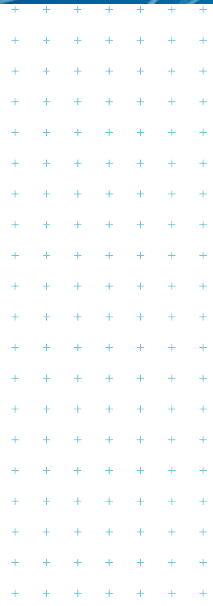




For a comprehensive understanding of this report, please also refer to the relevant s92 responses

# Auckland Regional Landfill Human Health Risk Assessment

**Prepared for**  
Waste Management NZ Ltd  
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## Executive summary

Tonkin & Taylor Ltd (T+T) has been commissioned by Waste Management NZ Ltd (WMNZ) to prepare a human health risk assessment (HHRA) for the proposed Auckland Regional Landfill. The main mechanisms by which contaminants can be released from the proposed Auckland Regional Landfill are via leachate and landfill gas. The purpose of this HHRA is to evaluate the potential risks to human health from possible exposure to leachate and landfill gas contaminants.

Some degree of emissions to air is unavoidable and will occur throughout the operational life of a landfill, progressively reducing once the landfill is completed. In contrast, the landfill lining system is designed to avoid the release of leachate and releases will only occur in the event of defects in the lining system. Conservative assumptions are typically made about the effectiveness of the lining system to recognise the possibility of defects, however these discharges may never occur in reality. Similarly, management measures will be in place to avoid leachate coming into contact with surface water, so ongoing release of leachate is not expected to occur. However, for the purpose of evaluating potential risks to human health, a range of conservative assumptions have been adopted, particularly in relation to potential releases of leachate to both groundwater and surface water.

The HHRA has considered the surface water features and land use in the vicinity of the proposed landfill and evaluated three representative receptor types and associated exposure pathways, namely:

- Residential receptor:
  - Inhalation of airborne contaminants;
  - Ingestion of drinking water supplied from roof collected water or the farm bore<sup>1</sup> (whichever generates the more conservatively high intake values);
  - Use of water from the farm bore to irrigate a vegetable garden combined with aerial deposition of contaminants onto soil, and subsequent ingestion of contaminants in both soil and home-grown produce.
- Wild food collector:
  - Harvesting of wild eels and watercress from the confluence of streams from Valley 1 and 2 (noting that in reality site management would discourage people from coming onto the landholdings in this manner); and
  - Incidental ingestion of a small quantity of surface water from the confluence of streams from Valley 1 and 2 during collection of eels or watercress.
- Public consumer eating beef or drinking cow's milk originating from a farm using the bore water from the WMNZ landholdings bore for stock watering.

The potential for health effects from cumulative exposure to compounds of potential concern in leachate and landfill gas has been considered for the first two receptors. For the public consumer, the assessment has estimated concentrations of per- and polyfluoroalkyl substances (PFAS) (as indicator substances) in beef meat and cow's milk and compared these to draft trigger levels for investigation set by Food Standards Australia and New Zealand.

The findings of the risk assessment are expressed in terms of the hazards associated with exposure to 'threshold compounds', which are substances that have a threshold of exposure (i.e. a safe level below which adverse effects are not expected to occur) and the incremental lifetime cancer risk associated with exposure to carcinogenic substances i.e. 'non-threshold compounds'.

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<sup>1</sup> The farm bore referred to here is the bore located within the WMNZ landholdings, as this is the closest groundwater take to the landfill.

The most significant exposure pathway identified in the risk assessment is the inhalation of airborne contaminants from the residual emissions from the flares and generators and fugitive escape of unburnt landfill gas. However, the cumulative hazard is estimated to be several orders of magnitude below the level where health effects would need to be investigated in more detail.

The potential for seepage of leachate to cause health effects from eating fish in the Kaipara Harbour has been identified as an issue that needs to be specifically addressed. As the health hazard associated with harvesting and eating eels at the stream confluence close to the landfill (within the WMNZ landholdings) is calculated to be acceptable, then it can be concluded that exposure from other fish species in the Hōteu River and Kaipara Harbour will also be acceptable (by a significantly greater margin).

Overall, the risk assessment has found that there are no unacceptable hazards or risks for any of the contaminants or pathways considered, both individually and cumulatively using a conservative screening approach.

## Acronyms and key terms

<b>Abbreviation</b>	<b>Meaning</b>
AAQC	Ambient air quality criteria
AEE	Assessment of Environmental Effects
ARL	Auckland Regional Landfill
AT	Averaging time
BCF	Bioconcentration factor
BW	Body weight
COPC	Contaminants of potential concern
CR	Consumption rate
DW	Dry weight
ED	Exposure duration
EF	Exposure frequency
ESLs	Effects Screening Levels
GCL	Geosynthetic Clay Liner layer
HHRA	Human Health Risk Assessment
HHRAP	Human Health Risk Assessment Protocol, published by the US EPA
HI	Hazard index
HQ	Hazard quotient
HRL	Health Risk Limits
IARC	International Agency for Research on Cancer
Kow	n-Octanol/Water Partition Coefficient
LFG	Landfill gas
LMP	Landfill Management Plan
LOD	Limit of detection
MAV	Maximum Acceptable Values
MDH	Minnesota Department of Health
MfE	Ministry for the Environment
MoH	Ministry of Health
MSW	Municipal solid waste
NEPC	National Environment Protection Council (Australia)
NES Soil	National Environmental Standard for Assessing and Managing Contaminants in Soil to Protect Human Health 2011
NMOCs	Non-methane organic compounds
NPS-FM	National Policy Statement for Freshwater Management
OEHHA	California Office of Environmental Health Hazard Assessment
PCBs	Polychlorinated biphenyls
PFAS	Per- and polyfluoroalkyl substances
POE	Point of Exposure
RBCA	Risk-Based Corrective Action software package
RMA	Resource Management Act
T+T	Tonkin & Taylor Ltd

TC	Tolerable concentration
TDI	Tolerable Daily Intake
TRS	Total reduced sulphur
UK EA	United Kingdom Environment Agency
US EPA	United States Environment Protection Agency
VOCs	Volatile organic compounds
WAC	Waste Acceptance criteria
WHO	World Health Organization
WMNZ	Waste Management NZ Ltd



## 1 Introduction

Waste Management NZ Ltd (WMNZ) has purchased land in the Wayby Valley area, north of Auckland, and proposes the construction and operation of a 25 Mm<sup>3</sup> landfill (known as the Auckland Regional Landfill) to provide for the disposal of municipal solid waste (MSW) for a period in excess of 35 years. A full description of the project is provided in Section 7 of the Assessment of Environmental Effects (AEE) Report. Tonkin & Taylor Ltd (T+T) has been commissioned by WMNZ to prepare a human health risk assessment (HHRA) for the Auckland Regional Landfill.

There are three main mechanisms for the release of contaminants from landfills, namely:

- Releases to air in the form of fugitive or partially combusted landfill gas;
- Seepage of leachate through the landfill lining system and the underlying soils into groundwater; and
- Escape of leachate into the site stormwater system (for example via a leachate breakout on a landfill face) and subsequent release to surface water via the stormwater treatment ponds.

Some degree of emissions to air is unavoidable and will occur throughout the operational life of the landfill, progressively reducing once the landfill is completed. In contrast, the landfill lining system is designed to avoid the release of leachate and releases will only occur in the event of defects in the lining system. Conservative assumptions are typically made about the effectiveness of the lining system to recognise the possibility of defects, however the associated discharges may never occur in reality. Similarly, management measures will be in place to avoid leachate coming into contact with surface water.

The purpose of the HHRA is to evaluate the potential risks to human health from possible exposure to these contaminants from the proposed Auckland Regional Landfill. This HHRA has been prepared to accompany the resource consent application for the landfill and, as such, considers the risk of off-site effects and does not consider possible effects on people working at the landfill (which are managed under the Health and Safety at Work Act).

Elements of the health risk assessment have been addressed in other technical reports prepared to support the AEE Report. These include:

- The Air Quality Assessment (Technical Report D), which evaluates the effects of odour, dust and combustion products in the exhaust from the generators and flare(s). The effects of combustion products are assessed using dispersion modelling and comparison with relevant ambient air quality standards and regional air quality targets; and
- The Hydrogeological Assessment (Technical Report E), which considers the potential for contaminants to seep through the lining system into soils and groundwater under the landfill. The concentrations of contaminants are estimated at various points of exposure using fate and transport modelling. The Hydrogeological Assessment compares the predicted exposure concentrations with ANZECC guidelines (for ecological effects and contact recreation) and drinking water guidelines.

The Ministry of Health has provided guidance (Ministry of Health, 1995) on how health impact assessments can be incorporated into assessments of environmental effects in the context of applications for resource consent under the Resource Management Act (RMA). Although this guidance is dated, the framework and key steps for undertaking a HHRA have not changed. A diagram outlining the key elements of a HHRA is shown in Figure 1.1 (reproduced from Environmental Health Australia, 2012). The key elements broadly comprise:

- Identifying the key issues for the risk assessment based on the nature of discharges and the potential exposure pathways and populations;

- Identifying the contaminants of potential concern and the dose response relationship;
- Estimating the potential exposure to contaminants via each of the relevant exposure pathways and media; and
- Characterising the risk associated with potential exposure to contaminants.

This report has been structured to address each of these key elements in turn.

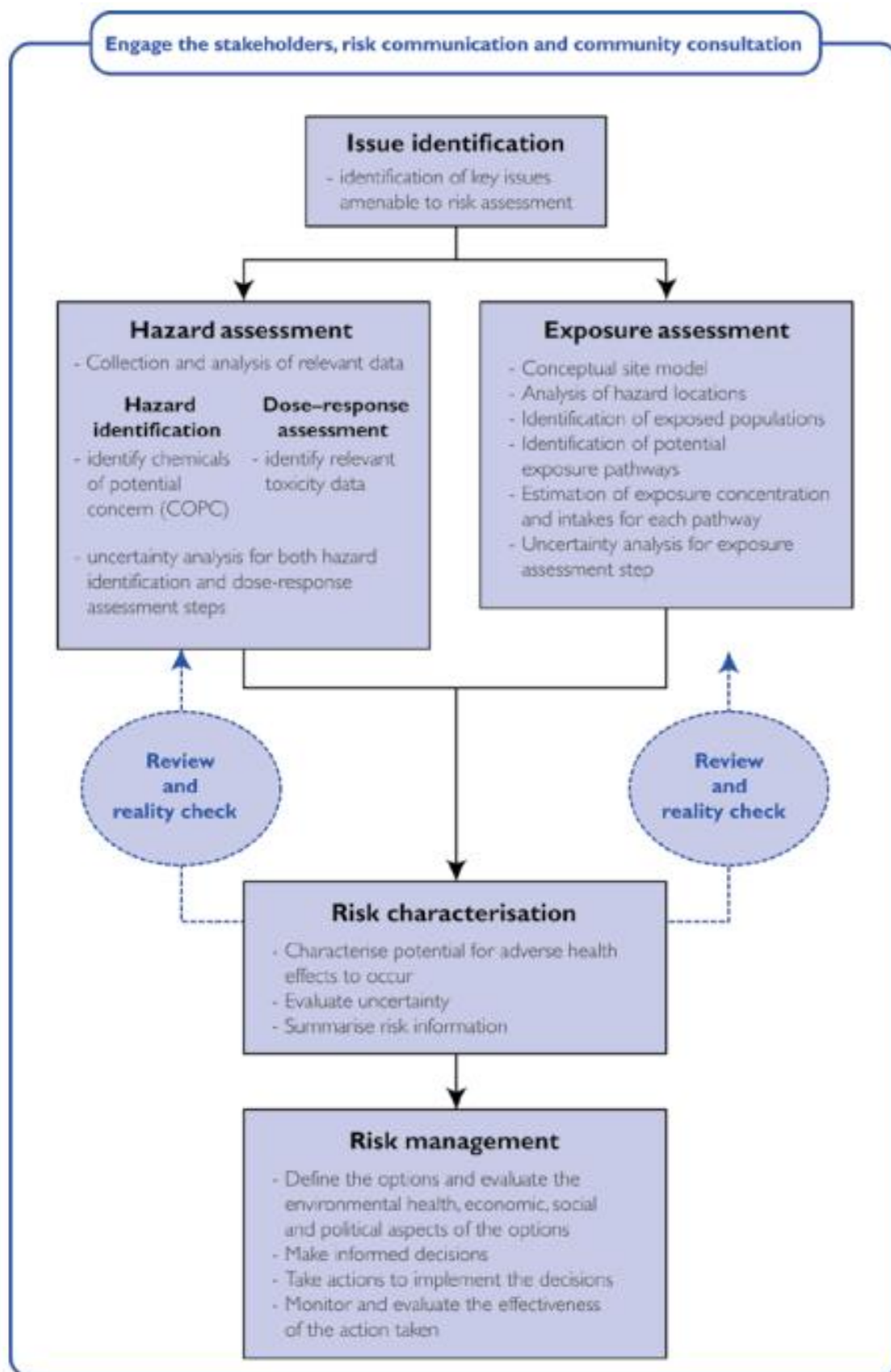


Figure 1.1: Environmental health risk assessment model (Environmental Health Australia, 2012)

## 2 Risk assessment methodology

This section provides a high level overview of the methodology adopted for this HHRA. Specific details of the methodology for each step of the risk assessment are set out in the relevant section of this report.

The most recent guidance on conducting HHRA in a New Zealand context is set out in the supporting documents for the development of the National Environmental Standard for Assessing and Managing Contaminants in Soil to Protect Human Health (NES Soil). The report *Methodology for deriving standards for contaminants in soil to protect human health* (Ministry for the Environment (MfE), 2011) sets out recommended default exposure (intake) parameters for a range of scenarios (for example residential or commercial exposures). For the contaminants addressed in the NES Soil, it also sets out recommended factors for the uptake of contaminants into home-grown produce. These recommended New Zealand-specific intake factors have been adopted in this HHRA.

The NES Soil is principally concerned with exposure to contaminants present in soil. It does not address fate and transport modelling to predict concentrations in groundwater or surface water that may result from a discharge. In this HHRA, the potential seepage of leachate through the landfill liner and underlying soils into groundwater has been modelled using the Groundwater Services Inc. Risk-Based Corrective Action (RBCA) software package. RBCA has been used to predict concentrations in groundwater and groundwater as it enters surface water at potential points of exposure (Hydrogeological Assessment, Technical Report E).

There is no New Zealand-specific guidance on detailed methods for undertaking risk assessments of discharges to air. The methodology used in this HHRA to evaluate exposure to airborne contaminants (from fugitive or partially combusted landfill gas) is based on the US EPA Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities (HHRAP) (US EPA, 2005). This is considered to be the most relevant international guidance as it includes detailed methods to estimate the concentration of contaminants derived from aerial transport and deposition, and a database of key compound-specific parameters for many of the contaminants of interest for this HHRA. The HHRAP was developed for use with the ISCST3 air dispersion model, but also provides for the use of other air dispersion models such as CALPUFF (US EPA, 2005. p3-6). For this HHRA, the CALPUFF dispersion model has been used to predict the transport of airborne pollutants emitted from the flares and generators, and fugitive emissions of landfill gas.

### 3 Previous health risk assessments for New Zealand landfills

Two health risk assessments have previously been undertaken for landfills in New Zealand – Hampton Downs Landfill (Woodward Clyde, 1999) and Redvale Landfill (T+T, 2003). The key characteristics of these landfills, in terms of scale and location, compared to the proposed Auckland Regional Landfill, are summarised in Table 3.1.

**Table 3.1: Comparison of landfill characteristics**

Landfill parameter	Hampton Downs Landfill	Redvale Landfill	Proposed Auckland Regional Landfill
Landfill footprint (ha)	Not available	59	58.5
Landfill volume (Mm <sup>3</sup> )	30 <sup>1</sup>	20	25
Waste input (Tonnes per Annum)	600,000 <sup>1</sup>	750,000	500,000
Distance from edge of footprint to closest sensitive receptor (m)	500	250	1,050

**Notes:**

1. Data from Waikato Regional Council website: <https://www.waikatoregion.govt.nz/services/regional-services/waste-hazardous-substances-and-contaminated-sites/solid-waste/what-happens-to-our-waste/waste-disposal-sites/>

Given the similarities between the scale of these landfills and the proposed Auckland Regional Landfill, we consider that the findings of the health risk assessments prepared for Hampton Downs and Redvale are broadly relevant to this project. Although it is noted that the Auckland Regional Landfill will have a significantly greater separation distance to sensitive receivers compared to these existing landfills.

The risk assessment for Hampton Downs only considered exposure to contaminants in landfill gas via inhalation. The report noted that exposure via deposition into roof collected drinking water was considered as a possible pathway, but preliminary calculations indicated it was not significant. The Redvale Landfill health risk assessment considered multiple exposure pathways to contaminants in landfill gas, including deposition onto roofs used for drinking water collection, and deposition into soil with subsequent dermal exposure and soil ingestion.

Both the Hampton Downs and Redvale Landfill health risk assessments concluded that:

- The discharges of threshold compounds does not pose a hazard to neighbouring residents; and
- The discharge of non-threshold compounds does not pose an unacceptable risk to neighbouring residents.

## **4 Identification of issues and receptor pathway assessment**

### **4.1 Introduction**

The main mechanisms by which contaminants can be released from a landfill are via leachate and landfill gas. In order for a contaminant to present a risk to an identified receptor, a complete exposure pathway must exist, i.e. a physical pathway for a receptor to be exposed to a contaminant. A pathway is considered to be incomplete if there is no practical way for the receptor to be exposed.

The following sub-sections consider the nature of contaminants associated with the different potential sources (landfill gas and leachate, which may be present in groundwater or surface water) and identifies the pathways that have the potential to pose an adverse human health effect.

### **4.2 Leachate**

#### **4.2.1 Management of leachate**

Leachate is the liquid produced when rain water percolates through the waste collecting dissolved and/or suspended matter from the waste as it passes through. The proposed Auckland Regional Landfill will incorporate a landfill lining system to contain the leachate, cover material to minimise the amount of water coming into contact with waste (and becoming leachate) and a collection system to capture the leachate for off-site disposal or recirculation back into the landfill (possibly after some of the water has been evaporated off to reduce its volume). The contaminants in leachate are predominantly metals (arsenic, cadmium, copper, chromium, lead, mercury, selenium and silver) and volatile organic compounds (VOCs) including benzene and chlorinated solvents.

The landfill lining system will comprise a high density polyethylene (HDPE) geomembrane overlying either a 600 mm compacted clay or a combination of a geosynthetic clay liner (GCL) and 600 mm compacted clay (see Engineering Report, Technical Report N). The various components of the composite lining system will work together to minimise the potential for leakage. For example, leakage through a pinhole in the geomembrane is expected to be blocked by the direct contact with the underlying GCL or clay layer. However, some leakage may occur through defects that may be present in the lining system, from either the installation or manufacturing process, although it is noted that modern manufacturing techniques have virtually eliminated manufacturing defects.

The rate of leakage through the lining system has been conservatively estimated assuming there are some defects in the geomembrane (refer Engineering Report, Technical Report N). The worst case scenario occurs at full development of the landfill (58.5 ha of lining system). The highest annual rate of potential leakage from the landfill under this scenario is approximately 3 m<sup>3</sup>, which is equivalent to an average of 8.2 L/day.

The low permeability clay layer will provide a further barrier to contaminants, as many of the chemical constituents in leachate will tend to bind onto the clay particles, and will not progress further through the soil profile. As a worst case scenario, this chemical attenuation has been conservatively ignored in calculating the source concentration of contaminants that could potentially enter the underlying groundwater system.

The concentrations of contaminants in leachate are expected to be greatest in the final years of waste placement and to decrease substantially during the aftercare period. The assumed concentrations in leachate adopted for this HHRA are based on data from Redvale Landfill, which is nearing completion and is therefore likely to be representative of worst case conditions. The leachate strength has been conservatively assumed to remain constant over time.

#### 4.2.2 Receptor pathway assessment for contaminants in leachate

A detailed description of the hydrogeological setting of the landfill and a contaminant exposure pathway assessment is set out in the Hydrogeological Assessment (Technical Report E). Any leachate that seeps through the lining system could reach groundwater in the geological unit known as the Upper Pakiri Formation under the landfill or migrate vertically through preferential pathways (assuming these exist), to the deeper regional groundwater system. The Hydrogeological Assessment evaluated five Points of Exposure (POE#1 to POE#5) to contaminants in leachate. These POE are referenced below and shown in Figure HG-F8, which is reproduced at the end of this report.

There is an existing farm bore located on 1232A State Highway 1. This bore is located approximately 1.9 km to the west of the proposed landfill footprint. No information is available about this bore, e.g. depth, pumping rate, etc. However, WMNZ has confirmed that they use the bore, which provides potable water to the farm cottage and woolshed (POE#5) as well as stock watering (POE#4). The regional groundwater flows to the west toward this farm bore. The use of water from the farm bore to feed domestic chickens has also been considered specifically in relation to per- and polyfluoroalkyl substances (PFAS), as these are expected to be the most sensitive contaminants given their mobility, persistence and toxicity, and their known ability to bioaccumulate in animals. This deeper groundwater could continue to migrate and eventually discharge into the Hōteō River (POE#3). The potential for contact recreation exposure has been assessed at POE#3.

Local groundwater in the Upper Pakiri Formation, directly beneath the landfill, follows the topography of the land and flows northwest. This groundwater could reach surface water in the vicinity of the confluence of streams from Valley 1 and 2, approximately 360 m from the landfill footprint (POE#1). This is also a potential point of exposure for leachate in stormwater, as discussed in the following sub-section. Direct human exposure to surface water at the confluence of streams from Valley 1 and 2 is unlikely as it is within the WMNZ landholding. However, this location has been considered in the HHRA as the worst case POE for uptake into freshwater food sources, such as eels (representative of all fish species) and water cress. The uptake of contaminants into eels has only been considered in relation to PFAS, for the reasons given in the previous paragraph related to chickens. The uptake into watercress has only been considered for arsenic as an indicator compound, due to the known ability of arsenic to bioaccumulate in water cress.

The Hōteō River discharges into the Kaipara Harbour, which is a large enclosed estuarine harbour that provides a key snapper fish breeding ground. The findings of the assessment of PFAS in eels in the stream confluence can be used to infer hazards associated with uptake of PFAS in other fish present in the stream, and of PFAS reaching the Kaipara Harbour and subsequent uptake into fish. PFAS concentrations in the Harbour as a result of leachate seepage would be many order of magnitude lower than in the stream confluence due to dilution.

Regional groundwater flows to the west toward the Hōteō River. Watercare currently supplies Wellsford with water taken from the Hōteō River, upstream of the confluence of the Valley 1 stream and the Hōteō River, at 362 Wayby Valley Road. There are a number of consented surface water takes from the Hōteō River, which are believed to be predominantly used for agricultural purposes, in particular for irrigation, and are unlikely to be used for potable water. Based on this, direct human exposure to contaminants that may reach the Hōteō River by entrainment in regional groundwater is likely to be restricted to recreational contact exposure. Use of water from the Hōteō River for irrigation of crops or stock watering has not been considered because the predicted concentration of contaminants in groundwater entering the River are lower than in the farm bore (i.e. the assessment of use of farm bore water for stock irrigation will be more conservative).

## **4.3 Landfill gas**

### **4.3.1 Management of landfill gas**

Landfill gas, which comprises an approximately 50:50 mixture of methane and carbon dioxide and traces of volatile organic compounds and reduced sulphur compounds, is generated as the waste decomposes. The Auckland Regional Landfill will incorporate a landfill gas collection system, comprising a network of collection wells and pipework to which a suction pressure will be applied. The lining system and landfill capping, including temporary capping on active working areas, increases the efficiency of the landfill gas collection system. Combustion of the captured landfill gas will occur in electricity generators at the Renewable Energy Centre, with any residual landfill gas being flared.

Landfill gas poses an on-site hazard due to its flammability which is a safety matter for the site to manage. The main constituents of landfill gas, methane and carbon dioxide, are not toxic at levels that would be experienced after dispersion beyond the WMNZ landholding. However, landfill gas can contain a range of other chemical constituents at low concentrations, principally VOCs and reduced sulphur compounds, which are described in Technical Report D. Discharges of landfill gas are minimised by the use of a network of landfill gas collection wells and combustion of the captured landfill gas to destroy methane and other compounds. The main sources of contaminant discharges to air from the Landfill will be:

- Fugitive landfill gas, particularly from the active tipping face; and
- Residual emissions from combustion of landfill gas in the flares and generators.

Emissions of combustion products (carbon monoxide, oxides of nitrogen, sulphur dioxide and fine particulate matter) from the flares and generators are considered in the Air Quality Assessment by dispersion modelling and comparison with relevant ambient air quality standards and guidelines. Therefore, potential health impacts of emissions of these combustion products have already been considered elsewhere and so are not considered further in this HHRA.

### **4.3.2 Receptor pathway assessment for contaminants in landfill gas**

Exposure to contaminants in landfill gas could occur through direct inhalation, from deposition of contaminants onto roofs used for drinking water supply, or deposition onto soils used to grow food or graze animals.

Areas to the northeast, east and south of the landholding are dominated by plantation forestry. Land uses to the west and north-west of the landholding are predominantly agricultural, comprising dairy, beef and sheep farms, and lifestyle blocks. The closest house to the Renewable Energy Centre and northern extent of the landfill footprint is at 302 Wilson Road (identified as receptor 302 in the dispersion modelling), with a separation distance of approximately 1590 m. The closest houses to the southern extent of the landfill footprint are 792, 776 and 762 State Highway 1 (near the intersection with Crowther Road) with separation distances in the range 1050 to 1160 m.

The dispersion and deposition modelling has predicted impacts at the two receptors identified as having the highest predicted concentrations in the Air Quality Assessment being Receptor 32 (302 Wilson Rd) and Receptor 28 (109 Waiwhiu Rd). The highest concentrations and deposition rates were predicted at Receptor 32 and this was adopted as the point of exposure for residential exposure to airborne contaminants.



## 4.4 Surface water

### 4.4.1 Management of surface water

The overall approach to surface water management as part of the landfill development is as follows:

- Any surface water that comes into contact with waste will be treated as leachate and is kept separate from clean surface water;
- Surface water from up-gradient of the landfill cells and development areas will be diverted around the landfill and development works areas; and
- All surface water (excluding any water treated as leachate because it has come in contact with waste) will pass through the stormwater treatment system where it will be monitored prior to release to the receiving environment.

Any surface water that drains onto the open working face and associated areas including the tipping pad will be treated as leachate and managed as such. No treatment or disposal of leachate via the stormwater ponds is proposed at the landfill.

Leachate will tend to percolate through the waste towards the liner and collection system, however there is the potential for localised perching of leachate above areas of lower permeability waste. Where this occurs, and there are defects in the cover material, there can be “breakouts” of leachate through the cover on sloped areas of the landfill surface. Procedures or systems will be implemented to monitor and identify potential leachate breakouts or contamination of surface water including:

- Weekly inspections of the landfill surface to look out for any evidence of leachate breakouts and any malfunctioning or leaking associated with the reticulation system;
- Continuous monitoring of conductivity at the inlet to the ponds as an indicator of the presence of leachate in surface water, including automated notification from site-operated telemetry system if pond inlet conductivity exceeds the trigger limits; and
- Routine monitoring of a broad range of contaminants at pond outlets including those that may indicate the presence of leachate and those included to monitor for changes in the receiving environment.

The proposed monitoring programme includes monitoring at the inlet to the ponds for leachate indicators, and regular outlet monitoring for a full suite of contaminants. The main indicators of leachate in surface water include changes in pH and elevated conductivity, boron, ammoniacal nitrogen (ammonia) and chlorides. Conductivity can be monitored continuously while ammonia, boron and sulphates require samples to be analysed at a laboratory. Analysis of pH can be done on a regular basis with a portable meter. Therefore, continuous conductivity monitoring and frequent pH monitoring e.g. weekly, is used at the inlet to the ponds as the first indicator of leachate in surface water.

If a leachate breakout is identified, or leachate is identified in surface water through the continuous monitoring, the landfill will have contingency procedures in place, which are included in the contingency section of the surface water section of the Landfill Management Plan (LMP) and the Emergency Management Plan. These measures include ceasing any discharges from the ponds with the pond outlets fitted with gate valves to prevent on-going discharges.

If monitoring of ammonia, sulphates or boron indicate elevated levels in the ponds or discharge, an investigation will be undertaken to identify the source. It would be unlikely for these parameters to be elevated due to leachate contamination without changes in the conductivity and pH to also occur.

The potential maximum volume of leachate that could be in the final pond at the discharge point has been calculated by assuming that the water is at the trigger level for ammonia and that the trigger

level is the published attribute state values in the National Policy Statement of Freshwater Management (NPS-FM). Trigger levels for the remaining indicator parameters will be based on the baseline monitoring which is currently still underway and therefore are not yet able to be determined.

Based on the typical ammonia concentration within leachate, the permanent water volume in the wetland and the rainfall and associated discharge volume, the maximum volume of leachate discharged from the site into surface water has been calculated as 5 L/day for the purpose of a worst case scenario in this HHRA.

#### **4.4.2 Receptor pathway assessment for contaminants in surface water**

While the proposed controls seek to avoid any discharge of leachate into the surface water system on-site and to undertake contingency actions in the event that this does occur, there is the potential for low levels of contaminants to be present in the ponds below the trigger levels.

The risk of leachate being discharged via surface water is low, and in the unlikely event it did occur, identification via the continuous monitoring would enable appropriate actions to be taken to avoid leachate being discharged. Nevertheless, the discharge of surface water containing low levels of leachate contamination (at levels below the trigger levels to avoid effects on the receiving environment) has been included within the receptor pathways.

Surface water from the site will discharge via the wetland into the immediate downstream environment which provides habitat for a range of fish species, including eels that may be collected for human consumption. As noted above, harvesting and eating eels collected from this stream immediately downstream of the landfill has been considered as an exposure pathway. The possibility of the stream immediately downstream of the landfill being used for stock-watering, for example on Spindler Road farms, has also been considered.

