Maryvale Energy from Waste Plant: Health Impact Assessment

Prepared for: Australian Paper and Jacobs

27 September 2018

Document History and Status

Report Reference	J/18/EWR001
Revision	B - Final
Date	27 September 2018
Previous Revisions	A – Draft issued on 20 September 2018

Limitations

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

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

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

This report was prepared in July to September 2018 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.

Table of Contents

Appendices

Glossary of Terms and Abbreviations

Term	Definition
ABS	Australian Bureau of Statistics
Acute exposure	Contact with a substance that occurs once or for only a short time (up to 14 days)
Absorption	The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs
Adverse health effect	A change in body function or cell structure that might lead to disease or health problems
ATSDR	Agency for Toxic Substances and Disease Register
AAQ	Ambient air quality
ANZECC	Australia and New Zealand Environment and Conservation Council
Background level	An average or expected amount of a substance or material in a specific environment, or typical amounts of substances that occur naturally in an environment.
BaP	Benzo(a)pyrene
Biodegradation	Decomposition or breakdown of a substance through the action of micro-organisms (such as bacteria or fungi) or other natural physical processes (such as sunlight).
Body burden	The total amount of a substance in the body. Some substances build up in the body because they are stored in fat or bone or because they leave the body very slowly.
Carcinogen	A substance that causes cancer.
CCME	Canadian Council of Ministers of the Environment
Chronic exposure	Contact with a substance or stressor that occurs over a long time (more than one year) [compare with acute exposure and intermediate duration exposure].
CO	Carbon monoxide
dB(A)	Decibels (A-weighted)
DECCW	NSW Department of Environment, Climate Change and Water
DEFRA	Department for Environment, Food & Rural Affairs
DEH	Australian Department of Environment and Heritage
Detection limit	The lowest concentration of a substance that can reliably be distinguished from a zero concentration.
Dose	The amount of a substance to which a person is exposed over some time period. Dose is a measurement of exposure. Dose is often expressed as milligram (amount) per kilogram (a measure of body weight) per day (a measure of time) when people eat or drink contaminated water, food, or soil. In general, the greater the dose, the greater the likelihood of an effect. An ‘exposure dose’ is how much of a substance is encountered in the environment. An ‘absorbed dose’ is the amount of a substance that actually got into the body through the eyes, skin, stomach, intestines, or lungs.
EIS	Environmental Impact Statement
Exposure	Contact with a substance by swallowing, breathing, or touching the skin or eyes. Also includes contact with a stressor such as noise or vibration. Exposure may be short term [acute exposure], of intermediate duration, or long term [chronic exposure].
Exposure assessment	The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.

Term	Definition
Exposure pathway	The route a substance takes from its source (where it began) to its endpoint (where it ends), and how people can come into contact with (or get exposed) to it. An exposure pathway has five parts: a source of contamination (such as chemical substance leakage into the subsurface); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.
Genotoxic carcinogen	These are carcinogens that have the potential to result in genetic (DNA) damage (gene mutation, gene amplification, chromosomal rearrangement). Where this occurs, the damage may be sufficient to result in the initiation of cancer at some time during a lifetime.
Guideline value	Guideline value is a concentration in soil, sediment, water, biota or air (established by relevant regulatory authorities such as the NSW Department of Environment and Conservation (DEC) or institutions such as the National Health and Medical Research Council (NHMRC), Australia and New Zealand Environment and Conservation Council (ANZECC) and World Health Organization (WHO)), that is used to identify conditions below which no adverse effects, nuisance or indirect health effects are expected. The derivation of a guideline value utilises relevant studies on animals or humans and relevant factors to account for inter and intra-species variations and uncertainty factors. Separate guidelines may be identified for protection of human health and the environment. Dependent on the source, guidelines would have different names, such as investigation level, trigger value and ambient guideline.
HIA	Health impact assessment
HI	Hazard Index
IARC	International Agency for Research on Cancer
Inhalation	The act of breathing. A hazardous substance can enter the body this way [see route of exposure].
Intermediate exposure Duration	Contact with a substance that occurs for more than 14 days and less than a year [compare with acute exposure and chronic exposure].
LGA	Local Government Area
LOR	Limit of Reporting
Metabolism	The conversion or breakdown of a substance from one form to another by a living organism.
NCAs	Noise catchment areas
NCG	Noise Criteria Guideline (various, as referenced in the report)
NEPC	National Environment Protection Council
NEPM	National Environment Protection Measure
NHMRC	National Health and Medical Research Council
NO_2	Nitrogen dioxide
NOx	Nitrogen oxides
NSW	New South Wales
NSW EPA	NSW Environment Protection Authority
OEH	NSW Office of Environment and Heritage
OEHHA	Office of Environmental Health Hazard Assessment, California Environment Protection Agency (Cal EPA)
PAH	Polycyclic aromatic hydrocarbon
PM	Particulate matter
$PM_{2.5}$	Particulate matter of aerodynamic diameter 2.5 µm and less
PM_{10}	Particulate matter of aerodynamic diameter 10 µm and less

Term	Definition
Point of exposure	The place where someone can come into contact with a substance present in the environment [see exposure pathway].
Population	A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).
Receptor population	People who could come into contact with hazardous substances [see exposure pathway].
Risk	The probability that something would cause injury or harm.
Roads and Maritime	NSW Roads and Maritime Services
Route of exposure	The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].
SEIFA	Socio-Economic Index for Areas
SO_2	Sulfur dioxide
TCEQ	Texas Commission on Environmental Quality
TEQ	Toxicity equivalent
Toxicity	The degree of danger posed by a substance to human, animal or plant life.
Toxicity data	Characterisation or quantitative value estimated (by recognised authorities) for each individual chemical substance for relevant exposure pathway (inhalation, oral or dermal), with special emphasis on dose-response characteristics. The data are based on based on available toxicity studies relevant to humans and/or animals and relevant safety factors.
Toxicological profile	An assessment that examines, summarises, and interprets information about a hazardous substance to determine harmful levels of exposure and associated health effects. A toxicological profile also identifies significant gaps in knowledge on the substance and describes areas where further research is needed.
Toxicology	The study of the harmful effects of substances on humans or animals.
TSP	Total suspended particulates
UK	United Kingdom
US	United States
USEPA	United States Environmental Protection Agency
VOC	Volatile organic compound
WHO	World Health Organization
$\mu g/m^3$	Micrograms per cubic metre

Executive Summary

Introduction

The project, proposed by Paper Australia Pty Ltd also known as Australian Paper (AP), involves the construction and operation of an energy from waste (EfW) plant on its existing pulp and paper mill site in Maryvale, located between Tanjil East and Traralgon West roads near the townships of Traralgon and Morwell, Victoria (the '**site**') (**Figure 1**).

The proposed facility will process an estimated 650,000 tonnes per annum of municipal solid waste and commercial and industrial waste sourced from the greater Melbourne Metropolitan area along with the local Gippsland region. Waste will be transported to the site via rail and road in sealed 40 foot containers, with waste from the Gippsland region delivered via refuse collection vehicles. The plant will provide both steam and power to the existing Maryvale Mill operations of the order of 30 Megawatts electricity (MWe) per annum and 130 tonnes per hour of high pressure steam. Any energy created in excess of these needs, will be placed into the national electricity market.

This Health Impact Assessment (HIA) has been developed for Australian Paper by identifying and estimating the health impacts of the proposed project on the health of the surrounding (local and regional) community.

Assessment Approach

The HIA assessment has been conducted as a desktop assessment in accordance with national guidelines available from the Centre for Health Equity Training, Research and Evaluation (CHETRE) (Harris 2007) and enHealth (enHealth 2001, 2012a). The HIA has been undertaken on the basis of the information provided in the *Maryvale Energy from Waste Plant – Works Approval Application, Jacobs -23 April 2018.*

The conduct of an HIA is intended to provide a structured, solution-focused and action-oriented approach to maximising the positive and minimising the negative health impacts of a proposed project. This HIA has therefore been conducted to identify and address potential social, economic and environmental impacts of the project on health and provide recommendations to enhance positive impacts and mitigate negative impacts.

Outcomes of the HIA

The HIA has considered the operation of the proposed project and potential impacts to the health of the off-site community. The assessment has considered a range of issues that have the potential to affect the health of the community (either positive or negative), which relate to changes to air quality, odour, noise, water, traffic, hazardous materials, economic and social environment.

Based on the assessment undertaken, the project is associated with some benefits to the community, particularly in relation to employment. Where negative impacts have been identified, these are considered to be negligible in terms of community health.

Table ES-1 presents a summary of the HIA undertaken.

Table ES-1: Summary of HIA outcomes and enhancement/mitigation measures

Health Aspect/Issue	Reference in HIA	Potential Health Impacts Considered	Impact Identified (positive or negative and significance)	Types of measures that could be implemented to enhance positive impacts or mitigate negative impacts
Air quality – Inhalation exposures	Section 5.4	Range of health effects associated with exposure to pollutants released to air from the proposed facility	**All exposures: Negative but negligible** More specifically: ▪ No acute risk issues of concern ▪ No chronic risk issues of concern Particulate exposures are negligible and essentially representative of zero risk ▪ Incremental carcinogenic risks are negligible and essentially representative of zero risk	The proper operation and maintenance, and monitoring, of the pollution control/flue gas equipment.
Air quality – Multiple pathway exposures	Section 5.5	Range of health effects associated with exposure to pollutants released to air from the proposed facility, that may then deposit and accumulate in soil, homegrown fruit and vegetables and other farm produce (eggs, beef and milk)	**All exposures: Negative but negligible** More specifically: ▪ No chronic risk issues of concern for multiple pathway exposures ▪ All calculated risks for individual exposure pathways are negligible and essentially representative of zero risk ▪ All calculated risks for combined multiple pathway exposures are negligible and essentially representative of zero risk	The proper operation and maintenance, and monitoring, of the pollution control/flue gas equipment.
Odour	Section 5.6	Annoyance, stress, anxiety	**Not significant and negligible**	The proper operation of the tipping hall as proposed to ensure fugitive odour emissions are effectively managed.
Noise	Section 6	Sleep disturbance, annoyance, children's school performance and cardiovascular health	**Modelled noise impacts: negligible potential for health impacts**	Additional assessment of the project detailed design is required, and application of appropriate and reasonable mitigation measures is required so as not to increase noise levels at the nearest sensitive receivers from current levels.
Economic Environment	Section 7	Reduction in anxiety, stress and feelings of insecurity	**Positive improvements in health and wellbeing**	The identified positive outcomes in the local community can be enhanced by encouraging employment of people who live within the local community

Health Aspect/Issue	Reference in HIA	Potential Health Impacts Considered	Impact Identified (positive or negative and significance)	Types of measures that could be implemented to enhance positive impacts or mitigate negative impacts
Traffic and transport	Section 7	Injury or death, stress and anxiety.	**Negative but minimal**	Details to be determined at the detailed design phase of the project
Discovery and disposal of hazardous waste	Section 7	Possible injury if incorrectly disposed of	**Negative but minimal**	Further development of the feedstock delivery protocol into an operational management plan to address the discovery and proper disposal of this material
Community and social	Section 7	Wellbeing, changes in levels of stress and anxiety	**Positive outcomes** enhancing feelings of wellbeing for aspects such as sustainability **Negative outcomes** for potential changes to amenity and community feelings of control related to perceived risks rather than actual risks	These health impacts relate to community perceptions and trust. It is therefore important that the positive impacts associated with the project are enhanced within the local community and community consultation is continued and uses a range of techniques that are tailored to the various sub-populations that have particular areas of concern or particular characteristics that make normal methods of communication less effective. It is important that an effective communication/ community consultation program is maintained throughout the construction, commissioning and operational phases of the project.

Section 1. Introduction

1.1 Background

The project, proposed by Paper Australia Pty Ltd also known as Australian Paper (AP), involves the construction and operation of an energy from waste (EfW) plant on its existing pulp and paper mill site in Maryvale, located between Tanjil East and Traralgon West roads near the townships of Traralgon and Morwell, Victoria (the '**site**') (**Figure 1.1**).

The proposed facility will process an estimated 650,000 tonnes per annum of municipal solid waste and commercial and industrial waste sourced from the greater Melbourne Metropolitan area along with the local Gippsland region. Waste will be transported to the site via rail and road in sealed 40 foot containers, with waste from the Gippsland region delivered via refuse collection vehicles. The plant will provide both steam and power to the existing Maryvale Mill operations of the order of 30 Megawatts electric (MWe) per annum and 130 tonnes per hour of high pressure steam. Any energy created in excess of these needs will be placed into the national electricity market.

Works approval is being sought for the facility development which includes (**Figure 1.2**):

- Weighbridges and gatehouse
- Energy from waste facility building
- Condenser, turbine and generator
- Road infrastructure
- Car park and hard stand area.

Legend

- Population centre
- Maryvale Mill
- Project Area
- LGA Boundary

Figure 1.1: Site location

RO097400
GDA 1994 MGA Zone 55

0 1 2
Kilometers

DATA SOURCES
© Commonwealth of Australia (Geoscience Australia) 2006 Geodata Topo 250k Series 3; Vicmap Data © State of Victoria 2017; Department of Environment, Land, Water and Planning 23/04/2017; Jacobs 2017DATA SOURCES © ESRI BaseMap

Australian Paper

JACOBS

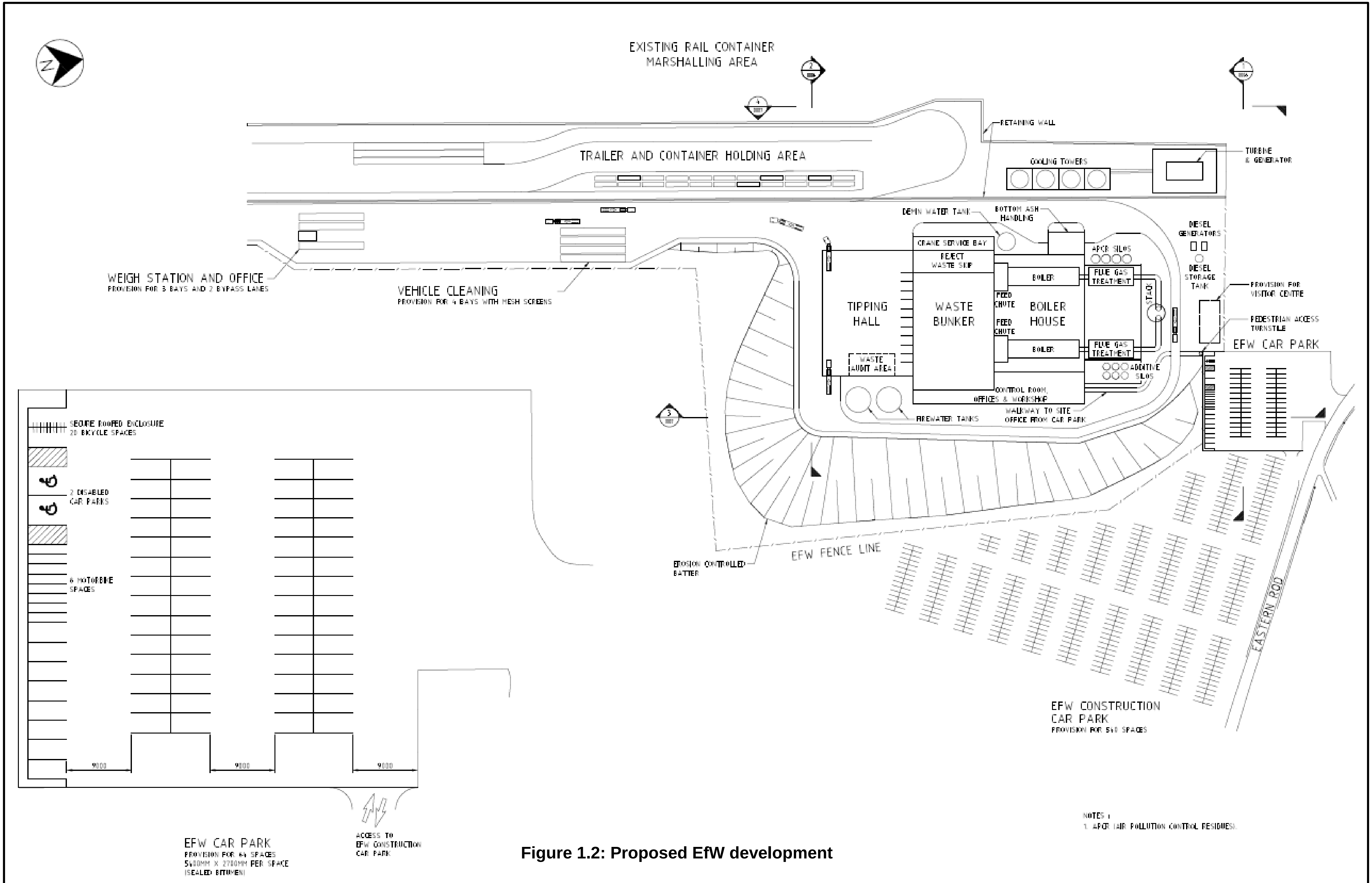


Figure 1.2: Proposed EfW development

1.2 Objectives

This Health Impact Assessment (HIA) has been developed for Australian Paper with the aim of identifying and estimating the health impacts (both positive and negative) of the project within the surrounding community, as specified in the Approach and scope of works (**Section 1.3**).

1.3 Approach and scope of works

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, 2001. Health Impact Assessment Guidelines (enHealth 2001);
- enHealth, 2012. Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a).

The above guidance requires the consideration of impacts that relate to a wider definition of health and well-being within the community. Health and health inequalities are affected by a wide range of factors, as illustrated below. These factors may be affected by a specific project in different ways. In some cases, the changes will result in negative impacts on health (and hence the HIA needs to determine what these impacts are and how they can be minimised) or positive impacts or benefits (and it is important that the HIA identify these and determine if these benefits can be enhanced).

Figure 1.4: Wider determinants of health, as presented by Harris et al (2007)

In accordance with this guidance the HIA has been undertaken as a desk-top assessment, based on information available (refer to **Section 1.5**). The HIA has evaluated positive and negative impacts from predicted air, noise and water emissions, increased transport, social and economic consequences. These predicted impacts have been sourced from the *Maryvale Energy from Waste Plant – Works Approval Application, Jacobs -23 April 2018.*

1.4 Definitions

For the conduct of the HIA the following definitions are relevant and should be considered when reading this report.

Health:

The World Health Organisation defines health as "*a (dynamic) state of complete physical, mental and social wellbeing and not merely the absence of disease or infirmity*".

Hence the assessment of health should include both the traditional/medical definition that focuses on illness and disease as well as the more broad social definition that includes the general health and wellbeing of a population.

Health Hazard:

These are aspects of a Project, or specific activities that present a hazard or source of negative risk to health or well-being.

In relation to the HIA these hazards may be associated with specific aspects of the proposed development/construction or operational activities, incidents or circumstances that have the potential to directly affect health. In addition, some activities may have a flow-on effect that results in some effect on health. Hence health hazards may be identified on the basis of the potential for both direct and indirect effects on health.

Health Outcomes:

These are the effects of the activity on health. These outcomes can be negative (such as injury, disease or disadvantage), or positive (such as good quality of life, physical and mental wellbeing, reduction in injury, diseases or disadvantage).

It is noted that where health effects are considered these are also associated with a time or duration with some effects being experienced for a short period of time (acute) and other for a long period of time (chronic). The terminology relevant to acute and chronic effects is most often applied to the assessment of negative/adverse effects as these are typically the focus of technical evaluations of various aspects of the project.

Likelihood:

This refers to how likely it is that an effect or health outcome will be experienced. It is often referred to as the probability of an impact occurring.

Risk:

This is the chance of something happening that will have an impact on objectives. In relation to the proposed project and the conduct of the HIA, the concept of risk more specifically relates to the chance that some aspect of the project will result in a reduction or improvement in the health and/or well-being of the local community. The assessment of risk has been undertaken on a quantitative basis for air, water and noise emissions and a qualitative basis for all other impacts. This is in line with the methods and levels of evidence currently available to assess risk.

***Equity*:**

Equity relates to the potential for the project to lead to impacts that are differentially distributed in the surrounding population. Population groups may be advantaged or disadvantaged based on age, gender, socioeconomic status, geographic location, cultural background, aboriginality, and current health status and existing disability.

1.5 Available information

In relation to the proposed project, and potential for impacts within the local community, this HIA has been developed on the basis of information provided within the chapters of the following report:

- Maryvale Energy from Waste Plant – Works Approval Application, Jacobs – 23 April 2018
- Additional outputs from air dispersion modelling (conducted for the Works Approval Application).

Section 2. Project description

2.1 Site description and location

The project, proposed by Australian Paper (AP), involves the construction and operation of an EfW plant on its existing pulp and paper mill site located between Tanjil East Road and Traralgon West Road near the townships of Traralgon and Morwell, Victoria (the '**site**'). The site is located approximately 7 kilometres to west of Traralgon and 7 kilometres to the north east of Morwell (**Figures 1.1 and 1.2**). The Mill is situated in the centre of the Latrobe Valley, adjacent to the La Trobe River.

The site is surrounded by large industrial premises including several open cut brown coal mines and associated power stations, water treatment plants, quarries, a dairy processing facility and numerous light industrial premises. The site is located within a planning zone designated for industrial activities (Industrial 2 Zone, IN2Z) and is surrounded by farming zones and special use zones (predominantly for coal mining and power generation activities) (**Figure 2.1**).

