Appendix E

Health Impact Assessment

REGIS RESOURCES LTD

McPhillamys Gold Project: Health Impact Assessment

Prepared for: LFB Resources NL

25 August 2020

EnRiskS

Document History and Status

Report Reference	EMM/19/HIAR001
Revision	C - Final
Date	25 August 2020
Previous Revisions	A – Draft issued 21 November 2019 B – Revised draft issued 23 July 2020

Limitations

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

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

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

This report was prepared in October and November 2019 and updated in July and August 2020 and is based on the information provided and reviewed at that time. Environmental Risk Sciences disclaims responsibility for any changes that may have occurred after this time.

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

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Glossary of Terms and Abbreviations

Term	Definition
AAQ	Ambient air quality.
ABS	Australian Bureau of Statistics.
Acute exposure	Contact with a substance that occurs once or for only a short time (up to 14 days).
Absorption	The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs.
Adverse health effect	A change in body function or cell structure that might lead to disease or health problems.
Aerodynamic diameter	Airborne particles have irregular shapes, their aerodynamic behaviour is expressed in terms of the diameter of an idealised spherical particle.
AIHW	Australian Institute of Health and Welfare.
ANZECC	Australia and New Zealand Environment and Conservation Council.
AQGGA	Air Quality and Greenhouse Gas Assessment.
ATSDR	Agency for Toxic Substances and Disease Register.
Background level	An average or expected amount of a substance or material in a specific environment, or typical amounts of substances that occur naturally in an environment.
Biodegradation	Decomposition or breakdown of a substance through the action of micro-organisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).
Body burden	The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.
Carcinogen	A substance that causes cancer.
CCME	Canadian Council of Ministers of the Environment.
Chronic exposure	Contact with a substance or stressor that occurs over a long time (more than one year) [compare with acute exposure and intermediate duration exposure].
COMEAP	Committee on the Medical Effects of Air Pollutants.
dB(A)	Decibels (A-weighted).
DEC	NSW Department of Environment and Conservation.
DECC	NSW Department of Environment and Climate Change.
DECCW	NSW Department of Environment, Climate Change and Water.
DEFRA	Department for Environment, Food & Rural Affairs.
DEH	Australian Department of Environment and Heritage.
Detection limit	The lowest concentration of a substance that can reliably be distinguished from a zero concentration.
Dose	The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An ‘exposure dose’ is how much of a substance is encountered in the environment. An ‘absorbed dose’ is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.
DPIE	Department of Planning, Infrastructure and Environment
EIS	Environmental Impact Statement.
EL	Exploration Licence.
EPHC	Environment Protection and Heritage Council.
EU	European Union.

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

Term	Definition
NEPM	National Environment Protection Measure.
NHMRC	National Health and Medical Research Council.
NO_2	Nitrogen dioxide.
NOA	Naturally occurring asbestos.
NOx	Nitrogen oxides.
NSW	New South Wales.
NSW EPA	NSW Environment Protection Authority.
OEH	NSW Office of Environment and Heritage.
OEHHA	Office of Environmental Health Hazard Assessment, California Environment Protection Agency (Cal EPA).
PM	Particulate matter.
PM_1	Particulate matter of aerodynamic diameter 1 micrometre (µm) and less (termed ultrafine particles).
$PM_{2.5}$	Particulate matter of aerodynamic diameter 2.5 micrometres (µm) and less.
PM_{10}	Particulate matter of aerodynamic diameter 10 micrometres (µm) and less.
Point of exposure	The place where someone can come into contact with a substance present in the environment [see exposure pathway].
Population	A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).
RBL	Rating Background Level.
Receptor population	People who could come into contact with hazardous substances [see exposure pathway].
Risk	The probability that something would cause injury or harm.
ROM	Run-of-mine.
Route of exposure	The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].
SEARs	Secretary's Environmental Assessment Requirements.
SEIFA	Socio-Economic Index for Areas.
SIA	Social Impact Assessment.
TCEQ	Texas Commission on Environmental Quality.
Toxicity	The degree of danger posed by a substance to human, animal or plant life.
Toxicity data	Characterisation or quantitative value estimated (by recognised authorities) for each individual chemical substance for relevant exposure pathway (inhalation, oral or dermal), with special emphasis on dose-response characteristics. The data are based on based on available toxicity studies relevant to humans and/or animals and relevant safety factors.
Toxicological profile	An assessment that examines, summarises, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.
Toxicology	The study of the harmful effects of substances on humans or animals.
TSP	Total suspended particulates.
UK	United Kingdom.
US	United States of America.
USEPA	United States Environmental Protection Agency.
WHO	World Health Organization.
$\mu g/m^3$	Micrograms per cubic metre.
µm	Micrometre.

Executive Summary

Environmental Risk Sciences Pty Ltd (enRiskS) has been engaged to undertake a health impact assessment (HIA) to provide an assessment of health impacts, relevant to the project as detailed in the Environmental Impact Statement (EIS) (EMM 2019) and Amended Project report (EMM 2020).

It is understood that there was no requirement for the completion of an HIA in the Department of Planning, Infrastructure and Environments (DPIEs) Secretary's Environmental Assessment Requirements (SEARs), however, as the mine is proposed to be located relatively close to a number of rural residences issues relating to health impacts were raised during consultation carried out to inform the preparation of the EIS and subsequently through submissions on the EIS.

The HIA has been undertaken in accordance with current Australian guidance (enHealth 2012b, 2017; Harris 2007) and has focus on potential impacts on community health in relation to project related impacts on air quality, noise, water, hazardous materials, lighting and stress/anxiety.

Based on the available information, and with consideration of the uncertainties identified no health risk issues of concern have been identified for the off-site community. More specifically, the following table presents a summary of the health impact assessment and mitigation measures relevant to ensuring impacts are minimised or mitigated.

Table ES1: Summary of health impacts

Air emissions	
Impacts	Based on the available data and information in relation to emissions to air of dust, naturally occurring asbestos (NOA), metals and metalloids that may be present on the dust, nitrogen dioxide and hydrogen cyanide from the project, potential impacts on the health of the community have been assessed. The impact assessment has concluded there are no health risk issues of concern relevant to the project.
Mitigation	A range of mitigation measures have been identified in the EIS and Amended Project report that would minimise dust emissions, diesel combustion emissions and blast fumes. In addition, air quality monitoring is proposed to be continued for the duration of the project, which includes dust-deposition monitoring and real-time particulate monitoring. An air quality management plan, which includes a monitoring plan, would be developed for the project, documenting locations, monitoring methods and reporting responsibilities.
Noise emissions	
Impacts	Based on the predicted noise levels and the proposed mitigation and management measures, including additional monitoring and management as detailed in the EIS and Amended Project report, 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	A range of noise mitigation and management measures, including monitoring and additional management measures are outlined in the EIS and Amended Project report. These mitigation and management measures would be detailed in a site Noise Management Plan relevant to the mine development site and the pipeline (construction phase) to guide, quantify and control noise emissions from the project. These plans may include provisions to undertake noise monitoring. In addition, a complaints handling process will be implemented for the mine development site and the works associated with the pipeline construction.

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 (in relation to quantity and quality of water) as a result of the project, including the pipeline, is considered to be negligible.
Mitigation	Implementation of the proposed Water Management Plans which would include a monitoring program covering both onsite and neighbouring bore/water sources plus regular public reporting.
Hazardous materials	
Impacts	Based on the assessments undertaken there are no impacts in the off-site community. This includes the transport, storage and use of a range of dangerous goods, including explosives, cyanide and LPG. Where there are no impacts, there are no risks to community health.
Mitigation	Transport in accordance with the Australian Transport Code and the storage and use of dangerous good in accordance with all relevant regulations and codes.
Lighting	
Impacts	Based on the assessments undertaken, it is expected that lighting would be visible in various areas surrounding the site, however the potential for lighting to directly intrude into residential homes and adversely affect sleep and hence health is considered negligible.
Mitigation	A range of mitigation measures related to lighting are outlined in the EIS and Amended Project report to minimise the potential for light spill that may affect the off-site areas.
Stress and anxiety	
Impacts	The potential for the project to result in increased levels of stress and anxiety in the community has been identified by the community and recognised as an area of key concern in the SIA.
Mitigation	Management measures, principally related to engaging in and maintain transparent, evidence based and ongoing dialogue with concerned property owners and other community members based on the outcomes of the EIS and Amended Project report.

Section 1. Introduction

1.1 Background

LFB Resources NL is seeking State Significant Development consent for the construction and operation of the McPhillamys Gold Project, a greenfield open cut gold mine and associated water supply pipeline in the Central West of New South Wales (NSW). LFB Resources NL is a 100% owned subsidiary of Regis Resources Limited (herein referred to as Regis).

The McPhillamys Gold Project comprises two key components (refer to **Figure 1.1**); the mine site where the ore will be extracted, processed and gold produced for distribution to the market (the mine development), and an associated water pipeline that will enable the supply of water from approximately 90 km away near Lithgow to the mine site (the pipeline development). The mine development is around 8 km north-east of Blayney within the Blayney and Cabonne local government areas (LGAs). It is also in the upper reaches of the Belubula River catchment, within the greater Lachlan River catchment.

Up to 8.5 million tonnes per annum (Mtpa) of ore will be extracted from the McPhillamys gold deposit over a total project life of 15 years. The mine development will include a conventional carbon-in-leach processing facility, waste rock emplacement, an engineered tailings storage facility (TSF) and associated mine infrastructure including workshops, administration buildings, roads, water management infrastructure, laydown and hardstand areas, and topsoil stockpiles.

In accordance with the requirements of the NSW *Environmental Planning and Assessment Act 1979* (EP&A Act), the NSW *Environmental Planning & Assessment Regulation 2000* (EP&A Regulation) and the Secretary's Environmental Assessment Requirements (SEARs) for the project, an Environmental Impact Statement (EIS) was prepared to assess the potential environmental, economic and social impacts of the project. The development application and accompanying EIS were submitted to the NSW Department of Planning, Industry and Environment (DPIE) in August 2019 and publicly exhibited for six weeks, from 12 September 2019 to 24 October 2019.

During this exhibition period Regis received submissions from regulators, businesses and other organisations and the community regarding varying aspects of the project.

Environmental Risk Sciences Pty Ltd (enRiskS) has been engaged to undertake a health impact assessment (HIA) to provide an assessment of health impacts, relevant to the project as detailed in the Environmental Impact Statement (EIS) (EMM 2019) and Amended Project report (EMM 2020).

It is understood that there was no requirement for the completion of an HIA in the Department of Planning, Infrastructure and Environments (DPIEs) Secretary's Environmental Assessment Requirements (SEARs), however, as the mine is proposed to be located relatively close to a number of rural residences issues relating to health impacts were raised during consultation carried out to inform the preparation of the EIS and subsequently through submissions on the EIS.

1.2 Objectives

The overall objective of the HIA is to provide an assessment of potential impacts to human health in relation to the project, specifically in relation to impacts related to air quality, noise and vibration, hazardous incidents and water quality.

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

This report applies to the mine development component of the project, as it is this component where health impacts were raised in the submissions received. Therefore, references to 'the project' herein are referring to the mine development component, and the 'mine development project area' is referred to as the 'project area' or 'McPhillamys' throughout.

1.3 Submissions on the EIS

Concerns relevant to potential health impacts were raised by various interest groups in submissions received on the EIS. These concerns are listed in **Table 1.1** and have been considered in this assessment.

Table 1.1: Key comments received in submissions relating to health and how they have been addressed

Issue	Where addressed in this report
Concerns about the potential for the project to adversely affect human health. This included concerns that the EIS did not contain a standalone human health assessment prepared by a suitably qualified professional. – the potential for dust generated by the project to adversely affect human health. This included concerns related to the: – potential for the project to exacerbate existing respiratory health issues for neighbouring residents; and – lack of baseline investigations of potential dust-related health impacts (including inhalation and ingestion).	This concern has been addressed through the provision of the detailed health impact assessment presented in this report. The assessment includes information about baseline health of the community (**Section 3**) along with the potential for emissions of dust to result in health impacts within the community (**Section 4**).
Concerns regarding the potential for lighting impacts associated with the project to adversely impact people's health. This included concerns that light pollution from the project will cause sleep disturbance resulting in a number of health problems.	Addressed in **Section 7.2**.
Concerns about the potential for the project to adversely affect people's mental health and contribute to stress.	Addressed in **Section 7.3**.
Concerns about the potential disturbance of naturally occurring asbestos within the mine project area. This included concerns related to: – exposure of workers to naturally occurring asbestos during ongoing operations; and – the absence of asbestos management measures from the EIS.	This is addressed in **Sections 4.3 and 4.5.2.3** of this report.

1.4 Methodology

An HIA is a more broad assessment of potential health impacts related to a project. An HIA addresses impacts to both health and wellbeing of a community and considers both positive and negative impacts of a project on community health. As is often the case, the EIS process typically focuses on the negative impacts, and, as the HIA has been undertaken at the end of the EIS process (and revised during the mended Project/Submissions Report process), the HIA has largely focused on negative health impacts. Where positive impacts can be addressed, they have been included in the HIA.

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

- Harris, P., Harris-Roxas, B., Harris, E. & Kemp, L., Health Impact Assessment: A Practical Guide, Centre for Health Equity Training, Research and Evaluation (CHETRE). Part of the UNSW Research Centre for Primary Health Care and Equity. University of New South Wales, Sydney, 2007 (Harris 2007)
- enHealth, Health Impact Assessment Guidelines (enHealth 2017)
- enHealth Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012b).

In addition, the following guidance will be considered, where appropriate:

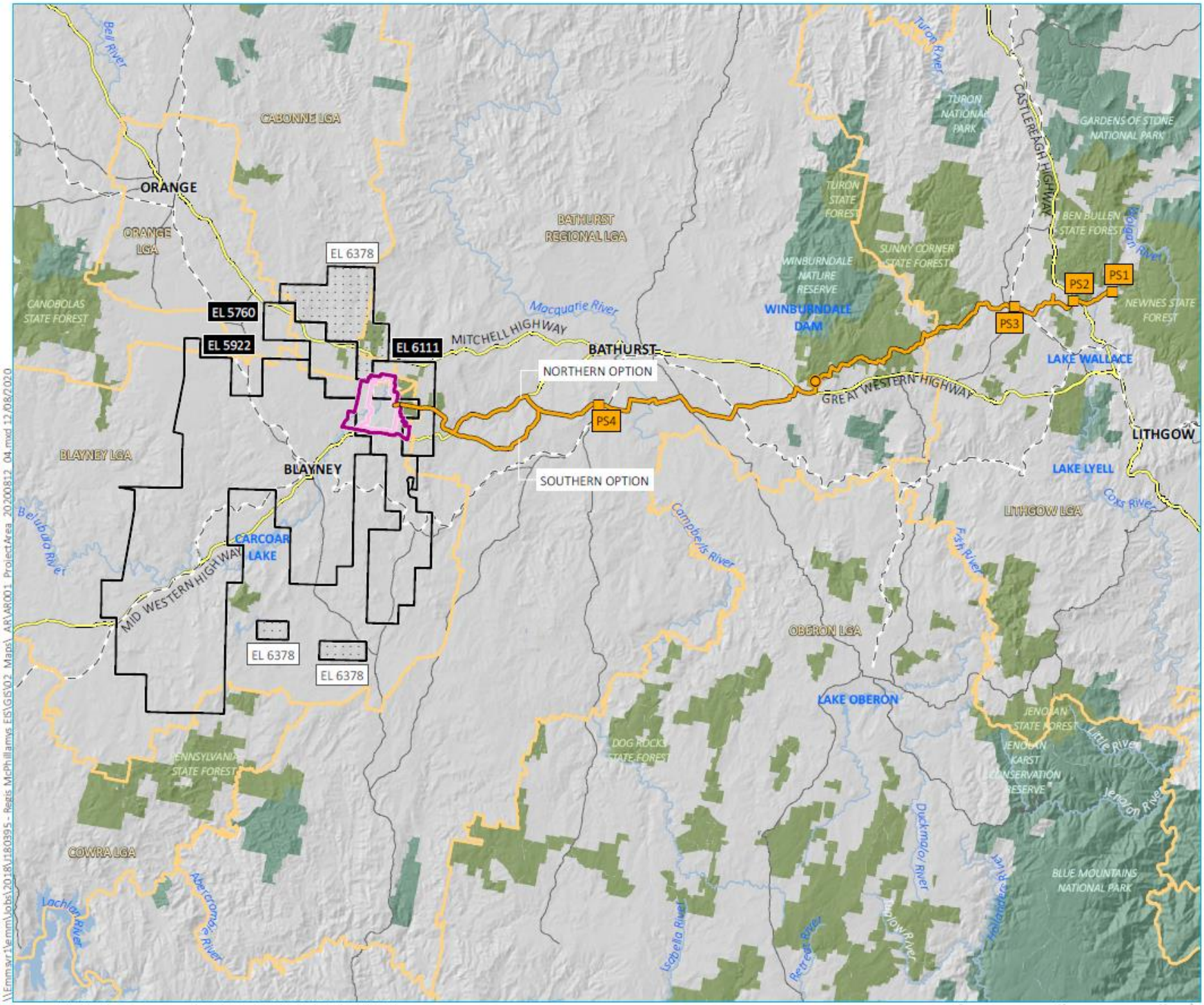
- SEPP No. 33 - Hazardous and Offensive Development (NSW Government 2014)
- NEPC National Environment Protection (Ambient Air Quality) Measure (NEPC 2016)
- National Environmental Protection Measure – Assessment of Site Contamination including:
    - Schedule B1 Investigation Levels for Soil and Groundwater (NEPC 1999 amended 2013e)
    - Schedule B4 Guideline on Health Risk Assessment Methodology (NEPC 1999 amended 2013d)
    - Schedule B6 Guideline on Risk Based Assessment of Groundwater Contamination (NEPC 1999 amended 2013c)
    - Schedule B7 Guideline on Health-Based Investigation Levels (NEPC 1999 amended 2013b)
    - 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 DEC 2005)
- NSW Industrial Noise Policy (NSW EPA 2000)
- NHMRC Australian Drinking Water Guidelines (NHMRC 2011 Updated 2016)
- 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 Environment Protection Agency (USEPA) and the World Health Organisation (WHO), consistent with current industry best practice.

1.5 Available information

The HIA has been prepared on the basis of information available in the EIS for the project, as detailed below:

- EMM 2019, McPhillamys Gold Project, Environmental Impact Statement. Prepared for LFB Resources NL, dated August 2019, and the following appendices:
  - HEC 2019, McPhillamys Gold Project, Mine Development, Surface Water Assessment. Prepared by Hydro Engineering & Consulting Pty Ltd (HEC) dated 27 August 2019. Appendix J of the EIS
  - EMM 2019a, McPhillamys Gold Project, Groundwater Assessment. Report dated August 2019. Appendix K of the EIS
  - MAC 2019a, Noise and Vibration Impact Assessment, McPhillamys Gold Project, Blayney, NSW. Report prepared by Muller Acoustic Consulting (MAC) dated August 2019. Appendix L of the EIS
  - EMM 2019, McPhillamys Gold Project, Air Quality and Greenhouse Gas Assessment. Report dated August 2019, Appendix M of the EIS
  - RM 2019, McPhillamys Preliminary Hazard Analysis. Report dated June 2019. Appendix R of the EIS
  - EMM 2019b, McPhillamys Gold Project, Pipeline Development Water Assessment. Report dated August 2019. Appendix X of the EIS
  - MAC 2019b, Noise and Vibration Impact Assessment, McPhillamys Gold Project – Pipeline Development Lithgow to Blayney, NSW. Report prepared by Muller Acoustic Consulting (MAC) dated July 2019. Appendix AA of the EIS
  - VPA 2019, Visual Impact Assessment, McPhillamys Gold Project. Report dated 19 August 2019. Appendix S of the EIS
  - Hansen Bailey 2019, Social Impact Assessment, McPhillamys Gold Project. Report dated July 2019. Appendix T of the EIS.
- EMM 2020, McPhillamys Gold Project, Amendment report, including the following appendices:
  - EMM 2020, McPhillamys Gold Project, Air Quality and Greenhouse Gas Assessment. Report dated July 2020, which is a supplementary report to the Air Quality and Greenhous Gas Assessment included in Appendix M of the EIS
  - VPA 2020, McPhillamys Gold Project, Visual Impact Assessment – Addendum. Report dated August 2020.
  - MAC 2020, McPhillamys Gold Project, Amended Noise and Vibration Impact Assessment. Report dated July 2020.
  - HEC 2020, McPhillamys Gold Project, Mine Development, Revised Surface Water Assessment. Report dated July 2020.
  - EMM 2020, McPhillamys Gold Project Amendment Report – Groundwater Assessment Addendum.
- Regis 2020, Potential for the Presence of Naturally Occurring Asbestos at the McPhillamys Gold Project, Kings Plains NSW. Report dated 14 May 2020.



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Source: EMM (2020); Regis Resources (2020); DPE (2018); DFSI (2017); GA (2011)

Regional setting – project application area – amended project

McPhillamys Gold Project

Figure 1.1

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Section 2. Project description

2.1 Project location

The project is located in the Central Tablelands region of NSW, approximately 8 km to the north-east of Blayney, 20 km to the west of Bathurst and 27 km to the south-east of Orange (refer to **Figure 2.1**). The project is mostly located within the Blayney local government area (LGA) with a small proportion extending into the Cabonne LGA. The pipeline runs through the LGAs of Lithgow, Bathurst and Blayney.

The project involves two key components, as shown on **Figure 1.1**:

- The mine site where the ore will be extracted and processed (referred to as the mine development); and
- An associated water pipeline (referred to as the pipeline development) which is a 90 km long pipeline, transferring surplus water from Centennial Coal's Angus Place Colliery (Angus Place) and Springvale Coal Services operations (SCSO), and Energy Australia's Mount Piper Power Station (MPPS) near Lithgow, to the mine.

The mine project area is zoned RU1 Primary Production and is surrounded by a variety of land uses. The predominant land use in the area is agriculture which includes rural residential properties, with other uses that include forestry and natural areas.

The pipeline corridor alignment is predominantly used for agriculture, with mostly cleared, open paddocks used for sheep and cattle grazing.

2.2 Project overview

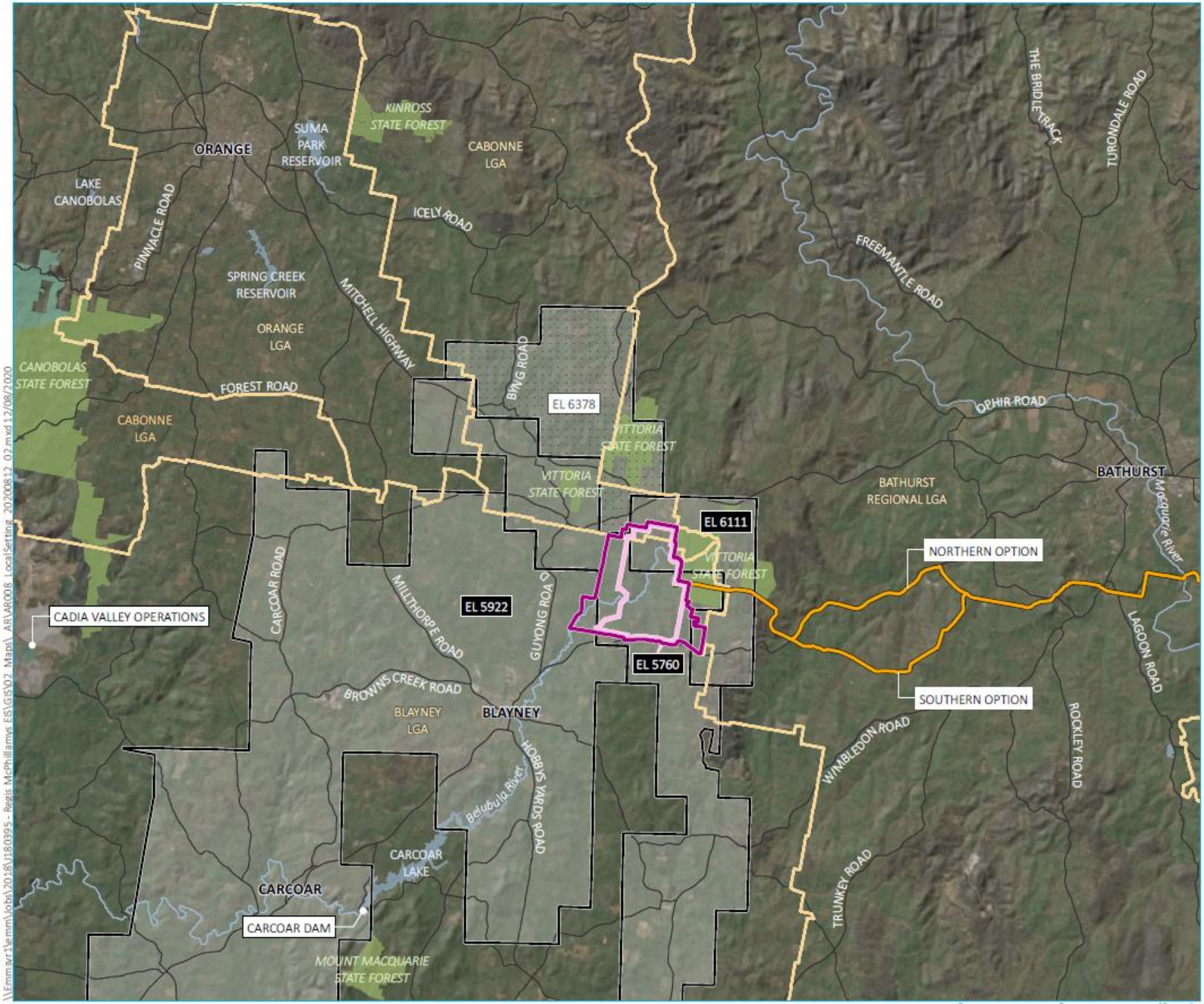
The key components of the project as described in the EIS (EMM 2019), and for which Regis is seeking development consent, include:

- development and operation of an open cut gold mine and associated infrastructure to support the mine over a 15 year project life, including ore processing, stockpiling, tailings management and on-site water management infrastructure;
- extraction of up to 8.5 Million tonnes per annum (Mtpa) of ore over the project life, and the use of a conventional carbon-in-leach processing facility with a processing rate of up to 7 Mtpa to produce approximately 200,000 ounces, and up to 250,000 ounces, per annum of product gold;
- construction and use of an engineered TSF to store tailings material;
- establishment of mine site access via a new intersection off the Mid Western Highway;
- development of ancillary infrastructure, including a mine site access road, internal haul roads, workshop, stores, administration buildings, explosives magazine and storage, soil stockpiles and other minor site infrastructure;
- progressive rehabilitation of the mine development; and
- construction and use of a water supply pipeline between the mine development and the Western Coalfields (i.e. the pipeline development).

In response to issues raised in submissions from the community, government agencies, businesses and other organisations, as well as a result of further detailed mine planning and stakeholder

engagement, Regis has made a number of amendments to the project since the public exhibition of the EIS. The main amendments relate to the following aspects:

- **Site access** – a new location for the site access intersection off the Mid Western Highway is proposed, approximately 1 km east of the original location assessed in the EIS, in response to feedback from Transport for NSW (TfNSW, former Roads and Maritime Services) and the community. A new alignment is subsequently proposed for the site access road to the mine administration and infrastructure area.
- **Mine and waste rock emplacement schedule** – revision of the mine schedule and the subsequent construction sequence of the waste rock emplacement has been undertaken, in particular consideration of predicted noise levels in Kings Plains, resulting in reduced early activity in the southern end of the mine development project area while extending the construction timeframe for the southern amenity bund.
- **Pit amenity bund** – optimisation of the open cut pit design and the improved location of the primary exit ramps for haul trucks (particularly from a noise perspective) allowed the size of the pit amenity bund to be reduced.
- **Tailings Storage Facility (TSF)** – amendments to the design include changes to the embankment design and construction timing, the TSF footprint and the TSF post closure landform to facilitate improved water management around the TSF.
- **Water management system –** the secondary water management facility (WMF) has been removed from the water management system resulting in an avoidance of impacts to a potential item of historic heritage (MGP 23 - Hallwood Farm Complex (Hallwood)). The size of the WMFs has also been revised to achieve a reduced likelihood of discharge from the storages within the operational water management system as part of a revised nil discharge design.
- **Mine administration and infrastructure area** – the layout of this area has been revised and optimised.
- **Mine development project area** – a very small change has been made to the mine development project area along the eastern boundary (an additional 1 ha, or 0.04% change), to accommodate the required clean water management system. The change takes the project area from 2,513 ha to 2,514 ha (refer to **Figure 2.2** for the mine layout).



KEY

Project application area
- Mine development project area
- Mining lease application area (Note: boundary offset for clarity)
- Pipeline

Existing environment
- Main road
- Named watercourse
- Named waterbody
- NPWS reserve
- State forest
- Local government area

Exploration lease boundaries (of interest)
- Held by LFB Resources NL (Regis)
- Held by others

Source: EMM (2020); Regis Resources (2020); DFSI (2017); GA (2011)

0 5 10 km

GDA 1994 MGA Zone 55

N

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Local setting of the mine development

McPhillamys Gold Project

Figure 2.1

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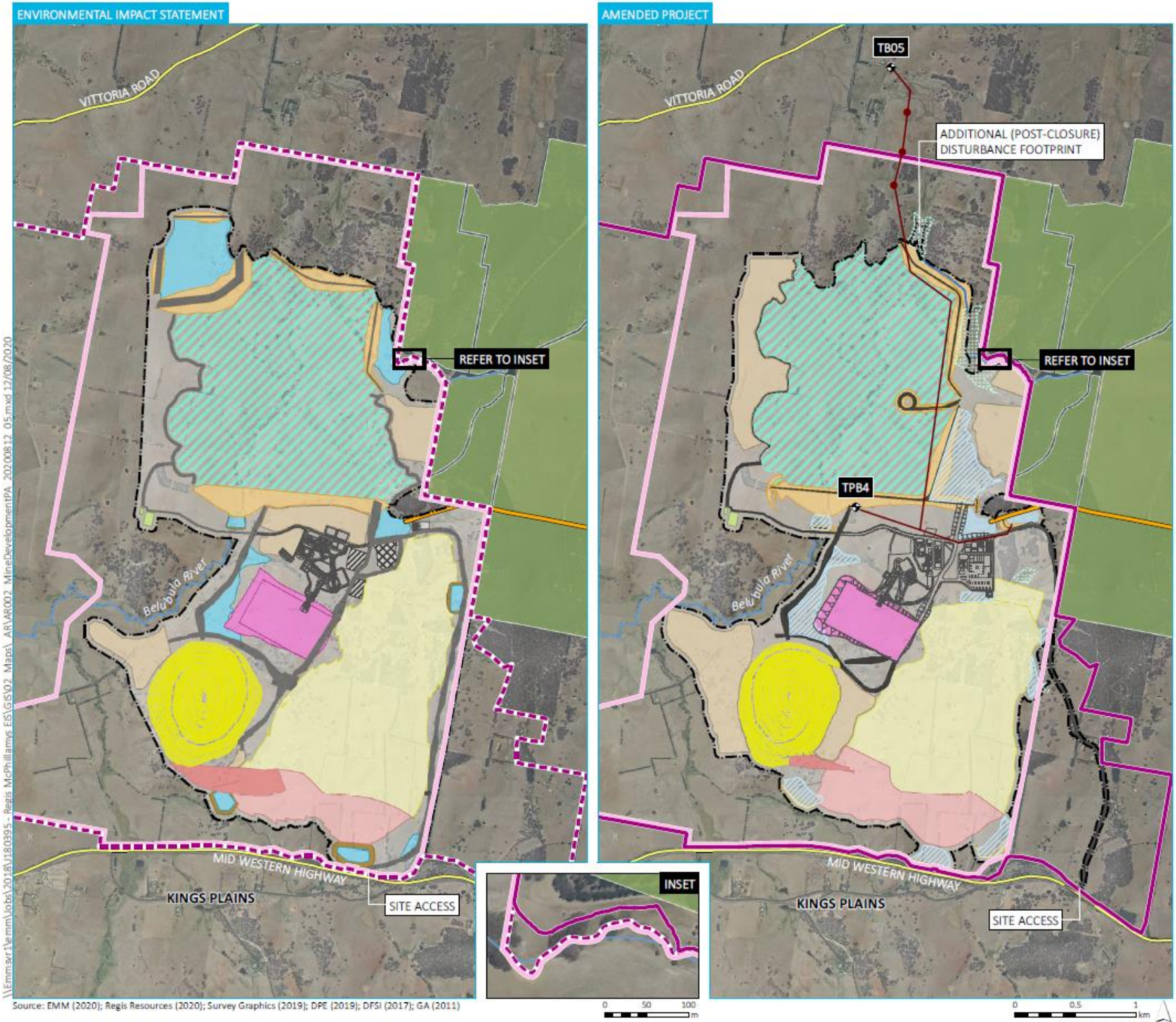

Source: EMM (2020); Regis Resources (2020); Survey Graphics (2019); DPE (2019); DFSI (2017); GA (2011)

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Amended mine development conceptual layout compared to EIS

McPhillamys Gold Project

Figure 2.2

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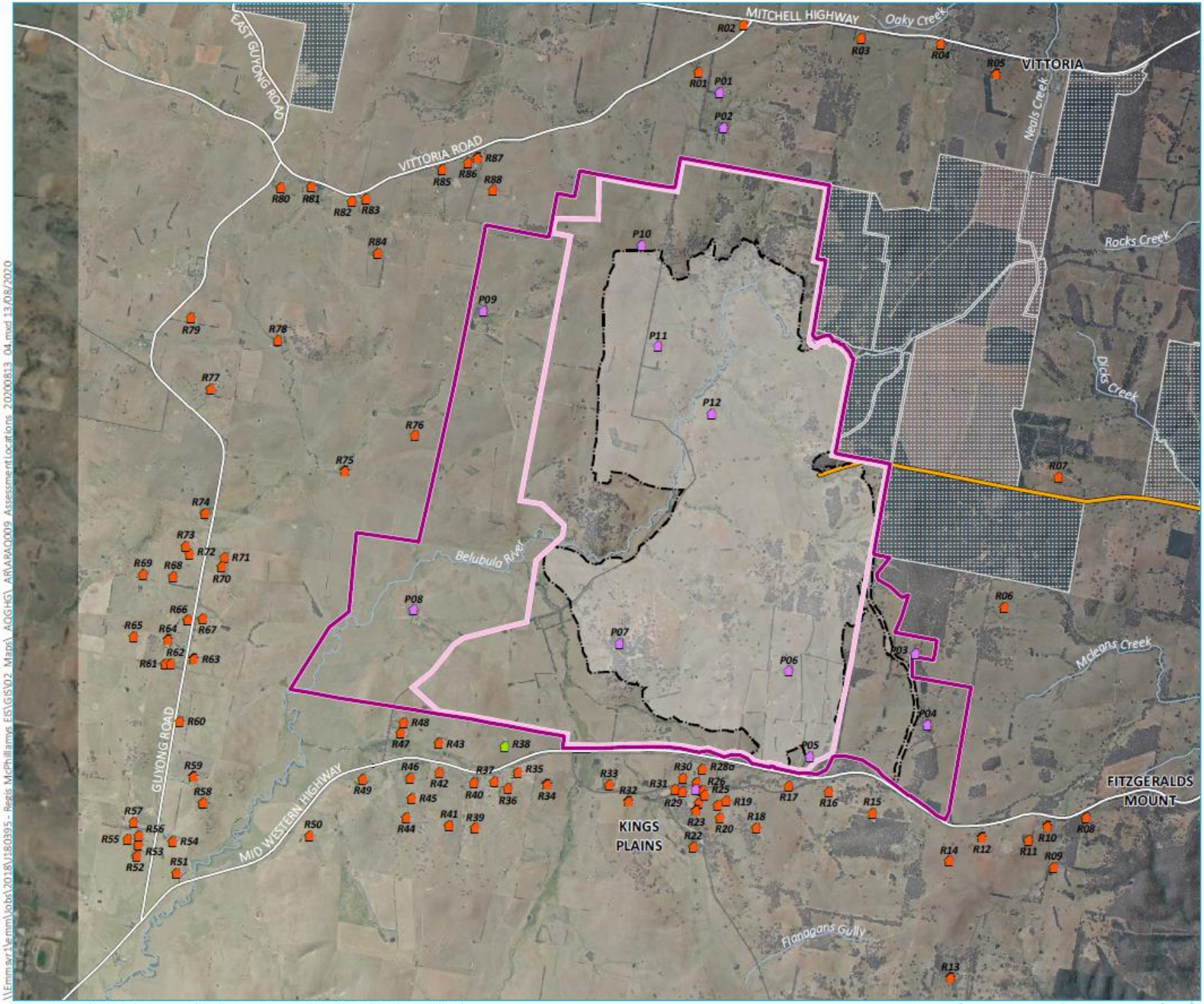
Section 3. Community profile

This section provides an overview of the community potentially impacted by the project. It is noted that the key focus of this assessment is the local community surrounding the site, and include the community areas that are likely to be impacted by the project.

The project is situated in an area that is dominated by existing rural residential and agricultural areas. Remote from the mine development area site are the larger population areas of Blayney, Bathurst and Orange, situated in the LGAs of Blayney and Cabonne with the Bathurst LGA located directly east.

The pipeline development runs through agricultural areas situated within the LGAs of Lithgow, Bathurst and Blayney. Once constructed the pipeline is a passive piece of infrastructure with no emissions to air or water and no noise and vibration impacts. Hence the focus of the assessment of impacts related to the project presented in the EIS relates to the community surrounding the mine development area.

The boundary of the community evaluated in this assessment has been determined based on modelling completed to evaluate key potential health impacts within the EIS, specifically air quality and noise. The assessment of air quality and noise has specifically addressed impacts 89 individual residences (also termed receptors) in the community surrounding the mine development site. These receptors are within 2 km of the mine development and are shown on **Figure 3.1**.



KEY

Project application area

- Mine development project area
- Mining lease application area (Note: boundary offset for clarity)
- Disturbance footprint
- Pipeline

Sensitive receptor

- Private
- Residences under option
- Project related (Regis-owned)

Existing environment

- Major road
- Minor road
- Named watercourse
- Vittoria State Forest

Source: EMM (2020); Regis Resources (2020); Survey Graphics (2019); DFSI (2017); ELVIS (2014)

0 1 2 km

GDA 1994 MGA Zone 55

N

Assessment locations

McPhillamys Gold Project

Figure 3.1

REGIS RESOURCES LTD

EMM creating opportunities

\\Emmsvr1\emm\Jobs\2018\J180395 - Regis McPhillamys EIS\GIS\02_Maps_AQGHG_AR\ARAQ009_AssessmentLocations_20200813_04.mxd 13/08/2020

Table 3.1 presents a general summary of the populations within the localities surrounding the mine development which include the suburbs[1] of Kings Plains (which includes the residences located to the south as well as most of the mine development site), Millthorpe (which included the residences to the west), Guyong (which includes the residences to the northwest), Vittoria (which includes residences to the north and the northern part of the mine development site) and Fitzgeralds Mount (which includes residences to the east). The table also includes data for the Blayney LGA (which encompasses most of these suburbs, particularly to the south and west), Cabonne LGA (that includes the suburbs to the north) Bathurst LGA (which include the suburbs to the east of the site) with comparison to NSW and Australia.

Based on the general statistics available for the population in areas surrounding the project, these populations are generally older, have a lower rate of unemployment and are considered to be less disadvantaged than the population of NSW and Australia. More specifically the smaller populations in suburbs surrounding the project, where data is available, suggests there may be a higher proportion of young children aged 0-4 years (who may be more vulnerable to impacts to air quality and noise), low rates of unemployment and socioeconomic disadvantage (which may indicate the population in general has a lower vulnerability to impacts). Due to the small size of the populations in many of the suburbs, insufficient data is available from some areas. These populations can be adequately characterised on the basis of population data reported for the LGAs of Blayney, Cabonne and Bathurst.

[1] Suburbs as defined by the Australian Bureau of statistics from which the data is derived.

Table 3.1: Summary of populations surrounding the project

Location	Population information/indicator												
	Total population	Population distribution for ages (% total)					Median age	Average household size	Unemployment rate		SEIFA IRSD (rank)	Indigenous	Born overseas
		0-4	5-19	20-64	65+	30+			2016	March 2019			
Suburbs													
Kings Plains	117	*	*	*	*	*	48	2.6	*	--	4	*	*
Millthorp	1,253	7.2%	23.7%	55%	14%	60%	40	2.7	4.0%	--	5	3.4%	13.9%
Guyong	127	7.1%	25.9%	51.9%	15%	63%	44	2.3	4.9%	--	5	0%	25.6%
Vittoria	62	*	*	*	*	*	56	2	*	--	4	*	*
Fitzgeralds Mount	54	*	*	*	*	*	43	2	*	--	4	*	*
LGAs													
Blayney	7,257	6.2%	20.7%	54.3%	18.7%	63.4%	42	2.5	5.8%	4.2%	3	3.7%	14.2%
Cabonne	13,386	6.2%	22.1%	51.9%	19.9%	63.9%	43	2.6	4.3%	2.8%	4	3.7%	13.7%
Bathurst	41,300	6.2%	20.1%	56.7%	16.4%	59%	37	2.5	6.0%	4.9%	3	5.4%	16.3%
Larger areas													
NSW	7,480,231	6.2%	18.3%	59.2%	16.3%	61.9%	38	2.6	6.3%		--	2.9%	34.5%
Australia	23,401,892	6.3%	18.5%	59.6%	15.7%	61.6%	38	2.6	6.9%		--	2.8%	26.3%

Notes:
Most data presented in the table derived from the ABS 2016 Census (ABS 2016).
* Data not available as the population size is too small for these details to be provided by the ABS (to protect privacy)
** Data presented for unemployment is based on available data (Australian Government 2018) https://docs.employment.gov.au/documents/lga-data-tables-small-area-labour-markets-march-quarter-2019 .
SEIFA IRSD = index of socioeconomic disadvantage, rank relates to rank in Australia that ranges from
1 = most disadvantaged to 5 = least disadvantaged. Ranks lower than 3 are more disadvantaged than NSW and Australia on average.

Shading relates to comparison against NSW:

- statistic/data suggestive of a potential higher vulnerability within the population to health stressors.
- statistic/data suggestive of a potential lower vulnerability within the population to health stressors.
- statistics/data materially different to that of NSW and Australia, however this indicator is not a clear determinant of higher or lower vulnerability to health stressors.

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

The population adjacent to the proposed site is relatively small and health data is not available that specifically relates to this population.

The project is located within the Western NSW Local Health District (LHD). This district covers a large rural/regional area of approximately 250,000 square kilometres, with the mine development area and surrounding populations located in the south-eastern portion. The LHD extends further to the west (beyond Cobar and Bourke) and north to the NSW boarder, and this area has a population of approximately 270,775.

Table 3.2 presents a summary of the general population health relevant to the area, based on currently available data. The table presents available information on health-related behaviours (i.e. key lifestyle and behaviours factors known to be important to health) and indicators for the burden of disease within the relevant LGAs (where available), the Western NSW LHD and NSW. The values noted in bold are those utilised in this assessment.

Table 3.2: Summary of health indicators/data

Health indicator/data[1]	LGAs			Western NSW LHD	NSW
	Blayney	Cabonne	Bathurst		
Health behaviours					
Adults - compliance with fruit consumption guidelines (2017)	47.0% [2]	48.1% [2]	47.5% [2]	46.2%	46.4%
Adults - compliance with vegetable consumption guidelines (2017)	--	--	--	7.3%	6.6%
Children - compliance with fruit consumption guidelines (2017-2018)	--	--	--	71.6%	64%
Children - compliance with vegetable consumption guidelines (2017)	--	--	--	6.3%	6.7%
Adults - increased lifetime risk of alcohol related harm (2018)	--	--	--	31.2%	31.5%
Adults - body weight (overweight) (2018)	27.1% [2]	29.3% [2]	29.0% [2]	28.3%	32.9%
Adults - body weight (obese) (2018)	43.3% [2]	39.6% [2]	37.1% [2]	30.1%	21.4%
Adults – sufficient physical activity (2018)	--	--	--	53.5%	60.2%
Children – overweight or obese (5-16 years) (2015-2018)	--	--	--	32.8%	22.4%
Children – adequate physical activity (2017-2018)	--	--	--	19.1%	24.2%
Current smoker, adults (2018)	21.0% [2]	17.6% [2]	18.0% [2]	12.4%	10.3%
Burden of disease (rate per 100,000 population unless stated otherwise)					
Morbidity - cardiovascular disease hospitalisations (2016/2018)	1748.7	1699.7	1663.7	1881.7	1671.1
Morbidity – respiratory disease hospitalisations (2017/2018)	1947.7 [2]	2146.0 [2]	2171.9 [2]	2261.1	1714.2
Mortality – all causes, all ages (2016-2017)	624.5	517.9	587.4	625.1	519.6
Mortality – cardiovascular (all ages) (2017)	--	--	--	173.9	134.4
Mortality – cardiovascular (0-74 years) (2011-2015)[3]	47.0	35.4	51.5	--	45.3
Mortality – respiratory (all ages) (2017)	--	--	--	81.4	51.4

Health indicator/data[1]	LGAs			Western NSW LHD	NSW
	Blayney	Cabonne	Bathurst		
Mortality – respiratory (0-74 years) (2011-2015)[3]	25.6	26.2	18.9		15.6
Adults - prevalence of high blood pressure (2013)				37.6%	28.4%
Adult asthma – prevalence (2018)	--	--	--	13.6%	10.5%
Adolescent (2-15 years) – prevalence of current asthma (2016/2017)	--	--	--	12.9%	12.9%

1 Data from NSW Health (2019) Statistics: http://www.healthstats.nsw.gov.au/.

2 Data for LGAs from the Social Health Atlas of Australia: NSW and ACT: http://phidu.torrens.edu.au/current/maps/sha-aust/lga-single-map/nsw-act/atlas.html It is noted that data for fruit consumption, current smoking and overweight and obesity from 2014-2015 and data for respiratory hospitalisations relates to 2016-2017

Shading relates to comparison against NSW:

- statistic/data suggestive of a potential higher vulnerability within the population to health stressors.
- statistic/data suggestive of a potential lower vulnerability within the population to health stressors.
- statistics/data materially different to that of NSW and Australia, however this indicator is not a clear determinant of higher or lower vulnerability to health stressors.

As described above, the Western NSW LHD covers a large area. However, the district comprises rural communities consistent with those present in the local area and hence the data available for the larger LHD are expected to also be representative of the smaller population considered in this project.

Data presented in **Table 3.2**, along with data presented in **Table 3.1**, suggest some of the population in the areas surrounding the site may be more vulnerable to health-related impacts associated with the project, where compared with the general population of NSW. The underlying reasons for this increased vulnerability are expected to be complex, and may include a broad range of lifestyle, behaviour and environmental factors (some of which are included in **Table 3.2**).

