhalation RfC of 0.0003 mg/m^3 for Cr VI particulates based on the same study as USEPA considered but the value derived was on the basis of an arithmetic average of benchmark concentrations for the pulmonary inflammation end point.

In addition, the following are also available:

- WHO (WHO 2000b) has derived a range of air guideline values based on an inhalation unit risk of 0.04 (μg/m3)$^{-1}$ derived from the mean of a number of occupational studies.
- USEPA (USEPA 1998) also derived a unit risk of 0.012 (μg/m3)$^{-1}$ derived from one occupational study (also considered by WHO).
- UK DEFRA & EA (UK DEFRA & EA 2002a) has derived an index dose of 0.001 µg/kg/day for Cr VI based on occupational inhalation studies based on a lung cancer end point, consideration of the WHO non-threshold approach and a target risk level of 10^{-4}.
- RIVM (Baars et al. 2001) has adopted a cancer risk value of 0.0025 µg/m^3 based on occupational inhalation studies based on a lung cancer end point, consideration of the WHO non-threshold approach and a target risk level of 10^{-4}. It is noted that a 10^{-4} target risk level is used for inhalation guidelines by (UK DEFRA & EA 2002a) and RIVM (Baars et al. 2001). The value results in guidelines that address background levels of Cr VI reported in ambient air, which range up to 30% of total chromium reported (up to 0.0015-0.0025 µg/m^3).
- ATSDR (ATSDR 2012b) has derived a chronic inhalation MRL for Cr VI aerosols and mists but this is not considered relevant to the derivation of toxicity reference values for soil contamination.

Recommendation

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

- Oral TRV (TRV_O) = 0.001 mg/kg/day (ATSDR 2012b);
- Inhalation TRV (TRV_i) = 0.0001 mg/m^3 (USEPA 1998); and
- Background intakes from other sources (as % of TRV) = 10% for oral/dermal intakes and 0% for inhalation.

C5 COBALT

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

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

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

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

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

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:

Toxicity reference values

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 Updated 2016)	No evaluation available	
International		
WHO DWG (WHO 2011a)	No evaluation available	
WHO (WHO 2006a)	TC = 0.0001 mg/m^3	The WHO (2006) derived a TC in air of 0.0001 mg/m^3 based on a NOAEC from an occupational inhalation study with conversions to address exposures by the general population. The WHO did not derive an oral threshold value due to the lack of suitable data
RIVM (Baars et al. 2001)	TDI = 0.0014 mg/kg/day TC = 0.0005 mg/m^3	RIVM (2001) derived a TDI of 0.0014 mg/kg/day based on a LOAEL of 0.04 mg/kg/day associated with cardiomyopathy from oral exposures in workers and an uncertainty factor of 30. TC based on a LOAEC of 0.005 mg/m^3 for interstitial lung disease in workers and an uncertainty factor of 100.
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 2006a) for oral and dermal routes of exposure; and
- Background intakes from other sources (as % of TRV) = 20% for oral intakes.

C6 COPPER

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 2018) 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 calculated separately based on available information on the existing environment.

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:

Toxicity reference values

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 Updated 2016)	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; and
- Background intakes for the general population = 0.08 mg/kg/day = 60% of the oral TRV.

C7 LITHIUM

Limited information is available for determining relevant toxicity reference values for lithium. The most current review is provided by the USEPA (USEPA 2008) and this is the only review that provides a quantitative value for the characterisation of toxicity. The following information is derived from the USEPA review.

Lithium (Li), an alkali metal, exists in two isotopic forms (^{7}Li and ^{6}Li) and is naturally present in soil and water. Lithium has numerous industrial and commercial uses including as a cell additive in electrolytic aluminium production, a catalyst of chemical reactors, a component of fluxes and brazing alloys, a component of batteries, specialized glass and ceramics, and a sanitizing agent for swimming pools, hot tubs and spas. Lithium carbonate and lithium citrate are also used for the therapeutic treatment of psychiatric disorders, primarily in the acute and long-term maintenance treatment of bipolar mood disorders.

The use of lithium as a long-term maintenance therapy in the treatment of bipolar affective disorders has led to an extensive body of literature on the adverse effects associated with oral lithium therapy. Adverse effects, which are observed in several organs and systems, are associated with the entire target therapeutic serum lithium concentration range, leading to treatment strategies based on a risk-benefit assessment for individual patients. Data reported in human studies are not sufficient to define the relationship between serum lithium concentrations and the development or severity of adverse effects, although it is generally accepted that the severity of adverse effects is related to serum lithium levels. Given the lack of adequate dose-response data, a single critical effect cannot be identified for lithium. Occupational and environmental oral exposure studies in humans are not available.

The most significant health effects identified in the human studies are adverse renal effects (specifically impaired renal concentrating ability and the production of excessively dilute urine), as well as neurological effects (lethargy, fatigue, weakness, tremor and cognitive impairment), endocrine and thyroid effects, cardiovascular effects (EKG changes), gastrointestinal effects (nausea, vomiting, diarrhea and abdominal cramping), haematological effects and developmental effects.

The available animal data provide supportive evidence that lithium produces adverse effects in several organs and systems at exposure levels that result in serum lithium concentrations in same range as that targeted for therapeutic use in humans.

Based on the available data, the USEPA has derived a provisional RfD for lithium. The value is provisional as the available data and animal studies are not as robust as normally available for determining the LOAEL or NOAEL.

The provisional RfD is based on a LOAEL for adverse effects in several organs and systems, from patient data. The LOAEL adopted is 2.1 mg/kg/day, and the USEPA has applied a 1000 fold uncertainty factor (10 for the use of a LOAEL, 10 for susceptible individuals and 10 for database deficiencies), resulting in an RfD of 0.002 mg/kg/day.

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

Where lithium is not being taken for therapeutic purposes, intakes from food and water are considered to be negligible (USEPA 2008).

Recommendation

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

- Oral TRV (TRV_O) = 0.002 mg/kg/day (USEPA 2008) for all routes of exposure; and
- Background intakes for the general population are negligible.

C8 MANGANESE

General

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

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

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

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

Background

Review of current information from Australia indicates the following:

- Review of manganese by FSANZ indicates that for young children aged 2-3 years, intakes range from a mean of 0.19 mg/kg/day to a 90th percentile of 0.26 mg/kg/day. Dietary intakes of manganese reported by the WHO are approximately 0.06 mg/kg/day for young children. Estimates provided by ATSDR suggest that adult intakes of food are 3.8 mg/day (or 0.05 mg/kg/day) (ATSDR 2012a; FSANZ 2011; Lindon & Sabordo 1996).
- Typical concentrations of manganese reported in the ADWG are less than 0.01 mg/L, resulting in an intake (1 L/day and body weight of 15.5 kg) by toddlers of 0.00076 mg/kg/day (NHMRC 2011 updated 2018).
- Based on the above background intakes for young children, it has been assumed that background oral intakes comprise 50% of the recommended oral TRV.
- Manganese was reported in ambient air data collected in NSW where concentrations (24-hour averages) in urban, regional and industrial areas assessed ranged from 3.7 to 119 ng/m^3 (average of 18 ng/m^3) (NSW DEC 2003). Typical concentrations in air have been reported by ATSDR to be 23 ng/m^3, consistent with that reported by NSW DEC (2003) (ATSDR 2012a). These background concentrations comprise (based on average concentrations) approximately 15% of the recommended inhalation TRV. A conservative background of 20% of the inhalation TRV could be assumed for intakes from air.

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

Classification

The International Agency for Research on Cancer (IARC) has not classified manganese. The USEPA has classified manganese as Group D: not classifiable.

Review of Available Values/Information

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

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

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 updated 2018)	Safe level of 10 mg/day	The ADWG (NHMRC 2011) derived a health based guideline of 0.5 mg/L based on a level of 10 mg/day which is the amount of manganese that can be safely consumed from all sources, referenced from WHO 1973 evaluation.
International		
WHO DWG (WHO 2017)	TDI = 0.05 mg/kg/day	The current WHO DWG (2017) has not established a guideline for drinking water as the compound is not considered to be of health concern at the levels found in drinking water. The review notes that a health-based guideline of 0.4 mg/L can be derived based on the upper range value of manganese intake of 11 mg/day from dietary studies (IOM 2001) and an uncertainty factor of 3 (to allow for the increased bioavailability of manganese from water), which results in a TDI of 0.05 mg/kg/day for 70kg adult. The guidance also notes that the presence of manganese in drinking water will be objectionable (water discolouration) above 0.05 mg/L.
WHO (WHO 1999a)	TC = 0.00015 mg/m^3	Tolerable concentration or guideline value derived by WHO on the basis of the same study considered by the USEPA (IRIS 2012) and ATSDR (2012), with the guideline value derived on the basis of a NOAEL of 0.03 mg/m^3 for neurotoxicological effects from a benchmark dose (BMD) analysis, adjustment for continuous exposure (5/7 x 8/24) and an uncertainty factor of 50. The value derived is similar to that from ATSDR (2012) with the main difference being the application of the BMD model. No oral guideline value was provided.
Health Canada (Health Canada 2010)	RfC = 0.00005 mg/m^3	RfC derived based on most sensitive benchmark dose analysis associated with neurotoxicological effects in an occupational inhalation study. A range of RfCs were derived that varied from 0.00005 to 0.00014 mg/m^3. The range derived is consistent with values derived from ATSDR and WHO.
ATSDR (ATSDR 2012a)	Interim oral value of 0.16 mg/kd/day Inhalation MRL = 0.0003 mg/m^3	No oral MRLs have been derived by ATSDR; however, they provide an interim guidance value of 0.16 mg/kg/day based on a tolerable upper intake level of 11 mg/day. Chronic inhalation MRL derived on the basis of a benchmark concentration (at the lower 95% confidence limit for the level of manganese exposure expected to result in 10% response rate) $BMCL_{10}$ (adjusted for continuous exposure) of 0.03 mg/m^3 associated with neurobehavioural effects in an occupational study and an uncertainty factor of 100.

Source	Value	Basis/Comments
International (Cont'd)		
USEPA (USEPA IRIS)	RfD = 0.14 mg/kg/day RfC = 0.00005 mg/m^3	RfD (last reviewed in 1993) based on a NOAEL of 0.14 mg/kg/day associated with CNS effects in a number of dietary human studies and an uncertainty factor of 1. The USEPA also note that individual requirements for and effects associated with manganese exposure may be highly variable and that some individuals may consume more than 10 mg/day of manganese without any cause for concern. RfC (last reviewed in 1993) based on the same study considered by ATSDR (2012) however the USEPA considered the LOAEL (HEC) of 0.05 mg/m^3 and applied an uncertainty factor of 1000.

As manganese toxicity via inhalation has been shown to be more significant than via oral intakes, it is reasonable that quantitative values for inhalation exposures are significantly lower than for oral exposures. Based on the available data, an oral threshold value of 0.16 mg/kg/day as derived by ATSDR (2012) in the most recent detailed review of manganese toxicity is recommended for use in this assessment. It is noted that the basis for the value is consistent with the upper range of manganese intake considered by the USEPA, NHMRC and WHO (NHMRC 2011 updated 2018; USEPA IRIS; WHO 2017) (especially if the additional uncertainty factor of 3 used in the WHO drinking water guidelines is not included for exposures from soil (based on increased bioavailability from water)).

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

Recommendation

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

- Oral TRV (TRV_O) = 0.16 mg/kg/day (ATSDR 2012a);
- Dermal absorption (DAF) = negligible (0%); and
- Inhalation TRV (TRV_I) = 0.00015 mg/m^3 (WHO 1999a).

C9 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 TI 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 2018) 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 1999a) of intakes associated with amalgam fillings in Australian children and adults (based on average number of fillings of 0.5 and 8 respectively) provides an reasonable estimate of daily mercury absorption per person of about 0.3 µg for children and 3.5 µg for adults. The estimate for children is expected to be conservative as 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 2009e) 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 calculated separately based on available information on the existing environment.

Health Effects

The following information is available from UK (UK EA 2002, 2009e) 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 1995a).

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 2012b) 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 2016)	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 increases in male rates was considered as the point of departure. A 100 fold uncertainty factor was applied.

Toxicity Reference Values for Inorganic and Elemental Mercury (Cont'd)

Source	Value	Basis/Comments
International (Cont'd)		
WHO (WHO 2000d)	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 2009e)	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 1991b). 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 2009e), 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); and
- Background intakes for the general population are 40% for ingestion and dermal and 10% for inhalation.

C9 Nickel

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

Nickel is a silvery white metal that is stable under environmental conditions. It occurs naturally in the earth's crust. It is the 24th most abundant element and is primarily found as oxides or sulfides (ATSDR 2005a). Nickel is extracted from mined ore via pyro- and hydrometallurgical refining processes. Most nickel is used for the production of stainless steel and other nickel alloys with high corrosion and temperature resistance. The primary sources of nickel emissions into the atmosphere are the combustion of coal and oil for heat or power generation, the incineration of waste and sewage sludge, nickel mining and primary production, steel manufacture, electroplating and cement manufacturing (WHO 1991a).

The chemistry of nickel is complex, and the toxicological properties of the various compounds depend on physicochemical characteristics, surface chemistry, solubility, geological history. Hence it is important that any site specific assessment of nickel consider these issues.

Background

Review of current information from Australia indicates the following:

- Dietary intakes of nickel have been assessed in the 22nd Australian Total Diet Survey (FSANZ 2008), where mean intakes reported for children aged 2-3 years was reported to be 83-91 µg/day, or 6.2 to 6.9 µg/kg/day. Estimates provided by (ATSDR 2005a) and UK (UK EA 2009c) suggest that adult intakes from food are 69-162 µg/day (up to 2.3 µg/kg/day) and 130 µg/day (1.9 µg/kg/day) respectively. Intakes for children (ATSDR 2005a) range from 6.9 µg/kg/day (6-11 months old) to 9.5 µg/kg/day (children aged less than 18).
- Typical concentrations of nickel reported in the ADWG (NHMRC 2011 updated 2018) are less than 0.01 mg/L. resulting in an intake (1 L/day and body weight of 15.5 kg) by toddlers of 0.6 µg/kg/day.
- Based on intakes estimated from Australian data, background intakes by young children are approximately 7 µg/kg/day, up to 60% of the recommended oral TRV.
- Nickel was reported in ambient air data collected in (NSW DEC 2003) where concentrations (24-hour averages) in urban, regional and industrial areas assessed ranged from 0.86 to 20 ng/m^3 (average of 3.5 ng/m^3). Typical background concentrations in air have been reported by (UK EA 2009c) to be from 0.3 to 4.5 ng/m^3, consistent with that reported by (NSW DEC 2003). These background concentrations comprise (based on average concentrations) approximately 17% of the recommended TC. A conservative background of 20% of the recommended inhalation TRV has been assumed for intakes from air.

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

Classification

(IARC 2012b) classified nickel compounds as Group 1: carcinogenic to humans. The IARC working group noted that the overall evaluation of nickel compounds as a group was undertaken on the basis of the combined results of epidemiological studies, carcinogenicity studies in experimental animals, and several types of other relevant data supported by the underlying assumption that nickel compounds can generate nickel ions at critical sites in their target cells.

It is noted that the USEPA has classified nickel refinery dust as Group A: human carcinogen.

Review of Available Values/Information

The toxicity of nickel is complex and appears to differ via the different routes of exposure and hence the following addresses oral exposures separately from inhalation exposures.

Oral

Review in the (WHO 2011a) concluded that there was no substantial evidence that nickel compounds may produce cancers other than in the lung or nose in occupationally exposed persons. Limited animal studies on carcinogenic effects after oral exposures to nickel compounds did not show any significant increase in tumours. Review by the UK (UK EA 2009c) noted that while not all expert groups (WHO, US EPA, EU) have explicitly concluded that there is no carcinogenic concern from ingested nickel, none of those evaluating oral exposure concluded that a non-threshold approach should be undertaken. Hence the assessment of oral intakes on the basis of a threshold approach is reasonable. The following quantitative values are available from Level 1 Australian and International sources:

Toxicity reference values – Oral

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 Updated 2016)	TDI = 0.005 mg/kg/day	The ADWG derived a health based guideline of 0.02 mg/L based on NOEL of 5 mg/kg/day associated with organ-to-body-weight ratios in a 2-year rat study and an uncertainty factor of 1000. An additional factor of 10 was not included to address carcinogenicity as this was only relevant for inhalation exposures, not oral exposures.
International		
WHO DWG (WHO 2011a)	TDI = 0.012 mg/kg/day	The current WHO DWG, based on a review conducted in 2005, derived a guideline of 0.07 mg/L based on a TDI of 0.012 mg/kg/day derived from a LOAEL of 0.012 mg/day established from a study associated with hand eczema in nickel-sensitised volunteers who had fasted prior to administration of the nickel salt ((Nielsen et al. 1999)). This study (using fasted patients) was considered conservative and an uncertainty factor of 1 was adopted. The review also noted that a general guideline value of 0.13 mg/L could also be derived from a TDI of 0.022 mg/kg/day on the basis of a two-generation study in rats where a NOAEL of 2.2 mg/kg/day could be determined for all end-points studied and an uncertainty factor of 100.
RIVM (Baars et al. 2001)	TDI = 0.05 mg/kg/day	TDI derived on the basis of a NOAEL of 5 mg/kg/day (same study considered in the ADWG) and an uncertainty factor of 100.
UK EA (UK EA 2009c))	TDI = 0.012 mg/kg/day	Adopted the WHO evaluation presented in the WHO DWG.
TERA (TERA 1999)	RfD = 0.008 mg/kg/day	RfD derived for soluble nickel salts on the basis of a LOAEL of 7.6 mg/kg/day associated with kidney effects in rats and an uncertainty factor of 1000. The value derived was in addition to the diet rather than total intake.
ATSDR (ATSDR 2005a)	No oral MRL derived	
USEPA (IRIS 2012)	RfD = 0.02 mg/kg/day	RfD (last reviewed in 1991) based on a NOAEL of 5 mg/kg/day (same study as considered in the ADWG) and an uncertainty factor of 300.

Inhalation

Inhalation exposures to nickel are complex, with the toxicity dependent on the form of nickel present. The most recent review of nickel toxicity by UK Environment Agency (UK EA 2009c) indicates the following with respect to the consideration of inhalation exposures:

- Nickel and compounds are established carcinogens via the inhalation route with tumours of the respiratory tract a consequence of occupational exposure to both soluble and insoluble nickel salts.
- Nickel compounds are generally considered to be genotoxic; however the mechanism of action associated is not well understood. The lack of understanding has resulted in a conservative approach that genotoxicity is critical in the development of tumours and that a non-threshold approach may be appropriate.
- Non-threshold assessments of inhalation cancer risk have relied on occupational studies to derive a quantitative value (unit risk). These occupational studies relate to specific nickel compounds in the occupational environment including nickel subsulfide (WHO 2000b) and nickel refinery dusts (USEPA IRIS).
- (WHO 1991a) notes that very high concentrations of nickel are required to produce teratogenic and genotoxic effects.
- Review by RIVM (Baars et al. 2001)suggested the mechanism of action appears to be via a cytotoxic effect and that a threshold was appropriate for inhalation exposure to nickel. Review by UK Environment Agency (UK EA 2009c) also suggested a non-genotoxic threshold mechanism of action and that a threshold can be considered.
- A threshold value can be adopted for inhalation exposure that is protective of both carcinogenic and non-carcinogenic effects. However it is noted that the assessment of carcinogenic issues relies on the non-threshold values available and acceptance of a 1 in 100,000 excess lifetime cancer risk.

Nickel is not volatile and hence inhalation exposures are only relevant for dust intakes. Carcinogenic end points are expected to be of particular importance if they are derived from nickel refinery dust or nickel subsulfide, but dust generated from soil contamination is not likely to be significant and hence the consideration of carcinogenic effects using a non-threshold approach may not be appropriate. It is therefore appropriate to consider intakes on the basis of a threshold approach associated with the most significant end point which includes both carcinogenic and non-carcinogenic effects. These issues were considered by UK Environment Agency (UK EA 2009c), where a threshold value was recommended that was considered protective of both carcinogenic and non-carcinogenic effects.

The following quantitative threshold values (including guideline values derived to be protective of carcinogenic effects) are available for the assessment of inhalation exposures from Level 1 Australian and International sources:

Toxicity reference values – Inhalation

Source	Value	Basis/Comments
Australian – No guidelines derived		
International		
WHO (WHO 2000b)	GV = 0.025 µg/m^3	Review by WHO established a range of air guideline values for nickel based on a non-threshold approach with a unit risk derived from occupational studies associated with nickel subsulfate. It has been assumed that the nickel ion is the active agent in the occupational studies and therefore the studies are relevant to all nickel exposures. The guideline value noted here is based on an excess lifetime cancer risk of 1 in 100 000.
Health Canada (Health Canada 1994)	TC = 0.0035 µg/m^3 TC05 = 0.07 mg/m^3	Tolerable concentration (TC) derived on the basis of a threshold approach from a LOAEC (HEC) of 0.0035 mg/m^3 associated with respiratory effects from nickel sulfate in rats, and an uncertainty factor of 1000. Health Canada also derived a tumorigenic concentration of 5%, TC05, based on epidemiology studies of exposed workers at two nickel refineries (based on nickel sulphate and nickel chloride), and derived from the non-threshold dose-response curves.
RIVM (Baars et al. 2001)	TC = 0.05 µg/m^3	Tolerable concentration (TC) derived on the basis of a threshold approach from a NOAEC (HEC) of 0.005 mg/m^3 associated with respiratory effects in rats, and an uncertainty factor of 100.
UK Air Quality Standards (UK Air Quality Standards 2010)	TC = 0.02 µg/m^3	TC derived assuming a threshold approach is appropriate, based on a LOAEL of 0.02 mg/m^3 associated with respiratory tract tumours in occupational nickel exposures, and an uncertainty factor of 1000. TC derived is similar to but slightly lower than that derived on the basis of inflammatory response in experimental animals.
UK EA (UK EA 2009c)	TC = 0.02 µg/m^3	Adopted evaluation of EPAQS, noting the value derived is protective of carcinogenic and non-carcinogenic effects.
OEHHA (OEHHA 2009)	REL = 0.014 µg/m^3	Chronic inhalation reference exposure level (REL) for nickel and nickel compounds (except nickel oxide where a higher REL is derived) based on a NOAEL (HEC) of 0.0016 mg/m^3 associated with respiratory/lung effects in a 104-week rat study, and an uncertainty factor of 30. OEHHA also provide a non-threshold unit risk for nickel and compounds.
TERA (TERA 1999)	RfC = 0.2 µg/m^3	RfC derived on the basis of a benchmark approach using a BMCL10 (HEC) of 0.0017 mg/m^3 associated with lung fibrosis from soluble nickel salts in a rat study and an uncertainty factor of 10. This is the same study as considered by the ATSDR.
ATSDR (ATSDR 2005a)	Inhalation MRL = 0.09 µg/m^3	Chronic inhalation MRL derived on the basis of a NOAEL (HEC) of 0.0027 mg/m^3 associated with lung effects in rats, and an uncertainty factor of 30.
US EPA IRIS (USEPA IRIS)	GV = 0.04 µg/m^3	Review by the US EPA (last reviewed in 1991) established a range of air guideline values for nickel based on a non-threshold approach with a unit risk derived from occupational studies associated with nickel refinery dust. The guideline value noted here is based on an excess lifetime cancer risk of 1 in 100 000.

Identified TRVs

With respect to oral exposures, the more recent review by the (WHO 2011a) is considered appropriate (and most current) and adequately protective of the most critical health effects. The threshold value recommended is considered adequately protective of hypersensitivity responses that may be associated with oral (and dermal) exposures.

With respect to inhalation exposures a number of evaluations are available that consider LOAELs/NOAELs that are similar, with the application of different uncertainty factors. It is recommended that the evaluation provided by (UK EA 2009c) be adopted, where the lower threshold value of 0.02 µg/m^3 is adopted, and is consistent with guidelines derived using a non-threshold approach (at an excess lifetime cancer risk level of 1 in 100 000).

Recommendation

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

- Oral TRV (TRV_O) = 0.012 mg/kg/day (WHO 2011a) for oral and dermal routes of exposure;
- Inhalation TRV (TRVi) = 0.00002 mg/m^3 (UK EA 2009c); and
- Background intakes from other sources (as % of TRV) = 60% for oral and dermal intakes and 20% for inhalation intakes.

C10 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 2018). 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 2018).

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 2018) for oral and dermal routes of exposure; and
- Background intakes from other sources (as % of TRV) = negligible.

C11 Zinc

General

Several comprehensive reviews of zinc in the environment and toxicity to humans are available (ATSDR 2005b; 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 2018).
- 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.

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

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 2005a). 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 2005a). 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 2018)	No health based guideline established	The ADWG (NHMRC 2011) 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 2005b)	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.
USEPA (USEPA 2005a)	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);
- Dermal absorption factor (DAF) = 0.001 (or 0.1%) (USEPA 1995b); and
- Background intakes from other sources (as % of TRV) = 80%.

C12 Respirable Crystalline Silica

General

The US Agency for Toxic Substances and Disease Registry (ATSDR) released an updated Toxicological Profile for Silica in September 2019 (ATSDR 2019a). This toxicity profile is based on the information presented in the ATSDR document, with support from other references where indicated.

Silica in the form of quartz is one of the most commonly occurring minerals on the Earth's surface, with over 95% of the earth's crust made of minerals containing silica. There are 2 forms of silica – crystalline silica and amorphous silica. Amorphous silica lacks a crystalline structure and is not further assessed. Two common forms of crystalline silica are quartz and cristobalite.

Silica from quartz is an odourless, white, black, purple or green solid and is generally considered to be insoluble in water and unreactive in the environment. RCS in dust is also considered stable.

Silica is naturally released into the environment through the weathering of rocks, volcanic activity and biogenic sources. Hence, background exposures (i.e. naturally occurring exposures outdoors or indoors – where there are no specific silica sources) may occur through air, indoor dust, food, water, soil and various consumer products. Crystalline silica has a wide variety of commercial and industrial uses including:

- To produce high-temperature or refractory silica brick, foundry moulds and cores for metal casings.
- To manufacture glass and pure silicon for computer chips.
- As a filler in asphalt, plastics, rubber and paint.
- As an abrasive (e.g. for blasting).
- In sand and gravel used for building roads and in concrete.
- In the water-sand mix used by the oil and gas industry to fracture rock.
- In bricks, mortar, plaster, calk, roofing granules and stone building materials (including benchtops).
- In art clay, glazes and gemstones in jewellery.
- In personal care products such as cleansers and talcum powder and cosmetics.
- In pet litter and furniture foam.

Given the wide range of sources of silica in the environment it is expected that there would be RCS in urban and rural environments.

Exposure, absorption and health effects

The exposure route of concern for RCS is inhalation. Exposure to RCS is known to occur in industrial and occupational settings, with RCS recognised as an important occupational inhalation hazard.

When RCS is inhaled it is the fine fractions that are of importance as these are small enough to penetrate into the lungs where they can be deposited. RCS particles are not soluble so they are not easily absorbed by the body and remain as solid particles once deposited in the lungs.

The presence of these particles in the lungs damages cells in the lungs, which impairs the ability of the lungs to clear or flush out the particles (which would normally occur through mucous or coughing). Hence these particles can become “stuck” or embedded in the lungs. As these particles can remain in the lungs for a long period of time the shape of the particles becomes important as this leads to inflammation which results in disease such as silicosis or fibrosis. The prolonged inflammation results in the formation of fibrotic scar tissue and degradation of the lung clearance mechanism. The improper repair of damaged lung tissue is essential for the development of chronic disease.

Acute exposures can also result in respiratory inflammation which stimulates a significant increase in alveolar macrophages, leading to elevated levels of reactive oxygen species (ROS), which plays an important part in inflammation and the production of antioxidant compounds

Health effects associated with occupational exposures include silicosis, lung cancer, renal toxicity and autoimmune diseases. The health effects that are generally of greatest concern to humans are silicosis and lung cancer.

Silicosis is a progressive and irreversible fibrotic lung disease that has been recognised since Roman and Greek times and is not caused by any substance other than RCS (including amorphous silica). A fibrotic lung disease is a disease where excess fibrous connective tissue is formed in an organ. This type of effect is also referred to as scarring when in response to an injury. Silicosis is caused by inhaling RCS, where the RCS is then deposited on the lungs. There is no known cure for silicosis. There are several types of silicosis:

- **Acute silicosis** is caused by intense exposure to fine RCS dust, such as those generated during blasting or tunnelling. With this disease, the alveolar (the tiny air sacks in the lungs which absorb oxygen) fill with a protein rich fluid containing damaged cells. Inflammation of the lung also occurs. Symptoms include laboured breathing, dry cough, decreased pulmonary function, fever and fatigue followed by cyanosis and respiratory failure.
- **Simple silicosis** is the most common type of silicosis and results from long periods (10 to >20 years) of continuous exposures to relatively low levels of RCS dust. Primary function and general health is typically not compromised in the early stages, however, intensity of cough and mucous discharge increases as the disease progresses. Decreases in lung function are often observed (including non-reversible air flow obstruction).
- **Progressive massive fibrosis (PMF)** is a progression of simple silicosis where nodular lung lesions (injuries) grow and come together to form masses of connective tissue that ultimately destroys the lung structures including the blood vessels. This leads to restricted lung volume and poor gas exchange.
- **Accelerated silicosis** is a progressive form of simple silicosis that develops 5 to 10 years after exposure and is typically associated with moderate exposures (as opposed to simple silicosis which is associated with lower level exposures). Symptoms are similar to those of simple silicosis.

Decreased lung function can also be observed in the absence of silicosis and may be caused by exposures to RCS. This is known as chronic obstructive pulmonary disease (COPD). COPD is characterised by limitation in airflow caused by chronic bronchitis, emphysema, asthma or peripheral airways disease (ATSDR 2019a; NIOSH 2002). Cigarette smoking is the main cause of COPD however occupational exposures to dust and community air pollution can also contribute and there are limited studies that link RCS and COPD. No studies have investigated a potential link between RCS and asthma and RCS is not known to cause asthma occupationally.

The most important factor for the development of silicosis is cumulative exposure to RCS. Time from first exposure to onset of symptoms can vary from a few weeks (for acute silicosis) to 20 years or more (for simple silicosis). Disease severity may also slowly increase following cessation of exposure, where RCS is retained in the lungs.

Carcinogenicity

Several studies have looked at whether exposure to RCS causes lung cancer and compared to other occupational lung carcinogens, the reported association is low. However, an increase risk to lung cancer in RCS workers has been reported, with risks dependant on cumulative (successive and ongoing) exposures over times. RCS workers are workers who are frequently exposed to crystalline silica at high levels as part of their occupation/work. This relates to workers being in frequent close proximity to or undertaking activities such as the crushing, cutting, drilling, grinding, sawing and polishing of stone or man-made products that contain silica[18].

Inhaled crystalline silica dust in the former of quartz or cristobalite, is classified as Group 1 (carcinogenic to humans) by the International Agency for Research on Cancer (IARC) (IARC 2012a), based on occupational data. TCEQ (TCEQ 2009) also emphasises that the identification of RCS as carcinogenic relates only to occupational exposures. The mechanisms for carcinogenicity is likely to be inflammation.

The classification of RCS as an occupational carcinogen is supported by ACGIH (2010). The ACGIH review identified that there was a consensus among US and international agencies is that there is a positive association between occupational silica exposures and lung cancer. Most agencies consider that silica does not directly act to initiate cancer, however, do agree that workers that have pulmonary fibrosis (following exposure to silica) are at risk of developing lung cancer (but does not prove that the fibrosis leads directly to lung cancer). However, ACGIH considers that a reduction in worker exposures such that risks from silicosis are eliminated will likely protect against the formation of lung cancer.

The available evidence indicates that RCS is genotoxic with the ability to cause mutagenicity and DNA damage (ATSDR 2019a).

No information is available in relation to the susceptibility of children to RCS as silicosis is generally considered to be an occupational disease the typically appears after prolonged exposures. The same adverse effects would be expected to appear in children where exposures were similar to adult workers. Individuals with underlying lung and health conditions such as asthma and emphysema may be more susceptible to adverse respiratory effects from inhaled RCS. The risk of silicosis in workers who smoke cigarettes is also higher than in workers who do not smoke.

[18] Refer to additional information available from SafeWork Australia in relation to work activities that represent a high risk exposure https://www.safeworkaustralia.gov.au/silica

Quantitative toxicity reference values

For RCS two types of toxicity values are available:

- **Occupational air guidelines:** these guidelines are applicable to individuals who are exposed to chemicals in the workplace through use or handling, that does not present an unacceptable risk to worker health or cause undue discomfort. These guidelines relate exposures by healthy workers in the workplace, during work hours. The guidelines are higher than ambient or community air guidelines and may be at levels that are mildly irritating.
- **Community air guidelines:** these guidelines represent the concentration of a chemical in air that, based on the current science, does not present an unacceptable risk to public or community health. These guidelines are based on a range of different studies conducted in animals and humans (from occupational studies or studies in large populations – epidemiological studies), with the application of an uncertainty factors to make sure that the guideline is relevant to the community who may have a range of sensitivities. The uncertainty factors may also take into account any limitations there are with the available studies.

This assessment has only further considered community air guidelines. The following quantitative values are available from Level 1 Australian and International sources.

Source	Value	Basis/Comments
Australian		
EPA Victoria (2007)	3 µg/m^3 ($PM_{2.5}$ fraction)	Annual average assessment criteria for mining and extractive industries for RCS. This is the total concentration of background plus emissions arising from activities at a site. The assessment criteria are used to evaluate the impact of any residual emissions following appropriate controls. The REL from the California EPA Office for Environmental Health Hazard Assessment (OEHHA) has been adopted (refer below).
International		
TCEQ (TCEQ 2009)	Non-cancer: = 2 µg/m^3 (PM_4 fraction) Cancer: 0.27 µg/m^3 (PM_4 fraction)	Effects Screening Levels (ESLs) for quartz, cristobalite, tripoli and tridymite. <u>Non-cancer effects:</u> Chronic Reference Value (ReV) based on epidemiological data from 2 studies. The key study involved South African gold miners (Hnizdo and Sluie-Cremer 1993; 2,235 individuals following 24 years exposure mainly to RCS as quartz). The supporting study involved Californian diatomaceous earth workers (Hughes et. al. 1998; 2,342 individuals exposed for at least 1 year to cristobalite). Bench-mark dose modelling was undertaken at the 1% response rate for both studies. The adopted point of departure was in the range 4 to 6 µg/m^3. The adopted uncertainty factor (UF) was 3 to account for susceptibility in the general population (including children and the elderly). An UF of 3 was assessed to be adequate as a $BMCL_{01}$ could be derived and the cohort examined was large and therefore assessed to cover sensitive sub-populations. However, the study only included male workers. The derived ReV was 2 µg/m^{3,} based on the rounding of results from both studies. A chronic non-cancer ESL of 0.6 µg/m^3 was also derived based on a Hazard Quotient (HQ) of 0.3 (this is not relevant to Australia where the applicable HQ is 1). <u>Cancer:</u> Unit Risk (UR) of $3.6x10^{-5}$ (µg/m^3)$^{-1}$ derived based on lung cancer mortality in silica-exposed workers (as pooled by Steenland etc. al. 2001; 65,980 workers from a range of industries) and RCS of ≤4 µm in diameter. The derived chronic ESL (cancer) was 0.00027 mg/m^3 at a target risk level of $1x10^5$.

Source	Value	Basis/Comments
International (Cont'd)		
Minnesota Department of Health (MDH 2013)	3 µg/m^3 (PM fraction not stated)	Non-cancer effects: Chronic Health Based Value (HBV) based on the same key epidemiological study evaluated by TCEQ (2009) (Hnizdo and Sluie-Cremer 1993), with a point of departure of 0.0098 mg/m^3 and an UF of 3. The main difference in the TCEQ and MDH assessments was the assumed %RCS in dust inhaled by the workers (30% by MDH versus 54% by TCEQ; a difference of around 2-fold). Cancer: No cancer HBV was calculated. MDH concluded that if exposure to silica is maintained at levels below the Chronic HBV the likelihood of increased risk of developing lung cancer is minimal.
California OEHHA (OEHHA 2005)	3 µg/m^3 (PM_4 fraction)	Non-cancer effects: Inhalation Reference Exposure Level (REL) based on the same key epidemiological study evaluated by TCEQ (2009) (Hnizdo and Sluie-Cremer 1993), with a point of departure of 9.8 µg/m^3 and an UF of 3. The assumed silica content in dust was 30%. Data from the Hughes et. al. (1998) study and 3 additional supporting studies (Chinese tin miners, Chen et. al., 2001; Dakota gold miners, Steenland and Brown 1995; South African gold miners, Churchyard etc. al. 2004) was also considered. Derived RELs were in the range 3 to 6 µg/m^3. The REL applies to the respirable fraction as defined occupationally by ACGIH (2004)/ISO (1995) which has a 50% cut-off point at the 4 µm particle aerodynamic diameter. Cancer: OEHHA notes that RELs are not derived based on cancer endpoints and there are no approved cancer potency factors for silica.
Vermont Agency of Natural Resources (2018)[19]	0.12 µg/m^3 (PM fraction not stated)	Hazardous ambient air standard (annual average) for crystalline silica as listed in the 2018 Air Pollution Control Regulations. No information available in relation to the derivation of the air standard (information was requested on 7 February 2020, but no information had been provided at the time of this HHRA). This guideline has not been considered further in the HHRA as no information is available in relation to how the guideline has been derived.

Based on the available guidelines and information related to the development of these guidelines, the following TRV or guideline has been adopted for this assessment:

- RCS in air as $PM_{2.5}$ (based on an annual average concentration) = 3 µg/m^3

This guideline is consistent with evaluations undertaken in California (OEHHA 2005), Texas (TCEQ 2009) and Minnesota (MDH 2013), and adopted by the Victorian EPA (EPA Victoria 2007) for the assessment of RCS exposures in the community surrounding mining and extractive industries (which includes quarry activities). The guideline is protective of all health effects for exposures by all members of the community.

International community air guidelines for RCS are similar (2 to 3 µg/m^3) and are all based on protection against silicosis from data from occupational studies. The guideline of 3 µg/m^3 was first derived by OEHHA (2005), was confirmed by the most recent review undertaken by MDH (2013) and adopted by EPA Victoria (2007). Hence, this guideline has been adopted in this HHRA. This means that exposures to RCS concentrations of less than 3 µg/m^3 are considered safe, or not associated with adverse health risks from RCS. A slightly lower guideline of 2 µg/m^3 has been derived by TCEQ (2009) but is noted to be based on the same key studies and is not significantly different to 3 µg/m^3.

[19] https://dec.vermont.gov/sites/dec/files/aqc/laws-regs/documents/AQCD%20Regulations%20ADOPTED_Dec132018.pdf

The OEHHA (2005) guideline specifically considered the protection of sensitive members of the population, especially children (as silica particles may penetrate further into the airways) and women (who may be more sensitive than men to the development of silicosis). For this reason, an UF of 3 (and not 1) was used for interspecies variation in the development of the air guideline, as the key studies primarily investigated effects in male workers. MDH (2013) notes that the derived guideline also considers general population exposures and is based on a benchmark concentration low_{01} ($BMCL_{01}$; a value similar to a NOAEL) which is the 95% lower bound estimate of the concentration at which 1% of the population will develop silicosis.

Except for TCEQ (2009), national and international guidelines for cancer effects have not been derived, as silicosis was determined to be the most sensitive effect. i.e. cancer was deemed unlikely to occur at concentrations of RCS below the guideline for silicosis. The rationale for the inclusion of the cancer guideline by TCEQ (2009) appears to be based on the TCEQ policy position in relation to the lack of a clearly identified mode of action for silica toxicity, including the potential uncertainties in the epidemiology studies. In their response to comments on the Draft document outlining the derivation of the guidelines, TCEQ indicate that[20]:

- *"There is not a consensus among the scientific community on whether the carcinogenic mode of action for silica is non-linear or linear or whether silicosis is necessary for the development of lung cancer".*

The opinion of TCEQ (2009) is not supported by the more recent MDH (2013) review who indicate the following:

- *Silica has been classified as a known human carcinogen…because of an observed increase in lung cancers in occupationally exposed workers. There is, however, a large body of evidence that indicates that lung cancer attributed to silica occurs only after repeated insult leads to silicosis. While some controversy remains, MDH has determined that if exposure to silica is maintained at levels below those that result in silicosis the likelihood of increased risk of developing lung cancer is minimal. MDH will continue to monitor this issue and reconsider this decision as new information becomes available.*

In the absence of a definitive mode of action, TCEQ guidance indicates that where chronic adverse effects are determined to be associated with a linear dose-response relationship in the low-dose region, which is typically for chronic exposures to carcinogens, a cancer evaluation should be undertaken. This determination is based on data or science policy default assumptions (TCEQ 2006).

Irrespective of the above, IARC is clear that the determination that RCS is carcinogenic relates only to occupational exposures. For this reason, the TCEQ (2009) cancer guideline has not been adopted in this HHRA.

In relation to the OEHHA (2005) community air guideline (REL), the background document notes that there is an absence of comprehensive data on the ability of different particle sizes to induce silicosis, hence, it is not possible to adjust the guideline for different size particle distributions (e.g. as might be measured at a particular site). Further, while silicosis is generally assumed to be induced by the fraction that reaches the alveoli (with the majority of particles around 4 µm), there is no data to confirm a lack of adverse effects for coarser particles of 4 to 10 µm. The guideline therefore applies to particles that are defined as "occupationally respirable".

20 https://www.tceq.texas.gov/assets/public/implementation/tox/dsd/final/october09/comments/responses_silica.pdf

Given this:

- $PM_{2.5}$ and PM_{10} concentrations ≤3 µg/m^3 would not be expected to be associated with adverse health effects.
- PM_{10} concentrations >3 µg/m^3 require further investigation and/or risk management.
- $PM_{2.5}$ >3 µg/m^3 < PM_{10} concentrations may require further investigation, including a more precise determination of the respirable fraction.

It is noted that Victoria (EPA Victoria 2007) has adopted the OEHHA (2005) guideline for RCS as $PM_{2.5}$. This approach has also been adopted in this assessment.

C13 Hydrogen Cyanide

General

Cyanides comprise a wide range of compounds of varying degrees of chemical complexity, all of which contain a CN moiety, to which humans are exposed in gas, liquid, and solid form from a broad range of natural and anthropogenic sources. While many chemical forms of cyanide are used in industrial application or are present in the environment, the cyanide anion CN^- is the primary toxic agent, regardless of origin (WHO 2004b).

Hydrogen cyanide (CAS No. 74-90-8) is a colourless or pale blue liquid or gas with a faint bitter almond-like odour. Hydrogen cyanide is used primarily in the production of substances such as adiponitrile, methyl methacrylate, chelating agents, cyanuric chloride, methionine and its hydroxylated analogues, and sodium and potassium cyanide. Hydrogen cyanide is also used as a fumigant in ships, railroad cars, large buildings, grain silos, and flour mills, as well as in the fumigation of peas and seeds in vacuum chambers (WHO 2004b).

Anthropogenic sources of cyanide release to the environment are diverse. Hydrogen cyanide is formed during the incomplete combustion of nitrogen-containing polymers, such as certain plastics, polyurethanes, and wool. Hydrogen cyanide is present in cigarette smoke (WHO 2004b).

Exposure and health effects

The majority of the population is exposed to very low levels of cyanide in the general environment. There are some subgroups with higher levels of exposure including occupational environments, those living near specific sources, active and passive smokers and fire-related smoke inhalation victims (WHO 2004b).

Cyanides are well absorbed via the gastrointestinal tract or skin and rapidly absorbed via the respiratory tract. Once absorbed, cyanide is rapidly and ubiquitously distributed throughout the body, although the highest levels are typically found in the liver, lungs, blood, and brain. There is no accumulation of cyanide in the blood or tissues following chronic or repeated exposure (WHO 2004b).

Approximately 80% of absorbed cyanide is metabolised to thiocyanate in the liver by the mitochondrial sulfur transferase enzyme rhodanese and other sulfur transferases. Thiocyanate is excreted in the urine (WHO 2004b).

The principal features of the toxicity profile for cyanide are its high acute toxicity by all routes of administration, with a very steep and rate-dependent dose–effect curve, and chronic toxicity, probably mediated through the main metabolite and detoxification product, thiocyanate (WHO 2004b).

The toxic effects of cyanide ion in humans and animals are generally similar (WHO 2004b).

The cyanide ion blocks oxidative respiration, causing tissue hypoxia; tissues with high metabolic demand such as the central nervous system (CNS)are therefore key targets for toxicity. Early features of systemic toxicity include non-specific CNS symptoms, muscular and neurological effects, tachypnoea and tachycardia. Late effects or those following larger exposures may include seizures, a rapid loss of consciousness, cardiorespiratory depression and collapse, pulmonary oedema and death. Lactate acidosis may also be noted (PHE 2016).

The primary targets of cyanide toxicity in humans and animals are the cardiovascular, respiratory, and central nervous systems. The endocrine system is also a potential target for long-term toxicity, as a function of continued exposure to thiocyanate, which prevents the uptake of iodine in the thyroid and acts as a goitrogenic agent (WHO 2004b).

Following chronic or repeated exposure optic neuropathy has been observed in some cases, including atrophy, amblyopia and colour deficits. Respiratory tract irritation, breathlessness, hoarse voice, chronic rhinitis and deafness have also been reported. Some gastrointestinal and skin effects have been observed, which are likely to be due to cyanide's irritant effects. There is some debate as to whether the effects observed on repeat exposure to cyanide are truly due to repeat dose toxicity or the result of acute intoxication (PHE 2016).

Exposure to a massive concentration of hydrogen cyanide gas may render an individual unconscious within seconds and may lead to coma and death within minutes. Some estimates of lethal concentrations are reported in the following table (PHE 2016).

Table: Time to death following hydrogen cyanide inhalation in humans (PHE 2016)

Dose		**Time to death**
mg/m^3	**ppm**	
120-150	110-135	30 mins to 1 hour or later
200	180	10 minutes
300	270	Immediate

Only mild effects may occur after exposure to 20–40 mg/m^3 for several hours; 50–60 mg/m^3 may be tolerated for 20 minutes to 1 hour without immediate or late effects (PHE 2016). Hydrogen cyanide is reported to have a characteristic odour of almonds or bitter almonds. However, not all individuals can detect this, so odour is not to be considered to be a reliable indicator of exposure.

Classification

IARC has notified hydrogen cyanide as to carcinogenicity.

The USEPA (USEPA 2010) has classified hydrogen cyanide as "inadequate information to assess carcinogenic potential".

There are limited studies in relation to genotoxicity, however review of the available studies indicates that based on the weight of evidence, cyanide is not genotoxic (ATSDR 2006; PHE 2016; WHO 2004b).

Toxicity Values

Review of available data with respect to hydrogen cyanide indicates that this chemical has not been determined to be carcinogenic (due to inadequate data) and is not genotoxic. Hence the quantification of effects associated with exposure to hydrogen cyanide is undertaken on the basis of a threshold.

Toxicity data relevant for use in the characterisation of risk to human health have been selected for hydrogen cyanide following review of the available information in general accordance with current Australian guidance (NEPC 1999 amended 2013c).

Table: Summary of published toxicity reference values: Hydrogen cyanide

Source	Value	Basis/Comments
Acute or short-term exposures		
NRC (NRC 2002)	1 hour average = 2.2 mg/m^3	Acute (1 hour average) AEGL-1 criteria based on a no effect concentration from an occupational study (that related to 8 hour exposures) and time scaling to a 1 hour exposure. No uncertainty factor was applies as a no effect level was adopted and resulted in similar levels as the use of lowest effect levels from other studies and application of a 3 fold uncertainty factor.
OEHHA (OEHHA 2008)	1 hour average = 0.34 mg/m^3	Acute reference exposure level (REL) has been derived on the basis of CNS effects (a loss of coordination and consciousness due to cellular hypoxia) in monkeys.
Chronic exposures		
OEHHA (OEHHA 2008)	REL = 0.009 mg/m^3	Chronic REL based on the same study as the USEPA with application of a 300 fold uncertainty factor.
RIVM (Baars et al. 2001)	TC = 0.025 mg/m^3	The RIVM tolerable concentration is based on the same study and endpoint as the USEPA, however RIVM has applied an uncertainty factor of 100.
USEPA (USEPA 2010; USEPA IRIS)	RfC = 0.0008 mg/m^3	The USEPA RfC is based on a LOAEL (HEC) of 2.5 mg/m^3 for thyroid effects (thyroid enlargement and altered iodide uptake) in a human epidemiological/occupation study and application of 3000 fold uncertainty factor, which includes a 10 fold factor to address database deficiencies. Confidence on the RfC is noted to be low to medium.