### **4.5 Other issues considered**

#### **4.5.1 Dust**

Dust can be generated from earthworks and construction activities or from placement of dusty waste in the landfill. Dust from earthworks and construction will generally be free of any appreciable contamination as it will be the movement of materials already existing onsite and will not be a source of exposure to contaminants. Dust emissions from placement of contaminated waste at the working face are managed by application of water, if needed, and rapid mixing and cover with other waste materials, as well as pre-acceptance checks to avoid dusty waste in the first place. Based on the use of these well-established controls, dust emissions from placement of waste will be negligible beyond the immediate working area.

Lightly contaminated soils are used as daily cover at Redvale Landfill and it is proposed that this will also be the case at the Auckland Regional Landfill. The potential for dust from these soils when used as cover materials to be blown beyond the landfill footprint in any appreciable quantity is considered negligible. This is because of low concentrations in the original soil, distance to receptors, and tendency for forests to filter the air flow.

WMNZ has developed acceptance criteria for soils to be used as cover material. The acceptance criteria are principally based on relevant New Zealand guidance, being:

- The acceptance criteria for soils for recreational land use in the NES Soil; and
- The acceptance criteria for soils for industrial/commercial land use in the Guidelines for Assessing and Managing Petroleum Hydrocarbon Contaminated Sites (MfE, 1999). The hydrocarbon guidelines do not include acceptance criteria for recreational land uses, and the

industrial/commercial criteria are considered to be more appropriate to the nature of the site than the residential criteria.

Where there is no relevant value in the NES Soil, the Australian National Environment Protection Council (NEPC) Health Investigation Levels for recreational land uses has been used. The acceptance criteria for soils to be used as daily cover are set out in Table 4.1.

**Table 4.1: Acceptance criteria for soil for cover material**

Compound	Acceptance criteria (mg/kg)	Source
Arsenic	80	NES Soil (Recreational)
Boron	<10,000	NES Soil (Recreational)
Cadmium	400	NES Soil (Recreational)
Chromium VI	2,700	NES Soil (Recreational)
Cobalt	300	NEPC (Recreational)
Copper	<10,000	NES Soil (Recreational)
Lead	880	NES Soil (Recreational)
Mercury	1,800	NES Soil (Recreational)
Nickel	1,200	NEPC (Recreational)
Zinc	30,000	NEPC (Recreational)
C7-C9	500	Hydrocarbon contaminated soil guidelines (commercial/industrial)
C10-C14	1700	Hydrocarbon contaminated soil guidelines (commercial/industrial)
Aldrin	10	NEPC (Recreational)
Dieldrin	70	NES Soil (Recreational)
4,4, DDT	400	NES Soil (Recreational)
B(a)P equiv.	40	NES Soil (Recreational)

These acceptance criteria are based on values that are protective of the public using recreational areas (or workers in the case of petroleum hydrocarbons). Combined with the negligible potential for dust to be transported beyond the landfill footprint, contaminants in soils used for cover material are considered very unlikely to cause off-site effects. Consequently exposure to contaminants derived from dust has not been considered as a relevant pathway in this HHRA.

#### 4.5.2 Direct exposure to waste

The landfill will not be open to the public and therefore direct exposure of the public to waste is not a complete pathway.

Landfills can attract seagulls and a range of vermin, including rodents, insects and wild cats. Larger animals, such as pigs or goats, will be present on the land around the landfill, including in the forestry block and neighbouring Sunnybrook Reserve. To minimise pest numbers, control measures will be undertaken around the landfill footprint and the bin exchange area. Control methods may include physical controls such as fencing or traps, or bait, which will be implemented for the duration of waste disposal activities at the site.

Smaller animals could be a potential vector for pathogens but are unlikely to be a source of human exposure to contaminants. Goats and pigs may be hunted for food and therefore the potential for these animals to be a dietary exposure pathway has been considered as follows.

To the extent that pest fencing is used, this will minimise access of larger animals to the landfill. Larger animals are also unlikely to approach the landfill working area i.e. the only place that waste is exposed during working hours due to the presence of people and machinery. Outside working hours, all waste at the working face will be covered with a minimum 150 mm of daily cover. Even if animals were to root around on the landfill surface, they are likely to target organic wastes (e.g. food scraps) rather than wastes containing appreciable levels of contaminants. There is minimal potential for animals to encounter leachate, as the leachate is collected from the base of the landfill and stored in an enclosed system. Taking all these factors into account, it is unlikely that larger animals would be exposed to any significant levels of contaminants and therefore, it is an even more remote possibility that eating wild animals, such as pigs or goats, could present any risk to human health.

#### 4.5.3 Unplanned releases

A Risk Management Assessment has been carried out for the Auckland Regional Landfill resource consent application (Technical Report S, Volume 2). The Risk Management Assessment identifies a number of scenarios that could give rise to unplanned discharges, such as:

- The lining system failing due to differential settlement, an earthquake or other mechanism;
- Discharge of contaminated stormwater if a stormwater pipe or swales fail/block, up-catchment stormwater cannot discharge, and dammed water accumulates resulting in interaction with waste, creating leachate;
- Air emissions associated with a sub-surface landfill fire; and
- Waste acceptance criteria not met and hazardous waste received, resulting in higher levels of contaminants in leachate or discharges to air.

Risk is a combination of the likelihood of an event occurring and the consequences if it did occur. The risk of each of the events has been evaluated, first without controls in place, and then considering the effectiveness of the proposed controls at reducing the likelihood and/or the consequences.

The events identified above were assessed as having a “Moderate” residual risk after controls have been implemented, and the Assessment concludes that the risk is therefore considered tolerable, provided there is active management to reduce risk to as low as reasonably practicable.

Exposure to contaminants in the event of a significant unplanned release would be short term and can be mitigated, if necessary, by measures such as temporary evacuation (e.g. in the event of a large fire) or restrictions on collecting food from surface water. On this basis, further quantification of the consequences, in terms of effects on people’s health if an unplanned discharge of this nature were to occur, is not considered necessary in the context of this HHRA.

#### 4.6 Summary of exposure pathway assessment

The source pathway receptor model is summarised in Table 4.2 and illustrated diagrammatically in Figure 4.1.

For the purposes of evaluating the hazard or risk associated with cumulative exposure, three receptor types have been identified:

- Residential receptor:
  - Inhalation of airborne contaminants;

- Ingestion of drinking water supplied from roof collected water or the farm bore (whichever generates the more conservatively high intake values); and
- Use of water from the farm bore to irrigate a vegetable garden combined with aerial deposition of contaminants onto soil, and subsequent ingestion of contaminants in both soil and home-grown produce.
- Wild food collector:
  - Harvesting of wild eels (representing other fish species present in the stream) and watercress from the confluence of streams from Valley 1 and 2; and
  - Incidental ingestion of a small quantity per day<sup>2</sup> of surface water from the stream.
- Public consumer eating beef or drinking cow's milk from a farm using either bore water or water from the confluence of streams from Valley 1 and 2 for stock watering.

Other exposure pathways, such as exposure via contact recreation, have been considered (see Table 4.2), but are not additive to these key receptor exposures.

The potential impact of PFAS on fish species in the Hōteō River and Kaipara Harbour have been considered as a pathway as follows. The uptake of PFAS into eels has been evaluated based on the modelled PFAS concentration at the stream confluence immediately downstream of the landfill (POE#1). The surface water from the stream will be diluted by large volumes of water in the Hōteō River, and by even larger volumes in the Kaipara Harbour. Therefore concentrations in the Hōteō River and Kaipara harbour will be significantly lower. In addition, the bioaccumulation of PFAS in eels is significantly higher than other fish species. Therefore, it is considered that uptake of PFAS by eels at the stream confluence represents the worst case exposure via freshwater fish or seafood.

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<sup>2</sup> 100ml

**Table 4.2: Summary of source pathway exposure assessment**

Source	Pathway	Receptor	Contaminants	Assessed point of exposure
Leachate within the landfill	Potential discharge of leachate from surface water runoff in the stormwater treatment system	Wild food collector of eels (fish) and water cress for consumption	PFAS in eel consumption (representing probable worst) Arsenic in water cress consumption (high bioaccumulation in freshwater vegetation)	Confluence of streams from Valley 1 and 2: POE#1 from Hydrogeological Assessment
		Public consumers of beef from cattle watered with groundwater from the stream	PFAS	POE#1
		Public consumers of milk from dairy cattle watered with groundwater from the stream	PFAS	POE#1
	Potential seepage of leachate through the landfill lining system and migration of contaminants via diffuse flow in the groundwater in the higher elevations of the Pakiri Formation or via preferential pathways	Wild food collector of eels (fish) and water cress for consumption	PFAS in eel consumption (representing probable worst) Arsenic in water cress consumption (high bioaccumulation in freshwater vegetation)	POE#1
	Potential seepage of leachate through the landfill lining system and migration of contaminants via the groundwater in the deep regional groundwater	Surface water users (swimming)	All priority contaminants in leachate	Recreational users in the Hōteu River: POE#3 from Hydrogeological Assessment
		Residential receptor using farm bore for domestic chickens and vegetable garden	All priority contaminants in leachate PFAS only for domestic chickens	Residential receptor. Farm bore supplying water for vegetable garden: POE#4 from Hydrogeological Assessment (Technical Report E)

Source	Pathway	Receptor	Contaminants	Assessed point of exposure
		Residential receptor with potable supply from groundwater	All priority contaminants in leachate	Farm bore supplying potable drinking water: POE#5 from Hydrogeological Assessment
		Public consumers of beef from cattle watered with groundwater from farm bore	PFAS only	POE#4
		Public consumers of milk from dairy cattle watered with groundwater from farm bore	PFAS only	POE#4
Landfill gas collected within the landfill and fugitive emissions through landfill surface	Residual emissions of contaminants from flare and generators and fugitive emissions of landfill gas from the landfill surface. Aerial transport of contaminants	Inhalation of airborne contaminants at residential receptor	All priority contaminants in landfill gas	Residential receptor Most sensitive residential receptor from dispersion modelling
	Deposition of airborne contaminants onto roof through wet and dry deposition processes	Residential receptor with potable supply from roof collected water	All priority contaminants in landfill gas	Residential receptor Most sensitive residential receptor from dispersion modelling
	Deposition of airborne contaminants onto soil through wet and dry deposition processes	Residential receptor with vegetable garden	All priority contaminants in landfill gas	Residential receptor Most sensitive residential receptor from dispersion modelling

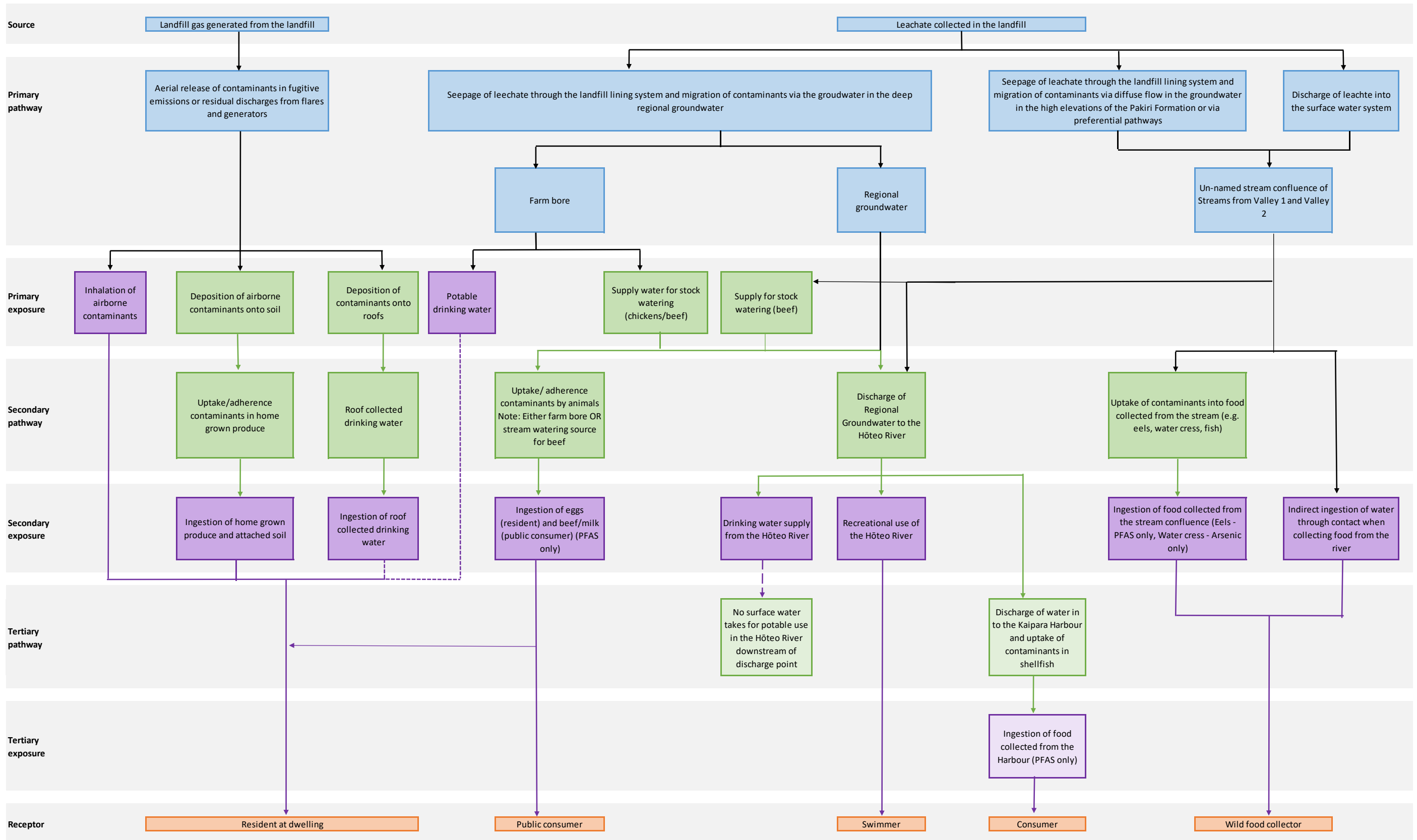


Figure 4.1: Diagram of conceptual source pathway receptor model



## 5 Contaminants of potential concern

### 5.1 Introduction

Monitoring data from Redvale Landfill has been used as the primary source of contaminant concentrations in air and water, supplemented by published data or other sources as described in the following sub-sections. Data from Redvale Landfill is expected to be broadly representative of landfill gas and leachate quality at the Auckland Regional Landfill during its operational life, based on similarities in the nature of waste accepted and landfill management practices. The rate of landfill gas generation, and the strength of leachate, will both reduce once the landfill is closed.

### 5.2 Contaminants and source concentrations in leachate

The contaminants of potential concern in leachate are those discussed in the report documenting the derivation of Waste Acceptance Criteria (Technical Report O) for the proposed Auckland Regional Landfill (see details in Section 8.3 of the Hydrogeology Assessment (Technical Report E)).

Contaminant concentrations in leachate have been adopted from the maximum concentrations measured in leachate at Redvale Landfill. Where concentrations were not detected, half of the detection limit was used.

Where compounds were measured in leachate but were below the limit of detection, the representative concentration was calculated from half the limit of detection.

For chlordane (total alpha and gamma), methoxychlor, toxaphene, 2,4-dichlorophenoxyacetic acid, 2,4,5-trichlorophenoxyacetic acid, and pyridine, the Waste Acceptance Criteria (WAC) was used to represent source leachate concentrations. This is a conservative estimate as it assumes that the leachate contains this contaminant at the maximum allowable concentration. These substances have been evaluated separately from the other priority contaminants to investigate the appropriateness of the Waste Acceptance Criteria (see Section 9.4).

### 5.3 Contaminants and source concentrations in landfill gas

Landfill gas contains trace amounts of a range of VOCs. Most of the VOCs in landfill gas occur because they were present in small amounts in the waste received, however smaller amounts can also be generated by biological processes and chemical reactions within the landfill. In relation to landfill gas, these compounds are sometimes also referred to as “non-methane organic compounds” (NMOCs). The US EPA default concentration for total NMOCs in landfill gas is 595 parts per million (expressed as hexane) - this value is higher than the 10 year average across Redvale and Whitford Landfills and is therefore considered a fair value for this report.

The US EPA has prepared list of default concentrations for a range of compounds that are known to be present in landfill gas. These are set out in the Compilation of Air Pollutant Emission Factors (AP42 database) (US EPA, 2008). WMNZ’s routine testing of landfill gas focusses on the contaminants identified in the AP42 database.

The UK Environment Agency (UK EA) has prepared a list of priority contaminants for monitoring in landfill gas, including maximum and average concentrations. The UK EA list is heavily influenced by data from landfills where there has been co-disposal of municipal solid waste with industrial wastes. Therefore the UK EA list includes compounds unlikely to be present in appreciable quantities at the Auckland Regional Landfill due to the proposed stricter Waste Acceptance Criteria. Where there are contaminants in the UK EA list that are not on the US EPA list, we have reviewed the likelihood of them being present at the Auckland Regional Landfill and included them in the screening assessment where appropriate.

The representative maximum concentrations were chosen based on measured concentrations in landfill gas at Redvale Landfill. As the concentrations are intended to represent the long term average, the second highest concentration from the data was generally adopted provided there was an adequate dataset. Where there was limited data, the highest value was generally adopted. Where a compound has been analysed in the landfill gas, but has never exceeded detection limits, the adopted concentration was half the limit of detection (LOD). There were two exceptions, methyl ethyl ketone and mercury, where the limit of detection of the method was too high to be useful and therefore the default concentrations in landfill gas from the UK EA were adopted.

## **5.4 Identification of contaminants of potential concern**

### **5.4.1 Introduction**

When conducting a risk assessment where there is potential for exposure to a wide range of substances, it is standard practice to undertake an initial screening assessment. The purpose of the screening assessment is to identify the priority contaminants (contaminants of potential concern or COPC) for the HHRA. The intent is to identify the most important contaminants taking into account their toxicity, concentration and fate and transport characteristics (particularly for contaminants in leachate). If the HHRA shows that the effects of this sub-set of priority contaminants are acceptable, then it can be inferred that the risks posed by contaminants that are present in smaller quantities and that are less toxic will also be acceptable.

### **5.4.2 Priority substances for consideration**

Contaminants of the following type were selected for detailed consideration in the HHRA without further screening:

- Genotoxic carcinogenic compounds (i.e. that can cause cancer by acting directly on the genetic material in cells) in either leachate or landfill gas (see Section 6.2.1); and
- Per- and polyfluoroalkyl substances (PFAS) were included because they are emerging contaminants of concern due to their mobility and environmental persistence. The groups of substances included in the assessment are PFOS, PFHxS and PFOA (see Appendix G for more detail).

### **5.4.3 Screening assessment based on source concentration and relative toxicity**

A sub-set of the remaining threshold compounds has been selected as COPC, taking into account the concentration of each compound in leachate and landfill gas (see previous sub-sections), and the toxicity of each compound. The screening methodology and list of screened priority contaminants is included in Appendix B. A summary of the methodology used is set out below.

Screening toxicity factors have been obtained from the following sources:

- For compounds in landfill gas - the 2016 Texas Effects Screening Levels (ESLs)<sup>3</sup>; and
- For compounds in leachate - New Zealand Maximum Acceptable Values (MAV) in drinking water (Ministry of Health (MoH), 2018) or World Health Organization drinking water guidelines, or values derived in the supporting information for the MAV or WHO guidelines. As a third tier source, the Minnesota Department of Health (MDH) chronic Health Risk Limits (HRL)<sup>4</sup> have been adopted.

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<sup>3</sup> <https://www.tceq.texas.gov/toxicology/esl>

<sup>4</sup> The MDH values were selected as a secondary source of screening toxicity factors for leachate as they are referenced extensively in the chemical datasheets that support the Ministry of Health Guidelines for drinking water quality (Ministry of Health, 2017). <https://www.health.state.mn.us/communities/environment/risk/guidance/hrltype.html#summary>.

The MDH values were selected as a secondary source of screening toxicity factors for leachate as they are referenced extensively in the chemical datasheets that support the Ministry of Health Guidelines for drinking water quality (Ministry of Health, 2017).

The ratio of the source concentration and the screening toxicity value has been calculated for each contaminant considered. The threshold compounds in leachate and landfill gas have then been separately ranked from greatest to least potential for health effects (not taking into account fate and transport characteristics) (see Appendix B).

The COPC have been selected as follows:

- The ten highest ranked compounds in landfill gas;
- The 15 highest ranked compounds in leachate, including the seven compounds evaluated based on Waste Acceptance Criteria; and
- Substances that are likely to be present in both landfill gas and leachate within the top 20 highest ranked substances (note: this did not add any new substances to the COPC list).

#### **5.4.4 Consideration of fate and transport characteristics**

The potential for substances to bioaccumulate has also been considered based on a review of the n-Octanol/Water Partition Coefficient (Kow). Values of Kow are unitless and usually expressed as logKow, a relative indicator of the tendency of an organic compound to adsorb to soil and living organisms. Substances with high logKow values tend to adsorb more readily to organic matter in soils or sediments. Chemicals with very high logKow values are of greater concern because they may have the potential to bio-concentrate in living organisms. All organic compounds with logKow greater than 4, with the exception of PCBs (polychlorinated biphenyls) have been included as COPC (see further discussion below regarding PCBs).

For contaminants present in leachate, consideration has also been given to the mobility of these contaminants in the environment. LogKow can also be used as a simple indicator of mobility, as organic compounds with a low logKow will tend to bind less strongly onto the soil layer beneath the landfill and therefore be more available for transport into groundwater. The COPC list has been checked to ensure that contaminants with a low logKow have also been included in the COPC.

#### **5.4.5 Polychlorinated biphenyls**

A programme to withdraw PCBs from service in New Zealand and export them for disposal overseas by high temperature incineration was initiated in the mid-1980's. Under the Hazardous Substances and New Organisms legislation, all PCBs were required to be withdrawn from use and destroyed no later than 2016. For these reasons, the amount of PCBs still present in New Zealand will be very small. There are specific requirements under the Hazardous Substances (Storage and Disposal of Persistent Organic Pollutants) Notice 2004 for the management of any residual PCBs that may be identified in the future. These requirements include not allowing PCBs to be disposed to landfill. Therefore, it is considered very unlikely that PCBs would be found in appreciable quantities in leachate at a new landfill in New Zealand. Annual tests for PCB at Redvale consistently do not find any PCB above the limit of detection.

#### **5.4.6 List of compounds of potential concern**

The final list of COPC, derived using the approach set out in the previous sub-sections, is summarised in Table 5.1. The table also identifies the classification of the substance by the International Agency for Research on Cancer (IARC) as to its carcinogenicity, whether the substance has been assessed as having a genotoxic mechanism for carcinogenicity (see Section 6.2) and whether the substance is present in landfill gas or leachate.

The IARC classification system is:

- Group 1: Carcinogenic to humans
- Group 2A: Probably carcinogenic to humans
- Group 2B: Possibly carcinogenic to humans
- Group 3: Not classifiable as to its carcinogenicity to humans

**Table 5.1: Contaminants of potential concern**

Compound	IARC classification	Assessed as genotoxic carcinogen	Source media	
			Landfill gas	Leachate
Acetaldehyde	Group 2B	Yes	X	-
Acrylonitrile	Group 2B	Yes	X	-
Arsenic	Group 1	Yes	-	X
Benzene	Group 1	Yes	X	X
Carbon tetrachloride (tetrachloromethane)	Group 2B		X	0
Chlorobenzene	Not assessed		X	0
Chloroethane	Group 3		X	0
Chromium	Depends on valence state		-	X
Cresols, total	Not assessed		-	X
1,2-dibromoethane (ethylene dibromide)	Group 2A	Yes	X	X
1,2-dichloroethane	Group 2B	Yes	X	X
Dichloromethane	Group 2A	Yes	X	X
1,2-dichloropropane	Group 1	Yes	X	X
Endrin	Group 3		-	X
Formaldehyde <sup>1</sup>	Group 1		X	NA
Hexachlorobenzene	Group 2B		NA	X
Hexachlorobutadiene	Group 3		NA	X
Hexachloroethane	Group 2B		NA	X
Lead	Group 2B		-	X
Methyl ethyl ketone (2-butanone)	Not assessed		0	X
Methyl isobutyl ketone	Group 2B		X	NA
PFOS/PFHxS	Not assessed		-	X
PFOA	Group 2B		-	X
Pentachlorophenol	Group 1	Yes	-	X
Styrene	Group 2A		X	0
1,1,1,2-tetrachloroethane	Group 2B		0	X
Tetrachloroethylene	Group 2A		X	0
Toluene	Group 3		X	0
Total reduced sulphur	Not assessed		X	-
Trichloroethylene	Group 1	Yes	X	X
Trichloromethane (chloroform)	Group 2B		X	0
Vinyl chloride	Group 1	Yes	X	X
Xylene	Group 3		X	0

See table notes and key on following page.

**Table notes:**

1. Formaldehyde is considered a genotoxic carcinogen at high doses via inhalation. However, the more sensitive health endpoint is respiratory effects.

**Table key:**

- X Identified as a priority contaminant through the screening process
- 0 Not identified as a priority contaminant in this media, but included because it is present in both media
- NA No analytical data available
- No analytical data and unlikely to be present in this media due to physical characteristics (e.g. volatility)

### 5.4.7 Evaluation of Waste Acceptance Criteria

As discussed in Section 5.2, there is a group of substances that have been included in the HHRA calculations for the purpose of evaluating the appropriateness of the Waste Acceptance Criteria. These compounds are listed in Table 5.2.

These substances all have low volatility and their acceptance into the landfill is controlled to ensure they are only present in leachate at low concentrations. Due to their low volatility, these compounds will not be present at any appreciable level in landfill gas.

**Table 5.2: Contaminants evaluated at Waste Acceptance Criteria limits**

Compound	IARC classification	Assessed as genotoxic carcinogen	Assessed as threshold compound
Chlordane	Group 2B		X
2,4-Dichlorophenoxyacetic acid	Group 2B		X
Heptachlor	Group 2B		X
Methoxychlor	Group 3		X
Pyridine	Group 3		X
Toxaphene	Group 2B	X	
2,4,5-Trichlorophenoxyacetic acid	Not assessed		X

## 6 Toxicity assessment

### 6.1 Introduction

The toxicity assessment in a HHRA considers the following matters for exposure to each of the identified priority contaminants:

- The nature of adverse effects related to the exposure;
- The dose-response relationship for various effects; and
- The weight of evidence for effects such as carcinogenicity.

There is a range of different potential health effects that may be associated with the different compounds in landfill and leachate. At the highest level of consideration, the compounds can be classified as either non-threshold genotoxic carcinogens, or threshold compounds. There is a different approach to assessing the potential effects of these two types of compounds, as discussed in the following sub-sections.

Epidemiological studies have been undertaken of effects on populations living near landfills, which have shown an increased risk of lung cancer, congenital abnormalities (birth defects) and low birth weight (see Appendix A). However, most of these studies did not distinguish between landfills that accept hazardous waste and municipal solid waste landfills. In a key study that did differentiate between landfill types, an increased risk of health effects was found for landfills that accept hazardous waste. However, there was no apparent increased risk of adverse effects (compared to the general population) in communities living near municipal solid waste landfills (i.e landfills that do not accept hazardous waste). These findings are expected to be relevant to Auckland Regional Landfill, which will be a municipal solid waste landfill<sup>5</sup>.

It is also noted that a correlation was found between predicted hydrogen sulphide concentrations (from landfill gas) and increased incidence of health effects. This suggests the effects seen in epidemiological studies are related to airborne contaminants rather than exposure to leachate or some other pathway.

### 6.2 Dose response relationship for carcinogens

#### 6.2.1 Mechanisms of carcinogenicity

Carcinogens can be categorised as genotoxic or non-genotoxic based on available information about the way in which they promote cancer. Genotoxic carcinogens are chemicals that exert carcinogenicity through the induction of mutations in cells. DNA reactivity is usually inferred from positive results in short term tests for genotoxicity (O'Brien 2006). Because they interact directly with DNA, there is thought to be no safe exposure threshold or dose. Hence, these substances are referred to as 'non-threshold' carcinogens.

Non-genotoxic carcinogens induce cancer through mechanisms other than mutations, such as hormonal effects, being toxic to cells or promoting cell proliferation, are thought to have a safe exposure threshold or dose.

These differing dose response models are illustrated in Figure 6.1.

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<sup>5</sup> The Auckland Regional Landfill will accept "potentially hazardous waste", which is defined as waste that contains properties that could potentially be hazardous or toxic, but has been tested and subsequently confirmed as not posing a risk prior to being accepted through the landfill gate. Potentially hazardous waste will only be accepted at the landfill where pre-acceptance screening has determined that the waste meets the waste acceptance criteria.

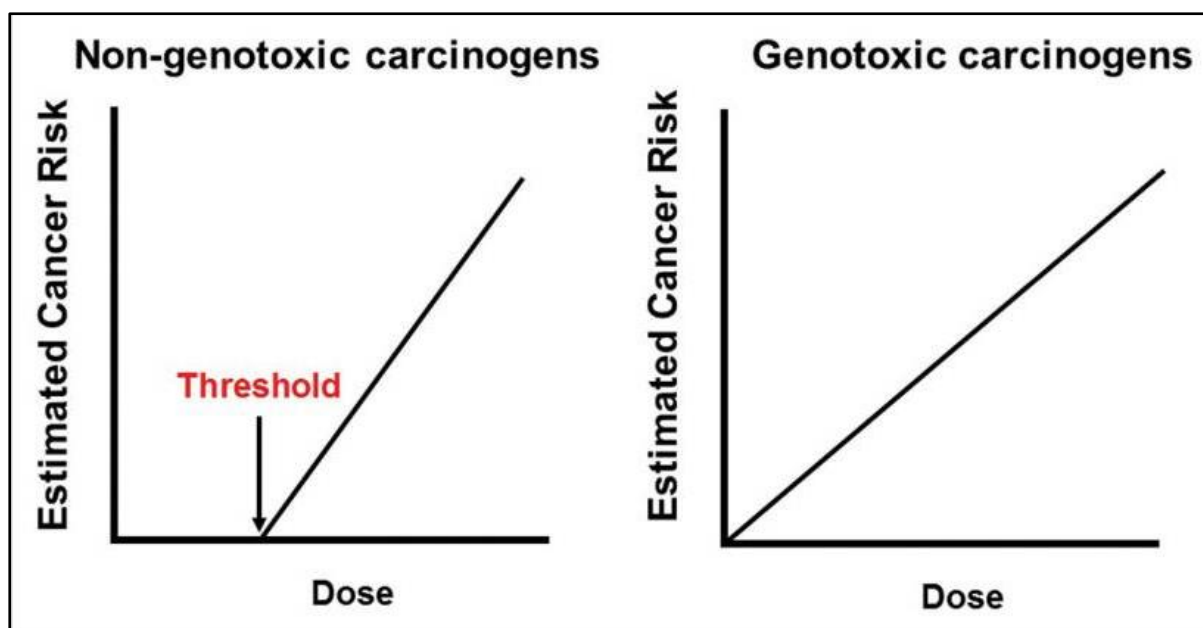


Figure 6.1: Models for dose-response curves of non-genotoxic and genotoxic carcinogens (Nohmi, T., 2018)

The WHO develops toxicity factors for carcinogens that are not genotoxic, or where there is insufficient evidence of a genotoxic mechanism, on the basis that there is a threshold of effects. These substances are assessed as threshold compounds using the approach discussed in the following sub-section. However, in contrast, the US EPA default approach for carcinogens where the mode of action is not established is to extrapolate using a low dose linear model (US EPA, 2005b. p3-23). This results in unit risk and slope factors being developed for some compounds that are evaluated by the WHO as threshold (non-genotoxic) carcinogens.