Section 4. Health impacts: Air emissions

4.1 Approach

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

- EMM 2020, McPhillamys Gold Project, Air Quality and Greenhouse Gas Assessment. Report dated July 2020, which is a supplementary report to the Air Quality and Greenhous Gas Assessment included in Appendix M of the EIS. These reports are referred to as the AQGGA.

This assessment does not involve a critique of the AQGGA, rather the information and data presented in that report have been assumed to be reliable for the further assessment of health impacts.

The focus of the AQGGA and this assessment of potential health impacts is the emissions to air of dust or particulate matter, metals bound to these particulates and dust that comprises naturally occurring asbestos (NOA). The assessment also addresses gaseous emissions, the focus of which are nitrogen dioxide (NO_2) from fuel combustion and blasting and hydrogen cyanide (HCN) from the processing of gold on the site.

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

4.2 Background on particulate matter

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

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

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

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

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

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

Smaller particles such as $PM_{2.5}$, however, are seen as more significant with respect to evaluating health effects, as a higher proportion of these particles penetrate deep into the

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

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

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

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

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

lungs. Very fine particles, specifically ultrafine particles (PM_1 or $PM_{0.1}$), are also considered to be of importance for the assessment of health effects as these particles penetrate the deepest into the respiratory system.

Figure 4.1 provides a general illustration to provide some context in relation to the size of different particles (discussed above) and relevance/importance for the assessment of inhalation exposures.

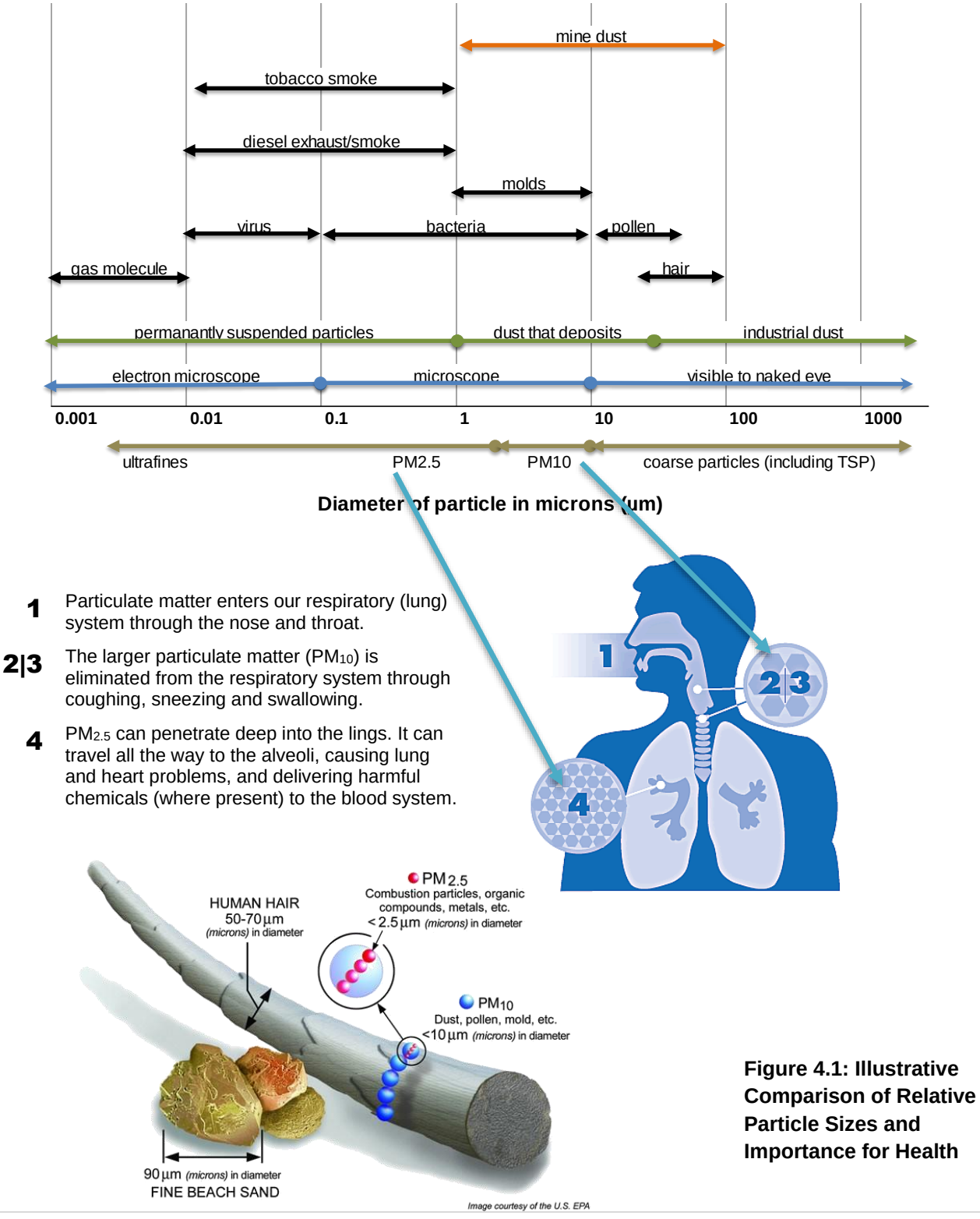


Figure 4.1: Illustrative Comparison of Relative Particle Sizes and Importance for Health

The composition of particulate matter relates to the concentration of metals and metalloids, or organic chemicals, that are bound to and make up the particulate. Different metals, metalloids and organic chemicals have different toxicities when inhaled. Hence when assessing exposures to particulates, especially where the particulates are generated from a mineralised area, it is also important to assess the inhalation toxicity of the individual metals and metalloids on the particulates.

4.3 Naturally occurring asbestos (NOA)

Asbestos is the generic name given to the fibrous variety of six naturally occurring minerals. These minerals have been used in a wide range of commercial products. The minerals are hydrated silicates including a serpentine mineral (*chrysotile*) (also known as 'white asbestos'), and five amphibole minerals (*actinolite, amosite* (also known as 'brown asbestos'), *anthophyllite, crocidolite* (also known as 'blue asbestos'), and *tremolite*) (enHealth 2005, 2013; IARC 1973; USGS 2001).

The structure of these silicate minerals depends on the conditions under which they were formed and they may be long, thin fibres or they may take a range of other shapes. It is when they are in the form of the long, thin fibres that are of most concern. The terms 'asbestos' or 'asbestiform minerals' refer only to those silicate minerals that occur in these fibres and as polyfilamentous bundles. The bundles are composed of extremely flexible fibres with a relatively small diameter and a large length. These fibre bundles have splaying ends, and the fibres are easily separated from one another (HSE 2005; USGS 2001).

The fibres are light and their shape means they do not settle out onto surfaces very quickly unlike larger particles or particles of different shapes so they remain airborne for some time (enHealth 2013).

Asbestos fibres are present in normal urban air. Such fibres are present due to historic uses including in brake pads in cars. They are also present because this is a naturally occurring material, where fibres can be disturbed from rocks containing the mineral deposits (enHealth 2013). Potential health effects associated with exposure to asbestos are further discussed in **Section 4.5.2.3**.

NOA may form in a wide range of rock types however larger accumulations of asbestiform minerals are typically associated with ultramafic rocks. Ultramafic rocks are typically dark rocks rich in magnesium and iron with low silica and potassium and composed mostly of minerals such as olivine and pyroxene. Mafic rocks may also host such minerals. These minerals can be altered by metamorphic processes and hydrothermal fluids to form asbestiform minerals such as chrysotile.

The EIS noted that the Anson Formation, over which the disturbance footprint associated with the mine development lies, has been categorised as having low potential for NOA and noted that the extensive exploration drilling program conducted to date on site had not identified any NOA to date in cores drilled. Notwithstanding the EIS outlined that Regis will follow appropriate procedures for NOA as recommended by SafeWork NSW and in accordance with Regis' naturally occurring asbestos procedure.

Subsequent to the EIS, to further quantify the potential risk of encountering NOA during the life of the project, Regis (2020) has undertaken a review of the project location, in conjunction with information and mapping on the potential presence of geological materials that have the potential to include NOA. This review identified that the project is hosted within intermediate rocks of the Anson

formation which is categorized as low potential for NOA. The high and medium potential NOA mafic units of the Blayney and Byng Volcanics mainly occur on the western side of a large fault known as the Godolphin Fault. This fault is located to the west of the McPhillamys Gold Deposit.

Small quantities of mafic rocks that fit the geological description of the Byng or Blayney Volcanics were intersected in drill core during the resource drilling program at McPhillamys in 2013 and 2016. These mafic rocks are confined to the southern end of the deposit, and the drill core from these units had no visible minerals with an asbestiform habit as documented in the geological logs compiled by Regis geologists. Further work has been undertaken in 2019 and 2020 to determine the potential presence of NOA in the project area, and provide an understanding of the potential location and extent of these materials (where present).

Based on the assessment undertaken by Regis (2020), the intermediate-mafic rock unit in the southern portion of the deposit from the surface may contain NOA. This unit has been extensively drilled and the majority of the material falls outside of the ore zone. It is expected that the rock unit reaches the surface based on modelling from drilling and extends to depths greater than 400m. The unit constitutes less than 1% of the total rock mass proposed to be mined. The location and mining of these materials has been further considered by the air quality modelling specialists, with modelled estimates of NOA in dust predicted and provided for use in this assessment.

4.4 Summary of air modelling

4.4.1 Existing air quality

Existing sources of air pollution in the local area have been identified based on data from the National Pollutant Inventory (NPI) and NSW EPA licence database. The key sources are the natural gas pipeline (APA Moomba to Sydney) located 5 km to the west-southwest of the site, the Cadia Valley Operations Dewatering Facility located 5 km to the south-southwest of the site and the Nestle Purina Pet Care factory located 7km to the west-southwest of the site. Particulate emissions from these facilities are low. Other sources of particulate emissions in the project area include agricultural practices, unsealed roads, combustion from on-road and non-road engines, seasonal emissions from household wood burning, vegetation fires and wind-blown dust (particularly during drought conditions).

Data on existing air quality in the local area has been derived from Regis monitoring of PM_{10} and dust deposition, as well as data on PM_{10} and $PM_{2.5}$ from the NSW Office of Environment and Heritage (OEH) air quality monitoring station at Bathurst. It is noted that the data reported from Bathurst reflects more influences from urban sources within Bathurst, which are not expected to be present in the project area.

Dust levels in the local area are typically low and comply with the NEPM guidelines for PM_{10} with some variability, however in more recent times, specifically 2018, dust levels have been significantly affected by windblown dust generated during drought conditions (including dust storms). Similarly, $PM_{2.5}$ concentrations are general low (based on data from Bathurst only) and in compliance with the NEPM guidelines, but show some variability, with somewhat higher levels in 2018 reflecting the drought conditions. Dust deposition levels in the local area reflect the same pattern with higher levels reported in 2018, but overall compliance with the guidelines.

No data is available in the local or regional area in relation to existing levels of NO_2 or HCN. NO_2 levels in the existing environment were modelled by EMM based on data available from the ACT.

4.4.2 Modelling impacts from the project

Modelling of air quality impacts requires consideration of the local area, specifically the local terrain and meteorological conditions, as well as emissions to air from the various activities relevant to the project.

The local meteorological conditions have been evaluated on the basis of data collected from the on-site meteorological monitoring station (maintained by Regis) as well as data from the Bureau of Meteorology (BoM) automated weather station (AWS) at Orange Airport.

The influence of the local terrain of the project areas and surrounding environments on meteorological conditions have also been taken into account.

Emissions from the project have been estimated on the basis of emission factors for all the relevant activities, volumes to be handled, equipment proposed and processing operations. The emission factors were adopted from NPI emission estimation techniques and USEPA emission factors. Dust control measures were considered in line with guidance from the NSW OEH.

The composition of metals and metalloids in dust emissions were determined ion the basis of geochemistry profiles relevant to waste rock, ore and tailings.

Modelling was undertaken using AEROMOD for the following stages of the project:

- Year 1;
- Year 2;
- Year 4;
- Year 6; and
- Year 8.

The modelling was used to predict air quality impacts over a 10 km x 10km area as well as 89 individual receptor locations reflecting local properties and towns (refer to **Figure 3.1**).

4.5 Assessment of impacts from dust emissions

4.5.1 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 needs to address the inhalation dust particles that are small enough to reach the lungs, namely PM_{10} and $PM_{2.5}$ (refer to **Section 4.2**). This assessment has considered potential health effects that are related to the particle size only, as well as health effects related to the inhalation of various metals (present in the soil and rock) bound to these particles, and the presence of NOA in dust.

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

- Antimony (Sb);
- Arsenic (As);
- Barium (Ba);
- Beryllium (Be)
- Cadmium (Cd);
- Chromium VI (Cr);
- Copper (Cu);
- Lead (Pb);
- Manganese (Mn);
- Mercury (Hg);
- Nickel (Ni);
- Silver (Ag);
- Zinc (Zn).

In addition, the dust generated from the project may also deposit onto the ground, where metals present in the dust may accumulate in topsoil or household dust or 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 4.2 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, as well as exposures that may occur as a result of dust emissions from the project,

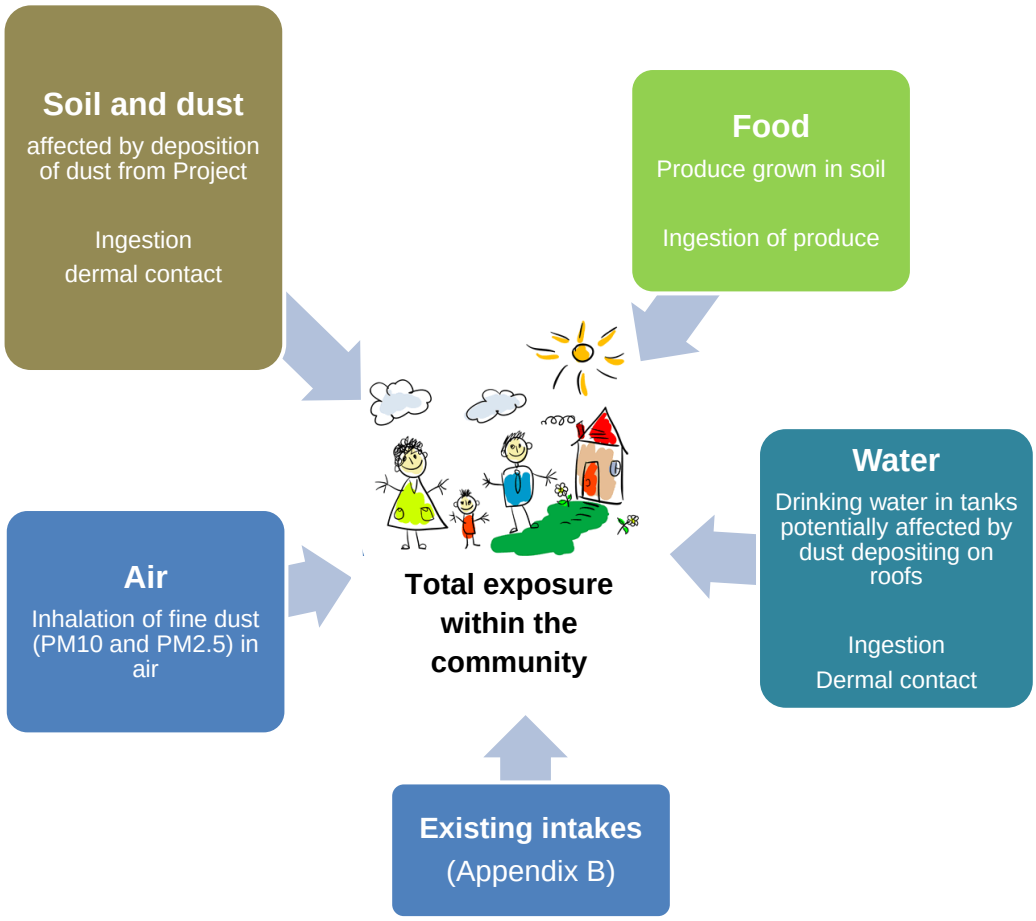


Figure 4.2: Media and pathways evaluated for assessing community exposures to dust emissions

4.5.2 Health effects of particulates

4.5.2.1 General

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 2009b, 2018; WHO 2003a).

4.5.2.2 Health effects of particle size only

There is strong evidence to conclude (USEPA 2012; WHO 2003a, 2013a) 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 effect 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 2009b; 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.

Appendix 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 AQGGA (EMM 2020) 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 private residences/receptors where the cumulative concentrations of PM_{10} exceed the NEPM air guideline for an annual average, noting the maximum predicted is 16.8 µg/m³ which is below the NEPM guideline of 25 µg/m³.

There are no private residences/receptors where the cumulative concentrations of PM_{10} exceed the NEPM air guideline for a 24-hour average, noting the maximum predicted is 46.1 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 **Appendix A**), which are further evaluated below.

$PM_{2.5}$:

There are no private residences where the cumulative concentrations of $PM_{2.5}$ exceed the NEPM air guideline for an annual average, noting the maximum predicted is 6.7 µg/m³ which is below the NEPM guideline of 8 µg/m³.

There are no private residences where the cumulative concentrations of $PM_{2.5}$ exceed the NEPM air guideline for a 24-hour average, noting the maximum predicted is 17.5 µg/m³ which is below the NEPM guideline of 25 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. **Appendix 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 all the receptors is calculated to be 2×10^{-5}, which is lower than the risk level outlined in the NSW EPA Approved Methods (NSW EPA 2016) as unacceptable. Hence there are no health impacts of concern that relate to exposure to $PM_{2.5}$, based on the particle size alone.

4.5.2.3 Health effects of NOA in dust

Where project operations involve disturbance of materials that include NOA, there is the potential for asbestos fibres to be released to air within the dust generated during these works. For asbestos, the health issues relate to inhalation of fibres in the air. Health effects that are of importance in relation to asbestos exposure include pleural plaques, asbestosis, lung cancer and mesothelioma (HACA 2016). It is noted that asbestos fibres are present in ambient air within urban and rural areas, regardless of the project. Hence all members of the population are exposed to some low level of asbestos fibres in air every day.

Asbestos fibres normally constitute only a relatively small fraction of the total fibrous aerosol in ambient air. The biologically more important so-called “critical” fibres are those equal to or longer than 5 µm and having diameters up to 3 µm with an aspect ratio equal to or greater than 3:1 (WHO 2000b).

The risk of developing an asbestos-related disease increases when a larger number of fibres are inhaled, which includes consideration of the duration of the exposure. The greater the exposure, and the longer the time of exposure, the greater the risk. Hence the duration of exposure to asbestos is an important aspect of the assessment of risk. The WHO (WHO 2000a) determined that the most sensitive health effect relates to mesothelioma, where a lifetime exposure to 0.0001 f/ml corresponds to a lifetime risk of 10-6 to 10-5, which is considered acceptable for the development of asbestos guidelines in Australia (NEPC 1999 amended 2013e; WA DOH 2009). For non-occupational exposures enHealth reference acceptable exposures to asbestos in air of <0.01 f/ml. The guideline derived from the WHO is generally consistent with lower levels of background concentrations of asbestos reported in urban air (WHO 2000b) (IARC 2012a).

For the assessment of potential health impacts of exposure to NOA derived from the project, information on the presence of NOA in the materials proposed to be mined during the project has been considered, for each of the scenarios (or model years) assessed for the project (refer to **Section 4.4.2**). As discussed in **Section 4.3**, it has been assumed that NOA is present in 1% of the materials proposed to be mined. This information has been used by the air quality assessment technical specialists to calculate a concentration of asbestos in air at each receptor location as a mass concentration (mg/m^3). The modelling assumes that NOA would be released to air as dust during all relevant activities considered in the project. The predicted concentration of asbestos in air is not specific to the characteristics of asbestos that are of importance to health, hence 100% of the NOA fibres predicted to be in air at each receptor have been assumed to be biologically important. It is also noted that the percentage of NOA fibres in dust generated from materials where NOA is assumed to be present is unknown, hence the modelling has assumed that 100% of these materials comprise NOA. This is a highly conservative assumption as NOA is expected to comprise a small fraction of the rock and mitigation measures proposed to be implemented during operations (to manage dust) would further reduce the amount of NOA generated. Based on the most common measurement technique (phase contrast light microscope) the conversion adopted in this assessment is 30 f/ml per mg/m^3 (NRC 1984; USEPA 1986).

The air guideline adopted relates to a lifetime exposure. Dust emissions that may contain NOA would occur, at most, over the duration of the project which is 15 years. If it was assumed that the generation of dust that included NOA occurred each year of operation an exposure concentration

can be calculated by multiplying the modelled concentrations of NOA in dust in air by a factor that is the ratio of the exposure duration (15 years) to the carcinogen averaging time (70 years as per enHealth guidance (enHealth 2012b). This calculation assumes that the exposure modelled in each project year occurs for 15 years. Actual exposures would be expected to be lower as NOA would not be encountered during every year of works (noting that this is the case for works in Year 8).

Based on the above assumptions and the air modelling undertaken, **Table 4.1** presents the maximum exposure concentration of NOA, relevant to the exposures over the life of the project, and compares this with the lifetime air guideline from the WHO.

Table 4.1: Review of predicted exposure concentrations for NOA – maximum from all receptors

Project year	Asbestos exposure concentration (f/ml)
Year 1	0.00003
Year 2	0.00007
Year 4	0.00008
Year 6	0.00008
Year 8	0 (no NOA predicted to be accessed during these works)
Asbestos air guideline for lifetime exposures	**0.0001**

Review of the above table indicates that the maximum predicted exposure concentrations are below the adopted guideline from the WHO. Hence risks associated with potential exposure to NOA in air as a result of the project are considered to be low and acceptable.

4.5.2.4 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).

Appendix B presents toxicity summaries for the metals evaluated in this assessment.

Table 4.2 presents a summary of the acute inhalation guidelines adopted in this assessment. **Table 4.3** 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 will occur.

Table 4.2: Summary of acute inhalation guidelines

Metal	Acute inhalation guideline (mg/m^3)	Averaging time	Source
Antimony	0.001	1-hour	ATSDR[1]
Arsenic	0.003	1-hour	TCEQ[2]
Barium	0.01	1-hour	OMECC[4]
Beryllium	0.00001	1-hour	OMECC[4]
Cadmium	0.0054	1-hour	TCEQ[2]
Chromium	0.0013	1-hour	TCEQ[2]
Copper	0.1	1-hour	OEHHA
Lead	0.0005	1-hour	OMECC[4]
Mercury	0.0006	1-hour	OEHHA
Manganese	0.0091	1-hour	TCEQ[2]
Nickel	0.0011	1-hour	TCEQ[2]
Silver	0.001	1-hour	OMECC[4]
Zinc	0.12	1-hour	OMECC[4]

Notes:

1 ATSDR acute inhalation value (ATSDR 2019b). It is noted that the ASTDR values relate to exposures from 1 hour up to 14 days, hence application of the guideline for peak 1 hour exposures is considered conservative

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 concentrations in particulates <10 microns in size, and have been adopted for arsenic (TCEQ 2012), cadmium (TCEQ 2016), manganese (TCEQ 2017), 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

4 Acute ambient air guideline value available from the Ontario Ministry of the Environment and Climate Change (OMECC): https://www.ontario.ca/page/ontarios-ambient-air-quality-criteria-sorted-contaminant-name. The value for barium, beryllium, lead, silver (assumed to be soluble compounds) and zinc (assuming zinc oxide on particulates) relates to a 24-hour average, which has been conservatively adopted for the assessment of a peak 1 hour average.

Table 4.3: Summary of chronic guidelines, toxicity reference values (TRV) (annual average) and dermal absorption parameters

Metal[2]	Inhalation TRV (mg/m^3)	Ingestion TRV (mg/kg/day)	Dermal TRV[4] (mg/kg/day)	Dermal absorption[3] – for contact with soil (unitless)	Dermal permeability[3] - for contact with water (cm/hr)
Antimony	0.003	0.0009	0.00014	Negligible	0.001
Arsenic	0.001	0.002	0.002	0.005	0.001
Barium	0.001	0.2	0.014	Negligible	0.001
Beryllium	0.00002	0.002	0.000014	Negligible	0.001
Cadmium	0.000005	0.0008	0.0008	Negligible	0.001
Chromium	0.0001	0.001	0.001	Negligible	0.002
Copper	0.49	0.14	0.14	Negligible	0.001
Lead	0.0005[1]	Children = 0.0014 Adults = 0.0006	Children = 0.0007 Adults = 0.0003	Negligible	0.0001
Manganese	0.00015	0.14	0.14	Negligible	0.001
Mercury	0.0002	0.0006	0.00004	0.001	0.001
Nickel	0.00002	0.012	0.012	0.005	0.0002
Silver	0.02	0.0057	0.00023	Negligible	0.0006
Zinc	1.75	0.5	0.5	0.001	0.0006

Notes:

1 NEPM Ambient Air Quality (NEPC 2016)

2 Refer to **Appendix B** for details in relation to the toxicity reference values adopted for these 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, 4% for silver, 7% for inorganic mercury and barium, 15% for antimony and 0.7% for beryllium (refer to **Appendix B**)

When undertaking any assessment of potential risks to human health, the threshold TRVs outlined above relate to intakes from all sources, not just the project. Metals are ubiquitous in the environment and hence for many of the metals evaluated these will be present in soil, water, food and air. In some cases, the concentration of metals naturally occurring in the environment is so low that it is considered negligible. However, for some more commonly occurring metals in the environment such as arsenic, cadmium, copper, manganese, mercury, nickel and zinc intakes from other sources are significant. **Appendix B** also includes a summary of the intakes assumed from other sources.

4.5.3 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 intake of the chemical. This assessment has also addressed acute inhalation exposures.

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

The exposure assessment involves the following:

- Identification of the population(s) that might be exposed – for this assessment residents in the surrounding community areas (i.e. at all receptor locations) have been addressed, which include adults and children. For this assessment the maximum impacts predicted at all receptors has been evaluated;
- 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 (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.

Appendix C 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. The appendix also includes the assumptions adopted for characterising exposures for adults and children, as well as the methodology used to estimate concentrations in soil, rainwater tanks and produce.

4.5.4 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 considers acceptable levels of risk outlined in the NEPM (NEPC 1999 amended 2013d), 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 Hazard or Risk Index (HI/RI), which is the sum of all ratios (termed Hazard or Risk Quotients (HQ/RQ)) over all relevant pathways of exposure. These are calculated using the following equations:

Inhalation exposures

$$\text{Risk Quotient(RQ)(Project)} = \frac{\text{Exposure Concentration (Project)}}{\text{(Inhalation toxicity reference value)*(100\%-\%TRV from background sources)}}$$

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

$$\text{Risk Quotient(RQ)(Project)} = \frac{\text{Daily Chemical Intake (Project)}}{\text{(Oral or dermal toxicity reference value)*(100\%-\%TRV from background sources)}}$$

Total risk

$$\text{Risk Index(RI)} = \sum_{\textit{All pathways}} \text{RQ}$$

The interpretation of an acceptable RI needs to 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 2013d);
- 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.

4.5.5 Acute inhalation exposures

The calculated RI for acute inhalation exposures to the maximum 1-hour average concentration of metals attached to particulates, for the maximum impacted receptor location, are presented in **Table 4.4**. The calculated RI relate to exposures by all members of the community, of all ages. The table presents the calculated total RI for exposure to all metals evaluated. The calculations are included in **Appendix D**.

Table 4.4: Calculated risk indices – Acute inhalation exposures to metals in air from the project

Project scenario	Calculated RI – Total for all metals
	Maximum of all receptors
Year 1	0.030
Year 2	0.060
Year 4	0.060
Year 6	0.048
Year 8	0.047
Acceptable RI	**≤ 1**

Review of **Table 4.4** indicates that all calculated RI, related to acute exposures to all metals in dust, at all locations, are well below 1 and hence there are no acute inhalation exposure risks of concern for the project.

It is noted that the table presents the maximum RI, with inhalation exposures at all other locations in the community, lower than presented in **Table 4.4**, and are therefore also not considered to be of concern.

4.5.6 Chronic exposures

4.5.6.1 Inhalation exposures from project emissions

The maximum calculated RI for chronic inhalation exposures of metals attached to particulates has been calculated for the maximum impacted receptor location. These calculations use an annual average air concentration, which has been provided by the air quality assessment technical specialists.

The calculated RI relate to exposures by all members of the community, of all ages. The detailed calculations are presented in **Appendix E**.

Table 4.5: Calculated risk indices – Chronic inhalation exposures to metals in air from the project

Project scenario	Calculated RI – Total for all metals Maximum of all receptors
Year 1	0.013
Year 2	0.026
Year 4	0.027
Year 6	0.023
Year 8	0.022
Acceptable RI	**≤ 1**

Review of **Table 4.5** indicates that all calculated RI, related to chronic inhalation exposures to all metals in dust emitted to air from the project, are well below 1. 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, are lower than presented in **Table 4.5** and are also considered to be negligible.

4.5.6.2 Multi-pathway exposures from project emissions

The assessment of multi-pathway exposures requires the consideration of the amount of dust that may deposit onto surfaces (soil and roof tops). The air quality assessment technical specialists have provided dust deposition rates for metals in TSP, as an annual average deposition rate. This has been used in this assessment.

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 private residence for each of the project scenarios, are presented in **Table 4.6** for young children and adults respectively. The table presents the total RI for exposure to all metals (as a sum). The RI's are presented to 1 significant figure, consistent with the level of

accuracy and uncertainty inherent in the calculations. The detailed calculations are presented in **Appendix F**.

Table 4.6: Calculated risk indices for multi-pathway exposures to metal deposited from the project

Project scenario	RI Calculated for each exposure pathway - maximum impacted private 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
Young children						
Year 1	0.07	0.009	0.03	0.0001	0.003	0.006
Year 2	0.2	0.02	0.07	0.0002	0.005	0.01
Year 4	0.2	0.02	0.08	0.0002	0.006	0.01
Year 6	0.2	0.02	0.08	0.0002	0.006	0.01
Year 8	0.2	0.02	0.08	0.0002	0.006	0.01
Adults						
Year 1	0.009	0.009	0.01	0.00005	0.001	0.002
Year 2	0.02	0.02	0.03	0.0001	0.002	0.003
Year 4	0.02	0.02	0.03	0.0001	0.003	0.004
Year 6	0.02	0.02	0.03	0.0001	0.002	0.004
Year 8	0.02	0.02	0.03	0.0001	0.002	0.004
Acceptable RI	**≤ 1**	**≤ 1**	**≤ 1**	**≤ 1**	**≤ 1**	**≤ 1**

Review of **Table 4.6** indicates that all calculated RI's for the most impacted receptor, related to chronic exposures to all metals that may be deposited to soil or other surfaces from dust emissions from the project are all below 1. This indicates that the incremental increase in exposure to metals via these multi-pathway exposures from dust generated from the mine is very low and would be considered negligible. In relation to emissions that occur over the different scenarios, or years of operation, there is little difference between the calculate RIs for the scenarios, however the calculated risks are observed to be slightly higher for Year 4.

Risks related to exposures at all other receptors surrounding the site are lower than presented in **Table 4.6**, and are also considered to be negligible.

It is noted that there is the potential for residents on a property surrounding the site to be exposed to dust impacts via a range of different pathways. For a representative and potentially higher impacted year, Year 4, **Table 4.7** presents potential RI's associate with combined exposures.

Table 4.7: Calculated risk indices for combined exposure pathways – Year 4, maximum receptor exposures

Exposure scenario	Calculated additive RI	
	Young children	Adults
Inhalation (I) + soil ingestion (SI) and soil dermal contact (SD)	0.2	0.05
I + SI + SD + water ingestion (WI) + water dermal contact (WD)	0.2	0.07
I + SI + SD + WI + WD + home consumption of fruit and vegetables (FV)	0.3	0.1
I + SI + SD + WI + WD + consumption of homegrown eggs (E)	0.2	0.07
I + SI + SD + WI + WD + FV + E	0.3	0.1
I + SI + SD + WI + WD + FV + E + home consumption of beef	0.3	0.1
I + SI + SD + WI + WD + FV + E + home consumption of milk	0.3	0.1

Review of **Table 4.7** indicates that even where there may be properties located in the community surrounding the site where multiple exposure may occur (which would depend on the use of these properties), the calculated RI's remain below 1 and are representative of negligible risks to human health.

4.6 Assessment of health impacts – nitrogen dioxide

4.6.1 General

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

In terms of health effects, nitrogen dioxide (NO_2) is the only oxide of nitrogen that may be of concern (WHO 2000f). NO_2 is a colourless and tasteless gas with a sharp odour. NO_2 can cause inflammation of the respiratory system and increase susceptibility to respiratory infection. Exposure to elevated levels of NO_2 has also been associated with increased mortality, particularly related to respiratory disease, and with increased hospital admissions for asthma and heart disease patients (WHO 2013b). Asthmatics, the elderly and people with existing cardiovascular and respiratory disease are particularly susceptible to the effects of NO_2 (Morgan, Broom & Jalaludin 2013; NEPC 2010). The health effects associated with exposure to NO_2 depend on the duration of exposure as well as the concentration.

The most current review undertaken by the USEPA (USEPA 2015) identified that exposure to NO_2 was causal in relation to respiratory effects (which are reflected in relationships established for all-cause mortality), that these effects were independent of exposure to particulates and there was no threshold for these effects. Hence the assessment of potential exposure to NO_2 has considered the available Australian guidance (NEPC 2016) that relates to cumulative exposures (background plus the project) and incremental exposures (risks related to project related emissions).

4.6.2 Assessment of cumulative exposures to nitrogen dioxide

The NEPC has established ambient air quality guidelines for the assessment of acute (short-term, based on 1-hour averages) and chronic (long-term, based on annual averages) exposures to NO_2 (NEPC 2016). These guidelines are designed to be protective of adverse health effects in all individuals (refer to **Appendix A**) for exposure to all sources of NO_2.

The modelling of air emissions from the project presented in the AQGGA estimated the following for cumulative concentrations of NO_2:

- Maximum 1-hour average concentration of NO_2 (relevant to all years of operation) of 171.7 $\mu g/m^3$, which is well below the current NEPC guideline of 246 $\mu g/m^3$; and
- Maximum annual average concentration of NO_2 (relevant to all years of operation) of 14.2 $\mu g/m^3$, which is well below the current NEPC guideline of 62 $\mu g/m^3$.

On the basis of the above there are no issues of concern in relation to cumulative exposures to NO_2 in the community surrounding the project.

4.6.3 Assessment of incremental impacts

In addition, a calculation of incremental changes in NO_2 exposures from the project alone has been undertaken, focusing on the key health endpoint, mortality (all causes and all ages). This health endpoint captures all other health effects found to be causally related to NO_2 exposure and is the most significant in terms of calculating risks related to changes in NO_2 exposures. **Appendix A** includes discussion on the methodology and calculations undertaken to determine an incremental risk. The maximum incremental risk for exposure to changes in NO_2 at all the receptors is calculated to be 2×10^{-5}, which is lower than the risk level defined in the NSW EPA Approved Methods (NSW EPA 2016) as unacceptable. Risks at all other receptor locations are lower than the maximum presented here. Hence there are no health impacts of concern that relate to exposure to project related emissions of NO_2.

4.7 Assessment of hydrogen cyanide exposures

The AQGGGA evaluated potential emissions to air of gaseous hydrogen cyanide from project operations. The AQGGA has predicted the maximum 1-hour average concentration, from all the receptors evaluated, of hydrogen cyanide in air as 69.6 µg/m³.

In air, cyanide is present as gaseous hydrogen cyanide, with a small amount present in fine dust particles. The majority of the population is exposed to very low levels of cyanide in the general environment. The principal features of the toxicity profile for cyanide are its high acute toxicity by all routes of exposure, with a very steep and rate-dependent dose–effect curve. The toxicity of hydrogen cyanide gas is dominated by the acute health effects, which commonly result in effects prior to determining any chronic health effects (WHO 2004c). Hence the protection of acute inhalation effects associated with hydrogen cyanide is expected to be protective of chronic health effects.

In relation to the assessment of acute inhalation exposures, a 1-hour guideline of 2000 µg/m³ is based on no adverse health effects in humans (NRC 2002), with a lower value of 340 µg/m³ available from OEHHA (OEHHA).

The maximum predicted concentration of hydrogen cyanide in air as a 1-hour average, is well below these health based levels.

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.

4.8 Uncertainties

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

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

The impact 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 assessment

methodology, uncertainties and limitations are inherent in the health impact assessment process. This section discusses the uncertainties and limitations associated with this health impact 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

No information is available to provide an understanding of existing (background) exposures to metals and metalloids in the environment. Hence generic information about potential levels that may be present in soil, water, air and dietary intakes have been adopted. While conservative assumptions have been adopted that are likely to overestimate intakes from existing sources, there is the potential for these intakes to be underestimated, particularly in naturally mineralised areas.

In relation to the assessment of impacts from the project, this assessment has relied on the modelling of emissions as presented in the AQGGA. The AQGGA 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 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. As a result, the predicted impacts from dust are expected to be an overestimate.

Exposure assessment

Risk and impact assessments require the adoption of several assumptions in order to assess potential human exposure. This 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 **Appendix C**. 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 updake 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 will 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 will 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 low (considered negligible) and exposures (including concentrations) would need to increase my many orders of magnitude for risks to be considered more 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 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.

4.9 Outcomes of health impact assessment

Table 4.8 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 4.8: Summary of health risks – air quality

Air emissions	
Impacts	Based on the available data and information in relation to emissions of dust, NOA, metals and metalloids that may be present on the dust, nitrogen dioxide and hydrogen cyanide from the project, potential impacts on the health of the community have been assessed. The impact assessment has concluded there are no health risk issues of concern relevant to the project.
Mitigation	The AQGGA has identified a range of mitigation measures that would minimise dust emissions, diesel combustion emissions and blast fumes. In addition, the AQGGA has outlined air quality monitoring proposed to be continued for the duration of the project, which includes dust-deposition monitoring and real-time particulate monitoring. An air quality management plan, which includes a monitoring plan, would be developed for the project, documenting locations, monitoring methods and reporting responsibilities.

Section 5. Health impacts: Noise

5.1 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:

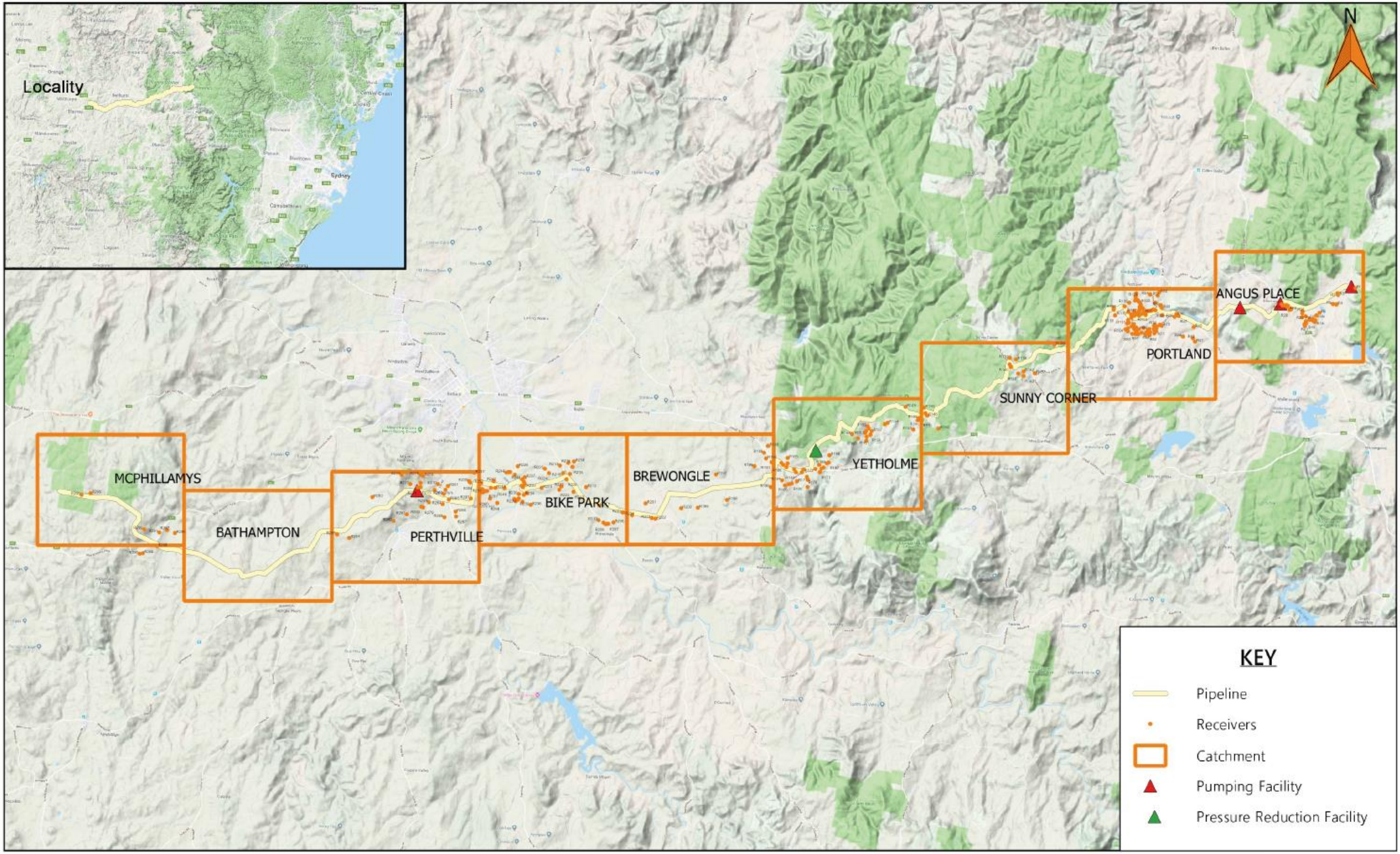
- MAC 2019a, Noise and Vibration Impact Assessment, McPhillamys Gold Project, Blayney, NSW. Prepared by Muller Acoustic Consulting (MAC) for EMM Consulting Pty Limited, dated August 2019. Appendix L of the EIS. This report is referred to as the NVIA.
- MAC 2019b, Noise and Vibration Impact Assessment, McPhillamys Gold Project – Pipeline Development Lithgow to Blayney, NSW. Report prepared by Muller Acoustic Consulting (MAC) dated July 2019. Appendix AA of the EIS. This report is referred to as the NVIA – Pipeline.
- MAC 2020a, Amended Noise and Vibration Impact Assessment. McPhillamys Gold Project, Blayney, NSW. Prepared by Muller Acoustic Consulting (MAC) for EMM Consulting Pty Limited, dated 17 July 2020.
- MAC 2020b, McPhillamys Gold Project – Pipeline Development, Lithgow to Blayney, NSW. Prepared by Muller Acoustic Consulting (MAC) for EMM Consulting Pty Limited, dated 1`4 August 2020.

This assessment does not involve a critique of the NVIA or the NVIA – Pipeline or the amended assessments, rather the information and data presented in that report have been assumed to be reliable for the further assessment of health impacts.

For the assessment of noise impacts relevant to the mine development area, the NVIA has considered the same 89 receptors (or receivers) that were evaluated within the air quality assessment (refer to **Figure 3.1**). These receptors have been grouped in the NVIA into catchments, with the following catchments defined:

- Distant Rural – which are rural receptors that are typically rural in nature with low background noise levels and are generally more than 2 km from the project area boundary. This includes receptors R01 to R14 and R52 to R88;
- Kings Plains – which are receptors situated in the Kings Plains locale that are in closer proximity to the Mid Western Highway. This includes receptors R15 to R17 and R25 to R33;
- Walkom Road – which are receptors in the Kings Plains locale that are not influenced by road traffic from the Mid Western Highway. This includes receptors R18-R24; and
- Sturgeon Hill – which are receptors to the south west of the project mine development. This includes receptors R34 to R51.

Existing, or background, noise levels in the community, at the receptors evaluated, have been determined on the basis of available noise monitoring data. The background noise levels adopted in the assessment, termed a Rating Background Level (RBL, which relates to noise over a 15-minute period) are 35 to 36 decibels (A-weighted) (dBA) during the day, range from 30 to 34 dBA during the evening and are 30 dBA at night for the mine development area.



MAC
Muller Acoustic Consulting

Figure 5.1

Receivers, Catchments, Pump Stations and Pipeline Alignment
REF: MAC180742

The RBLs for areas located along the pipeline range from 35 to 47 dBA during the day, 30 to 37 dBA during the evening and 30 dBA at night (in all areas).

The assessment of noise impacts associated with the construction and operation of the pipeline, presented in the NVIA – Pipeline, has evaluated impacts at 329 community receptors (receivers) located along the pipeline in areas potentially impacted by noise from the project, particularly during construction (as shown in **Figure 5.1**)

5.2 Health impacts associated with noise

Environmental noise has been identified (I-INCE 2011; WHO 2011b, 2018)[7] 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, 2011b, 2018):

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

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

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

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 5.2**.

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

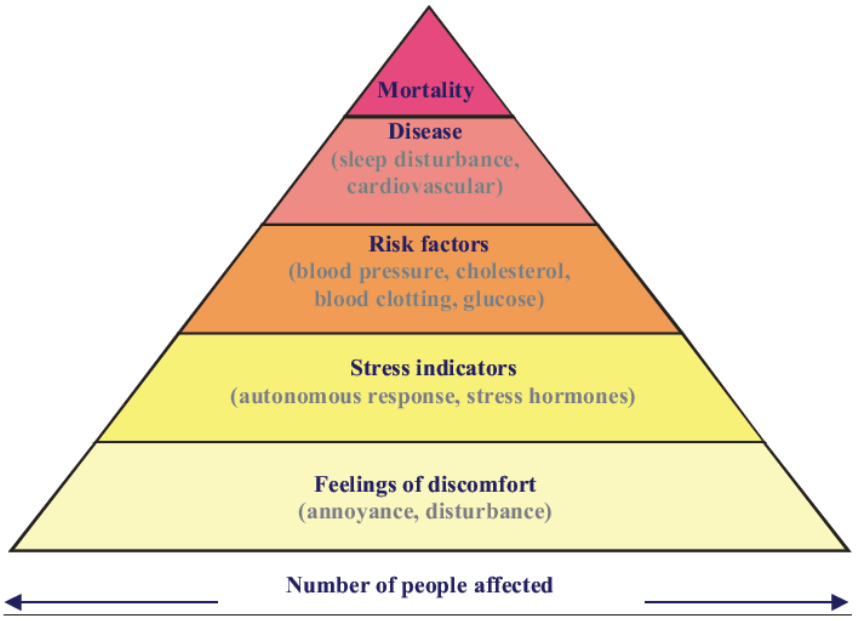


Figure 5.2: Schematic of severity of health effects of exposure to noise and the number of people affected (WHO 2011b)

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 2011b, 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 2011b, 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 2011b, 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 2011b, 2018).

