Appendix G: Per- and polyfluoroalkyl substances (PFAS)

G1 Scope of this Appendix

This Appendix G to the HHRA sets out relevant background information and detailed supporting calculations for the assessment of potential health effects from exposure to per- and polyfluoroalkyl substances (PFAS) from the landfill. This Appendix does not include the overall findings and conclusions with respect to the hazard from exposure to PFAS.

The overall findings in relation to exposure to PFAS compounds are set out in the following tables in the HHRA report:

- Table 9.4: Residential receptor using bore water for potable supply and to irrigate the vegetable garden.
- Table 9.5: Wild food collector eating eels and incidental ingestion of surface water.
- Table 9.7: Comparison of estimated concentrations in food with PFAS trigger points (for public consumers).

Revised versions of these tables, incorporating updated values for exposure to PFAS compounds, have been provided in S.92 response tranche 4 (question 99). The revisions do not alter the conclusions of the original HHRA.

G2 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.

There are over 4000 individual PFAS substances. 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 (PFAS)	Fluorotelomer sulfonic acids (FTSA)	8:2 Fluorotelomer sulfonic acid (8:2 FTS)	Surfactant/AFFF
	Fluorotelomer carboxylic acids (FTCA)	6:2 Fluorotelomer alcohol (6:2 FTC)	Immediate product
	Fluorotelomer alcohols	8:2 Fluorotelomer 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.

G3 Toxicity of PFAS

G3.1 Published toxicity values

Most research on the toxicity of PFAS has been carried out on long chain compounds, particularly:

- Perfluoroalkane sulfonic acids (PFSAs) with carbon chain lengths of 6 and higher (C6+), including perfluorohexane sulfonic acid (PFHxS) and perfluorooctane sulfonic acid (PFOS).
- Perfluorocarboxylic acids (PFCAs) with carbon chain lengths of 8 and higher (C8+), including perfluorooctanoic acid (PFOA).

Short chain PFAS compounds (e.g. PFSAs of ≤C5 and PFCAs ≤C7) have similar toxic endpoint to long chain PFAS compounds, but at higher doses, i.e. they have a lower toxicity, and they have a shorter half-life. However, short chain PFAS compounds are still persistent in the environment.

There is ongoing research into the toxicity of a number of other PFAS compounds (for example the US EPA, as shown in Appendix G Table 3). However, given the emerging nature of the science around these compounds, there are only a small number of peer-reviewed toxicity factors that are formally recognised.

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 different health effects, however 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 valuesError! Reference source not found.. 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.

In addition to the FSANZ values, we have reviewed toxicity values published by:

- US EPA.

- Oak Ridge National Laboratory provisional peer-reviewed toxicity values (PPRTV).
- European Food Safety Authority.

This has identified provisional values from the US EPA for GenX chemicals and PFBS (Appendix G Table 2).

Appendix G Table 2: Peer reviewed chronic oral toxicity values

Acronym	Name	Chronic oral toxicity values (µg/kg-day)		
		US EPA Oral Reference Dose	FSANZ Tolerable Daily Intake[c]	European EFSA Tolerable Weekly Intake [h]
Perfluoroalkyl carbonates				
PFOA	Perfluorooctanoic acid	0.02[d]	0.16	0.006 µg/kg-week
Perfluoroalkane sulfonates				
PFOS	Perfluorooctanesulfonic acid	0.02[e]	0.02*	0.013 µg/kg-week
PFHxS	Perfluorohexanesulfonic acid		0.02*	
PFBS	Perfluorobutanesulfonic acid	10[f**]		
Perfluoroalkyl ether carboxylates				
GenX	Perfluoro-2-methyl-3-oxahexanoic acid	0.08[g**]		

*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

**Draft

References:

[a] https://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=1117&tid=237

[b] https://hhpprtv.ornl.gov/quickview/pprtv.php

[c] https://www1.health.gov.au/internet/main/publishing.nsf/Content/ohp-pfas-hbgv.htm#final

[d] https://www.epa.gov/sites/production/files/2016-05/documents/pfoa_health_advisory_final-plain.pdf

[e] https://www.epa.gov/sites/production/files/2016-05/documents/pfos_health_advisory_final-plain.pdf

