

# **Per- and Poly- Fluorinated Alkyl Substances (PFAS) in selected New Zealand foods**

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# 1 Executive Summary

A survey for Per- and Poly-Fluorinated Alkyl Substances (PFAS) in 12 food groups, including vegetables, dairy product, meats, takeaway foods and seafood, was carried out on samples collected as part of the 2016 New Zealand Total Diet Study programme.

A single PFAS, perfluorohexanoic acid (PFHxA), was found in a beef rump steak sample, with no samples reporting the PFAS congeners related to current contaminated site investigations. Comparison of the analytical method performance to occurrence values for perfluorooctanoic acid (PFOA) and perfluorooctane sulphonic acid (PFOS) in overseas monitoring identified it was suitably sensitive to capture the potential ranges of occurrence in the food supply. Exposure of PFOS and PFOA, accounting for hypothetical levels up to the analytical limit of reporting (LOR), indicated negligible dietary risk.

While the source of the detected PFHxA is unknown, one possibility is migration from packaging or from cooking utensils. A margin of exposure (MOE) approach was used to characterise the potential dietary risk, as a health based guidance value has not been set for PFHxA in New Zealand. A toxicological point of departure of 15 mg/kg bw/day was selected from the critical chronic toxicity study in rats. PFHxA is reported to be much more rapidly eliminated in the body than PFOA and PFOS. PFHxA was also absent in a New Zealand blood monitoring study. Therefore, directly applying a point of departure from an animal study was justified. MOEs for all New Zealand population groups for PFHxA are sufficiently high to deem the dietary risk as negligible.

Considering these results, it is reasonable to use the current LORs as the New Zealand baseline to identify any dietary elevation. However, a site investigation may be needed to establish a localised baseline if there was to be attribution to an individual source within a region following the detection of an elevated level.

## 2 Background

Per- and Poly-Fluorinated Alkyl Substances (PFAS) are an emerging contaminant group with food safety concerns because of the degree of persistence and mobility in the environment, and the potential for accumulation in humans and long-term adverse health effects (EFSA, 2012; FSANZ, 2017). There are a vast number of different PFAS congeners (Table 1) that can enter the diet from environmental, processing and domestic applications. Dietary intake through migration or uptake into foods is expected to be a significant contributor to exposure to PFAS.

Three PFAS congeners (PFOS, PFHxS and PFOA) are currently being targeted in current environmental investigations in New Zealand.

Surveys or routine monitoring of PFAS congeners in the New Zealand diet have not previously been carried out, although there has been limited monitoring of food and biota around some site investigations. Consequently a current baseline for dietary levels, and therefore population exposures, does not exist. A survey of PFAS in twelve food types was therefore commissioned to establish occurrence in the New Zealand diet.

## 3 Method

### 3.1 SAMPLING

Samples for PFAS analysis were selected from the food types collected in the 2016 New Zealand Total Diet Study (Pearson *et al.*, 2018) on the basis of overseas reports of presence of PFAS congeners, and to obtain a spread of meat, shellfish, dairy products and vegetables.

For example, fish and sausages were selected as PFOS was detected in these food types in the 24<sup>th</sup> Australian Total Diet Study (FSANZ, 2016).

Table 1: List of PFAS congeners and abbreviated titles tested in a survey of New Zealand foods

Congener	Abbreviation
<b><u>Perfluoroalkylsulphonic acids</u></b>	
Perfluoropropane sulphonic acid	PFPrS
Perfluorobutane sulphonic acid	PFBS
Perfluoropentane sulphonic acid	PFPeS
Perfluorohexane sulphonic acid	PFHxS (*)
Perfluoroheptane sulphonic acid	PFHpS
Perfluorooctane sulphonic acid	PFOS (*)
Perfluorononane sulphonic acid	PFNS
Perfluorodecane sulphonic acid	PFDS
<b><u>Perfluorooctanesulphonamides</u></b>	
Perfluorooctanesulphonamide	PFOSA
N-ethylperfluoro-1-octanesulphonamide	NEtFOSA-M
N-methylperfluoro-1-octanesulphonamide	NMeFOSA-M
<b><u>Perfluorooctanesulphonamidoacetic acids</u></b>	
N-ethylperfluorooctanesulphonamidoacetic acid	NEtFOSAA
N-methylperfluorooctanesulphonamidoacetic acid	NMeFOSAA
<b><u>Perfluorooctanesulfonamidoethanols</u></b>	
2-(N-ethylperfluoro-1-octanesulphonamido)-ethanol	NEtFOSE-M
2-(N-methylperfluoro-1-octanesulphonamido)-ethanol	NMeFOSE-M
<b><u>Perfluoroalkylcarboxylic acids</u></b>	
Perfluorobutanoic acid	PFBA
Perfluoropentanoic acid	PFPrA
Perfluorohexanoic acid	PFHxA
Perfluoroheptanoic acid	PFHpA
Perfluorooctanoic acid	PFOA (*)
Perfluorononanoic acid	PFNA
Perfluorodecanoic acid	PFDA
Perfluoroundecanoic acid	PFUnDA
Perfluorododecanoic acid	PFDoDA
Perfluorotridecanoic acid	PFTriDA
Perfluorotetradecanoic acid	PFTeDA
<b><u>Telomer Sulphonic acids</u></b>	
1H,1H,2H,2H-perfluorohexanesulphonic acid	4:2 FTS
1H,1H,2H,2H-perfluorooctanesulphonic acid	6:2 FTS
1H,1H,2H,2H-perfluorodecanesulphonic acid	8:2 FTS