Limited TRVs are available for inhalation exposures to hydrogen cyanide. Assessment of short-term exposures can be undertaken on the basis of the value from OEHHA, which the USEPA value can be adopted for chronic exposures, noting that all available chronic criteria are based on the same study, with only the uncertainty factor differing.

Background intakes

In relation to background exposures there is no data available on background exposures to hydrogen cyanide in the Australian community. Some international data is available close to specific sources (ATSDR 2006; WHO 2004b). For the purpose of this assessment background intakes for the community have been assumed to be negligible.

Annexure D

Lead Bioaccessibility Testing of Soils

(Total No. of pages including blank pages = 58)

Note: This Annexure is only available on the digital version of this document

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University of South Australia

Determination of Pb Bioaccessibility in Bowdens Silver Soil Samples

Prepared for: RW Corkery & Co Pty Ltd,
Level 1, 12 Dangar Road,
Brooklyn, NSW, 2083

Attention: Scott Hollamby
Telephone: 0437 858 511
Email: scott@rwcorkery.com

Prepared by: Dr Albert Juhasz
Future Industries Institute
University of South Australia
Mawson Lakes Boulevard
Mawson Lakes, SA 5095

Telephone: 08 8302 5045
Facsimile: 08 8302 3057
Email: Albert.Juhasz@unisa.edu.au

Date of issue: 8 June 2017

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INTRODUCTION

This report was prepared for RW Corkery & Co Pty Ltd to assess lead bioaccessibility in impacted soil. The bioaccessibility testing was conducted at the Future Industries Institute, based at the Mawson Lakes Campus of the University of South Australia (UniSA). UniSA's Flagship Institute focuses on building knowledge and capacity in core research strengths of physical chemistry and environmental science and management. The Institute has four distinct yet inter-related strands: Minerals and Resources; Energy and Advanced Manufacturing; Environmental Science and Engineering; and Bioengineering and Nanomedicine. The Institute aggregates and builds upon existing expertise and infrastructure from the Ian Wark Research Institute, the Mawson Institute and the Centre for Environmental Risk Assessment and Remediation. The vision for the Future Industries Institute aligns strongly with South Australian and National economic and research priorities by building a critical mass of trans-disciplinary research capacity focused on pressing real-world challenges.

OBJECTIVES

The objective of this assessment was to:

- Assess the concentration of lead in the < 2 mm and < 250 μm soil particle size fractions; and
- Assess lead bioaccessibility in the < 250 μm soil particle size fraction using the gastric phase of the SBRC assay.

OUTCOMES AND DELIVERABLES

The expected outcome from this assessment was:

- A report assessing the bioaccessibility of lead in soil. The report was to include:
    - Assessment of lead concentration in the < 2 mm and < 250 μm soil particle size fractions;
    - Assessment of lead bioaccessibility in the < 250 μm soil particle size fractions using an vitro method;
    - Methodology procedures; and
    - QA/QC protocols

PROJECT BACKGROUND

Soil testing was initiated at the invitation of RW Corkery & Co Pty Ltd for an assessment of lead bioaccessibility in impacted soil. Human exposure to a contaminant may be through a number of pathways including inhalation, dermal absorption and ingestion. For many metal contaminants, the most significant metal exposure pathway is via soil ingestion. Generally, soil ingestion results from the accidental or, in the case of children less than 5 years old, the incidental ingestion of soil (< 250 µm particle size fraction) via hand-to-mouth contact (Basta et al., 2001). In assessing contaminant exposure, it is often assumed that the contaminant is 100% bioaccessible / bioavailable, however, there is growing evidence to suggest that contaminant bioaccessibility / bioavailability in soil may be less than 100%. Therefore, incorporation of metal bioaccessibility / bioavailability may reduce the uncertainty in estimating exposure associated with the incidental ingestion of contaminated soil.

Contaminant bioaccessibility may be estimated using *in vitro* assays that simulate processes that occur in the human body that lead to the release of contaminants from the soil matrix. A frequently used assay for the determination of contaminant bioaccessibility is the Solubility Bioaccessibility Research Consortium (SBRC) method (Kelly *et al.*, 2002). The gastric phase of this method (termed the Simplified Bioaccessibility Extraction Test [SBET] for arsenic or the Relative Bioavailability Leaching Procedure [RBALP] for lead) has been correlated to *in vivo* arsenic and lead relative bioavailability when determined using juvenile swine (Juhasz *et al.*, 2007; USEPA 2007).

FINDINGS

Total lead concentration for each sample is shown in Table 1 while lead bioaccessibility results are shown in Tables 2 and 3.

- Total lead concentration in the < 2 mm soil particle size fraction was < 100 mg kg^{-1} with the exception of samples 65 5-15 (375.5 mg kg^{-1}) and 65 30-60 (615.5 mg kg^{-1}) (Table 1). Similarly, total lead concentration in the < 250 µm soil particle size fraction was < 100 mg kg^{-1} with the exception of samples 65 5-15 (147.5mg kg^{-1}) and 65 30-60 (305.5 mg kg^{-1}) (Table 1).

- Lead bioaccessibility determined using gastric phase extraction (SBRC-G) ranged from 14.6% (sample 67 30-60) to 53.8% (sample 69) (Tables 2 and 3).

- Bioaccessibility values determined for QC1 (lead contaminated reference soil) was within the acceptable range for this reference material.

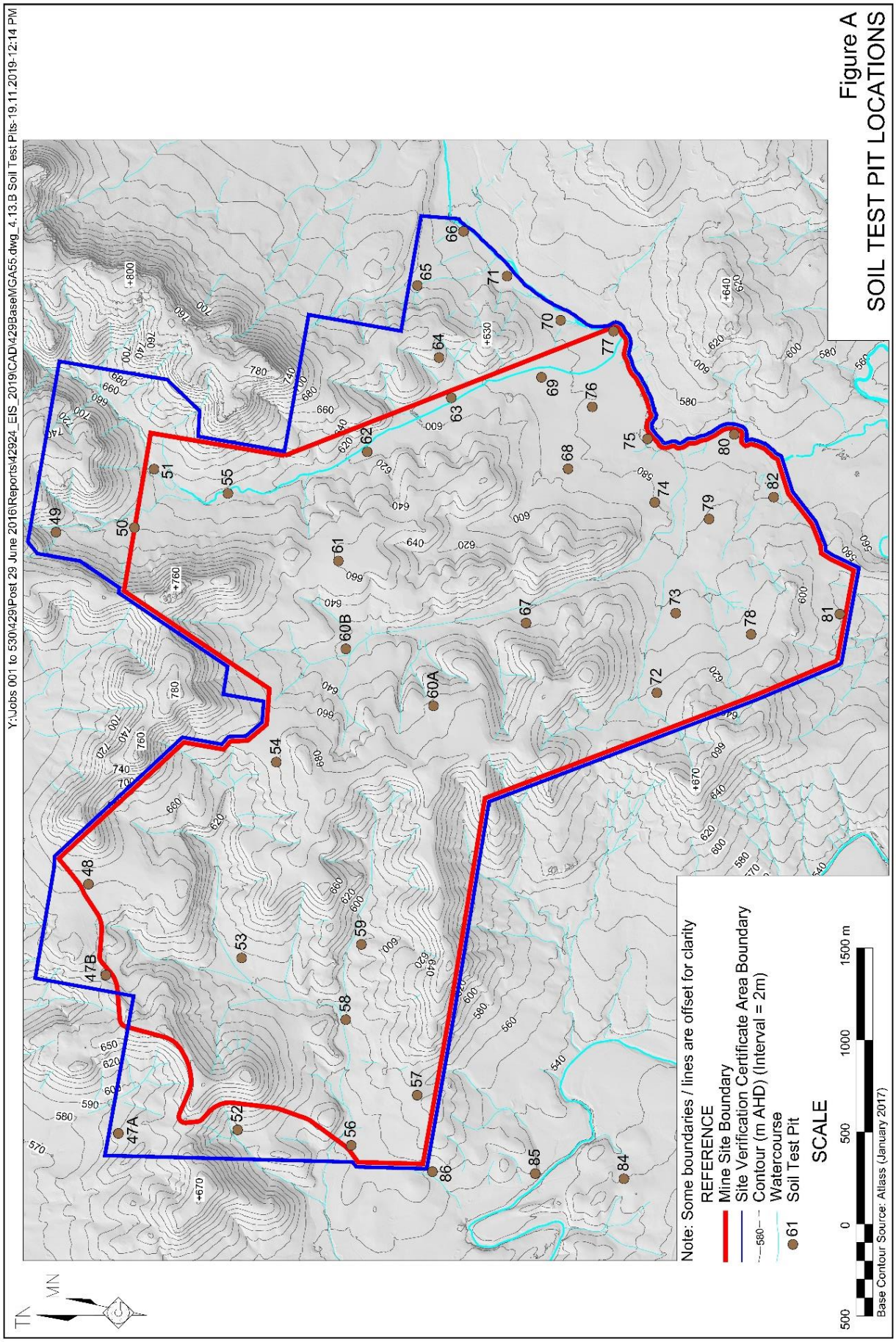
Figure A
SOIL TEST PIT LOCATIONS
Y:\Jobs 001 to 530\429\Post 29 June 2016\Reports\42924_EIS_2019\CAD\429BaseMGA55.dwg_4.13.B Soil Test Pits-19.11.2019-12:14 PM
TN
MN
Note: Some boundaries / lines are offset for clarity
REFERENCE
Mine Site Boundary
Site Verification Certificate Area Boundary
Contour (m AHD) (Interval = 2m)
Watercourse
Soil Test Pit
SCALE
500
0
500
1000
1500 m
Base Contour Source: Atlass (January 2017)

Table A
Record of Soil Test Pits and Samples Tested

Soil Landscape Unit	SITE	BSAL soil sampling depths		
		0-5cm	5-15cm	30-60cm
Steepland - acid volcanics	47A	√		
Steepland - congl./sandstone	47B	√		
<10% slope - acid volcanics	52	√		
<10% slope - acid volcanics	58	√		
<10% Slope - congl./sandstone	60A	√		
<10% Slope - congl./sandstone	61	√		
Steepland - acid volcanics	67		√	√
Lower slopes - Ordovician volcanics	68	√		
Alluvial - mixed parent materials	69	√		
<10% slope - acid volcanics	72	√		
<10% slope - acid volcanics	73		√	
Lower slopes - Ordovician volcanics	84	√		
Alluvial - mixed parent materials	85	√		

Soil samples for additional testing for bioaccessibility analysis (University of Adelaide)

Table 1. Total lead concentration in the < 2 mm and < 250 µm soil particle size fractions.

Soil	< 2 mm Soil Particle Size Fraction			< 250 µm Soil Particle Size Fraction		
	ID #	Pb (mg kg^{-1})	Mean Pb (mg kg^{-1})	ID #	Pb (mg kg^{-1})	Mean Pb (mg kg^{-1})
47A	47A-2A	21		47A-250A	25	
	47A-2B	22	21.5	47A-250B	24	24.5
47B	47B-2A	16		47B-250A	12	
	47B-2B	22	19	47B-250B	12	12
52	52-2A	16		52-250A	12	
	52-2B	16	16	52-250B	13	12.5
58	58-2A	13		58-250A	13	
	58-2B	11	12	58-250B	12	12.5
60A	60A-2A	10		60A-250A	8	
	60A-2B	9	9.5	60A-250B	8	8
61	61-2A	9		61-250A	16	
	61-2B	7	8	61-250B	16	16
67 5-15	67 5-15-2A	396		67 5-15-250A	152	
	67 5-15-2B	355	375.5	67 5-15-250B	143	147.5
67 30-60	67 30-60-2A	536		67 30-60-250A	310	
	67 30-60-2B	695	615.5	67 30-60-250B	301	305.5
68	68-2A	78		68-250A	83	
	68-2B	90	84	68-250B	80	81.5
69	69-2A	<5		69-250A	7	
	69-2B	<5	<5	69-250B	6	6.5
72	72-2A	10		72-250A	9	
	72-2B	11	10.5	72-250B	9	9
73	73-2A	9		73-250A	9	
	73-2B	8	8.5	73-250B	9	9
84	84-2A	8		84-250A	8	
	84-2B	7	7.5	84-250B	9	8.5
85	85-2A	12		85-250A	14	
	85-2B	12	12	85-250B	13	13.5

Table 2. Lead bioaccessibility in contaminated soils determined using gastric phase extraction (SBRC-G).

Soil	Sample #	ICP-AES Pb ($mg\ l^{-1}$)	Soil:Solution Ratio	Gastric Phase Pb Bioaccessibility ($mg\ kg^{-1}$)	Mean Gastric Phase Pb Bioaccessibility ($mg\ kg^{-1}$)
47A	47A-G1	0.08	100	8.0	
	47A-G2	0.09	100	9.0	8.5
47B	47B-G1	0.04	100	4.0	
	47B-G2	0.03	100	3.0	3.5
52	52-G1	0.04	100	4.0	
	52-G2	0.03	100	3.0	3.5
58	58-G1	0.05	100	5.0	
	58-G2	0.06	100	6.0	5.5
60A	60A-G1	0.03	100	3.0	
	60A-G2	0.02	100	2.0	2.5
61	61-G1	0.06	100	6.0	
	61-G2	0.06	100	6.0	6.0
67 5-15	67 5-15-G1	0.27	100	27.0	
	67 5-15-G2	0.28	100	28.0	27.5
67 30-60	67 30-60-G1	0.44	100	44.0	
	67 30-60-G2	0.45	100	45.0	44.5
68	68-G1	0.20	100	20.0	
	68-G2	0.19	100	19.0	19.5
69	69-G1	0.04	100	4.0	
	69-G2	0.03	100	3.0	3.5
72	72-G1	0.04	100	4.0	
	72-G2	0.04	100	4.0	4.0

73	73-G1	0.04	100	4.0			
	73-G2	0.04	100	4.0		4.0	
84	84-G1	0.02	100	2.0			
	84-G2	0.02	100	2.0		2.0	
85	85-G1	0.04	100	4.0			
	85-G2	0.04	100	4.0		4.0	
QC1†	QC1-G1	47.2	100	4720			
	QC1-G2	47.6	100	4760		4740	
QC2‡	QC2-G1	0.09	-	0.09			
	QC2-G2	0.03	-	0.03		0.06	

†QC1 comprised of a lead-contaminated (6400 mg Pb kg^{-1}) reference soil.
‡QC2 comprised of SBRC gastric phase solution without soil addition (assay blank).

Table 3. Total lead concentration and bioaccessible lead in impacted soils (< 250 µm soil particle size fraction).

Soil	Lead		
	Total (mg kg^{-1})	Bioaccessible (mg kg^{-1})	Bioaccessible (%)‡
47A	24.5	8.5	34.7
47B	12.0	3.5	29.2
52	12.5	3.5	28.0
58	12.5	5.5	44.0
60A	8.0	2.5	31.3
61	16.0	6.0	37.5
67 5-15	147.5	27.5	18.6
67 30-60	305.5	44.5	14.6
68	81.5	19.5	23.9
69	6.5	3.5	53.8
72	9.0	4.0	44.4
73	9.0	4.0	44.4
84	8.5	2.0	23.5
85	13.5	4.0	29.6
QC1	6400	4740	74.1Ω

‡Percentage lead bioaccessibility following gastric phase extraction was calculated by dividing bioaccessible lead (SBRC-G) by the total lead concentration multiplied by 100.

ΩLead bioaccessibility for the QC1 soil was within an acceptable range for this reference material for SBRC-G.

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CONFIDENTIALITY

We acknowledge the confidential nature of the results of this project and will treat the results and project reports with appropriate confidentiality and security.

APPENDIX 1 - METHODOLOGY

Soil samples

Samples supplied by RW Corkery & Co Pty Ltd were oven-dried at 105°C for 24 hours and sieved to obtain 2 soil particle size fractions; < 2 mm and < 250 µm. The < 250 µm soil particle size fraction was used to assess lead bioaccessibility.

Assessment of total Pb concentration in the < 2 mm and < 250 µm soil particle size fractions

Total lead concentration in the < 2 mm and < 250 µm soil fractions were determined by ALS Environmental Laboratories. A copy of the ALS Environmental Laboratories analytical report is included in Appendix 3.

Assessment of Pb bioaccessibility in the < 250 µm soil particle size fraction

A frequently used assay for the determination of contaminant bioaccessibility is the Solubility Bioaccessibility Research Consortium (SBRC) method (Kelly *et al.*, 2002). The gastric phase of this method (termed the Relative Bioavailability Leaching Procedure [RBALP] for lead) has been correlated to *in vivo* arsenic and lead relative bioavailability when determined using juvenile swine (USEPA 2007). Contaminated soil and gastric solution (30.03 g l^{-1} glycine adjusted to pH 1.5 with concentrated HCl) were combined in polyethylene screw cap flasks at a soil:solution ratio of 1:100. The pH was noted then the flasks were incubated at 37°C, 40 rpm on a Ratek suspension mixer. After 1 hour incubation, the pH was determined and gastric phase samples (10 ml) were collected, filtered through 0.45 µm filters and analysed by ICP-MS by ALS Environmental Laboratories. A copy of the ALS Environmental Laboratories analytical report is included in Appendix 3.

QA/QC procedures

ALS Environmental Laboratories conducted the analysis for total and bioaccessible lead concentrations for all samples. ALS Environmental Laboratories are a NATA accredited laboratory for the chemical testing of environmental materials. Quality Control results are reported in Appendix 2. Two additional samples were included in bioaccessibility assays for quality assurance and quality control. The samples consisted of:

a. QC1 – Lead-contaminated (6400 mg Pb kg^{-1}) reference soil.
b. QC2 – SBRC solution without soil addition (assay blank).

APPENDIX 2 – CHAIN OF CUSTODY FORMS

CHAIN OF CUSTODY – CLIENT

University of South Australia

Company: Bowdens Silver

Contact Person: Jane Munro or Scott Hollamby (R.W. Corkery & Co Pty Limited)

Project Manager: Jane Munro

Address: 68 Maloneys Road, Lue, NSW 2850

Phone: (02) 63736420

Email: janemunro@bowdenssilver.com.au and scott@rwcorkery.com

DELIVERY DETAILS
Attn: Albert Juhasz
University of South Australia
Future Industries Institute
Building X1-17, Mawson Lakes Campus
Mawson Lakes, Adelaide, 5095, SA
Tel: 08 8302 5045
Email: Albert.Juhasz@unisa.edu.au

Sample Information					Tests Required								Comments				
UniSA Sample ID	Client Sample ID	Depth	Date Collected	Sample Type	Lead bioacessibility	Total Lead											
	47A	0-5cm	Feb 2017	Soil	x	x											
	47B	0-5cm	Feb 2017	Soil	x	x											
	52	0-5cm	Feb 2017	Soil	x	x											
	58	0-5cm	Feb 2017	Soil	x	x											
	60A	0-5cm	Feb 2017	Soil	x	x											
	61	0-5cm	Feb 2017	Soil	x	x											
	67	5-15cm	Feb 2017	Soil	x	x											
	67	30-60cm	Feb 2017	Soil	x	x											
	68	0-5cm	Feb 2017	Soil	x	x											
	69	0-5cm	Feb 2017	Soil	x	x											
	72	0-5cm	Feb 2017	Soil	x	x											
	73	5-15cm	Feb 2017	Soil	x	x											
	84	0-5cm	Feb 2017	Soil	x	x											
	85	0-5cm	Feb 2017	Soil	x	x											

Relinquished by (Company): Soil Management Designs	Received by (Company) UniSA	Lab use only:
Print Name: David McKenzie	Print Name: ALBERT JUHASZ	Samples received – comments:
Date and Time: Weds 17 May 2017 10:30 am	Date and Time: 23/5/17	
Signature:	Signature:	

EnRiskS

EnRiskS

CHAIN OF CUSTODY

ALS Laboratory: please tick →

☐ Sydney: 277 Woodpark [illegible]hfield NSW 2176 Ph: 02 8784 8555 E:samples.sydney@alsenviro.com
☐ Newcastle: 5 Rosegum Rd, Warabrook NSW 2304 Ph:02 4968 9433 E:samples.newcastle@alsenviro.com
☐ Brisbane: 32 Shand St, Stafford QLD 4053 Ph:07 3243 7222 E:samples.brisbane@alsenviro.com
☐ Townsville: 14-15 Desma Ct, Bohle QLD 4818 Ph:07 4796 0600 E: townsville.environmental@alsenviro.com
☐ Melbo[illegible]4 Westall Rd, Springvale VIC 3171 Ph:03 8549 9600 E: samples.melbourne@alsenviro.com
☐ Adelaide: 2-1 Burma Rd, Pooraka SA 5095 Ph: 08 8359 0890 E:adelaide@alsenviro.com
FREIGHT [illegible]alsenviro.com
☐ [illegible]27 [illegible]ngton St, Launceston TAS 7250 Ph: 03 6331 2158 E: launceston@alsenviro.com

CLIENT:	CERAR University of South Australia	TURNAROUND REQUIREMENTS : (Standard TAT may be longer for some tests e.g., Ultra Trace Organics)	☒ Standard TAT (List due date): ☐ Non Standard or urgent TAT (List due date):
OFFICE:	Mawson Lakes Campus X1-17		
PROJECT:	RWC Pb BioAcc	ALS QUOTE NO.:	
ORDER NUMBER:			COC SEQUENCE NUMBER (Circle) COC: 1 2 3 4 5 6 7 8 OF: 1 2 3 4 5 6
PROJECT MANAGER:	Albert Juhasz	CONTACT PH: 08 8302 5045	
SAMPLER:	Albert Juhasz	SAMPLER MOBILE: 0418 818 121	
COC emailed to ALS? (NO)		EDD FORMAT (or default):	
Email Reports to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au			
Email Invoice to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au			

FOR LABORATORY USE ONLY (Circle) [illegible]

RELINQUISHED BY:	RECEIVED BY:	RELINQUISHED BY:	RECEIVED BY:
Albert Juhasz			[illegible] (ALS)
DATE/TIME: 30/5/17	DATE/TIME:	DATE/TIME:	DATE/TIME: 31/5, 9:00

COMMENTS/SPECIAL HANDLING/STORAGE OR DISPOSAL:

ALS USE ONLY	SAMPLE DETAILS MATRIX: Solid(S) Water(W)			CONTAINER INFORMATION		ANALYSIS REQUIRED including SUITES (NB. Suite Codes must be listed to attract suite price) Where Metals are required, specify Total (unfiltered bottle required) or Dissolved (field filtered bottle required).								Additional Information
LAB ID	SAMPLE ID	DATE / TIME	MATRIX	TYPE & PRESERVATIVE (refer to codes below)	TOTAL BOTTLES	Total Pb								Comments on likely contaminant levels, dilutions, or samples requiring specific QC analysis etc.
1	47A-2A	30/05/2017	S		1	1								Soils have been oven dried and sieved
2	47A-2B	30/05/2017	S		1	1								
3	47A-250A	30/05/2017	S		1	1								
4	47A-250B	30/05/2017	S		1	1								
5	47B-2A	30/05/2017	S		1	1								
6	47B-2B	30/05/2017	S		1	1								
7	47B-250A	30/05/2017	S		1	1								
8	47B-250B	30/05/2017	S		1	1								
9	52-2A	30/05/2017	S		1	1								
10	52-2B	30/05/2017	S		1	1								
11	52-250A	30/05/2017	S		1	1								
12	52-250B	30/05/2017	S		1	1								
				TOTAL	12	12								

Environmental Division
Melbourne
Work Order Reference
EM1706956

Telephone : + 61-3-8549 9600

Water Container Codes: P = Unpreserved Plastic; N = Nitric Preserved Plastic; ORC = Nitric Preserved ORC; SH = Sodium Hydroxide/Cd Preserved; S = Sodium Hydroxide Preserved Plastic; AG = Amber Glass Unpreserved; AP - Airfreight Unpreserved Plastic
V = VOA Vial HCl Preserved; VB = VOA Vial Sodium Bisulphate Preserved; VS = VOA Vial Sulfuric Preserved; AV = Airfreight Unpreserved Vial SG = Sulfuric Preserved Amber Glass; H = HCl preserved Plastic; HS = HCl preserved Speciation bottle; SP = Sulfuric Preserved Plastic; F = Formaldehyde Preserved Glass;
Z = Zinc Acetate Preserved Bottle; E = EDTA Preserved Bottles; ST = Sterile Bottle; ASS = Plastic Bag for Acid Sulphate Soils; B = Unpreserved Bag.

TC 31/5

CHAIN OF CUSTODY

ALS Laboratory: please tick →

☐ Sydney: 277 Woodpark Rd Smithfield NSW 2176 Ph: 02 8784 8555 E:samples.sydney@alsenviro.com
☐ Newcastle: 5 Rosegum Rd, Warabrook NSW 2304 Ph:02 4968 9433 E:samples.newcastle@alsenviro.com
☐ Brisbane: 32 Shand St, Stafford QLD 4053 Ph:07 3243 7222 E:samples.brisbane@alsenviro.com
☐ Townsville: 14-15 Desma Ct, Bohle QLD 4818 Ph:07 4796 0600 E: townsville.environmental@alsenviro.com
☐ Melb: 2-4 Westall Rd, Springvale VIC 3171 Ph:03 8549 9600 E: samples.melbourne@alsenviro.com
☐ Adelaide: 2-1 Burma Rd, Pooraka SA 5095 Ph: 08 8359 0890 E:adelaide@alsenviro.com
☐ Perth: 10 Hod Way, Malaga WA 6090 Ph: 08 9209 7655 E: samples.perth@alsenviro.com
☐ Launceston: 27 Wellington St, Launceston TAS 7250 Ph: 03 6331 2158 E: launceston@alsenviro.com

CLIENT:	CERAR University of South Australia
OFFICE:	Mawson Lakes Campus X1-17
PROJECT:	RWC Pb BioAcc
ORDER NUMBER:	
PROJECT MANAGER:	Albert Juhasz — CONTACT PH: 08 8302 5045
SAMPLER:	Albert Juhasz — SAMPLER MOBILE: 0418 818 121
COC emailed to ALS? (NO)	EDD FORMAT (or default):
Email Reports to (will default to PM if no other addresses are listed):	Albert.Juhasz@unisa.edu.au
Email Invoice to (will default to PM if no other addresses are listed):	Albert.Juhasz@unisa.edu.au

TURNAROUND REQUIREMENTS: (Standard TAT may be longer for some tests e.g. Ultra Trace Organics)
☒ Standard TAT (List due date):
☐ Non Standard or urgent TAT (List due date):

ALS QUOTE NO.:

COC SEQUENCE NUMBER (Circle)
COC: 1 (2) 3 4 5 6 7
OF: 1 2 3 4 5 6 (7)

FOR LABORATORY USE ONLY (Circle) [illegible]

RELINQUISHED BY:	RECEIVED BY:	RELINQUISHED BY:	RECEIVED BY:
Albert Juhasz [signature]			[signature]
DATE/TIME: 30/5/17	DATE/TIME:	DATE/TIME:	DATE/TIME: 31/5 9-10

COMMENTS/SPECIAL HANDLING/STORAGE OR DISPOSAL:

SAMPLE DETAILS — MATRIX: Solid(S) Water(W)
CONTAINER INFORMATION
ANALYSIS REQUIRED including SUITES (NB. Suite Codes must be listed to attract suite price) — Where Metals are required, specify Total (unfiltered bottle required) or Dissolved (field filtered bottle required).

LAB ID	SAMPLE ID	DATE / TIME	MATRIX	TYPE & PRESERVATIVE (refer to codes below)	TOTAL BOTTLES	Total Pb								Additional Information: Comments on likely contaminant levels, dilutions, or samples requiring specific QC analysis etc.
13	58-2A	30/05/2017	S		1	1								Soils have been oven dried and sieved
14	58-2B	30/05/2017	S		1	1								
15	58-250A	30/05/2017	S		1	1								
16	58-250B	30/05/2017	S		1	1								
17	60A-2A	30/05/2017	S		1	1								
18	60A-2B	30/05/2017	S		1	1								
19	60A-250A	30/05/2017	S		1	1								
20	60A-250B	30/05/2017	S		1	1								
21	61-2A	30/05/2017	S		1	1								
22	61-2B	30/05/2017	S		1	1								
23	61-250A	30/05/2017	S		1	1								
24	61-250B	30/05/2017	S		1	1								
				TOTAL	12	12								

Water Container Codes: P = Unpreserved Plastic; N = Nitric Preserved Plastic; ORC = Nitric Preserved ORC; SH = Sodium Hydroxide/Cd Preserved; S = Sodium Hydroxide Preserved Plastic; AG = Amber Glass Unpreserved; AP - Airfreight Unpreserved Plastic
V = VOA Vial HCl Preserved; VB = VOA Vial Sodium Bisulphate Preserved; VS = VOA Vial Sulfuric Preserved; AV = Airfreight Unpreserved Vial SG = Sulfuric Preserved Amber Glass; H = HCl preserved Plastic; HS = HCl preserved Speciation bottle; SP = Sulfuric Preserved Plastic; F = Formaldehyde Preserved Glass;
Z = Zinc Acetate Preserved Bottle; E = EDTA Preserved Bottles; ST = Sterile Bottle; ASS = Plastic Bag for Acid Sulphate Soils; B = Unpreserved Bag.

CHAIN OF CUSTODY

ALS Laboratory: please tick →

☐ Sydney: 277 Woodpark [illegible]mithfield NSW 2176 Ph: 02 8784 8555 E:samples...ydney@alsenviro.com
☐ Newcastle: 5 Rosegum Rd, Warabrook NSW 2304 Ph:02 4968 9433 E:samples.newcastle@alsenviro.com
☐ Brisbane: 32 Shand St, Stafford QLD 4053 Ph:07 3243 7222 E:samples.brisbane@alsenviro.com
☐ Townsville: 14-15 Desma Ct, Bohle QLD 4818 Ph:07 4796 0600 E: townsville environmental@alsenviro.com
☐ Melbu[illegible] 2-4 Westall Rd, Springvale VIC 3171 Ph:03 8549 9600 E: samples.melbourne@alsenviro.com
☐ Adelaide: 2-1 Burma Rd, Pooraka SA 5095 Ph: 08 8359 0890 E: adelaide@alsenviro.com
☐ Perth: 10 Hod Way, Malaga WA 6090 Ph: 08 9209 7655 E: samples.perth@alsenviro.com
☐ Launceston: 27 Wellington St, Launceston TAS 7250 Ph: 03 6331 2158 E: launceston@alsenviro.com

CLIENT:	CERAR University of South Australia	TURNAROUND REQUIREMENTS : (Standard TAT may be longer for some tests e.g., Ultra Trace Organics)	☒ Standard TAT (List due date): ☐ Non Standard or urgent TAT (List due date):
OFFICE:	Mawson Lakes Campus X1-17		
PROJECT:	RWC Pb BioAcc	ALS QUOTE NO.:	
ORDER NUMBER:			
PROJECT MANAGER:	Albert Juhasz	CONTACT PH: 08 8302 5045	
SAMPLER:	Albert Juhasz	SAMPLER MOBILE: 0418 818 121	
COC emailed to ALS? (NO)		EDD FORMAT (or default):	
Email Reports to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au			
Email Invoice to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au			

COC SEQUENCE NUMBER (Circle): COC: 1 2 (3) 4 5 6 7 OF: 1 2 3 4 5 6 (7)

FOR LABORATORY USE ONLY (Circle): Custody Seal Intact? Yes / No / N/A; Free ice / frozen packs present on receipt? Yes / No; Random Sample Temperature on Receipt: °C; Other comments:

RELINQUISHED BY:	RECEIVED BY:	RELINQUISHED BY:	RECEIVED BY:
Albert Juhasz			[illegible]
DATE/TIME: 30/5/17	DATE/TIME:	DATE/TIME:	DATE/TIME: 31/5 9-00

COMMENTS/SPECIAL HANDLING/STORAGE OR DISPOSAL:

LAB USE ONLY	SAMPLE DETAILS (MATRIX: Solid(S) Water(W))			CONTAINER INFORMATION		ANALYSIS REQUIRED including SUITES (NB. Suite Codes must be listed to attract suite price). Where Metals are required, specify Total (unfiltered bottle required) or Dissolved (field filtered bottle required).								Additional Information
LAB ID	SAMPLE ID	DATE / TIME	MATRIX	TYPE & PRESERVATIVE (refer to codes below)	TOTAL BOTTLES	Total Pb								Comments on likely contaminant levels, dilutions, or samples requiring specific QC analysis etc.
25	67 5-15-2A	30/05/2017	S		1	1								
26	67 5-15-2B	30/05/2017	S		1	1								Soils have been oven dried and sieved
27	67 5-15-250A	30/05/2017	S		1	1								
28	67 5-15-250B	30/05/2017	S		1	1								
29	67 30-60-2A	30/05/2017	S		1	1								
30	67 30-60-2B	30/05/2017	S		1	1								
31	67 30-60-250A	30/05/2017	S		1	1								
32	67 30-60-250B	30/05/2017	S		1	1								
33	68-2A	30/05/2017	S		1	1								
34	68-2B	30/05/2017	S		1	1								
35	68-250A	30/05/2017	S		1	1								
36	68-250B	30/05/2017	S		1	1								
				TOTAL	12	12								

Water Container Codes: P = Unpreserved Plastic; N = Nitric Preserved Plastic; ORC = Nitric Preserved ORC; SH = Sodium Hydroxide/Cd Preserved; S = Sodium Hydroxide Preserved Plastic; AG = Amber Glass Unpreserved; AP - Airfreight Unpreserved Plastic
V = VOA Vial HCl Preserved; VB = VOA Vial Sodium Bisulphate Preserved; VS = VOA Vial Sulfuric Preserved; AV = Airfreight Unpreserved Vial SG = Sulfuric Preserved Amber Glass; H = HCl preserved Plastic; HS = HCl preserved Speciation bottle; SP = Sulfuric Preserved Plastic; F = Formaldehyde Preserved Glass;
Z = Zinc Acetate Preserved Bottle; E = EDTA Preserved Bottles; ST = Sterile Bottle; ASS = Plastic Bag for Acid Sulphate Soils; B = Unpreserved Bag.

CHAIN OF CUSTODY

ALS Laboratory: please tick →

☐ Sydney: 277 Woodpark mithfield NSW 2176 Ph: 02 8784 8555 E:samples.sydney@alsenviro.com
☐ Newcastle: 5 Rosegum Rd, Warabrook NSW 2304 Ph:02 4968 9433 E:samples.newcastle@alsenviro.com
☐ Brisbane: 32 Shand St, Stafford QLD 4053 Ph:07 3243 7222 E:samples.brisbane@alsenviro.com
☐ Townsville: 14-15 Desma Ct, Bohle QLD 4818 Ph:07 4796 0600 E: townsville.environmental@alsenviro.com
☐ Melb. 2-4 Westall Rd, Springvale VIC 3171 Ph:03 8549 9600 E: samples.melbourne@alsenviro.com
☐ Adelaide: 2-1 Burma Rd, Pooraka SA 5095 Ph: 08 8359 0890 E:adelaide@alsenviro.com
☐ Perth: 16 Hod Way, Malaga WA 6090 Ph: 08 9209 7655 F: samples.perth@alsenviro.com
☐ Launceston: 27 Wellington St, Launceston TAS 7250 Ph: 03 6331 2158 E: launceston@alsenviro.com

CLIENT:	CERAR University of South Australia	TURNAROUND REQUIREMENTS : (Standard TAT may be longer for some tests e.g., Ultra Trace Organics)	☒ Standard TAT (List due date): ☐ Non Standard or urgent TAT (List due date):
OFFICE:	Mawson Lakes Campus X1-17		
PROJECT:	RWC Pb BioAcc	ALS QUOTE NO.:	COC SEQUENCE NUMBER (Circle) COC: 1 2 3 4 5 6 7 OF: 1 2 3 4 5 6 7 8
ORDER NUMBER:			
PROJECT MANAGER:	Albert Juhasz	CONTACT PH: 08 8302 5045	
SAMPLER:	Albert Juhasz	SAMPLER MOBILE: 0418 818 121	
COC emailed to ALS? (NO)		EDD FORMAT (or default):	
Email Reports to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au			
Email Invoice to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au			

RELINQUISHED BY:	RECEIVED BY:	RELINQUISHED BY:	RECEIVED BY:
Albert Juhasz			
DATE/TIME: 30/5/17	DATE/TIME:	DATE/TIME:	DATE/TIME: 31/5 9:00

FOR LABORATORY USE ONLY (Circle)
Custody seal intact? Yes No N/A
Free ice / frozen gel packs present upon receipt? Yes No N/A
Evidence of chilling upon receipt? Yes No N/A

COMMENTS/SPECIAL HANDLING/STORAGE OR DISPOSAL:

ALS USE ONLY	SAMPLE DETAILS MATRIX: Solid(S) Water(W)			CONTAINER INFORMATION		ANALYSIS REQUIRED including SUITES (NB. Suite Codes must be listed to attract suite price) Where Metals are required, specify Total (unfiltered bottle required) or Dissolved (field filtered bottle required).								Additional Information
LAB ID	SAMPLE ID	DATE / TIME	MATRIX	TYPE & PRESERVATIVE (refer to codes below)	TOTAL BOTTLES	Total Pb								Comments on likely contaminant levels, dilutions, or samples requiring specific QC analysis etc.
37	69-2A	30/05/2017	S		1	1								Soils have been oven dried and sieved
38	69-2B	30/05/2017	S		1	1								
39	69-250A	30/05/2017	S		1	1								
40	69-250B	30/05/2017	S		1	1								
41	72-2A	30/05/2017	S		1	1								
42	72-2B	30/05/2017	S		1	1								
43	72-250A	30/05/2017	S		1	1								
44	72-250B	30/05/2017	S		1	1								
45	73-2A	30/05/2017	S		1	1								
46	73-2B	30/05/2017	S		1	1								
47	73-250A	30/05/2017	S		1	1								
48	73-250B	30/05/2017	S		1	1								
				TOTAL	12	12								

Water Container Codes: P = Unpreserved Plastic; N = Nitric Preserved Plastic; ORC = Nitric Preserved ORC; SH = Sodium Hydroxide/Cd Preserved; S = Sodium Hydroxide Preserved Plastic; AG = Amber Glass Unpreserved; AP - Airfreight Unpreserved Plastic
V = VOA Vial HCl Preserved; VB = VOA Vial Sodium Bisulphate Preserved; VS = VOA Vial Sulfuric Preserved; AV = Airfreight Unpreserved Vial SG = Sulfuric Preserved Amber Glass; H = HCl preserved Plastic; HS = HCl preserved Speciation bottle; SP = Sulfuric Preserved Plastic; F = Formaldehyde Preserved Glass;
Z = Zinc Acetate Preserved Bottle; E = EDTA Preserved Bottles; ST = Sterile Bottle; ASS = Plastic Bag for Acid Sulphate Soils; B = Unpreserved Bag.

EnRiskS

CHAIN OF CUSTODY

ALS Laboratory: please tick →

☐ **Sydney**: 277 Woodpark ... mithfield NSW 2176 Ph: 02 8784 8555 E: samples.sydney@alsenviro.com
☐ **Newcastle**: 5 Rosegum Rd, Warabrook NSW 2304 Ph:02 4968 9433 E: samples.newcastle@alsenviro.com
☐ **Brisbane**: 32 Shand St, Stafford QLD 4053 Ph:07 3243 7222 E: samples.brisbane@alsenviro.com
☐ **Townsville**: 14-15 Desma Ct, Bohle QLD 4818 Ph:07 4796 0600 E: townsville.environmental@alsenviro.com
☐ **Melbo...** 2-4 Westall Rd, Springvale VIC 3171 Ph:03 8549 9600 E: samples.melbourne@alsenviro.com
☐ **Adelaide**: 2-1 Burma Rd, Pooraka SA 5095 Ph: 08 8359 0890 E:adelaide@alsenviro.com
☐ **Perth**: 19 Hod Way, Malaga WA 6090 Ph: 08 9209 7655 E: samples.perth@alsenviro.com
☐ **Launceston**: 27 Wellington St, Launceston TAS 7250 Ph: 03 6331 2158 E: launceston@alsenviro.com

CLIENT:	**CERAR University of South Australia**	**TURNAROUND REQUIREMENTS :** (Standard TAT may be longer for some tests e.g., Ultra Trace Organics)	☒ Standard TAT **(List due date):** ☐ Non Standard or urgent TAT **(List due date):**
OFFICE:	**Mawson Lakes Campus X1-17**		
PROJECT:	**RWC Pb BioAcc**	**ALS QUOTE NO.:**	
ORDER NUMBER:			
PROJECT MANAGER:	**Albert Juhasz**	**CONTACT PH: 08 8302 5045**	
SAMPLER:	**Albert Juhasz**	**SAMPLER MOBILE: 0418 818 121**	
COC emailed to ALS? (NO)		**EDD FORMAT (or default):**	
Email Reports to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au			
Email Invoice to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au			

COC SEQUENCE NUMBER (Circle)
COC: 1 2 3 4 (5) 6 7
OF: 1 2 3 4 5 6 (7)

FOR LABORATORY USE ONLY (Circle)
Custody seal intact? Yes No N/A
Preservation and containers as per relevant protocol(s)? Yes No N/A
Random Sample Temperature on Receipt
Other comments:

RELINQUISHED BY:	**RECEIVED BY:**	**RELINQUISHED BY:**	**RECEIVED BY:**
Albert Juhasz			
DATE/TIME: 30/5/17	DATE/TIME:	DATE/TIME:	DATE/TIME: 31/5 9-00

COMMENTS/SPECIAL HANDLING/STORAGE OR DISPOSAL:

ALS USE ONLY	SAMPLE DETAILS MATRIX: Solid(S) Water(W)			CONTAINER INFORMATION		ANALYSIS REQUIRED including SUITES (NB. Suite Codes must be listed to attract suite price) Where **Metals** are required, specify **Total** (unfiltered bottle required) or **Dissolved** (field filtered bottle required).								Additional Information
LAB ID	SAMPLE ID	DATE / TIME	MATRIX	TYPE & PRESERVATIVE *(refer to codes below)*	TOTAL BOTTLES	Total Pb								Comments on likely contaminant levels, dilutions, or samples requiring specific QC analysis etc.
49	84-2A	30/05/2017	S		1	1								Soils have been oven dried and sieved
50	84-2B	30/05/2017	S		1	1								
51	84-250A	30/05/2017	S		1	1								
52	84-250B	30/05/2017	S		1	1								
53	85-2A	30/05/2017	S		1	1								
54	85-2B	30/05/2017	S		1	1								
55	85-250A	30/05/2017	S		1	1								
56	85-250B	30/05/2017	S		1	1								
				TOTAL	8	8								

Water Container Codes: P = Unpreserved Plastic; N = Nitric Preserved Plastic; ORC = Nitric Preserved ORC; SH = Sodium Hydroxide/Cd Preserved; S = Sodium Hydroxide Preserved Plastic; AG = Amber Glass Unpreserved; AP - Airfreight Unpreserved Plastic
V = VOA Vial HCl Preserved; VB = VOA Vial Sodium Bisulphate Preserved; VS = VOA Vial Sulfuric Preserved; AV = Airfreight Unpreserved Vial SG = Sulfuric Preserved Amber Glass; H = HCl preserved Plastic; HS = HCl preserved Speciation bottle; SP = Sulfuric Preserved Plastic; F = Formaldehyde Preserved Glass;
Z = Zinc Acetate Preserved Bottle; E = EDTA Preserved Bottles; ST = Sterile Bottle; ASS = Plastic Bag for Acid Sulphate Soils; B = Unpreserved Bag.

CHAIN OF CUSTODY

ALS Laboratory: please tick →

☐ **Sydney**: 277 Woodpark ...mithfield NSW 2176 Ph: 02 8784 8555 E:samples.sydney@alsenviro.com
☐ **Newcastle**: 5 Rosegum Rd. Warabrook NSW 2304 Ph:02 4968 9433 E:samples.newcastle@alsenviro.com
☐ **Brisbane**: 32 Shand St. Stafford QLD 4053 Ph:07 3243 7222 E:samples.brisbane@alsenviro.com
☐ **Townsville**: 14-15 Desma Ct. Bohle QLD 4818 Ph 07 4796 0600 E townsville.environmental@alsenviro.com
☐ **Melbo...** 2-4 Westall Rd. Springvale VIC 317... Ph:03 8549 9600 E: samples.melbourne@alsenviro.com
☐ **Adelaide**: 2-1 Burma Rd. Pooraka SA 5095 Ph: 08 8359 0890 E:adelaide@alsenviro.com
☐ **Perth**: 10 Hod Way, Malaga WA 6090 Ph: 08 9209 7655 E: samples.perth@alsenviro.com
☐ **Launceston**: 27 Wellington St. Launceston TAS 7250 Ph: 03 6331 2158 E: launceston@alsenviro.com

CLIENT:	**CERAR University of South Australia**	**TURNAROUND REQUIREMENTS :** (Standard TAT may be longer for some tests e.g., Ultra Trace Organics)	☒ Standard TAT **(List due date):**	**FOR LABORATORY USE ONLY (Circle)**	
OFFICE:	**Mawson Lakes Campus X1-17**		☐ Non Standard or urgent TAT **(List due date):**		
PROJECT:	**RWC Pb BioAcc**	**ALS QUOTE NO.:**		**COC SEQUENCE NUMBER (Circle)** COC: 1 2 3 4 5 6 7 8 OF: 1 2 3 4 5 6	
ORDER NUMBER:					
PROJECT MANAGER:	**Albert Juhasz**	**CONTACT PH: 08 8302 5045**			
SAMPLER:	**Albert Juhasz**	**SAMPLER MOBILE: 0418 818 121**	**RELINQUISHED BY:** *Albert Juhasz*	**RECEIVED BY:**	**RELINQUISHED BY:**
COC emailed to ALS? (NO)		**EDD FORMAT (or default):**	DATE/TIME: 30/5/17	DATE/TIME:	DATE/TIME:
Email Reports to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au					
Email Invoice to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au					

RECEIVED BY: DATE/TIME: 31/5 9:10

COMMENTS/SPECIAL HANDLING/STORAGE OR DISPOSAL:

ALS USE ONLY	SAMPLE DETAILS MATRIX: Solid(S) Water(W)			CONTAINER INFORMATION		ANALYSIS REQUIRED including SUITES (NB. Suite Codes must be listed to attract suite price) Where **Metals** are required, specify **Total** (unfiltered bottle required) or **Dissolved** (field filtered bottle required).								Additional Information
LAB ID	SAMPLE ID	DATE / TIME	MATRIX	TYPE & PRESERVATIVE *(refer to codes below)*	TOTAL BOTTLES	Dissolved Pb								Comments on likely contaminant levels, dilutions, or samples requiring specific QC analysis etc.
57	47A-G1	30/05/2017	W		1	1								Samples have been filtered (0.45 um)
58	47A-G2	30/05/2017	W		1	1								and are ICP ready
59	47B-G1	30/05/2017	W		1	1								
60	47B-G2	30/05/2017	W		1	1								
61	52-G1	30/05/2017	W		1	1								
62	52-G2	30/05/2017	W		1	1								
63	58-G1	30/05/2017	W		1	1								
64	58-G2	30/05/2017	W		1	1								
65	60A-G1	30/05/2017	W		1	1								
66	60A-G2	30/05/2017	W		1	1								
67	61-G1	30/05/2017	W		1	1								
68	61-G2	30/05/2017	W		1	1								
				TOTAL	12	12								

Water Container Codes: P = Unpreserved Plastic; N = Nitric Preserved Plastic; ORC = Nitric Preserved ORC; SH = Sodium Hydroxide/Cd Preserved; S = Sodium Hydroxide Preserved Plastic; AG = Amber Glass Unpreserved; AP - Airfreight Unpreserved Plastic
V = VOA Vial HCl Preserved; VB = VOA Vial Sodium Bisulphate Preserved; VS = VOA Vial Sulfuric Preserved; AV = Airfreight Unpreserved Vial SG = Sulfuric Preserved Amber Glass; H = HCl preserved Plastic; HS = HCl preserved Speciation bottle; SP = Sulfuric Preserved Plastic; F = Formaldehyde Preserved Glass;
Z = Zinc Acetate Preserved Bottle; E = EDTA Preserved Bottles; ST = Sterile Bottle; ASS = Plastic Bag for Acid Sulphate Soils; B = Unpreserved Bag.