The nearest residents to the north, south, east and west of the site have been identified as shown in **Figure 2.2** (marked as locations 'North', 'East', 'South', 'West' with a yellow cross). Of these residents, the resident to the south is the closest at approximately 2 kilometres from the site, while the western resident is approximately 2.5 kilometres and the north and east residents are over 3 kilometres from the site.

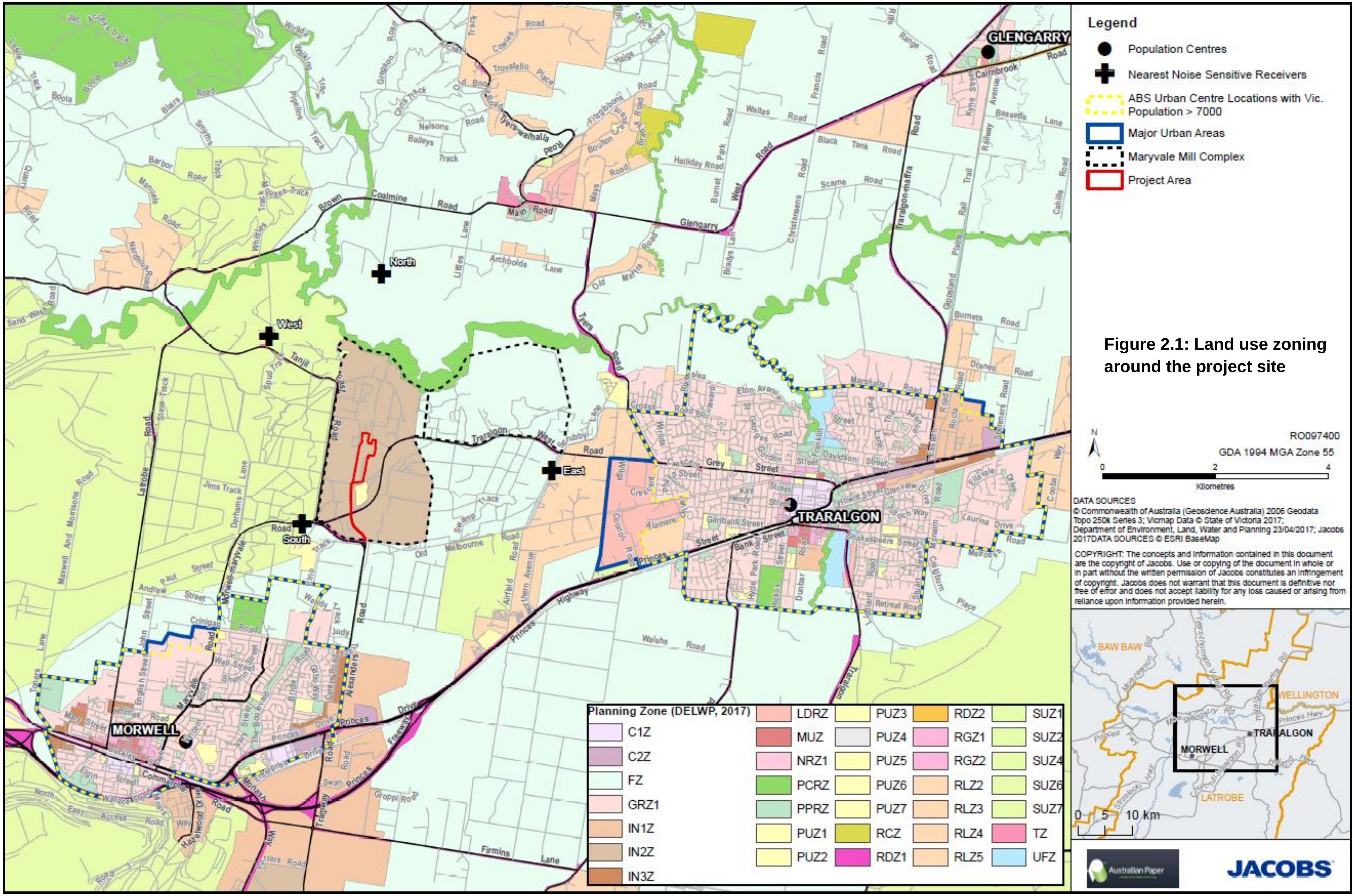
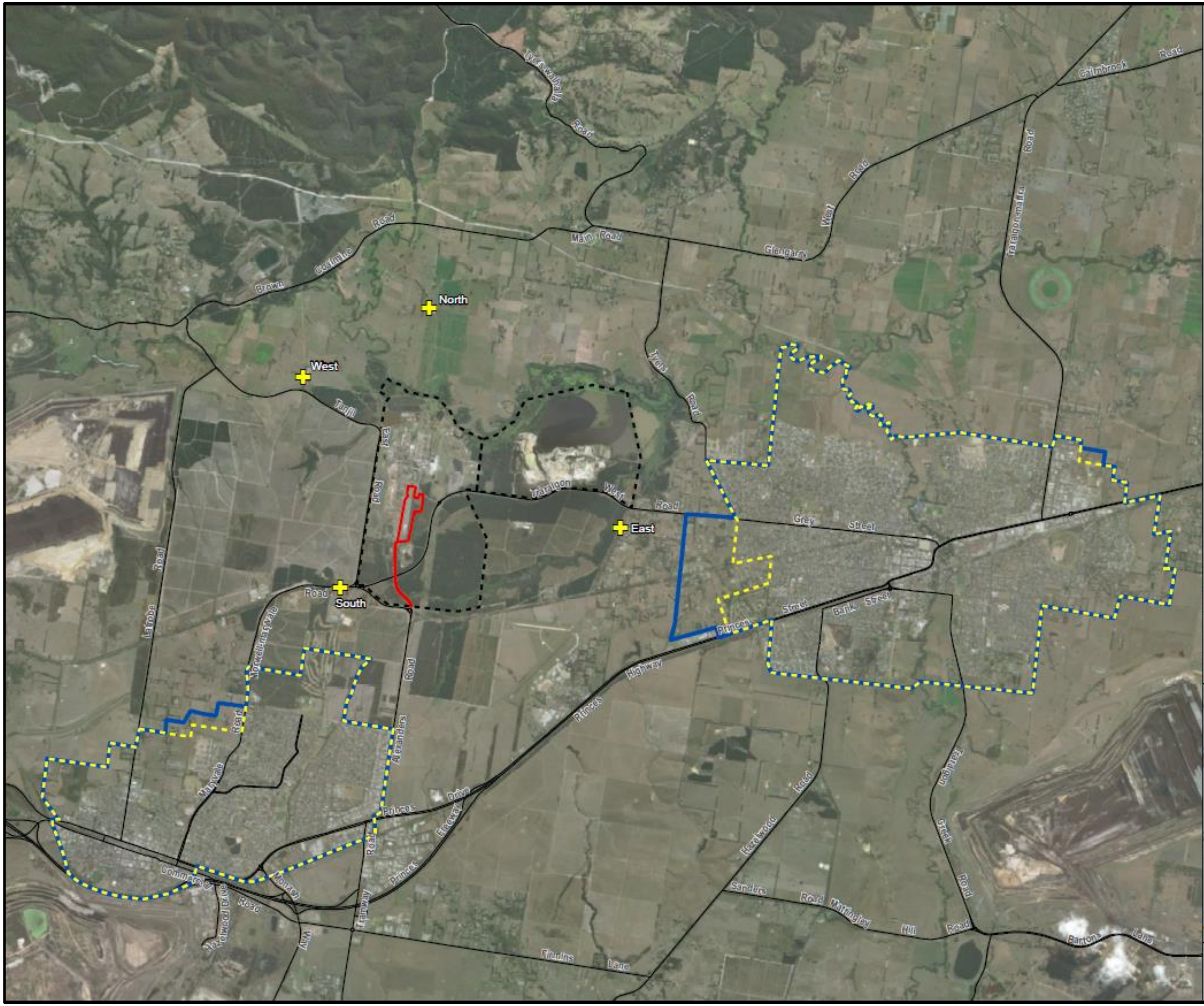


Figure 2.1: Land use zoning around the project site



Legend

- Nearest Noise Sensitive Areas
- ABS Urban Centre Locations with Vic. Population > 7000
- Major Urban Areas
- Maryvale Mill Complex
- Project Area

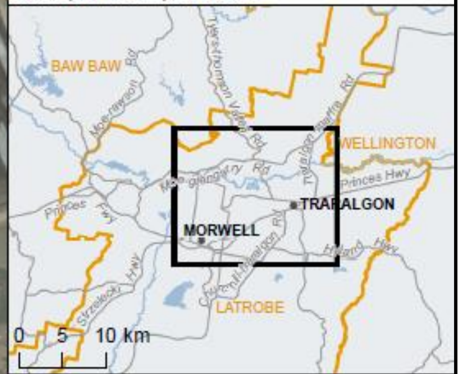
Figure 2.2: Location of nearest residents

RO097400

GDA 1994 MGA Zone 55

0 1 2 Kilometres

DATA SOURCES
© Commonwealth of Australia (Geoscience Australia) 2006 Geodata Topo 250k Series 3; Vicmap Data © State of Victoria 2017; Department of Environment, Land, Water and Planning 23/04/2017; Jacobs 2017DATA SOURCES © ESRI BaseMap



Australian Paper

JACOBS

2.2 Project infrastructure and layout

The following structures and infrastructure are intended to be located on the site:

- Weighbridges and gatehouse
- Energy from waste facility building
- Condenser, turbine and generator
- Road infrastructure
- Car park and hard stand area.

2.3 Process

In this application AP propose to use municipal solid waste (MSW) and commercial and industrial (C&I) waste for fuel stock for an energy from waste plant. **Figure 2.3** provides a diagram of the process with additional explanation of the process below the figure.

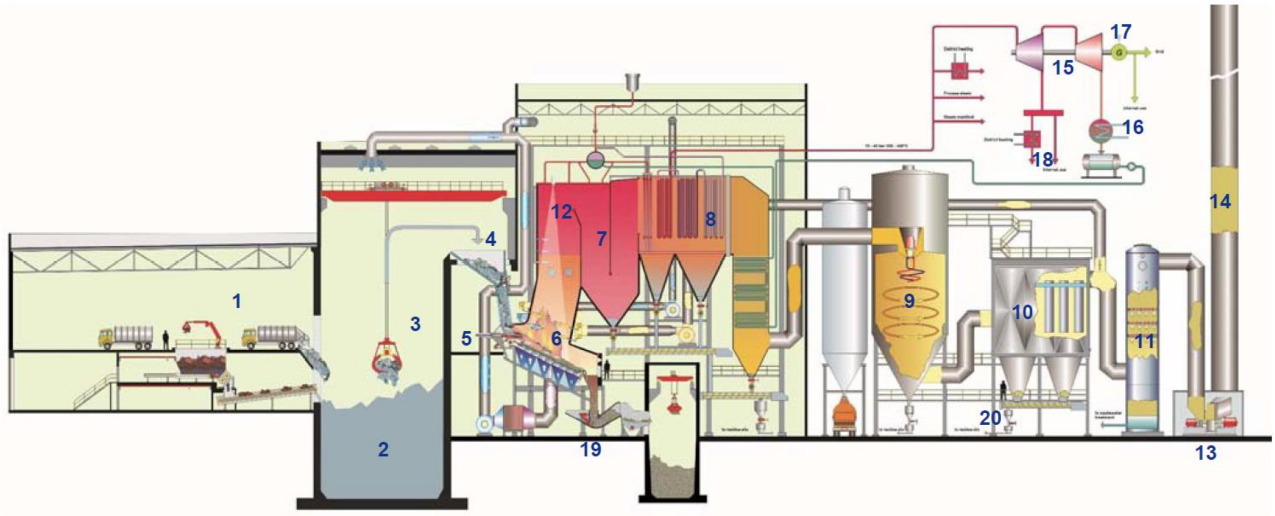


Figure 2.3: Diagram of the energy from waste process (courtesy Martin GMBH)

Wastes will be delivered via road and rail in sealed 40 foot containers as well as by refuse collection vehicles and other bulk solids handling vehicles that are delivering waste from waste transfer stations. Wastes will be manipulated in an enclosed and tipping hall held under negative pressure to control potential odours ((1) on **Figure 2.3**) and tipped into a waste bunker (2). Wastes will be mixed and lifted by overhead crane(s) (3) into the waste feed hopper (4). Waste is pushed from the bottom of the hopper onto the combustion grate (6) via a hydraulically driven ram feeder (5). Non-combustible material known as bottom ash falls off the end of the grate and is handled by the bottom ash extractor (19), where it is cooled and subjected to metal separation systems to remove ferrous and non-ferrous metals. These recycled metals can be resold as a commodity. The remaining bottom ash can then be loaded into vehicles and transported on or off-site for treatment or re-use as construction aggregate.

Heat is recovered from the flue gas to generate steam in the boiler and economiser pass (8), with flue gases leaving the boiler typically treated with powdered activated carbon to absorb volatile organic components and heavy metals such as mercury, and with a dry or semi-dry lime dosing and reactor system (9) to neutralise acid gas pollutants. Mobile ash particulates and flue gas treatment residues entrained in the flue gases are captured in the bag filter plant (10). The residues are collected in the bottom of the bag filters (20) and conveyed to a storage silo ready for disposal to an appropriate landfill capable of accepting hazardous waste.

Oxides of Nitrogen emissions are controlled by the injection of ammonia or urea into the flue gases at the top of the furnace (12). Furnace pressure is controlled by the induced draft fan (13), which then draws the cleaned flue gases up the chimney (14).

The high pressure steam produced in the boilers is piped to a single steam turbine generator (15), which generates electrical power via the turbogenerator (17). Steam exhausted from the turbine is cooled in a water cooled condenser (16).

Section 3. Community profile

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

The site is located in the Latrobe City Council Local Government Area, within an industrial land use zone and surrounded by farming and special use land zones (**Figure 2.1**). The closest resident is approximately 2 kilometres away, with the towns of Morwell and Traralgon approximately 7 kilometres from the site.

Table 3.1 presents a summary of the populations in the towns of Morwell and Traralgon (based on 2016 Census and 2016 Socio-Economic data from the Australian Bureau of Statistics) in comparison to the Victorian and Australian populations.

Table 3.1: Summary of populations surrounding the proposed project site

Indicator	Suburb or Statistical Area		Victoria	Australia
	Morwell	Traralgon		
Total population	13771	24933	5926624	23401892
Population 0 - 4 years	6.0% (828)	6.5% (1623)	6.3% (371220)	6.3% (1464779)
Population 5 - 19 years	16.7% (2298)	18.8% (4675)	18.0% (1066042)	18.5% (4321427)
Population 20 - 64 years	55.7% (7669)	57.4% (14309)	60.2% (3566775)	59.6% (13938918)
Population 65 years and over	21.6% (2976)	17.3% (4318)	15.6% (922598)	15.7% (3676758)
Median age	43	38	37	38
Household size	2.1	2.4	2.6	2.6
Unemployment	14.5%	7.7%	6.6%	6.9%
Tertiary education	36.1%	45.9%	50.4%	49.6%
SEIFA IRSAD	830	960	--	--
SEIFA rank	1	4	--	--
SEIFA IRSD	829	981	--	--
SEIFA rank	1	4	--	--
Indigenous	2.6%	1.2%	0.8%	2.8%
Born overseas	15.4%	11.9%	28.4%	26.3%
Speak other language at home	10.2%	7.1%	26.0%	20.8%

SEIFA IRSAD = index of socioeconomic advantage and disadvantage, rank relates to rank in Australia that ranges from 1 = most disadvantaged to 10 = least disadvantaged
SEIFA IRSD = index of socioeconomic disadvantage, rank relates to rank in Australia that ranges from 1 = most disadvantaged to 10 = least disadvantaged
Shading relates to comparison against Victoria: lower than; greater than

Sources of information:
http://quickstats.censusdata.abs.gov.au/census_services/getproduct/census/2016/communityprofile/SSC21757?opendocument **(for Morwell, Vic)**
http://quickstats.censusdata.abs.gov.au/census_services/getproduct/census/2016/communityprofile/SSC22556?opendocument **(for Traralgon, Vic)**

Based on the population data available and presented in **Table 3.1**, the community of Morwell is older, has higher unemployment, less tertiary education and a high socioeconomic disadvantage when compared to the general Victorian and Australian population. The town of Traralgon is more reflective of the general Victorian and Australian populations, however, is still subject to socioeconomic disadvantage. The indicators outlined in **Table 3.1** reflect the vulnerability of the population, its ability to adapt to environmental stresses, and are important to highlight from an equity point of view. The project will be implemented within a community with higher age profile and socially disadvantage relative to the rest of the state, so positive (such as employment) and negative impacts (such as air pollution) have the potential for a greater effect.

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. However, it is assumed that the health of the local community is consistent with that reported in the larger Latrobe City Council Local Government Area. The Latrobe City Council local government area has been selected as it contained the towns of Morwell and Traralgon and is the smallest unit for which health data is publicly available.

Table 3.2 presents a summary of the general population health considered relevant to the area. The table presents available information on health-related behaviours (i.e. key factors related to lifestyle and behaviours known to be of importance to health) and indicators for the burden of disease within the community compared to Victoria.

Table 3.2: Summary of health indicators/data

Health indicator/data	Latrobe City Council LGA	Victoria
Health behaviours		
Adults - compliance with fruit consumption guidelines (2014)[1]	45.3% (36.2% - 54.7%)	47.8% (46.6% - 49.0%)
Adults - compliance with vegetable consumption guidelines (2014)[1]	6.9% (3.9%- 11.9%)	6.4% (5.9% - 6.8%)
Children adequate consumption of fruit and vegetables (2009) [2]	35.4%	34.7%
Adults - increased lifetime risk of alcohol related harm (2014) [1]	61.0% (52.8% - 68.7%)	59.2% (58.0% - 60.3%)
Adults - body weight (preobese) (2014) [1]	36.6% (28.0% - 46.2%)	31.2% (30.2% - 32.3%)
Adults - body weight (obese) (2014) [1]	22.0% (16.0% - 29.4%)	18.8% (17.9% - 19.6%)
Adults – sufficient physical activity (2014) [1]	35.4% (27.2% – 44.5%)	41.4% (40.2% - 42.5%)
Children – adequate physical activity (2009) [2]	70.4%	60.3%
Current smoker (2014) [1]	24.4% (16.8% - 33.9%)	13.1% (12.3% - 14.0%)
Burden of disease		
Morbidity - cardiovascular disease hospitalisations (2014/15)[3]	2326.4*	2123.2*
Morbidity – respiratory disease hospitalisations (2014/15)[3]	2232.0*	1859.4*
Morbidity - prevalence of hypertension ≥18 years (2014/15)[3]	27700 (21000 – 34500)*	24100 (23400 – 24800)*
Adolescent (12 -17 years) – prevalence of asthma (2009)[3]	12.1%	11.6%
Children (school entrant) – prevalence of asthma (2016)[4]	16.2%	11.8%

* Rate per 100,000 population

1 Data from Victorian Population Health Survey 2014 (Department of Health and Human Services 2016)

2 Data from the City of Latrobe Early Childhood Community Profile (2010) and City of Latrobe Adolescent Community Profile (2010) (Gippsland region)

3 Age standardised ratio - data relevant to the years 2014-2015 from the Social Health Atlas of Australia, Victoria (as published April 2018)

4 Data available from School Entrant Health Questionnaire, 2016 https://www.education.vic.gov.au/about/research/Pages/reportdatahealth.aspx

Shading relates to comparison against Victoria: lower than, greater than

In general, the key indicators of health for the population in the Latrobe local government area are similar to those of Victoria with the exception of physical activity for children (LGA has more active children than Victorian average) and proportion of adult smokers (LGA has more smokers than Victorian average), which are statistically significant. There is also a possible greater burden of disease within the local community with higher reported rates of hospitalisations, hypertension and asthma, when compared with the rates for Victoria.

It is noted that the life expectancy for Latrobe LGA (male = 76.9 years, female = 82.2 years) is lower than other local government areas in the Gippsland region and lower than the Victorian average (male = 80.3 years, female = 84.4 years) (Department of Health 2013).

This data, along with data presented in **Table 3.1**, suggest the population in the areas surrounding the site are likely to be more susceptible to health-related impacts associated with the project, than the general population of Victoria.

Section 4. Community engagement

A community engagement program has been undertaken for this project, which commenced in May 2017 where it was discussed with community members through the Maryvale Community Consultation Committee. Since this time, a number of community focus groups and community forums have been held, a community information centre has been opened and meetings with government agencies have been undertaken. Activities relating to community engagement are detailed in *Maryvale Energy from Waste Plant – Works Approval Application, Jacobs (2018) – Chapter 2* including the updated information provided in **Table 4.1**.