(\*) Congeners of current regulatory interest in New Zealand

Eight samples were available of each of the 12 food types selected (Table 2). All samples were composite samples, pooled from usually four individually collected samples. The composite sample of each food was generally collected from each of four regional centres (Auckland, Napier, Christchurch and Dunedin) and on two occasions over the 2016 calendar year (January-February and June-July). The composite cheese samples were all collected from Christchurch in March-April and in October-November. Each composite comprised of pools of individual samples of a retail brand.

All food samples were prepared using standard cooking practices to the form in which they would usually be consumed (Table 2). All dry-frying was undertaken in polyfluorotetraethylene coated non-stick frying pans, in addition polyfluorotetraethylene based utensils were also used in sample preparation. As a result, the samples reflect the total PFAS concentrations that will be ingested through the diet, accounting for contribution from the environment, processing and food preparation.

Table 2: Food types analysed and details of food preparation processes (Pearson *et al.*, 2018)

Food type	Sample number	Sample preparation
Beef, rump	8	Fat trimmed, dry fried for 5 minutes each side until cooked, chopped, mixed and homogenised
Butter	8	Chopped, mixed and homogenised
Cheese	8	Chopped, mixed and homogenised
Egg	8	Boiled for 5 minutes, cooled, peeled, mixed and homogenised
Fish, fresh	8	Dry fried for 3 minutes each side until cooked, mixed and homogenised
Hamburger, plain	8	Chopped, mixed and homogenised
Lamb/mutton	8	Mutton chops: dry fried for 5- 8 minutes each side Lamb roast: roast in 180°C for 20-25 minutes per 500g Chopped, combined, mixed and homogenised
Lettuce	8	Inner leaves rinsed and homogenised
Mussels	8	Fresh mussels: Steamed in 2 cm of boiling water in a pot (mussels that remained closed were discarded), flesh scooped out, shells discarded Packaged mussels: Drained Combined, mixed and homogenised
Pork roast	8	Roasted at 160°C for 30-40 minutes (per 500 g) until cooked, cooled, flesh removed, mixed and homogenised
Potato, with skin	8	Scrubbed, baked at 200°C for 50-60 minutes until soft and homogenised
Sausages	8	Dry fried for 5 minutes on each side until cooked, chopped, mixed and homogenised

Samples were stored frozen in polycarbonate containers for up to 24 months prior to shipment to the testing laboratory. Fluorinated materials were not used subsequent to sample preparation to prevent inadvertent laboratory contamination. Appreciable loss was not expected during storage as PFAS congeners are highly stable.

## 3.2 ANALYSIS

All 96 food composite samples were analysed for PFAS (all 29 congeners; Table 1) using liquid chromatography and tandem mass spectrometry. Results for PFOS and PFHxS were presented as totals or individual for the branched and linear chain isomers. Only totals for each congener are reported as no difference in results were observed between the separate isomers and the total congener.

A total of 2698 results were reported. Extraction of some of the PFAS congeners was unreliable from a small proportion of the foods, with 86 results (3% of total analytical reporting) unable to be reported.

The majority (77%) of analyte/food type combinations had limits of reporting (LOR) of 0.1 - 0.25 µg/kg. However, there were a number of compound and sample combinations where a higher LOR was reported. All non-reported analytes and analytes with non-standard LORs are presented in Table 3.

Table 3: PFAS congeners and food types with results unable to be reported, or LORs above 0.25 µg/kg.