EnRiskS

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CHAIN OF CUSTODY

ALS Laboratory: **please tick** →

☐ **Sydney**: 277 Woodpark … Smithfield NSW 2176 Ph: 02 8784 8555 E:samples.sydney@alsenviro.com
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CLIENT:	**CERAR University of South Australia**	**TURNAROUND REQUIREMENTS :** (Standard TAT may be longer for some tests e.g., Ultra Trace Organics)	☒ Standard TAT **(List due date):** ☐ Non Standard or urgent TAT **(List due date):**
OFFICE:	**Mawson Lakes Campus X1-17**		
PROJECT:	**RWC Pb BioAcc**	**ALS QUOTE NO.:**	**COC SEQUENCE NUMBER (Circle)** COC: 1 2 3 4 5 6 7 OF: 1 2 3 4 5 6 8
ORDER NUMBER:			
PROJECT MANAGER:	**Albert Juhasz**	**CONTACT PH: 08 8302 5045**	
SAMPLER:	**Albert Juhasz**	**SAMPLER MOBILE: 0418 818 121**	
COC emailed to ALS? (NO)		**EDD FORMAT (or default):**	
Email Reports to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au			
Email Invoice to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au			

RELINQUISHED BY:	**RECEIVED BY:**	**RELINQUISHED BY:**	**RECEIVED BY:**
Albert Juhasz			
DATE/TIME: 30/5/17	DATE/TIME:	DATE/TIME:	DATE/TIME: 31/5 9-10

COMMENTS/SPECIAL HANDLING/STORAGE OR DISPOSAL:

	SAMPLE DETAILS MATRIX: Solid(S) Water(W)			**CONTAINER INFORMATION**		**ANALYSIS REQUIRED including SUITES** (NB. Suite Codes must be listed to attract suite price) Where **Metals** are required, specify **Total** (unfiltered bottle required) or **Dissolved** (field filtered bottle required).								**Additional Information**
LAB ID	**SAMPLE ID**	**DATE / TIME**	**MATRIX**	**TYPE & PRESERVATIVE** *(refer to codes below)*	**TOTAL BOTTLES**	**Dissolved Pb**								Comments on likely contaminant levels, dilutions, or samples requiring specific QC analysis etc
69	67 5-15-G1	30/05/2017	W		1	1								Samples have been filtered (0.45 um)
70	67 5-15-G2	30/05/2017	W		1	1								and are ICP ready
71	67 30-60-G1	30/05/2017	W		1	1								
72	67 30-60-G2	30/05/2017	W		1	1								
73	68-G1	30/05/2017	W		1	1								
74	68-G2	30/05/2017	W		1	1								
75	69-G1	30/05/2017	W		1	1								
76	69-G2	30/05/2017	W		1	1								
77	72-G1	30/05/2017	W		1	1								
78	72-G2	30/05/2017	W		1	1								
79	73-G1	30/05/2017	W		1	1								
80	73-G2	30/05/2017	W		1	1								
				TOTAL	12	12								

Water Container Codes: P = Unpreserved Plastic; N = Nitric Preserved Plastic; ORC = Nitric Preserved ORC; SH = Sodium Hydroxide/Cd Preserved; S = Sodium Hydroxide Preserved Plastic; AG = Amber Glass Unpreserved; AP - Airfreight Unpreserved Plastic
V = VOA Vial HCl Preserved; VB = VOA Vial Sodium Bisulphate Preserved; VS = VOA Vial Sulfuric Preserved; AV = Airfreight Unpreserved Vial SG = Sulfuric Preserved Amber Glass; H = HCl preserved Plastic; HS = HCl preserved Speciation bottle; SP = Sulfuric Preserved Plastic; F = Formaldehyde Preserved Glass;
Z = Zinc Acetate Preserved Bottle; E = EDTA Preserved Bottles; ST = Sterile Bottle; ASS = Plastic Bag for Acid Sulphate Soils; B = Unpreserved Bag.

CHAIN OF CUSTODY

ALS Laboratory: please tick →

☐ Sydney: 277 Woodpark... nithfield NSW 2176 Ph: 02 8784 8555 E: samples.sydney@alsenviro.com
☐ Newcastle: 5 Rosegum Rd, Warabrook NSW 2304 Ph: 02 4968 9433 E: samples.newcastle@alsenviro.com
☐ Brisbane: 32 Shand St, Stafford QLD 4053 Ph: 07 3243 7222 E: samples.brisbane@alsenviro.com
☐ Townsville: 14-15 Desma Ct, Bohle QLD 4818 Ph: 07 4796 0600 E: townsville.environmental@alsenviro.com
☐ Melbc... 2-4 Westall Rd, Springvale VIC 3171 Ph: 03 8549 9600 E: samples.melbourne@alsenviro.com
☐ Adelaide: 2-1 Burma Rd, Pooraka SA 5095 Ph: 08 8359 0890 E: adelaide@alsenviro.com
☐ Perth: 10 Hod Way, Malaga WA 6090 Ph: 08 9209 7655 E: samples.perth@alsenviro.com
☐ Launceston: 27 Wellington St, Launceston TAS 7250 Ph: 03 6331 2158 E: launceston@alsenviro.com

CLIENT:	CERAR University of South Australia	TURNAROUND REQUIREMENTS : (Standard TAT may be longer for some tests e.g., Ultra Trace Organics)	☒ Standard TAT (List due date): ☐ Non Standard or urgent TAT (List due date):
OFFICE:	Mawson Lakes Campus X1-17		
PROJECT:	RWC Pb BioAcc	ALS QUOTE NO.:	COC SEQUENCE NUMBER (Circle) COC: 1 2 3 4 5 6 OF: 1 2 3 4 5 6
ORDER NUMBER:			
PROJECT MANAGER:	Albert Juhasz	CONTACT PH: 08 8302 5045	
SAMPLER:	Albert Juhasz	SAMPLER MOBILE: 0418 818 121	
COC emailed to ALS? (NO)		EDD FORMAT (or default):	
Email Reports to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au			
Email Invoice to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au			

RELINQUISHED BY:	RECEIVED BY:	RELINQUISHED BY:	RECEIVED BY:
Albert Juhasz			
DATE/TIME: 30/5/17	DATE/TIME:	DATE/TIME:	DATE/TIME: 31/5 9-10

COMMENTS/SPECIAL HANDLING/STORAGE OR DISPOSAL:

	SAMPLE DETAILS MATRIX: Solid(S) Water(W)			CONTAINER INFORMATION		ANALYSIS REQUIRED including SUITES (NB. Suite Codes must be listed to attract suite price) Where Metals are required, specify Total (unfiltered bottle required) or Dissolved (field filtered bottle required).								Additional Information
LAB ID	SAMPLE ID	DATE / TIME	MATRIX	TYPE & PRESERVATIVE (refer to codes below)	TOTAL BOTTLES	Dissolved Pb								Comments on likely contaminant levels, dilutions, or samples requiring specific QC analysis etc.
81	84-G1	30/05/2017	W		1	1								Samples have been filtered (0.45 um)
82	84-G2	30/05/2017	W		1	1								and are ICP ready.
83	85-G1	30/05/2017	W		1	1								
84	85-G2	30/05/2017	W		1	1								
85	QC1-[redacted]1	30/05/2017	W		1	1								
86	QC1-[redacted]2	30/05/2017	W		1	1								
87	QC2-[redacted]1	30/05/2017	W		1	1								
88	QC2-[redacted]2	30/05/2017	W		1	1								
				TOTAL	8	8								

Water Container Codes: P = Unpreserved Plastic; N = Nitric Preserved Plastic; ORC = Nitric Preserved ORC; SH = Sodium Hydroxide/Cd Preserved; S = Sodium Hydroxide Preserved Plastic; AG = Amber Glass Unpreserved; AP - Airfreight Unpreserved Plastic
V = VOA Vial HCl Preserved; VB = VOA Vial Sodium Bisulphate Preserved; VS = VOA Vial Sulfuric Preserved; AV = Airfreight Unpreserved Vial SG = Sulfuric Preserved Amber Glass; H = HCl preserved Plastic; HS = HCl preserved Speciation bottle; SP = Sulfuric Preserved Plastic; F = Formaldehyde Preserved Glass;
Z = Zinc Acetate Preserved Bottle; E = EDTA Preserved Bottles; ST = Sterile Bottle; ASS = Plastic Bag for Acid Sulphate Soils; B = Unpreserved Bag.

EnRiskS

CHAIN OF CUSTODY

ALS Laboratory: please tick →

☐ **Sydney** 277 Woodpark Rd, Smithfield NSW 2176 Ph: 02 8784 8555 E:samples.sydney@alsenviro.com
☐ **Newcastle:** 5 Rosegum Rd, Warabrook NSW 2304 Ph:02 4968 9433 E:samples.newcastle@alsenviro.com
☐ **Brisbane:** 32 Shand St, Stafford QLD 4053 Ph:07 3243 7222 E:samples.brisbane@alsenviro.com
☐ **Townsville:** 14-15 Desma Ct, Bohle QLD 4818 Ph:07 4796 0600 E: townsville.environmental@alsenviro.com
☐ **Melbourne:** 2-4 Westall Rd, Springvale VIC 3171 Ph:03 8549 9600 E: samples.melbourne@alsenviro.com
☐ **Adelaide:** 2-1 Burma Rd, Pooraka SA 5095 Ph: 08 8359 0890 E:adelaide@alsenviro.com
☐ **Perth** 10 Hod Way, Malaga WA 6090 Ph: 08 9209 7655 E: samples.perth@alsenviro.com
☐ **Launceston** 27 Chesterman St, Launceston TAS 7250 Ph: 03 6323 2800 E: launceston@alsenviro.com

FREIGHT

CLIENT:	**CERAR University of South Australia**	**TURNAROUND REQUIREMENTS :** (Standard TAT may be longer for some tests e.g., Ultra Trace Organics)	☒ Standard TAT **(List due date):**	**FOR LABORATORY USE ONLY (Circle)**	
OFFICE:	**Mawson Lakes Campus X1-17**		☐ Non Standard or urgent TAT **(List due date):**	Custody Seal Intact?	Yes / No / N/A
PROJECT:	**RWC Pb BioAcc**	**ALS QUOTE NO.:**		Free Ice (frozen gel packs present upon receipt)?	Yes / No / N/A
ORDER NUMBER:			**COC SEQUENCE NUMBER (Circle)**	Random Sample Temperature upon Receipt:	°C
PROJECT MANAGER:	**Albert Juhasz**	**CONTACT PH: 08 8302 5045**	COC: (1) 2 3 4 5 6 7	Other comments:	
SAMPLER:	**Albert Juhasz**	**SAMPLER MOBILE: 0418 818 121**	OF: (1) 2 3 4 5 6 7		

RELINQUISHED BY:	**RECEIVED BY:**	**RELINQUISHED BY:**	**RECEIVED BY:**
Albert Juhasz			
DATE/TIME: 5/6/17	DATE/TIME:	DATE/TIME:	DATE/TIME: 6/6 1035

Email Reports to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au
Email Invoice to (will default to PM if no other addresses are listed): Albert.Juhasz@unisa.edu.au
COC emailed to ALS? (NO) **EDD FORMAT (or default):**

COMMENTS/SPECIAL HANDLING/STORAGE OR DISPOSAL:

ALS USE ONLY	SAMPLE DETAILS MATRIX: Solid(S) Water(W)			CONTAINER INFORMATION		ANALYSIS REQUIRED including SUITES (NB. Suite Codes must be listed to attract suite price) Where **Metals** are required, specify **Total** (unfiltered bottle required) or **Dissolved** (field filtered bottle required).								Additional Information
LAB ID	SAMPLE ID	DATE / TIME	MATRIX	TYPE & PRESERVATIVE *(refer to codes below)*	TOTAL BOTTLES	Total Pb								Comments on likely contaminant levels, dilutions, or samples requiring specific QC analysis etc.
9	52-2A	30/05/2017	S		1	1								Soils have been oven dried and sieved
19	60A-250A	30/05/2017	S		1	1								
41	72-2A	30/05/2017	S		1	1								
51	84-250A	30/05/2017	S		1	1								
				TOTAL	4	4								

Water Container Codes: P = Unpreserved Plastic; N = Nitric Preserved Plastic; ORC = Nitric Preserved ORC; SH = Sodium Hydroxide/Cd Preserved; S = Sodium Hydroxide Preserved Plastic; AG = Amber Glass Unpreserved; AP - Airfreight Unpreserved Plastic
V = VOA Vial HCl Preserved; VB = VOA Vial Sodium Bisulphate Preserved; VS = VOA Vial Sulfuric Preserved; AV = Airfreight Unpreserved Vial SG = Sulfuric Preserved Amber Glass; H = HCl preserved Plastic; HS = HCl preserved Speciation bottle; SP = Sulfuric Preserved Plastic; F = Formaldehyde Preserved Glass;
Z = Zinc Acetate Preserved Bottle; E = EDTA Preserved Bottles; ST = Sterile Bottle; ASS = Plastic Bag for Acid Sulphate Soils; B = Unpreserved Bag.

Albert Juhasz

From: Justin Wilson <justin.wilson@ALSGlobal.com>
Sent: Friday, 2 June 2017 12:12 PM
To: Albert Juhasz
Subject: RWC Pb BioAcc
Attachments: EM1706956_COC.pdf

Follow Up Flag: Follow up
Flag Status: Flagged

Hi Albert

Regarding the attached COC, due to an error in our laboratory, we are unable to complete the testing on four samples as listed below.

9 – 52-2A
19 – 60A-250A
41 – 72-2A
51 – 84-250A

Are you able to supply us with extra sample to complete these tests?

Sorry for any inconvenience caused.

Regards

Justin Wilson
Client Services Officer
Environmental

T +61 3 8549 9600 **D** +61 3 8549 9644
F +61 3 8549 9626
justin.wilson@alsglobal.com
2-4 Westall Rd
Springvale Vic 3171
Australia

APPENDIX 3 – ANALYTICAL RESULTS AND QA/QC

ALS Environmental

CERTIFICATE OF ANALYSIS

Work Order	: **EM1706956**	Page	: 1 of 21
Client	: **UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION**	Laboratory	: Environmental Division Melbourne
Contact	: MR ALBERT JUHASZ	Contact	:
Address	: UNIVERSITY OF SOUTH AUSTRALIA CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION BUILDING X MAWSON LAKES CAMPUS MAWSON LAKES SOUTH AUSTRALIA 5095	Address	: 4 Westall Rd Springvale VIC Australia 3171
Telephone	: +61 08 8302 6273	Telephone	: +61-3-8549 9600
Project	: RWC Pb BioAcc	Date Samples Received	: 31-May-2017 09:10
Order number	: ----	Date Analysis Commenced	: 31-May-2017
C-O-C number	: ----	Issue Date	: 08-Jun-2017 09:36
Sampler	: ALBERT JUHASZ		
Site	: ----		
Quote number	: ADBQ/011/10		
No. of samples received	: 88		
No. of samples analysed	: 88		

ilac-MRA NATA
Accreditation No. 825
Accredited for compliance with ISO/IEC 17025 - Testing

This report supersedes any previous report(s) with this reference. Results apply to the sample(s) as submitted. This document shall not be reproduced, except in full.

This Certificate of Analysis contains the following information:

- General Comments
- Analytical Results

Additional information pertinent to this report will be found in the following separate attachments: Quality Control Report, QA/QC Compliance Assessment to assist with Quality Review and Sample Receipt Notification.

Signatories

This document has been electronically signed by the authorized signatories below. Electronic signing is carried out in compliance with procedures specified in 21 CFR Part 11.

Signatories	*Position*	*Accreditation Category*
Chris Lemaitre	Non-Metals Team Leader	Melbourne Inorganics, Springvale, VIC
Dilani Fernando	Senior Inorganic Chemist	Melbourne Inorganics, Springvale, VIC

General Comments

The analytical procedures used by the Environmental Division have been developed from established internationally recognized procedures such as those published by the USEPA, APHA, AS and NEPM. In house developed procedures are employed in the absence of documented standards or by client request.

Where moisture determination has been performed, results are reported on a dry weight basis.

Where a reported less than (<) result is higher than the LOR, this may be due to primary sample extract/digestate dilution and/or insufficient sample for analysis.

Where the LOR of a reported result differs from standard LOR, this may be due to high moisture content, insufficient sample (reduced weight employed) or matrix interference.

When no sampling time is provided, the sampling time will default 00:00 on the date of sampling. If no sampling date is provided, the sampling date will be assumed by the laboratory and displayed in brackets without a time component.

Where a result is required to meet compliance limits the associated uncertainty must be considered. Refer to the ALS Contact for details.

Key : CAS Number = CAS registry number from database maintained by Chemical Abstracts Services. The Chemical Abstracts Service is a division of the American Chemical Society.
LOR = Limit of reporting
^ = This result is computed from individual analyte detections at or above the level of reporting
ø = ALS is not NATA accredited for these tests.
~ = Indicates an estimated value.

Page	:	3 of 21
Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: **SOIL** (Matrix: **SOIL**)				Client sample ID	47A-2A	47A-2B	47A-250A	47A-250B	47B-2A
				Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	*CAS Number*	*LOR*	*Unit*		**EM1706956-001**	**EM1706956-002**	**EM1706956-003**	**EM1706956-004**	**EM1706956-005**
					Result	Result	Result	Result	Result
EA055: Moisture Content									
Moisture Content (dried @ 103°C)	----	1	%		1.4	1.8	1.6	1.7	<1.0
EG005T: Total Metals by ICP-AES									
Lead	7439-92-1	5	mg/kg		21	22	25	24	16

Page	:	4 of 21
Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: **SOIL** (Matrix: **SOIL**)			*Client sample ID*	**47B-2B**	**47B-250A**	**47B-250B**	**52-2A**	**52-2B**
			Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	*CAS Number*	*LOR*	*Unit*	**EM1706956-006**	**EM1706956-007**	**EM1706956-008**	**EM1706956-009**	**EM1706956-010**
				Result	Result	Result	Result	Result
EA055: Moisture Content								
Moisture Content (dried @ 103°C)	----	1	%	<1.0	<1.0	<1.0	<1.0	**1.4**
EG005T: Total Metals by ICP-AES								
Lead	7439-92-1	5	mg/kg	**22**	**12**	**12**	**16**	**16**

Page	:	5 of 21
Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: **SOIL** (Matrix: **SOIL**)			Client sample ID	**52-250A**	**52-250B**	**58-2A**	**58-2B**	**58-250A**
			Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	*CAS Number*	*LOR*	*Unit*	**EM1706956-011**	**EM1706956-012**	**EM1706956-013**	**EM1706956-014**	**EM1706956-015**
				Result	Result	Result	Result	Result
EA055: Moisture Content								
Moisture Content (dried @ 103°C)	----	1	%	**1.1**	**1.7**	**1.1**	<1.0	<1.0
EG005T: Total Metals by ICP-AES								
Lead	7439-92-1	5	mg/kg	**12**	**13**	**13**	**11**	**13**

Analytical Results

Sub-Matrix: SOIL (Matrix: SOIL)				Client sample ID	58-250B	60A-2A	60A-2B	60A-250A	60A-250B
				Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	CAS Number	LOR	Unit		EM1706956-016	EM1706956-017	EM1706956-018	EM1706956-019	EM1706956-020
					Result	Result	Result	Result	Result
EA055: Moisture Content									
Moisture Content (dried @ 103°C)	----	1	%		<1.0	1.4	1.4	1.6	1.3
EG005T: Total Metals by ICP-AES									
Lead	7439-92-1	5	mg/kg		12	10	9	8	8

Page	:	7 of 21
Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: **SOIL** (Matrix: **SOIL**)				Client sample ID	61-2A	61-2B	61-250A	61-250B	67 5-15-2A
				Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	*CAS Number*	*LOR*	*Unit*		**EM1706956-021**	**EM1706956-022**	**EM1706956-023**	**EM1706956-024**	**EM1706956-025**
					Result	Result	Result	Result	Result
EA055: Moisture Content									
Moisture Content (dried @ 103°C)	----	1	%		<1.0	<1.0	**1.9**	**1.4**	<1.0
EG005T: Total Metals by ICP-AES									
Lead	7439-92-1	5	mg/kg		**9**	**7**	**16**	**16**	**396**

Analytical Results

Sub-Matrix: SOIL (Matrix: SOIL)				Client sample ID	67 5-15-2B	67 5-15-250A	67 5-15-250B	67 30-60-2A	67 30-60-2B
				Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	CAS Number	LOR	Unit		EM1706956-026	EM1706956-027	EM1706956-028	EM1706956-029	EM1706956-030
					Result	Result	Result	Result	Result
EA055: Moisture Content									
Moisture Content (dried @ 103°C)	----	1	%		<1.0	<1.0	<1.0	<1.0	<1.0
EG005T: Total Metals by ICP-AES									
Lead	7439-92-1	5	mg/kg		355	152	143	536	695

Page	:	9 of 21
Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: **SOIL** (Matrix: **SOIL**)				Client sample ID	67 30-60-250A	67 30-60-250B	68-2A	68-2B	68-250A
				Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	*CAS Number*	*LOR*	*Unit*		**EM1706956-031**	**EM1706956-032**	**EM1706956-033**	**EM1706956-034**	**EM1706956-035**
					Result	Result	Result	Result	Result
EA055: Moisture Content									
Moisture Content (dried @ 103°C)	----	1	%		<1.0	<1.0	1.1	1.3	1.0
EG005T: Total Metals by ICP-AES									
Lead	7439-92-1	5	mg/kg		310	301	78	90	83

Analytical Results

Sub-Matrix: SOIL (Matrix: SOIL)				Client sample ID	68-250B	69-2A	69-2B	69-250A	69-250B
				Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	CAS Number	LOR	Unit		EM1706956-036	EM1706956-037	EM1706956-038	EM1706956-039	EM1706956-040
					Result	Result	Result	Result	Result
EA055: Moisture Content									
Moisture Content (dried @ 103°C)	----	1	%		1.3	<1.0	<1.0	<1.0	<1.0
EG005T: Total Metals by ICP-AES									
Lead	7439-92-1	5	mg/kg		80	<5	<5	7	6

Page	:	11 of 21
Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: **SOIL** (Matrix: **SOIL**)				Client sample ID	72-2A	72-2B	72-250A	72-250B	73-2A
				Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	*CAS Number*	*LOR*	*Unit*		**EM1706956-041**	**EM1706956-042**	**EM1706956-043**	**EM1706956-044**	**EM1706956-045**
					Result	Result	Result	Result	Result
EA055: Moisture Content									
Moisture Content (dried @ 103°C)	----	1	%		<1.0	<1.0	<1.0	<1.0	<1.0
EG005T: Total Metals by ICP-AES									
Lead	7439-92-1	5	mg/kg		**10**	**11**	**9**	**9**	**9**

Analytical Results

Sub-Matrix: SOIL (Matrix: SOIL)			Client sample ID	73-2B	73-250A	73-250B	84-2A	84-2B
			Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	CAS Number	LOR	Unit	EM1706956-046	EM1706956-047	EM1706956-048	EM1706956-049	EM1706956-050
				Result	Result	Result	Result	Result
EA055: Moisture Content								
Moisture Content (dried @ 103°C)	----	1	%	<1.0	<1.0	<1.0	<1.0	<1.0
EG005T: Total Metals by ICP-AES								
Lead	7439-92-1	5	mg/kg	8	9	9	8	7

Page	:	13 of 21
Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: **SOIL** (Matrix: **SOIL**)				Client sample ID	84-250A	84-250B	85-2A	85-2B	85-250A
				Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	*CAS Number*	*LOR*	*Unit*		**EM1706956-051**	**EM1706956-052**	**EM1706956-053**	**EM1706956-054**	**EM1706956-055**
					Result	Result	Result	Result	Result
EA055: Moisture Content									
Moisture Content (dried @ 103°C)	----	1	%		**1.2**	<1.0	<1.0	<1.0	<1.0
EG005T: Total Metals by ICP-AES									
Lead	7439-92-1	5	mg/kg		**8**	**9**	**12**	**12**	**14**

Analytical Results

Sub-Matrix: **SOIL** (Matrix: **SOIL**)				*Client sample ID*	**85-250B**	----	----	----	----
				Client sampling date / time	30-May-2017 00:00	----	----	----	----
Compound	*CAS Number*	*LOR*	*Unit*		**EM1706956-056**	------	------	------	------
					Result	----	----	----	----
EA055: Moisture Content									
Moisture Content (dried @ 103°C)	----	1	%		<1.0	---	---	---	---
EG005T: Total Metals by ICP-AES									
Lead	7439-92-1	5	mg/kg		**13**	---	---	---	---

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Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: WATER (Matrix: WATER)				Client sample ID	47A-G1	47A-G2	47B-G1	47B-G2	52-G1
				Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	CAS Number	LOR	Unit		EM1706956-057	EM1706956-058	EM1706956-059	EM1706956-060	EM1706956-061
					Result	Result	Result	Result	Result
EG005F: Dissolved Metals by ICP-AES									
Lead	7439-92-1	0.01	mg/L		**0.09**	**0.08**	**0.04**	**0.03**	**0.04**

Analytical Results

Sub-Matrix: **WATER** (Matrix: **WATER**)			Client sample ID	52-G2	58-G1	58-G2	60A-G1	60A-G2
			Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	*CAS Number*	*LOR*	*Unit*	EM1706956-062	EM1706956-063	EM1706956-064	EM1706956-065	EM1706956-066
				Result	Result	Result	Result	Result
EG005F: Dissolved Metals by ICP-AES								
Lead	7439-92-1	0.01	mg/L	**0.03**	**0.05**	**0.06**	**0.03**	**0.02**

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Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: WATER (Matrix: WATER)				Client sample ID	61-G1	61-G2	67 5-15-G1	67 5-15-G2	67 30-60-G1
				Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	CAS Number	LOR	Unit		EM1706956-067	EM1706956-068	EM1706956-069	EM1706956-070	EM1706956-071
					Result	Result	Result	Result	Result
EG005F: Dissolved Metals by ICP-AES									
Lead	7439-92-1	0.01	mg/L		**0.06**	**0.06**	**0.27**	**0.28**	**0.44**

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Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: **WATER** (Matrix: **WATER**)			Client sample ID	**67 30-60-G2**	**68-G1**	**68-G2**	**69-G1**	**69-G2**
			Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	*CAS Number*	*LOR*	*Unit*	**EM1706956-072**	**EM1706956-073**	**EM1706956-074**	**EM1706956-075**	**EM1706956-076**
				Result	Result	Result	Result	Result
EG005F: Dissolved Metals by ICP-AES								
Lead	7439-92-1	0.01	mg/L	**0.45**	**0.20**	**0.19**	**0.04**	**0.03**

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Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: **WATER** (Matrix: **WATER**)				Client sample ID	72-G1	72-G2	73-G1	73-G2	84-G1
				Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	*CAS Number*	*LOR*	*Unit*		**EM1706956-077**	**EM1706956-078**	**EM1706956-079**	**EM1706956-080**	**EM1706956-081**
					Result	Result	Result	Result	Result
EG005F: Dissolved Metals by ICP-AES									
Lead	7439-92-1	0.01	mg/L		**0.04**	**0.04**	**0.04**	**0.04**	**0.02**

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Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: WATER (Matrix: WATER)				Client sample ID	84-G2	85-G1	85-G2	QC1-1	QC1-2
				Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00	30-May-2017 00:00
Compound	CAS Number	LOR	Unit		EM1706956-082	EM1706956-083	EM1706956-084	EM1706956-085	EM1706956-086
					Result	Result	Result	Result	Result
EG005F: Dissolved Metals by ICP-AES									
Lead	7439-92-1	0.01	mg/L		**0.02**	**0.04**	**0.04**	**47.2**	**47.6**

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Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Analytical Results

Sub-Matrix: **WATER** (Matrix: **WATER**)			Client sample ID	**QC2-1**	**QC2-2**	----	----	----
			Client sampling date / time	30-May-2017 00:00	30-May-2017 00:00	----	----	----
Compound	*CAS Number*	*LOR*	*Unit*	**EM1706956-087**	**EM1706956-088**	-------	-------	-------
				Result	Result	----	----	----
EG005F: Dissolved Metals by ICP-AES								
Lead	7439-92-1	0.01	mg/L	**0.09**	**0.03**	---	---	---

Environmental

QUALITY CONTROL REPORT

Work Order	: **EM1706956**	Page	: 1 of 3
Client	: **UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION**	Laboratory	: Environmental Division Melbourne
Contact	: MR ALBERT JUHASZ	Contact	:
Address	: UNIVERSITY OF SOUTH AUSTRALIA CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION BUILDING X MAWSON LAKES CAMPUS MAWSON LAKES SOUTH AUSTRALIA 5095	Address	: 4 Westall Rd Springvale VIC Australia 3171
Telephone	: +61 08 8302 6273	Telephone	: +61-3-8549 9600
Project	: RWC Pb BioAcc	Date Samples Received	: 31-May-2017
Order number	: ----	Date Analysis Commenced	: 31-May-2017
C-O-C number	: ----	Issue Date	: 08-Jun-2017
Sampler	: ALBERT JUHASZ		
Site	: ----		
Quote number	: ADBQ/011/10		
No. of samples received	: 88		
No. of samples analysed	: 88		

Accreditation No. 825
Accredited for compliance with ISO/IEC 17025 - Testing

This report supersedes any previous report(s) with this reference. Results apply to the sample(s) as submitted. This document shall not be reproduced, except in full.

This Quality Control Report contains the following information:

- Laboratory Duplicate (DUP) Report; Relative Percentage Difference (RPD) and Acceptance Limits
- Method Blank (MB) and Laboratory Control Spike (LCS) Report; Recovery and Acceptance Limits
- Matrix Spike (MS) Report; Recovery and Acceptance Limits

Signatories

This document has been electronically signed by the authorized signatories below. Electronic signing is carried out in compliance with procedures specified in 21 CFR Part 11.

Signatories	*Position*	*Accreditation Category*
Chris Lemaitre	Non-Metals Team Leader	Melbourne Inorganics, Springvale, VIC
Dilani Fernando	Senior Inorganic Chemist	Melbourne Inorganics, Springvale, VIC

Page	:	2 of 3
Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

General Comments

The analytical procedures used by the Environmental Division have been developed from established internationally recognized procedures such as those published by the USEPA, APHA, AS and NEPM. In house developed procedures are employed in the absence of documented standards or by client request.

Where moisture determination has been performed, results are reported on a dry weight basis.

Where a reported less than (<) result is higher than the LOR, this may be due to primary sample extract/digestate dilution and/or insufficient sample for analysis. Where the LOR of a reported result differs from standard LOR, this may be due to high

Key : Anonymous = Refers to samples which are not specifically part of this work order but formed part of the QC process lot
CAS Number = CAS registry number from database maintained by Chemical Abstracts Services. The Chemical Abstracts Service is a division of the American Chemical Society.
LOR = Limit of reporting
RPD = Relative Percentage Difference
# = Indicates failed QC

Laboratory Duplicate (DUP) Report

The quality control term Laboratory Duplicate refers to a randomly selected intralaboratory split. Laboratory duplicates provide information regarding method precision and sample heterogeneity. The permitted ranges for the Relative Percent Deviation (RPD) of Laboratory Duplicates are specified in ALS Method QWI-EN/38 and are dependent on the magnitude of results in comparison to the level of reporting: Result < 10 times LOR: No Limit; Result between 10 and 20 times LOR: 0% - 50%; Result > 20 times LOR: 0% - 20%.

Sub-Matrix: **SOIL**

				Laboratory Duplicate (DUP) Report					
Laboratory sample ID	*Client sample ID*	*Method: Compound*	*CAS Number*	*LOR*	*Unit*	*Original Result*	*Duplicate Result*	*RPD (%)*	*Recovery Limits (%)*
EA055: Moisture Content (QC Lot: 920581)									
EM1706956-009	52-2A	EA055-103: Moisture Content (dried @ 103°C)	----	1	%	<1.0	<1.0	0.00	No Limit
EM1706956-019	60A-250A	EA055-103: Moisture Content (dried @ 103°C)	----	1	%	1.6	1.7	9.24	No Limit
EA055: Moisture Content (QC Lot: 920582)									
EM1706956-021	61-2A	EA055-103: Moisture Content (dried @ 103°C)	----	1	%	<1.0	<1.0	0.00	No Limit
EM1706956-030	67 30-60-2B	EA055-103: Moisture Content (dried @ 103°C)	----	1	%	<1.0	<1.0	0.00	No Limit
EA055: Moisture Content (QC Lot: 920583)									
EM1706956-041	72-2A	EA055-103: Moisture Content (dried @ 103°C)	----	1	%	<1.0	<1.0	0.00	No Limit
EM1706956-051	84-250A	EA055-103: Moisture Content (dried @ 103°C)	----	1	%	1.2	1.2	0.00	No Limit
EG005T: Total Metals by ICP-AES (QC Lot: 920605)									
EM1706956-001	47A-2A	EG005T: Lead	7439-92-1	5	mg/kg	21	19	10.3	No Limit
EM1706956-010	52-2B	EG005T: Lead	7439-92-1	5	mg/kg	16	16	0.00	No Limit
EG005T: Total Metals by ICP-AES (QC Lot: 920606)									
EM1706956-021	61-2A	EG005T: Lead	7439-92-1	5	mg/kg	9	10	0.00	No Limit
EM1706956-030	67 30-60-2B	EG005T: Lead	7439-92-1	5	mg/kg	695	690	0.592	0% - 20%
EG005T: Total Metals by ICP-AES (QC Lot: 921372)									
EM1706982-002	Anonymous	EG005T: Lead	7439-92-1	5	mg/kg	64	58	9.67	0% - 50%
EM1706982-001	Anonymous	EG005T: Lead	7439-92-1	5	mg/kg	46	44	4.02	No Limit
EG005T: Total Metals by ICP-AES (QC Lot: 923931)									
EM1706956-009	52-2A	EG005T: Lead	7439-92-1	5	mg/kg	16	16	0.00	No Limit

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Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Method Blank (MB) and Laboratory Control Spike (LCS) Report

The quality control term Method / Laboratory Blank refers to an analyte free matrix to which all reagents are added in the same volumes or proportions as used in standard sample preparation. The purpose of this QC parameter is to monitor potential laboratory contamination. The quality control term Laboratory Control Spike (LCS) refers to a certified reference material, or a known interference free matrix spiked with target analytes. The purpose of this QC parameter is to monitor method precision and accuracy independent of sample matrix. Dynamic Recovery Limits are based on statistical evaluation of processed LCS.

Sub-Matrix: **SOIL**

				Method Blank (MB) Report	Laboratory Control Spike (LCS) Report			
					Spike	Spike Recovery (%)	Recovery Limits (%)	
Method: Compound	CAS Number	LOR	Unit	Result	Concentration	LCS	Low	High
EG005T: Total Metals by ICP-AES (QCLot: 920605)								
EG005T: Lead	7439-92-1	5	mg/kg	<5	40 mg/kg	97.3	85	107
EG005T: Total Metals by ICP-AES (QCLot: 920606)								
EG005T: Lead	7439-92-1	5	mg/kg	<5	40 mg/kg	97.6	85	107
EG005T: Total Metals by ICP-AES (QCLot: 921372)								
EG005T: Lead	7439-92-1	5	mg/kg	<5	40 mg/kg	100.0	85	107
EG005T: Total Metals by ICP-AES (QCLot: 923931)								
EG005T: Lead	7439-92-1	5	mg/kg	<5	40 mg/kg	94.0	85	107

Sub-Matrix: **WATER**

				Method Blank (MB) Report	Laboratory Control Spike (LCS) Report			
					Spike	Spike Recovery (%)	Recovery Limits (%)	
Method: Compound	CAS Number	LOR	Unit	Result	Concentration	LCS	Low	High
EG005F: Dissolved Metals by ICP-AES (QCLot: 918857)								
EG005F: Lead	7439-92-1	0.01	mg/L	<0.01	1 mg/L	99.6	84	111
EG005F: Dissolved Metals by ICP-AES (QCLot: 918858)								
EG005F: Lead	7439-92-1	0.01	mg/L	<0.01	1 mg/L	102	84	111

Matrix Spike (MS) Report

The quality control term Matrix Spike (MS) refers to an intralaboratory split sample spiked with a representative set of target analytes. The purpose of this QC parameter is to monitor potential matrix effects on analyte recoveries. Static Recovery Limits as per laboratory Data Quality Objectives (DQOs). Ideal recovery ranges stated may be waived in the event of sample matrix interference.

Sub-Matrix: **SOIL**

				Matrix Spike (MS) Report			
				Spike	SpikeRecovery(%)	Recovery Limits (%)	
Laboratory sample ID	Client sample ID	Method: Compound	CAS Number	Concentration	MS	Low	High
EG005T: Total Metals by ICP-AES (QCLot: 920605)							
EM1706956-002	47A-2B	EG005T: Lead	7439-92-1	50 mg/kg	92.6	76	124
EG005T: Total Metals by ICP-AES (QCLot: 920606)							
EM1706956-022	61-2B	EG005T: Lead	7439-92-1	50 mg/kg	95.2	76	124
EG005T: Total Metals by ICP-AES (QCLot: 921372)							
EM1706956-042	72-2B	EG005T: Lead	7439-92-1	50 mg/kg	82.8	76	124
EG005T: Total Metals by ICP-AES (QCLot: 923931)							
EM1706956-019	60A-250A	EG005T: Lead	7439-92-1	50 mg/kg	102	76	124

ALS Environmental

QA/QC Compliance Assessment to assist with Quality Review

Work Order	: **EM1706956**	Page	: 1 of 7
Client	: **UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION**	Laboratory	: Environmental Division Melbourne
Contact	: MR ALBERT JUHASZ	Telephone	: +61-3-8549 9600
Project	: RWC Pb BioAcc	Date Samples Received	: 31-May-2017
Site	: ----	Issue Date	: 08-Jun-2017
Sampler	: ALBERT JUHASZ	No. of samples received	: 88
Order number	: ----	No. of samples analysed	: 88

This report is automatically generated by the ALS LIMS through interpretation of the ALS Quality Control Report and several Quality Assurance parameters measured by ALS. This automated reporting highlights any non-conformances, facilitates faster and more accurate data validation and is designed to assist internal expert and external Auditor review. Many components of this report contribute to the overall DQO assessment and reporting for guideline compliance.

Brief method summaries and references are also provided to assist in traceability.

Summary of Outliers

Outliers : Quality Control Samples

This report highlights outliers flagged in the Quality Control (QC) Report.

- NO Method Blank value outliers occur.
- NO Duplicate outliers occur.
- NO Laboratory Control outliers occur.
- NO Matrix Spike outliers occur.
- For all regular sample matrices, NO surrogate recovery outliers occur.

Outliers : Analysis Holding Time Compliance

- NO Analysis Holding Time Outliers exist.

Outliers : Frequency of Quality Control Samples

- Quality Control Sample Frequency Outliers exist - please see following pages for full details.

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Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Outliers : Frequency of Quality Control Samples

Matrix: **WATER**

Quality Control Sample Type	Count		Rate (%)		Quality Control Specification
Method	QC	Regular	Actual	Expected	
Laboratory Duplicates (DUP)					
Dissolved Metals by ICP-AES	0	32	0.00	10.00	NEPM 2013 B3 & ALS QC Standard
Laboratory Control Samples (LCS)					
Dissolved Metals by ICP-AES	2	32	6.25	10.00	NEPM 2013 B3 & ALS QC Standard
Matrix Spikes (MS)					
Dissolved Metals by ICP-AES	0	32	0.00	5.00	NEPM 2013 B3 & ALS QC Standard

Analysis Holding Time Compliance

If samples are identified below as having been analysed or extracted outside of recommended holding times, this should be taken into consideration when interpreting results.

This report summarizes extraction / preparation and analysis times and compares each with ALS recommended holding times (referencing USEPA SW 846, APHA, AS and NEPM) based on the sample container provided. Dates reported represent first date of extraction or analysis and preclude subsequent dilutions and reruns. A listing of breaches (if any) is provided herein.

Holding time for leachate methods (e.g. TCLP) vary according to the analytes reported. Assessment compares the leach date with the shortest analyte holding time for the equivalent soil method. These are: organics 14 days, mercury 28 days & other metals 180 days. A recorded breach does not guarantee a breach for all non-volatile parameters.

Holding times for ***VOC in soils*** vary according to analytes of interest. Vinyl Chloride and Styrene holding time is 7 days; others 14 days. A recorded breach does not guarantee a breach for all VOC analytes and should be verified in case the reported breach is a false positive or Vinyl Chloride and Styrene are not key analytes of interest/concern.

Matrix: **SOIL**

Evaluation: × = Holding time breach ; ✓ = Within holding time.

Method	***Sample Date***	*Extraction / Preparation*			*Analysis*		
Container / *Client Sample ID(s)*		***Date extracted***	*Due for extraction*	*Evaluation*	***Date analysed***	*Due for analysis*	*Evaluation*
EA055: Moisture Content							
Plastic Tube (EA055-103)							

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Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Matrix: **SOIL**

Evaluation: × = Holding time breach ; ✓ = Within holding time.

Method / Container / Client Sample ID(s)		Sample Date	Extraction / Preparation			Analysis		
			Date extracted	Due for extraction	Evaluation	Date analysed	Due for analysis	Evaluation
EA055: Moisture Content - Continued								
47A-2A,	47A-2B,	30-May-2017	----	----	----	01-Jun-2017	13-Jun-2017	✓
47A-250A,	47A-250B,							
47B-2A,	47B-2B,							
47B-250A,	47B-250B,							
52-2A,	52-2B,							
52-250A,	52-250B,							
58-2A,	58-2B,							
58-250A,	58-250B,							
60A-2A,	60A-2B,							
60A-250A,	60A-250B,							
61-2A,	61-2B,							
61-250A,	61-250B,							
67 5-15-2A,	67 5-15-2B,							
67 5-15-250A,	68-2A, 67 5-15-250B,							
67 30-60-2A,	67 30-60-2B,							
67 30-60-250A,	67 30-60-250B,							
68-2B,								
68-250A,	68-250B,							
69-2A,	69-2B,							
69-250A,	69-250B,							
72-2A,	72-2B,							
72-250A,	72-250B,							
73-2A,	73-2B,							
73-250A,	73-250B,							
84-2A,	84-2B,							
84-250A,	84-250B,							
85-2A,	85-2B,							
85-250A,	85-250B							

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Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Matrix: **SOIL**

Evaluation: × = Holding time breach ; ✓ = Within holding time.

Method / Container / Client Sample ID(s)		Sample Date	Extraction / Preparation			Analysis		
			Date extracted	Due for extraction	Evaluation	Date analysed	Due for analysis	Evaluation
EG005T: Total Metals by ICP-AES								
Plastic Tube (EG005T)								
47A-2A,	47A-2B,	**30-May-2017**	**02-Jun-2017**	26-Nov-2017	✓	**02-Jun-2017**	26-Nov-2017	✓
47A-250A,	47A-250B,							
47B-2A,	47B-2B,							
47B-250A,	47B-250B,							
52-2B,	52-250A,							
52-250B,	58-2A,							
58-2B,	58-250A,							
58-250B,	60A-2A,							
60A-2B,	60A-250B,							
61-2A,	61-2B,							
61-250A,	61-250B,							
67 5-15-2A,	67 5-15-2B,							
67 5-15-250A,	67 5-15-250B,							
67 30-60-2A,	67 30-60-2B,							
67 30-60-250A,	67 30-60-250B,							
68-2A,	68-2B,							
68-250A,	68-250B,							
69-2A,	69-2B,							
69-250A,	69-250B							
Plastic Tube (EG005T)								
72-2B,	72-250A,	**30-May-2017**	**02-Jun-2017**	26-Nov-2017	✓	**05-Jun-2017**	26-Nov-2017	✓
72-250B,	73-2A,							
73-2B,	73-250A,							
73-250B,	84-2A,							
84-2B,	84-250B,							
85-2A,	85-2B,							
85-250A,	85-250B							
Plastic Tube (EG005T)								
52-2A,	60A-250A,	**30-May-2017**	**06-Jun-2017**	26-Nov-2017	✓	**07-Jun-2017**	26-Nov-2017	✓
72-2A,	84-250A							

Matrix: **WATER**

Evaluation: × = Holding time breach ; ✓ = Within holding time.

Method / Container / Client Sample ID(s)	Sample Date	Extraction / Preparation			Analysis		
		Date extracted	Due for extraction	Evaluation	Date analysed	Due for analysis	Evaluation

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Work Order	:	EM1706956
Client	:	UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	:	RWC Pb BioAcc

Matrix: **WATER**

Evaluation: × = Holding time breach ; ✓ = Within holding time.

Method / Container / Client Sample ID(s)		Sample Date	Extraction / Preparation			Analysis		
			Date extracted	Due for extraction	Evaluation	Date analysed	Due for analysis	Evaluation
EG005F: Dissolved Metals by ICP-AES								
Miscellanous Nitric Preserved - field filtered (EG005F)								
47A-G1,	47A-G2,	**30-May-2017**	**----**	----	----	**02-Jun-2017**	26-Nov-2017	✓
47B-G1,	47B-G2,							
52-G1,	52-G2,							
58-G1,	58-G2,							
60A-G1,	60A-G2,							
61-G1,	61-G2,							
67 5-15-G1,	67 5-15-G2,							
67 30-60-G1,	67 30-60-G2,							
68-G1,	68-G2,							
69-G1,	69-G2,							
72-G1,	72-G2,							
73-G1,	73-G2,							
84-G1,	84-G2,							
85-G1,	85-G2,							
QC1-1,	QC1-2,							
QC2-1,	QC2-2							

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Work Order	: EM1706956
Client	: UNISA - CENTRE FOR ENVIRONMENT RISK ASSESSMENT & REMEDIATION
Project	: RWC Pb BioAcc

Quality Control Parameter Frequency Compliance

The following report summarises the frequency of laboratory QC samples analysed within the analytical lot(s) in which the submitted sample(s) was(were) processed. Actual rate should be greater than or equal to the expected rate. A listing of breaches is provided in the Summary of Outliers.

Matrix: **SOIL**

Evaluation: × = Quality Control frequency not within specification ; ✓ = Quality Control frequency within specification.

Quality Control Sample Type		Count		Rate (%)			Quality Control Specification
Analytical Methods	Method	QC	Regular	Actual	Expected	Evaluation	
Laboratory Duplicates (DUP)							
Moisture Content	EA055-103	6	56	10.71	10.00	✓	NEPM 2013 B3 & ALS QC Standard
Total Metals by ICP-AES	EG005T	7	60	11.67	10.00	✓	NEPM 2013 B3 & ALS QC Standard
Laboratory Control Samples (LCS)							
Total Metals by ICP-AES	EG005T	4	60	6.67	5.00	✓	NEPM 2013 B3 & ALS QC Standard
Method Blanks (MB)							
Total Metals by ICP-AES	EG005T	4	60	6.67	5.00	✓	NEPM 2013 B3 & ALS QC Standard
Matrix Spikes (MS)							
Total Metals by ICP-AES	EG005T	4	60	6.67	5.00	✓	NEPM 2013 B3 & ALS QC Standard

Matrix: **WATER**

Evaluation: × = Quality Control frequency not within specification ; ✓ = Quality Control frequency within specification.

Quality Control Sample Type		Count		Rate (%)			Quality Control Specification
Analytical Methods	Method	QC	Regular	Actual	Expected	Evaluation	
Laboratory Duplicates (DUP)							
Dissolved Metals by ICP-AES	EG005F	0	32	0.00	10.00	×	NEPM 2013 B3 & ALS QC Standard
Laboratory Control Samples (LCS)							
Dissolved Metals by ICP-AES	EG005F	2	32	6.25	10.00	×	NEPM 2013 B3 & ALS QC Standard
Method Blanks (MB)							
Dissolved Metals by ICP-AES	EG005F	2	32	6.25	5.00	✓	NEPM 2013 B3 & ALS QC Standard
Matrix Spikes (MS)							
Dissolved Metals by ICP-AES	EG005F	0	32	0.00	5.00	×	NEPM 2013 B3 & ALS QC Standard

Brief Method Summaries

The analytical procedures used by the Environmental Division have been developed from established internationally recognized procedures such as those published by the US EPA, APHA, AS and NEPM. In house developed procedures are employed in the absence of documented standards or by client request. The following report provides brief descriptions of the analytical procedures employed for results reported in the Certificate of Analysis. Sources from which ALS methods have been developed are provided within the Method Descriptions.