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

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

5.3 Review of the noise guidelines adopted

Construction:

Construction noise criteria have been adopted from the Interim Construction Noise Guideline (ICNG) (NSW DECC 2009)[8] which provide management levels relevant to the assessment of noise impacts above the RBL.

For the mine development area the construction noise management levels during standard hours are RBL + 10 dBA = 45 to 46 dBA and outside standard hours the guidelines are RBL + 5 dBA = 35 to 39 dBA for evening and 35 dBA for the night. While these criteria may result in some construction noise being noticeable, the noise criteria adopted for the project will be protective of health, including annoyance and sleep disturbance, where they relate to outside noise levels (WHO 1999b, 2009a).

For areas located along the pipeline, the guidelines relevant to construction during standard hours are RBL + 10 dBA = 45 to 57 dBA and outside standard hours the guidelines are RBL + 5 dBA = 35 to 42 dBA during the evening and 35 dBA at night. Noise levels of 75 dBA and above are considered to be highly noise affected for locations along the pipeline. While these criteria may result in some construction noise being noticeable, the noise criteria adopted for the project will generally be protective of health, including annoyance and sleep disturbance, where they relate to outside noise levels (WHO 1999b, 2009a). The exception to this is the daytime guideline adopted for the Yetholme receptors where the guideline is 57 dBA, which is at a level at which there may be some interference with speech and speech intelligibility indoors, including within schools. The level of interference would depend on the reduction in noise from outside to inside. Where windows and doors are closed it is likely that the noise reduction would be sufficient to prevent construction noise from interfering with speech at receptors in this area.

[8] DECC – NSW Department of Environment and Climate Change.

Road traffic:

Road traffic noise was assessed on the basis of the NSW Road Noise Policy (NSW DECCW 2011)[9], as it applies to existing residence affected by additional traffic. This provides a guideline of 60 dBA as $LA_{eq,15\ hour}$ (day and evening) and 55 dBA as $LA_{eq,9\ hour}$ (night) for the Mid Western Highway as well as Castlereagh Highway and Great Western Highway, and 55 dBA (day and evening) and 50 dBA (night) for Dungeon Road as well as the numerous other local roads relevant to the pipeline corridor.

In addition, an increase in noise levels of more than 12 dB A from road traffic requires consideration of mitigation. 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, 2009a).

Operations:

Noise guidelines adopted in the Noise Impact 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 dBA. In addition, consideration has also been made to noise amenity, with the project noise trigger levels adopted based on the lower noise criteria relevant to intrusiveness and amenity.

For the mine development area, the intrusive noise trigger levels adopted were $LA_{eq,15\text{-}minute}$ of 40 to 41 dBA during the day, 35-39 dBA during the evening and 35 dBA during the night. In relation to the pipeline the only operation noise sources are pumps and the pressure reducing system, which may operate at night. Where this may occur the intrusive noise guideline adopted for all residential receptors is 35 dBA. These noise trigger levels are sufficiently low to be protective of health, based on available guidance from the WHO (WHO 1999b, 2011b). The NPfI provides guidance on the interpretation of noise impacts in relation to these trigger levels, particularly in relation to predicted/estimated changes in noise levels.

Noise amenity criteria for the mine development area (noted above) as $LA_{eq,15\text{-}minute}$ are 53 dBA during the day, 48 dBA during the evening and 43 dBA during the night-time period. The noise amenity criteria are more specifically used to evaluate cumulative noise from a number of industrial sources. It is noted that the lower of the criteria for intrusive noise and noise amenity is adopted for the assessment, which are the criteria for intrusive noise (discussed above).

Maximum noise levels were also established based on the NPfI guidance (NSW EPA 2017). The maximum noise criteria are set to protect residence from sleep disturbance and for this project, an LA_{max} of 52 dBA is relevant to the night-time period relevant to all areas (mine development area and pipeline). This maximum noise level is sufficiently low to be protective of health, based on available guidance from the WHO (WHO 1999b).

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

The NVIA also evaluated criteria provided in the Voluntary Land Acquisition and Mitigation Policy (NSW DPE 2018) to identify receptors that may be considered to have negligible, marginal, moderate or significant noise impacts from the project and where mitigation and/or voluntary land acquisition may be applicable.

Blasting impacts have been evaluated in accordance with criteria established to protect human annoyance (ANZEC 1990). Provided the human comfort criteria are met, there would be no concern in relation to health impacts.

5.4 Review and assessment of health impacts from noise

5.4.1 Noise modelling

Assessment of noise impacts during construction and operation at all locations involved consideration of the relevant construction activities (equipment used, hours of use and location of use), including blasting (where required). Noise impacts were modelled in the NVIA and NVIA – Pipeline and amended assessments using DGMR's iNoise noise modelling software, which takes into account the topography, meteorology, activities being undertaken, the ground type as well as the presence of any barriers. The modelling focused on worst-case meteorological conditions for the assessment of noise impacts.

Following submission of the EIS, additional detailed design has been undertaken, primarily to reduce noise emissions through optimisation of mobile equipment and the mining schedule. Project amendments to reduce noise impacts include:

- Selection of mobile equipment with lower noise emissions
- General reduction of the number of equipment in the mining fleet
- Revised waste schedule and the development of the Waste Rock Emplacement (WRE) to optimise night and daytime dump locations, while minimising noise emissions to Kings Plains
- Optimisation of haul roads by adding a northern exit from the pit
- Redesign of the processing plant and mine infrastructure areas, enhancing natural topographical shielding
- Scheduling of noise intensive works such as the construction of the pit amenity bund and Water Management Facilities (WMF) during the construction phase with works to occur during the daytime only.

These mitigation measures have been included in the modelling of operational noise impacts from the project.

5.4.2 Construction noise

Construction noise impacts were modelled for the first 6 months of Year 1.

Project

The Amended NVIA (MAC 2020a) modelled construction noise impacts at each of the receptors, without additional noise mitigation or management measures. All predicted noise impacts were below the relevant construction noise guidelines at all receptors.

Pipeline

The NVIA – Pipeline considered noise impacts from transient activities as well as static activities. During transient activities (such as clearing, grading, trenching, backfill and restoration) there modelling indicates there are exceedances of the relevant guidelines at all receptors in close proximity to the works, noting that for most these exceedances are only expected to be for a short duration (i.e. either one to two shifts or up to a few days). For the static construction activities, such as under boring, exceedances are anticipated to occur for a few shifts during peak construction activities. Notwithstanding, construction noise levels are predicted to satisfy the highly noise affected criteria 75 dBA as $L_{Aeq,15\text{-}minute}$ for all activities.

The Revised Pipeline Development Noise and Vibration Assessment (MAC 2020b) identifies a hierarchical strategy to minimise noise impacts during construction of the pipeline development. A formal complaints and grievances procedure will be developed for the construction phase of the pipeline development. This procedure will be aligned with that prepared for the mine development. Regis will communicate the procedure to all directly affected pipeline corridor property owners and near neighbours.

As the construction noise guidelines adopted for this assessment are protective of health, there are no health impacts of concern in relation to construction activities (where assessed separately) for most of the receptors surrounding the mine development area. It is expected, however that some construction noise may be noticeable at some receptors in this area. For receptors located along the pipeline, there is the potential for exceedance of these guidelines. These exceedances are expected to be of short duration only (up to a few days). In relation to health effects, these exceedances may result in annoyance, however the potential for longer term effects is consider low. It is therefore important that the noise mitigation measures outlined in the NVIA – Pipeline are implemented to minimise noise and health impacts.

5.4.3 Operational noise

Approach

The operational noise assessment presented in the Amended NVIA (MAC 2020a) for the mine development area has considered noise impacts from the project operations as well as road impacts. Noise modelling considered operations during:

- Year 1 (months 7 to 12), which includes some ongoing construction works and some operations.
- Year 2 Project fully operational. Waste rock hauled to the southern end of the waste rock emplacement during the daytime to build up the southern amenity bund and the northern end of the waste rock emplacement during the night time.
- Year 4 continued operations. Haulage 24/7 to both northern and southern end of the waste rock emplacement. Rehabilitation fleet continues to build southern amenity bund during daytime hours only.
- Year 6 Peak material movement occurs during this year with additional fleet required. Waste rock hauled to both southern and northern areas of waste rock emplacement 24/7. Southern amenity bund is completed.

- Year 8 Continued operations, waste rock generally placed in centre of waste rock emplacement 24/7. Progressive rehabilitation.
- Year 10-11 Continued operations with reduced mining fleet. Waste emplacement reached final landform. Progressive rehabilitation.

Activities that are proposed to be undertaken during these project years, including the time and location of operation, and sound power levels generated by the equipment/activities, have been considered in the noise model. The modelling has considered the noise mitigation and management measures proposed for the project.

In relation to the pipeline, operational noise associated with the operation of four pump stations has been evaluated. The assessment evaluated noise impacts at all receptors located within 1,000 m of each of these facilities.

Noise impacts – Mine development area

Predicted noise levels during the daytime are not expected to exceed the adopted noise guidelines throughout the Project. Predicted noise levels during evening and night time periods are below the adopted noise guidelines for all receivers except for the majority of receivers in the Kings Plains and Walkom Road NCAs during Project Year 1 and Project Year 4 where levels are expected to exceed the PNTLs by up to 2dB.

Where predicted noise levels exceed the adopted noise guidelines by up to 2dB, the residual impact is determined to be “Negligible” in accordance with NPI methodology. Hence, the residual noise impacts at those receivers during Project Year 1 and Project Year 4 would be considered negligible, being of a minor magnitude that can be managed accordingly in conjunction with the appropriate noise monitoring system and management controls.

A single receiver (R28a) is expected to experience noise levels up to 3dB over the adopted noise guidelines during the evening period for a brief period in the second half of Project Year 1, during the initial development of the pit. This exceedance of the PNTLs is predicted for a period of 2 to 3 months during the development of the pit amenity bund and as the pit develops from a depth where there is minimum natural shielding. The significance of this impact in accordance with NPI methodology is marginal and of short duration.

Predicted noise level in the EIS NVIA (MAC 2019a) resulted in numerous occurrences of noise levels exceeding the adopted noise guidelines by more than 3dB, but not exceeding 5dB. Predicted noise levels in the Amended NVIA (MAC 2020a) are generally within 2dB of the adopted noise guidelines at all receivers throughout the Project life and are lower than those presented in the EIS NVIA. The reduction in predicted noise levels at receivers in Kings Plains is attributable to the revised waste schedule and WRE design, selection of quieter mobile mining fleet and optimisation of that equipment.

Low frequency noise impacts were assessed to be negligible or imperceptible.

In addition to the above the following is noted in relation to the predicted noise levels at the surrounding receptors:

- Predicted $L_{Aeq,15\text{-}minute}$ noise predictions in the Amended NVIA can be converted to represent $LA_{eq,day}$, $LA_{eq,evening}$ or $LA_{eq,night}$[10].
- Using these noise levels, all predicted noise levels during the day (maximum $LA_{eq,day}$ = 38 dBA) and night (maxim $LA_{eq,night}$ = 35 dBA) (taken to be outdoor noise predictions at each receptor) are below health based noise guidelines.

Hence, from a health perspective, there are no noise impacts identified that would be considered to be of concern to community health.

There were no exceedances of the maximum noise level, at any of the receptors, for any of the project years and time periods evaluated. As these noise criteria are protective of health, there would be no health impacts of concern in relation to the maximum noise levels related to the project.

Noise impacts – Pipeline

The assessment of noise impacts associated with the operation of the pipeline, as presented in the Amended NVIA – Pipeline (MAC 2020b), did not identify any receptors where predicted noise levels would exceed the adopted noise guidelines.

Operational noise emissions from the pump stations are anticipated to be negligible at adjacent receivers to each site, although this assumes some form of container or enclosure is adopted for each pump station. As the project pipeline will be largely underground once it is operational, amenity or access impacts to landowners or the public from the pipeline itself are not expected. On this basis there are no health impacts of concern in relation to noise derived from the operation of the pipeline.

5.4.4 Road noise

For the mine development area, as presented in the NVIA (MAC 2019a) and Amended NVIA (MAC 2020a), assessment of road noise impacts considered expected road traffic volumes relevant to the project, on Dungeon Road and the Mid Western Highway. The assessment determined that noise at all receptors along these roads will comply with the relevant noise guideline.

In relation to the pipeline, assessment of road traffic noise during construction determined that noise at all receptors along relevant roads will comply with the adopted noise guidelines.

Hence there are no noise impacts of concern to community health, relevant to the project.

[10] Conversion of $LA_{eq,15\text{-}minute}$ to $LA_{eq,period}$ is outlined in the NPfI NSW EPA 2017, Noise Policy for Industry (and in the Noise Impact Assessment), where $LA_{eq,period}$ = $LA_{eq,15\text{-}minute}$ - 3 dB.

5.4.5 Blasting

For the mine development area, as presented in the NVIA (MAC 2019a) and Amended NVIA (MAC 2020a), air blast overpressure and vibration levels are predicted to be below the adopted guidelines at all receptors.

For the pipeline, the assessment of construction activities has identified offset distances at which the air blast overpressure and vibration guidelines are met. These offsets would be used to adjust the blasting activities at different locations such that the guidelines can be met at all receptors.

Based on the above, and where management measures outlined in the NVIA – Pipeline (2019b and 2020b) are implemented, there are no health impacts related to blasting activities relevant to the surrounding community.

5.5 Uncertainties

The assessment of presented in relation to potential noise impacts, and the potential for impacts on community health as a result of changes in noise as a result of the project 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 the selection of RBLs relevant to the off-site areas and consideration of the worst-case meteorological conditions and assuming these occur on a regular basis.

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

5.6 Outcomes of health impact assessment: noise

Table 5.1 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 5.1: Summary of health risks - noise

Noise emissions	
Impacts	Based on the predicted noise levels and the proposed mitigation and management measures, including additional monitoring and management as detailed in the NVIA and NVIA – Pipeline for the amended project, 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	A range of noise mitigation and management measures, including monitoring and additional management measures are outlined in the NVIA (Section 8) and the NVIA – Pipeline (Section 8), and further detailed in the Amended NVIA. These mitigation and management measures would be detailed in a site Noise Management Plan relevant to the mine development site and the pipeline (construction phase) to guide, quantify and control noise emissions from the project. These plans may include provisions to undertake noise monitoring. In addition, a complaints handling process will be implemented for the mine development site and the works associated with the pipeline construction.

Section 6. Health impact assessment: Water

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:

- EMM 2019a, McPhillamys Gold Project, Groundwater Assessment, dated August 2019.Appendix K of the EIS.
- EMM 2019b, McPhillamys Gold Project, Pipeline Development Water Assessment, dated August 2019. Appendix X of the EIS.
- HEC 2019, McPhillamys Gold Project, Mine Development, Surface Water Assessment. Prepared by Hydro Engineering & Consulting Pty Ltd, dated 27 August 2019. Appendix L of the EIS.
- HEC 2020, McPhillamys Gold Project, Mine development, Revised Surface Water Assessment.
- EMM 2020, McPhillamys Gold Project Amendment Report – Groundwater Assessment Addendum.

This assessment has not conducted a detailed review of the above reports. The assessments presented have been relied on for the purpose of assessing potential impacts on community health related to changes in surface water and groundwater.

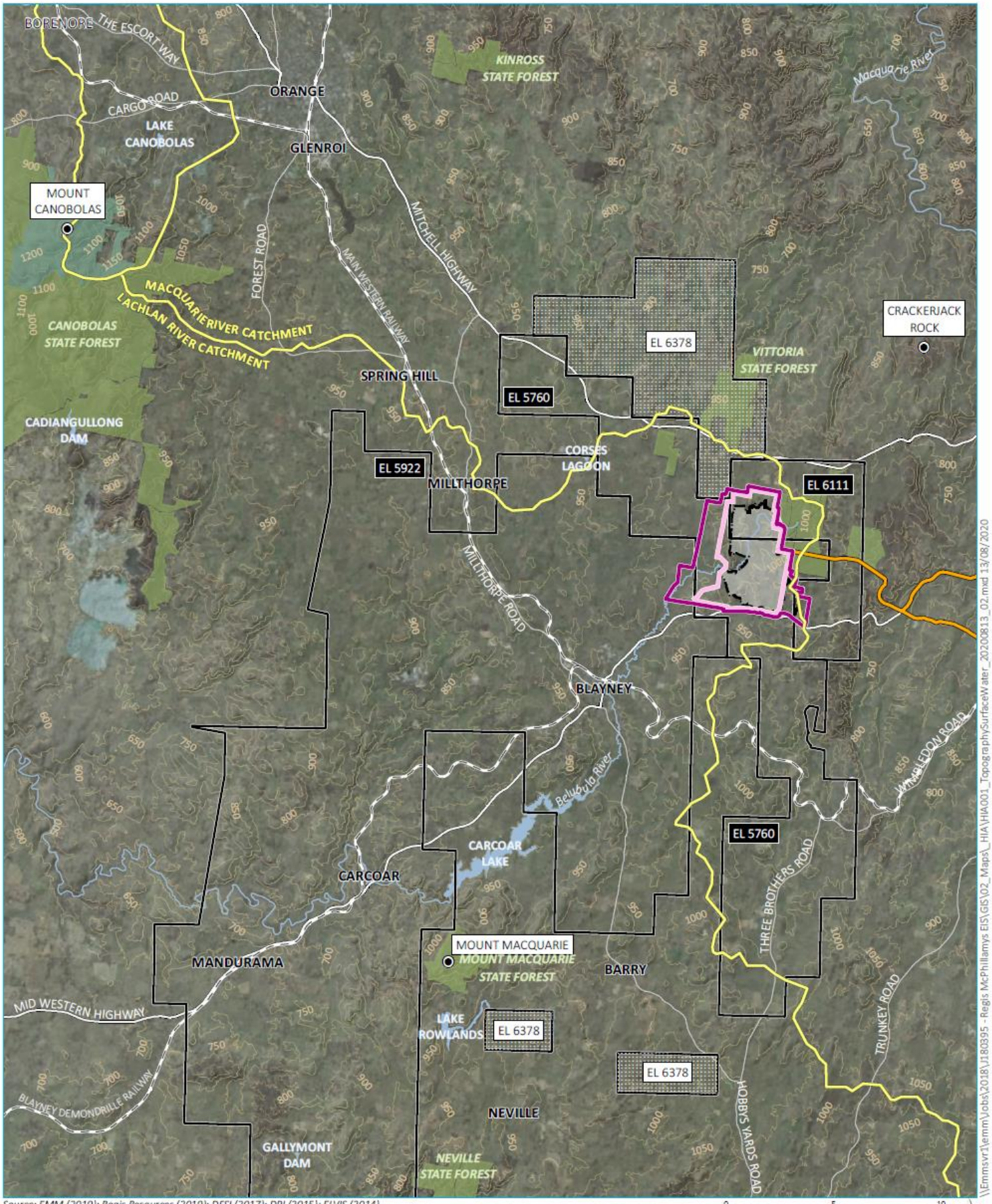
The assessment undertaken in relation to water, has involved a qualitative review of the available information to determine if there is the potential for the project to result in changes to surface water of groundwater quality or quantity, and where such changes may occur, if these may adversely affect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

6.2 Existing surface water and groundwater

The mine development is located in the upper reaches of the unregulated Belubula River which is located within the greater Lachlan River catchment. The Belubula River flows northeast to southwest through the project area into Carcoar Dam located 26 km from the project (refer to **Figure 6.1**). From the Carcoar Dam the river flows west to the Lachlan River which flows into the Great Cumbung Swamp near the banks of the Murrumbidgee River approximately 580 km to the west southwest of the project area.

A series of unnamed tributaries, referred to as Trib A to Trib K, flow into the Belubula River in the mine development site and are ephemeral with isolated, stagnant pools, generally flowing after periods of heavy rainfall. Trib A and Trib B combined are the most substantial tributaries, having a catchment area of approximately 24.4 km^2. These tributaries and the Bulubula River within the project area are shown on **Figure 6.2**.

Carcoar Dam has a catchment area of approximately 230 km2 and a storage capacity of 35.8 gigalitres (GL). The dam is managed by Water NSW and is primarily used for regulated releases for licenced extraction, environmental, stock and domestic purposes. The Belubula River contributes 10% of flows into the dam.



Source: EMM (2019); Regis Resources (2019); DFSI (2017); DPI (2015); ELVIS (2014)

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GDA 1994 MGA Zone 55

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KEY

Project application area
- Mine development project area
- Mining lease application area (Note: boundary offset for clarity)
- Disturbance footprint
- Pipeline

Exploration lease boundaries (of interest)
- Held by LFB Resources NL (Regis)
- Held by others

Existing environment
- Rail line
- Primary road
- Arterial road
- Contour (50 m)
- River
- Waterbody
- NPWS reserve
- State forest
- Catchment boundary

Topography and surface water catchments

McPhillamys Gold Project

Figure 6.1

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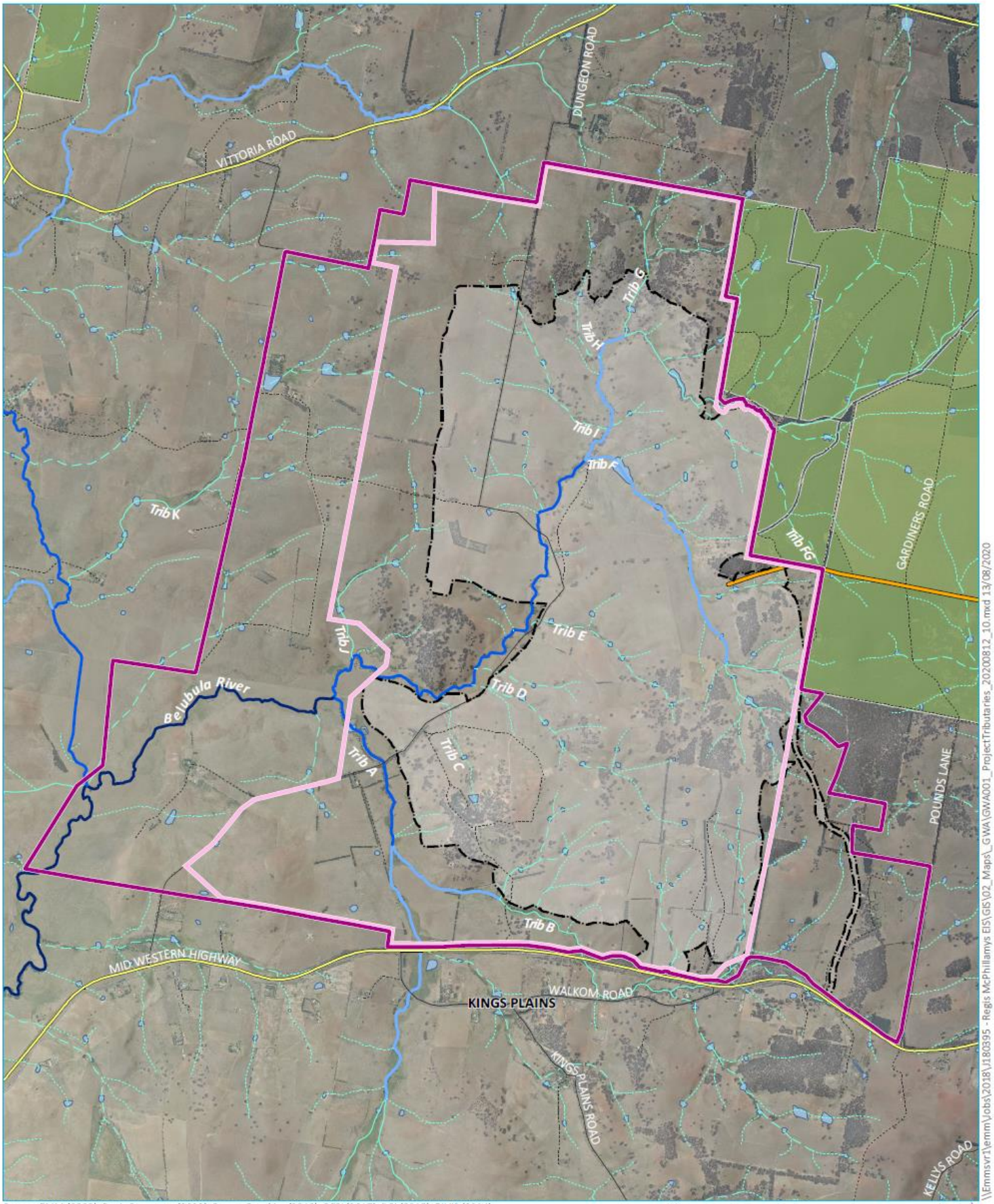
EMM creating opportunities



Source: EMM (2020); Regis Resources (2020); Survey Graphics (2019); DFSI (2017); DPI (2015); ELVIS (2014)

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KEY

Project application area
- Mine development project area
- Mining lease application area (Note: boundary offset for clarity)
- Disturbance footprint
- Pipeline

Strahler stream order
- 1st order
- 2nd order
- 3rd order
- 4th order
- 5th order
- 6th order

Existing environment
- Major road
- Minor road
- Vehicular track
- Waterbody
- Vittoria State Forest

Mine development surface drainage

McPhillamys Gold Project

Figure 6.2

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In relation to groundwater, the mine development is located within the Lachlan Fold Belt Murray Darling Basin Groundwater Source which is managed by the Water Sharing Plan for the NSW Murray-Darling Basin Fractured Rock Groundwater Sources 2011 (current to 2022).

The mine development is underlain by metasediments and volcanics, with the water table mainly located within the saprock (weathered bedrock, with water mainly located in fractures) and alluvium (where present, typically associated with watercourses and drainage lines). There is minimal fluctuation in the water table. The volcanics and metasediment materials weather to a clay-like material which, when present in fractures in the bedrock, do not act as conduits for groundwater flow. As a result, bore yields in the rock and alluvium aquifers is low and groundwater use in the area is limited to stock watering and domestic supplies.

Groundwater discharge to surface waterways occurs in isolated areas associated with the alluvium, geological structures or in the lower reaches of the Belubula River downstream of the mine development area.

In the mine development area groundwater discharges as springs and seeps are observed on the sides of hills and are typically dammed for agricultural use. The seeps are ephemeral and run dry. Groundwater is estimated to contribute approximately 5% of the overall surface flows in the Belubula River upstream of the confluence with Trib A.

There are no identified high-priority groundwater dependant ecosystems within or proximate to the mine development area.

In addition to the mine development area, an assessment of surface water impacts related to the water pipeline has also been undertaken. The pipeline extends over 90km and crosses or intersects 112 surface water drainage lines, 8 of which are associated with permanent streams. These include Coxs River, Pipers Flat Creek, Wangcol Creek, Salt Water Creek, Macquarie River, Queen Charlottes Creek, Evans Plains Creek, McLeans Creek. Dicks Creek and Spring Creek. The pipeline also traverses a multitude of geologies and number of groundwater resources including the Sydney Basin Coxs River Groundwater Source, Sydney Basin Murray Darling Basin Groundwater Source and the Lachlan Fold Belt Murry Darling Basin Groundwater Source. Water within the pipeline is expected to have an average total dissolved solids (TDS) of approximately 3,500 mg/L. This water will only be used within the project in a closed system with no discharges.

6.3 Project management and use of water

The project would include two overarching and adaptive Water Management Plans, one for construction and one for operations. The primary mitigation strategy for the project relates to the TSF and the potential for seepage from the tailings to leave the project area and affect groundwater and surface water resources.

Water will be supplied to the project via a pipeline 90 km long, transferring surplus water from coal mines and a power station near Lithgow.

The site is also located within the Water Sharing Plan for Lachlan Unregulated and Alluvial Water Sources zone.

A water management plan has been developed for the project to manage:

- Clean water (runoff from undisturbed or established rehabilitation areas);
- Development/construction water (runoff from disturbed areas and unestablished rehabilitation which may comprise sediments); and
- Operational water (runoff from mining areas such as haul roads, waste rock emplacement, hardstand areas and the open cut as well as pipeline water supply).

The operational water system will be comprised of a number of Water Management Facilities, the open cut and the TSF and a system of pumped transfers and drains. On average external water supplies comprise most (51%) of the total inflow of water to the project. The majority of water is used by the Process Plant followed by water required for haul road dust suppression.

Water imported onto the site via the pipeline will only be used for the project and this water will be contained within the proposed operational water management system, with this system designed for no discharge. The site will also be equipped with a Reverse Osmosis plant for the production of potable water.

6.4 Review of project impacts on surface water and groundwater

6.4.1 Groundwater and surface water – Mine development

Numerical modelling of potential drawdown due to the project has been undertaken on the amended project design by EMM (2020) to evaluate quantity and quality changes in groundwater resources and surface water resources.

Based on the modelling undertaken, and with consideration of the mitigation, monitoring and management measures proposed, the following impacts were identified and evaluated by EMM (2020):

- Groundwater bores:

  In relation to groundwater draw-down that may affect third-party users and groundwater supply (i.e. affecting groundwater levels and ability to extract groundwater for use), the impacts are considered to be minor, with the maximum drawdown estimated to be less than 2 m at all neighbouring bores. The NSW Aquifer Interference Policy make good provisions are not expected to be triggered.

  Impacts relating to the quality of water in third-party bores are considered to be minor as groundwater chemistry at all bores is not expected to be affected by the project.

  On this basis groundwater quality and quantity in bore on neighbouring properties is not expected to be affected by the project. Hence there would be no project related impacts that would affect community health where bore water is used.

- Belubula River:

  In relation to draw-down that may affect water flows in the Belubula River, the impacts are considered to be minor. Groundwater currently only contributes a small amount to surface water flows.

Drawdown has the potential to result in 'capture' of surface water, due to both reduced baseflow and increased leakage from surface water bodies. The Addendum Groundwater Assessment (EMM 2020) found:

- there is no predicted change to baseflow or river leakage along the Belubula River downstream of the confluence with Trib A;
- leakage from the Belubula River upstream of confluence with Trib A is predicted to increase by up to 10% between 5 and 10 years after mining (35 kL/day) when compared to the null (or no project) scenario,
- leakage from Trib A is predicted to increase by 5% (42 kL/day) when compared to the null (or no project) scenario
- baseflow reduction to the Belubula River upstream of the Trib A confluence (to the TSF embankment) is predicted to peak at 28 kL/day (15% reduction) at the end of mining.

These impacts would not be expected to affect the volume of water in the Belubula River.

Changes to the flow of water in springs are considered to be minor. Groundwater will not be removed from the flow system, however where these flows occur and where seeps/springs occur would change particularly within and on the boundary of the mine development area. The contribution of these springs to Belubula River are negligible and hence changes in these flows would not be of concern to the surface water flows.

On this basis the project would not impact on the quantity of water in the Belubula River, and therefore would not impact on community health.

- Seepage from project related activities:

Seepage from the TSF that may migrate outside of the mine development area and impact on the quality of groundwater and the Belubula River has been assessed, and impacts have been determined to be minor. With limited mitigation measures, seepage from the TSF is predicted to slowly migrate south-west and south of the TSF. Where not intercepted by the seepage management system, the seepage may migrate towards the pit, with some seepage predicted to move towards the Belubula River. TSF seepage is very slow and by the time it migrates through the ground and reaches the Belubula River, the seepage water will mix with groundwater, become diluted and will undergo other reactions with the geology and groundwater. Changes are unlikely to be measurable.

Where seepage from the TSF may migrate to the Belubula River, EMM (2020) has undertaken an assessment of potential changes to water quality. This review concluded that the seepage would not change existing water quality within groundwater or the Belubular River and its tributaries.

The seepage of leachate from the waste rock emplacement to groundwater has been assessed and impacts on groundwater quality considered minor. The geochemical characterisation confirmed that there is sufficient non-acid forming waste rock material to fully encapsulate the waste rock emplacement, minimising the potential for acid forming materials to be exposed. Leachate from the waste rock emplacement is designed to be captured and recirculated to the

operational water management system. Hence it is unlikely that leachate from the waste rock emplacement would impact on groundwater quality.

The seepage of water in water storage areas to groundwater has been assessed and impacted determined to be minor. The design of water storage areas will aim to reduce water seepage and these areas will not spill under all historical climate scenarios.

The release of chemicals from the storage of hazardous goods as runoff to surface water or seepage to groundwater has been assessed and impacts determined to be minor. The design of these areas would ensure these chemicals are in dedicated storage areas which would restrict the potential for surface water runoff (using containment).

On this basis the proposed TSF, waste rock emplacement, water storage areas and the storage of hazardous goods would not impact on the quality (or change existing water quality) of groundwater or surface water, and therefore would not impact on community health.

6.4.2 Groundwater and surface water – Pipeline

Impacts of the pipeline on groundwater and surface water resources along the length of the pipeline have been assessed by EMM (2019b). The assessment concluded that potential risks to groundwater resources from the pipeline development were negligible. During construction a number of mitigation measures would be implemented to manage potential impacts to groundwater and during operations isolation or section valves would isolate the pipeline in discrete sections such that only individual sections would be required to be dewatered for maintenance and provide security in the event of a leak. These valves would also be installed on either side of major water course crossings to minimise potential impacts to surface water.

Risks to surface water sources were evaluated as low and periodic monitoring of water quality is proposed along the pipeline route. During construction a number of mitigation measures would be implemented to manage potential impacts to surface water. In addition, the isolation valve would minimise impacts to surface water. The pipeline would be pressure tested for leaks during commissioning and leak detection would be implemented during operations.

All management measures relevant to the pipeline (including maintenance requirements) would be detailed.

Where these management measures are implemented the potential for the proposed water pipeline to adversely affect the quality of water accessed and used by the community located close to the pipeline (as groundwater or surface water) is negligible. Hence there are no health risk issues of concern in relation to the construction and operation of the pipeline.

6.5 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.

6.6 Outcomes of health impact 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 (in relation to quantity and quality of water) as a result of the project, including the pipeline, is considered to be negligible.
Mitigation	Implementation of the proposed Water Management Plans which would include a monitoring program covering both onsite and neighbouring bore/water sources plus regular public reporting.

Section 7. Health impact assessment: Other key issues

7.1 Hazardous incidents

Health impacts associated with potential impacts of the project relevant to the local community, associated with hazardous incidents, have been evaluated on the basis of information provided in the following report:

- RM 2019, McPhillamys Preliminary Hazard Analysis. Report dated June 2019. Appendix R of the EIS. Report is referred to as the PHA.

This assessment has not undertaken a detailed review of the PHA, rather the results have been relied on for the purpose of evaluating the potential for hazardous incidents to impact on the off-site community.

With the exception of explosives, cyanide and LPG, all of the dangerous goods on the site would not result in hazardous conditions that would extend beyond the immediate area of storage or use.

Further analysis of the impacts associated with explosives, cyanide and LPG determined the following:

Explosives – a worst-case evaluation (where all stored materials exploded assuming these materials were outdoors, not in the appropriate magazine stores) indicated that there would be no impacts from defragmentation or overpressure to any location off the site. It is noted that explosive management practices are intended to be adopted at the project which would prevent any unwanted explosive initiation, resulting in no risk.

Cyanide – a worst-case evaluation of the storage of sodium cyanide on the site has been undertaken. Where stored, as proposed, to meet the requirements of the International Cyanide Code, there is no plausible mechanism for this material to ignite or heat such that hydrogen cyanide gas is produced. For cyanide used in the processing circuit, this remains in an enclosed system and would not be able to generate hydrogen cyanide outside the site. In the event of inadvertent mixing of an acid (rather than caustic) occurred in the processing circuit, hydrogen cyanide gas would be released. The extent of the hydrogen cyanide plume would remain onsite and not extend to the off-site community. It is noted that cyanide (and other reagent) management practices are intended to be adopted at the project which would prevent such events occurring.

Stored flammable goods – a worst-case assessment of the storage of flammable goods, in particular LPG has been undertaken. In the event of a tank fire of LPG impacts would extend less than 200 m from the tank, which would remain within the project site. Review of a diesel fire indicated impacts lower than for the LPG fire. Hence there would be no impacts extending to the off-site community. It is noted that fuels and flammable materials would be managed on the site to prevent fire.

Off-site transport – assessment of the transport of materials relevant to the project has been undertaken. All goods would be transported in accordance with the Australian Code for the

Transport of Dangerous Goods by Road and Rail (Australian Transport Code). This would effectively manage any transport risks relevant to all dangerous goods transported to the site.

Based on the assessment presented within the PHA there are no hazardous incidents where there is the potential for impacts to occur within the off-site community. Where there are no off-site impacts there are no risks to community health.

7.2 Lighting

Lighting impacts associated with the proposed project is assessed in the following report:

- VPA 2019, Visual Impact Assessment, McPhillamys Gold Project. Report dated 19 August 2019. Appendix S of the EIS. Report is referred to as the VIA.
- VPA 2020, Visual Impact Assessment – Addendum, McPhillamys Gold Project. Report dated August 2020.

The presence of light that directly intrudes into residential homes has the potential to disrupt sleep and adversely affect health and wellbeing. The assessment undertaken has identified that lighting required for the project, will result in sky glow that would be visible from surrounding areas, particularly given the existing dark night skies. Sky glow effects also occur from lighting in local towns. A sky glow does not provide sufficient light to affect sleep. More specific lighting sources include:

- Lighting from the processing plant and mining infrastructure – these areas are separated from residential areas of Kings Plains by the prevailing topography (combined with the WRE as it progresses) hence there would be no direct lighting impacts from these sources.
- Light impacts from machinery moving on the waste emplacement, and trucks entering the pit. There will be no off-site haulage as the extracted ore would be processed on the site. The revised waste emplacement schedule provides for placement of waste rock in the southern portion of the waste rock emplacement during the day time and the northern portion of the waste rock emplacement at night, until the southern amenity bund is built to height (end of year 6). Therefore, there will be minimal direct lighting impacts associated with waste emplacement activities during the mine life.

Activities associated with the amended project would be expected to contribute to the project related sky glow, and specific mobile light sources may be visible from some locations at times, however the potential for direct intrusion of lighting into surrounding residential homes is considered low.

Mitigation measures have been identified in the VIA and VIA-Addendum to minimise impacts related to lighting. This includes restriction of night lighting to the minimum required for operations and safety, use of unidirectional lighting techniques, aiming of the lights to where needed, shielding to limit light spill, use of anti-reflective paint to limit light spill and the use of low beam on headlights.

Regis have, and continue to, plant on and off site landscape screens and are also in the process of negotiating visual mitigation agreements with key affected residences in Kings Plains.

These measured which would reduce the potential for light impacts to any off-site residential areas.

Hence, while there would be expected to be some visual impacts of lighting, direct intrusion of lighting into residential homes is not expected to occur and hence the potential for sleep disturbance, and impacts on health, is considered negligible.

7.3 Stress and anxiety

Consideration of factors that may more generally affect community wellbeing, in particular changes in levels of stress and anxiety associated with the proposed project is assessed in the following report:

- Hansen Bailey 2019, Social Impact Assessment, McPhillamys Gold Project. Report dated July 2019. Appendix T of the EIS. Report is referred to as the SIA.

Impacts of the project on stress and anxiety levels was identified as a key concern by the community. Existing data presented in the SIA indicated that hospitalisations for mental health conditions in the Blayney LGA were lower than the NSW average. Impacts of stress and anxiety associated with the proposed project was identified as a key issue particularly in relation to project uncertainty. More specifically, these uncertainties related to:

- Whether the project would proceed
- The mine life
- The potential impacts and the extent to which they may be impacts
- The ability of Regis to suitably manage or control impacts
- The opportunity for property acquisition and/or compensation
- The potential impacts on property values and saleability of property in the locality in the event that residents seek to relocate during the approvals process or following project determination.

In addition, residents expressed frustration with the amount of time the project was taking up in their daily life at the expense of more enjoyable or necessary activities.

Increased levels of stress and anxiety, both short-term acute events and chronic events/impacts are well recognised to affect health and wellbeing. Individuals experience a wide range of complex factors and issues influence health and wellbeing, specifically mental health. In addition, individuals respond to changes in stress and anxiety in different ways and hence it is not possible to quantify how the project would affect stress and anxiety levels in the community.

The potential for the project to impact on community stress and anxiety is acknowledged to be of high significance in the SIA. The primary strategy to manage stress and anxiety in relation to the project is for Regis to engage in and maintain transparent, evidence based and ongoing dialogue with concerned property owners and other community members, based on the results of the EIS. It is expected that the additional assessment presented in this report, that specifically addresses impacts on community health in relation to impacts to air quality (a key concern identified by the community) as well as other impacts such as noise and water, will further assist in addressing concerns of the community, which would assist in managing stress and anxiety.

Section 8. Conclusions

The HIA presented in this report has considered potential impacts on community health in relation to air quality, noise, water, hazardous materials, lighting and stress/anxiety.

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.

Table 8.1: Summary of health impacts

Air emissions	
Impacts	Based on the available data and information in relation to emissions to air of dust, naturally occurring asbestos (NOA), metals and metalloids that may be present on the dust, nitrogen dioxide and hydrogen cyanide from the project, potential impacts on the health of the community have been assessed. The impact assessment has concluded there are no health risk issues of concern relevant to the project.
Mitigation	A range of mitigation measures have been identified in the EIS and Amended Project report that would minimise dust emissions, diesel combustion emissions and blast fumes. In addition, air quality monitoring is proposed to be continued for the duration of the project, which includes dust-deposition monitoring and real-time particulate monitoring. An air quality management plan, which includes a monitoring plan, would be developed for the project, documenting locations, monitoring methods and reporting responsibilities.
Noise emissions	
Impacts	Based on the predicted noise levels and the proposed mitigation and management measures, including additional monitoring and management as detailed in the EIS and Amended Project report, 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	A range of noise mitigation and management measures, including monitoring and additional management measures are outlined in the EIS and Amended Project report. These mitigation and management measures would be detailed in a site Noise Management Plan relevant to the mine development site and the pipeline (construction phase) to guide, quantify and control noise emissions from the project. These plans may include provisions to undertake noise monitoring. In addition, a complaints handling process will be implemented for the mine development site and the works associated with the pipeline construction.
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 (in relation to quantity and quality of water) as a result of the project, including the pipeline, is considered to be negligible.
Mitigation	Implementation of the proposed Water Management Plans which would include a monitoring program covering both onsite and neighbouring bore/water sources plus regular public reporting.
Hazardous materials	
Impacts	Based on the assessments undertaken there are no impacts in the off-site community. This includes the transport, storage and use of a range of dangerous goods, including explosives, cyanide and LPG. Where there are no impacts, there are no risks to community health.
Mitigation	Transport in accordance with the Australian Transport Code and the storage and use of dangerous good in accordance with all relevant regulations and codes.

Lighting	
Impacts	Based on the assessments undertaken, it is expected that lighting would be visible in various areas surrounding the site, however the potential for lighting to directly intrude into residential homes and adversely affect sleep and hence health is considered negligible.
Mitigation	A range of mitigation measures related to lighting are outlined in the EIS and Amended Project report to minimise the potential for light spill that may affect the off-site areas.
Stress and anxiety	
Impacts	The potential for the project to result in increased levels of stress and anxiety in the community has been identified by the community and recognised as an area of key concern in the SIA.
Mitigation	Management measures, principally related to engaging in and maintain transparent, evidence based and ongoing dialogue with concerned property owners and other community members based on the outcomes of the EIS and Amended Project report.

Section 9. References

Project related references

EMM 2019, McPhillamys Gold Project, Environmental Impact Statement. Prepared for LFB Resources NL, dated August 2019, and the following appendices:

- HEC 2019, McPhillamys Gold Project, Mine Development, Surface Water Assessment. Prepared by Hydro Engineering & Consulting Pty Ltd (HEC) dated 27 August 2019. Appendix J of the EIS
- EMM 2019a, McPhillamys Gold Project, Groundwater Assessment. Report dated August 2019. Appendix K of the EIS
- MAC 2019a, Noise and Vibration Impact Assessment, McPhillamys Gold Project, Blayney, NSW. Report prepared by Muller Acoustic Consulting (MAC) dated August 2019. Appendix L of the EIS
- EMM 2019, McPhillamys Gold Project, Air Quality and Greenhouse Gas Assessment. Report dated August 2019, Appendix M of the EIS
- RM 2019, McPhillamys Preliminary Hazard Analysis. Report dated June 2019. Appendix R of the EIS
- EMM 2019b, McPhillamys Gold Project, Pipeline Development Water Assessment. Report dated August 2019. Appendix X of the EIS
- MAC 2019b, Noise and Vibration Impact Assessment, McPhillamys Gold Project – Pipeline Development Lithgow to Blayney, NSW. Report prepared by Muller Acoustic Consulting (MAC) dated July 2019. Appendix AA of the EIS
- VPA 2019, Visual Impact Assessment, McPhillamys Gold Project. Report dated 19 August 2019. Appendix S of the EIS
- Hansen Bailey 2019, Social Impact Assessment, McPhillamys Gold Project. Report dated July 2019. Appendix T of the EIS.

EMM 2020, McPhillamys Gold Project, Amendment report, including the following appendices:

- EMM 2020, McPhillamys Gold Project, Air Quality and Greenhouse Gas Assessment. Report dated July 2020, which is a supplementary report to the Air Quality and Greenhous Gas Assessment included in Appendix M of the EIS
- VPA 2020, McPhillamys Gold Project, Visual Impact Assessment – Addendum. Report dated August 2020.
- MAC 2020, McPhillamys Gold Project, Amended Noise and Vibration Impact Assessment. Report dated July 2020.
- HEC 2020, McPhillamys Gold Project, Mine Development, Revised Surface Water Assessment. Report dated July 2020.
- EMM 2020, McPhillamys Gold Project Amendment Report – Groundwater Assessment Addendum.

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Appendix A Assessment of health impacts of fine particulates and nitrogen dioxide

A1 Fine particles

A1.1 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 2009b, 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 2009b).

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 string 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 2006).

A1.2 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.

A1.3 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 1998a, 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/m^3	50 µg/m^3	50 µg/m^3 as limit value with 35 exceedances permitted each year	150 µg/m^3 (not to be exceeded more than once per year on average over 3 years)
	Annual	25 µg/m^3	20* µg/m^3	40 µg/m^3 as limit value	NA
$PM_{2.5}$	24-hour	25 µg/m^3 20 µg/m^3 (goal for 2025)	25 µg/m^3	NA	35 µg/m^3 (98th percentile, averaged over 3 years)
	Annual	8 µg/m^3 7 µg/m^3 (goal for 2025)	10* µg/m^3	25 µg/m^3 as target value from 2010 and limit value from 2015. 20 µg/m^3 as a 3 year average (average exposure indicator) from 2015 with requirements for ongoing percentage reduction and target of 18 µg/m^3 as 3 year average by 2020	12 µg/m^3 (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).

A1.4 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[11]. 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 $RR = \exp[\beta(X-X0)]$

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)[12] where

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

12 For regional guidance, such as that provided for Europe by the WHO WHO 2006, 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

the exposure-response relationships identified have been directly considered on the basis of the approach outlined below.

