Appendix F. Health impact assessment

Energy from Waste – Prospect Hill: Health Impact Assessment

Prepared for: Prospect Hill International and Jacobs

25 January 2021

EnRiskS

Document History and Status

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Limitations

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

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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)
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.
EPA	Environment Protection Authority
Exposure	Contact with a substance by swallowing, breathing, or touching the skin or eyes. Also includes contact with a stressor such as noise or vibration. Exposure may be short term [acute exposure], of intermediate duration, or long term [chronic exposure].
Exposure assessment	The process of finding out how people come into contact with a hazardous substance, how often and for how long they are in contact with the substance, and how much of the substance they are in contact with.
Exposure pathway	The route a substance takes from its source (where it began) to its endpoint (where it ends), and how people can come into contact with (or get exposed) to it. An exposure pathway has five parts: a source of contamination (such as chemical substance leakage into the subsurface); an environmental media and transport mechanism (such as movement through groundwater); a point of exposure (such as a private well); a route of exposure (eating, drinking, breathing, or touching), and a receptor population (people potentially or actually exposed). When all five parts are present, the exposure pathway is termed a completed exposure pathway.

Term	Definition
Genotoxic carcinogen	These are carcinogens that have the potential to result in genetic (DNA) damage (gene mutation, gene amplification, chromosomal rearrangement). Where this occurs, the damage may be sufficient to result in the initiation of cancer at some time during a lifetime.
Guideline value	Guideline value is a concentration in soil, sediment, water, biota or air (established by relevant regulatory authorities such as the 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.
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
Point of exposure	The place where someone can come into contact with a substance present in the environment [see exposure pathway].
Population	A group or number of people living within a specified area or sharing similar characteristics (such as occupation or age).
Receptor population	People who could come into contact with hazardous substances [see exposure pathway].
Risk	The probability that something would cause injury or harm.
Route of exposure	The way people come into contact with a hazardous substance. Three routes of exposure are breathing [inhalation], eating or drinking [ingestion], or contact with the skin [dermal contact].
SEIFA	Socio-Economic Index for Areas
SO_2	Sulfur dioxide
TCEQ	Texas Commission on Environmental Quality
TEQ	Toxicity equivalent

Term	Definition
Toxicity	The degree of danger posed by a substance to human, animal or plant life.
Toxicity data	Characterisation or quantitative value estimated (by recognised authorities) for each individual chemical substance for relevant exposure pathway (inhalation, oral or dermal), with special emphasis on dose-response characteristics. The data are based on 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
µg/m^3	Micrograms per cubic metre

Executive Summary

Introduction

The project, proposed by Prospect Hill International Pty Ltd involves the construction and operation of an energy from waste (EfW) facility at the Prospect Hill owned site at 164-200 McManus Road, Lara, Victoria (the 'site').

The site is approximately 395 x 400 m in size and is a greenfield undeveloped parcel of land located in an Industrial 2 Zone. The proposed facility will process an estimated 400,000 tonnes per annum of municipal solid waste (MSW), commercial and industrial (C&I) waste and residual waste generated from Material Recovery Facilities that cannot be recycled. The waste materials will be derived from a number of councils in Victoria. The plant would provide approximately 40.7 Megawatts electric (MWe) per annum, with approximately 36 MWe as output to the electricity grid.

This Health Impact Assessment (HIA) has been developed for the project 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 2012a, 2017). The HIA has been undertaken on the basis of technical assessments completed in relation to emissions to air, noise, waste management and transport.

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, traffic, waste management and the economic 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 low to 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 4.4	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 4.5	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 4.6	Annoyance, stress, anxiety	**Not significant and negligible**	The proper operation of the tipping hall as proposed to ensure fugitive odour emissions are effectively managed on-site.
Noise	Section 5	Sleep disturbance, annoyance, children's school performance and cardiovascular health	**Modelled noise impacts: low potential for health impacts**	The plant is currently in a concept stage of design. Further noise modelling and the inclusion of additional noise mitigation measures would be expected to be considered in the detailed design, which would result in a further reduction of noise impacts within the community.
Economic Environment	Section 6	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 (particularly in areas with higher levels of existing unemployment)

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 6	Injury or death, stress and anxiety.	**Negative but minimal**	Details to be determined at the detailed design phase of the project
Presence of hazardous waste in feedstock and generation of waste	Section 6	Possible injury if incorrectly disposed of	**Negative but minimal**	Further development of the proposed feedstock delivery protocols into an operational management plan to address the discovery and proper disposal of hazardous waste, should.it be present in feedstock. Appropriate testing and management of waste materials generated during operations, with compliance with all relevant current regulations in relation to waste disposal and/or re-use.

Section 1. Introduction

1.1 Background

The project, proposed by Prospect Hill International Pty Ltd involves the construction and operation of an energy from waste (EfW) facility at the Prospect Hill owned site at 164-200 McManus Road, Lara, Victoria (the 'site').

The site is approximately 395 x 400 m in size and is a greenfield undeveloped parcel of land located in an Industrial 2 Zone. The location of the site is shown below in **Figure 1.1** and a spatial image of the actual site footprint is shown in **Figure 1.2**.

The proposed facility will process an estimated 400,000 tonnes per annum of municipal solid waste (MSW), commercial and industrial (C&I) waste and residual waste generated from Material Recovery Facilities that cannot be recycled. The waste materials will be derived from a number of councils in Victoria.

Waste will be transported to the site via road in council collection compactor, 19 m semitrailer bulk waste vehicles and other trucks (b-doubles, a-doubles and high productivity freight vehicles as approved). The plant is proposed to include two boiler trains capable of processing 200,000 tonnes per annum (each), with the capacity to increase to three (if required in the future). The plant will provide approximately 40.7 Megawatts electric (MWe) per annum, with approximately 36 MWe as output to the electricity grid.

The proposed facility development includes (**Figure 1.3**):

- weighbridges and gatehouse
- silos
- energy from waste facility building which include the tipping hall, boilers, flue gas cleaning, steam turbine and cooling towers
- bottom ash pre-treatment, processing and storage building
- road infrastructure
- stormwater detention pond
- office, car park and hard stand area.

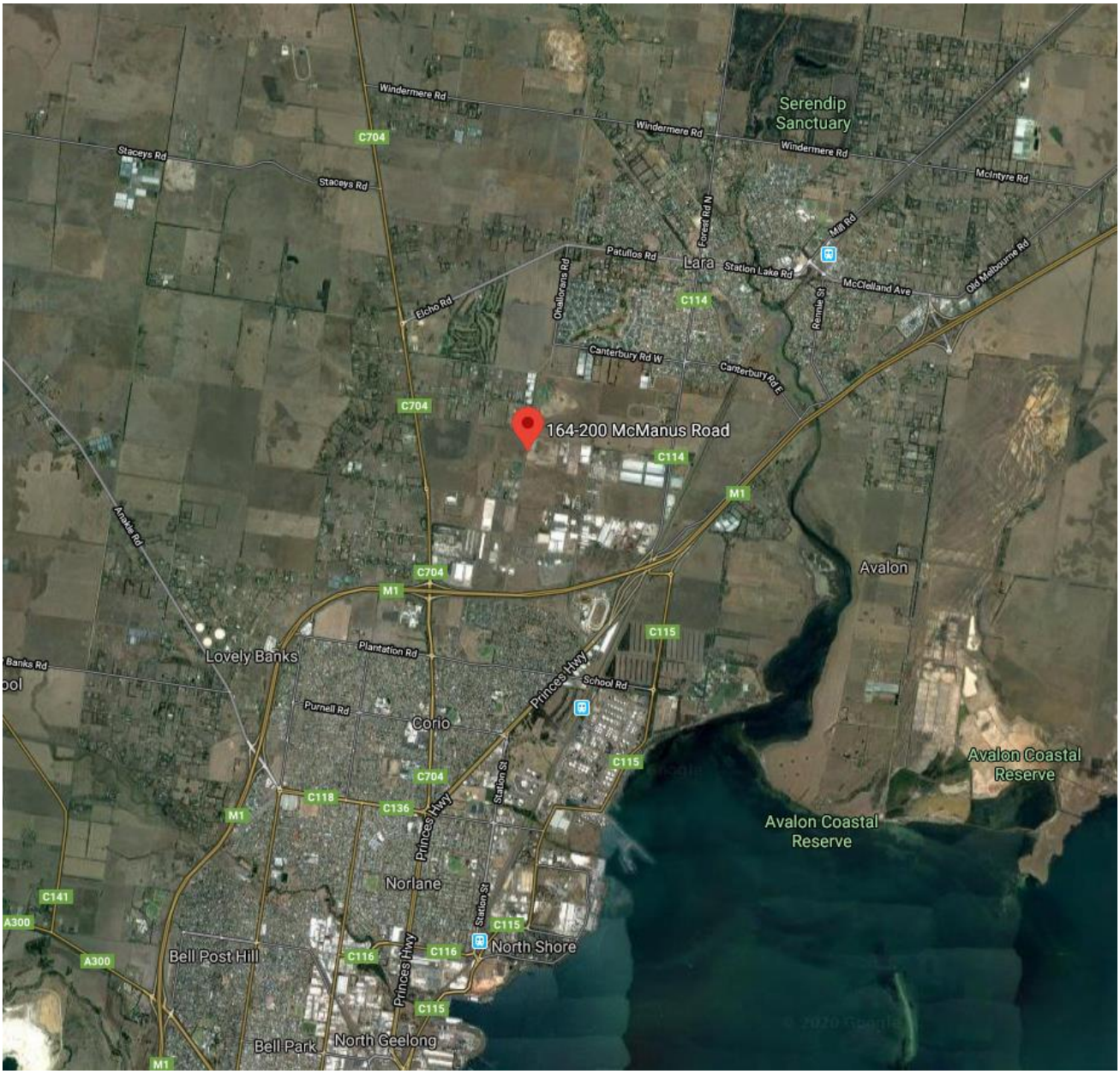
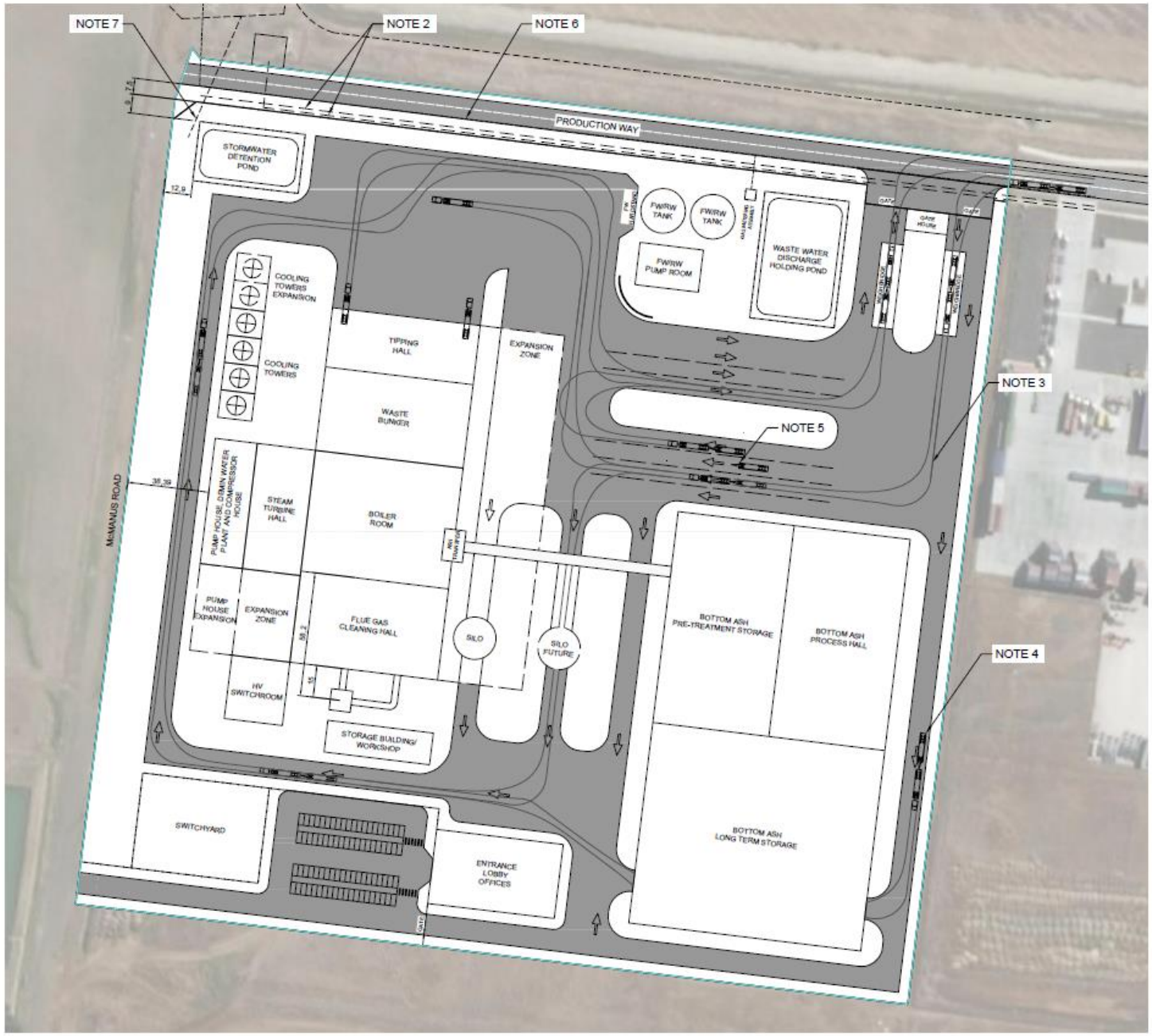


Figure 1.1: General site location

Figure 1.2: Spatial image of the site



NOTES:

1. ALL DIMENSIONS ARE IN METERS & ARE APPROXIMATE ONLY.
2. BARWON WATER POTABLE WATER AND SEWER PIPELINES ARE SHOWN AS INDICATIVE ONLY.
3. GENERAL ROAD LAYOUT DESIGN ALLOWS FOR 36.2 M TYPE 1 ROAD TRAIN (A-DOUBLE / B-TRIPLE) VEHICLES TRAVELLING BETWEEN 5-10 KM/H.
4. LANE WAY USED FOR VEHICLES TO ACCESS BOTTOM ASH LONG TERM STORAGE WAREHOUSE.
5. TRAILER DETACHMENT AND PARKING AREA.
6. AUSNET SERVICES GAS PIPELINE IS SHOWN AS INDICATIVE ONLY. LOCATION TO BE CONFIRMED DURING THE NEXT PHASE OF DESIGN.
7. CITY OF GEELONG COUNCIL 900MM STORMWATER CULVERT SHOWN AS INDICATIVE ONLY. LOCATION AND SIZE TO BE CONFIRMED DURING THE NEXT PHASE OF DESIGN.

Figure 1.3: Proposed site layout

LEGEND:

- BOUNDARY LINE
- FENCE LINE
- EASEMENT - POTABLE WATER AND SEWER
- EASEMENT - GAS
- EASEMENT - STORMWATER

SCALE 1:1000 (A1)

0 20 40 60 80 100m
20 10

CONCEPT

REV	DATE	APP'D	REVISION	DRAWING NUMBER	REFERENCE DRAWING TITLE
A	xx.05.20		PRELIMINARY		

Jacobs

CLIENT: PROSPECT HILL INTERNATIONAL
PROJECT: PROSPECT HILL ENERGY FROM WASTE PLANT, LARA

DRAWN	DRAWING CHECK	REVIEWED	APPROVED
R. INCAMPO			
DESIGNED	DESIGN REVIEW	DATE	DATE
K. IVANUSIC			

TITLE: PROSPECT HILL ENERGY FROM WASTE PLANT CONCEPT STUDY LAYOUT

SCALE	DRAWING NO.	REV
1:1000	IS305100-0000-MC-DRG-0001	A

A1

1.2 Objectives

This Health Impact Assessment (HIA) has been developed for Prospect Hill 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):

- enHealth, 2017. Health Impact Assessment Guidelines (enHealth 2017)
- 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, 2012. Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a)
- enHealth, 2012. Australian Exposure Factor Guidance – Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012b)
- Guidance and guidelines available from the National Environment Protection Council in relation to ambient air quality (NEPC 2016) and contaminated land (NEPC 1999 amended 2013a)
- Victorian State Environment Protection Policies relevant to the assessment of air quality (EPA Victoria 2001) and others as relevant to the assessment.

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).