Analytical Methods	Method	Matrix	Method Descriptions
Moisture Content	EA055-103	SOIL	In house: A gravimetric procedure based on weight loss over a 12 hour drying period at 103-105 degrees C. This method is compliant with NEPM (2013) Schedule B(3) Section 7.1 and Table 1 (14 day holding time).
Total Metals by ICP-AES	EG005T	SOIL	In house: Referenced to APHA 3120; USEPA SW 846 - 6010. Metals are determined following an appropriate acid digestion of the soil. The ICPAES technique ionises samples in a plasma, emitting a characteristic spectrum based on metals present. Intensities at selected wavelengths are compared against those of matrix matched standards. This method is compliant with NEPM (2013) Schedule B(3)
Dissolved Metals by ICP-AES	EG005F	WATER	In house: Referenced to APHA 3120; USEPA SW 846 - 6010. The ICPAES technique ionises the 0.45µm filtered samples, emitting a characteristic spectrum which is compared against matrix matched standards. This method is compliant with NEPM (2013) Schedule B(3)

Preparation Methods	Method	Matrix	Method Descriptions
Hot Block Digest for metals in soils sediments and sludges	EN69	SOIL	In house: Referenced to USEPA 200.2. Hot Block Acid Digestion 1.0g of sample is heated with Nitric and Hydrochloric acids, then cooled. Peroxide is added and samples heated and cooled again before being filtered and bulked to volume for analysis. Digest is appropriate for determination of selected metals in sludge, sediments, and soils. This method is compliant with NEPM (2013) Schedule B(3) (Method 202)

Annexure E

Characterisation of Exposure

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Note: This Annexure is only available on the digital version of this document

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E1 QUANTIFICATION OF INHALATION EXPOSURE

Intakes via inhalation has been assessed on the basis of the inhalation guidance available from the USEPA and recommended for use in the ASC NEPM and enHealth (enHealth 2012b; NEPC 1999 amended 2013e; 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 E1** provides details on the assumptions adopted in this assessment:

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

for $PM_{2.5}$ and PM_{10} where 100% of the inhaled particulates are assumed to reach the lungs

Table E1
Inhalation Exposure Assumptions

Parameter		Value adopted	Basis
Ca	Concentration of chemical substance in air (mg/m^3)	Existing exposures: as measured (refer to Section 4). Project emissions: Modelled in the Air Quality Assessment, where the maximum concentration from all receptors, maximum at all privately-owned residences and the maximum at each individual receptor has been evaluated. This assessment has considered the maximum 1-hour average concentration for the assessment of acute exposures and the annual average concentration for the assessment of chronic exposures.	Modelled ground level concentrations at each receptor.
ET	Exposure time (dependant on activity) (hours/day)	24 hours/day	Assume someone is exposed at the maximum location all day, every day of the year
EF	Exposure frequency (days/year)	365 days	
ED	Exposure duration (years)	35 years	Duration of residency as per enHealth (enHealth 2012a)
AT	Averaging time (hours)	ED x 365 days/year x 24 hours/day	As per enHealth (enHealth 2012b) guidance for threshold calculations (as is relevant in this assessment)

E2 MULTIPLE PATHWAY EXPOSURES

E2.1 Ingestion and Dermal Absorption

Chemical substances that are deposited on the ground have the potential to be ingested either directly through accidental consumption of dirt or indirectly through food grown or raised in the soil (fruit and vegetables, eggs, beef and milk) that is subsequently consumed.

The assessment of the potential ingestion of chemical substances has been undertaken using the approach presented by enHealth and the USEPA (enHealth 2012b; USEPA 1989). This approach is presented in the following equation, and parameters adopted in this assessment are presented in **Table E2**:

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

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 E2**:

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

For dermal contact with water, the equations are as follows (USEPA 2004):

$DA_{event} = K_p \times C_w \times CF \times t_{event}$ (mg/m^2 per event), relevant to inorganics

$$\text{Daily Chemical Intake}_{\text{Dermal}} = C_w \bullet \frac{SA \bullet DA_{event} \bullet EV \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

Table E2
Ingestion and Dermal Exposure Assumptions

Parameter		Value Adopted		Basis
		Young Children	Adults	
C_M	Concentration of chemical substance in media or relevance (soil, fruit and vegetables, eggs, beef or milk) (mg/kg)	Existing exposures: Based on measured concentrations in soil and tank water, and modelled levels in produce: Project emissions: Modelled based on deposition of particulates to soil (refer to Section E2.2)		Calculations undertaken on the basis of the maximum predicted impacts relevant to areas where multi-pathway exposures may occur
IR_M	Ingestion rate of media			
	Soil (mg/day)	100 mg/day	50 mg/day	Ingestion rate of outdoor soil and dust (tracked or deposited indoors) as per enHealth (enHealth 2012a)
	Water (L/day)	0.4 L/day	2 L/day	Water intakes from all sources (including food and bathing) (enHealth 2012a)
IR_M (Cont'd)	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 2013e)
	Eggs (kg/day)	0.006 kg/day	0.014 kg/day	Ingestion rate of eggs per day as per enHealth (enHealth 2012a), also consistent with P90 intakes from FSANZ (FSANZ 2017)
	Beef (kg/day)	0.085	0.16 kg/day	Ingestion rate for adults aged 19 years and older (enHealth 2012a), also consistent with P90 intakes from FSANZ (FSANZ 2017), Values for children from FSANZ (2017)
	Milk (kg/day)	1.097 kg/day	1.295 kg/day	Ingestion rate P90 intakes from FSANZ (FSANZ 2017)
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
	Water	100%	100%	Assume all water is from rainwater tanks on the property
	Fruit and vegetables	35%	35%	Rate assumed for rural area (higher than the default of 10% for urban areas)
	Eggs	200%	200%	Assume higher intake of home-produced eggs in rural areas (SAHC 1998)

Table E2 (Cont'd)
Ingestion and Dermal Exposure Assumptions

Parameter		Value Adopted		Basis
		Young Children	Adults	
FI	Beef	35%	35%	Rate assumed for rural area (higher than the default of 10% for urban areas)
	Milk	100%	100%	Assume all milk consumed each day is from the property
B	Bioavailability or absorption of chemical substance via ingestion	50% for lead 100% for all others	50% for lead 100% for all others	Conservative assumption
SA (soil)	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 2013e)
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 2013e)
SA (water)	Surface area of body exposed to water per day (cm^2/day)	6100	20000	Whole body gets wet each day during bathing (enHealth 2012a)
t_{event}	Exposure time per event, in water (hours/event)	1	0.58	Reasonable maximum time showering or wet each day (USEPA 2011)
EV	Events per day when wet	1	1	Assumed relevant to the use of rainwater
ABSd	Dermal absorption fraction (unitless)	Chemical specific		Refer to **Table 5.2**
Kp	Dermal permeability through skin (water) (cm/hr)	Chemical specific		Refer to **Table 5.2**

Table E2 (Cont'd)
Ingestion and Dermal Exposure Assumptions

Parameter		Value Adopted		Basis
		Young Children	Adults	
CF	Conversion factor			
	Soil	$1x10^{-6}$ to convert mg to kg		Conversion of units relevant to soil ingestion and dermal contact
	Water	0.001 to convert L to cm^3		Conversion for the assessment of dermal exposures to water
	Produce	1		No units conversion required for these calculations
BW	Body weight	70	15	As per enHealth (enHealth 2012a) and ASC NEPM (NEPC 1999 amended 2013e)
EF	Exposure frequency (days/year)	365	365	Assume residents exposed every day
ED	Exposure duration (years)	6 years	29	Duration of residency as per enHealth (enHealth 2012a) and split between young children and adults as per ASC NEPM (NEPC 1999 amended 2013e)
AT	Averaging time (days)	Threshold = ED x 365 days/year Non-threshold = 70 years x 365 days/year		As per enHealth (enHealth 2012b) guidance

E2.2 Calculation of Concentrations in Various Media

Potential Concentrations in Soil

The potential accumulation of persistent and bioaccumulative chemical substances in soil (relevant to Project emissions), which may be the result of deposition from a number of air emissions source, can be estimated using a soil accumulation model (OEHHA 2015; Stevens 1991).

The concentration in soil, which may be the result of deposition following emission of persistent chemical substances, can be calculated using the following equation, with assumptions adopted in this assessment presented in **Table E3**.

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

Table E3
Assumptions adopted to Estimate Soil Concentrations

Parameter		Value Adopted		Basis
		Surface Soil*	Agricultural Soil*	
DR	Particle deposition rate for TSP (mg/m^2/year)	Modelled for the facility. Adopted maximum deposition rate for discrete receptors		Relevant to areas where multi-pathway exposures may occur
k	Chemical-specific soil-loss constant (1/year) = ln(2)/$T^{0.5}$	Calculated	Calculated	
$T^{0.5}$	Chemical half-life in soil (years)	273973	273973	Default values for metals as per OEHHA (2015)
t	Accumulation time (years)	70 years	70 years	Default value (OEHHA 2015)
d	Soil mixing depth (m)	0.01 m	0.15 m	Default values (OEHHA 2015)
ρ	Soil bulk-density (g/m^3)	1600000	1600000	Default for fill material (CRC CARE 2011)
1000	Conversion from g to kg	Default conversion of units		

* Surface soil values adopted for the assessment of direct contact exposures. All other exposures including produce and meat/milk intakes utilise soil concentrations calculated for agricultural intakes (OEHHA 2015)

Homegrown Fruit and Vegetables

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

The potential concentration of persistent chemical substances that may be present within the plant following atmospheric deposition can be estimated using the following equation (Stevens 1991), with the parameters and assumptions adopted outlined in **Table E4**:

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

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 E4:**

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

Table E4
Assumptions Adopted to Estimate Concentration in Fruit and Vegetables

Parameter		Value adopted	Basis
DR	Particle deposition rate for TSP (mg/m^2/day)	Modelled in the Air Quality Assessment for each receptor	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 pollutants 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 E3**
RUF	Root uptake factor (unitless)	Chemical specific value adopted	Root uptake factors from RAIS (RAIS) (soil to wet weight of plant)

Eggs, Beef and Milk

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

The concentration (C_P) calculated in eggs, beef or milk is calculated using the following equation (OEHHA 2015), with parameters and assumptions adopted presented in **Table E5**:

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

Table E5
Assumptions Adopted to Estimate Concentration in Animal Produce

Parameter		**Value adopted**	**Basis**
FI	Fraction of grain/crop ingested by animals each day derived from the property (unitless)	100%	Assume all pasture/crops ingested by chickens and cows are grown on the property
IR_C	Ingestion rate of pasture/crops by each animal considered (kg/day)		
	Chickens	0.12 kg/day	Ingestion rate from OEHHA (2015)
	Beef cattle	9 kg/day	Ingestion rate from OEHHA (2015)
	Lactating cattle	22 kg/day	Ingestion rate for lactating cattle from OEHHA (2015)
C	Concentration of pollutant in crops consumed by animals (mg/kg)	Assume equal to that calculated in aboveground produce	Calculated as described above with assumptions in **Table E4**
IR_S	Ingestion rate of soil by animals each day (kg/day)		
	Chickens	0.0024 kg/day	Based on data from OEHHA 2015 (2% total produce intakes from soil)
	Beef cattle	0.45 kg/day	Based on data from OEHHA 2015 (5% total produce intakes from soil from pasture)
	Lactating cattle	1.1 kg/day	Based on data from OEHHA 2015 (5% total produce intakes from soil from pasture)
Cs	Concentration of pollutant in soil (mg/kg)	Calculated value for agricultural soil	Calculated as described above and assumptions in **Table E3**
B	Bioavailability of soil ingested (unitless)	100%	Conservative assumption
TF_P	Transfer factor for the produce of interest		
	Eggs	Chemical specific	Transfer factors adopted from OEHHA (2015), with the exception of chromium where the value was derived from an earlier OEHHA (OEHHA 2003) and the mean value from Leeman et al (Leeman, Van Den Berg & Houben 2007) adopted for silver, copper, manganese, zinc, cobalt and lithium
	Beef	Chemical specific	Transfer factors adopted from OEHHA (OEHHA 2003, 2015) and RAIS
	Milk	Chemical specific	Transfer factors adopted from OEHHA (2015), RAIS and Leeman et al (Leeman, Van Den Berg & Houben 2007)

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, NSW Health[21] 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 E6**:

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

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

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

Table E6

Assumptions adopted to estimate concentration in rainwater tank

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 for TSP ($mg/m^2/year$)	Modelled in the Air Quality Assessment for each receptor	Relevant to areas where multi-pathway exposures may occur
Area	Area of the roof (m^2)	200	Based on the average roof size for a 4 bedroom house in Australia (refer to Footnote 1)
VR	Volume of water collected from the roof each year (L)	calculated	Equation as above
R	Rainfall each year (mm)	663.2	Average rainfall at Mudgee Airport for all years of records (1994 – 2019). No first flush devise is considered, hence all rainfall is considered
Rc	Runoff coefficient	0.7	Assumes 30% loss in capture of water into the tank (Lizárraga-Mendiola et al. 2015)
1000	Conversion from m^3 to L Conversion from mm to m		
Kd	Soil-water partition coefficient (cm^3/g)	Chemical-specific	All values from RAIS (RAIS)
ρ	Soil bulk density (g/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			

Annexure F

Risk Calculations – Existing Exposures

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Inhalation - PM2.5 (Existing)

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

Parameters Relevant to Quantification of Community Exposures - Residents		
Exposure Time at Home (ET, hr/day)	24	Assume residents at home or on property 24 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Assume resident at the same property
Exposure Frequency (EF, days/yr)	365	Days at home, as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	35	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	306600	US EPA 2009

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk ($(mg/m^3)^{-1}$)	Chronic TC Air (mg/m^3)	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background) (mg/m^3)	Estimated Concentration in Air - Maximum all 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 (Ag)		2.0E-02		2.0E-02				--		--	
Lead (Pb)		2.0E-03		2.0E-03	1.0E-06	5.1E-07	1.0E-06	--		0.00051	0%
Arsenic (As)		1.0E-03		1.0E-03	2.3E-06	1.2E-06	2.3E-06	--		0.0023	1%
Cadmium (Cd)		5.0E-06		5.0E-06	4.6E-07	2.3E-07	4.6E-07	--		0.093	58%
Copper (Cu)		4.9E-01		4.9E-01	5.7E-06	2.9E-06	5.7E-06	--		0.000012	0%
Manganese (Mn)		1.5E-04		1.5E-04				--		--	
Zinc (Zn)		1.8E+00		1.8E+00	7.2E-06	3.6E-06	7.2E-06	--		0.0000041	0%
Cobalt (Co)		1.0E-04		1.0E-04				--		--	
Chromium (Cr)		1.0E-04		1.0E-04	1.5E-06	7.3E-07	1.5E-06	--		0.015	9%
Mercury (Hg)		2.0E-04		2.0E-04				--		--	
Lithium (Li)		7.0E-03		7.0E-03				--		--	
Nickel (Ni)		2.0E-05		2.0E-05	1.0E-06	5.1E-07	1.0E-06	--		0.051	31%
TOTAL										**0.16**	

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	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)
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 - All receptors

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Bioavailability (%)		NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	0.50	1.5E-07	3.6E-07	--		0.000063	0%
Lead (Pb)		6.0E-04		6.0E-04	16%	50.00	2.4E-06	5.8E-06	--		0.0097	22%
Arsenic (As)		2.0E-03		2.0E-03	100%	15.00	4.4E-06	1.1E-05	--		0.0054	12%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	0.13	3.8E-08	9.3E-08	--		0.00012	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	21.00	6.2E-06	1.5E-05	--		0.00011	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1113.00	3.3E-04	8.0E-04	--		0.0057	13%
Zinc (Zn)		5.0E-01		5.0E-01	100%	24.00	7.1E-06	1.7E-05	--		0.000034	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	9.50	2.8E-06	6.8E-06	--		0.00485	11%
Chromium (Cr)		1.0E-03		1.0E-03	100%	21.00	6.2E-06	1.5E-05	--		0.0150	34%
Mercury (Hg)		6.0E-04		6.0E-04	100%	0.03	8.6E-09	2.1E-08	--		0.000035	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	5.70	1.7E-06	4.1E-06	--		0.00204	5%
Nickel (Ni)		1.2E-02		1.2E-02	100%	12.00	3.6E-06	8.6E-06	--		0.000714	2%

Dermal Exposure to Chemicals via Contact with Soil

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

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

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04		0.50			--		--	
Lead (Pb)		3.0E-04		3.0E-04		50.00			--		--	
Arsenic (As)		2.0E-03		2.0E-03	0.005	15.00	1.4E-06	3.4E-06	--		0.00169	75%
Cadmium (Cd)		8.0E-04		8.0E-04		0.13			--		--	
Copper (Cu)		1.4E-01		1.4E-01		21.00			--		--	
Manganese (Mn)		1.4E-01		1.4E-01		1113.00			--		--	
Zinc (Zn)		5.0E-01		5.0E-01	0.001	24.00	4.5E-07	1.1E-06	--		0.0000022	0%
Cobalt (Co)		1.4E-03		1.4E-03	0.001	9.50	1.8E-07	4.3E-07	--		0.000305	14%
Chromium (Cr)		1.0E-03		1.0E-03		21.00			--		--	
Mercury (Hg)		4.2E-05		4.2E-05	0.001	0.03	5.4E-10	1.3E-09	--		0.00003	1%
Lithium (Li)		2.0E-03		2.0E-03		5.70			--		--	
Nickel (Ni)		1.2E-02		1.2E-02	0.005	12.00	1.1E-06	2.7E-06	--		0.000225	10%
								TOTAL			**0.0023**	

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 Chilrdren		
Ingestion Rate (IRs, mg/day)	100	Assumed daily soil ingestion rate for young children, enHealth (2012)
Fraction Ingested from Source (FI, unitless)	100%	Compound-specific as noted below
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)
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 - All receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	0.50	2.9E-07	3.3E-06	--		0.00058	0%
Lead (Pb)		1.4E-03		1.4E-03	16%	50.00	4.7E-06	5.4E-05	--		0.039	11%
Arsenic (As)		2.0E-03		2.0E-03	100%	15.00	8.6E-06	1.0E-04	--		0.050	14%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	0.13	7.4E-08	8.7E-07	--		0.0011	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	21.00	1.2E-05	1.4E-04	--		0.0010	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1113.00	6.4E-04	7.4E-03	--		0.053	15%
Zinc (Zn)		5.0E-01		5.0E-01	100%	24.00	1.4E-05	1.6E-04	--		0.00032	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	9.50	5.4E-06	6.3E-05	--		0.045	13%
Chromium (Cr)		1.0E-03		1.0E-03	100%	21.00	1.2E-05	1.4E-04	--		0.14	39%
Mercury (Hg)		6.0E-04		6.0E-04	100%	0.03	1.7E-08	1.9E-07	--		0.00032	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	5.70	3.3E-06	3.8E-05	--		0.019	5%
Nickel (Ni)		1.2E-02		1.2E-02	100%	12.00	6.9E-06	8.0E-05	--		0.0067	2%
								TOTAL			**0.36**	

Dermal Exposure to Chemicals via Contact with Soil

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

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

Maximum - All receptors

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04		0.50			--		--	
Lead (Pb)		7.0E-04		7.0E-04		50.00			--		--	
Arsenic (As)		2.0E-03		2.0E-03	0.005	15.00	5.8E-07	6.8E-06	--		0.0034	75%
Cadmium (Cd)		8.0E-04		8.0E-04		0.13			--		--	
Copper (Cu)		1.4E-01		1.4E-01		21.00			--		--	
Manganese (Mn)		1.4E-01		1.4E-01		1113.00			--		--	
Zinc (Zn)		5.0E-01		5.0E-01	0.001	24.00	1.9E-07	2.2E-06	--		0.0000043	0%
Cobalt (Co)		1.4E-03		1.4E-03	0.001	9.50	7.3E-08	8.6E-07	--		0.00061	14%
Chromium (Cr)		1.0E-03		1.0E-03		21.00			--		--	
Mercury (Hg)		4.2E-05		4.2E-05	0.001	0.03	2.2E-10	2.6E-09	--		0.000062	1%
Lithium (Li)		2.0E-03		2.0E-03		5.70			--		--	
Nickel (Ni)		1.2E-02		1.2E-02	0.005	12.00	4.6E-07	5.4E-06	--		0.00045	10%
								TOTAL			**0.0045**	

Exposure to Chemicals via Incidental Ingestion of Water

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate (Irw, L/day)	2	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10950	US EPA 1989 and CSMS 1996

Maximum - All receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%				--		--	
Lead (Pb)		6.0E-04		6.0E-04	50%	4.9E-03	3.0E-05	7.0E-05	--		0.12	46%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.8E-03	3.4E-05	8.0E-05	--		0.040	16%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	5.5E-04	6.7E-06	1.6E-05	--		0.020	8%
Copper (Cu)		1.4E-01		1.4E-01	100%				--		--	
Manganese (Mn)		1.4E-01		1.4E-01	100%	2.0E-03	2.4E-05	5.7E-05	--		0.00041	0%
Zinc (Zn)		5.0E-01		5.0E-01	100%	8.4E-01	1.0E-02	2.4E-02	--		0.048	19%
Cobalt (Co)		1.4E-03		1.4E-03	100%				--		--	
Chromium (Cr)		1.0E-03		1.0E-03	100%				--		--	
Mercury (Hg)		6.0E-04		6.0E-04	100%				--		--	
Lithium (Li)		2.0E-03		2.0E-03	100%				--		--	
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.3E-02	1.6E-04	3.8E-04	--		0.031	12%
TOTAL											**0.26**	

Dermal Exposure to Chemicals via Contact with Water

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Adults		
Surface Area (Saw, cm2)	20000	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	0.58	Reasonable maximum time spent showering or wet each day (ESEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10950	US EPA 1989 and CSMS 1996

Maximum - All receptors

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	6.00E-4					--		--	
Lead (Pb)		3.0E-04		3.0E-04	1.00E-4	4.90E-03	2.84E-10	3.5E-08	8.1E-08	--		2.7E-04	44%
Arsenic (As)		2.0E-03		2.0E-03	1.00E-3	2.80E-03	1.62E-09	2.0E-07	4.6E-07	--		2.3E-04	37%
Cadmium (Cd)		8.0E-04		8.0E-04	1.00E-3	5.50E-04	3.19E-10	3.9E-08	9.1E-08	--		1.1E-04	18%
Copper (Cu)		1.4E-01		1.4E-01	1.00E-3					--		--	
Manganese (Mn)		1.4E-01		1.4E-01	1.00E-3	2.00E-03	1.16E-09	1.4E-07	3.3E-07	--		2.4E-06	0%
Zinc (Zn)		5.0E-01		5.0E-01	6.00E-4	8.41E-01	2.93E-07	3.6E-05	8.4E-05	--		1.7E-04	27%
Cobalt (Co)		1.4E-03		1.4E-03	4.00E-4					--		--	
Chromium (Cr)		1.0E-03		1.0E-03	2.00E-3					--		--	
Mercury (Hg)		6.0E-04		6.0E-04	1.00E-3					--		--	
Lithium (Li)		2.0E-03		2.0E-03	1.00E-3					--		--	
Nickel (Ni)		1.2E-02		1.2E-02	2.00E-4	1.32E-02	1.53E-09	1.9E-07	4.4E-07	--		3.6E-05	6%
												6.2E-04	

Exposure to Chemicals via Incidental Ingestion of Water

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Children		
Ingestion Rate (Irw, L/day)	0.4	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	US EPA 1989 and CSMS 1996

Maximum - All receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%				--		--	
Lead (Pb)		1.4E-03		1.4E-03	50%	4.9E-03	5.6E-06	6.5E-05	--		0.047	26%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.8E-03	6.4E-06	7.5E-05	--		0.037	21%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	5.5E-04	1.3E-06	1.5E-05	--		0.018	10%
Copper (Cu)		1.4E-01		1.4E-01	100%				--		--	
Manganese (Mn)		1.4E-01		1.4E-01	100%	2.0E-03	4.6E-06	5.3E-05	--		0.00038	0%
Zinc (Zn)		5.0E-01		5.0E-01	100%	8.4E-01	1.9E-03	2.2E-02	--		0.045	25%
Cobalt (Co)		1.4E-03		1.4E-03	100%				--		--	
Chromium (Cr)		1.0E-03		1.0E-03	100%				--		--	
Mercury (Hg)		6.0E-04		6.0E-04	100%				--		--	
Lithium (Li)		2.0E-03		2.0E-03	100%				--		--	
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.3E-02	3.0E-05	3.5E-04	--		0.029	17%
								TOTAL			**0.18**	

Dermal Exposure to Chemicals via Contact with Water

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Children		
Surface Area (Saw, cm2)	6100	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	1	Reasonable maximum time spent showering or wet each day (ESEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	US EPA 1989 and CSMS 1996

Maximum - All receptors

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	6.00E-4					--		--	
Lead (Pb)		7.0E-04		7.0E-04	1.00E-4	4.90E-03	4.90E-10	1.7E-08	2.0E-07	--		2.8E-04	25%
Arsenic (As)		2.0E-03		2.0E-03	1.00E-3	2.80E-03	2.80E-09	9.8E-08	1.1E-06	--		5.7E-04	50%
Cadmium (Cd)		8.0E-04		8.0E-04	1.00E-3	5.50E-04	5.50E-10	1.9E-08	2.2E-07	--		2.8E-04	25%
Copper (Cu)		1.4E-01		1.4E-01	1.00E-3					--		--	
Manganese (Mn)		1.4E-01		1.4E-01	1.00E-3	2.00E-03	2.00E-09	7.0E-08	8.1E-07	--		5.8E-06	1%
Zinc (Zn)		5.0E-01		5.0E-01	6.00E-4	8.41E-01	5.05E-07	1.8E-05	2.1E-04	--		4.1E-04	36%
Cobalt (Co)		1.4E-03		1.4E-03	4.00E-4					--		--	
Chromium (Cr)		1.0E-03		1.0E-03	2.00E-3					--		--	
Mercury (Hg)		6.0E-04		6.0E-04	1.00E-3					--		--	
Lithium (Li)		2.0E-03		2.0E-03	1.00E-3					--		--	
Nickel (Ni)		1.2E-02		1.2E-02	2.00E-4	1.32E-02	2.64E-09	9.2E-08	1.1E-06	--		8.9E-05	8%

	1.1E-03

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Annexure G

Risk Calculations – Project Emissions

(Total No. of pages including blank pages = 106)

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Scenario 1: Site Establishment and Construction Stage

Predicted ground level concentrations and screening assessment - acute exposures

		PM2.5		Scenario 1			
		Air Concentration (ug/m3)		Air Concentration (1-hour average) (mg/m3)		Calculated HI	
COPC	**Acute air guideline (mg/m3)**	**Maximum anywhere**	**Maximum receptors**	**Maximum all receptors**	**Maximum private residences**	**Maximum all receptors**	**Maximum private residences**
Silver (Ag)	0.3	6.55E-03	8.12E-04	6.5E-06	8.1E-07	2.2E-05	2.7E-06
Lead (Pb)	0.15	2.04E-01	2.62E-02	2.0E-04	2.6E-05	1.4E-03	1.7E-04
Arsenic (As)	0.003	1.88E-02	2.76E-03	1.9E-05	2.8E-06	6.3E-03	9.2E-04
Cadmium (Cd)	0.0054	1.23E-03	1.62E-04	1.2E-06	1.6E-07	2.3E-04	3.0E-05
Copper (Cu)	0.1	1.63E-03	3.77E-04	1.6E-06	3.8E-07	1.6E-05	3.8E-06
Manganese (Mn)	0.0091	5.83E-01	1.18E-01	5.8E-04	1.2E-04	6.4E-02	1.3E-02
Zinc (Zn)	6	2.42E-01	7.65E-02	2.4E-04	7.6E-05	4.0E-05	1.3E-05
Cobalt (Co)	0.00069	2.90E-04	1.22E-04	2.9E-07	1.2E-07	4.2E-04	1.8E-04
Chromium (Cr)	0.0013	6.56E-03	8.40E-04	6.6E-06	8.4E-07	5.0E-03	6.5E-04
Mercury (Hg)	0.0006	3.06E-04	5.01E-05	3.1E-07	5.0E-08	5.1E-04	8.3E-05
Lithium (Li)	3.3	3.84E-03	6.01E-04	3.8E-06	6.0E-07	1.2E-06	1.8E-07
Nickel (Ni)	0.0011	5.95E-04	8.82E-05	5.9E-07	8.8E-08	5.4E-04	8.0E-05
						7.9E-02	1.5E-02

Chronic Exposures:

Inhalation - $PM_{2.5}$

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

Parameters Relevant to Quantification of Community Exposures - Residents		
Exposure Time at Home (ET, hr/day)	24	Assume residents at home or on property 24 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Assume resident at the same property
Exposure Frequency (EF, days/yr)	365	Days at home, as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	35	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	306600	US EPA 2009

	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
Key Chemical	Inhalation Unit Risk ($(mg/m^3)^{-1}$)	Chronic TC Air (mg/m^3)	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background) (mg/m^3)	Estimated Concentration in Air - Maximum all 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 (Ag)		2.0E-02		2.0E-02	2.3E-07	1.1E-07	2.3E-07	--		0.000011	0%
Lead (Pb)		2.0E-03		2.0E-03	7.3E-06	3.6E-06	7.3E-06	--		0.0036	2%
Arsenic (As)		1.0E-03		1.0E-03	7.2E-07	3.6E-07	7.2E-07	--		0.00072	0%
Cadmium (Cd)		5.0E-06		5.0E-06	4.4E-08	2.2E-08	4.4E-08	--		0.0089	5%
Copper (Cu)		4.9E-01		4.9E-01	6.6E-08	3.3E-08	6.6E-08	--		0.00000014	0%
Manganese (Mn)		1.5E-04		1.5E-04	2.2E-05	1.1E-05	2.2E-05	--		0.15	90%
Zinc (Zn)		1.8E+00		1.8E+00	8.5E-06	4.2E-06	8.5E-06	--		0.0000049	0%
Cobalt (Co)		1.0E-04		1.0E-04	1.3E-08	6.4E-09	1.3E-08	--		0.00013	0%
Chromium (Cr)		1.0E-04		1.0E-04	2.3E-07	1.2E-07	2.3E-07	--		0.0023	1%
Mercury (Hg)		2.0E-04		2.0E-04	1.1E-08	5.4E-09	1.1E-08	--		0.000054	0%
Lithium (Li)		7.0E-03		7.0E-03	1.4E-07	7.1E-08	1.4E-07	--		0.000020	0%
Nickel (Ni)		2.0E-05		2.0E-05	1.9E-08	9.6E-09	1.9E-08	--		0.00096	1%
TOTAL										**0.16**	

	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
Key Chemical	Inhalation Unit Risk	Chronic TC Air	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background)	Estimated Concentration in Air - Maximum private residences (Ca)	Inhalation Exposure Concentration - NonThreshold	Inhalation Exposure Concentration - Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
Silver (Ag)		2.0E-02		2.0E-02	7.3E-09	3.6E-09	7.3E-09	--		0.00000036	0%
Lead (Pb)		2.0E-03		2.0E-03	2.5E-07	1.3E-07	2.5E-07	--		0.00013	1%
Arsenic (As)		1.0E-03		1.0E-03	3.6E-08	1.8E-08	3.6E-08	--		0.000036	0%
Cadmium (Cd)		5.0E-06		5.0E-06	1.6E-09	8.0E-10	1.6E-09	--		0.00032	4%
Copper (Cu)		4.9E-01		4.9E-01	4.6E-09	2.3E-09	4.6E-09	--		0.0000000094	0%
Manganese (Mn)		1.5E-04		1.5E-04	1.2E-06	5.9E-07	1.2E-06	--		0.0079	92%
Zinc (Zn)		1.8E+00		1.8E+00	3.2E-07	1.6E-07	3.2E-07	--		0.00000018	0%
Cobalt (Co)		1.0E-04		1.0E-04	1.1E-09	5.5E-10	1.1E-09	--		0.000011	0%
Chromium (Cr)		1.0E-04		1.0E-04	8.0E-09	4.0E-09	8.0E-09	--		0.000080	1%
Mercury (Hg)		2.0E-04		2.0E-04	1.2E-09	6.1E-10	1.2E-09	--		0.0000061	0%
Lithium (Li)		7.0E-03		7.0E-03	1.2E-08	6.1E-09	1.2E-08	--		0.0000017	0%
Nickel (Ni)		2.0E-05		2.0E-05	1.6E-09	8.2E-10	1.6E-09	--		0.000082	1%
TOTAL										**0.0086**	

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 for TSP (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
Accumulation time (t)	years	70	70	As per OEHHA (2015) guidance

Chemical-specific Inputs and calculations - maximum private residences

Chemical	**Half-life in soil**	**Loss constant (K)**	**TSP Deposition Rate (DR)**	**Surface Concentration in Soil**	**Agricultural Concentration in Soil**
	years	**per year**	**mg/m^2/year**	**mg/kg**	**mg/kg**
Silver (Ag)	273973	2.5E-06	0.0215	9.4E-02	6.3E-03
Lead (Pb)	273973	2.5E-06	0.7667	3.4E+00	2.2E-01
Arsenic (As)	273973	2.5E-06	0.1191	5.2E-01	3.5E-02
Cadmium (Cd)	273973	2.5E-06	0.0052	2.3E-02	1.5E-03
Copper (Cu)	273973	2.5E-06	0.0152	6.6E-02	4.4E-03
Manganese (Mn)	273973	2.5E-06	3.8710	1.7E+01	1.1E+00
Zinc (Zn)	273973	2.5E-06	1.0394	4.5E+00	3.0E-01
Cobalt (Co)	273973	2.5E-06	0.0045	2.0E-02	1.3E-03
Chromium (Cr)	273973	2.5E-06	0.0241	1.1E-01	7.0E-03
Mercury (Hg)	273973	2.5E-06	0.0039	1.7E-02	1.1E-03
Lithium (Li)	273973	2.5E-06	0.0392	1.7E-01	1.1E-02
Nickel (Ni)	273973	2.5E-06	0.0058	2.6E-02	1.7E-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	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2012 and NEPM 2013
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	0.09	2.8E-08	6.7E-08	--		0.000012	1%
Lead (Pb)		6.0E-04		6.0E-04	16%	3.35	1.6E-07	3.9E-07	--		0.00065	58%
Arsenic (As)		2.0E-03		2.0E-03	100%	0.52	1.5E-07	3.7E-07	--		0.00019	16%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	0.02	6.7E-09	1.6E-08	--		0.000020	2%
Copper (Cu)		1.4E-01		1.4E-01	100%	0.07	2.0E-08	4.7E-08	--		0.00000034	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	16.934	5.0E-06	1.2E-05	--		0.000086	8%
Zinc (Zn)		5.0E-01		5.0E-01	100%	4.55	1.3E-06	3.2E-06	--		0.0000065	1%
Cobalt (Co)		1.4E-03		1.4E-03	100%	0.02	5.9E-09	1.4E-08	--		0.000010	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	0.11	3.1E-08	7.5E-08	--		0.000075	7%
Mercury (Hg)		6.0E-04		6.0E-04	100%	0.017	5.0E-09	1.2E-08	--		0.000020	2%
Lithium (Li)		2.0E-03		2.0E-03	100%	0.17	5.1E-08	1.2E-07	--		0.000061	5%
Nickel (Ni)		1.2E-02		1.2E-02	100%	0.026	7.6E-09	1.8E-08	--		0.0000015	0%
								TOTAL			**0.0011**	

Dermal Exposure to Chemicals via Contact with Soil

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

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/yr)	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 - Private residences

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04		9.4E-02			--		--	
Lead (Pb)		3.0E-04		3.0E-04		3.4E+00			--		--	
Arsenic (As)		2.0E-03		2.0E-03	0.005	5.2E-01	4.9E-08	1.2E-07	--		0.000059	75%
Cadmium (Cd)		8.0E-04		8.0E-04		2.3E-02			--		--	
Copper (Cu)		1.4E-01		1.4E-01		6.6E-02			--		--	
Manganese (Mn)		1.4E-01		1.4E-01		1.7E+01			--		--	
Zinc (Zn)		5.0E-01		5.0E-01	0.001	4.5E+00	8.5E-08	2.0E-07	--		0.00000041	1%
Cobalt (Co)		1.4E-03		1.4E-03	0.001	2.0E-02	3.7E-10	8.9E-10	--		0.00000064	1%
Chromium (Cr)		1.0E-03		1.0E-03		1.1E-01			--		--	
Mercury (Hg)		4.2E-05		4.2E-05	0.001	1.7E-02	3.2E-10	7.6E-10	--		0.000018	23%
Lithium (Li)		2.0E-03		2.0E-03		1.7E-01			--		--	
Nickel (Ni)		1.2E-02		1.2E-02	0.005	2.6E-02	2.4E-09	5.8E-09	--		0.00000048	1%
TOTAL											**0.000078**	

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 Chilrdren		
Ingestion Rate (IRs, mg/day)	100	Assumed daily soil ingestion rate for young children, enHealth (2012)
Fraction Ingested from Source (FI, unitless)	100%	Compound-specific as noted below
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)
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	0.09	5.4E-08	6.3E-07	--		0.00011	2%
Lead (Pb)		1.4E-03		1.4E-03	16%	3.35	3.1E-07	3.7E-06	--		0.0026	37%
Arsenic (As)		2.0E-03		2.0E-03	100%	0.52	3.0E-07	3.5E-06	--		0.0017	25%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	0.02	1.3E-08	1.5E-07	--		0.00019	3%
Copper (Cu)		1.4E-01		1.4E-01	100%	0.07	3.8E-08	4.4E-07	--		0.0000032	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	16.934	9.7E-06	1.1E-04	--		0.00081	11%
Zinc (Zn)		5.0E-01		5.0E-01	100%	4.55	2.6E-06	3.0E-05	--		0.000061	1%
Cobalt (Co)		1.4E-03		1.4E-03	100%	0.02	1.1E-08	1.3E-07	--		0.000094	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	0.11	6.0E-08	7.0E-07	--		0.00070	10%
Mercury (Hg)		6.0E-04		6.0E-04	100%	0.017	9.7E-09	1.1E-07	--		0.00019	3%
Lithium (Li)		2.0E-03		2.0E-03	100%	0.17	9.8E-08	1.1E-06	--		0.00057	8%
Nickel (Ni)		1.2E-02		1.2E-02	100%	0.026	1.5E-08	1.7E-07	--		0.000014	0%
								TOTAL			**0.0071**	

Dermal Exposure to Chemicals via Contact with Soil

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

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

Maximum - Private residences

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04		9.4E-02			--		--	
Lead (Pb)		7.0E-04		7.0E-04		3.4E+00			--		--	
Arsenic (As)		2.0E-03		2.0E-03	0.005	5.2E-01	2.0E-08	2.3E-07	--		0.00012	75%
Cadmium (Cd)		8.0E-04		8.0E-04		2.3E-02			--		--	
Copper (Cu)		1.4E-01		1.4E-01		6.6E-02			--		--	
Manganese (Mn)		1.4E-01		1.4E-01		1.7E+01			--		--	
Zinc (Zn)		5.0E-01		5.0E-01	0.001	4.5E+00	3.5E-08	4.1E-07	--		0.00000082	1%
Cobalt (Co)		1.4E-03		1.4E-03	0.001	2.0E-02	1.5E-10	1.8E-09	--		0.0000013	1%
Chromium (Cr)		1.0E-03		1.0E-03		1.1E-01			--		--	
Mercury (Hg)		4.2E-05		4.2E-05	0.001	1.7E-02	1.3E-10	1.5E-09	--		0.000036	23%
Lithium (Li)		2.0E-03		2.0E-03		1.7E-01			--		--	
Nickel (Ni)		1.2E-02		1.2E-02	0.005	2.6E-02	9.9E-10	1.2E-08	--		0.0000010	1%
								TOTAL			**0.00016**	

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 1 year
DR =	Deposition rate from model for TSP (mg/m^2/year)
Area =	Area of roof (m^2)
VR =	Volume of water collected from roof over year (L) = (R x Area x Rc x 1000)/1000
R =	Rainfall each year (mm)
ρ =	Soil bulk-density (g/cm^3)
Rc =	Runoff coefficient (unitless)
Kd =	Soil-water partition coefficient (cm^3/g)
1000 =	Conversion from mm to m; and conversion from m^3 to L

General Parameters			
Average rainfaill	mm	663.2	mean for all years (1994 - 2019) for Mudgee airport
Roof area	m^2	200	4 bedroom australian home
Runoff coefficient	-	0.7	assumes 30% loss in capture into tank
Volume of rainwater	L	92848	
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 private 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	Drininking water guideline	Proportion of DWG: Particulate	Proportion of DWG: Dissolved
	mg/m^2/year	mg	(cm3/g)	mg/L	mg/L	mg/L		
Silver (Ag)	0.0215	4.3	8.3	4.6E-05	1.1E-05	0.1	0%	0.011%
Lead (Pb)	0.7667	153.3	900	1.7E-03	3.7E-06	0.01	17%	0.037%
Arsenic (As)	0.1191	23.8	29	2.6E-04	1.8E-05	0.01	3%	0.18%
Cadmium (Cd)	0.0052	1.0	75	1.1E-05	3.0E-07	0.002	1%	0.015%
Copper (Cu)	0.0152	3.0	35	3.3E-05	1.9E-06	2	0%	0.000093%
Manganese (Mn)	3.8710	774.2	65	8.3E-03	2.6E-04	0.5	2%	0.051313%
Zinc (Zn)	1.0394	207.9	62	2.2E-03	7.2E-05	6	0%	0.0012%
Cobalt (Co)	0.0045	0.9	45	9.8E-06	4.3E-07	0.006	0%	0.0072%
Chromium (Cr)	0.0241	4.8	19	5.2E-05	5.5E-06	0.05	0%	0.011%
Mercury (Hg)	0.0039	0.8	52	8.4E-06	3.2E-07	0.001	1%	0.032%
Lithium (Li)	0.0392	7.8	300	8.4E-05	5.6E-07	0.04	0%	0.00141%
Nickel (Ni)	0.0058	1.2	65	1.3E-05	3.9E-07	0.02	0%	0.00194%

Apprach assumes all dust deposited on the roof ends up in the water tank - no first flush diversion

0.02 RSL for tap water from USEPA (2018) as no ADWG available

Exposure to Chemicals via Incidental Ingestion of Water

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate (Irw, L/day)	2	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	4.6E-05	5.5E-07	1.3E-06	--		0.00023	0%
Lead (Pb)	0.0E+00	6.0E-04	0%	6.0E-04	50%	1.7E-03	9.8E-06	2.4E-05	--		0.039	31%
Arsenic (As)	0.0E+00	2.0E-03	0%	2.0E-03	100%	2.6E-04	3.0E-06	7.3E-06	--		0.0037	3%
Cadmium (Cd)	0.0E+00	8.0E-04	0%	8.0E-04	100%	1.1E-05	1.3E-07	3.2E-07	--		0.00040	0%
Copper (Cu)	0.0E+00	1.4E-01	0%	1.4E-01	100%	3.3E-05	3.9E-07	9.3E-07	--		0.0000067	0%
Manganese (Mn)	0.0E+00	1.4E-01	0%	1.4E-01	100%	8.3E-03	9.9E-05	2.4E-04	--		0.0017	1%
Zinc (Zn)	0.0E+00	5.0E-01	0%	5.0E-01	100%	2.2E-03	2.7E-05	6.4E-05	--		0.00013	0%
Cobalt (Co)	0.0E+00	1.4E-03	0%	1.4E-03	100%	9.8E-06	1.2E-07	2.8E-07	--		0.00020	0%
Chromium (Cr)	0.0E+00	1.0E-03	0%	1.0E-03	100%	5.2E-05	6.1E-07	1.5E-06	--		0.0015	1%
Mercury (Hg)	0.0E+00	6.0E-04	0%	6.0E-04	100%	8.4E-06	9.9E-08	2.4E-07	--		0.00040	0%
Lithium (Li)	0.0E+00	2.0E-03	0%	2.0E-03	100%	8.4E-05	1.0E-06	2.4E-06	--		0.0012	1%
Nickel (Ni)	0.0E+00	1.2E-02	0%	1.2E-02	100%	1.3E-05	1.5E-07	3.6E-07	--		0.000030	0%
								TOTAL	**0.00E+00**		**0.049**	

Dermal Exposure to Chemicals via Contact with Water

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Adults		
Surface Area (Saw, cm2)	20000	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	0.58	Reasonable maximum time spent showering or wet each day (USEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	4.62E-05	1.61E-11	1.9E-09	4.6E-09	--		2.0E-05	5%
Lead (Pb)		3.0E-04		3.0E-04	1.00E-4	1.65E-03	9.58E-11	1.1E-08	2.7E-08	--		9.1E-05	24%
Arsenic (As)		2.0E-03		2.0E-03	1.00E-3	2.57E-04	1.49E-10	1.8E-08	4.3E-08	--		2.1E-05	6%
Cadmium (Cd)		8.0E-04		8.0E-04	1.00E-3	1.11E-05	6.44E-12	7.6E-10	1.8E-09	--		2.3E-06	1%
Copper (Cu)		1.4E-01		1.4E-01	1.00E-3	3.27E-05	1.90E-11	2.2E-09	5.4E-09	--		3.9E-08	0%
Manganese (Mn)		1.4E-01		1.4E-01	1.00E-3	8.34E-03	4.84E-09	5.7E-07	1.4E-06	--		9.9E-06	3%
Zinc (Zn)		5.0E-01		5.0E-01	6.00E-4	2.24E-03	7.79E-10	9.2E-08	2.2E-07	--		4.5E-07	0%
Cobalt (Co)		1.4E-03		1.4E-03	4.00E-4	9.76E-06	2.27E-12	2.7E-10	6.5E-10	--		4.6E-07	0%
Chromium (Cr)		1.0E-03		1.0E-03	2.00E-3	5.19E-05	6.02E-11	7.1E-09	1.7E-08	--		1.7E-05	5%
Mercury (Hg)		4.2E-05		4.2E-05	1.00E-3	8.35E-06	4.84E-12	5.7E-10	1.4E-09	--		3.3E-05	9%
Lithium (Li)		2.0E-03		2.0E-03	1.00E-3	8.44E-05	4.90E-11	5.8E-09	1.4E-08	--		7.0E-06	2%
Nickel (Ni)		1.2E-02		1.2E-02	2.00E-4	1.26E-05	1.46E-12	1.7E-10	4.2E-10	--		3.5E-08	0%
												1.4E-04	

Exposure to Chemicals via Incidental Ingestion of Water

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Children		
Ingestion Rate (Irw, L/day)	0.4	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	4.6E-05	1.1E-07	1.2E-06	--		0.00022	0%
Lead (Pb)	0.0E+00	1.4E-03	0%	1.4E-03	50%	1.7E-03	1.9E-06	2.2E-05	--		0.016	26%
Arsenic (As)	0.0E+00	2.0E-03	0%	2.0E-03	100%	2.6E-04	5.9E-07	6.8E-06	--		0.0034	6%
Cadmium (Cd)	0.0E+00	8.0E-04	0%	8.0E-04	100%	1.1E-05	2.5E-08	3.0E-07	--		0.00037	1%
Copper (Cu)	0.0E+00	1.4E-01	0%	1.4E-01	100%	3.3E-05	7.5E-08	8.7E-07	--		0.0000062	0%
Manganese (Mn)	0.0E+00	1.4E-01	0%	1.4E-01	100%	8.3E-03	1.9E-05	2.2E-04	--		0.0016	3%
Zinc (Zn)	0.0E+00	5.0E-01	0%	5.0E-01	100%	2.2E-03	5.1E-06	6.0E-05	--		0.00012	0%
Cobalt (Co)	0.0E+00	1.4E-03	0%	1.4E-03	100%	9.8E-06	2.2E-08	2.6E-07	--		0.00019	0%
Chromium (Cr)	0.0E+00	1.0E-03	0%	1.0E-03	100%	5.2E-05	1.2E-07	1.4E-06	--		0.0014	2%
Mercury (Hg)	0.0E+00	6.0E-04	0%	6.0E-04	100%	8.4E-06	1.9E-08	2.2E-07	--		0.00037	1%
Lithium (Li)	0.0E+00	2.0E-03	0%	2.0E-03	100%	8.4E-05	1.9E-07	2.3E-06	--		0.0011	2%
Nickel (Ni)	0.0E+00	1.2E-02	0%	1.2E-02	100%	1.3E-05	2.9E-08	3.4E-07	--		0.000028	0%
								TOTAL	**0.00E+00**		**0.025**	

