North West Treatment Hub Plant Upgrades: Human Health and Ecological Risk Assessment

Prepared for: Sydney Water

11 June 2024

Document History and Status

Report Reference	SW/24/R01
Revision	B – Final
Date	11 June 2024
Previous Revisions	A – Draft issued on 27 May 2024

Limitations

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

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

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

This report was prepared in May and June 2024 and is based on the information provided and reviewed at that time. Environmental Risk Sciences disclaims responsibility for any changes that may have occurred after this time.

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

Table of Contents

Appendices

Glossary of terms and abbreviations

Term	Definition
ABS	Australian Bureau of Statistics
Acute exposure	Contact with a substance that occurs once or for only a short time (up to 14 days)
Absorption	The process of taking in. For a person or an animal, absorption is the process of a substance getting into the body through the eyes, skin, stomach, intestines, or lungs
Adverse health effect	A change in body function or cell structure that might lead to disease or health problems
ATSDR	Agency for Toxic Substances and Disease Register
AAQ	Ambient air quality
ANZECC	Australia and New Zealand Environment and Conservation Council
Background level	An average or expected amount of a substance or material in a specific environment, or typical amounts of substances that occur naturally in an environment.
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
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	Environmental 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.
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.

Term	Definition
Guideline value	Guideline value is a concentration in soil, sediment, water, biota or air (established by relevant regulatory authorities such as the NSW Department of Environment and Conservation (DEC) or institutions such as the National Health and Medical Research Council (NHMRC), Australia and New Zealand Environment and Conservation Council (ANZECC) and World Health Organization (WHO)), that is used to identify conditions below which no adverse effects, nuisance or indirect health effects are expected. The derivation of a guideline value utilises relevant studies on animals or humans and relevant factors to account for inter and intra-species variations and uncertainty factors. Separate guidelines may be identified for protection of human health and the environment. Dependent on the source, guidelines would have different names, such as investigation level, trigger value and ambient guideline.
HI	Hazard Index
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].
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 hydrocarbons
PFAS	Per and poly-fluoroalkyl substances
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].
SO_2	Sulfur dioxide
TCEQ	Texas Commission on Environmental Quality
TEQ	Toxicity equivalent
Toxicity	The degree of danger posed by a substance to human, animal or plant life.
Toxicity data	Characterisation or quantitative value estimated (by recognised authorities) for each individual chemical substance for relevant exposure pathway (inhalation, oral or dermal), with special emphasis on dose-response characteristics. The data are based on based on available toxicity studies relevant to humans and/or animals and relevant safety factors.

Term	Definition
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
mg/m^3	Milligrams per cubic metre

Executive summary

Introduction

Environmental Risk Sciences Pty Ltd (enRiskS) has been engaged by Sydney Water to undertake a human health and environmental risk assessment (HHERA) in relation to the carbonisation plant and associated infrastructure at the Riverstone Wastewater Resource Recovery Facility (WRRF). This facility relates to upgrades associated with Sydney Water's North West Treatment Hub (NWTH).

This assessment has focused on impacts to human health and the environment from emissions to air derived from the carbonisation facility. The carbonisation facility would receive mixed sludge from the wastewater treatment plant, which is then dried and gasified. The product from this process is a solid product (Biochar). Off gasses from the process are treated prior to discharge to air.

Assessment Approach

The HHERA has been undertaken in accordance with Australian guidance on the assessment of environmental risks from enHealth (enHealth 2012a, 2012b), National Environment Protection Council, specifically relevant to the assessment of ambient air quality and contaminated land (NEPC 1999 amended 2013a, 2004, 2016), PFAS National Environmental Management Plan (the "PFAS NEMP") (HEPA 2020), Australian and New Zealand Water Quality Guidelines (ANZG 2018); and the NSW EPA Approved Methods for the Modelling and Assessment of Air Chemicals in New South Wales (NSW EPA 2016).

The HHERA has also drawn on international guidance where relevant.

The focus of the HHERA relates to the assessment of impacts to human health and the environment in relation to emissions to air as modelled by WSP (2024). The emissions evaluated include gases as well as other chemicals that may bound to particulates. The assessment has considered the following exposures:

- Human health
    - inhalation of all emissions by workers close to the site, recreational users of the area, and residents
    - deposition of metals, dioxins and furans, polycyclic aromatic hydrocarbons (PAHs) and per- and poly-fluoroalkyl substances to soil and:
        - ingestion and dermal contact with these chemicals in surface soil
        - ingestion of homegrown fruit and vegetables grown in soil where deposition occurs
        - ingestion of home produced eggs from chickens where livestock access soil where deposition occurs
    - deposition of metals, dioxins and furans, PAHs and PFAS to rainwater tanks used for non-potable purposes and waterbodies used for recreational use or stock water (pets and chickens).
- Ecological health

- direct toxicity effects related to air emissions of gases (particularly relevant to potential vegetation effects)
- deposition of metals dioxins and furans, PAHs and PFAS and
  - assessment of accumulation of these chemicals in soil and the potential for impacts on the terrestrial environment
  - assessment of the accumulation of these chemicals in surface water bodies and the potential for impacts on the aquatic environment.

Conclusions

Based on the available data, and with consideration of the uncertainties identified, the following can be concluded:

- there are no health risk issues of concern in relation to potential exposures by workers, recreational users of areas adjacent to the site
- there are no health risk issues of concern in relation to potential exposures by residents in the areas surrounding the site, including where residents consume homegrown fruit and vegetable and eggs
- there are no health risk issues of concern in relation to the health of pets or stock, such as chickens, where water from rainwater tanks is used
- there are no health risk issues of concern in relation to potential exposure to water in rainwater tanks by residents (noting reticulated potable water is available in the area)
- there are no health risk issues of concern in relation to potential recreational exposures in Eastern Creek (in relation to emissions to air from the facility)
- there are no risk issues of concern in relation to potential exposures by terrestrial and aquatic environments in areas adjacent to and surrounding the site.

Section 1. Introduction

1.1 Background

Sydney Water's North West Treatment Hub (NWTH) comprises of the Castle Hill Water Resource Recovery Facilities (WRRF), Rouse Hill WRRF and Riverstone Wastewater WRRF. The NWTH provides wastewater servicing to Sydney's northwest including the North West Growth Area (NWGA) and North West Urban Renewal Corridor along the new Metro North West Line.

In 2022, Sydney Water proposed the NWTH upgrades to address rapid growth, meet future regulatory requirements and provide a solution that minimises impacts to the community and the environment. The proposed works included:

- upgrading at Rouse Hill WRRF and Riverstone WRRF
- constructing a new sludge transfer system between the three WRRFs to centralise solid treatment at Riverstone.

The potential environmental impacts and mitigation measures for these works were assessed under the NWTH Upgrades and Sludge Transfer System – Growth Package, July 2022 (approved Review of Environmental Factors (REF)).

Following this, Sydney Water identified an opportunity to diversify methods for solids processing. A review of technology available for advanced processing of biosolids to reduce contaminants of concern found that carbonisation with upstream digestion, dewatering and drying was the preferred technology for the NWTH upgrade project.

The proposed changes to the approved REF include the following:

- Riverstone Wastewater Resource Recovery Facility (WRRF)
    - a new carbonisation plant and associated infrastructure including drying, heating and carbonisation systems, this will result in production of biochar rather than biosolids
    - no expansion of existing anaerobic digestion and no upgrade to waste gas burners
    - deletion of cogeneration unit.
- Rouse Hill WRRF
    - new dewatering and outloading building to cater for sludge treatment
    - expansion of the construction footprint to include a compound site in 7 Money Close, Rouse Hill (5/-/DP1158760) and new access roads into the facility
    - ongoing use of part of existing biological nutrient removal (BNR) treatment and existing aerobic digester.
- Sludge transfer systems
    - deletion of both sludge transfer pipelines (Rouse Hill WRRF to Riverstone WRRF, and Castle Hill WRRF to Rouse Hill WRRF).

Environmental Risk Sciences Pty Ltd (enRiskS) has been engaged by Sydney Water to undertake a human health and environmental risk assessment (HHERA) in relation to the carbonisation plant and associated infrastructure at the Riverstone Wastewater Resource Recovery Facility (WRRF).

1.2 Project description

This assessment specifically relates to proposed changes at the Riverstone WRRF, specifically the new carbonisation plant and associated infrastructure. This process involves the following:

- primary sludge is digested in the existing digesters (upgraded for improved performance)
- undigested waste activated sludge (WAS) and primary sludge are blended in the sludge mixing tank. Achieving consistent mixing of the two sludge types is critical to the success of the drying and gasification processes
- the mixed sludge is then dewatered using the dewatering centrifuges
- biogas generated from the digesters will continue to be used to heat the digesters via the existing compressors and gas heaters. The excess gas will now be utilised to provide supplementary heat to the dryer and gasification heat loop
- the dewatered sludge is dried and transferred to the gasifiers
- dried sludge is then gasified. This process involves:
    - dried sludge enters the gasification chamber where it is heated to between 600 – 700 ºC with limited oxygen supply
    - the organic content in the sludge is then gasified with syngas being transferred to a thermal oxidisation chamber via gas filtration. This burns the off gas generating heat
    - the solid product (Biochar) is discharged
- off gases are treated from each step of the process prior to discharge via their relevant stacks:
    - biological scrubbing and carbon for dewatered sludge gases (through the existing site odour control system)
    - chemical scrubbing for sludge dryer gases
    - chemical scrubbing and activated carbon for flue gas from the gasifier.

The final product is biochar. During some phases of operation, a dewatered sludge or dried sludge product may be extracted depending on the overall operational needs of the system.

The issues in relation to the assessment of impacts to human health and the environment in the area surrounding the process are emissions to air. Hence this assessment has specifically been undertaken to address impacts on the surrounding community as a result of emissions to air.

1.3 Objectives

The overall objective of the HHERA is to undertake an assessment of risks to human health and the environment in relation to emissions to air derived from the operation of the proposed gasification plant at the Riverstone WRRF.

More specifically the HHERA has addressed the following:

- assessment of emissions to air from the proposed facility based on outputs from the Air Quality Impact Assessment (AQIA), specifically in relation to ground level concentrations and deposition rates (relevant to particulate bound pollutants including metals and PFAS)
- assessment of potential risks to the health of the community surrounding the Riverstone WRRF. The assessment will address inhalation exposures as well as other relevant exposure pathways following deposition of pollutants, including contact with soil and dust,

uptake into and ingestion of homegrown produce such as fruit and vegetables, eggs and milk (for bioaccumulative chemicals) and impacts on the quality of water in rainwater tanks
- assessment of the risk to soil, water, vegetation and fauna relevant to the environment surrounding the Riverstone WRRF
- Where elevated risks are identified, emission limits to protect human health or the environment relevant to the proposed facility for specific compounds have been derived.

The assessment has only addressed risks to human health and the environment relevant to emissions to air from the proposed facility, as assessed and modelled in the AQIA. The HHERA has not addressed any other aspects of the NWTH.

In addition, the HHERA has not addressed impacts to workers within the proposed facility as these aspects would be managed under the *Work Health and Safety Act 2011* and *Work Health and Safety Regulation 2017* and all other relevant codes of practice as detailed by Work Safe NSW and Safe Work Australia.

1.4 Methodology

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

- enHealth (2012) Environmental Health Risk Assessment, Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a)
- enHealth (2012) Australian Exposure Factor Guide (enHealth 2012b)
- ASC NEPM (1999 amended 2013) National Environmental Protection Measure – Assessment of Site Contamination (NEPC 1999 amended 2013b)
- NEPM (Ambient Air Quality) (2021) (NEPC 2021)
- NEPM (Air Toxics) (2004) (NEPC 2004)
- NSW EPA (2022) Approved Methods for the Modelling and Assessment of Air Pollutants in New South Wales (NSW EPA 2022)
- Australian Drinking Water Guidelines (NHMRC 2011 updated 2022)
- Australia and New Zealand Fresh and Marine Water Quality Guidelines (ANZG 2018)
- PFAS National Environmental Management Plan (the “PFAS NEMP”), 2020 (HEPA 2020).

Additional guidance has been sought from international sources such as the USEPA or WHO, where necessary and where it is not inconsistent with Australian regulatory or policy setting.

1.5 Available information

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

- WSP 2024, Riverstone WRRF, Air Quality Impact Assessment. North West Treatment Hub Plant Upgrades – Growth Package. Draft report dated May 2024.

Section 2. Surrounding environment

2.1 Introduction

This section provides an overview of the environment surrounding the Riverstone WRRF, relevant to the assessment of potential risks to human health and the environment. This includes a summary of the community and the location of environmental receptors in the area surrounding the site.

2.2 Site location and surrounding environment

The Riverstone WRRF is located at 108 Bandon Road in Vineyard, New South Wales, which is approximately 40 km north-west of Sydney city centre (Figure 2.1). The site is approximately 230 m west of Riverstone Parade, approximately 240 m south-west of Vineyard Station, and approximately 2.8 km NW and 5 km south-east of the Riverstone and Windsor town centres, respectively.

Riverstone WRRF is located east of Eastern Creek which receives treated wastewater from the plant via a discharge point on the eastern bank of the creek. The creek is considered to be a key fish habitat. A range of aquatic vegetation (macrophytes) have been observed in and adjacent to Eastern Creek as part of works undertaken for the preparation of the Aquatic Impact Assessment for the NMTH[1]. Some smaller farm dams, and ephemeral creek/drainage lines have also been identified in land located between Eastern Creek and Riverstone WRRF. Other ephemeral creek lines and other smaller dams also located further west associated with South Creek.

Vegetation in the area surrounding the Riverstone WRRF includes rural/semi-rural agricultural land, with some areas of more dense vegetation. The residential areas located to the east of the Riverstone WRRF, and east of Riverstone Parade comprise larger semi-rural lots.

Windsor downs nature reserve is located further west of the Riverstone WRRF.

[1] Eco Logical Australia 2022, North West Treatment Hub Aquatic Impact Assessment. Report dated 28 June 2022.

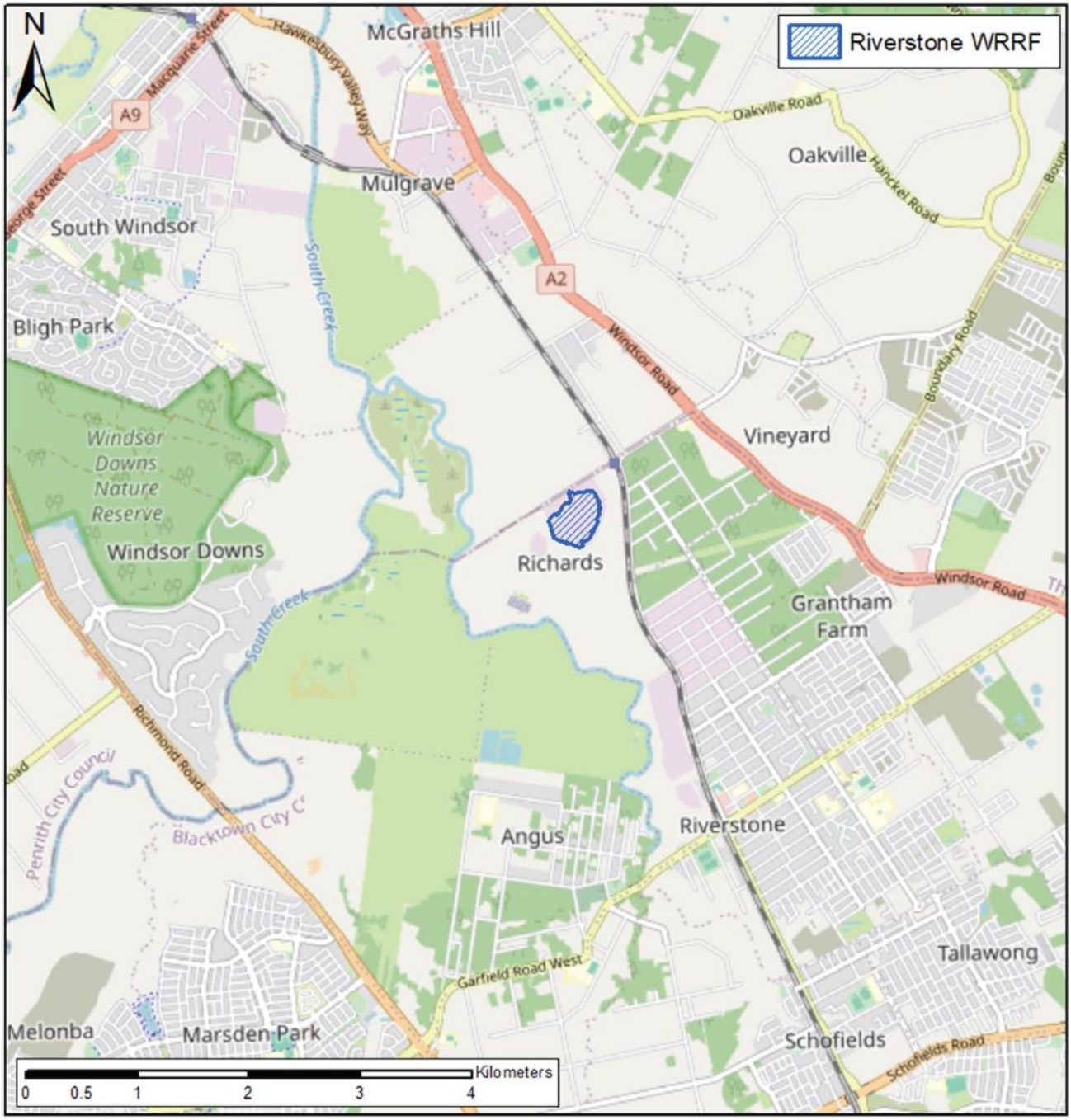

Figure 2.1: Location of Riverstone WRRF

For the assessment of potential impacts in the community surrounding the proposed facility, a number of individual receptors have been identified and evaluated in the AQIA. These are locations representative of where individuals in the community may live, or attend daycare/school, work or conduct recreational activities. A number of these locations are also relevant to assessing potential impacts on the environment. The assessment of impacts has focused on locations closest to the Riverstone WRRF, as shown on **Figure 2.2** and listed in **Table 2.1**. Exposures at locations further away from the Riverstone WRRF would be lower.

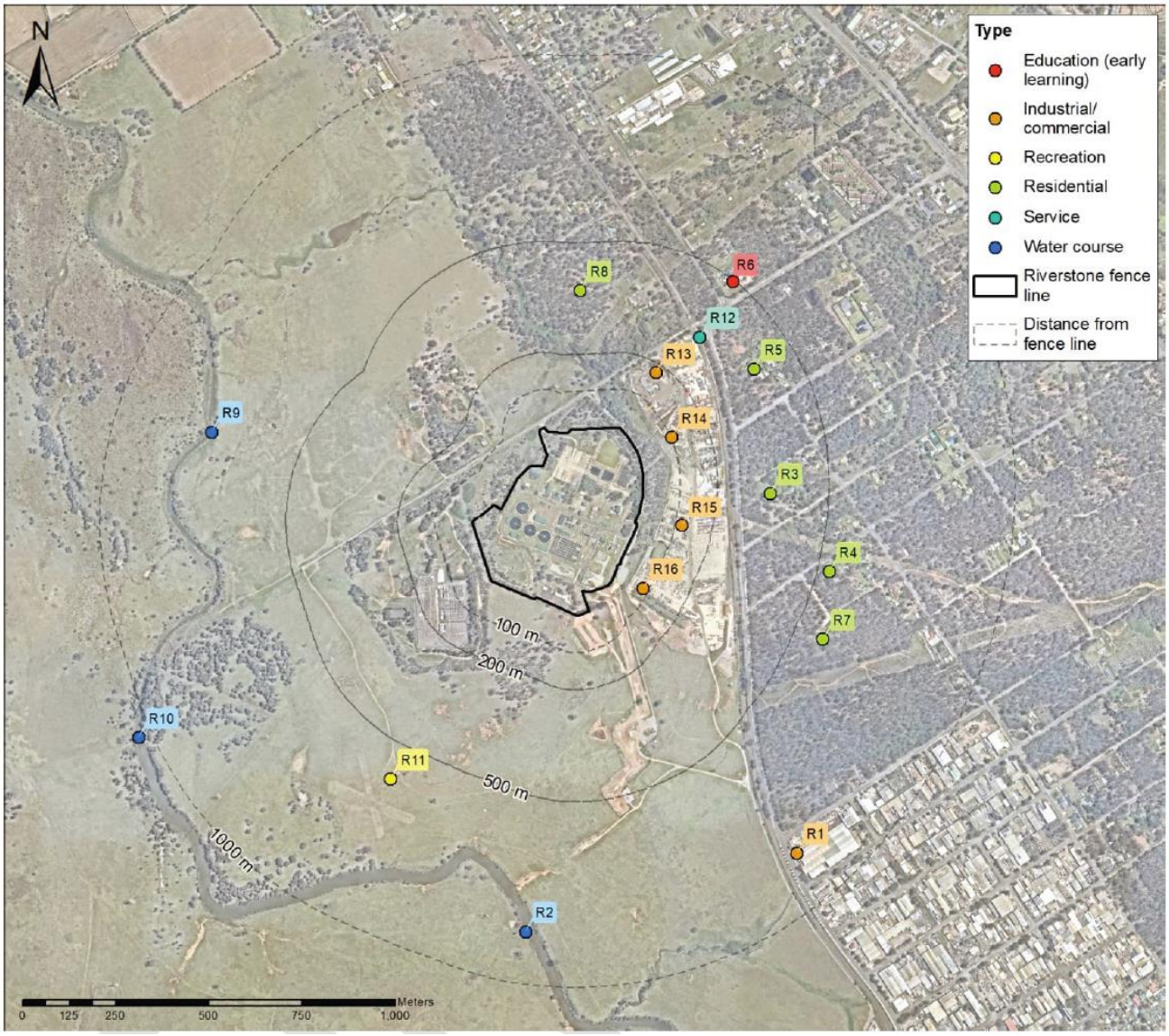

Figure 2.2: Location of individual receptor locations in the vicinity of Riverstone WRRF, as evaluated in the AQIA

Table 2.1: Individual receptor locations evaluated in AQIA (as shown on Figure 2.2)

Receptor number	Name	Type
R1	Victoria St Industry	Commercial/industrial (workplace)
R2	Eastern Creek 3	Waterway (Eastern Creek)
R3	Ashford Rd	Residential
R4	Otago St	Residential
R5	Camberwell Rd	Residential
R6	Vineyard Early Learning	Education (early learning)
R7	Brisbane Rd	Residential
R8	House North	Residential
R9	Eastern Creek 1	Waterway (Eastern Creek)
R10	Eastern Creek 2	Waterway (Eastern Creek)
R11	Hawkesbury Model Air Sports	Recreational area
R12	Vineyard Train Station	Public transport
R13	Western Storage 1	Commercial/industrial (workplace)
R14	Western Storage 2	Commercial/industrial (workplace)
R15	Western Storage 3	Commercial/industrial (workplace)
R16	Western Storage 4	Commercial/industrial (workplace)

Note: shading in the table reflects the land use as indicated in the table and shown on **Figure 2.2**

In addition to these individual receptor locations, the AQIA also modelled potential impacts at the following locations:

- grid centred on the proposed facility as follows (totalling 7165 locations):
  - 500 m from facility at 20 m resolution
  - 1000 m from the facility at 30 m spacing
  - 1500 m from the facility at 100 m spacing
  - 3000 m from the facility at 250 m spacing
  - 4000 m from the facility at 500 m spacing.

The Riverstone WRRF and surrounding areas evaluated in the AQIA are located on the boundary of the Hawkesbury and Blacktown Local Government Areas. In terms of statistical areas, the area is located in the Suburbs and Localities (SALs) of Richards, Vineyard, Riverstone and Grantham Farm; and the SA2 statistical areas of Riverstone and Pitt Town – McGraths Hill.

2.3 Demographics

Table 2.2 presents a summary of the population demographics for the SALs relevant to the community evaluated in this assessment. The data presented in **Table 2.2** also includes the Hawkesbury LGA information – as this LGA is considered more representative of the population in the area surrounding the site (which is semi-rural residential/recreational and commercial industrial) compared with Blacktown LGA that includes much larger and more densely populated areas.

These data are based on data available from the 2021 Census and 2021 Socio-Economic data from the Australian Bureau of Statistics (ABS). The data presented also addresses aspects relating to cultural and linguistic diversity (CALD) within the population. People born in some countries have higher rates of disease and health factors that may make them more vulnerable (NSW Health 2019). It is noted that migrant populations are often healthier than native-borne populations, with many having lower level of premature mortality and self-reported chronic conditions compared to those born in Australia (AIHW 2018).

Table 2.2 also provides some review of the demographics data to indicate where the population may be more or less vulnerable. The vulnerability of the population is considered to potentially reflect the ability of the population to adapt to environmental change and stressors. Communities with higher rates of unemployment, ranked more socioeconomically disadvantaged, with higher rates of young children or the elderly are considered to be potentially more vulnerable to the environmental stressors considered in this assessment.

Table 2.2: Summary of populations surrounding the proposed project site

Indicator	Suburb or Statistical Area					NSW	Australia
	Richards	Vineyard	Riverstone	Grantham Farm	Hawkesbury LGA		
Total population	37	1143	8627	3669	67207	8,072,163	25,422,788
Population 0 - 4 years	0%	3.2%	9.5%	13.4%	6.2%	5.8%	5.8%
Population 5 - 19 years	7.5%	17.5%	20.4%	18.5%	19.4%	18.1%	18.2%
Population 20 - 64 years	80%	54.1%	60.8%	64.4%	57.8%	58.5%	58.8%
Population 65 years and over	12.5%	25.2%	9.3%	3.8%	16.6%	17.6%	17.2%
Median age	52	50	32	30	39	39	38
Household size	1.6	2.4	2.9	3.2	2.8	2.6	2.5
Unemployment	3.5 %				3.6%	3.3%	3.7%
Tertiary education	0%	16.3%	19.8%	21.3%	21.1%	23.8%	23.3%
SEIFA IRSD	929	951	1001	1078	1026	--	--
SEIFA rank	2	2	5	9	8	--	--
Indigenous	16%	4.5%	4.9%	1.5%	4.8%	3.4%	3.2%
Born overseas	8%	29.4%	34.1%	46.1%	17.7%	29.3%	27.7%
Top 4 countries of birth	NA	England China Malta New Zealand	India Philippines Nepal New Zealand	India Philippines Nepal China	England New Zealand India Malta	China England India New Zealand	England India China New Zealand

Data from ABS 2021 Census and 2021 Socio-Economic Data
Unemployment rates for December quarter 2023, relevant to LGA and Statistical Area 2 region (SA2) of Pitt Town-McGraths Hill (no data available for Riverstone) as defined by the ABS, https://www.jobsandskills.gov.au/data/small-area-labour-markets
SEIFA IRSD = index of socioeconomic disadvantage, rank in deciles in Australia that ranges from:
1 = most disadvantaged to 10 = least disadvantaged
Shading relates to comparison against NSW (potential): more vulnerable; less vulnerable
Country of birth – where in blue there is the potential for higher levels of diabetes or complications from diabetes (relevant for England and Philippines) or higher rate of coronary heart disease and heart failure (relevant to Malta) (NSW Health 2019)

Based on the population data available and presented in **Table 2.2**, the community in the area surrounding the proposed facility comprise small populations where there is significant variability in the data. The population closest to the facility (Richards and Vineyard SAL) has a lower percentage of young children and older adults (for Richards SAL only), however this area also has a higher average age, when compared with Hawkesbury LGA and NSW. The population closest to the facility is considered more disadvantaged, with the larger population of the Hawkesbury LGA considered less disadvantaged than average in NSW.

The population data does not indicate any consistent indication that the community close to the proposed facility may be more vulnerable to project related impacts.

2.4 Existing community health

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 2.3**) generally reflect a wide range of these factors.

The population in the area surrounding the site is small and health data specifically relating to this population are not available.

The site is located on the boundary of the Western Sydney Local Health District (LHD) and the Nepean Blue Mountains LHD. Health statistics for both these LHDs have been included in this assessment, noting that these statistics provide only a general indication of the range of values that may be representative of the population in the area of the proposed facility.

Table 2.3 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 NSW.

Table 2.3: Summary of health indicators/data

Health indicator/data[1]	Western Sydney LHD	Nepean Blue Mountains LHD	NSW
Health behaviours (95% confidence interval)			
Adults - compliance with fruit consumption guidelines (2022)	32.1% (28.0% - 36.2%)	35.7% (30.6% - 40.8%)	37.7% (36.36% – 39%)
Adults - compliance with vegetable consumption guidelines (2022)	2.7% (1.1% - 4.3%)	5.2% (2.7% - 7.6%)	4.4% (3.9% – 4.9%)
Children - compliance with fruit consumption guidelines (2021-2022)	56.2% (47.3% - 65.1%)	56.9% (47.5% - 66.2%)	59.6% (56.9% - 62.3%)
Children - compliance with vegetable consumption guidelines (2021-2022)	2.1% (0.4% - 3.8%)	2.9% (0.0% - 5.7%)	5.9% (4.7% - 7.1%)
Adults - increased lifetime risk of alcohol related harm (2022)	23.5% (19.8% - 27.3%)	38.9% (33.6% - 44.2%)	32.4% (31.2% - 33.7%)
Adults - body weight (overweight) (2022)	31.0% (27.2% - 34.8%)	36.4% (31.3% - 41.6%)	33.8% (32.6% - 35.1%)
Adults - body weight (obese) (2022)	23.6% (19.7% - 27.4%)	31.5% (26.8% - 36.3%)	24.2% (23% - 25.3%)
Adults – insufficient physical activity (2022)	45.8% (41.3% - 50.3%)	41.6% (36.3% - 46.8%)	39.4% (38 – 40.8%)
Children – adequate physical activity (2019-2020)	16.5% (9.2% - 23.8%)	26.4% (16.7% - 36.1%)	20.5% (18.1% - 23%)
Current smoker, adult (2022)	12.6% (10% - 15.2%)	11% (7.9% - 14.2%)	11.4% (10.6% - 12.2%)
Burden of disease (95% confidence interval) as rate per 100,000 unless indicated otherwise			
Morbidity - cardiovascular disease hospitalisations (all ages, 2022/2023)	1425 (1402.2 – 1448)	1537.1 (1501.1 – 1573.8)	1522.7 (1515.1 – 1530.3)
Morbidity – respiratory disease hospitalisations (all ages, 2022/2023)	1486.3 (1463.1 – 1509.6)	1637 (1597.6 – 1677.2)	1505.6 (1497.3 – 1513.9)
Mortality – all causes, all ages (2021)	490.7 (476.9 – 504.8)	561.4 (539.4 – 584)	496.2 (492 – 500.4)
Mortality – all causes, all ages (2022)[2]	Hawkesbury LGA = 520		520
Mortality – respiratory (all ages) (2019 - 2021)	41 (38.6 – 43.4)	48.8 (45.1 – 52.8)	41.5 (40.8 – 42.2)
Adults – prevalence of high blood pressure (2021/2022)	26.8 (23.8 – 30.2)	15.8 (12.3 – 20.1)	26.4% (25.4% - 27.4%)
Adult asthma – prevalence (2019)	11.7% (8.7% - 14.8%)	18.9% (12.7% - 25.2%)	11.5% (10.5% – 12.5%)
Children (2 to15 years) – prevalence of current asthma (2017 – 2019)	10.4% (6.8% - 14.1%)	18.2% (12.2% - 24.3%)	13.1% (11.8% - 14.4%)

* Rate per 100,000 population.