There is some discussion of this difference in approach and the blurring of the boundaries between genotoxicity and non-genotoxicity in the NES Soil (MfE, 2011. p2). Although the Toxicology Advisory Group did not state a specific preferred approach, DDT and its derivatives were treated as threshold contaminants in the NES Soil “given the equivocal data on their genotoxicity” (in line with their default approach, the US EPA has developed an oral slope factor and inhalation unit risk for DDT). This implies a preference for the WHO’s approach.

In this HHRA, the WHO’s evaluation of genotoxicity has been adopted and toxicity factors developed by the WHO for substances evaluated as threshold carcinogens have been used to estimate lifetime incremental cancer risk.

### 6.2.2 Unit risk and slope factors

The toxicity factor for inhalation exposure to a non-threshold carcinogens is expressed as the unit risk. The unit risk is defined as the upper-bound probability of an individual developing cancer as a result of a lifetime of exposure to a concentration of  $1 \mu\text{g}/\text{m}^3$  of the substance. The unit risk is expressed in units of  $(\mu\text{g}/\text{m}^3)^{-1}$ .

The toxicity factor for ingestion exposure to a non-threshold carcinogen is expressed as the slope factor. The slope factor is an upper bound estimate (generally 95% confidence limit) of the excess cancer risk from lifetime exposure to a body-weight adjusted daily dose of substance. The slope factor is usually expressed in units of  $(\text{mg}/\text{kg bw}\text{-day})^{-1}$ , although in this HHRA has been expressed in



units of  $(\mu\text{g}/\text{kg bw}\cdot\text{day})^{-1}$  for ease of calculation because of the very low concentrations of the compounds of concern<sup>6</sup>.

### 6.3 Dose response relationship for threshold compounds

Substances that do not fall into the category of ‘non-threshold carcinogens’ can have varying potential health effects, in terms of the particular organs or systems in the body that can be affected. For these substances, the dose response relationship is assumed to have a threshold concentration (i.e. a safe level) below which adverse health effects do not occur. This threshold concentration can differ depending on the particular health endpoint being considered. For this risk assessment, only the health effect that occurs at the lowest concentration has been considered (i.e. the most sensitive health endpoint).

The toxicity factors for threshold compounds are expressed as a Tolerable Concentration (TC, expressed in units of  $\mu\text{g}/\text{m}^3$ ) for inhalation exposure or a Tolerable Daily Intake (TDI, expressed in units of  $\mu\text{g}/\text{kg bw}\cdot\text{day}$ ) for ingestion exposure. Dermal exposure is not analysed in detail as explained in section 8.1. No adverse effects are expected from exposure to a substance at or below the TC or TDI.

### 6.4 Toxicity factors

#### 6.4.1 Hierarchy of sources

Toxicity factors for all of the COPC in this HHRA have been established, either specifically for New Zealand or by overseas organisations such as the World Health Organization (WHO). Toxicity factors have been selected based on the hierarchy set out in Table 6.1.

**Table 6.1: Source of toxicity factors**

Source of toxicity factors	Inhalation toxicity factors	Ingestion toxicity factors
Preferred source	<ul style="list-style-type: none"> <li>New Zealand ambient air quality guidelines (MfE, 2002)</li> </ul>	<ul style="list-style-type: none"> <li>NES Soil and supporting documents (MfE, 2011)</li> <li>Drinking-water Standards for New Zealand (Ministry of Health, 2017).</li> </ul>
Secondary source	<ul style="list-style-type: none"> <li>Air Quality Guidelines for Europe (WHO, 2000)</li> <li>Other WHO guidance, e.g. CICADs or Environmental Health Criteria documents</li> </ul>	<ul style="list-style-type: none"> <li>WHO Guidelines for drinking-water quality (WHO, 2017).</li> <li>Other WHO guidance, e.g. CICADs or Environmental Health Criteria documents</li> </ul>
Other	<ul style="list-style-type: none"> <li>California Office of Environmental Health Hazard Assessment (OEHAA)</li> </ul>	<ul style="list-style-type: none"> <li>California OEHAA</li> </ul>

The toxicity factors are tabulated in Appendix C. Given the large number of COPC and the ready availability of published information about their toxicity, a detailed description of the toxic effects of each of them has not been included in this HHRA.

#### 6.4.2 Approach to differing toxicity factors

There are two compounds where the selection of appropriate toxicity factors was unclear and so they have been assessed as both threshold compounds and genotoxic carcinogens. This approach

<sup>6</sup> 1 milligram (mg) = 0.001 gram, and 1 microgram ( $\mu\text{g}$ ) = 0.000001 gram

adds further conservatism as it potentially risks “double-counting”, however it has not affected the conclusions of the HRRRA.

### **Trichloroethylene**

Trichloroethylene is present in both landfill gas and leachate. The WHO considers that trichloroethylene is a genotoxic carcinogen via inhalation. However for ingestion exposure, the Ministry of Health has assessed it as a threshold compound based on developmental toxicity. For completeness, we have assessed trichloroethylene as follows:

- As a genotoxic carcinogen by both inhalation and ingestion using the unit risk (for inhalation) developed by the WHO and the slope factor (for ingestion) developed by the California OEHHA; and
- As a threshold compound by ingestion only, using the tolerable daily intake proposed by the New Zealand Ministry of Health (MoH, 2017)

### **Dichloromethane**

WHO (2002) considered that carcinogenicity was not the critical endpoint for risk assessment purposes and that formation of carboxyhaemoglobin was a more direct indication of toxic effects. However, a more recent evaluation by the IARC (2017) found that there is extensive evidence of genotoxicity and overall that the mechanistic evidence for dichloromethane carcinogenesis is strong. For completeness, we have assessed dichloromethane as follows:

- As a genotoxic carcinogen by both inhalation and ingestion using the unit risk (for inhalation) and the slope factor (for ingestion) developed by the California OEHHA; and
- As a threshold compound by both inhalation and ingestion, using the tolerable concentration set by the WHO in the ambient air quality guidelines for Europe and tolerable daily intake proposed by the New Zealand Ministry of Health (MoH, 2017).

## 7 Media concentrations

### 7.1 Introduction

Exposure to contaminants can occur through a variety of environmental media and foods. The concentrations of COPC in water (groundwater and surface water) and air at the various points-of-exposure have been estimated with modelling software using landfill source concentrations and fate and transport effects. . These model estimated water and air point-of-exposure values can then be used to estimate the concentrations of COPC in other media, such as soil. The following sub-sections set out the approach used to estimate media concentrations of the COPC.

Additional potential exposure pathways have been considered for PFAS given their mobility, persistence and toxicity. These pathways are uptake into domestic chickens and cows, and wild-collected eels. Eels have been selected as representative of fish species because they are at the top of the ecological food chain and there is an established bioaccumulation factor. The estimates of media concentrations of PFAS are set out in Appendix G.

### 7.2 Concentrations in groundwater and surface water

Leachate will reach peak generation when the landfill is at full development but prior to closure. While every attempt is made to avoid leakage of leachate from a landfill, some leakage may occur through defects that may be present in the lining system, from either the manufacturing process or installation<sup>7</sup>. Defects in the lining may cause some leachate to leak in to the underlying soil, and potentially the underlying groundwater system. The potential leachate leakage has been modelled over a 50 year period to represent full development of the site, indicating a potential theoretical leakage rate of up to approximately 3 m<sup>3</sup>/year, which is an average of 8.2 L/day<sup>8</sup>. The leachate is assumed to contain contaminants at a constant concentration throughout this period. In reality, these concentrations would decrease significantly during the aftercare period and beyond.

Concentrations of the priority contaminants have been determined in groundwater and surface water, as appropriate, at each of the relevant points of exposure identified in Table 4.2. The fate and transport modelling has been carried out using the Groundwater Services Inc. RBCA software package. The modelling methodology and results are detailed in the Hydrogeology Assessment.

### 7.3 Air concentrations

The Auckland Regional Landfill will use a combination of generators and flares to combust landfill gas and recover energy. The combustion process in the generators and flares will destroy most VOCs in the captured landfill gas. Residual emissions have been based on an assumed destruction efficiency of 97% in the generators and flare(s)<sup>9</sup> which will be constructed to minimum standards prescribed in the National Environmental Standards for Air Quality. Fugitive emissions of landfill gas through the landfill surface will have the same level of VOC emissions as uncombusted landfill gas. It has been assumed that this worst-case exposure scenario occurs for the entire 30 year exposure duration considered in the HHRA (see Table 8.1 – for rural residential scenarios a person is assumed to live at the same house for 30 years, 6 years as a child and 24 years as an adult).

Exposure to airborne contaminants can occur through direct inhalation, and from deposition of contaminants into soil (and subsequent uptake into produce or home-grown chicken eggs) or onto roofs used to collect drinking water. Ambient air concentrations and wet/dry deposition rates of the

<sup>7</sup> Quality assurance measures to minimise potential for defects at the Auckland Regional Landfill are described in the Engineering Report prepared for the consent application (Technical Report N).

<sup>8</sup> As described in the Engineering Report, the modelled leakage rate is likely over-estimated due to conservatism in the modelling.

<sup>9</sup> USEPA AP42 default destruction efficiency for non-methane organic compounds is 97% for both flares and generators

priority contaminants have been calculated using the CALMET/CALPUFF suite of modelling software. The dispersion modelling set up is described in the Air Quality Assessment (Technical Report D). Further information specific to modelling the deposition of contaminants is set out in Appendix E.

Two receptor sites have been used in the dispersion modelling to represent the closest residential neighbours to the landfill (both of which are more than a kilometre away). The highest concentration/deposition rate from either site has been chosen to represent the residential exposure scenario.

## **7.4 Concentrations in surface water**

To calculate the concentration of leachate in surface water, it was assumed that the wetland where surface water will discharge into the receiving environment is at the trigger level above which it would require discharges from the wetland to cease. Ammonia has been used as the key indicator of leachate in the pond as this is a good indicator of leachate contamination, and there are robust published guideline values that can be used in place of the trigger levels which have yet to be developed. The National Policy Statement for Freshwater Management (August 2017) (NPS-FM) includes numerical attribute state for ammonia in freshwater.

Based on the NPS-FM numerical attribute state for ammonia as the maximum discharge concentration, the permanent water volume of the wetland, and the typical concentration of ammonia in leachate, the volume of leachate required to be discharged into the wetland to maintain the concentration in the wetland at the trigger level was calculated.

Rainfall data from 2014 to 2018 has been used to calculate the discharge volume of surface water and the total mass discharge of leachate over the same period to calculate an average annual in-stream concentration of leachate as a worst case scenario for the purposes of this HHRA.

The mass of individual contaminants in the stream confluence from surface water can then be calculated based on the composition of the leachate.

## **7.5 Concentrations in soil**

### **7.5.1 Accumulation of contaminants in soil from aerial deposition**

Contaminants can be deposited onto soil through wet and dry deposition processes. At the same time, contaminants can be lost from the soil through a variety of mechanisms, including volatilisation and breakdown of the contaminants by abiotic (e.g. sunlight) and biotic processes (i.e. by bacteria).

The methodology used to calculate the accumulation of contaminants in soil is based on the methodology sets out in the HHRAP and the detailed calculations are set out in Appendix I.

The annual deposition rate of contaminants is based on the maximum rate of emissions of combusted landfill gas and fugitive landfill gas. This annual deposition rate is assumed to be constant over a 30 year period. For threshold compounds, the representative soil concentration is assumed to be the cumulative concentration at the end of the 30 year accumulation period (the exposure duration), taking into account losses. For non-threshold compounds, the average concentration in soil over the exposure period has been used, i.e. the soil concentration at Year 30 minus the soil concentration at Year 1, divided by 30 years).

In both cases, this will over-estimate actual exposures, as the emission rates will only occur at the peak rate for a period in the order of a year.

### 7.5.2 Accumulation of contaminants in soil from irrigation

Neither the NES Soil nor the HHRAP set out a recommended methodology for calculating the accumulation of contaminants in soil in a domestic vegetable garden from the use of water containing contaminants.

We have estimated the concentration of contaminants in soil that might arise from the use of water from the farm bore using the following assumptions:

In order to provide 25% home-grown produce, it is assumed that a household has a 45 m<sup>2</sup> vegetable garden. It is assumed that the garden is watered for 60 minutes a day, 100 days a year using a garden hose (or sprinkler) at a water application rate of 10 L/min. The contaminants are assumed to be uniformly mixed in the soil to a depth of 20 cm. As with aerial deposition, the contaminants are assumed to accumulate over a period of 30 years to give the representative soil concentration.

There is assumed to be some loss of contaminants from the soil through biotic and abiotic processes, but none through infiltration through to below the root zone. The soil loss constants are based on values provided in the HHRAP (the same values used to estimate the losses from accumulation through aerial deposition).

### 7.6 Concentrations in roof collected drinking water

Neither the NES Soil nor the HHRAP set out a recommended methodology for calculating the concentration of contaminants in roof-collected drinking water. The method used to estimate the entrainment of contaminants deposited onto a roof into drinking water are shown in Appendix I3.

Airborne contaminants can be deposited onto a roof in the same way as they can be deposited onto soils (see Section 7.5.1). It has been assumed that these contaminants accumulate on the roof during dry periods (i.e. there is no sample loss off the roof surface through re-volatilisation). This is a conservative assumption for contaminants deposited by dry deposition, as most of the airborne contaminants are volatile.

An annual rainfall of 1,500 mm/year has been selected<sup>10</sup> as being representative of the area, but at the lower end of the range of annual rainfall to conservatively estimate the dilution of contaminants. The calculation is not sensitive to the roof area of a house, as both the deposition area for contaminants and the volume of rainfall intercepted are dependent on the roof area.

A proportion of rainwater that falls on the roof will be lost due to overflows from rainwater tanks and evaporation. The deposition of contaminants is dominated by dry deposition processes. Therefore, it has been assumed that there may be “first flush” effects, which means that the contaminants that have accumulated through dry deposition processes are entrained by the early stages of a rainfall event, while the later rainwater volumes are lost as overflow. To account for this, and for losses by evaporation/overflow, it has been assumed that the total annual deposition of contaminants onto a 300 m<sup>2</sup> roof area is collected in 50% of the potential rainwater volume (i.e. 225 m<sup>3</sup>). This will be a highly conservative assumption, particularly for those contaminants deposited by wet deposition (i.e. they are only deposited when it is raining and do not accumulate in the roof surface).

As previously noted, most of the contaminants in the aerial emissions are relatively volatile. While re-volatilisation off the roof has not been accounted for, losses by evaporation from the water sitting in the tank have been accounted for. Estimated volatilisation half-life data in still water (lake model) for most of the COPCs are available in the background information to the New Zealand guidelines for drinking-water quality management (MoH, 2017). The half-life data has been used to estimate the

<sup>10</sup> based on annual rainfall data in the Stormwater and Industrial and Trade Activity Report, Technical Report P

losses from drinking water through volatilisation using a similar first order decay equation to that recommended in the HHRAP for volatilisation losses from soils.

## **7.7 Concentrations in home-grown produce**

There are two possible pathways for vapour phase airborne contaminants to be taken up into home-grown produce:

- 1 Direct transfer of gases into above-ground produce (air-to-plant transfer); and
- 2 Uptake into the roots from soil (soil-to-plant transfer).

The HHRAP sets out a detailed methodology for calculating uptake into plants through both these mechanisms. The soil-to-plant calculations use the soil concentrations calculated using the methods described in Section 7.5.

As discussed in Section 7.5, the concentrations of contaminants in soil has been assumed to occur from both aerial deposition and use of water from the farm bore. This is a very conservative assumption given that the maximum emissions of airborne contaminants will occur during the operational phase of the landfill, whereas potential leachate seepage through soil and transport in groundwater is a much slower mechanism (occurring over decades if not hundreds of years).

The detailed calculations of concentrations in home-grown produce are set out in Appendix I.

## **7.8 Concentrations of PFAS in foods**

PFAS have been given special consideration in the HHRA because of their mobility, environmental persistence and tendency to bioconcentrate in animals and be transferred into foods such as chicken eggs and milk. Appendix G sets out a detailed consideration of the toxicity of PFAS and the calculations of PFAS concentrations in wild caught eel exposed to PFAS in surface water and in chicken eggs, beef tissue and milk from animals that have been watered using groundwater from the farm bore.

The relationship between PFAS concentrations in surface water and eel flesh has been estimated from published bioconcentration factors (BCF). The methodology adopted to calculate PFAS concentrations in chicken eggs, beef meat and cow's milk is based on the methodology and PFAS-specific factors used in the human health risk assessment prepared by Aecom for the RAAF Williamtown Airforce Base in New South Wales, Australia (Aecom, 2017).

## **7.9 Concentrations of arsenic in watercress**

Arsenic has been considered as an indicator compound for the possible bioconcentration of metals in aquatic plants, particularly water cress that can be harvested and eaten by people.

The calculations of arsenic concentration in water cress are set out in Appendix I, based on BCF factors for arsenic uptake into watercress developed in New Zealand.

## 8 Exposure assessment

### 8.1 Approach to estimating intake

Exposures to each of the COPCs via the different exposure pathways can be calculated using a form of the following generic intake equation (HHRAP Equation 6-1):

$$\frac{C_{gen} \times CR \times EF \times ED}{BW \times AT}$$

Where:

$C_{gen}$  is the COPC concentration in the medium of concern (e.g., mg/kg for soil)

CR is the consumption rate or the amount of contaminated medium consumed per unit of time (e.g. mg/day for soil ingestion)

EF is the exposure frequency (days/year)

ED is the exposure duration (years)

BW is body weight (kg)

AT is the period over which exposure is averaged (days). AT is calculated differently for threshold compounds and non-threshold compounds

For all of the COPCs, it has been assumed that there is 100% bioavailability, i.e. all of the substance consumed (inhaled or ingested) is available to the body (and not excreted or exhaled).

Dermal exposure to contaminants in soil or water is not typically recommended in the HHRAP as it is rarely a significant exposure pathway for aerial emissions. Given the very low concentrations of contaminants predicted in groundwater or soil at the residential receptor (Appendix I), or in surface water that could be used for contact recreation (Appendix D), dermal exposure is unlikely to be a significant pathway for this assessment and has not been considered further.

### 8.2 Averaging time and critical receptors

For threshold compounds, exposure is averaged only over the exposure duration. The critical receptor for exposure to threshold contaminants is generally a child because of their relatively low body weight compared to intake. Therefore the exposure duration is assumed to be 6 years and child values for consumption rates and body weight are used in the intake calculations.

For non-threshold substances, exposure is a pro-rated cumulative dose over a lifetime (75 years). The period of exposure is assumed to be 30 years for the rural residential/lifestyle block scenario, so the critical receptor becomes a combination of childhood and adult exposure (i.e. age-weighted consumption rates and body weight assuming 6 years as a child and 24 years as an adult).

### 8.3 General intake factors

The intake factors used in this HHRA are summarised in Table 8.1. These factors have been taken from the recommendations for the rural residential/lifestyle block scenario in the NES Soil, which represents the most recent guidance on health risk assessment in the New Zealand context.

The key differences between the intake factors for the rural residential / lifestyle block scenario and the standard urban residential scenario are:

- A longer period of residence (30 years compared 20 years);
- Higher percentage of home grown produce (25% compared to 10%); and
- Consumption of twice the average dietary intake of eggs.

**Table 8.1: General intake factors (from NES Soil for rural residential)**

Exposure factor	Units	Recommended values for rural residential exposure scenario
Exposure Frequency (EF)	days/year	350
Exposure Duration (ED)	Years (6 years as child, 24 years as adult)	30
Body Weight (BW)	Child (kg)	13
	Adult (kg)	70
Averaging Time (AT)	Non-threshold compounds (days) 365 days/year x 75 years	27375
	Threshold compounds (days) 365 days/year x 6 years	2190
Inhalation rate	Child (m <sup>3</sup> /day)	6.8
	Adult (m <sup>3</sup> /day)	13.3
Drinking water ingestion rate	Child (L/day)	1
	Adult (L/day)	2
Soil ingestion rate	Child (mg/day)	50
	Adult (mg/day)	25

#### 8.4 Intake factors for home-grown produce

The approach used to quantify ingestion of home-grown produce from the derivation of the NES Soil (MfE, 2011) has been adopted (see summary of intake factors from the NES Soil in Appendix I Table 4). The percentage of produce that is home-grown is assumed to be 25 %, based on the recommendation in MfE, 2011.

**Table 8.2: Produce consumption rates (from NES Soil for rural residential)**

Vegetable type	Produce consumption (vegetable types likely to be home-grown)		Home grown produce consumption		
	Adult	Child	Adult	Child	Weighted average
	g DW /day	g DW /day	g DW /day	g DW /day	g DW /day
Tuber vegetable	18.9	6.6	4.73	1.65	4.11
Root vegetable	1.9	1.0	0.48	0.25	0.43
Above ground vegetables (including cucurbits)	11.4	2.86	2.85	0.72	2.42
Total	32.2	10.46	8.05	2.62	6.96

The amount of soil attached to home-grown produce is based on 38 mg/day for an adult and 8 mg/day for a child for 100 % of produce grown at home, pro-rated to 25 % of produce being grown at home (MfE, 2011).



**Table 8.3: Ingestion of soil with home-grown produce**

Exposure factor	Units	Calculated values for rural residential exposure scenario
Soil ingested with homegrown produce	Child (mg/day)	2
	Adult (mg/day)	9.5

### 8.5 Intake factors for wild harvested eels and watercress

Dietary surveys undertaken for Te Arawa iwi found a maximum eel consumption rate of 93.3 g/day and a mean of 9.6 g/day (NIWA, 2011). A similar survey for Arowhenua iwi found a maximum eel consumption rate of 20 g/day. These data suggest that eel consumption rates are highly variable. The daily intake value adopted for this assessment is 70 g/day based on data collected for an assessment of exposure to dioxins in the Kopeopeo Canal (ToxConsult, 2013). This value was chosen as it was at the higher end of the reported range.

No specific data on eel ingestion by children could be found in the literature. MPI (2018) estimated the daily intake of fresh fish by New Zealand children to be 3 g/day. Assuming this fresh fish intake comprised entirely eel, adults would have a higher body-weight adjusted eel intake compared to a child.

NIWA (2011) found a maximum watercress consumption in a survey of Te Arawa iwi of 90 g/day with a mean of 15.8 g/day. An average consumption rate of 33 g/day was proposed by Golder Associates and NIWA (2009), cited in NIWA (2011). This average value was adopted for this HHRA. No consumption data for watercress by children was found in the literature.

Given the absence of reliable consumption data for children, the wild food collector receptor has been based on adult intake and exposure parameters.

### 8.6 Conservatism in assessment of cumulative exposure to contaminants in leachate and landfill gas

The assessment has adopted conservatively high concentrations of contaminants based on measured concentrations in leachate at Redvale Landfill. Leachate strength will reduce progressively after the landfill is closed. It is estimated that any leachate that seeps through the high density polyethylene geomembrane component of the lining system would take between approximately 6 and 20 years to enter groundwater under the landfill. Many of the contaminants in leachate tend to bind strongly to soil and this would mitigate the potential for contaminants to reach groundwater. There would be a significant further time lag for contaminants to be transported by groundwater to the farm bore (decades if not centuries).

In comparison, the highest landfill gas generation rates will occur around the time the landfill is close to being full and landfill gas generation will gradually decrease from this point.

Therefore, in reality, the worst case scenario for exposure to contaminants in landfill gas will not occur at the same time as the worst case scenario for exposure to contaminants in leachate (which will occur many years later due to the length of time taken to reach the farm bore as described above). The HHRA calculations conservatively assume that these exposures occur simultaneously.

## 9 Risk characterisation

### 9.1 Estimating risk from exposure to genotoxic carcinogens

#### 9.1.1 Calculating excess lifetime cancer risk

As outlined in Section 6, inhalation exposure to non-threshold carcinogens is assessed using the unit risk and ingestion exposure is assessed using the slope factor.

The excess lifetime cancer risk for exposure to a substance in air (via inhalation) can be calculated as follows:

$$\begin{aligned} \text{Excess lifetime cancer risk (via inhalation)} \\ = \text{unit risk } (\mu\text{g}/\text{m}^3)^{-1} \times \text{concentration in air } (\mu\text{g}/\text{m}^3) \end{aligned}$$

The excess lifetime cancer risk for exposure to a substance via ingestion (oral dose) can be calculated as follows:

$$\begin{aligned} \text{Excess lifetime cancer risk (via ingestion)} \\ = \text{slope factor } (\mu\text{g}/\text{kg bw-day})^{-1} \times \text{oral dose } (\mu\text{g}/\text{kg bw-day}) \end{aligned}$$

#### 9.1.2 Acceptable risk level

Because there is no “safe” dose for non-threshold carcinogens, the risk associated with exposure to these substances is based on the incremental lifetime cancer risk. The exposure evaluation to these non-threshold contaminants is based on an agreed acceptable increase in risk.

The Toxicology Advisory Group on the NES Soil recommended that the acceptable increased risk level for use in health risk assessments is 1 in 100,000 ( $10^{-5}$ ) (MfE, 2011. p4). This falls in the “mid-range” of acceptable risk levels used by international agencies, which range from one in a million (e.g. United States, Canada) to 1 in 10,000 (The Netherlands).

#### 9.1.3 Evaluating cumulative risk

It is common practice to assume cancer risks due to different genotoxic carcinogenic compounds are additive. The individual cancer risks are summed to estimate the total lifetime risk of developing cancer. However, it should be noted that this approach is particularly conservative as each of the unit risk estimates is based on the upper bound 95% confidence estimate. The greater the number of carcinogens considered, the more unlikely the true risk for each carcinogen lies near the upper bound estimate. Therefore the cumulative cancer risk derived by adding all of these upper bound estimates together may not actually be plausible. As such, this approach increases the conservatism of the conclusions drawn by this HHRA.

## 9.2 Estimating hazard from exposure to threshold compounds

### 9.2.1 Calculating the hazard quotient

The hazard quotient is the ratio of the potential exposure to a substance and the TDI (see Section 6), as follows:

$$\text{Hazard quotient (via ingestion)} = \frac{\text{Daily intake } (\mu\text{g}/\text{kg bw-day})}{\text{Tolerable daily intake } (\mu\text{g}/\text{kg bw-day})}$$

For exposure via inhalation, the same form of equation can be used. However, the time and body-weight factors in the daily intake and tolerable daily intake calculations are virtually identical and effectively cancel each other out. Therefore, a simplified form of the equation can be used as follows:

$$\text{Hazard quotient (via inhalation)} = \frac{\text{Concentration in air } (\mu\text{g}/\text{m}^3)}{\text{Tolerable concentration } (\mu\text{g}/\text{m}^3)}$$

If the Hazard Quotient is calculated to be less than unity (1.0), then no adverse health effects are expected as a result of exposure to the substance via the pathway under consideration.

### 9.2.2 Evaluating cumulative hazards

The HHRAP recommends a tiered approach to evaluating hazards from cumulative exposure to threshold compounds. In the first instance, a highly simplified approach of summing all Hazard Indices (HI) across all exposure pathways can be used. While this is not toxicologically appropriate it can provide an initial useful screen as if the single HI is less than 1.0, then no further evaluation is required.

Where the single HI exceeds a value of 1.0, it is recommended that the COPCs are segregated based on their toxicological similarity (e.g. the same target organ or systems). If the segregated HIs are all less than the 1.0, then health effects are not likely to result from combined exposure to the COPCs included in the HI.

The HHRAP notes that the segregated HI approach is still an over-simplification as ideally the HI would also be segregated according to the mechanisms of toxicity of the COPCs. However, this approach is beyond the scope of an initial risk evaluation.

## 9.3 Risk and hazard findings

### 9.3.1 Residential receptor

The COPC-specific hazard and risk estimates for the residential exposure scenario are set out in Table 9.2 (for genotoxic carcinogens) and Table 9.3 (for threshold compounds). An overall summary of the cumulative hazard and risk estimates via each pathway is shown in Table 9.1.

Using roof-collected drinking water for potable supply gave higher cumulative hazard index/incremental lifetime cancer risk values compared to the alternative of using groundwater from the farm bore for potable supply. These are the values presented in Tables 9.1 to 9.3.

The 'single HI' value calculated by summing all the individual COPC- and pathway- HQ values for the threshold compounds is 0.014. As this highly conservative screening value is less than 1.0, this indicates that health effects are not likely to result from combined exposure to the threshold COPCs by cumulative exposure over all pathways.

The cumulative lifetime incremental cancer risk value for the genotoxic carcinogens is  $2.34 \times 10^{-7}$ . This is well below the acceptable risk level of  $1 \times 10^{-5}$  (i.e. a lifetime incremental cancer risk of 0.23 in a million compared to the 10 in a million acceptable risk value).

These estimates do not include the contribution from the substances that have been evaluated at the Waste Acceptance Criteria limits, as these substances have been evaluated separately in Section 9.4. However, it is noted that inclusion of these substances would not alter the conclusions of the HHRA.