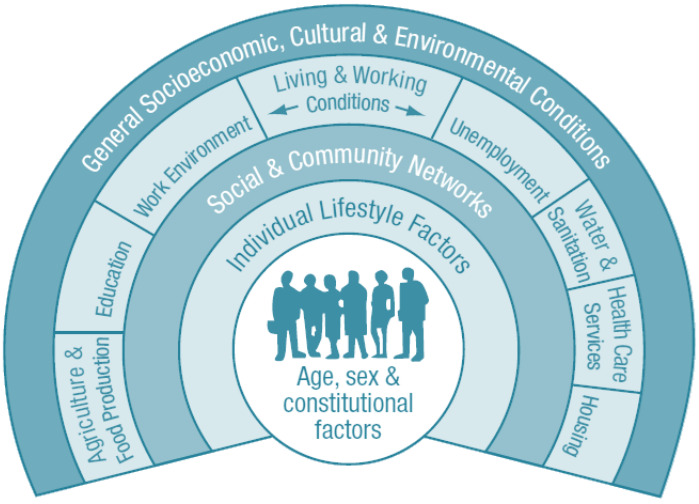


Figure 1.4: Wider determinants of health, as presented by Harris et al (2007)

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. The key focus of the HIA relates to air quality impacts, however the HIA has also addressed the transport of waste, odour and noise which are of relevance to the offsite community.

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:

- Concept design basis report, Jacobs – 15 April 2020
- Noise Assessment Report, Jacobs – 19 September 2020
- Traffic Impact Assessment, Jacobs – 12 June 2020
- Waste Chapter, Jacobs – 2020
- Air Quality Impact Assessment, Jacobs – 6 October 2020.

Section 2. Project description

2.1 Site description and location

The project, proposed by Prospect Hill, involves the construction and operation of an EfW plant on an undeveloped greenfields site located at 164-200 McManus Road, Lara, Victoria. The site is located to the north of Geelong with the M1 freeway and Avalon Airport located to the east of the area.

The site is in an area zoned Industrial 2 within the Greater City of Geelong. The area is currently a mix of undeveloped lots and lots previously developed for a range of industrial uses.

The industrial area is surrounded by larger residential and rural residential blocks to the northwest and west; residential areas in Lara, which includes low density residential homes with some rural residential blocks approximately 1.5 km to the north, northeast and northwest; and residential areas in Corio located approximately 1.75 km to the south.

The closest residential property is located at 180 Minyip Road, approximately 0.3 m from the site, located within an area zoned Rural Living Zone. This area, along with the urban areas of Lara and Corio are illustrated on **Figure 2.1**. **Figure 2.2** includes the zoning of the area, noting that land located to the north of the site is currently zoned as Farming Zone.

Figure 2.1: Surrounding areas

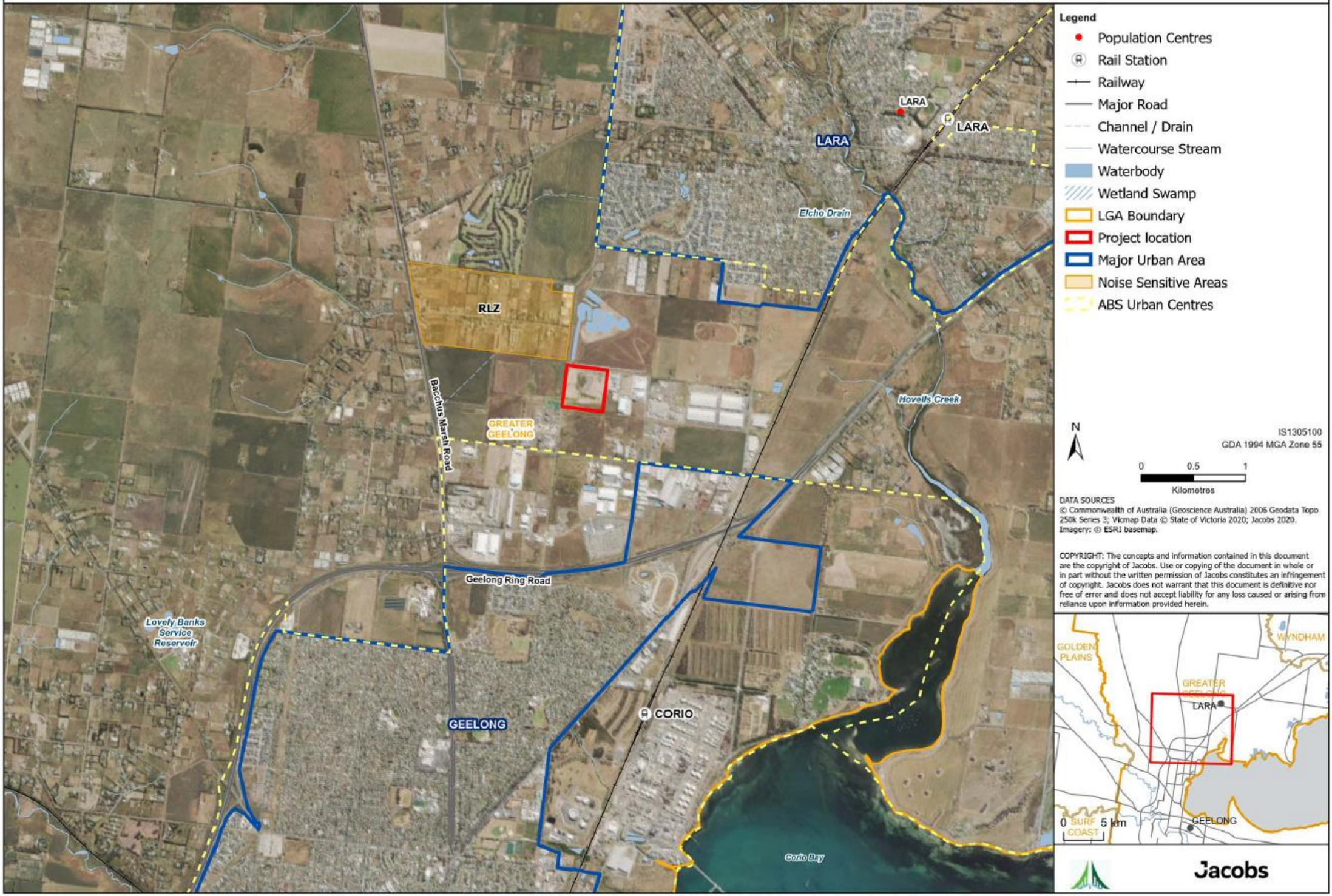
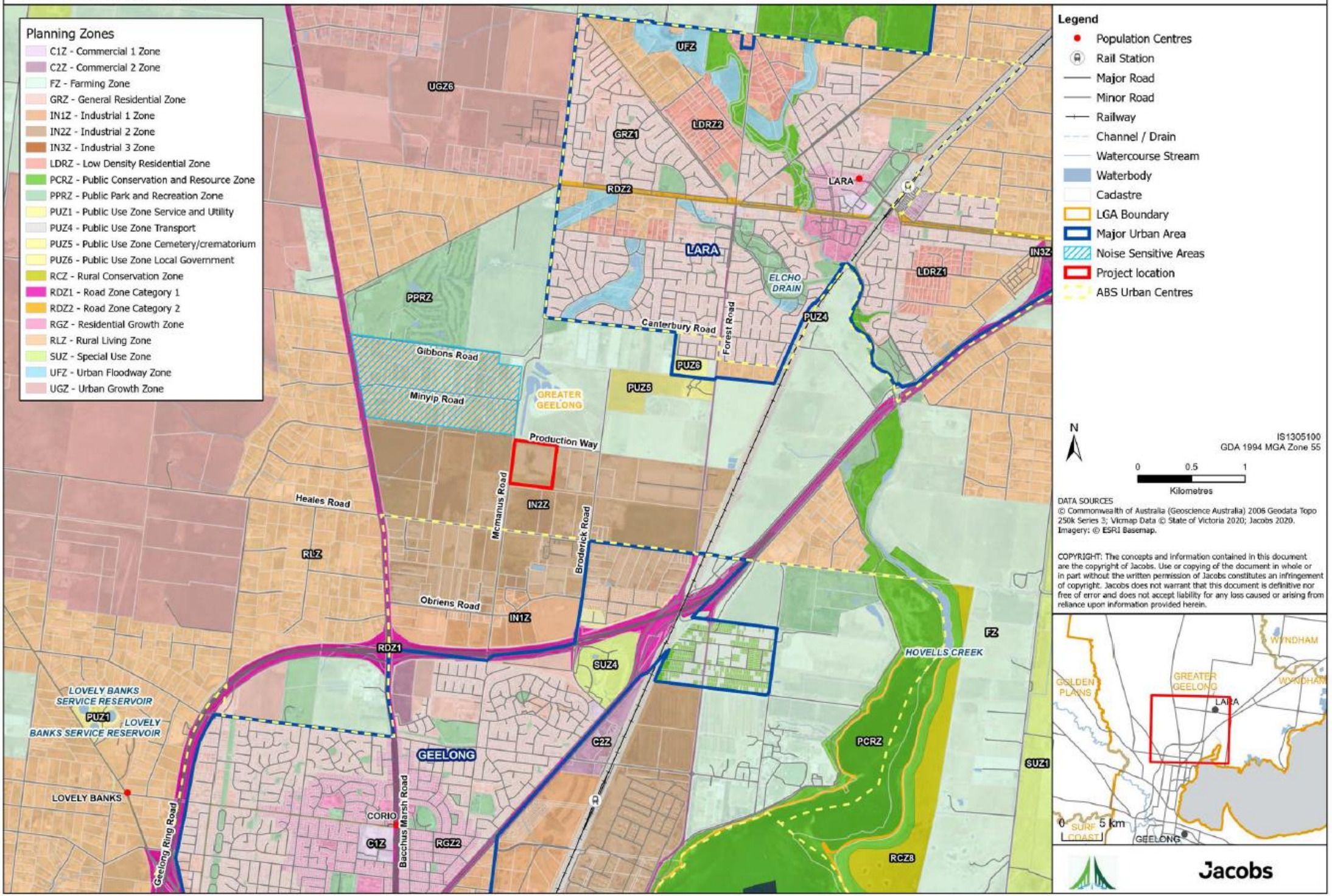


Figure 2.2: Landuse zonings



2.2 Project infrastructure and process

The project involves the thermal treatment of 400,000 tonnes per annum (TPA) of Municipal Solid Waste (MSW) and Commercial and Industrial (C&I) Waste, utilising a proven combustion grate technology with energy recovery in a steam boiler and turbine, and flue gas emission controls in accordance with the BAT recommendations of the 2019 EU Best Reference Document (BREF).

Flue gases leaving the boiler will be treated with powdered activated carbon to absorb toxic volatile organic components such as dioxins and furans and heavy metals such as mercury, and with a dry or semi-dry lime dosing and reactor system to neutralise acid gas pollutants such as hydrochloric acid, sulfuric oxides, etc. Oxides of nitrogen emissions are normally controlled by a Selective Non Catalytic Reduction (SNCR) system, which injects ammonia or urea into the flue gases at the top of the furnace.

Safe vacuum conditions are maintained in the furnace and boiler so that hot combustion gases do not escape to the atmosphere. Furnace pressure is controlled by the induced draft fan which then draws the cleaned flue gases up the stack. The stack will be designed to a height to disperse the gases to achieve ambient air state environment protection policy (SEPP) requirements.

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 City of Greater Geelong Local Government Area, within an industrial land use zone and surrounded by farming, rural and urban use land zones (**Figure 2.2**). The suburbs/towns of Lara and Corio are located to the northwest and south of the site.

Table 3.1 presents a summary of the populations in Lara, Corio, Greater Geelong (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
	Lara	Corio	Greater Geelong LGA		
Total population	16355	15296	233429	5926624	23401892
Population 0 - 4 years	6.8% (1114)	6.3% (965)	6% (14347)	6.3% (371220)	6.3% (1464779)
Population 5 - 19 years	19% (3138)	23% (3590)	18% (42358)	18.0% (1066042)	18.5% (4321427)
Population 20 - 64 years	61% (9997)	56% (8580)	57% (133357)	60.2% (3566775)	59.6% (13938918)
Population 65 years and over	13% (2108)	14% (2155)	19% (43363)	15.6% (922598)	15.7% (3676758)
Median age	37	35	40	37	38
Household size	2.7	2.5	2.4	2.6	2.6
Unemployment	4.8%	12.5%	6.4%	6.6%	6.9%
Tertiary education	9%	5.3%	15.2%	17.8%	16.1%
SEIFA IRSD	1028	832	994	--	--
SEIFA rank	4	4	4	--	--
Indigenous	1.4%	2%	1%	0.8%	2.8%
Born overseas	15.4%	23.3%	16.3%	34.9%	33.3%

SEIFA IRSD = index of socioeconomic disadvantage, rank relates to rank in Australia that ranges from 1 = most disadvantaged to 5 = least disadvantaged
Shading relates to comparison against Victoria: more vulnerable; less vulnerable

Based on the population data available and presented in **Table 3.1**, the community of Greater Geelong is older, however the smaller populations of Lara and Corio are more consistent when compared to the general Victorian and Australian population. The populations of Lara and Corio have a lower percentage with tertiary education and percentage born overseas. Corio has higher unemployment and Lara has lower unemployment, compared to the population of Victoria (which is the same as Australia). The populations in these areas are considered to be less socioeconomically disadvantaged. The indicators outlined in **Table 3.1** reflect the vulnerability of the population and is considered to potentially reflect the ability of the population to adapt to environmental change and stressors. These indicators are important to highlight from an equity point of view. In general, the population close to the site is considered somewhat less vulnerable (based on socioeconomic disadvantage) with some areas of higher unemployment and older groups potentially more vulnerable.

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 Greater Geelong Local Government Area, which includes the suburbs of Lara and Corio.

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	Greater Geelong LGA	Victoria
Health behaviours (rate with 95% confidence limits)		
Adults - compliance with fruit consumption guidelines (2017)[1]	47.1% (40.7% - 53.7%)	43.2% (42.3% - 44.1%)
Adults - compliance with vegetable consumption guidelines (2017)[1]	6.9% (4.4% - 10.7%)	5.4% (5.0% - 5.8%)
Children adequate consumption of fruit and vegetables (4-12 years) (2009) [2]	35.9%	34.7%
Adults - increased lifetime risk of alcohol related harm (2017) [1]	65.9% (60.0% - 71.5%)	59.5% (58.63% - 60.4%)
Adults - body weight (preobese) (2017) [1]	28.2% (23% - 34%)	31.5% (30.7% - 32.4%)
Adults - body weight (obese) (2017) [1]	21.7% (16.9% - 27.5%)	19.3% (18.6 – 20.0%)
Adults – insufficient physical activity (2017) [1]	41.3% (35.2% - 47.6%)	44.1% (43.2 – 45.0%)
Children – adequate physical activity (2009) [2]		60.3%
Current smoker (2017) [1]	18.1% (13.2% - 24.2%)	16.7% (16.0% - 17.5%)
Burden of disease		
Mortality – all causes (all ages) (2018)[5]	540*	500*
Mortality – cardiovascular (2013-2017) [3]	33.7*	40.4*
Mortality – respiratory (2013-2017) [3]	12.0*	14.0
Morbidity - cardiovascular disease hospitalisations (2016/17)[3]	1846.2*	2229.4*
Morbidity – respiratory disease hospitalisations (2016/17)[3]	1845.0*	1913.4*
Morbidity - prevalence of hypertension ≥18 years (2016/17)[3]	23600*	22700*
Adults – prevalence of asthma (2017)[1]	20.7%	20.0%
Children (school entrant) – prevalence of asthma (2019)[4]	11.3%	10.6%

* Rate per 100,000 population

1 Data from Victorian Population Health Survey 2017: https://www2.health.vic.gov.au/public-health/population-health-systems/health-status-of-victorians/survey-data-and-reports/victorian-population-health-survey/victorian-population-health-survey-2017

2 Data from the City of Greater Geelong Early Childhood Community Profile 2010 https://www.education.vic.gov.au/Documents/about/research/ecprofgreatgeelong.pdf

3 Age standardised ratio - data relevant to the years 2013-2018 from the Social Health Atlas of Australia, Victoria: http://phidu.torrens.edu.au/social-health-atlases

4 Data available from School Entrant Health Questionnaire, 2019 https://www.education.vic.gov.au/about/research/Pages/reportdatahealth.aspx

5 Data from the Australian Bureau of Statistics for Greater Geelong, expressed as rate per 100,000 population for all deaths reported in 2018

Shading relates to comparison against Victoria: more vulnerable, less vulnerable.

The key indicators of health for the population in the Greater Geelong local government area are similar to those of Victoria with none of the key indicators statistically significantly different from Victoria. The indicators related to the existing burden of disease indicate a lower rate of

cardiovascular and respiratory disease hospitalisations in the Greater Geelong area, compared with Victoria.