Table 4.1: Community engagement activities

Dates	Activity
13/01/17	State Minister Briefing
16/03/17	State Premier Briefing
11/05/17 10/08/17 09/11/17	Presentations to the Maryvale Community Consultation Committee
Throughout 2017	Regular briefings to staff of Australian Paper Maryvale Plant
21/08/17 – 22/08/17	Focus groups
25/09/17	Community forum
01/10/17	Regular advertisements in the Latrobe Valley Express
05/10/17 01/11/17 22/11/17 08/12/17	Meetings with EPA
19/10/17	Meeting with Regional Development Victoria
23/11/2017	Meeting with Latrobe City Council
01/12/17	Meeting with Latrobe Valley Authority and Regional Development Victoria
11/12/17	Information Centre opens to public
12/12/17 15/12/17 15/02/18	Meeting with Traralgon Chamber of Commerce
12/12/17	Meeting with Advance Morwell
16/12/17 14/03/18	Interview with Gippsland FM
11/01/18	Public notification on WIN news regarding information centre
19/02/18 23/02/18	Meeting with Committee for Gippsland
27/02/18	Morwell Business information night
14/03/18 to 17/03/18	Pop up information centre – Traralgon Centre Plaza
19/03/18	Opening of the Information Centre
21/3/18	DELWP Young Professional Network (YPN)
25/03/2018	Maryvale Mill Open Day
27/3/18	Committee for Gippsland
5 to 6/04/18	Moe Pop Up
09/04/18	Traralgon Central Rotary
10/04/18	LV Sustainability Network
11 to 12/04/18	Midvalley Morwell Pop Up
18/04/18	Nationals Meeting
10/05/18	Voices of the Valley
5, 6 and 19/06/18	Community Open House sessions

To date, the Project Office and Information Centre in Morwell CBD has had over 250 visitors and hosted 35 delegations.

Key issues raised during the community engagement activities that relate to community health (either directly or indirectly) for this proposal were as follows:

- Air quality
- Odour impacts
- Noise impacts
- Water quality
- Combustion by-products
- Employment opportunities.

Section 5. Health impacts: Air emissions

5.1 Approach

This section presents a review of impacts on health associated with predicted air emissions, relevant to the operation of the facility. The assessment presented has relied on the *Maryvale Energy from Waste Plant – Works Approval Application, Jacobs (2018) – Chapter 6* along with further modelling requested from and provided by Jacobs. The estimation of risk follows the general principles outlined in the enHealth document Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a).

5.2 Modelled air impacts

5.2.1 General

To predict the concentration of emissions from the energy from waste plant, a study area was defined (**Figure 5.1**) and predicted emissions from the stack were modelled using the AEROMOD air dispersion model. The AEROMOD air dispersion model is the regulatory air pollution model prescribed by EPA Victoria for the assessment of air quality impacts from all industrial developments including energy from waste facilities. This model uses air emissions estimates for energy from waste processes, plant design (for example stack height), local terrain and meteorological data to predict the ground level concentrations of emissions within the defined study area.

Background air concentrations are also used to determine the total emissions exposure in the study area. Background air data were obtained from EPA Victoria monitoring data acquired in the Latrobe Valley between 2012 and 2016. These estimated background concentrations are likely to be an overestimate of the current background concentrations because the historical data includes facilities that have since closed (Hazelwood Power Station, Morwell Power Station and Briquette Factory).

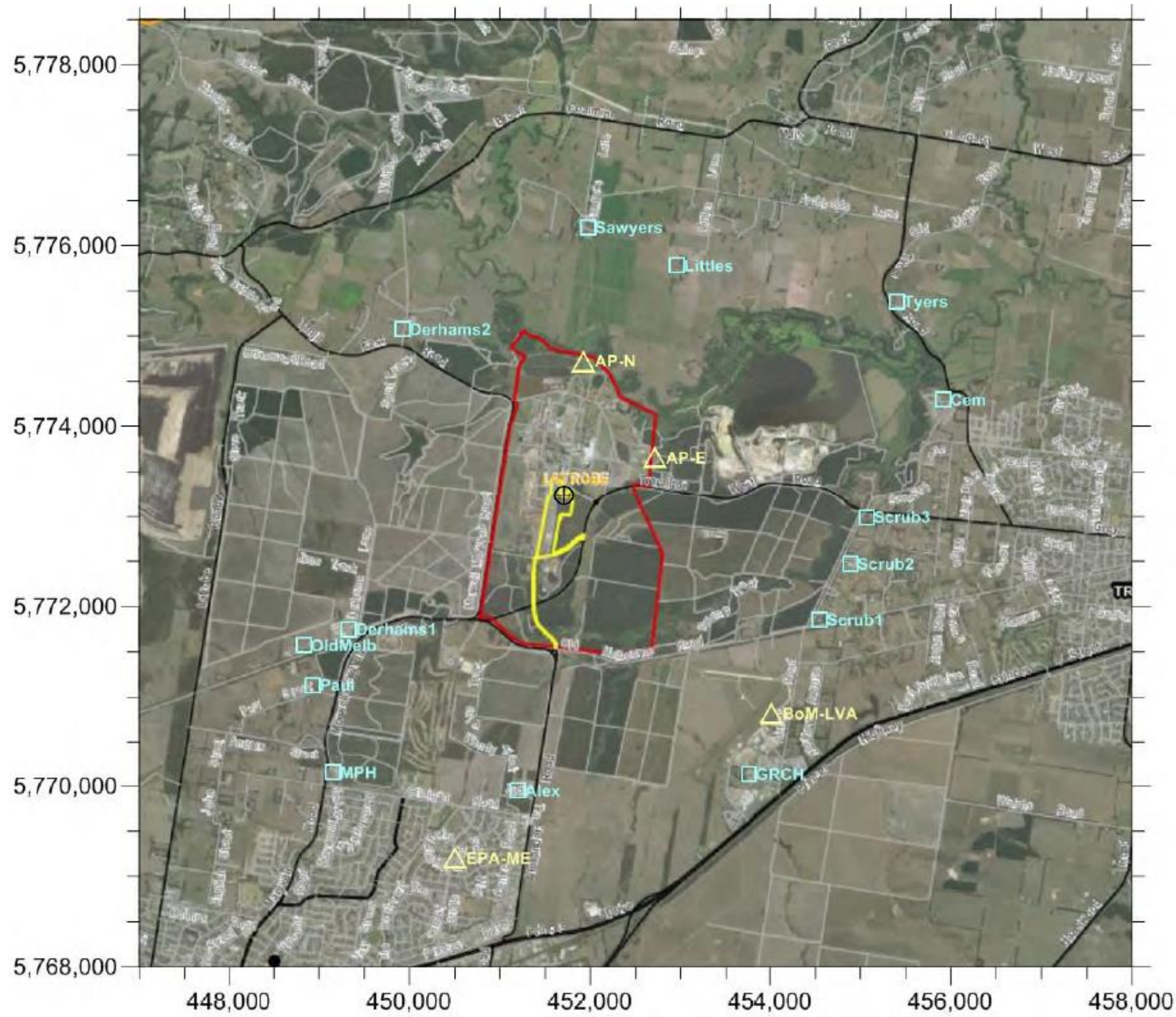


Figure 5.1: Air modelling study area

5.2.2 Air pollutants considered

The selection of pollutants to assess as part of the Works Approval Application for EPA Victoria was derived from the legislation, policies and guidelines applicable to air quality assessment for this type of development.

Proposed EfW feedstock

The proposed EfW facility will utilise MSW and C&I wastes as the fuel source to generate energy in the form of steam and electricity. The EfW feedstock would comprise primarily of residual MSW (approx. 80%) which represents a relatively predictable baseload feedstock having relatively consistent compositions. MSW materials would be supplemented with other residual waste sourced from the C&I sector (approx. 20%), but only from those businesses generating waste appropriate for treatment by the EfW facility – for example, MSW-like wastes from shopping centres, office blocks and schools. Wastes will not include prescribed industrial wastes such as asbestos, dangerous goods or clinical waste. These waste feedstocks are consistent with household waste treated in the United Kingdom and the waste composition of the Suffolk EfW reference plant.

The majority of these residual waste materials are currently collected by councils and private contractors for disposal at landfill. The benefits of EfW are realised when waste materials used as input feedstocks cannot be viably recovered for reuse and recycling. The project is seeking to target waste feedstocks which have limited potential for reuse or recycling and can be aggregated and transported along existing major transport routes.

For further information on the waste materials, refer to Chapter 10 of the Works Approval Application.

Legislation, policies and guidelines

A Works Approval is required by EPA Victoria under the *Environment Protection Act 1970* and the *Environment Protection (Schedule Premises) Regulations 2017*. The State Environment Protection Policy (Air Quality Management) (or "SEPP (AQM)") specifies emission limits from new stationary sourced for particulate matter (PM), carbon monoxide (CO) and oxides of nitrogen (NOx) and gas and solid fuel.

The EfW facility will also have to comply with EPA Publication 1559.1 (Energy from Waste Guideline, July 2017). In this guideline, reference is made to the European Union's Waste Incineration Directive 2000/76/EC (WID) as follows:

- *Emission discharges, under both steady and non-steady state operating conditions, meet all the emissions standards set in the European Union's Waste Incineration Directive 2000/76/EC (WID), which was recast into the Industrial Emissions Directive 2010/75/EU (IED). The IED sets stringent emission limits and monitoring requirements which include:*
    - *continuous emissions monitoring of total particulate matter (TPM); sulphur dioxide (SO_2); oxides of nitrogen (NOx); hydrogen chloride (HCl); carbon monoxide (CO); total organic carbon (TOC); hydrogen fluoride (HF)). In addition, there must be at least non-continuous air emission monitoring of other pollutants such as heavy metals, dioxins and furans, a minimum of two measurements per year, which should be more frequent during the initial operation of the plant. This monitoring should capture seasonal variability in waste feedstock and characteristics.*

The IED emission limits for such a facility relate to: total dust; TOC; gaseous emissions: HCl, HF, SO_2, NOx, nitrogen dioxide (NO_2) and CO; heavy metals and their compounds: cadmium (Cd), thallium (Tl), mercury (Hg), antimony (Sb), arsenic (As), lead (Pb), chromium (Cr), cobalt (Co), manganese (Mn), nickel (Ni) and vanadium (V); and dioxins and furans.

In addition, the modelling has considered emissions to air of polycyclic aromatic hydrocarbons (PAHs), specifically the carcinogenic PAHs as benzo(a)pyrene (BaP). Emissions of ammonia (NH_3) have also been assessed.

For further information, refer to Chapter 6 of the Works Approval Application.

The list of pollutants above have been considered within the air quality modelling.

Flue gas treatment

Air emissions from the main stack of the EfW facility are regulated, and the facility is required to have continuous (24/7) emission monitoring systems (CEMS) to ensure compliance. Within the flue gases emitted from the stack, the following types of emissions are found in low concentrations for MSW and C&I feedstock and for each emission type the techniques typically used to control these emissions (known as best available techniques (BAT)) which can achieve effective emission control:

- Oxides of Nitrogen (NOx) – controlled by combustion control (ensuring efficient combustion conditions) and selective non catalytic reduction (SNCR) with the injection of ammonia or urea into hot flue gases
- Oxides of Sulfur (SOx) – controlled by the injection of lime (alkaline) reagent into the flue gas to absorb and neutralise the acid gas compounds
- Halogens (e.g. HCl and HF) – also controlled by lime (alkaline) reagent injection, neutralisation and adsorption
- Particulates –ash and residues from the various air pollution control technologies are filtered out in the bag filter system
- Heavy Metals – controlled by the injection of activated carbon into the flue gas which is subsequently collected in the bag filter system
- Volatile organic compounds and dioxins and furans– which are destroyed by high temperature in the furnace, the reformation inhibited by controlling the flue gas cooling and the use of activated carbon injection and bag filters to absorb and remove any residuals.

The air modelling undertaken has included the operation of the above emissions control processes in the operation of the EfW facility.

For further details on the flue gas treatment system, refer to section 4.6 in the Works Approval Application.

5.2.3 Modelled impacts within the community

The assessment of air quality impacts within the off-site community considered impacts within the study area that is 15 km x 12.5 km in size, as illustrated in **Figure 5.1**. Impacts were modelled within this study area based on a grid with 100 m spacing.

Figure 5.1 also shows the locations of the air discharge stacks (black cross) and the meteorological stations (yellow triangles). In addition, a number of individual sensitive receptors have been evaluated. These are described in **Table 5.1** and shown on **Figure 5.1** as blue squares.

Table 5.1: Sensitive receptors

Receptor name	Location/description
MPH	Maryvale Private Hospital
Derhams1	Residence/farm Derhams Lane
OldMelb	Residence/farm Old Melbourne Road
Paul	Residence on Paul Street
Scrub1	Scrubbly Lane 1 – rural residential area
Scrub2	Scrubbly Lane 2 – rural residential area
Scrub3	Scrubbly Lane 3 – rural residential area

Receptor name	Location/description
Alex	Alexanders Road – residential property
GRCH	Gippsland Rotary Centenary House
Derhams2	Derhams Road – rural residential area
Sawyers	Sawyers Lane – rural residential properties
Littles	Littles Lane – rural residential properties
Tylers	Tylers Lane – rural residential properties
Cem	Cemetery – with rural residential properties in the vicinity

This assessment, of risks to human health, has considered the maximum predicted impacts at any location across the study area (regardless of the land use and presence (or otherwise) of a residential home), as well as each of the sensitive receptors.

5.3 Conceptual site model

Understanding how a community member may come into contact with pollutants released in air emissions from the proposed energy from waste facility is a vital step in assessing potential health risk from these emissions. A conceptual site model provides a holistic view of these exposures, outlining the ways a community may come in contact with these pollutants.

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

- inhalation (breathing it in)
- ingestion (eating or drinking it) or
- dermally (absorbing it through the skin).

For some of the emissions from the proposed EfW plant, inhalation is considered the only route of exposure. This is due to the substance's chemical properties, which make the other pathways inconsequential. In this instance, gases such as NO_2, SO_2, HCl, HF and CO as well as fine particulate matter as particulates less than 10 micrometres (PM_{10}) and particulate matter less than 2.5 micrometres ($PM_{2.5}$) that are so small they remain suspended in air could be considered in this class (i.e. inhalation only exposure pathway).

Other emissions may be inhaled, but also may be deposited on the ground. These emissions can then be ingested either directly through accidental consumption of soil or indirectly through food grown or raised in the soil (fruit, vegetables and eggs). Skin contact with the soil is also possible. Therefore, it is important with these emissions that all three exposure pathways are considered. In this instance, metals and dioxins that are bound to the heavier particulate matter that may fall out and deposit onto the ground could be considered in this class.

Table 5.2 lists the substances considered in the EfW emissions and the exposure pathway/s of potential concern. **Figure 5.2** provides a diagrammatical representation of the community exposures to emissions from the energy from waste facility (conceptual site model).

Table 5.2: Substances and routes of exposure

Substance	Route of exposure
Nitrogen dioxide	**Inhalation only** as these are gases
Sulfur dioxide	
Hydrogen chloride	
Hydrogen fluoride	
Carbon monoxide	
Ammonia	
PM_{10}	**Inhalation only** as these particulates are very small and will remain suspended in air. It is noted that other exposure pathways have also been assessed for the individual chemical substances bound to these particles. These other pathways relate to the individual chemical substances, rather than the physical size of the particulates.
$PM_{2.5}$	
Cadmium	**Inhalation** of these pollutants adhered to fine particulates **Ingestion and dermal contact** with these pollutants deposited to soil **Ingestion** of produce grown in soil potentially impacted by these pollutants (i.e. homegrown fruit and vegetables, eggs, milk and meat products – where the pollutants can be taken up/bioaccumulated into plants and animals)
Thallium	
Mercury	
Antimony	
Arsenic	
Lead	
Chromium	
Cobalt	
Copper	
Manganese	
Nickel	
Vanadium	
Dioxins / furans	

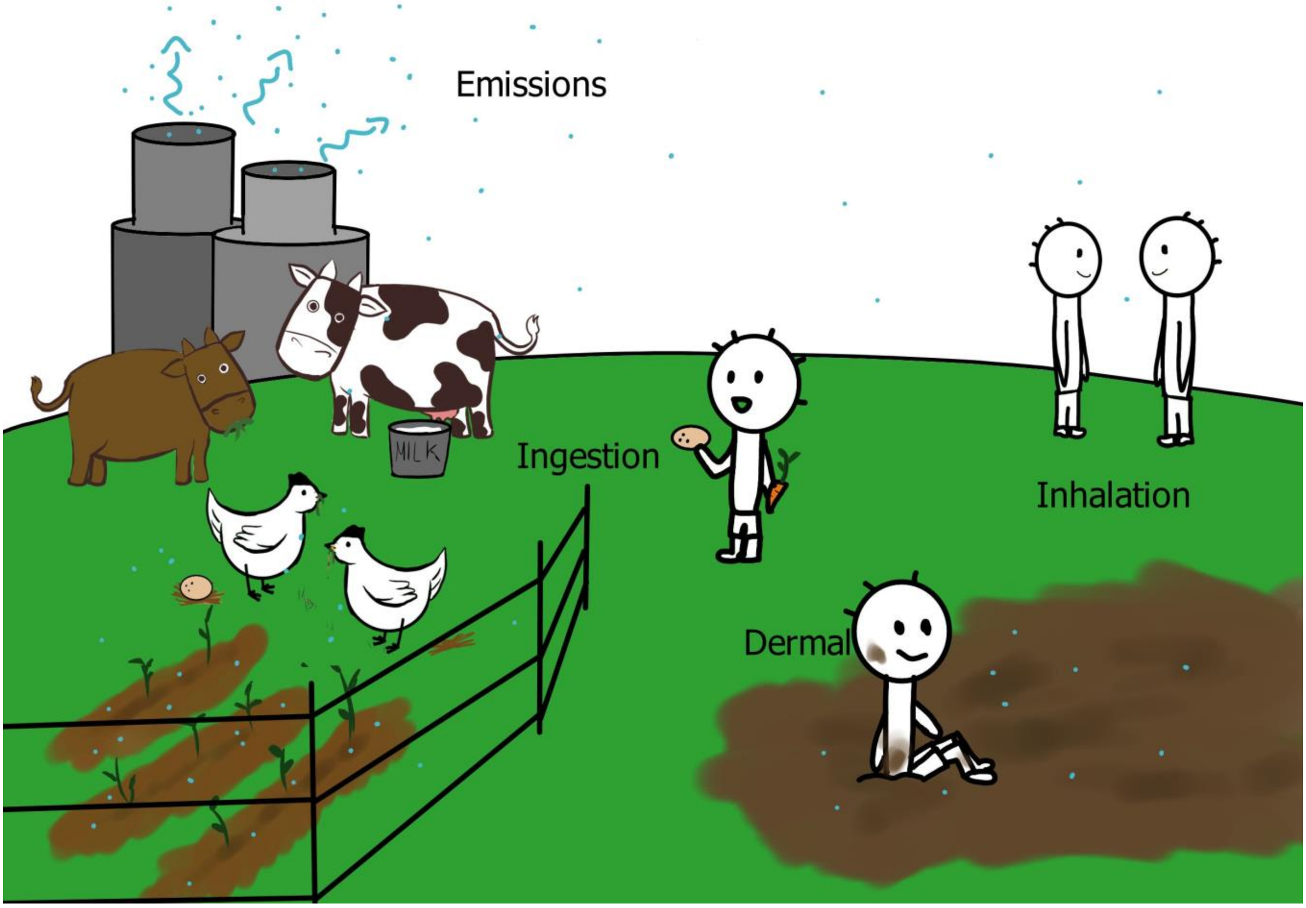


Figure 5.2: Conceptual site model (illustrative only)

5.4 Inhalation exposures

5.4.1 General

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

5.4.2 Particulates

The assessment of potential health impacts associated with exposure to particulate matter, based on the size of the particulate matter, rather than composition, has been undertaken and presented within the Air Quality Impact Assessment described in chapter 6 of the Works Approval Application. The assessment has focused on fine particulates, namely $PM_{2.5}$, which are small enough to reach deep into the lungs and have been linked with, and shown to be causal, for a wide range of health effects (USEPA 2012; WHO 2013). These health effects were considered in the derivation of the NEPM air guideline for $PM_{2.5}$ (NEPC 2016), which are consistent with the SEPP (AAQ)

The NEPM/SEPP criteria relate to total exposures to $PM_{2.5}$, that is background or existing levels as well as the additional impact from the proposed facility. Background levels of $PM_{2.5}$ relevant to the local area have been considered and are noted to be influenced by the Hazelwood Coal Mine Fire and prescribed burns as the existing data includes results for these periods. As a result, the air quality impact assessment has identified that, depending on the meteorological data year assessed and meteorological monitoring location, total exposures to $PM_{2.5}$ have the potential to exceed the NEPM/SEPP air criteria. These exceedances occur regardless of the project – i.e. they relate to background levels.

Table 5.3 provides a summary of the contribution of the project to the total $PM_{2.5}$ concentrations, and the NEPM/SEPP air criteria. This table shows that the worst-case $PM_{2.5}$ derived from the facility makes a negligible contribution to existing concentrations and only makes up a very small fraction of the NEPM/SEPP guideline.

Table 5.3: $PM_{2.5}$ impacts from the project – maximum impacts from all years and all meteorological monitoring stations

Parameter	$PM_{2.5}$ – as 24-hour average ($\mu g/m^3$)	$PM_{2.5}$ – as annual average ($\mu g/m^3$)
Maximum from all grid receptors		
Guideline (NEPM 2016)	25	8
Background	32.9	7.2
Contribution from project	0.032	0.003
% contribution of project to NEPM	0.13%	0.038%
% contribution of project to background	0.098%	0.042%

In addition to the analysis presented above, it is possible to also estimate the incremental individual risk associated with the change in $PM_{2.5}$ from the facility. This calculation has been undertaken on the basis of the most significant health indicator, namely mortality, for which changes in $PM_{2.5}$ have been identified to have a causal relationship. The health indicator also captures a wide range of other health effects associated with $PM_{2.5}$. The calculation has considered the baseline mortality rate for males in the Latrobe Valley LGA (which is higher than for females) from 2010 to 2014 (all ages and all causes), along with the exposure-response relationship relevant to assessing all-cause mortality. Further details and calculations are presented in **Appendix A**. These calculations assume that someone is present at the location of maximum increase in $PM_{2.5}$ from the facility for 24 hours a day, every day of the year.