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

2 Data from the Australian Bureau of Statistics (Deaths, Australia 2022 – released September 2023)

Shading relates to comparison against NSW:

statistic/data suggestive of a potential higher vulnerability within the population to health stressors.

statistic/data suggestive of a potential lower vulnerability within the population to health stressors.

The key indicators of health for the population in areas surrounding the site indicate the following, when compared with the data for NSW:

- The population in Western Sydney LHD, considered more relevant to the study area, is generally similar to NSW in terms of health-related behaviours, with the exception of a lower proportion of the adult population who consume the recommended intake of fruit, and a lower proportion of children who consume the recommended intake of vegetables and a higher proportion of adults with insufficient physical activity. In relation to health statistics this population is generally similar to NSW with a lower rate of cardiovascular hospitalisations.
- Mortality data for the Hawkesbury LGA is consistent with data for the NSW population and does not indicate an increased vulnerability.
- The population in Nepean Blue Mountains LHD is different to that of Western Sydney LHD and has a number of indicators that suggest increased vulnerability in relation to health-related behaviours. In addition, the health statistics indicate higher levels of respiratory disease hospitalisations, mortality and asthma. This LHD is very large and covers a large area that incorporates populations that may not be representative of the population in the study area. As the population in the study area is very small it is not possible to determine if the statistics from the Nepean Blue Mountains LHD influence the underlying health statistics for this area.

It is expected that, where the data for Western Sydney LHD is considered more representative, the population in the study area may not have any increased vulnerability to project related impacts. There is some uncertainty as to the influence of statistics from the Nepean Blue Mountains LHD population on the characteristics of the population in the study area.

No data are available for the smaller population in the areas immediately surrounding the project.

Section 3. Conceptual site model

3.1 Approach

This section presents a review of the conceptual site model relevant to the assessment of potential impacts associated with emissions to air from the operation proposed changes to the NWTH, specifically emissions to air from the carbonisation process. This includes a summary of the emission sources, modelling of air emissions, the nature of the chemicals being evaluated, and the potential for exposure to occur for a range of human and environmental receptors.

3.2 Emission sources

The approved REF included a range of emission sources relevant to the operation of the Riverstone facility. These emission sources mainly relate to odour emissions from wastewater treatment, and include the thickening building fan (TBFAN), dewatering building fan (DBFAN), odour control unit (OCU), biological reactor (BIOR) and three secondary clarifiers (SC1, SC2 and SC3). These emission sources remain unchanged from the approved REF and have been included in the assessment of air emissions (where relevant).

The new sources of emissions relate to the carbonisation process and include the following:

- carbonisation exhaust stacks (two stacks, GAS1 and GAS2)
- gas heaters (three heaters GH1, GH2 and GH3)
- the scrubber stack serving the two driers (DRIER).

The carbonisation process includes emissions control technology to minimise emissions to air from the site.

Figure 3.1 shows the location of the above emission sources on the site. The carbonisation process emission sources are all point source emissions where pollutants are released to air via a stack. The characteristics of the stacks relevant to each source are detailed in the AQIA (WSP 2024).

Figure 3.1: Riverstone WRRF layout, and emission source locations (from WSP 2024)

In relation to emissions to air, the AQIA (WSP 2024) has assessed a range of pollutants relevant to the carbonisation process. These pollutants include:

- nitrogen dioxide (NO_2)
- sulfur dioxide (SO_2)
- particulates as total suspended particulates (TSP) PM_{10} and $PM_{2.5}$
- carbon monoxide (CO)
- metals (bound to particulates) that include:
    - lead
    - arsenic
    - chromium VI
    - nickel
    - selenium
    - zinc
    - copper
    - cadmium
    - mercury
- organics (predominantly bound to particulates):
    - polycyclic aromatic hydrocarbons (PAHs)
    - dioxins and furans
    - per- and poly-fluoroalkyl substances (PFAS)
- other gases

- hydrogen fluoride (HF)
- hydrogen chloride (HCl)
- hydrogen sulfide (H_2S)
- ammonia
- volatile organic compounds (VOCs)

Emissions from the carbonisation process have been determined on the basis of:

- emission limits established in the *Protection of the Environment Operations Act* (*POEO Act*) for afterburners and other thermal treatment plant equipment, excluding flares
- emissions limits and data from an example carbonisation facility in Loganholme Queensland (including consideration of information from potential equipment suppliers)
- metals composition of existing Riverstone WRRF sludge based on sampling conducted in November 2023.

3.3 General concepts relevant to 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 3.2 and 3.3 illustrate the processes which govern how the emissions get mixed into the atmosphere.

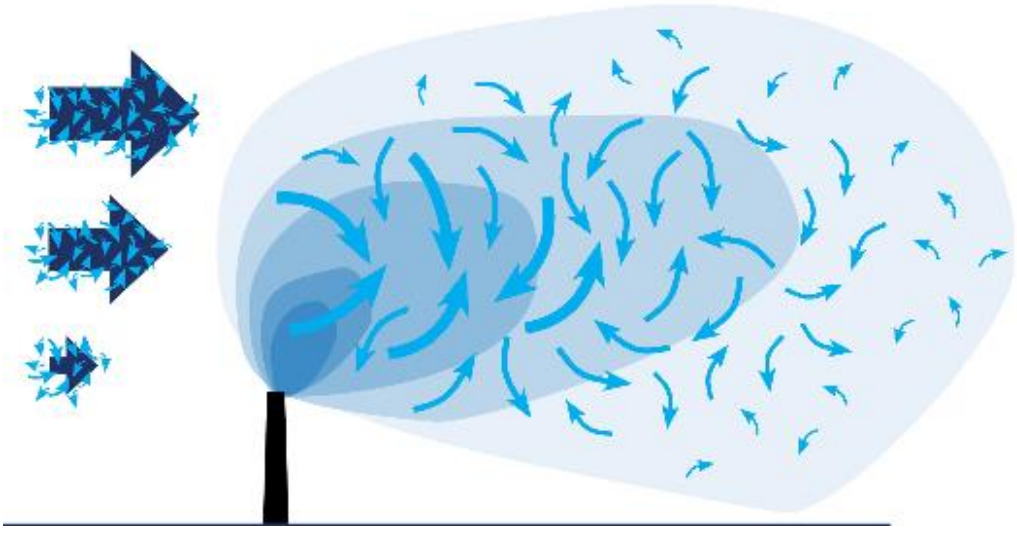

Figure 3.2: Turbulence in the air, how it mixes and dilutes pollutants emitted from a stack (NSW Chief Scientist 2018)

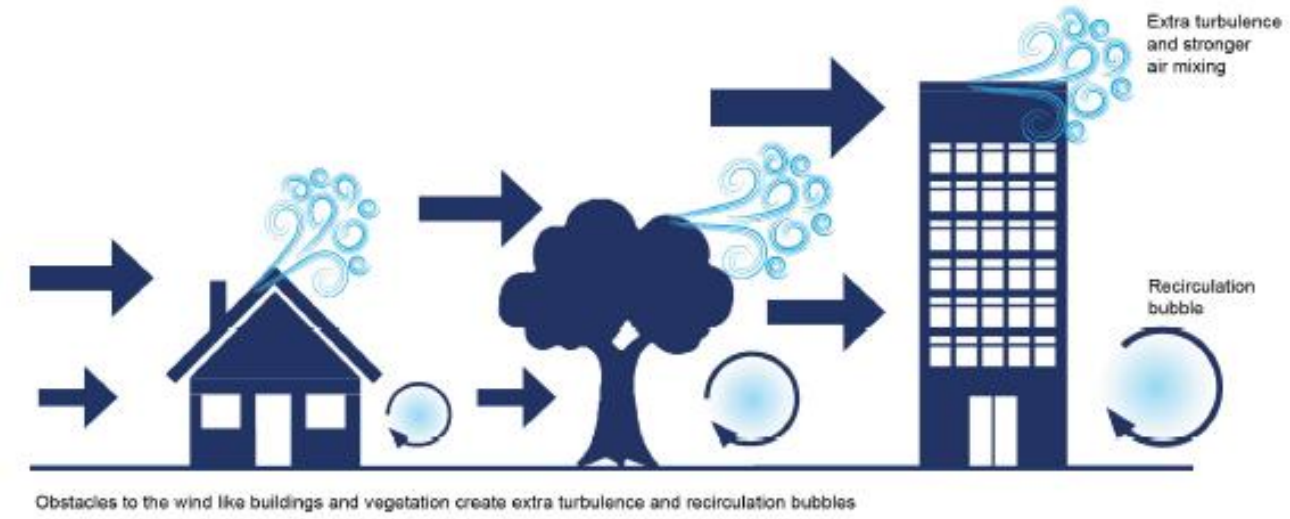

Figure 3.3: Turbulence in the air and how it is affected by buildings and vegetation (NSW Chief Scientist 2018)

Gases and particles such as PM_{10} and $PM_{2.5}$ are emitted at around 90 ^{o}C from the carboniser units, 50 ^{o}C from the scrubber and 170 ^{o}C from the gas heaters and are pushed out of the relevant stacks using fans (i.e. at some speed) so these gases and particles rise or are pushed up significant distances above the top of the stack – because hot gases rise and because these 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 particles cool and slow down they begin to interact with the wind above the stack (i.e. well above the stack heights). This mixes the gases and particles into the atmosphere decreasing the actual concentration present in any one particular place.

Figure 3.2 shows that most of the pollutants remain up in the atmosphere away from where people would 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 or water around the facility.

3.4 Overview of air modelling

To predict the concentration of emissions from the site, a study area was defined and shown in **Section 2.2** and predicted emissions from the relevant processes (stack emissions), along with all other emission sources relevant to the WRRF were modelled by WSP (2024) using the CALPUFF air dispersion model.

The CALPUFF air dispersion model is a regulatory air pollution model that was selected based on the need to evaluate complex terrain and heterogeneous land use (relevant to the area evaluated). This model uses air emission estimates for each of the sources evaluated, plant design (for example, stack height and building sizes), local terrain and meteorological data to predict the ground level concentrations of emissions within the defined study area. Meteorological data from Rouse Hill (consistent with the previous modelling for the approved REF) has been used in the model.

Background air quality is influenced by existing sources at or adjacent to the site. Background air quality has been assessed by WSP (2024) on the basis of data reported at the Rouse Hill Air Quality Monitoring Station. Maximum concentrations for the key criteria pollutants monitored at the station between 2019 and 2023, excluding data from the bushfire season in 2019/2020, was adopted as background levels relevant to the project area.

The modelling of air emissions was based on the available information on emissions relevant to the proposed process as summarised in Section 3.1 and in the AQIA (WSP 2024).

Full details on the emission rates adopted for these scenarios and the air model are presented in the AQIA (WSP 2024). This model is used to provide predicted air concentrations, and deposition rates, over the study area (as a grid including boundary locations) and at all the individual receptor locations (as detailed in **Section 2.2** and **Figure 2.2**), with the results averaged over different time periods. This data has been provided in spreadsheets from the air modelling for use in the HHRA.

3.5 Potential for exposure

Understanding how a community member or the environment may come into contact with pollutants released in air emissions from the proposed energy recovery 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 and/or the environment may come in contact with these pollutants.

There are five main ways a community member or the environment may be exposed to a chemical substance emitted from the facility:

- direct contact by vegetation with vapours and gases (including respiration by vegetation)
- inhalation of gases, vapour or fine particulate matter in air
- direct contact, which may include ingestion and/or dermal absorption of chemicals present in dust that may deposit onto surfaces or accumulate in water collected in rainwater tanks or water in recreational areas/aquatic environments
- accumulation into animals
- accumulation into produce that may be consumed. For the semi-rural area evaluated the produce considered includes home-grown fruit and vegetables and eggs.

For some of the emissions from the Project, inhalation is considered the only route of exposure. This is due to the substance's chemical properties, which make the other pathways inconsequential. This includes gases such as nitrogen dioxide (NO_2), sulfur dioxide (SO_2), carbon monoxide (CO), hydrogen chloride (HCl), hydrogen sulfide (H_2S), ammonia (NH_3), hydrogen fluoride (HF) and VOCs (assessed assuming it is all present as benzene) as well as fine particulate matter as particulates less than 2.5 micrometres ($PM_{2.5}$) that are so small they remain suspended in air (i.e. inhalation only exposure pathway).

Other chemicals emitted to air may be inhaled, but also may be deposited on the ground, where they may contribute to soil concentrations, or water bodies where they may contribute to water concentrations (including dam and rainwater collected in tanks). These chemicals can then be ingested directly through incidental ingestion of soil or water. Where the chemicals bioaccumulate exposure may occur indirectly through consumption of food grown or raised in the soil (fruit, vegetables, eggs, milk and meat) or consumed by higher order species. These pathways are of particular relevance to metals, dioxins and furans, PAHs and PFAS. Absorption through dermal contact with soil and water is also possible.

Table 3.1 lists the substances considered in the air emissions from the facility and the exposure pathway/s of potential concern for human health and the environment.

Table 3.1: Substances and routes of exposure

Substance	Route of exposure	
	Human health	**Environmental**
Nitrogen dioxide (NO_2)	**Inhalation only** as these are vapours/gases	**Direct contact /inhalation/ respiration with gases –** terrestrial environments only
Sulfur dioxide (SO_2)		
Hydrogen sulfide		
Hydrogen chloride		
Hydrogen fluoride		
Carbon monoxide		
VOCs (assumed to comprise 100% benzene)		
$PM_{2.5}$	**Inhalation only** as these particulates are very small and will remain suspended in air. It is noted that other exposure pathways have also been assessed for the individual chemical substances bound to these particles. These other pathways relate to the individual chemical substances, rather than the physical size of the particulates.	**NA**
Metals (as individual metals listed in **Section 3.2**)	**Inhalation** of these chemicals adhered to dust/particulates **Ingestion and dermal contact** with these chemicals deposited to soil and present in water (rainwater tanks or recreational water) **Ingestion** of produce grown in soil potentially impacted by these chemicals (where the chemicals can be taken up/bioaccumulated into plants and produce). For the surrounding semi-rural properties, home consumption of produce such as fruit and vegetables and eggs have been assessed.	**Direct contact and bioaccumulation for terrestrial environments –** with chemicals that are deposited to soil **Direct contact and bioaccumulation for aquatic environments –** with chemicals that are deposited to waterways
PAHs (assumed to comprise 100% benzo(a)pyrene, BaP)		
PFAS (assumed to comprise 100% PFOS)		
Dioxins / furans		

In relation to assessing exposures to these chemicals the following has been considered:

- For the assessment of potential exposure to chromium, the data provided relates to total chromium. In the environment, chromium is mainly present as chromium III (CrIII), however it can also be present as chromium VI (CrVI). Chromium VI is significantly more toxic form of chromium and as the proportion of CrVI of total chromium in the sludge proposed to be treated in the carbonisation process, or the emissions from the facility, is not known, it has been conservatively assumed that all emissions of chromium are as CrVI. Published data (Spanos et al. 2016) suggests that for sewage sludge, the chromium species that is most dominant is CrIII, with CrVI comprising up to 3.5% of total chromium. This illustrates how conservative it is to assume total chromium is 100% CrVI.
- There are a number of groups of chemicals evaluated. An assessment of risks to human health or the environment needs to consider information on the hazards or toxicity of the chemical. For groups of chemicals the toxicity of the individual chemicals will vary and the overall toxicity of the group of chemicals will then depend on which chemicals are present, the toxicity of these chemicals and the concentrations present (i.e. proportion present in the group). This information is not known for the three groups of chemicals included in this

assessment. To address this lack of information, a conservative approach is adopted where it has been assumed that the most toxic individual chemical that may be present in the group makes up 100% of the concentration for the whole group of chemicals. For this assessment the following has been assumed:

- Volatile organic compounds (VOCs) can comprise a large number of individual volatile chemicals. For this assessment 100% of the VOCs have been assumed to comprise benzene, which is expected to be the most toxic VOC chemical that may be present in emissions from the facility.
- PAHs comprise a large number of individual chemicals, noting that analysis often only focuses on a group of 16 key PAH compounds. These compounds include chemicals that are non-carcinogenic, and a small number that are carcinogenic (and genotoxic). The toxicity of the group of PAHs is dominated by the carcinogenic PAHs. The most toxic carcinogenic PAH is benzo(a)pyrene. The toxicity of other carcinogenic PAHs can be assessed on the basis of a toxicity equivalent or ratio of toxicity compared with BaP. The composition of these PAHs in emissions to air from the facility is not known, hence it has been conservatively assumed that the emissions comprise 100% BaP.
- PFAS comprise thousands of individual compounds, with the key individual PFAS commonly present from sources such as the use of fire fighting foams, being PFOS, PFHxS and PFOA (FSANZ 2017a, 2017b). Of these PFAS compounds PFOS is the most toxic in terms of human health and the environment. Some data is available on the presence of PFAS in wastewater treatment plants in Australia (Coggan et al. 2019). This data shows that PFOS is detected in most samples of wastewater and sludge, with the proportion of PFOS in the total PFAS detected less than 50%. Assuming total PFAS always comprises 100% PFOS is therefore conservative.

- It is assumed that the carbonisation process operates continuously, where inhalation exposures may occur, and chemicals may deposit to and accumulate in soil over 35 years.
- Following deposition to soil, exposures relating to direct contact and consumption of produce are assumed to occur for every year while living at the property as the chemicals remain, once deposited. For the chemicals assessed for accumulation in soil and water, and uptake into produce, degradation in the environment is not considered to be significant.
- When considering deposition to and accumulation in rainwater tanks and water bodies this has been assumed to occur over a 1 year period as an average noting that these water bodies would be flushed during periods of rain, and water in tanks used throughout the year and replenished with rainfall.

3.6 Use of air modelling data for the assessment of impacts to health and the environment

The air dispersion modelling has predicted ground level concentrations on the basis of different averaging times. Data has been provided for use in this assessment for the maximum 1 hour average, 24 hour average, and annual average for all receptors and across the modelling grid.

In addition, the assessment of impacts requires consideration of the deposition of metals, PAHs, PFAS, and dioxins/furans. Concentrations of these pollutants are assumed to be adhered to particulates, where the modelled air concentrations are assumed to relate to the total concentration in air (as TSP).

This assessment has considered impacts at the following locations, for the assessment of various exposures:

- **Maximum impacted location anywhere offsite** within the study area regardless of location and land use – this is a location on the site boundary. Impacts at this location have been assessed in relation to acute and chronic inhalation exposures (as workers may be present in this area) and direct toxicity effects to vegetation and deposition to and potential impacts on terrestrial environments.
- **Maximum impacted sensitive receptor**. This is the maximum impacted receptor from the individual sensitive receptors shown on **Figure 2.2**. These exposures more specifically relate to the closest residential areas and areas where the public may access for recreational purposes. Exposures in these areas are assumed to be consistent with rural residential where inhalation exposures may occur, along with deposition and exposure to impacts in rainwater tanks, surface soil and uptake into homegrown produce (fruit and vegetables and eggs). Exposures are assumed to occur for 24 hours per day, every day at this location.
- **Maximum impacted water body**. This relates to the closest water bodies to the boundary of the site, where water may be used for stock watering, or an aquatic environment may be present and of importance. The off-site areas comprise a range of environments, included rural residential areas. For the purpose of this assessment impacts at the closest receptor are assumed to also occur at the closest aquatic environment – namely Eastern Creek. Assessment of potential impacts to the off-site terrestrial environments has been undertaken on the basis of the maximum impacted location anywhere (noted above – which is conservative for the assessment of terrestrial environments further away from the site boundary).

Section 4. Human health impacts

4.1 General

This section presents a detailed assessment of potential risks to human health as a result of emissions to air from the project. The assessment of risk has relied on air modelling presented in the AQIA (WSP 2024), and evaluated the pathways of exposure relevant to the community surrounding the site (as detailed in **Section 3**) and follows the principles outlined in the enHealth document Environmental Health Risk Assessment: Guidelines for Assessing Human Health Risks from Environmental Hazards (enHealth 2012a).

The assessment has addressed inhalation exposures (**Section 4.2**) as well as multi-pathway exposures relevant to persistent and bioaccumulative chemicals (**Section 4.3**).

4.2 Inhalation exposures

4.2.1 General

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

It is noted that compliance with the guidelines adopted for assessing acute and chronic inhalation exposures, are also protective of buildings and structures.

4.2.2 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 (WSP 2024).

In relation to the assessment of human health, 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, 2019; WHO 2013a). These health effects were considered in the derivation of the NEPM air guideline for $PM_{2.5}$ (NEPC 2021).

The NEPM 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 detailed by WSP (2024)).

Table 4.1 provides a summary of the contribution of the project to the total $PM_{2.5}$ concentrations, and the NEPM air criteria.

Table 4.1: $PM_{2.5}$ impacts from the project – maximum from all receptors (regardless of landuse)

Parameter	$PM_{2.5}$ – as 24-hour average ($\mu g/m^3$)	$PM_{2.5}$ – as annual average ($\mu g/m^3$)
Guideline (NEPC 2021)	25 (20 as goal for 2025)	8 (7 as goal for 2025)
Background (existing)	40.5	6.7
Contribution from project	2.6	0.35
% contribution of project to NEPM	10%	4%
Total (project + background)	43.1	7.0

Table 4.1 shows that the worst-case $PM_{2.5}$ derived from the facility at any of the receptors evaluated makes a small contribution to existing concentrations. Based on the modelling undertaken there would be no exceedance of the NEPM standard for $PM_{2.5}$ as an annual average. However, exceedances have been identified for the 24-hour average as a result of elevated background concentrations. WSP (2024) conducted a more detailed (Level 2), contemporaneous assessment of $PM_{2.5}$ impacts that showed that there are no additional exceedances of the $PM_{2.5}$ standard as a result of the project. Hence changes in $PM_{2.5}$ associated with the project would not be expected to be of significance to compliance with the NEPM standard or community health.

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 Hawkesbury Local Government Area (LGA, all ages and all causes) available for 2022 (refer to **Table 2.3**) 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 residential location where the maximum increase in $PM_{2.5}$ from the facility is relevant for long-term exposures, and it is assumed that exposure occurs 24 hours a day, every day of the year.

For a maximum annual average increase at the residential (sensitive) receptor locations of $PM_{2.5}$ of 0.153 $\mu g/m^3$, this results in a maximum individual risk of 5×10^{-6}. This risk level is considered to be low and acceptable based on criteria established by the NEPC (NEPC 2011).

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

4.2.3 Sulfur dioxide

Sulfur oxides are formed during combustion when chemicals present in fuels (such as coal, gas, petrol etc) and which containing sulfur react with oxygen to form sulfur oxides. Burning of coal in power stations in Europe resulted in acid rain affecting forests. The acid rain was primarily a result of the formation of sulfur oxides as the coal was burnt. Sulfur oxides are also released from volcanos. Wildfires and other types of fires are also sources to the atmosphere of these chemicals (USEPA 2018).

Sulfur dioxide (SO_2) is the main sulfur oxide that can have impacts on people. Exposure to elevated levels can result in irritation of the respiratory system and can make breathing difficult. The most affected by exposure to these chemicals are people with asthma (USEPA 2018).

Guidelines are available from NSW EPA (NSW EPA 2022) and NEPC (NEPC 2021) which indicate concentrations of SO_2 considered to be acceptable by national health authorities. These are the guidelines adopted by WSP (2024).

These guidelines focus on the protection of health effects of short-term exposures for SO_2, which are the key health effects relevant to this pollutant. The NEPM standard is considered to be protective of adverse effects for all members of the population including sensitive populations like asthmatics, children and the elderly.

Table 4.2 presents a comparison of modelled SO_2 levels and the relevant NEPM guidelines for the project and for the cumulative case which includes the project + background.

Table 4.2: SO_2 impacts from the project - maximum from all receptors (regardless of landuse)

Parameter	SO_2 (µg/m^3)	
	1-hour average	24-hour average
Guideline (NEPM 2021)	286 (100 ppb)	57 (20 ppb)
Background	74	18
Contribution from project	17	9.7
% contribution of project to NEPM	8%	17%
Cumulative case (project + background)	91	27

Table 4.2 shows that all cumulative concentrations of SO_2 well are below the NEPM criteria. On this basis there are no risks to community health in relation to SO_2 emissions from the project.

4.2.4 Nitrogen dioxide

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

In Sydney, the NSW Government estimated that for calendar year 2013 on-road vehicles accounted for about 53% of emissions of nitrogen oxides, industrial facilities accounted for 12%, other mobile sources accounted for about 26%, with the remainder from domestic/ commercial or natural sources (Ewald et al. 2020; NSW EPA 2019).

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

Guidelines are available from NEPC (NEPC 2021) which indicate concentrations of nitrogen dioxide considered to be acceptable by national health authorities.

These guidelines are based on protection from adverse health effects following both short term (acute) and longer term (chronic) exposure for all members of the population including sensitive populations like asthmatics, children and the elderly.

Table 4.3 presents a comparison of the maximum modelled NO_2 concentrations (anywhere) and the relevant NEPM guidelines for the project and for the cumulative case which includes the project + background.

Table 4.3: NO_2 impacts from the project - maximum from all receptors (regardless of landuse)

Parameter	NO_2 (µg/m^3)	
	1-hour average	Annual average
Guideline (NEPM 2021)	164 (0.08 ppm)	31 (0.015 ppm)
Background	74	10
Contribution from project	73	12
% contribution of project to NEPM	45%	40%
Cumulative case (project + background)	143	22

Table 4.3 shows that emissions of NO_2 from the project have the potential to contribute to the short-term (maximum 1-hour average) and long-term average (annual average) NEPM standards. However, all cumulative concentrations of NO_2 are below the NEPM standards.

On this basis there are no risks to community health in relation to NO_2 emissions from the project.

4.2.5 Carbon monoxide

Motor vehicles are the dominant source of carbon monoxide in air (DECCW 2009). Carbon monoxide is produced during combustion when there is a limited supply of oxygen.

The sorts of effects that can be expected due to exposure to CO are those linked with carboxyhaemoglobin (COHb) in blood – i.e. where CO replaces oxygen in the blood preventing oxygen from being transported around the body. In addition, association between exposure to carbon monoxide and cardiovascular hospital admissions and mortality, especially in the elderly for cardiac failure, myocardial infarction and ischemic heart disease; and some birth outcomes (such as low birth weights) have been identified (NEPC 2010).

Guidelines are available from NSW EPA (NSW EPA 2022) and NEPC (NEPC 2021) which indicate concentrations of carbon monoxide considered to be acceptable by national health authorities. These guidelines are considered protective of health for all members of the population.

Table 4.4 presents a comparison of the maximum modelled CO concentrations (anywhere) and the relevant NEPM guidelines for the project and for the cumulative case which includes the project + background.

Table 4.4: CO impacts from the project - maximum from all receptors (regardless of landuse)

Parameter	CO (µg/m^3)	
	1-hour average	8-hour average
Guideline (NEPM 2021)	NA	10,000
Guideline (NSW EPA 2022)	30,000	10,000
Background	1872	1875
Contribution from project	28	19
% contribution of project to guideline	<1%	<1%
Cumulative case (project + background)	1940	1910

Table 4.4 shows that emissions of CO from the project are a very small contribution to the guidelines adopted from the NEPM and NSW EPA. All concentrations from the project and cumulative concentrations of CO below the relevant health protective criteria. On this basis there are no risks to community health in relation to CO emissions from the project.

4.2.6 All other chemicals

4.2.6.1 General

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

4.2.6.2 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:

$$HI = \frac{\text{Exposure concentration (maximum 1-hour average)}}{\text{(Acute health based guideline)}}$$

Where:

Exposure concentration = calculated from the concentration in air derived from the air modelling (mg/m^3) – note that for the assessment of pollutants bound to particulates (e.g. metals) the exposure concentration = maximum 1 hour average air concentration x 0.375, which is the proportion of dust in air that is respirable and is small enough to reach and be retained in the lungs as per enHealth and NEPM (enHealth 2012a; NEPC 1999 amended 2013c)

Acute health based guideline = health based guideline that is protective of short-duration exposures, as per **Appendix B** (mg/m^3)

Risks associated with acute exposures are considered to be acceptable where the individual and total HI's are less than or equal to 1.

For this assessment, the maximum predicted 1-hour average concentration at any location has been considered. This has been done to address acute inhalation exposures that may occur in any area. The calculations presented are conservative for all other locations.

The acute health based guidelines adopted in this assessment have been adopted on the basis of the approach detailed in **Appendix B**. It is noted that for exposures to dioxins and furans, PAHs and

PFAS, as well as some metals, there are no health based guidelines available as the hazards/health impacts for these chemicals relates to chronic exposures or long-term body burdens.

Table 4.5 presents a summary of the relevant acute health-based guideline, the predicted maximum 1-hour average concentration anywhere offsite and the maximum impacted receptor, and the calculated HI for each chemical.