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

Section 4. Health impacts: Air emissions

4.1 Approach

This section presents a review of impacts on health associated with predicted air emissions, relevant to the operation of the facility. The assessment presented has relied on the Air Quality Impact Assessment (AQIA) report prepared by Jacobs (2020) along with additional outputs from the 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).

4.2 Modelled air impacts

4.2.1 Air modelling

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

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

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

Figures 4.1 and 4.2 illustrate the processes which govern how the emissions get mixed into the atmosphere.

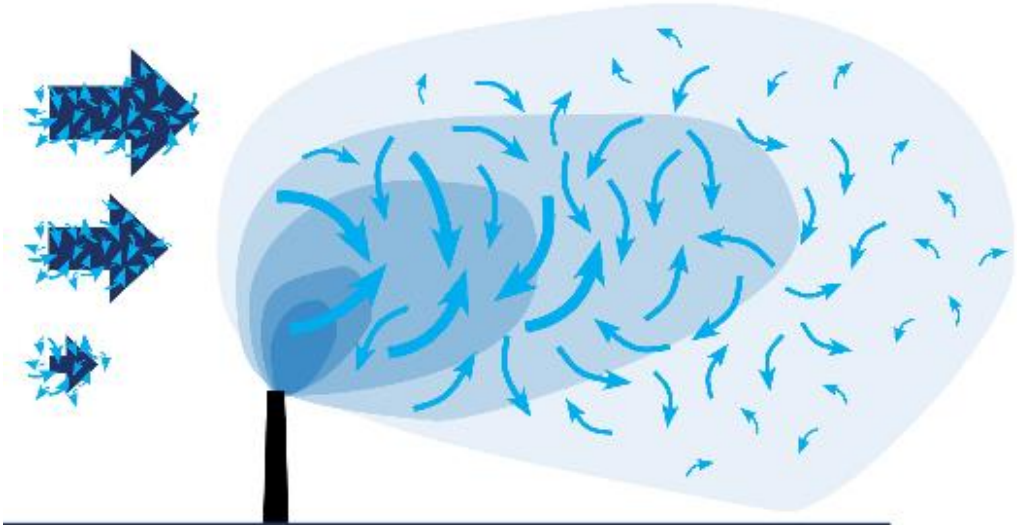


Figure 4.1: Turbulence in the air, how it mixes and dilutes pollutants emitted from a stack (NSW Chief Scientist 2018)

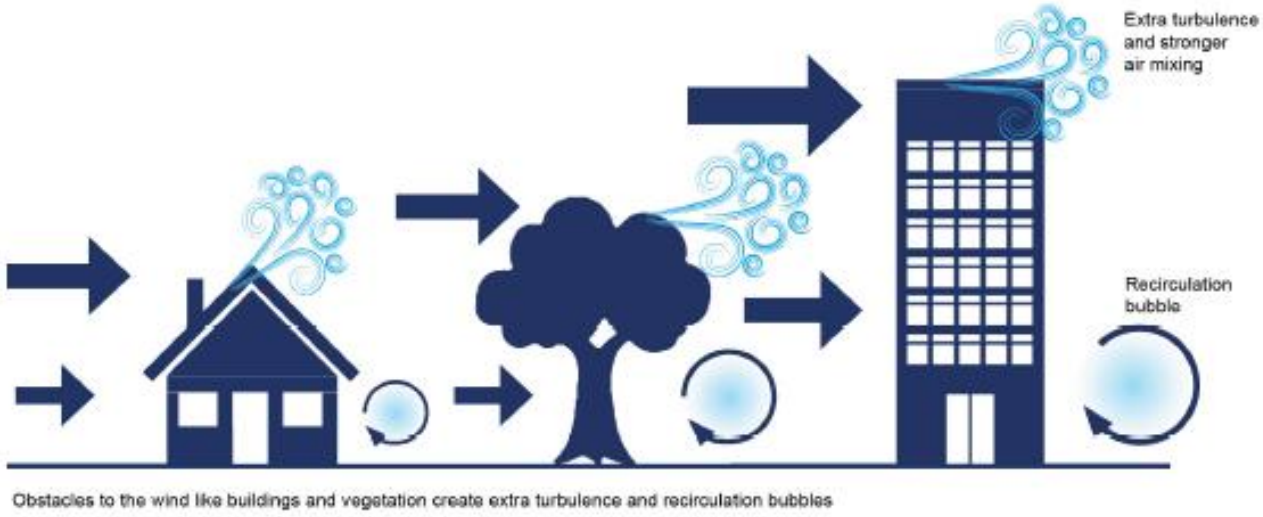


Figure 4.2: Turbulence in the air and how it is affected by buildings and vegetation (NSW Chief Scientist 2018)

Gases (and any fine particles that remain) are emitted at around 140ºC from the stack and they are pushed out of the stack using fans (i.e. at some speed) so these gases (and fine particles) rise or are pushed up significant distances above the top of the stack – because hot gases rise and because gases are travelling at a faster speed than the air surrounding the stack. This can be seen in the figures above.

As the gases (and fine particles) cool and slow down they begin to interact with the wind above the stack (i.e. well above the 80 m high stack). This mixes the gases (and fine particles) into the atmosphere decreasing the actual concentration present in any one particular place.

Figure 4.1 shows that most of the pollutants remain up in the atmosphere away from where people be exposed. However, small amounts do eventually reach ground level. The air dispersion modelling determines what proportion of the amount in the stack could reach ground level at different locations. Such modelling looks at worst case weather characteristics (that can actually occur – based on real meteorological data) to ensure that the amount that could reach ground level in areas where people live or work neighbouring the proposed facility are not underestimated. It is these ground level concentrations that are then used to assess potential for health impacts.

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

4.2.2 Overview of air modelling

To predict the concentration of emissions from the energy from waste plant, a study area was defined (**Figure 4.1**) and predicted emissions from the stack were modelled by Jacobs (2020) using the AERMOD air dispersion model.

The AERMOD 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. The modelling utilised 5 years of meteorological data from Avalon Airport (from 2015 to 2019) with the maximum impacts predicted from all these years presented and considered in this HIA.

For this project the air modelling was undertaken on the basis of the European Union EfW emissions limits as published in 2010 and 2019. These are considered to be the maximum emissions likely to occur, noting that the plant is designed to operate with emissions that are lower than the modelled emissions limits. Hence the modelling or air impacts as well as the modelling of risks to human health has consider the worst-case scenario, where it is assumed that the facility is operating at the emissions limits at all times.

Full details on the air model is presented in the AQIA (Jacobs 2020). This model is used to provide predicted air concentrations over a study area and at sensitive locations/receptors (described further below), with the results averaged over different time periods. The AQIA has presented the modelling results over a range of averaging periods that specifically relate to that assessment, and include 3-

minute averages, 1-hour averages, 24-hour averages and annual averages (as required for each pollutant). The assessment of impacts on community health requires the use of the modelling results from the same model for averaging periods relevant to characterising short-term (acute) and long-term (chronic) exposures. This means that the HIA has utilised modelled impacts for a 1-hour average and annual average for all pollutants. In addition, for the assessment of exposures to particulate matter 24-hour average concentrations are also used in the HIA.

The air modelling has predicted impacts across the whole study area. In addition, a number of individual or sensitive receptors were considered in the modelling. These represented the closest sensitive receptors to the site, as listed in **Table 4.1**.

Background air concentrations are also used to determine the total exposures for some of the key pollutants modelled in the study area. Background air data were obtained from EPA Victoria monitoring data from Geelong South between 2014 and 2019.

Figure 4.1: Air modelling study area

Table 4.1: Summary of sensitive receptors (as shown on Figure 4.1)

Label	Receptor
Minyip	Closest residence on Minyip Road
CN	Corio North
FMP	Flinders Memorial Park
SR	Stulle Reserve
EPGC	Elcho Park Golf Course
MC	Macgregor Court
RS	Rennie Street
BP	Beckley Park
MW	Minyip West
AD	Apollo Drive
FR	Frys Road

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.

4.3 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)
- 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 nitrogen dioxide (NO_2), sulfur dioxide (SO_2),carbon monoxide (CO), hydrogen chloride (HCl), ammonia (NH_3) and hydrogen fluoride (HF) as well as fine particulate matter as particulates less than 10 micrometres (PM_{10}) and particulate matter less than 2.5 micrometres ($PM_{2.5}$). $PM_{2.5}$ 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 with the deposition of dust. 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, polycyclic aromatic hydrocarbons (PAHs), 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 4.2 lists the substances considered in the EfW emissions and the exposure pathway/s of potential concern. **Figure 4.2** provides a diagrammatical representation of the community exposures to emissions from the energy from waste facility (conceptual site model).

Table 4.2: Substances and routes of exposure

Substance	Route of exposure
Nitrogen dioxide	**Inhalation only** as these are gases
Sulfur dioxide	
Hydrogen chloride	
Hydrogen fluoride[1]	
Carbon monoxide	
Ammonia	
Volatile organic compounds (VOCs) as formaldehyde	
PM_{10}	**Inhalation relevant for particulates based on particle size** as these particulates are very small and will remain suspended in air. It is noted that other exposure pathways have also been assessed for the individual chemical substances bound to these particles that may be deposited to the ground. These other pathways relate to the individual chemical substances, rather than the physical size of the particulates, however they do relate to the more coarse fractions of dust in PM_{10} (rather than $PM_{2.5}$) as some PM_{10} will deposit to the ground
$PM_{2.5}$	
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	
Polycyclic aromatic hydrocarbons as benzo(a)pyrene (BaP)	
Dioxins / furans	

The HIA has evaluated health impacts from all the contaminants listed in the European Union's Industrial Emissions Directive (EU IED). While these have all been modelled in the AQIA (Jacobs 2020) health impacts can only be quantified for individual chemicals or chemical groups with similar (or related) toxicities. In some cases, a conservative approach has been adopted for a group of chemicals where the composition is less well known. Hence the HIA has evaluated all individual pollutants and key pollutant groups:

- Dioxins and furans, assuming the group is characterised by the toxicity of the most potent compound, 2,3,7,8-TCDD
- Polycyclic aromatic hydrocarbons (PAHs), assuming the group is characterised by the toxicity of the one of the most potent and well understood compound, benzo(a)pyrene (BaP)
- The more general chemical group of volatile organic compounds (VOCs) includes a large number of individual volatile chemicals with individual varying toxicities. For this group it has

been conservatively assumed that this group comprises 100% formaldehyde, one of the more toxic (and likely) components of VOCs from EfW facilities.

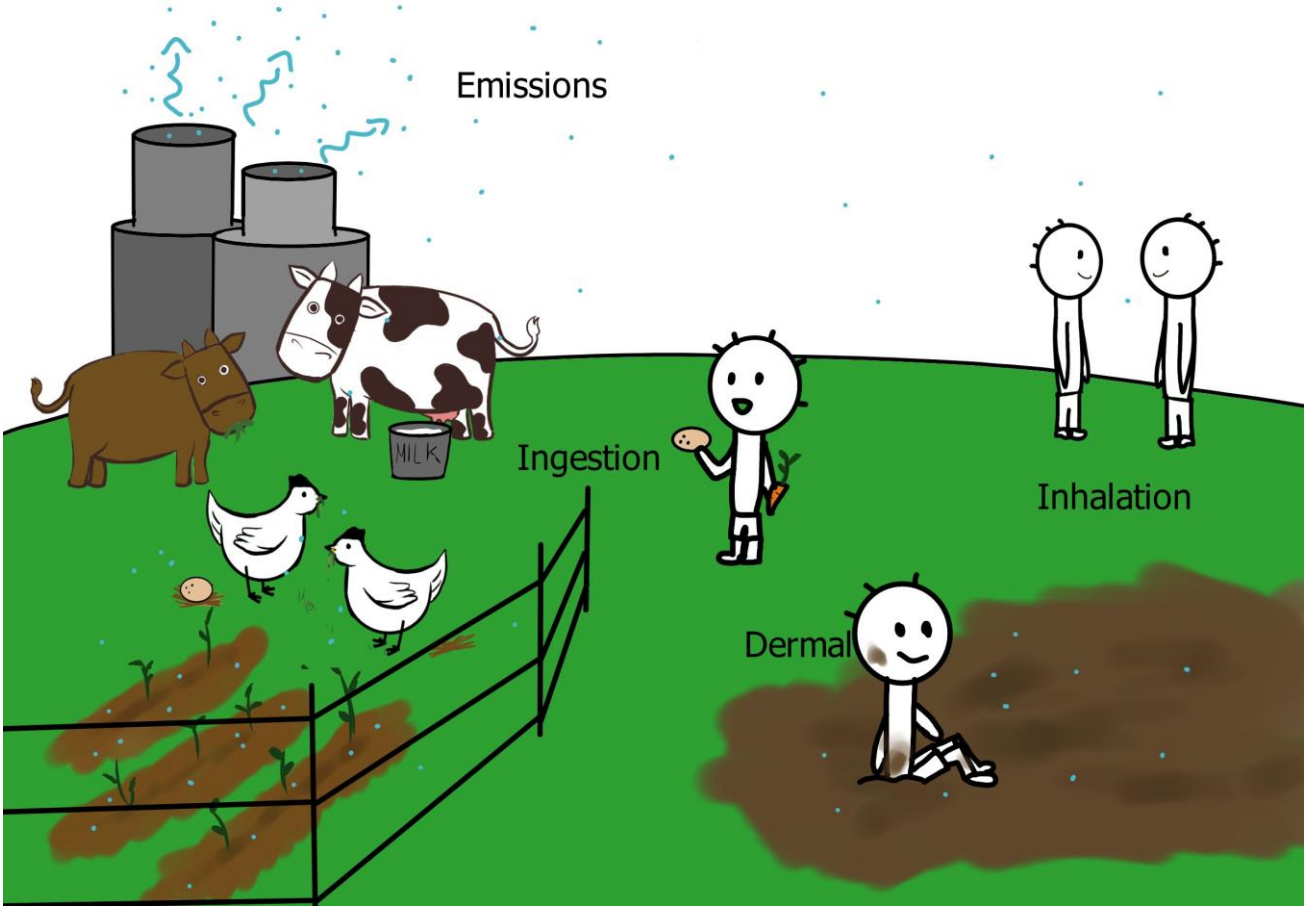


Figure 4.2: Conceptual site model (illustrative only)

4.4 Use of air modelling data in HIA

The air dispersion modelling has predicted ground level concentrations on the basis of the facility operating all of the time at the EU emission limits. Ground level concentrations have been predicted over each of the years of meteorological data considered, with the maximum from these years evaluated for short-duration exposures and annual average data provided separately for each of the modelled years.

It is noted that the modelling has presented results for averaging times relevant to the Victorian EPA Air Quality Management (AQM) SEPP (EPA Victoria 2001) and Victorian EPA Ambient Air Quality (AAQ) SEPP (EPA Victoria 1999 as varied to 2016), which include 3 minute averages, 1 hour averages, 24-hour averages for a wide range of pollutants with the AAQ SEPP also providing annual averages for particulate matter. The focus of this assessment relates to the evaluation of health impacts that may occur as a result of acute and chronic exposures to emissions from the facility. This requires the use of 1 hour average (for the assessment of acute exposures to most pollutants except particulates), 24 hour average data (for short-term exposures to particulates) and

annual average (for the assessment of chronic exposures) data. All data required for use in this assessment has been provided by Jacobs and are from the same model as presented in the AQIA. There has been no adjustments or post processing of the air modelling outputs for use in this assessment.

The modelling undertaken has provided estimated ground level concentrations. The HIA also utilises a deposition rate. Dust deposition was not specifically modelled by Jacobs (2020) however guidance on the assessment of multi-pathway exposures (OEHHA 2015) indicates that for facilities where particulate matter control devices are implemented (as is the case for the proposed facility), a default deposition velocity of 0.02 m/s can be adopted. Where emissions are uncontrolled, the default deposition velocity is 0.05 m/s (refer to **Appendix B4.1** for further detail on the use of this value). To ensure that the assessment of health impacts is sufficiently conservative, the worst-case deposition velocity of 0.05 m/s has been adopted in this assessment. Pollutant specific deposition rates have then been calculated based on the annual average air concentration and the particle deposition velocity of 0.05 m/s. The calculation is concentration (mg/m^3) x particle deposition velocity (m/s) = deposition rate ($mg/m^2/s$). This is then converted to mg/m^2/year for use in the risk calculations.

Risk calculations have been presented for the following locations within the community:

- **Maximum impacted location anywhere** within the study area regardless of location and land use – this is a location on the site or on the site boundary, however for the purpose of this assessment exposures that may occur 24 hours per day, every day have been assumed.
- **Maximum impacted sensitive receptor** – this is the maximum impacted receptor from the individual sensitive receptors listed in **Table 4.1** and shown on **Figure 4.1**. Exposures are assumed to occur for 24 hours per day, every day at this location.