**Table 9.1: Summary of risk assessment findings for residential receptor**

<b>Receptor-pathway</b>	<b>Lifetime incremental cancer risk (genotoxic carcinogens)</b>	<b>Hazard Index (threshold compounds)</b>
Inhalation of airborne contaminants at residential receptor	6.17E-08 (0.0617 per million)	0.0101
Residential receptor with potable supply from roof collected water	7.61E-08 (0.0761 per million)	0.00259
Direct ingestion of soil	1.22E-10 (0.000122 per million)	0.00000145
Ingestion of home-grown produce from vegetable garden and eggs from chickens at residential receptor subject to aerial deposition of contaminants and using farm bore for irrigation	9.59E-08 (0.0959 per million)	0.00163
Cumulative risk/hazard	2.34E-07 (0.234 per million)	0.0143
Acceptable risk/hazard level	1.00E-05 (10 per million)	1.0

**Table 9.2: Genotoxic carcinogens - Residential receptor drinking roof collected water and using bore water to irrigate the vegetable garden**

Contaminant	Incremental lifetime cancer risk				COPC cumulative risk
	Inhalation	Produce ingestion	Drinking water	Soil ingestion	
1,2-dibromoethane	3.42E-08	5.16E-08	2.77E-08	1.14E-10	1.13E-07
1,2-dichloroethane	1.32E-11	4.24E-14	1.74E-13	1.12E-17	1.34E-11
1,2-dichloropropane	2.07E-10	1.38E-12	1.31E-09	1.59E-15	1.52E-09
Acetaldehyde	4.21E-09	*	*	*	4.21E-09
Acrylonitrile	7.12E-09	2.44E-08	1.04E-08	3.08E-13	4.19E-08
Arsenic	-	2.75E-11	-	7.80E-12	3.53E-11
Benzene	6.03E-09	3.02E-11	2.82E-09	4.42E-14	8.88E-09
Dichloromethane	7.86E-09	1.91E-08	3.07E-09	6.07E-13	3.01E-08
Trichloroethylene	4.42E-10	6.22E-12	1.94E-10	1.58E-14	6.43E-10
Vinyl chloride	1.61E-09	7.86E-10	3.08E-08	9.92E-14	3.32E-08
Total cumulative incremental lifetime cancer risk	6.17E-08 0.0617 per million	9.59E-08 0.0959 per million	7.61E-08 0.0761 per million	1.23E-10 0.0001 per million	2.34E-07 0.234 per million
Acceptable risk level	1.00E-05 (10 per million)				

**Table note:**

Contaminants that have concentrations so low they are “indistinguishable from zero” based on RBCA modelling are not presented in this table.

As the values in this table are very small, they are expressed as exponentials for ease of reading. For example 3.24E-08 is the same as  $3.24 \times 10^{-8}$  or 0.0000000324

**Table key:**

- Indicates no exposure via this media (e.g. arsenic is present in leachate but not landfill gas and therefore there is no exposure via inhalation or roof collected drinking water)

\* Acetaldehyde is carcinogenic via inhalation, however no slope factors have been published to allow an evaluation of exposure to acetaldehyde via ingestion. Acetaldehyde has a high volatility and the main exposure pathway will be via inhalation

**Table 9.3: Threshold compounds - Residential receptor drinking roof collected water and using bore water to irrigate the vegetable garden)**

Contaminant	Hazard Quotient				COPC Hazard Index
	Inhalation	Produce ingestion	Drinking water	Soil ingestion	
Carbon tetrachloride	4.94E-04	2.22E-05	4.69E-04	8.80E-09	9.85E-04
Chlordane, total	-	1.95E-08	-	1.19E-09	2.07E-08
Chlorobenzene	7.55E-07	5.33E-07	9.71E-05	3.91E-10	9.84E-05
Chromium	-	1.17E-07	-	2.56E-08	1.42E-07
Dichloromethane	1.75E-05	1.43E-03	2.28E-04	3.80E-08	1.67E-03
Endrin	-	4.48E-07	-	1.33E-08	4.61E-07
Formaldehyde	3.17E-05	8.66E-05	2.28E-05	7.89E-10	1.41E-04
Hexachlorobutadiene	*	3.72E-07	1.14E-03	1.03E-06	1.14E-03
Lead	-	7.05E-10	-	3.03E-10	1.01E-09
Methyl ethyl ketone	1.17E-06	3.35E-05	1.13E-04	3.32E-09	1.48E-04
Methyl isobutyl ketone	9.06E-07	*	*	*	9.06E-07
PFOA	-	4.63E-08	-	6.92E-10	4.70E-08
PFOS/PFHxS	-	7.28E-08	-	9.13E-09	8.20E-08
Styrene	6.04E-03	1.77E-06	1.16E-04	2.97E-07	6.16E-03
1,1,2,2-tetrachloroethane	6.08E-06	5.96E-06	2.63E-06	3.41E-09	1.47E-05
Tetrachloroethylene	1.47E-05	2.34E-05	5.70E-05	1.71E-09	9.51E-05
Toluene	1.09E-04	4.03E-06	2.22E-05	2.15E-09	1.35E-04
Total reduced sulphur	3.26E-03	*	*	*	3.26E-03
Trichloroethylene	X	8.47E-06	1.41E-04	8.05E-09	1.50E-04
Trichloromethane (chloroform)	9.30E-05	9.31E-06	1.66E-04	4.93E-09	2.69E-04
Xylene	1.73E-05	6.02E-06	1.07E-05	2.53E-09	3.40E-05
Hazard Index	0.0101	0.0016	0.0026	0.0000015	0.0143
Hazard threshold	1.0				

**Table note:** Contaminants that are not present in this media, or have concentrations indistinguishable from zero, are not presented in this table.

As the values in this table are very small, they are expressed as exponentials for ease of reading. For example 4.94E-04 is the same as  $4.94 \times 10^{-4}$  or 0.000494.

**Table key:**

- Indicates no exposure via this media (e.g. lead is present in leachate but not landfill gas and therefore there is no exposure via inhalation or roof collected drinking water)

\* No toxicity factor available:

There are no established toxicity values for exposure to total reduced sulphur compounds via ingestion, and they are expected to have a low oral toxicity.

The WHO has established an oral toxicity factors for hexachlorobutadiene, however no relevant published inhalation toxicity factors could be identified

The WHO has established inhalation toxicity factor for methy isobutyl ketone, however no relevant published oral toxicity factors could be identified

X Trichloroethylene is assessed as a genotoxic carcinogen via inhalation

PFAS exposure for the representative residential receptor has been shown separately in Table 9.4. This exposure scenario differs from the standard residential receptor as it includes ingestion of home-grown eggs from chickens that have been watered using water from the farm bore and it also assumes that potable water supply is from the farm bore (rather than roof supply). The cumulative hazard index associated with PFAS exposure is  $1.07 \times 10^{-5}$  (0.0000107) compared to the hazard threshold value of 1.0. From this, it can be concluded there is no appreciable health risk posed by exposure to PFAS via the pathways considered.

**Table 9.4: PFAS - Residential receptor using bore water for potable supply and to irrigate the vegetable garden)**

Contaminant	Hazard Quotient				COPC Hazard Index
	Chicken egg ingestion	Produce ingestion	Drinking water	Soil ingestion	
PFOA	3.55E-07	4.63E-08	9.96E-08	6.92E-10	5.02E-07
PFOS/PFHxS	8.83E-06	7.28E-08	1.31E-06	9.13E-09	1.02E-05
Hazard Index	9.19E-06	1.19E-07	1.41E-06	9.82E-09	1.07E-05
Hazard threshold	1.0				

### 9.3.2 Wild food collector

The indicator compounds selected for exposure by a person collecting foods from surface water are PFAS (for ingestion of eels and incidental ingestion of water) and arsenic (for ingestion of water cress). The HI and lifetime incremental cancer risk calculations are shown in Table 9.5 and Table 9.6.

The HI for exposure to PFAS via ingestion of eels and incidental ingestion of surface water is  $2.19 \times 10^{-3}$  (0.00219). This value is well below 1.0 and therefore it can be concluded that health effects are not likely to result from exposure by a person collecting and eating eels.

The lifetime incremental cancer risk value for exposure to arsenic via ingestion of watercress and incidental ingestion of surface water is  $2.27 \times 10^{-9}$ . This is well below the acceptable risk level of  $1 \times 10^{-5}$  (i.e. a lifetime incremental cancer risk of 0.00227 in a million compared to the 10 in a million acceptable risk value).

**Table 9.5: PFAS substances – Wild food collector eating eels and incidental ingestion of surface water**

Contaminant	Hazard quotient		Compound-specific Hazard Index
	Ingestion eels	Ingestion water	
PFOA	2.67E-06	5.53E-08	2.73E-06
PFOS/PFHxS	2.19E-03	9.86E-07	2.19E-03
Hazard Index	2.19E-03	1.04E-06	2.19E-03
Hazard threshold	1.0		

**Table 9.6: Arsenic - Wild food collector eating water cress and indirect ingestion of surface water**

Contaminant	Incremental lifetime cancer risk		Cumulative risk	Acceptable risk level
	Ingestion water cress	Ingestion water		
Arsenic	8.95E-13	2.27E-09	2.27E-09 (0.00227 per million)	1.00E-05 (10 per million)

#### Fish in the Hōteō River and Kaipara Harbour

The potential for PFAS to impact on contaminant levels in fish in the Hōteō River and further downstream in the Kaipara Harbour has also been considered. Concentrations of PFAS will be diluted by the large volumes of water in the River and will be even further diluted in the Harbour. Combined with this, the bio-accumulation values for PFAS in eel are higher than for other fish. For these reasons, it is considered that uptake of PFAS by eels at the exposure concentrations calculated for the small stream close to the proposed Landfill are the worst case. As the health hazard associated with harvesting and eating eels at this location are calculated to be acceptable, then it can be concluded that exposure for other fish species in the Hōteō River and Kaipara Harbour will also be acceptable (by a significantly greater margin).

### 9.3.3 Public consumer

Some of the substances present in leachate can bioconcentrate in meat or milk from farmed stock animals. As outlined in Section 5.4.2, PFAS have been considered as a special category of substances because they are emerging contaminants of concern due to their mobility and environmental persistence. These PFAS compounds are considered to be the most sensitive substances of any of the contaminants in leachate for bioconcentration into terrestrial animals.

There are other substances considered in this HHRA that also have some of these characteristics, although generally to a lesser degree. If the assessment of PFAS suggests human health effects are unlikely then this conclusion will also apply to the other priority compounds with persistent and bioaccumulative properties.

The calculations of PFAS uptake into animals and subsequent concentrations in food (meat, milk and eggs) are set out in Appendix G. The calculated concentrations have been compared to the proposed trigger points for investigation set for PFAS in various foods by Food Standards Australia New Zealand (FSANZ, undated b) in Table 9.7. When comparing sources for stock watering, higher concentrations of PFAS are calculated in beef and dairy cattle being watered from the stream compared to the farm bore. These higher values are presented in the table. All of the calculated values for PFAS in food are well below the recommended trigger levels for further investigation. Consequently there is not expected to be any health effects associated with eating these foods.



**Table 9.7: Comparison of estimated concentrations in food with PFAS trigger points**

Contaminant	PFOS/PFHxS		PFOA	
	Estimated concentration (µg/kg)	Trigger point <sup>1</sup> (µg/kg)	Estimated concentration (µg/kg)	Trigger point (µg/kg)
Finfish	0.0536	5.2	0.00043	41
Mammalian animal meat (e.g. beef)	1.24E-04	3.5	6.12E-07	28
Milk	6.43E-08	0.4 <sup>2</sup>	8.67E-06	2.8
Poultry eggs	1.42E-04	11	4.59E-05	85

<sup>1</sup> Source: FSANZ, undated b

<sup>2</sup> or level of detection if higher

#### 9.4 Evaluation of waste acceptance criteria

As discussed in Section 5.4.7, there is a group of substances that have been included in the HHRA calculations for the purpose of evaluating the appropriateness of the Waste Acceptance Criteria. Because of their low volatility, these compounds are only present in leachate (they are not present in landfill gas), so inhalation is not a complete exposure pathway and there is no potential for aerial deposition and presence in roof-collected drinking water. Therefore, the reasonable maximum exposure scenario for these compounds is a residential receptor with potable water supply from groundwater and use of groundwater for irrigation of a vegetable garden.

The only substance where modelling predicts a quantifiable concentration at the farm bore is chlordane. The predicted concentrations of all other substances is so low it is indistinguishable from zero. Chlordane is assessed as a threshold compound, as shown in Table 9.8. The hazard index for exposure to chlordane is well below levels that would be of concern with respect to health effects.

Toxaphene is the only compound included in the Waste Acceptance Criteria that has been assessed as a genotoxic carcinogen. The predicted concentration of toxaphene in the farm bore is indistinguishable from zero and therefore the incremental lifetime cancer risk is too small to be calculated.

**Table 9.8: Threshold compounds in the Waste Acceptance Criteria for residential receptor drinking farm bore water**

Contaminant	Hazard quotient			COPC Hazard Index
	Produce ingestion	Child soil ingestion	Drinking water	
Chlordane, total	1.95E-08	9.29E-07	1.19E-09	9.50E-07
Hazard threshold	1.0			

## 10 Conclusions

The findings of the risk assessment are expressed in terms of the hazards associated with exposure to 'threshold compounds', which are substances that have a threshold of exposure (i.e. a safe level below which adverse effects are not expected to occur) and the incremental lifetime cancer risk associated with exposure to carcinogenic substances i.e. 'non-threshold compounds'.

Because there is no "safe" dose for non-threshold carcinogens, the risk associated with exposure to these substances is based on the incremental lifetime cancer risk. The generally agreed acceptable increase in risk in New Zealand is 1 in 100,000 (or 10 in a million).

The hazard associated with threshold compounds is based on the Hazard Quotient, which is the ratio between the daily intake and the Tolerable Daily Intake. If the Hazard Quotient is calculated to be less than unity (1.0), then no adverse health effects are expected as a result of exposure to the substance via the pathway under consideration. To consider the cumulative hazard from exposure to multiple contaminants, a highly simplified approach of summing all Hazard Indices (HI) across all exposure pathways can be used as an initial screening assessment. While this is not toxicologically appropriate it can provide an initial useful screen as if the single HI is less than 1.0, then no further evaluation is required.

The most significant exposure pathway identified in the risk assessment is the inhalation of airborne contaminants from the residual emissions from the flares and generators and fugitive escape of unburnt landfill gas.

For non-threshold compounds the cumulative incremental lifetime excess cancer risk for inhalation exposure by a residential receptor is calculated to be  $6.17 \times 10^{-8}$ , or 0.0617 per million, compared to the acceptable risk of 10 per million. For threshold compounds, the cumulative Hazard Index for inhalation exposure by a residential receptor is calculated to be 0.0101 compared to the hazard threshold of 1.0. The cumulative hazard and risk estimates are three orders of magnitude below the level where health effects would need to be investigated in more detail.

The cumulative risk and hazard values for all of the compounds and pathways considered for a representative residential receptor (using the conservative screening method outlined above) are 0.234 per million and 0.0143, respectively (which can be compared to assessment values of 10 per million and 1.0, respectively). This shows that there are no unacceptable hazards or risks for any of the contaminants or pathways considered for a representative residential receptor, both individually and cumulatively using a conservative screening approach.

Conservative estimates of PFAS concentrations in a range of food products, either home grown (e.g. chicken eggs), farmed (e.g. beef or cow's milk) or wild caught (e.g. eels), were well below the recommended trigger levels for further investigation. Consequently there is not expected to be any health effects associated with eating these foods.

The investigation of PFAS concentrations in eels also provides a conservative assessment of possible contaminants in other freshwater fish species, for example in the Hōteu River, or saltwater fish in the Kaipara Harbour. PFAS has been selected as the indicator contaminant because of its mobility and persistence in the environment and its known tendency to bioaccumulate in fin fish such as eels. The assessment used contaminant levels representative of the worst case concentrations in the stream close to the landfill footprint. Given the findings of the assessment that concentrations of PFAS in eels would be two orders of magnitude lower than the trigger levels, then it can be concluded that exposure for other fish species further downstream or in the Kaipara Harbour will also be acceptable (by a significantly greater margin).

## 11 References

- Ahrens L, Shoeib M, Harner T, Lee SC, Guo R, Reiner EJ. (2011). Wastewater treatment plant and landfills as sources of polyfluoroalkyl compounds to the atmosphere. *Environmental Science & Technology* 2011, 45, 8098-8105 (cited in Tian et al, 2018)
- Auckland Council. (2018). Draft Auckland Waste Management and Minimisation Plan 2018
- Australian Government Department of Health. (2017). Health based guidance values for PFAS for use in site investigations in Australia. Retrieved from: <https://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-pfas-hbgv.htm>
- Environmental Health Australia. (2012). Environmental health risk assessment: Guidelines for assessing human health risks from environmental hazards
- Food Standards Australia and New Zealand. (undated). Hazard assessment report – Perfluorooctane Sulfonate (PFOS), Perfluorooctanoic Acid (PFOA), Perfluorohexane Sulfonate (PFHxS)
- Food Standards Australia and New Zealand. (undated b). Perfluorinated chemicals in food
- Gallen C, Drage D, Eaglesham G, Grant S, Bowman M, Mueller J.F. (2017). Australia-wide assessment of perfluoroalkyl substances (PFASs) in landfill leachates. *Journal of Hazardous Materials* 331 (2017) 132–141
- Huber M, Laesecke A, and Friend D (2006). The Vapor Pressure of Mercury. National Institute of Standards and Technology. Boulder, Colorado
- Ministry for Primary Industries (2018). New Zealand Food Safety. 2016 New Zealand Total Diet Study
- Ministry for the Environment. (2011). Methodology for deriving standards for contaminants in soil to protect human health. Wellington: Ministry for the Environment
- Ministry for the Environment. (1999). Guidelines for Assessing and Managing Petroleum Hydrocarbon Contaminated Sites in New Zealand (Revised 2011). Appendix 4L - Toxicity Assessment. Wellington: Ministry for the Environment
- Ministry of Health. (1995). A Guide to Health Impact Assessment. Guidelines for public health services and resource management agencies and consent applications. Reprinted 1998
- Ministry of Health. (2017). Guidelines for Drinking-water Quality Management for New Zealand (3rd edn) Volume 3 Datasheets - Chemical and physical determinands. Updated June 2019 Wellington.
- Ministry of Health. (2018). Drinking-water Standards for New Zealand 2005 (revised 2018). Wellington: Ministry of Health
- Nohmi, T. (2018). Thresholds of Genotoxic and Non-Genotoxic Carcinogens. *Toxicol. Res.* 2018. 34(4): 281–290. PMID: 30370002
- Tian Y, Yao Y, Chang S, Zhao Z, Zhao Y, Yuan X, Wu F, Sun H. (2018) Occurrence and Phase Distribution of Neutral and Ionizable Per- and Polyfluoroalkyl Substances (PFASs) in the Atmosphere and Plant Leaves around Landfills: A Case Study in Tianjin, China. *Environ Sci Technol.* 2018 Feb 6; 52(3):1301-1310
- Tonkin + Taylor Ltd. (2003). Health risk assessment for discharges to air of volatile organic compounds at Redvale Landfill – Final Report
- ToxConsult. (2013). Kopeopeo Canal dioxin survey

- US EPA. (2005). Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities. Office of Solid Waste and Emergency Response. EPA530-R-05-006
- US EPA. (2005b). Guidelines for Carcinogen Risk Assessment. Risk Assessment Forum. EPA/630/P-03/001F
- USEPA. (2008). AP-42: Compilation of Air Emissions Factors Fifth Edition, Volume I Section 2.4 Municipal Solid Waste Landfills (Draft)
- Weinberg I, Dreyer A, Ebinghaus R. (2011). Landfills as sources of polyfluorinated compounds, polybrominated diphenyl ethers and musk fragrances to ambient air. Atmospheric Environment. 513 2011, 45, 935-941
- World Health Organisation (2003) Guidelines for safe recreational water environments. Volume 1, Coastal and fresh waters. Geneva
- Woodward Clyde NZ Ltd. (1999). Health risk assessment for proposed Hampton Downs Landfill

## 12      **Applicability**

This report has been prepared for the exclusive use of our client Waste Management NZ Ltd, with respect to the particular brief given to us and it may not be relied upon in other contexts or for any other purpose, or by any person other than our client, without our prior written agreement.

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**Appendix A: Literature review of health impacts in communities around landfills**

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There have been a number of systematic reviews of epidemiological studies of health impacts in communities living around landfills and other waste management facilities (incinerators, composting, etc). Key studies include:

- 1 Porta et al. (2009). Systematic review of epidemiological studies on health effects associated with management of solid waste.
- 2 Giusti L (2009) A review of waste management practices and their impact on human health Waste Management.
- 3 Matiello et al (2013). Health effects associated with the disposal of solid waste in landfills and incinerators in populations living in surrounding areas: a systematic review.

These reviews have all found that a limitation of many of the epidemiological studies of communities near landfills is that they do not make any distinction between the types of waste accepted at the landfill or landfilling practice, such as landfill cover/capping and landfill gas management. In particular, most studies do not differentiate between landfills accepting hazardous (special) wastes and those accepting only municipal solid waste (MSW).

A key study that considers the difference in effects between MSW and hazardous waste landfills is that by Elliott et al (2009), which investigated the association between the geographic density of landfills and frequency of congenital anomalies in communities within 2 km. This built on an earlier study by Elliott (2001), which found a statistically significant association between congenital anomalies and low birth weight in populations living at different distances from a large number of waste sites in the UK (Relative Risk (RR) = 1.05). However, the 2009 study found that the association disappeared for MSW landfills (RR = 0.99) when analysed separately from special waste landfills (RR = 1.08).

Matiello (2013) provides the most recent systematic review of studies of health impacts of landfills. It identifies 29 relevant, eligible papers evaluating health effects in communities living in the proximity of landfills. The outcomes considered in these studies were cancers, birth defects, respiratory diseases and total mortality. In summary, the findings of Matiello (2013) were that:

- 1 There was inadequate evidence to identify any relationship between landfills and increased cancer risk or total mortality.
- 2 There was evidence of risks of certain congenital anomalies (birth defects) and hospitalisation due to respiratory disease in communities near special waste landfills. However, the data suggested that there was no increased risk of these effects near landfills accepting only urban (i.e. municipal) solid waste.

In November 2015, the World Health Organization (WHO) held a meeting on “Waste and human health: evidence and needs”. The meeting report (WHO, 2015) highlighted that:

*In the most recent literature, health outcomes have been analysed that are less severe, but of a greater overall impact as more frequent in the exposed population. Several papers reported associations between exposure to odorous disposal facilities such as landfills, and respiratory symptoms and other non-specific symptoms in the population, such as noise and other problems due to annoyance.*

The WHO meeting report included suggested relative risk (RR) values that could be used to assess the order of magnitude of health impacts for alternative waste management scenarios. The health outcomes considered are congenital abnormalities and low birth weight in populations within 2 km of the landfill and respiratory disease in populations within 5 km of the landfill.

The RR values suggested for congenital abnormalities and low birth weight in populations within 2 km of a landfill are based on the earlier work of Elliott (2001) and do not reflect the more recent findings that these associations disappear for landfills that accept only MSW.



The value suggested for relative risks associated with respiratory disease<sup>11</sup> is based on the study by Matolini (2016), which found associations with mortality from lung cancer and respiratory diseases and with hospitalisation for respiratory diseases in communities living within 5 km around nine municipal solid waste landfills in the area of Lazio, Central Italy. Annual average exposures to H<sub>2</sub>S were predicted using generalised estimates of H<sub>2</sub>S emission rates (first order decay model) and a Lagrangian dispersion model. H<sub>2</sub>S was used as a tracer for emissions of all air pollutants from the landfill.

Matolini (2016) represents a more robust attempt to characterise exposure than many other studies that use distance from the landfill as a proxy for exposure. However, its relevance to potential health impacts of modern, well managed landfills is unclear. Only two of the landfills, representing 1% of the exposed cohort, were opened since 2000 (Viterbo and Roccasecca). The four largest landfill sites (by area) representing 63% of the exposed cohort, were opened in 1980 (Albano Laziale), 1987 (Roma) and 1991 (Latina and Guidonia) and. The study notes that only in the last two decades (i.e. since about 1995) have the landfills been required to adopt “*containments (including leachate collection and treatment, landfill cap construction and landfill gas collection and treatment)*”. Given the challenges of retrofitting collection systems into existing landfills, we consider it reasonable to assume that landfill gas controls at these landfills would not be as effective as at a new landfill, where these systems are incorporated into the landfill design and installed progressively. There is also no discussion of the operational controls to exclude hazardous waste. Consequently we consider that the relative risk estimates from this study should only be used as a screening assessment tool in the absence of a detailed, site-specific assessment.

In summary, the literature review found that the available information supports an increased risk of lung cancer, congenital abnormalities (birth defects) and low birth weight in populations living near landfills that accept hazardous waste. A correlation was found between predicted H<sub>2</sub>S concentrations and increased incidence of health effects, suggesting the effects are related to airborne contaminants. These effects were not evident around landfills accepting only MSW.

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<sup>11</sup> RR = 1.09 for an increment of 1 ng/m<sup>3</sup> hydrogen sulphide (H<sub>2</sub>S)

## Reference list for literature review

- Elliott P, Briggs D, Morris S, de Hoogh C, Hurt C, Jensen TK, Maitland I, Richardson S, Wakefield J, Jarup L (2001) Risk of adverse birth outcomes in populations living near landfill sites. *Br Med J* 323:363–368
- Elliott P., Richardson S., Abellan JJ., Thomson A., de Hoog C., Jaruo L., Briggs DJ. (2009) Geographic density of landfill sites and risk of congenital anomalies in England. *Occup Environ Med* 66:81–89
- Giusti L. A review of waste management practices and their impact on human health. (2009) *Waste Management*. 29(8), p 2227 –2239
- Mataloni F., Badaloni C., Golini MN., Bolignano A., Bucci S., Sozzi R., Forastiere F., Davoli M., Ancona C. (2016). Morbidity and mortality of people who live close to municipal waste landfills: a multisite cohort study. *International Journal of Epidemiology*, Volume 45, Issue 3, 1 June 2016, Pages 806–815
- Mattiello A., Chiodini P., Bianco E., Forgione N., Flammia I., Gallo C., Pizzuti R., Panicoet S. (2013) Health effects associated with the disposal of solid waste in landfills and incinerators in populations living in surrounding areas: a systematic review. *International Journal of Public Health*. 58(5): 725-735
- Porta D., Milani S., Lazzarino A., Perucci C. and Forastiere F. (2009) Systematic review of epidemiological studies on health effects associated with management of solid waste. *Environ Health*. 8: 60
- World Health Organization Meeting Report. “Waste and human health: evidence and needs” held in Bonn, Germany on 5-6 November 2015

**Appendix B: Ranking of threshold compounds in  
leachate and landfill gas**

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As outlined in section 5.4.3, a sub-set of the threshold contaminants in leachate in landfill gas have been selected using a screening assessment taking into account their toxicity and concentration. The ranking of threshold compounds in landfill gas is set out in Appendix B Table 1 and for leachate is set out in Appendix B Table 3. These lists do not include genotoxic compounds or PFAS, which have all been included for detailed evaluation as a matter of course.

A number of reduced sulphur compounds have been grouped together and evaluated against a single toxicity factor for “Total Reduced Sulphur”. These compounds are listed in Appendix B Table 2.