Table 4.5: Review of acute inhalation exposures and risks

Chemicals	Acute air guideline (1-hour average) (mg/m^3)	Maximum 1-hour average concentration (mg/m^3)		Calculated HI	
		Off-site	Receptors	Off-site	Receptors
Hydrogen chloride (HCl)	0.66 [1]	0.0019	0.00055	0.0029	0.0008
Hydrogen fluoride (HF)	0.06 [1]	8.0×10^{-5}	8.0×10^{-5}	0.0013	0.0013
Ammonia	0.59 [1]	0.0046	0.0021	0.0078	0.0036
Hydrogen sulfide (H_2S)	0.5 [3]	0.0058	0.0027	0.012	0.0054
Arsenic	0.0099 [1]	1.4×10^{-6}	4.1×10^{-7}	0.000054	0.000015
Cadmium	0.00055 [1]	2.0×10^{-5}	5.5×10^{-6}	0.013	0.0038
Chromium (Cr VI assumed)	0.0013 [1]	2.1×10^{-5}	6.0×10^{-6}	0.0061	0.0017
Copper	0.1 [2]	1.1×10^{-5}	4.7×10^{-6}	0.000042	0.000017
Nickel	0.0011 [1]	1.8×10^{-5}	5.1×10^{-6}	0.0061	0.0017
Selenium	0.025 [4]	6.1×10^{-6}	1.8×10^{-6}	0.000091	0.000027
Mercury (as elemental)	0.0006 [2]	1.9×10^{-5}	5.5×10^{-6}	0.012	0.0034
VOCs (as benzene)	0.17 [1]	0.023	0.011	0.14	0.062
			Total HI	**0.2**	**0.08**
			Acceptable HI	**≤ 1**	**≤ 1**

Notes:

Shaded HI = calculation of HI incorporates factor of 0.375 which is the proportion of dust in air that is respirable and is small enough to reach and be retained in the lungs as per enHealth and NEPM (enHealth 2012a; NEPC 1999 amended 2013c)

References for health-based acute air guidelines (1-hour average):
1 = Guideline available from the Texas Commission on Environmental Quality (TCEQ), https://www.tceq.texas.gov/toxicology/dsd/final.html
2 = 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
3 = Guideline available from the WA Department of Health (WA Department of Health 2009), converted from 24-hour average to 1 hour average using an averaging time conversion factor of 2.5.
4 = Guideline available from the Ontario Ministry of the Environment and Climate Change https://www.ontario.ca/page/ontarios-ambient-air-quality-criteria-sorted-contaminant-name converted from 24-hour average to 1 hour average using an averaging time conversion factor of 2.5

Table 4.5 indicates all maximum predicted concentrations of chemicals in air are below the health-based criteria protective of acute effects. For each of the individual chemicals evaluated the calculated HI is well below 1 indicating there is a significant margin of safety.

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

4.2.6.3 Chronic exposures

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

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

$$\text{Exposure concentration} = C_a \times \frac{ET \times FI \times LRF \times EF \times ED}{AT}$$

Where:
Exposure concentration = calculated based on the concentration in air and parameters relevant to characterising exposure (mg/m^3), based on the following:

- Ca = concentration in air at the point of exposure as an annual average (mg/m^3)
- ET = exposure time (hours/day)
- FI = fraction inhaled derived from source (unitless)
- LRF = lung retention factor (unitless – relevant to the inhalation of pollutants bound to dust/particulates)
- EF = exposure frequency (days/year)
- ED = years exposed (years)
- AT = averaging time relevant to the assessment of threshold and non-threshold effects (hours)

Health based criteria or TC = health-based threshold 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**)

Risks associated with chronic exposure to chemicals assessed on the basis of a threshold 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 exposures to benzene and PAHs (assumed to comprise 100% benzo(a)pyrene), this requires the calculation of an incremental lifetime cancer risk, as these chemicals are genotoxic carcinogens. This is a different calculation that only considers the incremental risk associated with exposures to benzene derived from the facility (i.e. no consideration of background). The calculation of risk is as follows:

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

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

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 considered to be acceptable (NEPC 1999 amended 2013b).

For this assessment, inhalation exposures have been evaluated on the basis of the maximum concentration predicted in the offsite industrial, residential and childcare receptors, and recreational receptors where the following exposures are considered relevant:

- Maximum concentration: commercial/industrial – this is a maximum that occurs at the commercial/industrial receptors located offsite. For workers at these locations, inhalation exposures are assumed to occur 8 hours per day for 240 days of the year, for 30 years, consistent with guidance from enHealth and NEPC (enHealth 2012a; NEPC 1999 amended 2013c). These exposure assumptions are conservative, and protective, of exposures by visitors and recreational users in areas close to the site.
- Maximum concentrations at residential or childcare receptors – this maximum has been assessed for residential exposures, which is 24 hours per day for 365 days of the year for 35 years consistent with guidance from enHealth and NEPC (enHealth 2012a; NEPC 1999 amended 2013c). These exposure assumptions are conservative, and protective, of exposures by exposures in a childcare setting and for visitors.
- Maximum concentrations at recreational receptors – this maximum relates to locations where people may spend time undertaking recreational activities, assumed to comprise 4 hours per day, 2 days per week for 35 years.

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, recreational and worker inhalation exposures.

Table 4.6 presents the calculated individual HI and the incremental lifetime cancer risk relevant to the assessment of chronic inhalation exposures, and the total risk (assuming additivity of effects).

Table 4.6: Calculated chronic inhalation risks

Chemical	Calculated Incremental Lifetime Risk			Calculated HI		
	Maximum C/I workers	Maximum recreational	Maximum residents	Maximum C/I workers	Maximum recreational	Maximum residents
Hydrogen chloride (HCl)	--	--	--	0.00038	0.000014	0.00062
Hydrogen fluoride (HF)	--	--	--	0.000034	1.2×10^{-6}	0.000056
Ammonia	--	--	--	0.00013	3.3×10^{-6}	0.00026
Hydrogen sulfide (H_2S)	--	--	--	0.0030	0.000077	0.0058
Arsenic	--	--	--	0.000044	1.6×10^{-6}	0.000078
Cadmium	--	--	--	0.010	0.00070	0.033
Chromium (Cr VI assumed)	--	--	--	0.00050	0.000032	0.0015
Copper	--	--	--	0.000000081	4.2×10^{-9}	0.00000024
Lead	--	--	--	0.000051	2.2×10^{-6}	0.00010
Nickel	--	--	--	0.0021	0.00014	0.0066
Selenium	--	--	--	0.00000077	5.0×10^{-8}	0.0000024
Mercury (as inorganic and elemental)	--	--	--	0.00024	0.000015	0.00069
Zinc	--	--	--	0.000000068	3.8×10^{-9}	0.00000024
VOCs (as 100% benzene)	5.0×10^{-7}	1.6×10^{-8}	1.2×10^{-6}	0.0077	0.00020	0.015
Total PFAS (as 100% PFOS)	--	--	--	0.000014	3.6×10^{-7}	0.000027
Dioxins and furans (WHO_{05} TEQ)	--	--	--	0.000011	3.7×10^{-7}	0.000018
PAHs (as 100% BaP)	9.5×10^{-11}	2.4×10^{-11}	1.1×10^{-9}	--	--	--
Total risk and HI	**5×10^{-7}**	**2×10^{-8}**	**1×10^{-6}**	**0.02**	**0.001**	**0.06**
Acceptable risk and HI	**≤1×10^{-5}**	**≤1×10^{-5}**	**≤1×10^{-5}**	**≤1**	**≤1**	**≤1**

Table 4.6 indicates the following:

- All calculated non-threshold risks associated with incremental lifetime risks associated with exposure to VOCs (assuming this is 100% benzene) and PAHs (assuming this is 100% BaP) are below the adopted criteria representative of acceptable risks ($1x10^{-5}$) and also equal to or below the criteria representative of negligible risks ($1x10^{-6}$). The calculated non-threshold risk is considered to be a highly conservative calculation due to the assumptions adopted in relation to the composition of VOCs and PAHs, where the most toxic compound has been assumed to comprise 100% of these chemical groups. Actual risks would be lower than presented in the table.
- The calculated HI for all individual chemicals significantly lower than 1, and the HI for exposure to all chemicals evaluated (assuming additivity of risk) is well below 1. This calculation assumes some conservative assumptions for some chemicals including VOCs (assumes to only comprise benzene, the most toxic VOC likely to be present), chromium (assumed to only be present as Cr VI, the most toxic form) and PFAS (assumed to only comprise PFOS, the most toxic PFAS compound).

It is noted that the margin of safety (MOS) relevant to inhalation exposures ranges from 16 to 1000 for the total HI, and 10 to 500 for the non-threshold risk, with the MOS significantly higher than this for many individual chemicals. This margin of safety, along with the conservative assumptions

adopted for the toxicity of chemical groups such as VOCs, PAHs and PFAS, is more than sufficient to address any likely changes in guidelines that may be applicable to these chemicals over time.

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

4.3 Multiple pathway exposures

4.3.1 General

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

- Deposition to water (refer to **Section 4.4**):
  - Eastern Creek where water may be used for recreational purposes, where ingestion and dermal contact may occur
  - rainwater tanks, where water may be used as non-potable water where ingestion and dermal contact may occur. This also includes irrigation of homegrown crops and stock (chickens).
- Deposition to soil:
  - incidental ingestion and dermal contact with soil (and dust indoors that is derived from outdoor soil or deposited particulates)
  - ingestion of homegrown fruit and vegetables where chemicals may deposit onto the plants and is also present in the soil where the plants are grown, and where chemicals are taken up into these plants
  - ingestion of eggs where chemicals may deposit onto pasture and be present in soil (which the soil present where backyard chickens are kept and ingested during feeding), and the chemicals are taken up into the eggs.

The above exposures are chronic or long-term exposures.

4.3.2 Assessment approach

In relation to exposures related to the deposition of emissions to soil, such exposures will only occur at the semi-rural residential properties where people live and where some homegrown produce is present. Risks associated with multiple pathway exposures have been calculated on the basis of the maximum predicted impacts relevant to waterways and residential properties/receptors. Risks will be lower for all other sensitive receptors.

The calculation of risks posed by multiple pathway exposures only relates to chemicals that are persistent and bioaccumulative. For these chemicals it is assumed that the chemicals deposited during the operation of the facility continue to accumulate and remain in soil for the duration of occupancy at the property. The calculations undertaken has utilised a deposition rate, which is derived from the air modelling as detailed in **Section 3.4**.

Appendix C 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 D**.

For the chemicals considered in this assessment, the risk calculations undertaken relate to a non-threshold risk (relevant to exposure to PAHs) and a threshold HI. As discussed in **Section 4.2.3**, the following criteria have been adopted for determining when risks are considered to be negligible or acceptable.

- **Non-threshold risk**: individual and total non-threshold risk summed over all relevant exposure pathways and chemicals ≤ $1x10^{-5}$ = negligible/acceptable risk to human health
- **HI:** the individual and total HI, where calculated as the sum over all relevant exposure pathways and chemicals ≤ 1 = negligible/acceptable risk to human health.

4.3.3 Calculated risks

Table 4.7 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 at the offsite residential properties in the surrounding community and provides a conservative estimation of risks relevant to other residential and recreational areas. The table presents the total HI for each exposure pathway, calculated as the sum over all the chemicals evaluated. The table also includes the calculated risks associated with inhalation exposures, as these exposures are additive to the other exposure pathways for residential properties.

Table 4.7: Summary of risks for multiple pathway exposures (maximum residential receptor)

Exposure pathway	Non threshold risk		HI	
	Adults	Young children	Adults	Young children
Individual exposure pathways				
Inhalation (I)	1.2×10^{-6}	1.2×10^{-6}	0.064	0.064
Soil ingestion (SI)	1.9×10^{-11}	3.6×10^{-11}	0.00038	0.0064
Soil dermal contact (SD)	7.0×10^{-11}	2.9×10^{-11}	0.000064	0.00015
Ingestion of homegrown fruit and vegetables (F&V)	5.7×10^{-11}	4.4×10^{-11}	0.0029	0.012
Ingestion of homegrown eggs (E)	1.3×10^{-14}	5.4×10^{-15}	0.00016	0.00035
Multiple pathways (i.e. combined exposure pathways)				
I + SI + SD	1.2×10^{-6}	1.2×10^{-6}	0.064	0.071
I + SI + SD + F&V	1.2×10^{-6}	1.2×10^{-6}	0.067	0.082
I + SI + SD + E	1.2×10^{-6}	1.2×10^{-6}	0.065	0.071
I + SI + SD + F&V + E	1.2×10^{-6}	1.2×10^{-6}	0.068	0.082
Acceptable risk/HI	**≤1 x 10^{-5}**		**≤1**	

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

Table 4.7 indicates that all calculated risks associated with each individual exposure pathway as well as a combination of multiple exposure pathways, remain well below the target risk levels considered representative of low and acceptable risk. The calculations presented are dominated by inhalation exposures and are largely unchanged with consideration of multi-pathway exposures.

In relation to the contribution of individual chemical exposures to the total HI (where most of the chemicals are assessed), **Figure 4.1** shows the contribution of each pathway (inhalation, soil ingestion and dermal contact, ingestion of homegrown fruit and vegetables and eggs) to the total HI for each chemical assessed in relation to exposures by adults and children.

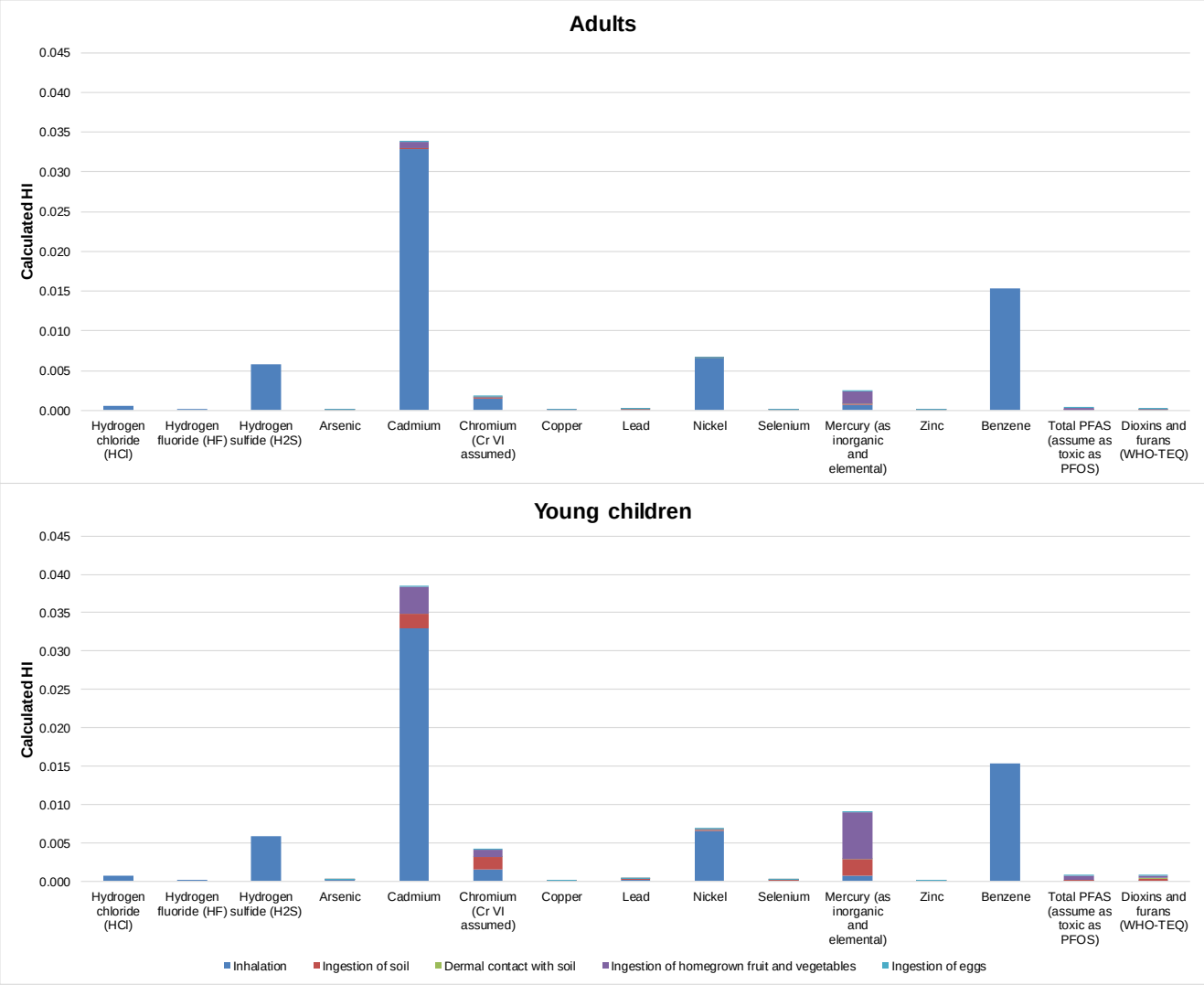

Figure 4.1: Calculated HI for each exposure pathway for each chemical for residential exposures to emissions from proposed carbonisation facility (adults and young children)

Figure 4.1 shows that the calculated HI is dominated by intakes of cadmium and VOCs as 100% benzene, mercury, nickel and hydrogen sulfide. The calculated HI for VOCs is highly conservative as it assumes this comprises 100% benzene, which would not be the case.

The margin of safety (MOS) relevant to the calculated multi-pathway exposures are similar to the MOS for inhalation exposures and are around 10 to 15 for the maximum impacted residential receptor. This, along with the conservative assumptions adopted, is more than sufficient to address any future changes in guidelines that may occur.

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 semi-rural residential areas surrounding the proposed facility.

4.4 Residential and recreational exposures to water

Where there may be deposition of chemicals that are persistent to water bodies located in areas surrounding the site, there is the potential for these chemicals to accumulate and impact on water quality in these areas, and these areas may be accessed and used for recreational purposes by the community.

A conservative approach has been undertaken where a potential worst-case concentration in water has been calculated. The area surrounding the site comprises open space recreational areas with semi-rural residential properties. The waterways in the surrounding area primary comprise Eastern Creek, with some smaller dams located close to the creek. To be conservative it has been assumed that a water body/dam in the waterway of Eastern Creek (i.e. no flow in the creek) or close to these waterways.

The calculation undertaken assumes that a water body is located at the most impacted waterway location and this water body is a standard pond size of 1 Ha (10,000 m^2) and 0.15 m deep (EPHC 2009), and chemicals may deposit to and mix within this water body over a full year, after which time it is assumed the that water would have flushed through as a result of rainfall (or running water in the creek). The maximum total (i.e. particulates plus dissolved phase) water concentration in the water body (which would be present at the end of the year of deposition) has then been directly compared with recreational water quality guidelines. The predicted concentrations have also been compared against stock water guidelines and drinking water guidelines. The drinking water guidelines are screening level guidelines protective of all uses, including irrigation. Where no stock water guidelines are available, drinking water guidelines have been adopted.

In addition to impacts on water quality in the surrounding waterway, semi-rural properties surrounding the site also include rainwater tanks. The deposition of chemicals to a roof, and accumulation in rainwater has been estimated for the maximum impacted receptor location, assuming the average rainfall for Seven Hills and Richmond RAAF (from the Bureau of Meteorology), a roof that is consistent with a 4 bedroom Australian home and the use of a first-flush device (noting that outcomes do not change if this devise is not included). Using this approach concentrations of chemicals in the water as suspended sediment and dissolved has been calculated. Rainwater tanks are designed such that suspended sediment deposits or settles and is not consumed. For the purpose of this assessment, it is assumed that both suspended sediment and dissolve phase concentrations may be present in the water used for non-potable purposes. It is noted that reticulated water is supplied to the surrounding community and hence rainwater would not be expected to be used for any potable purpose.

Predicted concentrations in in rainwater tanks have then been compared with drinking water guidelines, which are protective of all exposures relevant to non-potable, and potable, water use including ingestion, dermal contact, bathing and irrigation of homegrown crops that may be consumed. These guidelines are also protective of the health of livestock and pets.

Table 4.8 presents the maximum predicted concentrations in a water body that may be present in the surrounding environment, and in rainwater tanks with comparison against drinking water, recreational water and stock water guidelines. It is noted that most recreational water guidelines adopted are 10 times higher than drinking water guidelines, consistent with guidance provided by

NHMRC (NHMRC 2008) in relation to recreational exposures. The exception is PFAS where NHMRC has derived guidelines specific to recreational exposures (NHMRC 2019).

Appendices C and D present the calculated water concentrations.

Table 4.8: Summary and review of exposures to chemicals in water

Persistent and bioacumulative chemical	Calculated concentration in water – maximum total (mg/L)		Adopted water guideline (mg/L)		
	Recreational water (dam)	Rainwater tanks	Drinking water	Recreational water	Stock water
Arsenic	3.4×10^{-6}	5.2×10^{-7}	0.01 A	0.1 AX	0.5 S
Cadmium	4.4×10^{-5}	6.6×10^{-6}	0.002 A	0.02 AX	0.01 S
Chromium (Cr VI assumed)	5.0×10^{-5}	7.4×10^{-6}	0.05 A	0.5 AX	1 S
Copper	5.0×10^{-5}	7.3×10^{-6}	2 A	20 AX	0.5 S
Lead	2.9×10^{-5}	4.3×10^{-6}	0.01 A	0.1 AX	0.1 S
Nickel	4.3×10^{-5}	6.4×10^{-6}	0.02 A	0.2 AX	1 S
Selenium	1.5×10^{-5}	2.2×10^{-6}	0.01 A	0.1 AX	0.02 S
Mercury (as inorganic and elemental)	4.3×10^{-5}	6.4×10^{-6}	0.001 A	0.001 AX	0.002 S
Zinc	1.2×10^{-4}	1.9×10^{-5}	6 U	60 UX	20 S
Total PFAS (as PFOS)	1.9×10^{-7}	2.4×10^{-8}	0.00007 A	0.002 H	7×10^{-5} A
Dioxins and furans (WHO-TEQ)	4.8×10^{-11}	7.3×10^{-12}	1.6×10^{-8} A	1.6×10^{-7} AX	1.6×10^{-8} A
PAHs (as BaP)	4.3×10^{-7}	4.9×10^{-8}	1×10^{-5} A	1×10^{-4} AX	1×10^{-5} A

Refer to **Appendix B and C** for the calculation of water concentrations
A = Australian Drinking Water Guidelines (NHMRC 2011 updated 2022), with the exception of dioxins where the drinking water guideline in the recycled water guidelines has been adopted (NRMMC 2008)
U = Residential Tap Water Regional Screening Level available from USEPA (USEPA 2024)
AX = Recreational water guideline based on 10 x Australian drinking water guidelines (NHMRC 2011 updated 2022)
UX = Recreational water guideline based on 10 x Tap Water Regional Screening Level available from USEPA (USEPA 2024)
H = Recreational water guideline for PFAS (specifically PFOS+PFHxS and PFOS) derived by NHMRC (NHMRC 2019) and also presented in the NEMP (HEPA 2020). Total PFAS has been assessed assuming total PFAS (as a sum) is as toxic as PFOS+PFHxS
S = Stock water guideline as per ANZECC (ANZECC/ARMCANZ 2000) – with the lowest guideline adopted (noting that chickens are most likely to be the stock present). Note, where stock water guideline not available the drinking water guideline has been adopted.

Table 4.8 indicates that the predicted water concentrations (as total concentrations comprising both dissolved and particulates) in recreational water and rainwater tanks that may be used for non-potable purposes or for stock watering (e.g. pets and chickens) are all well below the adopted water guidelines. All concentrations are below 0.1% of the adopted guidelines. As a result, potential risks calculated for residential exposures (**Table 4.7**) do not change where the HI (ratio of water concentration to guideline) is added to the total HI.

The MOS relevant to these calculations is in the range of 200 to around 250,000. This is more than sufficient to address any variability in assumptions adopted to estimate the concentrations and any future changes to guidelines.

The calculations above relate to the contribution of the operation of the facility to existing water quality. Existing water quality, particularly in rainwater tanks is not known. However, the calculations undertaken also show that the contribution from the facility would not be measurable in any water analysis. Hence emissions from the facility would not result in any change in water quality relevant to the offsite waterways and rainwater tank quality.

Based on the assessment undertaken, there are no risk issues of concern in relation to potential exposures of persistent and bioaccumulative chemicals that may be deposited to waterways used for recreational purpose or rainwater tanks (used for any purpose).

4.5 Uncertainties

The quantification of human health risks has relied on the modelling of emissions to air and prediction of worst-case or maximum impacts in the off-site community. Hazards associated with potential exposure to the chemicals evaluated is based on current toxicological information relevant to the chemicals evaluated. This includes guidance provided in the PFAS NEMP (HEPA 2020). Quantification of risk has utilised a number of assumptions that are expected to overestimate actual exposure to chemicals derived from the facility.

Further review of some assumptions has been undertaken as detailed below.

Worst-case residential soil exposures

The focus of the assessment of deposition and multi-pathway exposures has been for the closest sensitive receptor, namely the closest semi-rural residential property. There may be the potential for the community to come into direct contact with chemicals deposited to surface soil in areas closer to the site when accessing and using these areas for recreational or commercial/industrial purposes. To address this aspect the maximum predicted surface soil concentrations (refer to **Appendices B and C**) relevant to the maximum impacted offsite receptor – regardless of land use have been compared against soil guidelines protective of both recreational and commercial/industrial land use (which is protective of ingestion, dermal contact and dust inhalation). This is presented in **Table 4.9**.

Table 4.9: Review of maximum predicted surface soil concentrations – recreational and commercial/ industrial land use

Persistent chemical	Maximum surface soil concentration from facility* (mg/kg)	Health based guideline (mg/kg)	
		Recreational	Commercial/ industrial
Arsenic	0.0067	300 [N]	3,000 [N]
Cadmium	0.085	90 [N]	900 [N]
Chromium (Cr VI assumed)	0.095	300 [N]	3,600 [N]
Copper	0.094	17,000 [N]	240,000 [N]
Lead	0.056	600 [N]	1,500 [N]
Nickel	0.083	1,200 [N]	6,000 [N]
Selenium	0.028	700 [N]	10,000 [N]
Mercury (inorganic)	0.083	80 [N]	730 [N]
Zinc	0.25	30,000 [N]	400,000 [N]
Total PFAS (as PFOS)	0.00023	1 [H]	20 [H]
Dioxins and furans (WHO-TEQ)	0.00000004	0.00005 [U]	0.00072 [U]
PAHs (as BaP)	0.00027	3 [N]	40 [N]

Refer to **Appendix B** for the methodology used to calculate soil concentrations
* Calculated on following deposition and accumulation in surface soil over 35 years – from the facility alone with no other sources of dust deposition considered
N = NEPM Health Investigation levels (HILs) for recreational and commercial/industrial land use (NEPC 1999 amended 2013a)
H = PFAS NEMP (HEPA 2020)
U = USEPA Regional Screening Levels (RSLs) for residential soil, adopted for the assessment of recreational exposures (adopting residential criteria in the absence of recreational values) and commercial/industrial worker exposures (USEPA 2024)

Table 4.9 indicates that the maximum predicted concentrations in soil relevant to recreational and commercial/industrial areas are well below the relevant soil guidelines protective health. In addition, the predicted concentrations would not be measurable and would not result in any change to existing soil concentrations in the surrounding environment.

Hence there would be no risk issues of concern in relation to the deposition of dust from the facility to soil in the surrounding recreational and commercial/industrial areas.

Duration of operation

One of the key assumptions relates to the duration of the operation of the facility. The calculations presented have assumed that the facility would operate for at least 35 years, the duration of residential occupation. Where the facility is operated continuously for a longer period of time, there is the potential for risks to be higher. However, this is unlikely to occur as the calculations undertaken have assumed that deposition to the ground from the facility would be the only contribution to soil – i.e. no other dust deposition occurs at all. This will not be the case as dust derived from the facility would be a small proportion of existing dust deposition (from wind erosion of surface soil, roads and other sources), which would mean the accumulated dust deposition from the facility would be mix with deposition from other sources. In addition, changes in technology relevant to managing emissions would be expected to change over this period of time, resulting in lower emissions to air. Hence the concentrations estimated after accumulation from the facility alone for 35 years would be conservative for longer term operations. It is also noted that it is likely that technology changes would result in lower emissions to air over time.

Where the facility operates for a shorter period of time, the overall risks will be lower.

For example, should the facility operate for 20 years rather than 35 years, risks for all exposure pathways evaluated (inhalation, ingestion and dermal contact with soil, ingestion of homegrown fruit and vegetables and ingestion of home-produced eggs) reduces to 7×10^{-7} as the maximum non-threshold risk and 0.076 for the maximum HI.

Assumptions regarding the nature of chemicals present

This assessment has adopted a number of conservative assumptions in relation to the chemicals that are preset in the emissions to air. This is discussed further in **Section 4.2.6.3**. The calculation of risk has been refined on the basis of assuming conservative, but not worst-case, assumptions regarding the proportion of the more toxic forms of the chemicals that may be present, as follows:

- Chromium VI: Limited data is available on the proportion of total chromium that is CrVI. One publication (Spanos et al. 2016) suggests up to 3.5%. Analysis of emissions from the Loganholme facility did not detect CrVI in emissions. For this review CrVI is assumed to comprise up to 10% of total Cr.
- VOCs: VOCs derived from the proposed facility are most likely to be dominated by the presence of light hydrocarbons propane, which is significantly less toxic than benzene. For this review it is assumed that benzene comprises 10% of the total VOCs (remains highly conservative).
- PAHs: While the proportion of BaP in total PAHs is unlikely to be 100%, the calculated risks derived from exposure to BaP emissions (non-threshold risk) are very low, with exposure to

PAHs adopting this conservative assumption is negligible. Adjusting this assumption to be less conservative is not considered necessary given the very low levels of risk.

- PFAS: No data is available data on the composition of PFAS likely to be present in air emissions from the proposed facility. Data on PFAS in wastewater and sludge (expected to be treated in the facility) suggests PFOS may be up to 50% of total PFAS. For this review it is assumed that PFOs represents 50% of total PFAS.

Where the above assumptions are adopted, risks to human health for the maximum impacted residential location have been revised. **Table 4.10** presents the revised risks for the exposure pathways evaluated, with comparison against the risks presented in **Table 4.7**.

Table 4.10: Revised risks where conservative but not worst-case assumptions regarding chemical composition is adopted (maximum sensitive receptor)

Exposure pathway	Non threshold risk		HI	
	Adults	Young children	Adults	Young children
Risks calculated and presented in Table 4.7 (worst-case scenario)				
Individual exposure pathways				
Inhalation (I)	1.2×10^{-6}	1.2×10^{-6}	0.064	0.064
Soil ingestion (SI)	1.9×10^{-11}	3.6×10^{-11}	0.00038	0.0064
Soil dermal contact (SD)	7.0×10^{-11}	2.9×10^{-11}	0.000064	0.00015
Ingestion of homegrown fruit and vegetables (F&V)	5.7×10^{-11}	4.4×10^{-11}	0.0029	0.012
Ingestion of homegrown eggs (E)	1.3×10^{-14}	5.4×10^{-15}	0.00016	0.00035
Multiple pathways (i.e. combined exposure pathways)				
I + SI + SD	1.2×10^{-6}	1.2×10^{-6}	0.064	0.071
I + SI + SD + F&V	1.2×10^{-6}	1.2×10^{-6}	0.067	0.082
I + SI + SD + E	1.2×10^{-6}	1.2×10^{-6}	0.065	0.071
I + SI + SD + F&V + E	1.2×10^{-6}	1.2×10^{-6}	0.068	0.082
Revised risks – conservative but refined assumptions for chromium, VOCs and PFAS				
Individual exposure pathways				
Inhalation (I)	1.3×10^{-7}	1.3×10^{-7}	0.049	0.049
Soil ingestion (SI)	1.9×10^{-11}	3.6×10^{-11}	0.00030	0.0049
Soil dermal contact (SD)	7.0×10^{-11}	2.9×10^{-11}	0.000064	0.00015
Ingestion of homegrown fruit and vegetables (F&V)	5.7×10^{-11}	4.4×10^{-11}	0.0027	0.0105
Ingestion of homegrown eggs (E)	1.3×10^{-14}	5.4×10^{-15}	0.00012	0.00028
Multiple pathways (i.e. combined exposure pathways)				
I + SI + SD	1.3×10^{-7}	1.3×10^{-7}	0.049	0.054
I + SI + SD + F&V	1.3×10^{-7}	1.3×10^{-7}	0.052	0.064
I + SI + SD + E	1.3×10^{-7}	1.3×10^{-7}	0.049	0.054
I + SI + SD + F&V + E	1.3×10^{-7}	1.3×10^{-7}	0.052	0.065
Acceptable risk/HI	**$\leq 1 \times 10^{-5}$**		**≤1**	

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

The calculated risks for the revised assumptions presented above are lower. While the total HI has reduced, the most significant change is for the calculated non-threshold risk where the calculated risk is reduced by a factor of 10. This is because these risks are dominated by inhalation exposures of VOCs, where the proportion of total VOCs that comprise benzene dominates the calculations. This illustrates the highly conservative nature of the assumptions adopted for the quantification of risk, in particular the non-threshold risk.