For a maximum annual increase of $PM_{2.5}$ of 0.003 $\mu g/m^3$, this results in a maximum individual risk of $1x10^{-7}$. This risk level is considered to be negligible, noting the enHealth (enHealth 2012a) considers risks less than $1x10^{-6}$ as negligible and essentially representative of zero risk.

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

5.4.3 All other pollutants

For all other pollutants, inhalation exposures have considered both short-term/acute exposures as well as chronic exposures.

Acute exposures

The assessment of acute exposures is based on comparing the maximum predicted 1-hour average concentration with health-based criteria relevant to an acute or short-term exposure, also based on a 1-hour average exposure time. The ratio of the maximum predicted concentration to the acute guideline is termed a hazard index (HI). For this assessment, the maximum predicted 1-hour average concentration across all the grid receptors (i.e. anywhere) as well as the maximum predicted at the discrete receptors have been considered. **Table 5.4** presents a summary of the relevant health-based guideline, the predicted maximum 1-hour average concentration and the calculated HI for each pollutant. The assessment of exposures to nitrogen dioxide and sulfur dioxide has utilised the NEPM guidelines that are protective of health. Risks associated with these pollutants are not considered to be additive. However, potential exposures to all other gases and chemical substances attached to fine particulates have been assumed to be additive and the total HI (the sum of all individual HI's) is also presented.

Risks associated with acute exposures are considered to be acceptable where the individual and total HI's are less than or equal to 1. Based on the assessment presented in **Table 5.4**, all the individual and total HI's are less than 1.

On this basis there are no acute risk issues of concern in relation to inhalation exposures.

Table 5.4: Review of acute exposures and risks

Pollutants	Acute air guideline (1-hour average) (mg/m^3)	1-hour average concentration (mg/m^3)		Calculated HI	
		Maximum anywhere	Maximum receptors	Maximum anywhere	Maximum receptors
NEPM pollutants					
Nitrogen dioxide (NO_2)	0.22[1]	7.6E-02	6.8E-02	3.4E-01	3.1E-01
Sulfur dioxide (SO_2)	0.5[1]	2.2E-01	2.2E-01	4.5E-01	4.5E-01
Other Pollutants					
Hydrogen chloride (HCl)	0.66[2]	6.4E-03	1.4E-03	9.7E-03	2.1E-03
Hydrogen fluoride (HF)	0.06[2]	3.4E-04	7.7E-05	5.7E-03	1.3E-03
Ammonia	0.59[2]	2.9E-02	6.6E-03	5.0E-02	1.1E-02
Cadmium	0.0054[2]	2.9E-06	6.6E-07	5.4E-04	1.2E-04
Thallium	0.06[4]	2.9E-06	6.6E-07	4.9E-05	1.1E-05
Mercury (as elemental)	0.0006[3]	3.5E-05	8.0E-06	5.9E-02	1.3E-02
Antimony	1.5[4]	9.8E-05	2.2E-05	6.5E-05	1.5E-05
Arsenic	0.003[2]	9.8E-07	2.2E-07	3.3E-04	7.4E-05
Lead	0.15[4]	4.3E-05	9.7E-06	2.9E-04	6.5E-05
Chromium (Cr VI assumed)	0.0013[2]	2.0E-06	4.4E-07	1.5E-03	3.4E-04
Cobalt	0.00069[2]	2.0E-06	4.4E-07	2.8E-03	6.4E-04
Copper	0.1[3]	9.8E-05	2.2E-05	9.8E-04	2.2E-04
Manganese	0.0091[2]	9.8E-05	2.2E-05	1.1E-02	2.4E-03
Nickel	0.0011[2]	9.8E-05	2.2E-05	8.9E-02	2.0E-02
Vanadium	0.03[3]	9.8E-05	2.2E-05	3.3E-03	7.4E-04
Dioxin	0.00013[4]	9.8E-11	2.2E-11	7.5E-07	1.7E-07
BaP	0.6[4]	1.3E-05	2.9E-06	2.2E-05	4.9E-06
			Total HI (for other pollutants)	**0.23**	**0.053**
			Target (acceptable/negligible HI)	**≤1**	**≤1**

References for health-based acute air guidelines (1-hour average):
1 = NEPM health based guideline (NEPC 2016)
2 = Guideline available from the Texas Commission on Environmental Quality (TCEQ), https://www.tceq.texas.gov/toxicology/dsd/final.html
3 = Guideline available from California Office of Environmental Health Hazard Assessment (OEHHA) https://oehha.ca.gov/air/general-info/oehha-acute-8-hour-and-chronic-reference-exposure-level-rel-summary
4 = Guideline available from the USEPA as Protective Action Criteria (PAC), where the most conservative value has been adopted https://www.energy.gov/ehss/protective-action-criteria-pac-aegls-erpgs-teels-rev-29-chemicals-concern-may-2016

Chronic exposures

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

$$HI = \frac{\text{Exposure Concentration}}{(\text{Health based criteria or Tolerable Concentration (TC)}) x (100\%\text{-Background})}$$

Where:
Exposure concentration = concentration in air relevant to the exposure period – annual average (mg/m^3)
Health based criteria or TC = health-based threshold protective of all health effects for the community (mg/m^3)
Background = proportion of the TC that may be derived from other sources/exposures such as water, soil or products (%)

For this assessment, it is assumed that a resident or rural resident spend 24 hours per day at home or working on the property, every day of the year, and that the maximum predicted concentration in air is present at the residence and on the property.

For the assessment of exposures to BaP, this requires the calculation of an incremental lifetime cancer risk, as BaP is a genotoxic carcinogen. This is a different calculation that only considers the incremental risk associated with exposures to BaP derived from the facility (i.e. no consideration of background). The calculation of risk is as follows:

$$\text{Incremental lifetime risk} = \text{Exposure concentration x inhalation unit risk}$$

Where:
Inhalation unit risk = health-based value relevant to calculating the risk associated with an inhalation exposure (relevant to exposures within the community) $(mg/m^3)^{-1}$

Appendix B presents the relevant health-based criteria and inhalation unit risk values adopted in these calculations, along with assumptions adopted for the assessment of background intakes.

Table 5.5 presents the calculated individual HI and the incremental lifetime cancer risk relevant to the assessment of chronic inhalation exposures. The table presents the calculations relevant to the maximum annual average concentration predicted across all the grid receptors (i.e. anywhere) as well as the maximum predicted at the discrete receptors.

The assessment of exposures to nitrogen dioxide and sulfur dioxide has utilised the NEPM guidelines that are protective of health. Risks associated with these pollutants are not considered to be additive. However, potential exposures to all other gases and chemical substances attached to fine particulates have been assumed to be additive and the total HI (the sum of all individual HI’s) is also presented.

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

For the assessment of incremental lifetime cancer risks, risks that are less than $1x10^{-6}$ are considered to be negligible or representative of an essentially zero risk (enHealth 2012a), while risks less than or equal to $1x10^{-5}$ are generally considered to be acceptable (NEPC 1999 amended 2013a).

Based on the assessment presented in **Table 5.5**, all the individual and total HI's are less than 1, and the calculated incremental carcinogenic risk is less than $1x10^{-6}$.

On this basis, there are no chronic risk issues of concern in relation to inhalation exposures.

Table 5.5: Calculated chronic risks

Pollutant	Calculated Incremental Lifetime Risk		Calculated HI	
	Maximum anywhere	Maximum receptors	Maximum anywhere	Maximum receptors
NEPM pollutants				
Nitrogen dioxide (NO_2)	--	--	0.24	0.23
Sulfur dioxide (SO_2)	--	--	0.093	0.092
Other pollutants				
Hydrogen chloride (HCl)	--	--	0.0036	0.0026
Hydrogen fluoride (HF)	--	--	0.00017	0.00012
Ammonia	--	--	0.0013	0.0010
Cadmium	--	--	0.019	0.018
Thallium	--	--	0.000012	0.000012
Mercury (as elemental)	--	--	0.0042	0.0031
Antimony	--	--	0.0073	0.0054
Arsenic	--	--	0.000031	0.000023
Lead	--	--	0.012	0.0091
Chromium (Cr VI assumed)	--	--	0.00050	0.00036
Cobalt	--	--	0.00040	0.00029
Copper	--	--	0.0000075	0.0000055
Manganese	--	--	0.021	0.015
Nickel	--	--	0.19	0.14
Vanadium	--	--	0.018	0.013
Dioxin	--	--	0.00038	0.00028
BaP	$3.8x10^{-8}$	$2.7x10^{-8}$		
		Total HI (other pollutants)	**0.26**	**0.19**
Negligible risk	**$\leq 1x10^{-6}$**	**$\leq 1x10^{-6}$**	**≤1**	**≤1**

5.5 Multiple pathway exposures

5.5.1 General

Where pollutants may be bound to particulates, are persistent in the environment and have the potential to bioaccumulate in plants or animals, it is relevant to also assess potential exposures that may occur as a result of particulates depositing to the environment where a range of other exposures may then occur. These include:

- Incidental ingestion and dermal contact with soil (and dust indoors that is derived from outdoor soil or deposited particulates);
- Ingestion of homegrown fruit and vegetables where particulates may deposit onto the plants and is also present in the soil where the plants are grown, and where pollutants bound to these particles are taken up into these plants;
- Ingestion of eggs, meat (beef) and milk (cows) where particulates may deposit onto pasture and be present in soil (which the pasture/feed grows in and animals also ingest when feeding), and the pollutants bound to these particles are taken up into the edible produce.

The above exposures are chronic or long-term exposures.

5.5.2 Assessment approach

In relation to these exposures, such exposures will only occur on residential or rural residential properties where people live and where homegrown produce or other agricultural activities can be undertaken. It is overly conservative to calculate risks associated with these exposures for the location where the maximum rate of particulate deposition occurs. The predicted particle deposition rates have been further reviewed, with **Figure 5.3** showing the modelled contours of the worst-case predicted annual average deposition rate (i.e. maximum from all modelled years and meteorological stations). This contour plot is similar to those from other years, in terms of the distribution/pattern of deposition.

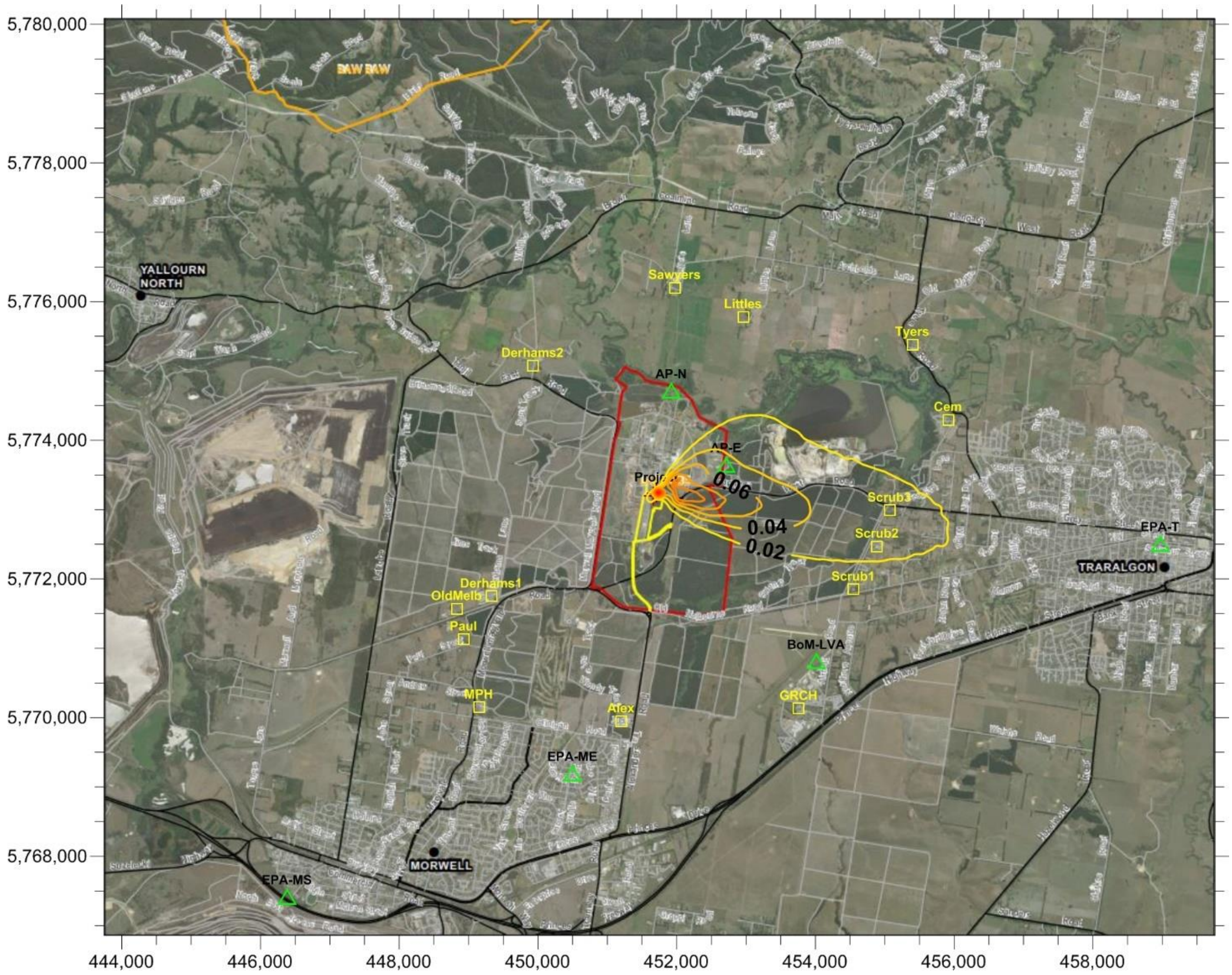


Figure 5.3: Contour plot of annual average particulate deposition rates

Review of **Figure 5.3** indicates the following:

- The highest rate of deposition occurs on the site
- The next highest rates of deposition are to the east within the tree plantation area, which is not residential/rural residential or used for any agricultural purpose
- The locations where the highest rate of deposition occurs that are residential and may also include agricultural uses (including home-grown produce) are the discrete receptors Scrub2 and Scrub3.

On the basis of the above, risks associated with multiple pathway exposures have been calculated on the basis of the maximum predicted deposition rate from all the discrete receptors.

The calculation of risks posed by multiple pathway exposures only relates to pollutants that are bound to the particulates.

Appendix B includes the equations and assumptions adopted for the assessment of potential exposures via these exposure pathways, with the calculation of risk for each of these exposure pathways presented in **Appendix C**.

For the pollutants considered in this assessment, the risk calculations undertaken predominantly relate to a threshold HI, with risks associated with exposure to BaP only calculated on the basis of an incremental lifetime cancer risk. As discussed in **Section 5.4.3**, the following criteria have been adopted for determining when risks are considered to be negligible or acceptable.

- **HI:** the individual and total HI, where calculated as the sum over all relevant exposure pathways and pollutants ≤ 1 = negligible/acceptable risk to human health
- **Incremental lifetime cancer risk:** the individual and total risk, calculated as the sum over all relevant exposure pathways and pollutants ≤ 1×10^{-6} = negligible risk, and ≤ 1×10^{-5} = acceptable risk

5.5.3 Calculated risks

Table 5.6 presents the calculated risks associated with these multiple pathway exposures relevant to both adults and children. These risks have been calculated on the basis of the maximum predicted deposition rate for all of the discrete receptors. The table presents the total HI for each exposure pathway, calculated as the sum over all the pollutants evaluated. The table also includes the calculated risks associated with inhalation exposures, as these exposures are additive to the other exposure pathways for residential/rural residential properties.

Depending on the use of the agricultural property, the types of exposures that may occur are likely to vary. For this assessment, a number of scenarios have been considered where a range of different exposures may occur. The sum of risks associated with these multiple exposures is presented in **Table 5.6**.

Table 5.6: Summary of risks for multiple pathway exposures

Exposure pathway	Calculated risks - Adults		Calculated risks - Children	
	Non-threshold Risk	HI	Non-threshold Risk	HI
Individual exposure pathways				
Inhalation (I)	2.7 $\times10^{-8}$	0.21	2.7 $\times10^{-8}$	0.21
Soil ingestion (SI)	1.2 $\times10^{-9}$	0.019	2.4 $\times10^{-9}$	0.18
Soil dermal contact (SD)	4.7 $\times10^{-9}$	0.0053	1.9 $\times10^{-9}$	0.011
Ingestion of homegrown fruit and vegetables (F&V)	3.3 $\times10^{-8}$	0.078	2.6 $\times10^{-8}$	0.18
Ingestion of homegrown eggs (E)	3.6 $\times10^{-12}$	0.0019	1.5 $\times10^{-12}$	0.0038
Ingestion of homegrown beef (B)	1.4 $\times10^{-8}$	0.028	7.3 $\times10^{-9}$	0.069
Ingestion of homegrown dairy milk (at property) (M)	1.1 $\times10^{-7}$	0.061	9.4 $\times10^{-8}$	0.24
Multiple pathways (i.e. combined exposure pathways)				
I + SI + SD	3.3 $\times10^{-8}$	0.23	3.2 $\times10^{-8}$	0.39
I + SI + SD + F&V	6.6 $\times10^{-8}$	0.31	5.7 $\times10^{-8}$	0.57
I + SI + SD + E	3.3 $\times10^{-8}$	0.23	3.2 $\times10^{-8}$	0.40
I + SI + SD + F&V + E	6.6 $\times10^{-8}$	0.31	5.7 $\times10^{-8}$	0.58
I + SI + SD + B	4.8 $\times10^{-8}$	0.26	3.9 $\times10^{-8}$	0.46
I + SI + SD + M	1.5 $\times10^{-7}$	0.29	1.3 $\times10^{-7}$	0.64
I + SI + SD + F&V + E + B	8.0 $\times10^{-8}$	0.34	6.5 $\times10^{-8}$	0.64
I + SI + SD + F&V + E + M	1.8 $\times10^{-7}$	0.37	1.5 $\times10^{-7}$	0.82
Negligible risk	**≤1 $\times10^{-6}$**	**≤1**	**≤1 $\times10^{-6}$**	**≤1**

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

Review of **Table 5.6** indicates that all calculated risks associated with each individual exposure pathway as well as a combination of multiple exposure pathways, remain below the target risk levels considered representative of negligible risks.

It is noted that the highest HI calculated for multiple exposure pathways (inhalation, soil ingestion, soil dermal contact, ingestion of homegrown fruit and vegetables, ingestion of home-grown eggs and ingestion of milk derived from the property) is 0.82, which is close to the target HI of 1. The calculated HI for the multiple pathway exposures, is dominated by potential exposures to dioxins and furans that may be accumulated in milk, and mercury that may be accumulated in fruit and vegetable crops. While these exposures dominate the calculated risks, the total risk relates to the maximum impacted location calculated using the maximum deposition rate from the different years of meteorological data and from the different meteorological stations. It is also assumed that the maximum deposition rate applies across the whole property, which is not the case particularly for larger properties as the deposition rate decreases with increasing distance from the proposed facility. This will provide a most conservative estimate of lifetime deposition and risks at the closest property. Impacts and hence risks at other properties will be lower. Hence the calculated risks are considered to be representative of a worst-case.

On the basis of the assessment undertaken there are no chronic risk issues of concern in relation to multiple pathway exposures that may be relevant to the rural residential use of the surrounding areas.

5.6 Odour

Predominate odour emissions that may occur from the energy from waste plant will be as a result of fugitive emissions from the tipping hall. To counter this, the tipping hall will be equipped with automatic roller doors that will open and close quickly as trucks enter and leave the tipping hall to minimise fugitive odour escaping the building. Further, the tipping hall will be held under negative air pressure to minimise fugitive emissions from the tipping hall doors and creating the ability to control emissions. It is expected that air from the tipping hall will be used as combustion air in the energy from waste boiler and through this process odorous molecules and hydrocarbons are expected to be destroyed. Owing to this, Chapter 6 – Air Quality of the Works Approval Application has concluded that there would be no significant fugitive odour emissions from the site.

5.7 Outcomes of health impact assessment

Table 5.7 presents a summary of the outcomes of the assessment undertaken in relation to the impacts of changes in air quality, associated with the proposed project, on community health.

Table 5.7: Summary of health impacts – air quality

Impacts associated with air emissions	
Benefits	There are no benefits to the off-site community in relation to air emissions of this type
Impacts	Based on the available data and information in relation to emissions to air from the proposed facility, potential impacts on the health of the community have been assessed. The impact assessment has concluded the following: ■ There are no acute inhalation exposure risks of concern ■ There are no chronic inhalation exposure risks of concern ■ There are no chronic risks of concern from exposure to pollutants from the facility via soil or ingestion of home-grown produce The design of the facility, specifically the tipping hall, will ensure that there are no significant fugitive odour emissions from the site.
Mitigation	The proper operation and maintenance, and monitoring, of the pollution control/flue gas equipment as described in **Section 5.2.2**. The proper operation of the tipping hall as proposed to ensure fugitive odour emissions are effectively managed.