Dermal Exposure to Chemicals via Contact with Water

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Children		
Surface Area (Saw, cm2)	6100	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	1	Reasonable maximum time spent showering or wet each day (USEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	4.62E-05	2.77E-11	9.7E-10	1.1E-08	--		4.9E-05	9%
Lead (Pb)		7.0E-04		7.0E-04	1.00E-4	1.65E-03	1.65E-10	5.8E-09	6.7E-08	--		9.6E-05	17%
Arsenic (As)		2.0E-03		2.0E-03	1.00E-3	2.57E-04	2.57E-10	8.9E-09	1.0E-07	--		5.2E-05	9%
Cadmium (Cd)		8.0E-04		8.0E-04	1.00E-3	1.11E-05	1.11E-11	3.9E-10	4.5E-09	--		5.6E-06	1%
Copper (Cu)		1.4E-01		1.4E-01	1.00E-3	3.27E-05	3.27E-11	1.1E-09	1.3E-08	--		9.5E-08	0%
Manganese (Mn)		1.4E-01		1.4E-01	1.00E-3	8.34E-03	8.34E-09	2.9E-07	3.4E-06	--		2.4E-05	4%
Zinc (Zn)		5.0E-01		5.0E-01	6.00E-4	2.24E-03	1.34E-09	4.7E-08	5.5E-07	--		1.1E-06	0%
Cobalt (Co)		1.4E-03		1.4E-03	4.00E-4	9.76E-06	3.91E-12	1.4E-10	1.6E-09	--		1.1E-06	0%
Chromium (Cr)		1.0E-03		1.0E-03	2.00E-3	5.19E-05	1.04E-10	3.6E-09	4.2E-08	--		4.2E-05	7%
Mercury (Hg)		4.2E-05		4.2E-05	1.00E-3	8.35E-06	8.35E-12	2.9E-10	3.4E-09	--		8.1E-05	14%
Lithium (Li)		2.0E-03		2.0E-03	1.00E-3	8.44E-05	8.44E-11	2.9E-09	3.4E-08	--		1.7E-05	3%
Nickel (Ni)		1.2E-02		1.2E-02	2.00E-4	1.26E-05	2.52E-12	8.8E-11	1.0E-09	--		8.5E-08	0%

	2.3E-04

Calculation of Concentrations in Plants

ref: Stevens B. (1991)

Uptake Due to Deposition in Aboveground Crops

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

where:
DR= Particle deposition rate for TSP (mg/m^2/day)
F= Fraction for the surface area of plant (unitless)
k= Chemical-specific soil-loss constant (1/years) = ln(2)/$T_{0.5}$
$T_{0.5}$= Chemical half-life as particulate on plant (days)
t= Deposition time (days)
Y= Crop yield (kg/m^2)

Uptake via Roots from Soil

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

where:
Cs = Concentration of persistent chemical in soil assuming 15cm mixing depth within gardens, calculated using Soil Equation for each chemical assessed (mg/kg)
RUF = Root uptake factor which differs for each Chemical (unitless)

General Parameters	Units	Value
Crop		Edible crops
Crop Yield (Y)	kg/m^2	2
Deposition Time (t)	days	70
Plant Interception fraction (F)	unitless	0.051

Chemical-specific Inputs and calculations - Maximum private residences

Chemical	Half-life in plant ($T_{0.5}$)	Loss constant (k)	Deposition Rate TSP (DR)	Aboveground Produce Concentration via Deposition	Root Uptake Factor (RUF)	Soil Concentration (Cs)	Below Ground Produce Concentration
	days	per day	mg/m^2/day	mg/kg ww	unitless	mg/kg	mg/kg ww
Silver (Ag)	14	0.05	0.0000588	2.9E-05	0.1	6.3E-03	6.3E-04
Lead (Pb)	14	0.05	0.0021006	1.0E-03	0.011	2.2E-01	2.5E-03
Cadmium (Cd)	14	0.05	0.0000141	7.1E-06	0.125	1.5E-03	1.9E-04
Copper (Cu)	14	0.05	0.0000416	2.1E-05	0.1	4.4E-03	4.4E-04
Manganese (Mn)	14	0.05	0.0106055	5.3E-03	0.0625	1.1E+00	7.1E-02
Zinc (Zn)	14	0.05	0.0028476	1.4E-03	0.0113	3.0E-01	3.4E-03
Cobalt (Co)	14	0.05	0.0000124	6.2E-06	0.005	1.3E-03	6.6E-06
Chromium (Cr)	14	0.05	0.0000660	3.3E-05	0.00188	7.0E-03	1.3E-05
Mercury (Hg)	14	0.05	0.0000106	5.3E-06	0.225	1.1E-03	2.5E-04
Lithium (Li)	14	0.05	0.0001074	5.4E-05	0.00625	1.1E-02	7.1E-05
Nickel (Ni)	14	0.05	0.0000160	8.0E-06	0.015	1.7E-03	2.6E-05

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

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 (%)	35%	Assumed for rural areas (higher than typical default)
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)				NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	2.9E-05	6.3E-04	1.6E-07	3.8E-07	--		6.7E-05	2%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.0E-03	2.5E-03	5.9E-07	1.4E-06	--		2.4E-03	71%
Arsenic (As)		2.0E-03		2.0E-03	100%	1.6E-04	3.5E-04	4.6E-08	1.1E-07	--		5.6E-05	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	7.1E-06	1.9E-04	4.6E-08	1.1E-07	--		1.4E-04	4%
Copper (Cu)		1.4E-01		1.4E-01	100%	2.1E-05	4.4E-04	1.1E-07	2.7E-07	--		1.9E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	5.3E-03	7.1E-02	1.9E-05	4.6E-05	--		3.3E-04	10%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.4E-03	3.4E-03	1.6E-06	3.9E-06	--		7.8E-06	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	6.2E-06	6.6E-06	5.2E-09	1.3E-08	--		9.0E-06	0%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.3E-05	1.3E-05	2.3E-08	5.5E-08	--		5.5E-05	2%
Mercury (Hg)		6.0E-04		6.0E-04	100%	5.3E-06	2.5E-04	6.0E-08	1.5E-07	--		2.4E-04	7%
Lithium (Li)		2.0E-03		2.0E-03	100%	5.4E-05	7.1E-05	4.8E-08	1.2E-07	--		5.8E-05	2%
Nickel (Ni)		1.2E-02		1.2E-02	100%	8.0E-06	2.6E-05	1.1E-08	2.5E-08	--		2.1E-06	0%
									TOTAL			**0.0033**	

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 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 (%)	35%	Assumed for rural areas (higher than typical default)
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)				NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	2.9E-05	6.3E-04	7.0E-08	8.2E-07	--		1.4E-04	3%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.0E-03	2.5E-03	3.6E-07	4.2E-06	--		3.0E-03	57%
Arsenic (As)		2.0E-03		2.0E-03	100%	1.6E-04	3.5E-04	2.0E-08	2.4E-07	--		1.2E-04	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	7.1E-06	1.9E-04	2.0E-08	2.4E-07	--		2.9E-04	6%
Copper (Cu)		1.4E-01		1.4E-01	100%	2.1E-05	4.4E-04	4.9E-08	5.8E-07	--		4.1E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	5.3E-03	7.1E-02	8.8E-06	1.0E-04	--		7.3E-04	14%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.4E-03	3.4E-03	9.8E-07	1.1E-05	--		2.3E-05	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	6.2E-06	6.6E-06	3.5E-09	4.1E-08	--		2.9E-05	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.3E-05	1.3E-05	1.7E-08	1.9E-07	--		1.9E-04	4%
Mercury (Hg)		6.0E-04		6.0E-04	100%	5.3E-06	2.5E-04	2.5E-08	2.9E-07	--		4.9E-04	9%
Lithium (Li)		2.0E-03		2.0E-03	100%	5.4E-05	7.1E-05	3.2E-08	3.7E-07	--		1.8E-04	4%
Nickel (Ni)		1.2E-02		1.2E-02	100%	8.0E-06	2.6E-05	6.1E-09	7.1E-08	--		5.9E-06	0%
									TOTAL			**0.0052**	

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 100% of crops consumed by chickens is grown in the same soil
IRc (ingestion rate of crops)	kg/day	0.12	Assumed ingestion rate from OEHHA 2015 (assume concentration the same as predicted for aboveground crops)
IRs (ingestion rate of soil)	kg/day	0.0024	Based on data from OEHHA 2015 (2% total produce intakes from soil)
B (bioavailability)	%	100%	Assumed to be 100% except for lead

Chemical-specific Inputs and calculations - Maximum private residences

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 (Ag)	2.9E-05	6.3E-03	3.8E-02	7.0E-07	
Lead (Pb)	1.0E-03	2.2E-01	4.0E-02	1.2E-05	
Arsenic (As)	1.6E-04	3.5E-02	7.0E-02	7.2E-06	
Cadmium (Cd)	7.1E-06	1.5E-03	1.0E-02	4.5E-08	
Copper (Cu)	2.1E-05	4.4E-03	3.8E-02	5.0E-07	
Manganese (Mn)	5.3E-03	1.1E+00	3.8E-02	1.3E-04	
Zinc (Zn)	1.4E-03	3.0E-01	3.8E-02	3.4E-05	
Cobalt (Co)	6.2E-06	1.3E-03	3.8E-02	1.5E-07	
Chromium (Cr)	3.3E-05	7.0E-03	9.2E-03	1.9E-07	OEHHA (2003)
Mercury (Hg)	5.3E-06	1.1E-03	8.0E-01	2.7E-06	
Lithium (Li)	5.4E-05	1.1E-02	3.8E-02	1.3E-06	
Nickel (Ni)	8.0E-06	1.7E-03	2.0E-02	1.0E-07	

Transfer factors from OEHHA 2015 unless otherwise noted
Mean transfer factor for heavy metals used in absense of specific data (Leeman et al 2007)

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 (%)	200%	Assumed for rural areas where a higher rate of egg ingestion expected
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	7.0E-07	1.2E-10	2.8E-10	--		4.9E-08	1%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.2E-05	1.0E-09	2.4E-09	--		4.0E-06	50%
Arsenic (As)		2.0E-03		2.0E-03	100%	7.2E-06	1.2E-09	2.9E-09	--		1.4E-06	18%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	4.5E-08	7.4E-12	1.8E-11	--		2.2E-08	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	5.0E-07	8.3E-11	2.0E-10	--		1.4E-09	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1.3E-04	2.1E-08	5.1E-08	--		3.6E-07	4%
Zinc (Zn)		5.0E-01		5.0E-01	100%	3.4E-05	5.7E-09	1.4E-08	--		2.7E-08	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.5E-07	2.5E-11	6.0E-11	--		4.3E-08	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	1.9E-07	3.2E-11	7.7E-11	--		7.7E-08	1%
Mercury (Hg)		6.0E-04		6.0E-04	100%	2.7E-06	4.4E-10	1.1E-09	--		1.8E-06	22%
Lithium (Li)		2.0E-03		2.0E-03	100%	1.3E-06	2.1E-10	5.1E-10	--		2.6E-07	3%
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.0E-07	1.7E-11	4.0E-11	--		3.4E-09	0%
								TOTAL			**0.0000081**	

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 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 (%)	200%	Assumed for rural areas where a higher rate of egg ingestion expected
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 - Private residences

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 (Ag)		5.7E-03		5.7E-03	100%	7.0E-07	4.8E-11	5.6E-10	--		9.9E-08	1%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.2E-05	4.1E-10	4.8E-09	--		3.4E-06	30%
Arsenic (As)		2.0E-03		2.0E-03	100%	7.2E-06	4.9E-10	5.8E-09	--		2.9E-06	25%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	4.5E-08	3.1E-12	3.6E-11	--		4.5E-08	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	5.0E-07	3.4E-11	4.0E-10	--		2.8E-09	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1.3E-04	8.7E-09	1.0E-07	--		7.3E-07	6%
Zinc (Zn)		5.0E-01		5.0E-01	100%	3.4E-05	2.3E-09	2.7E-08	--		5.5E-08	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.5E-07	1.0E-11	1.2E-10	--		8.5E-08	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	1.9E-07	1.3E-11	1.5E-10	--		1.5E-07	1%
Mercury (Hg)		6.0E-04		6.0E-04	100%	2.7E-06	1.8E-10	2.1E-09	--		3.6E-06	31%
Lithium (Li)		2.0E-03		2.0E-03	100%	1.3E-06	8.8E-11	1.0E-09	--		5.1E-07	4%
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.0E-07	6.9E-12	8.1E-11	--		6.7E-09	0%
								TOTAL			**0.000012**	

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%	Assumed to be 100% except for lead

Chemical-specific Inputs and calculations - maximum private residences

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 (Ag)	2.9E-05	6.3E-03	3.0E-03	9.2E-06	RAIS
Lead (Pb)	1.0E-03	2.2E-01	3.0E-04	1.3E-05	
Arsenic (As)	1.6E-04	3.5E-02	2.0E-03	3.4E-05	
Cadmium (Cd)	7.1E-06	1.5E-03	2.0E-04	1.5E-07	
Copper (Cu)	2.1E-05	4.4E-03	1.0E-02	2.2E-05	RAIS
Manganese (Mn)	5.3E-03	1.1E+00	4.0E-04	2.2E-04	RAIS
Zinc (Zn)	1.4E-03	3.0E-01	1.0E-01	1.5E-02	RAIS
Cobalt (Co)	6.2E-06	1.3E-03	2.0E-02	1.3E-05	RAIS
Chromium (Cr)	3.3E-05	7.0E-03	9.2E-03	3.2E-05	OEHHA (2003)
Mercury (Hg)	5.3E-06	1.1E-03	4.0E-04	2.2E-07	
Lithium (Li)	5.4E-05	1.1E-02	1.0E-02	5.6E-05	RAIS
Nickel (Ni)	8.0E-06	1.7E-03	3.0E-04	2.5E-07	

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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	9.2E-06	3.1E-09	7.4E-09	--		1.3E-06	1%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.3E-05	2.1E-09	5.1E-09	--		8.5E-06	8%
Arsenic (As)		2.0E-03		2.0E-03	100%	3.4E-05	1.1E-08	2.7E-08	--		1.4E-05	13%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.5E-07	4.9E-11	1.2E-10	--		1.5E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	2.2E-05	7.2E-09	1.7E-08	--		1.2E-07	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	2.2E-04	7.4E-08	1.8E-07	--		1.3E-06	1%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.5E-02	4.9E-06	1.2E-05	--		2.4E-05	23%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.3E-05	4.3E-09	1.0E-08	--		7.4E-06	7%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.2E-05	1.1E-08	2.5E-08	--		2.5E-05	24%
Mercury (Hg)		6.0E-04		6.0E-04	100%	2.2E-07	7.4E-11	1.8E-10	--		3.0E-07	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	5.6E-05	1.9E-08	4.5E-08	--		2.3E-05	22%
Nickel (Ni)		1.2E-02		1.2E-02	100%	2.5E-07	8.3E-11	2.0E-10	--		1.7E-08	0%
								TOTAL			**0.00010**	

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 - Private residences

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 (Ag)		5.7E-03		5.7E-03	100%	9.2E-06	1.6E-09	1.8E-08	--		3.2E-06	1%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.3E-05	1.1E-09	1.3E-08	--		9.0E-06	4%
Arsenic (As)		2.0E-03		2.0E-03	100%	3.4E-05	5.8E-09	6.8E-08	--		3.4E-05	14%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.5E-07	2.5E-11	2.9E-10	--		3.7E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	2.2E-05	3.7E-09	4.3E-08	--		3.1E-07	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	2.2E-04	3.8E-08	4.4E-07	--		3.1E-06	1%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.5E-02	2.5E-06	3.0E-05	--		5.9E-05	24%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.3E-05	2.2E-09	2.6E-08	--		1.8E-05	7%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.2E-05	5.4E-09	6.3E-08	--		6.3E-05	26%
Mercury (Hg)		6.0E-04		6.0E-04	100%	2.2E-07	3.8E-11	4.4E-10	--		7.4E-07	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	5.6E-05	9.6E-09	1.1E-07	--		5.6E-05	23%
Nickel (Ni)		1.2E-02		1.2E-02	100%	2.5E-07	4.3E-11	5.0E-10	--		4.2E-08	0%
TOTAL											**0.00025**	

Calculation of Concentrations in Dairy Milk

Uptake in to milk (dairy cows)

$$C_E = (FI \times IR_C \times C + IR_S \times C_S \times B) \times TF_B \quad \text{(mg/L)}$$

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/L)

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%	Assumed to be 100% except for lead

Chemical-specific Inputs and calculations - Maximum private residences

Chemical	Concentration in crops ingested by cattle mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to milk day/L	Milk Concentration mg/L	
Silver (Ag)	2.9E-05	6.3E-03	5.0E-03	3.8E-05	Median transfer factor for metals (Leeman et al 2007)
Lead (Pb)	1.0E-03	2.2E-01	6.0E-05	6.2E-06	
Cadmium (Cd)	7.1E-06	1.5E-03	5.0E-06	9.0E-09	
Copper (Cu)	2.1E-05	4.4E-03	1.5E-03	8.0E-06	RAIS
Manganese (Mn)	5.3E-03	1.1E+00	3.5E-04	4.8E-04	RAIS
Zinc (Zn)	1.4E-03	3.0E-01	2.7E-09	9.8E-10	RAIS
Cobalt (Co)	6.2E-06	1.3E-03	2.0E-03	3.2E-06	RAIS
Chromium (Cr)	3.3E-05	7.0E-03	9.0E-06	7.6E-08	
Mercury (Hg)	5.3E-06	1.1E-03	7.0E-05	9.5E-08	
Lithium (Li)	5.4E-05	1.1E-02	5.0E-03	6.9E-05	Median transfer factor for metals (Leeman et al 2007)
Nickel (Ni)	8.0E-06	1.7E-03	3.0E-05	6.2E-08	

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Milk

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

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Milk (IRM) (kg/day)	1.295	Ingestion rate of cows milk for adults (P90 value from FSANZ 2017)
Fraction ingested that is homegrown (%)	100%	Assume all milk consumed is from the dairy farm
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	3.8E-05	2.9E-07	7.0E-07	--		1.2E-04	12%
Lead (Pb)		6.0E-04		6.0E-04	50%	6.2E-06	2.4E-08	5.7E-08	--		9.6E-05	10%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.1E-06	1.6E-08	3.9E-08	--		1.9E-05	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	9.0E-09	6.9E-11	1.7E-10	--		2.1E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	8.0E-06	6.1E-08	1.5E-07	--		1.1E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	4.8E-04	3.6E-06	8.8E-06	--		6.3E-05	6%
Zinc (Zn)		5.0E-01		5.0E-01	100%	9.8E-10	7.5E-12	1.8E-11	--		3.6E-11	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	3.2E-06	2.4E-08	5.9E-08	--		4.2E-05	4%
Chromium (Cr)		1.0E-03		1.0E-03	100%	7.6E-08	5.8E-10	1.4E-09	--		1.4E-06	0%
Mercury (Hg)		6.0E-04		6.0E-04	100%	9.5E-08	7.3E-10	1.8E-09	--		2.9E-06	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	6.9E-05	5.3E-07	1.3E-06	--		6.4E-04	65%
Nickel (Ni)		1.2E-02		1.2E-02	100%	6.2E-08	4.7E-10	1.1E-09	--		9.5E-08	0%
								TOTAL			**0.00098**	

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) (kg/day)	1.097	Ingestion rate of cows milk for children aged 2-6 years (P90 value from FSANZ 2017)
Fraction ingested that is homegrown (%)	100%	Assume all milk consumed is from the dairy farm
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Silver (Ag)		5.7E-03		5.7E-03	100%	3.8E-05	2.4E-07	2.8E-06	--		4.8E-04	13%
Lead (Pb)		1.4E-03		1.4E-03	100%	6.2E-06	3.9E-08	4.5E-07	--		3.2E-04	8%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.1E-06	1.3E-08	1.5E-07	--		7.6E-05	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	9.0E-09	5.7E-11	6.6E-10	--		8.3E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	8.0E-06	5.0E-08	5.8E-07	--		4.2E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	4.8E-04	3.0E-06	3.5E-05	--		2.5E-04	6%
Zinc (Zn)		5.0E-01		5.0E-01	100%	9.8E-10	6.1E-12	7.1E-11	--		1.4E-10	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	3.2E-06	2.0E-08	2.3E-07	--		1.7E-04	4%
Chromium (Cr)		1.0E-03		1.0E-03	100%	7.6E-08	4.8E-10	5.6E-09	--		5.6E-06	0%
Mercury (Hg)		6.0E-04		6.0E-04	100%	9.5E-08	6.0E-10	7.0E-09	--		1.2E-05	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	6.9E-05	4.3E-07	5.0E-06	--		2.5E-03	66%
Nickel (Ni)		1.2E-02		1.2E-02	100%	6.2E-08	3.9E-10	4.5E-09	--		3.8E-07	0%
								TOTAL			**0.0038**	

Scenario 2: Year 3

Predicted ground level concentrations and screening assessment - acute exposures

		PM2.5		Scenario 2			
		Air Concentration (ug/m3)		Air Concentration (1-hour average) (mg/m3)		Calculated HI	
COPC	**Acute air guideline (mg/m3)**	**Maximum anywhere**	**Maximum receptors**	**Maximum all receptors**	**Maximum private residences**	**Maximum all receptors**	**Maximum private residences**
Silver (Ag)	0.3	2.47E-03	5.75E-04	2.5E-06	5.8E-07	8.2E-06	1.9E-06
Lead (Pb)	0.15	7.69E-02	1.84E-02	7.7E-05	1.8E-05	5.1E-04	1.2E-04
Arsenic (As)	0.003	7.13E-03	2.00E-03	7.1E-06	2.0E-06	2.4E-03	6.7E-04
Cadmium (Cd)	0.0054	4.64E-04	1.13E-04	4.6E-07	1.1E-07	8.6E-05	2.1E-05
Copper (Cu)	0.1	6.77E-04	2.04E-04	6.8E-07	2.0E-07	6.8E-06	2.0E-06
Manganese (Mn)	0.0091	2.20E-01	6.35E-02	2.2E-04	6.3E-05	2.4E-02	7.0E-03
Zinc (Zn)	6	9.17E-02	2.25E-02	9.2E-05	2.2E-05	1.5E-05	3.7E-06
Cobalt (Co)	0.00069	1.39E-04	4.45E-05	1.4E-07	4.5E-08	2.0E-04	6.5E-05
Chromium (Cr)	0.0013	2.47E-03	5.92E-04	2.5E-06	5.9E-07	1.9E-03	4.6E-04
Mercury (Hg)	0.0006	1.24E-04	4.02E-05	1.2E-07	4.0E-08	2.1E-04	6.7E-05
Lithium (Li)	3.3	1.46E-03	4.69E-04	1.5E-06	4.7E-07	4.4E-07	1.4E-07
Nickel (Ni)	0.0011	1.64E-04	4.68E-05	1.6E-07	4.7E-08	1.5E-04	4.3E-05
						3.0E-02	8.4E-03

Chronic Exposures

Inhalation - $PM_{2.5}$

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

Parameters Relevant to Quantification of Community Exposures - Residents		
Exposure Time at Home (ET, hr/day)	24	Assume residents at home or on property 24 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Assume resident at the same property
Exposure Frequency (EF, days/yr)	365	Days at home, as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	35	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	306600	US EPA 2009

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk	Chronic TC Air	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background)	Estimated Concentration in Air - Maximum all receptors (Ca)	Inhalation Exposure Concentration - NonThreshold	Inhalation Exposure Concentration - Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/m^3)^{-1}$	(mg/m^3)		(mg/m^3)	(mg/m^3)	(mg/m^3)	(mg/m^3)	(unitless)		(unitless)	
Silver (Ag)		2.0E-02		2.0E-02	2.3E-07	1.1E-07	2.3E-07	--		0.000011	0%
Lead (Pb)		2.0E-03		2.0E-03	7.2E-06	3.6E-06	7.2E-06	--		0.0036	1%
Arsenic (As)		1.0E-03		1.0E-03	7.2E-07	3.6E-07	7.2E-07	--		0.00072	0%
Cadmium (Cd)		5.0E-06		5.0E-06	4.4E-08	2.2E-08	4.4E-08	--		0.0088	4%
Copper (Cu)		4.9E-01		4.9E-01	1.0E-07	5.2E-08	1.0E-07	--		0.00000021	0%
Manganese (Mn)		1.5E-04		1.5E-04	3.5E-05	1.7E-05	3.5E-05	--		0.23	93%
Zinc (Zn)		1.8E+00		1.8E+00	8.7E-06	4.4E-06	8.7E-06	--		0.0000050	0%
Cobalt (Co)		1.0E-04		1.0E-04	1.3E-08	6.7E-09	1.3E-08	--		0.00013	0%
Chromium (Cr)		1.0E-04		1.0E-04	2.3E-07	1.2E-07	2.3E-07	--		0.0023	1%
Mercury (Hg)		2.0E-04		2.0E-04	1.1E-08	5.5E-09	1.1E-08	--		0.000055	0%
Lithium (Li)		7.0E-03		7.0E-03	1.4E-07	7.1E-08	1.4E-07	--		0.000020	0%
Nickel (Ni)		2.0E-05		2.0E-05	1.2E-08	5.9E-09	1.2E-08	--		0.00059	0%
TOTAL										**0.25**	

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk	Chronic TC Air	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background)	Estimated Concentration in Air - Maximum private residences (Ca)	Inhalation Exposure Concentration - NonThreshold	Inhalation Exposure Concentration - Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
Silver (Ag)		2.0E-02		2.0E-02	1.0E-08	5.0E-09	1.0E-08	--		0.00000050	0%
Lead (Pb)		2.0E-03		2.0E-03	3.3E-07	1.7E-07	3.3E-07	--		0.00017	1%
Arsenic (As)		1.0E-03		1.0E-03	3.7E-08	1.9E-08	3.7E-08	--		0.000037	0%
Cadmium (Cd)		5.0E-06		5.0E-06	2.1E-09	1.0E-09	2.1E-09	--		0.00041	3%
Copper (Cu)		4.9E-01		4.9E-01	6.9E-09	3.4E-09	6.9E-09	--		0.000000014	0%
Manganese (Mn)		1.5E-04		1.5E-04	2.1E-06	1.0E-06	2.1E-06	--		0.014	95%
Zinc (Zn)		1.8E+00		1.8E+00	4.1E-07	2.1E-07	4.1E-07	--		0.00000024	0%
Cobalt (Co)		1.0E-04		1.0E-04	9.7E-10	4.9E-10	9.7E-10	--		0.000010	0%
Chromium (Cr)		1.0E-04		1.0E-04	1.1E-08	5.3E-09	1.1E-08	--		0.00011	1%
Mercury (Hg)		2.0E-04		2.0E-04	9.5E-10	4.7E-10	9.5E-10	--		0.0000047	0%
Lithium (Li)		7.0E-03		7.0E-03	1.0E-08	5.0E-09	1.0E-08	--		0.0000014	0%
Nickel (Ni)		2.0E-05		2.0E-05	1.2E-09	6.0E-10	1.2E-09	--		0.000060	0%
TOTAL										**0.015**	

Calculation of Concentrations in Soil

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

ref: Stevens B. (1991)

where:

DR=	Particle deposition rate for TSP (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
Accumulation time (t)	years	70	70	As per OEHHA (2015) guidance

Chemical-specific Inputs and calculations - maximum private residences

Chemical	**Half-life in soil years**	**Loss constant (K) per year**	**TSP Deposition Rate (DR) mg/m^2/year**	**Surface Concentration in Soil mg/kg**	**Agricultural Concentration in Soil mg/kg**
Silver (Ag)	273973	2.5E-06	0.0190	8.3E-02	5.6E-03
Lead (Pb)	273973	2.5E-06	0.7225	3.2E+00	2.1E-01
Arsenic (As)	273973	2.5E-06	0.1124	4.9E-01	3.3E-02
Cadmium (Cd)	273973	2.5E-06	0.0049	2.1E-02	1.4E-03
Copper (Cu)	273973	2.5E-06	0.0150	6.6E-02	4.4E-03
Manganese (Mn)	273973	2.5E-06	3.9020	1.7E+01	1.1E+00
Zinc (Zn)	273973	2.5E-06	0.9826	4.3E+00	2.9E-01
Cobalt (Co)	273973	2.5E-06	0.0037	1.6E-02	1.1E-03
Chromium (Cr)	273973	2.5E-06	0.0227	9.9E-02	6.6E-03
Mercury (Hg)	273973	2.5E-06	0.0037	1.6E-02	1.1E-03
Lithium (Li)	273973	2.5E-06	0.0369	1.6E-01	1.1E-02
Nickel (Ni)	273973	2.5E-06	0.0048	2.1E-02	1.4E-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	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2012 and NEPM 1913
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 - Private residences

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Bioavailability (%)		NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	0.08	2.5E-08	5.9E-08	--		0.000010	1%
Lead (Pb)		6.0E-04		6.0E-04	16%	3.16	1.5E-07	3.7E-07	--		0.00061	57%
Arsenic (As)		2.0E-03		2.0E-03	100%	0.49	1.5E-07	3.5E-07	--		0.00018	16%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	0.02	6.3E-09	1.5E-08	--		0.000019	2%
Copper (Cu)		1.4E-01		1.4E-01	100%	0.07	1.9E-08	4.7E-08	--		0.00000033	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	17.070	5.1E-06	1.2E-05	--		0.000087	8%
Zinc (Zn)		5.0E-01		5.0E-01	100%	4.30	1.3E-06	3.1E-06	--		0.0000061	1%
Cobalt (Co)		1.4E-03		1.4E-03	100%	0.02	4.8E-09	1.2E-08	--		0.0000082	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	0.10	2.9E-08	7.1E-08	--		0.000071	7%
Mercury (Hg)		6.0E-04		6.0E-04	100%	0.016	4.7E-09	1.1E-08	--		0.000019	2%
Lithium (Li)		2.0E-03		2.0E-03	100%	0.16	4.8E-08	1.2E-07	--		0.000058	5%
Nickel (Ni)		1.2E-02		1.2E-02	100%	0.021	6.2E-09	1.5E-08	--		0.0000012	0%
								TOTAL			**0.0011**	

Dermal Exposure to Chemicals via Contact with Soil

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

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/yr)	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 - Private residences

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04		8.3E-02			--		--	
Lead (Pb)		3.0E-04		3.0E-04		3.2E+00			--		--	
Arsenic (As)		2.0E-03		2.0E-03	0.005	4.9E-01	4.6E-08	1.1E-07	--		0.000055	75%
Cadmium (Cd)		8.0E-04		8.0E-04		2.1E-02			--		--	
Copper (Cu)		1.4E-01		1.4E-01		6.6E-02			--		--	
Manganese (Mn)		1.4E-01		1.4E-01		1.7E+01			--		--	
Zinc (Zn)		5.0E-01		5.0E-01	0.001	4.3E+00	8.0E-08	1.9E-07	--		0.00000039	1%
Cobalt (Co)		1.4E-03		1.4E-03	0.001	1.6E-02	3.0E-10	7.3E-10	--		0.00000052	1%
Chromium (Cr)		1.0E-03		1.0E-03		9.9E-02			--		--	
Mercury (Hg)		4.2E-05		4.2E-05	0.001	1.6E-02	3.0E-10	7.2E-10	--		0.000017	23%
Lithium (Li)		2.0E-03		2.0E-03		1.6E-01			--		--	
Nickel (Ni)		1.2E-02		1.2E-02	0.005	2.1E-02	2.0E-09	4.7E-09	--		0.00000039	1%
								TOTAL			**0.000074**	

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 Chilrdren		
Ingestion Rate (IRs, mg/day)	100	Assumed daily soil ingestion rate for young children, enHealth (2012)
Fraction Ingested from Source (FI, unitless)	100%	Compound-specific as noted below
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)
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	0.08	4.8E-08	5.6E-07	--		0.000097	1%
Lead (Pb)		1.4E-03		1.4E-03	16%	3.16	3.0E-07	3.4E-06	--		0.0025	37%
Arsenic (As)		2.0E-03		2.0E-03	100%	0.49	2.8E-07	3.3E-06	--		0.0016	24%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	0.02	1.2E-08	1.4E-07	--		0.00018	3%
Copper (Cu)		1.4E-01		1.4E-01	100%	0.07	3.7E-08	4.4E-07	--		0.0000031	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	17.070	9.8E-06	1.1E-04	--		0.00081	12%
Zinc (Zn)		5.0E-01		5.0E-01	100%	4.30	2.5E-06	2.9E-05	--		0.000057	1%
Cobalt (Co)		1.4E-03		1.4E-03	100%	0.02	9.2E-09	1.1E-07	--		0.000077	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	0.10	5.7E-08	6.6E-07	--		0.00066	10%
Mercury (Hg)		6.0E-04		6.0E-04	100%	0.016	9.2E-09	1.1E-07	--		0.00018	3%
Lithium (Li)		2.0E-03		2.0E-03	100%	0.16	9.2E-08	1.1E-06	--		0.00054	8%
Nickel (Ni)		1.2E-02		1.2E-02	100%	0.021	1.2E-08	1.4E-07	--		0.000012	0%
								TOTAL			**0.0067**	

Dermal Exposure to Chemicals via Contact with Soil

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

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

Maximum - Private residences

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04		8.3E-02			--		--	
Lead (Pb)		7.0E-04		7.0E-04		3.2E+00			--		--	
Arsenic (As)		2.0E-03		2.0E-03	0.005	4.9E-01	1.9E-08	2.2E-07	--		0.00011	75%
Cadmium (Cd)		8.0E-04		8.0E-04		2.1E-02			--		--	
Copper (Cu)		1.4E-01		1.4E-01		6.9E-02			--		--	
Manganese (Mn)		1.4E-01		1.4E-01		2.0E-01			--		--	
Zinc (Zn)		5.0E-01		5.0E-01	0.001	4.3E+00	3.3E-08	3.9E-07	--		0.00000077	1%
Cobalt (Co)		1.4E-03		1.4E-03	0.001	1.6E-02	1.2E-10	1.5E-09	--		0.0000010	1%
Chromium (Cr)		1.0E-03		1.0E-03		9.9E-02			--		--	
Mercury (Hg)		4.2E-05		4.2E-05	0.001	1.6E-02	1.2E-10	1.4E-09	--		0.000034	23%
Lithium (Li)		2.0E-03		2.0E-03		1.6E-01			--		--	
Nickel (Ni)		1.2E-02		1.2E-02	0.005	2.1E-02	8.1E-10	9.4E-09	--		0.00000079	1%
								TOTAL			**0.00015**	

Dermal Exposure to Chemicals via Contact with Soil

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

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

Maximum - Private residences

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04		8.3E-02			--		--	
Lead (Pb)		7.0E-04		7.0E-04		3.2E+00			--		--	
Arsenic (As)		2.0E-03		2.0E-03	0.005	4.9E-01	1.9E-08	2.2E-07	--		0.00011	75%
Cadmium (Cd)		8.0E-04		8.0E-04		2.1E-02			--		--	
Copper (Cu)		1.4E-01		1.4E-01		6.6E-02			--		--	
Manganese (Mn)		1.4E-01		1.4E-01		1.7E+01			--		--	
Zinc (Zn)		5.0E-01		5.0E-01	0.001	4.3E+00	3.3E-08	3.9E-07	--		0.00000077	1%
Cobalt (Co)		1.4E-03		1.4E-03	0.001	1.6E-02	1.2E-10	1.5E-09	--		0.0000010	1%
Chromium (Cr)		1.0E-03		1.0E-03		9.9E-02			--		--	
Mercury (Hg)		4.2E-05		4.2E-05	0.001	1.6E-02	1.2E-10	1.4E-09	--		0.000034	23%
Lithium (Li)		2.0E-03		2.0E-03		1.6E-01			--		--	
Nickel (Ni)		1.2E-02		1.2E-02	0.005	2.1E-02	8.1E-10	9.4E-09	--		0.00000079	1%
								TOTAL			**0.00015**	

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 1 year
DR =	Deposition rate from model for TSP (mg/m^2/year)
Area =	Area of roof (m^2)
VR =	Volume of water collected from roof over year (L) = (R x Area x Rc x 1000)/1000
R =	Rainfall each year (mm)
ρ =	Soil bulk-density (g/cm^3)
Rc =	Runoff coefficient (unitless)
Kd =	Soil-water partition coefficient (cm^3/g)
1000 =	Conversion from mm to m; and conversion from m^3 to L

General Parameters			
Average rainfaill	mm	663.2	mean for all years (1994 - 2019) for Mudgee airport
Roof area	m^2	200	4 bedroom australian home
Runoff coefficient	-	0.7	assumes 30% loss in capture into tank
Volume of rainwater	L	92848	
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 private 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	Drininking water guideline	Proportion of DWG: Particulate	Proportion of DWG: Dissolved
	mg/m^2/year	mg	(cm3/g)	mg/L	mg/L	mg/L		
Silver (Ag)	0.0190	3.8	8.3	4.1E-05	9.9E-06	0.1	0%	0.010%
Lead (Pb)	0.7225	144.5	900	1.6E-03	3.5E-06	0.01	16%	0.035%
Arsenic (As)	0.1124	22.5	29	2.4E-04	1.7E-05	0.01	2%	0.17%
Cadmium (Cd)	0.0049	1.0	75	1.1E-05	2.8E-07	0.002	1%	0.014%
Copper (Cu)	0.0150	3.0	35	3.2E-05	1.8E-06	2	0%	0.000092%
Manganese (Mn)	3.9020	780.4	65	8.4E-03	2.6E-04	0.5	2%	0.051723%
Zinc (Zn)	0.9826	196.5	62	2.1E-03	6.8E-05	6	0%	0.0011%
Cobalt (Co)	0.0037	0.7	45	7.9E-06	3.5E-07	0.006	0%	0.0059%
Chromium (Cr)	0.0227	4.5	19	4.9E-05	5.2E-06	0.05	0%	0.010%
Mercury (Hg)	0.0037	0.7	52	7.9E-06	3.0E-07	0.001	1%	0.030%
Lithium (Li)	0.0369	7.4	300	8.0E-05	5.3E-07	0.04	0%	0.00133%
Nickel (Ni)	0.0048	1.0	65	1.0E-05	3.2E-07	0.02	0%	0.00159%

Apprach assumes all dust deposited on the roof ends up in the water tank - no first flush diversion

0.02 RSL for tap water from USEPA (2018) as no ADWG available

Exposure to Chemicals via Incidental Ingestion of Water

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate (Irw, L/day)	2	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10950	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	4.1E-05	5.0E-07	1.2E-06	--		0.00020	0%
Lead (Pb)	0.0E+00	6.0E-04	0%	6.0E-04	50%	1.6E-03	9.5E-06	2.2E-05	--		0.037	24%
Arsenic (As)	0.0E+00	2.0E-03	0%	2.0E-03	100%	2.4E-04	3.0E-06	6.9E-06	--		0.0035	2%
Cadmium (Cd)	0.0E+00	8.0E-04	0%	8.0E-04	100%	1.1E-05	1.3E-07	3.0E-07	--		0.00038	0%
Copper (Cu)	0.0E+00	1.4E-01	0%	1.4E-01	100%	3.2E-05	4.0E-07	9.2E-07	--		0.0000066	0%
Manganese (Mn)	0.0E+00	1.4E-01	0%	1.4E-01	100%	8.4E-03	1.0E-04	2.4E-04	--		0.0017	1%
Zinc (Zn)	0.0E+00	5.0E-01	0%	5.0E-01	100%	2.1E-03	2.6E-05	6.0E-05	--		0.00012	0%
Cobalt (Co)	0.0E+00	1.4E-03	0%	1.4E-03	100%	7.9E-06	9.7E-08	2.3E-07	--		0.00016	0%
Chromium (Cr)	0.0E+00	1.0E-03	0%	1.0E-03	100%	4.9E-05	6.0E-07	1.4E-06	--		0.0014	1%
Mercury (Hg)	0.0E+00	6.0E-04	0%	6.0E-04	100%	7.9E-06	9.7E-08	2.3E-07	--		0.00038	0%
Lithium (Li)	0.0E+00	2.0E-03	0%	2.0E-03	100%	8.0E-05	9.7E-07	2.3E-06	--		0.0011	1%
Nickel (Ni)	0.0E+00	1.2E-02	0%	1.2E-02	100%	1.0E-05	1.3E-07	3.0E-07	--		0.000025	0%
								TOTAL	**0.00E+00**		**0.046**	

Dermal Exposure to Chemicals via Contact with Water

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Adults		
Surface Area (Saw, cm2)	20000	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	0.58	Reasonable maximum time spent showering or wet each day (ESEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10950	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	4.10E-05	1.43E-11	1.7E-09	4.1E-09	--		1.8E-05	4%
Lead (Pb)		3.0E-04		3.0E-04	1.00E-4	1.56E-03	9.03E-11	1.1E-08	2.6E-08	--		8.6E-05	19%
Arsenic (As)		2.0E-03		2.0E-03	1.00E-3	2.42E-04	1.40E-10	1.7E-08	4.0E-08	--		2.0E-05	4%
Cadmium (Cd)		8.0E-04		8.0E-04	1.00E-3	1.05E-05	6.09E-12	7.5E-10	1.7E-09	--		2.2E-06	0%
Copper (Cu)		1.4E-01		1.4E-01	1.00E-3	3.23E-05	1.87E-11	2.3E-09	5.3E-09	--		3.8E-08	0%
Manganese (Mn)		1.4E-01		1.4E-01	1.00E-3	8.41E-03	4.87E-09	6.0E-07	1.4E-06	--		9.9E-06	2%
Zinc (Zn)		5.0E-01		5.0E-01	6.00E-4	2.12E-03	7.37E-10	9.0E-08	2.1E-07	--		4.2E-07	0%
Cobalt (Co)		1.4E-03		1.4E-03	4.00E-4	7.94E-06	1.84E-12	2.3E-10	5.3E-10	--		3.8E-07	0%
Chromium (Cr)		1.0E-03		1.0E-03	2.00E-3	4.90E-05	5.68E-11	7.0E-09	1.6E-08	--		1.6E-05	4%
Mercury (Hg)		4.2E-05		4.2E-05	1.00E-3	7.89E-06	4.58E-12	5.6E-10	1.3E-09	--		3.1E-05	7%
Lithium (Li)		2.0E-03		2.0E-03	1.00E-3	7.95E-05	4.61E-11	5.6E-09	1.3E-08	--		6.6E-06	1%
Nickel (Ni)		1.2E-02		1.2E-02	2.00E-4	1.03E-05	1.20E-12	1.5E-10	3.4E-10	--		2.9E-08	0%

	1.4E-04

Exposure to Chemicals via Incidental Ingestion of Water

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Children		
Ingestion Rate (Irw, L/day)	0.4	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	4.1E-05	9.4E-08	1.1E-06	--		0.00019	0%
Lead (Pb)	0.0E+00	1.4E-03	0%	1.4E-03	50%	1.6E-03	1.8E-06	2.1E-05	--		0.015	20%
Arsenic (As)	0.0E+00	2.0E-03	0%	2.0E-03	100%	2.4E-04	5.5E-07	6.5E-06	--		0.0032	4%
Cadmium (Cd)	0.0E+00	8.0E-04	0%	8.0E-04	100%	1.1E-05	2.4E-08	2.8E-07	--		0.00035	0%
Copper (Cu)	0.0E+00	1.4E-01	0%	1.4E-01	100%	3.2E-05	7.4E-08	8.6E-07	--		0.0000061	0%
Manganese (Mn)	0.0E+00	1.4E-01	0%	1.4E-01	100%	8.4E-03	1.9E-05	2.2E-04	--		0.0016	2%
Zinc (Zn)	0.0E+00	5.0E-01	0%	5.0E-01	100%	2.1E-03	4.8E-06	5.6E-05	--		0.00011	0%
Cobalt (Co)	0.0E+00	1.4E-03	0%	1.4E-03	100%	7.9E-06	1.8E-08	2.1E-07	--		0.00015	0%
Chromium (Cr)	0.0E+00	1.0E-03	0%	1.0E-03	100%	4.9E-05	1.1E-07	1.3E-06	--		0.0013	2%
Mercury (Hg)	0.0E+00	6.0E-04	0%	6.0E-04	100%	7.9E-06	1.8E-08	2.1E-07	--		0.00035	0%
Lithium (Li)	0.0E+00	2.0E-03	0%	2.0E-03	100%	8.0E-05	1.8E-07	2.1E-06	--		0.0011	1%
Nickel (Ni)	0.0E+00	1.2E-02	0%	1.2E-02	100%	1.0E-05	2.4E-08	2.8E-07	--		0.000023	0%
								TOTAL	**0.00E+00**		**0.023**	

Dermal Exposure to Chemicals via Contact with Water

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Children		
Surface Area (Saw, cm2)	6100	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	1	Reasonable maximum time spent showering or wet each day (ESEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	4.10E-05	2.46E-11	8.6E-10	1.0E-08	--		4.4E-05	6%
Lead (Pb)		7.0E-04		7.0E-04	1.00E-4	1.56E-03	1.56E-10	5.4E-09	6.3E-08	--		9.0E-05	13%
Arsenic (As)		2.0E-03		2.0E-03	1.00E-3	2.42E-04	2.42E-10	8.4E-09	9.8E-08	--		4.9E-05	7%
Cadmium (Cd)		8.0E-04		8.0E-04	1.00E-3	1.05E-05	1.05E-11	3.7E-10	4.3E-09	--		5.3E-06	1%
Copper (Cu)		1.4E-01		1.4E-01	1.00E-3	3.23E-05	3.23E-11	1.1E-09	1.3E-08	--		9.4E-08	0%
Manganese (Mn)		1.4E-01		1.4E-01	1.00E-3	8.41E-03	8.41E-09	2.9E-07	3.4E-06	--		2.4E-05	3%
Zinc (Zn)		5.0E-01		5.0E-01	6.00E-4	2.12E-03	1.27E-09	4.4E-08	5.2E-07	--		1.0E-06	0%
Cobalt (Co)		1.4E-03		1.4E-03	4.00E-4	7.94E-06	3.18E-12	1.1E-10	1.3E-09	--		9.2E-07	0%
Chromium (Cr)		1.0E-03		1.0E-03	2.00E-3	4.90E-05	9.79E-11	3.4E-09	4.0E-08	--		4.0E-05	6%
Mercury (Hg)		4.2E-05		4.2E-05	1.00E-3	7.89E-06	7.89E-12	2.7E-10	3.2E-09	--		7.6E-05	11%
Lithium (Li)		2.0E-03		2.0E-03	1.00E-3	7.95E-05	7.95E-11	2.8E-09	3.2E-08	--		1.6E-05	2%
Nickel (Ni)		1.2E-02		1.2E-02	2.00E-4	1.03E-05	2.07E-12	7.2E-11	8.4E-10	--		7.0E-08	0%

	2.1E-04

Calculation of Concentrations in Plants

ref: Stevens B. (1991)

Uptake Due to Deposition in Aboveground Crops

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

where:
DR= Particle deposition rate for TSP (mg/m^2/day)
F= Fraction for the surface area of plant (unitless)
k= Chemical-specific soil-loss constant (1/years) = ln(2)/$T_{0.5}$
$T_{0.5}$= Chemical half-life as particulate on plant (days)
t= Deposition time (days)
Y= Crop yield (kg/m^2)

Uptake via Roots from Soil

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

where:
Cs = Concentration of persistent chemical in soil assuming 15cm mixing depth within gardens, calculated using Soil Equation for each chemical assessed (mg/kg)
RUF = Root uptake factor which differs for each Chemical (unitless)

General Parameters	Units	Value
Crop		Edible crops
Crop Yield (Y)	kg/m^2	2
Deposition Time (t)	days	70
Plant Interception fraction (F)	unitless	0.051