An additional risk can be calculated as:

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

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 receptors. For this assessment the change in $PM_{2.5}$ relates to the maximum change in annual average air concentrations and the value considered in this assessment is 0.6 µg/m^3
- Baseline incidence of the key health endpoints that are relevant to the population exposed (namely mortality all causes – all ages) – the assessment undertaken has considered the baseline mortality data relevant to the Western NSW LHD (which has a similar value as the LGAs, but is the highest rate, so most conservative value, refer to **Table 3.3**) of 625.1 as the rate per 100,000 people
- 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

exposure. These impact functions are simpler to use than the approach adopted in this assessment, however in utilising this approach it is assumed that the baseline incidence of the health effects is consistent throughout the whole population (as used in the studies) and is specifically applicable to the sub-population group being evaluated. For the assessment of exposures in the areas evaluated surrounding the project it is more relevant to utilise local data in relation to baseline incidence rather than assume that the population is similar to that in Europe (where these relationships are derived).

undertaken in Australia and New Zealand (EPHC 2010). Using a RR of 1.06, results in a β = 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).

Based on the above:

Risk = 0.006 x 0.6 x 625.1/100000 = 2 x 10^{-5}

This incremental risk is below the unacceptable risk level of 10^{-4} outlined in the NSW EPA Approved Methods (NSW EPA 2016). Population risks for the wider community will be lower than this maximum risk. 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.

A2 Nitrogen dioxide

A2.1 Cumulative exposures

The NEPC ambient air quality guideline for the assessment of acute (short-term) exposures to NO_2 relates to the maximum predicted total (cumulative) 1-hour average concentration in air. The guideline of 246 µg/m^3 (or 0.12 parts per million [ppm]) is based on a lowest-observed-adverse-effect level (LOAEL) of 409–613 µg/m^3derived from statistical reviews of epidemiological data suggesting an increased incidence of lower respiratory tract symptoms in children and aggravation of asthma. An uncertainty factor of two to protect susceptible people (i.e. asthmatic children) was applied to the LOAEL (NEPC 1998a). On this basis, the NEPC acute guideline is protective of adverse health effects in all individuals, including sensitive individuals.

The NEPC ambient air quality standard for the assessment of chronic (long-term) exposures to NO_2 relates to the maximum predicted total (cumulative) annual average concentration in air. The standard of 62 µg/m^3 (or 0.03 ppm) is based on a LOAEL of the order of 40–80 parts per billion by volume (around 75–150 µg/m^3). This relates to the early and middle childhood years when exposure can lead to the development of recurrent upper and lower respiratory tract symptoms, such as recurrent ‘colds’, a productive cough and an increased incidence of respiratory infection with resultant absenteeism from school.

An uncertainty factor of two was applied to the LOAEL to account for susceptible people within the population resulting in a guideline of 20-40 parts per billion by volume (38–75 µg/m^3) (NEPC 1998a). On this basis, the NEPC standard is protective of adverse health effects in all individuals, including sensitive individuals.

A2.2 Incremental exposures

The approach adopted for the assessment of exposures and impacts is consistent with that adopted for particulates as outlined above. This involves the calculation of a change in relative risk relevant to the most impacted receptor in the off-site community.

The assessment has focused on the most significant health end point, all-cause mortality for all ages. This has utilised the exposure-response relationship derived for from modelling undertaken for 5 cities in Australia and 1 day lag (EPHC 2010; Golder 2013), which has a β coefficient of 0.00188 for a 1 µg/m^3 increase in NO_2 exposure.

This relationship has been used in conjunction with the baseline mortality data adopted for particulates (as detailed above) and the modelled maximum increase in annual average NO_2 (as presented in the AQGGA) of 2.0 µg/m^3.

Based on the above:

$$\text{Risk} = 0.00188 \times 2.0 \times 625.1/100000 = 2 \times 10^{-5}$$

This incremental risk is below the unacceptable risk level of 10^{-4} outlined in the NSW EPA Approved Methods (NSW EPA 2016). Population risks for the wider community will be lower than this maximum risk.

Appendix B Toxicity summaries

B1 Antimony

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

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

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

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

Antimony enters the environment during the mining and processing of its ores and in the production of antimony metal, alloys, antimony oxide, and combinations of antimony with other substances. Little or no antimony is mined in the United States, Antimony ore and impure metals are brought into this country from other countries for processing. Small amounts of antimony are also released into the environment by incinerators and coal-burning power plants. The antimony that comes out of the smoke stacks of these plants is attached to very small particles that settle to the ground or are washed out of the air by rain. It usually takes many days for antimony to be removed from the air. Antimony attached to very small particles may stay in the air for more than a month. Antimony cannot be destroyed in the environment. It can only change its form or become attached to or separated from particles. Most antimony will end up in the soil or sediment, where it attaches strongly to particles that contain iron, manganese, or aluminium (ATSDR 1992).

Background

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

- Intakes of antimony were addressed by FSANZ (FSANZ 2003). Estimated dietary intakes for infants and 2-3 year olds ranged from 0.01 to 0.25 µg/kg bw/day which ranges from 3 to 61% of the adopted tolerable intake – 0.4 µg/kg bw/day – taken from USEPA IRIS summary for antimony (USEPA IRIS). The average intake of antimony is estimated to be 0.13 µg/kg/day for 2-3 year olds, approximately 20% of the TDI from the ADWG ((NHMRC 2011 Updated 2016)) – the recommended oral TRV.
- Antimony was reported in ambient air data collected in (NSW DEC 2003) where concentrations in urban, regional and industrial areas assessed ranged from 0.04 to 4.6 ng/m^3. Intakes associated with these are concentrations are negligible compared with intakes from food.

Classification

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

Review of Available Values/Information

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

Toxicity reference values for antimony

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 updated 2018)	TDI = 0.00086 mg/kg/day	The ADWG derived a guideline of 0.003 mg/L based on a lowest effect level of 0.43 mg/kg/d from a lifetime study in rats showing decreased lifespan and altered blood levels of glucose and cholesterol and a safety factor of 500 (10 for interspecies, 10 for intraspecies and 5 as result was a lowest observed effect level rather than a no effect level.
International		
WHO DWG (WHO 2011c)	TDI = 0.00086 mg/kg/day	The WHO DWG derived a guideline of 0.005 mg/L using the same study as the ADWG but including rounding.
ATSDR (ATSDR 1992)	No MRL derived	
USEPA IRIS (USEPA IRIS)	RfD = 0.0004 mg/kg/d	The USEPA IRIS entry (last reviewed in 1991) derived an oral RfD of 0.0004 mg/kg/day based on a LOAEL of 0.35 mg/kg/day from the same study in rats used in the ADWG with an uncertainty factor of 1000. The confidence level in the study, database and RfD is noted to be low.

There are no inhalation guidelines available for antimony.

It is recommended that the oral TDI from the Australian Drinking Water Guidelines be adopted for use in this assessment for all pathways of exposure

Recommendation

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

- Oral TRV (TRV_O) = 0.0009 mg/kg/day (NHMRC 2011 updated 2018)
- Background intakes from other sources (as % of TRV) = 20% for all intakes

B2 Arsenic

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

Arsenic is a metalloid which can exist in four valence states (-3, 0, +3 and +5) and forms a steel gray, brittle solid in elemental form (ATSDR 2007a). Under reducing conditions arsenite (AsIII) is the dominant form and in well oxygenated environments, arsenate (AsV) predominates (WHO 2001d). 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 2011a). On the basis of the range of intake estimations available, a reasonable estimation of 50% of the oral toxicity reference value (TRV) from sources other than soil has been assumed.
- Intakes from inhalation exposures are low (around 0.0017 µg/kg/day (APVMA 2005)), comprising <1% of the inhalation TRV adopted.

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 2012a).

Identification of Toxicity Reference Values

Oral

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

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

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

Revision to the WHO guidelines on drinking water (WHO 2011c) 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 2005a) as there remains uncertainty on the carcinogenic MOA for arsenic (Sams et al. 2007). Further research is required to define and review the MOA prior to the USA revising the dose-response approach currently adopted. Inherent in the current US approach (where a non-threshold slope factor is derived) are some key uncertainties that likely result in an overestimate of risk, which include:

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

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

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

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

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

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

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

Assessment of End-Points – Oral Exposures

Existing Oral Dose-Response Approaches - Australia

Oral intakes of arsenic were considered in Australia in (Langley 1991) and the Australian Drinking Water Guidelines (ADWG) (NHMRC 2011 Updated 2016). The following can be noted from these guidelines:

- The derivation of the previous HIL for arsenic was dated and considers all intakes of arsenic on the basis of a threshold PTWI established by the WHO in 1983, and reconfirmed in 1988 (Langley 1991; WHO 1989). The PTWI adopted was 15 µg/kg/week. In setting the PTWI it was noted that there is "a narrow margin between the PTWI and intakes reported to have toxic effects in epidemiological studies" (WHO 1989). The PTWI was withdrawn by JECFA (WHO 2011a) following further review (refer to discussion below).
- The previous ADWG (NHMRC 2004) derived a guideline of 7 µg/L for inorganic arsenic in drinking water based on the former WHO PTWI (noted above) converted to a daily intake (provisional maximum tolerable daily intake) of 2 µg/kg/day. The current ADWG (NHMRC 2011 Updated 2016) has adopted a guideline of 10 µg/L based on a "practicable achievable" approach supported by contemporary epidemiological studies in which elevated cancer risks and other adverse effects are not demonstrable at arsenic concentrations around 10 µg/L. It is noted that this level is equivalent to an adult (70 kg) intake of 0.28 µg/kg/day.

A review of arsenic toxicity was conducted by the APVMA (APVMA 2005) where a threshold approach was considered appropriate (noted above). A threshold value of 3 µg/kg/day was derived by the Australian and New Zealand Food Authority (ANZFA now Food Standards Australia New Zealand (FSANZ)) in 1999, and considered in the APVMA (APVMA 2005) review. The review considered that skin cancers appear to be the most sensitive indicator of carcinogenicity of inorganic arsenic in humans and based on epidemiological studies a threshold of 2.9 µg/kg/day (rounded to 3 µg/kg/day) can be obtained. This threshold is the value adopted as a provisional tolerable daily intake (PTDI) by FSANZ (FSANZ 2003), similar to the former PTWI available from the WHO (noted above). This approach has been considered by APVMA for all intakes of arsenic (oral, dermal and inhalation). The evaluation has not been further updated.

Oral Dose-Response Approaches - International

Evaluation of arsenic by JECFA (WHO 2011a) considered the available epidemiological data in relation to the increased incidence of lung cancer and urinary tract cancer associated with exposure to arsenic in water and food. Using the data associated with these endpoints, JECFA derived a benchmark dose lower confidence limit for a 0.5% increased incidence ($BMDL_{0.5}$) of lung cancer (most sensitive endpoint) of 3 µg/kg/day (ranging from 2-7 µg/kg/day). Uncertainties associated with the assumptions related to total exposure, extrapolation of the $BMDL_{0.5}$ and influences of the existing health status of the population were identified. Given the uncertainties and that the $BMDL_{0.5}$ was the essentially equal to the PTWI (WHO 1989), the PTWI was withdrawn. No alternative threshold values were suggested by JECFA as the application of the BMDL needs to be addressed on a regulatory level, including when establishing guideline levels.

The review conducted by JECFA is generally consistent with that conducted by the European Food Safety Authority (EFSA) Panel on Contaminants in the Food Chain (CONTAM) (EFSA 2010b). The

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

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

- The studies considered in the derivation of the different ranges of BMDL values (EFSA 2010b; WHO 2011a) are based on drinking water studies. No studies considered are derived from other sources including soil. There are uncertainties inherent in the epidemiological studies considered by the WHO and EFSA (EFSA 2010b; WHO 2011a). These uncertainties include limitations or absence of information on levels of individual exposure or arsenic intake (from drinking water), limited quantification of arsenic intakes from other sources including food, size or the studies (variable) and the assumption that arsenic intake is the single cause of all endpoints identified.
- The drinking water studies are primarily associated with populations that have poorer nutritional status (i.e. Taiwan and Bangladesh). Studies (as summarised by EFSA (EFSA 2010b)) have shown that populations with poor nutrition (and health status) are more susceptible to the prevalence and severity of arsenic-related health effects.
- The largest of the studies conducted was within rural Asian populations which differ from Australian populations with respect to generic lifestyle factors.

In view of the above, consideration of the lower end of the range of BMDL values available from WHO and EFSA (EFSA 2010b; WHO 2011a) is not considered appropriate for the Australian population.

Based on the above considerations a TRV of 2 µg/kg/day has been adopted. The TRV has been selected on the basis of the following:

- The TRV is at the lower end of the range derived from JECFA, and also lies within, but is not at the lower end of the range presented by EFSA (EFSA 2010b; WHO 2011a);
- The value is within the range of no observable adverse effect levels (NOAELs) identified by RIVM (Baars et al. 2001), US EPA (USEPA IRIS) and ATSDR (ATSDR 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 2013b).

Inhalation

Less data is available with respect to inhalation exposures to arsenic, though trivalent arsenic has been shown to be carcinogenic via inhalation exposures (with lung cancer as the end point). Review of the relevant mechanisms for carcinogenicity by RIVM (Baars et al. 2001) suggests that the mechanism for arsenic carcinogenicity is the same regardless of the route of exposure. Hence a threshold is also considered relevant for the assessment of inhalation exposures. This is consistent with the approach adopted in the derivation of the previous arsenic HIL (Langley 1991) and in the review undertaken by APVMA (APVMA 2005). While NEPC (previous HIL) and APVMA adopted the oral PTWI as relevant for all routes of exposure, RIVM has derived an inhalation-specific threshold value. (Baars et al. 2001) identified that the critical effect associated with chronic inhalation exposures in humans was lung cancer. The lowest observable adverse effect concentration (LOAEC) for trivalent arsenic associated with these effects is 10 µg/m^3 (based on the review (ATSDR 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 2000c) 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 2001d) 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 2001d)) 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 2001d) 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
- Background Intakes from other sources (as % of TRV) = 50% for oral and dermal

B3 Barium

General

Potential exposures to barium and the toxicity of barium have been evaluated and summarised in a number of reviews available from the WHO (WHO 1990, 2001c, 2017), Unites States (ATSDR 2007b; USEPA 2005c) and Canada (Health Canada 2018). The following provides a summary of information available from these reviews.

Barium occurs in various compounds in the environment either naturally or from human activities. While the main use of barium is as a drilling fluid additive in oil and gas exploration, it is also used as a contrast agent in X-ray diagnostic tests and in a wide array of products, including plastics, rubbers, paint, glass, carpets, ceramics, sealants, furniture, fertilizers and pesticides. Naturally occurring barium can be found in most types of rocks and can enter surface and groundwater by leaching and eroding from sedimentary rocks (Health Canada 2018). A total of over 20 radioactive barium isotopes, with various degrees of stability and radioactivity, have been identified in the environment. However, the focus of this review is limited to barium's chemical properties. No assessment of radioactive barium compounds is included.

Barium is not considered to be an essential element. Different barium compounds have differing solubilities in water and body fluids, which influences their toxicity. The Ba^{2+} ion and the soluble compounds of barium (mainly chloride, nitrate, and hydroxide) are toxic to humans and animals. Barium carbonate, although relatively insoluble in water, is soluble in the gastrointestinal tract, allowing uptake into serum and tissues thereby capable of causing effects. Insoluble barium compounds, such as barium sulphate, serve as inefficient sources of Ba^{2+} ion and are therefore generally non-toxic to humans. The non-toxic nature of barium sulphate has made it useful in medical applications. However, barium sulphate or other insoluble barium compounds can become toxic if the gastrointestinal tract is compromised (e.g., in the case of colon cancer), thereby allowing barium to enter the bloodstream.

Exposure

Food and drinking water represent the main sources of exposure to barium for the general population; however, the available data indicate that contributions from these sources can be highly variable. Other sources include soil and air (where there is a specific source). Soluble barium compounds are rapidly absorbed by the body and is distributed to the muscles, lungs and bone. The primary route to excretion is via faeces.

In Australia, concentrations of barium in drinking water is reported to be between <0.002 and 1.1 mg/L (NHMRC 2011 updated 2018). Data from the US indicates a median concentration of 0.43 mg/L (WHO 2001c). Assuming the median is more representative of typical upper intakes from drinking water, this would represent an intake of approximately 0.012 mg/kg/day. There is no data available in relation to levels of barium in food products in Australia. Dietary intakes from Canada (Health Canada 2018) were in the range of 0.0166 to 0.0266 for children aged 7 months to 4 years and 0.0051 to 0.01 mg/kg/day for persons aged 20 and older. Data on levels of barium in air suggests these intakes are very low, <1 ng/m^3 (where not associated with a specific source) (Health

Canada 2018). Intakes from drinking water and dietary sources are low but may comprise around 10% of the adopted oral TRV. Inhalation intakes are negligible.

Health effects

At low doses, barium acts as a muscle stimulant and at higher doses affects the nervous system eventually leading to paralysis. Acute and subchronic oral doses of barium cause vomiting and diarrhea, followed by decreased heart rate and elevated blood pressure. Higher doses result in cardiac irregularities, weakness, tremors, anxiety, and dyspnea. A drop in serum potassium may account for some of the symptoms. Death can occur from cardiac and respiratory failure. Acute doses around 0.8 grams can be fatal to humans (RAIS).

Subchronic and chronic oral or inhalation exposure primarily affects the cardiovascular system resulting in elevated blood pressure. Subchronic and chronic inhalation exposure of human populations to barium-containing dust can result in a benign pneumoconiosis called "baritosis." This condition is often accompanied by an elevated blood pressure but does not result in a change in pulmonary function.

Barium toxicity is caused by the free cation, and highly soluble barium compounds are more toxic than insoluble compounds. In rodents, kidney toxicity appears to be the most sensitive effect, whereas in humans, cardiovascular (hypertension) effects have been of prime concern.

The International Agency for Research on Cancer (IARC) has not classified barium as to its carcinogenicity. The USEPA (USEPA 2005c) concluded that barium is considered not likely to be carcinogenic to humans via oral intake. Other agencies have concluded that there is no evidence that barium is carcinogenic (WHO 2001c). In addition, the weight of evidence supports that barium is not genotoxic.

Toxicity reference values

The following are available from relevant1 Australian and International sources:

Toxicity reference values for barium

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 updated 2018)	TDI = 0.2 mg/kg/day	The ADWG derived a guideline of 5.6 mg/L for barium in drinking water based on a BMDL(05) of 60 mg/kg/day for kidney effects in a chronic mice study, and application of a 300 fold uncertainty factor (10 for interspecies variation, 10 for intraspecies variation and 3 for database deficiencies).
International		
WHO DWG (WHO 2017)	TDI = 0.21 mg/kg/day	The WHO DWG derived a guideline of 1.3 mg/L using the same study as the ADWG but allowing for a different proportion of intakes from drinking water.
ATSDR (ATSDR 2007b)	MRL = 0.02 mg/kg/day	The MRL is based on the same study and approach adopted in the ADWG.
RIVM (Baars et al. 2001)	TDI = 0.2 mg/kg/day TC = 0.001 mg/m^3	TDI based on a NOEL of 0.2 mg/kg/day associated with cardiovascular effects in a human study, and application of a 10 fold uncertainty factor. TC is based on an adjusted NOAEC of 0.11 mg/m^3 for cardiovascular effects in rates from exposure to an insoluble barium carbonate dust and application of a 100 fold uncertainty factor.

Source	Value	Basis/Comments
USEPA IRIS (USEPA 2005c; USEPA IRIS)	RfD = 0.2 mg/kg/d	The USEPA IRIS entry (last reviewed in 1991) derived an oral RfD of 0.0004 mg/kg/day based on a LOAEL of 0.35 mg/kg/day from the same study in rats used in the ADWG with an uncertainty factor of 1000. The confidence level in the study, database and RfD is noted to be low.

Based on the above table there is consensus across a wide number of evaluations that an oral TDI of 0.2 mg/kg/day as adopted in the ADWG (NHMRC 2011 updated 2018). There is only one inhalation TRV available which may or may not be relevant to soluble barium compounds. The inhalation TRV is more conservative than would be adopted from route extrapolation of the oral TRV. Hence the RIVM inhalation TRV has been adopted.

Recommendation

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

- Oral TRV = 0.2 mg/kg/day for oral and dermal intakes, where 10% of intakes is derived from background/other sources
- Inhalation TRV = 0.001 mg/m^3, where background intakes are negligible
- Oral Bioavailability of 100% assumed

B4 Beryllium

General

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

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

Occupational exposure to beryllium has been associated with acute and chronic lung diseases. The acute disease is normally associated with inhalation exposures to high levels of soluble beryllium salts (e.g. Sulphate, chloride) and beryllium oxide (BeO) and may lead to chronic disease. The chronic disease is associated with long-term inhalation exposures to dust particles containing beryllium, has an immunological component and a latent period which varies depending on the beryllium species. Dermatological effects may also occur on skin contact (Di Marco & Buckett 1996).

Exposure

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

Oral Bioavailability

While oral bioavailability has been considered in the previous HIL, insufficient data are available to adequately define the bioavailability of beryllium in the range of contaminated sites that may need to be considered in Australia. On this basis a default approach of assuming 100% oral bioavailability has been adopted. It is noted that a site-specific assessment of bioavailability can be undertaken where required. It is noted that the assessment provided by Di Marco and Buckett (1996) utilised 1% bioavailability for beryllium.

Dermal absorption:

In humans and animals sensitised to beryllium, contact with beryllium and its soluble and insoluble compounds can cause dermatitis and skin granulomas. In general, the more soluble the compound the greater the sensitising potential. Dermal effects usually occur on abraded skin. Dermal absorption of beryllium is assumed to be poor and would not likely cause further systemic effects. While it is noted that absorption through damaged/injured skin is expected to be higher, review of dermal absorption of beryllium (Deubner et al. 2001) noted that absorption through intact skin is

considered negligible (<< 1%). Hence the assumption of 0.1% dermal absorption considered in the previous HIL is considered appropriate. The value is consistent with the default presented by RAIS (RAIS).

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

Inhalation:

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

Plant Uptake:

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

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

Intakes from Other Sources – Background:

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

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

Health effects

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

few days/weeks and a slower second phase, which may vary from a few weeks/months for the soluble compounds to months/years for the sparingly soluble compounds (WHO 2001a).

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

There are no reliable data on the oral toxicity of beryllium in humans. Acute oral exposures to single doses of soluble beryllium compounds are moderately toxic; however, in the case of sparingly soluble beryllium compounds, no oral single-dose studies are available. Short-, medium-, and long-term studies in animals showed that the gastrointestinal and skeletal systems are target organs for beryllium following oral exposure (WHO 2001a).

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

The inhalation data led the International Agency for Research on Cancer to conclude that beryllium and beryllium compounds are carcinogenic to humans (Group 1, sufficient evidence of carcinogenicity in humans and sufficient evidence in animals) (IARC 1993). The USEPA has classified beryllium as B1 – probable human carcinogen. The WHO (WHO 2001a) also classified beryllium as carcinogenic based on occupational inhalation studies. It is noted that the evidence is limited because of relatively small increases in lung cancer risks, poorly defined estimates of beryllium exposure, incomplete smoking data, and lack of control for potential exposure to other carcinogens, including co-exposure to sulfuric or hydrofluoric acid mists during employment in the beryllium industry (WHO 2001a).

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

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

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

This is supported by a more recent review by Hollins et al. (Hollins et al. 2009)(2009) where it was concluded that "*the increase in potential risk of lung cancer was observed among those exposed to very high levels of beryllium and that beryllium's carcinogenic potential in humans at exposure levels that exist in modern industrial settings should be considered either inadequate or marginally suggestive*".

Further review of genotoxicity by IARC (IARC 2012b) indicates that the evidence for mutagenic activity was weak or negative, however review of the available studies indicates that the underlying mechanism for carcinogenesis is complex and likely to involve several possible interactive mechanisms. Hence the evidence for a genotoxic mode of action is not clear, however there may be some mechanisms that relate to genotoxicity that affect carcinogenicity.

Based on the available data carcinogenic effects of inhaled beryllium in non-occupational environments are not genotoxic and a threshold can be adopted.

There is, however, no clear evidence that the compounds are carcinogenic when administered orally. Beryllium was not mutagenic in tests with different strains of bacteria, but caused chromosomal aberrations and gene mutations in cultured mammalian cells. Hence a threshold is adopted for the assessment of oral exposures (NHMRC 2011 updated 2018).

Toxicity reference values

The following are available from relevant Australian and International sources:

Toxicity reference values for beryllium

Source	Value	Basis/Comments
Australian		
ADWG (NHMRC 2011 updated 2018)	TDI = 0.002 mg/kg/day	The ADWG derived a guideline of 0.06 mg/L for beryllium in drinking water based on a BMD of 0.46 mg/kg/day for gastrointestinal effects in a chronic dog study and application of a 300 fold uncertainty factor (10 for interspecies variation, 10 for intraspecies variation and 3 for database deficiencies).
International		
WHO DWG (WHO 2017)	TDI = 0.002 mg/kg/day	The WHO DWG did not present a drinking water guideline, however they note a health based value of 12 mg/L may be derived using the same study as the ADWG but allowing for a different proportion of intakes from drinking water.
WHO (WHO 2001a)	TC = 0.00002 mg/m^3	TC based on CDB, characterised by the formation of granulomas. The TC is derived from a duration adjusted LOAEL or occupationally exposed workers and application of a 10 fold uncertainty factor. The WHO has also derived a non-threshold value for inhalation exposures, unit risk = 0.0024 (mg/m^3)$^{-1}$. This value has not been utilised in this assessment as it was derived on the basis of data relevant to a specific occupational exposure and there is insufficient evidence to indicate that beryllium in non-occupational environments is genotoxic and

Source	Value	Basis/Comments
		a non-threshold approach is applicable. The value also includes a significant level of uncertainty, particularly in relation to the estimation of beryllium exposures in the workplace.
ATSDR (ATSDR 2002)	MRL = 0.002 mg/kg/day	The MRL is based on the same study and approach adopted in the ADWG.
USEPA IRIS (USEPA 1998a)	RfD = 0.002 mg/kg/d RfC = 0.00002 mg/m^3	The oral RfD based on the same study and approach as outlined in the ADWG. RfC is based on CBD effects in humans and application of an uncertainty factor of 10. This is the same study and approach adopted by WHO

Based on the above table there is consensus across a wide number of evaluations that an oral TDI of 0.002 mg/kg/day as adopted in the ADWG (NHMRC 2011 updated 2018). In addition, there is consensus that the appropriate threshold inhalation TRV is 0.00002 mg/m^3.

Recommendation

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

- Oral TRV = 0.002 mg/kg/day for oral and dermal intakes, where derived from background/other sources are negligible
- Gastrointestinal absorption factor = 0.7%
- Inhalation TRV = 0.00002 mg/m^3, where background intakes are negligible
- Oral Bioavailability of 100% assumed

B5 Cadmium

General

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

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

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 2012b).

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

Background

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

Cadmium was detected in air samples collected from urban and rural areas in NSW (NSW DEC 2003). The average concentration reported was 0.17 ng/m^3, ranging from 0.3 to 1 ng/m^3. These concentrations constitute <5% to 20% of the recommended inhalation TRV in air (also considered as an international target in the DEC document). Background levels for cadmium in air can be conservatively assumed to comprise 20% of the recommended inhalation TRV.

Classification

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

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 2012b). IARC (2012) concluded that ionic cadmium causes genotoxic effects in a variety of eukaryotic cells, including human cells, although positive results were often weak and/or seen at high concentrations that also caused cytotoxicity. Based on the weight of evidence, MfE considered there to be weak evidence for the genotoxicity of cadmium.

On the basis of the available information, TRVs relevant for oral (and dermal) intakes and inhalation intakes have been considered separately.

Oral (and Dermal) Intakes

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

Source	Value	Basis/Comments
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.
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 2012b)	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 2012a). While cadmium is thought to be potentially genotoxic, the weight of evidence is not clear. In addition, epidemiology studies associated with lung cancer have confounding issues that limit useful interpretation (WHO 2000b). 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 and dermal TRV (TRV_O) = 0.0008 mg/kg/day (WHO 2010), with 60% intake from background sources
- Dermal absorption (DAF) = negligible (0%)
- Inhalation TRV (TRV_I) = 0.000005 mg/m^3 (WHO 2000b), with 20% intake from background sources

B6 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 2012c; UK DEFRA & EA 2002b).

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

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 2012c).

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

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

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 (Baars et al. 2001) to 3 ng/m^3 (UK DEFRA & EA 2002b). It is noted that concentrations of Cr VI in Europe and the UK are expected to be higher than in Australia due to the potential for long-range atmospheric transport from a greater proportion of industry in these general regions. Based on the recommended TRV for particulate phase Cr VI, these conservative air concentrations comprise less than 1% of the TC and are assumed negligible.

Classification

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

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

Review of Available Values/Information

Oral

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

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

Toxicity reference values – 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.
International		
WHO DWG (WHO 2011c)	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 2002b)	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 2012c)	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 1998b)	RfD = 0.003 mg/kg/day	The USEPA IRIS entry (last reviewed in 1998) derived an oral RfD of 0.003 mg/kg/day based on a NOAEL of 2.5 mg/kg/day from a 1-year drinking water study in rats and an uncertainty factor of 300 and modifying factor of 3 to address uncertainties in the study. The confidence level in the study, database and RfD is noted to be low.

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

Inhalation

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

Chromium is not volatile and hence inhalation exposures are only relevant to dust intakes. These are not likely to be significant for soil contamination and hence the consideration of carcinogenic effects using a non-threshold approach may not be appropriate. It is appropriate to consider intakes on the basis of a threshold approach associated with the most significant end-point. In addition inhalation exposures relating to soil contamination (dust) are expected to differ from the occupation

studies from which the non-threshold criteria are derived (where inhalation of fine dust and chromic acid mists occurs). These issues were considered by ITER (ITER 1998) in the derivation of an RfC that is relevant for environmental exposures only, not to occupational exposures associated with mists and aerosols, and USEPA (USEPA 1998b) in the derivation of an RfC.

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

- No Australian guideline values are available for Cr VI.
- The USEPA (USEPA 1998b) 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 2000c) 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 1998b) 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 2002b) 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 2002b) 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 2012c) 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 2012c)
- Inhalation TRV (TRV_i) = 0.0001 mg/m^3 (USEPA 1998b)
- Background intakes from other sources (as % of TRV) = 10% for oral/dermal intakes and 0% for inhalation.

B7 Copper

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

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 2016) are 0.05 mg/L, resulting in an intake (1 L/day and body weight of 15.5 kg) by toddlers of 0.004 mg/kg/day. It is noted that intakes of copper in drinking water supplies in New Zealand (MfE 2011b) were higher, with intakes by a young child estimated to be 0.013 mg/kg/day.
- Copper was reported in ambient air data collected in (NSW DEC 2003) where concentrations in urban, regional and industrial areas assessed ranged from 2.4 to 28 ng/m^3. Intakes associated with these are concentrations are negligible compared with intakes from food.

(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.

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 the consideration of 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 2011c)	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 2004)	No chronic MRLs available	
US EPA 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 2011c) for all routes of exposure
- Background intakes for the general population = 0.08 mg/kg/day = 60% of the oral TRV and is negligible for inhalation intakes

B8 Lead

General

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

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 2007c).

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 2007c).

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). Ingestion of soil and dust is considered a significant pathway of exposure where soil has raised lead concentrations.

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

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

Background Intake (Exposure)

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

- Dietary intakes of lead have been reported from (FSANZ 2003, 2011). Intakes reported in this study range from 0.02-0.4 µg/kg/day for adults to 0.01-1.2 µg/kg/day for infants. These data are the most current from FSANZ;
- The ADWG (NHMRC 2011 updated 2017) notes that lead concentrations in drinking water range up to 0.01 mg/L with typical concentrations less than 0.005 mg/L. Data available from South Australia (based on 5 years of data) suggest concentrations of lead in drinking water are on average 0.0007 mg/L, with a maximum of 0.014 mg/L. Intakes derived for a young child (consuming 1 L/day and a body weight of 15.5 kg) are approximately 0.04 µg/kg/day.
- Concentrations of lead in air have been derived from Australian data on lead levels in urban, suburban and rural areas. (NSW DEC 2003) report concentrations of lead in air that range from 2.4 to 99 ng/m^3 with an average of 30 ng/m^3. Intakes derived from urban air are considered negligible in comparison with that derived from dietary and water sources;
- Total intakes from sources other than soil are estimated to be 0.44 µg/kg/day for adults based on intakes from dietary and water sources. This comprises approximately 6% of the adopted threshold value;
- 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, these are <10% of the adopted threshold value. For this assessment 10% intake from other sources has been considered for oral and dermal exposures.

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 2007c).

Absorption - Inhalation

For inhalation, absorption of inorganic lead will be influenced by particle size, solubility and age-related factors that determine breathing patterns. Larger particles (>2.5 µm) that are deposited in the ciliated airways (nasopharyngeal and tracheobronchial regions) can be transferred by mucociliary transport into the esophagus and swallowed. Smaller particles (<1 µm), which can be deposited in the alveolar region, can be absorbed after extracellular dissolution or ingestion by

phagocytic cells (ATSDR 2007c). Several studies have shown lead particles deposited in the alveoli of the lung are absorbed relatively quickly and completely. Most of the lead deposited in the alveoli is absorbed into the systemic circulation and little is brought up by cilliary action and swallowed (Safe Work Australia 2014a). This is in contrast to the larger particles (>2.5 µm) that are transferred within hours by mucociliary transport into the oesophagus and mainly swallowed, meaning the digestive tract can also be an important avenue of lead absorption following inhalation (Safe Work Australia 2014a).

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

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 2007c). 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 2000d). The gastrointestinal absorption of lead appears higher for children than adults, while the presence of food in the gastrointestinal tract decreases lead absorption. Deficiencies in dietary iron and calcium is believed to be related to higher lead absorption, as is pregnancy. The intake of lead via the oral route is considered a capacity limiting process, where the percentage of absorption may decrease with increased intake. Smaller lead particles are believed to be absorbed more readily, while lead in soil is absorbed less than dissolved lead (ATSDR 2007c).

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

- RIVM (Baars et al. 2001) use a relative bioavailability (the bioavailability from a soil matrix with respect to the bioavailability from the matrix in toxicity studies used to assess tolerable intakes) for lead of 0.6 (60%) in the derivation of serious (human health) risk concentrations.
- UK and US agencies have developed models based on the relationship between exposure and blood lead concentrations to derive soil guideline values.
    - The IEUBK model was developed in the US to describe the exposure of children to lead from multiple sources, and incorporates data on the toxicokinetics of lead – five exposure pathways are considered (air, water, diet, soil and dust). Using the various generic default parameters, including absorption factors of 0.3 for soil and dust, and 0.5 for food and water, a soil guideline value of 400 mg/kg is derived, and is considered appropriate for use in a residential scenario.

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

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

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

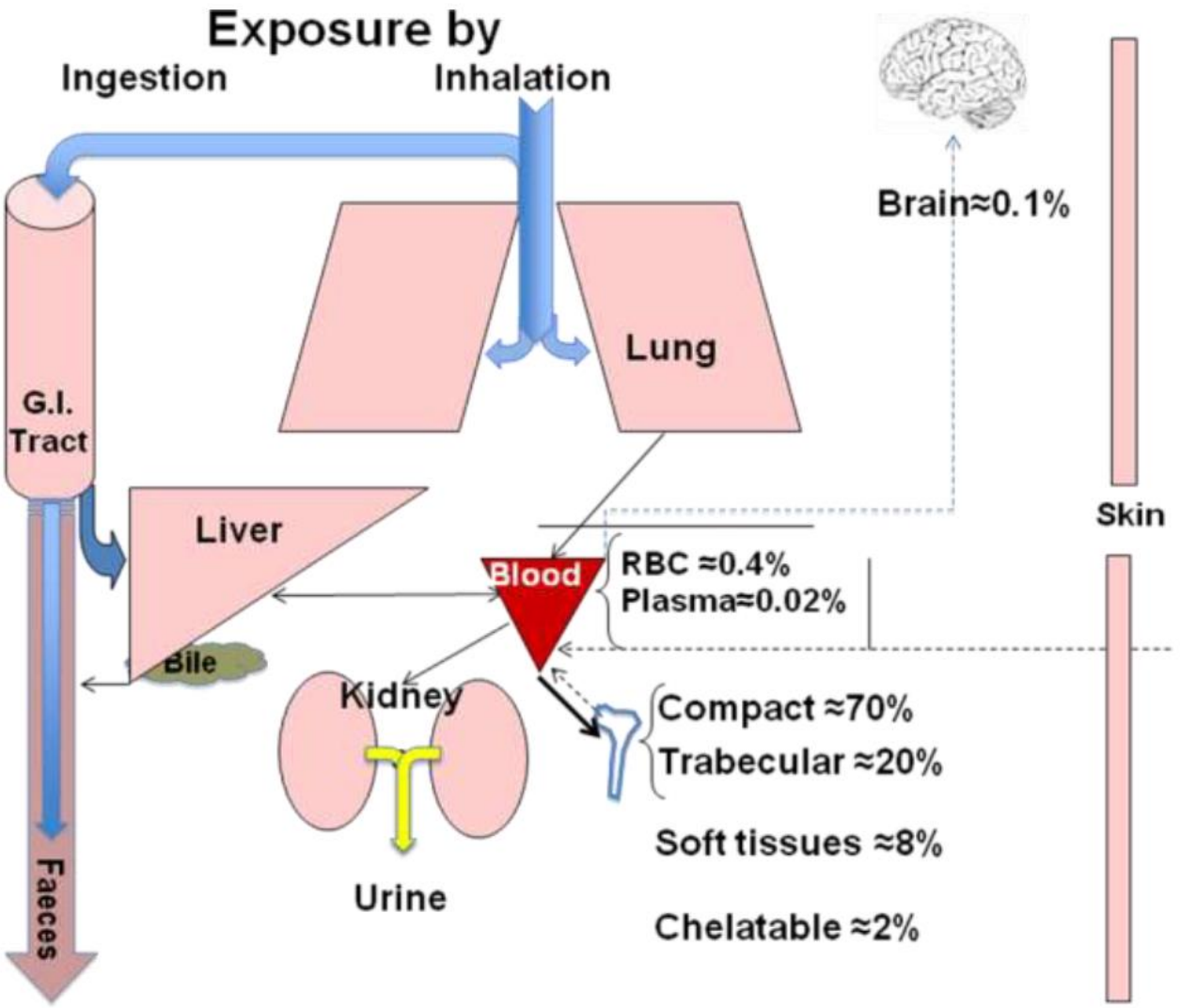
Where site specific bioaccessibility is available the bioavailability is adjusted to be 50% absorption x bioaccessible fraction.

Absorption - Dermal

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

Distribution

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



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

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

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

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

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

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

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

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

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 2007c).

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

Metabolism

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

Elimination

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

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

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 2007c).

Health Effects

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

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

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

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

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

The International Agency for Research on Cancer (IARC 2006) has classified inorganic lead as Group 2A: probably carcinogenic to humans. Organic lead was classified as Group 3: not classifiable (IARC 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 2011c). 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 2007c; Baars et al. 2001; UK DEFRA & EA 2002a; USEPA IRIS), however, blood lead goals and associated intakes have been identified by various agencies for the assessment of lead exposures by the general public. The NHMRC has noted that there are no benefits of human exposure to lead and that all demonstrated effects of exposure are adverse.

For the assessment of lead exposures in Australia, the current advice/statement from NHMRC on the evidence of health effects from lead, released in 2015 has been considered. This statement identified that the average Australian blood lead level was less than 5 micrograms per decilitre (µg/dL). Therefore, if an Australian had a blood lead level of 5 µg/dL or greater, and were not in a lead endemic area, this is a positive indicator of a non-background exposure to lead. Given that lead is not beneficial to human health, the NHMRC recommended that the non-background source be investigated and reduced (NHMRC 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 have acknowledged that health effects from blood lead levels greater than 10 µg/dL are well established. These effects include increased blood pressure, abnormally low haemoglobin, abnormal kidney function, long-term kidney damage and abnormal brain function. These health effects are summarised in the following figure (NHMRC 2015b).

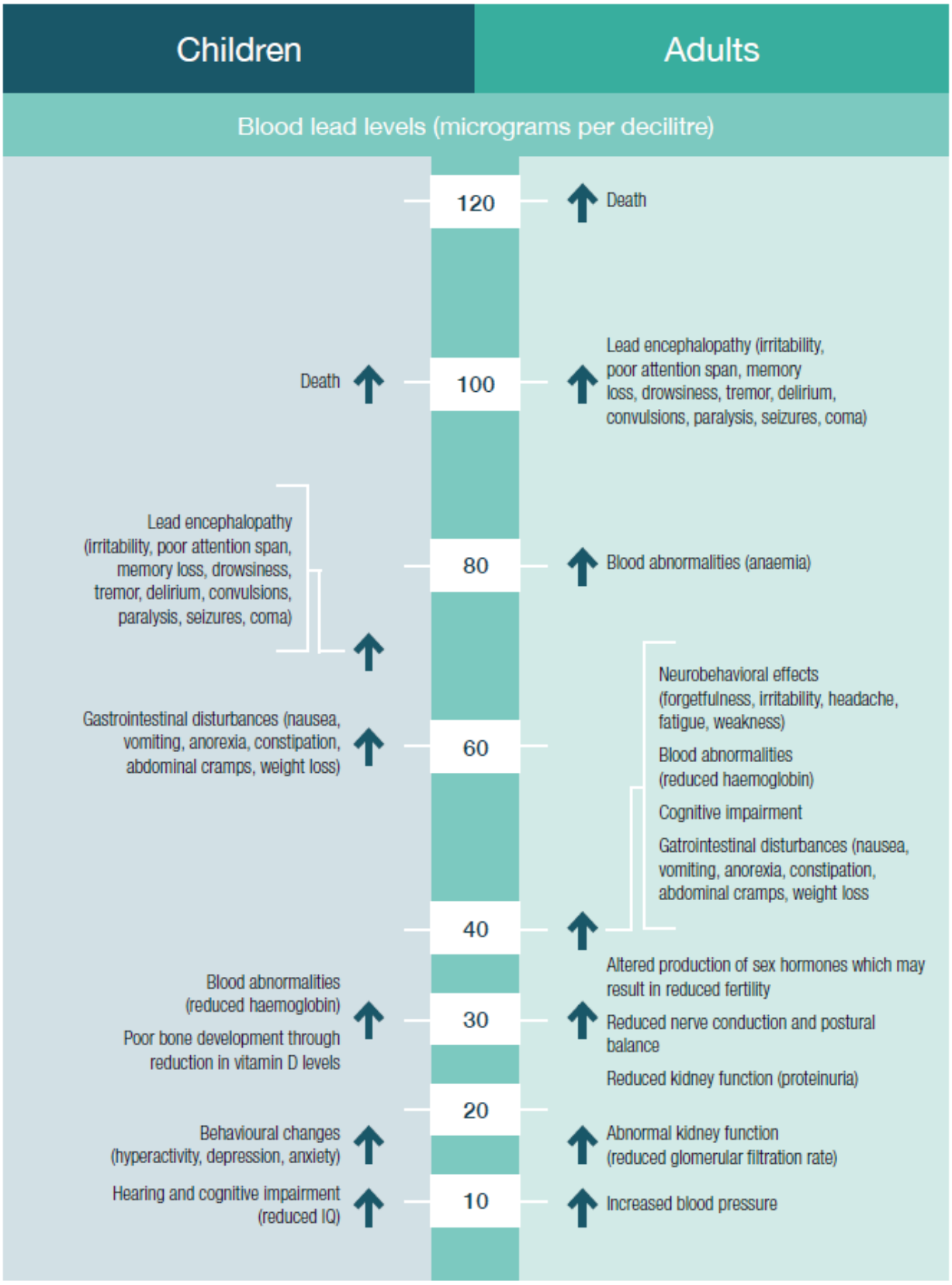


Figure: Summary of health effects of lead exposure above 10 µg/dL

However, for blood lead levels less than 10 µg/dL the evidence is less clear and must be treated with caution (Armstrong et al. 2014). This is because those studies that found a relationship (association) between blood lead levels below 10 µg/dL and health effects (such as reduced Intelligence Quotient) failed to account for other factors that may be responsible for the health effects (Armstrong et al. 2014). Further, for blood lead levels less than 10 µg/dL and cardiovascular effects it was concluded that *the clinical significance of the finding regarding increased blood pressure and increased risk of hypertension among adults and pregnant women may be minimal* (Armstrong et al. 2014). As a result, with regard to blood lead levels less than 10 µg/dL the NHMRC concluded that there is insufficient evidence that blood lead at this level caused any of the health effects observed (NHMRC 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 (2015). An upper concentration limit of lead, based on the protection of adverse health effect can be estimated using the IEUBK lead model as undertaken in the Contaminated sites NEPM (NEPC 1999 amended 2013b) and the blood lead criteria of 10 µg/dL, however this should not preclude the consideration of taking reasonable and feasible approaches to reduce exposures (where possible).

Approaches for the characterisation of hazards/toxicity

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

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.

Source	Value	Basis/Comments
NEPM (NEPC 1998b)	Air Quality Goal = 0.5 µg/m^3	Air guideline (based on an annual average) set by NEPM. Basis or the value is not stated; however, it is the same as that set by the WHO Air Quality Guidelines.
Safe Work Australia (Safe Work Australia 2014b)	Target PbB goals of 20 µg/dL Blood lead removal level 30 µg/dL	Relevant for nearly all workers, including females of non-reproductive capacity and males. For females of reproductive capacity, a lower blood lead goal is recommended, namely 10 µg/dL.
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 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 2000c)	TC = 0.5 µg/m^3	Air guideline (based on an annual average) established for lead based on an objective of 98% of the general population having a blood lead concentration of < 10 µg/dL, where the median blood lead levels would be no more than 5.4 µg/dL.
EFSA (EFSA 2010a)	PbB levels relevant for critical health effects Developmental effects in children: 1.2 µg/dL Renal effects in adults: 1.5 µg/dL Cardiovascular effects in adults: 3.6 µg/dL	Based on benchmark dose response levels for 1% change in IQ or blood pressure (BMDL01) and a 10% change in prevalence of CKD (considered significant for population health effects) (BMD10). EFSA also converted the blood lead goals to an intake using blood lead modelling.
UK DEFRA (DEFRA 2014)	PbB goals of 1.6 to 5 µg/dL	Conversion of blood lead criteria to intake dose levels of lead based on the IEUBK model for children and two different adult lead models for adults, refer to further discussion below.
CDC (CDC 2012)	PbB goal of 5 µg/dL	Recommends that the PbB goal be used to identify children aged 1-5 years may have elevated blood lead levels. The level is intended to trigger education, investigation and monitoring.

The more recent reviews of lead completed by EFSA (EFSA 2010a) and the UK DEFRA (UK DEFRA & EA 2014) have focused on the critical health endpoints for adults and children, using benchmark dose (BMD) modelling methods to identify blood lead levels associated with points of

departure considered to represent significant health outcomes, and the use of blood lead modelling to determine the intake (external intake of lead) that corresponds to the blood lead levels. The most detailed review of this process is presented by DEFRA (UK DEFRA & EA 2014), which is noted to be consistent with the EFSA evaluation, where the following can be summarised for the critical health endpoints identified.