4.5 Inhalation exposures

4.5.1 General

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

4.5.2 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 AQIA (Jacobs 2020). This 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 included in the modelling (as time varying concentrations).

Table 4.3 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 very small contribution to existing concentrations and only makes up a small fraction of the NEPM/SEPP guideline. It is noted that background concentrations of $PM_{2.5}$ are already elevated above the NEPM/SEPP guideline. Elevated background levels of $PM_{2.5}$ are the result of emissions from other regional sources that include road traffic, domestic wood burning, occasional controlled burns and bushfires. The review conducted by Jabobs identified that emissions from the facility are unlikely to change the number of exceedances of the NEPM/SEPP guideline.

Table 4.3: $PM_{2.5}$ impacts from the project – maximum impacts (at any location)**

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 from NEPM/SEPP (NEPC 2016)	25 (20 as goal for 2025)	8 (7 as goal for 2025)
Background (max from all years of data)*	32.7	8.6
Contribution from project (from all years)	0.339	0.018
% contribution of project to NEPM/SEPP	1.3% (1.7% for 2025)	0.2% (0.3% for 2025)
% contribution of project to background	1%	0.2%

* Background concentration based on time-varying data from Geelong South as modelled and assessed by Jacobs (2020). It is noted that this is the maximum from all years of data, namely during 2015. The annual average reported for other years (2016 to 2019) was in the range of 6 to 7 $\mu g/m^3$ which is below the NEPM/SEPP guideline.

**Maximum predicted concentration at any location modelled in the study area

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 the Greater Geelong LGA (all ages and all causes – refer to **Table 3.2**), 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 0.018 $\mu g/m^3$, this results in a maximum individual risk of $6x10^{-7}$. 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.

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

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

Where:
Exposure concentration = calculated from the concentration in air derived from the air modelling (mg/m^3)
Acute TRV = health based toxicity reference value (TRV) or guideline that is protective of short-duration exposures for all members of the community including sensitive individuals, as per **Appendix B** (mg/m^3)

The above calculation relates to acute inhalation exposures from the proposed facility only. The assessment of acute exposures included background intakes for NEPM pollutants (i.e. where data is available to assess short-duration background exposures). It should be noted that in relation to the NEPM pollutants, background data as outlined by Jabobs (2020) has been considered in this review.

For this assessment, the maximum predicted 1-hour average concentration at any location within the study area, as well as the maximum predicted 1-hour average concentration at the sensitive receptors has been considered. This has been done to address acute inhalation exposures that may occur in these areas.

The acute health based guidelines or TRVs adopted in this assessment have been adopted on the basis of the approach detailed in **Appendix B**.

Table 4.4 presents a summary of the relevant health-based guideline, the predicted maximum 1-hour average concentrations, and the maximum impacted receptor, and the calculated HI for each pollutant. Exposures at all other locations, including the other sensitive receptors will be lower than presented in **Table 4.4**.

To address additive exposures, for pollutants where there is sufficient information available to understand how these chemicals may cause health effects, and where these can be calculated separately (i.e. no interactions or additive effects with other air pollutants) the HI has been presented separately. This applies to the assessment of exposures to nitrogen dioxide (NO_2), sulfur dioxide (SO_2) and carbon monoxide (CO), where the NEPM guidelines have been adopted. The NEPM guidelines apply to individual pollutants and 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, less is well understood and hence the approach outlined by enHealth (enHealth 2012a)

has been adopted to address additive exposures, where the individual HI have been summed, as follows:

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

The total HI is presented in **Table 4.4**. Risks associated with acute exposures are considered to be acceptable where the individual and total HI's are less than or equal to 1.

Table 4.4: 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 sensitive receptors	Maximum anywhere*	Maximum sensitive receptors
NEPM pollutants					
Nitrogen dioxide (NO_2)	0.22[1]	9.6E-02	9.6E-02	4.4E-01	4.4E-01
Sulfur dioxide (SO_2)	0.5[1]	1.0E-01	7.8E-02	2.1E-01	1.6E-01
Carbon monoxide (CO)	30[1]	3.6E+00	3.6E+00	1.2E-01	1.2E-01
Other Pollutants					
Hydrogen chloride (HCl)	0.66[2]	1.7E-02	8.0E-03	2.6E-02	1.2E-02
Hydrogen fluoride (HF)	0.06[2]	1.2E-03	5.3E-04	1.9E-02	8.8E-03
Ammonia	0.59[2]	8.6E-03	4.0E-03	1.5E-02	6.8E-03
VOCs and formaldehyde	0.05[2]	5.7E-03	2.5E-03	1.1E-01	5.0E-02
Cadmium	0.0054[2]	5.7E-06	2.7E-06	4.0E-04	4.9E-04
Mercury (as elemental)	0.0006[3]	5.7E-07	2.7E-07	3.6E-04	4.4E-04
Antimony	0.001[4]	8.6E-06	4.0E-06	3.2E-03	4.0E-03
Arsenic	0.003[2]	1.7E-05	8.0E-06	2.1E-03	2.7E-03
Chromium (Cr VI assumed)	0.0013[2]	1.7E-05	8.0E-06	5.0E-03	6.1E-03
Cobalt	0.00069[2]	8.6E-07	4.0E-07	4.7E-04	5.8E-04
Copper	0.1[3]	8.6E-05	4.0E-05	3.2E-04	4.0E-04
Manganese	0.0091[2]	1.7E-05	8.0E-06	7.1E-04	8.8E-04
Nickel	0.0011[2]	1.7E-05	8.0E-06	5.9E-03	7.2E-03
Vanadium	0.03[3]	8.6E-07	4.0E-07	1.1E-05	1.3E-05
			Total HI (for other pollutants)	**0.19**	**0.10**
			Target (acceptable/negligible HI)	**≤1**	**≤1**

* Maximum anywhere is the maximum concentration (and calculated HI) at any of the modelled locations within the whole study area, regardless of land use or the presence of a residential property. This is different to the sensitive receptors which is where there are existing residential, or other sensitive uses

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 Agency for Toxic Substances and Disease Registry (ATSDR), as an acute air guideline (relevant to exposures from 1 hour to 14 days) https://www.atsdr.cdc.gov/mrls/index.html

Based on the assessment presented in **Table 4.4**, all the individual and total HI's are less than 1. For NO_2, SO_2 and CO the calculated acute exposure risks remain acceptable even where the limited background concentrations are considered (as discussed above, which would result in an individual HI of 0.4 for NO_2, 0.2 for SO_2 and 0.1 for CO).

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

Chronic exposures

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

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

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

For the assessment of exposures to benzo(a)pyrene (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:

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

Where:
TRV = non-threshold toxicity reference value relevant to calculating the carcinogenic risk associated with an inhalation exposure (relevant to exposures within the community, and protective of all members of the community) (refer to **Appendix B**) $(mg/m^3)^{-1}$

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.

Appendix B presents the relevant health-based values adopted in these calculations, along with assumptions adopted for the assessment of background intakes and the quantification of inhalation exposures for the calculation of the HI and incremental lifetime risk. **Appendix C** presents the calculations undertaken for residential and industrial inhalation exposures.

Table 4.5 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 in the whole study area (i.e. anywhere) as well as the maximum predicted at the sensitive receptors.

To address additive exposures, for pollutants where there is sufficient information available to understand how these chemicals may cause health effects, and where these can be calculated separately (i.e. no interactions or additive effects with other air pollutants) the HI has been presented separately. This applies to the assessment of exposures to nitrogen dioxide (NO_2) and sulfur dioxide (SO_2), where the NEPM guidelines have been adopted. The NEPM guidelines apply to individual pollutants and 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, less is well understood and hence the approach outlined by enHealth (enHealth 2012a) has been adopted to address additive exposures, where the individual HI have been summed, as follows:

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

The total HI is presented in **Table 4.5**.

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 4.5**, 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 4.5: Calculated chronic risks*

Pollutant	Calculated Incremental Lifetime Risk		Calculated HI	
	Maximum anywhere**	Maximum sensitive receptors	Maximum anywhere**	Maximum sensitive receptors
NEPM pollutants				
Nitrogen dioxide (NO_2)	--	--	0.22	0.22
Sulfur dioxide (SO_2)	--	--	0.026	0.025
Other pollutants				
Hydrogen chloride (HCl)	--	--	0.0017	0.0010
Hydrogen fluoride (HF)	--	--	0.00024	0.00014
Ammonia	--	--	0.00023	0.00013
VOCs as formaldehyde			0.0066	0.0037
Cadmium	--	--	0.0068	0.0038
Thallium	--	--	0.0000049	0.0000027
Mercury (as elemental)	--	--	0.000015	0.0000085
Antimony	--	--	0.00020	0.00012
Arsenic	--	--	0.00008	0.000046
Lead	--	--	0.00082	0.00046
Chromium (Cr VI assumed)	--	--	0.00082	0.00046
Cobalt	--	--	0.000041	0.000023
Copper	--	--	0.00000084	0.00000047
Manganese	--	--	0.00068	0.00038
Nickel	--	--	0.0051	0.0029
Vanadium	--	--	0.000041	0.000023
Dioxin	--	--	0.000059	0.000033
BaP	1.5×10^{-8}	5.0×10^{-9}	--	--
Total HI (other pollutants)			**0.024**	**0.013**
Negligible risk	**≤1x10^{-6}**	**≤1x10^{-6}**	**≤1**	**≤1**

* Refer to **Appendix C** for detailed calculations of the risk and HI, and **Appendix B** for the toxicity reference values adopted in the calculations

** Maximum anywhere is the maximum concentration (and calculated HI) at any of the modelled locations within the whole study area, regardless of land use or the presence of a residential property. This is different to the sensitive receptors which is where there are existing residential, or other sensitive uses

It is noted that the margin of safety (MOS) relevant to inhalation exposures ranges from 40 to 80 for the total HI[1], with the MOS higher than this for many individual pollutants. This is more than sufficient to address any likely changes in guidelines that may be applicable to these pollutants over time.

[1] The MOS is calculated as the ratio of the target/acceptable HI: calculated total HI. Hence for the assessment of exposures at the maximum impacted location anywhere the MOS is calculated to be 1/0.024 = 40 (rounding to 1 significant figure). For the assessment of exposures at the maximum impacted sensitive receptor the MOS is calculated as 1/0.013 = 80 (rounded to 1 significant figure).

4.6 Multiple pathway exposures

4.6.1 General

Where pollutants may be bound to particulates (as PM_{10}), 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.

4.6.2 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. The maximum impacts predicted from the facility are located on the site, or on the site boundary, where exposures evaluated for residential and agricultural type exposures cannot occur. Hence risks associated with multiple pathway exposures are of most relevance to the assessment of impacts at the off-site sensitive receptors. It is noted that for this site, the closest rural residential property (Minyip Road) is located close to the site boundary, approximately 0.3 km. As a result ,there is little difference in the calculated risks for the maximum sensitive receptor and the maximum anywhere in the study area.

Assessment of multi-pathway exposures for the closest sensitive receptor are relevant (give the rural residential use of the area), and also protective of these exposures should they occur at any of the other sensitive receptor areas.

The calculation of risks posed by multiple pathway exposures only relates to pollutants that are bound to the particulates. The calculations undertaken has utilised a deposition rate, which is derived from the air modelling as detailed in **Section 4.4**.

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 4.5.3**, 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 ≤ $1x10^{-6}$ = negligible risk, and ≤ $1x10^{-5}$ = acceptable risk.

4.6.3 Calculated risks

Table 4.6 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 sensitive receptors in the surrounding community as this is representative of the maximum impacted rural residential location, and provides a conservative estimation of risks relevant to other rural residential and urban residential areas. 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 4.6**.

Table 4.6: Summary of risks for multiple pathway exposures (maximum sensitive receptor)*

Exposure pathway	Calculated risks - Adults		Calculated risks - Children	
	Non-threshold Risk	HI	Non-threshold Risk	HI
Individual exposure pathways				
Inhalation (I)	$5.0 x10^{-9}$	0.0095	$5.0 x10^{-9}$	0.0095
Soil ingestion (SI)	$5.1 x10^{-10}$	0.0048	$9.8 x10^{-10}$	0.045
Soil dermal contact (SD)	$1.9 x10^{-9}$	0.00088	$7.9 x10^{-10}$	0.0018
Ingestion of homegrown fruit and vegetables (F&V)	$3.9 x10^{-9}$	0.0032	$3.0 x10^{-9}$	0.0082
Ingestion of homegrown eggs (E)	$9.4 x10^{-13}$	0.00080	$3.9 x10^{-13}$	0.0016
Ingestion of homegrown beef (B)	$5.8 x10^{-9}$	0.011	$3.0 x10^{-9}$	0.028
Ingestion of homegrown dairy milk (at property) (M)	$4.7 x10^{-8}$	0.021	$3.5 x10^{-8}$	0.084
Multiple pathways (i.e. combined exposure pathways)				
I + SI + SD	$7.4 x10^{-9}$	0.015	$6.7 x10^{-9}$	0.056
I + SI + SD + F&V	$1.1 x10^{-8}$	0.018	$9.7 x10^{-9}$	0.064
I + SI + SD + E	$7.4 x10^{-9}$	0.016	$6.7 x10^{-9}$	0.058
I + SI + SD + F&V + E	$1.1 x10^{-8}$	0.019	$9.7 x10^{-9}$	0.066
I + SI + SD + B	$1.3 x10^{-8}$	0.026	$9.7 x10^{-9}$	0.084
I + SI + SD + M	$5.4 x10^{-8}$	0.036	$4.5 x10^{-8}$	0.14
I + SI + SD + F&V + E + B	$1.7 x10^{-8}$	0.030	$1.3 x10^{-8}$	0.093
I + SI + SD + F&V + E + M	$5.8 x10^{-8}$	0.024	$4.8 x10^{-8}$	0.15
Negligible risk	**≤1 $x10^{-6}$**	**≤1**	**≤1 $x10^{-6}$**	**≤1**

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

Review of **Table 4.6** 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.

The MOS relevant to the calculated multi-pathway risks range from 7 to 67 for the maximum impacted sensitive receptor[2], which is the most reasonable calculation for these exposures.

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 existing rural residential and residential use of the surrounding areas.

4.7 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 maintained under negative pressure, which will provide management of odour emissions while one of the boilers is operations.

In the situation where there may be a short-term outage of the boiler, odours from the bunker would be minimised by a stack ventilation shutdown system to maintain negative pressure in the bunker and tipping hall, and an odour filtration system prior to the discharge point located on the facility roof for good dispersion.

4.8 Outcomes of health impact assessment

Table 4.7 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 4.7: 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. The proper operation of the tipping hall as proposed to ensure fugitive odour emissions are effectively managed.

[2] The MOS is calculated as the ratio of the target/acceptable HI: calculated total HI. Hence for the assessment of exposures at the maximum impacted location anywhere the MOS is calculated to be 1/0.024 = 40 (rounding to 1 significant figure). For the assessment of exposures at the maximum impacted sensitive receptor the MOS is calculated as 1/0.013 = 80 (rounded to 1 significant figure).

Section 5. Health impacts: Noise

5.1 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 Noise Assessment Report (Jacobs 2020).

As discussed in **Section 2.1**, The site is located within an Industrial 2 zone within the Greater City of Geelong. The nearest sensitive receptors to the proposed project have been identified (**Figure 4.1**) include a number of rural residential properties along Munyip Road and Gibbons Road to the north-west of the site, noting that the closest residential property is 0.3 km from the site.

5.2 Summary of noise assessment

5.2.1 General

The noise assessment was based on the *State Environment Protection Policy (Control of Noise from Commerce Industry and Trade) No. N-1* (SEPP N-1), with criteria outlined in the guideline – Noise from Industry in Regional Victoria (NIRV, Publication 1411, October 2011) applicable for the area evaluated. 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 22 off-site sensitive receptor locations along Minyip Road and Gibbons Road. Both the RMNLs and ERMNLs were developed to ensure compliance with the NIRV guideline.

5.2.2 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 expected to be used (as per the proposed design) and the likely operating hours. All equipment was assumed to be operating continuously and simultaneously for the day, evening and night periods. The location of these equipment on the site, including within buildings were considered. Standard noise mitigation measures where considered in the assessment.

Noise modelling was undertaken using the acoustic software package SoundPLAN (version 8.0). The modelling considered neutral and adverse meteorological conditions relevant to the day, evening and night periods.

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) at all receptors.

It is expected that more specific noise mitigation measures can be incorporated into the facility during the detailed design phase, which would be expected to reduce noise levels further. These additional mitigation measures are detailed in the noise assessment.

5.3 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 5.1**.