Chemical-specific Inputs and calculations - Maximum private residences

Chemical	Half-life in plant ($T_{0.5}$)	Loss constant (k)	Deposition Rate TSP (DR)	Aboveground Produce Concentration via Deposition	Root Uptake Factor (RUF)	Soil Concentration (Cs)	Below Ground Produce Concentration
	days	per day	mg/m^2/day	mg/kg ww	unitless	mg/kg	mg/kg ww
Silver (Ag)	14	0.05	0.0000521	2.6E-05	0.1	5.6E-03	5.6E-04
Lead (Pb)	14	0.05	0.0019793	9.9E-04	0.011	2.1E-01	2.3E-03
Cadmium (Cd)	14	0.05	0.0000134	6.7E-06	0.125	1.4E-03	1.8E-04
Copper (Cu)	14	0.05	0.0000410	2.0E-05	0.1	4.4E-03	4.4E-04
Manganese (Mn)	14	0.05	0.0106903	5.3E-03	0.0625	1.1E+00	7.1E-02
Zinc (Zn)	14	0.05	0.0026920	1.3E-03	0.0113	2.9E-01	3.2E-03
Cobalt (Co)	14	0.05	0.0000101	5.0E-06	0.005	1.1E-03	5.4E-06
Chromium (Cr)	14	0.05	0.0000623	3.1E-05	0.00188	6.6E-03	1.2E-05
Mercury (Hg)	14	0.05	0.0000100	5.0E-06	0.225	1.1E-03	2.4E-04
Lithium (Li)	14	0.05	0.0001012	5.0E-05	0.00625	1.1E-02	6.7E-05
Nickel (Ni)	14	0.05	0.0000131	6.6E-06	0.015	1.4E-03	2.1E-05

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

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 (%)	35%	Assumed for rural areas (higher than typical default)
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)				NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	2.6E-05	5.6E-04	1.4E-07	3.4E-07	--		5.9E-05	2%
Lead (Pb)		6.0E-04		6.0E-04	50%	9.9E-04	2.3E-03		1.3E-06	--		2.2E-03	71%
Arsenic (As)		2.0E-03		2.0E-03	100%	1.5E-04	3.3E-04	4.4E-08	1.1E-07	--		5.3E-05	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	6.7E-06	1.8E-04	4.4E-08	1.1E-07	--		1.3E-04	4%
Copper (Cu)		1.4E-01		1.4E-01	100%	2.0E-05	4.4E-04	1.1E-07	2.7E-07	--		1.9E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	5.3E-03	7.1E-02	1.9E-05	4.6E-05	--		3.3E-04	10%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.3E-03	3.2E-03	1.5E-06	3.7E-06	--		7.4E-06	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	5.0E-06	5.4E-06	4.2E-09	1.0E-08	--		7.3E-06	0%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.1E-05	1.2E-05	2.2E-08	5.2E-08	--		5.2E-05	2%
Mercury (Hg)		6.0E-04		6.0E-04	100%	5.0E-06	2.4E-04	5.7E-08	1.4E-07	--		2.3E-04	7%
Lithium (Li)		2.0E-03		2.0E-03	100%	5.0E-05	6.7E-05	4.6E-08	1.1E-07	--		5.5E-05	2%
Nickel (Ni)		1.2E-02		1.2E-02	100%	6.6E-06	2.1E-05	8.7E-09	2.1E-08	--		1.7E-06	0%
									TOTAL			**0.0032**	

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 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 (%)	35%	Assumed for rural areas (higher than typical default)
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)				NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	2.6E-05	5.6E-04	6.2E-08	7.2E-07	--		1.3E-04	3%
Lead (Pb)		1.4E-03		1.4E-03	50%	9.9E-04	2.3E-03		3.9E-06	--		2.8E-03	57%
Arsenic (As)		2.0E-03		2.0E-03	100%	1.5E-04	3.3E-04	1.9E-08	2.2E-07	--		1.1E-04	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	6.7E-06	1.8E-04	1.9E-08	2.2E-07	--		2.8E-04	6%
Copper (Cu)		1.4E-01		1.4E-01	100%	2.0E-05	4.4E-04	4.9E-08	5.7E-07	--		4.1E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	5.3E-03	7.1E-02	8.9E-06	1.0E-04	--		7.4E-04	15%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.3E-03	3.2E-03	9.2E-07	1.1E-05	--		2.2E-05	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	5.0E-06	5.4E-06	2.9E-09	3.3E-08	--		2.4E-05	0%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.1E-05	1.2E-05	1.6E-08	1.8E-07	--		1.8E-04	4%
Mercury (Hg)		6.0E-04		6.0E-04	100%	5.0E-06	2.4E-04	2.4E-08	2.8E-07	--		4.6E-04	9%
Lithium (Li)		2.0E-03		2.0E-03	100%	5.0E-05	6.7E-05	3.0E-08	3.5E-07	--		1.7E-04	4%
Nickel (Ni)		1.2E-02		1.2E-02	100%	6.6E-06	2.1E-05	5.0E-09	5.8E-08	--		4.8E-06	0%
									TOTAL			**0.0049**	

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 100% of crops consumed by chickens is grown in the same soil
IRc (ingestion rate of crops)	kg/day	0.12	Assumed ingestion rate from OEHHA 2015 (assume concentration the same as predicted for aboveground crops)
IRs (ingestion rate of soil)	kg/day	0.0024	Based on data from OEHHA 2015 (2% total produce intakes from soil)
B (bioavailability)	%	100%	Assumed to be 100% except for lead

Chemical-specific Inputs and calculations - Maximum private residences

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 (Ag)	2.6E-05	5.6E-03	3.8E-02	6.2E-07	
Lead (Pb)	9.9E-04	2.1E-01	4.0E-02	1.1E-05	
Arsenic (As)	1.5E-04	3.3E-02	7.0E-02	6.8E-06	
Cadmium (Cd)	6.7E-06	1.4E-03	1.0E-02	4.2E-08	
Copper (Cu)	2.0E-05	4.4E-03	3.8E-02	4.9E-07	
Manganese (Mn)	5.3E-03	1.1E+00	3.8E-02	1.3E-04	
Zinc (Zn)	1.3E-03	2.9E-01	3.8E-02	3.2E-05	
Cobalt (Co)	5.0E-06	1.1E-03	3.8E-02	1.2E-07	
Chromium (Cr)	3.1E-05	6.6E-03	9.2E-03	1.8E-07	OEHHA (2003)
Mercury (Hg)	5.0E-06	1.1E-03	8.0E-01	2.5E-06	
Lithium (Li)	5.0E-05	1.1E-02	3.8E-02	1.2E-06	
Nickel (Ni)	6.6E-06	1.4E-03	2.0E-02	8.3E-08	

Transfer factors from OEHHA 2015 unless otherwise noted
Mean transfer factor for heavy metals used in absense of specific data (Leeman et al 2007)

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 (%)	200%	Assumed for rural areas where a higher rate of egg ingestion expected
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	6.2E-07	1.0E-10	2.5E-10	--		4.4E-08	1%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.1E-05	9.4E-10	2.3E-09	--		3.8E-06	50%
Arsenic (As)		2.0E-03		2.0E-03	100%	6.8E-06	1.1E-09	2.7E-09	--		1.4E-06	18%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	4.2E-08	7.0E-12	1.7E-11	--		2.1E-08	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	4.9E-07	8.1E-11	2.0E-10	--		1.4E-09	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1.3E-04	2.1E-08	5.1E-08	--		3.7E-07	5%
Zinc (Zn)		5.0E-01		5.0E-01	100%	3.2E-05	5.3E-09	1.3E-08	--		2.6E-08	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.2E-07	2.0E-11	4.8E-11	--		3.5E-08	0%
Chromium (Cr)		1.0E-03		1.0E-03	100%	1.8E-07	3.0E-11	7.2E-11	--		7.2E-08	1%
Mercury (Hg)		6.0E-04		6.0E-04	100%	2.5E-06	4.2E-10	1.0E-09	--		1.7E-06	22%
Lithium (Li)		2.0E-03		2.0E-03	100%	1.2E-06	2.0E-10	4.8E-10	--		2.4E-07	3%
Nickel (Ni)		1.2E-02		1.2E-02	100%	8.3E-08	1.4E-11	3.3E-11	--		2.8E-09	0%
								TOTAL			**0.0000076**	

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 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 (%)	200%	Assumed for rural areas where a higher rate of egg ingestion expected
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 - Private residences

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 (Ag)		5.7E-03		5.7E-03	100%	6.2E-07	4.3E-11	5.0E-10	--		8.7E-08	1%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.1E-05	3.9E-10	4.5E-09	--		3.2E-06	30%
Arsenic (As)		2.0E-03		2.0E-03	100%	6.8E-06	4.7E-10	5.4E-09	--		2.7E-06	25%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	4.2E-08	2.9E-12	3.4E-11	--		4.2E-08	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	4.9E-07	3.4E-11	3.9E-10	--		2.8E-09	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1.3E-04	8.8E-09	1.0E-07	--		7.3E-07	7%
Zinc (Zn)		5.0E-01		5.0E-01	100%	3.2E-05	2.2E-09	2.6E-08	--		5.2E-08	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.2E-07	8.3E-12	9.7E-11	--		6.9E-08	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	1.8E-07	1.2E-11	1.4E-10	--		1.4E-07	1%
Mercury (Hg)		6.0E-04		6.0E-04	100%	2.5E-06	1.7E-10	2.0E-09	--		3.4E-06	31%
Lithium (Li)		2.0E-03		2.0E-03	100%	1.2E-06	8.3E-11	9.7E-10	--		4.8E-07	4%
Nickel (Ni)		1.2E-02		1.2E-02	100%	8.3E-08	5.7E-12	6.6E-11	--		5.5E-09	0%
								TOTAL			**0.000011**	

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%	Assumed to be 100% except for lead

Chemical-specific Inputs and calculations - maximum private residences

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 (Ag)	2.6E-05	5.6E-03	3.0E-03	8.2E-06	RAIS
Lead (Pb)	9.9E-04	2.1E-01	3.0E-04	1.2E-05	
Arsenic (As)	1.5E-04	3.3E-02	2.0E-03	3.2E-05	
Cadmium (Cd)	6.7E-06	1.4E-03	2.0E-04	1.4E-07	
Copper (Cu)	2.0E-05	4.4E-03	1.0E-02	2.2E-05	RAIS
Manganese (Mn)	5.3E-03	1.1E+00	4.0E-04	2.2E-04	RAIS
Zinc (Zn)	1.3E-03	2.9E-01	1.0E-01	1.4E-02	RAIS
Cobalt (Co)	5.0E-06	1.1E-03	2.0E-02	1.1E-05	RAIS
Chromium (Cr)	3.1E-05	6.6E-03	9.2E-03	3.0E-05	OEHHA (2003)
Mercury (Hg)	5.0E-06	1.1E-03	4.0E-04	2.1E-07	
Lithium (Li)	5.0E-05	1.1E-02	1.0E-02	5.3E-05	RAIS
Nickel (Ni)	6.6E-06	1.4E-03	3.0E-04	2.1E-07	

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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	8.2E-06	2.7E-09	6.6E-09	--		1.1E-06	1%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.2E-05	2.0E-09	4.8E-09	--		8.0E-06	8%
Arsenic (As)		2.0E-03		2.0E-03	100%	3.2E-05	1.1E-08	2.6E-08	--		1.3E-05	13%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.4E-07	4.6E-11	1.1E-10	--		1.4E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	2.2E-05	7.1E-09	1.7E-08	--		1.2E-07	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	2.2E-04	7.4E-08	1.8E-07	--		1.3E-06	1%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.4E-02	4.7E-06	1.1E-05	--		2.3E-05	23%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.1E-05	3.5E-09	8.5E-09	--		6.0E-06	6%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.0E-05	9.9E-09	2.4E-08	--		2.4E-05	25%
Mercury (Hg)		6.0E-04		6.0E-04	100%	2.1E-07	7.0E-11	1.7E-10	--		2.8E-07	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	5.3E-05	1.8E-08	4.2E-08	--		2.1E-05	22%
Nickel (Ni)		1.2E-02		1.2E-02	100%	2.1E-07	6.8E-11	1.7E-10	--		1.4E-08	0%
								TOTAL			**0.00010**	

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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	8.2E-06	1.4E-09	1.6E-08	--		2.8E-06	1%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.2E-05	1.0E-09	1.2E-08	--		8.5E-06	4%
Arsenic (As)		2.0E-03		2.0E-03	100%	3.2E-05	5.5E-09	6.4E-08	--		3.2E-05	14%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.4E-07	2.4E-11	2.8E-10	--		3.5E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	2.2E-05	3.7E-09	4.3E-08	--		3.0E-07	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	2.2E-04	3.8E-08	4.4E-07	--		3.2E-06	1%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.4E-02	2.4E-06	2.8E-05	--		5.6E-05	24%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.1E-05	1.8E-09	2.1E-08	--		1.5E-05	6%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.0E-05	5.1E-09	6.0E-08	--		6.0E-05	26%
Mercury (Hg)		6.0E-04		6.0E-04	100%	2.1E-07	3.6E-11	4.2E-10	--		7.0E-07	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	5.3E-05	9.0E-09	1.1E-07	--		5.3E-05	23%
Nickel (Ni)		1.2E-02		1.2E-02	100%	2.1E-07	3.5E-11	4.1E-10	--		3.4E-08	0%
								TOTAL			**0.00023**	

Calculation of Concentrations in Dairy Milk

Uptake in to milk (dairy cows)

$$C_E = (FI \times IR_C \times C + IR_S \times C_S \times B) \times TF_B \quad \text{(mg/L)}$$

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/L)

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%	Assumed to be 100% except for lead

Chemical-specific Inputs and calculations - Maximum private residences

Chemical	Concentration in crops ingested by cattle mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to milk day/L	Milk Concentration mg/L	
Silver (Ag)	2.6E-05	5.6E-03	5.0E-03	3.3E-05	Median transfer factor for metals (Leeman et al 2007)
Lead (Pb)	9.9E-04	2.1E-01	6.0E-05	5.8E-06	
Cadmium (Cd)	6.7E-06	1.4E-03	5.0E-06	8.6E-09	
Copper (Cu)	2.0E-05	4.4E-03	1.5E-03	7.9E-06	RAIS
Manganese (Mn)	5.3E-03	1.1E+00	3.5E-04	4.8E-04	RAIS
Zinc (Zn)	1.3E-03	2.9E-01	2.7E-09	9.2E-10	RAIS
Cobalt (Co)	5.0E-06	1.1E-03	2.0E-03	2.6E-06	RAIS
Chromium (Cr)	3.1E-05	6.6E-03	9.0E-06	7.2E-08	
Mercury (Hg)	5.0E-06	1.1E-03	7.0E-05	9.0E-08	
Lithium (Li)	5.0E-05	1.1E-02	5.0E-03	6.5E-05	Median transfer factor for metals (Leeman et al 2007)
Nickel (Ni)	6.6E-06	1.4E-03	3.0E-05	5.1E-08	

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Milk

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

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Milk (IRM) (kg/day)	1.295	Ingestion rate of cows milk for adults (P90 value from FSANZ 2017)
Fraction ingested that is homegrown (%)	100%	Assume all milk consumed is from the dairy farm
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	3.3E-05	2.6E-07	6.2E-07	--		1.1E-04	12%
Lead (Pb)		6.0E-04		6.0E-04	50%	5.8E-06	2.2E-08	5.4E-08	--		9.0E-05	10%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.0E-06	1.5E-08	3.6E-08	--		1.8E-05	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	8.6E-09	6.6E-11	1.6E-10	--		2.0E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	7.9E-06	6.0E-08	1.5E-07	--		1.0E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	4.8E-04	3.7E-06	8.9E-06	--		6.3E-05	7%
Zinc (Zn)		5.0E-01		5.0E-01	100%	9.2E-10	7.1E-12	1.7E-11	--		3.4E-11	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	2.6E-06	2.0E-08	4.8E-08	--		3.4E-05	4%
Chromium (Cr)		1.0E-03		1.0E-03	100%	7.2E-08	5.5E-10	1.3E-09	--		1.3E-06	0%
Mercury (Hg)		6.0E-04		6.0E-04	100%	9.0E-08	6.9E-10	1.7E-09	--		2.8E-06	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	6.5E-05	5.0E-07	1.2E-06	--		6.0E-04	65%
Nickel (Ni)		1.2E-02		1.2E-02	100%	5.1E-08	3.9E-10	9.3E-10	--		7.8E-08	0%
								TOTAL			**0.00092**	

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) (kg/day)	1.097	Ingestion rate of cows milk for children aged 2-6 years (P90 value from FSANZ 2017)
Fraction ingested that is homegrown (%)	100%	Assume all milk consumed is from the dairy farm
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Silver (Ag)		5.7E-03		5.7E-03	100%	3.3E-05	2.1E-07	2.4E-06	--		4.3E-04	12%
Lead (Pb)		1.4E-03		1.4E-03	50%	5.8E-06	1.8E-08	2.1E-07	--		1.5E-04	4%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.0E-06	1.2E-08	1.4E-07	--		7.2E-05	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	8.6E-09	5.4E-11	6.3E-10	--		7.8E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	7.9E-06	4.9E-08	5.8E-07	--		4.1E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	4.8E-04	3.0E-06	3.5E-05	--		2.5E-04	7%
Zinc (Zn)		5.0E-01		5.0E-01	100%	9.2E-10	5.8E-12	6.8E-11	--		1.4E-10	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	2.6E-06	1.6E-08	1.9E-07	--		1.4E-04	4%
Chromium (Cr)		1.0E-03		1.0E-03	100%	7.2E-08	4.5E-10	5.2E-09	--		5.2E-06	0%
Mercury (Hg)		6.0E-04		6.0E-04	100%	9.0E-08	5.6E-10	6.6E-09	--		1.1E-05	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	6.5E-05	4.1E-07	4.7E-06	--		2.4E-03	69%
Nickel (Ni)		1.2E-02		1.2E-02	100%	5.1E-08	3.2E-10	3.7E-09	--		3.1E-07	0%
								TOTAL			**0.0034**	

Scenario 3: Year 8

Predicted ground level concentrations and screening assessment - acute exposures

		PM2.5		Scenario 3			
		Air Concentration (ug/m3)		Air Concentration (1-hour average) (mg/m3)		Calculated HI	
COPC	**Acute air guideline (mg/m3)**	**Maximum anywhere**	**Maximum receptors**	**Maximum all receptors**	**Maximum private residences**	**Maximum all receptors**	**Maximum private residences**
Silver (Ag)	0.3	2.11E-03	5.98E-04	2.1E-06	6.0E-07	7.0E-06	2.0E-06
Lead (Pb)	0.15	6.59E-02	1.93E-02	6.6E-05	1.9E-05	4.4E-04	1.3E-04
Arsenic (As)	0.003	6.11E-03	2.09E-03	6.1E-06	2.1E-06	2.0E-03	7.0E-04
Cadmium (Cd)	0.0054	3.97E-04	1.19E-04	4.0E-07	1.2E-07	7.4E-05	2.2E-05
Copper (Cu)	0.1	7.23E-04	2.24E-04	7.2E-07	2.2E-07	7.2E-06	2.2E-06
Manganese (Mn)	0.0091	1.99E-01	6.83E-02	2.0E-04	6.8E-05	2.2E-02	7.5E-03
Zinc (Zn)	6	7.86E-02	2.35E-02	7.9E-05	2.4E-05	1.3E-05	3.9E-06
Cobalt (Co)	0.00069	1.65E-04	4.69E-05	1.6E-07	4.7E-08	2.4E-04	6.8E-05
Chromium (Cr)	0.0013	2.12E-03	6.07E-04	2.1E-06	6.1E-07	1.6E-03	4.7E-04
Mercury (Hg)	0.0006	1.61E-04	4.33E-05	1.6E-07	4.3E-08	2.7E-04	7.2E-05
Lithium (Li)	3.3	1.70E-03	4.94E-04	1.7E-06	4.9E-07	5.1E-07	1.5E-07
Nickel (Ni)	0.0011	2.02E-04	5.63E-05	2.0E-07	5.6E-08	1.8E-04	5.1E-05
						2.7E-02	9.0E-03

Chronic Exposures

Inhalation - PM2.5

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

Parameters Relevant to Quantification of Community Exposures - Residents		
Exposure Time at Home (ET, hr/day)	24	Assume residents at home or on property 24 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Assume resident at the same property
Exposure Frequency (EF, days/yr)	365	Days at home, as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	35	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	306600	US EPA 2009

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk	Chronic TC Air	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background)	Estimated Concentration in Air - Maximum all receptors (Ca)	Inhalation Exposure Concentration - NonThreshold	Inhalation Exposure Concentration - Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/m^3)^{-1}$	(mg/m^3)		(mg/m^3)	(mg/m^3)	(mg/m^3)	(mg/m^3)	(unitless)		(unitless)	
Silver (Ag)		2.0E-02		2.0E-02	1.9E-07	9.5E-08	1.9E-07	--		0.000010	0%
Lead (Pb)		2.0E-03		2.0E-03	6.1E-06	3.0E-06	6.1E-06	--		0.0030	2%
Arsenic (As)		1.0E-03		1.0E-03	6.1E-07	3.1E-07	6.1E-07	--		0.00061	0%
Cadmium (Cd)		5.0E-06		5.0E-06	3.7E-08	1.9E-08	3.7E-08	--		0.0074	5%
Copper (Cu)		4.9E-01		4.9E-01	5.9E-08	3.0E-08	5.9E-08	--		0.00000012	0%
Manganese (Mn)		1.5E-04		1.5E-04	1.9E-05	9.7E-06	1.9E-05	--		0.13	90%
Zinc (Zn)		1.8E+00		1.8E+00	7.4E-06	3.7E-06	7.4E-06	--		0.0000042	0%
Cobalt (Co)		1.0E-04		1.0E-04	1.1E-08	5.7E-09	1.1E-08	--		0.00011	0%
Chromium (Cr)		1.0E-04		1.0E-04	2.0E-07	9.8E-08	2.0E-07	--		0.0020	1%
Mercury (Hg)		2.0E-04		2.0E-04	9.6E-09	4.8E-09	9.6E-09	--		0.000048	0%
Lithium (Li)		7.0E-03		7.0E-03	1.2E-07	6.2E-08	1.2E-07	--		0.000018	0%
Nickel (Ni)		2.0E-05		2.0E-05	1.0E-08	5.2E-09	1.0E-08	--		0.00052	0%
							TOTAL			**0.14**	

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk	Chronic TC Air	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background)	Estimated Concentration in Air - Maximum private residences (Ca)	Inhalation Exposure Concentration - NonThreshold	Inhalation Exposure Concentration - Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
Silver (Ag)		2.0E-02		2.0E-02	5.6E-09	2.8E-09	5.6E-09	--		0.00000028	0%
Lead (Pb)		2.0E-03		2.0E-03	2.0E-07	9.8E-08	2.0E-07	--		0.00010	1%
Arsenic (As)		1.0E-03		1.0E-03	3.3E-08	1.7E-08	3.3E-08	--		0.000033	0%
Cadmium (Cd)		5.0E-06		5.0E-06	1.3E-09	6.7E-10	1.3E-09	--		0.00027	3%
Copper (Cu)		4.9E-01		4.9E-01	4.8E-09	2.4E-09	4.8E-09	--		0.000000010	0%
Manganese (Mn)		1.5E-04		1.5E-04	1.2E-06	6.0E-07	1.2E-06	--		0.0079	93%
Zinc (Zn)		1.8E+00		1.8E+00	2.7E-07	1.4E-07	2.7E-07	--		0.00000016	0%
Cobalt (Co)		1.0E-04		1.0E-04	1.2E-09	5.8E-10	1.2E-09	--		0.000012	0%
Chromium (Cr)		1.0E-04		1.0E-04	6.3E-09	3.1E-09	6.3E-09	--		0.000063	1%
Mercury (Hg)		2.0E-04		2.0E-04	1.2E-09	6.0E-10	1.2E-09	--		0.0000060	0%
Lithium (Li)		7.0E-03		7.0E-03	1.2E-08	5.9E-09	1.2E-08	--		0.0000017	0%
Nickel (Ni)		2.0E-05		2.0E-05	1.6E-09	7.8E-10	1.6E-09	--		0.000078	1%
							TOTAL			**0.0085**	

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 TSP (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
Accumulation time (t)	years	70	70	As per OEHHA (2015) guidance

Chemical-specific Inputs and calculations - maximum private residences

Chemical	**Half-life in soil**	**Loss constant (K)**	**TSP Deposition Rate (DR)**	**Surface Concentration in Soil**	**Agricultural Concentration in Soil**
	years	**per year**	**mg/m^2/year**	**mg/kg**	**mg/kg**
Silver (Ag)	273973	2.5E-06	0.0219	9.6E-02	6.4E-03
Lead (Pb)	273973	2.5E-06	0.8421	3.7E+00	2.5E-01
Arsenic (As)	273973	2.5E-06	0.1349	5.9E-01	3.9E-02
Cadmium (Cd)	273973	2.5E-06	0.0057	2.5E-02	1.7E-03
Copper (Cu)	273973	2.5E-06	0.0185	8.1E-02	5.4E-03
Manganese (Mn)	273973	2.5E-06	4.7390	2.1E+01	1.4E+00
Zinc (Zn)	273973	2.5E-06	1.1568	5.1E+00	3.4E-01
Cobalt (Co)	273973	2.5E-06	0.0044	1.9E-02	1.3E-03
Chromium (Cr)	273973	2.5E-06	0.0256	1.1E-01	7.5E-03
Mercury (Hg)	273973	2.5E-06	0.0045	2.0E-02	1.3E-03
Lithium (Li)	273973	2.5E-06	0.0451	2.0E-01	1.3E-02
Nickel (Ni)	273973	2.5E-06	0.0059	2.6E-02	1.7E-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	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2012 and NEPM 2013
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	0.10	2.8E-08	6.8E-08	--		0.000012	1%
Lead (Pb)		6.0E-04		6.0E-04	16%	3.68	1.8E-07	4.3E-07	--		0.00072	57%
Arsenic (As)		2.0E-03		2.0E-03	100%	0.59	1.7E-07	4.2E-07	--		0.00021	17%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	0.03	7.4E-09	1.8E-08	--		0.000022	2%
Copper (Cu)		1.4E-01		1.4E-01	100%	0.08	2.4E-08	5.8E-08	--		0.00000041	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	20.731	6.1E-06	1.5E-05	--		0.00011	8%
Zinc (Zn)		5.0E-01		5.0E-01	100%	5.06	1.5E-06	3.6E-06	--		0.0000072	1%
Cobalt (Co)		1.4E-03		1.4E-03	100%	0.02	5.8E-09	1.4E-08	--		0.000010	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	0.11	3.3E-08	8.0E-08	--		0.000080	6%
Mercury (Hg)		6.0E-04		6.0E-04	100%	0.020	5.8E-09	1.4E-08	--		0.000023	2%
Lithium (Li)		2.0E-03		2.0E-03	100%	0.20	5.8E-08	1.4E-07	--		0.000071	6%
Nickel (Ni)		1.2E-02		1.2E-02	100%	0.026	7.6E-09	1.8E-08	--		0.0000015	0%
								TOTAL			**0.0013**	

Dermal Exposure to Chemicals via Contact with Soil

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

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/yr)	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 - Private residences

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04		9.6E-02			--		--	
Lead (Pb)		3.0E-04		3.0E-04		3.7E+00			--		--	
Arsenic (As)		2.0E-03		2.0E-03	0.005	5.9E-01	5.5E-08	1.3E-07	--		0.000066	75%
Cadmium (Cd)		8.0E-04		8.0E-04		2.5E-02			--		--	
Copper (Cu)		1.4E-01		1.4E-01		8.1E-02			--		--	
Manganese (Mn)		1.4E-01		1.4E-01		2.1E+01			--		--	
Zinc (Zn)		5.0E-01		5.0E-01	0.001	5.1E+00	9.4E-08	2.3E-07	--		0.00000046	1%
Cobalt (Co)		1.4E-03		1.4E-03	0.001	1.9E-02	3.6E-10	8.8E-10	--		0.00000063	1%
Chromium (Cr)		1.0E-03		1.0E-03		1.1E-01			--		--	
Mercury (Hg)		4.2E-05		4.2E-05	0.001	2.0E-02	3.7E-10	8.9E-10	--		0.000021	24%
Lithium (Li)		2.0E-03		2.0E-03		2.0E-01			--		--	
Nickel (Ni)		1.2E-02		1.2E-02	0.005	2.6E-02	2.4E-09	5.8E-09	--		0.00000048	1%
								TOTAL			**0.000089**	

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 Chilrdren		
Ingestion Rate (IRs, mg/day)	100	Assumed daily soil ingestion rate for young children, enHealth (2012)
Fraction Ingested from Source (FI, unitless)	100%	Compound-specific as noted below
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)
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	0.10	5.5E-08	6.4E-07	--		0.00011	1%
Lead (Pb)		1.4E-03		1.4E-03	16%	3.68	3.4E-07	4.0E-06	--		0.0029	36%
Arsenic (As)		2.0E-03		2.0E-03	100%	0.59	3.4E-07	3.9E-06	--		0.0020	25%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	0.03	1.4E-08	1.7E-07	--		0.00021	3%
Copper (Cu)		1.4E-01		1.4E-01	100%	0.08	4.6E-08	5.4E-07	--		0.000004	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	20.731	1.2E-05	1.4E-04	--		0.00099	12%
Zinc (Zn)		5.0E-01		5.0E-01	100%	5.06	2.9E-06	3.4E-05	--		0.000067	1%
Cobalt (Co)		1.4E-03		1.4E-03	100%	0.02	1.1E-08	1.3E-07	--		0.000093	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	0.11	6.4E-08	7.5E-07	--		0.00075	9%
Mercury (Hg)		6.0E-04		6.0E-04	100%	0.020	1.1E-08	1.3E-07	--		0.00022	3%
Lithium (Li)		2.0E-03		2.0E-03	100%	0.20	1.1E-07	1.3E-06	--		0.00066	8%
Nickel (Ni)		1.2E-02		1.2E-02	100%	0.026	1.5E-08	1.7E-07	--		0.000014	0%
								TOTAL			**0.0079**	

Dermal Exposure to Chemicals via Contact with Soil

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

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

Maximum - Private residences

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04		9.6E-02			--		--	
Lead (Pb)		7.0E-04		7.0E-04		3.7E+00			--		--	
Arsenic (As)		2.0E-03		2.0E-03	0.005	5.9E-01	2.3E-08	2.7E-07	--		0.00013	75%
Cadmium (Cd)		8.0E-04		8.0E-04		2.5E-02			--		--	
Copper (Cu)		1.4E-01		1.4E-01		8.1E-02			--		--	
Manganese (Mn)		1.4E-01		1.4E-01		2.1E+01			--		--	
Zinc (Zn)		5.0E-01		5.0E-01	0.001	5.1E+00	3.9E-08	4.6E-07	--		0.00000091	1%
Cobalt (Co)		1.4E-03		1.4E-03	0.001	1.9E-02	1.5E-10	1.8E-09	--		0.0000013	1%
Chromium (Cr)		1.0E-03		1.0E-03		1.1E-01			--		--	
Mercury (Hg)		4.2E-05		4.2E-05	0.001	2.0E-02	1.5E-10	1.8E-09	--		0.000042	24%
Lithium (Li)		2.0E-03		2.0E-03		2.0E-01			--		--	
Nickel (Ni)		1.2E-02		1.2E-02	0.005	2.6E-02	9.9E-10	1.2E-08	--		0.0000010	1%
								TOTAL			**0.00018**	

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 1 year
DR =	Deposition rate from model for TSP (mg/m^2/year)
Area =	Area of roof (m^2)
VR =	Volume of water collected from roof over year (L) = (R x Area x Rc x 1000)/1000
R =	Rainfall each year (mm)
ρ =	Soil bulk-density (g/cm^3)
Rc =	Runoff coefficient (unitless)
Kd =	Soil-water partition coefficient (cm^3/g)
1000 =	Conversion from mm to m; and conversion from m^3 to L

General Parameters			
Average rainfaill	mm	663.2	mean for all years (1994 - 2019) for Mudgee airport
Roof area	m^2	200	4 bedroom australian home
Runoff coefficient	-	0.7	assumes 30% water loss in capture into tank (includes first flush)
Volume of rainwater	L	92848	
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 private 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	Drininking water guideline	Proportion of DWG: Particulate	Proportion of DWG: Dissolved
	mg/m^2/year	mg	(cm3/g)	mg/L	mg/L	mg/L		
Silver (Ag)	0.0219	4.4	8.3	4.7E-05	1.1E-05	0.1	0%	0.011%
Lead (Pb)	0.8421	168.4	900	1.8E-03	4.0E-06	0.01	18%	0.040%
Arsenic (As)	0.1349	27.0	29	2.9E-04	2.0E-05	0.01	3%	0.20%
Cadmium (Cd)	0.0057	1.1	75	1.2E-05	3.3E-07	0.002	1%	0.016%
Copper (Cu)	0.0185	3.7	35	4.0E-05	2.3E-06	2	0%	0.000114%
Manganese (Mn)	4.7390	947.8	65	1.0E-02	3.1E-04	0.5	2%	0.062819%
Zinc (Zn)	1.1568	231.4	62	2.5E-03	8.0E-05	6	0%	0.0013%
Cobalt (Co)	0.0044	0.9	45	9.6E-06	4.3E-07	0.006	0%	0.0071%
Chromium (Cr)	0.0256	5.1	19	5.5E-05	5.8E-06	0.05	0%	0.012%
Mercury (Hg)	0.0045	0.9	52	9.7E-06	3.7E-07	0.001	1%	0.037%
Lithium (Li)	0.0451	9.0	300	9.7E-05	6.5E-07	0.04	0%	0.00162%
Nickel (Ni)	0.0059	1.2	65	1.3E-05	3.9E-07	0.02	0%	0.00194%

Apprach assumes all dust deposited on the roof ends up in the water tank - no first flush diversion

0.02 RSL for tap water from USEPA (2018) as no ADWG available

Exposure to Chemicals via Incidental Ingestion of Water

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate (Irw, L/day)	2	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10950	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	4.7E-05	5.8E-07	1.3E-06	--		0.00024	0%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.8E-03	1.1E-05	2.6E-05	--		0.043	4%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.9E-04	3.6E-06	8.3E-06	--		0.0042	0%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.2E-05	1.5E-07	3.5E-07	--		0.00044	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	4.0E-05	4.9E-07	1.1E-06	--		0.0000081	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1.0E-02	1.2E-04	2.9E-04	--		0.0021	0%
Zinc (Zn)		5.0E-01		5.0E-01	100%	2.5E-03	3.1E-05	7.1E-05	--		0.00014	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	9.6E-06	1.2E-07	2.7E-07	--		0.00020	0%
Chromium (Cr)		1.0E-03		1.0E-03	100%	5.5E-05	6.8E-07	1.6E-06	--		0.0016	0%
Mercury (Hg)		6.0E-04		6.0E-04	100%	9.7E-06	1.2E-07	2.8E-07	--		0.00046	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	9.7E-05	1.2E-06	2.8E-06	--		0.0014	0%
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.3E-05	1.5E-07	3.6E-07	--		0.000030	0%
								TOTAL			**0.054**	

Dermal Exposure to Chemicals via Contact with Water

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Adults		
Surface Area (Saw, cm2)	20000	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	0.58	Reasonable maximum time spent showering or wet each day (ESEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10950	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	4.71E-05	1.64E-11	2.0E-09	4.7E-09	--		2.0E-05	1%
Lead (Pb)		3.0E-04		3.0E-04	1.00E-4	1.81E-03	1.05E-10	1.3E-08	3.0E-08	--		1.0E-04	3%
Arsenic (As)		2.0E-03		2.0E-03	1.00E-3	2.91E-04	1.68E-10	2.1E-08	4.8E-08	--		2.4E-05	1%
Cadmium (Cd)		8.0E-04		8.0E-04	1.00E-3	1.23E-05	7.16E-12	8.8E-10	2.0E-09	--		2.6E-06	0%
Copper (Cu)		1.4E-01		1.4E-01	1.00E-3	3.99E-05	2.31E-11	2.8E-09	6.6E-09	--		4.7E-08	0%
Manganese (Mn)		1.4E-01		1.4E-01	1.00E-3	1.02E-02	5.92E-09	7.2E-07	1.7E-06	--		1.2E-05	0%
Zinc (Zn)		5.0E-01		5.0E-01	6.00E-4	2.49E-03	8.67E-10	1.1E-07	2.5E-07	--		5.0E-07	0%
Cobalt (Co)		1.4E-03		1.4E-03	4.00E-4	9.58E-06	2.22E-12	2.7E-10	6.4E-10	--		4.5E-07	0%
Chromium (Cr)		1.0E-03		1.0E-03	2.00E-3	5.51E-05	6.40E-11	7.8E-09	1.8E-08	--		1.8E-05	1%
Mercury (Hg)		4.2E-05		4.2E-05	1.00E-3	9.72E-06	5.64E-12	6.9E-10	1.6E-09	--		3.8E-05	1%
Lithium (Li)		2.0E-03		2.0E-03	1.00E-3	9.72E-05	5.64E-11	6.9E-09	1.6E-08	--		8.1E-06	0%
Nickel (Ni)		1.2E-02		1.2E-02	2.00E-4	1.26E-05	1.47E-12	1.8E-10	4.2E-10	--		3.5E-08	0%

	1.6E-04

Exposure to Chemicals via Incidental Ingestion of Water

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Children		
Ingestion Rate (Irw, L/day)	0.4	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	4.7E-05	1.1E-07	1.3E-06	--		0.00022	0%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.8E-03	2.1E-06	2.4E-05	--		0.017	3%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.9E-04	6.6E-07	7.7E-06	--		0.0039	1%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.2E-05	2.8E-08	3.3E-07	--		0.00041	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	4.0E-05	9.1E-08	1.1E-06	--		0.0000076	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1.0E-02	2.3E-05	2.7E-04	--		0.0019	0%
Zinc (Zn)		5.0E-01		5.0E-01	100%	2.5E-03	5.7E-06	6.6E-05	--		0.00013	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	9.6E-06	2.2E-08	2.6E-07	--		0.00018	0%
Chromium (Cr)		1.0E-03		1.0E-03	100%	5.5E-05	1.3E-07	1.5E-06	--		0.0015	0%
Mercury (Hg)		6.0E-04		6.0E-04	100%	9.7E-06	2.2E-08	2.6E-07	--		0.00043	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	9.7E-05	2.2E-07	2.6E-06	--		0.0013	0%
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.3E-05	2.9E-08	3.4E-07	--		0.000028	0%
								TOTAL			**0.027**	

Dermal Exposure to Chemicals via Contact with Water

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Children		
Surface Area (Saw, cm2)	6100	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	1	Reasonable maximum time spent showering or wet each day (ESEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	4.71E-05	2.83E-11	9.9E-10	1.1E-08	--		5.0E-05	1%
Lead (Pb)		7.0E-04		7.0E-04	1.00E-4	1.81E-03	1.81E-10	6.3E-09	7.4E-08	--		1.1E-04	2%
Arsenic (As)		2.0E-03		2.0E-03	1.00E-3	2.91E-04	2.91E-10	1.0E-08	1.2E-07	--		5.9E-05	1%
Cadmium (Cd)		8.0E-04		8.0E-04	1.00E-3	1.23E-05	1.23E-11	4.3E-10	5.0E-09	--		6.3E-06	0%
Copper (Cu)		1.4E-01		1.4E-01	1.00E-3	3.99E-05	3.99E-11	1.4E-09	1.6E-08	--		1.2E-07	0%
Manganese (Mn)		1.4E-01		1.4E-01	1.00E-3	1.02E-02	1.02E-08	3.6E-07	4.2E-06	--		3.0E-05	1%
Zinc (Zn)		5.0E-01		5.0E-01	6.00E-4	2.49E-03	1.50E-09	5.2E-08	6.1E-07	--		1.2E-06	0%
Cobalt (Co)		1.4E-03		1.4E-03	4.00E-4	9.58E-06	3.83E-12	1.3E-10	1.6E-09	--		1.1E-06	0%
Chromium (Cr)		1.0E-03		1.0E-03	2.00E-3	5.51E-05	1.10E-10	3.8E-09	4.5E-08	--		4.5E-05	1%
Mercury (Hg)		4.2E-05		4.2E-05	1.00E-3	9.72E-06	9.72E-12	3.4E-10	4.0E-09	--		9.4E-05	2%
Lithium (Li)		2.0E-03		2.0E-03	1.00E-3	9.72E-05	9.72E-11	3.4E-09	4.0E-08	--		2.0E-05	0%
Nickel (Ni)		1.2E-02		1.2E-02	2.00E-4	1.26E-05	2.53E-12	8.8E-11	1.0E-09	--		8.6E-08	0%
												2.5E-04	

Calculation of Concentrations in Plants

ref: Stevens B. (1991)

Uptake Due to Deposition in Aboveground Crops

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

where:
DR= Particle deposition rate for TSP (mg/m^2/day)
F= Fraction for the surface area of plant (unitless)
k= Chemical-specific soil-loss constant (1/years) = ln(2)/$T_{0.5}$
$T_{0.5}$= Chemical half-life as particulate on plant (days)
t= Deposition time (days)
Y= Crop yield (kg/m^2)

Uptake via Roots from Soil

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

where:
Cs = Concentration of persistent chemical in soil assuming 15cm mixing depth within gardens, calculated using Soil Equation for each chemical assessed (mg/kg)
RUF = Root uptake factor which differs for each Chemical (unitless)

General Parameters	Units	Value
Crop		Edible crops
Crop Yield (Y)	kg/m^2	2
Deposition Time (t)	days	70
Plant Interception fraction (F)	unitless	0.051

Chemical-specific Inputs and calculations - Maximum private residences

Chemical	Half-life in plant ($T_{0.5}$)	Loss constant (k)	Deposition Rate TSP (DR)	Aboveground Produce Concentration via Deposition	Root Uptake Factor (RUF)	Soil Concentration (Cs)	Below Ground Produce Concentration
	days	per day	mg/m^2/day	mg/kg ww	unitless	mg/kg	mg/kg ww
Silver (Ag)	14	0.05	0.0000599	3.0E-05	0.1	6.4E-03	6.4E-04
Lead (Pb)	14	0.05	0.0023072	1.2E-03	0.011	2.5E-01	2.7E-03
Cadmium (Cd)	14	0.05	0.0000157	7.8E-06	0.125	1.7E-03	2.1E-04
Copper (Cu)	14	0.05	0.0000507	2.5E-05	0.1	5.4E-03	5.4E-04
Manganese (Mn)	14	0.05	0.0129836	6.5E-03	0.0625	1.4E+00	8.6E-02
Zinc (Zn)	14	0.05	0.0031692	1.6E-03	0.0113	3.4E-01	3.8E-03
Cobalt (Co)	14	0.05	0.0000122	6.1E-06	0.005	1.3E-03	6.5E-06
Chromium (Cr)	14	0.05	0.0000701	3.5E-05	0.00188	7.5E-03	1.4E-05
Mercury (Hg)	14	0.05	0.0000124	6.2E-06	0.225	1.3E-03	3.0E-04
Lithium (Li)	14	0.05	0.0001236	6.2E-05	0.00625	1.3E-02	8.2E-05
Nickel (Ni)	14	0.05	0.0000161	8.0E-06	0.015	1.7E-03	2.6E-05

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

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 (%)	35%	Assumed for rural areas (higher than typical default)
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)				NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	3.0E-05	6.4E-04	1.6E-07	3.9E-07	--		6.8E-05	2%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.2E-03	2.7E-03	6.5E-07	1.6E-06	--		2.6E-03	70%
Arsenic (As)		2.0E-03		2.0E-03	100%	1.8E-04	3.9E-04	5.1E-08	1.2E-07	--		6.2E-05	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	7.8E-06	2.1E-04	5.1E-08	1.2E-07	--		1.6E-04	4%
Copper (Cu)		1.4E-01		1.4E-01	100%	2.5E-05	5.4E-04	1.4E-07	3.3E-07	--		2.3E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	6.5E-03	8.6E-02	2.3E-05	5.6E-05	--		4.0E-04	11%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.6E-03	3.8E-03	1.8E-06	4.4E-06	--		8.7E-06	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	6.1E-06	6.5E-06	5.1E-09	1.2E-08	--		8.8E-06	0%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.5E-05	1.4E-05	2.4E-08	5.9E-08	--		5.9E-05	2%
Mercury (Hg)		6.0E-04		6.0E-04	100%	6.2E-06	3.0E-04	7.0E-08	1.7E-07	--		2.8E-04	8%
Lithium (Li)		2.0E-03		2.0E-03	100%	6.2E-05	8.2E-05	5.6E-08	1.3E-07	--		6.7E-05	2%
Nickel (Ni)		1.2E-02		1.2E-02	100%	8.0E-06	2.6E-05	1.1E-08	2.6E-08	--		2.1E-06	0%
							TOTAL					**0.0037**	

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 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 (%)	35%	Assumed for rural areas (higher than typical default)
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)				NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	3.0E-05	6.4E-04	7.1E-08	8.3E-07	--		1.5E-04	2%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.2E-03	2.7E-03	3.9E-07	4.6E-06	--		3.3E-03	56%
Arsenic (As)		2.0E-03		2.0E-03	100%	1.8E-04	3.9E-04	2.2E-08	2.6E-07	--		1.3E-04	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	7.8E-06	2.1E-04	2.2E-08	2.6E-07	--		3.3E-04	6%
Copper (Cu)		1.4E-01		1.4E-01	100%	2.5E-05	5.4E-04	6.0E-08	7.0E-07	--		5.0E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	6.5E-03	8.6E-02	1.1E-05	1.3E-04	--		9.0E-04	15%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.6E-03	3.8E-03	1.1E-06	1.3E-05	--		2.5E-05	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	6.1E-06	6.5E-06	3.4E-09	4.0E-08	--		2.9E-05	0%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.5E-05	1.4E-05	1.8E-08	2.1E-07	--		2.1E-04	4%
Mercury (Hg)		6.0E-04		6.0E-04	100%	6.2E-06	3.0E-04	2.9E-08	3.4E-07	--		5.7E-04	10%
Lithium (Li)		2.0E-03		2.0E-03	100%	6.2E-05	8.2E-05	3.6E-08	4.2E-07	--		2.1E-04	4%
Nickel (Ni)		1.2E-02		1.2E-02	100%	8.0E-06	2.6E-05	6.1E-09	7.1E-08	--		5.9E-06	0%
									TOTAL			**0.0058**	

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 100% of crops consumed by chickens is grown in the same soil
IRc (ingestion rate of crops)	kg/day	0.12	Assumed ingestion rate from OEHHA 2015 (assume concentration the same as predicted for aboveground crops)
IRs (ingestion rate of soil)	kg/day	0.0024	Based on data from OEHHA 2015 (2% total produce intakes from soil)
B (bioavailability)	%	100%	Assumed to be 100% except for lead

Chemical-specific Inputs and calculations - Maximum private residences

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 (Ag)	3.0E-05	6.4E-03	3.8E-02	7.2E-07	
Lead (Pb)	1.2E-03	2.5E-01	4.0E-02	1.3E-05	
Arsenic (As)	1.8E-04	3.9E-02	7.0E-02	8.2E-06	
Cadmium (Cd)	7.8E-06	1.7E-03	1.0E-02	5.0E-08	
Copper (Cu)	2.5E-05	5.4E-03	3.8E-02	6.1E-07	
Manganese (Mn)	6.5E-03	1.4E+00	3.8E-02	1.6E-04	
Zinc (Zn)	1.6E-03	3.4E-01	3.8E-02	3.8E-05	
Cobalt (Co)	6.1E-06	1.3E-03	3.8E-02	1.5E-07	
Chromium (Cr)	3.5E-05	7.5E-03	9.2E-03	2.0E-07	OEHHA (2003)
Mercury (Hg)	6.2E-06	1.3E-03	8.0E-01	3.1E-06	
Lithium (Li)	6.2E-05	1.3E-02	3.8E-02	1.5E-06	
Nickel (Ni)	8.0E-06	1.7E-03	2.0E-02	1.0E-07	

Transfer factors from OEHHA 2015 unless otherwise noted
Mean transfer factor for heavy metals used in absense of specific data (Leeman et al 2007)

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 (%)	200%	Assumed for rural areas where a higher rate of egg ingestion expected
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 - Private residences