Neurobehavioral effects in children

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

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

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

Cardiovascular effects (hypertension) in adults

The evaluation considered 4 human studies that relate 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), as this 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, which suggests some variability, and an average of 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.

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 2010a) and the

ALM is consistent with the modelling undertaken in Australia for adult lead exposures (NEPC 1999 amended 2013b).

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

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

The modelling undertaken was based on a 10% response level (BMD10) in having 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. It is acknowledged that the nature of the GFR endpoint is complex and causation at 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 DRFRA 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 2010a) and the ALM is consistent with the modelling undertaken in Australia for adult lead exposures (NEPC 1999 amended 2013b).

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 was 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 and cardiovascular effects in adults.

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
- Adults: 0.6 µg/kg/day
- Background intakes are 10% for oral and dermal exposures and negligible for inhalation exposures

B9 Manganese

General

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

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

Classification

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

Review of Available Values/Information

Insufficient data are available to assess whether manganese is carcinogenic to humans. Some *in vitro* and *in vivo* assays are available for manganese, with studies providing conflicting results. Overall review of the data shows that some chemical forms of manganese have mutagenic potential, however, most results are inconsistent and hence no overall conclusion as to the genotoxic potential associated with exposure to manganese can be determined (ATSDR 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.
USEPA (USEPA IRIS)	RfD = 0.14 mg/kg/day RfC = 0.00005 mg/m^3	RfD (last reviewed in 1993) based on a NOAEL of 0.14 mg/kg/day associated with CNS effects in a number of dietary human studies and an uncertainty factor of 1. The USEPA also note that individual requirements for and effects associated with manganese exposure may be highly variable and that some individuals may consume more than 10 mg/day of manganese without any cause for concern. RfC (last reviewed in 1993) based on the same study considered by ATSDR (2012) however the USEPA considered the LOAEL (HEC) of 0.05 mg/m^3 and applied an uncertainty factor of 1000.

As manganese toxicity via inhalation has been shown to be more significant than via oral intakes, it is reasonable that quantitative values for inhalation exposures are significantly lower than for oral exposures. Based on the available data an oral threshold value of 0.16 mg/kg/day as derived by ATSDR (2012) in the most recent detailed review of manganese toxicity. It is noted that the basis for the value is consistent with the upper range of manganese intake considered by the USEPA, NHMRC and WHO (NHMRC 2011 updated 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), 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), with 50% intakes from background sources
- Dermal absorption (DAF) = negligible (0%)
- Inhalation TRV (TRV_I) = 0.00015 mg/m^3 (WHO 1999a), with 20% intake from background sources

B10 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, noting that discussion relating to elemental mercury are also included.

Mercury occurs naturally as a mineral is widely distributed by natural and anthropogenic processes. The most significant natural source of atmospheric mercury is the degassing of the Earth's crust and oceans and emissions from volcanoes. Man-made sources such as mining, fossil fuel combustion and industrial emissions generally contribute less on a global scale, but more on a local scale. Wet and dry deposition to land and surface water result in mercury sorption to soil and sediments (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 2016) 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 1999) 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 is declining.
- 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 on speciation between elemental an inorganic). These concentrations comprise up to 10% of the recommended inhalation TRV.

Health Effects

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

Elemental Mercury (Hg^0)

General

Limited data is available concerning the absorption of elemental mercury. Inhaled mercury vapour by humans indicates approximately 80% of the vapour crosses the alveolar membranes into the blood. Ingested elemental mercury is poorly absorbed from the gastrointestinal tract (with approximately 0.01% absorbed, WHO 2003) unless there is an unusual delay in passage through the gastrointestinal tract or a gastrointestinal abnormality. This is partly due to the formation of sulfur laden compounds on the surface of the metal which prevents absorption. The processes of absorption in the gastrointestinal tract via sorption of mercury vapour (following partitioning in the GI tract to a vapour phase) have not been demonstrated in the available studies or case studies associated with accidental ingestion of elemental mercury. When evaluating exposures to elemental mercury, absorption following ingestion is too low to be of significance as the vapour inhalation pathway is of most importance.

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 mercuric mercury. Approximately 80% is excreted as mercuric mercury 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 2011a) has noted that "*Mercury reacts with skin proteins, and as a result penetration does not increase commensurably with increasing exposure concentration but rather approaches a plateau value. Mercury has a permeability coefficient in the order of 10^{-5} cm/h (Guy et al., 1999), which compares to permeability coefficients in the order of 10^{-4} cm/h for lead*." ATSDR (1999) note that absorption of mercurous salts in animals can occur through the skin, however no quantitative data are available, hence a default value of 0.1% has been adopted based on the lower end of the range for metals (USEPA 1995b).

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 2011c). The current US evaluation (USEPA IRIS) evaluation of mercuric chloride indicates that a linear low-dose extrapolation is not appropriate as kidney tumour seen in mice occurred at doses that were also nephrotoxic. On this basis, 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 2011c)	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.
WHO (WHO 2000e)	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. As inhalation is the most significant pathway of exposure relevant to this form of mercury, no values have been adopted for oral and dermal exposures.

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

B11 Nickel

Several comprehensive reviews of nickel in the environment and toxicity to humans are available (ATSDR 2005a; UK EA 2009b; 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 2009b) suggest that adult intakes from food are 69-162 µg/day (up to 2.3 µg/kg/day) and 130 µg/day (1.9 µg/kg/day) respectively. Intakes for children (ATSDR 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 2016) 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 2009b) 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.

Classification

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

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

Review of Available Values/Information

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

Oral

Review in the (WHO 2011c) 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 2009b)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 2011c)	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 2009b))	TDI = 0.012 mg/kg/day	Adopted the WHO evaluation presented in the WHO DWG.
TERA (TERA 1999)	RfD = 0.008 mg/kg/day	RfD derived for soluble nickel salts on the basis of a LOAEL of 7.6 mg/kg/day associated with kidney effects in rats and an uncertainty factor of 1000. The value derived was in addition to the diet rather than total intake.
ATSDR (ATSDR 2005a)	No oral MRL derived	
US EPA (IRIS 2012)	RfD = 0.02 mg/kg/day	RfD (last reviewed in 1991) based on a NOAEL of 5 mg/kg/day (same study as considered in the ADWG) and an uncertainty factor of 300.

Inhalation

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

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

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

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

Toxicity reference values – Inhalation

Source	Value	Basis/Comments
Australian – No guidelines derived		
International		
WHO (WHO 2000c)	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 2009b)	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 2011c) 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 2009b) 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 2011c) for oral and dermal routes of exposure
- Inhalation TRV (TRVi) = 0.00002 mg/m^3 (UK EA 2009b)
- Background intakes from other sources (as % of TRV) = 60% for oral and dermal intakes and 20% for inhalation intakes.

B12 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 in the form of these compounds 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.

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

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

B13 Zinc

General

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

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 2001b).

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 2001b).

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 2001b).

Zinc is an essential element for all living things, including man. 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 and 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 old's, 9 mg/day for 8-11 year old's and 12 mg/day for 12-18

year old's (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 2001b) 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 2001b). For a 2 year old child these intakes range from 0.4 to 0.9 mg/kg/day (80% to greater than 100% of the recommended TD). Based on mean intakes from Australian data, background intakes can be assumed to comprise up to 80% of the recommended oral TRV.

Classification

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

It is noted that the USEPA has evaluated zinc in their 2005 review (USEPA 2005d). 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. Other reviews of genotoxicity studies for zinc by EU and USEPA are equivocal (EU 2003; USEPA 2005d). 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 zinc intakes 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 2005d)	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 1995a)
- Background intakes have been assumed to be 80% for all exposures

Appendix C Characterisation of exposure

C1 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 2013b; USEPA 2009a).

This guidance requires the calculation of an exposure concentration which is based on the concentration in air and the time/duration spent in the area of impact. It is not dependent on age or body weight. The following equation outlines the calculation of an inhalation exposure concentration, and **Table C1** provides details on the assumptions adopted in this assessment:

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

for metals attached to dust as TSP.

Table C1: Inhalation exposure assumptions

Parameter		Value adopted	Basis
Ca	Concentration of chemical substance in air (mg/m^3)	Modelled in the Air Quality Assessment, where the maximum concentration from all receptors, maximum at all private 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.
RF	Retention factor	0.375	Fraction of TSP inhaled that can reach the lungs as per enHealth (enHealth 2012a)
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)

C2 Multiple pathway exposures

C2.1 Ingestion and dermal absorption

Chemical substances that are deposited on the ground have the potential to be ingested either directly through accidental consumption of dirt or indirectly through food grown or raised in the soil (fruit and vegetables, eggs, beef and milk) that is subsequently consumed.

The assessment of the potential ingestion of chemical substances has been undertaken using the approach presented by enHealth and the USEPA (enHealth 2012b; USEPA 1989). This approach is presented in the following equation, and parameters adopted in this assessment are presented in **Table C2**:

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

Chemical substances that are deposited on the ground have the potential to be absorbed through the skin when skin comes in contact with soil or dust.

The assessment of the potential dermal absorption of chemical substances has been generally undertaken using the approach presented by the USEPA (USEPA 1989, 2004). The USEPA define a simple approach to the evaluation of dermal absorption associated with soil contact. This is presented in the following equation and parameters adopted in this assessment are presented in **Table C2**:

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

For dermal contact with water, the equations are as follows (USEPA 2004):

$$DA_{event} = K_p \times C_w \times CF \times t_{event} \text{ (mg/m}^2\text{ per event), relevant to inorganics}$$

$$\text{Daily Chemical Intake}_{\text{Dermal}} = C_w \bullet \frac{SA \bullet DA_{event} \bullet EV \bullet EF \bullet ED}{BW \bullet AT} \quad \text{(mg/kg/day)}$$

Table C2: Ingestion and dermal exposure assumptions

Parameter		Value adopted		Basis
		Young children	Adults	
C_M	Concentration of chemical substance in media or relevance (soil, fruit and vegetables, eggs, beef or milk) (mg/kg)	Modelled based on deposition of particulates to soil (refer to **Section C2.2**)		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)
	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 2013b)
	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)
	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 2013b)
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 2013b)

Parameter		Value adopted		Basis
		Young children	Adults	
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 4.3**
Kp	Dermal permeability through skin (water) (cm/hr)	Chemical specific		Refer to **Table 4.3**
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 2013b)
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 2013b)
AT	Averaging time (days)	Threshold = ED x 365 days/year Non-threshold = 70 years x 365 days/year		As per enHealth (enHealth 2012b) guidance

C2.2 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 C3**.

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

Table C3: Assumptions adopted to estimate soil concentrations

Parameter		Value adopted		Basis
		Surface soil*	Agricultural soil*	
DR	Particle deposition rate ($mg/m^2/year$)	Calculated based on the maximum deposition rate of TSP and proportion of metals in TSP		Relevant to areas where multi-pathway exposures may occur
k	Chemical-specific soil-loss constant (1/year) = $ln(2)/T^{0.5}$	Calculated	Calculated	
$T^{0.5}$	Chemical half-life in soil (years)	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 C4**:

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

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

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

Table C4: Assumptions adopted to estimate concentration in fruit and vegetables

Parameter		Value adopted	Basis
DR	Particle deposition rate for accidental release (mg/m^2/day)	Calculated based on the maximum deposition rate of TSP and proportion of metals in TSP	Relevant to areas where multi-pathway exposures may occur
F	Fraction for the surface area of plant (unitless)	0.051	Relevant to aboveground exposed crops as per Stevens (1991) and OEHHA (OEHHA 2012)
k	Chemical-specific loss constant for particles on plants (1/days) = ln(2)/T$^{0.5}$	calculated	
T$^{0.5}$	Chemical half-life on plant (day)	14 days	Weathering of particulates on plant surfaces does occur and in the absence of measured data, it is generally assumed that 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 C3**
RUF	Root uptake factor (unitless)	Chemical specific value adopted	Root uptake factors from RAIS (RAIS) (soil to wet weight of plant)

Eggs, beef and milk

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

The concentration (C_P) calculated in eggs, beef or milk is calculated using the following equation (OEHHA 2015), with parameters and assumptions adopted presented in **Table C5**:

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

Table C5: Assumptions adopted to estimate concentration in animal produce

Parameter		Value adopted	Basis
FI	Fraction of grain/crop ingested by animals each day derived from the property (unitless)	100%	Assume all pasture/crops ingested by chickens and cows are grown on the property
IR_C	Ingestion rate of pasture/crops by each animal considered (kg/day)		
	Chickens	0.12 kg/day	Ingestion rate from OEHHA (2015)
	Beef cattle	9 kg/day	Ingestion rate from OEHHA (2015)
	Lactating cattle	22 kg/day	Ingestion rate for lactating cattle from OEHHA (2015)
C	Concentration of pollutant in crops consumed by animals (mg/kg)	Assume equal to that calculated in aboveground produce	Calculated as described above with assumptions in **Table C4**
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 C3**
B	Bioavailability of soil ingested (unitless)	100%	Conservative assumption
TF_P	Transfer factor for the produce of interest		
	Eggs	Chemical specific	Transfer factors adopted from OEHHA (2015), with the exception of chromium where the value was derived from an earlier OEHHA (OEHHA 2003) 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. The concentration in rainwater for Project related emissions, which may be used for all household purposes is calculated as follows, where the parameters adopted for this assessment are detailed in **Table C6**:

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

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

Table C6: Assumptions adopted to estimate concentration in rainwater tank

Parameter		Value adopted	Basis
DM	Mass of dust deposited on the roof each year (mg)	DR x Area	
DR	Particle deposition rate for accidental release (mg/m^2/year)	Modelled in the Air Quality Assessment for each receptor	Relevant to areas where multi-pathway exposures may occur
Area	Area of the roof (m^2)	200	Based on the average roof size for a 4 bedroom house in Australia (refer to Footnote 1)
VR	Volume of water collected from the roof each year	calculated	Equation as above
R	Rainfall each year (mm)	663.2	Average rainfall at Mudgee Airport for all years of records (1994 – 2019). No first flush devise is 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 mm to m		
Kd	Soil-water partition coefficient (cm^3/g)	Chemical-specific	All values from RAIS (RAIS)
ρ	Soil bulk density (g/m^3)	0.5	Assumed for loose deposited dust on roof (upper end measured for powders)

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

Calculations

All calculations relevant to the estimation of pollutant concentrations in soil, fruit and vegetables as well as animal products, and in rainwater tanks, are presented in **Appendix F.**

Appendix D Risk calculations – acute inhalation exposures

Predicted ground level concentrations and screening assessment - acute exposures

COPC	Acute air guideline (mg/m3)	Air Concentration - maximum from all receptors (mg/m^3)					Calculated HI				
		Year 1	Year 2	Year 4	Year 6	Year 8	Year 1	Year 2	Year 4	Year 6	Year 8
Antimony (Sb)	0.001	1.16E-07	2.34E-07	2.35E-07	1.86E-07	1.85E-07	1.2E-04	2.3E-04	2.4E-04	1.9E-04	1.8E-04
Arsenic (As)	0.003	6.21E-06	1.32E-05	1.33E-05	1.05E-05	1.06E-05	2.1E-03	4.4E-03	4.4E-03	3.5E-03	3.5E-03
Barium (Ba)	0.01	5.71E-06	1.14E-05	1.15E-05	9.10E-06	9.06E-06	5.7E-04	1.1E-03	1.2E-03	9.1E-04	9.1E-04
Beryllium (Be)	0.00001	1.06E-08	1.98E-08	1.96E-08	1.55E-08	1.52E-08	1.1E-03	2.0E-03	2.0E-03	1.5E-03	1.5E-03
Cadmium (Cd)	0.0054	1.31E-07	2.58E-07	2.58E-07	2.04E-07	2.02E-07	2.4E-05	4.8E-05	4.8E-05	3.8E-05	3.7E-05
Chromium (Cr)	0.0013	2.66E-06	4.80E-06	4.87E-06	3.84E-06	3.78E-06	2.0E-03	3.7E-03	3.7E-03	3.0E-03	2.9E-03
Copper (Cu)	0.1	1.94E-05	5.11E-05	5.32E-05	4.20E-05	4.34E-05	1.9E-04	5.1E-04	5.3E-04	4.2E-04	4.3E-04
Lead (Pb)	0.0005	4.10E-06	7.99E-06	8.12E-06	6.41E-06	6.37E-06	8.2E-03	1.6E-02	1.6E-02	1.3E-02	1.3E-02
Manganese (Mn)	0.0091	1.28E-04	2.62E-04	2.63E-04	2.08E-04	2.06E-04	1.4E-02	2.9E-02	2.9E-02	2.3E-02	2.3E-02
Mercury (Hg)	0.0006	1.78E-08	3.50E-08	3.50E-08	2.76E-08	2.73E-08	3.0E-05	5.8E-05	5.8E-05	4.6E-05	4.6E-05
Silver (Ag)	0.001	5.36E-08	1.23E-07	1.25E-07	9.86E-08	9.98E-08	5.4E-05	1.2E-04	1.2E-04	9.9E-05	1.0E-04
Nickel (Ni)	0.0011	1.35E-06	2.50E-06	2.54E-06	2.01E-06	1.98E-06	1.2E-03	2.3E-03	2.3E-03	1.8E-03	1.8E-03
Zinc (Zn)	0.12	4.10E-05	7.78E-05	7.73E-05	6.11E-05	6.00E-05	3.4E-04	6.5E-04	6.4E-04	5.1E-04	5.0E-04
						Total HI	**3.0E-02**	**6.0E-02**	**6.0E-02**	**4.8E-02**	**4.7E-02**

Appendix E Risk calculations – Chronic inhalation exposures

Inhalation exposures

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

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
Lung retention factor (RF, unitless)	0.375	Proportion of dust inhaled that reaches lungs, as per NEPM (1999 amended 2013)
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

Year 1

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)	
Antimony (Sb)	0.0E+00	3.2E-03	20%	2.5E-03	3.2E-09	6.0E-10	1.2E-09	--		0.00000048	0%
Arsenic (As)	0.0E+00	1.0E-03	0%	1.0E-03	1.7E-07	3.2E-08	6.5E-08	--		0.000065	1%
Barium (Ba)	0.0E+00	1.0E-03	0%	1.0E-03	1.6E-07	3.0E-08	5.9E-08	--		0.000059	0%
Beryllium (Be)	0.0E+00	2.0E-05	0%	2.0E-05	2.9E-10	5.5E-11	1.1E-10	--		0.0000055	0%
Cadmium (Cd)	0.0E+00	5.0E-06	20%	4.0E-06	3.6E-09	6.8E-10	1.4E-09	--		0.00034	3%
Chromium (Cr)	0.0E+00	1.0E-04	0%	1.0E-04	7.4E-08	1.4E-08	2.8E-08	--		0.00028	2%
Copper (Cu)	0.0E+00	4.9E-01	0%	4.9E-01	5.4E-07	1.0E-07	2.0E-07	--		0.00000041	0%
Lead (Pb)	0.0E+00	5.0E-04	0%	5.0E-04	1.1E-07	2.1E-08	4.3E-08	--		0.000085	1%
Manganese (Mn)	0.0E+00	1.5E-04	20%	1.2E-04	3.6E-06	6.7E-07	1.3E-06	--		0.011	87%
Mercury (Hg)	0.0E+00	2.0E-04	10%	1.8E-04	4.9E-10	9.2E-11	1.8E-10	--		0.0000010	0%
Silver (Ag)	0.0E+00	2.0E-02	0%	2.0E-02	1.5E-09	2.8E-10	5.6E-10	--		0.000000028	0%
Nickel (Ni)	0.0E+00	2.0E-05	20%	1.6E-05	3.8E-08	7.0E-09	1.4E-08	--		0.00088	7%
Zinc (Zn)	0.0E+00	1.8E+00	80%	3.5E-01	1.1E-06	2.1E-07	4.3E-07	--		0.0000012	0%
							TOTAL	**0.0E+00**		**0.013**	

Year 2

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)	
Antimony (Sb)	0.0E+00	3.2E-03	20%	2.5E-03	6.6E-09	1.2E-09	2.5E-09	--		0.0000010	0%
Arsenic (As)	0.0E+00	1.0E-03	0%	1.0E-03	3.7E-07	6.9E-08	1.4E-07	--		0.00014	1%
Barium (Ba)	0.0E+00	1.0E-03	0%	1.0E-03	3.2E-07	6.0E-08	1.2E-07	--		0.00012	0%
Beryllium (Be)	0.0E+00	2.0E-05	0%	2.0E-05	5.6E-10	1.0E-10	2.1E-10	--		0.000010	0%
Cadmium (Cd)	0.0E+00	5.0E-06	20%	4.0E-06	7.3E-09	1.4E-09	2.7E-09	--		0.00068	3%
Chromium (Cr)	0.0E+00	1.0E-04	0%	1.0E-04	1.3E-07	2.5E-08	5.1E-08	--		0.00051	2%
Copper (Cu)	0.0E+00	4.9E-01	0%	4.9E-01	1.4E-06	2.7E-07	5.4E-07	--		0.0000011	0%
Lead (Pb)	0.0E+00	5.0E-04	0%	5.0E-04	2.2E-07	4.2E-08	8.4E-08	--		0.00017	1%
Manganese (Mn)	0.0E+00	1.5E-04	20%	1.2E-04	7.4E-06	1.4E-06	2.8E-06	--		0.023	88%
Mercury (Hg)	0.0E+00	2.0E-04	10%	1.8E-04	9.8E-10	1.8E-10	3.7E-10	--		0.0000020	0%
Silver (Ag)	0.0E+00	2.0E-02	0%	2.0E-02	3.4E-09	6.5E-10	1.3E-09	--		0.000000065	0%
Nickel (Ni)	0.0E+00	2.0E-05	20%	1.6E-05	7.0E-08	1.3E-08	2.6E-08	--		0.0016	6%
Zinc (Zn)	0.0E+00	1.8E+00	80%	3.5E-01	2.2E-06	4.1E-07	8.2E-07	--		0.0000023	0%
							TOTAL	**0.0E+00**		**0.026**	

Year 4

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)	
Antimony (Sb)	0.0E+00	3.2E-03	20%	2.5E-03	6.7E-09	1.3E-09	2.5E-09	--		0.0000010	0%
Arsenic (As)	0.0E+00	1.0E-03	0%	1.0E-03	3.8E-07	7.1E-08	1.4E-07	--		0.00014	1%
Barium (Ba)	0.0E+00	1.0E-03	0%	1.0E-03	3.3E-07	6.1E-08	1.2E-07	--		0.00012	0%
Beryllium (Be)	0.0E+00	2.0E-05	0%	2.0E-05	5.6E-10	1.0E-10	2.1E-10	--		0.000010	0%
Cadmium (Cd)	0.0E+00	5.0E-06	20%	4.0E-06	7.3E-09	1.4E-09	2.8E-09	--		0.00069	3%
Chromium (Cr)	0.0E+00	1.0E-04	0%	1.0E-04	1.4E-07	2.6E-08	5.2E-08	--		0.00052	2%
Copper (Cu)	0.0E+00	4.9E-01	0%	4.9E-01	1.5E-06	2.8E-07	5.7E-07	--		0.0000012	0%
Lead (Pb)	0.0E+00	5.0E-04	0%	5.0E-04	2.3E-07	4.3E-08	8.7E-08	--		0.00017	1%
Manganese (Mn)	0.0E+00	1.5E-04	20%	1.2E-04	7.5E-06	1.4E-06	2.8E-06	--		0.023	87%
Mercury (Hg)	0.0E+00	2.0E-04	10%	1.8E-04	1.0E-09	1.9E-10	3.7E-10	--		0.0000021	0%
Silver (Ag)	0.0E+00	2.0E-02	0%	2.0E-02	3.6E-09	6.7E-10	1.3E-09	--		0.000000067	0%
Nickel (Ni)	0.0E+00	2.0E-05	20%	1.6E-05	7.2E-08	1.4E-08	2.7E-08	--		0.0017	6%
Zinc (Zn)	0.0E+00	1.8E+00	80%	3.5E-01	2.2E-06	4.1E-07	8.2E-07	--		0.0000024	0%
							TOTAL	**0.0E+00**		**0.027**	

Year 6

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)	
Antimony (Sb)	0.0E+00	3.2E-03	20%	2.5E-03	5.6E-09	1.1E-09	2.1E-09	--		0.00000084	0%
Arsenic (As)	0.0E+00	1.0E-03	0%	1.0E-03	3.2E-07	6.0E-08	1.2E-07	--		0.00012	0%
Barium (Ba)	0.0E+00	1.0E-03	0%	1.0E-03	2.8E-07	5.2E-08	1.0E-07	--		0.00010	0%
Beryllium (Be)	0.0E+00	2.0E-05	0%	2.0E-05	4.7E-10	8.8E-11	1.8E-10	--		0.0000088	0%
Cadmium (Cd)	0.0E+00	5.0E-06	20%	4.0E-06	6.2E-09	1.2E-09	2.3E-09	--		0.00058	2%
Chromium (Cr)	0.0E+00	1.0E-04	0%	1.0E-04	1.2E-07	2.2E-08	4.4E-08	--		0.00044	2%
Copper (Cu)	0.0E+00	4.9E-01	0%	4.9E-01	1.3E-06	2.4E-07	4.8E-07	--		0.0000010	0%
Lead (Pb)	0.0E+00	5.0E-04	0%	5.0E-04	1.9E-07	3.6E-08	7.3E-08	--		0.00015	1%
Manganese (Mn)	0.0E+00	1.5E-04	20%	1.2E-04	6.3E-06	1.2E-06	2.4E-06	--		0.020	74%
Mercury (Hg)	0.0E+00	2.0E-04	10%	1.8E-04	8.4E-10	1.6E-10	3.1E-10	--		0.0000017	0%
Silver (Ag)	0.0E+00	2.0E-02	0%	2.0E-02	3.0E-09	5.6E-10	1.1E-09	--		0.00000006	0%
Nickel (Ni)	0.0E+00	2.0E-05	20%	1.6E-05	6.1E-08	1.1E-08	2.3E-08	--		0.0014	5%
Zinc (Zn)	0.0E+00	1.8E+00	80%	3.5E-01	1.9E-06	3.5E-07	6.9E-07	--		0.0000020	0%
							TOTAL	**0.0E+00**		**0.023**	

Year 8

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)	
Antimony (Sb)	0.0E+00	3.2E-03	20%	2.5E-03	5.6E-09	1.1E-09	2.1E-09	--		0.00000083	0%
Arsenic (As)	0.0E+00	1.0E-03	0%	1.0E-03	3.2E-07	6.0E-08	1.2E-07	--		0.00012	1%
Barium (Ba)	0.0E+00	1.0E-03	0%	1.0E-03	2.7E-07	5.2E-08	1.0E-07	--		0.00010	0%
Beryllium (Be)	0.0E+00	2.0E-05	0%	2.0E-05	4.6E-10	8.6E-11	1.7E-10	--		0.0000086	0%
Cadmium (Cd)	0.0E+00	5.0E-06	20%	4.0E-06	6.1E-09	1.1E-09	2.3E-09	--		0.00057	3%
Chromium (Cr)	0.0E+00	1.0E-04	0%	1.0E-04	1.1E-07	2.2E-08	4.3E-08	--		0.00043	2%
Copper (Cu)	0.0E+00	4.9E-01	0%	4.9E-01	1.3E-06	2.5E-07	4.9E-07	--		0.0000010	0%
Lead (Pb)	0.0E+00	5.0E-04	0%	5.0E-04	1.9E-07	3.6E-08	7.2E-08	--		0.00014	1%
Manganese (Mn)	0.0E+00	1.5E-04	20%	1.2E-04	6.3E-06	1.2E-06	2.3E-06	--		0.020	87%
Mercury (Hg)	0.0E+00	2.0E-04	10%	1.8E-04	8.3E-10	1.6E-10	3.1E-10	--		0.0000017	0%
Silver (Ag)	0.0E+00	2.0E-02	0%	2.0E-02	3.0E-09	5.7E-10	1.1E-09	--		0.000000057	0%
Nickel (Ni)	0.0E+00	2.0E-05	20%	1.6E-05	6.0E-08	1.1E-08	2.3E-08	--		0.0014	6%
Zinc (Zn)	0.0E+00	1.8E+00	80%	3.5E-01	1.8E-06	3.4E-07	6.8E-07	--		0.0000019	0%
							TOTAL	**0.0E+00**		**0.022**	

Appendix F Risk calculations – Multipathway exposures

Calculation of Concentrations in Soil

$$C_s = \frac{DR \bullet \left[1 - e^{-k \bullet t}\right]}{d \bullet \rho \bullet k} \bullet 1000 \quad \textbf{(mg/kg)}$$ ref: Stevens B. (1991)

where:

DR=	Particle deposition rate (mg/m^2/year)
K =	Chemical-specific soil-loss constant (1/year) = ln(2)/T0.5
T0.5 =	Chemical half-life in soil (years)
t =	Accumulation time (years)
d =	Soil mixing depth (m)
ρ =	Soil bulk-density (g/m^3)
1000 =	Conversion from g to kg

General Parameters		Surface (for direct contact)	Depth (for agricultural pathways)	
Soil bulk density (ρ)	g/m^3	1600000	1600000	Default for fill materials
General mixing depth (d)	m	0.01	0.15	As per OEHHA (2015) guidance
Duration of deposition (T)	years	70	70	As per OEHHA (2015) guidance

Year 1

Chemical-specific Inputs and calculations - maximum all receptors

Chemical	Half-life in soil years	Loss constant (K) per year	Deposition Rate (DR) mg/m^2/year	Surface Concentration in Soil mg/kg	Agricultural Concentration in Soil mg/kg
Antimony (Sb)	273973	2.5E-06	9.2E-03	4.0E-02	2.7E-03
Arsenic (As)	273973	2.5E-06	4.9E-01	2.2E+00	1.4E-01
Barium (Ba)	273973	2.5E-06	4.5E-01	2.0E+00	1.3E-01
Beryllium (Be)	273973	2.5E-06	8.4E-04	3.7E-03	2.5E-04
Cadmium (Cd)	273973	2.5E-06	1.0E-02	4.5E-02	3.0E-03
Chromium (Cr)	273973	2.5E-06	2.1E-01	9.2E-01	6.2E-02
Copper (Cu)	273973	2.5E-06	1.5E+00	6.7E+00	4.5E-01
Lead (Pb)	273973	2.5E-06	3.3E-01	1.4E+00	9.5E-02
Manganese (Mn)	273973	2.5E-06	1.0E+01	4.5E+01	3.0E+00
Mercury (Hg)	273973	2.5E-06	1.4E-03	6.2E-03	4.1E-04
Silver (Ag)	273973	2.5E-06	4.3E-03	1.9E-02	1.2E-03
Nickel (Ni)	273973	2.5E-06	1.1E-01	4.7E-01	3.1E-02
Zinc (Zn)	273973	2.5E-06	3.3E+00	1.4E+01	9.5E-01

Year 2

Chemical-specific Inputs and calculations - maximum all receptors

Chemical	Half-life in soil years	Loss constant (K) per year	Deposition Rate (DR) mg/m^2/year	Surface Concentration in Soil mg/kg	Agricultural Concentration in Soil mg/kg
Antimony (Sb)	273973	2.5E-06	1.9E-02	8.3E-02	5.5E-03
Arsenic (As)	273973	2.5E-06	1.1E+00	4.7E+00	3.1E-01
Barium (Ba)	273973	2.5E-06	9.2E-01	4.0E+00	2.7E-01
Beryllium (Be)	273973	2.5E-06	1.6E-03	7.0E-03	4.7E-04
Cadmium (Cd)	273973	2.5E-06	2.1E-02	9.1E-02	6.1E-03
Chromium (Cr)	273973	2.5E-06	3.9E-01	1.7E+00	1.1E-01
Copper (Cu)	273973	2.5E-06	4.1E+00	1.8E+01	1.2E+00
Lead (Pb)	273973	2.5E-06	6.5E-01	2.8E+00	1.9E-01
Manganese (Mn)	273973	2.5E-06	2.1E+01	9.3E+01	6.2E+00
Mercury (Hg)	273973	2.5E-06	2.8E-03	1.2E-02	8.2E-04
Silver (Ag)	273973	2.5E-06	9.9E-03	4.3E-02	2.9E-03
Nickel (Ni)	273973	2.5E-06	2.0E-01	8.8E-01	5.9E-02
Zinc (Zn)	273973	2.5E-06	6.3E+00	2.8E+01	1.8E+00

Year 4

Chemical-specific Inputs and calculations - maximum all receptors					
Chemical	**Half-life in soil years**	**Loss constant (K) per year**	**Deposition Rate (DR) mg/m^2/year**	**Surface Concentration in Soil mg/kg**	**Agricultural Concentration in Soil mg/kg**
Antimony (Sb)	273973	2.5E-06	2.2E-02	9.8E-02	6.5E-03
Arsenic (As)	273973	2.5E-06	1.3E+00	5.6E+00	3.7E-01
Barium (Ba)	273973	2.5E-06	1.1E+00	4.8E+00	3.2E-01
Beryllium (Be)	273973	2.5E-06	1.9E-03	8.2E-03	5.5E-04
Cadmium (Cd)	273973	2.5E-06	2.5E-02	1.1E-01	7.2E-03
Chromium (Cr)	273973	2.5E-06	4.6E-01	2.0E+00	1.4E-01
Copper (Cu)	273973	2.5E-06	5.1E+00	2.2E+01	1.5E+00
Lead (Pb)	273973	2.5E-06	7.7E-01	3.4E+00	2.3E-01
Manganese (Mn)	273973	2.5E-06	2.5E+01	1.1E+02	7.3E+00
Mercury (Hg)	273973	2.5E-06	3.3E-03	1.5E-02	9.7E-04
Silver (Ag)	273973	2.5E-06	1.2E-02	5.2E-02	3.5E-03
Nickel (Ni)	273973	2.5E-06	2.4E-01	1.1E+00	7.1E-02
Zinc (Zn)	273973	2.5E-06	7.4E+00	3.2E+01	2.2E+00

Year 6

Chemical-specific Inputs and calculations - maximum all receptors					
Chemical	**Half-life in soil years**	**Loss constant (K) per year**	**Deposition Rate (DR) mg/m^2/year**	**Surface Concentration in Soil mg/kg**	**Agricultural Concentration in Soil mg/kg**
Antimony (Sb)	273973	2.5E-06	2.2E-02	9.4E-02	6.3E-03
Arsenic (As)	273973	2.5E-06	1.2E+00	5.3E+00	3.6E-01
Barium (Ba)	273973	2.5E-06	1.1E+00	4.6E+00	3.1E-01
Beryllium (Be)	273973	2.5E-06	1.8E-03	7.9E-03	5.2E-04
Cadmium (Cd)	273973	2.5E-06	2.4E-02	1.0E-01	6.9E-03
Chromium (Cr)	273973	2.5E-06	4.5E-01	1.9E+00	1.3E-01
Copper (Cu)	273973	2.5E-06	4.9E+00	2.1E+01	1.4E+00
Lead (Pb)	273973	2.5E-06	7.4E-01	3.2E+00	2.2E-01
Manganese (Mn)	273973	2.5E-06	2.4E+01	1.1E+02	7.0E+00
Mercury (Hg)	273973	2.5E-06	3.2E-03	1.4E-02	9.3E-04
Silver (Ag)	273973	2.5E-06	1.1E-02	5.0E-02	3.3E-03
Nickel (Ni)	273973	2.5E-06	2.3E-01	1.0E+00	6.8E-02
Zinc (Zn)	273973	2.5E-06	7.1E+00	3.1E+01	2.1E+00

Year 8

Chemical-specific Inputs and calculations - maximum all receptors					
Chemical	**Half-life in soil years**	**Loss constant (K) per year**	**Deposition Rate (DR) mg/m^2/year**	**Surface Concentration in Soil mg/kg**	**Agricultural Concentration in Soil mg/kg**
Antimony (Sb)	273973	2.5E-06	2.1E-02	9.4E-02	6.2E-03
Arsenic (As)	273973	2.5E-06	1.2E+00	5.4E+00	3.6E-01
Barium (Ba)	273973	2.5E-06	1.1E+00	4.6E+00	3.1E-01
Beryllium (Be)	273973	2.5E-06	1.8E-03	7.7E-03	5.1E-04
Cadmium (Cd)	273973	2.5E-06	2.3E-02	1.0E-01	6.8E-03
Chromium (Cr)	273973	2.5E-06	4.4E-01	1.9E+00	1.3E-01
Copper (Cu)	273973	2.5E-06	5.0E+00	2.2E+01	1.5E+00
Lead (Pb)	273973	2.5E-06	7.4E-01	3.2E+00	2.2E-01
Manganese (Mn)	273973	2.5E-06	2.4E+01	1.0E+02	7.0E+00
Mercury (Hg)	273973	2.5E-06	3.2E-03	1.4E-02	9.2E-04
Silver (Ag)	273973	2.5E-06	1.2E-02	5.1E-02	3.4E-03
Nickel (Ni)	273973	2.5E-06	2.3E-01	1.0E+00	6.7E-02
Zinc (Zn)	273973	2.5E-06	7.0E+00	3.0E+01	2.0E+00

Calculation of Concentrations in Rainwater tank

CW = DM/(VR*Kd*ρ) **(mg/L)**

where:

DM =	Mass of dust deposited on roof each year (mg) = DR x Area
DR =	Deposition rate from model (mg/m2/year)
Area =	Area of roof (m2)
VR =	Volume of water collected from roof over year (L) = R x Area x Rc/1000
R =	Rainfall each year (mm)
ρ =	Soil bulk-density (g/m^3)
Rc =	Runoff coefficient (unitless)
Kd =	Soil-water partition coefficient (cm3/g)
1000 =	Conversion from mm to m

General Parameters			
Average rainfaill	mm/year	663.2	mean for all years (1994 - 2019) for Mudgee airport
Roof area	m2	200	4 bedroom australian home
Runoff coefficient	-	0.7	assumes 30% water loss in capture into tank
Volume of rainwater	m3/year	92.848	calculated
Volume of rainwater	L/year	92848	
Bulk density of deposited dust	g/cm3	0.5	assumed for loose deposited dust on roof (similar to upper end measured for powders)

Year 1

Chemical-specific Inputs and calculations - maximum all receptors

Chemical	PM10 Deposition Rate (DR) mg/m^2/year	PM10 Mass deposited each year (DM) mg	Kd (cm3/g)	Particulate Concentration in water mg/L	Dissolved Concentration in water mg/L
Antimony (Sb)	0.0092	1.8	45.0	2.0E-05	8.8E-07
Arsenic (As)	0.4933	98.7	29	1.1E-03	7.3E-05
Barium (Ba)	0.4534	90.7	41	9.8E-04	4.8E-05
Beryllium (Be)	0.0008	0.2	790	1.8E-06	4.6E-09
Cadmium (Cd)	0.0104	2.1	75	2.2E-05	6.0E-07
Chromium (Cr)	0.2110	42.2	19	4.5E-04	4.8E-05
Copper (Cu)	1.5379	307.6	35	3.3E-03	1.9E-04
Lead (Pb)	0.3254	65.1	900	7.0E-04	1.6E-06
Manganese (Mn)	10.1993	2039.9	65	2.2E-02	6.8E-04
Mercury (Hg)	0.0014	0.3	52	3.0E-06	1.2E-07
Silver (Ag)	0.0043	0.9	8	9.2E-06	2.2E-06
Nickel (Ni)	0.1076	21.5	65	2.3E-04	7.1E-06
Zinc (Zn)	3.2592	651.8	62	7.0E-03	2.3E-04

Year 2

Chemical-specific Inputs and calculations - maximum private residences

Chemical	PM10 Deposition Rate (DR) mg/m^2/year	PM10 Mass deposited each year (DM) mg	Kd (cm3/g)	Particulate Concentration in water mg/L	Dissolved Concentration in water mg/L
Antimony (Sb)	0.0189	3.8	45	4.1E-05	1.8E-06
Arsenic (As)	1.0641	212.8	29	2.3E-03	1.6E-04
Barium (Ba)	0.9230	184.6	41	2.0E-03	9.7E-05
Beryllium (Be)	0.0016	0.3	790	3.4E-06	8.7E-09
Cadmium (Cd)	0.0209	4.2	75	4.5E-05	1.2E-06
Chromium (Cr)	0.3881	77.6	19	8.4E-04	8.8E-05
Copper (Cu)	4.1292	825.8	35	8.9E-03	5.1E-04
Lead (Pb)	0.6460	129.2	900	1.4E-03	3.1E-06
Manganese (Mn)	21.1831	4236.6	65	4.6E-02	1.4E-03
Mercury (Hg)	0.0028	0.6	52	6.1E-06	2.3E-07
Silver (Ag)	0.0099	2.0	8	2.1E-05	5.1E-06
Nickel (Ni)	0.2022	40.4	65	4.4E-04	1.3E-05
Zinc (Zn)	6.2868	1257.4	62	1.4E-02	4.4E-04

Year 4

Chemical-specific Inputs and calculations - maximum private residences					
Chemical	PM10 Deposition Rate (DR) mg/m^2/year	PM10 Mass deposited each year (DM) mg	Kd (cm3/g)	Particulate Concentration in water mg/L	Dissolved Concentration in water mg/L
Antimony (Sb)	0.0224	4.5	45	4.8E-05	2.1E-06
Arsenic (As)	1.2734	254.7	29	2.7E-03	1.9E-04
Barium (Ba)	1.1002	220.0	41	2.4E-03	1.2E-04
Beryllium (Be)	0.0019	0.4	790	4.0E-06	1.0E-08
Cadmium (Cd)	0.0246	4.9	75	5.3E-05	1.4E-06
Chromium (Cr)	0.4642	92.8	19	1.0E-03	1.1E-04
Copper (Cu)	5.0780	1015.6	35	1.1E-02	6.3E-04
Lead (Pb)	0.7743	154.9	900	1.7E-03	3.7E-06
Manganese (Mn)	25.0891	5017.8	65	5.4E-02	1.7E-03
Mercury (Hg)	0.0033	0.7	52	7.2E-06	2.8E-07
Silver (Ag)	0.0119	2.4	8	2.6E-05	6.2E-06
Nickel (Ni)	0.2425	48.5	65	5.2E-04	1.6E-05
Zinc (Zn)	7.3778	1475.6	62	1.6E-02	5.1E-04

Year 6

Chemical-specific Inputs and calculations - maximum private residences					
Chemical	PM10 Deposition Rate (DR) mg/m^2/year	PM10 Mass deposited each year (DM) mg	Kd (cm3/g)	Particulate Concentration in water mg/L	Dissolved Concentration in water mg/L
Antimony (Sb)	0.0215	4.3	45	4.6E-05	2.1E-06
Arsenic (As)	1.2215	244.3	29	2.6E-03	1.8E-04
Barium (Ba)	1.0553	211.1	41	2.3E-03	1.1E-04
Beryllium (Be)	0.0018	0.4	790	3.9E-06	9.8E-09
Cadmium (Cd)	0.0236	4.7	75	5.1E-05	1.4E-06
Chromium (Cr)	0.4453	89.1	19	9.6E-04	1.0E-04
Copper (Cu)	4.8708	974.2	35	1.0E-02	6.0E-04
Lead (Pb)	0.7427	148.5	900	1.6E-03	3.6E-06
Manganese (Mn)	24.0657	4813.1	65	5.2E-02	1.6E-03
Mercury (Hg)	0.0032	0.6	52	6.9E-06	2.7E-07
Silver (Ag)	0.0114	2.3	8	2.5E-05	5.9E-06
Nickel (Ni)	0.2326	46.5	65	5.0E-04	1.5E-05
Zinc (Zn)	7.0768	1415.4	62	1.5E-02	4.9E-04

Year 8

Chemical-specific Inputs and calculations - maximum private residences					
Chemical	PM10 Deposition Rate (DR) mg/m^2/year	PM10 Mass deposited each year (DM) mg	Kd (cm3/g)	Particulate Concentration in water mg/L	Dissolved Concentration in water mg/L
Antimony (Sb)	0.0214	4.3	45	4.6E-05	2.1E-06
Arsenic (As)	1.2249	245.0	29	2.6E-03	1.8E-04
Barium (Ba)	1.0500	210.0	41	2.3E-03	1.1E-04
Beryllium (Be)	0.0018	0.4	790	3.8E-06	9.6E-09
Cadmium (Cd)	0.0234	4.7	75	5.0E-05	1.3E-06
Chromium (Cr)	0.4385	87.7	19	9.4E-04	9.9E-05
Copper (Cu)	5.0350	1007.0	35	1.1E-02	6.2E-04
Lead (Pb)	0.7386	147.7	900	1.6E-03	3.5E-06
Manganese (Mn)	23.9336	4786.7	65	5.2E-02	1.6E-03
Mercury (Hg)	0.0032	0.6	52	6.8E-06	2.6E-07
Silver (Ag)	0.0116	2.3	8	2.5E-05	6.0E-06
Nickel (Ni)	0.2300	46.0	65	5.0E-04	1.5E-05
Zinc (Zn)	6.9509	1390.2	62	1.5E-02	4.8E-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 accidental release (mg/m^2/day)
F= Fraction for the surface area of plant (unitless)
k= Chemical-specific soil-loss constant (1/years) = ln(2)/$T_{0.5}$
$T_{0.5}$= Chemical half-life as particulate on plant (days)
t= Deposition time (days)
Y= Crop yield (kg/m^2)

Uptake via Roots from Soil

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

where:
Cs = Concentration of persistent chemical in soil assuming 15cm mixing depth within gardens, calculated using Soil Equation for each chemical assessed (mg/kg)
RUF = Root uptake factor which differs for each Chemical (unitless)

General Parameters	Units	Value
Crop		Edible crops
Crop Yield (Y)	kg/m^2	2
Deposition Time (t)	days	70
Plant Interception fraction (F)	unitless	0.051

Year 1

Chemical-specific Inputs and calculations - All receptors

Chemical	Half-life in plant ($T_{0.5}$)	Loss constant (k)	Deposition Rate (DR)	Aboveground Produce Concentration via Deposition	Root Uptake Factor (RUF)	Soil Concentration (Cs)	Below Ground Produce Concentration
	days	per day	mg/m^2/day	mg/kg ww	unitless	mg/kg	mg/kg ww
Antimony (Sb)	14	0.05	0.0000252	1.3E-05	0.05	2.7E-03	1.3E-04
Arsenic (As)	14	0.05	0.0013515	6.7E-04	0.01	1.4E-01	1.4E-03
Barium (Ba)	14	0.05	0.0012423	6.2E-04	0.0375	1.3E-01	5.0E-03
Beryllium (Be)	14	0.05	0.0000023	1.1E-06	0.0025	2.5E-04	6.1E-07
Cadmium (Cd)	14	0.05	0.0000285	1.4E-05	0.125	3.0E-03	3.8E-04
Chromium (Cr)	14	0.05	0.0005782	2.9E-04	0.00187	6.2E-02	1.2E-04
Copper (Cu)	14	0.05	0.0042134	2.1E-03	0.1	4.5E-01	4.5E-02
Lead (Pb)	14	0.05	0.0008915	4.4E-04	0.0112	9.5E-02	1.1E-03
Manganese (Mn)	14	0.05	0.0279433	1.4E-02	0.0625	3.0E+00	1.9E-01
Mercury (Hg)	14	0.05	0.0000039	1.9E-06	0.225	4.1E-04	9.3E-05
Silver (Ag)	14	0.05	0.0000117	5.8E-06	0.1	1.2E-03	1.2E-04
Nickel (Ni)	14	0.05	0.0002947	1.5E-04	0.015	3.1E-02	4.7E-04
Zinc (Zn)	14	0.05	0.0089293	4.5E-03	0.264	9.5E-01	2.5E-01