**Appendix B Table 1: Ranking of threshold compounds in landfill gas**

	Compound	Concentration adopted for HHRA ( $\mu\text{g}/\text{m}^3$ )	Long term Texas ESL ( $\mu\text{g}/\text{m}^3$ )	Ratio	Number of analyses in landfill gas	Rationale for adopted HHRA concentrations
Top 10 threshold compounds	Total reduced sulphur (see table below)	1043877				
	Trichloromethane (chloroform)	207845	10	20784.5	15	Second highest measured value
	Carbon tetrachloride (tetrachloromethane)	48075	13	3698.08	14	Second highest measured value
	Tetrachloroethylene	58491	26	2249.65	17	Second highest measured value
	Formaldehyde	4558	3.3	1381.21	11	Second highest measured value
	Xylene	239600	180	1331.11	6	Second highest measured value
	Methyl isobutyl ketone	43373	82	528.94	17	Second highest measured value
	Toluene	452400	1200	377.00	17	Second highest measured value
	Chlorobenzene	12053	46	262.02	15	Second highest measured value
	Chloroethane	66250	270	245.37	15	Second highest measured value
Top 20 threshold compounds	Ethylbenzene	114800	570	201.40	6	Second highest measured value
	Styrene	25071	140	179.08	17	Second highest measured value
	Hexane	27190	200	135.95	7	Second highest measured value
	Ethanol	170703	1880	90.80	17	Second highest measured value
	1-butanethiol	150	1.8	83.33	6	Second highest measured value
	Chloromethane	4958	103	48.14	16	Second highest measured value
	1,1,2,2-tetrachloroethane	330	7	47.14	6	Half Limit of Detection (LOD)
	Methyl ethyl ketone	93461	2600	35.95	18	Not detected in any samples and LOD too high to be useful. Value adopted is mean from UK Environment Agency
	1,3-butadiene	265	9.9	26.77	11	Second highest measured value

	Compound	Concentration adopted for HHRA ( $\mu\text{g}/\text{m}^3$ )	Long term Texas ESL ( $\mu\text{g}/\text{m}^3$ )	Ratio	Number of analyses in landfill gas	Rationale for adopted HHRA concentrations
	Isopropyl alcohol	11990	492	24.37	6	95th percentile
Other threshold compounds	Mercury	0.58	0.025	23.20	1	Not detected in any samples and LOD too high to be useful. Value adopted is mean from UK Environment Agency
	Dichlorobenzenes	3000	160	18.75	4	Highest
	1-propanethiol	20	1.6	12.50	6	Second highest measured value
	Acetone	54450	4800	11.34	17	Second highest measured value
	Benzyl chloride	30	5	6.00	6	Only result above LOD
	Pentane	37870	7100	5.33	6	Second highest measured value
	Dichlorodifluoromethane	10870	5000	2.17	6	Second highest measured value
	1,2-dichloroethene	1410	790	1.78	6	Second highest measured value
	Bromodichloromethane	30	70	0.43	6	Second highest measured value
	Trans-1,2-dichloroethene	330	790	0.42	6	Second highest measured value
	Dichlorofluoromethane	670	4200	0.16	6	Second highest measured value
	1,1-dichloroethylene	10	100	0.10	5	Second highest measured value
	1,1-dichloroethane	4	400	0.010	6	Only result greater than LOD
	Trichlorofluoromethane	40	5600	0.007	6	Second highest measured value
	Chlorodifluoromethane	10	1800	0.006	6	Second highest measured value
	1,1,1-trichloroethane	6.5	1500	0.004	7	Half Limit of Detection (LOD)
	Butane	6485	No value*	-	12	Half Limit of Detection (LOD)
Ethane	3355	No value*	-	13	Half Limit of Detection (LOD)	
Propane	29530	No value*	-	13	Second highest measured value	

\* Simple asphyxiants, essentially non-toxic

**Appendix B Table 2: Compounds assessed as total reduced sulphur**

<b>Compound</b>	<b>Number of analyses</b>	<b>Concentration adopted for HHRA (<math>\mu\text{g}/\text{m}^3</math>)</b>	<b>Rationale for adopted HHRA concentrations</b>
Carbon disulphide	18	18963	Second highest measured value
Carbonyl sulphide	13	13140	Second highest measured value
Dimethyl disulphide	17	5887	Second highest measured value
Dimethyl sulphide	17	10817	Second highest measured value
Ethyl mercaptan	17	19970	95th percentile of measured values
Hydrogen sulphide	38	999730	Second highest measured value
Methyl mercaptan	18	5150	Second highest measured value





**Appendix B Table 3: Ranking of threshold compounds in leachate**

	Compound	Concentration adopted for HHRA (mg/L)	Drinking water guideline (mg/L)	Ratio	Number of analyses	Rationale for adopted HHRA concentration	Drinking water guideline reference
Top 15 threshold compounds	Pyridine	5	0.002	2500	1	Waste acceptance criteria	US-EPA Region 3
	Heptachlor	0.025	0.00003	833	60	Half Limit of Detection (LOD)	WHO (2017)
	Methoxychlor	10	0.02	500	0	Waste acceptance criteria	NZ MAV
	2,4-Dichlorophenoxyacetic acid	10	0.04	250	0	Waste acceptance criteria	NZ MAV
	Chlordane	0.03	0.0002	150	0	Waste acceptance criteria	NZ MAV
	2,4,5-Trichlorophenoxyacetic acid	1	0.01	100	0	Waste acceptance criteria	NZ MAV
	Cresols, total	13	0.2	65	97	Highest measured concentration	US-EPA Region 3
	Hexachlorobutadiene	0.025	0.0007	36	61	Half LOD	NZ MAV
	Hexachlorobenzene	0.0068	0.0002	34	1	Highest measured concentration	MDH
	Chromium total	1.4	0.05	28	135	Highest measured concentration	NZ MAV
	Endrin	0.025	0.001	25	61	Half LOD	NZ MAV
	Hexachloroethane	0.025	0.001	25	61	Half LOD	US EPA cited in MoH (2019)
	Lead total	0.18	0.01	18	130	Highest measured concentration	NZ MAV
	Methyl ethyl ketone, total	58.4	4	15	27	Highest measured concentration	Minnesota Department of Health
	1,1,2,2-tetrachloroethane	0.025	0.002	13	61	Half LOD	US EPA cited in MoH (2019)

	Compound	Concentration adopted for HHRA (mg/L)	Drinking water guideline (mg/L)	Ratio	Number of analyses	Rationale for adopted HHRA concentration	Drinking water guideline reference
Top 20 threshold compounds	Barium	6.4	0.7	9.1	15	Highest measured concentration	NZ MAV
	PCB (Polychlorinated biphenyls)	0.003	0.0005	6.0	2	Highest measured concentration (sum of congeners)	US EPA cited in MoH (2019)
	2,4,5-Trichlorophenol	0.005	0.001	5.0	62	Highest measured concentration	US EPA cited in MoH (2019)
	2,4-Dinitrotoluene	0.025	0.005	5.0	61	Half LOD	EPA health advisory level
	Carbon tetrachloride	0.02	0.005	4.0	1	Highest measured concentration	NZ MAV
Other threshold compounds	Selenium total	0.038	0.01	3.8	1	Highest measured concentration	NZ MAV
	Cadmium total	0.012	0.004	3.0	60	Highest measured concentration	NZ MAV
	styrene	0.079	0.03	2.6	23	Highest measured concentration	NZ MAV
	ethylbenzene	0.649	0.3	2.2	52	Highest measured concentration	NZ MAV
	Xylenes, total	1.13	0.6	1.9	111	Highest measured concentration	NZ MAV
	Toluene	1.11	0.8	1.4	49	Highest measured concentration	NZ MAV
	acetone	3.8	3	1.3	29	Half 2 <sup>nd</sup> highest LOD	MDH
	Nitrobenzene	0.009	0.008	1.1	1	Highest measured concentration	Derived in MoH (2019)

Compound	Concentration adopted for HHRA (mg/L)	Drinking water guideline (mg/L)	Ratio	Number of analyses	Rationale for adopted HHRA concentration	Drinking water guideline reference
Mercury total	0.0065	0.007	0.93	26	Highest measured concentration	NZ MAV
1,1-Dichloroethene	0.025	0.03	0.83	60	Half LOD	DWSNZ (2015) cited in MOH (2019)
bromodichloromethane	0.025	0.06	0.42	61	Half LOD	NZ MAV
trans-1,2-dichloroethene	0.025	0.06	0.42	61	Half LOD	NZ MAV
cis-1,2-dichloroethene	0.02	0.06	0.33	18	Highest measured concentration	NZ MAV
1,1-dichloroethane	0.022	0.08	0.28	2	Highest measured concentration	Minnesota Department of Health
trichlorofluoromethane	0.05	0.2	0.25	61	Half LOD	Minnesota Department of Health
Cyanide	0.098	0.6	0.16	0	Maximum value from MWI RUST	NZ MAV
carbon disulphide	0.095	0.7	0.14	4	Highest measured concentration	Minnesota Department of Health
dichlorodifluoromethane	0.05	0.5	0.10	60	Half LOD	Minnesota Department of Health
Chloroform	0.025	0.4	0.06	60	Half LOD	NZ MAV
Chlorobenzene	0.006	0.1	0.06	18	Highest measured concentration	Minnesota Department of Health

	Compound	Concentration adopted for HHRA (mg/L)	Drinking water guideline (mg/L)	Ratio	Number of analyses	Rationale for adopted HHRA concentration	Drinking water guideline reference
	Tetrachloroethylene (tetrachloroethene)	0.002	0.05	0.04	2	Highest measured concentration	NZ MAV
	1,4-Dichlorobenzene	0.0143	0.4	0.04	29	Highest measured concentration	NZ MAV
	2,4,6-Trichlorophenol	0.0029	0.2	0.01	1	Highest measured concentration	NZ MAV
	Silver total	0.0011	0.1	0.01	8	Half LOD	DWSNZ 2005
	1,1,1-trichloroethane	0.001	5	0.00	1	Highest measured concentration	Minnesota Department of Health
	chloromethane	0.05	-	-	4	Highest measured concentration	
	Dichlorobenzenes, total	0.084	-	-	61	Highest measured concentration	

- indicates no data.

## **Appendix C: Toxicity factors**

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- **Unit risk and slope factors for genotoxic carcinogens**
- **Inhalation toxicity factors (threshold compounds)**
- **Ingestion toxicity factors (threshold compounds)**

## C1 Unit risk and slope factors for genotoxic carcinogens

Compound	Inhalation unit risk ( $\mu\text{g}/\text{m}^3$ ) <sup>-1</sup>	Source	Ingestion slope factor ( $\mu\text{g}/\text{kg}\cdot\text{day}$ ) <sup>-1</sup>	Source	Comments
Acetaldehyde	$9 \times 10^{-7}$	MfE, 2002			No appreciable exposure via ingestion.
Acrylonitrile	$2 \times 10^{-5}$	WHO, 2000	0.001	OEHHA	
Arsenic	$1.5 \times 10^{-3}$	WHO, 2000	0.00116	MfE, 2011	Ingestion slope factor converted from risk specific oral dose in MfE (2011) of 0.0086 $\mu\text{g}/\text{kg}\cdot\text{day}$ ( $10^{-5}$ risk level)
Benzene	$6 \times 10^{-6}$	WHO, 2000	$3.5 \times 10^{-5}$	MoH, 2017	
1,2-dibromoethane	$7.1 \times 10^{-5}$	OEHHA	$7.5 \times 10^{-4}$	WHO, 2017	
1,2-dichloroethane	$2.1 \times 10^{-5}$	OEHHA	$1.0 \times 10^{-5}$	MoH, 2017	Sum of cis- and trans-isomers. WHO considered there was insufficient data to set inhalation unit risk, so OEHHA value has been used.
Dichloromethane	$1 \times 10^{-6}$	OEHHA	$1.4 \times 10^{-5}$	OEHHA	IARC considered that there is extensive evidence of genotoxicity and overall that the mechanistic evidence for dichloromethane carcinogenesis is strong. WHO (2002) considered that carcinogenicity was not the critical endpoint for risk assessment purposes and that formation of COHb was a more direct indication of toxic effects. On this basis, dichloromethane has also been evaluated as a threshold carcinogen for oral and inhalation exposure.
1,2-dichloropropane	$1 \times 10^{-5}$	OEHHA	$3.6 \times 10^{-5}$	OEHHA	Recently re-evaluated by IARC and classified as a Class 1 carcinogen. Has not been re-assessed by WHO.
Pentachlorophenol			$3.3 \times 10^{-5}$	WHO, 2017	No appreciable exposure via inhalation
Toxaphene	$3.2 \times 10^{-4}$	US EPA IRIS database	$1.1 \times 10^{-3}$	US EPA IRIS database	
Trichloroethylene	$4.3 \times 10^{-7}$	WHO, 2000	$5.9 \times 10^{-6}$	OEHHA	
Vinyl chloride	$1 \times 10^{-6}$	WHO, 2000	0.001	MOH, 2017	

## C2 Inhalation toxicity factors (threshold compounds)

Compound	Tolerable concentration ( $\mu\text{g}/\text{m}^3$ )	Source	Comments
Carbon tetrachloride (tetrachloromethane)	6.1	WHO, 1999	Based on the weight of evidence it can be concluded that the hepatic tumours are induced by an indirect mechanism and that a tolerable daily intake or concentration can be derived.
Chlorobenzene	1000	OEHHA	
Chloroethane	NA		In a review of toxicity data for chloroethane (US EPA, 2007), the US EPA was able to determine a sub-chronic (14 days) reference concentration of 4000 $\mu\text{g}/\text{m}^3$ . There was no data available to assess the carcinogenic mode of action or calculate a unit risk.
Dichloromethane	450	WHO, 2000	
Formaldehyde	9	OEHHA	The New Zealand ambient air quality guideline (MfE, 2002) of 100 $\mu\text{g}/\text{m}^3$ is set for acute exposure (30 minute average) to avoid sensory irritation. OEHHA chronic REL based on respiratory effects has been adopted.
Hexachlorobutadiene			
Mercury	0.33	MfE, 2002	
Methyl ethyl ketone (2-butanone)	5000	US EPA IRIS database	US EPA Reference Concentration for inhalation exposure.
Methyl isobutyl ketone	3000	US EPA IRIS database	US EPA Reference Concentration for inhalation exposure.
Styrene	0.26	WHO, 2000	Although genotoxic effects in humans have been observed at relatively low concentrations, they were not considered by WHO (2000) as critical endpoints for development of a guideline, in view of the equivocal evidence for the carcinogenicity of styrene.
1,1,2,2-tetrachloroethane	3.4	WHO, 1998	See discussion under oral toxicity factors.
Tetrachloroethylene	250	WHO, 2000	WHO was unable to set an air quality guideline based on cancer effects and therefore based their guideline on non-neoplastic effects.
Toluene	260	WHO, 2000	
Total reduced sulphur (hydrogen sulphide, carbonyl	20	WHO, 2003	



Compound	Tolerable concentration ( $\mu\text{g}/\text{m}^3$ )	Source	Comments
sulphide, dimethyl disulphide ethyl mercaptan, methyl mercaptan)			
Trichloromethane (chloroform)	140	WHO, 2004	
Xylene	870	WHO, 1997	

### C3 Ingestion toxicity factors (threshold compounds)

Compound	Tolerable daily intake (µg/kg bw-day)	Source	Comment
Carbon tetrachloride	1.4	MOH, 2017	MoH (2017) states that on the basis of available data, carbon tetrachloride can be considered to be a non-genotoxic compound and therefore it has been assessed as a threshold compound based on hepatotoxic effects.
Chlordane	0.5	MOH, 2017	
Chlorobenzene	85.7	WHO, 2004c	
Chloroethane			In a review of toxicity data for chloroethane (US EPA, 2007), the US EPA was able to determine a sub-chronic (14 days) oral Reference Dose of 100 µg/kg-day. There was no data available to assess the carcinogenic mode of action or calculate a slope factor.
Chromium III	1500	MfE, 2011	
Chromium VI	3	MfE, 2011	MfE (2011) states that while Chromium (VI) is classified as a known human carcinogen via the inhalation route, there is limited data on carcinogenicity for exposure via the oral route. Supported by the indication for a greater reducing capacity of chromium (VI) to chromium (III) via the oral route as compared to the inhalation route, the threshold approach has been used.
Cresols	50	MOH, 2017	No MAV set in New Zealand. Based on USEPA Reference Dose (RfD) cited in MoH, 2017.
Dichloromethane	6	MOH, 2017	MoH (2017) states that the balance of evidence suggests that dichloromethane is not genotoxic and therefore it has been assessed as a threshold compound.
2,4-Dichlorophenoxyacetic acid	10	MOH, 2017	
Endrin	0.2	WHO, 2017	
Formaldehyde	150	MOH, 2017	MoH (2017) states that although formaldehyde is considered to be probably carcinogenic to humans by inhalation, the weight of evidence indicates that formaldehyde is not carcinogenic by the oral route.
Heptachlor	0.125	MOH, 2017	
Hexachlorobutadiene	0.2	WHO, 2017	

Compound	Tolerable daily intake (µg/kg bw-day)	Source	Comment
Hexachloroethane	1	MOH, 2017	No MAV set in New Zealand. Based on USEPA Reference Dose (RfD) cited in MoH, 2017.
Hexachlorobenzene	0.014	WHO, 2004	Value has been calculated using two different approaches as set out in WHO (2004b) and the lower value based on neoplastic effects has been adopted as a threshold (i.e. this is lower than the value equivalent to a 10 <sup>-5</sup> excess cancer risk).
Lead	1.9	MfE, 2011	
Methoxychlor	5	MOH, 2017	
Methyl ethyl ketone (2-butanone)	600	MOH, 2017	No MAV set in New Zealand. Based on USEPA Reference Dose (RfD) cited in MoH, 2017.
Methyl isobutyl ketone			No MAV set in New Zealand. No drinking water guidelines set by WHO. No oral RfD set by US EPA.
Pentachlorophenol	0.3	MfE, 2011	MfE (2011) recommends pentachlorophenol should be assessed as a threshold compound.
PFOA	0.16	Australian DoH, 2017	
PFOS/PFHxS	0.02	Australian DoH, 2017	
Pyridine	1	US EPA Oral Reference dose (IRIS)	No MAV set in New Zealand and no WHO drinking water guideline. Based on USEPA Reference Dose (RfD) cited in MoH, 2017.
Styrene	7.7	MOH, 2017	
1,1,2,2-tetrachloroethane	1.2	WHO, 1998	WHO (1998) states that guidance values have been determined on the basis of the potency of 1,1,2,2-tetrachloroethane to induce liver tumours in mice, as this is the toxicological end-point for which the dose–response relationship is best characterized. However, there are suggestive but incomplete data indicating that tumours may be induced by a non-genotoxic mechanism. The TDI values are derived based on the lower end of the range equivalent to a 10 <sup>-5</sup> risk.
Tetrachloroethylene	14	MOH, 2017	In view of the overall evidence for non-genotoxicity and evidence for a saturable metabolic pathway leading to kidney tumours in rats, it is considered appropriate to use a tolerable daily intake approach for the derivation of a MAV for tetrachloroethene in drinking-water.

Compound	Tolerable daily intake (µg/kg bw-day)	Source	Comment
Toluene	223	MOH, 2017	
Trichloroethylene	1.46	MOH, 2017	Inhalation exposure assessed as carcinogenic by WHO. MoH (2017) assessed trichloroethylene as a threshold compound based on developmental toxicity.
Trichloromethane (chloroform)	15	WHO, 2017	IARC classified chloroform as possibly carcinogenic to humans (Class 2B). According to WHO (2017) the weight of evidence for genotoxicity of chloroform is considered negative.
2,4,5-Trichlorophenoxyacetic acid	3	MOH, 2017	
Xylene	250	MOH, 2017	

## **C4 Discussion of toxicity factors for reduced sulphur compounds**

Two Canadian environmental agencies (Alberta Environment and Ontario Ministry of the Environment (MoE)) have reviewed the toxicity of reduced sulphur compounds for the purpose of developing ambient air quality objectives/standards.

Alberta Environment (2004) reviewed relevant toxicity data and found that there was insufficient effects information available to support the development of guidelines for specific reduced sulphur compounds.

The Ontario MoE (2007) similarly found that there was inadequate data to characterise the dose-response relationships of individual total reduced sulphur species or for a mixture of total reduced sulphur species (TRS). However, they found that recent epidemiological studies suggest that exposure to TRS exhibits similar targets for adverse effects as that observed with exposure to H<sub>2</sub>S (e.g., irritation, respiratory and CNS effects). Therefore they concluded that H<sub>2</sub>S may provide an appropriate surrogate for the toxicological assessment and in the development of air quality standards for TRS. The ambient air quality criteria (AAQC) for H<sub>2</sub>S recommended in the review was also recommended to be used as an AAQC for TRS in the vicinity of specific sources such as municipal sewage treatment plants, where the majority of TRS is made up of H<sub>2</sub>S.

The Ontario MoE adopted an AAQC for H<sub>2</sub>S of 7 µg/m<sup>3</sup> using the same study used by the WHO (2003) to develop a Tolerable Concentration of 20 µg/m<sup>3</sup> (a 90 day inhalation study in rats where the critical endpoint was nasal lesions in the olfactory mucosa). The differences between the two recommended values derives from differences in conversion of the equivalent gas dosage and uncertainty factors.

For this study, the Tolerable Concentration for H<sub>2</sub>S developed by the WHO of 20 µg/m<sup>3</sup> has been adopted for the mixture of total reduced sulphur species (carbon disulphide, carbonyl sulphide, dimethyl disulphide, dimethyl sulphide, ethyl mercaptan, hydrogen sulphide, methyl mercaptan).

The WHO states that ingestion of hydrogen sulphide is of no relevance for humans and there are no human ingestion toxicity data (WHO, 2003). Consequently ingestion of reduced sulphur species has not been considered in this study.

## **C5 References for toxicity factors**

Alberta Environment. (2004). Assessment Report on Reduced Sulphur Compounds for Developing Ambient Air Quality Objectives. Prepared by AMEC Earth & Environmental Limited and University of Calgary

Ministry of Health. (2017). Guidelines for Drinking-water Quality Management for New Zealand (3rd edn) Volume 3 Datasheets - Chemical and physical determinands. Updated June 2019. Wellington

Ministry of Health. (2018). Drinking-water Standards for New Zealand 2005 (revised 2018). Wellington

Ministry for the Environment. (2002). Ambient Air Quality Guidelines. 2002 Update. Wellington

Ministry for the Environment. (1999). Guidelines for Assessing and Managing Petroleum Hydrocarbon Contaminated Sites in New Zealand (Revised 2011). Appendix 4L - Toxicity Assessment. Wellington

Ministry for the Environment. (2011). Methodology for Deriving Standards for Contaminants in Soil to Protect Human Health. Wellington

Ontario Ministry of the Environment. (2007). Ontario Air Standard For Total Reduced Sulphur

- United States Environmental Protection Agency. (2007). Provisional Peer Reviewed Toxicity Values for Chloroethane (CASRN 75-00-3). Office of Research and Development, Cincinnati
- World Health Organization. (1997). Environmental Health Criteria 190 Xylenes. Geneva
- World Health Organization. (1998). Concise International Chemical Assessment Document 3. 1,1,2,2-tetrachloroethane. Geneva
- World Health Organization. (1999). Environmental Health Criteria 208 Carbon Tetrachloride. Geneva
- World Health Organization. (2000). Air Quality Guidelines for Europe Second Edition. WHO Regional Publications, European Series, No. 91
- World Health Organization. (2003). Concise International Chemical Assessment Document 53. Hydrogen Sulfide: Human health aspects. Geneva
- World Health Organization. (2004). Concise International Chemical Assessment Document 58 Chloroform. Geneva
- World Health Organization. (2004b). Hexachlorobenzene in Drinking-water. Background document for development of WHO Guidelines for Drinking-water Quality. Geneva
- World Health Organization. (2004c). Monochlorobenzene in Drinking-water. Background document for development of WHO Guidelines for Drinking-water Quality. Geneva
- World Health Organization. (2017). Guidelines for drinking-water quality: fourth edition incorporating the first addendum. Geneva
- Australian Government Department of Health. (2017). Health based guidance values for PFAS for use in site investigations in Australia. Retrieved from:  
<https://www.health.gov.au/internet/main/publishing.nsf/Content/ohp-pfas-hbgv.htm>

**Appendix D: Contact recreational exposure -  
screening assessment of**

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According to WHO (2003) exposure to contaminants in surface waters during contact recreation (e.g. swimming) is primarily driven by (largely unintended) ingestion. WHO suggests that environmental quality standards for chemicals in recreational waters could be based on the assumption that recreational water makes only a relatively minor contribution to intake. The contact recreation water quality guidelines have been set at 10% of the drinking water guidelines on the basis of an assumed 200 mL/day intake via unintended ingestion, i.e. 10% of the assumed drinking water intake of 2 L/day.

An initial screening assessment has been undertaken by comparing the predicted concentrations in the Hōteu River (POE #3) against a value of 10% the drinking-water guidelines. As shown Appendix D Table 1, the maximum concentration of any contaminant is less than 1% the screening criterion. For this reason, a detailed assessment of exposure via contact recreation has not been carried out.

**Appendix D Table 1: Screening assessment for recreational exposure to estimated contaminant concentrations in the Hōteu River.**

Chemical of concern	Hoteu River	Drinking water guideline	10% drinking water guideline	% Concentration of drinking water guideline
	mg/L	mg/L	mg/L	%
1,2-dibromoethane	4.30E-40	0.0004	0.00004	1.08E-33
1,2-dichloroethane	1.10E-246	0.03	0.003	3.67E-242
1,2-dichloropropane	7.60E-10	0.05	0.005	1.52E-05
Arsenic	7.60E-06	0.01	0.001	0.76
Chlordane, total	4.70E-08	0.0002	0.00002	0.235
Chromium	3.90E-06	0.05	0.005	0.078
Dichloromethane	2.70E-27	0.02	0.002	1.35E-22
Endrin	3.10E-08	0.001	0.0001	0.031
Hexachlorobutadiene	4.30E-09	0.0007	0.00007	0.00614
Lead	8.00E-06	0.01	0.001	0.80
PFOA	1.76E-10	560	56	3.14E-10
PFOS/PFHxS	2.92E-10	70	7	4.17E-9
Styrene	5.00E-252	0.03	0.003	1.67E-247
Toxaphene	8.60E-08	0.003	0.0003	0.0287
Trichloroethylene	3.70E-247	0.02	0.002	1.85E-242
Trichloromethane (chloroform)	9.90E-119	0.4	0.04	2.48E-115
Xylene	5.82E-52	0.6	0.06	9.70E-49

Table note:

Compounds with modelled concentrations that are so low they are “indistinguishable from zero” are not included in the table



## **Appendix E: Modelling the wet and dry deposition of airborne gas-phase contaminants**

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## **E1 Introduction**

Dispersion modelling has been used to predict wet and dry deposition rates of contaminant emissions from the working face, generators and flare from the proposed landfill. Dispersion modelling has been undertaken using the most recent version of the CALPUFF air dispersion model (version 7.2.1).

The Air Quality Assessment Report (Technical Report D) describes the modelling methodology used, including meteorological parameters, selection of model period, meteorological surface observation input data, terrain and land cover and discharge parameters for area and point sources.

## **E2 Emission rates**

The sources included in the modelling are:

- The residual emissions from the flare and generators assuming 97% destruction efficiency for the contaminants of interest (based on the US EPA AP 42 default destruction efficiency).
- Fugitive landfill gas emissions from an assumed 10,000 m<sup>2</sup> working face.

The peak rate of landfill gas generation, which will occur around the year of landfill closure, is estimated to be between 8,800 and 11,100 m<sup>3</sup>/hour. The rate of emissions from the flares and generators is the same as that adopted in the Air Quality Assessment, i.e. 8,200 m<sup>3</sup>/hour landfill gas combusted in the generators and the balance of 2,900 m<sup>3</sup>/hour combusted in a flare.

Once a landfill is closed, the landfill gas collection efficiency is assumed to be 95%, with the balance of (uncollected) landfill gas percolating through the cap and being treated by bioremediation i.e. negligible fugitive emissions.

While the landfill is still being filled, the quantum of uncontrolled landfill gas emissions can be calculated on an area coverage basis. In the year immediately prior to closure, the maximum open area of the landfill will be 1 hectare (10,000 m<sup>2</sup>) compared to the total capped area of 60 hectares. Therefore, it is assumed, in the first instance that 59/60 or 98.33% of the landfill gas (10,915 m<sup>3</sup>/hour) is either collected by the treatment system or treated by bioremediation through the cap. Further consideration is then required of the balance of 185 m<sup>3</sup>/hour landfill gas.

The Norfolk Pine Landfill gas collection well system used by WMNZ is progressively developed as the landfill is filled, meaning that there will be effective capture of landfill gas in the deeper, older areas of waste beneath the current filling area. The approximate depth of waste in the last area to be filled will be 90 m, which will be filled, at different times, in lifts of the order of 30 m. Therefore, fugitive landfill gas emissions from the working face is conservatively assumed to comprise 1/3 of the volume of landfill gas generated in the waste volume under this area. The reason this is a conservative is that it takes about 6 months for landfill gas to be generated within the waste. Overall, the estimated volume of landfill gas that could be released, untreated through the working face is 55.5 m<sup>3</sup>/hour.

For the purposes of a conservative assessment, this untreated landfill gas emission rate has been doubled, and a fugitive landfill gas emission rate of 110 m<sup>3</sup>/hour has been used in the dispersion modelling. For a 10,000 m<sup>2</sup> working face, this equates to a surface emission rate of 0.011 m<sup>3</sup>/m<sup>2</sup>-hour.

The modelling has been carried out assuming a unitary emission rate (e.g. 1 g/s), which is then adjusted in the post-processing based on the measured concentration of each contaminant in the landfill gas.

### E3 Dry deposition model parameters

A full resistance model is provided in CALPUFF for the computation of dry deposition rates of gases and particulate matter. Dry deposition is calculated as a function of geophysical parameters, meteorological conditions, and properties of the pollutant species.

CALPUFF calculates pollutant specific dry deposition fluxes from various user-inputted parameters as shown in Appendix E Table 1.

**Appendix E Table 1: Model parameters for vapour phase dry deposition**

Parameter	Description	Value
Pollutant diffusivity in air	This parameter characterizes the ability of a gas to mix spontaneously with and spread throughout another gas.	Variable (refer to Appendix E Table 3)
Aqueous phase dissociation constant	This is the solubility enhancement factor due to the aqueous phase dissociation of the pollutant.	Constant at 1 (dimensionless) Note: This parameter is only used by the model where pollutants interface with a water surface. No water surfaces are included in the modelled area, so this parameter is ignored by the model.
Pollutant reactivity	Reactivity characterizes the pollutant reactivity of the depositing gas with a surface.	8 (dimensionless) The model default pollutant reactivity value for xylene/toluene has been used.
Mesophyll resistance	This is the resistance to dissolution or reaction of the pollutant in the mesophyll cells and depends on the solubility and reactivity of the pollutant.	Set to model default 0 s/sm The model default pollutant reactivity value for xylene/toluene has been used.
Henry's Law coefficient	This parameter is the ratio of gas to liquid phase concentration of the pollutant.	Variable (refer to Appendix E Table 3) Note: This parameter is only used by the model where pollutants interface with a water surface. No water surfaces are included in the modelled area, so this parameter is ignored by the model.

### E4 Wet deposition model parameters

An empirical scavenging coefficient approach is used in CALPUFF to compute the depletion and wet deposition fluxes due to precipitation scavenging. Wet deposition flux is calculated by multiplying the scavenging ratio by a vertically integrated concentration. The scavenging ratio is the product of a scavenging coefficient, precipitation rate, and precipitation type (i.e. liquid or frozen precipitation).

Scavenging of vapour phase pollutants during rain events is likely to be related to the pollutant's solubility in water, which is represented in part by the pollutant specific Henry's law coefficient. The dimensionless Henry's law coefficient increases with decreasing solubility of the pollutant in water. Wet deposition has also only been modelled for certain pollutants (see Appendix E Table 3), based on an evaluation of the each pollutant's Henry's law coefficient and solubility in water.

Due to limited data to calculate pollutant-specific scavenging coefficients, pollutant coefficients have been based on that of a small particle (of PM<sub>0.56</sub>). Parameters used by the CALPUFF modelling suite for the calculation of wet deposition are provided in Appendix E Table 2.

**Appendix E Table 2: Model parameters for wet deposition**

Parameter	Description	Value
Scavenging coefficient (liquid precipitation)	The pollutant specific scavenging coefficient for liquid precipitation used to determine estimates of wet removal.	Constant at $4.71 \times 10^{-5} \text{ s}^{-1}$
Scavenging coefficient (frozen precipitation)	The pollutant specific scavenging coefficient for frozen precipitation used to determine estimates of wet removal.	Constant at $1.57 \times 10^{-5} \text{ s}^{-1}$ Note: no frozen precipitation is likely within modelled area.

## E5 COPC-specific parameters

Non-default parameters used in the calculation of dry deposition flux and the inclusion of wet deposition modelling of specific pollutants is described in Appendix E Table 3.