Section 5. Ecological impacts

5.1 General

This section presents a review and further assessment of potential impacts to the environment in relation to emissions to air from the proposed carbonisation facility.

As detailed in **Section 2**, the key risk issues of concern in relation to the off-site environment are as follows:

- direct toxicity effects of air emissions, specifically the potential for vegetation effects of direct exposure to gases released from the facility
- deposition of chemicals to soil and water, and
    - direct toxicity effects relevant to terrestrial and aquatic environments
    - potential effects associated with bioaccumulation.

These risk issues are further assessed in the following sections.

5.2 Direct toxicity – Air emissions

Emissions to air, particularly in relation to gases, have the potential to result in direct toxicity to vegetation. A range of gases predicted to be released to air from the facility are known to have effects on vegetation. These effects are not of concern for emissions to air of metals, PAHs, dioxins and PFAS as the key issues relating to these chemicals relate to long-term effects to terrestrial and aquatic organisms, where deposited to these environments (assessed in **Sections 5.3 and 5.4**).

In relation to potential direct effects on vegetation, these related to concentrations of SO_2, HCl, HF and ammonia in air. Guidelines are available for these gases that are protective of vegetation effects. The guidelines address a wide range of effects and hence relate to a range of different averaging periods.

Table 5.1 presents a summary of the available guidelines and key effects of concern addressed by these guidelines, that are protective of vegetation effects, the averaging time relevant to these guidelines and the maximum predicted concentration in air (anywhere – likely on the site boundary) for the averaging period.

Review of **Table 5.1** indicates that there are no exceedances of the guidelines relevant to the protection of vegetation from direct contact with concentrations in air.

Table 5.1: Review of potential direct effects on vegetation

Gas released to air from the facility	Vegetation effects	Air guideline – Protective of vegetation effects (mg/m^3)	Averaging time	Maximum predicted air concentration from facility for relevant averaging time (mg/m^3)	Comments
Ammonia (total)	Ammonia is naturally occurring in the atmosphere and is used by plants as a source of nitrogen. Depending on the ambient air concentration, plants can either absorb or be an emitter of ammonia. At high concentrations in air ammonia can cause direct toxic effects on plants by leaf etching or yellowing of foliage. These effects occur at a point at which the plants assimilation capacity for ammonia is overwhelmed (TCEQ 2014). Review by the WHO included consideration of effects on plant growth (longer term effects) and is considered provisional as there are a wide range of aspects that are uncertain (WHO 2000a)	2 (TCEQ 2014) 0.27 (WHO 2000a) 0.008 (WHO 2000a)	1-hour average (short-term effects) 24-hour average (short-term effects) Annual average (long-term effects)	0.0046 0.0018 0.00036	The maximum concentration of total ammonia (NH_3 and NH_4) is below the guideline for ammonia (NH_3) relevant to the protection of short-term effects (1-hour and 24-hour average) and long-term effects. The proportion of NH_3 in the emissions is not known, however it is unlikely that 100% of total ammonia would be in the form NH_3. This assessment is therefore conservative.
Hydrogen chloride (HCl)	Hydrogen chloride can cause damage to vegetation, however only at high concentrations. The threshold for vegetation damage is noted to be higher than for human health effects. The available data indicate a range from 1 ppm for no effects in several plant species exposed for 5 hours to 3 ppm for 4 hours that results in some visible injury (TCEQ 2015b).	1.5 to 6 (TCEQ 2015b)	1-hour average (short-term effect)	0.0019	The maximum concentration is well below the relevant guideline.
Hydrogen fluoride (HF)	Fluoride (F) is a phytotoxic air chemical. HF and F produce a wide range of effects such as reduction in plant growth, induction of leaf chlorosis, effects on photosynthesis, respiration and enzyme activity. F is an accumulative toxicant hence guidelines are available for both short-term exposures and long-term exposures. The long-term guideline is also protective of effects (specifically fluorosis) in cattle (TCEQ 2015a)	0.003 0.0006 (TCEQ 2015a)	24-hour average Annual average	0.00003 0.0000045	The maximum concentrations of HF are well below the short-term and long-term guidelines.

Gas released to air from the facility	Vegetation effects	Air guideline – Protective of vegetation effects (mg/m^3)	Averaging time	Maximum predicted air concentration from facility for relevant averaging time (mg/m^3)	Comments
Sulfur dioxide	High concentrations of sulfur dioxide can produce acute injury in the form of foliar necrosis, even after relatively short duration exposure. However, such effects are far less important in the field than chronic injury, which results from long-term exposure to much lower concentrations of the gas and is essentially cumulative in nature, taking the form of reduced growth and yield and increased senescence, often with no clear visible symptoms or else with some degree of chlorosis (WHO 2000a). The chronic guidelines established by the WHO is also protective of acute effects.	0.01 (WHO 2000a)	Annual average	0.00135 (including background)	The maximum concentration of SO_2 (facility plus background) is below the relevant guideline.

5.3 Terrestrial environments

Where there may be deposition of chemicals that are persistent and bioaccumulative to land, these chemicals may accumulate in the soil and have the potential for adverse effects to terrestrial environments, where present. There is no terrestrial environment present on the site, however all areas surrounding the site have terrestrial environments that are expected to warrant some level of protection.

To assess the potential for terrestrial impacts, a conservative approach has been adopted in this assessment. The maximum predicted deposition rate at all the offsite receptors (regardless of land use) has been adopted to determine the maximum concentration that may be present in the soil profile (i.e. top 0.15 m) that is relevant to plant and soil health. This calculation is presented in **Appendix C**.

Table 5.2 presents the calculated soil concentrations and provides comparison against relevant guidelines based on the protection of terrestrial environments. Where available the terrestrial guidelines adopted that are relevant to the land use of the area, namely parklands and urban/suburban areas. In relation to the assessment of PFAS, guidelines are available that address direct toxicity effects as well as indirect effects, which include effects on secondary consumers (HEPA 2020). Both guidelines have been adopted in this review.

Table 5.2: Review of potential terrestrial impacts

Persistent and bioacumulative chemical	Calculated concentration in soil (mixed to 0.15m) closest to site (mg/kg)	Terrestrial guideline (mg/kg)	
		Direct toxicity – parkland and urban areas	Indirect effects (where relevant)
Arsenic	0.00084	100 [N]	--
Cadmium	0.011	0.36 [R4]	--
Chromium (Cr VI assumed)	0.012	190 [N]	--
Copper	0.011	95 [N]	--
Lead	0.0071	470 [N]	--
Nickel	0.01	30 [N]	--
Selenium	0.0036	0.52 [R4]	--
Mercury (as inorganic and elemental)	0.011	0.013 [R4]	--
Zinc	0.028	230 [N]	--
Total PFAS (assume as toxic as PFOS)	0.000025	1 [U]	0.01 [U]
Dioxins and furans (WHO-TEQ)	7×10^{-9}	3.15×10^{-6} [R4]	--
PAHs (as BaP)	0.000042	33 [C]	--

Refer to **Appendix C** for the calculation of soil concentrations
N = NEPM ecological investigation level for sensitive environments adopted for arsenic, lead, chromium (as added contaminant level assuming 1% clay content), copper and nickel (as added contaminant levels assuming cation exchange capacity [CEC] of 5 $cmol_c/kg$) and zinc (as added contaminant level assuming CEC of 5 $cmol_c/kg$ and pH of 6). The soil properties adopted are generic values that are conservative (as no data is available specific to the off-site soil). It is expected that actual soil conditions would differ, and the guidelines are expected to be less conservative.
H = PFAS NEMP (HEPA 2020)
C = Ecological screening level of BaP as per CRC CARE (CRC CARE 2017)
U = USEPA Soil Screening Level (SSL) for cadmium, selenium and chromium (USEPA 2005b, 2007b, 2007a)
R4 = USEPA Region 4 ecological screening criteria protective of all species (updated in 2018)
https://rais.ornl.gov/documents/era_regional_supplemental_guidance_report-march-2018_update.pdf

Table 5.2 indicates that all predicted soil concentrations derived from the operation of the proposed facility, continually for 35 years, are well below the guidelines available for the protection of terrestrial environments.

Predicted concentrations in soil derived from the facility are considered negligible and would not impact on existing background levels in soil in the area. Hence there are no risk issues of concern in relation to terrestrial effects from deposition of emissions from the facility to soil.

5.4 Aquatic environments

Where there may be deposition of chemicals that are persistent and bioaccumulative to water bodies located in areas surrounding the facility, there is the potential for these chemicals to accumulate and impact on water quality in these areas. Where this may occur, and aquatic species are present, there is the potential for effects.

Section 4.4 presents the approach adopted for estimating water concentrations in the closest water body to the site. The dams noted to be located in the surrounding area, particularly in parkland areas closer to eastern Creek do not have aquatic environments, however the aquatic environment of Eastern Creek is of potential relevance. For the offsite waterways the water concentration has been estimated on the basis of the presence of a dam, or ephemeral waterway which is flushed once per year. Where Eastern Creek flows, such calculations would be highly conservative. However during drier conditions, it is assumed that the calculation represents a worst-case concentration that could be present in the creek, or ponds within the creek.

When assessing potential impacts to aquatic species, the dissolved phase concentration in the waterway is relevant.

Table 5.3 presents the maximum predicted dissolved phased concentrations in the closest water body, with comparison against water quality guidelines relevant to the protection of freshwater environments. The guidelines adopted relate to the 95% species protection level, except where the 99% species protection level is recommended to address bioaccumulation or other issues. The water quality guidelines, as default water quality guidelines have been adopted from the Australian and New Zealand Guidelines for Fresh and Marine Water Quality (ANZG 2018). It is noted there are currently no finalised Australian water quality guidelines for PFAS. Hence the interim criteria presented in the NEMP (HEPA 2020) have been adopted.

It is noted that concentrations in water bodies located further from the site will be lower than presented in **Table 5.3**, as the rate of deposition will be lower.

Table 5.3: Summary and review of impacts to aquatic environments

Persistent and bioacumulative chemical	Calculated concentration in closest water body – dissolved (mg/L)	Marine water guideline [A] (mg/L)	
		95% species protection	99% species protection where bioaccumulation is important
Arsenic	2.4×10^{-7}	0.013 [A]	--
Cadmium	1.2×10^{-6}	0.0002 [A]	--
Chromium (Cr VI assumed)	5.5×10^{-11}	0.001 [A]	--
Copper	2.8×10^{-6}	0.0014 [A]	--
Lead	6.5×10^{-8}	0.0034 [A]	--
Nickel	1.3×10^{-6}	0.011 [A]	--
Selenium	5.9×10^{-6}	0.011 [A]	0.005 [A]
Mercury (as inorganic and elemental)	1.7×10^{-6}	0.0006 [A]	0.0006 [A]
Zinc	3.9×10^{-6}	0.008 [A]	--
Total PFAS (assume as toxic as PFOS)	3.4×10^{-8}	0.00013 [N]	2.3×10^{-7} [N]
Dioxins and furans (WHO-TEQ)	1.5×10^{-15}	5×10^{-9} [A]	2×10^{-10} [A]
PAHs (as BaP)	1.4×10^{-10}	0.0002 [A]	0.0001 [A]

Refer to **Appendix B and C** for the calculation of water concentrations
A = Freshwater quality guidelines available from the Australian and New Zealand Fresh and Marine Water Guidelines (as default toxicant guidelines) (ANZG 2018). These values have been adopted unless noted otherwise.
N = Freshwater guidelines as presented in the NEMP (HEPA 2020). Total PFAS has been assessed assuming total PFAS (as a sum) is as toxic as PFOS, which is highly conservative particularly in relation to bioaccumulation as many other PFAS do not bioaccumulate)

Review of **Table 5.3** indicates that all predicted concentrations in water derived from the proposed facility, assuming a worst-case scenario in terms of flows in Eastern Creek, are below the relevant guidelines for protection of direct toxicity and bioaccumulation effects in the closest aquatic environment.

Predicted concentrations in water derived from the facility are considered negligible and would not impact on existing background levels in water in the area. Hence there are no risk issues of concern from emissions to air from the facility in relation to the protection of aquatic environments.

5.5 Uncertainties

The assessment undertaken in relation to potential terrestrial and aquatic effects that may occur as a result of the operation of the facility is conservative as it is assumed that impacts assessed on the boundary at the closest receptor (regardless of land use) or in the closest water body (for aquatic effects) are representative of the larger off-site terrestrial and aquatic environments. Concentrations and deposition rates reduce significantly with increasing distance from the site, and hence the potential or adverse impacts would be lower.

One aspect that may affect the assessment of potential impacts is the duration of the operation of the facility. Where the facility operates for a longer period of time there may be greater potential for deposition to result in a higher accumulation of chemicals in the environment, however this may not occur as a result of technology changes with emissions controls. This is only relevant to the assessment of potential soil impacts as it is assumed that waterbodies are flushed once per year, which is conservative for Eastern Creek which is expected to flow more often.

Section 6. Conclusions

This assessment has considered potential impacts to human health and the environment in relation to emissions to air from the operation of the proposed carbonisation facility at the Riverstone WRRF.

The assessment has considered the outcomes of modelling emissions to air from the proposed facility and the potential for exposure to occur close to and in areas surrounding the site. The assessment has considered the maximum predicted concentrations in air, along with the deposition and potential accumulation of metals, dioxins and furans, polycyclic aromatic hydrocarbons (PAHs) and per- and polyfluoroalkyl substances (PFAS) in the semi-rural residential, commercial/industrial, open space, terrestrial and aquatic environments.

Based on the available data, and with consideration of the uncertainties identified, the following can be concluded:

- there are no health risk issues of concern in relation to potential exposures by workers, recreational users of areas adjacent to the site
- there are no health risk issues of concern in relation to potential exposures by residents in the areas surrounding the site, including where residents consume homegrown fruit and vegetable and eggs
- there are no health risk issues of concern in relation to the health of pets or stock, such as chickens, where water from rainwater tanks is used
- there are no health risk issues of concern in relation to potential exposure to water in rainwater tanks by residents (noting reticulated potable water is available in the area)
- there are no health risk issues of concern in relation to potential recreational exposures in Eastern Creek (in relation to emissions to air from the facility)
- there are no risk issues of concern in relation to potential exposures by terrestrial and aquatic environments in areas adjacent to and surrounding the site.

Section 7. References

Australian Bureau of Statistics, 2024. Selected characteristics retrieved from QuickStats, TableBuilder and DataPacks. www.abs.gov.au. Accessed 2024.

AIHW 2018, *Autralia's Health 2018*, Australian Institute of Health and Welfare, Canberra.

ANZECC/ARMCANZ 2000, *Australian and New Zealand Guidelines for Fresh and Marine Water Quality*, Australian and New Zealand Environment and Conservation Council and Agriculture and Resource Management Council of Australia and New Zealand.

ANZG 2018, *Australian and New Zealand Guidelines for Fresh and Marine Water Quality*, A joint initiative of the Australian and New Zealand Governments in partnership with the Australian state and territory governments, Online. viewed August 2018, <http://www.waterquality.gov.au/anz-guidelines>.

ATSDR 2003, *Toxicological Profile for Selenium*, U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry.

ATSDR 2012, *Toxicological Profile for Chromium*, Agency for Toxic Substances and Disease Registry, United States Department of Health and Human Services, Atlanta, Georgia, USA. viewed 2015, <http://www.atsdr.cdc.gov/ToxProfiles/tp7.pdf>.

ATSDR 2016, *Toxicological Profile for Hydrogen Sulfide and Carbonyl Sulfide*, Agency for Toxic Substances and Disease Registry, Public Health Service, US Department of Health and Human Services. <https://www.atsdr.cdc.gov/ToxProfiles/tp.asp?id=389&tid=67>.

Coggan, TL, Moodie, D, Kolobaric, A, Szabo, D, Shimeta, J, Crosbie, ND, Lee, E, Fernandes, M & Clarke, BO 2019, 'An investigation into per- and polyfluoroalkyl substances (PFAS) in nineteen Australian wastewater treatment plants (WWTPs)', *Heliyon,* vol. 5, no. 8, 2019/08/01/, p. e02316.

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

CRC CARE 2017, *Risk-based management and remediation guidance for benzo(a)pyrene, Technical Report No. 39*, Cooperative Research Centre for Contamination Assessment and Remediation of the Environment, Newcastle.

DEH 2004, *National Dioxin Program: Dioxins in Australia: A summary of the findings of studies conducted form 2001 to 2004*, Department of the Environment and Heritage, Australian Government.

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

enHealth 2010, *Guidance on use of rainwater tanks*, Commonwealth of Australia. <https://www1.health.gov.au/internet/main/publishing.nsf/Content/0D71DB86E9DA7CF1CA257BF0001CBF2F/$File/enhealth-raintank.pdf>.

enHealth 2012a, *Environmental Health Risk Assessment, Guidelines for assessing human health risks from environmental hazards*, Commonwealth of Australia, Canberra. <https://www1.health.gov.au/internet/main/publishing.nsf/Content/A12B57E41EC9F326CA257BF0001F9E7D/$File/Environmental-health-Risk-Assessment.pdf>.

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

EPHC 2005, *National Dioxins Program - National Action Plan for Addressing Dioxins in Australia*, Environment Protection and Heritage Council. <http://www.nepc.gov.au/system/files/resources/74b7657d-04ce-b214-d5d7-51dcbce2a231/files/cmgt-rev-national-dioxins-program-national-action-plan-addressing-dioxins-australia-200510.pdf>.

EPHC 2009, *Environmental Risk Assessment Guidance Manual for Agricultural and Veterinary Chemicals*, Environment Protection and Heritage Council. <http://www.scew.gov.au/publications/pubs/chemicals/cmgt_nchem__eragm_for_agricultural_and_veterinary_chemicals_200902.pdf>.

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

Ewald, B, Knibbs, LD, Campbell, R & Marks, GB 2020, 'Public health opportunities in the Australian air quality standards review', *Australian and New Zealand Journal of Public Health,* vol. n/a, no. n/a.

FAO/WHO 2018, *Joint FAO/WHO Food Standards Programme. Codex Committee on Contaminants in Foods. 12th Session, Utrecht, 12–16 March 2018. Proposed draft revision of the Code of Practice for the Prevention and Reduction of Dioxins and Dioxin-like PCBs in Food and Feed*, Food and Agriculture Organzation and World Health Organization. <http://www.fao.org/fao-who-codexalimentarius/sh-proxy/en/?lnk=1&url=https%253A%252F%252Fworkspace.fao.org%252Fsites%252Fcodex%252FMeetings%252FCX-735-12%252FWD%252Fcf12_08e.pdf>.

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

FSANZ 2008, *The 22nd Australian Total Diet Study*, Food Standards Australia and New Zealand. <http://www.foodstandards.gov.au/Pages/default.aspx>.

FSANZ 2011, *The 23rd Australian Total Diet Study*, Food Standards Australia and New Zealand. <http://www.foodstandards.gov.au/Pages/default.aspx>.

FSANZ 2017a, *Consolidated Report - Perfluorinated chemicals in food*, Food Standards Australia and New Zealand. <http://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-pfas-hbgv.htm#final>.

FSANZ 2017b, *Hazard assessment report - Perfluorooctane sulfonate (PFOS), Perfluorooctanoic acid (PFOA), Perfluorohexane sulfonate (PFHxS)*, Food Standards Australia and New Zealand.

FSANZ 2019, *25th Australian Total Diet Study*, Food Standards Australia New Zealand (FSANZ).

FSANZ 2020, *26th Australian Total Diet Study*, Food Standards Australia New Zealand. <https://www.foodstandards.gov.au/publications/Pages/26th-Australian-Total-Diet-Study.aspx>.

FSANZ 2021, *27th Australian Total Diet Study. Per- and poly-fluoroalkyl substances*, Food Standards Australia and New Zealand.

HEPA 2020, *PFAS National Environmental Management Plan Version 2.0*, The National Chemicals Working Group (NCWG) of the Heads of EPAs Australia and New Zealand (HEPA). <http://www.environment.gov.au/protection/chemicals-management/pfas>.

IARC 2012, *IARC Monographs on the Evaluation of Carcinogenic Risks to Humans, Volume 100C, Arsenic, Metals, Fibres and Dusts*. <http://monographs.iarc.fr/ENG/Monographs/vol100C/index.php>.

ITER 1998, *ITER Peer Review on Hexavalent Chromium Meeting Summary, April 16, 1998*. <http://www.tera.org/Peer/HexavalentChromium1998MeetingReport.pdf>.

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

Krewski, D, Jerrett, M, Burnett, RT, Ma, R, Hughes, E, Shi, Y, Turner, MC, Pope, CA, 3rd, Thurston, G, Calle, EE, Thun, MJ, Beckerman, B, DeLuca, P, Finkelstein, N, Ito, K, Moore, DK, Newbold, KB, Ramsay, T, Ross, Z, Shin, H & Tempalski, B 2009, 'Extended follow-up and spatial analysis of the American Cancer Society study linking particulate air pollution and mortality', *Research report*, no. 140, May, pp. 5-114; discussion 15-36.

Kus, B, Kandasamy, J, Vigneswaran, S & Shon, HK 2010, 'Analysis of first flush to improve the water quality in rainwater tanks', *Water Sci Technol,* vol. 61, no. 2, pp. 421-8.

Leeman, WR, Van Den Berg, KJ & Houben, GF 2007, 'Transfer of chemicals from feed to animal products: The use of transfer factors in risk assessment', *Food Additives & Contaminants,* vol. 24, no. 1, 2007/01/01, pp. 1-13.

Lizárraga-Mendiola, L, Vázquez-Rodríguez, G, Blanco-Piñón, A, Rangel-Martínez, Y & González-Sandoval, M 2015, 'Estimating the Rainwater Potential per Household in an Urban Area: Case Study in Central Mexico', *Water,* vol. 7, no. 9, pp. 4622-37.

Lowe, JA, Dietrich, W & Alberts, MT 1991, 'Health Risk Assessment for Waste-To-Energy Projects in California', in HA Hattemer-Frey & C Travis (eds), *Health Effects of Municipal Waste Incineration*, CRC Press, pp. 163-200.

MacLachlan, DJ 2011, 'Estimating the transfer of contaminants in animal feedstuffs to livestock tissues, milk and eggs: a review', *Animal Production Science,* vol. 51, no. 12, pp. 1067-78.

Martinson, B & Thomas, T 2009, *Quantifying the first-flush phenomenon: effects of first-flush on water yield and quality*, In: 14th International Rainwater Catchment Systems Conference, Kuala Lumpur.

MfE 2011, *Toxicological intake values for priority contaminants in soil*, New Zealand Ministry for the Environment, Wellington. <http://mfe.govt.nz/publications/hazards/toxicological-intake-values-priority-contaminants-soil>.

Morgan, G, Broom, R & Jalaludin, B 2013, *Summary for Policy Makers of the Health Risk Assessment on Air Pollution in Australia*, Prepared for National Environment Protection Council by the University Centre for Rural Health, North Coast, Education Research Workforce, A collaboration between The University of Sydney, Southern Cross University, The University of Western Sydney, The University of Wollongong, Canberra.

NEPC 1999 amended 2013a, *Schedule B1, Guideline on Investigation Levels For Soil and Groundwater, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 1999 amended 2013b, *Schedule B4, Guideline on Site-Specific Health Risk Assessment Methodology, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 1999 amended 2013c, *Schedule B7, Guideline on Derivation of Health-Based Investigation Levels, National Environment Protection (Assessment of Site Contamination) Measure*, National Environment Protection Council. <https://www.legislation.gov.au/Details/F2013L00768/Download>.

NEPC 2004, *National Environment Protection (Air Toxics) Measure*, National Environment Protection Council. <http://scew.gov.au/nepms/air-toxics>.

NEPC 2010, *Review of the National Environment Protection (Ambient Air Quality) Measure, Discussion Paper, Air Quality Standards*, National Environmental Protection Council.

NEPC 2011, *Methodology for setting air quality standards in Australia Part A*, National Environment Protection Council, Adelaide.

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

NEPC 2021, *National Environment Protection (Ambient Air Quality) Measure*, Australian Government. <https://www.legislation.gov.au/Details/F2021C00475>.

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

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

NHMRC 2006, *Nutrient Reference Values for Australia and New Zealand, Including Recommended Dietary Intakes*, National Health and Medical Research Council and New Zealand Ministry of Health,

NHMRC 2008, *Guidelines for Managing Risks in Recreational Water*, National Health and Medical Research Council, Canberra.

NHMRC 2011 updated 2022, *Australian Drinking Water Guidelines 6, Version 3.8 Updated September 2022, National Water Quality Management Strategy*, National Health and Medical Research Council, National Resource Management Ministerial Council., Canberra.

NHMRC 2019, *Guidance on Per and Polyfluoroalkyl substances (PFAS) in Recreational Water*, Australian Government National Health and Medical Research Council.

NRMMC 2008, *Australian Guidelines for Water Recycling: Managing Health and Environmental Risks (Phase 2) Augmentation of Drinking Water Supplies*, Natural Resource Management Ministerial Council, Environment Protection and Heritage Council and National Health and Medical Research Council. <http://waterquality.gov.au/guidelines/recycled-water>.

NSW Chief Scientist 2018, *Advisory Committee on Tunnel Air Quality - Technical Paper 5: Road Tunnel Stack Emissions*, Advisory Committee on Tunnel Air Quality, NSW Chief Scientist and Engineer. <https://chiefscientist.nsw.gov.au/reports/advisory-committee-on-tunnel-air-quality>.

NSW DEC 2003, *Ambient Air Quality Research Project (1996-2001), Internal working paper no. 4, Ambient concentrations of heavy metals in NSW*, Department of Environment and Conservation (NSW).

NSW EPA 2004, *Ambient Air Quality Research Project (1996-2001). Internal working paper no. 2, Ambient concentrations of toxic organic compounds in NSW*, Department of Environment and Conservation.

NSW EPA 2013, *Air Emissions in My Community web tool, Substance information*, NSW Environment Protection Authority. <http://www.epa.nsw.gov.au/resources/air/130841AEsubstance.pdf >.

NSW EPA 2016, *NSW EPA Approved Methods for the Modelling and Assessment of Air Pollutants in NSW*, NSW Environment Protection Authority, 2016.

NSW EPA 2019, *Air Emissions Inventory for the Greater Metropolitan Region in New South Wales 2013 Calendar Year Consolidated Natural and Human-Made Emissions: Results*, NSW Environment Protection Authority, NSW Government. <https://www.epa.nsw.gov.au/-/media/epa/corporate-site/resources/air/19p1917-air-emissions-inventory-2013.pdf?la=en&hash=9217ADF2C8D5647147FF00F447258319D00BB75D>.

NSW EPA 2022, *Approved Methods for the Modelling and Assessment of Air Pollutants in New South Wales*, State of NSW and Environment Protection Authority, Parramatta. <https://www.epa.nsw.gov.au/-/media/epa/corporate-site/resources/air/22p3963-approved-methods-for-modelling-and-assessment-of-air-pollutants.pdf>.

NSW Health 2019, *NSW Plan for Healthy Culturally and Linguistically Diverse Communities, 2019-2023*. <https://www1.health.nsw.gov.au/pds/ActivePDSDocuments/PD2019_018.pdf; https://www.mhcs.health.nsw.gov.au/about-us/cald-community>.

OEHHA, *OEHHA Acute, 8-hour and Chronic Reference Exposure Level (REL) Summary*, Office of Expoure and health Hazard Assessment. <https://oehha.ca.gov/air/general-info/oehha-acute-8-hour-and-chronic-reference-exposure-level-rel-summary>.

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

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

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

Ontario MfE 2004, *Air Dispersion Modelling Guideline for Ontario*, Standards Development Branch, Ministry of the Environment.

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

Pope, IC, Burnett, RT, Thun, MJ & et al. 2002, 'Lung cancer, cardiopulmonary mortality, and long-term exposure to fine particulate air pollution', *JAMA,* vol. 287, no. 9, pp. 1132-41.

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

Soares, ME, Vieira, E & Bastos Mde, L 2010, 'Chromium speciation analysis in bread samples', *J Agric Food Chem,* vol. 58, no. 2, Jan 27, pp. 1366-70.

Spanos, T, Ene, A, Patronidou, CS & Xatzixristou, C 2016, 'Temporal variability of sewage sludge heavy metal content from Greek wastewater treatment plants', *Ecological Chemistry and Engineering S,* vol. 23, no. 2, pp. 271-83.

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

TCEQ 2012, *Arsenic and Inorganic Arsenic Compounds, Development Support Document*, Texas Commission on Environmental Quality.

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

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

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

TCEQ 2016, *Cadmium and Cadmium Compounds, Development Support Document*, Texas Comission on Environmental Quality.

Thompson, J, Lorber, M, Toms, L-ML, Kato, K, Calafat, AM & Mueller, JF 2010, 'Use of simple pharmacokinetic modeling to characterize exposure of Australians to perfluorooctanoic acid and perfluorooctane sulfonic acid', *Environment international,* vol. 36, no. 4, 2010/05/01/, pp. 390-97.

Toms, LML, Thompson, J, Rotander, A, Hobson, P, Calafat, AM, Kato, K, Ye, X, Broomhall, S, Harden, F & Mueller, JF 2014, 'Decline in perfluorooctane sulfonate and perfluorooctanoate serum concentrations in an Australian population from 2002 to 2011', *Environment international,* vol. 71, 10//, pp. 74-80.

Toms, LML, Bräunig, J, Vijayasarathy, S, Phillips, S, Hobson, P, Aylward, LL, Kirk, MD & Mueller, JF 2019, 'Per- and polyfluoroalkyl substances (PFAS) in Australia: Current levels and estimated population reference values for selected compounds', *International journal of hygiene and environmental health,* vol. 222, no. 3, 2019/04/01/, pp. 387-94.