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

Year 2

Chemical-specific Inputs and calculations - All receptors

Chemical	Half-life in plant ($T_{0.5}$)	Loss constant (k)	Deposition Rate (DR)	Aboveground Produce Concentration via Deposition	Root Uptake Factor (RUF)	Soil Concentration (Cs)	Below Ground Produce Concentration
	days	per day	mg/m^2/day	mg/kg ww	unitless	mg/kg	mg/kg ww
Antimony (Sb)	14	0.05	0.0000517	2.6E-05	0.05	5.5E-03	2.8E-04
Arsenic (As)	14	0.05	0.0029152	1.5E-03	0.01	3.1E-01	3.1E-03
Barium (Ba)	14	0.05	0.0025288	1.3E-03	0.0375	2.7E-01	1.0E-02
Beryllium (Be)	14	0.05	0.0000044	2.2E-06	0.0025	4.7E-04	1.2E-06
Cadmium (Cd)	14	0.05	0.0000571	2.9E-05	0.125	6.1E-03	7.6E-04
Chromium (Cr)	14	0.05	0.0010634	5.3E-04	0.00187	1.1E-01	2.1E-04
Copper (Cu)	14	0.05	0.0113130	5.6E-03	0.1	1.2E+00	1.2E-01
Lead (Pb)	14	0.05	0.0017700	8.8E-04	0.0112	1.9E-01	2.1E-03
Manganese (Mn)	14	0.05	0.0580358	2.9E-02	0.0625	6.2E+00	3.9E-01
Mercury (Hg)	14	0.05	0.0000077	3.9E-06	0.225	8.2E-04	1.9E-04
Silver (Ag)	14	0.05	0.0000271	1.4E-05	0.1	2.9E-03	2.9E-04
Nickel (Ni)	14	0.05	0.0005539	2.8E-04	0.015	5.9E-02	8.8E-04
Zinc (Zn)	14	0.05	0.0172241	8.6E-03	0.264	1.8E+00	4.8E-01

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

Year 4

Chemical-specific Inputs and calculations - All receptors							
Chemical	**Half-life in plant ($T_{0.5}$)**	**Loss constant (k)**	**Deposition Rate (DR)**	**Aboveground Produce Concentration via Deposition**	**Root Uptake Factor (RUF)**	**Soil Concentration (Cs)**	**Below Ground Produce Concentration**
	days	**per day**	**mg/m^2/day**	**mg/kg ww**	**unitless**	**mg/kg**	**mg/kg ww**
Antimony (Sb)	14	0.05	0.0000615	3.1E-05	0.05	6.5E-03	3.3E-04
Arsenic (As)	14	0.05	0.0034888	1.7E-03	0.01	3.7E-01	3.7E-03
Barium (Ba)	14	0.05	0.0030143	1.5E-03	0.0375	3.2E-01	1.2E-02
Beryllium (Be)	14	0.05	0.0000051	2.6E-06	0.0025	5.5E-04	1.4E-06
Cadmium (Cd)	14	0.05	0.0000675	3.4E-05	0.125	7.2E-03	9.0E-04
Chromium (Cr)	14	0.05	0.0012718	6.3E-04	0.00187	1.4E-01	2.5E-04
Copper (Cu)	14	0.05	0.0139123	6.9E-03	0.1	1.5E+00	1.5E-01
Lead (Pb)	14	0.05	0.0021213	1.1E-03	0.0112	2.3E-01	2.5E-03
Manganese (Mn)	14	0.05	0.0687374	3.4E-02	0.0625	7.3E+00	4.6E-01
Mercury (Hg)	14	0.05	0.0000091	4.6E-06	0.225	9.7E-04	2.2E-04
Silver (Ag)	14	0.05	0.0000326	1.6E-05	0.1	3.5E-03	3.5E-04
Nickel (Ni)	14	0.05	0.0006644	3.3E-04	0.015	7.1E-02	1.1E-03
Zinc (Zn)	14	0.05	0.0202130	1.0E-02	0.264	2.2E+00	5.7E-01

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

Year 6

Chemical-specific Inputs and calculations - All receptors							
Chemical	**Half-life in plant ($T_{0.5}$)**	**Loss constant (k)**	**Deposition Rate (DR)**	**Aboveground Produce Concentration via Deposition**	**Root Uptake Factor (RUF)**	**Soil Concentration (Cs)**	**Below Ground Produce Concentration**
	days	**per day**	**mg/m^2/day**	**mg/kg ww**	**unitless**	**mg/kg**	**mg/kg ww**
Antimony (Sb)	14	0.05	0.0000590	2.9E-05	0.05	6.3E-03	3.1E-04
Arsenic (As)	14	0.05	0.0033465	1.7E-03	0.01	3.6E-01	3.6E-03
Barium (Ba)	14	0.05	0.0028913	1.4E-03	0.0375	3.1E-01	1.2E-02
Beryllium (Be)	14	0.05	0.0000049	2.5E-06	0.0025	5.2E-04	1.3E-06
Cadmium (Cd)	14	0.05	0.0000647	3.2E-05	0.125	6.9E-03	8.6E-04
Chromium (Cr)	14	0.05	0.0012199	6.1E-04	0.00187	1.3E-01	2.4E-04
Copper (Cu)	14	0.05	0.0133448	6.7E-03	0.1	1.4E+00	1.4E-01
Lead (Pb)	14	0.05	0.0020348	1.0E-03	0.0112	2.2E-01	2.4E-03
Manganese (Mn)	14	0.05	0.0659335	3.3E-02	0.0625	7.0E+00	4.4E-01
Mercury (Hg)	14	0.05	0.0000088	4.4E-06	0.225	9.3E-04	2.1E-04
Silver (Ag)	14	0.05	0.0000313	1.6E-05	0.1	3.3E-03	3.3E-04
Nickel (Ni)	14	0.05	0.0006373	3.2E-04	0.015	6.8E-02	1.0E-03
Zinc (Zn)	14	0.05	0.0193885	9.7E-03	0.264	2.1E+00	5.4E-01

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

Year 8

Chemical-specific Inputs and calculations - All receptors							
Chemical	**Half-life in plant ($T_{0.5}$)**	**Loss constant (k)**	**Deposition Rate (DR)**	**Aboveground Produce Concentration via Deposition**	**Root Uptake Factor (RUF)**	**Soil Concentration (Cs)**	**Below Ground Produce Concentration**
	days	**per day**	**mg/m^2/day**	**mg/kg ww**	**unitless**	**mg/kg**	**mg/kg ww**
Antimony (Sb)	14	0.05	0.0000587	2.9E-05	0.05	6.2E-03	3.1E-04
Arsenic (As)	14	0.05	0.0033560	1.7E-03	0.01	3.6E-01	3.6E-03
Barium (Ba)	14	0.05	0.0028768	1.4E-03	0.0375	3.1E-01	1.1E-02
Beryllium (Be)	14	0.05	0.0000048	2.4E-06	0.0025	5.1E-04	1.3E-06
Cadmium (Cd)	14	0.05	0.0000641	3.2E-05	0.125	6.8E-03	8.5E-04
Chromium (Cr)	14	0.05	0.0012013	6.0E-04	0.00187	1.3E-01	2.4E-04
Copper (Cu)	14	0.05	0.0137945	6.9E-03	0.1	1.5E+00	1.5E-01
Lead (Pb)	14	0.05	0.0020236	1.0E-03	0.0112	2.2E-01	2.4E-03
Manganese (Mn)	14	0.05	0.0655714	3.3E-02	0.0625	7.0E+00	4.4E-01
Mercury (Hg)	14	0.05	0.0000087	4.3E-06	0.225	9.2E-04	2.1E-04
Silver (Ag)	14	0.05	0.0000317	1.6E-05	0.1	3.4E-03	3.4E-04
Nickel (Ni)	14	0.05	0.0006300	3.1E-04	0.015	6.7E-02	1.0E-03
Zinc (Zn)	14	0.05	0.0190435	9.5E-03	0.264	2.0E+00	5.4E-01

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

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

Year 1

Chemical-specific Inputs and calculations - Maximum all receptors

Chemical	Concentration in crops ingested by chickens mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to eggs day/kg	Egg Concentration mg/kg ww	
Antimony (Sb)	1.3E-05	2.7E-03	3.8E-02	3.0E-07	
Arsenic (As)	6.7E-04	1.4E-01	7.0E-02	3.0E-05	
Barium (Ba)	6.2E-04	1.3E-01	3.8E-02	1.5E-05	
Beryllium (Be)	1.1E-06	2.5E-04	9.0E-02	6.5E-08	
Cadmium (Cd)	1.4E-05	3.0E-03	1.0E-02	9.0E-08	
Chromium (Cr)	2.9E-04	6.2E-02	9.2E-03	1.7E-06	OEHHA (2003)
Copper (Cu)	2.1E-03	4.5E-01	3.8E-02	5.0E-05	
Lead (Pb)	4.4E-04	9.5E-02	4.0E-02	1.1E-05	
Manganese (Mn)	1.4E-02	3.0E+00	3.8E-02	3.3E-04	
Mercury (Hg)	1.9E-06	4.1E-04	8.0E-01	9.8E-07	
Silver (Ag)	5.8E-06	1.2E-03	3.8E-02	1.4E-07	
Nickel (Ni)	1.5E-04	3.1E-02	2.0E-02	1.9E-06	
Zinc (Zn)	4.5E-03	9.5E-01	3.8E-02	1.1E-04	

Transfer factors from OEHHA 2015 unless otherwise noted
Mean transfer factor for heavy metals used in absense of specific data (Leeman et al 2007)

Year 2

Chemical-specific Inputs and calculations - Maximum all receptors

Chemical	Concentration in crops ingested by chickens mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to eggs day/kg	Egg Concentration mg/kg ww	
Antimony (Sb)	2.6E-05	5.5E-03	3.8E-02	6.2E-07	
Arsenic (As)	1.5E-03	3.1E-01	7.0E-02	6.4E-05	
Barium (Ba)	1.3E-03	2.7E-01	3.8E-02	3.0E-05	
Beryllium (Be)	2.2E-06	4.7E-04	9.0E-02	1.2E-07	
Cadmium (Cd)	2.9E-05	6.1E-03	1.0E-02	1.8E-07	
Chromium (Cr)	5.3E-04	1.1E-01	9.2E-03	3.1E-06	OEHHA (2003)
Copper (Cu)	5.6E-03	1.2E+00	3.8E-02	1.4E-04	
Lead (Pb)	8.8E-04	1.9E-01	4.0E-02	2.2E-05	
Manganese (Mn)	2.9E-02	6.2E+00	3.8E-02	7.0E-04	
Mercury (Hg)	3.9E-06	8.2E-04	8.0E-01	2.0E-06	
Silver (Ag)	1.4E-05	2.9E-03	3.8E-02	3.3E-07	
Nickel (Ni)	2.8E-04	5.9E-02	2.0E-02	3.5E-06	
Zinc (Zn)	8.6E-03	1.8E+00	3.8E-02	2.1E-04	

Transfer factors from OEHHA 2015 unless otherwise noted
Mean transfer factor for heavy metals used in absense of specific data (Leeman et al 2007)

Year 4

Chemical-specific Inputs and calculations - Maximum all receptors					
Chemical	**Concentration in crops ingested by chickens mg/kg ww**	**Soil Concentration - Agriculture (Cs) mg/kg**	**Transfer factor to eggs day/kg**	**Egg Concentration mg/kg ww**	
Antimony (Sb)	3.1E-05	6.5E-03	3.8E-02	7.4E-07	
Arsenic (As)	1.7E-03	3.7E-01	7.0E-02	7.7E-05	
Barium (Ba)	1.5E-03	3.2E-01	3.8E-02	3.6E-05	
Beryllium (Be)	2.6E-06	5.5E-04	9.0E-02	1.5E-07	
Cadmium (Cd)	3.4E-05	7.2E-03	1.0E-02	2.1E-07	
Chromium (Cr)	6.3E-04	1.4E-01	9.2E-03	3.7E-06	OEHHA (2003)
Copper (Cu)	6.9E-03	1.5E+00	3.8E-02	1.7E-04	
Lead (Pb)	1.1E-03	2.3E-01	4.0E-02	2.7E-05	
Manganese (Mn)	3.4E-02	7.3E+00	3.8E-02	8.2E-04	
Mercury (Hg)	4.6E-06	9.7E-04	8.0E-01	2.3E-06	
Silver (Ag)	1.6E-05	3.5E-03	3.8E-02	3.9E-07	
Nickel (Ni)	3.3E-04	7.1E-02	2.0E-02	4.2E-06	
Zinc (Zn)	1.0E-02	2.2E+00	3.8E-02	2.4E-04	

Transfer factors from OEHHA 2015 unless otherwise noted

Mean transfer factor for heavy metals used in absense of specific data (Leeman et al 2007)

Year 6

Chemical-specific Inputs and calculations - Maximum all receptors					
Chemical	**Concentration in crops ingested by chickens mg/kg ww**	**Soil Concentration - Agriculture (Cs) mg/kg**	**Transfer factor to eggs day/kg**	**Egg Concentration mg/kg ww**	
Antimony (Sb)	2.9E-05	6.3E-03	3.8E-02	7.1E-07	
Arsenic (As)	1.7E-03	3.6E-01	7.0E-02	7.4E-05	
Barium (Ba)	1.4E-03	3.1E-01	3.8E-02	3.5E-05	
Beryllium (Be)	2.5E-06	5.2E-04	9.0E-02	1.4E-07	
Cadmium (Cd)	3.2E-05	6.9E-03	1.0E-02	2.0E-07	
Chromium (Cr)	6.1E-04	1.3E-01	9.2E-03	3.5E-06	OEHHA (2003)
Copper (Cu)	6.7E-03	1.4E+00	3.8E-02	1.6E-04	
Lead (Pb)	1.0E-03	2.2E-01	4.0E-02	2.6E-05	
Manganese (Mn)	3.3E-02	7.0E+00	3.8E-02	7.9E-04	
Mercury (Hg)	4.4E-06	9.3E-04	8.0E-01	2.2E-06	
Silver (Ag)	1.6E-05	3.3E-03	3.8E-02	3.8E-07	
Nickel (Ni)	3.2E-04	6.8E-02	2.0E-02	4.0E-06	
Zinc (Zn)	9.7E-03	2.1E+00	3.8E-02	2.3E-04	

Transfer factors from OEHHA 2015 unless otherwise noted

Mean transfer factor for heavy metals used in absense of specific data (Leeman et al 2007)

Year 8

Chemical-specific Inputs and calculations - Maximum all receptors					
Chemical	**Concentration in crops ingested by chickens mg/kg ww**	**Soil Concentration - Agriculture (Cs) mg/kg**	**Transfer factor to eggs day/kg**	**Egg Concentration mg/kg ww**	
Antimony (Sb)	2.9E-05	6.2E-03	3.8E-02	7.0E-07	
Arsenic (As)	1.7E-03	3.6E-01	7.0E-02	7.4E-05	
Barium (Ba)	1.4E-03	3.1E-01	3.8E-02	3.4E-05	
Beryllium (Be)	2.4E-06	5.1E-04	9.0E-02	1.4E-07	
Cadmium (Cd)	3.2E-05	6.8E-03	1.0E-02	2.0E-07	
Chromium (Cr)	6.0E-04	1.3E-01	9.2E-03	3.5E-06	OEHHA (2003)
Copper (Cu)	6.9E-03	1.5E+00	3.8E-02	1.7E-04	
Lead (Pb)	1.0E-03	2.2E-01	4.0E-02	2.6E-05	
Manganese (Mn)	3.3E-02	7.0E+00	3.8E-02	7.9E-04	
Mercury (Hg)	4.3E-06	9.2E-04	8.0E-01	2.2E-06	
Silver (Ag)	1.6E-05	3.4E-03	3.8E-02	3.8E-07	
Nickel (Ni)	3.1E-04	6.7E-02	2.0E-02	4.0E-06	
Zinc (Zn)	9.5E-03	2.0E+00	3.8E-02	2.3E-04	

Transfer factors from OEHHA 2015 unless otherwise noted

Mean transfer factor for heavy metals used in absense of specific data (Leeman et al 2007)

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

Year 1

Chemical-specific Inputs and calculations - maximum all receptors

Chemical	Concentration in crops ingested by cattle mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to beef day/kg	Beef Concentration mg/kg ww	
Antimony (Sb)	1.3E-05	2.7E-03	1.0E-03	1.3E-06	RAIS
Arsenic (As)	6.7E-04	1.4E-01	2.0E-03	1.4E-04	
Barium (Ba)	6.2E-04	1.3E-01	1.5E-04	9.8E-06	RAIS
Beryllium (Be)	1.1E-06	2.5E-04	3.0E-04	3.6E-08	
Cadmium (Cd)	1.4E-05	3.0E-03	2.0E-04	3.0E-07	
Chromium (Cr)	2.9E-04	6.2E-02	9.2E-03	2.8E-04	OEHHA (2003)
Copper (Cu)	2.1E-03	4.5E-01	1.0E-02	2.2E-03	RAIS
Lead (Pb)	4.4E-04	9.5E-02	3.0E-04	1.4E-05	
Manganese (Mn)	1.4E-02	3.0E+00	4.0E-04	5.9E-04	RAIS
Mercury (Hg)	1.9E-06	4.1E-04	4.0E-04	8.1E-08	
Silver (Ag)	5.8E-06	1.2E-03	3.0E-03	1.8E-06	RAIS
Nickel (Ni)	1.5E-04	3.1E-02	3.0E-04	4.6E-06	
Zinc (Zn)	4.5E-03	9.5E-01	1.0E-01	4.7E-02	RAIS

Transfer factors from OEHHA 2015 unless otherwise noted

Year 2

Chemical-specific Inputs and calculations - maximum all receptors

Chemical	Concentration in crops ingested by cattle mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to beef day/kg	Beef Concentration mg/kg ww	
Antimony (Sb)	2.6E-05	5.5E-03	1.0E-03	2.7E-06	RAIS
Arsenic (As)	1.5E-03	3.1E-01	2.0E-03	3.1E-04	
Barium (Ba)	1.3E-03	2.7E-01	1.5E-04	2.0E-05	RAIS
Beryllium (Be)	2.2E-06	4.7E-04	3.0E-04	6.9E-08	
Cadmium (Cd)	2.9E-05	6.1E-03	2.0E-04	6.0E-07	
Chromium (Cr)	5.3E-04	1.1E-01	9.2E-03	5.1E-04	OEHHA (2003)
Copper (Cu)	5.6E-03	1.2E+00	1.0E-02	5.9E-03	RAIS
Lead (Pb)	8.8E-04	1.9E-01	3.0E-04	2.8E-05	
Manganese (Mn)	2.9E-02	6.2E+00	4.0E-04	1.2E-03	RAIS
Mercury (Hg)	3.9E-06	8.2E-04	4.0E-04	1.6E-07	
Silver (Ag)	1.4E-05	2.9E-03	3.0E-03	4.3E-06	RAIS
Nickel (Ni)	2.8E-04	5.9E-02	3.0E-04	8.7E-06	
Zinc (Zn)	8.6E-03	1.8E+00	1.0E-01	9.0E-02	RAIS

Transfer factors from OEHHA 2015 unless otherwise noted

Year 4

Chemical-specific Inputs and calculations - maximum all receptors					
Chemical	**Concentration in crops ingested by cattle mg/kg ww**	**Soil Concentration - Agriculture (Cs) mg/kg**	**Transfer factor to beef day/kg**	**Beef Concentration mg/kg ww**	
Antimony (Sb)	3.1E-05	6.5E-03	1.0E-03	3.2E-06	RAIS
Arsenic (As)	1.7E-03	3.7E-01	2.0E-03	3.7E-04	
Barium (Ba)	1.5E-03	3.2E-01	1.5E-04	2.4E-05	RAIS
Beryllium (Be)	2.6E-06	5.5E-04	3.0E-04	8.1E-08	
Cadmium (Cd)	3.4E-05	7.2E-03	2.0E-04	7.1E-07	
Chromium (Cr)	6.3E-04	1.4E-01	9.2E-03	6.1E-04	OEHHA (2003)
Copper (Cu)	6.9E-03	1.5E+00	1.0E-02	7.3E-03	RAIS
Lead (Pb)	1.1E-03	2.3E-01	3.0E-04	3.3E-05	
Manganese (Mn)	3.4E-02	7.3E+00	4.0E-04	1.4E-03	RAIS
Mercury (Hg)	4.6E-06	9.7E-04	4.0E-04	1.9E-07	
Silver (Ag)	1.6E-05	3.5E-03	3.0E-03	5.1E-06	RAIS
Nickel (Ni)	3.3E-04	7.1E-02	3.0E-04	1.0E-05	
Zinc (Zn)	1.0E-02	2.2E+00	1.0E-01	1.1E-01	RAIS

Transfer factors from OEHHA 2015 unless otherwise noted

Year 6

Chemical-specific Inputs and calculations - maximum all receptors					
Chemical	**Concentration in crops ingested by cattle mg/kg ww**	**Soil Concentration - Agriculture (Cs) mg/kg**	**Transfer factor to beef day/kg**	**Beef Concentration mg/kg ww**	
Antimony (Sb)	2.9E-05	6.3E-03	1.0E-03	3.1E-06	RAIS
Arsenic (As)	1.7E-03	3.6E-01	2.0E-03	3.5E-04	
Barium (Ba)	1.4E-03	3.1E-01	1.5E-04	2.3E-05	RAIS
Beryllium (Be)	2.5E-06	5.2E-04	3.0E-04	7.7E-08	
Cadmium (Cd)	3.2E-05	6.9E-03	2.0E-04	6.8E-07	
Chromium (Cr)	6.1E-04	1.3E-01	9.2E-03	5.9E-04	OEHHA (2003)
Copper (Cu)	6.7E-03	1.4E+00	1.0E-02	7.0E-03	RAIS
Lead (Pb)	1.0E-03	2.2E-01	3.0E-04	3.2E-05	
Manganese (Mn)	3.3E-02	7.0E+00	4.0E-04	1.4E-03	RAIS
Mercury (Hg)	4.4E-06	9.3E-04	4.0E-04	1.8E-07	
Silver (Ag)	1.6E-05	3.3E-03	3.0E-03	4.9E-06	RAIS
Nickel (Ni)	3.2E-04	6.8E-02	3.0E-04	1.0E-05	
Zinc (Zn)	9.7E-03	2.1E+00	1.0E-01	1.0E-01	RAIS

Transfer factors from OEHHA 2015 unless otherwise noted

Year 8

Chemical-specific Inputs and calculations - maximum all receptors					
Chemical	**Concentration in crops ingested by cattle mg/kg ww**	**Soil Concentration - Agriculture (Cs) mg/kg**	**Transfer factor to beef day/kg**	**Beef Concentration mg/kg ww**	
Antimony (Sb)	2.9E-05	6.2E-03	1.0E-03	3.1E-06	RAIS
Arsenic (As)	1.7E-03	3.6E-01	2.0E-03	3.5E-04	
Barium (Ba)	1.4E-03	3.1E-01	1.5E-04	2.3E-05	RAIS
Beryllium (Be)	2.4E-06	5.1E-04	3.0E-04	7.6E-08	
Cadmium (Cd)	3.2E-05	6.8E-03	2.0E-04	6.7E-07	
Chromium (Cr)	6.0E-04	1.3E-01	9.2E-03	5.8E-04	OEHHA (2003)
Copper (Cu)	6.9E-03	1.5E+00	1.0E-02	7.2E-03	RAIS
Lead (Pb)	1.0E-03	2.2E-01	3.0E-04	3.2E-05	
Manganese (Mn)	3.3E-02	7.0E+00	4.0E-04	1.4E-03	RAIS
Mercury (Hg)	4.3E-06	9.2E-04	4.0E-04	1.8E-07	
Silver (Ag)	1.6E-05	3.4E-03	3.0E-03	5.0E-06	RAIS
Nickel (Ni)	3.1E-04	6.7E-02	3.0E-04	9.9E-06	
Zinc (Zn)	9.5E-03	2.0E+00	1.0E-01	1.0E-01	RAIS

Transfer factors from OEHHA 2015 unless otherwise noted

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

Year 1

Chemical-specific Inputs and calculations - Maximum all receptors

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	
Antimony (Sb)	1.3E-05	2.7E-03	1.0E-04	3.2E-07	RAIS
Arsenic (As)	6.7E-04	1.4E-01	5.0E-05	8.7E-06	
Barium (Ba)	6.2E-04	1.3E-01	3.5E-04	5.6E-05	RAIS
Beryllium (Be)	1.1E-06	2.5E-04	9.0E-07	2.7E-10	
Cadmium (Cd)	1.4E-05	3.0E-03	5.0E-06	1.8E-08	
Chromium (Cr)	2.9E-04	6.2E-02	9.0E-06	6.7E-07	
Copper (Cu)	2.1E-03	4.5E-01	1.5E-03	8.1E-04	RAIS
Lead (Pb)	4.4E-04	9.5E-02	6.0E-05	6.9E-06	
Manganese (Mn)	1.4E-02	3.0E+00	3.5E-04	1.3E-03	RAIS
Mercury (Hg)	1.9E-06	4.1E-04	7.0E-05	3.5E-08	
Silver (Ag)	5.8E-06	1.2E-03	2.7E-02	4.0E-05	Median transfer factor for metals (Leeman et al 2007)
Nickel (Ni)	1.5E-04	3.1E-02	3.0E-05	1.1E-06	
Zinc (Zn)	4.5E-03	9.5E-01	2.7E-09	3.1E-09	RAIS

Transfer factors from OEHHA 2015 unless otherwise noted

Year 2

Chemical-specific Inputs and calculations - Maximum all receptors

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	
Antimony (Sb)	2.6E-05	5.5E-03	1.0E-04	6.6E-07	RAIS
Arsenic (As)	1.5E-03	3.1E-01	5.0E-05	1.9E-05	
Barium (Ba)	1.3E-03	2.7E-01	3.5E-04	1.1E-04	RAIS
Beryllium (Be)	2.2E-06	4.7E-04	9.0E-07	5.1E-10	
Cadmium (Cd)	2.9E-05	6.1E-03	5.0E-06	3.7E-08	
Chromium (Cr)	5.3E-04	1.1E-01	9.0E-06	1.2E-06	
Copper (Cu)	5.6E-03	1.2E+00	1.5E-03	2.2E-03	RAIS
Lead (Pb)	8.8E-04	1.9E-01	6.0E-05	1.4E-05	
Manganese (Mn)	2.9E-02	6.2E+00	3.5E-04	2.6E-03	RAIS
Mercury (Hg)	3.9E-06	8.2E-04	7.0E-05	6.9E-08	
Silver (Ag)	1.4E-05	2.9E-03	2.7E-02	9.4E-05	Median transfer factor for metals (Leeman et al 2007)
Nickel (Ni)	2.8E-04	5.9E-02	3.0E-05	2.1E-06	
Zinc (Zn)	8.6E-03	1.8E+00	2.7E-09	6.0E-09	RAIS

Transfer factors from OEHHA 2015 unless otherwise noted

Year 4

Chemical-specific Inputs and calculations - Maximum all receptors					
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**	
Antimony (Sb)	3.1E-05	6.5E-03	1.0E-04	7.9E-07	RAIS
Arsenic (As)	1.7E-03	3.7E-01	5.0E-05	2.2E-05	
Barium (Ba)	1.5E-03	3.2E-01	3.5E-04	1.4E-04	RAIS
Beryllium (Be)	2.6E-06	5.5E-04	9.0E-07	5.9E-10	
Cadmium (Cd)	3.4E-05	7.2E-03	5.0E-06	4.3E-08	
Chromium (Cr)	6.3E-04	1.4E-01	9.0E-06	1.5E-06	
Copper (Cu)	6.9E-03	1.5E+00	1.5E-03	2.7E-03	RAIS
Lead (Pb)	1.1E-03	2.3E-01	6.0E-05	1.6E-05	
Manganese (Mn)	3.4E-02	7.3E+00	3.5E-04	3.1E-03	RAIS
Mercury (Hg)	4.6E-06	9.7E-04	7.0E-05	8.2E-08	
Silver (Ag)	1.6E-05	3.5E-03	2.7E-02	1.1E-04	Median transfer factor for metals (Leeman et al 2007)
Nickel (Ni)	3.3E-04	7.1E-02	3.0E-05	2.6E-06	
Zinc (Zn)	1.0E-02	2.2E+00	2.7E-09	7.0E-09	RAIS

Transfer factors from OEHHA 2015 unless otherwise noted

Year 6

Chemical-specific Inputs and calculations - Maximum all receptors					
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**	
Antimony (Sb)	2.9E-05	6.3E-03	1.0E-04	7.6E-07	RAIS
Arsenic (As)	1.7E-03	3.6E-01	5.0E-05	2.1E-05	
Barium (Ba)	1.4E-03	3.1E-01	3.5E-04	1.3E-04	RAIS
Beryllium (Be)	2.5E-06	5.2E-04	9.0E-07	5.7E-10	
Cadmium (Cd)	3.2E-05	6.9E-03	5.0E-06	4.1E-08	
Chromium (Cr)	6.1E-04	1.3E-01	9.0E-06	1.4E-06	
Copper (Cu)	6.7E-03	1.4E+00	1.5E-03	2.6E-03	RAIS
Lead (Pb)	1.0E-03	2.2E-01	6.0E-05	1.6E-05	
Manganese (Mn)	3.3E-02	7.0E+00	3.5E-04	3.0E-03	RAIS
Mercury (Hg)	4.4E-06	9.3E-04	7.0E-05	7.9E-08	
Silver (Ag)	1.6E-05	3.3E-03	2.7E-02	1.1E-04	Median transfer factor for metals (Leeman et al 2007)
Nickel (Ni)	3.2E-04	6.8E-02	3.0E-05	2.4E-06	
Zinc (Zn)	9.7E-03	2.1E+00	2.7E-09	6.7E-09	RAIS

Transfer factors from OEHHA 2015 unless otherwise noted

Year 8

Chemical-specific Inputs and calculations - Maximum all receptors					
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**	
Antimony (Sb)	2.9E-05	6.2E-03	1.0E-04	7.5E-07	RAIS
Arsenic (As)	1.7E-03	3.6E-01	5.0E-05	2.1E-05	
Barium (Ba)	1.4E-03	3.1E-01	3.5E-04	1.3E-04	RAIS
Beryllium (Be)	2.4E-06	5.1E-04	9.0E-07	5.5E-10	
Cadmium (Cd)	3.2E-05	6.8E-03	5.0E-06	4.1E-08	
Chromium (Cr)	6.0E-04	1.3E-01	9.0E-06	1.4E-06	
Copper (Cu)	6.9E-03	1.5E+00	1.5E-03	2.7E-03	RAIS
Lead (Pb)	1.0E-03	2.2E-01	6.0E-05	1.6E-05	
Manganese (Mn)	3.3E-02	7.0E+00	3.5E-04	2.9E-03	RAIS
Mercury (Hg)	4.3E-06	9.2E-04	7.0E-05	7.8E-08	
Silver (Ag)	1.6E-05	3.4E-03	2.7E-02	1.1E-04	Median transfer factor for metals (Leeman et al 2007)
Nickel (Ni)	3.1E-04	6.7E-02	3.0E-05	2.4E-06	
Zinc (Zn)	9.5E-03	2.0E+00	2.7E-09	6.6E-09	RAIS

Transfer factors from OEHHA 2015 unless otherwise noted

Adults

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

Year 1

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)	
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	4.0E-02	1.2E-08	2.9E-08	--		4.0E-05	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	2.2E+00	6.4E-07	1.5E-06	--		5.1E-03	60%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	2.0E+00	5.9E-07	1.4E-06	--		7.9E-06	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	3.7E-03	1.1E-09	2.6E-09	--		1.3E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	4.5E-02	1.3E-08	3.2E-08	--		1.0E-04	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	9.2E-01	2.7E-07	6.6E-07	--		7.3E-04	8%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	6.7E+00	2.0E-06	4.8E-06	--		8.6E-05	1%
Lead (Pb)		6.0E-04	10%	5.4E-04	100%	1.4E+00	4.2E-07	1.0E-06	--		1.9E-03	22%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	4.5E+01	1.3E-05	3.2E-05	--		4.6E-04	5%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	6.2E-03	1.8E-09	4.4E-09	--		1.2E-05	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.9E-02	5.5E-09	1.3E-08	--		2.3E-06	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.7E-01	1.4E-07	3.4E-07	--		7.0E-05	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	1.4E+01	4.2E-06	1.0E-05	--		1.0E-04	1%
TOTAL											**8.6E-03**	

Year 2

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	8.3E-02	2.4E-08	5.9E-08	--		8.2E-05	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	4.7E+00	1.4E-06	3.3E-06	--		1.1E-02	62%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	4.0E+00	1.2E-06	2.9E-06	--		1.6E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	7.0E-03	2.1E-09	5.0E-09	--		2.5E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	9.1E-02	2.7E-08	6.5E-08	--		2.0E-04	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.7E+00	5.0E-07	1.2E-06	--		1.3E-03	7%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.8E+01	5.3E-06	1.3E-05	--		2.3E-04	1%
Lead (Pb)		6.0E-04	10%	5.4E-04	100%	2.8E+00	8.4E-07	2.0E-06	--		3.7E-03	21%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	9.3E+01	2.7E-05	6.6E-05	--		9.5E-04	5%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.2E-02	3.7E-09	8.8E-09	--		2.5E-05	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	4.3E-02	1.3E-08	3.1E-08	--		5.4E-06	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	8.8E-01	2.6E-07	6.3E-07	--		1.3E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	2.8E+01	8.1E-06	2.0E-05	--		2.0E-04	1%
								TOTAL			**1.8E-02**	

Year 4

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	9.8E-02	2.9E-08	7.0E-08	--		9.7E-05	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	5.6E+00	1.6E-06	4.0E-06	--		1.3E-02	62%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	4.8E+00	1.4E-06	3.4E-06	--		1.9E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	8.2E-03	2.4E-09	5.8E-09	--		2.9E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.1E-01	3.2E-08	7.7E-08	--		2.4E-04	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	2.0E+00	6.0E-07	1.5E-06	--		1.6E-03	7%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.2E+01	6.6E-06	1.6E-05	--		2.8E-04	1%
Lead (Pb)		6.0E-04	10%	5.4E-04	100%	3.4E+00	1.0E-06	2.4E-06	--		4.5E-03	21%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.1E+02	3.2E-05	7.8E-05	--		1.1E-03	5%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.5E-02	4.3E-09	1.0E-08	--		2.9E-05	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	5.2E-02	1.5E-08	3.7E-08	--		6.5E-06	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.1E+00	3.1E-07	7.6E-07	--		1.6E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	3.2E+01	9.6E-06	2.3E-05	--		2.3E-04	1%
								TOTAL			**2.2E-02**	

Year 6

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	9.4E-02	2.8E-08	6.7E-08	--		9.3E-05	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	5.3E+00	1.6E-06	3.8E-06	--		1.3E-02	59%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	4.6E+00	1.4E-06	3.3E-06	--		1.8E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	7.9E-03	2.3E-09	5.6E-09	--		2.8E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.0E-01	3.1E-08	7.4E-08	--		2.3E-04	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.9E+00	5.8E-07	1.4E-06	--		1.5E-03	7%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.1E+01	6.3E-06	1.5E-05	--		2.7E-04	1%
Lead (Pb)		6.0E-04	10%	5.4E-04	100%	3.2E+00	9.6E-07	2.3E-06	--		4.3E-03	20%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.1E+02	3.1E-05	7.5E-05	--		1.1E-03	5%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.4E-02	4.1E-09	1.0E-08	--		2.8E-05	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	5.0E-02	1.5E-08	3.6E-08	--		6.2E-06	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.0E+00	3.0E-07	7.3E-07	--		1.5E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	3.1E+01	9.2E-06	2.2E-05	--		2.2E-04	1%
								TOTAL			**2.1E-02**	

Year 8

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	9.4E-02	2.8E-08	6.7E-08	--		9.3E-05	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	5.4E+00	1.6E-06	3.8E-06	--		1.3E-02	62%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	4.6E+00	1.4E-06	3.3E-06	--		1.8E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	7.7E-03	2.3E-09	5.5E-09	--		2.7E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.0E-01	3.0E-08	7.3E-08	--		2.3E-04	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.9E+00	5.7E-07	1.4E-06	--		1.5E-03	7%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.2E+01	6.5E-06	1.6E-05	--		2.8E-04	1%
Lead (Pb)		6.0E-04	10%	5.4E-04	100%	3.2E+00	9.6E-07	2.3E-06	--		4.3E-03	21%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.0E+02	3.1E-05	7.5E-05	--		1.1E-03	5%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.4E-02	4.1E-09	9.9E-09	--		2.7E-05	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	5.1E-02	1.5E-08	3.6E-08	--		6.3E-06	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.0E+00	3.0E-07	7.2E-07	--		1.5E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	3.0E+01	9.0E-06	2.2E-05	--		2.2E-04	1%
								TOTAL			**2.1E-02**	

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

Year 1

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		1.4E-04	20%	1.1E-04		4.0E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	2.2E+00	2.0E-07	4.9E-07	--		4.9E-04	92%
Barium (Ba)		1.4E-02	10%	1.3E-02		2.0E+00			--		--	
Beryllium (Be)		1.4E-05		1.4E-05		3.7E-03			--		--	
Cadmium (Cd)		8.0E-04	60%	3.2E-04		4.5E-02			--		--	
Chromium (Cr)		1.0E-03	10%	9.0E-04		9.2E-01			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		6.7E+00			--		--	
Lead (Pb)		3.0E-04	10%	2.7E-04		1.4E+00			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		4.5E+01			--		--	
Mercury (Hg)		4.2E-05	40%	2.5E-05	0.001	6.2E-03	1.2E-10	2.8E-10	--		1.1E-05	2%
Silver (Ag)		2.3E-04		2.3E-04		1.9E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03	0.005	4.7E-01	4.4E-08	1.1E-07	--		2.2E-05	4%
Zinc (Zn)		5.0E-01	80%	1.0E-01	0.001	1.4E+01	2.7E-07	6.4E-07	--		6.4E-06	1%
TOTAL											**5.3E-04**	

Year 2

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)	Dermal Absorption (ABS)	(mg/kg)	Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		1.4E-04	20%	1.1E-04		8.3E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	4.7E+00	4.3E-07	1.0E-06	--		1.0E-03	93%
Barium (Ba)		1.4E-02	10%	1.3E-02		4.0E+00			--		--	
Beryllium (Be)		1.4E-05		1.4E-05		7.0E-03			--		--	
Cadmium (Cd)		8.0E-04	60%	3.2E-04		9.1E-02			--		--	
Chromium (Cr)		1.0E-03	10%	9.0E-04		1.7E+00			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		1.8E+01			--		--	
Lead (Pb)		3.0E-04	10%	2.7E-04		2.8E+00			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		9.3E+01			--		--	
Mercury (Hg)		4.2E-05	40%	2.5E-05	0.001	1.2E-02	2.3E-10	5.6E-10	--		2.2E-05	2%
Silver (Ag)		2.3E-04		2.3E-04		4.3E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03	0.005	8.8E-01	8.2E-08	2.0E-07	--		4.1E-05	4%
Zinc (Zn)		5.0E-01	80%	1.0E-01	0.001	2.8E+01	5.1E-07	1.2E-06	--		1.2E-05	1%
								TOTAL			**1.1E-03**	

Year 4

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)	Dermal Absorption (ABS)	(mg/kg)	Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		1.4E-04	20%	1.1E-04		9.8E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	5.6E+00	5.2E-07	1.3E-06	--		1.3E-03	93%
Barium (Ba)		1.4E-02	10%	1.3E-02		4.8E+00			--		--	
Beryllium (Be)		1.4E-05		1.4E-05		8.2E-03			--		--	
Cadmium (Cd)		8.0E-04	60%	3.2E-04		1.1E-01			--		--	
Chromium (Cr)		1.0E-03	10%	9.0E-04		2.0E+00			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		2.2E+01			--		--	
Lead (Pb)		3.0E-04	10%	2.7E-04		3.4E+00			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		1.1E+02			--		--	
Mercury (Hg)		4.2E-05	40%	2.5E-05	0.001	1.5E-02	2.7E-10	6.6E-10	--		2.6E-05	2%
Silver (Ag)		2.3E-04		2.3E-04		5.2E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03	0.005	1.1E+00	9.9E-08	2.4E-07	--		5.0E-05	4%
Zinc (Zn)		5.0E-01	80%	1.0E-01	0.001	3.2E+01	6.0E-07	1.5E-06	--		1.5E-05	1%
								TOTAL			**1.3E-03**	

Year 6

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		1.4E-04	20%	1.1E-04		9.4E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	5.3E+00	5.0E-07	1.2E-06	--		1.2E-03	93%
Barium (Ba)		1.4E-02	10%	1.3E-02		4.6E+00			--		--	
Beryllium (Be)		1.4E-05		1.4E-05		7.9E-03			--		--	
Cadmium (Cd)		8.0E-04	60%	3.2E-04		1.0E-01			--		--	
Chromium (Cr)		1.0E-03	10%	9.0E-04		1.9E+00			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		2.1E+01			--		--	
Lead (Pb)		3.0E-04	10%	2.7E-04		3.2E+00			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		1.1E+02			--		--	
Mercury (Hg)		4.2E-05	40%	2.5E-05	0.001	1.4E-02	2.6E-10	6.3E-10	--		2.5E-05	2%
Silver (Ag)		2.3E-04		2.3E-04		5.0E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03	0.005	1.0E+00	9.5E-08	2.3E-07	--		4.8E-05	4%
Zinc (Zn)		5.0E-01	80%	1.0E-01	0.001	3.1E+01	5.8E-07	1.4E-06	--		1.4E-05	1%
								TOTAL			**1.3E-03**	

Year 8

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		1.4E-04	20%	1.1E-04		9.4E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	5.4E+00	5.0E-07	1.2E-06	--		1.2E-03	93%
Barium (Ba)		1.4E-02	10%	1.3E-02		4.6E+00			--		--	
Beryllium (Be)		1.4E-05		1.4E-05		7.7E-03			--		--	
Cadmium (Cd)		8.0E-04	60%	3.2E-04		1.0E-01			--		--	
Chromium (Cr)		1.0E-03	10%	9.0E-04		1.9E+00			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		2.2E+01			--		--	
Lead (Pb)		3.0E-04	10%	2.7E-04		3.2E+00			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		1.0E+02			--		--	
Mercury (Hg)		4.2E-05	40%	2.5E-05	0.001	1.4E-02	2.6E-10	6.2E-10	--		2.5E-05	2%
Silver (Ag)		2.3E-04		2.3E-04		5.1E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03	0.005	1.0E+00	9.4E-08	2.3E-07	--		4.7E-05	4%
Zinc (Zn)		5.0E-01	80%	1.0E-01	0.001	3.0E+01	5.7E-07	1.4E-06	--		1.4E-05	1%
								TOTAL			**1.3E-03**	

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

Year 1

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)	
Antimony (Sb)	0.0E+00	9.0E-04	20%	7.2E-04	100%	8.8E-07	1.1E-08	2.5E-08	--		3.5E-05	0%
Arsenic (As)	0.0E+00	6.0E-04	50%	3.0E-04	100%	7.3E-05	9.0E-07	2.1E-06	--		7.0E-03	76%
Barium (Ba)	0.0E+00	2.0E-01	10%	1.8E-01	100%	4.8E-05	5.8E-07	1.4E-06	--		7.6E-06	0%
Beryllium (Be)	0.0E+00	2.0E-03	0%	2.0E-03	100%	4.6E-09	5.6E-11	1.3E-10	--		6.6E-08	0%
Cadmium (Cd)	0.0E+00	8.0E-04	60%	3.2E-04	100%	6.0E-07	7.3E-09	1.7E-08	--		5.3E-05	1%
Chromium (Cr)	0.0E+00	1.0E-03	10%	9.0E-04	100%	4.8E-05	5.9E-07	1.4E-06	--		1.5E-03	17%
Copper (Cu)	0.0E+00	1.4E-01	60%	5.6E-02	100%	1.9E-04	2.3E-06	5.4E-06	--		9.7E-05	1%
Lead (Pb)	0.0E+00	6.0E-04	10%	5.4E-04	50%	1.6E-06	1.9E-08	4.5E-08	--		8.2E-05	1%
Manganese (Mn)	0.0E+00	1.4E-01	50%	7.0E-02	100%	6.8E-04	8.3E-06	1.9E-05	--		2.8E-04	3%
Mercury (Hg)	0.0E+00	6.0E-04	40%	3.6E-04	100%	1.2E-07	1.4E-09	3.3E-09	--		9.3E-06	0%
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	2.2E-06	2.7E-08	6.3E-08	--		1.1E-05	0%
Nickel (Ni)	0.0E+00	1.2E-02	60%	4.8E-03	100%	7.1E-06	8.7E-08	2.0E-07	--		4.2E-05	0%
Zinc (Zn)	0.0E+00	5.0E-01	80%	1.0E-01	100%	2.3E-04	2.8E-06	6.5E-06	--		6.5E-05	1%
TOTAL									**0.00E+00**		**9.2E-03**	

Year 2

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)	
Antimony (Sb)	0.0E+00	9.0E-04	20%	7.2E-04	100%	1.8E-06	2.2E-08	5.2E-08	--		7.2E-05	0%
Arsenic (As)	0.0E+00	6.0E-04	50%	3.0E-04	100%	1.6E-04	1.9E-06	4.5E-06	--		1.5E-02	78%
Barium (Ba)	0.0E+00	2.0E-01	10%	1.8E-01	100%	9.7E-05	1.2E-06	2.8E-06	--		1.5E-05	0%
Beryllium (Be)	0.0E+00	2.0E-03	0%	2.0E-03	100%	8.7E-09	1.1E-10	2.5E-10	--		1.2E-07	0%
Cadmium (Cd)	0.0E+00	8.0E-04	60%	3.2E-04	100%	1.2E-06	1.5E-08	3.4E-08	--		1.1E-04	1%
Chromium (Cr)	0.0E+00	1.0E-03	10%	9.0E-04	100%	8.8E-05	1.1E-06	2.5E-06	--		2.8E-03	14%
Copper (Cu)	0.0E+00	1.4E-01	60%	5.6E-02	100%	5.1E-04	6.2E-06	1.5E-05	--		2.6E-04	1%
Lead (Pb)	0.0E+00	6.0E-04	10%	5.4E-04	50%	3.1E-06	3.8E-08	8.8E-08	--		1.6E-04	1%
Manganese (Mn)	0.0E+00	1.4E-01	50%	7.0E-02	100%	1.4E-03	1.7E-05	4.0E-05	--		5.7E-04	3%
Mercury (Hg)	0.0E+00	6.0E-04	40%	3.6E-04	100%	2.3E-07	2.9E-09	6.7E-09	--		1.9E-05	0%
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	5.1E-06	6.3E-08	1.5E-07	--		2.6E-05	0%
Nickel (Ni)	0.0E+00	1.2E-02	60%	4.8E-03	100%	1.3E-05	1.6E-07	3.8E-07	--		8.0E-05	0%
Zinc (Zn)	0.0E+00	5.0E-01	80%	1.0E-01	100%	4.4E-04	5.3E-06	1.2E-05	--		1.2E-04	1%
								TOTAL	**0.00E+00**		**1.9E-02**	