[f] http://www.epa.gov/pfas/genx-pfbs

[g] http://www.epa.gov/pfas/genx-pfbs

[h] https://www.efsa.europa.eu/en/efsajournal/pub/5194

Appendix G Table 3: PFAS chemicals with toxicity assessments in progress by US EPA[a]

Acronym	Name
Perfluoroalkyl carbonates	
PFDA	Perfluorodecanoic acid
PFNA	Perfluorononanoic acid
PFHxA	Perfluorohexanoic acid
PFBA	Perfluorobutanoic acid
Perfluoroalkane sulfonates	
PFHxS	Perfluorohexanesulfonic acid

[a]. https://www.epa.gov/sites/production/files/2019-05/documents/pfas_research_list.pdf

G3.2 Toxicity of PFAS in landfill gas

As will be discussed in Section G4.3, the most abundant PFAS substances in landfill gas (prior to combustion) are expected to be FTOHs and PFBA. We have not identified any published toxicity values for fluorotelomer alcohols and they are not on the US EPA's work plan for development of toxicity factors.

It is our understanding that FTOHs are not in themselves particularly toxic but are of concern because they are precursors to the formation of PFCAs, including PFOA[1]. FTOHs can contribute to the body burden of PFOA (the PFCA of greatest concern) via several mechanisms, including:

- Biotransformation of 8:2FTOH to PFOA, combined with the long half-life of PFOA in humans (two to four years) (European Chemicals Agency, 2012[2]).
- Atmospheric oxidation of FTOHs released to air through a complex series of reactions initiated by hydroxyl (OH) radicals (Ellis et al, 2004).

If there were data available on the concentration of FTOHs in landfill gas, then a conservative assessment could be carried out by assuming that all FTOHs are converted to the equivalent PFCA through biotransformation and/or atmospheric oxidation. It is likely that emissions of 8:2 FTOH converting to PFOA would be the most important potential exposure. However, as previously discussed, we are not aware of any measurements of FTOHs in landfill gas in Australasia that would enable a quantitative assessment of fugitive emissions of these substances in landfill gas.

G3.3 Toxicity of PFAS found in leachate

Analysis of leachate from Australian landfills (Gallen et al, 2017 and Vic EPA[3]) showed there were five dominant PFAS compounds in both studies. In addition to PFOS, PFOA and PFHxS, two PFCA's (perfluorohexanoic acid (PFHxA) and perfluoroheptanoic acid (PFHpA)) were present in significant concentrations in leachate both studies. In the Vic EPA investigations, perfluorobutanesulfonic acid (PFBS) and perfluoropentanoic acid (PFPeA) were also found to be important.

The US EPA has developed a toxicity factor for PFBS that is 500 times higher than its toxicity factor for PFOS/PFOA. PFHxA is on the US EPA's work list for undertaking a toxicity assessment.

The Australian Department of Health (DoH) has undertaken human health assessments for a number of PFAS compounds under its Inventory Multi-tiered Assessment and Prioritisation (IMAP) framework. While these assessments do not provide toxicity factors that can be used in a quantitative risk assessment, they indicate the comparative toxicity for many of the PFAS compounds. With regard to PFBS, PFHxA and PFHpA, the IMAP assessments state the following:

- Data available indicate that PFBS has a more favourable toxicological profile and bioaccumulation potential than the long chain perfluoroalkyl substances. Chronic low-level effects on human health have not been identified.
- PFHxA and PFPeA are classed as a short chain (C4 – C6) perfluorocarboxylic acid. This class of compounds have potentially better human health outcomes and bioaccumulation than long-chain perfluoroalkyl substances. Chronic low-level effects on human health have not been identified. There is no evidence of significant hepatotoxicity or carcinogenicity in repeated dose toxicity studies. Compared with PFOA, the chemicals in the short-chain PFCA group showed developmental effects in mice at much higher doses[4].

[1] https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment_id=1687#cas-A_678-39-7

[2] European Chemicals Agency (2012). CLH Report for 8:2 FTOH

[3] https://ref.epa.vic.gov.au/your-environment/land-and-groundwater/pfas-in-victoria

[4] https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment_id=1686#cas-A_307-24-4

- The toxicity profile of PFHpA (C7) is expected to be intermediate to those of PFHxA and PFOA. Data for the critical effects of hepatotoxicity following repeated exposure, developmental toxicity and carcinogenicity are not available. Therefore, there are insufficient data to use in place of toxicity data for PFOA. Based on toxicokinetic data, it is anticipated that long-term effects would occur at higher doses compared with PFOA. However, a separate no-observed adverse effect level (NOAEL) cannot be established[5].