UK DEFRA & EA 2002, *Contaminants in Soil: Collation of Toxicological and Intake Data for Humans: Chromium*. <http://webarchive.nationalarchives.gov.uk/20140328084622/http://www.environment-agency.gov.uk/static/documents/Research/chromium_old_approach_2028660.pdf>.

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

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

USEPA 1998, *Toxicological Review of Hexavalent Chromium*. <http://www.epa.gov/iris/toxreviews/0144tr.pdf>.

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

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

USEPA 2005b, *Ecological Soil Screening Levels for Cadmium Interim Final*, US Environmental Protection Agency. <https://www.epa.gov/chemical-research/interim-ecological-soil-screening-level-documents>.

USEPA 2007a, *Ecological Soil Screening Levels for Manganese Interim Final*, US Environmental Protection Agency. <https://www.epa.gov/chemical-research/interim-ecological-soil-screening-level-documents>.

USEPA 2007b, *Ecological Soil Screening Levels for Selenium Interim Final*, US Environmental Protection Agency. <https://www.epa.gov/chemical-research/interim-ecological-soil-screening-level-documents>.

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

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

USEPA 2018, *Risk and Exposure Assessment for the Review of thePrimary National Ambient Air Quality Standard for Sulfur Oxides*, U.S. Environmental Protection Agency Office of Air Quality Planning and Standards Health and Environmental Impacts Division Research Triangle Park, NC. <https://www.epa.gov/sites/production/files/2018-05/documents/primary_so2_naaqs_-_final_rea_-_may_2018.pdf>.

USEPA 2019, *Integrated Science Assessment (ISA) for Particulate Matter (Final Report, 2019)*, U.S. Environmental Protection Agency, Washington, DC, EPA/600/R-19/188.

USEPA 2023, *Regional Screening Levels (RSL), May 2023*, United States Environmental Protection Agency. <https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables>.

USEPA 2024, *Regional Screening Levels (RSL), May 2024*, United States Environmental Protection Agency. <https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables>.

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

WA Department of Health 2009, *Environmental Health Guide - Hydrogen Sulfide and Public Health*, Department of Health, Government of Western Australia. <https://ww2.health.wa.gov.au/Articles/F_I/Hydrogen-sulfide-and-public-health>.

WHO 1991, *Environmental Health Criteria 108. Nickel.* <http://inchem.org/documents/ehc/ehc/ehc108.htm>.

WHO 2000a, *Air Quality Guidelines for Europe, Second Edition (CD ROM Version)*, Copenhagen. <https://www.euro.who.int/en/health-topics/environment-and-health/air-quality/publications/pre2009/who-air-quality-guidelines-for-europe,-2nd-edition,-2000-cd-rom-version>.

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

WHO 2000c, *Guidelines for Air Quality*, World Health Organisation, Geneva. <https://iris.who.int/bitstream/handle/10665/66537/WHO_SDE_OEH_00.02-eng.pdf?sequence=18>.

WHO 2000d, *WHO air quality guidelines for Europe, 2nd edition, 2000 (CD ROM version)*, World Health Organisation.

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

WHO 2003b, *Hydrogen Sulfide: Human Health Aspects Concise International Chemical Assessment Document No. 53*, World Health Organisation. <http://inchem.org/documents/cicads/cicads/cicad53.htm#6.1>.

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

WHO 2010, *WHO Guidelines for Indoor Air Quality, Selected Pollutants*, WHO Regional Office for Europe.

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

WHO 2013b, *Review of evidence on health aspects of air pollution - REVIHAAP Project, Technical Report*, World Health Organization, Regional Office for Europe.

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

WHO 2019, *Exposure to Dioxins and Dioxin-like Substances: A Major Public Health Concern*, World Health Organization.

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[2]. 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:

---

2 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)[3] 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 Project, with the maximum change from all residential receptors (where regional air quality is of most relevance) adopted in this calculation. For this assessment the change in $PM_{2.5}$ relates to the change in annual average air concentrations.
- 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 Hawkesbury LGA, with the most recent data indicating a rate of 520 per 100,000 as an age standardised rate which has been adopted in this assessment.

3 For regional guidance, such as that provided for Europe by the WHO WHO 2006, Health risks or particulate matter from long-range transboundary air pollution regional background incidence data for relevant health endpoints are combined with exposure-response functions to present an impact function, which is expressed as the number/change in incidence/new cases per 100,000 population exposed per microgram per cubic metre change in particulate matter 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 10 $\mu g/m^3$ 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 10 $\mu g/m^3$ 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):

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

$$= 0.153 \times 0.0052 \times 0.0058$$

$$= 5 \times 10^{-6}$$

Appendix B Toxicity of key chemicals evaluated

B1 Approach to the identification of toxicity reference values

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

For chemical substances that are not carcinogenic, a threshold exists below which there are no adverse effects (for all relevant end-points). The threshold typically adopted in risk calculations (a tolerable daily intake [TDI] or tolerable concentration [TC]) is based on the lowest no observed adverse effect level (NOAEL), typically from animal or human (e.g. occupational) studies, and the application of a number of safety or uncertainty factors. Intakes/exposures lower than the TDI/TC is considered safe, or not associated with an adverse health risk (NHMRC 1999). The threshold is more generically termed a toxicity reference value (TRV) and is protective of all adverse health effects, including respiratory, cardiovascular, developmental and reproductive[4] effects.

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**.

[4] Reproductive and development toxicity refers to an adverse effects of a substance on any part of the reproductive cycle which includes impairment of reproductive function in males and females as well as effects on the foetus or offspring, including lactation and breastfeeding, and development.

B2 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 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. Western Australian Guidelines for ammonia and protection of public health (WA Department of Health 2009), with the guideline adopted for 24-hours converted to a 1-hour average guideline
  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 2013c, 2004) by the WHO (WHO 2000c, 2000a, 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.
  4. Ontario Ministry of the Environment and Climate Change, with 24-hour average guidelines converted to 1-hour average guidelines.

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

The equation used to convert between different averaging times is:

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

Where

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

These stability classes are as follows:

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

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

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

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

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

For this assessment, almost all air concentrations have been provided from the AQIA model, for the correct averaging periods that need to be evaluated. Data for hydrogen fluoride has not been provided as a 1 hour average, hence the 24-hour average concentration has been used, along with an adjustment factor of 2.5 to calculate the 1 hour average concentrations for assessing acute exposures.

Based on the above the following acute TRVs have been adopted in this assessment. It is noted that no acute TRVs are available for a number of chemicals, specifically lead, zinc, PAHs, dioxins and furans and PAHs as these chemicals are either not acute toxicants or no suitable acute inhalation TRVs are available. All these chemicals have been assessed in relation to chronic exposures.

Some of the acute guidelines are based on a 24-houyr exposure period. The adjustment factors noted above have been used to convert the 24 hour average guideline to a 1 hour average guideline for use in this assessment.

Table B1: Acute TRVs adopted in this assessment

Chemicals	Acute air guideline (1-hour average) (mg/m^3)
Hydrogen chloride (HCl)	0.66 [1]
Hydrogen fluoride (HF)	0.06 [1]
Ammonia	0.59 [1]
Hydrogen sulfide (H_2S)	0.5 [3]
Arsenic	0.0099 [1]
Cadmium	0.00055 [1]
Chromium (Cr VI assumed)	0.0013 [1]
Copper	0.1 [2]
Nickel	0.0011 [1]
Selenium	0.025 [4]
Mercury (as elemental)	0.0006 [2]
VOCs (as benzene)	0.17 [1]

References for health-based acute air guidelines (1-hour average):
1 = Guideline available from the Texas Commission on Environmental Quality (TCEQ), https://www.tceq.texas.gov/toxicology/dsd/final.html
2 = 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
3 = Guideline available from the WA Department of Health (WA Department of Health 2009), converted from 24-hour average to 1 hour average using an averaging time conversion factor of 2.5 (discussed above).
4 = Guideline available from the Ontario Ministry of the Environment and Climate Change https://www.ontario.ca/page/ontarios-ambient-air-quality-criteria-sorted-contaminant-name converted from 24-hour average to 1 hour average using an averaging time conversion factor of 2.5 (discussed above)

B3 Values adopted for the assessment of chronic exposures

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

For carcinogens, this guidance requires consideration of the mechanism of action for the development of cancer. Some cancers are caused by a threshold mechanism, where there needs to be sufficient exposures to trigger the damage that results in or promotes the development of cancer. Other carcinogens are genotoxic/mutagenic and act in a way such that any level of exposure is assumed to result in damage that may increase the lifetime risk of cancer (i.e. no threshold exists). Not all carcinogenic (and not all mutagenic) pollutants cause cancer in the same way and hence the mechanism of action has been considered in the identification of appropriate TRVs for use in this assessment.

For the gaseous chemicals considered in this assessment, only inhalation TRVs have been adopted. For inorganics as well as dioxins and furans, PAHs and PFAS, TRVs relevant to all exposure pathways have been adopted. Background intakes of these chemicals have been estimated on the basis of the available information as detailed and presented in **Section B4**.

Tables B2 and B3 present the TRVs adopted for the assessment of chronic health effects associated with exposure to the other chemicals 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 chemicals – threshold effects

Chemical	Inhalation TRV (mg/m^3)	Oral TRV (mg/kg/day)	GI absorption factor*	Dermal TRV#	Dermal absorption from soil (unitless)*	Dermal permeability (water)** (cm/hr)
Hydrogen chloride (HCl)	0.026 T	NA (gaseous chemical)				
Hydrogen fluoride (HF)	0.029 T	NA (gaseous chemical)				
Hydrogen sulfide (H_2S)	0.02 W	NA (gaseous chemical)				
Ammonia	0.32 T	NA (gaseous chemical)				
Benzene	0.03 U	NA (gaseous chemical)				
Arsenic	0.000067 T	0.002 N	100%	0.002	0.005	0.001
Cadmium	0.000005 W	0.0008 W	100%	0.0008	0.0001	0.001
Chromium (Cr VI assumed)	0.0001 U	0.0009 A	100%	0.0009	0.0001	0.002
Copper	0.49 R	0.14 W	100%	0.14	0.0001	0.001
Lead	0.0005 N	0.0035 NH	100%	0.0035	0.0001	0.0001
Mercury (as inorganic and elemental)	0.0002 W	0.0006 W	7%	0.000042	0.001	0.001
Nickel	0.00002 E	0.012 W	100%	0.012	0.005	0.0002
Selenium	0.02 R	0.006 N1	100%	0.006	0.0001	0.001
Zinc	1.75 R	0.5 NH	100%	0.5	0.14	0.0006
PFAS – assumed 100% PFOS	0.00007 R	0.00002 H	100%	0.00002	Negligible	4.7×10^{-7}
Dioxins and furans, including dioxin-like PCBs assumed to be WHO_{05} TEQs	8.05×10^{-9} R	2.3×10^{-9} NH	100%	2.3×10^{-9}	0.03	0.81

Table B3: Summary of chronic TRVs adopted for chemicals – non-threshold effects

Chemical	Non-threshold inhalation TRV $(mg/m^3)^{-1}$	Non-threshold oral/dermal TRV $(mg/kg/day)^{-1}$	Dermal absorption*	Dermal permeability ** (cm/hr)
Benzene	0.006 W	NA (gaseous chemical)		
PAHs assuming 100% as BaP	0.6 U	0.233 N	0.06	0.71

Notes for Tables B2 and B3

* GI factor and dermal absorption values adopted from RAIS (accessed in 2023) (RAIS) noting that where values for dermal absorption from soil are not available a default of 0.0001 for inorganics has been adopted as per enHealth guidance (enHealth 2012a)

** Dermal permeability for water and dermal absorption from soil from USEPA RSLs chemical parameters (USEPA 2023)

= Dermal TRV = Oral TRV x GI absorption factor as per enHealth guidance

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 2012)

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

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

NH = Dioxin value (and background intakes, which includes natural soil) adopted from NHMRC (NHMRC 2002) and Environment Australia (DEH 2005; EPHC 2005), and other values consistent with that adopted by NHMRC to assess intakes in drinking water (NHMRC 2011 updated 2022)

N1 = Upper level of intake for the protection of adverse effects from selenium intakes (NHMRC 2006), and consistent with the approach adopted by WHO (WHO 2017). The inhalation value adopted is based on route extrapolation, however it is the same as the chronic inhalation value from OEHHA (OEHHA)

T = TRV available from TCEQ, relevant to chronic inhalation exposures (and HI=1) (TCEQ 2012, 2014, 2015b, 2015a)

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

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

All chronic TRVs adopted for the assessment of chronic exposures are protective of all adverse health effects (including respiratory, cardiovascular, developmental and reproductive effects) for all members of the community including sensitive groups such as children and the elderly.

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 2013c). Hence a threshold TRV has been adopted in the NEPM for the assessment of exposure to arsenic. In relation to inhalation exposures, the WHO indicates that a linear (non-threshold) dose–response relationship for lung cancer is supported by the occupational and epidemiological studies. It is difficult to mix threshold and non-threshold approaches, hence a threshold value for the assessment of chronic inhalation exposure to arsenic has been adopted based on the chronic air guideline developed by TCEQ (TCEQ 2012) that is protective of lung cancer effects (based on a non-threshold approach and adopting a 1 in 100,000 incremental risk consistent with the approach adopted in this assessment). This approach ensures all adverse effects are appropriately addressed and risks from multi-pathway exposures added.

Cadmium:

Inhalation of cadmium has been associated with carcinogenic effects (as well as other effects). Sufficient evidence is available (IARC 1993) to conclude that cadmium can produce lung cancers via inhalation (IARC 2012). While cadmium is thought to be potentially genotoxic, the weight of evidence is not clear. In addition, epidemiology studies associated with lung cancer have confounding issues that limit useful interpretation (WHO 2000b). It is noted that the USEPA derived their inhalation unit risk on the basis of the same study that the WHO dismissed due to confounding factors. Further, most of the epidemiological data available also include co-exposures with zinc and, in some cases, both zinc and lead.

Cadmium is not volatile and hence inhalation exposures are only relevant to dust intakes. These are not likely to be significant for soil contamination and hence the consideration of carcinogenic effects (where the mode of action is not clear) using a non-threshold approach is not considered appropriate. It is appropriate to consider intakes on the basis of a threshold approach associated with the most significant end-point. This is consistent with the approach noted by RIVM (2001) and considered by the WHO (2000) and UK EA (2009) where a threshold value for inhalation based on the protection of kidney toxicity (the most significant endpoint) has been considered. The value derived was then reviewed (based on the US cancer value) and considered to be adequately protective of lung cancer effects. On this basis, the WHO (2000) derived a guideline value of 0.005 µg/m^3 and the UK EA (2009) derived an inhalation TDI of 0.0014 µg/kg/day (which can be converted to a guideline value of 0.005 µg/m^3 – the same as the WHO value).

It is also noted that, where carcinogenic effects are evaluated using a non-threshold approach, the air guideline is higher (less conservative) than that calculated using a threshold (TCEQ 2016). The threshold TRV adopted in this assessment is lower than that evaluated by TCEQ (2016). Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.

Chromium VI:

The available data suggests chromium VI (Cr VI)may have some genotoxic potential, however, review by NEPC (NEPC 1999 amended 2013c) indicates that carcinogenicity is likely to act on the basis of a threshold mode of action, which has been adopted in the NEPM.

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

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

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

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

The threshold value from the USEPA has been adopted for the assessment of chronic inhalation exposures. This is considered protective of all adverse health effects.

Nickel:

The available data indicate that nickel 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 2013c). Hence the threshold TRV adopted is protective of all health effects including carcinogenicity.

Dioxins and furans (including dioxin-like PCBs), as 2,3,7,8-TCDD:

Reviews of carcinogenicity by NHMRC (NHMRC 2002) and the WHO (FAO/WHO 2018; WHO 2019) indicate 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.

B4 Background intakes

B4.1 General

The quantification of threshold risks requires consideration of background intakes, or intakes that are derived from existing sources, rather than those related to the project under investigation. This is particularly relevant to the assessment of chronic exposure to metals, PFAS and dioxin-like compounds. The presence of PAHs in emissions derived from the Project are assessed on the basis of a non-threshold risk calculation where background intakes are not included or relevant to the assessment approach (consistent with enHealth guidance).

It is important to recognise that everyone is exposed to metals in their daily lives. Metals are present in soil and can be taken up into food or washed off into waterways. They are naturally occurring and are widespread throughout our environment. There are some locations where particular metals are at elevated levels – mineral rich areas such as those where mining is likely to occur.

It is also important to note that many of the metals evaluated are essential elements for healthy lives, where health effects occur as a result of deficiency as well as where excessive may occur. For these metals (which include copper, chromium, nickel, selenium and zinc), background intakes (mainly from the diet) commonly comprise a significant proportion of the tolerable daily intake or an upper limit (based on health) to ensure recommended daily intakes or minimum intakes are maintained by populations for the protection of health.

Dioxin-like compounds are always produced whenever materials are burned – i.e. naturally occurring fires like bushfires. They are also in the emissions from wood heaters, gas stoves, cars, cigarettes, power stations and a wide range of other combustion sources. They can also be produced in chemical manufacturing and bleaching processes. Everyone is also exposed to these chemicals on a daily basis.

PFAS have been used in a range of products for many decades and hence, while these are man-made chemicals, these chemicals are present throughout the environment, particularly in urban and suburban areas.

When undertaking assessments like this, it is the difference between normal levels of exposure and exposure due to the project of interest that is the focus. This is why assessment of normal or background levels of exposure is an important part of the process so that difference can be determined.

Background intakes relate to existing exposures to key chemicals that are persistent in the environment and can be present in soil, water, air and food (dietary intakes), as a result of naturally occurring sources of metals, PFAS and dioxin-like compounds (mineralogy and combustion sources including bushfires, hazard reduction, agricultural burning and domestic fires).

The ASC NEPM includes an approach similar to that described below to determine relevant estimates for background intakes of metals, in particular, for use in contaminated land assessments. They are commonly used across Australia in contaminated land assessments and in assessments like this one for large projects. It is not common for regulators/governments to require a site-specific assessment of background exposures as these estimates developed within the ASC NEPM are normally considered to be sufficiently conservative. However, a site-specific assessment of background exposures has been undertaken in this assessment.

Background intakes of gaseous compounds, metals and dioxin-like compounds have been assessed separately as detailed below.

B4.2 Metals

The following provides further detail on how background intakes were estimated for this assessment.

Soil

Limited data are available in relation to background levels of the key chemicals in soil in the off-site community areas evaluated in this assessment.

JBS&G conducted an assessment for UrbanGrowth NSW of soil in relation to the Riverstone Scheduled Lands Precinct A redevelopment area [5]. This is land that was used for rural residential purpose, industrial workshops and open space parklands similar to existing used in the areas surrounding the Riverstone WRRF. Soil data from this area has therefore been considered to be representative of likely existing soil concentration in the area of this project. The average concentration reported from this data has been assumed representative of existing concentrations in the area.

For metals with no data from the previously discussed sources of information, typical background levels for these metals in soil from ATSDR or WHO reviews have been adopted, as referenced in **Table B4**.

[5] https://www.landcom.com.au/assets/Riverstone/riverstone-environmental-site-assessment-part-1.pdf

Background intakes of key metals in soil as a result of ingestion and dermal absorption have been calculated based on the concentrations listed in **Table B4** and adopting the same intake parameters detailed in **Table C2** (refer to **Section C3.1**). These calculations are included in **Section B5**. The calculated intakes are also provided in **Table B4**.

Table B4: Summary of intakes of metals from background/existing soil

Metals evaluated	Background levels in soil as average (min to max) (mg/kg)	Calculated background intakes – sum of ingestion and dermal absorption (mg/kg/day)	
		Young children	Adults
Arsenic	10 (<2 to 66)	0.000067	0.0000072
Cadmium	1 (<0.4 to 16)	0.0000067	0.00000072
Chromium (Cr VI assumed)	52 (7 to 2000)	--	--
Copper	35 (5 to 1000)	0.00023	0.000025
Lead	61 (6 to 760)	0.00041	0.000044
Nickel	14 (<5 to 140)	0.000093	0.000010
Selenium	0.2 [D]	0.0000013	0.00000014
Mercury (inorganic)	0.08 (<0.05 to 0.6)	0.00000053	0.000000058
Zinc	131 (8 to 1200)	0.000013	0.000099

Notes:

A = Average (min-max) value reported for existing soil for similar land use areas in Riverstone (as noted in text above table). It is also noted that no data is available for chromium VI in soil, with the data provided by JBS&G relevant to total chromium only. Unless there is a specific source of chromium VI, which is not the case for background soil in semi-rural areas, the form of chromium in soil would be chromium III which is significantly less toxic. Chromium VI is expected to be negligible, hence no background intakes of chromium VI from soil have been calculated

D = Values representative of general background levels of these metals in soil from international sources (as no data is available for the local region or rural areas in Australia). These values have been adopted for selenium (ATSDR 2003).

Water and dietary intakes

Food Standards Australia New Zealand (FSANZ) undertakes assessments of the population dietary intakes of a range of chemicals that may be present in food consumed by the population. This includes intakes from drinking/potable water. It is expected that soil in the agricultural areas surrounding the site are consistent with typical background levels that relate to where produce is grown in Australia. Hence dietary intakes of produce, included in the assessments undertaken by FSANZ are considered to represent background intakes in all produce from of the key chemicals evaluated in this assessment by members of the surrounding community. The data adopted in this study have been preferentially selected to adopt data from the most recent study, with data from other studies adopted in reverse date order.

Table B5 includes the background dietary intakes adopted from the most current studies conducted by FSANZ as well as the background intakes from drinking/potable water.

Table B5: Summary of intakes of metals from background water and dietary sources

Metals evaluated	Background dietary (including potable water) intakes (mg/kg/day)	
	Young children	Adults
Arsenic (inorganic)	0.00012 F1	0.000069 F1
Cadmium	0.0005 F1	0.0002 F1
Chromium (Cr VI assumed)	0.0005 F2	0.00016 F2
Copper	0.069 F2	0.02 F2
Lead	0.00038 F1	0.00016 F1
Nickel	0.0065 F3	0.002 F3
Selenium	0.0036 F2	0.0015 F2
Mercury (inorganic)	0.00034 F1	0.00014 F1
Zinc	0.3 F2	0.14 F2

Notes:

F1 = Data for dietary intakes from FSANZ 25th Australian Total Diet Survey (FSANZ 2019), with the upper bound value of the mean range adopted

F2 = Data for dietary intakes from FSANZ 23rd Australian Total Diet Survey (FSANZ 2011) with the mean (male/female combined) intake adopted (young children data are mean for ages 2-3 and 4-8 years), with intakes per unit body weight calculated based on body weight of child of 15 kg and adult as 70 kg. It is noted that the data presented by FSANZ for chromium relates to total chromium. Dietary intakes of total chromium may comprise a significant portion of the TDI for Cr VI. However, it is noted that the most common form of chromium in fresh produce is Cr III. Cr VI has been found to comprise approximately 10% of the total Cr intake from the diet ((Soares, Vieira & Bastos Mde 2010)) – this has been utilised in this assessment to estimate Cr VI intakes from the diet. In relation to manganese, FSANZ indicates that less than 5% of ingested manganese is absorbed by the body, hence the intake derived from the diet relevant to the assessment of health is 5% of that calculated in the FSANZ survey. Similarly, for cobalt, FSANZ indicates that absorption via the gut is around 50%, hence dietary intakes have been adjusted by 50%. The factor correcting for absorption has not been adopted for soil or water intakes making the calculations more conservative. In relation to zinc, FSANZ has evaluated these intakes in terms of the % of the tolerable daily intake (threshold relevant to zinc and health protection) and determined an average of 60% for young children and 28% for adults. These proportions from the diet, have been applied to the more conservative TRV adopted in this assessment. In relation to selenium, FSANZ has evaluated these intakes in terms of the % of the tolerable daily intake (threshold relevant to selenium and health protection) and determined an average of around 60% for young children and 25% for adults. These proportions from the diet are conservative based on data from EFSA and have been applied to the more conservative TRV adopted in this assessment.

F3 = Data for dietary intakes from FSANZ 22nd Australian Total Diet Survey (FSANZ 2008) with the upper bound mean (male/female combined) intake adopted (young children data are mean for ages 2-3 and 4-8 years), with intakes per unit body weight calculated based on body weight of child of 15 kg and adult as 70 kg

Air

For the metals, background intakes from air have been included in the air modelling, where data is available. For most metals, background levels have not been considered. Data collected by the NSW EPA (NSW DEC 2003) indicates that measured concentrations of arsenic, cadmium, copper, lead, nickel, selenium and zinc in air at Richmond are very low. Chromium and mercury were not detected.

Intakes of metals derived from air has been calculated based on the average concentration reported in air assuming a 70 kg individual inhales 20 m3 air per day. This intake is very low and has been assumed relevant to both adults and children. The calculated intake is included in Table B6.

Table B6: Intakes of metals from air (background/ambient)

Metals evaluated	Concentration in air - Average from Richmond (NSW DEC 2003) (ng/m^3)	Intake via inhalation (mg/kg/day)
Arsenic (inorganic)	0.4	0.00000011
Cadmium	0.06	0.000000017
Chromium (Cr VI assumed)	--	--
Copper	3.9	0.00000111
Lead	8.9	0.0000025
Nickel	5.2	0.0000015
Selenium	0.16	0.000000046
Mercury (inorganic)	--	--
Zinc	26	0.0000074

Intakes from all sources

Table B7 presents a summary of the total background intake for the off-site community from soil, diet/water and air. Total background intake as a proportion of the adopted threshold TRV are also presented in the table. These background intakes have been included in this assessment, for the quantification of risk.

Table B7: Summary of all background intakes and proportion of TRV

Key chemical	Background intakes (mg/kg/day)					
	Ingestion and dermal contact with soil	Intake from diet and water	Intakes from air	Total intake	TRV	%TRV
Young children						
Arsenic (inorganic)	0.000067	0.00012	0.00000011	0.00019	0.002	9%
Cadmium	0.0000067	0.0005	0.000000017	0.00051	0.0008	63%
Chromium (Cr VI assumed)	--	0.0005	--	0.00050	0.0009	56%
Copper	0.00023	0.069	0.00000111	0.069	0.14	49%
Lead	0.00041	0.00038	0.0000025	0.00079	0.0035	23%
Nickel	0.000093	0.0065	0.0000015	0.0066	0.012	55%
Selenium	0.0000013	0.0036	0.000000046	0.0036	0.006	60%
Mercury (inorganic)	0.00000053	0.00034	--	0.00034	0.0006	57%
Zinc	0.000013	0.3	0.0000074	0.30	0.5	60%
Adults						
Arsenic (inorganic)	0.0000072	0.000069	0.00000011	0.000076	0.002	4%
Cadmium	0.00000072	0.0002	0.000000017	0.00020	0.0008	25%
Chromium (Cr VI assumed)	--	0.00016	--	0.000160	0.0009	18%
Copper	0.000025	0.02	0.00000111	0.020	0.14	14%
Lead	0.000044	0.00016	0.0000025	0.00021	0.0035	6%
Nickel	0.000010	0.002	0.0000015	0.0020	0.012	17%
Selenium	0.00000014	0.0015	0.000000046	0.0015	0.006	25%
Mercury (inorganic)	0.000000058	0.00014	--	0.00014	0.0006	23%
Zinc	0.000099	0.14	0.0000074	0.14	0.5	28%

The above background intakes, as a %TRV have been adopted in this assessment.

B4.3 Dioxin-like compounds

Assessment of potential risks associated with dioxin-like compounds has adopted a threshold TRV from NHMRC (NHMRC 2002) that is based on the body burden of dioxin- like compounds. This threshold TRV is 70 pg/kg/month, which equates to 2.3 pg/kg/day as presented in **Table B3**.

Background intakes of dioxin-like compounds have been estimated for the Australian population based on data collected on levels in air, soil, water and the diet (DEH 2005). For the general population, over 95 % of exposure to dioxin-like compounds is through the diet, with foods of animal origin such as meat, dairy products and fish being the main sources. These intakes of dioxins into the human body are illustrated below.

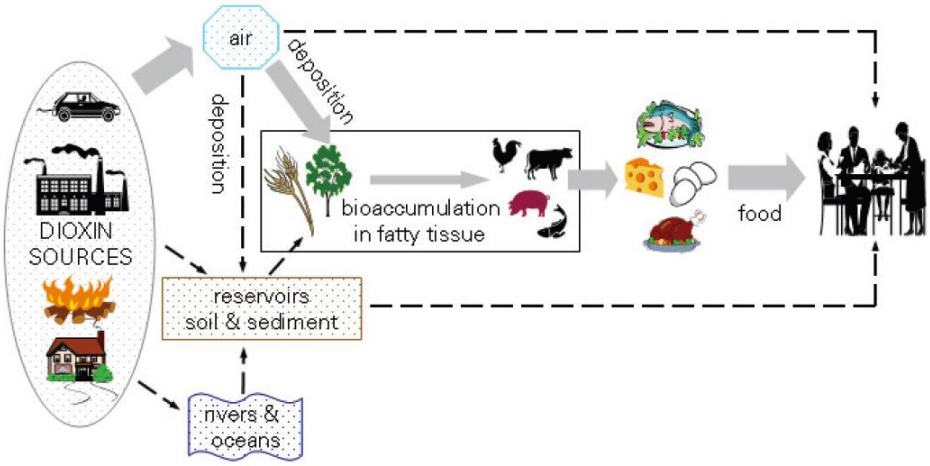

Figure B1: Pathway for dioxins entering our bodies (DEH 2004)

Based on the dietary study of dioxins, the intake of dioxins for the Australian population is lower than in most other countries. The risk assessment (DEH 2005) found that for Australians aged 2 years or older, the monthly intake of dioxins was between 3.9–15.8 pg TEQ/kg bw/month. These data would indicate background intakes from all these sources comprise between 5% and 23% of the TRV adopted (based on the TRV as a value per month). More recent dietary intakes of dioxin-like compounds by FSANZ (FSANZ 2020) indicates that mean intakes as a proportion of the tolerable monthly intake (same as adopted in this assessment) range from 9% to 25%, similar to the above.

Estimates of intake based on serum concentrations (DEH 2005; EPHC 2005), which relates to all intakes from all background sources, suggests that during approximately the last 25 years the average intake was probably close to 1.3 pg WHO-TEQ/kg bw/day. Where this intake is considered, this comprises around 54% of the adopted tolerable intake.

Intakes are lower in females than males for the same age, and decline with age in both sexes, the most rapid decline occurring after puberty. Infants and toddlers have the higher intakes. For this assessment, background intakes (all sources) are assumed to comprise 54% of the TRV adopted for both adults and children, adopting the higher estimate of intake detailed above. It is noted that this background intake is expected to be conservative for adults.