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 (Ag)		5.7E-03		5.7E-03	100%	7.2E-07	1.2E-10	2.9E-10	--		5.0E-08	1%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.3E-05	1.1E-09	2.6E-09	--		4.4E-06	48%
Arsenic (As)		2.0E-03		2.0E-03	100%	8.2E-06	1.4E-09	3.3E-09	--		1.6E-06	18%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	5.0E-08	8.2E-12	2.0E-11	--		2.5E-08	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	6.1E-07	1.0E-10	2.4E-10	--		1.7E-09	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1.6E-04	2.6E-08	6.2E-08	--		4.4E-07	5%
Zinc (Zn)		5.0E-01		5.0E-01	100%	3.8E-05	6.3E-09	1.5E-08	--		3.0E-08	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.5E-07	2.4E-11	5.8E-11	--		4.2E-08	0%
Chromium (Cr)		1.0E-03		1.0E-03	100%	2.0E-07	3.4E-11	8.1E-11	--		8.1E-08	1%
Mercury (Hg)		6.0E-04		6.0E-04	100%	3.1E-06	5.2E-10	1.2E-09	--		2.1E-06	23%
Lithium (Li)		2.0E-03		2.0E-03	100%	1.5E-06	2.5E-10	5.9E-10	--		3.0E-07	3%
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.0E-07	1.7E-11	4.1E-11	--		3.4E-09	0%
								TOTAL			**0.0000091**	

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 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 (%)	200%	Assumed for rural areas where a higher rate of egg ingestion expected
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 - Private residences

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 (Ag)		5.7E-03		5.7E-03	100%	7.2E-07	4.9E-11	5.7E-10	--		1.0E-07	1%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.3E-05	4.5E-10	5.3E-09	--		3.8E-06	29%
Arsenic (As)		2.0E-03		2.0E-03	100%	8.2E-06	5.6E-10	6.5E-09	--		3.3E-06	25%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	5.0E-08	3.4E-12	4.0E-11	--		5.0E-08	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	6.1E-07	4.2E-11	4.9E-10	--		3.5E-09	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1.6E-04	1.1E-08	1.2E-07	--		8.9E-07	7%
Zinc (Zn)		5.0E-01		5.0E-01	100%	3.8E-05	2.6E-09	3.0E-08	--		6.1E-08	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.5E-07	1.0E-11	1.2E-10	--		8.3E-08	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	2.0E-07	1.4E-11	1.6E-10	--		1.6E-07	1%
Mercury (Hg)		6.0E-04		6.0E-04	100%	3.1E-06	2.1E-10	2.5E-09	--		4.2E-06	32%
Lithium (Li)		2.0E-03		2.0E-03	100%	1.5E-06	1.0E-10	1.2E-09	--		5.9E-07	5%
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.0E-07	6.9E-12	8.1E-11	--		6.8E-09	0%
								TOTAL			**0.000013**	

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%	Assumed to be 100% except for lead

Chemical-specific Inputs and calculations - maximum private residences

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 (Ag)	3.0E-05	6.4E-03	3.0E-03	9.4E-06	RAIS
Lead (Pb)	1.2E-03	2.5E-01	3.0E-04	1.4E-05	
Arsenic (As)	1.8E-04	3.9E-02	2.0E-03	3.9E-05	
Cadmium (Cd)	7.8E-06	1.7E-03	2.0E-04	1.6E-07	
Copper (Cu)	2.5E-05	5.4E-03	1.0E-02	2.7E-05	RAIS
Manganese (Mn)	6.5E-03	1.4E+00	4.0E-04	2.7E-04	RAIS
Zinc (Zn)	1.6E-03	3.4E-01	1.0E-01	1.7E-02	RAIS
Cobalt (Co)	6.1E-06	1.3E-03	2.0E-02	1.3E-05	RAIS
Chromium (Cr)	3.5E-05	7.5E-03	9.2E-03	3.4E-05	OEHHA (2003)
Mercury (Hg)	6.2E-06	1.3E-03	4.0E-04	2.6E-07	
Lithium (Li)	6.2E-05	1.3E-02	1.0E-02	6.5E-05	RAIS
Nickel (Ni)	8.0E-06	1.7E-03	3.0E-04	2.5E-07	

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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	9.4E-06	3.1E-09	7.5E-09	--		1.3E-06	1%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.4E-05	2.3E-09	5.6E-09	--		9.3E-06	8%
Arsenic (As)		2.0E-03		2.0E-03	100%	3.9E-05	1.3E-08	3.1E-08	--		1.5E-05	13%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.6E-07	5.5E-11	1.3E-10	--		1.6E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	2.7E-05	8.8E-09	2.1E-08	--		1.5E-07	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	2.7E-04	9.0E-08	2.2E-07	--		1.6E-06	1%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.7E-02	5.5E-06	1.3E-05	--		2.7E-05	23%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.3E-05	4.2E-09	1.0E-08	--		7.3E-06	6%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.4E-05	1.1E-08	2.7E-08	--		2.7E-05	23%
Mercury (Hg)		6.0E-04		6.0E-04	100%	2.6E-07	8.6E-11	2.1E-10	--		3.5E-07	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	6.5E-05	2.1E-08	5.2E-08	--		2.6E-05	23%
Nickel (Ni)		1.2E-02		1.2E-02	100%	2.5E-07	8.4E-11	2.0E-10	--		1.7E-08	0%
								TOTAL			**0.00012**	

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 - Private residences

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 (Ag)		5.7E-03		5.7E-03	100%	9.4E-06	1.6E-09	1.9E-08	--		3.3E-06	1%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.4E-05	1.2E-09	1.4E-08	--		9.9E-06	4%
Arsenic (As)		2.0E-03		2.0E-03	100%	3.9E-05	6.6E-09	7.7E-08	--		3.8E-05	14%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.6E-07	2.8E-11	3.3E-10	--		4.1E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	2.7E-05	4.5E-09	5.3E-08	--		3.8E-07	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	2.7E-04	4.6E-08	5.4E-07	--		3.9E-06	1%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.7E-02	2.8E-06	3.3E-05	--		6.6E-05	24%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.3E-05	2.2E-09	2.5E-08	--		1.8E-05	7%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.4E-05	5.7E-09	6.7E-08	--		6.7E-05	25%
Mercury (Hg)		6.0E-04		6.0E-04	100%	2.6E-07	4.4E-11	5.1E-10	--		8.6E-07	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	6.5E-05	1.1E-08	1.3E-07	--		6.4E-05	24%
Nickel (Ni)		1.2E-02		1.2E-02	100%	2.5E-07	4.3E-11	5.0E-10	--		4.2E-08	0%
								TOTAL			**0.00027**	

Calculation of Concentrations in Dairy Milk

Uptake in to milk (dairy cows)

$$C_E=(FI \times IR_C \times C+IR_S \times C_S \times B) \times TF_B \quad \text{(mg/L)}$$

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/L)

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%	Assumed to be 100% except for lead

Chemical-specific Inputs and calculations - Maximum private residences

Chemical	Concentration in crops ingested by cattle mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to milk day/L	Milk Concentration mg/L	
Silver (Ag)	3.0E-05	6.4E-03	5.0E-03	3.8E-05	Median transfer factor for metals (Leeman et al 2007)
Lead (Pb)	1.2E-03	2.5E-01	6.0E-05	6.8E-06	
Cadmium (Cd)	7.8E-06	1.7E-03	5.0E-06	1.0E-08	
Copper (Cu)	2.5E-05	5.4E-03	1.5E-03	9.7E-06	RAIS
Manganese (Mn)	6.5E-03	1.4E+00	3.5E-04	5.8E-04	RAIS
Zinc (Zn)	1.6E-03	3.4E-01	2.7E-09	1.1E-09	RAIS
Cobalt (Co)	6.1E-06	1.3E-03	2.0E-03	3.1E-06	RAIS
Chromium (Cr)	3.5E-05	7.5E-03	9.0E-06	8.1E-08	
Mercury (Hg)	6.2E-06	1.3E-03	7.0E-05	1.1E-07	
Lithium (Li)	6.2E-05	1.3E-02	5.0E-03	7.9E-05	Median transfer factor for metals (Leeman et al 2007)
Nickel (Ni)	8.0E-06	1.7E-03	3.0E-05	6.2E-08	

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Milk

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

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Milk (IRM) (kg/day)	1.295	Ingestion rate of cows milk for adults (P90 value from FSANZ 2017)
Fraction ingested that is homegrown (%)	100%	Assume all milk consumed is from the dairy farm
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	3.8E-05	2.9E-07	7.1E-07	--		1.2E-04	11%
Lead (Pb)		6.0E-04		6.0E-04	50%	6.8E-06	2.6E-08	6.3E-08	--		1.1E-04	9%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.4E-06	1.8E-08	4.4E-08	--		2.2E-05	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.0E-08	7.7E-11	1.9E-10	--		2.3E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	9.7E-06	7.5E-08	1.8E-07	--		1.3E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	5.8E-04	4.5E-06	1.1E-05	--		7.7E-05	7%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.1E-09	8.3E-12	2.0E-11	--		4.0E-11	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	3.1E-06	2.4E-08	5.8E-08	--		4.1E-05	4%
Chromium (Cr)		1.0E-03		1.0E-03	100%	8.1E-08	6.2E-10	1.5E-09	--		1.5E-06	0%
Mercury (Hg)		6.0E-04		6.0E-04	100%	1.1E-07	8.5E-10	2.0E-09	--		3.4E-06	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	7.9E-05	6.1E-07	1.5E-06	--		7.3E-04	66%
Nickel (Ni)		1.2E-02		1.2E-02	100%	6.2E-08	4.7E-10	1.1E-09	--		9.5E-08	0%
								TOTAL			**0.0011**	

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) (kg/day)	1.097	Ingestion rate of cows milk for children aged 2-6 years (P90 value from FSANZ 2017)
Fraction ingested that is homegrown (%)	100%	Assume all milk consumed is from the dairy farm
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	3.8E-05	2.4E-07	2.8E-06	--		4.9E-04	12%
Lead (Pb)		1.4E-03		1.4E-03	50%	6.8E-06	2.1E-08	2.5E-07	--		1.8E-04	4%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.4E-06	1.5E-08	1.7E-07	--		8.7E-05	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.0E-08	6.3E-11	7.4E-10	--		9.2E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	9.7E-06	6.1E-08	7.1E-07	--		5.1E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	5.8E-04	3.6E-06	4.3E-05	--		3.0E-04	7%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.1E-09	6.8E-12	8.0E-11	--		1.6E-10	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	3.1E-06	2.0E-08	2.3E-07	--		1.6E-04	4%
Chromium (Cr)		1.0E-03		1.0E-03	100%	8.1E-08	5.1E-10	5.9E-09	--		5.9E-06	0%
Mercury (Hg)		6.0E-04		6.0E-04	100%	1.1E-07	6.9E-10	8.1E-09	--		1.4E-05	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	7.9E-05	5.0E-07	5.8E-06	--		2.9E-03	70%
Nickel (Ni)		1.2E-02		1.2E-02	100%	6.2E-08	3.9E-10	4.5E-09	--		3.8E-07	0%
								TOTAL			**0.0041**	

Scenario 4: Year 9

Predicted ground level concentrations and screening assessment - acute exposures

		PM2.5		Scenario 4			
		Air Concentration (ug/m3)		Air Concentration (1-hour average) (mg/m3)		Calculated HI	
COPC	**Acute air guideline (mg/m3)**	**Maximum anywhere**	**Maximum receptors**	**Maximum all receptors**	**Maximum private residences**	**Maximum all receptors**	**Maximum private residences**
Silver (Ag)	0.3	2.27E-03	6.06E-04	2.3E-06	6.1E-07	7.6E-06	2.0E-06
Lead (Pb)	0.15	7.05E-02	2.02E-02	7.1E-05	2.0E-05	4.7E-04	1.3E-04
Arsenic (As)	0.003	6.48E-03	2.44E-03	6.5E-06	2.4E-06	2.2E-03	8.1E-04
Cadmium (Cd)	0.0054	4.24E-04	1.27E-04	4.2E-07	1.3E-07	7.9E-05	2.4E-05
Copper (Cu)	0.1	5.94E-04	2.85E-04	5.9E-07	2.8E-07	5.9E-06	2.8E-06
Manganese (Mn)	0.0091	2.02E-01	8.16E-02	2.0E-04	8.2E-05	2.2E-02	9.0E-03
Zinc (Zn)	6	8.39E-02	2.53E-02	8.4E-05	2.5E-05	1.4E-05	4.2E-06
Cobalt (Co)	0.00069	1.31E-04	6.29E-05	1.3E-07	6.3E-08	1.9E-04	9.1E-05
Chromium (Cr)	0.0013	2.02E-03	6.44E-04	2.0E-06	6.4E-07	1.6E-03	5.0E-04
Mercury (Hg)	0.0006	1.25E-04	6.03E-05	1.3E-07	6.0E-08	2.1E-04	1.0E-04
Lithium (Li)	3.3	1.36E-03	6.54E-04	1.4E-06	6.5E-07	4.1E-07	2.0E-07
Nickel (Ni)	0.0011	1.70E-04	8.14E-05	1.7E-07	8.1E-08	1.5E-04	7.4E-05
						2.7E-02	1.1E-02

Chronic Exposures

Inhalation - $PM_{2.5}$

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

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
Exposure Frequency (EF, days/yr)	365	Days at home, as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	35	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	306600	US EPA 2009

	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
Key Chemical	Inhalation Unit Risk	Chronic TC Air	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background)	Estimated Concentration in Air - Maximum all receptors (Ca)	Inhalation Exposure Concentration - NonThreshold	Inhalation Exposure Concentration - Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/m^3)^{-1}$	(mg/m^3)		(mg/m^3)	(mg/m^3)	(mg/m^3)	(mg/m^3)	(unitless)		(unitless)	
Silver (Ag)		2.0E-02		2.0E-02	1.8E-07	9.0E-08	1.8E-07	--		0.0000090	0%
Lead (Pb)		2.0E-03		2.0E-03	5.8E-06	2.9E-06	5.8E-06	--		0.0029	2%
Arsenic (As)		1.0E-03		1.0E-03	5.9E-07	2.9E-07	5.9E-07	--		0.00059	0%
Cadmium (Cd)		5.0E-06		5.0E-06	3.5E-08	1.8E-08	3.5E-08	--		0.0071	5%
Copper (Cu)		4.9E-01		4.9E-01	5.8E-08	2.9E-08	5.8E-08	--		0.00000012	0%
Manganese (Mn)		1.5E-04		1.5E-04	1.9E-05	9.4E-06	1.9E-05	--		0.13	90%
Zinc (Zn)		1.8E+00		1.8E+00	7.0E-06	3.5E-06	7.0E-06	--		0.0000040	0%
Cobalt (Co)		1.0E-04		1.0E-04	1.1E-08	5.7E-09	1.1E-08	--		0.00011	0%
Chromium (Cr)		1.0E-04		1.0E-04	1.9E-07	9.3E-08	1.9E-07	--		0.0019	1%
Mercury (Hg)		2.0E-04		2.0E-04	9.6E-09	4.8E-09	9.6E-09	--		0.000048	0%
Lithium (Li)		7.0E-03		7.0E-03	1.2E-07	6.1E-08	1.2E-07	--		0.000017	0%
Nickel (Ni)		2.0E-05		2.0E-05	1.2E-08	5.8E-09	1.2E-08	--		0.00058	0%
TOTAL										**0.14**	

	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
Key Chemical	Inhalation Unit Risk	Chronic TC Air	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background)	Estimated Concentration in Air - Maximum private residences (Ca)	Inhalation Exposure Concentration - NonThreshold	Inhalation Exposure Concentration - Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
Silver (Ag)		2.0E-02		2.0E-02	5.8E-09	2.9E-09	5.8E-09	--		0.00000029	0%
Lead (Pb)		2.0E-03		2.0E-03	2.1E-07	1.0E-07	2.1E-07	--		0.00010	1%
Arsenic (As)		1.0E-03		1.0E-03	4.2E-08	2.1E-08	4.2E-08	--		0.000042	0%
Cadmium (Cd)		5.0E-06		5.0E-06	1.5E-09	7.6E-10	1.5E-09	--		0.00031	3%
Copper (Cu)		4.9E-01		4.9E-01	6.3E-09	3.2E-09	6.3E-09	--		0.000000013	0%
Manganese (Mn)		1.5E-04		1.5E-04	1.5E-06	7.6E-07	1.5E-06	--		0.010	94%
Zinc (Zn)		1.8E+00		1.8E+00	3.1E-07	1.6E-07	3.1E-07	--		0.00000018	0%
Cobalt (Co)		1.0E-04		1.0E-04	1.6E-09	7.9E-10	1.6E-09	--		0.000016	0%
Chromium (Cr)		1.0E-04		1.0E-04	6.7E-09	3.3E-09	6.7E-09	--		0.000067	1%
Mercury (Hg)		2.0E-04		2.0E-04	1.7E-09	8.3E-10	1.7E-09	--		0.0000083	0%
Lithium (Li)		7.0E-03		7.0E-03	1.6E-08	8.0E-09	1.6E-08	--		0.0000023	0%
Nickel (Ni)		2.0E-05		2.0E-05	2.2E-09	1.1E-09	2.2E-09	--		0.00011	1%
TOTAL										**0.011**	

Calculation of Concentrations in Soil

$$C_s = \frac{DR \bullet \left[1 - e^{-k \bullet t}\right]}{d \bullet \rho \bullet k} \bullet 1000 \quad \textbf{(mg/kg)}$$ ref: Stevens B. (1991)

where:

DR=	Particle deposition rate for TSP ($mg/m^2/year$)
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
Accumulation time (t)	years	70	70	As per OEHHA (2015) guidance

Chemical-specific Inputs and calculations - maximum private residences

Chemical	**Half-life in soil**	**Loss constant (K)**	**TSP Deposition Rate (DR)**	**Surface Concentration in Soil**	**Agricultural Concentration in Soil**
	years	**per year**	**$mg/m^2/year$**	**mg/kg**	**mg/kg**
Silver (Ag)	273973	2.5E-06	0.0210	9.2E-02	6.1E-03
Lead (Pb)	273973	2.5E-06	0.8760	3.8E+00	2.6E-01
Arsenic (As)	273973	2.5E-06	0.1601	7.0E-01	4.7E-02
Cadmium (Cd)	273973	2.5E-06	0.0062	2.7E-02	1.8E-03
Copper (Cu)	273973	2.5E-06	0.0233	1.0E-01	6.8E-03
Manganese (Mn)	273973	2.5E-06	5.7322	2.5E+01	1.7E+00
Zinc (Zn)	273973	2.5E-06	1.2567	5.5E+00	3.7E-01
Cobalt (Co)	273973	2.5E-06	0.0057	2.5E-02	1.7E-03
Chromium (Cr)	273973	2.5E-06	0.0267	1.2E-01	7.8E-03
Mercury (Hg)	273973	2.5E-06	0.0059	2.6E-02	1.7E-03
Lithium (Li)	273973	2.5E-06	0.0580	2.5E-01	1.7E-02
Nickel (Ni)	273973	2.5E-06	0.0078	3.4E-02	2.3E-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	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2012 and NEPM 2013
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	0.09	2.7E-08	6.5E-08	--		0.000011	1%
Lead (Pb)		6.0E-04		6.0E-04	16%	3.83	1.9E-07	4.5E-07	--		0.00075	54%
Arsenic (As)		2.0E-03		2.0E-03	100%	0.70	2.1E-07	5.0E-07	--		0.00025	18%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	0.03	8.0E-09	1.9E-08	--		0.000024	2%
Copper (Cu)		1.4E-01		1.4E-01	100%	0.10	3.0E-08	7.3E-08	--		0.00000052	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	25.076	7.4E-06	1.8E-05	--		0.00013	9%
Zinc (Zn)		5.0E-01		5.0E-01	100%	5.50	1.6E-06	3.9E-06	--		0.0000079	1%
Cobalt (Co)		1.4E-03		1.4E-03	100%	0.03	7.4E-09	1.8E-08	--		0.000013	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	0.12	3.4E-08	8.3E-08	--		0.000083	6%
Mercury (Hg)		6.0E-04		6.0E-04	100%	0.026	7.7E-09	1.9E-08	--		0.000031	2%
Lithium (Li)		2.0E-03		2.0E-03	100%	0.25	7.5E-08	1.8E-07	--		0.000091	7%
Nickel (Ni)		1.2E-02		1.2E-02	100%	0.034	1.0E-08	2.4E-08	--		0.0000020	0%
								TOTAL			**0.0014**	

Dermal Exposure to Chemicals via Contact with Soil

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

Parameters Relevant to Quantification of Exposure by Adults		
Surface Area (SAs, cm^2)	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/yr)	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 - Private residences

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04		9.2E-02			--		--	
Lead (Pb)		3.0E-04		3.0E-04		3.8E+00			--		--	
Arsenic (As)		2.0E-03		2.0E-03	0.005	7.0E-01	6.5E-08	1.6E-07	--		0.000079	73%
Cadmium (Cd)		8.0E-04		8.0E-04		2.7E-02			--		--	
Copper (Cu)		1.4E-01		1.4E-01		1.0E-01			--		--	
Manganese (Mn)		1.4E-01		1.4E-01		2.5E+01			--		--	
Zinc (Zn)		5.0E-01		5.0E-01	0.001	5.5E+00	1.0E-07	2.5E-07	--		0.00000049	0%
Cobalt (Co)		1.4E-03		1.4E-03	0.001	2.5E-02	4.7E-10	1.1E-09	--		0.00000081	1%
Chromium (Cr)		1.0E-03		1.0E-03		1.2E-01			--		--	
Mercury (Hg)		4.2E-05		4.2E-05	0.001	2.6E-02	4.8E-10	1.2E-09	--		0.000028	26%
Lithium (Li)		2.0E-03		2.0E-03		2.5E-01			--		--	
Nickel (Ni)		1.2E-02		1.2E-02	0.005	3.4E-02	3.2E-09	7.7E-09	--		0.00000064	1%
								TOTAL			**0.00011**	

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 Chilrdren		
Ingestion Rate (IRs, mg/day)	100	Assumed daily soil ingestion rate for young children, enHealth (2012)
Fraction Ingested from Source (FI, unitless)	100%	Compound-specific as noted below
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)
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	0.09	5.2E-08	6.1E-07	--		0.00011	1%
Lead (Pb)		1.4E-03		1.4E-03	16%	3.83	3.6E-07	4.2E-06	--		0.0030	33%
Arsenic (As)		2.0E-03		2.0E-03	100%	0.70	4.0E-07	4.7E-06	--		0.0023	26%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	0.03	1.5E-08	1.8E-07	--		0.00023	3%
Copper (Cu)		1.4E-01		1.4E-01	100%	0.10	5.8E-08	6.8E-07	--		0.000005	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	25.076	1.4E-05	1.7E-04	--		0.0012	13%
Zinc (Zn)		5.0E-01		5.0E-01	100%	5.50	3.1E-06	3.7E-05	--		0.000073	1%
Cobalt (Co)		1.4E-03		1.4E-03	100%	0.03	1.4E-08	1.7E-07	--		0.00012	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	0.12	6.7E-08	7.8E-07	--		0.00078	9%
Mercury (Hg)		6.0E-04		6.0E-04	100%	0.026	1.5E-08	1.7E-07	--		0.00029	3%
Lithium (Li)		2.0E-03		2.0E-03	100%	0.25	1.4E-07	1.7E-06	--		0.00085	9%
Nickel (Ni)		1.2E-02		1.2E-02	100%	0.034	2.0E-08	2.3E-07	--		0.000019	0%
								TOTAL			**0.0090**	

Dermal Exposure to Chemicals via Contact with Soil

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

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

Maximum - Private residences

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04		9.2E-02			--		--	
Lead (Pb)		7.0E-04		7.0E-04		3.8E+00			--		--	
Arsenic (As)		2.0E-03		2.0E-03	0.005	7.0E-01	2.7E-08	3.2E-07	--		0.00016	73%
Cadmium (Cd)		8.0E-04		8.0E-04		2.7E-02			--		--	
Copper (Cu)		1.4E-01		1.4E-01		1.0E-01			--		--	
Manganese (Mn)		1.4E-01		1.4E-01		2.5E+01			--		--	
Zinc (Zn)		5.0E-01		5.0E-01	0.001	5.5E+00	4.2E-08	4.9E-07	--		0.0000010	0%
Cobalt (Co)		1.4E-03		1.4E-03	0.001	2.5E-02	1.9E-10	2.3E-09	--		0.0000016	1%
Chromium (Cr)		1.0E-03		1.0E-03		1.2E-01			--		--	
Mercury (Hg)		4.2E-05		4.2E-05	0.001	2.6E-02	2.0E-10	2.3E-09	--		0.000056	26%
Lithium (Li)		2.0E-03		2.0E-03		2.5E-01			--		--	
Nickel (Ni)		1.2E-02		1.2E-02	0.005	3.4E-02	1.3E-09	1.5E-08	--		0.0000013	1%
								TOTAL			**0.00022**	

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 1 year
DR =	Deposition rate from model for TSP (mg/m^2/year)
Area =	Area of roof (m^2)
VR =	Volume of water collected from roof over year (L) = (R x Area x Rc x 1000)/1000
R =	Rainfall each year (mm)
ρ =	Soil bulk-density (g/cm^3)
Rc =	Runoff coefficient (unitless)
Kd =	Soil-water partition coefficient (cm^3/g)
1000 =	Conversion from mm to m; and conversion from m^3 to L

General Parameters			
Average rainfaill	mm	663.2	mean for all years (1994 - 2019) for Mudgee airport
Roof area	m^2	200	4 bedroom australian home
Runoff coefficient	-	0.7	assumes 30% loss in capture into tank
Volume of rainwater	L	92848	
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 private 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	Drininking water guideline	Proportion of DWG: Particulate	Proportion of DWG: Dissolved
	mg/m^2/year	mg	(cm3/g)	mg/L	mg/L	mg/L		
Silver (Ag)	0.0210	4.2	8.3	4.5E-05	1.1E-05	0.1	0%	0.011%
Lead (Pb)	0.8760	175.2	900	1.9E-03	4.2E-06	0.01	19%	0.042%
Arsenic (As)	0.1601	32.0	29	3.4E-04	2.4E-05	0.01	3%	0.24%
Cadmium (Cd)	0.0062	1.2	75	1.3E-05	3.6E-07	0.002	1%	0.018%
Copper (Cu)	0.0233	4.7	35	5.0E-05	2.9E-06	2	0%	0.000143%
Manganese (Mn)	5.7322	1146.4	65	1.2E-02	3.8E-04	0.5	2%	0.075985%
Zinc (Zn)	1.2567	251.3	62	2.7E-03	8.7E-05	6	0%	0.0015%
Cobalt (Co)	0.0057	1.1	45	1.2E-05	5.5E-07	0.006	0%	0.0092%
Chromium (Cr)	0.0267	5.3	19	5.7E-05	6.0E-06	0.05	0%	0.012%
Mercury (Hg)	0.0059	1.2	52	1.3E-05	4.9E-07	0.001	1%	0.049%
Lithium (Li)	0.0580	11.6	300	1.2E-04	8.3E-07	0.04	0%	0.00208%
Nickel (Ni)	0.0078	1.6	65	1.7E-05	5.2E-07	0.02	0%	0.00260%

Apprach assumes all dust deposited on the roof ends up in the water tank - no first flush diversion

0.02 RSL for tap water from USEPA (2018) as no ADWG available

Exposure to Chemicals via Incidental Ingestion of Water

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate (Irw, L/day)	2	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10950	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	4.5E-05	5.5E-07	1.3E-06	--		0.00023	0%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.9E-03	1.2E-05	2.7E-05	--		0.045	19%
Arsenic (As)		2.0E-03		2.0E-03	100%	3.4E-04	4.2E-06	9.9E-06	--		0.0049	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.3E-05	1.6E-07	3.8E-07	--		0.00048	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	5.0E-05	6.1E-07	1.4E-06	--		0.000010	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1.2E-02	1.5E-04	3.5E-04	--		0.0025	1%
Zinc (Zn)		5.0E-01		5.0E-01	100%	2.7E-03	3.3E-05	7.7E-05	--		0.00015	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.2E-05	1.5E-07	3.5E-07	--		0.00025	0%
Chromium (Cr)		1.0E-03		1.0E-03	100%	5.7E-05	7.0E-07	1.6E-06	--		0.0016	1%
Mercury (Hg)		6.0E-04		6.0E-04	100%	1.3E-05	1.6E-07	3.7E-07	--		0.00061	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	1.2E-04	1.5E-06	3.6E-06	--		0.0018	1%
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.7E-05	2.1E-07	4.8E-07	--		0.000040	0%
								TOTAL			**0.058**	

Dermal Exposure to Chemicals via Contact with Water

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Adults		
Surface Area (Saw, cm2)	20000	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	0.58	Reasonable maximum time spent showering or wet each day (ESEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	30	As per NEPM (1999 amended 2013)
Body Weight (BW, kg)	70	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10950	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	4.51E-05	1.57E-11	1.9E-09	4.5E-09	--		2.0E-05	3%
Lead (Pb)		3.0E-04		3.0E-04	1.00E-4	1.89E-03	1.09E-10	1.3E-08	3.1E-08	--		1.0E-04	15%
Arsenic (As)		2.0E-03		2.0E-03	1.00E-3	3.45E-04	2.00E-10	2.4E-08	5.7E-08	--		2.9E-05	4%
Cadmium (Cd)		8.0E-04		8.0E-04	1.00E-3	1.33E-05	7.74E-12	9.5E-10	2.2E-09	--		2.8E-06	0%
Copper (Cu)		1.4E-01		1.4E-01	1.00E-3	5.02E-05	2.91E-11	3.6E-09	8.3E-09	--		5.9E-08	0%
Manganese (Mn)		1.4E-01		1.4E-01	1.00E-3	1.23E-02	7.16E-09	8.8E-07	2.0E-06	--		1.5E-05	2%
Zinc (Zn)		5.0E-01		5.0E-01	6.00E-4	2.71E-03	9.42E-10	1.2E-07	2.7E-07	--		5.4E-07	0%
Cobalt (Co)		1.4E-03		1.4E-03	4.00E-4	1.24E-05	2.87E-12	3.5E-10	8.2E-10	--		5.9E-07	0%
Chromium (Cr)		1.0E-03		1.0E-03	2.00E-3	5.74E-05	6.66E-11	8.2E-09	1.9E-08	--		1.9E-05	3%
Mercury (Hg)		4.2E-05		4.2E-05	1.00E-3	1.28E-05	7.42E-12	9.1E-10	2.1E-09	--		5.1E-05	7%
Lithium (Li)		2.0E-03		2.0E-03	1.00E-3	1.25E-04	7.24E-11	8.9E-09	2.1E-08	--		1.0E-05	2%
Nickel (Ni)		1.2E-02		1.2E-02	2.00E-4	1.69E-05	1.96E-12	2.4E-10	5.6E-10	--		4.7E-08	0%

	1.7E-04

Exposure to Chemicals via Incidental Ingestion of Water

$$Daily\ Chemical\ Intake_{IW} = C_W \bullet \frac{IR_W \bullet FI \bullet B \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(L/kg/day)}$$

Parameters Relevant to Quantification of Exposure by Children		
Ingestion Rate (Irw, L/day)	0.4	Water intakes from all sources (incl. food and bathing) enHealth 2012
Fraction Ingested from Source	100%	Assumed to be 100%
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Concentration in Water (Cw)	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/L)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	4.5E-05	1.0E-07	1.2E-06	--		0.00021	0%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.9E-03	2.2E-06	2.5E-05	--		0.018	16%
Arsenic (As)		2.0E-03		2.0E-03	100%	3.4E-04	7.9E-07	9.2E-06	--		0.0046	4%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.3E-05	3.0E-08	3.6E-07	--		0.00044	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	5.0E-05	1.1E-07	1.3E-06	--		0.0000096	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1.2E-02	2.8E-05	3.3E-04	--		0.0024	2%
Zinc (Zn)		5.0E-01		5.0E-01	100%	2.7E-03	6.2E-06	7.2E-05	--		0.00014	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.2E-05	2.8E-08	3.3E-07	--		0.00024	0%
Chromium (Cr)		1.0E-03		1.0E-03	100%	5.7E-05	1.3E-07	1.5E-06	--		0.0015	1%
Mercury (Hg)		6.0E-04		6.0E-04	100%	1.3E-05	2.9E-08	3.4E-07	--		0.00057	1%
Lithium (Li)		2.0E-03		2.0E-03	100%	1.2E-04	2.9E-07	3.3E-06	--		0.0017	2%
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.7E-05	3.9E-08	4.5E-07	--		0.000038	0%
								TOTAL			**0.030**	

Dermal Exposure to Chemicals via Contact with Water

$$DA_{event} = K_p \times C_w \times t_{event}$$

mg/cm2 per event (for inorganics)

$$DAD = \frac{DA_{event} \times EV \times ED \times EF \times SA}{BW \times AT}$$

mg/kg bw/day

Parameters Relevant to Quantification of Exposure to Children		
Surface Area (Saw, cm2)	6100	Whole body as per enHealth (2012)
Exposure Time per event (tevent, hr/event)	1	Reasonable maximum time spent showering or wet each day (ESEPA)
Conversion Factor (CF, L/cm3)	1.E-03	Conversion of units
Dermal Permeability (cm/hr)	Chemical-specific (as below)	
Event Frequency (EV, events/day)	1	Assumed relevant to exposure being evaluated
Exposure Frequency (EF, days/yr)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	US EPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	US EPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data					Concentration in Water (Cw)	DAevent	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Permeability (Kp)			Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	4.51E-05	2.71E-11	9.4E-10	1.1E-08	--		4.8E-05	5%
Lead (Pb)		7.0E-04		7.0E-04	1.00E-4	1.89E-03	1.89E-10	6.6E-09	7.7E-08	--		1.1E-04	11%
Arsenic (As)		2.0E-03		2.0E-03	1.00E-3	3.45E-04	3.45E-10	1.2E-08	1.4E-07	--		7.0E-05	7%
Cadmium (Cd)		8.0E-04		8.0E-04	1.00E-3	1.33E-05	1.33E-11	4.6E-10	5.4E-09	--		6.8E-06	1%
Copper (Cu)		1.4E-01		1.4E-01	1.00E-3	5.02E-05	5.02E-11	1.8E-09	2.0E-08	--		1.5E-07	0%
Manganese (Mn)		1.4E-01		1.4E-01	1.00E-3	1.23E-02	1.23E-08	4.3E-07	5.0E-06	--		3.6E-05	3%
Zinc (Zn)		5.0E-01		5.0E-01	6.00E-4	2.71E-03	1.62E-09	5.7E-08	6.6E-07	--		1.3E-06	0%
Cobalt (Co)		1.4E-03		1.4E-03	4.00E-4	1.24E-05	4.95E-12	1.7E-10	2.0E-09	--		1.4E-06	0%
Chromium (Cr)		1.0E-03		1.0E-03	2.00E-3	5.74E-05	1.15E-10	4.0E-09	4.7E-08	--		4.7E-05	4%
Mercury (Hg)		4.2E-05		4.2E-05	1.00E-3	1.28E-05	1.28E-11	4.5E-10	5.2E-09	--		1.2E-04	12%
Lithium (Li)		2.0E-03		2.0E-03	1.00E-3	1.25E-04	1.25E-10	4.4E-09	5.1E-08	--		2.5E-05	2%
Nickel (Ni)		1.2E-02		1.2E-02	2.00E-4	1.69E-05	3.38E-12	1.2E-10	1.4E-09	--		1.1E-07	0%
												2.7E-04	

Calculation of Concentrations in Plants

ref: Stevens B. (1991)

Uptake Due to Deposition in Aboveground Crops

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

where:
DR= Particle deposition rate for TSP (mg/m^2/day)
F= Fraction for the surface area of plant (unitless)
k= Chemical-specific soil-loss constant (1/years) = ln(2)/$T_{0.5}$
$T_{0.5}$= Chemical half-life as particulate on plant (days)
t= Deposition time (days)
Y= Crop yield (kg/m^2)

Uptake via Roots from Soil

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

where:
Cs = Concentration of persistent chemical in soil assuming 15cm mixing depth within gardens, calculated using Soil Equation for each chemical assessed (mg/kg)
RUF = Root uptake factor which differs for each Chemical (unitless)

General Parameters	Units	Value
Crop		Edible crops
Crop Yield (Y)	kg/m^2	2
Deposition Time (t)	days	70
Plant Interception fraction (F)	unitless	0.051

Chemical-specific Inputs and calculations - Maximum private residences

Chemical	Half-life in plant ($T_{0.5}$)	Loss constant (k)	Deposition Rate TSP (DR)	Aboveground Produce Concentration via Deposition	Root Uptake Factor (RUF)	Soil Concentration (Cs)	Below Ground Produce Concentration
	days	per day	mg/m^2/day	mg/kg ww	unitless	mg/kg	mg/kg ww
Silver (Ag)	14	0.05	0.0000574	2.9E-05	0.1	6.1E-03	6.1E-04
Lead (Pb)	14	0.05	0.0023999	1.2E-03	0.011	2.6E-01	2.8E-03
Cadmium (Cd)	14	0.05	0.0000170	8.5E-06	0.125	1.8E-03	2.3E-04
Copper (Cu)	14	0.05	0.0000639	3.2E-05	0.1	6.8E-03	6.8E-04
Manganese (Mn)	14	0.05	0.0157047	7.8E-03	0.0625	1.7E+00	1.0E-01
Zinc (Zn)	14	0.05	0.0034430	1.7E-03	0.0113	3.7E-01	4.1E-03
Cobalt (Co)	14	0.05	0.0000157	7.9E-06	0.005	1.7E-03	8.4E-06
Chromium (Cr)	14	0.05	0.0000730	3.6E-05	0.00188	7.8E-03	1.5E-05
Mercury (Hg)	14	0.05	0.0000163	8.1E-06	0.225	1.7E-03	3.9E-04
Lithium (Li)	14	0.05	0.0001588	7.9E-05	0.00625	1.7E-02	1.1E-04
Nickel (Ni)	14	0.05	0.0000215	1.1E-05	0.015	2.3E-03	3.4E-05

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

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 (%)	35%	Assumed for rural areas (higher than typical default)
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)				NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	2.9E-05	6.1E-04	1.5E-07	3.7E-07	--		6.5E-05	2%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.2E-03	2.8E-03	6.8E-07	1.6E-06	--		2.7E-03	67%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.2E-04	4.7E-04	5.6E-08	1.3E-07	--		6.7E-05	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	8.5E-06	2.3E-04	5.6E-08	1.3E-07	--		1.7E-04	4%
Copper (Cu)		1.4E-01		1.4E-01	100%	3.2E-05	6.8E-04	1.7E-07	4.1E-07	--		3.0E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	7.8E-03	1.0E-01	2.8E-05	6.8E-05	--		4.8E-04	12%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.7E-03	4.1E-03	2.0E-06	4.7E-06	--		9.5E-06	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	7.9E-06	8.4E-06	6.6E-09	1.6E-08	--		1.1E-05	0%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.6E-05	1.5E-05	2.5E-08	6.1E-08	--		6.1E-05	2%
Mercury (Hg)		6.0E-04		6.0E-04	100%	8.1E-06	3.9E-04	9.2E-08	2.2E-07	--		3.7E-04	9%
Lithium (Li)		2.0E-03		2.0E-03	100%	7.9E-05	1.1E-04	7.2E-08	1.7E-07	--		8.6E-05	2%
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.1E-05	3.4E-05	1.4E-08	3.4E-08	--		2.8E-06	0%
									TOTAL			**0.0041**	

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 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 (%)	35%	Assumed for rural areas (higher than typical default)
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)				NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	2.9E-05	6.1E-04	6.8E-08	8.0E-07	--		1.4E-04	2%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.2E-03	2.8E-03	4.1E-07	4.8E-06	--		3.4E-03	53%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.2E-04	4.7E-04	2.4E-08	2.8E-07	--		1.4E-04	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	8.5E-06	2.3E-04	2.4E-08	2.8E-07	--		3.5E-04	5%
Copper (Cu)		1.4E-01		1.4E-01	100%	3.2E-05	6.8E-04	7.6E-08	8.9E-07	--		6.3E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	7.8E-03	1.0E-01	1.3E-05	1.5E-04	--		1.1E-03	17%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.7E-03	4.1E-03	1.2E-06	1.4E-05	--		2.8E-05	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	7.9E-06	8.4E-06	4.4E-09	5.2E-08	--		3.7E-05	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.6E-05	1.5E-05	1.8E-08	2.2E-07	--		2.2E-04	3%
Mercury (Hg)		6.0E-04		6.0E-04	100%	8.1E-06	3.9E-04	3.9E-08	4.5E-07	--		7.5E-04	12%
Lithium (Li)		2.0E-03		2.0E-03	100%	7.9E-05	1.1E-04	4.7E-08	5.5E-07	--		2.7E-04	4%
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.1E-05	3.4E-05	8.1E-09	9.5E-08	--		7.9E-06	0%
							TOTAL					**0.0064**	

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 100% of crops consumed by chickens is grown in the same soil
IRc (ingestion rate of crops)	kg/day	0.12	Assumed ingestion rate from OEHHA 2015 (assume concentration the same as predicted for aboveground crops)
IRs (ingestion rate of soil)	kg/day	0.0024	Based on data from OEHHA 2015 (2% total produce intakes from soil)
B (bioavailability)	%	100%	Assumed to be 100% except for lead

Chemical-specific Inputs and calculations - Maximum private residences

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 (Ag)	2.9E-05	6.1E-03	3.8E-02	6.9E-07	
Lead (Pb)	1.2E-03	2.6E-01	4.0E-02	1.4E-05	
Arsenic (As)	2.2E-04	4.7E-02	7.0E-02	9.7E-06	
Cadmium (Cd)	8.5E-06	1.8E-03	1.0E-02	5.4E-08	
Copper (Cu)	3.2E-05	6.8E-03	3.8E-02	7.7E-07	
Manganese (Mn)	7.8E-03	1.7E+00	3.8E-02	1.9E-04	
Zinc (Zn)	1.7E-03	3.7E-01	3.8E-02	4.1E-05	
Cobalt (Co)	7.9E-06	1.7E-03	3.8E-02	1.9E-07	
Chromium (Cr)	3.6E-05	7.8E-03	9.2E-03	2.1E-07	OEHHA (2003)
Mercury (Hg)	8.1E-06	1.7E-03	8.0E-01	4.1E-06	
Lithium (Li)	7.9E-05	1.7E-02	3.8E-02	1.9E-06	
Nickel (Ni)	1.1E-05	2.3E-03	2.0E-02	1.4E-07	

Transfer factors from OEHHA 2015 unless otherwise noted
Mean transfer factor for heavy metals used in absense of specific data (Leeman et al 2007)

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 (%)	200%	Assumed for rural areas where a higher rate of egg ingestion expected
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	6.9E-07	1.1E-10	2.8E-10	--		4.8E-08	0%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.4E-05	1.1E-09	2.8E-09	--		4.6E-06	44%
Arsenic (As)		2.0E-03		2.0E-03	100%	9.7E-06	1.6E-09	3.9E-09	--		1.9E-06	19%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	5.4E-08	8.9E-12	2.1E-11	--		2.7E-08	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	7.7E-07	1.3E-10	3.1E-10	--		2.2E-09	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1.9E-04	3.1E-08	7.5E-08	--		5.4E-07	5%
Zinc (Zn)		5.0E-01		5.0E-01	100%	4.1E-05	6.8E-09	1.7E-08	--		3.3E-08	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.9E-07	3.1E-11	7.5E-11	--		5.4E-08	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	2.1E-07	3.5E-11	8.5E-11	--		8.5E-08	1%
Mercury (Hg)		6.0E-04		6.0E-04	100%	4.1E-06	6.8E-10	1.6E-09	--		2.7E-06	26%
Lithium (Li)		2.0E-03		2.0E-03	100%	1.9E-06	3.2E-10	7.6E-10	--		3.8E-07	4%
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.4E-07	2.2E-11	5.4E-11	--		4.5E-09	0%
								TOTAL			**0.000010**	

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 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 (%)	200%	Assumed for rural areas where a higher rate of egg ingestion expected
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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	6.9E-07	4.7E-11	5.5E-10	--		9.6E-08	1%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.4E-05	4.7E-10	5.5E-09	--		3.9E-06	25%
Arsenic (As)		2.0E-03		2.0E-03	100%	9.7E-06	6.6E-10	7.7E-09	--		3.9E-06	25%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	5.4E-08	3.7E-12	4.3E-11	--		5.4E-08	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	7.7E-07	5.2E-11	6.1E-10	--		4.4E-09	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	1.9E-04	1.3E-08	1.5E-07	--		1.1E-06	7%
Zinc (Zn)		5.0E-01		5.0E-01	100%	4.1E-05	2.8E-09	3.3E-08	--		6.6E-08	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.9E-07	1.3E-11	1.5E-10	--		1.1E-07	1%
Chromium (Cr)		1.0E-03		1.0E-03	100%	2.1E-07	1.5E-11	1.7E-10	--		1.7E-07	1%
Mercury (Hg)		6.0E-04		6.0E-04	100%	4.1E-06	2.8E-10	3.3E-09	--		5.5E-06	35%
Lithium (Li)		2.0E-03		2.0E-03	100%	1.9E-06	1.3E-10	1.5E-09	--		7.6E-07	5%
Nickel (Ni)		1.2E-02		1.2E-02	100%	1.4E-07	9.3E-12	1.1E-10	--		9.0E-09	0%
								TOTAL			**0.000016**	

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%	Assumed to be 100% except for lead

Chemical-specific Inputs and calculations - maximum private residences

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 (Ag)	2.9E-05	6.1E-03	3.0E-03	9.0E-06	RAIS
Lead (Pb)	1.2E-03	2.6E-01	3.0E-04	1.5E-05	
Arsenic (As)	2.2E-04	4.7E-02	2.0E-03	4.6E-05	
Cadmium (Cd)	8.5E-06	1.8E-03	2.0E-04	1.8E-07	
Copper (Cu)	3.2E-05	6.8E-03	1.0E-02	3.3E-05	RAIS
Manganese (Mn)	7.8E-03	1.7E+00	4.0E-04	3.3E-04	RAIS
Zinc (Zn)	1.7E-03	3.7E-01	1.0E-01	1.8E-02	RAIS
Cobalt (Co)	7.9E-06	1.7E-03	2.0E-02	1.6E-05	RAIS
Chromium (Cr)	3.6E-05	7.8E-03	9.2E-03	3.5E-05	OEHHA (2003)
Mercury (Hg)	8.1E-06	1.7E-03	4.0E-04	3.4E-07	
Lithium (Li)	7.9E-05	1.7E-02	1.0E-02	8.3E-05	RAIS
Nickel (Ni)	1.1E-05	2.3E-03	3.0E-04	3.4E-07	

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 - Private residences

Key Chemical	Toxicity Data					Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Bioavailability (%)		NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	9.0E-06	3.0E-09	7.2E-09	--		1.3E-06	1%
Lead (Pb)		6.0E-04		6.0E-04	50%	1.5E-05	2.4E-09	5.8E-09	--		9.7E-06	7%
Arsenic (As)		2.0E-03		2.0E-03	100%	4.6E-05	1.5E-08	3.7E-08	--		1.8E-05	14%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.8E-07	5.9E-11	1.4E-10	--		1.8E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	3.3E-05	1.1E-08	2.7E-08	--		1.9E-07	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	3.3E-04	1.1E-07	2.6E-07	--		1.9E-06	1%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.8E-02	6.0E-06	1.4E-05	--		2.9E-05	22%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.6E-05	5.5E-09	1.3E-08	--		9.4E-06	7%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.5E-05	1.2E-08	2.8E-08	--		2.8E-05	21%
Mercury (Hg)		6.0E-04		6.0E-04	100%	3.4E-07	1.1E-10	2.7E-10	--		4.5E-07	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	8.3E-05	2.8E-08	6.7E-08	--		3.3E-05	25%
Nickel (Ni)		1.2E-02		1.2E-02	100%	3.4E-07	1.1E-10	2.7E-10	--		2.3E-08	0%
								TOTAL			**0.00013**	