Year 4

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)	
Antimony (Sb)	0.0E+00	9.0E-04	20%	7.2E-04	100%	2.1E-06	2.6E-08	6.1E-08	--		8.5E-05	0%
Arsenic (As)	0.0E+00	6.0E-04	50%	3.0E-04	100%	1.9E-04	2.3E-06	5.4E-06	--		1.8E-02	78%
Barium (Ba)	0.0E+00	2.0E-01	10%	1.8E-01	100%	1.2E-04	1.4E-06	3.3E-06	--		1.8E-05	0%
Beryllium (Be)	0.0E+00	2.0E-03	0%	2.0E-03	100%	1.0E-08	1.2E-10	2.9E-10	--		1.5E-07	0%
Cadmium (Cd)	0.0E+00	8.0E-04	60%	3.2E-04	100%	1.4E-06	1.7E-08	4.0E-08	--		1.3E-04	1%
Chromium (Cr)	0.0E+00	1.0E-03	10%	9.0E-04	100%	1.1E-04	1.3E-06	3.0E-06	--		3.3E-03	14%
Copper (Cu)	0.0E+00	1.4E-01	60%	5.6E-02	100%	6.3E-04	7.7E-06	1.8E-05	--		3.2E-04	1%
Lead (Pb)	0.0E+00	6.0E-04	10%	5.4E-04	50%	3.7E-06	4.5E-08	1.1E-07	--		2.0E-04	1%
Manganese (Mn)	0.0E+00	1.4E-01	50%	7.0E-02	100%	1.7E-03	2.0E-05	4.8E-05	--		6.8E-04	3%
Mercury (Hg)	0.0E+00	6.0E-04	40%	3.6E-04	100%	2.8E-07	3.4E-09	7.9E-09	--		2.2E-05	0%
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	6.2E-06	7.6E-08	1.8E-07	--		3.1E-05	0%
Nickel (Ni)	0.0E+00	1.2E-02	60%	4.8E-03	100%	1.6E-05	2.0E-07	4.6E-07	--		9.6E-05	0%
Zinc (Zn)	0.0E+00	5.0E-01	80%	1.0E-01	100%	5.1E-04	6.3E-06	1.5E-05	--		1.5E-04	1%
								TOTAL	**0.00E+00**		**2.3E-02**	

Year 6

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)	
Antimony (Sb)	0.0E+00	9.0E-04	20%	7.2E-04	100%	2.1E-06	2.5E-08	5.9E-08	--		8.2E-05	0%
Arsenic (As)	0.0E+00	6.0E-04	50%	3.0E-04	100%	1.8E-04	2.2E-06	5.2E-06	--		1.7E-02	75%
Barium (Ba)	0.0E+00	2.0E-01	10%	1.8E-01	100%	1.1E-04	1.4E-06	3.2E-06	--		1.8E-05	0%
Beryllium (Be)	0.0E+00	2.0E-03	0%	2.0E-03	100%	9.8E-09	1.2E-10	2.8E-10	--		1.4E-07	0%
Cadmium (Cd)	0.0E+00	8.0E-04	60%	3.2E-04	100%	1.4E-06	1.7E-08	3.9E-08	--		1.2E-04	1%
Chromium (Cr)	0.0E+00	1.0E-03	10%	9.0E-04	100%	1.0E-04	1.2E-06	2.9E-06	--		3.2E-03	14%
Copper (Cu)	0.0E+00	1.4E-01	60%	5.6E-02	100%	6.0E-04	7.3E-06	1.7E-05	--		3.1E-04	1%
Lead (Pb)	0.0E+00	6.0E-04	10%	5.4E-04	50%	3.6E-06	4.4E-08	1.0E-07	--		1.9E-04	1%
Manganese (Mn)	0.0E+00	1.4E-01	50%	7.0E-02	100%	1.6E-03	2.0E-05	4.6E-05	--		6.5E-04	3%
Mercury (Hg)	0.0E+00	6.0E-04	40%	3.6E-04	100%	2.7E-07	3.2E-09	7.6E-09	--		2.1E-05	0%
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	5.9E-06	7.3E-08	1.7E-07	--		3.0E-05	0%
Nickel (Ni)	0.0E+00	1.2E-02	60%	4.8E-03	100%	1.5E-05	1.9E-07	4.4E-07	--		9.2E-05	0%
Zinc (Zn)	0.0E+00	5.0E-01	80%	1.0E-01	100%	4.9E-04	6.0E-06	1.4E-05	--		1.4E-04	1%
								TOTAL	**0.00E+00**		**2.2E-02**	

Year 8

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)	
Antimony (Sb)	0.0E+00	9.0E-04	20%	7.2E-04	100%	2.1E-06	2.5E-08	5.9E-08	--		8.1E-05	0%
Arsenic (As)	0.0E+00	6.0E-04	50%	3.0E-04	100%	1.8E-04	2.2E-06	5.2E-06	--		1.7E-02	78%
Barium (Ba)	0.0E+00	2.0E-01	10%	1.8E-01	100%	1.1E-04	1.4E-06	3.2E-06	--		1.8E-05	0%
Beryllium (Be)	0.0E+00	2.0E-03	0%	2.0E-03	100%	9.6E-09	1.2E-10	2.7E-10	--		1.4E-07	0%
Cadmium (Cd)	0.0E+00	8.0E-04	60%	3.2E-04	100%	1.3E-06	1.6E-08	3.8E-08	--		1.2E-04	1%
Chromium (Cr)	0.0E+00	1.0E-03	10%	9.0E-04	100%	9.9E-05	1.2E-06	2.8E-06	--		3.2E-03	14%
Copper (Cu)	0.0E+00	1.4E-01	60%	5.6E-02	100%	6.2E-04	7.6E-06	1.8E-05	--		3.2E-04	1%
Lead (Pb)	0.0E+00	6.0E-04	10%	5.4E-04	50%	3.5E-06	4.3E-08	1.0E-07	--		1.9E-04	1%
Manganese (Mn)	0.0E+00	1.4E-01	50%	7.0E-02	100%	1.6E-03	1.9E-05	4.5E-05	--		6.5E-04	3%
Mercury (Hg)	0.0E+00	6.0E-04	40%	3.6E-04	100%	2.6E-07	3.2E-09	7.5E-09	--		2.1E-05	0%
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	6.0E-06	7.4E-08	1.7E-07	--		3.0E-05	0%
Nickel (Ni)	0.0E+00	1.2E-02	60%	4.8E-03	100%	1.5E-05	1.9E-07	4.4E-07	--		9.1E-05	0%
Zinc (Zn)	0.0E+00	5.0E-01	80%	1.0E-01	100%	4.8E-04	5.9E-06	1.4E-05	--		1.4E-04	1%
								TOTAL	**0.00E+00**		**2.2E-02**	

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

Year 1

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)	
Antimony (Sb)		1.4E-04	20%	1.1E-04	1.00E-3	8.8E-07	5.11E-13	6.3E-11	1.5E-10	--		1.4E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	7.3E-05	4.25E-11	5.2E-09	1.2E-08	--		1.2E-05	33%
Barium (Ba)		1.4E-02	10%	1.3E-02	1.00E-3	4.8E-05	2.76E-11	3.4E-09	7.9E-09	--		6.3E-07	2%
Beryllium (Be)		1.4E-05		1.4E-05	1.00E-3	4.6E-09	2.66E-15	3.3E-13	7.6E-13	--		5.4E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	6.0E-07	3.46E-13	4.2E-11	9.9E-11	--		3.1E-07	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	2.00E-3	4.8E-05	5.55E-11	6.8E-09	1.6E-08	--		1.8E-05	48%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	1.9E-04	1.10E-10	1.3E-08	3.1E-08	--		5.6E-07	2%
Lead (Pb)		3.0E-04	10%	2.7E-04	1.00E-4	1.6E-06	9.03E-14	1.1E-11	2.6E-11	--		9.6E-08	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	6.8E-04	3.92E-10	4.8E-08	1.1E-07	--		1.6E-06	4%
Mercury (Hg)		4.2E-05	40%	2.5E-05	1.00E-3	1.2E-07	6.79E-14	8.3E-12	1.9E-11	--		7.7E-07	2%
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	2.2E-06	7.69E-13	9.4E-11	2.2E-10	--		9.6E-07	3%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	7.1E-06	8.27E-13	1.0E-10	2.4E-10	--		4.9E-08	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	6.00E-4	2.3E-04	7.88E-11	9.7E-09	2.3E-08	--		2.3E-07	1%
												3.6E-05	

Year 2

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)	
Antimony (Sb)		1.4E-04	20%	1.1E-04	1.00E-3	1.8E-06	1.05E-12	1.3E-10	3.0E-10	--		2.8E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	1.6E-04	9.17E-11	1.1E-08	2.6E-08	--		2.6E-05	36%
Barium (Ba)		1.4E-02	10%	1.3E-02	1.00E-3	9.7E-05	5.63E-11	6.9E-09	1.6E-08	--		1.3E-06	2%
Beryllium (Be)		1.4E-05		1.4E-05	1.00E-3	8.7E-09	5.06E-15	6.2E-13	1.4E-12	--		1.0E-07	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	1.2E-06	6.95E-13	8.5E-11	2.0E-10	--		6.2E-07	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	2.00E-3	8.8E-05	1.02E-10	1.3E-08	2.9E-08	--		3.2E-05	45%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	5.1E-04	2.95E-10	3.6E-08	8.4E-08	--		1.5E-06	2%
Lead (Pb)		3.0E-04	10%	2.7E-04	1.00E-4	3.1E-06	1.79E-13	2.2E-11	5.1E-11	--		1.9E-07	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	1.4E-03	8.14E-10	1.0E-07	2.3E-07	--		3.3E-06	5%
Mercury (Hg)		4.2E-05	40%	2.5E-05	1.00E-3	2.3E-07	1.36E-13	1.7E-11	3.9E-11	--		1.5E-06	2%
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	5.1E-06	1.79E-12	2.2E-10	5.1E-10	--		2.2E-06	3%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	1.3E-05	1.55E-12	1.9E-10	4.4E-10	--		9.3E-08	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	6.00E-4	4.4E-04	1.52E-10	1.9E-08	4.3E-08	--		4.3E-07	1%
												7.3E-05	

Year 4

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)	
Antimony (Sb)		1.4E-04	20%	1.1E-04	1.00E-3	2.1E-06	1.25E-12	1.5E-10	3.6E-10	--		3.3E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	1.9E-04	1.10E-10	1.3E-08	3.1E-08	--		3.1E-05	36%
Barium (Ba)		1.4E-02	10%	1.3E-02	1.00E-3	1.2E-04	6.71E-11	8.2E-09	1.9E-08	--		1.5E-06	2%
Beryllium (Be)		1.4E-05		1.4E-05	1.00E-3	1.0E-08	5.92E-15	7.2E-13	1.7E-12	--		1.2E-07	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	1.4E-06	8.21E-13	1.0E-10	2.3E-10	--		7.3E-07	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	2.00E-3	1.1E-04	1.22E-10	1.5E-08	3.5E-08	--		3.9E-05	45%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	6.3E-04	3.63E-10	4.4E-08	1.0E-07	--		1.8E-06	2%
Lead (Pb)		3.0E-04	10%	2.7E-04	1.00E-4	3.7E-06	2.15E-13	2.6E-11	6.1E-11	--		2.3E-07	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	1.7E-03	9.64E-10	1.2E-07	2.8E-07	--		3.9E-06	5%
Mercury (Hg)		4.2E-05	40%	2.5E-05	1.00E-3	2.8E-07	1.60E-13	2.0E-11	4.6E-11	--		1.8E-06	2%
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	6.2E-06	2.15E-12	2.6E-10	6.1E-10	--		2.7E-06	3%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	1.6E-05	1.86E-12	2.3E-10	5.3E-10	--		1.1E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	6.00E-4	5.1E-04	1.78E-10	2.2E-08	5.1E-08	--		5.1E-07	1%
												8.7E-05	

Year 6

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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		1.4E-04	20%	1.1E-04	1.00E-3	2.1E-06	1.20E-12	1.5E-10	3.4E-10	--		3.2E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	1.8E-04	1.05E-10	1.3E-08	3.0E-08	--		3.0E-05	35%
Barium (Ba)		1.4E-02	10%	1.3E-02	1.00E-3	1.1E-04	6.43E-11	7.9E-09	1.8E-08	--		1.5E-06	2%
Beryllium (Be)		1.4E-05		1.4E-05	1.00E-3	9.8E-09	5.68E-15	7.0E-13	1.6E-12	--		1.2E-07	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	1.4E-06	7.87E-13	9.6E-11	2.2E-10	--		7.0E-07	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	2.00E-3	1.0E-04	1.17E-10	1.4E-08	3.3E-08	--		3.7E-05	43%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	6.0E-04	3.48E-10	4.3E-08	9.9E-08	--		1.8E-06	2%
Lead (Pb)		3.0E-04	10%	2.7E-04	1.00E-4	3.6E-06	2.06E-13	2.5E-11	5.9E-11	--		2.2E-07	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	1.6E-03	9.25E-10	1.1E-07	2.6E-07	--		3.8E-06	4%
Mercury (Hg)		4.2E-05	40%	2.5E-05	1.00E-3	2.7E-07	1.54E-13	1.9E-11	4.4E-11	--		1.7E-06	2%
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	5.9E-06	2.06E-12	2.5E-10	5.9E-10	--		2.6E-06	3%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	1.5E-05	1.79E-12	2.2E-10	5.1E-10	--		1.1E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	6.00E-4	4.9E-04	1.71E-10	2.1E-08	4.9E-08	--		4.9E-07	1%
												8.3E-05	

Year 8

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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		1.4E-04	20%	1.1E-04	1.00E-3	2.1E-06	1.19E-12	1.5E-10	3.4E-10	--		3.1E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	1.8E-04	1.06E-10	1.3E-08	3.0E-08	--		3.0E-05	36%
Barium (Ba)		1.4E-02	10%	1.3E-02	1.00E-3	1.1E-04	6.40E-11	7.8E-09	1.8E-08	--		1.5E-06	2%
Beryllium (Be)		1.4E-05		1.4E-05	1.00E-3	9.6E-09	5.56E-15	6.8E-13	1.6E-12	--		1.1E-07	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	1.3E-06	7.79E-13	9.5E-11	2.2E-10	--		7.0E-07	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	2.00E-3	9.9E-05	1.15E-10	1.4E-08	3.3E-08	--		3.7E-05	44%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	6.2E-04	3.59E-10	4.4E-08	1.0E-07	--		1.8E-06	2%
Lead (Pb)		3.0E-04	10%	2.7E-04	1.00E-4	3.5E-06	2.05E-13	2.5E-11	5.9E-11	--		2.2E-07	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	1.6E-03	9.20E-10	1.1E-07	2.6E-07	--		3.8E-06	5%
Mercury (Hg)		4.2E-05	40%	2.5E-05	1.00E-3	2.6E-07	1.52E-13	1.9E-11	4.3E-11	--		1.7E-06	2%
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	6.0E-06	2.09E-12	2.6E-10	6.0E-10	--		2.6E-06	3%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	1.5E-05	1.77E-12	2.2E-10	5.1E-10	--		1.1E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	6.00E-4	4.8E-04	1.68E-10	2.1E-08	4.8E-08	--		4.8E-07	1%
												8.3E-05	

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

Year 1

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)				NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	1.26E-05	1.34E-04	3.8E-08	9.1E-08	--		1.3E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	6.74E-04	1.44E-03	7.3E-07	1.8E-06	--		5.9E-03	44%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	6.20E-04	4.96E-03	8.3E-10	2.0E-09	--		1.1E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	1.15E-06	6.13E-07	8.3E-10	2.0E-09	--		1.0E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.42E-05	3.79E-04	9.3E-08	2.3E-07	--		7.0E-04	5%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	2.88E-04	1.15E-04	2.0E-07	4.8E-07	--		5.4E-04	4%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.10E-03	4.49E-02	1.1E-05	2.7E-05	--		4.9E-04	4%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	4.45E-04	1.06E-03	5.1E-07	1.2E-06	--		2.3E-03	17%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.39E-02	1.86E-01	5.0E-05	1.2E-04	--		1.7E-03	13%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.93E-06	9.28E-05	2.2E-08	5.3E-08	--		1.5E-04	1%
Silver (Ag)		5.7E-03		5.7E-03	100%	5.82E-06	1.24E-04	3.1E-08	7.6E-08	--		1.3E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.47E-04	4.71E-04	1.9E-07	4.7E-07	--		9.8E-05	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	4.46E-03	2.51E-01	5.9E-05	1.4E-04	--		1.4E-03	11%
									TOTAL			**1.3E-02**	

Year 2

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	2.6E-05	2.8E-04	7.7E-08	1.9E-07	--		2.6E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	1.5E-03	3.1E-03	1.6E-06	3.8E-06	--		1.3E-02	45%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.3E-03	1.0E-02	1.6E-09	3.8E-09	--		2.1E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	2.2E-06	1.2E-06	1.6E-09	3.8E-09	--		1.9E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.9E-05	7.6E-04	1.9E-07	4.5E-07	--		1.4E-03	5%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	5.3E-04	2.1E-04	3.7E-07	8.9E-07	--		9.9E-04	4%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	5.6E-03	1.2E-01	3.0E-05	7.3E-05	--		1.3E-03	5%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	8.8E-04	2.1E-03	1.0E-06	2.4E-06	--		4.5E-03	16%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	2.9E-02	3.9E-01	1.0E-04	2.5E-04	--		3.6E-03	13%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	3.9E-06	1.9E-04	4.4E-08	1.1E-07	--		2.9E-04	1%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.4E-05	2.9E-04	7.3E-08	1.8E-07	--		3.1E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.8E-04	8.8E-04	3.6E-07	8.8E-07	--		1.8E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	8.6E-03	4.8E-01	1.1E-04	2.7E-04	--		2.7E-03	10%
									TOTAL			**2.8E-02**	

Year 4

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	3.1E-05	3.3E-04	9.2E-08	2.2E-07	--		3.1E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	1.7E-03	3.7E-03	1.9E-06	4.5E-06	--		1.5E-02	45%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.5E-03	1.2E-02	1.9E-09	4.5E-09	--		2.5E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	2.6E-06	1.4E-06	1.9E-09	4.5E-09	--		2.2E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.4E-05	9.0E-04	2.2E-07	5.3E-07	--		1.7E-03	5%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	6.3E-04	2.5E-04	4.4E-07	1.1E-06	--		1.2E-03	4%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	6.9E-03	1.5E-01	3.7E-05	9.0E-05	--		1.6E-03	5%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	1.1E-03	2.5E-03	1.2E-06	2.9E-06	--		5.4E-03	16%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.4E-02	4.6E-01	1.2E-04	3.0E-04	--		4.2E-03	13%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	4.6E-06	2.2E-04	5.2E-08	1.2E-07	--		3.5E-04	1%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.6E-05	3.5E-04	8.8E-08	2.1E-07	--		3.7E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.3E-04	1.1E-03	4.4E-07	1.1E-06	--		2.2E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	1.0E-02	5.7E-01	1.3E-04	3.2E-04	--		3.2E-03	10%
									TOTAL			**3.3E-02**	

Year 6

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	2.9E-05	3.1E-04	8.8E-08	2.1E-07	--		3.0E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	1.7E-03	3.6E-03	1.8E-06	4.4E-06	--		1.5E-02	44%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.4E-03	1.2E-02	1.8E-09	4.3E-09	--		2.4E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	2.5E-06	1.3E-06	1.8E-09	4.3E-09	--		2.1E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.2E-05	8.6E-04	2.1E-07	5.1E-07	--		1.6E-03	5%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	6.1E-04	2.4E-04	4.2E-07	1.0E-06	--		1.1E-03	3%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	6.7E-03	1.4E-01	3.6E-05	8.6E-05	--		1.5E-03	5%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	1.0E-03	2.4E-03	1.2E-06	2.8E-06	--		5.2E-03	15%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.3E-02	4.4E-01	1.2E-04	2.8E-04	--		4.1E-03	12%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	4.4E-06	2.1E-04	5.0E-08	1.2E-07	--		3.3E-04	1%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.6E-05	3.3E-04	8.4E-08	2.0E-07	--		3.5E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.2E-04	1.0E-03	4.2E-07	1.0E-06	--		2.1E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	9.7E-03	5.4E-01	1.3E-04	3.1E-04	--		3.1E-03	9%
									TOTAL			**3.2E-02**	

Year 8

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	2.9E-05	3.1E-04	8.8E-08	2.1E-07	--		2.9E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	1.7E-03	3.6E-03	1.8E-06	4.4E-06	--		1.5E-02	46%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.4E-03	1.1E-02	1.7E-09	4.2E-09	--		2.3E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	2.4E-06	1.3E-06	1.7E-09	4.2E-09	--		2.1E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.2E-05	8.5E-04	2.1E-07	5.1E-07	--		1.6E-03	5%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	6.0E-04	2.4E-04	4.2E-07	1.0E-06	--		1.1E-03	3%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	6.9E-03	1.5E-01	3.7E-05	8.9E-05	--		1.6E-03	5%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	1.0E-03	2.4E-03	1.2E-06	2.8E-06	--		5.1E-03	16%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.3E-02	4.4E-01	1.2E-04	2.8E-04	--		4.0E-03	13%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	4.3E-06	2.1E-04	4.9E-08	1.2E-07	--		3.3E-04	1%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.6E-05	3.4E-04	8.5E-08	2.1E-07	--		3.6E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.1E-04	1.0E-03	4.2E-07	1.0E-06	--		2.1E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	9.5E-03	5.4E-01	1.3E-04	3.0E-04	--		3.0E-03	9%
									TOTAL			**3.2E-02**	

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

Year 1

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	3.02E-07	5.0E-11	1.2E-10	--		1.7E-07	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	2.98E-05	4.9E-09	1.2E-08	--		4.0E-05	75%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	6.54E-08	1.1E-11	2.6E-11	--		1.5E-10	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	6.54E-08	1.1E-11	2.6E-11	--		1.3E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	8.97E-08	1.5E-11	3.6E-11	--		1.1E-07	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.68E-06	2.8E-10	6.7E-10	--		7.5E-07	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	5.05E-05	8.4E-09	2.0E-08	--		3.6E-07	1%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	1.12E-05	1.9E-09	4.5E-09	--		8.3E-06	16%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.35E-04	5.5E-08	1.3E-07	--		1.9E-06	4%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	9.77E-07	1.6E-10	3.9E-10	--		1.1E-06	2%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.40E-07	2.3E-11	5.6E-11	--		9.8E-09	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.86E-06	3.1E-10	7.4E-10	--		1.5E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	1.07E-04	1.8E-08	4.3E-08	--		4.3E-07	1%
TOTAL											**5.3E-05**	

Year 2

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	6.2E-07	1.0E-10	2.5E-10	--		3.4E-07	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	6.4E-05	1.1E-08	2.6E-08	--		8.6E-05	76%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	3.0E-05	5.0E-09	1.2E-08	--		6.7E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	1.2E-07	2.1E-11	5.0E-11	--		2.5E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.8E-07	3.0E-11	7.2E-11	--		2.3E-07	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	3.1E-06	5.1E-10	1.2E-09	--		1.4E-06	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.4E-04	2.2E-08	5.4E-08	--		9.7E-07	1%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	2.2E-05	3.7E-09	8.9E-09	--		1.7E-05	15%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	7.0E-04	1.2E-07	2.8E-07	--		4.0E-06	4%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	2.0E-06	3.2E-10	7.8E-10	--		2.2E-06	2%
Silver (Ag)		5.7E-03		5.7E-03	100%	3.3E-07	5.4E-11	1.3E-10	--		2.3E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.5E-06	5.8E-10	1.4E-09	--		2.9E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	2.1E-04	3.4E-08	8.3E-08	--		8.3E-07	1%
								TOTAL			**1.1E-04**	

Year 4

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	7.4E-07	1.2E-10	2.9E-10	--		4.1E-07	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	7.7E-05	1.3E-08	3.1E-08	--		1.0E-04	76%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	3.6E-05	6.0E-09	1.4E-08	--		8.0E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	1.5E-07	2.4E-11	5.8E-11	--		2.9E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.1E-07	3.5E-11	8.5E-11	--		2.7E-07	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	3.7E-06	6.1E-10	1.5E-09	--		1.6E-06	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.7E-04	2.8E-08	6.7E-08	--		1.2E-06	1%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	2.7E-05	4.4E-09	1.1E-08	--		2.0E-05	15%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	8.2E-04	1.4E-07	3.3E-07	--		4.7E-06	3%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	2.3E-06	3.8E-10	9.2E-10	--		2.6E-06	2%
Silver (Ag)		5.7E-03		5.7E-03	100%	3.9E-07	6.5E-11	1.6E-10	--		2.7E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.2E-06	6.9E-10	1.7E-09	--		3.5E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	2.4E-04	4.0E-08	9.7E-08	--		9.7E-07	1%
								TOTAL			**1.3E-04**	

Year 6

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	7.1E-07	1.2E-10	2.8E-10	--		3.9E-07	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	7.4E-05	1.2E-08	3.0E-08	--		9.8E-05	73%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	3.5E-05	5.7E-09	1.4E-08	--		7.7E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	1.4E-07	2.3E-11	5.6E-11	--		2.8E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.0E-07	3.4E-11	8.2E-11	--		2.6E-07	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	3.5E-06	5.9E-10	1.4E-09	--		1.6E-06	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.6E-04	2.7E-08	6.4E-08	--		1.1E-06	1%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	2.6E-05	4.3E-09	1.0E-08	--		1.9E-05	14%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	7.9E-04	1.3E-07	3.2E-07	--		4.5E-06	3%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	2.2E-06	3.7E-10	8.9E-10	--		2.5E-06	2%
Silver (Ag)		5.7E-03		5.7E-03	100%	3.8E-07	6.2E-11	1.5E-10	--		2.6E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.0E-06	6.7E-10	1.6E-09	--		3.3E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	2.3E-04	3.9E-08	9.3E-08	--		9.3E-07	1%
								TOTAL			**1.3E-04**	

Year 8

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	7.0E-07	1.2E-10	2.8E-10	--		3.9E-07	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	7.4E-05	1.2E-08	3.0E-08	--		9.9E-05	76%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	3.4E-05	5.7E-09	1.4E-08	--		7.7E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	1.4E-07	2.3E-11	5.5E-11	--		2.7E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.0E-07	3.3E-11	8.1E-11	--		2.5E-07	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	3.5E-06	5.8E-10	1.4E-09	--		1.5E-06	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.7E-04	2.7E-08	6.6E-08	--		1.2E-06	1%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	2.6E-05	4.2E-09	1.0E-08	--		1.9E-05	15%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	7.9E-04	1.3E-07	3.1E-07	--		4.5E-06	3%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	2.2E-06	3.6E-10	8.8E-10	--		2.4E-06	2%
Silver (Ag)		5.7E-03		5.7E-03	100%	3.8E-07	6.3E-11	1.5E-10	--		2.7E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.0E-06	6.6E-10	1.6E-09	--		3.3E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	2.3E-04	3.8E-08	9.1E-08	--		9.1E-07	1%
								TOTAL			**1.3E-04**	

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

Year 1

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	1.3E-06	4.4E-10	1.1E-09	--		1.5E-06	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	1.4E-04	4.7E-08	1.1E-07	--		3.8E-04	36%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	9.8E-06	3.2E-09	7.8E-09	--		4.3E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	3.6E-08	1.2E-11	2.9E-11	--		1.4E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.0E-07	9.9E-11	2.4E-10	--		7.5E-07	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	2.8E-04	9.2E-08	2.2E-07	--		2.5E-04	23%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.2E-03	7.3E-07	1.8E-06	--		3.2E-05	3%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	1.4E-05	4.6E-09	1.1E-08	--		2.1E-05	2%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	5.9E-04	1.9E-07	4.7E-07	--		6.7E-06	1%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	8.1E-08	2.7E-11	6.5E-11	--		1.8E-07	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.8E-06	6.1E-10	1.5E-09	--		2.6E-07	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.6E-06	1.5E-09	3.7E-09	--		7.7E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	4.7E-02	1.6E-05	3.7E-05	--		3.7E-04	35%
TOTAL											**1.1E-03**	

Year 2

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	2.7E-06	9.0E-10	2.2E-09	--		3.0E-06	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	3.1E-04	1.0E-07	2.4E-07	--		8.1E-04	39%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	2.0E-05	6.6E-09	1.6E-08	--		8.8E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	6.9E-08	2.3E-11	5.5E-11	--		2.8E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	6.0E-07	2.0E-10	4.8E-10	--		1.5E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	5.1E-04	1.7E-07	4.1E-07	--		4.6E-04	22%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	5.9E-03	2.0E-06	4.7E-06	--		8.5E-05	4%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	2.8E-05	9.2E-09	2.2E-08	--		1.8E-05	1%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.2E-03	4.0E-07	9.7E-07	--		1.4E-05	1%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.6E-07	5.4E-11	1.3E-10	--		3.6E-07	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	4.3E-06	1.4E-09	3.4E-09	--		6.0E-07	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	8.7E-06	2.9E-09	7.0E-09	--		1.5E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	9.0E-02	3.0E-05	7.2E-05	--		7.2E-04	34%
								TOTAL			**2.1E-03**	

Year 4

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	3.2E-06	1.1E-09	2.6E-09	--		3.6E-06	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	3.7E-04	1.2E-07	2.9E-07	--		9.7E-04	39%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	2.4E-05	7.9E-09	1.9E-08	--		1.1E-07	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	8.1E-08	2.7E-11	6.4E-11	--		3.2E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	7.1E-07	2.3E-10	5.7E-10	--		1.8E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	6.1E-04	2.0E-07	4.9E-07	--		5.4E-04	22%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	7.3E-03	2.4E-06	5.8E-06	--		1.0E-04	4%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	3.3E-05	1.1E-08	2.7E-08	--		2.1E-05	1%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.4E-03	4.8E-07	1.2E-06	--		1.6E-05	1%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.9E-07	6.4E-11	1.5E-10	--		4.3E-07	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	5.1E-06	1.7E-09	4.1E-09	--		7.2E-07	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.0E-05	3.5E-09	8.4E-09	--		1.7E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	1.1E-01	3.5E-05	8.5E-05	--		8.5E-04	34%
								TOTAL			**2.5E-03**	

Year 6

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	3.1E-06	1.0E-09	2.5E-09	--		3.4E-06	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	3.5E-04	1.2E-07	2.8E-07	--		9.4E-04	37%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	2.3E-05	7.5E-09	1.8E-08	--		1.0E-07	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	7.7E-08	2.6E-11	6.2E-11	--		3.1E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	6.8E-07	2.2E-10	5.4E-10	--		1.7E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	5.9E-04	1.9E-07	4.7E-07	--		5.2E-04	21%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	7.0E-03	2.3E-06	5.6E-06	--		1.0E-04	4%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	3.2E-05	1.1E-08	2.6E-08	--		2.0E-05	1%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.4E-03	4.6E-07	1.1E-06	--		1.6E-05	1%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.8E-07	6.1E-11	1.5E-10	--		4.1E-07	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	4.9E-06	1.6E-09	3.9E-09	--		6.9E-07	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.0E-05	3.3E-09	8.0E-09	--		1.7E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	1.0E-01	3.4E-05	8.1E-05	--		8.1E-04	32%
								TOTAL			**2.4E-03**	

Year 8

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	3.1E-06	1.0E-09	2.5E-09	--		3.4E-06	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	3.5E-04	1.2E-07	2.8E-07	--		9.4E-04	39%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	2.3E-05	7.5E-09	1.8E-08	--		1.0E-07	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	7.6E-08	2.5E-11	6.1E-11	--		3.0E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	6.7E-07	2.2E-10	5.4E-10	--		1.7E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	5.8E-04	1.9E-07	4.6E-07	--		5.1E-04	21%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	7.2E-03	2.4E-06	5.8E-06	--		1.0E-04	4%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	3.2E-05	1.1E-08	2.5E-08	--		2.0E-05	1%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.4E-03	4.6E-07	1.1E-06	--		1.6E-05	1%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.8E-07	6.0E-11	1.5E-10	--		4.0E-07	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	5.0E-06	1.7E-09	4.0E-09	--		7.0E-07	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	9.9E-06	3.3E-09	7.9E-09	--		1.7E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	1.0E-01	3.3E-05	8.0E-05	--		8.0E-04	33%
								TOTAL			**2.4E-03**	

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

Year 1

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
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	3.2E-07	2.5E-09	6.0E-09	--		8.3E-06	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	8.7E-06	6.6E-08	1.6E-07	--		5.3E-04	35%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	5.6E-05	4.3E-07	1.0E-06	--		5.7E-06	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	2.7E-10	2.0E-12	4.9E-12	--		2.5E-09	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.8E-08	1.4E-10	3.4E-10	--		1.1E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	6.7E-07	5.1E-09	1.2E-08	--		1.4E-05	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	8.1E-04	6.2E-06	1.5E-05	--		2.7E-04	17%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	6.9E-06	5.3E-08	1.3E-07	--		2.3E-04	15%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.3E-03	9.6E-06	2.3E-05	--		3.3E-04	22%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	3.5E-08	2.7E-10	6.4E-10	--		1.8E-06	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	4.0E-05	3.1E-07	7.5E-07	--		1.3E-04	9%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.1E-06	8.7E-09	2.1E-08	--		4.4E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	3.1E-09	2.4E-11	5.7E-11	--		5.7E-10	0%
TOTAL											**1.5E-03**	

Year 2

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
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	6.6E-07	5.1E-09	1.2E-08	--		1.7E-05	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	1.9E-05	1.4E-07	3.5E-07	--		1.2E-03	34%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.1E-04	8.7E-07	2.1E-06	--		1.2E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	5.1E-10	3.9E-12	9.3E-12	--		4.7E-09	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.7E-08	2.8E-10	6.8E-10	--		2.1E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.2E-06	9.4E-09	2.3E-08	--		2.5E-05	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.2E-03	1.7E-05	4.0E-05	--		7.2E-04	21%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	1.4E-05	1.0E-07	2.5E-07	--		4.7E-04	14%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	2.6E-03	2.0E-05	4.8E-05	--		6.9E-04	20%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	6.9E-08	5.3E-10	1.3E-09	--		3.6E-06	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	9.4E-05	7.2E-07	1.7E-06	--		3.0E-04	9%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.1E-06	1.6E-08	3.9E-08	--		8.2E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	6.0E-09	4.6E-11	1.1E-10	--		1.1E-09	0%
								TOTAL			**3.4E-03**	

Year 4

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
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	7.9E-07	6.0E-09	1.5E-08	--		2.0E-05	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	2.2E-05	1.7E-07	4.1E-07	--		1.4E-03	34%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.4E-04	1.0E-06	2.5E-06	--		1.4E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	5.9E-10	4.5E-12	1.1E-11	--		5.5E-09	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	4.3E-08	3.3E-10	8.0E-10	--		2.5E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.5E-06	1.1E-08	2.7E-08	--		3.0E-05	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.7E-03	2.0E-05	4.9E-05	--		8.8E-04	22%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	1.6E-05	1.2E-07	3.0E-07	--		5.6E-04	14%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.1E-03	2.4E-05	5.7E-05	--		8.1E-04	20%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	8.2E-08	6.3E-10	1.5E-09	--		4.2E-06	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.1E-04	8.6E-07	2.1E-06	--		3.7E-04	9%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.6E-06	2.0E-08	4.7E-08	--		9.8E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	7.0E-09	5.4E-11	1.3E-10	--		1.3E-09	0%
								TOTAL			**4.1E-03**	

Year 6

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
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	7.6E-07	5.8E-09	1.4E-08	--		1.9E-05	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	2.1E-05	1.6E-07	4.0E-07	--		1.3E-03	32%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.3E-04	9.9E-07	2.4E-06	--		1.3E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	5.7E-10	4.3E-12	1.0E-11	--		5.2E-09	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	4.1E-08	3.2E-10	7.7E-10	--		2.4E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.4E-06	1.1E-08	2.6E-08	--		2.9E-05	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.6E-03	2.0E-05	4.7E-05	--		8.5E-04	21%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	1.6E-05	1.2E-07	2.9E-07	--		5.4E-04	13%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.0E-03	2.3E-05	5.5E-05	--		7.8E-04	19%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	7.9E-08	6.0E-10	1.5E-09	--		4.0E-06	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.1E-04	8.3E-07	2.0E-06	--		3.5E-04	9%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.4E-06	1.9E-08	4.5E-08	--		9.4E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	6.7E-09	5.1E-11	1.2E-10	--		1.2E-09	0%
								TOTAL			**3.9E-03**	

Year 8

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
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	7.5E-07	5.8E-09	1.4E-08	--		1.9E-05	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	2.1E-05	1.6E-07	4.0E-07	--		1.3E-03	34%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.3E-04	9.9E-07	2.4E-06	--		1.3E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	5.5E-10	4.3E-12	1.0E-11	--		5.1E-09	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	4.1E-08	3.1E-10	7.6E-10	--		2.4E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.4E-06	1.1E-08	2.6E-08	--		2.8E-05	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.7E-03	2.0E-05	4.9E-05	--		8.8E-04	22%
Lead (Pb)		6.0E-04	10%	5.4E-04	50%	1.6E-05	1.2E-07	2.9E-07	--		5.3E-04	14%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	2.9E-03	2.3E-05	5.4E-05	--		7.8E-04	20%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	7.8E-08	6.0E-10	1.4E-09	--		4.0E-06	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.1E-04	8.4E-07	2.0E-06	--		3.5E-04	9%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.4E-06	1.9E-08	4.5E-08	--		9.3E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	6.6E-09	5.0E-11	1.2E-10	--		1.2E-09	0%
								TOTAL			**3.9E-03**	

Young children

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 Children		
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

Year 1

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)	
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	4.0E-02	2.3E-08	2.7E-07	--		3.7E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	2.2E+00	1.2E-06	1.4E-05	--		4.8E-02	68%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	2.0E+00	1.1E-06	1.3E-05	--		7.3E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	3.7E-03	2.1E-09	2.5E-08	--		1.2E-05	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	4.5E-02	2.6E-08	3.0E-07	--		9.5E-04	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	9.2E-01	5.3E-07	6.2E-06	--		6.8E-03	10%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	6.7E+00	3.8E-06	4.5E-05	--		8.0E-04	1%
Lead (Pb)		1.4E-03	10%	1.3E-03	100%	1.4E+00	8.1E-07	9.5E-06	--		7.5E-03	11%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	4.5E+01	2.5E-05	3.0E-04	--		4.2E-03	6%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	6.2E-03	3.5E-09	4.1E-08	--		1.1E-04	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.9E-02	1.1E-08	1.2E-07	--		2.2E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.7E-01	2.7E-07	3.1E-06	--		6.5E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	1.4E+01	8.1E-06	9.5E-05	--		9.5E-04	1%
TOTAL											**7.1E-02**	

Year 2

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	8.3E-02	4.7E-08	5.5E-07	--		7.7E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	4.7E+00	2.7E-06	3.1E-05	--		1.0E-01	70%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	4.0E+00	2.3E-06	2.7E-05	--		1.5E-04	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	7.0E-03	4.0E-09	4.7E-08	--		2.3E-05	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	9.1E-02	5.2E-08	6.1E-07	--		1.9E-03	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.7E+00	9.7E-07	1.1E-05	--		1.3E-02	8%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.8E+01	1.0E-05	1.2E-04	--		2.2E-03	1%
Lead (Pb)		1.4E-03	10%	1.3E-03	100%	2.8E+00	1.6E-06	1.9E-05	--		1.5E-02	10%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	9.3E+01	5.3E-05	6.2E-04	--		8.8E-03	6%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.2E-02	7.1E-09	8.2E-08	--		2.3E-04	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	4.3E-02	2.5E-08	2.9E-07	--		5.1E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	8.8E-01	5.1E-07	5.9E-06	--		1.2E-03	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	2.8E+01	1.6E-05	1.8E-04	--		1.8E-03	1%
								TOTAL			**1.5E-01**	

Year 4

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	9.8E-02	5.6E-08	6.5E-07	--		9.1E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	5.6E+00	3.2E-06	3.7E-05	--		1.2E-01	70%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	4.8E+00	2.8E-06	3.2E-05	--		1.8E-04	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	8.2E-03	4.7E-09	5.5E-08	--		2.7E-05	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.1E-01	6.2E-08	7.2E-07	--		2.2E-03	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	2.0E+00	1.2E-06	1.4E-05	--		1.5E-02	8%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.2E+01	1.3E-05	1.5E-04	--		2.6E-03	1%
Lead (Pb)		1.4E-03	10%	1.3E-03	100%	3.4E+00	1.9E-06	2.3E-05	--		1.8E-02	10%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.1E+02	6.3E-05	7.3E-04	--		1.0E-02	6%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.5E-02	8.3E-09	9.7E-08	--		2.7E-04	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	5.2E-02	3.0E-08	3.5E-07	--		6.1E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.1E+00	6.1E-07	7.1E-06	--		1.5E-03	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	3.2E+01	1.8E-05	2.2E-04	--		2.2E-03	1%
								TOTAL			**1.8E-01**	

Year 6

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	9.4E-02	5.4E-08	6.3E-07	--		8.7E-04	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	5.3E+00	3.1E-06	3.6E-05	--		1.2E-01	67%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	4.6E+00	2.6E-06	3.1E-05	--		1.7E-04	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	7.9E-03	4.5E-09	5.2E-08	--		2.6E-05	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.0E-01	5.9E-08	6.9E-07	--		2.2E-03	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.9E+00	1.1E-06	1.3E-05	--		1.4E-02	8%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.1E+01	1.2E-05	1.4E-04	--		2.5E-03	1%
Lead (Pb)		1.4E-03	10%	1.3E-03	100%	3.2E+00	1.9E-06	2.2E-05	--		1.7E-02	10%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.1E+02	6.0E-05	7.0E-04	--		1.0E-02	6%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.4E-02	8.0E-09	9.3E-08	--		2.6E-04	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	5.0E-02	2.9E-08	3.3E-07	--		5.8E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.0E+00	5.8E-07	6.8E-06	--		1.4E-03	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	3.1E+01	1.8E-05	2.1E-04	--		2.1E-03	1%
								TOTAL			**1.7E-01**	

Year 8

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	9.4E-02	5.4E-08	6.2E-07	--		8.7E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	5.4E+00	3.1E-06	3.6E-05	--		1.2E-01	70%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	4.6E+00	2.6E-06	3.1E-05	--		1.7E-04	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	7.7E-03	4.4E-09	5.1E-08	--		2.6E-05	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.0E-01	5.8E-08	6.8E-07	--		2.1E-03	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.9E+00	1.1E-06	1.3E-05	--		1.4E-02	8%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.2E+01	1.3E-05	1.5E-04	--		2.6E-03	2%
Lead (Pb)		1.4E-03	10%	1.3E-03	100%	3.2E+00	1.8E-06	2.2E-05	--		1.7E-02	10%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.0E+02	6.0E-05	7.0E-04	--		1.0E-02	6%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.4E-02	7.9E-09	9.2E-08	--		2.6E-04	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	5.1E-02	2.9E-08	3.4E-07	--		5.9E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.0E+00	5.7E-07	6.7E-06	--		1.4E-03	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	3.0E+01	1.7E-05	2.0E-04	--		2.0E-03	1%
								TOTAL			**1.7E-01**	

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

Year 1

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		1.4E-04	20%	1.1E-04		4.0E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	2.2E+00	8.3E-08	9.7E-07	--		9.7E-04	92%
Barium (Ba)		1.4E-02	10%	1.3E-02		2.0E+00			--		--	
Beryllium (Be)		1.4E-05		1.4E-05		3.7E-03			--		--	
Cadmium (Cd)		8.0E-04	60%	3.2E-04		4.5E-02			--		--	
Chromium (Cr)		1.0E-03	10%	9.0E-04		9.2E-01			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		6.7E+00			--		--	
Lead (Pb)		7.0E-04	10%	6.3E-04		1.4E+00			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		4.5E+01			--		--	
Mercury (Hg)		4.2E-05	40%	2.5E-05	0.001	6.2E-03	4.8E-11	5.6E-10	--		2.2E-05	2%
Silver (Ag)		2.3E-04		2.3E-04		1.9E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03	0.005	4.7E-01	1.8E-08	2.1E-07	--		4.4E-05	4%
Zinc (Zn)		5.0E-01	80%	1.0E-01	0.001	1.4E+01	1.1E-07	1.3E-06	--		1.3E-05	1%
TOTAL											**1.1E-03**	

Year 2

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		1.4E-04	20%	1.1E-04		8.3E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	4.7E+00	1.8E-07	2.1E-06	--		2.1E-03	93%
Barium (Ba)		1.4E-02	10%	1.3E-02		4.0E+00			--		--	
Beryllium (Be)		1.4E-05		1.4E-05		7.0E-03			--		--	
Cadmium (Cd)		8.0E-04	60%	3.2E-04		9.1E-02			--		--	
Chromium (Cr)		1.0E-03	10%	9.0E-04		1.7E+00			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		1.8E+01			--		--	
Lead (Pb)		7.0E-04	10%	6.3E-04		2.8E+00			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		9.3E+01			--		--	
Mercury (Hg)		4.2E-05	40%	2.5E-05	0.001	1.2E-02	9.5E-11	1.1E-09	--		4.4E-05	2%
Silver (Ag)		2.3E-04		2.3E-04		4.3E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03	0.005	8.8E-01	3.4E-08	4.0E-07	--		8.3E-05	4%
Zinc (Zn)		5.0E-01	80%	1.0E-01	0.001	2.8E+01	2.1E-07	2.5E-06	--		2.5E-05	1%
								TOTAL			**2.2E-03**	