The majority of organic compounds have similar physical parameters to xylene and so, for ease of computation, have been modelled as xylene. Reduced sulphur compounds, ethylene dibromide and vinyl chloride have been modelled using their own specific parameters.

**Appendix E Table 3: Pollutant specific model parameters**

Pollutant	Pollutant diffusivity in air ( $\text{cm}^2/\text{s}$ )	Henry's Law coefficient (dimensionless)	Wet deposition modelled?
Acetaldehyde	0.1509*	0.04*	Yes
Acetone	0.1509*	0.04*	Yes
Acrylonitrile	0.1509*	0.04*	Yes
Benzene	0.1509*	0.04*	No
Bromodichloromethane	0.1509*	0.04*	No
Carbon tetrachloride	0.1509*	0.04*	No
Carbonyl sulphide	0.131	0.0492	No
Chloroform	0.1509*	0.04*	No
Dichloroethane, 1,2-	0.1509*	0.04*	No
Dichloroethylene, cis-1,2-	0.1509*	0.04*	No
Dichloroethylene, trans-1,2	0.1509*	0.04*	No
Dichloropropane, 1,2-	0.1509*	0.04*	No
Dimethyl disulphide	0.1509*	0.04*	No
Ethyl benzene	0.1509*	0.04*	No
Ethyl mercaptan	0.1509*	0.04*	No
Ethylene dibromide	0.0217	0.0293	No
Formaldehyde	0.1509*	0.04*	Yes
Hydrogen sulphide	0.174	0.00869	No
Methyl ethyl ketone	0.1509*	0.04*	Yes
Methyl mercaptan	0.13	0.00312	Yes
Methylene chloride	0.1509*	0.04*	No
Styrene	0.1509*	0.04*	No

Pollutant	Pollutant diffusivity in air (cm <sup>2</sup> /s)	Henry's Law coefficient (dimensionless)	Wet deposition modelled?
Tetrachloroethylene	0.1509*	0.04*	No
Trichloroethylene	0.1509*	0.04*	No
Vinyl chloride	0.106	3.49	No
Xylene	0.1509*	0.04*	No

\* = The model default dry deposition parameters for xylene have been used for this pollutant.

## **Appendix F: Surface water media concentration**

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The primary indicators of leachate contamination in surface water include conductivity, pH, ammoniacal nitrogen (ammonia), boron and chloride. The proposed monitoring programme includes both continuous monitoring and regular grab samples at the outlet for laboratory analysis. The monitoring results will be compared to set trigger levels.

The proposed trigger levels for conductivity, boron and sulphate will be developed based on the baseline monitoring results which commenced in 2018 and is continuing. The trigger level for ammonia will be based on the National Policy Statement for Freshwater Management (August 2017) which sets a value based on the attribute state of the stream and is not directly linked back to the site specific baseline values. Therefore, we consider the use of ammonia as an indicator of leachate in the ponds is appropriate.

The potential concentrations within the immediate downstream environment has been undertaken based using ammonia as an indicator of leachate discharges into the wetland (Appendix F Table 1).

The calculation has been undertaken on the following basis:

- The concentration of ammonia in the leachate has been based on sampling undertaken at Redvale Landfill;
- The wetland volume has been based on the permanent water volume;
- The ammonia discharge concentration has been based on the National Policy Statement for Freshwater Management (August 2017), a value of 0.05 mg/l has been chosen which is the maximum value for an Attribute State A stream, and is within the annual minimum for an Attribute State B stream;
- Daily rainfall data for the period 2014 to 2018 from Mahurangi RAW has been used to calculate the mass of stormwater discharge during each rain event;
- It has been assumed that the maximum mass discharge during any rainfall event equals the total mass present in the wetland;
- The total volume of water discharged during each rainfall event has been calculated based on the rational method;
- A 50% dilution factor has been added which represents the contributing volume the adjacent Valley 2; and
- To calculate a long term average concentration of leachate present in the receiving environment, the total mass of leachate discharged over the period has been divided by the total volume of water discharged during the same period with the dilution factor applied.

It is considered that the approach undertaken is highly conservative as it assumes that the concentration is always discharged at the maximum allowable concentration. As outlined in the surface water assessment, in the event that monitoring indicates the presence of leachate, remedial actions are required to be undertaken including ceasing or avoiding any discharges from the wetland.

**Appendix F Table 1: Contaminant concentrations in the stream confluence (POE#1) from leachate in potential surface water run-off.**

Contaminant	Concentration in leachate	Concentration in stream confluence from surface water run-off
	mg/L-leachate	mg/L-water
1,1,2,2-tetrachloroethane	0.025	7.60E-08
1,2-dibromoethane	0.02	6.08E-08
1,2-dichloroethane	0.0018	5.47E-09
1,2-dichloropropane	0.0016	4.86E-09
2,4,5-Trichlorophenoxyacetic acid	1	3.04E-06
2,4-Dichlorophenoxyacetic acid	10	3.04E-05
Acetaldehyde	-	-
Acrylonitrile	-	-
Arsenic	0.34	1.03E-06
Benzene	0.024	7.30E-08
Carbon tetrachloride	0.02	6.08E-08
Carbonyl sulphide	-	-
Chlordane, total	0.03	9.12E-08
Chlorobenzene	0.006	1.82E-08
Chloroethane	-	-
Chromium	1.4	4.26E-06
Cresols, total	13	3.95E-05
Dichloromethane	0.09	2.74E-07
Endrin	0.025	7.60E-08
Formaldehyde	-	-
Heptachlor	0.02	6.08E-08
Hexachlorobenzene	0.0068	2.07E-08
Hexachlorobutadiene	0.025	7.60E-08
Hexachloroethane	0.025	7.60E-08
Lead	0.18	5.47E-07
Methoxychlor	10	3.04E-05
Methyl ethyl ketone	58.4	1.78E-04
Methyl isobutyl ketone	-	-
Pentachlorophenol	0.005	1.52E-08
PFOA	1330	4.04E-03
PFOS/PFHxS	3900	1.19E-02
Pyridine	5	1.52E-05
Styrene	0.079	2.40E-07
Tetrachloroethylene	0.002	6.08E-09
Toluene	1.11	3.37E-06
Total reduced sulphur	-	-



Contaminant	Concentration in leachate	Concentration in stream confluence from surface water run-off
	mg/L-leachate	mg/L-water
Toxaphene	0.5	1.52E-06
Trichloroethylene	0.015	4.56E-08
Trichloromethane (chloroform)	0.025	7.60E-08
Vinyl chloride	0.084	2.55E-07
Xylene	1.13	3.44E-06

## **Appendix G: Per- and polyfluoroalkyl substances (PFAS)**

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## G1 Introduction

Per- and polyfluoroalkyl substances (PFAS) are a group of synthetic chemicals that have been in use since the 1940s. PFAS have been used in many consumer products to make them resistant to heat, stains, grease and/or water. Applications include keeping food from sticking to cookware, making sofas and carpets resistant to stains, making clothes and mattresses more waterproof, and making some food packaging resistant to grease absorption. They have also been used in some specialist firefighting foams. Because PFAS help reduce friction, they are also used in a variety of industries, including automotive, building and construction, and electronics.

A description of some of the most common types of PFAS compounds is listed in Appendix G Table 1.

**Appendix G Table 1: Common types of PFAS compounds (reproduced from Rumsby and Manning (2018))**

Family	Class	Examples	Uses
Perfluoroalkyl acids (PFAA)	Perfluoroalkyl carboxylic acids (PFCA)	PFOA	Surfactant
	Perfluoroalkyl sulfonic acids (PFSA)	PFOS	Surfactant
Polyfluorinated alkyl substance	Fluorotelemer sulfonic acids (FTSA)	8:2 Fluorotelemer sulfonic acid (8:2 FTS)	Surfactant/AFFF
	Fluorotelemer carboxylic acids (FTCA)	6:2 Fluorotelemer alcohol (6:2 FTC)	Immediate product
	Fluorotelemer alcohols	8:2 Fluorotelemer carboxylic acid (8:2 FTOH)	Use for manufacturing PFCA and PFSA
	Polyfluorinated alkyl phosphates (PAP)	Zonyl	Paper and food packaging materials

The most studied PFAS are PFOS (perfluorooctane sulphionate), PFOA (perfluorooctanoic acid) and PFHxS (perfluorohexane sulphonic acid).

When released into the environment, PFOS and PFOA are stable and resist typical environmental degradation processes.

Human epidemiological studies have found associations between PFOA exposure and high cholesterol, increased liver enzymes, decreased vaccination response, thyroid disorders, pregnancy-induced hypertension and preeclampsia, and cancer (testicular and kidney). Human health effects associated with PFOS exposure include high cholesterol and adverse reproductive and developmental effects.

In 2009, the Stockholm Convention on Persistent Organic Pollutants added PFOS to Annex B, restricting its production and use. PFOA has been recommended for listing and PFHxS is currently under review.

## G2 Toxicity values for PFAS

Food Standards Australia New Zealand (FSANZ) has undertaken a comprehensive review of toxicity information on PFOS, PFOA and PFHxS to support the derivation of health-based guidance values (FSANZ, undated). FSANZ noted that epidemiological studies have reported associations between PFOS exposure and several health effects, however that the findings are inconsistent between studies and the biological significance of a number of the observed effects is questionable.

FSANZ developed Tolerable Daily Intake (TDI) values based on extrapolation from toxicological studies in laboratory animals. The most sensitive health endpoints were determined to be:

- PFOS - decreased parental and offspring body weight gain in a reproductive toxicity study in rats.
- PFOA - for foetal toxicity in a developmental and reproductive study in mice.

These TDI values were used by the Australian Department of Health as the basis for setting drinking water and recreational water quality guideline values (Appendix G Table 2). Neither New Zealand nor the World Health Organization has set maximum acceptable values for PFAS. However the New Zealand Ministry of Health has accepted the Australian drinking water guideline values for PFOS and PFOA as interim guidance values in New Zealand.

**Appendix G Table 2: Toxicity reference values for PFOS, PFOA and PFHxS (Australian Government Department of Health (2017))**

Toxicity reference value	Units	PFOS/PFHxS*	PFOA
Tolerable daily intake	ng / (kg-bw · day)	20	160
Drinking water guidelines value	ng/L	70	560
Recreational water quality guideline value	ng/L	700	5600

\*Note: For PFHxS, FSANZ concluded that there was not enough toxicological and epidemiological information to justify establishing a tolerable daily intake. However, as a precaution, and for the purposes of site investigations, the PFOS tolerable daily intake should apply to PFHxS. In practice, this means that the level of PFHxS exposure should be added to the level of PFOS exposure; and this combined level be compared to the tolerable daily intake for PFOS.

### **G3 PFAS in landfills**

#### **G3.1 Overview**

PFAS is present in landfills because it is associated with a wide range of consumer products that are disposed in municipal solids waste, as described in Section G1 above.

Specifically in relation to fire-fighting foams, the EPA (NZ) has determined that wastes with a PFOS concentration of 50 ppm or greater must be treated as a Persistent Organic Pollutant and be managed in accordance with the Stockholm Convention and the Basel Convention. It is possible that waste handlers might in future be requested to consider acceptance of PFAS (including PFOS) wastes with a concentration less than 50ppm (for example contaminated soil).

Waste acceptance criteria for PFAS (including PFOS)-containing wastes have not been specifically developed for the Auckland Regional Landfill at this time. However, the acceptance of any such waste would be on the basis that it did not materially increase the mass PFAS in the landfill, given that municipal waste will be the main source of PFAS being placed at the landfill.

#### **G3.2 PFAS in leachate**

Gallen et al (2017) present the results of analysis of leachate from 27 Australian landfills for nine PFAS (including PFOA, PFOS and PFHxS). Higher mean concentrations of PFAS were measured in landfill accepting primarily construction and demolition waste compared to municipal solid waste landfills, and in operating landfills compared to older, closed landfills. The authors postulated that the higher levels of PFAS in leachate from younger landfills may be related to the time-lag between purchasing, using and disposing of PFAS-containing products with long lifetimes, such as textiles and carpets.

Mean concentrations and standard deviations of PFAS in landfill have been grouped by the operational status of the landfill (open or closed) and dominant waste type accepted (>50% municipal solid waste or greater than 50% constructions and demolition waste).

Approximately 40% (by weight) of waste going to landfill in the Auckland region is construction and demolition waste (Auckland Council, 2018). Therefore, the leachate data from Gallen *et al* (2017) considered to be most representative of the Auckland Regional Landfill is that for operating landfills accepting more than 50% municipal solid waste. We have selected the 95% upper confidence limit of this data (mean plus 2 x standard deviations) as the source concentration in leachate for this HHRA (Appendix G Table 3).

Leachate from Redvale Landfill has been analysed on one occasion and the results for the relevant groupings of PFAS are shown in the following table compared with the adopted values. This comparison suggests that the values adopted for the HHRA should be conservatively high.

**Appendix G Table 3: Source concentrations of selected PFAS in leachate**

PFAS compounds	Concentration in Redvale leachate	Source concentration in leachate
	ng/L	ng/L
PFOS	560	960
PFHxS	890	2940
PFOA	920	1330

### G3.3 PFAS in landfill gas

Compared to PFOS and PFAS, the fluorotelomer alcohols (FTOHs) have relatively low water solubility's and relatively high vapour pressure and are therefore more likely to volatilise into landfill gas.

We were not able to locate any direct measurements of PFAS in landfill gas and only three studies relating to the fate of PFASs in the atmosphere of landfills. Ahrens (2011) investigated the atmospheric concentrations of PFAS around a wastewater treatment plant and two landfills in Ontario, Canada. The sum of PFAS over the landfills was  $2.8 \times 10^3$  pg/m<sup>3</sup> to  $2.6 \times 10^4$  pg/m<sup>3</sup> and comprised more than 90% fluorotelomer alcohols (FTOHs) and perfluorobutanoate (PFBA).

Weinberg (2011) measured neutral PFAS in the air over two landfills in Germany. Concentrations were in the range 84 pg/m<sup>3</sup> to 706 pg/m<sup>3</sup> and were dominated by FTOH (average 82%). Trace levels of PFOS were recorded.

A more recent study by Tian (2018) investigated concentrations of PFAS in the air, dry deposition and plant leaves at two different landfills and one suburban reference site in Tianjin, China. The maximum concentrations of all PFAS in air above the two landfills was 9.5 ng/m<sup>3</sup>, 4.1 µg/g in dry deposition, and 48 µg/g lipid in leaves. The dominant fluorocarbon species were ultra-short chain perfluoroalkyl carboxylic acids (trifluoroacetic acid and perfluoropropionic acid), which accounted for 71%-94% of all fluorocarbons

The gas collected at the Auckland Regional Landfill will be combusted in a flare with a design residence time of at least 0.5 seconds at 750°C. While PFAS' are generally known for their resistance to degradation, there is likely to be a relatively high destruction efficiency in the flares and generators for the more volatile PFAS compounds likely to be present in landfill gas, e.g. FTOHs.

Given that the PFAS species in landfill gas predominantly comprise FTOHs, which are of less concern and do not have recognised toxicity factors, the very low concentrations expected in landfill gas and

the likely effectiveness of the flare and generators at destroying FTOHs, the possible contribution of PFAS in landfill gas emissions has not been considered further in the HHRA.

## G4 PFAS media concentrations

### G4.1 PFAS in groundwater for potable supply

As presented in Appendix G Table 4, concentrations of PFAS have been determined in groundwater at the closest groundwater take for possible potable supply (POE#4). The following table sets out the calculations of intake and hazard associated with drinking water from the bore. The calculations are based on a 13 kg child drinking 1 L water per day.

**Appendix G Table 4: Intake and hazard calculations for PFAS in drinking water**

PFAS Compounds	Conc in farm bore	Ingestion rate	Tolerable daily intake	Hazard index
	µg/L	ng/kg-day	ng/(kg bw-day)	Unitless
PFOA	2.16E-07	1.59E-05	160	9.96E-08
PFOS	1.99E-07	1.47E-05	20	7.34E-07
PFHxS	1.57E-07	1.16E-05	20	5.79E-07

### G4.2 PFAS in surface water

As presented in Appendix G Table 5, concentrations of PFAS have been determined in groundwater as it enters surface water, as appropriate, at each of the relevant points of exposure identified in Table 4.2, i.e:

- Stream confluence (360 m) Valley 1 and 2 stream (POE#1).
- Hōteō river - regional (2100 m) (POE#2 and POE#3).
- Waiteraire Stream (1000 m) (POE#6).

The RBCA modelling predicts that the highest concentrations of PFAS compounds in surface water as a result of groundwater, would be from shallow groundwater discharging to the Valley 1/Valley 2 stream confluence (POE#1).

**Appendix G Table 5: Predicted PFAS concentrations at POE#1**

Contaminant	Contribution from shallow groundwater	Contribution from surface water run-off	Concentration in surface water
	mg/L	mg/L	mg/L
PFOA	2.80E-09	3.40E-09	6.20E-09
PFHxS	2.71E-09	2.50E-09	5.21E-09
PFOS	1.70E-09	6.90E-09	8.60E-09

## G5 PFAS exposure pathways

### G5.1 Ingestion of eel (tuna) flesh

#### G5.1.1 PFAS concentration in eel flesh

Studies carried out in New Zealand (see Rumsby A. and Manning T., 2018)) and Europe (Effrosyni Zafeiraki et al, 2019) indicate that significant bio-accumulation of PFAS can occur in eels. Bio-concentration factors (BCF) were developed from the NZ data (the ratio of the concentration of contaminant measured in flesh to the concentration of contaminant measured in water) for eels for the PFAS compounds by Rumsby and Manning. BCFs for eels varied significantly with values of up to 727 recorded for PFHxS and PFOS in shortfin eels. BCFs for eels were much higher than other fish species.

Review of the European eel flesh data and PFAS water quality information (RIWA, 2017) indicates the following BCF factors (Appendix G Table 6 below).

**Appendix G Table 6: BCF for eels for selected PFAS based on European data**

Locale	BCF for PFOA	BCF for PFOS	BCF for PFHxS
Lobith	-	3700	-
Nieuwegein	-	423	174
Nieuwersluis	-	5288	-
Andijk	-	5796	-

Eel flesh concentrations were predicted using the most conservative (highest) BCF from the NZ and European data, as shown in Appendix G Table 7.

**Appendix G Table 7: BCF for eels adopted for this HHRA**

PFAS Compounds	BCF	Source
PFOA	5796	RIWA, 2017
PFHxS	727	Developed from PDP, 2018
PFOS	69	Developed from PDP, 2018

The predicted eel flesh concentrations are presented in Appendix G Table 8.

**Appendix G Table 8: Predicted eel flesh concentrations**

PFAS Compounds	Predicted eel flesh concentration (ng/kg)
PFOA	0.428
PFHxS	3.78
PFOS	49.9

## G5.1.2 PFAS intake from eel consumption

The amount of PFAS that could be ingested from eating eels can be calculated using standard dietary intake calculations and NES Soil intake factors. The parameter most difficult to assess is eel consumption. The selection of representative intake factors is discussed in Section 8.5.

Daily intake values for PFAS from consumption of eel flesh are presented in Appendix G Table 9 for both adults and children. The daily intake of PFAS from eel flesh are all well below the FSANZ TDI values.

**Appendix G Table 9: Eel flesh intake calculations and TDI**

PFAS Compounds	Adult	Child	Tolerable Daily Intake
	ng/(kg bw-day)	ng/(kg bw-day)	ng/(kg bw-day)
PFOS	0.060	0.050	20
PFHxS	0.0045	0.00038	20
PFOS + PFHxS	0.065	0.054	20
PFOA	5.1E-03	4.3E-04	160

## G5.2 Ingestion of chicken eggs

### G5.2.1 PFAS concentration in chicken eggs

PFAS is known to accumulate in birds, including chickens, and can be transferred to chicken eggs. The primary exposure pathway for domestic chickens considered in this HHRA is the use of water from the farm bore as drinking water. Other potential exposure pathways could include ingestion of soil or plants contaminated with PFAS through aerial deposition. There is expected to be negligible aerial deposition of PFAS and therefore the potential for chickens to ingest soil via these pathways is considered negligible.

A study commissioned by the Australian Department of Defence as part of their investigation into PFAS contamination at the Williamstown RAAF Base (Aecom, 2017) investigated the relationship between PFAS concentrations (PFOS, PFHxS and PFOA) in chicken eggs and in their drinking water. The study found that 100% of the PFOS ingested by a chicken was transferred to the egg. The percentage transfer was lower for other PFHxS and PFOA (see Appendix G Table 10).

The concentration of PFAS in chicken eggs can be calculated as follows:

$$C_{egg}(ng/g) = \frac{C_{drinking\ water} \times Intake_{drinking\ water} \times \%transfer}{Laying\ rate \times Mass_{egg}}$$

Where:

$C_{drinking\ water}$  is the concentration of PFAS in the chicken's drinking water (ng/L)

$Intake_{drinking\ water}$  is the amount of water the chicken drinks each day (L/day)

% transfer is the percentage of PFAS transferred to the egg

$Mass_{egg}$  is the weight of edible portion of egg (g)

Laying rate is the average number of eggs a chicken lays per day (eggs/day)



**Appendix G Table 10: Chicken egg intake parameters**

Parameter	Value	Source
Drinking water intake of the chicken (L/day)	0.208	Aecom, 2017
Percentage transferred to egg	PFOS: 100% PFHxS: 69% PFOA: 46%	Aecom, 2017
Edible weight of egg (g/egg)	50	NES Soil
Egg laying rate (eggs per day)	0.9	Aecom, 2017

Calculations for the concentration of PFAS in chicken eggs are summarised Appendix G Table 11.

**Appendix G Table 11: Egg concentration calculations**

PFAS Compounds	Concentration in drinking water	Percentage transferred to egg	Mass in egg	Concentration in egg
	mg/L	%	ng	ng/g
PFOS	1.99E-10	100	0.0046	9.20E-05
PFHxS	1.57E-10	69	0.00265	5.01E-05
PFOA	2.16E-10	46	0.0023	4.59E-05

### G5.2.2 PFAS intake from egg consumption

The amount of PFAS that could be ingested from eating home-grown eggs can be calculated using standard dietary intake calculations. The NES Soil includes consideration of chicken eggs as a potential exposure pathway. The dietary intake parameters adopted in the NES Soil have been used in this HHRA as summarised in Appendix G Table 12.

**Appendix G Table 12: Dietary intake parameters**

Parameter	Value	Source
Average egg consumption (adult) (eggs per year)	200	NES Soil
Average egg consumption (adult) (g/day)	27.4	NES Soil
Average egg consumption (child) (g/day)	8	NES Soil

Calculations for the daily intake of PFAS in chicken eggs are summarised in Appendix G Table 13. Some studies have shown that households that keep their own chickens can eat up to twice as many eggs as the normal consumer. Daily intake values are presented for both adults and children at average and 'double the average' egg consumption rates.

**Appendix G Table 13: Chicken egg dietary intake calculations**

PFAS Compounds	Adult average intake	Adult double average intake	Child average intake	Child double average intake
	ng/(kg bw-day)	ng/(kg bw-day)	ng/(kg bw-day)	ng/(kg bw-day)
PFOS	3.60E-05	7.20E-05	5.66E-05	1.13E-04
PFHxS	1.96E-05	3.92E-05	3.08E-05	6.16E-05
PFOA	1.80E-05	3.60E-05	2.83E-05	5.65E-05

### G5.3 Concentration of PFAS in beef tissue

Cattle could ingest PFAS if it is present in groundwater from the farm bore or water from the stream confluence downstream of the landfill footprint used for stock watering. The potential for PFAS to accumulate in beef meet has been calculated. The resulting concentrations in beef meat can be compared to proposed trigger levels for further investigation set by FASANZ.

The concentration in beef is calculated using a three step process, as set out in Appendix O of Aecom (2017). The formulae for each of the calculation steps are set out below:

$$CDI_{livestock} = \frac{C_{DW} * IngRate_{dw} * CF}{BW}$$

$$C_{serum/plasma} = \frac{CDI_{livestock} * t_{1/2}}{0.693 * V_d}$$

$$C_{tissue} = TSR * C_{serumORplasma} * CF$$

Where:

$CDI_{livestock}$  is the chronic daily intake for the livestock (mg/kg-day)

$C_{dw}$  is the concentration of PFAS in the stock drinking water (ng/L)

$IngRate_{dw}$  is the amount of water the animal drinks each day (L/day)

$BW$  is the average livestock body weight at slaughter (kg)

$C_{serum/plasma}$  is the steady state concentration of PFAS in beef steer blood serum

$t_{1/2}$  is the COPC specific beef steer serum elimination half life (days)

$V_d$  is the apparent volume of distribution in beef steers (L/kg)

$C_{tissue}$  is the livestock tissue concentration of PFAS (mg/kg)

$TSR$  is the COPC specific empirical tissue/serum ratio

$CF$  is the serum density conversion factor (L/kg)

The relevant parameters are set out in the following tables:

**Appendix G Table 14: Livestock dietary intake parameters**

Parameter	Value	Source
Livestock water intake per day (L/day) ( $IngRate_{dw}$ )	100	DPI (2014) – taken from Aecom, 2017
Apparent volume of distribution for beef steers (L/kg) ( $V_d$ )	0.21	Aecom (2017)
Serum density conversion factor (L/kg) (CF)	0.97	ToxConsult (2016) - taken from Aecom, 2017
Livestock body weight (kg)	540	API (2004) and CCME (1999) - taken from AECOM, 2017

**Appendix G Table 15: Serum half-life and tissue/serum ratio from (Aecom, 2017)**

PFAS Compounds	Beef steer tissue/serum ratio (TSR)	Beef steer serum half life ( $t_{1/2}$ ) (days)
PFOA	0.1	0.8
PFHxS	0.05	1
PFOS	0.1	0.0114

The resulting concentrations of PFAS in beef tissue are set out below.

**Appendix G Table 16: Concentration of PFAS in beef tissue (use of farm bore for stock watering)**

PFAS compounds	Concentration in drinking water (farm bore)	Chronic daily intake by livestock	Steady state concentration in blood serum	Concentration in tissue
	mg/L	mg/kg-day	mg/L	µg/kg
PFOA	2.16E-10	4.00E-11	2.20E-10	2.13E-08
PFHxS	1.57E-10	2.91E-11	2.00E-09	9.69E-08
PFOS	1.99E-10	3.69E-11	2.89E-08	2.80E-06

**Appendix G Table 17: Concentration of PFAS in beef tissue (use of stream water for stock watering)**

PFAS compounds	Concentration in drinking water (stream)	Chronic daily intake by livestock	Steady state concentration in blood serum	Concentration in tissue
	mg/L	mg/kg-day	mg/L	µg/kg
PFOA	6.20E-09	1.15E-09	6.31E-09	6.12E-07
PFHxS	5.21E-09	9.64E-10	6.62E-08	3.21E-06
PFOS	8.60E-09	1.59E-09	1.25E-06	1.21E-04

## G5.4 Concentration of PFAS in cow's milk

Dairy cows could also ingest PFAS if it is present in groundwater from the farm bore or stream used for stock watering. The potential for PFAS to accumulate in dairy cows and be transferred into milk has been calculated. The resulting concentrations in milk can be compared to proposed trigger levels for further investigation set by FASANZ.

The concentration in cow's milk is calculated using a three step process, as set out in Appendix O of Aecom (2017). These calculation steps are similar to those set out in the previous sub-section for beef. However, the factors (e.g. serum half-life, etc) for dairy cows differ to those for beef steers. The dairy cow livestock body weight and water intake factors are the same as for beef steers.

$$CDI_{mother} = CDI_{ing}$$

$$C_{serum/plasma} = \frac{CDI_{mother} * t_{1/2}}{0.693 * V_d}$$

$$C_{milk} = MSR * C_{serum/plasma}$$

Where:

$CDI_{mother}$  is the chronic daily intake for the dairy cow (mg/kg-day)

$t_{1/2}$  is the COPC specific dairy cow serum elimination half life (days)

$V_d$  is the apparent volume of distribution in dairy cows (0.26 L/kg)

MSR is the COPC specific empirical tissue/serum ratio

The relevant parameters are set out in the following tables.

**Appendix G Table 18: Serum half-life and tissue/serum ratio (from Aecom, 2017)**

PFAS Compounds	Milk/serum ratio (MSR)	Dairy cow serum half life ( $t_{1/2}$ ) (days)
PFOA	8.00E-03	1.3
PFHxS	1.00E-02	10
PFOS	1.90E-02	50

The resulting concentrations of PFAS in dairy milk are set out below.

**Appendix G Table 19: Milk concentration calculations (use of farm bore for stock watering)**

PFAS compounds	Concentration in drinking water (farm bore)	Chronic daily intake by dairy cow	Steady state concentration in blood serum	Concentration in milk	Concentration in milk
	mg/L	mg/kg/day	mg/L	mg/L	µg/kg
PFOA	2.16E-10	4.00E-11	2.89E-10	2.31E-12	2.24E-09
PFHxS	1.57E-10	2.91E-11	1.61E-09	1.61E-11	1.57E-08
PFOS	1.99E-10	3.69E-11	1.02E-08	1.94E-10	1.89E-07

**Appendix G Table 20: Milk concentration calculations (use of stream water for stock watering)**

PFAS compounds	Concentration in drinking water (farm bore)	Chronic daily intake by dairy cow	Steady state concentration in blood serum	Concentration in milk	Concentration in milk
	mg/L	mg/kg/day	mg/L	mg/L	µg/kg
PFOA	6.20E-09	1.15E-09	8.28E-09	6.62E-11	6.43E-08
PFHxS	5.21E-09	9.64E-10	5.35E-08	5.35E-10	5.19E-07
PFOS	8.60E-09	1.59E-09	4.42E-07	8.40E-09	8.15E-06

## **G6      References for PFAS**

- Aecom, 2017, Off-site Human Health Risk Assessment. RAAF Base Williamtown. Stage 2B Environmental Investigation.
- Effrosyni Zafeiraki et al, 2019, Occurrence of perfluoroalkyl substances (PFASs) in a large number of wild and farmed aquatic animals collected in the Netherlands, Chemosphere 232 (2019) 415e423.
- Ministry for Primary Industries (2018). New Zealand Food Safety. 2016 New Zealand Total Diet Study.
- NIWA. (2011). Contaminants in kai – Te Arawa rohe. Part 1: Data Report. NIWA Report HAM2011-021.
- Rijncommissie Waterleidingsbedrijven (RIWA), 2017, Vereniging van Rivierwaterbedrijven, Jaarrapport 2017, De Rijn.
- Rumsby A. and Manning T. (2018, Impact of per and poly and fluoralkyl substances on ecosystems. Prepared by Pattle Delamore Partners for Ministry for the Environment.
- US EPA (1989) Risk Assessment Guidance for SuperFund Volume 1 Human Health Evaluation Manual (Part A).