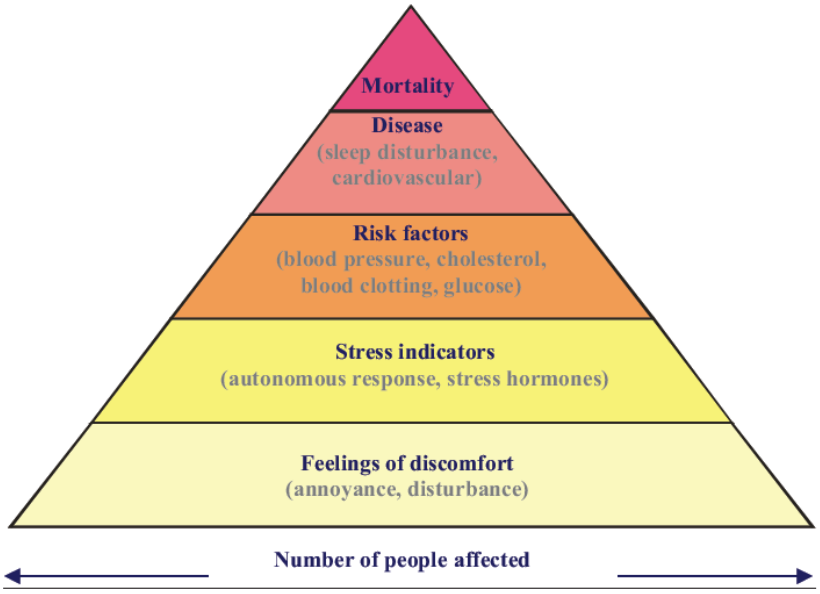


Figure 6.1: Schematic of severity of health effects of exposure to noise and the number of people affected (WHO 2011)

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 standard mitigation measures considered. Based on the assessment undertaken, noise levels at all sensitive receptors comply with the relevant guidelines and would be protective of health.

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

5.4 Outcomes of health impact assessment: noise

Table 5.1 presents a summary of the outcomes of the assessment undertaken in relation to the impacts of changes in noise, associated with the proposed project, on community health.

Table 5.1: 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 noise levels 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 low
Mitigation	The plant is currently in a concept stage of design. Further noise modelling and the inclusion of additional noise mitigation measures would be expected to be considered in the detailed design, which would result in a further reduction of noise from the facility.

Section 6. Health impact assessment: Economics, waste and transport

6.1 Approach

Health impacts associated with other aspects of the proposed project, including the management and handling of waste and traffic impacts. This review has relied on information available in the following reports:

- Concept design basis report, Jacobs – 15 April 2020
- Traffic Impact Assessment, Jacobs – 12 June 2018
- Waste Chapter, Jacobs – 27 May 2020.

6.2 Overview and assessment of issues

Economics

The proposed project will result in the direct employment of 300-400 full time staff during the construction phase and 50-60 full time staff during the operational phase. It is expected that there would also be an increase in 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. Lara and Greater Geelong have lower rates of unemployment than Victoria, while Corio has a higher rate of unemployment. Therefore, improvements in health and wellbeing in the local community can be enhanced by encouraging local employment, particularly from areas where unemployment is higher, 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 a peak of 400 light vehicles (for staff) and 390 heavy vehicles a day (for plant and equipment) with most movements occurring during peak hour periods.

When in operation, the predicted increase is expected to be 40 light vehicles (for staff) and 93 heavy vehicles (delivering waste, consumable and chemicals, and ash and scrap metal removed).

A review of the traffic movements related to the project concluded that the existing road performance would not be adversely impacts and "*it is likely that the traffic generated from this project will have negligible adverse impact to traffic performance*" at key intersections during construction. Further traffic impact assessment works, including the preparation of traffic

management plan(s) are proposed in relation to the next stages of the planning process for the project.

Hazardous waste

The proposed project would not procure waste streams as feedstock that do not meet the requirements of the Victorian EPA Energy from Waste Guidelines. That is, any waste streams that can be feasibly reused or recycled will not be targeted for the purposed of recovery by thermal processing. Hence the following input types will not be targeted:

- source separated household, commercial and industrial recycling streams
- bulky / drop off household wastes and other municipal wastes such as street sweepings
- commercial and industrial waste types that are not considered to be appropriate feedstocks, such as medical wastes
- construction and demolition waste
- prescribed industrial wastes (e.g. asbestos, unprocessed used cooking fats and oils).

Prescribed industrial waste (PIW) such as asbestos, PVC, treated wood waste, dangerous goods and clinical waste will not be sought as a procurement option. EfW facilities operating to a temperature of 850°C must also meet with feedstock criteria which states that halogenated organic substances, expressed as chlorine, should comprise of no more than 1% of the feedstock. PIW can often contain high levels of chlorine (or other hazardous substances in elevated concentrations) which is why the aforementioned wastes have not been targeted, and no hazardous waste will be accepted as feedstock.

Quality assurance processes will also be implemented to reduce the potential for contamination or the presence of recyclable materials to be present.

Feedstock will be managed during operation of the proposed facility. The management measures would include:

1. Waste Acceptance Criteria. This would detail the waste that is deemed hazardous and not accepted by the facility.
2. Waste inspection. This would occur at the waste transfer as well as at the weighbridge upon entry to the facility. If a problem or hazard is suspected the material would be further inspected at an inspection area. Any waste classified as hazardous would be separated and disposed separately. The feedstock would also inspected upon tipping into the bunker.
3. Periodic auditing and independent auditing of feedstock to ensure incoming materials comply with EPA regulatory requirements.

Where these measures are implemented the potential for hazardous waste to be present in feedstock is minimised, and no further assessment of potential health impacts for off-site communities is required.

Generated waste

The operation of the proposed facility would generate the following waste materials:

- Incinerator Bottom Ash (IBA), this is the solid residue removed from the combustion chamber after the waste has been thermally treated
- boiler ash, the part of the fly ash that is removed from the boiler
- Air Pollution Control (APC) residues (also known as Flue Gas Treatment (FGT) residues) from the APC equipment.

These waste materials would be categorised appropriately (in accordance with EPA waste classification guidelines) and appropriately disposed (potentially following treatment for some materials) or re-used as permitted. In relation to the IBA, these materials may be treated for re-use as an aggregate material for the construction industry. In addition, it is possible to extract ferrous and non-ferrous metals from bottom ash for recycling.

Where these waste materials are appropriately tested, categorised, treated, disposed or re-used in accordance with relevant current regulations and guidance, they would not be of concern to community health.

Section 7. Summary of HIA Outcomes

Based on the evaluations presented in **Section 4** to **6**, **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 low to 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 7.1**.

Table 7.1: 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 4.4	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 4.5	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 4.6	Annoyance, stress, anxiety	**Not significant and negligible**	The proper operation of the tipping hall as proposed to ensure fugitive odour emissions are effectively managed on-site.
Noise	Section 5	Sleep disturbance, annoyance, children's school performance and cardiovascular health	**Modelled noise impacts: low potential for health impacts**	The plant is currently in a concept stage of design. Further noise modelling and the inclusion of additional noise mitigation measures would be expected to be considered in the detailed design, which would result in a further reduction of noise impacts within the community.
Economic Environment	Section 6	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 (particularly in areas with higher levels of existing unemployment).

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 6	Injury or death, stress and anxiety.	**Negative but minimal**	Details to be determined at the detailed design phase of the project.
Presence of hazardous waste in feedstock and generation of waste	Section 6	Possible injury if incorrectly disposed of	**Negative but minimal**	Further development of the proposed feedstock delivery protocols into an operational management plan to address the discovery and proper disposal of hazardous waste, should.it be present in feedstock. Appropriate testing and management of waste materials generated during operations, with compliance with all relevant current regulations in relation to waste disposal and/or re-use.

Section 8. References

Project specific reports:

Jacobs 2020, Concept design basis report, Prospect Hill Energy from Waste Plant. Memorandum prepared by Jacobs dated 15 April 2020.

Jacobs 2020, Energy from Waste, Noise Assessment Report. Report prepared by Jacobs for Prospect Hill International Pty Ltd, dated 19 September 2020.

Jacobs 2020, Prospect Hill Energy from Waste Plant, Traffic Impact Assessment, Report prepared by Jacobs for Prospect Hill International Pty Ltd (PHI), dated 12 June 2020.

Jacobs 2020, Prospect Hill EfW Plant, Waste Chapter.

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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>.

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

Calculation of risk: $PM_{2.5}$

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

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

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

The assessment of potential risks associated with exposure to particulate matter involves the calculation of a relative risk (RR). For the purpose of this assessment the shape of the exposure-response function used to calculate the relative risk is assumed to be linear[3]. The calculation of a relative risk based on the change in relative risk exposure concentration from baseline/existing (ie based on incremental impacts from the project) can be calculated on the basis of the following equation (Ostro 2004):

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

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

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

Equation 2

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

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

The assessment of health impacts for a particular population associated with exposure to particulate matter has been undertaken utilising the methodology presented by the WHO (Ostro 2004)[4] where the exposure-response relationships identified have been directly considered on the basis of the approach outlined below.

An additional risk can be calculated as:

Equation 3 **Risk=β x ΔX x B**

Where:
β = slope coefficient relevant to the per cent change in response to a 1 µg/m³ change in exposure
ΔX = change (increment) in exposure concentration in µg/m³ relevant to the project at the point of exposure
B = baseline incidence of a given health effect per person (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) adopted in this calculation. For this assessment the change in $PM_{2.5}$ relates to the change in annual average air concentrations and the value considered in this assessment is 0.018 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 Greater Geelong LGA, with the most recent data indicating a rate of 540 per 100,000 as an age standardised rate which has been adopted in this assessment.

4 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).

- 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 (RR) of all-cause mortality of 1.06 per 10µg/m^3 change in $PM_{2.5}$, and that this risk relationship was in the form of an exponential function. Based on a RR of 1.06 per 10µg/m^3 change in $PM_{2.5}$, this results in a $\beta = 0.0058$. It is noted that the exposure response relationship established in this study was re-affirmed in a follow-up study (that included approximately 500,000 participants in the US) (Krewski et al. 2009) and is consistent with findings from California (Ostro et al. 2006). The relationship is also more conservative than a study undertaken in Australia and New Zealand (EPHC 2010).

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

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

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

$$= 0.018 \times 0.00540 \times 0.0058$$

$$= 6 \times 10^{-7}$$

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 Identification of 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 for the assessment of acute exposures

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

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

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

- Acute guidelines relevant to a 1-hour average exposure period are preferred.
- The TRVs have been selected on the basis of the following hierarchy:
  1. NEPM Ambient Air Quality guideline, relevant to 1-hour average exposures (NEPC 2016).
  2. Texas Commission on Environmental Quality (TCEQ) Acute Reference Value (Acute ReV), which is based on a target HI of 1, consistent with the target HI adopted in the derivation of guidelines in Australia (enHealth 2012a; NEPC 1999 amended 2013b, 2004) by the WHO (WHO 2000a, 2000b, 2010). These are used as the primary source of acute guidelines as they specifically relate to and consider studies relevant to a 1-hour exposure and they have undergone the most recent detailed review process.
  3. California Office of Environmental Health Hazard Assessment (OEHHA) acute Reference Exposure Level (REL), which are all based on a target HI of 1 with RELs relevant to 1-hour average exposures adopted.

Some pollutants are not considered to be acute toxicants, which means that they have a very low acute toxicity and as a result there are no suitable and robust acute inhalation guidelines available. For these pollutants the assessment of chronic exposure is of most importance, which is evaluated on the basis of appropriate chronic toxicity values (discussed below). The pollutants where acute inhalation exposures have not been quantified are thallium, lead, dioxins and furans and PAHs.

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

Table B1: Acute TRVs adopted in this assessment

Pollutants	Acute air guideline (1-hour average) (mg/m^3)
NEPM pollutants	
Nitrogen dioxide (NO_2)	0.22[1]
Sulfur dioxide (SO_2)	0.5[1]
Carbon monoxide (CO)	30[1]
Other pollutants	
Hydrogen chloride (HCl)	0.66[2]
Hydrogen fluoride (HF)	0.06[2]
Ammonia	0.59[2]
VOCs as formaldehyde	0.05[2]
Cadmium	0.0054[2]
Thallium	NA – Not an acute toxicant
Mercury	0.0006[3]
Antimony	0.001[4]
Arsenic	0.003[2]
Lead	NA – Not an acute toxicant
Chromium (Cr VI assumed)	0.0013[2]
Cobalt	0.00069[2]
Copper	0.1[3]
Manganese	0.0091[2]
Nickel	0.0011[2]
Vanadium	0.03[3]
Dioxins and furans	NA – Not an acute toxicant
PAHs (as BaP)	NA – Not an acute toxicant

References

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 Agency for Toxic Substances and Disease Registry (ATSDR), as an acute air guideline (relevant to exposures from 1 hour to 14 days) https://www.atsdr.cdc.gov/mrls/index.html

Values adopted for the assessment of chronic exposures

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

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

For this assessment the following pollutants have been classified as class 1 carcinogens by the International Agency for Research on Cancer (IARC), and a review has been undertaken on the mechanism of action relevant to the way in which they cause cancer as follows:

- Arsenic – the mechanism by which cancer is caused does not appear to be mutagenic with a threshold mode of action identified for the assessment of cancer (where damage to cells and sufficient exposure to result in cancer proliferation required) (NEPC 1999 amended 2013b). Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.
- Beryllium - review of genotoxicity by IARC (IARC 2012) indicates that the evidence for mutagenic activity was weak or negative (i.e. non-genotoxic), and review by NHMRC and NEPC (NEPC 1999 amended 2013b; NHMRC 2011 updated 2018) indicates that a threshold mode of action is relevant for the assessment of cancer. Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.
- Benzo(a)pyrene – this is considered to be a genotoxic carcinogen and has been assessed on the basis of a non-threshold TRV (NEPC 1999 amended 2013b)
- Cadmium – the available data suggests only weak evidence of genotoxicity and review by NEPC (NEPC 1999 amended 2013b) indicates that a threshold mode of action is relevant for the assessment of cancer. Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.
- Chromium VI – the available data suggests the compound may have some genotoxic potential however review by NEPC (NEPC 1999 amended 2013b) indicates that carcinogenicity is likely to act on the basis of a threshold mode of action. Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.
- Formaldehyde – the available information suggests the compound is a weak genotoxin, the mechanism of action for carcinogenicity is considered to be via a threshold (being a point of contact carcinogen with complex mechanisms such as cell damage/death required prior to the induction of cancer) (TCEQ 2013). Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.
- Nickel – the available data indicates that the compound may be genotoxic, however the mechanism of action is not well understood. The WHO (WHO 1991) indicates that very high concentrations of nickel are required to produce genotoxic effects (after cell damage/death) and hence a threshold mode of action is considered appropriate (NEPC 1999 amended 2013b). Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.
- Dioxins and furans, as 2,3,7,8-TCDD – review of carcinogenicity by NHMRC (NHMRC 2002) and the WHO (FAO/WHO 2018; WHO 2019) indicates that TCDD is not genotoxic and hence a threshold approach is considered appropriate. Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.

All chronic TRVs adopted for the assessment of chronic exposures are protective of all adverse health effects for all members of the community including sensitive groups such as children and the elderly.

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 B2 and B3 present the TRVs adopted for the assessment of chronic health effects associated with exposure to the other pollutants considered in this assessment. **Table B2** presents the threshold TRVs, while **Table B3** presents the non-threshold TRVs.

Table B2: 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)	
					Oral/dermal**	Inhalation**
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%
VOCs as formaldehyde	0.011 T	NA (gaseous pollutant)			0%	0%
Cadmium	0.000005 W	0.0008 W	100%	0	60%	20%
Thallium	0.0028 R	0.0008 U	3%	0	0%	0%
Mercury (as inorganic and elemental)	0.0002 W	0.0006 W	7%	0.001	40%	10%
Antimony	0.0002 U	0.00086 NH	15%	0	0%	4%
Arsenic	0.001 D	0.002 N	100%	0.005	50%	0%
Lead	0.0005 N	0.0035 NH	100%	0	50%	0%
Chromium (Cr VI assumed)	0.0001 U	0.001 A	100%	0	10%	0%
Cobalt	0.0001 W	0.0014 D	100%	0.001	20%	0%
Copper	0.49 R	0.14 W	100%	0	60%	0%
Manganese	0.00015 W	0.14 A	100%	0	50%	20%
Nickel	0.00002 E	0.012 W	100%	0.005	60%	20%
Vanadium	0.0001 A	0.002 D	100%	0	0%	20
Dioxins and furans	8.05E-09 R	2.3E-09 NH	100%	0.03	54%	54%

Table B3: 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 B2 and B3:

* 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 2012b, 2012a, 2012c)

D = TRV available from RIVM (Baars et al. 2001; van Vlaardingen, Posthumus & Posthuma-Doodeman 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 2013, 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, 2000b, 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 B4** 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 B4: 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
FI	Fraction inhaled from site	100%	All exposures occur at the same location
RF	Dust lung retention factor (unitless)	0.375 for pollutants bound to particles (as PM_{10}) 1 for gasses	Percentage of respirable dust as PM_{10} that is small enough to reach and be retained in the lungs (NEPC 1999 amended 2013b) 100% of gases assumed to reach the lungs
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 Particle deposition modelling

The assessment of multi-pathway exposures has utilised the default deposition velocity from OEHHA (OEHHA 2015). This means the deposition rate is calculated from the predicted/ modelled air concentration and a deposition velocity. The following, as an extract from Lowe et al 1991 (Lowe, Dietrich & Alberts 1991), provides additional information on the use of this approach.