Section 6. Health impacts: Noise

6.1 Approach

This section presents a review and further assessment of impacts on health associated with noise, relevant to the operation of the facility. The assessment presented has relied on the information provided in the *Maryvale Energy from Waste Plant – Works Approval Application, Jacobs (2018) – Chapter 8 – Noise emissions*.

The site is located within an Industrial 2 Zone (IZ2) and surrounded by commercial and special use zones. The nearest sensitive receptors to the proposed project have been identified (**Figure 6**) to the north, east, south and west of the project site at approximately 3.1, 3.2, 1.7 and 2.6 kilometres respectively.

6.2 Summary of noise assessment

6.2.1 General

The noise assessment was based on criteria outlined in the guideline – Noise from Industry in Regional Victoria (NIRV, Publication 1411, October 2011). This guideline provides a process for calculating the recommended maximum noise levels for industry in regional Victoria. From this guideline and in consultation with EPA Victoria, both recommended maximum noise levels (RMNLs) and effective recommended maximum noise levels (ERMNLs) were determined for the four nearest receptors. Both the RMNLs and ERMNLs were developed to ensure compliance with the NIRV guideline.

6.2.2 Site noise assessment

Noise impact from the project was estimated by noise associated with energy from waste plant equipment, along with likely truck movements within the facility. Noise generation from the equipment was estimated from a noise database of common plant equipment, design details sourced from published material on similar energy from waste plants as well as industry recognised data sources. The site was estimated to have three trucks in operation during the day, two during the evening and one at night. No potential mitigation measures were included in the modelling.

Based on the incremental modelled noise impacts, that is the noise generated purely from the project without consideration of background noise, the project is predicted to be in compliance with the RMNLs and ERMNLs (i.e. the noise guidelines for Victoria).

It is noted that given the feasibility phase of the project not all design parameters that have been used in the noise model have been confirmed. Therefore, as highlighted by Jacobs (2018), further refinement of predicted noise impacts and mitigation measures should be incorporated into the design during the design phase. This includes masking the noise of the energy from waste plant within the current noise environment.

6.2.3 Cumulative noise impact

Cumulative noise impact refers to the impact of the noise from the project along with background noise. Further analysis was undertaken for this report by Jacobs, and the following cumulative noise levels were predicted at the nearest receptors (**Table 6.1**).

The *overall* noise level, calculated by logarithmic addition, was based upon the noise level generated by the development plus the background noise level at the affected sensitive receptor. Background noise levels have been previously assessed in 2015 as part of AP Works Application for a De-Inking Plant with confirmation that these levels have not changed (Jacobs 2018).

Typically, due to fluctuations in the background noise environment, masking of the development's noise level will result in a perceived level less than that calculated in **Table 6.1**. In relation to potential health impacts, the predicted noise levels presented in **Table 6.1** have been further considered in **Section 6.3**, where guidelines available from the World Health Organization guideline values have been considered.

Table 6.1: Modelled cumulative noise impact for the four sensitive receptors

Location	Overall noise level (dBA)		
	Day	Evening	Night
North – Sawyer Lane	45.1	39.3	35.0
East – Scrubby Lane	45.0	39.1	39.1
South – Maryvale Rd	52.0	47.1	42.3
West – Derhams Lane	47.0	42.1	37.2

6.3 Health impacts associated with noise

Environmental noise has been identified (I-INCE 2011; WHO 2011) as a growing concern in urban areas because it has negative effects on quality of life and well-being and it has the potential for causing harmful physiological health effects. With increasingly urbanised societies impacts of noise on communities have the potential to increase over time.

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

- Sleep disturbance (sleep fragmentation that can affect psychomotor performance, memory consolidation, creativity, risk-taking behaviour and risk of accidents)
- Annoyance
- Hearing impairment
- Interference with speech and other daily activities
- Impacts on children's school performance (through effects on memory and concentration)
- Impacts on cardiovascular health.

Other effects for which evidence of health impacts exists, but for which the evidence is weaker, include:

- Effects on mental health (usually in the form of exacerbation of existing issues for vulnerable populations rather than direct effects)
- Tinnitus (which can also result in sleep disturbance, anxiety, depression, communication and listening problems, frustration, irritability, inability to work, reduced efficiency and a restricted participation in social life)

- Cognitive impairment in children (including deficits in long term memory and reading comprehension)
- Some evidence of indirect effects such as impacts on the immune system.

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

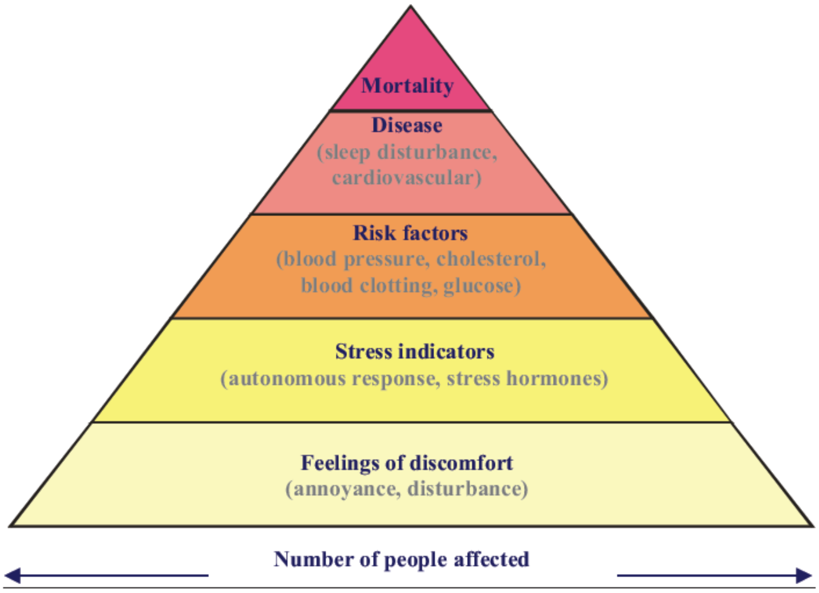


Figure 6.1: Schematic of severity of health effects of exposure to noise and the number of people affected (WHO 2011)

Often, annoyance is the major consideration because it reflects the community's dislike of noise and their concerns about the full range of potential negative effects, and it affects the greatest number of people in the population.

There are many possible reasons for noise annoyance in different situations. Noise can interfere with communication or other desired activities. Noise can contribute to sleep disturbance, which can obviously be very annoying and has the potential to lead to 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.

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 (for example in their kitchen when preparing a meal) may be considered completely unacceptable by that same person in another context (for example in their bedroom when they are trying to sleep). In this case the annoyance relates, in part, to the intrusion from the noise. Similarly, a noise level, which is considered to be completely unacceptable by one person, may be of little consequence to another even if they are in essentially the same room. In this case, the annoyance depends almost entirely on the personal preferences, lifestyles and attitudes of the listeners concerned.

In relation to this project, potential noise impacts have been assessed against criteria developed by the World Health Organization (WHO 1999a, 2009) that have been established on the basis of the relationship between noise and health impacts, where annoyance and sleep disturbance are of most significance. The predicted noise impacts are those that would be outside of a dwelling. These predicted impacts are all below the World Health Organization guideline values that are protective of adverse health effects.

It should be noted that the predicted values are based on modelled impacts for the plant with no mitigation measures considered. There is an understanding and commitment from AP that these mitigation measures will result in no increase in noise levels above background at the nearest receptors (Jacobs 2018). Therefore, it is likely that, following the implementation of noise mitigation measures, noise impacts will be lower than predicted.

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

6.4 Outcomes of health impact assessment: noise

Table 6.2 presents a summary of the outcomes of the assessment undertaken in relation to the impacts of changes in noise, associated with the proposed project, on community health.

Table 6.2: Summary of health impacts - noise

Health impacts associated with noise emissions	
Benefits	There are no benefits to the off-site community in relation to noise emissions
Impacts	Based on the predicted cumulative noise levels and potential mitigation measures, the potential for adverse health impacts within the off-site community associated with noise generated from the operation of the facility is considered to be negligible
Mitigation	The plant is currently in a feasibility stage of design. Further noise modelling and intervention will need to be undertaken if the project moves to the design phase. This includes: ■ Undertaking confirmation of impacts once the design of the plant is confirmed ■ Apply appropriate and reasonable mitigation measures, to be determined at the design phase so as not to increase the noise levels at the nearest receptors from current levels. These mitigation measures may include: ○ Selection of quiet plant and equipment ○ "line of sight' with noise sensitive areas reduced as far as practicably possible ○ Application of acoustic attenuation in the form of noise 'barrier' walls or enclosure. ○ Application of acoustic insulating constructions for building door and walls ○ Use of attenuators on extraction systems

Section 7. Health impact assessment: Water, economics, transport, hazardous waste, community and social aspects

7.1 Approach

Health impacts associated with other aspects of the proposed project, including wastewater, economics, transport, pestilence, community and social aspects have been addressed in this section. The assessment presented has relied on the *Maryvale Energy from Waste Plant – Works Approval Application, Jacobs (2018)*. The assessment has been undertaken as a qualitative evaluation, to identify benefits and impacts associated with the project.

7.2 Overview and assessment of issues

Water

The use and discharge of water is described in Chapter 9 of the Works Approval Application. The chapter describes the current water treatment facility used for the Pulp and Paper Mill next to the site including its current discharge licence.

The energy from waste plant will require water for the following uses:

- Cooling tower water make-up
- Ash handling
- Flue gas treatment (if a semi dry system is used)
- Production of demineralised water from the generation of steam
- Boiler chemistry control and online boiler cleans (soot blowing)
- Fire service system.

A general description has been provided for the backwash water from the filtration plant and use of water for the cooling tower including its proposed connection to the current water treatment facility for the Pulp and Paper Mill. Both uses are isolated from any interaction with the waste stock used as fuel in the EfW plant and so the discharged water from these two processes will be different from the discharged water used for the other identified purposes. Further, current discharge water from the Pulp and Paper Mill's Power Plant operations will be similar to that of the EfW plant. With reduced Power Plant operations, the EfW operations water discharge will substitute a proportion of volumes currently processed.

The discharge of water from the EfW plant is further described in chapter 9.5.4 of the Works Approval Application. Review of this information indicates that water discharges from the EfW plant, either in volume of discharge and general water quality (i.e. total dissolved solids, total suspended solids and biocides), will not alter the overall water discharges from the Mill. It is noted that water discharges from the water treatment facility at the Mill are currently conducted under an EPA licence. It is expected that the licence will continue to apply and will include water discharges from the EfW plant.

On this basis no further, detailed assessment of water discharges from the proposed EfW plant is required.

Economics

The proposed project will result in estimated Victorian employment opportunities of more than 1600 jobs during the construction phase and 440 jobs during the operational phase, including direct and indirect employment.

The most significant health outcomes in the community are expected to be benefits associated with job creation. While there is evidence to support that finding employment has health benefits, most studies are related to the negative impacts of unemployment. It would seem reasonable that if unemployment has a range of negative effects then finding employment would have positive effects. Health outcomes from unemployment include increases in the risk of illness and premature death and there are impacts on a range of mental health issues (anxiety, stress etc.) and social aspects of life (lower self-esteem, feelings of insecurity etc.). Finding employment is expected to be associated with improvements in these aspects of health and wellbeing. This is especially important for the local community which is likely to be more susceptible to health-related impacts associated with the project (**Section 3**). The region also has higher than average unemployment. Therefore, improvements in health and wellbeing in the local community can be enhanced by encouraging local employment at the facility.

Transport

A high-level assessment of the proposed traffic generation and traffic impacts of the proposed energy from waste plant was undertaken. Construction traffic was predicted to increase vehicle movements in the local area by over 800 vehicles a day, with most of these being from construction workers. When in operation, the predicted increase is likely to be around 100 vehicles per day. A review of the operational traffic movements leads to the conclusion that 'the number of vehicles added by (the energy from waste) development will have a minimal traffic impact upon the local road network. The energy from waste project would add small amounts of truck traffic to roads in the local area relative to current volumes. The only location at which site volumes would be significant is Alexanders Road where the site would increase trucks volumes by 16%, and overall traffic by 2%.'

Increased traffic congestion has the potential to decrease road safety and increase levels of stress and anxiety in the community. The assessment concluded that no significant reduction in travel times along Alexanders Road or any other local road is expected, however, this will need to be confirmed during the next design phase. Based on current information, the health impacts from increased traffic are considered to be minimal.

Discovery and disposal of hazardous waste

It is inevitable that during operations the discovery of hazardous waste will occur. Hazardous waste includes smoke alarms, batteries (household, car, phone, laptop and rechargeable) and light bulbs. AP has committed to establishing detailed inspection and management procedures for the waste feedstock including:

1. Waste Acceptance Criteria. This specification will be captured in the Waste Supply contracts and the responsibility of ensuring nil contaminants and hazardous materials will reside with the supplier.
2. Waste Inspection Procedures.

a. Inspection of materials being loaded into containers and trucks will seek to detect contaminants and hazardous materials. When detected, pre-prepared operating procedures shall be initiated.
b. Inspection of materials delivered into the EfW bunker will include Bunker Operator observations and dedicated CCTV cameras and recording devices.
c. An audit mechanism will be established to periodically divert waste deliveries to an inspection zone where waste can be thoroughly inspected and reported.

2. Hazardous Waste Management Procedures. If hazardous materials are detected, then procedures will be initiated to segregated and appropriately dispose of the materials.
3. Non-Conformance Reporting. Detected hazardous materials will be reported to the relevant supplier and transport organisations as a non-conformance requiring assessment, appropriate countermeasures and formal response.

These procedures need to be further developed into a comprehensive operational plan, to account for the incorrect handling and disposal of such waste that can lead to inappropriate human exposures. A comprehensive operational plan for the discovery and correct disposal of hazardous waste will minimise these exposures. It is anticipated that such procedures and processes are readily transferable from EfW facilities operating in Europe.

Community and Social

There are a range of benefits the overall project offers to the community, specifically:

- A high diversion of waste from landfill estimated to be 650,000 tonnes annually
- A net reduction in greenhouse gas emissions of approximately 550,000 tonnes per year
- Improved energy security by returning approximately 3-4 PJ of natural gas per annum to the broader market

These aspects offer benefits to the community by improving the sustainability of fuels. For some individuals, sustainability is an important factor in community wellbeing and for these individuals the project has the potential to enhance feelings of wellbeing which may be linked with a reduced risk of mental health issues.

Changes to the amenity of a street, suburb or town can negatively impact on a sense of belonging and identity of its residents and consequently their community cohesion. The project is to be located in an existing industrial area adjacent to a pulp and paper mill and approximately 2 kilometres from the nearest receptor. It is not anticipated that the plant will significantly change the current viewscape of the immediate area.

Community issues may also arise for particular developments as a result of feelings of control, or lack of control, over decisions. This can result in increased levels of stress and anxiety particularly where there are perceptions that a particular development may affect the wellbeing and amenity of the community. These issues relate to perceived risks, rather than actual risks for this project and can be mitigated through the maintenance of community consultation throughout the construction, commissioning and operation of the facility.

As outlined in **Sections 5** and **6** there are no impacts on the off-site community in relation to changes in air quality, odour or noise that would adversely affect the health of the off-site community, provided appropriate migration measures are undertaken. Hence there are no equity issues that require further consideration in relation to the distribution of health-related impacts in the off-site areas.

Section 8. Summary of HIA Outcomes

Based on the evaluations presented in **Section 5** to **7**, **a range of outcomes (both positive and negative) have been assessed in relation to health impacts relevant to the off-site community. Where negative impacts have been identified, these are considered to be negligible in terms of community health.**

These outcomes, along with measures that could be implemented to enhance or mitigate the identified health impacts, are summarised in **Table 8.1**.

Table 8.1: Summary of HIA Outcomes and Enhancement/Mitigation Measures

Health Aspect/Issue	Reference in HIA	Potential Health Impacts Considered	Impact Identified (positive or negative and significance)	Types of measures that could be implemented to enhance positive impacts or mitigate negative impacts
Air quality – Inhalation exposures	Section 5.4	Range of health effects associated with exposure to pollutants released to air from the proposed facility	**All exposures: Negative but negligible** More specifically: ▪ No acute risk issues of concern ▪ No chronic risk issues of concern Particulate exposures are negligible and essentially representative of zero risk ▪ Incremental carcinogenic risks are negligible and essentially representative of zero risk	The proper operation and maintenance, and monitoring, of the pollution control/flue gas equipment.
Air quality – Multiple pathway exposures	Section 5.5	Range of health effects associated with exposure to pollutants released to air from the proposed facility, that may then deposit and accumulate in soil, homegrown fruit and vegetables and other farm produce (eggs, beef and milk)	**All exposures: Negative but negligible** More specifically: ▪ No chronic risk issues of concern for multiple pathway exposures ▪ All calculated risks for individual exposure pathways are negligible and essentially representative of zero risk ▪ All calculated risks for combined multiple pathway exposures are negligible and essentially representative of zero risk	The proper operation and maintenance, and monitoring, of the pollution control/flue gas equipment.
Odour	Section 5.6	Annoyance, stress, anxiety	**Not significant and negligible**	The proper operation of the tipping hall as proposed to ensure fugitive odour emissions are effectively managed.
Noise	Section 6	Sleep disturbance, annoyance, children's school performance and cardiovascular health	**Modelled noise impacts: negligible potential for health impacts**	Additional assessment of the project detailed design is required, and application of appropriate and reasonable mitigation measures is required so as not to increase noise levels at the nearest sensitive receivers from current levels.
Economic Environment	Section 7	Reduction in anxiety, stress and feelings of insecurity	**Positive improvements in health and wellbeing**	The identified positive outcomes in the local community can be enhanced by encouraging employment of people who live within the local community

Health Aspect/Issue	Reference in HIA	Potential Health Impacts Considered	Impact Identified (positive or negative and significance)	Types of measures that could be implemented to enhance positive impacts or mitigate negative impacts
Traffic and transport	Section 7	Injury or death, stress and anxiety.	**Negative but minimal**	Details to be determined at the detailed design phase of the project
Discovery and disposal of hazardous waste	Section 7	Possible injury if incorrectly disposed of	**Negative but minimal**	Further development of the feedstock delivery protocol into an operational management plan to address the discovery and proper disposal of this material
Community and social	Section 7	Wellbeing, changes in levels of stress and anxiety	**Positive outcomes** enhancing feelings of wellbeing for aspects such as sustainability **Negative outcomes** for potential changes to amenity and community feelings of control related to perceived risks rather than actual risks	These health impacts relate to community perceptions and trust. It is therefore important that the positive impacts associated with the project are enhanced within the local community and community consultation is continued and uses a range of techniques that are tailored to the various sub-populations that have particular areas of concern or particular characteristics that make normal methods of communication less effective. It is important that an effective communication/ community consultation program is maintained throughout the construction, commissioning and operational phases of the project.

Section 9. References

Australian Bureau of Statistics, 2011. Selected characteristics retrieved from QuickStats, TableBuilder and DataPacks. www.abs.gov.au. Accessed December 2016.

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

ATSDR 2012b, *Toxicological Profile for Manganese*, US Department of Health and Human Services, Agency for Toxic Substances and Disease Registry.

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

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

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

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

Department of Health 2013, *2012 Regional health status profiles, Gippsland region*, Victorian Government Department of Health.

Department of Health and Human Services 2016, *Victorian Population Health Survey 2014: Modifiable risk factors contributing to chronic disease*, State Government of Victoria, Melbourne.

enHealth 2001, *Health Impact Assessment Guidelines*, Commonwealth Department of Health and Aged Care.

enHealth 2012a, *Environmental Health Risk Assessment, Guidelines for assessing human health risks from environmental hazards*, Commonwealth of Australia, Canberra. <http://www.health.gov.au/internet/main/publishing.nsf/content/804F8795BABFB1C7CA256F1900045479/$File/DoHA-EHRA-120910.pdf >.

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

EPHC 2005, *National Dioxins Program - National Action Plan for Addressing Dioxins in Australia*, Environment Protection and Heritage Council.

<http://www.nepc.gov.au/system/files/resources/74b7657d-04ce-b214-d5d7-51dcbce2a231/files/cmgt-rev-national-dioxins-program-national-action-plan-addressing-dioxins-australia-200510.pdf>.

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

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

Harris, P, Harris-Roxas, B., Harris, E. & Kemp, L. 2007, *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.

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

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

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

NEPC 1999 amended 2013a, *Schedule B4, Guideline on Health Risk Assessment Methodology, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <http://scew.gov.au/nepms/assessment-site-contamination>.

NEPC 1999 amended 2013b, *Schedule B7, Guideline on Health-Based Investigation Levels, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <http://scew.gov.au/nepms/assessment-site-contamination>.

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

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

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

NHMRC 2011 updated 2018, *Australian Drinking Water Guidelines 6, Version 3.5 Updated August 2018, National Water Quality Management Strategy*, National Health and Medical Research Council, National Resource Management Ministerial Council, Canberra.

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Calculation of risk: $PM_{2.5}$

A quantitative assessment of risk for these endpoints uses a mathematical relationship between an exposure concentration (ie 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[1]. The calculation of a relative risk based on the change in relative risk exposure concentration from baseline/existing (ie based on incremental impacts from the project) can be calculated on the basis of the following equation (Ostro 2004):

Equation 1 **RR = exp[β(X-X0)]**

Where:
X-X0 = the change in particulate matter concentration to which the population is exposed (µg/m³)
β = regression/slope coefficient, or the slope of the exposure-response function which can also be expressed as the per cent change in response per 1 µg/m³ increase in particulate matter exposure.