## **Appendix H: Impact of leachate evaporator on mercury emissions**

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## H1 Introduction

Specific consideration has been given to mercury because of its complex chemistry and the possible implications for mercury emissions to air from the use of a low temperature leachate evaporator, similar to the system currently used at Redvale Landfill.

## H2 Forms of mercury in leachate and landfill gas

Mercury can enter the landfill in the form of disposed products such as batteries, fluorescent bulbs, thermostats and other switches, and in thermometers. Elemental mercury could be released when the casings of these electronic goods break down in the landfill. Mercury can occur as one of three oxidation states (0, +1, and +2) and as different compounds based on the conditions and constituents of the environmental medium. Different mercury compounds have different volatility and solubility, for example elemental mercury can be present in the gaseous phase, while mercury (+2) will tend to form strong bonds with ligands such as chloride, sulphide and organic matter and remain sequestered within the waste mass. Mercury is likely to exist primarily as inorganic forms in landfill leachate (rather than the more toxic organic forms), for example as mercury oxide which is slightly soluble.

In landfill gas, the gaseous form of mercury is principally elemental mercury (Hg(0)). Gaseous mercury can also exist in landfill gas in the form of methylated mercury species, which are more toxic than elemental mercury. Studies have shown that average concentrations of monomethyl and dimethyl mercury in landfill gas are typically less than 2% of the total mercury. In the flare and generators, methylated mercury compounds will decompose to elemental mercury, reducing their toxicity.

## H3 Fate of mercury in the leachate evaporator

### H3.1 Leachate evaporator characteristics

The leachate evaporator is described in Section 4.6 of the Air Quality Assessment. The purpose of the leachate evaporator is to evaporate water from the collected leachate, to reduce the volume of liquid that needs to be managed. The leachate evaporator will operate at temperatures below 90°C, so only relatively volatile contaminants (mainly organic compounds) will be released with the water vapour. This water vapour is then passed to the flare, where the organic contaminants will be destroyed. However, any mercury that is volatilised from the leachate will pass through the flare and be emitted as elemental mercury.

### H3.2 Rate of mercury evaporation and release into flare

The leachate evaporator is designed to reduce the volume of leachate by 60 m<sup>3</sup>/day. Ignoring the presence of other contaminants in the leachate (i.e. assuming the leachate comprises only water and mercury), the rate of mercury evaporation can be estimated from Raoult's law and the Ideal Gas law, as follows:

$$\text{Loss rate of mercury} = \frac{\text{Vap pressure}_{\text{mercury}} \times \text{Vap mole fraction}_{\text{mercury}} \times \text{Total vap loss} \times \text{MW}_{\text{mercury}}}{\text{Ideal gas law constant (R)} \times \text{Temperature}}$$

**Vapour pressure<sub>mercury</sub>** is the partial pressure of mercury above the leachate at 90°C. This can be calculated from the vapour pressure of pure mercury at 90°C multiplied by the mole fraction of mercury in the leachate. The vapour pressure of mercury at 90°C is 0.02167 kPa using the mercury vapour pressure correlation developed by Huber M, Laesecke A, and Friend D (2006) (see Figure Appendix H.1).



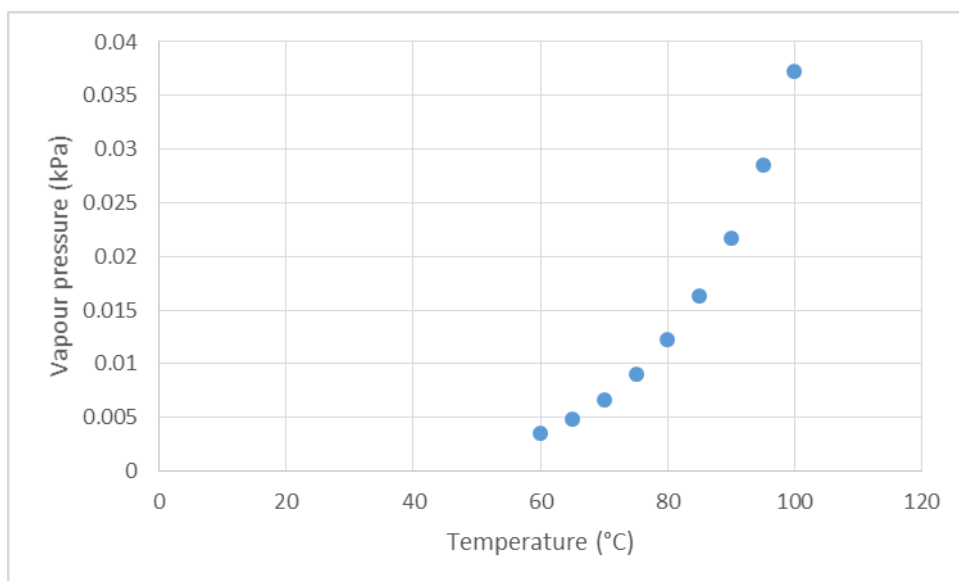


Figure Appendix H.1: Vapour pressure of mercury at varying temperatures (based on Huber M, Laesecke A, and Friend D (2006))

The maximum measured concentration of mercury in leachate at Redvale Landfill is 0.0065 g/m<sup>3</sup>. The **vapour mole fraction**<sub>mercury</sub> can be calculated by determining the moles of mercury and water in 1kg of leachate. The molar ratio of mercury and water in the vapour will be the same as the molar ratio in the leachate.

The total vapour losses are in units of L/s and, because the rate of water evaporation is known, this can be determined iteratively. The calculation of mass emissions of water (known to be 60 kg/day) and mercury, using the equation set out above, are summarised in Appendix H Table 1.

**Appendix H Table 1: Calculation of losses of mercury and water vapour from leachate into the flare (via the leachate evaporator)**

Component	Vapour pressure	Molecular weight	Mass fraction	Moles per kg of leachate	Vapour mole fraction	Loss rate	Loss rate
	kPa	g/mol	g/kg	mol/kg	Unitless	g/s	kg/day
Water	70.182	18.015	0.9999935	0.055509	0.99999942	0.6944	60
Mercury	0.02167	200.59	0.0000065	3.24E-08	5.84E-07	1.39E-09	1.20E-07

\* Assuming evaporator is sized to evaporate 60L/day water

If all of the mercury in the 60 m<sup>3</sup>/day leachate treated through the evaporator were volatilised, this would equate to 0.39 g/day of mercury being released into the flare along with the water vapour. However, calculations based on partial vapour pressure show that approximately 0.00012g/day of mercury, which is 0.03% of the mercury in the leachate, is likely to be lost to the flare and the balance will remain in the leachate and be recirculated back into the landfill (or disposed off-site).

### H3.3 Impact of the leachate evaporator on mercury emissions to air

The upper bound estimate of landfill gas that will be collected at the Auckland Regional Landfill is 10,089 m<sup>3</sup>/hr. There has been only one analysis of mercury in landfill gas at Redvale Landfill. This

was carried out in 2002 and the method used had a very high detection limit ( $2775 \mu\text{g}/\text{m}^3$ ) relative to the concentration that would typically be expected in landfill gas (of the order of  $1 \mu\text{g}/\text{m}^3$ ).

In the absence of site specific data, we have relied on published data on concentrations of mercury in landfill gas. The maximum concentration of mercury in landfill gas recorded at UK landfills is  $1.33 \mu\text{g}/\text{m}^3$  with an average concentration of  $0.58 \mu\text{g}/\text{m}^3$  and the US EPA AP 42 default emission factor for mercury is  $2.435 \mu\text{g}/\text{m}^3$ .

Adopting a mercury concentration of  $2.435 \mu\text{g}/\text{m}^3$  in landfill gas, this would equate to a mass emission rate of  $0.59 \text{ g/day}$  mercury from the flares/generators at the Auckland Regional landfill based on the maximum amount of landfill gas likely to be collected and combusted. The additional  $0.00012 \text{ g/day}$  mercury that may be released from the leachate evaporator into the flare is negligible ( $0.02\%$ ) compared to this larger amount released to air from combustion of landfill gas. Therefore, the leachate evaporator is not considered to have any material impact on mercury emissions from the Auckland Regional Landfill.

## **Appendix I: Detailed exposure concentration calculations**

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## I1 Accumulation of contaminants in soil from airborne deposition

### I1.1 Introduction

The concentration of a contaminant in soil as a result of aerial deposition is a function of the rate of deposition, the rate at which contaminants are lost from the soil and the duration over which the deposition occurs.

Mercury is a special case. The vast majority of mercury exiting the stack is expected to be elemental mercury that does not readily deposit, but is vertically diffused to the free atmosphere. While deposition rates can be theoretically calculated, there are no uptake values from soil to plants or vapour to plants cited in the US EPA database on the basis that mercury is assumed not to deposit onto soils. Therefore it has been assumed there is no deposition of mercury onto soils.

### I1.2 Deposition term

Airborne contaminants emitted from the flares/generators or fugitive landfill gas emissions from the working face are dominated by volatile organic compounds. These compounds are assumed to be present entirely in the vapour phase. Deposition of vapour phase contaminants can occur through wet and dry deposition processes. Dry deposition is driven by gravity and diffusion while wet deposition is controlled by the rate and intensity of precipitation (rainfall).

The wet and dry deposition fluxes have been calculated using the CALPUFF dispersion model. Wet deposition of vapours requires the gas phase contaminants to dissolve in falling raindrops. The tendency of contaminants to be captured by wet deposition processes is related to solubility. Therefore, wet deposition fluxes have only been calculated for relatively soluble contaminants.

The equation given in the HHRAP for the deposition term ( $D_s$ ) (expressed in mg COPC/m<sup>2</sup> per year) is as follows (HHRAP Equation 5-11):

$$D_s = \left[ \frac{100 \cdot Q}{Z_s \cdot BD} \right] \cdot [F_v \cdot (D_{ydv} + D_{yww}) + (D_{ydp} + D_{ywp}) \cdot (1 - F_v)]$$

As the COPC are assumed to be entirely in the vapour phase ( $F_v = 1$ ), the particulate deposition terms ( $D_{ydp}$  and  $D_{ywp}$ ) fall away.

The outputs of the CALPUFF dispersion modelling have already been adjusted to account for the concentrations of each COPC in the emissions, and therefore  $Q$  (the COPC emission rate in g/s) also falls away. Therefore, the equation can be simplified as follows:

$$D_s \text{ (mg/kg soil - year)} = \frac{(D_{dry} + D_{wet})}{Z_s \times BD}$$

Where

$D_{dry}$  is the annual dry deposition flux of the contaminant (mg COPC /m<sup>2</sup>-year)

$D_{wet}$  is the annual wet deposition flux of the contaminant (mg COPC/m<sup>2</sup>-year)

$Z_s$  is the soil mixing depth (20 cm for tilled soil and 2 cm for untilled soil)

$BD$  is the soil bulk density (1500 kg/m<sup>3</sup> soil)

### 11.3 Soil losses

The HHRAP describes five mechanisms by which contaminants can be lost from soils, as shown in Appendix I Table 1.

**Appendix I Table 1: Soil loss constants**

Soil loss constant	Description	Included
k <sub>sg</sub>	Soil losses due to biotic and abiotic degradation (year) <sup>-1</sup>	Yes
K <sub>se</sub>	Soil losses due to soil erosion (year) <sup>-1</sup>	No
k <sub>sre</sub>	Soil losses due to surface runoff (year) <sup>-1</sup>	No
k <sub>sl</sub>	Soil losses due to leaching (year) <sup>-1</sup>	No
k <sub>sv</sub>	Soil losses due to volatilisation (year) <sup>-1</sup>	Yes

As the COPC for aerial deposition are predominantly volatile or semi-volatile organic compounds, they will be subject to volatilisation and to biotic and abiotic degradation processes. The other soil loss mechanisms have been ignored. On this basis, the total soil loss factor (k<sub>v</sub>) (HHRAP Equation 5-2A) can be simplified to:

$$k_s(\text{year})^{-1} = k_{sg} + k_v$$

The USEPA provides estimates of the soil loss constant due to biotic and abiotic degradation processes (k<sub>sg</sub>) for many of the priority contaminants for this HHRA. Where no published k<sub>sg</sub> value could be found, it has been assumed that there are no losses by this mechanism.

The HHRAP includes a recommended equation for calculating k<sub>sv</sub> based on a number of generic factors and COPC-specific factors provided in the HHRAP companion database (HHRAP Equation 5-7A).

$$k_{sv} = \left( \frac{3.1536 \times 10^7 \cdot H}{Z_s \cdot Kd_s \cdot R \cdot T_a \cdot BD} \right) \cdot \left( \frac{D_a}{Z_s} \right) \cdot \left[ 1 - \left( \frac{BD}{\rho_{soil}} \right) - \Phi_{sw} \right]$$

Where

Z<sub>s</sub> is the soil mixing depth (20 cm for tilled soil and 2 cm for untilled soil)

K<sub>d<sub>s</sub></sub> is the COPC-specific soil/water partition coefficient (ml/g)

R is the universal gas constant (atm·m<sup>3</sup>/mol·K)

T<sub>a</sub> is the ambient air temperature = 298.1 K

BD is the soil bulk density = 1.5 g soil/cm<sup>3</sup> soil)

H is the COPC-specific Henry's law constant (atm·m<sup>3</sup>/mol)

D<sub>a</sub> is the COPC-specific diffusivity in air (cm<sup>2</sup>/s)

ρ<sub>soil</sub> is the solids particle density = 2.7 g/cm<sup>3</sup>

Φ<sub>sw</sub> is the soil volumetric water content (ml/cm<sup>3</sup> soil) = 0.2 ml/cm<sup>3</sup>

#### 11.4 Cumulative soil concentration

For threshold compounds, the concentration in soil used in the intake equations is based on the worst year of exposure, which is assumed to be Year 30 (i.e. cumulative 30 years of deposition). This is calculated using the following equation (HRRAP Equation 5-1E):

$$C_{s_{tD}} = \frac{Ds \cdot [1 - \exp(-ks \cdot tD)]}{ks}$$

Where:

$C_{s_{tD}}$  is the soil concentration in year 30 (mg COPC/kg soil)

$Ds$  is the yearly deposition rate of contaminants by wet and dry deposition (mg COPC/kg soil-year)

$ks$  is the soil loss constant due to all processes (year)<sup>-1</sup>

$tD$  is the time period over which deposition occurs (30 years)

For non-threshold compounds, the average concentration in soil over 30 years of exposure used in the intake equations. This is calculated using the following equation (HRRAP Equation 5-1C):

$$Cs = \frac{Ds}{ks \cdot (tD - T_1)} \cdot \left[ \left( tD + \frac{\exp(-ks \cdot tD)}{ks} \right) - \left( T_1 + \frac{\exp(-ks \cdot T_1)}{ks} \right) \right]$$

Where:

$T_1$  is the first year of deposition (Year 1)

## 12 Uptake into home-grown produce from aerial contaminants

### 12.1 Uptake mechanisms into home-grown produce

The HHRAP describes three mechanisms for the possible uptake of COPC into home-grown produce, as illustrated in the following figure.

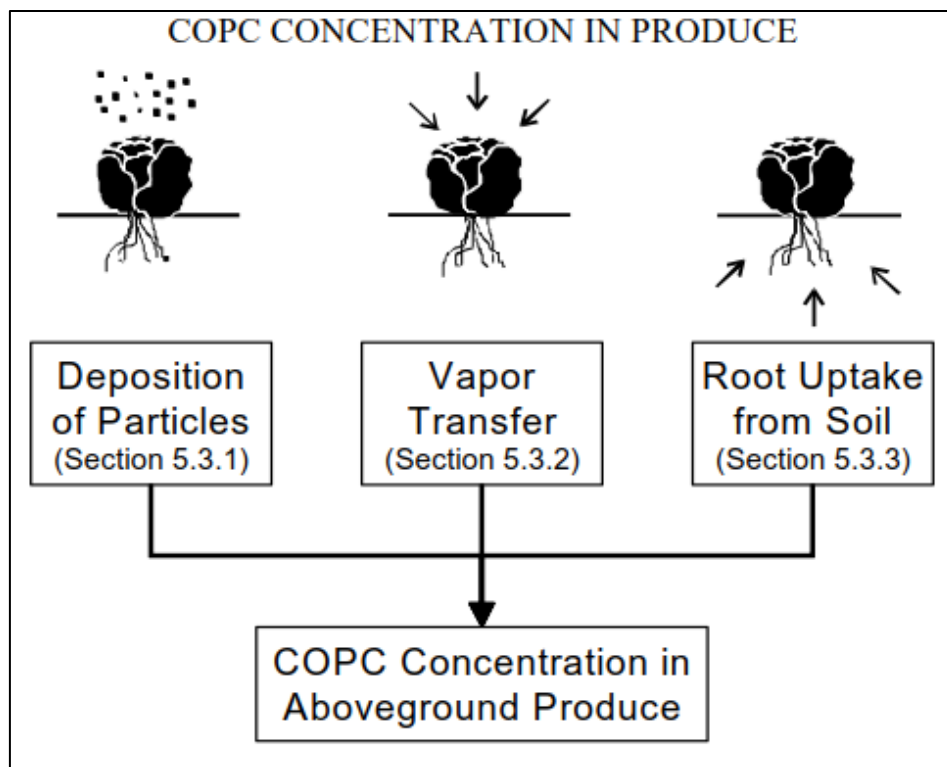


Figure Appendix I.1: Uptake of COPC into produce (reproduced from US EPA, 2005. p5-23)

The two mechanisms relevant to air borne contaminants from the flares/generators and fugitive landfill gas emissions (which are all in the vapour phase) are vapour transfer and root uptake from soil. The HHRAP considers home-grown produce in three categories, as shown in Appendix I Table 2. Relevant bioaccumulation factors are provided on the basis of these produce types (e.g. root vegetables (carrots) are not differentiated from tubers (potatoes)).

**Appendix I Table 2: HHRAP home-grown produce categories and uptake mechanisms**

Home-grown produce type	Recommended uptake pathways
Exposed above-ground produce	Sum of contamination occurring through all three of these mechanisms
Protected above-ground produce (e.g. peas, corn, melons)	Root uptake is considered to be the primary mechanism
Below ground produce (roots and tubers)	Root uptake (assume that root or tuber is protected from contact with contaminants in the vapour phase)

These categories do not fully align with the home-grown produce categories used in the NES Soil, for which there are recommended ingestion factors for a New Zealand rural residential setting.

Therefore it has been necessary to make some conservative assumptions to align with the New Zealand dietary information and recommendations in the NES Soil (Appendix I Table 3).

**Appendix I Table 3: HHRAP home-grown produce categories and uptake mechanisms**

Home-grown produce types used in HHRA (from NES Soil)	Equivalent HHRAP category	Comment
Aboveground produce	Exposed and protected above-ground produce	Will overstate COPC uptake in protected aboveground produce due to air-plant transfer
Root produce	Below ground produce	COPC-specific uptake factors in the HHRAP do not differentiate between roots and tubers.
Tuber produce		

## 12.2 Air-to-plant transfer

The method recommended in the HHRAP for estimating the concentration of contaminants in aboveground produce via air-to-plant transfer ( $P_v$  expressed in units of  $\mu\text{g COPC/g DW}$ ) is described by the following equation (HHRAP Equation 5-18):

$$P_v = Q \cdot F_v \cdot \frac{C_{yv} \cdot B_{v_{ag}} \cdot VG_{ag}}{\rho_a}$$

The COPC are assumed to be entirely in the vapour phase ( $F_v = 1$ ) and the outputs of the CALPUFF dispersion modelling have already been adjusted to account for the concentrations of each COPC in the emissions. Therefore  $Q$  (the COPC emission rate in  $\text{g/s}$ ) and  $F_v$  fall away to be replaced by  $C_{yv}$  expressed as the annual average concentration in air ( $\mu\text{g/m}^3$ ).

Where the remaining terms are:

$B_{v_{ag}}$  is the COPC-specific air-to-plant biotransfer factor ( $[\text{mg COPC/g DW plant}]/[\text{mg COPC/g air}]$ )

$VG_{ag}$  is COPC-specific empirical correction factor

$\rho_a$  is the density of air ( $\text{g/m}^3$ )

$VG_{ag}$  for aboveground produce is intended to account for the potential to over-estimate the transfer of lipophilic COPCs to the inner portion of bulk produce. The HHRAP recommends a value of 0.01 for COPCs with a  $\log K_{ow}$  great than 4, and 1.0 for COPCs with a  $\log K_{ow}$  less than 4.

## 12.3 Produce concentration due to root uptake

The HHRAP recommends the following equation to estimate the uptake of contaminants into exposed and protected aboveground produce (HHRAP Equation 5-20A):

$$P_r = C_s \cdot B_r$$



Where:

Cs is the average soil concentration over the exposure duration (mg COPC/kg soil)

Br is the COPC-specific plant-soil bioconcentration factor for produce

The following equation is recommended to be used for belowground produce (HHRAP Equation 5-20B):

$$Pr = \frac{Cs \cdot RCF \cdot VG_{rootveg}}{Kd_s \cdot 1 \text{ kg/L}}$$

Where:

RCF is the COPC-specific root concentration factor

VG<sub>rootveg</sub> is the empirical correction factor for belowground produce

Kd<sub>s</sub> is the soil/water partition coefficient (L/kg)

As for aboveground produce, the HHRAP recommends a value of 0.01 for COPCs with a logKow great than 4, and 1.0 for COPCs with a logKow less than 4

## 12.4 Home-grown produce ingestion factors

The home-grown produce ingestion factors used in the HHRAP are based on the rural residential scenario in the NES Soil, reproduced in Appendix I Table 4.

**Appendix I Table 4: Home-grown produce ingestion factors (NES Soil)**

Exposure factor	Units	Recommended values for rural residential exposure scenario
Produce ingestion rate	Child kg/day (DW)	0.0105
	Adult kg/day (DW)	0.0322
Proportion of above-ground produce	dimensionless	0.3
Proportion of root (not tuber) produce	dimensionless	0.1
Proportion of tuber produce	dimensionless	0.6
Percentage of home grown produce	percent	25
Soil ingested with homegrown produce (assuming 100% homegrown produce)	Child (mg/day)	38
	Adult (mg/day)	8

### 13      **Entrainment of contaminants in roof collected drinking water**

The average daily deposition rate of contaminants onto a roof area can be calculated using the following equation:

$$D_{dw} (\mu g/day) = \frac{(D_{dry} + D_{wet}) \times 3600 \times 24}{Area}$$

Where:

$D_{dry}$  is the average dry deposition flux of the contaminant ( $\mu g$  COPC/ $m^2$ -second)

$D_{wet}$  is the average wet deposition flux of the contaminant ( $\mu g$  COPC/ $m^2$ -second)

Area is the roof area ( $m^2$ )

It has been assumed that the entire mass of deposited material is entrained in the roof water, i.e. that there are no volatilisation losses from the roof. However, the losses of volatile contaminants from the rainwater storage tank ( $k_{wv}$ ) can be estimated from published data on the COPC-specific half-life in water due to volatilisation,  $t_{1/2}$  (days), using the following equation:

$$k_{wv}(\text{day})^{-1} = \frac{\ln(2)}{t_{1/2}}$$

The resulting annual average concentration in drinking water can be calculated using a first order decay equation as follows:

$$C_{dw} (\mu g/m^3) = \frac{D_{dw}/\text{Rainfall volume} \times (1 - \exp(-k_{wv} \times 365))}{k_{wv}}$$

Where:

Rainfall volume is the annual rainfall (1.5 m/year) x roof area (300  $m^2$ ). This has been conservatively multiplied by a factor of 0.5 to account for losses of water due to overflows and evaporation

### 14      **Uptake of arsenic into watercress**

Leachate contains arsenic at low concentrations. Therefore arsenic could be present in surface water as a result of leachate seepage and migration in shallow groundwater and/or seepage into the landfill's stormwater system.

Arsenic is known to bioaccumulate in watercress. Robinson et al (2003)<sup>12</sup> studied the uptake of arsenic into watercress in the Waikato River as well as conducting greenhouse trials with watercress grown in beakers containing arsenic. The study found that, at equilibrium, the arsenic concentrations in the greenhouse samples were approximately fivefold less than in plants taken from the Waikato River, even though both were in arsenic solutions of similar concentrations. It was postulated that this may be due to the watercress in the experiment being free floating, whereas in the river it is rooted to the sediment layer, which may contain higher levels of arsenic. In order to obtain a conservative result, an arsenic-to-water cress BCF of 2000 L/kg has been used in this HHRA

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<sup>12</sup> Robinson B, Duwig C, Bolan N, Kannathasan M, Saravanan A. (2003). Uptake of arsenic by New Zealand watercress (*Lepidium sativum*). The Science of the Total Environment 301 (2003) 67–73.

(based on the ratio of arsenic in water and water cress samples in the Waikato River, Appendix I Table 5).

The concentration of arsenic in water cress is calculated using the BCF and a conservative value for the concentration of arsenic in surface water from both shallow groundwater ( $7.70 \times 10^{-7}$  mg/L) and stormwater ( $1.03 \times 10^{-6}$  mg/L).

**Appendix I Table 5: Concentration of arsenic in water cress**

Contaminant	Concentration in stream water	Bioconcentration Factor	Concentration in water cress
	mg/L	L/kg	mg/kg FW
Arsenic	1.80E-06	2000	3.61E-03

## **Appendix J: Detailed risk and hazard calculations**

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## J1 Inhalation

**Appendix J Table 1: Calculations of incremental lifetime cancer risk from inhalation of genotoxic carcinogens.**

Contaminant	Maximum air concentration	Inhalation unit risk	Incremental lifetime cancer risk
	$\mu\text{g}/\text{m}^3$	$(\mu\text{g}/\text{m}^3)^{-1}$	Unitless
1,2-dibromoethane	4.81E-04	7.10E-05	3.42E-08
1,2-dichloroethane	6.27E-07	2.00E-05	1.25E-11
1,2-dichloropropane	2.07E-05	1.00E-05	2.07E-10
Acetaldehyde	4.68E-03	9.00E-07	4.21E-09
Acrylonitrile	3.56E-04	2.00E-05	7.12E-09
Benzene	1.01E-03	6.00E-06	6.03E-09
Dichloromethane	7.86E-03	1.00E-06	7.86E-09
Trichloroethylene	1.03E-03	4.30E-07	4.42E-10
Vinyl chloride	1.61E-03	1.00E-06	1.61E-09
Total cumulative incremental lifetime cancer risk			6.17E-08 (0.0617 per million)
Acceptable risk level			1.0E-05 (10 per million)

**Table note:** Contaminants that are not present in this media, or have concentrations indistinguishable from zero, are not presented in this table.

**Appendix J Table 2: Calculation of Hazard Index from inhalation of threshold compounds.**

Contaminant	Maximum air concentration	Tolerable concentration (TC)	Hazard Quotient
	$\mu\text{g}/\text{m}^3$	$\mu\text{g}/\text{m}^3$	Unitless
Carbon tetrachloride	3.01E-03	6.1	4.94E-04
Chlorobenzene	7.55E-04	1000	7.55E-07
Dichloromethane	7.86E-03	450	1.75E-05
Formaldehyde	2.86E-04	9	3.17E-05
Methyl ethyl ketone	5.86E-03	5000	1.17E-06
Methyl isobutyl ketone	2.72E-03	3000	9.06E-07
Styrene	1.57E-03	0.26	6.04E-03
1,1,2,2-tetrachloroethane	2.07E-05	3.4	6.08E-06
Tetrachloroethylene	3.66E-03	250	1.47E-05
Toluene	2.83E-02	260	1.09E-04
Total reduced sulphur	6.53E-02	20	3.26E-03
Trichloromethane (chloroform)	1.30E-02	140	9.30E-05
Xylene	1.50E-02	870	1.73E-05
Hazard Index			0.0101
Hazard threshold			1.0

Table note: Contaminants that are not present in landfill gas, or there is no available toxicity factor for evaluation are not shown in this table.



## J2 Drinking water

### J2.1 Roof supplied drinking water

Appendix J Table 3: Calculation of incremental lifetime cancer risk for ingestion of genotoxic carcinogens in roof collected drinking water.

Parameters	
Body weight (kg)	58.6
Time related ratio (unitless)	0.38
Ingestion rate L/day	1.8
Half annual rainfall volume (m <sup>3</sup> )	225
Roof area (m <sup>2</sup> )	300

\*Age adjusted

\*Based on 1500 mm/year on 300 m<sup>2</sup> roof area

Contaminant	Wet deposition	Dry deposition	Total deposition	Dw	Half life	Ks	Cw without loss	Cw with loss	Cw	Daily intake	Ingestion slope factor	Incremental lifetime cancer risk
	µg /m <sup>2</sup> /s	µg /m <sup>2</sup> /s	µg /m <sup>2</sup> /s	µg /day	days	1/day	µg/m <sup>3</sup>	µg/m <sup>3</sup>	µg/m <sup>3</sup>	µg /kg-day	(µg /kg-day) <sup>-1</sup>	Unitless
1,2-dibromoethane	X	1.18E-06	1.18E-06	3.06E+01	1.60E+01	4.33E-02	4.96E+01	3.14E+00	3.14E+00	3.70E-05	7.50E-04	2.77E-08
1,2-dichloroethane	X	2.23E-09	2.23E-09	5.77E-02	4.00E+00	1.73E-01	9.36E-02	1.48E-03	1.48E-03	1.74E-08	1.00E-05	1.74E-13
1,2-dichloropropane	X	7.35E-08	7.35E-08	1.90E+00	ND	0.00E+00	3.09E+00	3.09E+00	3.09E+00	3.64E-05	3.60E-05	1.31E-09
Acrylonitrile	6.23E-08	1.27E-06	1.33E-06	3.44E+01	4.00E+00	1.73E-01	5.59E+01	8.83E-01	8.83E-01	1.04E-05	1.00E-03	1.04E-08
Benzene	X	3.57E-06	3.57E-06	9.26E+01	1.15E+01	6.03E-02	1.50E+02	6.83E+00	6.83E+00	8.04E-05	3.50E-05	2.82E-09
Dichloromethane	X	2.80E-05	2.80E-05	7.25E+02	4.00E+00	1.73E-01	1.18E+03	1.86E+01	1.86E+01	2.19E-04	1.40E-05	3.07E-09
Trichloroethylene	X	3.66E-06	3.66E-06	9.48E+01	4.60E+00	1.51E-01	1.54E+02	2.80E+00	2.80E+00	3.29E-05	5.90E-06	1.94E-10
Vinyl chloride	X	5.25E-06	5.25E-06	1.36E+02	3.00E+00	2.31E-01	2.21E+02	2.62E+00	2.62E+00	3.08E-05	1.00E-03	3.08E-08
Total cumulative incremental lifetime cancer risk												7.63E-08 (0.076 per million)
Acceptable risk level												1.0E-05 (10 per million)

Table note: Contaminants that are not present in landfill gas or there is no toxicity factor for assessment (e.g. oral toxicity factor for acetaldehyde), are not presented in this table.