Deposition of particles onto the ground is used to evaluate exposures through the food chain. Pollutant deposition is assumed to be proportional to concentration of pollutants in air. Hence, deposition is estimated using a proportionality constant referred to as a deposition velocity. The deposition velocity is expressed in units of centimetres per second. The methods for evaluating deposition are critical and greatly influence the results of a risk assessment. The assumptions used and the values selected for deposition velocities tend to over predict the magnitude of particle deposition from WTE facilities.

Deposition of particles emitted from WTE facilities is a function of particle size, density, meteorological conditions, and terrain. Deposition is not constant over time changes with variations in wind speed, stability, and vegetative canopy. All of these factors are considered in estimating the rate of deposition of facility-emitted pollutants.

Existing techniques for estimating the rate of deposition are not adequate to provide a realistic simulation. The most commonly used dispersion models were not specifically designed to simulate dispersion of fine particulate matter. EPA-preferred dispersion models typically have a deposition algorithm in which the user specifies a settling velocity computed from Stokes' law and a reflection (or resuspension) coefficient dependent on settling velocity. This type of deposition model, however, is not recommended for particles with a diameter of less than 20 µm for two reasons: (1) the dominant mechanism of deposition for particles with diameters less than 20 µm is not gravity but diffusion and (2) the reflection coefficient for particles with a settling velocity of 3 to 5 cm/s (i.e., particles that are 10 to 20 µm in diameter) is essentially 100%. Hence, the dispersion model used in many regulatory applications assumes 100% reflection, which means that particles do not deposit onto environmental surfaces. It essentially treats emitted particles as gases. The main problem with this approach is that it is not mass conservative, because the model does not subtract mass from the plume. Thus, particulate emitted from a WTE facility are assumed to both deposit from the plume (which is estimated by using a deposition velocity) and are allowed to disperse throughout the air as well. This approach also neglects the influence of meteorology and variations in particle size and terrain in estimating deposition.

However, modern WTE facilities equipped with a dry scrubber and baghouse filter emit a very small fraction of particles in the 20 to 50 µm size range; most emitted particles are Jess than 20 µm in diameter. In the absence of approved models and techniques, several alternative models have been developed for simulating particle deposition. The most common method for simulating dispersion and deposition of particulate matter involves multiplying the modelled concentration in air by a chemical-specific deposition velocity, typically 1 to 2 cm/s. This approach is used in screening

analyses but is considered conservative, since it tends to overestimate deposition and ground-level air concentrations.

A more realistic approach utilizes the work of Sehmel and Hodgson (Sehmel & Hodgson 1978) to estimate a deposition velocity that is dependent on particle size, meteorological conditions, and surface roughness (terrain effects). They also developed (Sehmel & Hodgson 1978) a series of nomographs that relate particle diameter and density to wind speed, stability, and surface roughness. These nomographs have been incorporated into a computer model that was developed by ARB. This model uses hourly meteorological data and a size distribution of the emitted particles to obtain an hourly, size-dependent estimate of deposition velocity. These values are then used to compute deposition fluxes on an hourly basis, which are averaged over 1 year to determine annual average deposition rates. This method is also not mass conservative, but it does provide deposition velocities that are more realistic and reflect changing weather conditions. For a typical WTE facility equipped with a baghouse, the deposition velocity for emitted pollutants can range from 0.05 to approximately 1 cm/s, depending on the particle size distribution used in the model.

Another approach to deposition modelling is currently under development (Tesche et al. 1978). In this approach, Sehmel and Hodgson 's nomographs are used to calculate a deposition rate, but a site-specific reflection coefficient is specified by the user. The difficulty of this approach is selection of a justifiable reflection coefficient, as this task involves an extensive research effort that is not feasible for health risk assessments prepared to support regulatory permitting. To date, this method has not been used or proposed for use in California.

Facility emissions in the Milliken WTE health risk assessment were modelled using the Industrial Source Complex-Short Term (ISCST) dispersion model. Deposition of pollutants onto environmental surfaces was modelled using a modified version of ISCST developed by the Radian Corporation[5] (based on Sehmel and Hodgson 1978), in which deposition is calculated as a function of particle size and meteorological conditions. Particle size distributions were estimated from tests conducted at the WTE facility in Wurzburg, West Germany. Two size distributions were used in the modelling to account for the effects of fine particulate enrichment of organic compounds. Metals were assumed to be an integral part of the fly ash and to be evenly distributed on a mass basis. Semi-volatile organics were assumed to adsorb to the surface of particles during cooling of the flue gas and, thus, would distribute according to surface area. The surface area-weighted distribution (Table 4 below) indicates that over 99% of the total available surface area is in the 0 to 2 µm range. As a result of the different weighing schemes, metals had an estimated deposition rate of 0.6 cm/s, while the semi-volatile organics had an estimated rate of 0.06 cm/s.

Estimated health risks were developed for two exposure scenarios. These scenarios describe the best estimate of upper-bound risk to the maximally exposed individual. These results represent the bounds of estimated risks, given the ranges of values for the data inputs. A deposition velocity of 1 cm/s, recommended by the California Air Resources Board, provides an upper-bound estimate of particle deposition. The lower bound estimate (0.6 cm/s for trace metals and 0.06 cm/s for semi-volatile organic compounds) accounts for the distribution of organic emissions onto particle surface areas.[5]

TABLE 4
Particle Size Distribution Used in Estimating Deposition Velocities[54]

Particle size (μm)	Mass fraction (%)[a]	Area (%)
0.1	13.25	47.1
0.18	13.25	26.2
0.33	13.25	14.3
0.60	13.25	7.9
1.0	4.0	1.4
1.6	3.0	0.7
3.2	10.0	1.1
5.0	8.0	0.6
7.3	8.0	0.4
10.8	6.0	0.2
40.0	8.0	0.1

[a] Original data listed 53.7% by mass less than 0.6 μm. Mass was distributed over the range of 0.1 to 0.6 using a log normal distribution.

End of extract.

Additional comments in relation to deposition velocity

The current default deposition velocities adopted by OEHHA (2015), which come from a review by ARM in 1989 are 5 cm/s for uncontrolled facilities and 2 cm/s for facilities with verifiable particulate matter control devices or facilities that only emit $PM_{2.5}$ (e.g. internal combustion engines) (OEHHA 2015).

A more recent review of deposition velocity which includes the Sehmel–Hodgson model (Mariraj Mohan 2016), further outlines the complexities in modelling deposition of fine particulates. Deposition velocities listed in this paper from literature for particulates that are predominantly PM_{10} are in the range 0.19 to 8.17 cm/s.

Similarly review by Saylor et al (Saylor et al. 2019) also outlines the complexities. The paper indicates that modelled and measured deposition velocities for PM_{10} typically sit in the range of 1 to 10 cm/s with some variability depending on the method used.

The adopted value of 5 cm/s is considered appropriately conservative for use in this assessment, as applied to modelled PM_{10} air concentrations.

B4.2 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 B5**:

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

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 B5**:

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

Table B5: Ingestion and dermal exposure assumptions

Parameter		Value adopted		Basis
		Young children	Adults	
C_M	Concentration of chemical substance in media or relevance (soil [C_S], fruit and vegetables [C_P and C_{RP}], eggs [C_E], beef [C_B] or milk [C_M]) (mg/kg)	Modelled based on deposition of particulates to soil (refer to **Section B4.3**), 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)
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			

Parameter		Value adopted		Basis
		Young children	Adults	
	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

B4.3 Calculation of concentrations in various media

Potential Concentrations in Soil

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

The concentration in soil, which may be the result of deposition following emission of persistent chemical substances, can be calculated using the following equation from Stevens (1991), with assumptions adopted in this assessment presented in **Table B6**.

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

Table B6: 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 = C_A x DV x 86400 x 365		Relevant to areas where multi-pathway exposures may occur
C_A	Concentration in air (mg/m^3)	As modelled for total dust or PM_{10} (based on an annual average)		
DV	Particle deposition velocity (m/s)	0.05 m/s		Default for the deposition of fine particulates for uncontrolled facilities (OEHHA 2015), refer to **Section B4.1**
86400	Conversion from seconds to days	Default conversion of units		
365	Conversion from days to year	Default conversion of units		
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 B7**:

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

The potential uptake of persistent chemical substances into edible crops via the roots can be estimated using the following equation (OEHHA 2015; USEPA 2005), with the parameters and assumptions adopted outlined in **Table B7:**

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

Table B7: 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 B6**
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 B8**:

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

Where P = E for eggs, B = beef and M = milk

Table B8: 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	0.01 kg/day	As per OEHHA (2015) and advice from Ag Vic
	Beef cattle	0.45 kg/day	Based on data from OEHHA 2015 (5% total produce intakes from soil from pasture)
	Lactating cattle	1.1 kg/day	Based on data from OEHHA 2015 (5% total produce intakes from soil from pasture)
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. The value for cobalt is from Australian data (MacLachlan 2011). Other values are the 95% value for the transfer of heavy metals into eggs (Leeman, Van Den Berg & Houben 2007)
	Beef	Chemical specific	Transfer factors adopted from OEHHA (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

Inhalation exposures

Inhalation - gases and particulates

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

Parameters Relevant to Quantification of Community Exposures - Residents		
Exposure Time at Home (ET, hr/day)	24	Assume residents at home or on property 24 hours per day
Fraction Inhaled from Source (FI, unitless)	1	Assume resident at the same property
Dust lung retention factor (unitless)	0.375	Percentage of respirable dust that is small enough to reach and be retained in the lungs (NEPM 1999 amended 2013) - NA for gasses
Exposure Frequency - normal conditions (EF, days/yr)	365	Days at home (normal conditions), 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

Maximum anywhere

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk	Chronic TC Air	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background)	Estimated Concentration in Air - Maximum anywhere (Ca)	Inhalation Exposure Concentration - NonThreshold	Inhalation Exposure Concentration - Threshold	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	1.2E-02	6.2E-03	1.2E-02	--		0.22	
Sulfur dioxide (SO2)	0.0E+00	5.0E-02	0%	5.0E-02	1.3E-03	6.5E-04	1.3E-03	--		0.026	
Hydrogen chloride (HCl)	0.0E+00	2.6E-02	0%	2.6E-02	4.4E-05	2.2E-05	4.4E-05	--		0.0017	10%
Hydrogen fluoride (HF)	0.0E+00	2.9E-02	0%	2.9E-02	7.0E-06	3.5E-06	7.0E-06	--		0.00024	1%
Ammonia	0.0E+00	3.2E-01	0%	3.2E-01	7.3E-05	3.6E-05	7.3E-05	--		0.00023	1%
Cadmium	0.0E+00	5.0E-06	20%	4.0E-06	7.3E-08	1.4E-08	2.7E-08	--		0.0068	40%
Thallium	0.0E+00	2.8E-03	0%	2.8E-03	3.6E-08	6.8E-09	1.4E-08	--		0.0000049	0%
Mercury (as inorganic and element	0.0E+00	2.0E-04	10%	1.8E-04	7.3E-09	1.4E-09	2.7E-09	--		0.000015	0%
Antimony	0.0E+00	2.0E-04	0%	2.0E-04	1.1E-07	2.0E-08	4.1E-08	--		0.00020	1%
Arsenic	0.0E+00	1.0E-03	0%	1.0E-03	2.2E-07	4.1E-08	8.2E-08	--		0.00008	0%
Lead	0.0E+00	5.0E-04	0%	5.0E-04	1.1E-06	2.0E-07	4.1E-07	--		0.00082	5%
Chromium (Cr VI assumed)	0.0E+00	1.0E-04	0%	1.0E-04	2.2E-07	4.1E-08	8.2E-08	--		0.00082	5%
Cobalt	0.0E+00	1.0E-04	0%	1.0E-04	1.1E-08	2.0E-09	4.1E-09	--		0.000041	0%
Copper	0.0E+00	4.9E-01	0%	4.9E-01	1.1E-06	2.0E-07	4.1E-07	--		0.00000084	0%
Manganese	0.0E+00	1.5E-04	20%	1.2E-04	2.2E-07	4.1E-08	8.2E-08	--		0.00068	4%
Nickel	0.0E+00	2.0E-05	20%	1.6E-05	2.2E-07	4.1E-08	8.2E-08	--		0.0051	30%
Vanadium	0.0E+00	1.0E-04	0%	1.0E-04	1.1E-08	2.0E-09	4.1E-09	--		0.000041	0%
Dioxins and furans	0.0E+00	8.1E-09	54%	3.7E-09	5.8E-13	1.1E-13	2.2E-13	--		0.000059	0%
PAHs (as BaP)	6.0E-01	0.0E+00	0%	0.0E+00	1.3E-07	2.4E-08	4.9E-08	1.5E-8		--	
							TOTAL	**1.5E-08**		**0.017**	

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk	Chronic TC Air	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background)	Estimated Concentration in Air - Maximum 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	1.2E-02	6.1E-03	1.2E-02	--		0.22	
Sulfur dioxide (SO2)	0.0E+00	5.0E-02	0%	5.0E-02	1.3E-03	6.3E-04	1.3E-03	--		0.025	
Hydrogen chloride (HCl)	0.0E+00	2.6E-02	0%	2.6E-02	2.5E-05	1.3E-05	2.5E-05	--		0.0010	10%
Hydrogen fluoride (HF)	0.0E+00	2.9E-02	0%	2.9E-02	4.0E-06	2.0E-06	4.0E-06	--		0.00014	1%
Ammonia	0.0E+00	3.2E-01	0%	3.2E-01	4.1E-05	2.1E-05	4.1E-05	--		0.00013	1%
Cadmium	0.0E+00	5.0E-06	20%	4.0E-06	4.1E-08	7.7E-09	1.5E-08	--		0.0038	40%
Thallium	0.0E+00	2.8E-03	0%	2.8E-03	2.0E-08	3.8E-09	7.7E-09	--		0.0000027	0%
Mercury (as inorganic and element	0.0E+00	2.0E-04	10%	1.8E-04	4.1E-09	7.7E-10	1.5E-09	--		0.0000085	0%
Antimony	0.0E+00	2.0E-04	0%	2.0E-04	6.1E-08	1.2E-08	2.3E-08	--		0.00012	1%
Arsenic	0.0E+00	1.0E-03	0%	1.0E-03	1.2E-07	2.3E-08	4.6E-08	--		0.000046	0%
Lead	0.0E+00	5.0E-04	0%	5.0E-04	6.1E-07	1.2E-07	2.3E-07	--		0.00046	5%
Chromium (Cr VI assumed)	0.0E+00	1.0E-04	0%	1.0E-04	1.2E-07	2.3E-08	4.6E-08	--		0.00046	5%
Cobalt	0.0E+00	1.0E-04	0%	1.0E-04	6.1E-09	1.2E-09	2.3E-09	--		0.000023	0%
Copper	0.0E+00	4.9E-01	0%	4.9E-01	6.1E-07	1.2E-07	2.3E-07	--		0.00000047	0%
Manganese	0.0E+00	1.5E-04	20%	1.2E-04	1.2E-07	2.3E-08	4.6E-08	--		0.00038	4%
Nickel	0.0E+00	2.0E-05	20%	1.6E-05	1.2E-07	2.3E-08	4.6E-08	--		0.0029	30%
Vanadium	0.0E+00	1.0E-04	0%	1.0E-04	6.1E-09	1.2E-09	2.3E-09	--		0.000023	0%
Dioxins and furans	0.0E+00	8.1E-09	54%	3.7E-09	3.3E-13	6.2E-14	1.2E-13	--		0.000033	0%
PAHs (as BaP)	6.0E-01	0.0E+00	0%	0.0E+00	4.4E-08	8.3E-09	1.7E-08	5.0E-9		--	
							TOTAL	**5.0E-09**		**0.0095**	

Multi-pathway exposures for maximum sensitive receptor

Soil exposures

Calculation of Concentrations in Soil

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

where:

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

General Parameters		Surface (for direct contact)	Depth (for agricultural pathways)	
Soil bulk density (p)	g/m^3	1600000	1600000	Default for fill materials
General mixing depth (d)	m	0.01	0.15	As per OEHHA (2015) guidance
Duration of deposition (T)	years	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	6.5E-02	2.8E-01	1.9E-02
Thallium	273973	2.5E-06	3.2E-02	1.4E-01	9.4E-03
Mercury (as inorganic and eleme	273973	2.5E-06	6.5E-03	2.8E-02	1.9E-03
Antimony	273973	2.5E-06	9.7E-02	4.2E-01	2.8E-02
Arsenic	273973	2.5E-06	1.9E-01	8.5E-01	5.7E-02
Lead	273973	2.5E-06	9.7E-01	4.2E+00	2.8E-01
Chromium (Cr VI assumed)	273973	2.5E-06	1.9E-01	8.5E-01	5.7E-02
Cobalt	273973	2.5E-06	9.7E-03	4.2E-02	2.8E-03
Copper	273973	2.5E-06	9.7E-01	4.2E+00	2.8E-01
Manganese	273973	2.5E-06	1.9E-01	8.5E-01	5.7E-02
Nickel	273973	2.5E-06	1.9E-01	8.5E-01	5.7E-02
Vanadium	273973	2.5E-06	9.7E-03	4.2E-02	2.8E-03
Dioxins and furans		0.069	5.2E-07	4.7E-07	3.1E-08
PAHs (as BaP)	1.18	0.588	6.9E-02	7.4E-03	4.9E-04

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	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Conversion Factor (CF)	1.00E-06	conversion from mg to kg
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium		8.0E-04	60%	3.2E-04	100%	2.8E-01	8.4E-08	2.0E-07	--		6.3E-04	13%
Thallium		8.0E-04		8.0E-04	100%	1.4E-01	4.2E-08	1.0E-07	--		1.3E-04	3%
Mercury (as inorganic and e		6.0E-04	40%	3.6E-04	100%	2.8E-02	8.4E-09	2.0E-08	--		5.6E-05	1%
Antimony		8.6E-04		8.6E-04	100%	4.2E-01	1.3E-07	3.0E-07	--		3.5E-04	7%
Arsenic		2.0E-03	50%	1.0E-03	100%	8.5E-01	2.5E-07	6.1E-07	--		6.1E-04	13%
Lead		3.5E-03	50%	1.8E-03	100%	4.2E+00	1.3E-06	3.0E-06	--		1.7E-03	36%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	8.5E-01	2.5E-07	6.1E-07	--		7.5E-04	16%
Cobalt		1.4E-03	20%	1.1E-03	100%	4.2E-02	1.3E-08	3.0E-08	--		2.7E-05	1%
Copper		1.4E-01	60%	5.6E-02	100%	4.2E+00	1.3E-06	3.0E-06	--		5.4E-05	1%
Manganese		1.4E-01	50%	7.0E-02	100%	8.5E-01	2.5E-07	6.1E-07	--		8.6E-06	0%
Nickel		1.2E-02	60%	4.8E-03	100%	8.5E-01	2.5E-07	6.1E-07	--		1.3E-04	3%
Vanadium		2.0E-03		2.0E-03	100%	4.2E-02	1.3E-08	3.0E-08	--		1.5E-05	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	4.7E-07	1.4E-13	3.3E-13	--		3.1E-04	7%
PAHs (as BaP)	2.3E-01				100%	7.4E-03	2.2E-09	5.3E-09	5.1E-10	100%	--	
								TOTAL	**5.1E-10**		**4.8E-03**	

Exposure to Chemicals via Incidental Ingestion of Soil

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

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

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	(mg/kg-day)$^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium		8.0E-04	60%	3.2E-04	100%	2.8E-01	1.6E-07	1.9E-06	--		5.9E-03	13%
Thallium		8.0E-04		8.0E-04	100%	1.4E-01	8.1E-08	9.4E-07	--		1.2E-03	3%
Mercury (as inorganic and e		6.0E-04	40%	3.6E-04	100%	2.8E-02	1.6E-08	1.9E-07	--		5.2E-04	1%
Antimony		8.6E-04		8.6E-04	100%	4.2E-01	2.4E-07	2.8E-06	--		3.3E-03	7%
Arsenic		2.0E-03	50%	1.0E-03	100%	8.5E-01	4.8E-07	5.7E-06	--		5.7E-03	13%
Lead		3.5E-03	50%	1.8E-03	100%	4.2E+00	2.4E-06	2.8E-05	--		1.6E-02	36%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	8.5E-01	4.8E-07	5.7E-06	--		7.0E-03	16%
Cobalt		1.4E-03	20%	1.1E-03	100%	4.2E-02	2.4E-08	2.8E-07	--		2.5E-04	1%
Copper		1.4E-01	60%	5.6E-02	100%	4.2E+00	2.4E-06	2.8E-05	--		5.0E-04	1%
Manganese		1.4E-01	50%	7.0E-02	100%	8.5E-01	4.8E-07	5.7E-06	--		8.1E-05	0%
Nickel		1.2E-02	60%	4.8E-03	100%	8.5E-01	4.8E-07	5.7E-06	--		1.2E-03	3%
Vanadium		2.0E-03		2.0E-03	100%	4.2E-02	2.4E-08	2.8E-07	--		1.4E-04	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	4.7E-07	2.7E-13	3.1E-12	--		2.9E-03	7%
PAHs (as BaP)	2.3E-01				100%	7.4E-03	4.2E-09	4.9E-08	9.8E-10	100%	--	
								TOTAL	**9.8E-10**		**4.5E-02**	

Dermal Exposure to Chemicals via Contact with Soil

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

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

Maximum from sensitive receptors

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium		8.0E-04	60%	3.2E-04		2.8E-01			--		--	
Thallium		2.1E-05		2.1E-05		2.5E-01			--		--	
Mercury (as inorganic and elem		4.2E-05	40%	2.5E-05	0.001	2.8E-02	5.3E-10	1.3E-09	--		5.0E-05	6%
Antimony		1.3E-04		1.3E-04		4.2E-01			--		--	
Arsenic		2.0E-03	50%	1.0E-03	0.005	8.5E-01	7.9E-08	1.9E-07	--		1.9E-04	22%
Lead		3.5E-03	50%	1.8E-03		4.2E+00			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		8.5E-01			--		--	
Cobalt		1.4E-03	20%	1.1E-03	0.001	4.2E-02	7.9E-10	1.9E-09	--		1.7E-06	0%
Copper		1.4E-01	60%	5.6E-02		4.2E+00			--		--	
Manganese		1.4E-01	50%	7.0E-02		8.5E-01			--		--	
Nickel		1.2E-02	60%	4.8E-03	0.005	8.5E-01	7.9E-08	1.9E-07	--		4.0E-05	5%
Vanadium		2.0E-03		2.0E-03		4.2E-02			--		--	
Dioxins and furans		2.3E-09	54%	1.1E-09	0.03	4.7E-07	2.6E-13	6.3E-13	--		5.9E-04	68%
PAHs (as BaP)	2.3E-01				0.06	7.4E-03	8.2E-09	2.0E-08	1.9E-9	100%	--	
TOTAL									**1.9E-9**		**8.8E-04**	

Dermal Exposure to Chemicals via Contact with Soil

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

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

Maximum from sensitive receptors

Key Chemical	Toxicity Data					Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)	Dermal Absorption (ABS)		Non-Threshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium		8.0E-04	60%	3.2E-04		2.8E-01			--		--	
Thallium		2.1E-05		2.1E-05		2.5E-01			--		--	
Mercury (as inorganic and elem		4.2E-05	40%	2.5E-05	0.001	2.8E-02	2.2E-10	2.5E-09	--		1.0E-04	6%
Antimony		1.3E-04		1.3E-04		4.2E-01			--		--	
Arsenic		2.0E-03	50%	1.0E-03	0.005	8.5E-01	3.3E-08	3.8E-07	--		3.8E-04	22%
Lead		3.5E-03	50%	1.8E-03		4.2E+00			--		--	
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04		8.5E-01			--		--	
Cobalt		1.4E-03	20%	1.1E-03	0.001	4.2E-02	3.3E-10	3.8E-09	--		3.4E-06	0%
Copper		1.4E-01	60%	5.6E-02		4.2E+00			--		--	
Manganese		1.4E-01	50%	7.0E-02		8.5E-01			--		--	
Nickel		1.2E-02	60%	4.8E-03	0.005	8.5E-01	3.3E-08	3.8E-07	--		7.9E-05	5%
Vanadium		2.0E-03		2.0E-03		4.2E-02			--		--	
Dioxins and furans		2.3E-09	54%	1.1E-09	0.03	4.7E-07	1.1E-13	1.3E-12	--		1.2E-03	68%
PAHs (as BaP)	2.3E-01				0.06	7.4E-03	3.4E-09	4.0E-08	7.9E-10	100%	--	
TOTAL									**7.9E-10**		**1.8E-03**	

Homegrown fruit and vegetables

Calculation of Concentrations in Plants

ref: Stevens B. (1991)

Uptake Due to Deposition in Aboveground Crops

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

where:
DR= Particle deposition rate for accidental release (mg/m^2/day)
F= Fraction for the surface area of plant (unitless)
k= Chemical-specific soil-loss constant (1/years) = ln(2)/$T_{0.5}$
$T_{0.5}$= Chemical half-life as particulate on plant (days)
t= Deposition time (days)
Y= Crop yield (kg/m^2)

Uptake via Roots from Soil

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

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

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

Chemical-specific Inputs and calculations - Maximum sensitive receptors

Chemical	Half-life in plant ($T_{0.5}$)	Loss constant (k)	Deposition Rate (DR)	Aboveground Produce Concentration via Deposition	Root Uptake Factor (RUF)	Soil Concentration (Cs)	Below Ground Produce Concentration
	days	per day	mg/m^2/day	mg/kg ww	unitless	mg/kg	mg/kg ww
Cadmium	14	0.05	1.8E-04	8.8E-05	0.125	1.9E-02	2.4E-03
Thallium	14	0.05	8.8E-05	4.4E-05	0.001	1.7E-02	1.7E-05
Mercury (as inorganic and eleme	14	0.05	1.8E-05	8.8E-06	0.225	1.9E-03	4.2E-04
Antimony	14	0.05	2.7E-04	1.3E-04	0.05	2.8E-02	1.4E-03
Arsenic	14	0.05	5.3E-04	2.6E-04	0.01	5.7E-02	5.7E-04
Lead	14	0.05	2.7E-03	1.3E-03	0.0113	2.8E-01	3.2E-03
Chromium (Cr VI assumed)	14	0.05	5.3E-04	2.6E-04	0.00188	5.7E-02	1.1E-04
Cobalt	14	0.05	2.7E-05	1.3E-05	0.005	2.8E-03	1.4E-05
Copper	14	0.05	2.7E-03	1.3E-03	0.1	2.8E-01	2.8E-02
Manganese	14	0.05	5.3E-04	2.6E-04	0.0625	5.7E-02	3.5E-03
Nickel	14	0.05	5.3E-04	2.6E-04	0.015	5.7E-02	8.5E-04
Vanadium	14	0.05	2.7E-05	1.3E-05	0.00138	2.8E-03	3.9E-06
Dioxins and furans	14	0.05	1.4E-09	7.1E-10	0.000876	3.1E-08	2.7E-11
PAHs (as BaP)	14	0.05	0.0001901	9.5E-05	0.00214	1.5E-03	3.1E-06

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

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables

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

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

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	Root crops concentrations	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	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	60%	3.2E-04	100%	8.8E-05	2.4E-03	1.7E-07	4.0E-07	--		1.3E-03	39%
Thallium		8.0E-04		8.0E-04	100%	4.4E-05	1.7E-05	8.7E-09	2.1E-08	--		2.6E-05	1%
Mercury (as inorganic and element		6.0E-04	40%	3.6E-04	100%	8.8E-06	4.2E-04	2.9E-08	6.9E-08	--		1.9E-04	6%
Antimony		8.6E-04		8.6E-04	100%	1.3E-04	1.4E-03	1.1E-07	2.7E-07	--		3.2E-04	10%
Arsenic		2.0E-03	50%	1.0E-03	100%	2.6E-04	5.7E-04	8.2E-08	2.0E-07	--		2.0E-04	6%
Lead		3.5E-03	50%	1.8E-03	100%	1.3E-03	3.2E-03	4.3E-07	1.0E-06	--		6.0E-04	19%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.6E-04	1.1E-04	5.3E-08	1.3E-07	--		1.6E-04	5%
Cobalt		1.4E-03	20%	1.1E-03	100%	1.3E-05	1.4E-05	3.2E-09	7.7E-09	--		6.9E-06	0%
Copper		1.4E-01	60%	5.6E-02	100%	1.3E-03	2.8E-02	2.0E-06	4.9E-06	--		8.8E-05	3%
Manganese		1.4E-01	50%	7.0E-02	100%	2.6E-04	3.5E-03	2.7E-07	6.6E-07	--		9.4E-06	0%
Nickel		1.2E-02	60%	4.8E-03	100%	2.6E-04	8.5E-04	1.0E-07	2.4E-07	--		5.0E-05	2%
Vanadium		2.0E-03		2.0E-03	100%	1.3E-05	3.9E-06	2.5E-09	6.1E-09	--		3.1E-06	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	7.1E-10	2.7E-11	1.2E-13	3.0E-13	--		2.8E-04	9%
PAHs (as BaP)	2.3E-01				100%	9.5E-05	3.1E-06	1.7E-08	4.0E-08	3.9E-9	100%	--	
									TOTAL	**3.9E-9**		**3.2E-03**	

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables

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

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 (%)	10%	Relevant to urban areas as per NEPM (2013)
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Above ground produce concentration	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	60%	3.2E-04	100%	8.8E-05	2.4E-03	7.2E-08	8.4E-07	--		2.6E-03	32%
Thallium		8.0E-04		8.0E-04	100%	4.4E-05	1.7E-05	6.4E-09	7.4E-08	--		9.3E-05	1%
Mercury (as inorganic and element		6.0E-04	40%	3.6E-04	100%	8.8E-06	4.2E-04	1.2E-08	1.4E-07	--		3.9E-04	5%
Antimony		8.6E-04		8.6E-04	100%	1.3E-04	1.4E-03	5.4E-08	6.3E-07	--		7.3E-04	9%
Arsenic		2.0E-03	50%	1.0E-03	100%	2.6E-04	5.7E-04	5.0E-08	5.8E-07	--		5.8E-04	7%
Lead		3.5E-03	50%	1.8E-03	100%	1.3E-03	3.2E-03	2.6E-07	3.0E-06	--		1.7E-03	21%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	2.6E-04	1.1E-04	3.8E-08	4.5E-07	--		5.5E-04	7%
Cobalt		1.4E-03	20%	1.1E-03	100%	1.3E-05	1.4E-05	2.1E-09	2.5E-08	--		2.2E-05	0%
Copper		1.4E-01	60%	5.6E-02	100%	1.3E-03	2.8E-02	9.0E-07	1.1E-05	--		1.9E-04	2%
Manganese		1.4E-01	50%	7.0E-02	100%	2.6E-04	3.5E-03	1.3E-07	1.5E-06	--		2.1E-05	0%
Nickel		1.2E-02	60%	4.8E-03	100%	2.6E-04	8.5E-04	5.7E-08	6.7E-07	--		1.4E-04	2%
Vanadium		2.0E-03		2.0E-03	100%	1.3E-05	3.9E-06	1.9E-09	2.2E-08	--		1.1E-05	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	7.1E-10	2.7E-11	9.6E-14	1.1E-12	--		1.1E-03	13%
PAHs (as BaP)	2.3E-01				100%	9.5E-05	3.1E-06	1.3E-08	1.5E-07	3.0E-9	100%	--	
									TOTAL	**3.0E-9**		**8.2E-03**	

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	0.01	As per OEHHA (2015) ad advice from AgVIC
B (bioavailability)	%	100%	

Chemical-specific Inputs and calculations - Maximum sensitive receptors

Chemical	Concentration in crops ingested by chickens mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to eggs day/kg	Egg Concentration mg/kg ww	
Cadmium	8.8E-05	1.9E-02	1.0E-02	2.0E-06	
Thallium	4.4E-05	9.4E-03	1.7E-01	1.7E-05	95% from Leeman et al (2007)
Mercury (as inorganic and elementa	8.8E-06	1.9E-03	8.0E-01	1.6E-05	
Antimony	1.3E-04	2.8E-02	1.7E-01	5.1E-05	95% from Leeman et al (2007)
Arsenic	2.6E-04	5.7E-02	7.0E-02	4.2E-05	
Lead	1.3E-03	2.8E-01	4.0E-02	1.2E-04	
Chromium (Cr VI assumed)	2.6E-04	5.7E-02	9.2E-03	5.5E-06	OEHHA (2003)
Cobalt	1.3E-05	2.8E-03	3.3E-03	9.8E-08	MacLachlan (2011)
Copper	1.3E-03	2.8E-01	1.7E-01	5.1E-04	95% from Leeman et al (2007)
Manganese	2.6E-04	5.7E-02	1.7E-01	1.0E-04	95% from Leeman et al (2007)
Nickel	2.6E-04	5.7E-02	2.0E-02	1.2E-05	
Vanadium	1.3E-05	2.8E-03	1.7E-01	5.1E-06	95% from Leeman et al (2007)
Dioxins and furans	7.1E-10	3.1E-08	1.0E+01	4.0E-09	
PAHs (as BaP)	9.5E-05	4.9E-04	3.0E-03	4.9E-08	