Based on this evaluation, PFBS and PFHxA do not warrant further consideration in the HHRA as they a relatively lower toxicity compared to PFOA and their inclusion is unlikely to alter the conclusions of the assessment, however PFHpA could be conservatively assessed in the HHRA using the toxicity factor for PFOA.

G3.4 Summary of toxicity values for relevant PFAS

Appendix G Table 4 summarises the toxicity values adopted for the quantitative assessment.

Appendix G Table 4: Toxicity values

PFAS compound(s)	Tolerable daily intake ng /(kg-bw · day)
∑PFOS and PFHxS	20
∑PFOA and PFHpA	160
PFBS	10,000

G4 PFAS in landfills

G4.1 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 G2 above, and in certain other waste streams such as biosolids from municipal wastewater treatment plants.

Specifically, in relation to fire-fighting foams, the EPA (NZ) has determined that waste 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) waste with a concentration less than 50 ppm (for example contaminated soil).

Waste acceptance criteria for PFAS (including PFOS)-containing waste 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.

G4.2 PFAS in leachate

Gallen et al (2017) present the results of analysis of leachate from 27 Australian landfills for nine PFAS. Higher mean concentrations of PFAS were measured in landfills 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.

[5] https://www.nicnas.gov.au/chemical-information/imap-assessments/imap-group-assessment-report?assessment_id=1689#cas-A_375-85-9

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 5).

PFBS was not included in the analytical suite in Gallen et al (2017), however was measured by Vic EPA in their study of Victorian landfills[6]. The highest measured concentration of PFBS (once an outlier value of 23 µg/L was removed) was 1.23 µg/L or 1230 ng/L. As there is a two to three order of magnitude difference in the toxicity values, PFBS has not been carried forward into the detailed HHRA calculations. If the HHRA findings were close to acceptable levels, then the contribution of PFBS may need to be considered.

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 5: Source concentrations of selected PFAS in leachate

PFAS compounds	Concentration in Redvale leachate	Adopted source concentration in leachate
	ng/L	ng/L
PFOA	920	1330[a]
PFHxS	890	2940[a]
PFOS	560	960[a]
PFHpA	370	1080[a]
PFBS	4600	-

[a] Source: Gallen et al (2017)

G4.3 PFAS in landfill gas

PFAS species in landfill gas are expected to be dominated by compounds with relatively low water solubility and relatively high vapour pressure. Based on monitoring of ambient air around landfills in Germany (Weinberg, 2010), Canada (Ahrens et al, 2011) and China (Tian, 2018), PFAS species will mainly comprise volatile and semi-volatile neutral PFAS compounds such as fluorotelomer alcohols (FTOHs) and, to a lesser extent, perfluorobutanoate (PFBA).

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 2.8×10^3 pg/m^3 to 2.6×10^4 pg/m^3 and comprised more than 90% fluorotelomer alcohols (FTOHs) and perfluorobutanoate (PFBA).

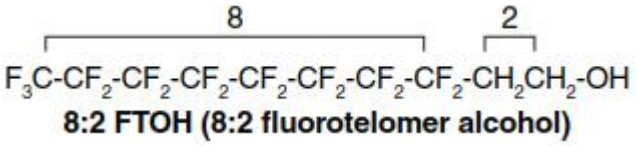
[6] https://ref.epa.vic.gov.au/your-environment/land-and-groundwater/pfas-in-victoria

Weinberg (2011) measured neutral PFAS in the air over two landfills in Germany. Concentrations were in the range 84 pg/m^3 to 706 pg/m^3 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^3, 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

Under ideal combustion conditions, FTOHs in landfill gas would be completely degraded to CO_2, HF and H_2O in the flares and generators. Studies have confirmed that this complete combustion occurs at a temperature of 1,000°C and 2 seconds residence time, which are the typical conditions for hazardous waste incinerators. The proposed flare(s) at the ARL will operate in accordance with the requirements of the NESAQ, which are a minimum of 0.5 seconds residence time at 750°C. We are not aware of any published data on the extent of complete combustion (i.e. the destruction efficiency) for PFAS substances under these conditions. Flares and generators are known to have a high destruction efficiency for a range of non-methane organic compounds (i.e. breaking of C-H and C-C bonds). However, due to the stability of the C-F bond, there is the potential for partial degradation products to be released.