Congener	Food types	LOR (µg/kg)
NEtFOSAA	Egg, lettuce	2.5
NEtFOSAA	Potato	12.0
NEtFOSA-M	Lettuce	2.5
NEtFOSA-M	Egg, fish, mussels (5 out of 8), potato	12.0
NEtFOSE-M	Beef rump, hamburger, lamb/mutton, mussels (3 out of 8), pork roast, sausage	2.5
NEtFOSE-M	Egg, fish, lettuce, mussels (5 out of 8), potato	12.0
NMeFOSAA	Egg, lettuce	2.5
NMeFOSAA	Potato	12.0
NMeFOSA-M	Beef rump, hamburger, lamb/mutton, lettuce, mussels (3 out of 8), pork roast, sausage	2.5
NMeFOSA-M	Egg, fish, mussels (5 out of 8), potato	12.0
NMeFOSE-M	Beef rump, hamburger (7 out of 8), lamb/mutton, mussels (3 out of 8), pork roast, sausage	2.5
NMeFOSE-M	Egg, fish, lettuce, mussels (5 out of 8), potato	12.0
PFBA	Egg, lettuce, potato	0.5
PFBA	Beef rump, cheese, hamburger, lamb/mutton, pork roast	Not able to be reported
PFDODA	Butter, cheese	1.2
PFDODA	Beef rump, fish, hamburger, lamb/mutton, mussels, pork roast, sausage	12.0
PFDODA	Egg, lettuce, potato	50.0
PFDS	Egg, lettuce	2.5
PFTeDA	Butter, cheese	5.0
PFTeDA	Pork roast	12.0
PFTeDA	Beef rump, fish, hamburger, lamb/mutton, lettuce, mussels (5 out of 8)	50.0
PFTeDA	Egg, mussels (3 out of 8), sausage	Not able to be reported
PFTrDA	Butter	1.2
PFTrDA	Beef rump, egg, fish, hamburger, lamb/mutton, lettuce, mussels (5 out of 8), pork roast, potato	12.0
PFTrDA	Mussels (3 out of 8), sausage	Not able to be reported
4:2 FTS	Lettuce, hamburger	Not able to be reported
8:2 FTS	Egg	0.5

## 4 Results

A PFAS congener was recorded in one of the 96 food samples; PFHxA in a beef rump steak sample (Table 4).

None of the three PFAS congeners targeted in current New Zealand investigations (PFOA, PFOS and PFHxS) were detected above the analytical LOR. This included individual tested isomers of PFOS and PFHxS (linear and branched chains) and for the total of the isomers.

Table 4: Detected PFAS concentration in a survey of New Zealand foods.

Food type	Congener	Concentration (µg/kg)	LOR (µg/kg)
Beef, rump	PFHxA	0.42	0.25

## 5 Discussion

### 5.1 PFOS, PFHXS AND PFOA

#### 5.1.1 Hazard characterisation

Health based guidance values, in the form of tolerable daily intakes, have been established recently for the sum of PFOS and PFHxS, and for PFOA, by Food Standards Australia New Zealand (FSANZ, 2017). These values are:

Sum of PFOS and PFHxS: 20 ng/kg bw/day.  
PFOA: 160 ng/kg bw/day.

Both values have been used for the hazard characterisation of theoretical exposure to PFOA, PFOS and PFHxS in the New Zealand diet.

#### 5.1.2 Exposure assessment

While PFOA, PFOS and PFHxS were not present above the LOR in the survey of 96 food samples, it was still possible to establish the theoretical range of dietary exposures. The approach taken substituted the LOR for either zero, to establish a lower-bound (LB) mean-exposure, or for the value of the LOR (0.1 µg/kg for butter and cheese; and 0.25 µg/kg for all other foods) to establish an upper-bound (UB) mean-exposure. The potential exposure was then anticipated to fall within this range and the risk of the theoretical exposure calculated.

Exposure assessments were undertaken based on the intakes of each food type for different population cohorts reported in the 2016 New Zealand Total Diet Study (Table 5). The intakes were developed as part of a simulated fortnightly diet. A longer term pattern of intake is the most appropriate measure to estimate exposure because these three PFAS congeners are regulated as sub-chronic to chronic toxicants.

Table 5: Daily average intake amounts (in g/day) of different population cohorts for the tested food types (Pearson *et al.*, 2018).