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

1 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.

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

Equation 2

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

The assessment of health impacts for a particular population associated with exposure to particulate matter has been undertaken utilising the methodology presented by the WHO (Ostro 2004)[2] where the exposure-response relationships identified have been directly considered on the basis of the approach outlined below.

An additional risk can be calculated as:

Equation 3 **Risk=β x ∆X x B**

Where:
β = slope coefficient relevant to the per cent change in response to a 1 µg/m³ change in exposure
ΔX = change (increment) in exposure concentration in µg/m³ relevant to the project at the point of exposure
B = baseline incidence of a given health effect per person (eg annual mortality rate)

The calculation of the incremental individual risk for relevant health endpoints associated with exposure to particulate matter as outlined by the WHO (Ostro 2004) has considered the following four elements:

- Estimates of the changes in particulate matter exposure levels (ie incremental impacts) due to the project for the relevant modelled scenarios – these have been modelled for the proposed project, with the maximum change from all locations (grid receptors). For this assessment the change in $PM_{2.5}$ relates to the change in annual average air concentrations and the value considered in this assessment is 0.003 µg/m³
- Baseline incidence of the key health endpoints that are relevant to the population exposed – the assessment undertaken has considered the baseline mortality data relevant to the Latrobe Valley (with the highest rate for males, all ages, all causes adopted). The data has been obtained from the Gippsland PHN Population Health Planning Hub, with the mortality

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

rate for males (based on data from 2010 to 2014) for the Latrobe LGA being 774 as an age standardised rate (per 100,000). The rate for females is reported to be 556. This calculation has used the higher value for males in the Latrobe area

- 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 et al. 2002). The study found a relative risk of all-cause mortality of 1.06 per 10µg/m^3 change in $PM_{2.5}$, and that this risk relationship was in the form of an exponential function. It is noted that the exposure response relationship established in this study was re-affirmed in a follow-up study (that included approximately 500,000 participants in the US) (Krewski et al. 2009) and is consistent with findings from California (Ostro et al. 2006). The relationship is also more conservative than a study undertaken in Australia and New Zealand (EPHC 2010).

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

Based on the calculations undertaken the calculated incremental individual risk is $1x10^{-7}$.

Appendix B Methodology and assumptions

B1 Introduction

This appendix presents the methodology and assumptions adopted in the calculation of risk related to the assessment of chronic risks via inhalation or other pathways that may occur following deposition of chemical substances that are persistent.

B2 Chronic toxicity reference values

Approach

The quantitative assessment of potential risks to human health for any substance requires the consideration of the health end-points and where carcinogenicity is identified; the mechanism of action needs to be understood. This will determine whether the chemical substance is considered a threshold or non-threshold chemical substance. A threshold chemical has a concentration below which health effects are not considered to occur. A non-threshold chemical substance is believed to theoretically cause health effects at any concentration, and it is the level of health risk posed by the concentration of the chemical substance that is assessed. The following paragraphs provide further context around these concepts.

For chemical substances that are not carcinogenic, a threshold exists below which there are no adverse effects (for all relevant end-points). The threshold typically adopted in risk calculations (a tolerable daily intake [TDI] or tolerable concentration [TC]) is based on the lowest no observed adverse effect level (NOAEL), typically from animal or human (e.g. occupational) studies, and the application of a number of safety or uncertainty factors. Intakes/exposures lower than the TDI/TC is considered safe, or not associated with an adverse health risk (NHMRC 1999).

Where the chemical substance has the potential for carcinogenic effects the mechanism of action needs to be understood as this defines the way that the dose-response is assessed. Carcinogenic effects are associated with multi-step and multi-mechanism processes that may include genetic damage, altering gene expression and stimulating proliferation of transformed cells. Some carcinogens have the potential to result in genetic (DNA) damage (gene mutation, gene amplification, chromosomal rearrangement) and are termed genotoxic carcinogens. For these carcinogens it is assumed that any exposure may result in one mutation or one DNA damage event that is considered sufficient to initiate the process for the development of cancer sometime during a lifetime (NHMRC 1999). Hence no safe-dose or threshold is assumed and assessment of exposure is based on a linear non-threshold approach using slope factors or unit risk values.

For other (non-genotoxic) carcinogens, while some form of genetic damage (or altered cell growth) is still necessary for cancer to develop, it is not the primary mode of action for these chemical substances. For these chemical substances carcinogenic effects are associated with indirect mechanisms (that do not directly interact with genetic material) where a threshold is believed to exist.

In the case of particulate matter (PM_{10} or $PM_{2.5}$), current health evidence has not been able to find a concentration below which health impacts do not exist. Thus, the quantification of risk for $PM_{2.5}$ follows a non-threshold approach as described in **Appendix A**.

Values adopted

Chronic toxicity reference values (TRVs) associated with inhalation, ingestion and dermal exposures have been adopted from credible peer-reviewed sources as detailed in the NEPM (NEPC 1999 amended 2013a) and enHealth (enHealth 2012a).

For the gaseous pollutants considered in this assessment, only inhalation TRVs have been adopted. For inorganics as well as dioxins and BaP, TRVs relevant to all exposure pathways have been adopted. Background intakes of these pollutants have been estimated on the basis of existing available information as noted.

The assessment of chronic exposures has considered pollutants that are listed under the NEPM (NEPC 2016), namely NO_2 and SO_2, where the assessment requires comparison of the total intake (background plus the project) to the NEPM air criteria, relevant to an annual average. This has been undertaken separately to the other pollutants, and these pollutants have only been assessed on the basis of inhalation exposures.

Tables B1 and B2 present the TRVs adopted for the assessment of chronic health effects associated with exposure to the other pollutants considered in this assessment. **Table B1** presents the threshold TRVs, while **Table B2** presents the non-threshold TRVs.

Table B1: Summary of chronic TRVs adopted for pollutants – threshold effects

Pollutant	Inhalation TRV (mg/m^3)	Oral/dermal TRV (mg/kg/day)	GI absorption factor*	Dermal absorption*	Background intakes (as percentage of TRV)	
					Other sources**	Including natural soil***
Hydrogen chloride (HCl)	0.026 T	NA (gaseous pollutant)			0%	0%
Hydrogen fluoride (HF)	0.029 T	NA (gaseous pollutant)			0%	0%
Ammonia	0.32 T	NA (gaseous pollutant)			0%	0%
Cadmium	0.000005 W	0.0008 W	100%	0	60%	66%
Thallium	0.0028 R	0.0008 U	3%	0	0%	4%
Mercury (as inorganic and elemental)	0.0002 W	0.0006 W	7%	0.001	40%	40%
Antimony	0.0002 U	0.00086 NH	15%	0	0%	4%
Arsenic	0.001 D	0.002 N	100%	0.005	50%	55%
Lead	0.0005 N	0.0035 NH	100%	0	50%	90%
Chromium (Cr VI assumed)	0.0001 U	0.001 A	100%	0	10%	43%
Cobalt	0.0001 W	0.0014 D	100%	0.001	20%	30%
Copper	0.49 R	0.14 W	100%	0	60%	62%
Manganese	0.00015 W	0.14 A	100%	0	50%	54%
Nickel	0.00002 E	0.012 W	100%	0.005	60%	63%
Vanadium	0.0001 A	0.002 D	100%	0	0%	21%
Dioxins and furans	8.05E-09 R	2.3E-09 NH	100%	0.03	54%	54%

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

Pollutant	Inhalation TRV $(mg/m^3)^{-1}$	Oral/dermal TRV $(mg/kg/day)^{-1}$	GI absorption factor*	Dermal absorption*	Background intakes
BaP	0.4 U	0.233 N	100%	0.06	NA for non-threshold risk calculations

Notes for Tables B1 and B2:

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

** Background intakes relate to intakes from inhalation, drinking water and food products. The values adopted based on information provided in the ASC-NEPM (NEPC 1999 amended 2013b) and relevant sources as noted for the TRVs. Gaseous pollutant background intakes are not known and hence for this assessment they have been assumed to be negligible

*** As the background intakes of inorganics as provided within the ASC-NEPM does not include natural soil, calculated intakes associated with ingestion of soil, adopting background concentrations of inorganics in soil from Morwell (maximum value from sites assessed by EPA Victoria in 2014, https://www.epa.vic.gov.au/our-work/monitoring-the-environment/hazelwood-recovery-effort/testing-during-the-hazelwood-fire/soil-testing-data-during-the-fire), has been included. Calculations relevant to these intakes are presented in **Appendix C**

R = No inhalation-specific TRV available, hence inhalation exposures assessed on the basis of route-extrapolation from the oral TRV, as per USEPA guidance (USEPA 2009)

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

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

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

N = Inhalation guideline adopted for lead from the NEPM (NEPC 2016), and arsenic oral/dermal value as adopted in ASC-NEPM (NEPC 1999 amended 2013b). The value adopted for BaP is also consistent with the recommendation provided in the ASC-NEPM

NH = Dioxin value (and background intakes, which includes natural soil) adopted from NHMRC (NHMRC 2002) and Environment Australia (DEH 2005; EPHC 2005), and antimony and lead value consistent with that adopted by NHMRC to assess intakes in drinking water (NHMRC 2011 updated 2018)

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

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

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

B3 Quantification of inhalation exposure

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

This guidance requires the calculation of an exposure concentration which is based on the concentration in air and the time/duration spent in the area of impact. It is not dependent on age or body weight. The following equation outlines the calculation of an inhalation exposure concentration, and **Table B3** provides details on the assumptions adopted in this assessment:

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

Table B3: Inhalation exposure assumptions

Parameter		Value adopted	Basis
Ca	Concentration of chemical substance in air (mg/m^3)	Modelled from facility, adopting the maximum predicted anywhere (all grid receptors) and the maximum from all discrete receptors	Calculations undertaken on the basis of the maximum predicted impacts
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 2012b)
AT	Averaging time (hours)	Threshold = ED x 365 days/year x 24 hours/day Non-threshold = 70 years x 365 days/year x 24 hours/day	As per enHealth (enHealth 2012a) guidance

B4 Multiple pathway exposures

B4.1 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 2012a; USEPA 1989). This approach is presented in the following equation, and parameters adopted in this assessment are presented in **Table B4**:

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

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 B4**:

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

Table B4: Ingestion and dermal exposure assumptions

Parameter		Value adopted		Basis
		Young children	Adults	
C_M	Concentration of chemical substance in media or relevance (soil, fruit and vegetables, eggs, beef or milk) (mg/kg)	Modelled based on deposition of particulates to soil (refer to **Section 4.2**), adopting the maximum from all discrete receptors		Calculations undertaken on the basis of the maximum predicted impacts relevant to areas where multi-pathway exposures may occur
IR_M	Ingestion rate of media			
	Soil (mg/day)	100 mg/day	50 mg/day	Ingestion rate of outdoor soil and dust (tracked or deposited indoors) as per enHealth (enHealth 2012b)
	Fruit and vegetables (kg/day)	0.28 kg/day 85% from aboveground crops 16% from root crops	0.4 kg/day 73% from aboveground crops 27% from root crops	Total fruit and vegetable intakes per day as per ASC NEPM (NEPC 1999 amended 2013b)
	Eggs (kg/day)	0.006 kg/day	0.014 kg/day	Ingestion rate of eggs per day as per enHealth (enHealth 2012b), 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 2012b), 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
	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	100%	100%	Conservative assumption
SA	Surface area of body exposed to soil per day (cm^2/day)	2700	6300	Exposed skin surface area relevant to adults as per ASC NEPM (NEPC 1999 amended 2013b)

Parameter		Value adopted		Basis
		Young children	Adults	
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)
ABSd	Dermal absorption fraction (unitless)	Chemical specific		Refer to **Tables B1 and B2**
CF	Conversion factor			
	Soil	$1x10^{-6}$ to convert mg to kg		Conversion of units relevant to soil ingestion and dermal contact
	Produce	1		No units conversion required for these calculations
BW	Body weight	70	15	As per enHealth (enHealth 2012b) 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 2012b) 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 2012a) guidance

B4.2 Calculation of concentrations in various media

Potential Concentrations in Soil

The potential accumulation of persistent and bioaccumulative chemical substances in soil, which may be the result of deposition from a number of air emissions source, can be estimated using a soil accumulation model (OEHHA 2015; Stevens 1991).

The concentration in soil, which may be the result of deposition following emission of persistent chemical substances, can be calculated using the following equation, with assumptions adopted in this assessment presented in **Table B5**.

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

Table B5: Assumptions adopted to estimate soil concentrations

Parameter		Value adopted		Basis
		Surface soil*	**Agricultural soil***	
DR	Particle deposition rate for accidental release ($mg/m^2/year$)	Modelled for the facility. Adopted maximum deposition rate for discrete receptors		Relevant to areas where multi-pathway exposures may occur
k	Chemical-specific soil-loss constant (1/year) = $\ln(2)/T^{0.5}$	Calculated	Calculated	
$T^{0.5}$	Chemical half-life in soil (years)	Chemical specific	Chemical specific	Default values adopted for pollutants considered as per OEHHA (2015)
t	Accumulation time (years)	70 years	70 years	Default value (OEHHA 2015)
d	Soil mixing depth (m)	0.01 m	0.15 m	Default values (OEHHA 2015)
ρ	Soil bulk-density (g/m^3)	1600000	1600000	Default for fill material (CRC CARE 2011)
1000	Conversion from g to kg	Default conversion of units		

* Surface soil values adopted for the assessment of direct contact exposures. All other exposures including produce and meat/milk intakes utilise soil concentrations calculated for agricultural intakes (OEHHA 2015)

Homegrown fruit and vegetables

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

The potential concentration of persistent chemical substances that may be present within the plant following atmospheric deposition can be estimated using the following equation (Stevens 1991), with the parameters and assumptions adopted outlined in **Table B6**:

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

The potential uptake of persistent chemical substances into edible crops via the roots can be estimated using the following equation (OEHHA 2015; USEPA 2005), with the parameters and assumptions adopted outlined in **Table B6:**

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

Table B6: Assumptions adopted to estimate concentration in fruit and vegetables

Parameter		Value adopted	Basis
DR	Particle deposition rate for accidental release (mg/m^2/day)	Modelled for the facility. Adopted maximum deposition rate for discrete receptors	Relevant to areas where multi-pathway exposures may occur
F	Fraction for the surface area of plant (unitless)	0.051	Relevant to aboveground exposed crops as per Stevens (1991) and OEHHA (OEHHA 2012)
k	Chemical-specific loss constant for particles on plants (1/days) = ln(2)/$T^{0.5}$	calculated	
$T^{0.5}$	Chemical half-life on plant (day)	14 days	Weathering of particulates on plant surfaces does occur and in the absence of measured data, it is generally assumed that organics deposited onto the outer portion of plant surfaces have a weathering half life of 14 days (Stevens, 1991)
t	Deposition time or length of growing season (days)	70 days	Relevant to aboveground crops based on the value relevant to tomatoes, consistent with the value adopted by Stevens (1991)
Y	Crop yield (kg/m^2)	2 kg/m^2	Value for aboveground crops (OEHHA 2015)
Cs	Concentration of pollutant in soil (mg/kg)	Calculated value for agricultural soil	Calculated as described above and assumptions in **Table B5**
RUF	Root uptake factor (unitless)	Chemical specific value adopted	Root uptake factors from RAIS (RAIS) (soil to wet weight of plant)

Eggs, beef and milk

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

The concentration (C_P) calculated in eggs, beef or milk is calculated using the following equation (OEHHA 2015), with parameters and assumptions adopted presented in **Table B7**:

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

Table B7: Assumptions adopted to estimate concentration in animal produce

Parameter		Value adopted	Basis
FI	Fraction of grain/crop ingested by animals each day derived from the property (unitless)	100%	Assume all pasture/crops ingested by chickens and cows are grown on the property
IR_C	Ingestion rate of pasture/crops by each animal considered (kg/day)		
	Chickens	0.12 kg/day	Ingestion rate from OEHHA (2015)
	Beef cattle	9 kg/day	Ingestion rate from OEHHA (2015)
	Lactating cattle	22 kg/day	Ingestion rate for lactating cattle from OEHHA (2015)
C	Concentration of pollutant in crops consumed by animals (mg/kg)	Assume equal to that calculated in aboveground produce	Calculated as described above with assumptions in **Table B6**
IR_S	Ingestion rate of soil by animals each day (kg/day)		
	Chickens	0.0024 kg/day	Based on data from OEHHA 2015 (2% total produce intakes from soil)
	Beef cattle	0.45 kg/day	Based on data from OEHHA 2015 (5% total produce intakes from soil from pasture)
	Lactating cattle	1.1 kg/day	Based on data from OEHHA 2015 (5% total produce intakes from soil from pasture)
Cs	Concentration of pollutant in soil (mg/kg)	Calculated value for agricultural soil	Calculated as described above and assumptions in **Table B5**
B	Bioavailability of soil ingested (unitless)	100%	Conservative assumption
TF_P	Transfer factor for the produce of interest		
	Eggs	Chemical specific	Transfer factors adopted from OEHHA (2015), with the exception of chromium where the value was derived from an earlier OEHHA (OEHHA 2003) evaluation and the value for antimony has been calculated from a fat transfer factor as per OEHHA (OEHHA 2012)
	Beef	Chemical specific	Transfer factors adopted from OEHHA (2015) and RAIS
	Milk	Chemical specific	Transfer factors adopted from OEHHA (2015) and RAIS

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

Appendix C Risk calculations

Calculation of background intakes from natural soil

Exposure to Chemicals via Incidental Ingestion of Soil

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

Parameters Relevant to Quantification of Exposure by Young Children		
Ingestion Rate (IRs, mg/day)	100	Assumed daily soil ingestion rate for young children, enHealth (2012)
Fraction Ingested from Source (FI, unitless)	100%	Compound-specific as noted below
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	5	Duration as young child
Body Weight (BW, kg)	15	As per enHealth 2012 - mean for children aged 2-3 years
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)	1825	USEPA 1989 and CSMS 1996

Key Chemical	Toxicity Data				Bioavailability (%)	Background soil concnetration - Morwell	Daily Intake			
	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)	Chronic Hazard Quotient (unitless)	Intake as % TDI
Cadmium		8.0E-04		8.0E-04	100%	6.8	3.2E-06	4.5E-05	0.057	5.7%
Thallium		8.0E-04		8.0E-04	100%	5	2.4E-06	3.3E-05	0.042	4.2%
Mercury (as inorganic and ele		6.0E-04		6.0E-04	100%	0.11	5.2E-08	7.3E-07	0.00122	0.1%
Antimony		8.6E-04		8.6E-04	100%	5	2.4E-06	3.3E-05	0.039	3.9%
Arsenic		2.0E-03		2.0E-03	100%	14	6.7E-06	9.3E-05	0.047	4.7%
Lead		3.5E-03		3.5E-03	100%	210	1.0E-04	1.4E-03	0.40	40.0%
Chromium (Cr VI assumed)		1.0E-03		1.0E-03	100%	50	2.4E-05	3.3E-04	0.33	33.3%
Cobalt		1.4E-03		1.4E-03	100%	20	9.5E-06	1.3E-04	0.095	9.5%
Copper		1.4E-01		1.4E-01	100%	390	1.9E-04	2.6E-03	0.0186	1.9%
Manganese		1.4E-01		1.4E-01	100%	910	4.3E-04	6.1E-03	0.043	4.3%
Nickel		1.2E-02		1.2E-02	100%	52	2.5E-05	3.5E-04	0.029	2.9%
Vanadium		2.0E-03		2.0E-03	100%	62	3.0E-05	4.1E-04	0.21	20.7%

Soil concentrations are maximum reported by EPA Victoria for locations in Morwell (sampled after the Hazelwood fire). Values in red are the analytical limit of reporting as the analyte was not detected

Inhalation exposures

Inhalation - gases and fine particulates

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

Parameters Relevant to Quantification of Community Exposures - Residents		
Exposure Time at Home (ET, hr/day)	24	Assume residents at home or on property 24 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Assume resident at the same property
Exposure Frequency (EF, days/yr)	365	Days at home, as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	35	As per NEPM (1999 amended 2013)
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	306600	US EPA 2009