**Table key:**

ND No half life data available – assumed no losses by volatilisation.

X Assumed no deposition via wet deposition processes due to insolubility.



Appendix J Table 4: Calculation of Hazard Index for ingestion of threshold compounds in roof collected drinking water.

Parameters	
Body weight - child (kg)	13
Ingestion rate (L/day)	1
Half annual rainfall volume (m <sup>3</sup> )	225
Roof area (m <sup>2</sup> )	300

\*Based on 1500 mm/year on 300 m<sup>2</sup> roof area

Contaminant	Wet deposition	Dry deposition	Total deposition	Dw	Half life	Ks	Cw without loss	Cw with loss	Cw	Daily intake	Tolerable daily intake	Hazard Quotient
	µg /m <sup>2</sup> /s	µg /m <sup>2</sup> /s	µg /m <sup>2</sup> /s	µg /day	days	1/day	µg/m <sup>3</sup>	µg/m <sup>3</sup>	µg/m <sup>3</sup>	µg /kg-day	µg /kg-day	Unitless
Carbon tetrachloride	X	1.07E-05	1.07E-05	2.78E+02	5.00E+00	1.39E-01	4.50E+02	8.90E+00	8.90E+00	6.56E-04	1.40E+00	4.69E-04
Chlorobenzene	X	2.68E-06	2.68E-06	6.96E+01	ND	0.00E+00	1.13E+02	1.13E+02	1.13E+02	8.33E-03	8.57E+01	9.71E-05
Dichloromethane	X	2.80E-05	2.80E-05	7.25E+02	4.00E+00	1.73E-01	1.18E+03	1.86E+01	1.86E+01	1.37E-03	6.00E+00	2.28E-04
Formaldehyde	5.00E-08	1.05E-06	1.10E-06	2.86E+01	ND	0.00E+00	4.64E+01	4.64E+01	4.64E+01	3.42E-03	1.50E+02	2.28E-05
Hexachlorobutadiene	X	7.35E-08	7.35E-08	1.90E+00	ND	0.00E+00	3.09E+00	3.09E+00	3.09E+00	2.28E-04	2.00E-01	1.14E-03
Methyl ethyl ketone	1.02E-06	2.08E-05	2.18E-05	5.66E+02	ND	0.00E+00	9.18E+02	9.18E+02	9.18E+02	6.77E-02	6.00E+02	1.13E-04
Styrene	X	5.58E-06	5.58E-06	1.45E+02	1.30E+01	5.33E-02	2.35E+02	1.21E+01	1.21E+01	8.90E-04	7.70E+00	1.16E-04
1,1,2,2-tetrachloroethane	X	7.35E-08	7.35E-08	1.90E+00	3.50E+00	1.98E-01	3.09E+00	4.27E-02	4.27E-02	3.15E-06	1.20E+00	2.63E-06
Tetrachloroethylene	X	1.30E-05	1.30E-05	3.38E+02	5.00E+00	1.39E-01	5.48E+02	1.08E+01	1.08E+01	7.98E-04	1.40E+01	5.70E-05
Toluene	X	1.01E-04	1.01E-04	2.61E+03	4.00E+00	1.73E-01	4.24E+03	6.70E+01	6.70E+01	4.94E-03	2.23E+02	2.22E-05
Trichloroethylene	X	3.66E-06	3.66E-06	9.48E+01	4.60E+00	1.51E-01	1.54E+02	2.80E+00	2.80E+00	2.06E-04	1.46E+00	1.41E-04
Trichloromethane (chloroform)	X	4.63E-05	4.63E-05	1.20E+03	4.40E+00	1.58E-01	1.95E+03	3.38E+01	3.38E+01	2.50E-03	1.50E+01	1.66E-04
Xylene	X	5.34E-05	5.34E-05	1.38E+03	4.10E+00	1.69E-01	2.24E+03	3.64E+01	3.64E+01	2.68E-03	2.50E+02	1.07E-05
Hazard Index												2.59E-03 (0.00259)
Hazard threshold												1.0

Table note: Contaminants that are not present in landfill gas or there is no toxicity factor for assessment (e.g. oral toxicity factors for chloroethane, methyl isobutyl ketone and total reduced sulphur), are not presented in this table.

Table key:

ND No half life data available – assumed no losses by volatilisation.

X Assumed no deposition via wet deposition processes due to insolubility.

## J2.2 Farm bore supplying potable drinking water

**Appendix J Table 5: Calculation of incremental lifetime risk for ingestion of genotoxic carcinogens in potable drinking water from farm bore.**

Parameters	
Body weight (kg)	58.6
Time related ratio (unitless)	0.38
Ingestion rate L/day	1.8

Contaminant	Concentration in farm bore	Concentration in farm bore	Daily intake	Ingestion slope factor	Incremental lifetime cancer risk
	mg/L	µg/L	µg/kg-day	(µg/kg-day) <sup>-1</sup>	Unitless
Arsenic	6.90E-08	6.90E-05	8.13E-07	0.00116	9.43E-10
Acceptable risk level					1.0E-05 (10 per million)

**Table note:** Contaminants that have modelled concentrations that are so low they are indistinguishable from zero are not presented in this table.

**Appendix J Table 6: Calculation of Hazard Index for ingestion of threshold compounds in potable drinking water from farm bore.**

Parameters	
Body weight - child (kg)	13
Ingestion rate (L/day)	1

Contaminant	Concentration in farm bore	Concentration in farm bore	Daily intake	Tolerable daily intake	Hazard Quotient
	mg/L	µg/L	µg/kg-day	µg/kg-day	Unitless
Chlordane, total	6.30E-09	6.30E-06	4.65E-07	0.5	9.29E-07
Chromium	1.50E-07	1.50E-04	1.11E-05	3	3.69E-06
Endrin	5.20E-09	5.20E-06	3.84E-07	0.2	1.92E-06
Hexachlorobutadiene	5.20E-09	5.20E-06	3.84E-07	0.2	1.92E-06
Lead	3.80E-08	3.80E-05	2.80E-06	1.9	1.48E-06
PFOA	2.16E-10	2.16E-07	1.59E-08	0.16	9.96E-08
PFOS/PFHxS	3.56E-10	3.56E-07	2.63E-08	0.02	1.31E-06
Hazard Index					1.13E-05 (0.0000113)
Hazard threshold					1.0

**Table note:** Contaminants that are not present leachate, or have modelled concentrations that are so low they are indistinguishable from zero, are not presented in this table.



**J3 Ingestion of home-grown produce with COPC from use of bore water for irrigation and aerial deposition of contamination**

**Appendix J Table 7: Calculation of incremental lifetime cancer risk for ingestion of genotoxic carcinogens in home-grown produce.**

Parameters	Soil (mg/day)	Above ground (g/day)	Roots (g/day)	Tubers (g/day)
Ingestion rate	8	2.85	0.43	4.11
Ingestion rate (kg/day)	0.000008	0.00285	0.00043	0.00411
Time related ratio (unitless)	0.38			
Body weight (kg) (time weighted)	58.6			
Duration of release (years)	30			
T1 (year)	1			
Density of air (kg/m <sup>3</sup> )	1.2754			
Soil bulk density (kg/m <sup>3</sup> )	1500			
Mixing depth (m)	0.2			
Watering rate L/min	10			
Water period (min/day)	60			
Watering days (days/year)	100			
Garden size (m <sup>2</sup> )	45			

Contaminants	Kow (log)	Root veg factor	Maximum air concentration		Wet flux deposition	Dry flux deposition	Wet flux deposition	Dry flux deposition	Farm bore concentration	Mass (garden)	Ks	Ds (aerial deposition)	Ds (watered garden)	Cs without losses	Cs (losses) calc term 1	Cs (losses) calc term 2	Cs with losses
		VG rootveg	µg/m <sup>3</sup>	mg/kg air	µg/m <sup>2</sup> /s	µg/m <sup>2</sup> /s	mg/m <sup>2</sup> /year	mg/m <sup>2</sup> /year	mg/L	mg	1/year	mg/kg soil-year	mg/kg-soil year	mg/kg soil	unitless	unitless	mg/kg soil
1,2-dibromoethane	2	1.00	4.81E-04	3.77E-07	X	1.18E-06	X	3.72E-02	*	*	5.98E+00	1.24E-04	*	3.72E-03	7.15E-07	28.9996	2.07E-05
1,2-dichloroethane	1.5	1.00	6.27E-07	4.91E-10	X	2.23E-09	X	7.02E-05	*	*	1.53E+03	2.34E-07	*	7.02E-06	5.27E-12	29.0000	1.53E-10
1,2-dichloropropane	2	1.00	2.07E-05	1.62E-08	X	7.35E-08	X	2.32E-03	*	*	1.28E+03	7.73E-06	*	2.32E-04	2.08E-10	29.0000	6.04E-09
Acrylonitrile	0.25	1.00	3.56E-04	2.79E-07	6.23E-08	1.27E-06	1.97E-03	3.99E-02	-	-	3.31E+03	1.40E-04	*	4.19E-03	1.45E-09	29.0000	4.22E-08
Arsenic	0.68	1.00	-	-	-	-	-	-	6.90E-08	4.14E-03	ND	-	3.07E-06	9.20E-05	ND	ND	ND
Benzene	2.1	1.00	1.01E-03	7.88E-07	X	3.57E-06	X	1.13E-01	*	*	2.17E+03	3.76E-04	*	1.13E-02	5.96E-09	29.0000	1.73E-07
Dichloromethane	1.3	1.00	7.86E-03	6.17E-06	X	2.80E-05	X	8.81E-01	*	*	4.96E+02	2.94E-03	*	8.81E-02	2.04E-07	29.0000	5.93E-06
Trichloroethylene	2.4	1.00	1.03E-03	8.07E-07	X	3.66E-06	X	1.15E-01	*	*	1.26E+03	3.84E-04	*	1.15E-02	1.05E-08	2.90E+01	3.05E-07
Vinyl chloride	1.4	1.00	1.61E-03	1.26E-06	X	5.25E-06	X	1.66E-01	*	*	4.06E+04	5.52E-04	*	1.66E-02	4.68E-10	29.0000	1.36E-08

Appendix J Table 7 continued.

Contaminants				Soil-to-plant transfer			Plant concentration		Air-to-plant transfer		Daily intake						Incremental lifetime cancer risk
	Cs	RCF	Ksd w	Above ground	Roots	Tubers	Above ground	Roots and tubers	Above ground	Above ground	Soil ingestion	Above ground	Roots	Tubers	Daily Intake	Ingestion slope factor	
	mg/kg soil	(µg/g)/(µg/µL)	L/kg	(mg/kg DW plant)/(mg/kg soil)	(mg/kg DW plant)/(mg/kg soil)	(mg/kg DW plant)/(mg/kg soil)	mg/kg	mg/kg	(mg/kg DW plant)/(mg/kg air)	mg/kg	µg/kg bw-day	µg/kg bw-day	µg/kg bw-day	µg/kg bw-day	µg/kg bw-day	(µg/kg bw-day) <sup>-1</sup>	
1,2-dibromoethane	2.07E-05	8.05	6.94	2.7	94.8	94.8	5.59E-05	2.28E-03	1.02E-02	3.85E-09	1.09E-09	1.04E-06	6.41E-06	6.13E-05	6.88E-05	7.50E-04	5.16E-08
1,2-dichloroethane	1.53E-10	9.63	2.85	5.26	275	275	8.04E-10	1.42E-07	2.26E-03	1.11E-12	8.00E-15	1.50E-11	4.00E-10	3.82E-09	4.24E-09	1.00E-05	4.24E-14
1,2-dichloropropane	6.04E-09	8.05	3.53	2.7	92.6	92.6	1.63E-08	1.28E-06	2.69E-03	4.36E-11	3.16E-13	3.05E-10	3.59E-09	3.43E-08	3.82E-08	3.60E-05	1.38E-12
Acrylonitrile	4.22E-08	6.67	0.13	8.38	379	379	3.53E-07	8.20E-04	1.00E-03	2.79E-10	2.21E-12	6.60E-09	2.31E-06	2.21E-05	2.44E-05	1.00E-03	2.44E-08
Arsenic	9.20E-05	ND	29	0.011	0.011	0.01	1.01E-06	0.00E+00	-	-	4.82E-09	1.89E-08	0.00E+00	0.00E+00	2.37E-08	1.16E-03	2.75E-11
Benzene	1.73E-07	9.62	4.6275	2.37	80.1	80.1	4.09E-07	2.88E-05	1.72E-03	1.36E-09	9.05E-12	7.66E-09	8.10E-08	7.74E-07	8.63E-07	3.50E-05	3.02E-11
Dichloromethane	5.93E-06	8.64	0.4	6.86	359	359	4.07E-05	4.60E-02	6.16E-04	3.80E-09	3.10E-10	7.59E-07	1.29E-04	1.24E-03	1.37E-03	1.40E-05	1.91E-08
Trichloroethylene	3.05E-07	16.4	7.07	1.59	49.6	49.6	4.86E-07	3.51E-05	2.01E-03	1.62E-09	1.60E-11	9.09E-09	9.89E-08	9.45E-07	1.05E-06	5.90E-06	6.22E-12
Vinyl chloride	1.36E-08	9.09	1.15	6.01	246	246	8.16E-08	2.64E-05	6.41E-05	8.10E-11	7.11E-13	1.52E-09	7.43E-08	7.10E-07	7.86E-07	1.00E-03	7.86E-10
Total cumulative incremental lifetime cancer risk																9.59E-08 (0.0959 per million)	
Acceptable risk level																1.0E-05 (10 per million)	

Table note: Contaminants where there is no toxicity factor for assessment (e.g. oral toxicity factor for acetaldehyde), are not presented in this table.

Table key:

- Indicates pathway not relevant for this compound (e.g. arsenic is not relevant to aerial deposition pathway but is relevant to water from the farm bore).
- X Not modelled in wet deposition due to insolubility.
- \* Indicates contaminants that have modelled concentrations in groundwater at the farm bore so low they are indistinguishable from zero

Appendix J Table 8: Calculation of the Hazard Index for ingestion of threshold compounds in home-grown produce.

Parameters	Soil (mg/day)	Above ground (g/day)	Roots (g/day)	Tubers (g/day)											
Ingestion rate	8	2.85	0.43	4.11											
Ingestion rate (kg/day)	0.000008	0.00285	0.00043	0.00411											
Body weight (kg) (time weighted)	13														
Duration of release (years)	30														
T1 (year)	1														
Density of air (kg/m <sup>3</sup> )	1.2754														
Soil bulk density (kg/m <sup>3</sup> )	1500														
Mixing depth (m)	0.2														
Mixing depth – non tilled (m)	0.02														
Watering rate (L/min)	10														
Watering duration (min/day)	60														
Watering period (day/year)	100														
Garden size (m <sup>2</sup> )	45														
Contaminants	Kow (log)	Root veg factor	Maximum air concentration		Wet flux deposition	Dry flux deposition	Wet flux deposition	Dry flux deposition	Farm bore concentration	Mass (watered garden)	Ks	Ds (aerial deposition)	Ds (watered garden)	Cs without losses	Cs with losses
		VG rootveg	µg/m <sup>3</sup>	mg/kg air	µg/m <sup>2</sup> /s	µg/m <sup>2</sup> /s	mg/m <sup>2</sup> /year	mg/m <sup>2</sup> /year	mg/L	mg	1/year	mg/kg soil-year	mg/kg soil-year	mg/kg soil	unitless
Carbon tetrachloride	2.8	1.00	3.01E-03	2.36E-06	X	1.07E-05	X	3.38E-01	*	*	3.51E+03	1.13E-03	*	3.38E-02	3.20E-07
Chlordane, total	5.5	0.01	-	-	-	-	-	-	6.30E-09	3.78E-04	1.80E-01	-	2.80E-08	8.40E-07	1.55E-07
Chlorobenzene	2.8	1.00	7.55E-04	5.92E-07	X	2.68E-06	X	8.46E-02	*	*	3.24E+02	2.82E-04	*	8.46E-03	8.70E-07
Chromium	0.23	1.00	-	-	-	-	-	-	1.50E-07	9.00E-03	ND	-	6.67E-07	2.00E-05	ND
Dichloromethane	1.3	1.00	7.86E-03	6.17E-06	X	2.80E-05	X	8.81E-01	*	*	4.96E+02	2.94E-03	*	8.81E-02	5.93E-06
Endrin	4.6	0.01	-	-	-	-	-	-	5.20E-09	3.12E-04	ND	-	2.31E-08	6.93E-07	ND
Formaldehyde	0.35	1.00	2.86E-04	2.24E-07	5.00E-08	1.05E-06	1.58E-03	3.32E-02	-	-	3.77E+01	1.16E-04	-	3.48E-03	3.08E-06
Hexachlorobutadiene	4.8	0.01	2.07E-05	1.62E-08	X	7.35E-08	X	2.32E-03	5.20E-09	3.12E-04	1.44E+00	7.73E-06	2.31E-08	2.32E-04	5.38E-06
Lead	0.73	1.00	-	-	-	-	-	-	3.80E-08	2.28E-03	1.13E+00	-	1.69E-07	5.07E-06	1.50E-07
Methyl ethyl ketone	0.29	1.00	5.86E-03	4.59E-06	1.02E-06	2.08E-05	3.23E-02	6.56E-01	*	*	4.43E+01	2.30E-03	*	6.89E-02	5.18E-05
PFOA	0	1.00	-	-	-	-	-	-	2.16E-10	1.30E-05	ND	-	9.60E-10	2.88E-08	ND
PFOS/PFHxS	0	1.00	-	-	-	-	-	-	3.56E-10	2.14E-05	ND	-	1.58E-09	4.75E-08	ND
Styrene	3	1.00	1.57E-03	1.23E-06	X	5.58E-06	X	1.76E-01	*	*	9.87E+00	5.87E-04	*	1.76E-02	5.95E-05
1,1,2,2-tetrachloroethane	2.4	1.00	2.07E-05	1.62E-08	X	7.35E-08	X	2.32E-03	*	*	7.25E+01	7.73E-06	*	2.32E-04	1.07E-07
Tetrachloroethylene	3.4	1.00	3.66E-03	2.87E-06	X	1.30E-05	X	4.11E-01	*	*	2.20E+03	1.37E-03	*	4.11E-02	6.23E-07
Toluene	2.7	1.00	2.83E-02	2.22E-05	X	1.01E-04	X	3.18E+00	*	*	8.50E+02	1.06E-02	*	3.18E-01	1.25E-05
Trichloroethylene	2.4	1.00	1.03E-03	8.07E-07	X	3.66E-06	X	1.15E-01	*	*	1.26E+03	3.84E-04	*	1.15E-02	3.05E-07
Trichloromethane (chloroform)	2	1.00	1.30E-02	1.02E-05	X	4.63E-05	X	1.46E+00	*	*	2.53E+03	4.87E-03	*	1.46E-01	1.92E-06
Xylene	3.2	1.00	1.50E-02	1.18E-05	X	5.34E-05	X	1.68E+00	*	*	3.41E+02	5.61E-03	*	1.68E-01	1.65E-05

Appendix J Table 8 continued.

Contaminants	Cs	RCF	Ksd w	Soil-to-plant transfer			Plant concentration		Air-to-plant transfer		Daily intake						Hazard Quotient
				Above ground	Roots	Tubers	Above ground	Roots and tubers	Above ground	Above ground	Soil ingestion	Above ground	Roots	Tubers	Daily Intake	Tolerable daily intake	
	mg/kg soil	(µg/g)/(µg/µL)	L/kg	(mg/kg DW plant)/(mg/kg soil)	(mg/kg DW plant)/(mg/kg soil)	(mg/kg DW plant)/(mg/kg soil)	mg/kg	mg/kg	(mg/kg DW plant)/(mg/kg air)	mg/kg	µg/kg bw-day	µg/kg bw-day	µg/kg bw-day	µg/kg bw-day	µg/kg bw-day	µg/kg bw-day	
Carbon tetrachloride	3.20E-07	33.3	11.4	0.932	95.1	95.1	2.99E-07	8.90E-05	1.79E-03	4.23E-09	4.93E-11	5.64E-08	2.94E-06	2.81E-05	3.11E-05	1.4	2.22E-05
Chlordane, total	1.55E-07	ND	ND	0.0256	16.6	16.6	3.96E-09	2.57E-08	822	-	2.38E-11	7.38E-10	8.50E-10	8.12E-09	9.73E-09	0.5	1.95E-08
Chlorobenzene	8.70E-07	33.3	16.8	0.932	75.6	75.6	8.11E-07	1.30E-04	1.45E-02	8.59E-09	1.34E-10	1.53E-07	4.31E-06	4.12E-05	4.57E-05	85.7	5.33E-07
Chromium	2.00E-05	ND	ND	0.0324	0.0324	0.0324	6.48E-07	6.48E-07	ND	-	3.08E-09	1.21E-07	2.14E-08	2.05E-07	3.50E-07	3	1.17E-07
Dichloromethane	5.93E-06	8.64	0.75	6.86	359	359	4.07E-05	2.45E-02	6.16E-04	3.80E-09	9.12E-10	7.58E-06	8.11E-04	7.75E-03	8.57E-03	6	1.43E-03
Endrin	6.93E-07	ND	ND	0.085	32.4	32.4	5.89E-08	2.25E-07	5.91E+02	-	1.07E-10	1.10E-08	7.43E-09	7.10E-08	8.95E-08	0.2	4.48E-07
Formaldehyde	3.08E-06	6.74	0.17	8.38	305	305	2.58E-05	3.72E-02	3.92E-01	8.78E-08	4.73E-10	4.82E-06	1.23E-03	1.18E-02	1.30E-02	150	8.66E-05
Hexachlorobutadiene	5.38E-06	1150	568.3	0.065	0.152	0.152	3.49E-07	1.65E-08	8.93E-01	1.45E-08	8.27E-10	6.78E-08	5.47E-10	5.23E-09	7.44E-08	0.2	3.72E-07
Lead	1.50E-07	ND	ND	0.019	0.015	0.005	2.85E-09	2.25E-09	ND	-	2.31E-11	5.31E-10	7.44E-11	7.11E-10	1.34E-09	1.9	7.05E-10
Methyl ethyl ketone	5.18E-05	6.7	0.14	8.38	23.1	23.1	4.34E-04	5.72E-02	2.03E-03	9.32E-09	7.97E-09	8.09E-05	1.89E-03	1.81E-02	2.01E-02	600	3.35E-05
PFOA	2.88E-08	ND	ND	0.48	0.48	0.48	1.38E-08	1.38E-08	ND	-	4.43E-12	2.58E-09	4.57E-10	4.37E-09	7.41E-09	0.16	4.63E-08
PFOS/PFHxS	4.75E-08	ND	ND	0.057	0.057	0.057	2.71E-09	2.71E-09	ND	-	7.30E-12	5.04E-10	8.95E-11	8.55E-10	1.46E-09	0.02	7.28E-08
Styrene	5.95E-05	47.4	68.4	0.714	0.395	0.395	4.25E-05	1.63E-05	3.24E-02	3.99E-08	9.15E-09	7.92E-06	5.38E-07	5.15E-06	1.36E-05	7.7	1.77E-06
1,1,2,2-tetrachloroethane	1.07E-07	50	22.54	1.59	86.2	86.2	1.69E-07	2.04E-05	5.91E-02	9.58E-10	1.64E-11	3.18E-08	6.74E-07	6.44E-06	7.15E-06	1.2	5.96E-06
Tetrachloroethylene	6.23E-07	96.4	19.88	0.42	311	311	2.62E-07	9.40E-04	1.30E-02	3.74E-08	9.59E-11	5.57E-08	3.11E-05	2.97E-04	3.28E-04	14	2.34E-05
Toluene	1.25E-05	27.9	10.5	1.07	77.4	77.4	1.33E-05	2.56E-03	6.36E-03	1.41E-07	1.92E-09	2.51E-06	8.48E-05	8.11E-04	8.98E-04	223	4.03E-06
Trichloroethylene	3.05E-07	16.4	7.07	1.59	49.6	49.6	4.86E-07	3.51E-05	2.01E-03	1.62E-09	4.70E-11	9.08E-08	1.16E-06	1.11E-05	1.24E-05	1.46	8.47E-06
Trichloromethane (chloroform)	1.92E-06	8.05	3.94	2.7	101	101	5.19E-06	3.97E-04	2.04E-03	2.08E-08	2.96E-10	9.72E-07	1.31E-05	1.26E-04	1.40E-04	15	9.31E-06
Xylene	1.65E-05	56.6	18.08	0.548	83.5	83.5	9.03E-06	4.31E-03	1.96E-02	2.31E-07	2.53E-09	1.73E-06	1.42E-04	1.36E-03	1.51E-03	250	6.02E-06
Hazard Index																	1.63E-03 (0.00163)
Hazard threshold																	1.0

Table note: Contaminants where there is no toxicity factor for assessment (e.g. oral toxicity factors for chloroethane, methyl isobutyl ketone and total reduced sulphur), are not presented in this table

Table key:

- X Not modelled in wet deposition due to insolubility
- Indicates pathway not relevant for this compound (e.g. chromium is not relevant to aerial deposition pathway but is relevant to water from the farm bore).
- ND No physical parameter data available.
- \* Indicates contaminants that have modelled concentrations in groundwater at the farm bore so low they are indistinguishable from zero

## J4 Ingestion of soil

Appendix J Table 9: Calculations for lifetime incremental cancer risk from direct ingestion of genotoxic carcinogens in soil.

Parameters				
Age adjusted ingestion (mg/day)	30			
Age adjusted (kg/day)	0.00003			
Time ratio (unitless)	0.38			
Duration of release (years)	30			
Chemicals of concern	Concentration in soil <sup>1</sup> (from Table 10)	Daily intake	Ingestion slope factor	Incremental lifetime cancer risk
	mg/kg soil	ug/kg bw-day	(ug/kg bw-day) <sup>-1</sup>	Unitless
1,2-dibromoethane	2.07E-04	1.82E-07	7.50E-04	1.36E-10
1,2-dichloroethane	1.53E-09	1.34E-12	1.00E-05	1.34E-17
1,2-dichloropropane	6.04E-08	5.30E-11	3.60E-05	1.91E-15
Acrylonitrile	4.22E-07	3.70E-10	1.00E-03	3.70E-13
Arsenic	9.20E-06	8.07E-09	1.16E-03	9.36E-12
Benzene	1.73E-06	1.51E-09	3.50E-05	5.30E-14
Dichloromethane	5.93E-05	5.20E-08	1.40E-05	7.28E-13
Trichloroethylene	3.05E-06	2.68E-09	5.90E-06	1.58E-14
Vinyl chloride	1.36E-07	1.19E-10	1.00E-03	1.19E-13
Total cumulative incremental lifetime cancer risk				<b>1.47E-10 (0.000147 per million)</b>
Acceptable risk level				<b>1.0E-5 (10 per million)</b>

Table note:

1. The concentration in soil is taken from Appendix J Table 7, but is ten times higher as the soil is assumed to be untilled (i.e. mixing depth in vegetable garden is 20 cm and for untilled soil is 2 cm)



Appendix J Table 10: Calculation of Hazard Index for ingestion of threshold compounds in soil.

Parameters				
Ingestion rate	2			
Ingestion rate (kg/day)	0.000002			
Child ingestion (mg/day)	50			
Child ingestion (kg/day)	0.00005			
Chemicals of concern	Concentration in soil <sup>1</sup> (from Table 10)	Daily intake	Tolerable daily intake	Hazard index
	mg/kg soil	µg/kg bw-day	µg/kg bw-day	Unitless
Carbon tetrachloride	3.20E-06	1.23E-08	1.4	8.80E-09
Chlordane, total	1.55E-07	5.95E-10	0.5	1.19E-09
Chlorobenzene	8.70E-06	3.35E-08	85.7	3.91E-10
Chromium	2.00E-05	7.69E-08	3	2.56E-08
Dichloromethane	5.93E-05	2.28E-07	6	3.80E-08
Endrin	6.93E-07	2.67E-09	0.2	1.33E-08
Formaldehyde	3.08E-05	1.18E-07	150	7.89E-10
Hexachlorobutadiene	5.36E-05	2.06E-07	0.2	1.03E-06
Lead	1.50E-07	5.76E-10	1.9	3.03E-10
Methyl ethyl ketone	5.18E-04	1.99E-06	600	3.32E-09
PFOA	2.88E-08	1.11E-10	0.16	6.92E-10
PFOS/PFHxS	4.75E-08	1.83E-10	0.02	9.13E-09
Styrene	5.95E-04	2.29E-06	7.7	2.97E-07
1,1,2,2-tetrachloroethane	1.07E-06	4.10E-09	1.2	3.41E-09
Tetrachloroethylene	6.23E-06	2.40E-08	14	1.71E-09
Toluene	1.25E-04	4.79E-07	223	2.15E-09
Trichloroethylene	3.05E-06	1.17E-08	1.46	8.05E-09
Trichloromethane (chloroform)	1.92E-05	7.40E-08	15	4.93E-09
Xylene	1.65E-04	6.34E-07	250	2.53E-09

Table note:

1. The concentration in soil is taken from Appendix J Table 8, but is ten times higher as the soil is assumed to be untilled (i.e. mixing depth in vegetable garden is 20 cm and for untilled soil is 2 cm)