Background intakes for New Zealand populations have been estimated (MfE 2011) to be 10 pg/kg/month (i.e., 33% of the tolerable monthly intake adopted in New Zealand) based on the dietary intake of adult males, assumed to be also relevant to children.

B4.4 PFAS

There are a number of ways to determine background intakes of PFOS and PFOA in Australia.

FSANZ undertook a total diet survey for PFAS in food which was published in 2021 (FSANZ 2021). The findings indicated very low levels of these chemicals in food in Australia.

Testing of PFAS in blood has also been undertaken in Australia by researchers in Queensland. (Thompson et al. 2010; Toms et al. 2014) (Toms et al. 2019). It is noted that blood levels of PFAS are reflective of all intakes from consumer products, drinking water and the environment in general, hence no further specific evaluation of other potential sources of PFAS in the environment is required as part of this assessment. This is because PFOS accumulates in the blood serum, hence concentrations in the blood represent intakes from other sources in the environment. The findings of these studies indicate that an assumption that people in Australia are exposed to background levels of PFOS, in particular, at 10% of the TRV adopted by FSANZ, and also adopted in this assessment, is appropriate for use in risk assessment.

B4.5 Gaseous compounds

These chemicals are only assessed in relation to inhalation exposures.

Benzene

Background intakes of benzene relevant for urban and rural areas are based on inhalation exposure being the major contributor. Data collected in Sydney (NSW EPA 2004) for the period 1996 to 2001 reported a range of average concentrations that included 0.0074 mg/m^3 in Sydney CBD, 0.0035 mg/m^3 in Rozelle (inner city area) and 0.00128 mg/m^3 in western Sydney (St Marys). These concentrations comprise between 4% and 25% of the adopted TRV. Concentrations of benzene in other cities are noted to contribute 2.5% (in Perth) and 6% (Melbourne) of the TRV. It is noted that the CRC CARE (Friebel & Nadebaum 2011) derivation of HSLs adopted the maximum background level of 20% from the DEC (2004) study for Sydney CBD.

The data reported by DEC (2004) is dated and is not considered to reflect more current benzene emissions. Specifically, since 2006 the national cleaner fuel standards required that refineries reduce benzene levels in petrol from around four per cent to less than one per cent (Fuel Standard (Petrol) Determination 2001). This has resulted in lower levels of benzene in ambient air in all cities in Australia. Monitoring of benzene at 5 locations in Sydney in 2006, and 2 sites in 2008-2009 reported lower levels of benzene in the range 0.0006 to 0.0016 mg/m^3 (NSW EPA 2013) (consistent with the lowering of benzene content in fuel). These levels are more relevant to current levels of benzene in urban air and comprise up to 5% of the inhalation TRV. To be conservative a background intake of 10% of the TRV has been considered where the threshold TRVs are adopted.

Hydrogen sulfide

Limited data is available regarding environmental levels of hydrogen sulfide in Sydney. Environmental concentrations are reported in the various reviews noted above. WHO have reported

levels ranging from 0.00003 to 0.0004 ppm in air (WHO 2003b). Close to a sulfurous lake in New Zealand levels ranged from 0.2 to 5 mg/m^3 (WHO 2003b). ATSDR reported that concentrations range from 0.0001 to 0.0003 ppm in most locations away from geothermal sources and, in urban areas, are generally less than 0.001 ppm (0.002 mg/m^3) (ATSDR 2016).

If it assumed that the concentration in urban air in Sydney could be 0.001 ppm (0.002 mg/m^3) this would be 10% of the tolerable concentration adopted in this assessment, hence a background intake of 10% can be assumed, which is conservative.

Ammonia, hydrogen chloride and hydrogen fluoride

Background intakes for these gasses, from inhalation exposures, are not known as data is not available or the available data has not detected concentrations on ambient air, and hence for this assessment they have been assumed to be negligible.

B5 Background intake calculations

The following presents the intake calculations relating to ingestion and dermal contact with existing soil and inhalation of metals in ambient air, and the calculation of total background intakes.

Exposure to Chemicals via Incidental Ingestion of Soil - Existing soil

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

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

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Arsenic						10		7.1E-06	--			
Cadmium						1		7.1E-07	--			
Chromium (Cr VI assumed)						52		3.7E-05	--			
Copper						35		2.5E-05	--			
Lead						61		4.4E-05	--			
Nickel						14		1.0E-05	--			
Selenium						0.2		1.4E-07	--			
Mercury (as inorganic and e						0.08		5.7E-08	--			
Zinc						131		9.4E-05	--			

Exposure to Chemicals via Incidental Ingestion of Soil - Existing soil

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

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

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Arsenic						10		6.7E-05	--			
Cadmium						1		6.7E-06	--			
Chromium (Cr VI assumed)						52		3.5E-04	--			
Copper						35		2.3E-04	--			
Lead						61		4.1E-04	--			
Nickel						14		9.3E-05	--			
Selenium						0.2		1.3E-06	--			
Mercury (as inorganic and e						0.08		5.3E-07	--			
Zinc						2.5E-01		1.7E-06	--			

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)	
Arsenic					0.0001	10		4.5E-08	--			
Cadmium					0.0001	1		4.5E-09	--			
Chromium (Cr VI assumed)					0.0001	52		2.3E-07	--			
Copper					0.0001	35		1.6E-07	--			
Lead					0.0001	61		2.7E-07	--			
Nickel					0.0001	14		6.3E-08	--			
Selenium					0.0001	0.2		9.0E-10	--			
Mercury (as inorganic and elem					0.0001	0.08		3.6E-10	--			
Zinc					0.001	131		5.9E-06	--			

Dermal Exposure to Chemicals via Contact with Soil

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

Parameters Relevant to Quantification of Exposure by 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)	
Arsenic					0.0001	10		9.0E-08	--			
Cadmium					0.0001	1		9.0E-09	--			
Chromium (Cr VI assumed)					0.0001	52		4.7E-07	--			
Copper					0.0001	35		3.2E-07	--			
Lead					0.0001	61		5.5E-07	--			
Nickel					0.0001	14		1.3E-07	--			
Selenium					0.0001	0.2		1.8E-09	--			
Mercury (as inorganic and elem					0.0001	0.08		7.2E-10	--			
Zinc					0.001	131		1.2E-05	--			

Intakes from inhalation of metals in ambient air (data from NSW DEC 2003)

Key chemical	Ambient air concentration - Richmond		Intake via inhalation assuming 20 m^3 air inhaled per day and body weight of 70 kg
	(ng/m^3)	(mg/m^3)	(mg/kg/day)
Arsenic	0.4	0.00000040	0.00000011
Cadmium	0.06	0.000000060	0.000000017
Chromium (Cr VI assumed)			
Copper	3.9	0.0000039	0.00000111
Lead	8.9	0.0000089	0.0000025
Nickel	5.2	0.0000052	0.0000015
Selenium	0.16	0.00000016	0.000000046
Mercury (as inorganic and elemental)			
Zinc	26	0.000026	0.0000074

Calculation of total intake from existing/background sources

Key chemical	Children						Adults					
	soil	diet/water	air	total	TRV	%TRV	soil	diet/water	air	total	TRV	%TRV
Arsenic	6.7E-05	0.00012	1.1E-07	0.00019	0.002	9%	7.2E-06	0.000069	1.1E-07	0.000076	0.002	4%
Cadmium	6.7E-06	0.0005	1.7E-08	0.00051	0.0008	63%	7.2E-07	0.0002	1.7E-08	0.00020	0.0008	25%
Chromium (Cr VI assumed)		0.0005		0.00050	0.0009	56%		0.00016		0.00016	0.0009	18%
Copper	2.3E-04	0.069	1.1E-06	0.069	0.14	49%	2.5E-05	0.02	1.1E-06	0.020	0.14	14%
Lead	4.1E-04	0.00038	2.5E-06	0.00079	0.0035	23%	4.4E-05	0.00016	2.5E-06	0.00021	0.0035	6%
Nickel	9.3E-05	0.0065	1.5E-06	0.0066	0.012	55%	1.0E-05	0.002	1.5E-06	0.0020	0.012	17%
Selenium	1.3E-06	0.0036	4.6E-08	0.0036	0.006	60%	1.4E-07	0.0015	4.6E-08	0.0015	0.006	25%
Mercury (as inorganic and elemental)	5.3E-07	0.00034		0.00034	0.0006	57%	5.8E-08	0.00014		0.00014	0.0006	23%
Zinc	1.3E-05	0.3	7.4E-06	0.30	0.5	60%	9.9E-05	0.14	7.4E-06	0.14	0.5	28%

Appendix C Methodology and assumptions

C1 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.

C2 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 2013c; 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 C1** provides details on the assumptions adopted in this assessment:

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

Table C1: Inhalation exposure assumptions

Parameter		Value adopted	Basis
Ca	Concentration of chemical substance in air (mg/m^3)	Maximum from receptors modelled	Calculations undertaken on the basis of the maximum predicted impacts
FI	Fraction inhaled from site	100%	All exposures occur at the same location
RF	Dust lung retention factor (unitless)	Gasses = 1 Particulate bound chemicals = 0.375	100% of gases reach the lungs. For particulates, these assessed on the basis of the concentration bound to dust, assumed to be consistent with TSP
ET	Exposure time (dependant on activity) (hours/day)	Residents = 24 hours/day Workers = 8 hours/day Recreational = 4 hours/day	Residents: Assume someone is exposed at the maximum location all day, every day of the year. Workers: Working 8 hours per day, 5 days per week for 48 weeks of the year (enHealth 2012a) Recreational users: Assume to spend 4 hours in the recreational location, 2 days every week (conservative estimate)
EF	Exposure frequency (days/year)	Residents = 365 days/year Workers = 240 days/year Recreational = 104 days/year	
ED	Exposure duration (years)	Residents = 35 years Workers = 30 years Recreational = 35 years	Time at the same residence and workplace as per enHealth (enHealth 2012a). Residential value adopted for the assessment of recreational exposures
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

C3 Multiple pathway exposures

C3.1 Ingestion and dermal absorption

Chemical substances that are deposited on the ground have the potential to be ingested either directly through accidental consumption of dirt or indirectly through food grown or raised in the soil (fruit and vegetables, eggs) 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 C2**:

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

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

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

Table C2: Ingestion and dermal exposure assumptions

Parameter		Value adopted		Basis
		Young children	Adults	
C_M	Concentration of chemical substance in media or relevance (soil, fruit and vegetables, eggs) (mg/kg or mg/L)	Modelled based on deposition of particulates to soil, adopting the maximum from all sensitive receptors		Calculations undertaken on the basis of the maximum predicted impacts relevant to areas where multi-pathway exposures may occur
IR_M	Ingestion rate of media			
	Soil (mg/day)	100 mg/day	50 mg/day	Ingestion rate of outdoor soil and dust (tracked or deposited indoors) as per enHealth (enHealth 2012b)
	Fruit and vegetables (kg/day)	0.28 kg/day 85% from aboveground crops 16% from root crops	0.4 kg/day 73% from aboveground crops 27% from root crops	Total fruit and vegetable intakes per day as per ASC NEPM (NEPC 1999 amended 2013c)
	Eggs (kg/day)	0.006 kg/day	0.014 kg/day	Ingestion rate of eggs per day as per enHealth (enHealth 2012b)
FI	Fraction of media ingested derived from impacted media, or fraction of produce consumed each day derived from the property			

Parameter		Value adopted		Basis
		Young children	Adults	
	Soil	100%	100%	Assume all soil contact occurs on the one property
	Fruit and vegetables	35%	35%	Default of 35% for rural areas (NEPC 1999 amended 2013c) – adopted for semi-rural areas to be conservative
	Eggs	100%	100%	Assume all eggs and milk are 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)	2,700	6,300	Exposed skin surface area relevant to adults as per ASC NEPM (NEPC 1999 amended 2013c)
SA_W	Surface area of body exposed to water per day (cm^2/event)	7,000	20,000	Whole body area gets wet with bathing each day, values based on data from enHealth (2012)
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 2013c)
ABSd	Dermal absorption fraction for soil (unitless)	Chemical specific		Refer to **Tables B2 and B3**
CF	Conversion factor			
	Soil	$1x10^{-6}$ to convert mg to kg		Conversion of units relevant to soil ingestion and dermal contact
	Produce	1		No units conversion required for these calculations
BW	Body weight	15	70	As per enHealth (enHealth 2012b) and ASC NEPM (NEPC 1999 amended 2013c)
EF	Exposure frequency (days/year)	365	365	Assume residents exposed every day
ED	Exposure duration (years)	6	29	Duration of residency as per enHealth (enHealth 2012b) and ASC NEPM (NEPC 1999 amended 2013c)
AT	Averaging time (days)	Threshold = ED x 365 days/year Non-threshold = 70 years x 365 days/year		As per enHealth (enHealth 2012a) guidance

C3.2 Calculation of concentrations in various media

Potential Concentrations in Soil

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

The concentration in soil, which may be the result of deposition following emission of persistent chemical substances, can be calculated using the following equation from Stevens (1991), with assumptions adopted in this assessment presented in **Table C3**.

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

Table C3: Assumptions adopted to estimate soil concentrations

Parameter		Value adopted		Basis
		Surface soil*	Agricultural soil*	
DR	Particle deposition rate for accidental release (mg/m^2/year)	Modelled for the particulates emitted from the facility based on the deposition of TSP		Relevant to areas where multi-pathway exposures may occur
k	Chemical-specific soil-loss constant (1/year) = $ln(2)/T^{0.5}$	Calculated	Calculated	
$T^{0.5}$	Chemical half-life in soil (years)	Chemical specific	Chemical specific	Default values adopted for pollutants considered as per OEHHA (2015) with the value for dioxins from Lowe (Lowe, Dietrich & Alberts 1991)
t	Accumulation time (years)	35 years	35 years	Conservative estimation of time the facility will operate continuously
d	Soil mixing depth (m)	0.01 m	0.15 m	Default values (OEHHA 2015)
ρ	Soil bulk-density (g/m^3)	1600000	1600000	Default for fill material (CRC CARE 2011)
1000	Conversion from g to kg	Default conversion of units		

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

Homegrown fruit and vegetables

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

The potential concentration of persistent chemical substances that may be present within the plant following atmospheric deposition can be estimated using the following equation (Stevens 1991), with the parameters and assumptions adopted outlined in **Table C4**:

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

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

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

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

Parameter		Value adopted	Basis
DR	Particle deposition rate for accidental release (mg/m^2/day)	Modelled for the particulates emitted from the facility based on the deposition of TSP	Relevant to areas where multi-pathway exposures may occur
F	Fraction for the surface area of plant (unitless)	0.051	Relevant to aboveground exposed crops as per Stevens (1991) and OEHHA (OEHHA 2012)
k	Chemical-specific loss constant for particles on plants (1/days) = ln(2)/$T^{0.5}$	calculated	
$T^{0.5}$	Chemical half-life on plant (day)	14 days	Weathering of particulates on plant surfaces does occur and in the absence of measured data, it is generally assumed that organics deposited onto the outer portion of plant surfaces have a weathering half-life of 14 days (Stevens, 1991)
t	Deposition time or length of growing season (days)	70 days	Relevant to aboveground crops based on the value relevant to tomatoes, consistent with the value adopted by Stevens (1991)
Y	Crop yield (kg/m^2)	2 kg/m^2	Value for aboveground crops (OEHHA 2015)
Cs	Concentration of pollutant in soil (mg/kg)	Calculated value for agricultural soil	Calculated as described above and assumptions in **Table C3**
RUF for root crops	Root uptake factor (unitless)	Chemical specific value adopted	Root uptake factors from RAIS (RAIS) (soil to wet weight of plant)

Eggs

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

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

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

Table C5: Assumptions adopted to estimate concentration in animal produce

Parameter		Value adopted	Basis
FI	Fraction of grain/crop ingested by animals each day derived from the property (unitless)	100%	Assume pasture is grown on the property
IR_C	Ingestion rate of pasture/crops by each animal considered (kg/day)		
	Chickens	0.12	As per OEHHA (2015)
C	Concentration of chemical in crops consumed by animals (mg/kg)	Assume equal to that calculated in aboveground produce	Calculated as described above with assumptions in **Table C4**
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
Cs	Concentration of chemical in soil (mg/kg)	Calculated value for agricultural soil	Calculated as described above and assumptions in **Table C3**
B	Bioavailability of soil ingested (unitless)	100%	Conservative assumption
TF_P	Transfer factor for the produce of interest		
	Eggs	Chemical specific	Transfer factors adopted from OEHHA (2015), with the exception of chromium where the value was derived from an earlier OEHHA (OEHHA 2003) evaluation and cobalt where the uptake value from an Australian database has been used (MacLachlan 2011). Other values are the 95% value for the transfer of heavy metals into eggs (Leeman, Van Den Berg & Houben 2007).

All calculations relevant to the estimation of chemical concentrations in soil, fruit and vegetables and eggs are presented in **Appendix D**.

Rainwater tanks

The concentration in rainwater tanks depends on the deposition rate of dust, the size of the roof, the volume of rainfall each year and how much of the rain that falls onto the roof is captured in the tank. When dust is deposited onto a roof, some will be remobilised into air (wind) and blown off the roof before it can be washed into the tank. This has not been considered in this assessment.

In addition, health authorities[6] recommends the use of first flush devices to minimise the movement of accumulated dust, bird droppings and organic matter into the tank which can affect water quality (contamination and bacterial load). The use of a first flush device has not been considered in this assessment as it is unknown how many existing tanks use this device. For rainwater tanks used for drinking water purposes, it is expected that these would be maintained appropriately, in line with NSW Health and enHealth guidance (enHealth 2010), which includes the regular cleaning of tanks to remove accumulated sediments, maintaining roof materials, gutters and tank inlet, use of first flush devices and disinfection. The proper maintenance of rainwater tanks (specifically the cleaning out of sediments) would further reduce concentrations below those estimated in this assessment.

Based on mass balance modelling undertaken on rainwater tanks with first flush devices (Martinson & Thomas 2009) and measurements conducted in Australia (Kus et al. 2010), first flush devices can reduce concentrations in rainwater tanks by 90% or more. As noted above the use of a first flush devise has not been considered in this assessment.

The concentration in rainwater for project related emissions, which may be used for all household purposes is calculated as follows, where the parameters adopted for this assessment are detailed in **Table C6**:

$$C_W = \frac{DM}{VR}$$ as total mass in water (dissolved and particulate phase)

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

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

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

Table C6: Assumptions adopted to estimate concentration in rainwater tanks

Parameter		Value adopted	Basis
DM	Mass of dust deposited on the roof each year that would enter the tank (mg)	DR x Area x 1 year	Conservative assumption that 100% of the dust deposited on the roof for a full year, washes into the rainwater tank (i.e., there is no first flush device and no dust is blown of the roof before being washed into the tank)
DR	Particle deposition rate ($mg/m^2/year$)	Relevant to the maximum sensitive receptor (for deposition of chemicals attached to TSP)	Relevant to areas where multi-pathway exposures may occur
Area	Area of the roof (m^2)	200	Based on the average roof size for a 4-bedroom house in Australia (refer to Footnote 1)
VR	Volume of water collected from the roof each year (L)	calculated	Equation as above
R	Rainfall each year (mm)	843.7	Average rainfall reported at Seven Hills (data from 1950 to 2024) and Richmond RAAF (data from 1994 to 2024) met stations
Rc	Runoff coefficient	0.7	Assumes 30% loss in capture of water into the tank (Lizárraga-Mendiola et al. 2015)
1000	Conversion from m^3 to L Conversion from mm to m		
Kd	Soil-water partition coefficient (cm^3/g)	Chemical-specific	All values for metals from RAIS (RAIS). For organics Kd has been calculated as Kd = Koc x Foc. Koc values obtained from RAIS (for BaP), PubChem (for dioxins) and ITRC (for PFOS – average value adopted). Foc (fraction of organic carbon) assumed to be 1%.
ρ	Soil bulk density (g/cm^3)	0.5	Assumed for loose deposited dust on roof (upper end measured for powders)

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

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

Dam water concentrations

The concentrations in a water body have been calculated on the basis of the following equation, which provides a total concentration in water (dissolved and particulate phase):

Cw, total = DR x Area x t/v (mg/L)

DR = Particle deposition rate (mg/m^2/year)

Area = Area of water body (m^2)

t = Accumulation time (years)

v = Volume of water body for deposited mass to mix (L)

For this assessment a default water body has been adopted, which is 1 Ha in area and 0.15 m deep (EPHC 2009). The accumulation time adopted is 1 year, which is highly conservative as the water bodies surrounding the site comprise Eastern Creek (which is flowing most of the time) and some smaller dams that would be expected to be flushed during rainfall events.

The dissolved phase concentration that may be present in a dam is calculated as follows:

$$C_{W,\ dissolved} = \frac{C_{w,\ total}}{Kd \times \rho}$$

With the Kd and bulk density (ρ) as per **Table C6**.

Appendix D Risk calculations

Data from air modelling

					Incremental Results																
					Industrial/ commercial	Water course	Residential	Residential	Residential	Education (early learning)	Residential	Residential	Water course	Water course	Recreation	Service	Industrial/ commercial	Industrial/ commercial	Industrial/ commercial	Industrial/ commercial	
Model	Pollutant	Output (units)	Averaging Peric	Percentile mode	R1	R2	R3	R4	R5	R6	R7	R8	R9	R10	R11	R12	R13	R14	R15	R16	MAX
Impact Assessment Pollutants	CO	Concentration (ug/m3)	15-Minutes	100th Percentile	1.07E-05	9.04E-05	7.61E-05	4.04E-04	2.72E-04	3.14E-04	2.38E-04	2.26E-04	1.74E-04	7.66E-05	8.25E-05	1.30E-04	3.18E-04	4.41E-04	5.71E-04	7.32E-04	N/A
	CO	Concentration (ug/m3)	1-Hour	100th Percentile	4.19E+00	2.82E+00	1.10E+01	1.32E+01	1.31E+01	1.07E+01	1.35E+01	9.72E+00	3.55E+00	2.48E+00	4.13E+00	1.30E+01	1.62E+01	2.02E+01	2.08E+01	1.61E+01	N/A
	CO	Concentration (ug/m3)	8-Hour	100th Percentile	2.91E+00	1.52E+00	7.27E+00	5.60E+00	1.03E+01	9.00E+00	6.40E+00	8.87E+00	1.61E+00	9.54E-01	2.71E+00	1.11E+01	1.38E+01	1.93E+01	1.44E+01	1.22E+01	N/A
	CO	Concentration (ug/m3)	Annual	100th Percentile	5.91E-02	7.87E-02	2.05E-01	1.03E-01	6.34E-01	5.68E-01	1.16E-01	3.55E-01	6.60E-02	5.54E-02	2.14E-01	7.71E-01	9.33E-01	1.30E+00	3.54E-01	4.32E-01	N/A
	DUST	Deposition (g/m2/month)	Annual	100th Percentile	5.15E-06	1.01E-05	2.92E-05	1.12E-05	6.07E-05	4.28E-05	1.01E-05	3.13E-05	4.92E-06	4.27E-06	1.81E-05	6.14E-05	8.00E-05	1.56E-04	5.49E-05	4.71E-05	N/A
	HF	Concentration (ug/m3)	07-Days	100th Percentile	7.67E-04	9.00E-04	4.46E-03	7.59E-04	4.98E-03	3.61E-03	7.40E-04	3.53E-03	6.30E-04	4.06E-04	1.52E-03	4.92E-03	6.15E-03	1.20E-02	3.59E-03	5.18E-03	N/A
	HF	Concentration (ug/m3)	24-Hour	100th Percentile	3.63E-03	2.78E-03	1.17E-02	4.46E-03	1.27E-02	1.01E-02	5.43E-03	9.94E-03	2.23E-03	1.50E-03	4.92E-03	1.31E-02	1.37E-02	3.21E-02	1.64E-02	2.10E-02	N/A
	HF	Concentration (ug/m3)	30-Days	100th Percentile	3.70E-04	7.16E-04	2.18E-03	6.03E-04	4.45E-03	2.33E-03	5.07E-04	2.29E-03	4.42E-04	3.16E-04	1.30E-03	3.17E-03	3.84E-03	1.05E-02	2.53E-03	3.28E-03	N/A
	HF	Concentration (ug/m3)	90-Days	100th Percentile	2.95E-04	5.01E-04	1.08E-03	4.10E-04	2.67E-03	1.96E-03	3.50E-04	1.88E-03	3.48E-04	2.38E-04	8.32E-04	2.66E-03	2.69E-03	7.15E-03	1.73E-03	2.23E-03	N/A
	HF	Concentration (ug/m3)	Annual	100th Percentile	1.86E-04	3.59E-04	5.69E-04	2.66E-04	1.61E-03	1.31E-03	2.52E-04	1.12E-03	2.31E-04	2.00E-04	7.51E-04	1.83E-03	2.33E-03	4.47E-03	1.19E-03	1.61E-03	N/A
	NO2 (see notes)	Concentration (ug/m3)	1-Hour	100th Percentile	6.97E+01	6.97E+01	6.97E+01	6.97E+01	7.22E+01	7.26E+01	7.15E+01	7.00E+01	6.97E+01	6.97E+01	6.97E+01	7.29E+01	7.29E+01	7.28E+01	7.31E+01	7.29E+01	N/A
	NO2 (see notes)	Concentration (ug/m3)	Annual	100th Percentile	1.02E+01	1.02E+01	1.04E+01	1.02E+01	1.11E+01	1.10E+01	1.03E+01	1.07E+01	1.02E+01	1.02E+01	1.05E+01	1.13E+01	1.16E+01	1.23E+01	1.07E+01	1.09E+01	N/A
	PB	Concentration (ug/m3)	1-Hour	100th Percentile	7.69E-04	5.54E-04	1.58E-03	1.30E-03	1.44E-03	1.30E-03	1.36E-03	1.75E-03	6.97E-04	5.09E-04	7.95E-04	1.64E-03	2.04E-03	3.13E-03	3.68E-03	3.56E-03	N/A
	PB	Concentration (ug/m3)	Annual	100th Percentile	1.19E-05	2.26E-05	3.72E-05	1.72E-05	1.06E-04	8.60E-05	1.65E-05	7.35E-05	1.44E-05	1.23E-05	4.74E-05	1.21E-04	1.54E-04	2.92E-04	7.78E-05	1.05E-04	N/A
	PB	Deposition (g/m2/month)	Annual	100th Percentile	1.64E-07	3.63E-07	1.00E-06	3.83E-07	2.12E-06	1.39E-06	2.81E-07	1.19E-06	2.20E-07	2.00E-07	7.32E-07	2.16E-06	3.07E-06	7.10E-06	2.24E-06	2.07E-06	N/A
	PM10	Concentration (ug/m3)	24-Hour	100th Percentile	4.08E-01	3.08E-01	2.53E+00	6.95E-01	1.91E+00	1.44E+00	7.08E-01	1.27E+00	1.96E-01	1.34E-01	4.64E-01	1.82E+00	2.14E+00	3.95E+00	2.45E+00	2.10E+00	N/A
	PM10	Concentration (ug/m3)	Annual	100th Percentile	2.39E-02	4.14E-02	8.96E-02	3.99E-02	2.32E-01	1.85E-01	4.28E-02	1.34E-01	2.05E-02	1.58E-02	7.31E-02	2.54E-01	3.17E-01	5.34E-01	1.49E-01	1.80E-01	N/A
	PM2.5	Concentration (ug/m3)	24-Hour	100th Percentile	2.63E-01	1.95E-01	1.61E+00	4.69E-01	1.24E+00	9.91E-01	4.79E-01	8.58E-01	1.28E-01	8.72E-02	3.08E-01	1.25E+00	1.45E+00	2.58E+00	1.56E+00	1.38E+00	N/A
	PM2.5	Concentration (ug/m3)	Annual	100th Percentile	1.55E-02	2.60E-02	5.78E-02	2.62E-02	1.53E-01	1.24E-01	2.84E-02	8.77E-02	1.38E-02	1.07E-02	4.84E-02	1.71E-01	2.12E-01	3.46E-01	9.65E-02	1.16E-01	N/A
	SO2	Concentration (ug/m3)	1-Hour	100th Percentile	3.58E+00	2.64E+00	7.26E+00	5.91E+00	6.60E+00	5.96E+00	6.27E+00	8.19E+00	3.33E+00	2.48E+00	3.71E+00	7.52E+00	9.35E+00	1.49E+01	1.70E+01	1.65E+01	N/A
	SO2	Concentration (ug/m3)	24-Hour	100th Percentile	1.10E+00	8.37E-01	3.55E+00	1.35E+00	3.86E+00	3.06E+00	1.65E+00	2.99E+00	6.74E-01	4.51E-01	1.48E+00	3.96E+00	4.16E+00	9.69E+00	4.97E+00	6.35E+00	N/A
	SO2	Concentration (ug/m3)	Annual	100th Percentile	5.63E-02	1.08E-01	1.72E-01	8.06E-02	4.90E-01	3.98E-01	7.66E-02	3.40E-01	6.99E-02	6.05E-02	2.27E-01	5.57E-01	7.06E-01	1.35E+00	3.60E-01	4.86E-01	N/A
	TSP	Concentration (ug/m3)	Annual	100th Percentile	3.42E-02	6.05E-02	1.27E-01	5.63E-02	3.27E-01	2.58E-01	5.98E-02	1.91E-01	2.96E-02	2.29E-02	1.05E-01	3.55E-01	4.45E-01	7.67E-01	2.14E-01	2.60E-01	N/A
Odour	ODOR (see notes)	Concentration (OU)	1-Hour	99th Percentile	3.10E-01	2.25E-01	1.11E+00	6.88E-01	1.33E+00	1.09E+00	6.62E-01	1.06E+00	2.07E-01	1.83E-01	3.93E-01	1.36E+00	1.53E+00	2.10E+00	1.58E+00	2.04E+00	