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 - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	9.0E-06	1.5E-09	1.8E-08	--		3.1E-06	1%
Lead (Pb)		1.4E-03		1.4E-03	50%	1.5E-05	1.2E-09	1.4E-08	--		1.0E-05	3%
Arsenic (As)		2.0E-03		2.0E-03	100%	4.6E-05	7.8E-09	9.1E-08	--		4.6E-05	15%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.8E-07	3.0E-11	3.5E-10	--		4.4E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	3.3E-05	5.7E-09	6.6E-08	--		4.7E-07	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	3.3E-04	5.6E-08	6.5E-07	--		4.7E-06	1%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.8E-02	3.1E-06	3.6E-05	--		7.2E-05	23%
Cobalt (Co)		1.4E-03		1.4E-03	100%	1.6E-05	2.8E-09	3.3E-08	--		2.3E-05	7%
Chromium (Cr)		1.0E-03		1.0E-03	100%	3.5E-05	6.0E-09	7.0E-08	--		7.0E-05	22%
Mercury (Hg)		6.0E-04		6.0E-04	100%	3.4E-07	5.8E-11	6.8E-10	--		1.1E-06	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	8.3E-05	1.4E-08	1.7E-07	--		8.3E-05	26%
Nickel (Ni)		1.2E-02		1.2E-02	100%	3.4E-07	5.7E-11	6.7E-10	--		5.6E-08	0%
								TOTAL			**0.00031**	

Calculation of Concentrations in Dairy Milk

Uptake in to milk (dairy cows)

$$C_E=(FI \times IR_C \times C+IR_S \times C_S \times B) \times TF_B \quad \text{(mg/L)}$$

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/L)

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%	Assumed to be 100% except for lead

Chemical-specific Inputs and calculations - Maximum private residences

Chemical	Concentration in crops ingested by cattle mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to milk day/L	Milk Concentration mg/L	
Silver (Ag)	2.9E-05	6.1E-03	5.0E-03	3.7E-05	Median transfer factor for metals (Leeman et al 2007)
Lead (Pb)	1.2E-03	2.6E-01	6.0E-05	7.1E-06	
Cadmium (Cd)	8.5E-06	1.8E-03	5.0E-06	1.1E-08	
Copper (Cu)	3.2E-05	6.8E-03	1.5E-03	1.2E-05	RAIS
Manganese (Mn)	7.8E-03	1.7E+00	3.5E-04	7.0E-04	RAIS
Zinc (Zn)	1.7E-03	3.7E-01	2.7E-09	1.2E-09	RAIS
Cobalt (Co)	7.9E-06	1.7E-03	2.0E-03	4.0E-06	RAIS
Chromium (Cr)	3.6E-05	7.8E-03	9.0E-06	8.4E-08	
Mercury (Hg)	8.1E-06	1.7E-03	7.0E-05	1.5E-07	
Lithium (Li)	7.9E-05	1.7E-02	5.0E-03	1.0E-04	Median transfer factor for metals (Leeman et al 2007)
Nickel (Ni)	1.1E-05	2.3E-03	3.0E-05	8.3E-08	

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Milk

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

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Milk (IRM) (kg/day)	1.295	Ingestion rate of cows milk for adults (P90 value from FSANZ 2017)
Fraction ingested that is homegrown (%)	100%	Assume all milk consumed is from the dairy farm
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Silver (Ag)		5.7E-03		5.7E-03	100%	3.7E-05	2.8E-07	6.8E-07	--		1.2E-04	9%
Lead (Pb)		6.0E-04		6.0E-04	50%	7.1E-06	2.7E-08	6.6E-08	--		1.1E-04	8%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.8E-06	2.2E-08	5.2E-08	--		2.6E-05	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.1E-08	8.3E-11	2.0E-10	--		2.5E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	1.2E-05	9.4E-08	2.3E-07	--		1.6E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	7.0E-04	5.4E-06	1.3E-05	--		9.3E-05	7%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.2E-09	9.1E-12	2.2E-11	--		4.4E-11	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	4.0E-06	3.1E-08	7.5E-08	--		5.3E-05	4%
Chromium (Cr)		1.0E-03		1.0E-03	100%	8.4E-08	6.5E-10	1.6E-09	--		1.6E-06	0%
Mercury (Hg)		6.0E-04		6.0E-04	100%	1.5E-07	1.1E-09	2.7E-09	--		4.5E-06	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	1.0E-04	7.8E-07	1.9E-06	--		9.4E-04	70%
Nickel (Ni)		1.2E-02		1.2E-02	100%	8.3E-08	6.3E-10	1.5E-09	--		1.3E-07	0%
								TOTAL			**0.0013**	

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) (kg/day)	1.097	Ingestion rate of cows milk for children aged 2-6 years (P90 value from FSANZ 2017)
Fraction ingested that is homegrown (%)	100%	Assume all milk consumed is from the dairy farm
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum - Private residences

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Silver (Ag)		5.7E-03		5.7E-03	100%	3.7E-05	2.3E-07	2.7E-06	--		4.7E-04	9%
Lead (Pb)		1.4E-03		1.4E-03	50%	7.1E-06	2.2E-08	2.6E-07	--		1.9E-04	4%
Arsenic (As)		2.0E-03		2.0E-03	100%	2.8E-06	1.8E-08	2.1E-07	--		1.0E-04	2%
Cadmium (Cd)		8.0E-04		8.0E-04	100%	1.1E-08	6.8E-11	7.9E-10	--		9.9E-07	0%
Copper (Cu)		1.4E-01		1.4E-01	100%	1.2E-05	7.7E-08	9.0E-07	--		6.4E-06	0%
Manganese (Mn)		1.4E-01		1.4E-01	100%	7.0E-04	4.4E-06	5.1E-05	--		3.7E-04	7%
Zinc (Zn)		5.0E-01		5.0E-01	100%	1.2E-09	7.4E-12	8.6E-11	--		1.7E-10	0%
Cobalt (Co)		1.4E-03		1.4E-03	100%	4.0E-06	2.5E-08	2.9E-07	--		2.1E-04	4%
Chromium (Cr)		1.0E-03		1.0E-03	100%	8.4E-08	5.3E-10	6.2E-09	--		6.2E-06	0%
Mercury (Hg)		6.0E-04		6.0E-04	100%	1.5E-07	9.1E-10	1.1E-08	--		1.8E-05	0%
Lithium (Li)		2.0E-03		2.0E-03	100%	1.0E-04	6.4E-07	7.4E-06	--		3.7E-03	73%
Nickel (Ni)		1.2E-02		1.2E-02	100%	8.3E-08	5.2E-10	6.0E-09	--		5.0E-07	0%
								TOTAL			**0.0051**	

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Annexure H

Noise Impact Figures for all Scenarios

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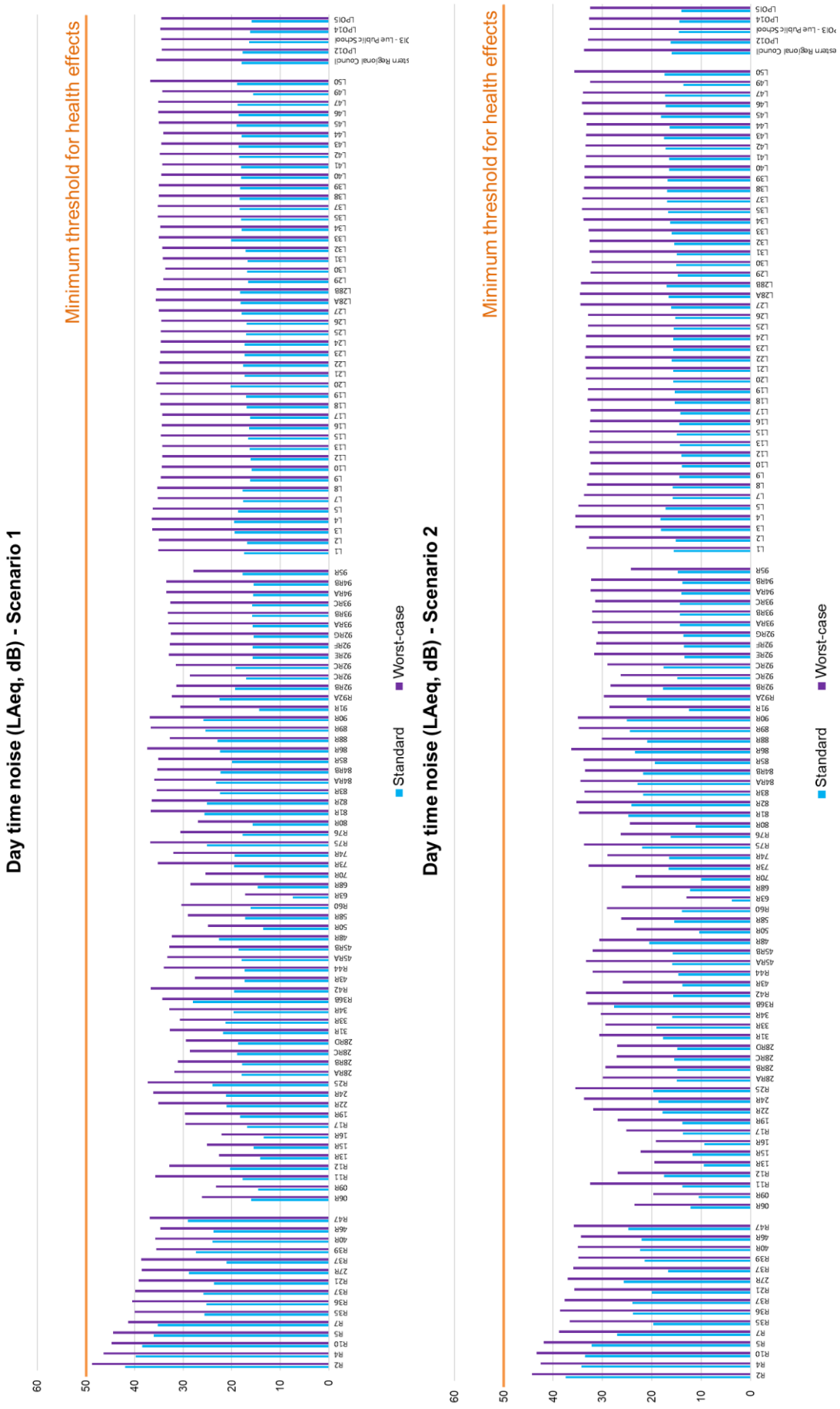
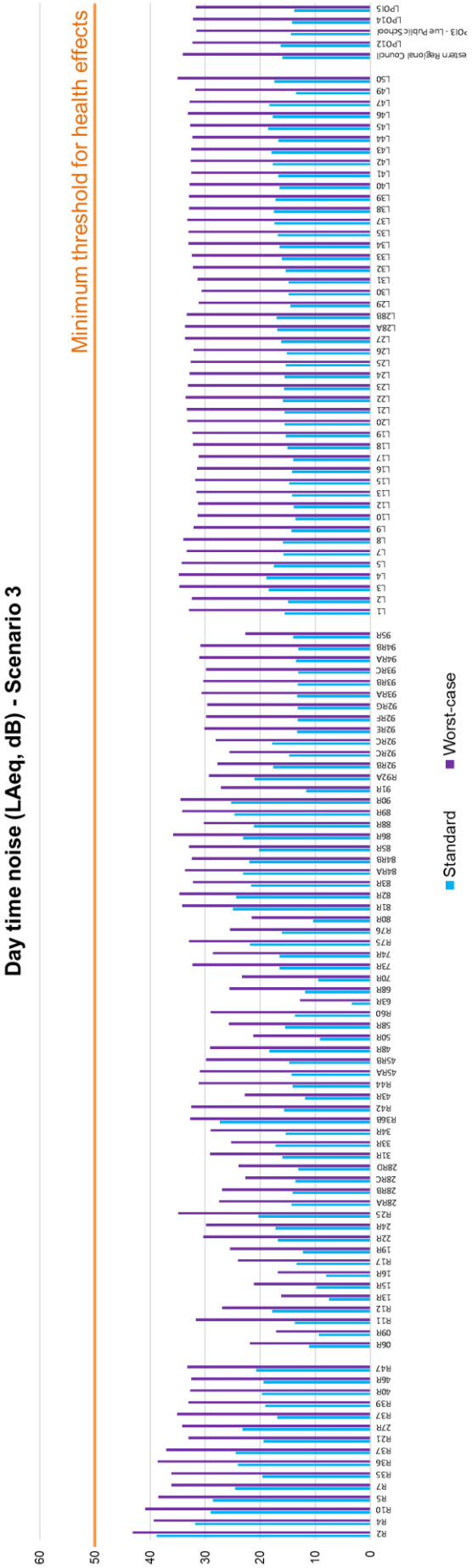
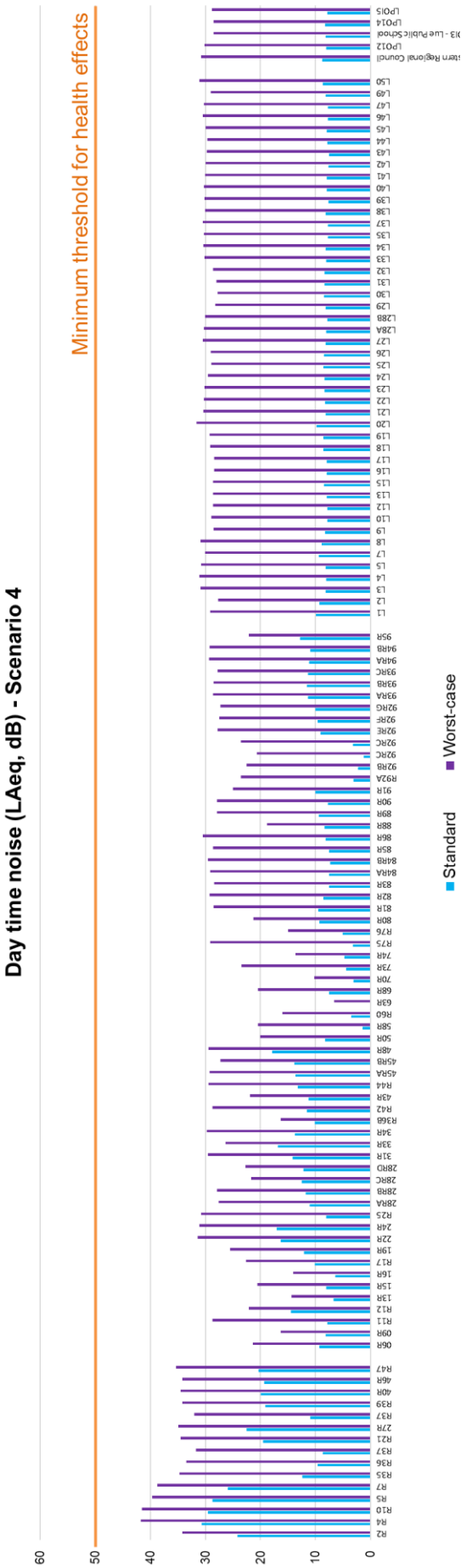
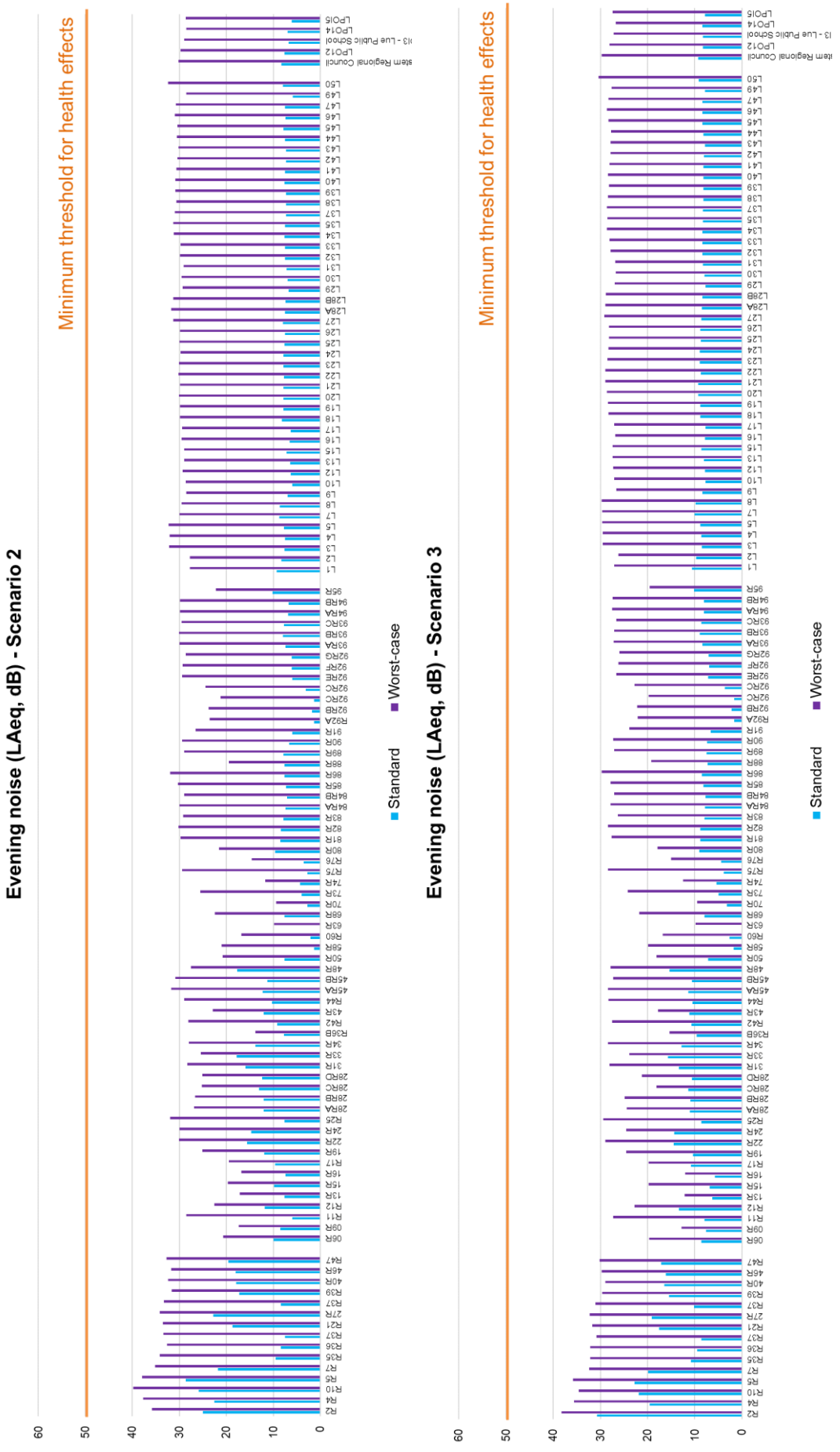
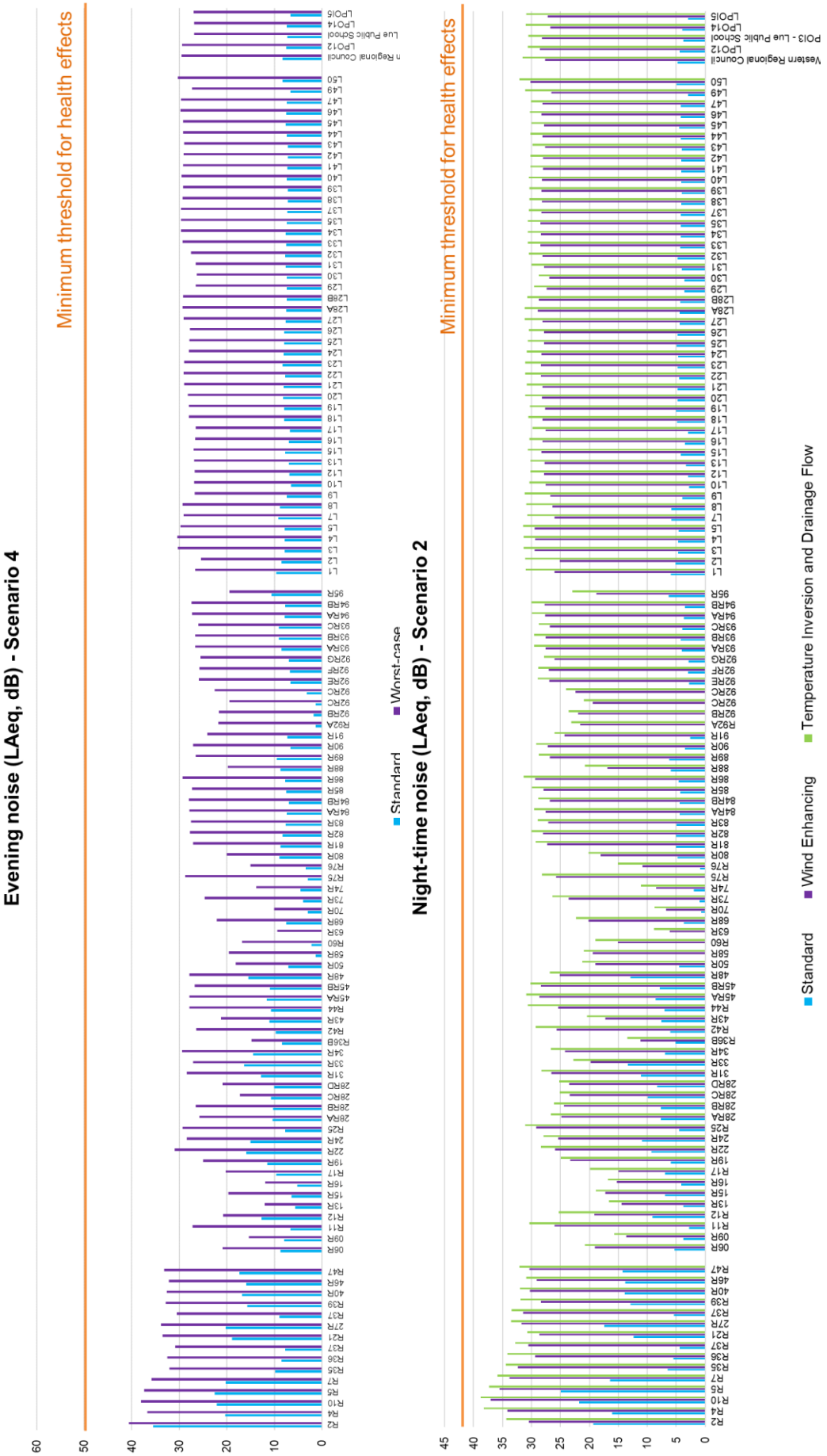
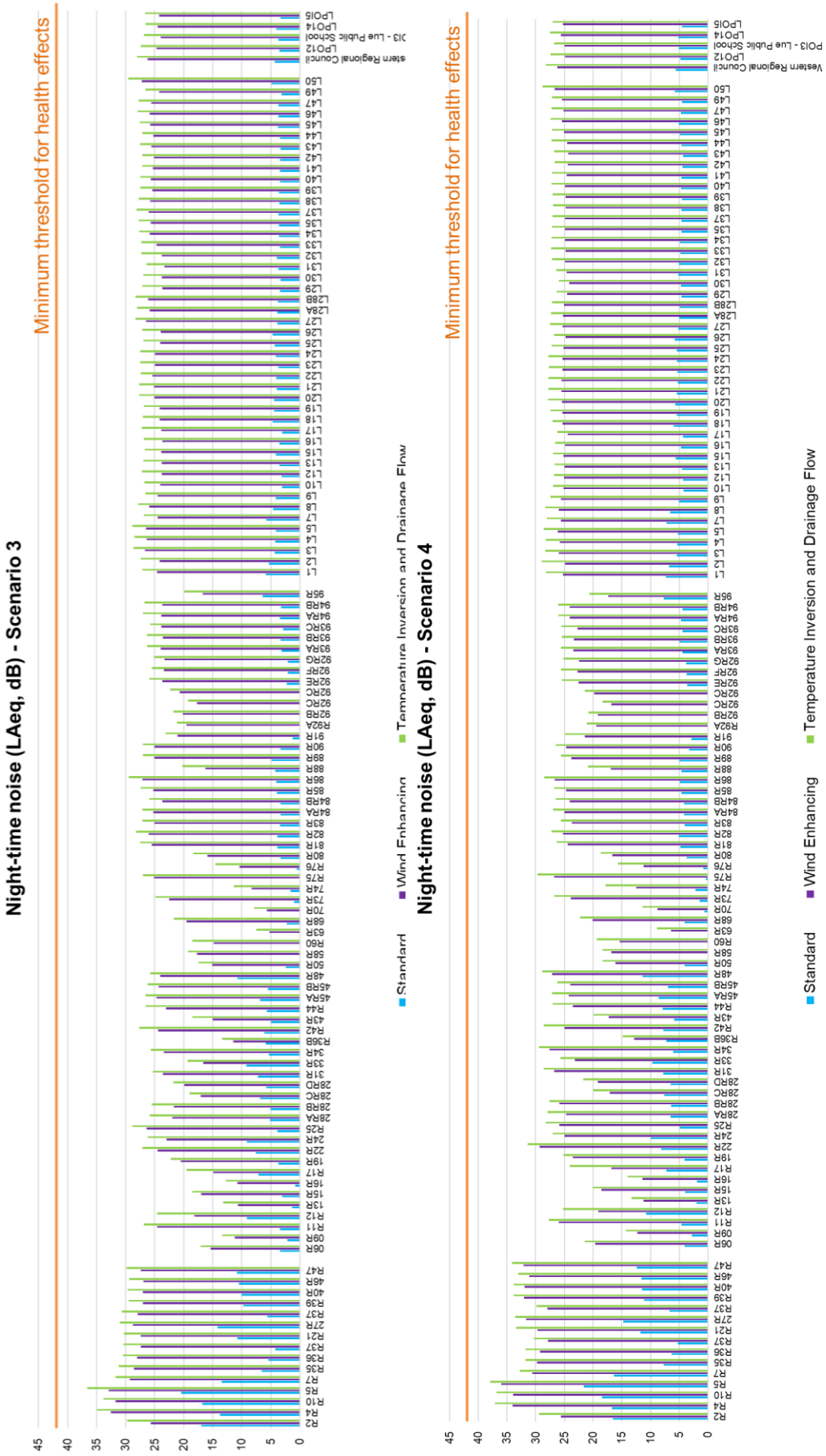

Day time noise (LAeq, dB) - Scenario 1
Minimum threshold for health effects
Standard
Worst-case
Day time noise (LAeq, dB) - Scenario 2
Minimum threshold for health effects
Standard
Worst-case


Day time noise (LAeq, dB) - Scenario 3
Minimum threshold for health effects
Worst-case
Standard
0
10
20
30
40
50
60


Day time noise (LAeq, dB) - Scenario 4
Minimum threshold for health effects
Worst-case
Standard
0
10
20
30
40
50
60


Evening noise (LAeq, dB) - Scenario 2
Minimum threshold for health effects
Standard
Worst-case
Evening noise (LAeq, dB) - Scenario 3
Minimum threshold for health effects
Standard
Worst-case


Evening noise (LAeq, dB) - Scenario 4
Minimum threshold for health effects
Standard
Worst-case
Night-time noise (LAeq, dB) - Scenario 2
Minimum threshold for health effects
Western Regional Council
LPO12
PO13 - Lue Public School
LPO14
LPO15
Standard
Wind Enhancing
Temperature Inversion and Drainage Flow


Night-time noise (LAeq, dB) - Scenario 3
Minimum threshold for health effects
Standard
Wind Enhancing
Temperature Inversion and Drainage Flow
Night-time noise (LAeq, dB) - Scenario 4
Minimum threshold for health effects
Standard
Wind Enhancing
Temperature Inversion and Drainage Flow

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Annexure I

Peer Review
Prepared by Brian G. Priestly (Environmental Risk Sciences)

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PRIESTLY TOXICOLOGY CONSULTING
ABN 62 201 868 314

8B Sturt Ave
Toorak Gardens
SA 5065

Phone: 0413 607 285
e-mail: brianpriestly8@gmail.com

Peer Review
EnRiskS Technical Report

Report reference: BSM/17/HHRA001
Draft - November 2019
Final – January 2020

Human Health Risk Assessment

Bowdens Silver Pty Ltd

Bowdens Silver Project
Report No. 429/25
Specialist Consultant Studies
Part 7: Human Health Risk Assessment

Brian G. Priestly M.Pharm, PhD, FACTRA

Draft prepared 11 December 2019
Final version, addressing comments received, completed 8 January 2020

Executive Summary

This peer review report has been prepared as part of the Environmental Impact Statement (EIS) process relating to the proposed development of an open cut silver mine near Lue (NSW) by Bowdens Silver Pty Limited. It primarily considers the approach taken by Environmental Risk Sciences (EnRiskS) in its Human Health Risk Assessment (HHRA) report on the project.

The EnRiskS HHRA report addresses potential health impacts on nearby communities associated with the planned mining operations in both the site establishment and construction and operational phases of the Project. It is focussed primarily on potential changes in air and water quality, noise impacts and health risks associated with the more significant components of generated dusts (metals, including lead, silica, and particulates) and airborne hydrogen cyanide emissions.

In summary of my peer review, I find the EnRiskS report to be a thorough, professional and well-presented review of the potential **incremental** health risks associated with the proposed development. It generally follows prescribed national (enHealth, NHMRC, NEPC & NEPM) and NSW guidance on how the HHRA component of an EIS is to be structured. It focusses on the likely exposure pathways by which residents of local communities may be exposed to airborne dusts and metals associated with current activities in the region and estimates the extent to which the proposed mine operations may result in incremental risks for adverse health effects, over and above the health risks associated with current exposures to these elements.

The EnRiskS HHRA report has been comprehensive and systematic in reviewing the geological characteristics of the region, as well as the geographic relationship of the proposed mine with nearby residential areas and the demographics of the region. Particular attention has been paid to the township of Lue where there has been a history of community concerns raised about the Project.

The overall outcomes of the risk assessments are summarised at the end of each section, with an overall summary in Table 8.1 in relation to air and water quality, and noise impacts, along with the recommended risk mitigation strategies.

The main conclusions from the EnRiskS HHRA report

- for dust-borne risks are that "*intakes of metals by all members of the public are dominated by existing exposures to metals in the environment and … dust emissions from the Project would make a negligible contribution to these intakes and there would be no Project-related exposures that are considered to result in any health impacts for any member of the community*"
- for water-borne risks, are that "*the assessment has not identified any impacts to groundwater or surface water that would have the potential to adversely affect community health. The quantity and quality of water that may be accessed by the community is not expected to be significantly affected by Project activities*"
- for the health impacts of noise, are that "*the assessment of noise impacts from the Project has not identified noise levels within the community that exceed health-based WHO guidelines for the protection of health, during the day, evening or night"*

I find that these overall conclusions of the report are measured and reasonable and I can find no reason to challenge any of them.

1. Introduction

The purpose of this peer review report is primarily to consider the approach taken by Environmental Risk Sciences (EnRiskS) in its Human Health Risk Assessment (HHRA) report relating to the proposed development and operation of an open cut silver mine near Lue (NSW) by Bowdens Silver Pty Limited. The EnRiskS HHRA report has been prepared as a component of the suite of Environmental Impact Statement (EIS) reports mandated under Section 4.12(8) of the NSW *Environmental Planning and Assessment Act 1979*, and Schedule 2 of the *Environmental Planning and Assessment regulation 2000* to meet the Secretary's Environmental Assessment Requirements (SEARs) for this State Significant Development.

It addresses potential health impacts on nearby communities associated with the planned mining operations in both the site establishment and construction and operational phases of the Project. It is focussed primarily on potential changes in air and water quality, noise impacts and health risks associated with the more significant components of generated dusts (metals, including lead, silica, and particulates) and airborne hydrogen cyanide emissions. The EnRisk Report is part of a larger suite of technical reports prepared by various consultants that address issues relating to traffic management, air and water quality, noise impacts, including blasting operations at the mine, social and economic impacts and other environmental issues. My peer review report only addresses the HHRA aspects of the EIS, as contained in the EnRiskS report (Specialist Consultant Studies Compendium, Part 7).

The EnRiskS report includes a summary of the relevant legislative requirements for an EIS relating to such a major development project and includes citations of the various ambient air, water, soil and noise impact guidance against which the potential health impacts are measured.

2. Peer review process

Priestly Toxicology Consulting was engaged, in May 2018, to peer review HHRA reports prepared by EnRiskS for RW Corkery & Co. Pty Ltd, acting for Bowdens Silver Pty Ltd to co-ordinate the health-risk components of the EIS. The expertise and experience of the peer reviewer is outlined in Appendix A.

The documents initially provided to scope the project included:

1. A copy of the Preliminary Environmental Assessment for the Bowdens Silver Project.
2. A tabulated summary of the lead/health-related SEARs requirements provided by DPE (Table A3.1), Greater Western Area Health Service and Mid-Western Regional Council (Table A3.2) and the local community (including the Lue Action Group) (Table A3.2).
3. A copy of correspondence from the Greater Western Area Health Service – now known as the Western NSW Local Health District.
4. A copy of the Scope of Work provided to EnRiskS for the assessment.

Initially, the plan was to undertake the peer review in three stages:

Stage 1 – a review of the existing Constraints Report and the proposed approach to the overall assessment of the Project (target date 31 May 2018)
Stage 2 – a review of the Draft Assessment Report (target date 10 August 2018)
Stage 3 – a review of the Final Assessment Report (target date 10 September 2018)

Ultimately, due to delays in preparation of the HHRA reports, the staged approach was abandoned, and a single peer review was prepared for a draft final HHRA report dated November 2019.

A draft peer review report, dated 11 December 2019, was prepared following a review of the draft November 2019 EnRiskS report. It did not require a site visit, nor engagement with other specialists working on the Project. The peer review report was completed on 8 January 2020, with consideration of the EnRiskS responses to the draft peer review.

3. Peer review findings

3.1 General Comments and overall assessment

In summary, I find the EnRiskS report to be a thorough, professional and well-presented review of the potential health risks to nearby communities that could be associated with the proposed development. It generally follows prescribed national (enHealth, NHMRC, NEPC & NEPM) and NSW guidance on how the HHRA component of an EIS is to be structured. There is extensive collation of the data used and appropriately detailed descriptions of the methods used to calculate potential exposures and the benchmarking of these exposure estimates against appropriately chosen health-based environmental standards. The report includes a useful suite of illustrations, tabulated data and discussion. Quite correctly, it focusses on potential health impacts on the local community, and notes that issues of occupational health and safety for workers on the Project are out of scope and addressed by other regulatory measures.

The layout of the report is standard. The Table of Contents allows for easy location of the key elements, and there is a comprehensive list of Figures and Tables. There is also a useful list of common acronyms and an explanation of common terminology.

It is noted that the report has been comprehensive and systematic in reviewing the geological characteristics of the region, as well as the geographic relationship of the proposed mine with nearby residential areas and the demographics of the region. Particular attention has been paid to the township of Lue where there has been a history of community concerns[1] raised about the Project.

The amount of data presented in the report is very extensive, and I will not attempt to comment on all of it. While I generally have confidence in the approaches outlined in Annexures A to E and H in relation to the calculation of potential exposures and incremental risks associated with fine particulates (PM_{10} and $PM_{2.5}$), lead, various metals and noise, I have not attempted to verify all the calculations based on the equations, as outlined in Annexures F & G, other than to check a few of the more critical ones. This is consistent with the general disclaimer outlined by EnRiskS in Sections 1.7 and 1.8, indicating reliance on, but no responsibility for, the validity of the data used in the various reports.

The overall outcomes of the risk assessments are summarised at the end of each section, with an overall summary in Table 8.1 in relation to air and water quality, and noise impacts, along with the recommended risk mitigation strategies.

The overall conclusions drawn are measured and reasonable and I can find no reason to challenge any of them.

[1] Outlined to some extent is Sections 1.6 and 3.4, but also generally available via internet blogs

3.2 Comments on selected elements of the EnRiskS Report

3.2.1 Baseline environmental data

Sections 3.2 and 3.3 of the HHRA include the geographic and demographic characteristics of the regions surrounding the proposed mine site, while Sections 4.1 to 4.4 include details of the geological features of the area, with Tables 4.1 and 4.2 detailing the levels of key metals in soil and relevant dust samples. It is particularly noteworthy that levels of certain metals (notably, lead, zinc and the metalloid arsenic) currently exceed relevant health-based soil and dust levels. Table 4.2 is particularly informative because it includes data on **current** lead (Pb) levels in indoor dust in and around sites in Lue (including the Lue Public School). It suggests that Pb-based paints may have contributed to these baseline levels, as well as dusts from geological soil sources in the regions[2].

Table 4.3 establishes that levels of a more extensive range of metals is surface water and groundwaters can exceed relevant health (drinking water) and recreational water guidelines at some sites. While these types of water resources may represent limited exposure potential for nearby residential areas (except through the use of bores), more relevant are the measures of key metals in selected home rainwater tanks (Table 4.4[3]). These follow a similar pattern of selective exceedances of health-based guidelines. The EnRiskS report draws attention to the fact that the measured levels of metals in rainwater tank water and sediments may be partly attributable to the nature of the tank construction materials, as well as dusts settling on collecting roof surfaces.

Section 4.7 outlines current air quality characteristic of the region, as extracted from the report of another consultant (Ramboll 2019). The average air quality criteria and dust deposition data are more generally within health-based guidelines than the corresponding data on water and soil quality. The data in Table 4.7 indicate that current metal ingestion via locally grown foodstuffs is not a matter for concern[4]. Section 4.9 confirms that current noise levels in the region are typical of a relatively undeveloped rural environment.

These sections are an important element of the HHRA, because they outline the existing health status of the area, along with some of the demographic and geological characteristics that define it. It is used to establish the baseline against which incremental risks associated with the mine development can be understood.

3.2.2 Data inputs

Data on exposures to airborne metals and particulates, water-borne metals and noise are largely drawn from reports and modelling prepared by others[5]. However, the exposure assessments include various assumptions that are crucial to the EnRiskS HHRA, such as intakes associated with the soil, water, airborne and deposited dusts, as well as the locations of sensitive receptors. I have reviewed these and consider

[2] It is noted that the dust levels in Table 4.2 are benchmarked against current NSW criteria. The US EPA has flagged more stringent criteria (see https://www.federalregister.gov/documents/2019/07/09/2019-14024/review-of-the-dust-lead-hazard-standards-and-the-definition-of-lead-based-paint) that could be introduced in 2020. If more stringent dust Pb levels are subsequently adopted by NSW, this may exacerbate the level of concern about the current level of community exposure to Pb, but it should not alter the estimates or impacts of **incremental** risks associated with the proposed project. Perhaps the projected US EPA changes could be added to the discussion of Pb in Annexure B.
[3] There is an error in Table 4.4 in that the legend indicates that exceedances of health-based guidelines are highlighted by blue text. The highlighting is missing is this version of the Table.
[4] It may have been helpful to include in Table 4.7, FSANZ-derived TDI values for these metals, where available.
[5] Part 3.1 of my report includes standard disclaimers about reliance on data developed by others.

them to be fit-for-purpose and that they have been chosen using appropriately conservative assumptions. Figure 5.1 is an example of how the report addresses the integration of all significant exposure sources.

3.2.3 Air quality and dust-borne assessments

The approach taken by EnRiskS has been to compare maximum predicted airborne concentrations of particulate dusts (PM_{10} and $PM_{2.5}$) with recognised Australian (NEPM) air quality standards for both acute (24h) and chronic (annual average) exposures[6]. In addition, calculations of the incremental risk attributable to predicted changes in $PM_{2.5}$ (the more significant measure for health effects from dust) associated with the Project have been set out in Annexure A. The risk measure chosen was the incremental risk of all-cause mortality for a suitably chosen residential site. The estimate (3×10^{-5}) is within guidelines established by the NSW EPA.

In addition, and perhaps of more significance to the HHRA for this Project, EnRiskS have calculated incremental health risks predicted for exposures to lead (Annexure B) and other metals (Annexure C) via airborne and deposited dusts. The exposures have been estimated (Section 5.2.3) and benchmarked against appropriate health-based guidance values (HBGVs), as set out in Tables 5.1 and 5.2. Annexure E sets out the parameters used and the assumptions made to arrive at estimates of Reasonable Maximal Exposure (RME). I agree these methodologies have been appropriate and adequately described. I further support the use of the Risk Index (RI) approach to integrating the risk estimates across all the metals considered in the report (Section 5.2.4)

Section 5.2.6.1 and Annexure F make an important contribution to the HHRA report because they establish the baseline risks associated with exposures to lead and the relevant metals in existing soil and dust in the region. Table 4.1 suggests that these risks are relatively high (RI between 0.5 – 1.1) for lead, arsenic, cadmium zinc, nickel and manganese for young children, although lower for adults. These baseline risks are generally at least an order of magnitude higher than estimates of **incremental** risk associated with the Project (Tables 5.2 and 5.3). These differences are graphically illustrated in Figures 5.5 and 5.6.

Similar conclusions have been drawn about the negligible health impacts of inhaled silica (Section 5.3) and hydrogen cyanide (Section 5.4).

3.2.4 Focus on lead

Given the concerns raised in local communities, there is an appropriate focus on risks associated with lead (Pb) in dusts, water and foodstuffs. There is an extensive discussion (Annexure B) of the health effects of Pb and the derivation of various national and international Toxicity Reference Values (TRVs) and HBGVs. The EnRiskS HHRA report proposes the use of TRVs 1.3 μg/kg/d for children and 0.6 μg/kg/d, based on different endpoints (neurobehavioural effects in children and renal effects in adults)[7]. I am comfortable with these proposals and note they are

[6] It is noted that the estimated PM_{10} and $PM_{2.5}$ levels have been benchmarked against current NEPM guideline values. Table A1 in Annexure A notes that goal values for $PM_{2.5}$ are proposed to be reduced in 2025, which is within the lifespan of the Project. Perhaps some discussion could be included acknowledging that the estimated particulate levels projected for the Project should still meet the more stringent standard.

[7] Perhaps some thought could be given to a further explanation of why the proposed TRV for children is higher than that for adults. This seems counter-intuitive because it is generally considered that children are more susceptible to Pb toxicity.

probably more conservative than some of the HBGVs in current use, as outlined in the (un-numbered) Table in Annexure B.

3.2.5 Water quality vs water availability

A primary focus of the HHRA in relation to water quality is to determine the incremental risks associated with mine-related activities and impacts on local water quality (Section 6). The conclusion (Section 6.5) that "*impacts to surface water and groundwater as a result of the Project is considered to be negligible*" is supported by calculations of incremental risk in Annexure G. In addition, there has been some consideration of possible impacts on water use and availability (Section 6.3). These estimated impacts rely on a different approach to that used in conventional HHRA, so I am unable to verify or comment on the conclusions in the EnRiskS report relating to water availability. It is advised that peer reviews have also been completed for the groundwater and surface water assessments and are appended to those respective reports.

3.2.6 Noise and vibration impacts

Assessment of noise impacts is also a process somewhat different from standard HHRA processes, so my expertise in this area is limited. It is advised that a peer review has also been completed for the noise and vibration assessment. Nevertheless, the EnRiskS HHRA report, relying on separate estimates by SLR (2019) of noise generated during construction and mine operations (including blasting) appears reasonable. It flags some potential disturbances that exceed NSW EPA guidelines at certain residences, although none of these is likely to result in adverse health effects. The outcome is a proposal to implement relevant noise reduction plans.

3.2.7 Road traffic noise impacts

Section 7.3 of the HHRA report deals with health impacts associated with noise from increases in road traffic (including heavy vehicle traffic). These impacts are assessed largely in terms of the extra noise as measured against NSW DECCW policies. The potential health effects attributable to the risk of road accidents associated with increased heavy vehicle traffic is not considered by the HHRA. Rather, road safety is addressed separately as part of the Traffic and Transport Assessment (TTPP, 2019).

3.2.8 Treatment of elements of uncertainty

The issue of how the HHRA deals with uncertainties in the reviewed data and information is explained in some detail in Section 5.5 in relation to air quality, Section 6.4 in relation to surface and groundwater impacts, and Section 7.5 in relation to noise impacts. These parts of the EnRiskS report set out clearly the ways in which these uncertainties have been managed and stresses the conservatism that is normally built into the models and paradigms that inform the health risk estimates.

3.2.9 Selection of emission standards and other Health-Based Guidance Values (HBGVs)

In any health risk assessment, the benchmark against which the risk estimates are compared is a crucial element. In some cases, where a threshold is deemed to exist,

the benchmark is generally conservatively[8] set as a Tolerable Daily Intake (TDI) and this may have been used to establish HBGVs, such as NHMRC drinking water guidelines, or air/soil/dust standards included in relevant NEPMs. Estimated exposures less than the TDI or HBGV are generally assumed to have no appreciable health impacts over a lifetime of exposure. They should therefore be protective against shorter-term exposures, such as those likely to be associated with the Project.

Where the exposure-response relationships do not exhibit a clear threshold (e.g. for carcinogens and some air pollutants), the exposure-response relationship is extrapolated down to a defined level of excess risk (e.g. 1 in 10,000 or 10^{-4} to 1 in 1,000,000 or 10^{-6}). This defined risk level is termed 'acceptable' in the EnRiskS HIA report, although it is sometimes referred to as a 'target risk level' in some cases to avoid argument over to whom the risk level meets 'acceptable' criteria (see also discussion in Section 5.10 enHealth 2012).

The same concept applies to integrated estimates of risk, such as the Risk Index (RI), where estimates <1 are considered to be "acceptable".

In the case of noise annoyance and vibration, where the risk assessments are based on endpoints relating to noise levels that have been equated with health effects, such as sleep disturbances, impacts on hearing, cardiovascular health and general annoyances. The risk estimates have been based on noise projected to be generated by traffic and equipment, during construction activities and during projected mine operations, and in particular, blasting operations. These noise and vibration estimates have been benchmarked against statutory guidance published by NSW EPA and WHO.

4. **Peer reviewer recommendations**

i. The HHRA report does not require any substantive changes. It represents a comprehensive and fair assessment of likely health impacts associated with projected mine operations during both construction and operational phases. It includes estimates of incremental risks associated with airborne dusts and hydrogen cyanide vapours, water- and food-borne metals, as well as potential impacts of noise and water resource management.

ii. Some minor edits, or suggested additional discussion, have been identified at footnotes 2, 3, 4, 6 and 7.

iii. The response by EnRiskS to issues raised in the draft peer review are summarised in Appendix B.

iv. There are no issues raised in the EnRiskS response that alter the opinion of the peer reviewer. The minor issues raised in (ii) above have been appropriately addressed.

[8] There is a useful discussion of the application of the term 'conservative' in the list of defined terms. It notes how such estimates may over-estimate risks and how they may incorporate 'safety factors' to further mitigate actual risks.

Appendix A – Credentials of peer reviewer

This peer review was prepared by Brian G. Priestly M.Pharm, PhD, FACTRA, Principal of my own consultancy company, Priestly Toxicology Consulting.

The relevant experience brought to this task by the peer reviewer includes:

- Fifteen years of leadership of the Australian Centre for Human Health Risk Assessment at Monash University (part time from 2009 to 2018)
- Experience in regulatory toxicology in former leadership appointments to the Commonwealth Department of Health in areas of toxicological assessment of agricultural & veterinary chemicals, regulation of medicines, and assessment of chemicals for poisons scheduling
- More than 45 years experience with government expert committees and panels assessing chemical toxicity and chemicals risk management, including issues of air, water and soil quality assessment
- Management of the re-writing and updating of seminal Australian guidance on health risk assessment of environmental hazards (enHealth 2012).
- Preparation of reports on health-related issues associated with the Oz Minerals mine at Rosebery, Tasmania (2008-09), Costerfield antimony mine Victoria (2014), water quality relating to the Redmont mining camp, WA (2015) and a peer review of a report on dust issues relating to multi-industry and port operations at Port Hedland WA (2015).
- Peer-reviewed recognition as a Fellow of the Australasian College of Toxicology & Risk Assessment (ACTRA), a professional organisation that I helped to found and for which I served as its inaugural President.

The opinions set out in this report are my own, and do not reflect views of any previous employers.

Appendix B – EnRiskS response to draft peer review

In relation to the peer review comments – the following has been undertaken to address the comments received:

- Footnote 1: Noted
- Footnote 2: The reference to the NSW guidelines has included the word current as these are the current NSW guidelines for lead in dust. In addition a footnote has been included in Section 4.3 where elevated levels of lead in dust indoors is discussed in relation to the school.
- Footnote 3: The table has been corrected to include the blue text (as noted in the footnote).
- Footnote 4: Intakes as a % of the TDI or nutrient upper limit (from FSANZ) has been included in the table, where available.
- Footnote 5: Noted
- Footnote 6: The 2025 goal has been included in the discussion in Section 5.2.2.2 as well as in Table A1 in Annexure A
- Footnote 7: Some additional text has been included in Annexure B

The peer reviewer notes that these changes adequately address points raised in the draft peer review.