FTOHs can be described by the formula $CF_{2n+1}CH_2CH_2OH$. The most common FTOHs detected in landfill gas are 6:2 FTOH and 8:2 FTOH. The chemical structure of 8:2 FTOH is shown below.



8:2 FTOH (8:2 fluorotelomer alcohol)

8:2 FTOH comprises 8 fully fluorinated carbon atoms and a non-fluorinated ethyl alcohol "tail". The weakest bonds in this structure are the bonds in the ethyl alcohol tail, and between the tail and the telemer group. Therefore, under thermal oxidation conditions (with excess air), the plausible partial degradation products for FTOHs are compounds containing the following fragments: $\bullet CF_3$ (trifluoromethyl functional group) and $\bullet CF_2CH{=}CH_2$ (olefin (alkene) functional group). The formation of compounds containing the $\bullet CF_2CH{=}CH_2$ fragment (i.e. fluorotelomer olefins or FTOs) was confirmed in tests of the thermal degradation products of fluorotelomer treated articles at 200°C and 600°C (Yamada et al, 2005).

We have reviewed available data on the fate of FTOs released to the air. FTOs are expected to partition to the atmospheric aqueous phase and undergo photooxidation to form the equivalent PFCA[7] (i.e. 8:2 FTO is likely form PFOA and 6:2 FTO would form PFHxA).

The absence of data on FTOH concentrations in landfill gas in Australasia makes it difficult to undertake a meaningful assessment of the contribution of these emissions to potential health impacts, either directly or through partial decomposition to PFCA precursor species. However, as discussed later, exposure to PFAS compounds could be nearly 10,000 times greater than estimated in the HHRA before levels would be of concern with respect to potential health impacts.

[7] UNEP (2016). Persistent Organic Pollutants Review Committee Twelfth meeting

G5 PFAS media concentrations

G5.1 PFAS in groundwater for potable supply

As presented in Appendix G Table 6, concentrations of PFAS have been determined in groundwater at the closest groundwater take for possible potable supply (POE#5). 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 6: Intake and hazard calculations for PFAS in drinking water

PFAS Compounds	Conc in farm bore	Ingestion rate	Tolerable daily intake	Hazard index
	ng/L	ng/kg-day	ng/(kg bw-day)	Unitless
PFOA	3.13E-04	2.41E-05	160	1.50E-07
PFHxS	6.92E-04	5.32E-05	20	2.66E-06
PFOS	2.08E-04	1.60E-05	20	8.00E-07
PFHpA	2.96E-04	2.28E-05	160	1.42E-07

G5.2 PFAS in surface water

As presented in Appendix G Table 7, 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 of the HHRA report, i.e.:

- Stream confluence (360 m) Valley 1 and 2 stream (POE#1).
- Hōteo 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). The concentration at the stream confluence is also conservatively assumed to include contributions from leachate present in storm water run-off (discussed in Appendix F).

Appendix G Table 7: 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	3.48E-09	3.48E-09	7.52E-09
PFHxS	7.69E-09	7.69E-09	1.66E-08
PFOS	2.32E-09	2.32E-09	5.24E-09
PFHpA	3.29E-09	3.29E-09	6.57E-09

G6 PFAS exposure pathways

G6.1 Ingestion of eel (tuna) flesh

G6.1.1 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 8 below).

Appendix G Table 8: 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 9.

Appendix G Table 9: BCF for eels adopted for this HHRA

PFAS Compounds	BCF	Source
PFOA	69	Developed from PDP, 2018
PFHxS	727	Developed from PDP, 2018
PFOS	5796	RIWA, 2017
PFHpA	69	Assumed the same as PFOA

The predicted eel flesh concentrations are presented in Appendix G Table 10.

Appendix G Table 10: Predicted eel flesh concentrations

PFAS Compounds	Predicted eel flesh concentration (ng/kg)
PFOA	0.519
PFHxS	12.09
PFOS	30.36
PFHpA	0.454

G6.1.2 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 of the main HHRA report.