					Cumulative Results															
					Industrial/ commercial	Water course	Residential	Residential	Residential	Education (early learning)	Residential	Residential	Water course	Water course	Recreation	Service	Industrial/ commercial	Industrial/ commercial	Industrial/ commercial	Industrial/ commercial
Model	Pollutant	Output (units)	Averaging Peri	Percentile mode	R1	R2	R3	R4	R5	R6	R7	R8	R9	R10	R11	R12	R13	R14	R15	R16
Impact Assessment Pollutants	CO	Concentration (ug/m3)	15-Minutes	100th Percentile	2474.08	2474.08	2474.08	2474.08	2474.08	2474.08	2474.08	2474.08	2474.08	2474.08	2474.08	2474.08	2474.08	2474.08	2474.08	2474.08
	CO	Concentration (ug/m3)	1-Hour	100th Percentile	1879.19	1877.82	1886.00	1888.19	1888.11	1885.66	1888.54	1884.72	1878.55	1877.48	1879.13	1887.99	1891.15	1895.19	1895.75	1891.09
	CO	Concentration (ug/m3)	8-Hour	100th Percentile	1877.91	1876.52	1882.27	1880.60	1885.31	1884.00	1881.40	1883.87	1876.61	1875.95	1877.71	1886.13	1888.78	1894.28	1889.45	1887.21
	CO	Concentration (ug/m3)	Annual	100th Percentile	0.06	0.08	0.20	0.10	0.63	0.57	0.12	0.35	0.07	0.06	0.21	0.77	0.93	1.30	0.35	0.43
	DUST	Deposition (g/m2/month)	Annual	100th Percentile	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	HF	Concentration (ug/m3)	07-Days	100th Percentile	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.01	0.00	0.01
	HF	Concentration (ug/m3)	24-Hour	100th Percentile	0.00	0.00	0.01	0.00	0.01	0.01	0.01	0.01	0.00	0.00	0.00	0.01	0.01	0.03	0.02	0.02
	HF	Concentration (ug/m3)	30-Days	100th Percentile	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.00	0.00
	HF	Concentration (ug/m3)	90-Days	100th Percentile	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.01	0.00	0.00
	HF	Concentration (ug/m3)	Annual	100th Percentile	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	NO2 (see notes)	Concentration (ug/m3)	1-Hour	100th Percentile	139.41	139.41	139.41	139.41	141.90	142.35	139.41	139.66	139.41	139.41	139.41	142.56	142.60	142.54	139.41	139.41
	NO2 (see notes)	Concentration (ug/m3)	Annual	100th Percentile	20.25	20.31	20.48	20.31	21.18	21.04	20.33	20.78	20.25	20.24	20.55	21.37	21.66	22.40	20.77	20.95
	PB	Concentration (ug/m3)	1-Hour	100th Percentile	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	PB	Concentration (ug/m3)	Annual	100th Percentile	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00	0.00
	PB	Deposition (g/m2/month)	Annual	100th Percentile																
	PM10	Concentration (ug/m3)	24-Hour	100th Percentile	51.97	51.87	54.09	52.25	53.46	53.00	52.27	52.83	51.75	51.69	52.02	53.38	53.70	55.51	54.01	53.66
	PM10	Concentration (ug/m3)	Annual	100th Percentile	15.40	15.42	15.46	15.41	15.61	15.56	15.42	15.51	15.39	15.39	15.45	15.63	15.69	15.91	15.52	15.55
	PM2.5	Concentration (ug/m3)	24-Hour	100th Percentile	40.78	40.71	42.13	40.98	41.75	41.50	40.99	41.37	40.64	40.60	40.82	41.77	41.97	43.09	42.07	41.89
	PM2.5	Concentration (ug/m3)	Annual	100th Percentile	6.68	6.69	6.72	6.69	6.81	6.79	6.69	6.75	6.68	6.67	6.71	6.83	6.87	7.01	6.76	6.78
	SO2	Concentration (ug/m3)	1-Hour	100th Percentile	77.98	77.04	81.66	80.31	81.00	80.36	80.67	82.59	77.73	76.88	78.11	81.92	83.75	89.34	91.38	90.94
	SO2	Concentration (ug/m3)	24-Hour	100th Percentile	18.80	18.54	21.25	19.05	21.56	20.76	19.35	20.69	18.37	18.15	19.18	21.66	21.86	27.39	22.67	24.05
	SO2	Concentration (ug/m3)	Annual	100th Percentile	0.06	0.11	0.17	0.08	0.49	0.40	0.08	0.34	0.07	0.06	0.23	0.56	0.71	1.35	0.36	0.49
	TSP	Concentration (ug/m3)	Annual	100th Percentile	30.78	30.81	30.87	30.80	31.07	31.01	30.81	30.94	30.78	30.77	30.85	31.10	31.19	31.51	30.96	31.01

					Incremental Results																
					Industrial/ commercial	Water course	Residential	Residential	Residential	Education (early learning)	Residential	Residential	Water course	Water course	Recreation	Service	Industrial/ commercial	Industrial/ commercial	Industrial/ commercial	Industrial/ commercial	
Model	Pollutant	Output (units)	Averaging Peric	Percentile mode	R1	R2	R3	R4	R5	R6	R7	R8	R9	R10	R11	R12	R13	R14	R15	R16	MAX
Toxic Air Pollutants	AS	Concentration (ug/m3)	1-Hour	99.9th Percentile	8.61E-05	5.45E-05	1.82E-04	1.47E-04	1.68E-04	1.53E-04	1.36E-04	1.93E-04	6.25E-05	5.20E-05	8.35E-05	1.88E-04	2.38E-04	3.21E-04	4.07E-04	3.89E-04	0.0014
	AS	Concentration (ug/m3)	Annual	100th Percentile	1.40E-06	2.59E-06	4.46E-06	2.07E-06	1.27E-05	1.04E-05	2.02E-06	8.62E-06	1.67E-06	1.42E-06	5.48E-06	1.45E-05	1.84E-05	3.42E-05	9.11E-06	1.22E-05	0.0001
	AS	Deposition (g/m2/month)	Annual	100th Percentile	1.96E-08	4.31E-08	1.21E-07	4.62E-08	2.55E-07	1.69E-07	3.44E-08	1.43E-07	2.61E-08	2.38E-08	8.75E-08	2.62E-07	3.72E-07	8.39E-07	2.69E-07	2.44E-07	
	CD	Concentration (ug/m3)	1-Hour	99.9th Percentile	1.13E-03	7.42E-04	2.36E-03	1.91E-03	2.15E-03	1.96E-03	1.75E-03	2.56E-03	8.65E-04	7.25E-04	1.11E-03	2.43E-03	3.09E-03	4.26E-03	5.53E-03	5.27E-03	0.0195
	CD	Concentration (ug/m3)	Annual	100th Percentile	1.82E-05	3.45E-05	5.67E-05	2.64E-05	1.62E-04	1.32E-04	2.53E-05	1.13E-04	2.23E-05	1.91E-05	7.30E-05	1.86E-04	2.36E-04	4.49E-04	1.19E-04	1.61E-04	0.0009
	CD	Deposition (g/m2/month)	Annual	100th Percentile	2.50E-07	5.52E-07	1.52E-06	5.81E-07	3.24E-06	2.13E-06	4.26E-07	1.82E-06	3.37E-07	3.07E-07	1.12E-06	3.31E-06	4.69E-06	1.09E-05	3.40E-06	3.15E-06	
	CR	Concentration (ug/m3)	1-Hour	99.9th Percentile	1.24E-03	8.04E-04	2.61E-03	2.12E-03	2.40E-03	2.18E-03	1.93E-03	2.82E-03	9.31E-04	7.80E-04	1.23E-03	2.69E-03	3.42E-03	4.71E-03	6.04E-03	5.71E-03	0.0211
	CR	Concentration (ug/m3)	Annual	100th Percentile	2.02E-05	3.82E-05	6.37E-05	2.95E-05	1.81E-04	1.47E-04	2.85E-05	1.25E-04	2.45E-05	2.09E-05	8.03E-05	2.07E-04	2.63E-04	4.97E-04	1.32E-04	1.78E-04	0.0010
	CR	Deposition (g/m2/month)	Annual	100th Percentile	2.80E-07	6.20E-07	1.72E-06	6.56E-07	3.64E-06	2.39E-06	4.84E-07	2.04E-06	3.75E-07	3.42E-07	1.25E-06	3.71E-06	5.26E-06	1.21E-05	3.84E-06	3.53E-06	
	CU	Concentration (ug/m3)	1-Hour	99.9th Percentile	9.92E-04	6.62E-04	2.66E-03	1.95E-03	2.10E-03	1.93E-03	1.72E-03	2.12E-03	5.44E-04	4.16E-04	9.89E-04	2.36E-03	3.01E-03	3.76E-03	4.66E-03	4.12E-03	0.0113
	CU	Concentration (ug/m3)	Annual	100th Percentile	1.75E-05	3.24E-05	6.27E-05	2.76E-05	1.63E-04	1.28E-04	2.83E-05	1.02E-04	1.69E-05	1.34E-05	5.87E-05	1.78E-04	2.26E-04	4.14E-04	1.14E-04	1.44E-04	0.0008
	CU	Deposition (g/m2/month)	Annual	100th Percentile	2.79E-07	6.22E-07	1.92E-06	6.91E-07	3.59E-06	2.30E-06	5.41E-07	1.92E-06	3.36E-07	3.02E-07	1.13E-06	3.53E-06	4.98E-06	1.09E-05	4.12E-06	3.35E-06	
	H2S	Concentration (ug/m3)	1-Hour	99.9th Percentile	5.75E-01	4.53E-01	2.36E+00	1.32E+00	1.45E+00	1.25E+00	1.20E+00	1.23E+00	3.80E-01	2.87E-01	6.13E-01	1.58E+00	1.93E+00	2.46E+00	2.72E+00	2.27E+00	5.7583
	H2S	Concentration (ug/m3)	Annual	100th Percentile	1.09E-02	1.99E-02	4.39E-02	1.86E-02	1.05E-01	7.94E-02	2.00E-02	5.90E-02	7.54E-03	5.23E-03	2.91E-02	1.10E-01	1.38E-01	2.44E-01	6.96E-02	8.13E-02	0.4580
	H2S	Deposition (g/m2/month)	Annual	100th Percentile	2.77E-05	5.55E-05	1.29E-04	5.16E-05	2.75E-04	2.01E-04	5.15E-05	1.58E-04	2.28E-05	1.78E-05	8.41E-05	2.86E-04	3.77E-04	6.86E-04	2.32E-04	2.41E-04	
	HCL	Concentration (ug/m3)	1-Hour	99.9th Percentile	1.13E-01	7.62E-02	2.31E-01	1.88E-01	2.10E-01	1.91E-01	1.71E-01	2.56E-01	8.70E-02	7.43E-02	1.13E-01	2.38E-01	3.02E-01	4.23E-01	5.51E-01	5.20E-01	1.9303
	HCL	Concentration (ug/m3)	Annual	100th Percentile	1.86E-03	3.59E-03	5.69E-03	2.66E-03	1.61E-02	1.31E-02	2.52E-03	1.12E-02	2.31E-03	2.00E-03	7.51E-03	1.83E-02	2.33E-02	4.47E-02	1.19E-02	1.61E-02	0.0901
	HCL	Deposition (g/m2/month)	Annual	100th Percentile	4.33E-06	8.87E-06	1.55E-05	6.86E-06	3.98E-05	3.11E-05	6.14E-06	2.71E-05	5.54E-06	4.91E-06	1.82E-05	4.48E-05	5.92E-05	1.18E-04	3.38E-05	4.19E-05	
	HG	Concentration (ug/m3)	1-Hour	99.9th Percentile	1.11E-03	7.41E-04	2.30E-03	1.87E-03	2.10E-03	1.90E-03	1.71E-03	2.55E-03	8.52E-04	7.19E-04	1.10E-03	2.37E-03	3.01E-03	4.21E-03	5.49E-03	5.19E-03	0.0193
	HG	Concentration (ug/m3)	Annual	100th Percentile	1.78E-05	3.41E-05	5.54E-05	2.57E-05	1.58E-04	1.28E-04	2.44E-05	1.11E-04	2.20E-05	1.88E-05	7.19E-05	1.80E-04	2.30E-04	4.41E-04	1.17E-04	1.58E-04	0.0009
	HG	Deposition (g/m2/month)	Annual	100th Percentile	2.44E-07	5.41E-07	1.48E-06	5.67E-07	3.16E-06	2.06E-06	4.13E-07	1.78E-06	3.29E-07	3.00E-07	1.09E-06	3.20E-06	4.55E-06	1.06E-05	3.32E-06	3.10E-06	
	NH3	Concentration (ug/m3)	1-Hour	99.9th Percentile	4.54E-01	3.58E-01	1.88E+00	1.04E+00	1.15E+00	9.84E-01	9.48E-01	9.66E-01	3.01E-01	2.28E-01	4.82E-01	1.25E+00	1.52E+00	1.93E+00	2.12E+00	1.77E+00	4.5931
	NH3	Concentration (ug/m3)	Annual	100th Percentile	8.56E-03	1.56E-02	3.46E-02	1.46E-02	8.27E-02	6.22E-02	1.58E-02	4.61E-02	5.81E-03	3.99E-03	2.26E-02	8.59E-02	1.08E-01	1.91E-01	5.45E-02	6.35E-02	0.3577
	NH3	Deposition (g/m2/month)	Annual	100th Percentile	2.18E-05	4.35E-05	1.02E-04	4.06E-05	2.16E-04	1.58E-04	4.06E-05	1.24E-04	1.77E-05	1.38E-05	6.55E-05	2.24E-04	2.96E-04	5.38E-04	1.83E-04	1.89E-04	
	NI	Concentration (ug/m3)	1-Hour	99.9th Percentile	1.07E-03	6.83E-04	2.26E-03	1.82E-03	2.08E-03	1.90E-03	1.68E-03	2.42E-03	7.93E-04	6.61E-04	1.05E-03	2.34E-03	2.96E-03	4.02E-03	5.13E-03	4.89E-03	0.0179
	NI	Concentration (ug/m3)	Annual	100th Percentile	1.74E-05	3.25E-05	5.51E-05	2.56E-05	1.57E-04	1.28E-04	2.49E-05	1.07E-04	2.10E-05	1.79E-05	6.87E-05	1.80E-04	2.28E-04	4.26E-04	1.14E-04	1.52E-04	0.0009
	NI	Deposition (g/m2/month)	Annual	100th Percentile	2.42E-07	5.34E-07	1.49E-06	5.69E-07	3.15E-06	2.08E-06	4.22E-07	1.77E-06	3.24E-07	2.96E-07	1.08E-06	3.24E-06	4.59E-06	1.04E-05	3.32E-06	3.03E-06	
	PAH	Concentration (ug/m3)	1-Hour	99.9th Percentile	6.79E-05	4.57E-05	1.39E-04	1.13E-04	1.26E-04	1.14E-04	1.03E-04	1.54E-04	5.22E-05	4.46E-05	6.76E-05	1.43E-04	1.81E-04	2.54E-04	3.31E-04	3.12E-04	0.0012
	PAH	Concentration (ug/m3)	Annual	100th Percentile	1.12E-06	2.16E-06	3.41E-06	1.59E-06	9.69E-06	7.85E-06	1.51E-06	6.75E-06	1.39E-06	1.20E-06	4.51E-06	1.10E-05	1.40E-05	2.68E-05	7.14E-06	9.66E-06	0.0001
	PAH	Deposition (g/m2/month)	Annual	100th Percentile	2.60E-09	5.32E-09	9.28E-09	4.12E-09	2.39E-08	1.87E-08	3.69E-09	1.63E-08	3.32E-09	2.95E-09	1.09E-08	2.69E-08	3.55E-08	7.10E-08	2.03E-08	2.51E-08	
	PFAS	Concentration (ug/m3)	1-Hour	99.9th Percentile	2.45E-05	1.89E-05	9.60E-05	5.54E-05	6.09E-05	5.25E-05	4.98E-05	5.17E-05	1.58E-05	1.18E-05	2.62E-05	6.65E-05	8.19E-05	1.03E-04	1.16E-04	9.60E-05	0.0002
	PFAS	Concentration (ug/m3)	Annual	100th Percentile	4.65E-07	8.52E-07	1.84E-06	7.85E-07	4.46E-06	3.37E-06	8.38E-07	2.53E-06	3.37E-07	2.40E-07	1.28E-06	4.66E-06	5.88E-06	1.04E-05	2.96E-06	3.50E-06	0.00002
	PFAS	Deposition (g/m2/month)	Annual	100th Percentile	1.18E-09	2.35E-09	5.41E-09	2.17E-09	1.16E-08	8.52E-09	2.15E-09	6.75E-09	9.95E-10	7.87E-10	3.64E-09	1.21E-08	1.60E-08	2.92E-08	9.81E-09	1.03E-08	
	SE	Concentration (ug/m3)	1-Hour	99.9th Percentile	3.64E-04	2.41E-04	7.51E-04	6.27E-04	6.97E-04	6.31E-04	5.56E-04	8.25E-04	2.66E-04	2.25E-04	3.69E-04	7.87E-04	1.01E-03	1.40E-03	1.79E-03	1.68E-03	0.0061
	SE	Concentration (ug/m3)	Annual	100th Percentile	5.97E-06	1.14E-05	1.89E-05	8.69E-06	5.32E-05	4.29E-05	8.35E-06	3.67E-05	7.13E-06	6.07E-06	2.35E-05	6.03E-05	7.68E-05	1.47E-04	3.91E-05	5.26E-05	0.0003
	SE	Deposition (g/m2/month)	Annual	100th Percentile	8.35E-08	1.86E-07	5.18E-07	1.96E-07	1.08E-06	7.00E-07	1.44E-07	6.02E-07	1.11E-07	1.01E-07	3.68E-07	1.09E-06	1.54E-06	3.59E-06	1.15E-06	1.05E-06	
	Dioxins and furans (TEQ)	Concentration (ug/m3)	1-Hour	99.9th Percentile	1.23E-09	7.61E-10	2.67E-09	2.11E-09	2.46E-09	2.23E-09	2.08E-09	2.73E-09	9.11E-10	7.52E-10	1.14E-09	2.74E-09	3.41E-09	4.54E-09	5.69E-09	5.58E-09	2.02E-08
	Dioxins and furans (TEQ)	Concentration (ug/m3)	Annual	100th Percentile	1.97E-11	3.58E-11	6.24E-11	2.93E-11	1.81E-10	1.50E-10	2.89E-11	1.22E-10	2.39E-11	2.04E-11	7.80E-11	2.09E-10	2.64E-10	4.81E-10	1.28E-10	1.71E-10	9.59E-10
	Dioxins and furans (TEQ)	Deposition (g/m2/month)	Annual	100th Percentile	2.73E-13	5.94E-13	1.66E-12	6.42E-13	3.58E-12	2.41E-12	4.79E-13	2.02E-12	3.69E-13	3.37E-13	1.24E-12	3.75E-12	5.31E-12	1.18E-11	3.71E-12	3.37E-12	
	VOC	Concentration (ug/m3)	1-Hour	99.9th Percentile	2.27E+00	1.79E+00	9.40E+00	5.20E+00	5.73E+00	4.92E+00	4.74E+00	4.83E+00	1.50E+00	1.14E+00	2.41E+00	6.24E+00	7.60E+00	9.66E+00	1.06E+01	8.85E+00	22.9650
	VOC	Concentration (ug/m3)	Annual	100th Percentile	4.28E-02	7.81E-02	1.73E-01	7.32E-02	4.13E-01	3.11E-01	7.88E-02	2.31E-01	2.90E-02	1.99E-02	1.13E-01	4.29E-01	5.42E-01	9.53E-01	2.73E-01	3.17E-01	1.7886
	VOC	Deposition (g/m2/month)	Annual	100th Percentile	1.09E-04	2.18E-04	5.10E-04	2.03E-04	1.08E-03	7.90E-04	2.03E-04	6.21E-04	8.84E-05	6.89E-05	3.27E-04	1.12E-03	1.48E-03	2.69E-03	9.13E-04	9.45E-04	
	ZN	Concentration (ug/m3)	1-Hour	99.9th Percentile	2.67E-03	1.62E-03	7.15E-03	5.73E-03	6.85E-03	6.08E-03	6.35E-03	5.79E-03	1.39E-03	1.22E-03	2.44E-03	7.39E-03	8.86E-03	1.00E-02	1.13E-02	1.06E-02	0.0277
	ZN	Concentration (ug/m3)	Annual	100th Percentile	4.49E-05	7.30E-05	1.63E-04	7.49E-05	4.51E-04	3.75E-04	8.09E-05	2.66E-04	4.43E-05	3.54E-05	1.51E-04	5.17E-04	6.42E-04	1.04E-03	2.86E-04	3.53E-04	0.0019
	ZN	Deposition (g/m2/month)	Annual	100th Percentile	7.13E-07	1.52E-06	4.78E-06	1.78E-06	9.44E-06	6.51E-06	1.43E-06	5.17E-06	8.90E-07	8.02E-07	3.07E-06	1.00E-05	1.41E-05	2.79E-05	1.03E-05	8.11E-06	

Inhalation exposures

Predicted ground level concentrations and screening assessment - acute exposures

			Air Concentration - Maximum 1 hour average (ug/m^3)		Air Concentration - Maximum 1 hour average (mg/m^3)		Calculated HI	
COPC	**Acute air guideline - health (mg/m^3)**	**Air guideline - vegetation (mg/m^3)**	**Maximum anywhere off-site**	**Maximum at receptors**	**Maximum anywhere off-site**	**Maximum at receptors**	**Maximum anywhere off-site**	**Maximum at receptors**
Hydrogen chloride (HCl)	0.66	1.5 to 6	1.93E+00	5.51E-01	1.9E-03	5.5E-04	0.0029	0.0008
Hydrogen fluoride (HF)	0.06	0.003	8.03E-02	8.03E-02	8.0E-05	8.0E-05	0.0013	0.0013
Ammonia	0.59	2	4.59E+00	2.12E+00	4.6E-03	2.1E-03	0.0078	0.0036
Hydrogen sulfide (H2S)	0.5	NA	5.76E+00	2.72E+00	5.8E-03	2.7E-03	0.012	0.0054
Arsenic	0.0099		1.42E-03	4.07E-04	1.4E-06	4.1E-07	0.000054	0.000015
Cadmium	0.00055		1.95E-02	5.53E-03	2.0E-05	5.5E-06	0.013	0.0038
Chromium (Cr VI assumed)	0.0013		2.11E-02	6.04E-03	2.1E-05	6.0E-06	0.0061	0.0017
Copper	0.1		1.13E-02	4.66E-03	1.1E-05	4.7E-06	0.000042	0.000017
Nickel	0.0011		1.79E-02	5.13E-03	1.8E-05	5.1E-06	0.0061	0.0017
Selenium	0.025		6.09E-03	1.79E-03	6.1E-06	1.8E-06	0.000091	0.000027
Mercury (as inorganic and el	0.0006		1.93E-02	5.49E-03	1.9E-05	5.5E-06	0.012	0.0034
Benzene	0.17		2.30E+01	1.06E+01	2.3E-02	1.1E-02	0.14	0.062
							0.2	0.08

Calculated HI incorporates a factor of 0.375 to obtain the respirable fraction from TSP (or total concentration) - relevant to the assessment of health impacts

Inhalation - gases and particulates

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

Parameters Relevant to Quantification of Community Exposures - Commercial/Industrial Off-site		
Exposure Time at Home (ET, hr/day)	8	Workers present for 8 hours per day as per enHealth (2012)
Fraction Inhaled from Source (FI, unitless)	1	Assume workers at the same location each day
Dust lung retention factor (LRF, unitless)	0.375	Percentage of respirable dust that is small enough to reach and be retained in the lungs (NEPM 1999 amended 2013). Value of 1 adopted for gasses
Exposure Frequency - normal conditions (EF, days/yr)	240	Days at work (normal conditions), as per NEPM (1999 amended 2013)
Exposure Duration (ED, years)	30	Duration of work at same location as per NEPM
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	262800	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 C/I (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)	
Hydrogen chloride (HCl)	0.0E+00	2.6E-02	0%	2.6E-02	4.5E-05	4.2E-06	9.8E-06	--		0.00038	2%
Hydrogen fluoride (HF)	0.0E+00	2.9E-02	0%	2.9E-02	4.5E-06	4.2E-07	9.8E-07	--		0.000034	0%
Ammonia	0.0E+00	3.2E-01	0%	3.2E-01	1.9E-04	1.8E-05	4.2E-05	--		0.00013	1%
Hydrogen sulfide (H2S)	0.0E+00	2.0E-02	10%	1.8E-02	2.4E-04	2.3E-05	5.3E-05	--		0.0030	12%
Arsenic	0.0E+00	6.7E-05	4%	6.4E-05	3.4E-08	1.2E-09	2.8E-09	--		0.000044	0%
Cadmium	0.0E+00	5.0E-06	25%	3.8E-06	4.5E-07	1.6E-08	3.7E-08	--		0.010	41%
Chromium (Cr VI assumed)	0.0E+00	1.0E-04	18%	8.2E-05	5.0E-07	1.8E-08	4.1E-08	--		0.00050	2%
Copper	0.0E+00	4.9E-01	14%	4.2E-01	4.1E-07	1.5E-08	3.4E-08	--		0.000000081	0%
Lead	0.0E+00	5.0E-04	6%	4.7E-04	2.9E-07	1.0E-08	2.4E-08	--		0.000051	0%
Nickel	0.0E+00	2.0E-05	17%	1.7E-05	4.3E-07	1.5E-08	3.5E-08	--		0.0021	9%
Selenium	0.0E+00	2.1E-02	25%	1.6E-02	1.5E-07	5.2E-09	1.2E-08	--		0.00000077	0%
Mercury (as inorganic and element	0.0E+00	2.0E-04	23%	1.5E-04	4.4E-07	1.6E-08	3.6E-08	--		0.00024	1%
Zinc	0.0E+00	1.8E+00	28%	1.3E+00	1.0E-06	3.7E-08	8.5E-08	--		0.000000068	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	9.5E-04	9.0E-05	2.1E-04	5.4E-7	100%	0.0077	32%
Total PFAS (assume as toxic as P	0.0E+00	7.0E-05	10%	6.3E-05	1.0E-08	3.7E-10	8.6E-10	--		0.000014	0%
Dioxins and furans (WHO-TEQ)	0.0E+00	8.1E-09	54%	3.7E-09	4.8E-13	1.7E-14	4.0E-14	--		0.000011	0%
PAHs (assuming BaP)	6.0E-01	0.0E+00	0%	0.0E+00	4.5E-09	1.6E-10	3.7E-10	9.5E-11	0%	--	
							TOTAL	**5E-07**		**2E-02**	

Background intakes - relevant to adults

Inhalation - gases and particulates

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

Parameters Relevant to Quantification of Community Exposures - Recreational areas		
Exposure Time at Home (ET, hr/day)	4	Assumed time spent in recreational areas each time
Fraction Inhaled from Source (FI, unitless)	1	Assume workers at the same location each day
Dust lung retention factor (LRF, unitless)	0.375	Percentage of respirable dust that is small enough to reach and be retained in the lungs (NEPM 1999 amended 2013). Value of 1 adopted for gasses
Exposure Frequency - normal conditions (EF, days/yr)	104	Assume use of recreational area 2 days per week
Exposure Duration (ED, years)	35	Assumed relevant for using recreational area
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 $(mg/m^3)^{-1}$	Chronic TC Air (mg/m^3)	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background) (mg/m^3)	Estimated Concentration in Air - Maximum recreational (Ca) (mg/m^3)	Inhalation Exposure Concentration - NonThreshold (mg/m^3)	Inhalation Exposure Concentration - Threshold (mg/m^3)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Hydrogen chloride (HCl)	0.0E+00	2.6E-02	0%	2.6E-02	7.5E-06	1.8E-07	3.6E-07	--		1.4E-05	1%
Hydrogen fluoride (HF)	0.0E+00	2.9E-02	0%	2.9E-02	7.5E-07	1.8E-08	3.6E-08	--		1.2E-06	0%
Ammonia	0.0E+00	3.2E-01	0%	3.2E-01	2.3E-05	5.4E-07	1.1E-06	--		3.3E-06	0%
Hydrogen sulfide (H2S)	0.0E+00	2.0E-02	10%	1.8E-02	2.9E-05	6.9E-07	1.4E-06	--		7.7E-05	6%
Arsenic	0.0E+00	6.7E-05	9%	6.1E-05	5.5E-09	4.9E-11	9.8E-11	--		1.6E-06	0%
Cadmium	0.0E+00	5.0E-06	63%	1.9E-06	7.3E-08	6.5E-10	1.3E-09	--		7.0E-04	59%
Chromium (Cr VI assumed)	0.0E+00	1.0E-04	56%	4.4E-05	8.0E-08	7.1E-10	1.4E-09	--		3.2E-05	3%
Copper	0.0E+00	4.9E-01	49%	2.5E-01	5.9E-08	5.2E-10	1.0E-09	--		4.2E-09	0%
Lead	0.0E+00	5.0E-04	23%	3.9E-04	4.7E-08	4.2E-10	8.4E-10	--		2.2E-06	0%
Nickel	0.0E+00	2.0E-05	55%	9.0E-06	6.9E-08	6.1E-10	1.2E-09	--		1.4E-04	11%
Selenium	0.0E+00	2.1E-02	60%	8.4E-03	2.4E-08	2.1E-10	4.2E-10	--		5.0E-08	0%
Mercury (as inorganic and element	0.0E+00	2.0E-04	57%	8.6E-05	7.2E-08	6.4E-10	1.3E-09	--		1.5E-05	1%
Zinc	0.0E+00	1.8E+00	60%	7.0E-01	1.5E-07	1.3E-09	2.7E-09	--		3.8E-09	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	1.1E-04	2.7E-06	5.4E-06	1.6E-8	100%	2.0E-04	17%
Total PFAS (assume as toxic as P	0.0E+00	7.0E-05	10%	6.3E-05	1.3E-09	1.1E-11	2.3E-11	--		3.6E-07	0%
Dioxins and furans (WHO-TEQ)	0.0E+00	8.1E-09	54%	3.7E-09	7.8E-14	6.9E-16	1.4E-15	--		3.7E-07	0%
PAHs (assuming BaP)	6.0E-01	0.0E+00	0%	0.0E+00	4.5E-09	4.0E-11	8.0E-11	2.4E-11	0%	--	
							TOTAL	**2E-08**		**1E-03**	

Background intakes - values for young children adopted

Inhalation - gases and particulates

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

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 (LRF, unitless)	0.375	Percentage of respirable dust that is small enough to reach and be retained in the lungs (NEPM 1999 amended 2013). Value of 1 adopted 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	Duration of residency
Averaging Time - NonThreshold (Atc, hours)	613200	US EPA 2009
Averaging Time - Threshold (Atn, hours)	306600	US EPA 2009