Year 4

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		1.4E-04	20%	1.1E-04		9.8E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	5.6E+00	2.1E-07	2.5E-06	--		2.5E-03	93%
Barium (Ba)		1.4E-02	10%	1.3E-02		4.8E+00			--		--	
Beryllium (Be)		1.4E-05		1.4E-05		8.2E-03			--		--	
Cadmium (Cd)		8.0E-04	60%	3.2E-04		1.1E-01			--		--	
Chromium (Cr)		1.0E-03	10%	9.0E-04		2.0E+00			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		2.2E+01			--		--	
Lead (Pb)		7.0E-04	10%	6.3E-04		3.4E+00			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		1.1E+02			--		--	
Mercury (Hg)		4.2E-05	40%	2.5E-05	0.001	1.5E-02	1.1E-10	1.3E-09	--		5.2E-05	2%
Silver (Ag)		2.3E-04		2.3E-04		5.2E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03	0.005	1.1E+00	4.1E-08	4.8E-07	--		9.9E-05	4%
Zinc (Zn)		5.0E-01	80%	1.0E-01	0.001	3.2E+01	2.5E-07	2.9E-06	--		2.9E-05	1%
								TOTAL			**2.7E-03**	

Year 6

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		1.4E-04	20%	1.1E-04		9.4E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	5.3E+00	2.1E-07	2.4E-06	--		2.4E-03	93%
Barium (Ba)		1.4E-02	10%	1.3E-02		4.6E+00			--		--	
Beryllium (Be)		1.4E-05		1.4E-05		7.9E-03			--		--	
Cadmium (Cd)		8.0E-04	60%	3.2E-04		1.0E-01			--		--	
Chromium (Cr)		1.0E-03	10%	9.0E-04		1.9E+00			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		2.1E+01			--		--	
Lead (Pb)		7.0E-04	10%	6.3E-04		3.2E+00			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		1.1E+02			--		--	
Mercury (Hg)		4.2E-05	40%	2.5E-05	0.001	1.4E-02	1.1E-10	1.3E-09	--		5.0E-05	2%
Silver (Ag)		2.3E-04		2.3E-04		5.0E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03	0.005	1.0E+00	3.9E-08	4.6E-07	--		9.5E-05	4%
Zinc (Zn)		5.0E-01	80%	1.0E-01	0.001	3.1E+01	2.4E-07	2.8E-06	--		2.8E-05	1%
								TOTAL			**2.6E-03**	

Year 8

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		1.4E-04	20%	1.1E-04		9.4E-02			--		--	
Arsenic (As)		2.0E-03	50%	1.0E-03	0.005	5.4E+00	2.1E-07	2.4E-06	--		2.4E-03	93%
Barium (Ba)		1.4E-02	10%	1.3E-02		4.6E+00			--		--	
Beryllium (Be)		1.4E-05		1.4E-05		7.7E-03			--		--	
Cadmium (Cd)		8.0E-04	60%	3.2E-04		1.0E-01			--		--	
Chromium (Cr)		1.0E-03	10%	9.0E-04		1.9E+00			--		--	
Copper (Cu)		1.4E-01	60%	5.6E-02		2.2E+01			--		--	
Lead (Pb)		7.0E-04	10%	6.3E-04		3.2E+00			--		--	
Manganese (Mn)		1.4E-01	50%	7.0E-02		1.0E+02			--		--	
Mercury (Hg)		4.2E-05	40%	2.5E-05	0.001	1.4E-02	1.1E-10	1.2E-09	--		4.9E-05	2%
Silver (Ag)		2.3E-04		2.3E-04		5.1E-02			--		--	
Nickel (Ni)		1.2E-02	60%	4.8E-03	0.005	1.0E+00	3.9E-08	4.5E-07	--		9.4E-05	4%
Zinc (Zn)		5.0E-01	80%	1.0E-01	0.001	3.0E+01	2.3E-07	2.7E-06	--		2.7E-05	1%
								TOTAL			**2.6E-03**	

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

Year 1

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)	
Antimony (Sb)	0.0E+00	9.0E-04	20%	7.2E-04	100%	8.8E-07	2.0E-09	2.3E-08	--		3.3E-05	0%
Arsenic (As)	0.0E+00	6.0E-04	50%	3.0E-04	100%	7.3E-05	1.7E-07	2.0E-06	--		6.5E-03	76%
Barium (Ba)	0.0E+00	2.0E-01	10%	1.8E-01	100%	4.8E-05	1.1E-07	1.3E-06	--		7.1E-06	0%
Beryllium (Be)	0.0E+00	2.0E-03	0%	2.0E-03	100%	4.6E-09	1.0E-11	1.2E-10	--		6.1E-08	0%
Cadmium (Cd)	0.0E+00	8.0E-04	60%	3.2E-04	100%	6.0E-07	1.4E-09	1.6E-08	--		5.0E-05	1%
Chromium (Cr)	0.0E+00	1.0E-03	10%	9.0E-04	100%	4.8E-05	1.1E-07	1.3E-06	--		1.4E-03	17%
Copper (Cu)	0.0E+00	1.4E-01	60%	5.6E-02	100%	1.9E-04	4.3E-07	5.0E-06	--		9.0E-05	1%
Lead (Pb)	0.0E+00	1.4E-03	10%	1.3E-03	50%	1.6E-06	3.6E-09	4.2E-08	--		3.3E-05	0%
Manganese (Mn)	0.0E+00	1.4E-01	50%	7.0E-02	100%	6.8E-04	1.5E-06	1.8E-05	--		2.6E-04	3%
Mercury (Hg)	0.0E+00	6.0E-04	40%	3.6E-04	100%	1.2E-07	2.7E-10	3.1E-09	--		8.7E-06	0%
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	2.2E-06	5.1E-09	5.9E-08	--		1.0E-05	0%
Nickel (Ni)	0.0E+00	1.2E-02	60%	4.8E-03	100%	7.1E-06	1.6E-08	1.9E-07	--		4.0E-05	0%
Zinc (Zn)	0.0E+00	5.0E-01	80%	1.0E-01	100%	2.3E-04	5.2E-07	6.0E-06	--		6.0E-05	1%
TOTAL									**0.00E+00**		**8.5E-03**	

Year 2

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)	
Antimony (Sb)	0.0E+00	9.0E-04	20%	7.2E-04	100%	1.8E-06	4.1E-09	4.8E-08	--		6.7E-05	0%
Arsenic (As)	0.0E+00	6.0E-04	50%	3.0E-04	100%	1.6E-04	3.6E-07	4.2E-06	--		1.4E-02	78%
Barium (Ba)	0.0E+00	2.0E-01	10%	1.8E-01	100%	9.7E-05	2.2E-07	2.6E-06	--		1.4E-05	0%
Beryllium (Be)	0.0E+00	2.0E-03	0%	2.0E-03	100%	8.7E-09	2.0E-11	2.3E-10	--		1.2E-07	0%
Cadmium (Cd)	0.0E+00	8.0E-04	60%	3.2E-04	100%	1.2E-06	2.7E-09	3.2E-08	--		1.0E-04	1%
Chromium (Cr)	0.0E+00	1.0E-03	10%	9.0E-04	100%	8.8E-05	2.0E-07	2.3E-06	--		2.6E-03	15%
Copper (Cu)	0.0E+00	1.4E-01	60%	5.6E-02	100%	5.1E-04	1.2E-06	1.4E-05	--		2.4E-04	1%
Lead (Pb)	0.0E+00	1.4E-03	10%	1.3E-03	50%	3.1E-06	7.1E-09	8.2E-08	--		6.5E-05	0%
Manganese (Mn)	0.0E+00	1.4E-01	50%	7.0E-02	100%	1.4E-03	3.2E-06	3.7E-05	--		5.3E-04	3%
Mercury (Hg)	0.0E+00	6.0E-04	40%	3.6E-04	100%	2.3E-07	5.4E-10	6.2E-09	--		1.7E-05	0%
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	5.1E-06	1.2E-08	1.4E-07	--		2.4E-05	0%
Nickel (Ni)	0.0E+00	1.2E-02	60%	4.8E-03	100%	1.3E-05	3.1E-08	3.6E-07	--		7.4E-05	0%
Zinc (Zn)	0.0E+00	5.0E-01	80%	1.0E-01	100%	4.4E-04	1.0E-06	1.2E-05	--		1.2E-04	1%
								TOTAL	**0.00E+00**		**1.8E-02**	

Year 4

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)	
Antimony (Sb)	0.0E+00	9.0E-04	20%	7.2E-04	100%	2.1E-06	4.9E-09	5.7E-08	--		8.0E-05	0%
Arsenic (As)	0.0E+00	6.0E-04	50%	3.0E-04	100%	1.9E-04	4.3E-07	5.0E-06	--		1.7E-02	78%
Barium (Ba)	0.0E+00	2.0E-01	10%	1.8E-01	100%	1.2E-04	2.6E-07	3.1E-06	--		1.7E-05	0%
Beryllium (Be)	0.0E+00	2.0E-03	0%	2.0E-03	100%	1.0E-08	2.3E-11	2.7E-10	--		1.4E-07	0%
Cadmium (Cd)	0.0E+00	8.0E-04	60%	3.2E-04	100%	1.4E-06	3.2E-09	3.8E-08	--		1.2E-04	1%
Chromium (Cr)	0.0E+00	1.0E-03	10%	9.0E-04	100%	1.1E-04	2.4E-07	2.8E-06	--		3.1E-03	15%
Copper (Cu)	0.0E+00	1.4E-01	60%	5.6E-02	100%	6.3E-04	1.4E-06	1.7E-05	--		3.0E-04	1%
Lead (Pb)	0.0E+00	1.4E-03	10%	1.3E-03	50%	3.7E-06	8.5E-09	9.9E-08	--		7.8E-05	0%
Manganese (Mn)	0.0E+00	1.4E-01	50%	7.0E-02	100%	1.7E-03	3.8E-06	4.4E-05	--		6.3E-04	3%
Mercury (Hg)	0.0E+00	6.0E-04	40%	3.6E-04	100%	2.8E-07	6.3E-10	7.4E-09	--		2.0E-05	0%
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	6.2E-06	1.4E-08	1.6E-07	--		2.9E-05	0%
Nickel (Ni)	0.0E+00	1.2E-02	60%	4.8E-03	100%	1.6E-05	3.7E-08	4.3E-07	--		8.9E-05	0%
Zinc (Zn)	0.0E+00	5.0E-01	80%	1.0E-01	100%	5.1E-04	1.2E-06	1.4E-05	--		1.4E-04	1%
								TOTAL	**0.00E+00**		**2.1E-02**	

Year 6

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)	
Antimony (Sb)	0.0E+00	9.0E-04	20%	7.2E-04	100%	2.1E-06	4.7E-09	5.5E-08	--		7.6E-05	0%
Arsenic (As)	0.0E+00	6.0E-04	50%	3.0E-04	100%	1.8E-04	4.1E-07	4.8E-06	--		1.6E-02	75%
Barium (Ba)	0.0E+00	2.0E-01	10%	1.8E-01	100%	1.1E-04	2.5E-07	3.0E-06	--		1.6E-05	0%
Beryllium (Be)	0.0E+00	2.0E-03	0%	2.0E-03	100%	9.8E-09	2.2E-11	2.6E-10	--		1.3E-07	0%
Cadmium (Cd)	0.0E+00	8.0E-04	60%	3.2E-04	100%	1.4E-06	3.1E-09	3.6E-08	--		1.1E-04	1%
Chromium (Cr)	0.0E+00	1.0E-03	10%	9.0E-04	100%	1.0E-04	2.3E-07	2.7E-06	--		3.0E-03	14%
Copper (Cu)	0.0E+00	1.4E-01	60%	5.6E-02	100%	6.0E-04	1.4E-06	1.6E-05	--		2.9E-04	1%
Lead (Pb)	0.0E+00	1.4E-03	10%	1.3E-03	50%	3.6E-06	8.1E-09	9.5E-08	--		7.5E-05	0%
Manganese (Mn)	0.0E+00	1.4E-01	50%	7.0E-02	100%	1.6E-03	3.6E-06	4.3E-05	--		6.1E-04	3%
Mercury (Hg)	0.0E+00	6.0E-04	40%	3.6E-04	100%	2.7E-07	6.1E-10	7.1E-09	--		2.0E-05	0%
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	5.9E-06	1.4E-08	1.6E-07	--		2.8E-05	0%
Nickel (Ni)	0.0E+00	1.2E-02	60%	4.8E-03	100%	1.5E-05	3.5E-08	4.1E-07	--		8.6E-05	0%
Zinc (Zn)	0.0E+00	5.0E-01	80%	1.0E-01	100%	4.9E-04	1.1E-06	1.3E-05	--		1.3E-04	1%
								TOTAL	**0.00E+00**		**2.1E-02**	

Year 8

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)	
Antimony (Sb)	0.0E+00	9.0E-04	20%	7.2E-04	100%	2.1E-06	4.7E-09	5.5E-08	--		7.6E-05	0%
Arsenic (As)	0.0E+00	6.0E-04	50%	3.0E-04	100%	1.8E-04	4.2E-07	4.9E-06	--		1.6E-02	79%
Barium (Ba)	0.0E+00	2.0E-01	10%	1.8E-01	100%	1.1E-04	2.5E-07	2.9E-06	--		1.6E-05	0%
Beryllium (Be)	0.0E+00	2.0E-03	0%	2.0E-03	100%	9.6E-09	2.2E-11	2.6E-10	--		1.3E-07	0%
Cadmium (Cd)	0.0E+00	8.0E-04	60%	3.2E-04	100%	1.3E-06	3.1E-09	3.6E-08	--		1.1E-04	1%
Chromium (Cr)	0.0E+00	1.0E-03	10%	9.0E-04	100%	9.9E-05	2.3E-07	2.7E-06	--		2.9E-03	14%
Copper (Cu)	0.0E+00	1.4E-01	60%	5.6E-02	100%	6.2E-04	1.4E-06	1.7E-05	--		3.0E-04	1%
Lead (Pb)	0.0E+00	1.4E-03	10%	1.3E-03	50%	3.5E-06	8.1E-09	9.4E-08	--		7.5E-05	0%
Manganese (Mn)	0.0E+00	1.4E-01	50%	7.0E-02	100%	1.6E-03	3.6E-06	4.2E-05	--		6.0E-04	3%
Mercury (Hg)	0.0E+00	6.0E-04	40%	3.6E-04	100%	2.6E-07	6.0E-10	7.0E-09	--		1.9E-05	0%
Silver (Ag)	0.0E+00	5.7E-03	0%	5.7E-03	100%	6.0E-06	1.4E-08	1.6E-07	--		2.8E-05	0%
Nickel (Ni)	0.0E+00	1.2E-02	60%	4.8E-03	100%	1.5E-05	3.5E-08	4.1E-07	--		8.5E-05	0%
Zinc (Zn)	0.0E+00	5.0E-01	80%	1.0E-01	100%	4.8E-04	1.1E-06	1.3E-05	--		1.3E-04	1%
								TOTAL	**0.00E+00**		**2.1E-02**	

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

Year 1

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)	
Antimony (Sb)		1.4E-04	20%	1.1E-04	1.00E-3	8.8E-07	8.81E-13	3.1E-11	3.6E-10	--		3.3E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	7.3E-05	7.33E-11	2.6E-09	3.0E-08	--		3.0E-05	33%
Barium (Ba)		1.4E-02	10%	1.3E-02	1.00E-3	4.8E-05	4.76E-11	1.7E-09	1.9E-08	--		1.5E-06	2%
Beryllium (Be)		1.4E-05		1.4E-05	1.00E-3	4.6E-09	4.59E-15	1.6E-13	1.9E-12	--		1.3E-07	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	6.0E-07	5.97E-13	2.1E-11	2.4E-10	--		7.6E-07	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	2.00E-3	4.8E-05	9.57E-11	3.3E-09	3.9E-08	--		4.3E-05	48%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	1.9E-04	1.89E-10	6.6E-09	7.7E-08	--		1.4E-06	2%
Lead (Pb)		3.0E-04	10%	2.7E-04	1.00E-4	1.6E-06	1.56E-13	5.4E-12	6.3E-11	--		2.3E-07	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	6.8E-04	6.76E-10	2.4E-08	2.7E-07	--		3.9E-06	4%
Mercury (Hg)		4.2E-05	40%	2.5E-05	1.00E-3	1.2E-07	1.17E-13	4.1E-12	4.8E-11	--		1.9E-06	2%
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	2.2E-06	1.33E-12	4.6E-11	5.4E-10	--		2.4E-06	3%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	7.1E-06	1.43E-12	5.0E-11	5.8E-10	--		1.2E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	6.00E-4	2.3E-04	1.36E-10	4.7E-09	5.5E-08	--		5.5E-07	1%
												8.9E-05	

Year 2

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)	
Antimony (Sb)		1.4E-04	20%	1.1E-04	1.00E-3	1.8E-06	1.81E-12	6.3E-11	7.4E-10	--		6.8E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	1.6E-04	1.58E-10	5.5E-09	6.4E-08	--		6.4E-05	36%
Barium (Ba)		1.4E-02	10%	1.3E-02	1.00E-3	9.7E-05	9.70E-11	3.4E-09	3.9E-08	--		3.1E-06	2%
Beryllium (Be)		1.4E-05		1.4E-05	1.00E-3	8.7E-09	8.73E-15	3.0E-13	3.5E-12	--		2.5E-07	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	1.2E-06	1.20E-12	4.2E-11	4.9E-10	--		1.5E-06	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	2.00E-3	8.8E-05	1.76E-10	6.1E-09	7.2E-08	--		8.0E-05	45%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	5.1E-04	5.08E-10	1.8E-08	2.1E-07	--		3.7E-06	2%
Lead (Pb)		3.0E-04	10%	2.7E-04	1.00E-4	3.1E-06	3.09E-13	1.1E-11	1.3E-10	--		4.7E-07	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	1.4E-03	1.40E-09	4.9E-08	5.7E-07	--		8.2E-06	5%
Mercury (Hg)		4.2E-05	40%	2.5E-05	1.00E-3	2.3E-07	2.34E-13	8.2E-12	9.5E-11	--		3.8E-06	2%
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	5.1E-06	3.08E-12	1.1E-10	1.3E-09	--		5.5E-06	3%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	1.3E-05	2.68E-12	9.3E-11	1.1E-09	--		2.3E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	6.00E-4	4.4E-04	2.62E-10	9.1E-09	1.1E-07	--		1.1E-06	1%
												1.8E-04	

Year 4

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)	
Antimony (Sb)		1.4E-04	20%	1.1E-04	1.00E-3	2.1E-06	2.15E-12	7.5E-11	8.7E-10	--		8.1E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	1.9E-04	1.89E-10	6.6E-09	7.7E-08	--		7.7E-05	36%
Barium (Ba)		1.4E-02	10%	1.3E-02	1.00E-3	1.2E-04	1.16E-10	4.0E-09	4.7E-08	--		3.7E-06	2%
Beryllium (Be)		1.4E-05		1.4E-05	1.00E-3	1.0E-08	1.02E-14	3.6E-13	4.1E-12	--		3.0E-07	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	1.4E-06	1.42E-12	4.9E-11	5.8E-10	--		1.8E-06	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	2.00E-3	1.1E-04	2.11E-10	7.3E-09	8.6E-08	--		9.5E-05	45%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	6.3E-04	6.25E-10	2.2E-08	2.5E-07	--		4.5E-06	2%
Lead (Pb)		3.0E-04	10%	2.7E-04	1.00E-4	3.7E-06	3.71E-13	1.3E-11	1.5E-10	--		5.6E-07	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	1.7E-03	1.66E-09	5.8E-08	6.8E-07	--		9.7E-06	5%
Mercury (Hg)		4.2E-05	40%	2.5E-05	1.00E-3	2.8E-07	2.77E-13	9.6E-12	1.1E-10	--		4.5E-06	2%
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	6.2E-06	3.71E-12	1.3E-10	1.5E-09	--		6.6E-06	3%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	1.6E-05	3.21E-12	1.1E-10	1.3E-09	--		2.7E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	6.00E-4	5.1E-04	3.08E-10	1.1E-08	1.3E-07	--		1.3E-06	1%
												2.1E-04	

Year 6

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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		1.4E-04	20%	1.1E-04	1.00E-3	2.1E-06	2.06E-12	7.2E-11	8.4E-10	--		7.8E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	1.8E-04	1.81E-10	6.3E-09	7.4E-08	--		7.4E-05	35%
Barium (Ba)		1.4E-02	10%	1.3E-02	1.00E-3	1.1E-04	1.11E-10	3.9E-09	4.5E-08	--		3.6E-06	2%
Beryllium (Be)		1.4E-05		1.4E-05	1.00E-3	9.8E-09	9.79E-15	3.4E-13	4.0E-12	--		2.8E-07	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	1.4E-06	1.36E-12	4.7E-11	5.5E-10	--		1.7E-06	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	2.00E-3	1.0E-04	2.02E-10	7.0E-09	8.2E-08	--		9.1E-05	43%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	6.0E-04	6.00E-10	2.1E-08	2.4E-07	--		4.4E-06	2%
Lead (Pb)		3.0E-04	10%	2.7E-04	1.00E-4	3.6E-06	3.56E-13	1.2E-11	1.4E-10	--		5.4E-07	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	1.6E-03	1.60E-09	5.6E-08	6.5E-07	--		9.3E-06	4%
Mercury (Hg)		4.2E-05	40%	2.5E-05	1.00E-3	2.7E-07	2.65E-13	9.2E-12	1.1E-10	--		4.3E-06	2%
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	5.9E-06	3.56E-12	1.2E-10	1.4E-09	--		6.3E-06	3%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	1.5E-05	3.08E-12	1.1E-10	1.3E-09	--		2.6E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	6.00E-4	4.9E-04	2.95E-10	1.0E-08	1.2E-07	--		1.2E-06	1%
												2.0E-04	

Year 8

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
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)	(cm/hr)	(mg/L)	(mg/cm2 per event)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		1.4E-04	20%	1.1E-04	1.00E-3	2.1E-06	2.05E-12	7.1E-11	8.3E-10	--		7.7E-06	4%
Arsenic (As)		2.0E-03	50%	1.0E-03	1.00E-3	1.8E-04	1.82E-10	6.3E-09	7.4E-08	--		7.4E-05	36%
Barium (Ba)		1.4E-02	10%	1.3E-02	1.00E-3	1.1E-04	1.10E-10	3.8E-09	4.5E-08	--		3.6E-06	2%
Beryllium (Be)		1.4E-05		1.4E-05	1.00E-3	9.6E-09	9.58E-15	3.3E-13	3.9E-12	--		2.8E-07	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	1.00E-3	1.3E-06	1.34E-12	4.7E-11	5.5E-10	--		1.7E-06	1%
Chromium (Cr)		1.0E-03	10%	9.0E-04	2.00E-3	9.9E-05	1.99E-10	6.9E-09	8.1E-08	--		9.0E-05	44%
Copper (Cu)		1.4E-01	60%	5.6E-02	1.00E-3	6.2E-04	6.20E-10	2.2E-08	2.5E-07	--		4.5E-06	2%
Lead (Pb)		3.0E-04	10%	2.7E-04	1.00E-4	3.5E-06	3.54E-13	1.2E-11	1.4E-10	--		5.3E-07	0%
Manganese (Mn)		1.4E-01	50%	7.0E-02	1.00E-3	1.6E-03	1.59E-09	5.5E-08	6.5E-07	--		9.2E-06	5%
Mercury (Hg)		4.2E-05	40%	2.5E-05	1.00E-3	2.6E-07	2.62E-13	9.1E-12	1.1E-10	--		4.2E-06	2%
Silver (Ag)		2.3E-04		2.3E-04	6.00E-4	6.0E-06	3.60E-12	1.3E-10	1.5E-09	--		6.4E-06	3%
Nickel (Ni)		1.2E-02	60%	4.8E-03	2.00E-4	1.5E-05	3.05E-12	1.1E-10	1.2E-09	--		2.6E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	6.00E-4	4.8E-04	2.90E-10	1.0E-08	1.2E-07	--		1.2E-06	1%
												2.0E-04	

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

Year 1

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)				NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	1.26E-05	1.34E-04	1.8E-08	2.1E-07	--		2.9E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	6.74E-04	1.44E-03	4.5E-07	5.2E-06	--		1.7E-02	54%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	6.20E-04	4.96E-03	6.0E-10	7.0E-09	--		3.9E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	1.15E-06	6.13E-07	6.0E-10	7.0E-09	--		3.5E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.42E-05	3.79E-04	4.1E-08	4.7E-07	--		1.5E-03	5%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	2.88E-04	1.15E-04	1.5E-07	1.7E-06	--		1.9E-03	6%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.10E-03	4.49E-02	5.0E-06	5.8E-05	--		1.0E-03	3%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	4.45E-04	1.06E-03	3.0E-07	3.6E-06	--		2.8E-03	9%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.39E-02	1.86E-01	2.3E-05	2.7E-04	--		3.9E-03	12%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.93E-06	9.28E-05	9.2E-09	1.1E-07	--		3.0E-04	1%
Silver (Ag)		5.7E-03		5.7E-03	100%	5.82E-06	1.24E-04	1.4E-08	1.6E-07	--		2.8E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.47E-04	4.71E-04	1.1E-07	1.3E-06	--		2.7E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	4.46E-03	2.51E-01	2.5E-05	2.9E-04	--		2.9E-03	9%
									TOTAL			**3.2E-02**	

Year 2

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	2.6E-05	2.8E-04	3.7E-08	4.3E-07	--		6.0E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	1.5E-03	3.1E-03	9.6E-07	1.1E-05	--		3.7E-02	55%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.3E-03	1.0E-02	1.1E-09	1.3E-08	--		7.3E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	2.2E-06	1.2E-06	1.1E-09	1.3E-08	--		6.6E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.9E-05	7.6E-04	8.2E-08	9.5E-07	--		3.0E-03	4%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	5.3E-04	2.1E-04	2.7E-07	3.1E-06	--		3.5E-03	5%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	5.6E-03	1.2E-01	1.3E-05	1.6E-04	--		2.8E-03	4%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	8.8E-04	2.1E-03	6.0E-07	7.1E-06	--		5.6E-03	8%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	2.9E-02	3.9E-01	4.8E-05	5.6E-04	--		8.0E-03	12%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	3.9E-06	1.9E-04	1.8E-08	2.2E-07	--		6.0E-04	1%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.4E-05	2.9E-04	3.2E-08	3.8E-07	--		6.6E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.8E-04	8.8E-04	2.1E-07	2.4E-06	--		5.1E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	8.6E-03	4.8E-01	4.7E-05	5.5E-04	--		5.5E-03	8%
									TOTAL			**6.8E-02**	

Year 4

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	3.1E-05	3.3E-04	4.4E-08	5.1E-07	--		7.1E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	1.7E-03	3.7E-03	1.2E-06	1.3E-05	--		4.5E-02	55%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.5E-03	1.2E-02	1.3E-09	1.5E-08	--		8.6E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	2.6E-06	1.4E-06	1.3E-09	1.5E-08	--		7.7E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.4E-05	9.0E-04	9.6E-08	1.1E-06	--		3.5E-03	4%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	6.3E-04	2.5E-04	3.2E-07	3.7E-06	--		4.2E-03	5%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	6.9E-03	1.5E-01	1.7E-05	1.9E-04	--		3.4E-03	4%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	1.1E-03	2.5E-03	7.2E-07	8.5E-06	--		6.7E-03	8%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.4E-02	4.6E-01	5.7E-05	6.7E-04	--		9.5E-03	12%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	4.6E-06	2.2E-04	2.2E-08	2.5E-07	--		7.1E-04	1%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.6E-05	3.5E-04	3.9E-08	4.5E-07	--		7.9E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.3E-04	1.1E-03	2.5E-07	2.9E-06	--		6.1E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	1.0E-02	5.7E-01	5.6E-05	6.5E-04	--		6.5E-03	8%
									TOTAL			**8.1E-02**	

Year 6

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	2.9E-05	3.1E-04	4.2E-08	4.9E-07	--		6.8E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	1.7E-03	3.6E-03	1.1E-06	1.3E-05	--		4.3E-02	53%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.4E-03	1.2E-02	1.3E-09	1.5E-08	--		8.2E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	2.5E-06	1.3E-06	1.3E-09	1.5E-08	--		7.4E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.2E-05	8.6E-04	9.2E-08	1.1E-06	--		3.4E-03	4%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	6.1E-04	2.4E-04	3.1E-07	3.6E-06	--		4.0E-03	5%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	6.7E-03	1.4E-01	1.6E-05	1.9E-04	--		3.3E-03	4%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	1.0E-03	2.4E-03	6.9E-07	8.1E-06	--		6.4E-03	8%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.3E-02	4.4E-01	5.5E-05	6.4E-04	--		9.1E-03	11%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	4.4E-06	2.1E-04	2.1E-08	2.4E-07	--		6.8E-04	1%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.6E-05	3.3E-04	3.7E-08	4.3E-07	--		7.6E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.2E-04	1.0E-03	2.4E-07	2.8E-06	--		5.9E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	9.7E-03	5.4E-01	5.3E-05	6.2E-04	--		6.2E-03	8%
									TOTAL			**7.7E-02**	

Year 8

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	2.9E-05	3.1E-04	4.2E-08	4.9E-07	--		6.8E-04	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	1.7E-03	3.6E-03	1.1E-06	1.3E-05	--		4.3E-02	56%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.4E-03	1.1E-02	1.2E-09	1.5E-08	--		8.1E-08	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	2.4E-06	1.3E-06	1.2E-09	1.5E-08	--		7.3E-06	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.2E-05	8.5E-04	9.1E-08	1.1E-06	--		3.3E-03	4%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	6.0E-04	2.4E-04	3.0E-07	3.5E-06	--		3.9E-03	5%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	6.9E-03	1.5E-01	1.6E-05	1.9E-04	--		3.4E-03	4%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	1.0E-03	2.4E-03	6.9E-07	8.1E-06	--		6.4E-03	8%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.3E-02	4.4E-01	5.4E-05	6.4E-04	--		9.1E-03	12%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	4.3E-06	2.1E-04	2.1E-08	2.4E-07	--		6.7E-04	1%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.6E-05	3.4E-04	3.8E-08	4.4E-07	--		7.7E-05	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.1E-04	1.0E-03	2.4E-07	2.8E-06	--		5.8E-04	1%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	9.5E-03	5.4E-01	5.2E-05	6.1E-04	--		6.1E-03	8%
									TOTAL			**7.7E-02**	

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

Year 1

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	3.02E-07	2.1E-11	2.4E-10	--		3.4E-07	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	2.98E-05	2.0E-09	2.4E-08	--		8.0E-05	82%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	6.54E-08	4.5E-12	5.2E-11	--		2.9E-10	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	6.54E-08	4.5E-12	5.2E-11	--		2.6E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	8.97E-08	6.2E-12	7.2E-11	--		2.2E-07	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.68E-06	1.2E-10	1.3E-09	--		1.5E-06	2%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	5.05E-05	3.5E-09	4.0E-08	--		7.2E-07	1%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	1.12E-05	7.7E-10	9.0E-09	--		7.1E-06	7%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.35E-04	2.3E-08	2.7E-07	--		3.8E-06	4%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	9.77E-07	6.7E-11	7.8E-10	--		2.2E-06	2%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.40E-07	9.6E-12	1.1E-10	--		2.0E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.86E-06	1.3E-10	1.5E-09	--		3.1E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	1.07E-04	7.3E-09	8.6E-08	--		8.6E-07	1%
								TOTAL			**9.7E-05**	

Year 2

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	6.2E-07	4.3E-11	5.0E-10	--		6.9E-07	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	6.4E-05	4.4E-09	5.1E-08	--		1.7E-04	83%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	3.0E-05	2.1E-09	2.4E-08	--		1.3E-07	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	1.2E-07	8.5E-12	1.0E-10	--		5.0E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.8E-07	1.2E-11	1.4E-10	--		4.5E-07	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	3.1E-06	2.1E-10	2.5E-09	--		2.7E-06	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.4E-04	9.3E-09	1.1E-07	--		1.9E-06	1%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	2.2E-05	1.5E-09	1.8E-08	--		1.4E-05	7%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	7.0E-04	4.8E-08	5.6E-07	--		7.9E-06	4%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	2.0E-06	1.3E-10	1.6E-09	--		4.3E-06	2%
Silver (Ag)		5.7E-03		5.7E-03	100%	3.3E-07	2.2E-11	2.6E-10	--		4.6E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	3.5E-06	2.4E-10	2.8E-09	--		5.8E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	2.1E-04	1.4E-08	1.7E-07	--		1.7E-06	1%
								TOTAL			**2.1E-04**	

Year 4

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	7.4E-07	5.1E-11	5.9E-10	--		8.2E-07	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	7.7E-05	5.3E-09	6.2E-08	--		2.1E-04	83%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	3.6E-05	2.5E-09	2.9E-08	--		1.6E-07	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	1.5E-07	1.0E-11	1.2E-10	--		5.8E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.1E-07	1.5E-11	1.7E-10	--		5.3E-07	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	3.7E-06	2.5E-10	3.0E-09	--		3.3E-06	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.7E-04	1.1E-08	1.3E-07	--		2.4E-06	1%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	2.7E-05	1.8E-09	2.1E-08	--		1.7E-05	7%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	8.2E-04	5.6E-08	6.6E-07	--		9.4E-06	4%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	2.3E-06	1.6E-10	1.8E-09	--		5.1E-06	2%
Silver (Ag)		5.7E-03		5.7E-03	100%	3.9E-07	2.7E-11	3.1E-10	--		5.5E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.2E-06	2.9E-10	3.4E-09	--		7.0E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	2.4E-04	1.7E-08	1.9E-07	--		1.9E-06	1%
								TOTAL			**2.5E-04**	

Year 6

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	7.1E-07	4.8E-11	5.7E-10	--		7.9E-07	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	7.4E-05	5.1E-09	5.9E-08	--		2.0E-04	80%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	3.5E-05	2.4E-09	2.8E-08	--		1.5E-07	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	1.4E-07	9.6E-12	1.1E-10	--		5.6E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.0E-07	1.4E-11	1.6E-10	--		5.1E-07	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	3.5E-06	2.4E-10	2.8E-09	--		3.1E-06	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.6E-04	1.1E-08	1.3E-07	--		2.3E-06	1%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	2.6E-05	1.8E-09	2.1E-08	--		1.6E-05	7%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	7.9E-04	5.4E-08	6.3E-07	--		9.0E-06	4%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	2.2E-06	1.5E-10	1.8E-09	--		4.9E-06	2%
Silver (Ag)		5.7E-03		5.7E-03	100%	3.8E-07	2.6E-11	3.0E-10	--		5.3E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.0E-06	2.8E-10	3.2E-09	--		6.7E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	2.3E-04	1.6E-08	1.9E-07	--		1.9E-06	1%
								TOTAL			**2.4E-04**	

Year 8

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	7.0E-07	4.8E-11	5.6E-10	--		7.8E-07	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	7.4E-05	5.1E-09	5.9E-08	--		2.0E-04	83%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	3.4E-05	2.4E-09	2.8E-08	--		1.5E-07	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	1.4E-07	9.4E-12	1.1E-10	--		5.5E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	2.0E-07	1.4E-11	1.6E-10	--		5.1E-07	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	3.5E-06	2.4E-10	2.8E-09	--		3.1E-06	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	1.7E-04	1.1E-08	1.3E-07	--		2.4E-06	1%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	2.6E-05	1.8E-09	2.0E-08	--		1.6E-05	7%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	7.9E-04	5.4E-08	6.3E-07	--		9.0E-06	4%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	2.2E-06	1.5E-10	1.8E-09	--		4.9E-06	2%
Silver (Ag)		5.7E-03		5.7E-03	100%	3.8E-07	2.6E-11	3.0E-10	--		5.3E-08	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.0E-06	2.7E-10	3.2E-09	--		6.6E-07	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	2.3E-04	1.6E-08	1.8E-07	--		1.8E-06	1%
								TOTAL			**2.4E-04**	

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

Year 1

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	1.3E-06	2.2E-10	2.6E-09	--		3.6E-06	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	1.4E-04	2.4E-08	2.8E-07	--		9.4E-04	36%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	9.8E-06	1.7E-09	1.9E-08	--		1.1E-07	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	3.6E-08	6.2E-12	7.2E-11	--		3.6E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.0E-07	5.1E-11	5.9E-10	--		1.8E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	2.8E-04	4.7E-08	5.5E-07	--		6.1E-04	24%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.2E-03	3.8E-07	4.4E-06	--		7.8E-05	3%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	1.4E-05	2.4E-09	2.8E-08	--		2.2E-05	1%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	5.9E-04	1.0E-07	1.2E-06	--		1.7E-05	1%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	8.1E-08	1.4E-11	1.6E-10	--		4.5E-07	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.8E-06	3.1E-10	3.6E-09	--		6.4E-07	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	4.6E-06	7.9E-10	9.2E-09	--		1.9E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	4.7E-02	8.0E-06	9.3E-05	--		9.3E-04	36%
TOTAL											**2.6E-03**	

Year 2

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	2.7E-06	4.6E-10	5.4E-09	--		7.5E-06	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	3.1E-04	5.2E-08	6.1E-07	--		2.0E-03	39%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	2.0E-05	3.4E-09	3.9E-08	--		2.2E-07	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	6.9E-08	1.2E-11	1.4E-10	--		6.8E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	6.0E-07	1.0E-10	1.2E-09	--		3.7E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	5.1E-04	8.7E-08	1.0E-06	--		1.1E-03	22%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	5.9E-03	1.0E-06	1.2E-05	--		2.1E-04	4%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	2.8E-05	4.7E-09	5.5E-08	--		4.4E-05	1%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.2E-03	2.1E-07	2.4E-06	--		3.4E-05	1%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.6E-07	2.8E-11	3.2E-10	--		8.9E-07	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	4.3E-06	7.2E-10	8.5E-09	--		1.5E-06	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	8.7E-06	1.5E-09	1.7E-08	--		3.6E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	9.0E-02	1.5E-05	1.8E-04	--		1.8E-03	34%
								TOTAL			**5.2E-03**	

Year 4

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	3.2E-06	5.5E-10	6.4E-09	--		8.9E-06	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	3.7E-04	6.2E-08	7.3E-07	--		2.4E-03	39%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	2.4E-05	4.0E-09	4.7E-08	--		2.6E-07	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	8.1E-08	1.4E-11	1.6E-10	--		8.0E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	7.1E-07	1.2E-10	1.4E-09	--		4.4E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	6.1E-04	1.0E-07	1.2E-06	--		1.4E-03	22%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	7.3E-03	1.2E-06	1.4E-05	--		2.6E-04	4%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	3.3E-05	5.7E-09	6.6E-08	--		5.2E-05	1%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.4E-03	2.4E-07	2.9E-06	--		4.1E-05	1%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.9E-07	3.3E-11	3.8E-10	--		1.1E-06	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	5.1E-06	8.7E-10	1.0E-08	--		1.8E-06	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.0E-05	1.8E-09	2.1E-08	--		4.3E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	1.1E-01	1.8E-05	2.1E-04	--		2.1E-03	34%
								TOTAL			**6.2E-03**	

Year 6

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	3.1E-06	5.3E-10	6.1E-09	--		8.5E-06	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	3.5E-04	6.0E-08	7.0E-07	--		2.3E-03	37%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	2.3E-05	3.9E-09	4.5E-08	--		2.5E-07	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	7.7E-08	1.3E-11	1.5E-10	--		7.7E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	6.8E-07	1.2E-10	1.3E-09	--		4.2E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	5.9E-04	1.0E-07	1.2E-06	--		1.3E-03	21%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	7.0E-03	1.2E-06	1.4E-05	--		2.5E-04	4%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	3.2E-05	5.4E-09	6.3E-08	--		5.0E-05	1%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.4E-03	2.3E-07	2.7E-06	--		3.9E-05	1%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.8E-07	3.1E-11	3.6E-10	--		1.0E-06	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	4.9E-06	8.4E-10	9.8E-09	--		1.7E-06	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.0E-05	1.7E-09	2.0E-08	--		4.1E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	1.0E-01	1.7E-05	2.0E-04	--		2.0E-03	32%
								TOTAL			**6.0E-03**	

Year 8

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	3.1E-06	5.2E-10	6.1E-09	--		8.5E-06	0%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	3.5E-04	6.0E-08	7.0E-07	--		2.3E-03	39%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	2.3E-05	3.8E-09	4.5E-08	--		2.5E-07	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	7.6E-08	1.3E-11	1.5E-10	--		7.5E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	6.7E-07	1.1E-10	1.3E-09	--		4.2E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	5.8E-04	9.8E-08	1.1E-06	--		1.3E-03	21%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	7.2E-03	1.2E-06	1.4E-05	--		2.6E-04	4%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	3.2E-05	5.4E-09	6.3E-08	--		5.0E-05	1%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.4E-03	2.3E-07	2.7E-06	--		3.9E-05	1%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	1.8E-07	3.1E-11	3.6E-10	--		1.0E-06	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	5.0E-06	8.5E-10	9.9E-09	--		1.7E-06	0%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	9.9E-06	1.7E-09	2.0E-08	--		4.1E-06	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	1.0E-01	1.7E-05	2.0E-04	--		2.0E-03	33%
								TOTAL			**5.9E-03**	

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

Year 1

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
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	3.2E-07	2.0E-09	2.4E-08	--		3.3E-05	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	8.7E-06	5.4E-08	6.3E-07	--		2.1E-03	38%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	5.6E-05	3.5E-07	4.1E-06	--		2.3E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	2.7E-10	1.7E-12	1.9E-11	--		9.7E-09	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	1.8E-08	1.1E-10	1.3E-09	--		4.2E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	6.7E-07	4.2E-09	4.9E-08	--		5.4E-05	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	8.1E-04	5.1E-06	5.9E-05	--		1.1E-03	19%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	6.9E-06	4.3E-08	5.0E-07	--		4.0E-04	7%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	1.3E-03	7.9E-06	9.2E-05	--		1.3E-03	24%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	3.5E-08	2.2E-10	2.5E-09	--		7.1E-06	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	4.0E-05	2.5E-07	2.9E-06	--		5.2E-04	9%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	1.1E-06	7.1E-09	8.3E-08	--		1.7E-05	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	3.1E-09	1.9E-11	2.3E-10	--		2.3E-09	0%
TOTAL											**5.5E-03**	

Year 2

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
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	6.6E-07	4.2E-09	4.8E-08	--		6.7E-05	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	1.9E-05	1.2E-07	1.4E-06	--		4.6E-03	37%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.1E-04	7.1E-07	8.3E-06	--		4.6E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	5.1E-10	3.2E-12	3.7E-11	--		1.8E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	3.7E-08	2.3E-10	2.7E-09	--		8.4E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.2E-06	7.7E-09	9.0E-08	--		1.0E-04	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.2E-03	1.4E-05	1.6E-04	--		2.8E-03	23%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	1.4E-05	8.5E-08	9.9E-07	--		7.9E-04	6%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	2.6E-03	1.6E-05	1.9E-04	--		2.7E-03	22%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	6.9E-08	4.4E-10	5.1E-09	--		1.4E-05	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	9.4E-05	5.9E-07	6.9E-06	--		1.2E-03	10%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.1E-06	1.3E-08	1.6E-07	--		3.2E-05	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	6.0E-09	3.7E-11	4.4E-10	--		4.4E-09	0%
								TOTAL			**1.2E-02**	

Year 4

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
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	7.9E-07	4.9E-09	5.8E-08	--		8.0E-05	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	2.2E-05	1.4E-07	1.6E-06	--		5.4E-03	37%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.4E-04	8.5E-07	9.9E-06	--		5.5E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	5.9E-10	3.7E-12	4.3E-11	--		2.2E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	4.3E-08	2.7E-10	3.2E-09	--		9.9E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.5E-06	9.2E-09	1.1E-07	--		1.2E-04	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.7E-03	1.7E-05	2.0E-04	--		3.5E-03	23%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	1.6E-05	1.0E-07	1.2E-06	--		9.5E-04	6%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.1E-03	1.9E-05	2.3E-04	--		3.2E-03	22%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	8.2E-08	5.1E-10	6.0E-09	--		1.7E-05	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.1E-04	7.1E-07	8.3E-06	--		1.4E-03	10%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.6E-06	1.6E-08	1.9E-07	--		3.9E-05	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	7.0E-09	4.4E-11	5.1E-10	--		5.1E-09	0%
								TOTAL			**1.5E-02**	

Year 6

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
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	7.6E-07	4.7E-09	5.5E-08	--		7.7E-05	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	2.1E-05	1.3E-07	1.6E-06	--		5.2E-03	35%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.3E-04	8.1E-07	9.5E-06	--		5.3E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	5.7E-10	3.6E-12	4.1E-11	--		2.1E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	4.1E-08	2.6E-10	3.0E-09	--		9.5E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.4E-06	8.8E-09	1.0E-07	--		1.1E-04	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.6E-03	1.6E-05	1.9E-04	--		3.3E-03	23%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	1.6E-05	9.8E-08	1.1E-06	--		9.1E-04	6%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	3.0E-03	1.9E-05	2.2E-04	--		3.1E-03	21%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	7.9E-08	4.9E-10	5.8E-09	--		1.6E-05	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.1E-04	6.8E-07	7.9E-06	--		1.4E-03	9%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.4E-06	1.5E-08	1.8E-07	--		3.7E-05	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	6.7E-09	4.2E-11	4.9E-10	--		4.9E-09	0%
								TOTAL			**1.4E-02**	

Year 8

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
Antimony (Sb)		9.0E-04	20%	7.2E-04	100%	7.5E-07	4.7E-09	5.5E-08	--		7.6E-05	1%
Arsenic (As)		6.0E-04	50%	3.0E-04	100%	2.1E-05	1.3E-07	1.6E-06	--		5.2E-03	36%
Barium (Ba)		2.0E-01	10%	1.8E-01	100%	1.3E-04	8.1E-07	9.4E-06	--		5.2E-05	0%
Beryllium (Be)		2.0E-03		2.0E-03	100%	5.5E-10	3.5E-12	4.1E-11	--		2.0E-08	0%
Cadmium (Cd)		8.0E-04	60%	3.2E-04	100%	4.1E-08	2.6E-10	3.0E-09	--		9.4E-06	0%
Chromium (Cr)		1.0E-03	10%	9.0E-04	100%	1.4E-06	8.7E-09	1.0E-07	--		1.1E-04	1%
Copper (Cu)		1.4E-01	60%	5.6E-02	100%	2.7E-03	1.7E-05	1.9E-04	--		3.5E-03	24%
Lead (Pb)		1.4E-03	10%	1.3E-03	50%	1.6E-05	9.7E-08	1.1E-06	--		9.0E-04	6%
Manganese (Mn)		1.4E-01	50%	7.0E-02	100%	2.9E-03	1.8E-05	2.1E-04	--		3.1E-03	21%
Mercury (Hg)		6.0E-04	40%	3.6E-04	100%	7.8E-08	4.9E-10	5.7E-09	--		1.6E-05	0%
Silver (Ag)		5.7E-03		5.7E-03	100%	1.1E-04	6.9E-07	8.0E-06	--		1.4E-03	10%
Nickel (Ni)		1.2E-02	60%	4.8E-03	100%	2.4E-06	1.5E-08	1.8E-07	--		3.7E-05	0%
Zinc (Zn)		5.0E-01	80%	1.0E-01	100%	6.6E-09	4.1E-11	4.8E-10	--		4.8E-09	0%
								TOTAL			**1.4E-02**	