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk $(mg/m^3)^{-1}$	**Chronic TC Air** (mg/m^3)	**Background Intake (% Chronic TC)**	**Chronic TC Allowable for Assessment (TC-Background)** (mg/m^3)	**Estimated Concentration in Air - Maximum anywhere (Ca)** (mg/m^3)	**Inhalation Exposure Concentration -** NonThreshold (mg/m^3)	**Inhalation Exposure Concentration -** Threshold (mg/m^3)	**Non-Threshold Risk** (unitless)	**% Total Risk**	**Chronic Hazard Quotient** (unitless)	**% Total HI**
Nitrogen dioxide (NO2)	0.0E+00	5.6E-02	0%	5.6E-02	1.3E-02	6.7E-03	1.3E-02	--		0.24	
Sulfur dioxide (SO2)	0.0E+00	5.0E-02	0%	5.0E-02	4.6E-03	2.3E-03	4.6E-03	--		0.093	
Hydrogen chloride (HCl)	0.0E+00	2.6E-02	0%	2.6E-02	9.2E-05	4.6E-05	9.2E-05	--		0.0036	1%
Hydrogen fluoride (HF)	0.0E+00	2.9E-02	0%	2.9E-02	4.9E-06	2.5E-06	4.9E-06	--		0.00017	0%
Ammonia	0.0E+00	3.2E-01	0%	3.2E-01	4.2E-04	2.1E-04	4.2E-04	--		0.0013	0%
Cadmium	0.0E+00	5.0E-06	66%	1.7E-06	3.2E-08	1.6E-08	3.2E-08	--		0.019	7%
Thallium	0.0E+00	2.8E-03	4%	2.7E-03	3.2E-08	1.6E-08	3.2E-08	--		0.000012	0%
Mercury (as inorganic and elementa	0.0E+00	2.0E-04	40%	1.2E-04	5.1E-07	2.5E-07	5.1E-07	--		0.0042	2%
Antimony	0.0E+00	2.0E-04	4%	1.9E-04	1.4E-06	7.1E-07	1.4E-06	--		0.0073	3%
Arsenic	0.0E+00	1.0E-03	55%	4.5E-04	1.4E-08	7.1E-09	1.4E-08	--		0.000031	0%
Lead	0.0E+00	5.0E-04	90%	5.0E-05	6.2E-07	3.1E-07	6.2E-07	--		0.0124	4%
Chromium (Cr VI assumed)	0.0E+00	1.0E-04	43%	5.7E-05	2.8E-08	1.4E-08	2.8E-08	--		0.00050	0%
Cobalt	0.0E+00	1.0E-04	30%	7.0E-05	2.8E-08	1.4E-08	2.8E-08	--		0.00040	0%
Copper	0.0E+00	4.9E-01	62%	1.9E-01	1.4E-06	7.1E-07	1.4E-06	--		0.0000075	0%
Manganese	0.0E+00	1.5E-04	54%	6.9E-05	1.4E-06	7.1E-07	1.4E-06	--		0.021	7%
Nickel	0.0E+00	2.0E-05	63%	7.4E-06	1.4E-06	7.1E-07	1.4E-06	--		0.19	68%
Vanadium	0.0E+00	1.0E-04	21%	7.9E-05	1.4E-06	7.1E-07	1.4E-06	--		0.018	6%
Dioxin	0.0E+00	8.1E-09	54%	3.7E-09	1.4E-12	7.1E-13	1.4E-12	--		0.00038	0%
BaP	4.0E-01	0.0E+00	0%	0.0E+00	1.9E-07	9.4E-08	1.9E-07	3.8E-8	100%	--	
							TOTAL	**3.8E-08**		**0.28**	

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 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)	
Nitrogen dioxide (NO2)	0.0E+00	5.6E-02	0%	5.6E-02	1.3E-02	6.6E-03	1.3E-02	--		0.23	
Sulfur dioxide (SO2)	0.0E+00	5.0E-02	0%	5.0E-02	4.6E-03	2.3E-03	4.6E-03	--		0.092	
Hydrogen chloride (HCl)	0.0E+00	2.6E-02	0%	2.6E-02	6.8E-05	3.4E-05	6.8E-05	--		0.0026	1%
Hydrogen fluoride (HF)	0.0E+00	2.9E-02	0%	2.9E-02	3.6E-06	1.8E-06	3.6E-06	--		0.00012	0%
Ammonia	0.0E+00	3.2E-01	0%	3.2E-01	3.1E-04	1.5E-04	3.1E-04	--		0.0010	0%
Cadmium	0.0E+00	5.0E-06	66%	1.7E-06	3.1E-08	1.5E-08	3.1E-08	--		0.0180	9%
Thallium	0.0E+00	2.8E-03	4%	2.7E-03	3.1E-08	1.5E-08	3.1E-08	--		0.000012	0%
Mercury (as inorganic and elementa	0.0E+00	2.0E-04	40%	1.2E-04	3.7E-07	1.9E-07	3.7E-07	--		0.0031	1%
Antimony	0.0E+00	2.0E-04	4%	1.9E-04	1.0E-06	5.2E-07	1.0E-06	--		0.0054	3%
Arsenic	0.0E+00	1.0E-03	55%	4.5E-04	1.0E-08	5.2E-09	1.0E-08	--		0.000023	0%
Lead	0.0E+00	5.0E-04	90%	5.0E-05	4.5E-07	2.3E-07	4.5E-07	--		0.0091	4%
Chromium (Cr VI assumed)	0.0E+00	1.0E-04	43%	5.7E-05	2.1E-08	1.0E-08	2.1E-08	--		0.00036	0%
Cobalt	0.0E+00	1.0E-04	30%	7.0E-05	2.1E-08	1.0E-08	2.1E-08	--		0.00029	0%
Copper	0.0E+00	4.9E-01	62%	1.9E-01	1.0E-06	5.2E-07	1.0E-06	--		0.0000055	0%
Manganese	0.0E+00	1.5E-04	54%	6.9E-05	1.0E-06	5.2E-07	1.0E-06	--		0.015	7%
Nickel	0.0E+00	2.0E-05	63%	7.4E-06	1.0E-06	5.2E-07	1.0E-06	--		0.14	67%
Vanadium	0.0E+00	1.0E-04	21%	7.9E-05	1.0E-06	5.2E-07	1.0E-06	--		0.013	6%
Dioxin	0.0E+00	8.1E-09	54%	3.7E-09	1.0E-12	5.2E-13	1.0E-12	--		0.00028	0%
BaP	4.0E-01	0.0E+00	0%	0.0E+00	1.4E-07	6.9E-08	1.4E-07	2.7E-8	100%	--	
							TOTAL	**2.7E-08**		**0.21**	

Soil exposures

Calculation of Concentrations in Soil

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

where:

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

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

Chemical-specific Inputs and calculations - maximum 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
Cadmium	273973	2.5E-06	0.0380	1.7E-01	1.1E-02
Thallium	273973	2.5E-06	0.0380	1.7E-01	1.1E-02
Mercury (as inorganic and eleme	273973	2.5E-06	0.4500	2.0E+00	1.3E-01
Antimony	273973	2.5E-06	1.3000	5.7E+00	3.8E-01
Arsenic	273973	2.5E-06	0.0130	5.7E-02	3.8E-03
Lead	273973	2.5E-06	0.5500	2.4E+00	1.6E-01
Chromium (Cr VI assumed)	273973	2.5E-06	0.0250	1.1E-01	7.3E-03
Cobalt	273973	2.5E-06	0.0250	1.1E-01	7.3E-03
Copper	273973	2.5E-06	1.3000	5.7E+00	3.8E-01
Manganese	273973	2.5E-06	1.3000	5.7E+00	3.8E-01
Nickel	273973	2.5E-06	1.3000	5.7E+00	3.8E-01
Vanadium	273973	2.5E-06	1.3000	5.7E+00	3.8E-01
Dioxin		0.069	0.0000	1.2E-06	7.8E-08
BaP	1.18	0.588	0.1700	1.8E-02	1.2E-03

Half-life in soil: dioxin loss constant from Lowe et al (1991) and half-life for remainder from OEHHA (2015)

Exposure to Chemicals via Incidental Ingestion of Soil

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

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

Maximum - Discrete receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium		8.0E-04	66%	2.7E-04	100%	1.7E-01	4.9E-08	1.2E-07	--		0.00043	2%
Thallium		8.0E-04	4%	7.7E-04	100%	1.7E-01	4.9E-08	1.2E-07	--		0.00015	1%
Mercury (as inorganic and ele		6.0E-04	40%	3.6E-04	100%	2.0E+00	5.8E-07	1.4E-06	--		0.0039	21%
Antimony		8.6E-04	4%	8.3E-04	100%	5.7E+00	1.7E-06	4.1E-06	--		0.0049	26%
Arsenic		2.0E-03	55%	9.1E-04	100%	5.7E-02	1.7E-08	4.1E-08	--		0.000045	0%
Lead		3.5E-03	90%	3.5E-04	100%	2.4E+00	7.1E-07	1.7E-06	--		0.0049	26%
Chromium (Cr VI assumed)		1.0E-03	43%	5.7E-04	100%	1.1E-01	3.2E-08	7.8E-08	--		0.00014	1%
Cobalt		1.4E-03	30%	9.9E-04	100%	1.1E-01	3.2E-08	7.8E-08	--		0.000079	0%
Copper		1.4E-01	62%	5.3E-02	100%	5.7E+00	1.7E-06	4.1E-06	--		0.000076	0%
Manganese		1.4E-01	54%	6.4E-02	100%	5.7E+00	1.7E-06	4.1E-06	--		0.000064	0%
Nickel		1.2E-02	63%	4.5E-03	100%	5.7E+00	1.7E-06	4.1E-06	--		0.00091	5%
Vanadium		2.0E-03	21%	1.6E-03	100%	5.7E+00	1.7E-06	4.1E-06	--		0.0026	13%
Dioxin		2.3E-09	54%	1.1E-09	100%	1.2E-06	3.4E-13	8.3E-13	--		0.00079	4%
BaP	2.3E-01				100%	1.8E-02	5.3E-09	1.3E-08	1.2E-9	11%	--	
								TOTAL	1.2E-9		0.019	

Exposure to Chemicals via Incidental Ingestion of Soil

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

Parameters Relevant to Quantification of Exposure by Young Children		
Ingestion Rate (IRs, mg/day)	100	Assumed daily soil ingestion rate for young children, enHealth (2012)
Fraction Ingested from Source (FI, unitless)	100%	Compound-specific as noted below
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum - Discrete receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium		8.0E-04	66%	2.7E-04	100%	1.7E-01	9.5E-08	1.1E-06	--		0.0040	2%
Thallium		8.0E-04	4%	7.7E-04	100%	1.7E-01	9.5E-08	1.1E-06	--		0.0014	1%
Mercury (as inorganic and el		6.0E-04	40%	3.6E-04	100%	2.0E+00	1.1E-06	1.3E-05	--		0.037	21%
Antimony		8.6E-04	4%	8.3E-04	100%	5.7E+00	3.2E-06	3.8E-05	--		0.046	26%
Arsenic		2.0E-03	55%	9.1E-04	100%	5.7E-02	3.2E-08	3.8E-07	--		0.00042	0%
Lead		3.5E-03	90%	3.5E-04	100%	2.4E+00	1.4E-06	1.6E-05	--		0.046	26%
Chromium (Cr VI assumed)		1.0E-03	43%	5.7E-04	100%	1.1E-01	6.2E-08	7.3E-07	--		0.00129	1%
Cobalt		1.4E-03	30%	9.9E-04	100%	1.1E-01	6.2E-08	7.3E-07	--		0.00074	0%
Copper		1.4E-01	62%	5.3E-02	100%	5.7E+00	3.2E-06	3.8E-05	--		0.00071	0%
Manganese		1.4E-01	54%	6.4E-02	100%	5.7E+00	3.2E-06	3.8E-05	--		0.00059	0%
Nickel		1.2E-02	63%	4.5E-03	100%	5.7E+00	3.2E-06	3.8E-05	--		0.0085	5%
Vanadium		2.0E-03	21%	1.6E-03	100%	5.7E+00	3.2E-06	3.8E-05	--		0.024	13%
Dioxin		2.3E-09	54%	1.1E-09	100%	1.2E-06	6.6E-13	7.8E-12	--		0.0073	4%
BaP	2.3E-01				100%	1.8E-02	1.0E-08	1.2E-07	2.4E-9	11%	--	
								TOTAL	2.4E-9		0.18	

Dermal Exposure to Chemicals via Contact with Soil

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

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

Maximum - Discrete receptors

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor $(mg/kg\text{-}day)^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)	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
Cadmium		8.0E-04	66%	2.7E-04		1.7E-01			--		--	
Mercury (as inorganic and eleme		4.2E-05	40%	2.5E-05	0.001	2.0E+00	3.7E-08	8.9E-08	--		0.0035	66%
Antimony		1.3E-04	4%	1.2E-04		5.7E+00			--		--	
Arsenic		2.0E-03	55%	9.1E-04	0.005	5.7E-02	5.3E-09	1.3E-08	--		0.000014	0%
Lead		3.5E-03	90%	3.5E-04		2.4E+00			--		--	
Chromium (Cr VI assumed)		1.0E-03	43%	5.7E-04		1.1E-01			--		--	
Cobalt		1.4E-03	30%	9.9E-04	0.001	1.1E-01	2.0E-09	4.9E-09	--		0.0000050	0%
Copper		1.4E-01	62%	5.3E-02		5.7E+00			--		--	
Manganese		1.4E-01	54%	6.4E-02		5.7E+00			--		--	
Nickel		1.2E-02	63%	4.5E-03	0.005	5.7E+00	5.3E-07	1.3E-06	--		0.00029	5%
Vanadium		2.0E-03	21%	1.6E-03		5.7E+00			--		--	
Dioxin		2.3E-09	54%	1.1E-09	0.03	1.2E-06	6.5E-13	1.6E-12	--		0.0015	28%
BaP	2.3E-01				0.06	1.8E-02	2.0E-08	4.9E-08	4.7E-9	100%	--	

TOTAL	**4.7E-9**	**0.0053**

Dermal Exposure to Chemicals via Contact with Soil

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

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

Maximum - Discrete receptors

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)	Dermal Absorption (ABS)	(mg/kg)	Non-Threshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium		8.0E-04	66%	2.7E-04		1.7E-01			--		--	
Thallium		2.1E-05	4%	2.0E-05		1.7E-01			--		--	
Mercury (as inorganic and eleme		4.2E-05	40%	2.5E-05	0.001	2.0E+00	1.5E-08	1.8E-07	--		0.0070	66%
Antimony		1.3E-04	4%	1.2E-04		5.7E+00			--		--	
Arsenic		2.0E-03	55%	9.1E-04	0.005	5.7E-02	2.2E-09	2.6E-08	--		0.000028	0%
Lead		3.5E-03	90%	3.5E-04		2.4E+00			--		--	
Chromium (Cr VI assumed)		1.0E-03	43%	5.7E-04		1.1E-01			--		--	
Cobalt		1.4E-03	30%	9.9E-04	0.001	1.1E-01	8.4E-10	9.8E-09	--		0.0000100	0%
Copper		1.4E-01	62%	5.3E-02		5.7E+00			--		--	
Manganese		1.4E-01	54%	6.4E-02		5.7E+00			--		--	
Nickel		1.2E-02	63%	4.5E-03	0.005	5.7E+00	2.2E-07	2.6E-06	--		0.00057	5%
Vanadium		2.0E-03	21%	1.6E-03		5.7E+00			--		--	
Dioxin		2.3E-09	54%	1.1E-09	0.03	1.2E-06	2.7E-13	3.1E-12	--		0.0030	28%
BaP	2.3E-01				0.06	1.8E-02	8.4E-09	9.8E-08	1.9E-9	100%	--	
								TOTAL	**1.9E-9**		**0.011**	

Homegrown fruit and vegetables

Calculation of Concentrations in Plants

ref: Stevens B. (1991)

Uptake Due to Deposition in Aboveground Crops	Uptake via Roots from Soil
$$C_p = \frac{DR \bullet F \bullet \left[1 - e^{-k \bullet t}\right]}{Y \bullet k}$$ (mg/kg plant – wet weight)	$$C_{rp} = C_s \bullet RUF$$ (mg/kg plant – wet weight)
where: DR= Particle deposition rate for accidental release (mg/m^2/day) F= Fraction for the surface area of plant (unitless) k= Chemical-specific soil-loss constant (1/years) = ln(2)/$T_{0.5}$ $T_{0.5}$= Chemical half-life as particulate on plant (days) t= Deposition time (days) Y= Crop yield (kg/m^2)	**where**: Cs = Concentration of persistent chemical in soil assuming 15cm mixing depth within gardens, calculated using Soil Equation for each chemical assessed (mg/kg) RUF = Root uptake factor which differs for each Chemical (unitless)

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

Chemical-specific Inputs and calculations - Maximum discrete receptors

Chemical	Half-life in plant ($T_{0.5}$) days	Loss constant (k) per day	Deposition Rate (DR) mg/m^2/day	Aboveground Produce Concentration via Deposition mg/kg ww	Root Uptake Factor (RUF) unitless	Soil Concentration (Cs) mg/kg	Below Ground Produce Concentration mg/kg ww
Cadmium	14	0.05	0.0001041	5.2E-05	0.125	1.1E-02	1.4E-03
Thallium	14	0.05	0.0001041	5.2E-05	0.001	1.1E-02	1.1E-05
Mercury (as inorganic and eleme	14	0.05	0.0012329	6.2E-04	0.225	1.3E-01	3.0E-02
Antimony	14	0.05	0.0035616	1.8E-03	0.05	3.8E-01	1.9E-02
Arsenic	14	0.05	0.0000356	1.8E-05	0.04	3.8E-03	1.5E-04
Lead	14	0.05	0.0015068	7.5E-04	0.0113	1.6E-01	1.8E-03
Chromium (Cr VI assumed)	14	0.05	0.0000685	3.4E-05	0.00188	7.3E-03	1.4E-05
Cobalt	14	0.05	0.0000685	3.4E-05	0.005	7.3E-03	3.6E-05
Copper	14	0.05	0.0035616	1.8E-03	0.1	3.8E-01	3.8E-02
Manganese	14	0.05	0.0035616	1.8E-03	0.0625	3.8E-01	2.4E-02
Nickel	14	0.05	0.0035616	1.8E-03	0.015	3.8E-01	5.7E-03
Vanadium	14	0.05	0.0035616	1.8E-03	0.00138	3.8E-01	5.2E-04
Dioxin	14	0.05	0.0000000	1.8E-09	0.000876	7.8E-08	6.8E-11
BaP	14	0.05	0.0004658	2.3E-04	0.00214	1.2E-03	2.6E-06

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

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables

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

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Produce (IRp) (kg/day)	0.4	Total fruit and vegetable consumption rate for adults as per NEPM (2013)
Proportion of total intake from aboveground crops (%A)	73%	Proportions as per NEPM (2013)
Proportion of total intake from root crops (%R)	27%	Proportions as per NEPM (2013)
Fraction ingested that is homegrown (%)	35%	Assumed for rural areas (higher than typical default)
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum - Discrete receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)				NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium		8.0E-04	66%	2.7E-04	100%	5.2E-05	1.4E-03	3.4E-07	8.2E-07	--		0.0030	4%
Thallium		8.0E-04	4%	7.7E-04	100%	5.2E-05	1.1E-05	3.4E-08	8.2E-08	--		0.00011	0%
Mercury (as inorganic and elementa		6.0E-04	40%	3.6E-04	100%	6.2E-04	3.0E-02	7.0E-06	1.7E-05	--		0.047	60%
Antimony		8.6E-04	4%	8.3E-04	100%	1.8E-03	1.9E-02	5.3E-06	1.3E-05	--		0.016	20%
Arsenic		2.0E-03	55%	9.1E-04	100%	1.8E-05	1.5E-04	4.5E-08	1.1E-07	--		0.00012	0%
Lead		3.5E-03	90%	3.5E-04	100%	7.5E-04	1.8E-03	8.6E-07	2.1E-06	--		0.0059	8%
Chromium (Cr VI assumed)		1.0E-03	43%	5.7E-04	100%	3.4E-05	1.4E-05	2.4E-08	5.7E-08	--		0.000101	0%
Cobalt		1.4E-03	30%	9.9E-04	100%	3.4E-05	3.6E-05	2.9E-08	7.0E-08	--		0.000071	0%
Copper		1.4E-01	62%	5.3E-02	100%	1.8E-03	3.8E-02	9.6E-06	2.3E-05	--		0.00043	1%
Manganese		1.4E-01	54%	6.4E-02	100%	1.8E-03	2.4E-02	6.4E-06	1.5E-05	--		0.00024	0%
Nickel		1.2E-02	63%	4.5E-03	100%	1.8E-03	5.7E-03	2.3E-06	5.7E-06	--		0.0013	2%
Vanadium		2.0E-03	21%	1.6E-03	100%	1.8E-03	5.2E-04	1.2E-06	2.9E-06	--		0.0018	2%
Dioxin		2.3E-09	54%	1.1E-09	100%	1.8E-09	6.8E-11	1.1E-12	2.6E-12	--		0.0025	3%
BaP	2.3E-01				100%	2.3E-04	2.6E-06	1.4E-07	3.4E-07	3.3E-8	11%	--	
									TOTAL	3.3E-8		0.078	