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Eggs

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

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Eggs (IRE) (kg/day)	0.014	Ingestion rate of eggs relevant for adults as per enHealth (2012)
Fraction ingested that is homegrown (%)	100%	Assume all eggs consumed in urban area are from backyard chickens
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	29	Time at one residence as adult as per enHealth 2002 and NEPM 1999
Body Weight (BW, kg)	70	For male and females combined (enHealth 2012)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	10585	USEPA 1989 and CSMS 1996

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium		8.0E-04	60%	3.2E-04	100%	2.0E-06	1.6E-10	4.0E-10	--		1.2E-06	0%
Thallium		8.0E-04		8.0E-04	100%	1.7E-05	1.4E-09	3.4E-09	--		4.2E-06	1%
Mercury (as inorganic and element		6.0E-04	40%	3.6E-04	100%	1.6E-05	1.3E-09	3.2E-09	--		8.8E-06	1%
Antimony		8.6E-04		8.6E-04	100%	5.1E-05	4.2E-09	1.0E-08	--		1.2E-05	1%
Arsenic		2.0E-03	50%	1.0E-03	100%	4.2E-05	3.5E-09	8.4E-09	--		8.4E-06	1%
Lead		3.5E-03	50%	1.8E-03	100%	1.2E-04	9.9E-09	2.4E-08	--		1.4E-05	2%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	5.5E-06	4.6E-10	1.1E-09	--		1.4E-06	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	9.8E-08	8.2E-12	2.0E-11	--		1.8E-08	0%
Copper		1.4E-01	60%	5.6E-02	100%	5.1E-04	4.2E-08	1.0E-07	--		1.8E-06	0%
Manganese		1.4E-01	50%	7.0E-02	100%	1.0E-04	8.4E-09	2.0E-08	--		2.9E-07	0%
Nickel		1.2E-02	60%	4.8E-03	100%	1.2E-05	9.9E-10	2.4E-09	--		5.0E-07	0%
Vanadium		2.0E-03		2.0E-03	100%	5.1E-06	4.2E-10	1.0E-09	--		5.1E-07	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	4.0E-09	3.3E-13	7.9E-13	--		7.5E-04	93%
PAHs (as BaP)	2.3E-01				100%	4.9E-08	4.1E-12	9.8E-12	9.4E-13	100%	--	
TOTAL									**9.4E-13**		**8.0E-04**	

Exposure to Chemicals via Ingestion of Eggs

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

Parameters Relevant to Quantification of Exposure by Young children		
Ingestion Rate of Eggs (IRE) (kg/day)	0.006	Ingestion rate of eggs relevant for young children as per enHealth (2012)
Fraction ingested that is homegrown (%)	100%	Assume all eggs consumed in urban area are from backyard chickens
Matrix effect (unitless)	1	Assume chemicals ingested in produce is 100% bioavailable
Exposure Frequency (EF, days/year)	365	Days at home (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	6	Duration as young child
Body Weight (BW, kg)	15	Representative weight as per NEPM (2013)
Averaging Time - NonThreshold (Atc, days)	25550	USEPA 1989 and CSMS 1996
Averaging Time - Threshold (Atn, days)	2190	USEPA 1989 and CSMS 1996

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium		8.0E-04	60%	3.2E-04	100%	2.0E-06	6.8E-11	8.0E-10	--		2.5E-06	0%
Thallium		8.0E-04		8.0E-04	100%	1.7E-05	5.8E-10	6.8E-09	--		8.5E-06	1%
Mercury (as inorganic and element		6.0E-04	40%	3.6E-04	100%	1.6E-05	5.5E-10	6.4E-09	--		1.8E-05	1%
Antimony		8.6E-04		8.6E-04	100%	5.1E-05	1.7E-09	2.0E-08	--		2.4E-05	1%
Arsenic		2.0E-03	50%	1.0E-03	100%	4.2E-05	1.4E-09	1.7E-08	--		1.7E-05	1%
Lead		3.5E-03	50%	1.8E-03	100%	1.2E-04	4.1E-09	4.8E-08	--		2.7E-05	2%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	5.5E-06	1.9E-10	2.2E-09	--		2.7E-06	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	9.8E-08	3.4E-12	3.9E-11	--		3.5E-08	0%
Copper		1.4E-01	60%	5.6E-02	100%	5.1E-04	1.7E-08	2.0E-07	--		3.6E-06	0%
Manganese		1.4E-01	50%	7.0E-02	100%	1.0E-04	3.5E-09	4.1E-08	--		5.8E-07	0%
Nickel		1.2E-02	60%	4.8E-03	100%	1.2E-05	4.1E-10	4.8E-09	--		9.9E-07	0%
Vanadium		2.0E-03		2.0E-03	100%	5.1E-06	1.7E-10	2.0E-09	--		1.0E-06	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	4.0E-09	1.4E-13	1.6E-12	--		1.5E-03	93%
PAHs (as BaP)	2.3E-01				100%	4.9E-08	1.7E-12	2.0E-11	3.9E-13	100%	--	
								TOTAL	**3.9E-13**		**1.6E-03**	

Calculation of Concentrations in Homegrown Beef

Uptake in to beef meat

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

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

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

Chemical-specific Inputs and calculations - maximum sensitive receptors

Chemical	Concentration in crops ingested by cattle mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to beef day/kg	Beef Concentration mg/kg ww	
Cadmium	8.8E-05	1.9E-02	2.0E-03	1.9E-05	
Thallium	4.4E-05	9.4E-03	4.0E-02	1.9E-04	RAIS
Mercury (as inorganic and eleme	8.8E-06	1.9E-03	4.0E-04	3.7E-07	
Antimony	1.3E-04	2.8E-02	1.0E-03	1.4E-05	RAIS
Arsenic	2.6E-04	5.7E-02	2.0E-03	5.6E-05	
Lead	1.3E-03	2.8E-01	3.0E-04	4.2E-05	
Chromium (Cr VI assumed)	2.6E-04	5.7E-02	5.5E-03	1.5E-04	RAIS
Cobalt	1.3E-05	2.8E-03	2.0E-02	2.8E-05	RAIS
Copper	1.3E-03	2.8E-01	1.0E-02	1.4E-03	RAIS
Manganese	2.6E-04	5.7E-02	4.0E-04	1.1E-05	RAIS
Nickel	2.6E-04	5.7E-02	3.0E-04	8.3E-06	
Vanadium	1.3E-05	2.8E-03	2.5E-03	3.5E-06	RAIS
Dioxins and furans	7.1E-10	3.1E-08	7.0E-01	1.4E-08	
PAHs (as BaP)	9.5E-05	4.9E-04	7.0E-02	7.5E-05	

Exposure to Chemicals via Ingestion of Beef

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

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

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium		8.0E-04	60%	3.2E-04	100%	1.9E-05	6.1E-09	1.5E-08	--		4.6E-05	0%
Thallium		8.0E-04		8.0E-04	100%	5.6E-05	1.8E-08	4.5E-08	--		5.6E-05	0%
Mercury (as inorganic and element		6.0E-04	40%	3.6E-04	100%	3.7E-07	1.2E-10	3.0E-10	--		8.2E-07	0%
Antimony		8.6E-04		8.6E-04	100%	1.4E-05	4.6E-09	1.1E-08	--		1.3E-05	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	5.6E-05	1.8E-08	4.5E-08	--		4.5E-05	0%
Lead		3.5E-03	50%	1.8E-03	100%	4.2E-05	1.4E-08	3.3E-08	--		1.9E-05	0%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	1.5E-04	5.1E-08	1.2E-07	--		1.5E-04	1%
Cobalt		1.4E-03	20%	1.1E-03	100%	2.8E-05	9.2E-09	2.2E-08	--		2.0E-05	0%
Copper		1.4E-01	60%	5.6E-02	100%	1.4E-03	4.6E-07	1.1E-06	--		2.0E-05	0%
Manganese		1.4E-01	50%	7.0E-02	100%	1.1E-05	3.7E-09	8.9E-09	--		1.3E-07	0%
Nickel		1.2E-02	60%	4.8E-03	100%	8.3E-06	2.8E-09	6.7E-09	--		1.4E-06	0%
Vanadium		2.0E-03		2.0E-03	100%	3.5E-06	1.2E-09	2.8E-09	--		1.4E-06	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.4E-08	4.7E-12	1.1E-11	--		1.1E-02	96%
PAHs (as BaP)	2.3E-01				100%	7.5E-05	2.5E-08	6.0E-08	5.8E-9	100%	--	
TOTAL									**5.8E-9**		**1.1E-02**	

Exposure to Chemicals via Ingestion of Beef

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

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

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Beef concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Cadmium		8.0E-04	60%	3.2E-04	100%	1.9E-05	3.2E-09	3.7E-08	--		1.1E-04	0%
Thallium		8.0E-04		8.0E-04	100%	5.6E-05	9.5E-09	1.1E-07	--		1.4E-04	0%
Mercury (as inorganic and element		6.0E-04	40%	3.6E-04	100%	3.7E-07	6.3E-11	7.4E-10	--		2.0E-06	0%
Antimony		8.6E-04		8.6E-04	100%	1.4E-05	2.4E-09	2.8E-08	--		3.2E-05	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	5.6E-05	9.5E-09	1.1E-07	--		1.1E-04	0%
Lead		3.5E-03	50%	1.8E-03	100%	4.2E-05	7.1E-09	8.3E-08	--		4.7E-05	0%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	1.5E-04	2.6E-08	3.0E-07	--		3.7E-04	1%
Cobalt		1.4E-03	20%	1.1E-03	100%	2.8E-05	4.7E-09	5.5E-08	--		4.9E-05	0%
Copper		1.4E-01	60%	5.6E-02	100%	1.4E-03	2.4E-07	2.8E-06	--		4.9E-05	0%
Manganese		1.4E-01	50%	7.0E-02	100%	1.1E-05	1.9E-09	2.2E-08	--		3.2E-07	0%
Nickel		1.2E-02	60%	4.8E-03	100%	8.3E-06	1.4E-09	1.7E-08	--		3.4E-06	0%
Vanadium		2.0E-03		2.0E-03	100%	3.5E-06	5.9E-10	6.9E-09	--		3.4E-06	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.4E-08	2.4E-12	2.8E-11	--		2.7E-02	96%
PAHs (as BaP)	2.3E-01				100%	7.5E-05	1.3E-08	1.5E-07	3.0E-9	100%	--	
								TOTAL	**3.0E-9**		**2.8E-02**	

Calculation of Concentrations in Dairy Milk

Uptake in to milk (dairy cows)

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

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

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

Chemical-specific Inputs and calculations - maximum sensitive receptors

Chemical	Concentration in crops ingested by cattle mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to milk day/kg	Milk Concentration mg/kg ww	
Cadmium	8.8E-05	1.9E-02	2.0E-03	4.5E-05	
Thallium	4.4E-05	9.4E-03	2.0E-03	2.3E-05	RAIS
Mercury (as inorganic and eleme	8.8E-06	1.9E-03	7.0E-05	1.6E-07	
Antimony	1.3E-04	2.8E-02	1.0E-04	3.4E-06	RAIS
Arsenic	2.6E-04	5.7E-02	5.0E-05	3.4E-06	
Lead	1.3E-03	2.8E-01	6.0E-05	2.0E-05	
Chromium (Cr VI assumed)	2.6E-04	5.7E-02	9.0E-06	6.1E-07	
Cobalt	1.3E-05	2.8E-03	2.0E-03	6.8E-06	RAIS
Copper	1.3E-03	2.8E-01	1.5E-03	5.1E-04	RAIS
Manganese	2.6E-04	5.7E-02	3.5E-04	2.4E-05	RAIS
Nickel	2.6E-04	5.7E-02	3.0E-05	2.0E-06	
Vanadium	1.3E-05	2.8E-03	2.0E-05	6.8E-08	RAIS
Dioxins and furans	7.1E-10	3.1E-08	2.0E-02	1.0E-09	
PAHs (as BaP)	9.5E-05	4.9E-04	1.0E-02	2.6E-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 from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/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	60%	3.2E-04	100%	4.5E-05	3.5E-07	8.4E-07	--		2.6E-03	12%
Thallium		8.0E-04		8.0E-04	100%	2.3E-05	1.7E-07	4.2E-07	--		5.2E-04	2%
Mercury (as inorganic and element		6.0E-04	40%	3.6E-04	100%	1.6E-07	1.2E-09	2.9E-09	--		8.2E-06	0%
Antimony		8.6E-04		8.6E-04	100%	3.4E-06	2.6E-08	6.3E-08	--		7.3E-05	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	3.4E-06	2.6E-08	6.3E-08	--		6.3E-05	0%
Lead		3.5E-03	50%	1.8E-03	100%	2.0E-05	1.6E-07	3.8E-07	--		2.2E-04	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	6.1E-07	4.7E-09	1.1E-08	--		1.4E-05	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	6.8E-06	5.2E-08	1.3E-07	--		1.1E-04	1%
Copper		1.4E-01	60%	5.6E-02	100%	5.1E-04	3.9E-06	9.4E-06	--		1.7E-04	1%
Manganese		1.4E-01	50%	7.0E-02	100%	2.4E-05	1.8E-07	4.4E-07	--		6.3E-06	0%
Nickel		1.2E-02	60%	4.8E-03	100%	2.0E-06	1.6E-08	3.8E-08	--		7.9E-06	0%
Vanadium		2.0E-03		2.0E-03	100%	6.8E-08	5.2E-10	1.3E-09	--		6.3E-07	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.0E-09	7.6E-12	1.8E-11	--		1.7E-02	82%
PAHs (as BaP)	2.3E-01				100%	2.6E-05	2.0E-07	4.9E-07	4.7E-8	100%	--	
								TOTAL	**4.7E-8**		**2.1E-02**	

Exposure to Chemicals via Ingestion of Milk

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

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

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Milk concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	Threshold TDI	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background)			NonThreshold	Threshold	Non-Threshold Risk	% Total Risk	Chronic Hazard Quotient	% Total HI
	$(mg/kg\text{-}day)^{-1}$	(mg/kg/day)		(mg/kg/day)		(mg/kg wet weight)	(mg/kg/day)	(mg/kg/day)	(unitless)		(unitless)	
Cadmium		8.0E-04	60%	3.2E-04	100%	4.5E-05	2.8E-07	3.3E-06	--		1.0E-02	12%
Thallium		8.0E-04		8.0E-04	100%	2.3E-05	1.4E-07	1.7E-06	--		2.1E-03	2%
Mercury (as inorganic and element		6.0E-04	40%	3.6E-04	100%	1.6E-07	9.9E-10	1.2E-08	--		3.2E-05	0%
Antimony		8.6E-04		8.6E-04	100%	3.4E-06	2.1E-08	2.5E-07	--		2.9E-04	0%
Arsenic		2.0E-03	50%	1.0E-03	100%	3.4E-06	2.1E-08	2.5E-07	--		2.5E-04	0%
Lead		3.5E-03	50%	1.8E-03	100%	2.0E-05	1.3E-07	1.5E-06	--		8.5E-04	1%
Chromium (Cr VI assumed)		9.0E-04	10%	8.1E-04	100%	6.1E-07	3.8E-09	4.5E-08	--		5.5E-05	0%
Cobalt		1.4E-03	20%	1.1E-03	100%	6.8E-06	4.3E-08	5.0E-07	--		4.4E-04	1%
Copper		1.4E-01	60%	5.6E-02	100%	5.1E-04	3.2E-06	3.7E-05	--		6.7E-04	1%
Manganese		1.4E-01	50%	7.0E-02	100%	2.4E-05	1.5E-07	1.7E-06	--		2.5E-05	0%
Nickel		1.2E-02	60%	4.8E-03	100%	2.0E-06	1.3E-08	1.5E-07	--		3.1E-05	0%
Vanadium		2.0E-03		2.0E-03	100%	6.8E-08	4.3E-10	5.0E-09	--		2.5E-06	0%
Dioxins and furans		2.3E-09	54%	1.1E-09	100%	1.0E-09	6.2E-12	7.3E-11	--		6.9E-02	82%
PAHs (as BaP)	2.3E-01				100%	2.6E-05	1.6E-07	1.9E-06	3.8E-8	100%	--	
TOTAL									**3.8E-8**		**8.4E-02**	