Key Chemical	Toxicity Data				Concentration	Daily Exposure		Calculated Risk			
	Inhalation Unit Risk $(mg/m^3)^{-1}$	Chronic TC Air (mg/m^3)	Background Intake (% Chronic TC)	Chronic TC Allowable for Assessment (TC-Background) (mg/m^3)	Estimated Concentration in Air - Maximum residential (Ca) (mg/m^3)	Inhalation Exposure Concentration - NonThreshold (mg/m^3)	Inhalation Exposure Concentration - Threshold (mg/m^3)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Hydrogen chloride (HCl)	0.0E+00	2.6E-02	0%	2.6E-02	1.6E-05	8.1E-06	1.6E-05	--		0.00062	1%
Hydrogen fluoride (HF)	0.0E+00	2.9E-02	0%	2.9E-02	1.6E-06	8.1E-07	1.6E-06	--		0.000056	0%
Ammonia	0.0E+00	3.2E-01	0%	3.2E-01	8.3E-05	4.1E-05	8.3E-05	--		0.00026	0%
Hydrogen sulfide (H2S)	0.0E+00	2.0E-02	10%	1.8E-02	1.1E-04	5.3E-05	1.1E-04	--		0.0058	9%
Arsenic	0.0E+00	6.7E-05	9%	6.1E-05	1.3E-08	2.4E-09	4.8E-09	--		0.000078	0%
Cadmium	0.0E+00	5.0E-06	63%	1.9E-06	1.6E-07	3.0E-08	6.1E-08	--		0.033	51%
Chromium (Cr VI assumed)	0.0E+00	1.0E-04	56%	4.4E-05	1.8E-07	3.4E-08	6.8E-08	--		0.0015	2%
Copper	0.0E+00	4.9E-01	49%	2.5E-01	1.6E-07	3.1E-08	6.1E-08	--		0.00000024	0%
Lead	0.0E+00	5.0E-04	23%	3.9E-04	1.1E-07	2.0E-08	4.0E-08	--		0.00010	0%
Nickel	0.0E+00	2.0E-05	55%	9.0E-06	1.6E-07	2.9E-08	5.9E-08	--		0.0066	10%
Selenium	0.0E+00	2.1E-02	60%	8.4E-03	5.3E-08	1.0E-08	2.0E-08	--		0.0000024	0%
Mercury (as inorganic and element	0.0E+00	2.0E-04	57%	8.6E-05	1.6E-07	3.0E-08	5.9E-08	--		0.00069	1%
Zinc	0.0E+00	1.8E+00	60%	7.0E-01	4.5E-07	8.5E-08	1.7E-07	--		0.00000024	0%
Benzene	6.0E-03	3.0E-02	10%	2.7E-02	4.1E-04	2.1E-04	4.1E-04	1.2E-6	100%	0.015	24%
Total PFAS (assume as toxic as P	0.0E+00	7.0E-05	10%	6.3E-05	4.5E-09	8.4E-10	1.7E-09	--		0.000027	0%
Dioxins and furans (WHO-TEQ)	0.0E+00	8.1E-09	54%	3.7E-09	1.8E-13	3.4E-14	6.8E-14	--		0.000018	0%
PAHs (assuming BaP)	6.0E-01	0.0E+00	0%	0.0E+00	9.7E-09	1.8E-09	3.6E-09	1.1E-9	0%	--	
							TOTAL	**1E-06**		**6E-02**	

Background intakes - values for young children adopted

Multi-pathway exposures for maximum sensitive receptor

Soil

Calculation of Concentrations in Soil

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

where:

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

General Parameters		Surface (for direct contact)	Depth (for agricultural pathways)	
Soil bulk density (p)	g/m^3	1600000	1600000	Default for fill materials
General mixing depth (d)	m	0.01	0.15	As per OEHHA (2015) guidance
Duration of deposition (T)	years	35	35	Duration of operation

Chemical-specific Inputs and calculations - maximum residential receptors

Chemical	Half-life in soil years	Loss constant (K) per year	Deposition Rate (DR) mg/m^2/year	Surface Concentration in Soil mg/kg	Agricultural Concentration in Soil mg/kg
Arsenic	273973	2.5E-06	3.1E-03	6.7E-03	4.5E-04
Cadmium	273973	2.5E-06	3.9E-02	8.5E-02	5.7E-03
Chromium (Cr VI assumed)	273973	2.5E-06	4.4E-02	9.5E-02	6.4E-03
Copper	273973	2.5E-06	4.3E-02	9.4E-02	6.3E-03
Lead	273973	2.5E-06	2.5E-02	5.6E-02	3.7E-03
Nickel	273973	2.5E-06	3.8E-02	8.3E-02	5.5E-03
Selenium	273973	2.5E-06	1.3E-02	2.8E-02	1.9E-03
Mercury (as inorganic and eleme	273973	2.5E-06	3.8E-02	8.3E-02	5.5E-03
Zinc	273973	2.5E-06	1.1E-01	2.5E-01	1.7E-02
Total PFAS (assume as toxic as	41	0.017	1.4E-04	2.3E-04	1.5E-05
Dioxins and furans (WHO-TEQ)	12	0.058	4.3E-08	4.0E-08	2.7E-09
PAHs (assuming BaP)	12	0.058	2.9E-04	2.7E-04	1.8E-05

Half-life in soil: dioxin value from Lowe et al (1991), PFAS value for PFOS from Environment Canada (2006) and metals from OEHHA (2015)

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
Arsenic	273973	2.5E-06	1.0E-02	2.2E-02	1.5E-03
Cadmium	273973	2.5E-06	1.3E-01	2.9E-01	1.9E-02
Chromium (Cr VI assumed)	273973	2.5E-06	1.5E-01	3.2E-01	2.1E-02
Copper	273973	2.5E-06	1.3E-01	2.9E-01	1.9E-02
Lead	273973	2.5E-06	8.5E-02	1.9E-01	1.2E-02
Nickel	273973	2.5E-06	1.3E-01	2.7E-01	1.8E-02
Selenium	273973	2.5E-06	4.3E-02	9.4E-02	6.3E-03
Mercury (as inorganic and eleme	273973	2.5E-06	1.3E-01	2.8E-01	1.9E-02
Zinc	273973	2.5E-06	3.3E-01	7.3E-01	4.9E-02
Total PFAS (assume as toxic as	41	0.017	3.5E-04	5.8E-04	3.9E-05
Dioxins and furans (WHO-TEQ)	12	0.058	1.4E-07	1.3E-07	8.8E-09
PAHs (assuming BaP)	12	0.058	8.5E-04	8.0E-04	5.3E-05

Half-life in soil: dioxin value from Lowe et al (1991), PFAS value for PFOS from Environment Canada (2006) and metals 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)	
Arsenic		2.0E-03	4%	1.9E-03	100%	6.7E-03	2.0E-09	4.8E-09	--		2.5E-06	1%
Cadmium		8.0E-04	25%	6.0E-04	100%	8.5E-02	2.5E-08	6.1E-08	--		1.0E-04	26%
Chromium (Cr VI assumed)		9.0E-04	18%	7.4E-04	100%	9.5E-02	2.8E-08	6.8E-08	--		9.2E-05	24%
Copper		1.4E-01	14%	1.2E-01	100%	9.4E-02	2.8E-08	6.7E-08	--		5.6E-07	0%
Lead		3.5E-03	6%	3.3E-03	100%	5.6E-02	1.6E-08	4.0E-08	--		1.2E-05	3%
Nickel		1.2E-02	17%	1.0E-02	100%	8.3E-02	2.4E-08	5.9E-08	--		5.9E-06	2%
Selenium		6.0E-03	25%	4.5E-03	100%	2.8E-02	8.4E-09	2.0E-08	--		4.5E-06	1%
Mercury (as inorganic and e		6.0E-04	23%	4.6E-04	100%	8.3E-02	2.5E-08	5.9E-08	--		1.3E-04	33%
Zinc		5.0E-01	28%	3.6E-01	100%	2.5E-01	7.3E-08	1.8E-07	--		4.9E-07	0%
Total PFAS (assume as tox		2.0E-05	10%	1.8E-05	100%	2.3E-04	6.8E-11	1.6E-10	--		9.1E-06	2%
Dioxins and furans (WHO-TE		2.3E-09	54%	1.1E-09	100%	4.0E-08	1.2E-14	2.9E-14	--		2.7E-05	7%
PAHs (assuming BaP)	2.3E-01				100%	2.7E-04	8.0E-11	1.9E-10	1.9E-11		--	
								TOTAL	**1.9E-11**		**3.8E-4**	

Exposure to Chemicals via Incidental Ingestion of Soil

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

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

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Soil Concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor	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)	
Arsenic		2.0E-03	9%	1.8E-03	100%	6.7E-03	3.8E-09	4.5E-08	--		2.5E-05	0%
Cadmium		8.0E-04	63%	3.0E-04	100%	8.5E-02	4.9E-08	5.7E-07	--		1.9E-03	30%
Chromium (Cr VI assumed)		9.0E-04	56%	4.0E-04	100%	9.5E-02	5.5E-08	6.4E-07	--		1.6E-03	25%
Copper		1.4E-01	49%	7.1E-02	100%	9.4E-02	5.4E-08	6.3E-07	--		8.8E-06	0%
Lead		3.5E-03	23%	2.7E-03	100%	5.6E-02	3.2E-08	3.7E-07	--		1.4E-04	2%
Nickel		1.2E-02	55%	5.4E-03	100%	8.3E-02	4.7E-08	5.5E-07	--		1.0E-04	2%
Selenium		6.0E-03	60%	2.4E-03	100%	2.8E-02	1.6E-08	1.9E-07	--		7.9E-05	1%
Mercury (as inorganic and e		6.0E-04	57%	2.6E-04	100%	8.3E-02	4.7E-08	5.5E-07	--		2.1E-03	34%
Zinc		5.0E-01	60%	2.0E-01	100%	2.5E-01	1.4E-07	1.7E-06	--		8.3E-06	0%
Total PFAS (assume as tox		2.0E-05	10%	1.8E-05	100%	2.3E-04	1.3E-10	1.5E-09	--		8.5E-05	1%
Dioxins and furans (WHO-TE		2.3E-09	54%	1.1E-09	100%	4.0E-08	2.3E-14	2.7E-13	--		2.5E-04	4%
PAHs (assuming BaP)	2.3E-01				100%	2.7E-04	1.5E-10	1.8E-09	3.6E-11		--	
								TOTAL	**3.6E-11**		**6.4E-3**	

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)	
Arsenic		2.0E-03	4%	1.9E-03	0.0001	6.7E-03	1.2E-11	3.0E-11	--		1.6E-8	0%
Cadmium		8.0E-04	25%	6.0E-04	0.0001	8.5E-02	1.6E-10	3.8E-10	--		6.4E-07	1%
Chromium (Cr VI assumed)		9.0E-04	18%	7.4E-04	0.0001	9.5E-02	1.8E-10	4.3E-10	--		5.8E-07	1%
Copper		1.4E-01	14%	1.2E-01	0.0001	9.4E-02	1.8E-10	4.2E-10	--		3.5E-09	0%
Lead		3.5E-03	6%	3.3E-03	0.0001	5.6E-02	1.0E-10	2.5E-10	--		7.6E-08	0%
Nickel		1.2E-02	17%	1.0E-02	0.0001	8.3E-02	1.5E-10	3.7E-10	--		3.7E-08	0%
Selenium		6.0E-03	25%	4.5E-03	0.0001	2.8E-02	5.3E-11	1.3E-10	--		2.8E-08	0%
Mercury (as inorganic and elem		4.2E-05	23%	3.2E-05	0.0001	8.3E-02	1.5E-10	3.7E-10	--		1.2E-05	18%
Zinc		5.0E-01	28%	3.6E-01	0.001	2.5E-01	4.6E-09	1.1E-08	--		3.1E-08	0%
Total PFAS (assume as toxic a		2.0E-05	10%	1.8E-05		2.3E-04			--		--	
Dioxins and furans (WHO-TEQ)		2.3E-09	54%	1.1E-09	0.03	4.0E-08	2.3E-14	5.4E-14	--		5.1E-05	80%
PAHs (assuming BaP)	2.3E-01				0.06	2.7E-04	3.0E-10	7.3E-10	7.0E-11		--	
								TOTAL	**7.0E-11**		**6.4E-05**	

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)	
Arsenic		2.0E-03	9%	1.8E-03	0.0001	6.7E-03	5.2E-12	6.0E-11	--		3.3E-8	0%
Cadmium		8.0E-04	63%	3.0E-04	0.0001	8.5E-02	6.6E-11	7.6E-10	--		2.6E-06	2%
Chromium (Cr VI assumed)		9.0E-04	56%	4.0E-04	0.0001	9.5E-02	7.4E-11	8.6E-10	--		2.2E-06	1%
Copper		1.4E-01	49%	7.1E-02	0.0001	9.4E-02	7.3E-11	8.5E-10	--		1.2E-08	0%
Lead		3.5E-03	23%	2.7E-03	0.0001	5.6E-02	4.3E-11	5.0E-10	--		1.9E-07	0%
Nickel		1.2E-02	55%	5.4E-03	0.0001	8.3E-02	6.4E-11	7.4E-10	--		1.4E-07	0%
Selenium		6.0E-03	60%	2.4E-03	0.0001	2.8E-02	2.2E-11	2.5E-10	--		1.1E-07	0%
Mercury (as inorganic and elem		4.2E-05	57%	1.8E-05	0.0001	8.3E-02	6.4E-11	7.5E-10	--		4.1E-05	28%
Zinc		5.0E-01	60%	2.0E-01	0.001	2.5E-01	1.9E-09	2.2E-08	--		1.1E-07	0%
Total PFAS (assume as toxic a		2.0E-05	10%	1.8E-05		2.3E-04			--		--	
Dioxins and furans (WHO-TEQ)		2.3E-09	54%	1.1E-09	0.03	4.0E-08	9.3E-15	1.1E-13	--		1.0E-04	69%
PAHs (assuming BaP)	2.3E-01				0.06	2.7E-04	1.2E-10	1.5E-09	2.9E-11		--	
								TOTAL	**2.9E-11**		**1.5E-04**	

Produce

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
Arsenic	14	0.05	8.4E-06	4.2E-06	0.01	4.5E-04	4.5E-06
Cadmium	14	0.05	1.1E-04	5.3E-05	0.125	5.7E-03	7.1E-04
Chromium (Cr VI assumed)	14	0.05	1.2E-04	6.0E-05	0.00188	6.4E-03	1.2E-05
Copper	14	0.05	1.2E-04	5.9E-05	0.1	6.3E-03	6.3E-04
Lead	14	0.05	7.0E-05	3.5E-05	0.0113	3.7E-03	4.2E-05
Nickel	14	0.05	1.0E-04	5.2E-05	0.015	5.5E-03	8.3E-05
Selenium	14	0.05	3.5E-05	1.8E-05	0.00625	1.9E-03	1.2E-05
Mercury (as inorganic and eleme	14	0.05	1.0E-04	5.2E-05	0.225	5.5E-03	1.2E-03
Zinc	14	0.05	3.1E-04	1.5E-04	0.264	1.7E-02	4.4E-03
Total PFAS (assume as toxic as	14	0.05	3.8E-07	1.9E-07	0.51	1.5E-05	7.8E-06
Dioxins and furans (WHO-TEQ)	14	0.05	1.2E-10	5.9E-11	0.000876	2.7E-09	2.4E-12
PAHs (assuming BaP)	14	0.05	7.9E-07	3.9E-07	0.00214	1.8E-05	3.8E-08

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

Exposure to Chemicals via Ingestion of Homegrown Fruit and Vegetables

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

Parameters Relevant to Quantification of Exposure by Adults		
Ingestion Rate of Produce (IRp) (kg/day)	0.4	Total fruit and vegetable consumption rate for adults as per NEPM (2013)
Proportion of total intake from aboveground crops (%A	73%	Proportions as per NEPM (2013)
Proportion of total intake from root crops (%R)	27%	Proportions as per NEPM (2013)
Fraction ingested that is homegrown (%)	35%	Relevant to rural 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)	
Arsenic		2.0E-03	4%	1.9E-03	100%	4.2E-06	4.5E-06	3.5E-09	8.5E-09	--		4.4E-06	0%
Cadmium		8.0E-04	25%	6.0E-04	100%	5.3E-05	7.1E-04	1.9E-07	4.6E-07	--		7.7E-04	26%
Chromium (Cr VI assumed)		9.0E-04	18%	7.4E-04	100%	6.0E-05	1.2E-05	3.9E-08	9.4E-08	--		1.3E-04	4%
Copper		1.4E-01	14%	1.2E-01	100%	5.9E-05	6.3E-04	1.8E-07	4.3E-07	--		3.5E-06	0%
Lead		3.5E-03	6%	3.3E-03	100%	3.5E-05	4.2E-05	3.0E-08	7.3E-08	--		2.2E-05	1%
Nickel		1.2E-02	17%	1.0E-02	100%	5.2E-05	8.3E-05	5.0E-08	1.2E-07	--		1.2E-05	0%
Selenium		6.0E-03	25%	4.5E-03	100%	1.8E-05	1.2E-05	1.3E-08	3.2E-08	--		7.2E-06	0%
Mercury (as inorganic and element		6.0E-04	23%	4.6E-04	100%	5.2E-05	1.2E-03	3.1E-07	7.5E-07	--		1.6E-03	56%
Zinc		5.0E-01	28%	3.6E-01	100%	1.5E-04	4.4E-03	1.1E-06	2.6E-06	--		7.2E-06	0%
Total PFAS (assume as toxic as P		2.0E-05	10%	1.8E-05	100%	1.9E-07	7.8E-06	1.9E-09	4.5E-09	--		2.5E-04	9%
Dioxins and furans (WHO-TEQ)		2.3E-09	54%	1.1E-09	100%	5.9E-11	2.4E-12	3.6E-14	8.7E-14	--		8.2E-05	3%
PAHs (assuming BaP)	2.3E-01				100%	3.9E-07	3.8E-08	2.5E-10	5.9E-10	5.7E-11		--	
									TOTAL	**5.7E-11**		**2.9E-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 (%)	35%	Relevant to rural 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)	
Arsenic		2.0E-03	9%	1.8E-03	100%	4.2E-06	4.5E-06	2.4E-09	2.8E-08	--		1.5E-05	0%
Cadmium		8.0E-04	63%	3.0E-04	100%	5.3E-05	7.1E-04	8.8E-08	1.0E-06	--		3.5E-03	30%
Chromium (Cr VI assumed)		9.0E-04	56%	4.0E-04	100%	6.0E-05	1.2E-05	2.9E-08	3.4E-07	--		8.6E-04	7%
Copper		1.4E-01	49%	7.1E-02	100%	5.9E-05	6.3E-04	8.4E-08	9.8E-07	--		1.4E-05	0%
Lead		3.5E-03	23%	2.7E-03	100%	3.5E-05	4.2E-05	2.0E-08	2.3E-07	--		8.7E-05	1%
Nickel		1.2E-02	55%	5.4E-03	100%	5.2E-05	8.3E-05	3.2E-08	3.7E-07	--		6.9E-05	1%
Selenium		6.0E-03	60%	2.4E-03	100%	1.8E-05	1.2E-05	9.4E-09	1.1E-07	--		4.6E-05	0%
Mercury (as inorganic and element		6.0E-04	57%	2.6E-04	100%	5.2E-05	1.2E-03	1.4E-07	1.6E-06	--		6.1E-03	53%
Zinc		5.0E-01	60%	2.0E-01	100%	1.5E-04	4.4E-03	4.6E-07	5.4E-06	--		2.7E-05	0%
Total PFAS (assume as toxic as P		2.0E-05	10%	1.8E-05	100%	1.9E-07	7.8E-06	7.9E-10	9.2E-09	--		5.1E-04	4%
Dioxins and furans (WHO-TEQ)		2.3E-09	54%	1.1E-09	100%	5.9E-11	2.4E-12	2.8E-14	3.2E-13	--		3.1E-04	3%
PAHs (assuming BaP)	2.3E-01				100%	3.9E-07	3.8E-08	1.9E-10	2.2E-09	4.4E-11		--	
									TOTAL	**4.4E-11**		**1.2E-02**	

Calculation of Concentrations in Eggs

Uptake in to chicken eggs

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

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

General Parameters	Units	Value	
FI (fraction of crops ingested from property)		1	Assume pasture is grown on the site
IRc (ingestion rate of crops)	kg/day	0.12	As per OEHHA (2015)
IRs (ingestion rate of soil)	kg/day	0.01	As per OEHHA (2015) and advice from AgVIC
B (bioavailability)	%	100%	

Chemical-specific Inputs and calculations - Maximum sensitive receptors

Chemical	Concentration in crops ingested by chickens mg/kg ww	Soil Concentration - Agriculture (Cs) mg/kg	Transfer factor to eggs day/kg	Egg Concentration mg/kg ww	
Arsenic	4.2E-06	4.5E-04	7.0E-02	3.5E-07	
Cadmium	5.3E-05	5.7E-03	1.0E-02	6.3E-07	
Chromium (Cr VI assumed)	6.0E-05	6.4E-03	9.2E-03	6.5E-07	OEHHA (2003)
Copper	5.9E-05	6.3E-03	1.7E-01	1.2E-05	95% from Leeman et al (2007)
Lead	3.5E-05	3.7E-03	4.0E-02	1.7E-06	
Nickel	5.2E-05	5.5E-03	2.0E-02	1.2E-06	
Selenium	1.8E-05	1.9E-03	3.0E+00	6.3E-05	
Mercury (as inorganic and elementa	5.2E-05	5.5E-03	8.0E-01	4.9E-05	
Zinc	1.5E-04	1.7E-02	1.7E-01	3.1E-05	95% from Leeman et al (2007)
Total PFAS (assume as toxic as PI	1.9E-07	1.5E-05	3.4E+01	6.0E-06	Transfer factors from Australian Study (AECOM 2017)
Dioxins and furans (WHO-TEQ)	5.9E-11	2.7E-09	1.0E+01	3.4E-10	
PAHs (assuming BaP)	3.9E-07	1.8E-05	3.0E-03	6.8E-10	

Transfer factors from OEHHA 2015 unless otherwise noted

Exposure to Chemicals via Ingestion of Eggs

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

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

Maximum from sensitive receptors

Key Chemical	Toxicity Data				Bioavailability (%)	Egg concentration	Daily Intake		Calculated Risk			
	Non-Threshold Slope Factor (mg/kg-day)$^{-1}$	Threshold TDI (mg/kg/day)	Background Intake (% TDI)	TDI Allowable for Assessment (TDI-Background) (mg/kg/day)		(mg/kg wet weight)	NonThreshold (mg/kg/day)	Threshold (mg/kg/day)	Non-Threshold Risk (unitless)	% Total Risk	Chronic Hazard Quotient (unitless)	% Total HI
Arsenic		2.0E-03	4%	1.9E-03	100%	3.5E-07	2.9E-11	6.9E-11	--		3.6E-08	0%
Cadmium		8.0E-04	25%	6.0E-04	100%	6.3E-07	5.2E-11	1.3E-10	--		2.1E-07	0%
Chromium (Cr VI assumed)		9.0E-04	18%	7.4E-04	100%	6.5E-07	5.4E-11	1.3E-10	--		1.8E-07	0%
Copper		1.4E-01	14%	1.2E-01	100%	1.2E-05	9.9E-10	2.4E-09	--		2.0E-08	0%
Lead		3.5E-03	6%	3.3E-03	100%	1.7E-06	1.4E-10	3.3E-10	--		1.0E-07	0%
Nickel		1.2E-02	17%	1.0E-02	100%	1.2E-06	1.0E-10	2.5E-10	--		2.5E-08	0%
Selenium		6.0E-03	25%	4.5E-03	100%	6.3E-05	5.2E-09	1.3E-08	--		2.8E-06	2%
Mercury (as inorganic and element		6.0E-04	23%	4.6E-04	100%	4.9E-05	4.1E-09	9.8E-09	--		2.1E-05	14%
Zinc		5.0E-01	28%	3.6E-01	100%	3.1E-05	2.6E-09	6.2E-09	--		1.7E-08	0%
Total PFAS (assume as toxic as P		2.0E-05	10%	1.8E-05	100%	6.0E-06	5.0E-10	1.2E-09	--		6.7E-05	43%
Dioxins and furans (WHO-TEQ)		2.3E-09	54%	1.1E-09	100%	3.4E-10	2.8E-14	6.8E-14	--		6.4E-05	41%
PAHs (assuming BaP)	2.3E-01				100%	6.8E-10	5.6E-14	1.4E-13	1.3E-14		--	
TOTAL									**1.3E-14**		**1.6E-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
Arsenic		2.0E-03	9%	1.8E-03	100%	3.5E-07	1.2E-11	1.4E-10	--		7.6E-08	0%
Cadmium		8.0E-04	63%	3.0E-04	100%	6.3E-07	2.2E-11	2.5E-10	--		8.5E-07	0%
Chromium (Cr VI assumed)		9.0E-04	56%	4.0E-04	100%	6.5E-07	2.2E-11	2.6E-10	--		6.6E-07	0%
Copper		1.4E-01	49%	7.1E-02	100%	1.2E-05	4.1E-10	4.8E-09	--		6.7E-08	0%
Lead		3.5E-03	23%	2.7E-03	100%	1.7E-06	5.7E-11	6.6E-10	--		2.5E-07	0%
Nickel		1.2E-02	55%	5.4E-03	100%	1.2E-06	4.2E-11	4.9E-10	--		9.1E-08	0%
Selenium		6.0E-03	60%	2.4E-03	100%	6.3E-05	2.2E-09	2.5E-08	--		1.1E-05	3%
Mercury (as inorganic and element		6.0E-04	57%	2.6E-04	100%	4.9E-05	1.7E-09	2.0E-08	--		7.6E-05	22%
Zinc		5.0E-01	60%	2.0E-01	100%	3.1E-05	1.1E-09	1.2E-08	--		6.2E-08	0%
Total PFAS (assume as toxic as P		2.0E-05	10%	1.8E-05	100%	6.0E-06	2.1E-10	2.4E-09	--		1.3E-04	38%
Dioxins and furans (WHO-TEQ)		2.3E-09	54%	1.1E-09	100%	3.4E-10	1.2E-14	1.4E-13	--		1.3E-04	37%
PAHs (assuming BaP)	2.3E-01				100%	6.8E-10	2.3E-14	2.7E-13	5.4E-15		--	
TOTAL									**5.4E-15**		**3.5E-04**	

Summary of Risks - Residential

Calculated HI for each CoPC	Adults					
	Exposure pathway					Sum over all pathways
	Inhalation	Ingestion of soil	Dermal contact with soil	Ingestion of home-grown fruit and vegetables	Ingestion of eggs	
Hydrogen chloride (HCl)	6.2E-04					6.2E-04
Hydrogen fluoride (HF)	5.6E-05					5.6E-05
Hydrogen sulfide (H2S)	5.8E-03					5.8E-03
Arsenic	7.8E-05	2.5E-06	1.6E-08	4.4E-06	3.6E-08	8.5E-05
Cadmium	3.3E-02	1.0E-04	6.4E-07	7.7E-04	2.1E-07	3.4E-02
Chromium (Cr VI assumed)	1.5E-03	9.2E-05	5.8E-07	1.3E-04	1.8E-07	1.8E-03
Copper	2.4E-07	5.6E-07	3.5E-09	3.5E-06	2.0E-08	4.4E-06
Lead	1.0E-04	1.2E-05	7.6E-08	2.2E-05	1.0E-07	1.4E-04
Nickel	6.6E-03	5.9E-06	3.7E-08	1.2E-05	2.5E-08	6.6E-03
Selenium	2.4E-06	4.5E-06	2.8E-08	7.2E-06	2.8E-06	1.7E-05
Mercury (as inorganic and elemental)	6.9E-04	1.3E-04	1.2E-05	1.6E-03	2.1E-05	2.5E-03
Zinc	2.4E-07	4.9E-07	3.1E-08	7.2E-06	1.7E-08	8.0E-06
Benzene	1.5E-02	--	--	--	--	1.5E-02
Total PFAS (assume as toxic as PFOS)	2.7E-05	9.1E-06	--	2.5E-04	6.7E-05	3.5E-04
Dioxins and furans (WHO-TEQ)	1.8E-05	2.7E-05	5.1E-05	8.2E-05	6.4E-05	2.4E-04
PAHs (assuming BaP)	--	--	--	--	--	0.0E+00
Calculated non-threshold risk for each CoPC						
Benzene	1.2E-06	--	--	--	--	1.2E-06
PAHs (assuming BaP)	1.1E-09	1.9E-11	7.0E-11	5.7E-11	1.3E-14	1.2E-09

Calculated HI for each CoPC	Young children					
	Exposure pathway					Sum over all pathways
	Inhalation	Ingestion of soil	Dermal contact with soil	Ingestion of home-grown fruit and vegetables	Ingestion of eggs	
Hydrogen chloride (HCl)	6.2E-04					6.2E-04
Hydrogen fluoride (HF)	5.6E-05					5.6E-05
Hydrogen sulfide (H2S)	5.8E-03					5.8E-03
Arsenic	7.8E-05	2.5E-05	3.3E-08	1.5E-05	7.6E-08	1.2E-04
Cadmium	3.3E-02	1.9E-03	2.6E-06	3.5E-03	8.5E-07	3.8E-02
Chromium (Cr VI assumed)	1.5E-03	1.6E-03	2.2E-06	8.6E-04	6.6E-07	4.0E-03
Copper	2.4E-07	8.8E-06	1.2E-08	1.4E-05	6.7E-08	2.3E-05
Lead	1.0E-04	1.4E-04	1.9E-07	8.7E-05	2.5E-07	3.3E-04
Nickel	6.6E-03	1.0E-04	1.4E-07	6.9E-05	9.1E-08	6.7E-03
Selenium	2.4E-06	7.9E-05	1.1E-07	4.6E-05	1.1E-05	1.4E-04
Mercury (as inorganic and elemental)	6.9E-04	2.1E-03	4.1E-05	6.1E-03	7.6E-05	9.1E-03
Zinc	2.4E-07	8.3E-06	1.1E-07	2.7E-05	6.2E-08	3.6E-05
Benzene	1.5E-02	--	--	--	--	1.5E-02
Total PFAS (assume as toxic as PFOS)	2.7E-05	8.5E-05	--	5.1E-04	1.3E-04	7.6E-04
Dioxins and furans (WHO-TEQ)	1.8E-05	2.5E-04	1.0E-04	3.1E-04	1.3E-04	8.1E-04
PAHs (assuming BaP)	--	--	--	--	--	0.0E+00
Calculated non-threshold risk for each CoPC						
Benzene	1.2E-06	--	--	--	--	1.2E-06
PAHs (assuming BaP)	1.1E-09	3.6E-11	2.9E-11	4.4E-11	5.4E-15	1.2E-09

Exposure pathway	Non threshold risk		HI	
	Adults	Young children	Adults	Young children
Individual exposure pathways				
Inhalation (I)	1.2E-06	1.2E-06	0.064	0.064
Soil ingestion (SI)	1.9E-11	3.6E-11	0.00038	0.0064
Soil dermal contact (SD)	7.0E-11	2.9E-11	0.000064	0.00015
Ingestion of homegrown fruit and vegetables (F&	5.7E-11	4.4E-11	0.0029	0.012
Ingestion of homegrown eggs (E)	1.3E-14	5.4E-15	0.00016	0.00035
Multiple pathways (i.e. combined exposure pathways)				
I + SI + SD	1.2E-06	1.2E-06	0.064	0.071
I + SI + SD + F&V	1.2E-06	1.2E-06	0.067	0.082
I + SI + SD + E	1.2E-06	1.2E-06	0.065	0.071
I + SI + SD + F&V + E	1.2E-06	1.2E-06	0.068	0.082