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 Young Children		
Ingestion Rate of Produce (IRp) (kg/day)	0.28	Total fruit and vegetable consumption rate for children as per NEPM (2013)
Proportion of total intake from aboveground crops (%A)	84%	Proportions as per NEPM (2013)
Proportion of total intake from root crops (%R)	16%	Proportions as per NEPM (2013)
Fraction ingested that is homegrown (%)	35%	Assumed for rural areas (higher than typical default)
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum - Discrete receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium		8.0E-04	66%	2.7E-04	100%	5.2E-05	1.4E-03	1.5E-07	1.7E-06	--		0.0063	4%
Thallium		8.0E-04	4%	7.7E-04	100%	5.2E-05	1.1E-05	2.5E-08	3.0E-07	--		0.00039	0%
Mercury (as inorganic and elementa		6.0E-04	40%	3.6E-04	100%	6.2E-04	3.0E-02	2.9E-06	3.4E-05	--		0.095	54%
Antimony		8.6E-04	4%	8.3E-04	100%	1.8E-03	1.9E-02	2.5E-06	3.0E-05	--		0.036	20%
Arsenic		2.0E-03	55%	9.1E-04	100%	1.8E-05	1.5E-04	2.2E-08	2.6E-07	--		0.00028	0%
Lead		3.5E-03	90%	3.5E-04	100%	7.5E-04	1.8E-03	5.2E-07	6.0E-06	--		0.017	10%
Chromium (Cr VI assumed)		1.0E-03	43%	5.7E-04	100%	3.4E-05	1.4E-05	1.7E-08	2.0E-07	--		0.00036	0%
Cobalt		1.4E-03	30%	9.9E-04	100%	3.4E-05	3.6E-05	1.9E-08	2.3E-07	--		0.00023	0%
Copper		1.4E-01	62%	5.3E-02	100%	1.8E-03	3.8E-02	4.2E-06	4.9E-05	--		0.00092	1%
Manganese		1.4E-01	54%	6.4E-02	100%	1.8E-03	2.4E-02	3.0E-06	3.5E-05	--		0.00054	0%
Nickel		1.2E-02	63%	4.5E-03	100%	1.8E-03	5.7E-03	1.3E-06	1.6E-05	--		0.0035	2%
Vanadium		2.0E-03	21%	1.6E-03	100%	1.8E-03	5.2E-04	8.8E-07	1.0E-05	--		0.0065	4%
Dioxin		2.3E-09	54%	1.1E-09	100%	1.8E-09	6.8E-11	8.4E-13	9.8E-12	--		0.0093	5%
BaP	2.3E-01				100%	2.3E-04	2.6E-06	1.1E-07	1.3E-06	2.6E-8	11%	--	
									TOTAL	**2.6E-8**		**0.18**	

Ingestion of eggs, beef and milk

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%	

Chemical-specific Inputs and calculations - Maximum discrete 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	
Cadmium	5.2E-05	1.1E-02	1.0E-02	3.3E-07	
Thallium	5.2E-05	1.1E-02	1.7E-02	5.5E-07	
Mercury (as inorganic and eleme	6.2E-04	1.3E-01	8.0E-01	3.1E-04	
Antimony	1.8E-03	3.8E-01	4.2E-04	4.7E-07	Calculated from fat transfer factors TF = $10^{logTFfat}$ x 0.08
Arsenic	1.8E-05	3.8E-03	7.0E-02	7.9E-07	
Lead	7.5E-04	1.6E-01	4.0E-02	1.9E-05	
Chromium (Cr VI assumed)	3.4E-05	7.3E-03	9.2E-03	2.0E-07	OEHHA (2003)
Cobalt	3.4E-05	7.3E-03		0.0E+00	
Copper	1.8E-03	3.8E-01		0.0E+00	
Manganese	1.8E-03	3.8E-01		0.0E+00	
Nickel	1.8E-03	3.8E-01	2.0E-02	2.2E-05	
Vanadium	1.8E-03	3.8E-01		0.0E+00	
Dioxin	1.8E-09	7.8E-08	1.0E+01	4.0E-09	
BaP	2.3E-04	1.2E-03	3.0E-03	9.2E-08	

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Eggs

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

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Eggs (IRE) (kg/day)	0.014	Ingestion rate of eggs relevant for adults as per enHealth (2012)
Fraction ingested that is homegrown (%)	200%	Assumed for rural areas where a higher rate of egg ingestion expected
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum - Discrete receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium		8.0E-04	66%	2.7E-04	100%	3.3E-07	5.4E-11	1.3E-10	--		0.00000048	0%
Thallium		8.0E-04	4%	7.7E-04	100%	5.5E-07	9.1E-11	2.2E-10	--		0.00000029	0%
Mercury (as inorganic and elementa		6.0E-04	40%	3.6E-04	100%	3.1E-04	5.2E-08	1.2E-07	--		0.00035	2%
Antimony		8.6E-04	4%	8.3E-04	100%	4.7E-07	7.8E-11	1.9E-10	--		0.00000023	0%
Arsenic		2.0E-03	55%	9.1E-04	100%	7.9E-07	1.3E-10	3.1E-10	--		0.00000035	0%
Lead		3.5E-03	90%	3.5E-04	100%	1.9E-05	3.1E-09	7.6E-09	--		0.000022	1%
Chromium (Cr VI assumed)		1.0E-03	43%	5.7E-04	100%	2.0E-07	3.3E-11	7.9E-11	--		0.00000014	0%
Cobalt		1.4E-03	30%	9.9E-04	100%				--		--	
Copper		1.4E-01	62%	5.3E-02	100%				--		--	
Manganese		1.4E-01	54%	6.4E-02	100%				--		--	
Nickel		1.2E-02	63%	4.5E-03	100%	2.2E-05	3.7E-09	9.0E-09	--		0.0000020	0%
Vanadium		2.0E-03	21%	1.6E-03	100%				--		--	
Dioxin		2.3E-09	54%	1.1E-09	100%	4.0E-09	6.6E-13	1.6E-12	--		0.0015	80%
BaP	2.3E-01				100%	9.2E-08	1.5E-11	3.7E-11	3.6E-12	11%	--	
								TOTAL	3.6E-12		0.0019	

Exposure to Chemicals via Ingestion of Eggs

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

Parameters Relevant to Quantification of Exposure by Young Children		
Ingestion Rate of Eggs (IRE) (kg/day)	0.006	Ingestion rate of eggs relevant for young children as per enHealth (2012)
Fraction ingested that is homegrown (%)	200%	Assumed for rural areas where a higher rate of egg ingestion expected
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum - Discrete receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium		8.0E-04	66%	2.7E-04	100%	3.3E-07	2.3E-11	2.6E-10	--		0.00000096	0%
Thallium		8.0E-04	4%	7.7E-04	100%	5.5E-07	3.8E-11	4.4E-10	--		0.00000058	0%
Mercury (as inorganic and elementa		6.0E-04	40%	3.6E-04	100%	3.1E-04	2.1E-08	2.5E-07	--		0.00069	18%
Antimony		8.6E-04	4%	8.3E-04	100%	4.7E-07	3.2E-11	3.8E-10	--		0.00000046	0%
Arsenic		2.0E-03	55%	9.1E-04	100%	7.9E-07	5.4E-11	6.3E-10	--		0.00000069	0%
Lead		3.5E-03	90%	3.5E-04	100%	1.9E-05	1.3E-09	1.5E-08	--		0.000043	1%
Chromium (Cr VI assumed)		1.0E-03	43%	5.7E-04	100%	2.0E-07	1.4E-11	1.6E-10	--		0.00000028	0%
Cobalt		1.4E-03	30%	9.9E-04	100%				--		--	
Copper		1.4E-01	62%	5.3E-02	100%				--		--	
Manganese		1.4E-01	54%	6.4E-02	100%				--		--	
Nickel		1.2E-02	63%	4.5E-03	100%	2.2E-05	1.5E-09	1.8E-08	--		0.0000040	0%
Vanadium		2.0E-03	21%	1.6E-03	100%				--		--	
Dioxin		2.3E-09	54%	1.1E-09	100%	4.0E-09	2.7E-13	3.2E-12	--		0.0030	80%
BaP	2.3E-01				100%	9.2E-08	6.3E-12	7.4E-11	1.5E-12	11%	--	
								TOTAL	1.5E-12		0.0038	

Calculation of Concentrations in Homegrown Beef

Uptake in to beef meat

$$C_E=(FI \times IR_C \times C+IR_S \times C_S \times B) \times TF_B$$ (mg/kg beef – wet weight)

where:
FI = Fraction of grain/crop ingested by cattle each day (unitless)
IRc = Ingestion rate of grain/crop by cattle each day (kg/day)
C = Concentration of chemical in grain/crop eaten by cattle (mg/kg)
IRs = Ingestion rate of soil by cattle each day (kg/day)
Cs = Concentration in soil the cattle ingest (mg/kg)
B = Bioavailability of soil ingested by cattle (%)
TFE = Transfer factor from ingestion to beef (day/kg)

General Parameters	Units	Value	
FI (fraction of crops ingested from property)		1	Assume 100% of pasture consumed by cattle is grown in the same soil
IRc (ingestion rate of crops)	kg/day	9	Assumed ingestion rate from OEHHA 2015 (assume concentration the same as predicted for aboveground crops)
IRs (ingestion rate of soil)	kg/day	0.45	Based on data from OEHHA 2015 (5% total produce intakes from soil from pasture)
B (bioavailability)	%	100%	

Chemical-specific Inputs and calculations - maximum discrete 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	
Cadmium	5.2E-05	1.1E-02	2.0E-03	1.1E-05	
Thallium	5.2E-05	1.1E-02	4.0E-02	2.2E-04	RAIS
Mercury (as inorganic and eleme	6.2E-04	1.3E-01	4.0E-04	2.6E-05	
Antimony	1.8E-03	3.8E-01	1.0E-03	1.9E-04	RAIS
Arsenic	1.8E-05	3.8E-03	2.0E-03	3.7E-06	
Lead	7.5E-04	1.6E-01	3.0E-04	2.4E-05	
Chromium (Cr VI assumed)	3.4E-05	7.3E-03	5.5E-03	2.0E-05	RAIS
Cobalt	3.4E-05	7.3E-03	2.0E-02	7.2E-05	RAIS
Copper	1.8E-03	3.8E-01	1.0E-02	1.9E-03	RAIS
Manganese	1.8E-03	3.8E-01	4.0E-04	7.5E-05	RAIS
Nickel	1.8E-03	3.8E-01	3.0E-04	5.6E-05	
Vanadium	1.8E-03	3.8E-01	2.5E-03	4.7E-04	RAIS
Dioxin	1.8E-09	7.8E-08	7.0E-01	3.6E-08	
BaP	2.3E-04	1.2E-03	7.0E-02	1.8E-04	

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Beef

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

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Beef (IRB) (kg/day)	0.16	Ingestion rate of beef for adults >19 years (enHealth 2012, noted to be the same as P90 from FSANZ 2017)
Fraction ingested that is homegrown (%)	35%	Assume 35% beef intakes from home-sourced meat
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum - Discrete receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium		8.0E-04	66%	2.7E-04	100%	1.1E-05	3.6E-09	8.7E-09	--		0.000032	0%
Thallium		8.0E-04	4%	7.7E-04	100%	2.2E-04	7.2E-08	1.7E-07	--		0.00023	1%
Mercury (as inorganic and elementa		6.0E-04	40%	3.6E-04	100%	2.6E-05	8.6E-09	2.1E-08	--		0.000058	0%
Antimony		8.6E-04	4%	8.3E-04	100%	1.9E-04	6.2E-08	1.5E-07	--		0.00018	1%
Arsenic		2.0E-03	55%	9.1E-04	100%	3.7E-06	1.2E-09	3.0E-09	--		0.0000033	0%
Lead		3.5E-03	90%	3.5E-04	100%	2.4E-05	7.8E-09	1.9E-08	--		0.000054	0%
Chromium (Cr VI assumed)		1.0E-03	43%	5.7E-04	100%	2.0E-05	6.5E-09	1.6E-08	--		0.000028	0%
Cobalt		1.4E-03	30%	9.9E-04	100%	7.2E-05	2.4E-08	5.7E-08	--		0.000058	0%
Copper		1.4E-01	62%	5.3E-02	100%	1.9E-03	6.2E-07	1.5E-06	--		0.000028	0%
Manganese		1.4E-01	54%	6.4E-02	100%	7.5E-05	2.5E-08	6.0E-08	--		0.00000093	0%
Nickel		1.2E-02	63%	4.5E-03	100%	5.6E-05	1.9E-08	4.5E-08	--		0.000010	0%
Vanadium		2.0E-03	21%	1.6E-03	100%	4.7E-04	1.5E-07	3.7E-07	--		0.00024	1%
Dioxin		2.3E-09	54%	1.1E-09	100%	3.6E-08	1.2E-11	2.8E-11	--		0.027	97%
BaP	2.3E-01				100%	1.8E-04	6.1E-08	1.5E-07	1.4E-8	11%	--	

TOTAL	**1.4E-8**	**0.028**

Exposure to Chemicals via Ingestion of Beef

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

Parameters Relevant to Quantification of Exposure by Children		
Ingestion Rate of Beef (IRB) (kg/day)	0.085	Ingestion rate of beef by children aged 2-6 years (P90 value) FSANZ (2017)
Fraction ingested that is homegrown (%)	35%	Assume 35% beef intakes from home-sourced meat
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum - Discrete receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium		8.0E-04	66%	2.7E-04	100%	1.1E-05	1.9E-09	2.2E-08	--		0.000079	0%
Thallium		8.0E-04	4%	7.7E-04	100%	2.2E-04	3.7E-08	4.3E-07	--		0.00056	1%
Mercury (as inorganic and elementa		6.0E-04	40%	3.6E-04	100%	2.6E-05	4.4E-09	5.1E-08	--		0.00014	0%
Antimony		8.6E-04	4%	8.3E-04	100%	1.9E-04	3.2E-08	3.7E-07	--		0.00045	1%
Arsenic		2.0E-03	55%	9.1E-04	100%	3.7E-06	6.3E-10	7.4E-09	--		0.0000082	0%
Lead		3.5E-03	90%	3.5E-04	100%	2.4E-05	4.0E-09	4.7E-08	--		0.00013	0%
Chromium (Cr VI assumed)		1.0E-03	43%	5.7E-04	100%	2.0E-05	3.4E-09	3.9E-08	--		0.000069	0%
Cobalt		1.4E-03	30%	9.9E-04	100%	7.2E-05	1.2E-08	1.4E-07	--		0.00014	0%
Copper		1.4E-01	62%	5.3E-02	100%	1.9E-03	3.2E-07	3.7E-06	--		0.000069	0%
Manganese		1.4E-01	54%	6.4E-02	100%	7.5E-05	1.3E-08	1.5E-07	--		0.0000023	0%
Nickel		1.2E-02	63%	4.5E-03	100%	5.6E-05	9.5E-09	1.1E-07	--		0.000025	0%
Vanadium		2.0E-03	21%	1.6E-03	100%	4.7E-04	7.9E-08	9.3E-07	--		0.00058	1%
Dioxin		2.3E-09	54%	1.1E-09	100%	3.6E-08	6.1E-12	7.1E-11	--		0.067	97%
BaP	2.3E-01				100%	1.8E-04	3.1E-08	3.7E-07	7.3E-9	11%	--	
								TOTAL	**7.3E-9**		**0.069**	

Calculation of Concentrations in Dairy Milk

Uptake in to milk (dairy cows)

$$C_E = (FI \times IR_C \times C + IR_S \times C_S \times B) \times TF_B$$

(mg/kg beef – wet weight)

where:
FI = Fraction of grain/crop ingested by cattle each day (unitless)
IRc = Ingestion rate of grain/crop by cattle each day (kg/day)
C = Concentration of chemical in grain/crop eaten by cattle (mg/kg)
IRs = Ingestion rate of soil by cattle each day (kg/day)
Cs = Concentration in soil the cattle ingest (mg/kg)
B = Bioavailability of soil ingested by cattle (%)
TFE = Transfer factor from ingestion to milk (day/kg)

General Parameters	Units	Value	
FI (fraction of crops ingested from property)		1	Assume 100% of pasture consumed by cattle is grown in the same soil
IRc (ingestion rate of crops)	kg/day	22	Assumed ingestion rate from OEHHA 2015 for lactating cattle (assume concentration the same as predicted for aboveground crops)
IRs (ingestion rate of soil)	kg/day	1.1	Based on data from OEHHA 2015 (5% total produce intakes from soil from pasture)
B (bioavailability)	%	100%	

Chemical-specific Inputs and calculations - maximum discrete receptors

Chemical	Concentration in crops ingested by cattle mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to milk day/kg	Milk Concentration mg/kg ww	
Cadmium	5.2E-05	1.1E-02	2.0E-03	2.7E-05	
Thallium	5.2E-05	1.1E-02	4.0E-02	5.3E-04	RAIS
Mercury (as inorganic and eleme	6.2E-04	1.3E-01	7.0E-05	1.1E-05	
Antimony	1.8E-03	3.8E-01	1.0E-04	4.6E-05	RAIS
Arsenic	1.8E-05	3.8E-03	5.0E-05	2.3E-07	
Lead	7.5E-04	1.6E-01	6.0E-05	1.2E-05	
Chromium (Cr VI assumed)	3.4E-05	7.3E-03	9.0E-06	7.9E-08	
Cobalt	3.4E-05	7.3E-03	2.0E-03	1.8E-05	RAIS
Copper	1.8E-03	3.8E-01	1.5E-03	6.8E-04	RAIS
Manganese	1.8E-03	3.8E-01	3.5E-04	1.6E-04	RAIS
Nickel	1.8E-03	3.8E-01	3.0E-05	1.4E-05	
Vanadium	1.8E-03	3.8E-01	2.0E-05	9.1E-06	RAIS
Dioxin	1.8E-09	7.8E-08	2.0E-02	2.5E-09	
BaP	2.3E-04	1.2E-03	1.0E-02	6.4E-05	

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Milk

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

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Milk (IRM) (kg/day)	1.295	Ingestion rate of cows milk for adults (P90 value from FSANZ 2017)
Fraction ingested that is homegrown (%)	100%	Assume all milk consumed is from the dairy farm
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum - Discrete receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium		8.0E-04	66%	2.7E-04	100%	2.7E-05	2.0E-07	4.9E-07	--		0.0018	3%
Thallium		8.0E-04	4%	7.7E-04	100%	5.3E-04	4.1E-06	9.9E-06	--		0.013	21%
Mercury (as inorganic and elementa		6.0E-04	40%	3.6E-04	100%	1.1E-05	8.5E-08	2.0E-07	--		0.00057	1%
Antimony		8.6E-04	4%	8.3E-04	100%	4.6E-05	3.5E-07	8.4E-07	--		0.0010	2%
Arsenic		2.0E-03	55%	9.1E-04	100%	2.3E-07	1.7E-09	4.2E-09	--		0.0000047	0%
Lead		3.5E-03	90%	3.5E-04	100%	1.2E-05	8.9E-08	2.1E-07	--		0.00061	1%
Chromium (Cr VI assumed)		1.0E-03	43%	5.7E-04	100%	7.9E-08	6.1E-10	1.5E-09	--		0.0000026	0%
Cobalt		1.4E-03	30%	9.9E-04	100%	1.8E-05	1.3E-07	3.2E-07	--		0.00033	1%
Copper		1.4E-01	62%	5.3E-02	100%	6.8E-04	5.2E-06	1.3E-05	--		0.00024	0%
Manganese		1.4E-01	54%	6.4E-02	100%	1.6E-04	1.2E-06	3.0E-06	--		0.000046	0%
Nickel		1.2E-02	63%	4.5E-03	100%	1.4E-05	1.0E-07	2.5E-07	--		0.000057	0%
Vanadium		2.0E-03	21%	1.6E-03	100%	9.1E-06	7.0E-08	1.7E-07	--		0.000106	0%
Dioxin		2.3E-09	54%	1.1E-09	100%	2.5E-09	1.9E-11	4.6E-11	--		0.044	71%
BaP	2.3E-01				100%	6.4E-05	4.9E-07	1.2E-06	1.1E-7	11%	--	
								TOTAL	1.1E-7		0.061	

Exposure to Chemicals via Ingestion of Milk

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

Parameters Relevant to Quantification of Exposure by Children		
Ingestion Rate of Milk (IRM) (kg/day)	1.097	Ingestion rate of cows milk for children aged 2-6 years (P90 value from FSANZ 2017)
Fraction ingested that is homegrown (%)	100%	Assume all milk consumed is from the dairy farm
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Exposure occurs every day
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum - Discrete receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium		8.0E-04	66%	2.7E-04	100%	2.7E-05	1.7E-07	2.0E-06	--		0.0071	3%
Thallium		8.0E-04	4%	7.7E-04	100%	5.3E-04	3.3E-06	3.9E-05	--		0.051	21%
Mercury (as inorganic and elementa		6.0E-04	40%	3.6E-04	100%	1.1E-05	6.9E-08	8.1E-07	--		0.0022	1%
Antimony		8.6E-04	4%	8.3E-04	100%	4.6E-05	2.9E-07	3.3E-06	--		0.0040	2%
Arsenic		2.0E-03	55%	9.1E-04	100%	2.3E-07	1.4E-09	1.7E-08	--		0.000018	0%
Lead		3.5E-03	90%	3.5E-04	100%	1.2E-05	7.3E-08	8.5E-07	--		0.0024	1%
Chromium (Cr VI assumed)		1.0E-03	43%	5.7E-04	100%	7.9E-08	4.9E-10	5.8E-09	--		0.0000102	0%
Cobalt		1.4E-03	30%	9.9E-04	100%	1.8E-05	1.1E-07	1.3E-06	--		0.0013	1%
Copper		1.4E-01	62%	5.3E-02	100%	6.8E-04	4.3E-06	5.0E-05	--		0.00094	0%
Manganese		1.4E-01	54%	6.4E-02	100%	1.6E-04	1.0E-06	1.2E-05	--		0.00018	0%
Nickel		1.2E-02	63%	4.5E-03	100%	1.4E-05	8.6E-08	1.0E-06	--		0.00022	0%
Vanadium		2.0E-03	21%	1.6E-03	100%	9.1E-06	5.7E-08	6.7E-07	--		0.00042	0%
Dioxin		2.3E-09	54%	1.1E-09	100%	2.5E-09	1.6E-11	1.8E-10	--		0.17	71%
BaP	2.3E-01				100%	6.4E-05	4.0E-07	4.7E-06	9.4E-8	11%	--	
								TOTAL	9.4E-8		0.24	