Daily intake values for PFAS from consumption of eel flesh are presented in Appendix G Table 11 for both adults and children. The daily intake of PFAS from eel flesh are all well below the FSANZ TDI values.

Appendix G Table 11: 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)
PFOA	5.19E-04	1.20E-04	160
PFHpA	4.54E-04	1.05E-04	
PFOA + PFHpA	9.73E-04	2.24E-04	
PFHxS	0.0121	0.0028	20
PFOS	0.0304	0.0070	
PFOS + PFHxS	0.0425	0.0098	

G6.2 Ingestion of chicken eggs

G6.2.1 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 (Appendix G Table 12).

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 12: Chicken egg intake parameters

Parameter	Value	Source
Drinking water intake of the chicken (L/day)	0.208	Aecom, 2017
Percentage transferred to egg	PFOS/PFHpA*: 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

*Assumed to have the same values as PFOA as no PFHpA specific data was available.

Calculations for the concentration of PFAS in chicken eggs are summarised Appendix G Table 13.

Appendix G Table 13: Egg concentration calculations

PFAS Compounds	Concentration in drinking water	Percentage transferred to egg	Mass in egg	Concentration in egg
	mg/L	%	ng	ng/g
PFOA	3.13E-10	46	0.0033	6.66E-05
PFHxS	6.92E-10	69	0.0110	2.21E-04
PFOS	2.08E-10	100	0.0048	9.61E-05
PFHpA	2.96E-10	46	0.0031	6.29E-05

G6.2.2 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 14.

Appendix G Table 14: 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 15. 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 15: 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)
PFOA	2.60E-05	5.21E-05	4.10E-05	8.19E-05
PFHxS	8.64E-05	1.73E-04	1.36E-04	2.72E-04
PFOS	3.76E-05	7.53E-05	5.92E-05	1.18E-04
PFHpA	2.46E-05	4.93E-05	3.87E-05	7.75E-05

G6.3 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)

Cdw 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 16: 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) (Vd)	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 17: 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/ PFHpA*	0.1	0.8
PFHxS	0.05	10
PFOS	0.1	114

*Assumed to have the same values as PFOA as no PFHpA specific data was available

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

Appendix G Table 18: 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	3.13E-10	5.80E-11	3.19E-10	3.09E-08
PFHxS	6.92E-10	1.28E-10	8.81E-09	4.27E-07
PFOS	2.08E-10	3.85E-11	3.02E-08	2.93E-06
PFHpA	2.96E-10	5.48E-11	3.01E-10	2.92E-08

Appendix G Table 19: 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	7.52E-09	1.39E-09	7.66E-09	7.43E-07
PFHxS	1.66E-08	3.08E-09	2.12E-07	1.03E-05
PFOS	5.24E-09	9.70E-10	7.60E-07	7.37E-05
PFHpA	6.57E-09	1.22E-09	6.69E-09	6.49E-07

G6.4 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 20: Serum half-life and milk/serum ratio (from Aecom, 2017)

PFAS Compounds	Milk/serum ratio (MSR)	Dairy cow serum half life ($t_{1/2}$) (days)
PFOA/PFHpA*	1.90E-01	150
PFHxS	1.00E-02	10
PFOS	8.00E-03	1.3

*Assumed to have the same values as PFOA as no PFHpA specific data was available

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

Appendix G Table 21: 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	3.13E-10	5.80E-11	1.61E-08	3.06E-10	2.97E-07
PFHxS	6.92E-10	1.28E-10	7.11E-09	7.11E-11	6.91E-08
PFOS	2.08E-10	3.85E-11	2.78E-10	2.22E-12	2.16E-09
PFHpA	2.96E-10	5.48E-11	1.52E-08	2.89E-10	2.81E-07

Appendix G Table 22: 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	7.52E-09	1.39E-09	3.87E-07	7.35E-09	7.13E-06
PFHxS	1.66E-08	3.08E-09	1.71E-07	1.71E-09	1.66E-06
PFOS	5.24E-09	9.70E-10	7.00E-09	5.60E-11	5.44E-08
PFHpA	6.57E-09	1.22E-09	3.38E-07	6.42E-09	6.23E-06

G7 References for PFAS

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