Food type	Adult females	Adult males	Teenage boys	Teenage girls	Children	Toddler	Infant
Beef, rump steak	8	12	14	11	6	4	2
Butter	9	10	7	5	5	4	3
Cheese	13	16	15	15	7	10	8
Eggs	20	19	16	14	11	8	4
Fish, fresh	14	17	12	5	3	2	1
Hamburger, plain	7	17	21	19	7	6	3
Lamb/mutton	8	10	7	6	3	3	2
Lettuce	9	9	14	9	2	1	0
Mussels	4	6	1	1	0	0	0
Pork roast	6	8	8	6	3	1	1
Potatoes, with skin	18	30	26	32	20	17	11
Sausages	7	17	21	14	14	11	5

Estimates of exposure for each of the population cohorts is presented in Table 6. Minimal difference in exposure was reported between adult males (25 years and above) and either young adult males (19-24 years of age) or adult males of Pacific Island ethnicity, and hence these were combined, as were adult females and adult females of Pacific Island ethnicity.

Table 6: Estimated LB and UB mean exposure ranges (in ng/kg bw/day) to PFOS+PFHxS and PFOA, and expressed as a percentage of the tolerable daily intake.

Congener(s)	Adult females	Adult males	Teenage boys	Teenage girls	Children	Toddler	Infant
PFOS+PFHxS	0-0.8	0-0.9	0-1.4	0-1.2	0-1.6	0-2.2	0-1.9
As %TDI	0-3.8	0-4.4	0-6.8	0-5.8	0-8.0	0-11.2	0-9.2
PFOA	0-0.4	0-0.5	0-0.7	0-0.6	0-0.8	0-1.1	0-0.9
As %TDI	0-0.3	0-0.3	0-0.4	0-0.4	0-0.5	0-0.7	0-0.6

### 5.1.3 Risk characterisation

The theoretical UB mean exposure to PFOS and PFHxS did not exceed 12% of the TDI for any of the population cohorts, while that for PFOA was below 1% of the TDI (Table 6). Exposure at the UB is the worst-case and a more realistic exposure will fall within the range of LB to UB mean exposures. However, even with the UB exposures, consumers are highly unlikely to see a dietary risk from PFOS, PFHxS and PFOA in the tested foods.

A full dietary exposure cannot be calculated as only a proportion of the foods in a normal varied diet have been tested. However, a European Food Safety Authority (EFSA) report suggests that adult mean dietary exposures to PFOS typically lie below 5.2 ng/kg bw/day (UB-mean consumer exposure for all adult age groups) of which fish and other seafood contributed 70-80% of exposure (EFSA, 2012). If this ratio to other foods is consistent between Europe and New Zealand, then the absence of PFAS congeners in fish and seafood in New Zealand suggests that other foods are highly unlikely to be large contributors to exposure.

In contrast, exposure sources for PFOA were more consistent across the European diet, with adult mean dietary exposures of 4.3 ng/kg bw/day (UB-mean consumer exposure for all adult age groups; EFSA, 2012), this is potential the case in New Zealand. However, based on the reported exposure in Europe the risk of exceeding the TDI for PFOA is highly unlikely.

### 5.1.4 Characterisation dietary baseline level

All of the LORs for PFOA, PFOS and PFHxS in the current survey were all in the range of 0.1 - 0.25 µg/kg which are sufficient to identify concentrations that could present a dietary concern. However, for the purposes of identifying the dietary baseline failure to detect PFOA, PFOS and PFHxS at concentrations above the LOR in the New Zealand diet could reflect a true low-level baseline, or insensitivity of the method.

Evaluation of published data from Australia and Europe provides confidence that the methods used in New Zealand were sufficiently sensitive for most foods to establish a baseline for appreciable entry into the diet, and to allow comparison with international studies.

In an Australian survey, PFOS was detected in only two of 304 samples, from 50 foods types, and PFOA was not detected (FSANZ, 2016). LORs in this study were typically 0.1-0.6 µg/kg, although water, sugar and UHT milk were lower. The reported results were of 1.0 µg/kg in fish fillets and 0.2 µg/kg in sausages. Samples were not tested for PFHxS.

EFSA has compiled food monitoring data on PFAS from across Europe (EFSA, 2012). The concentration ranges observed for PFOS and PFOA for the comparable food types to those tested in New Zealand were within the LORs for methods used in New Zealand (Table 7).

Table 7: Comparison of New Zealand analytical method sensitivity to reported occurrence ranges for PFOS, PFHxS and PFOA in European food monitoring (EFSA, 2012).

Food type	New Zealand LOR ( $\mu\text{g}/\text{kg}$ )	Concentration ranges for reported detections of PFAS congeners in European food surveys ( $\mu\text{g}/\text{kg}$ )		
		PFOS	PFHxS	PFOA
Butter	<0.10	Not detected	0.01	0.023
Dairy	<0.10	0.005-1.2	Not detected	0.007-3.7
Eggs	<0.25	0.002-6.4	0.005	0.006-25.5
Fish meat	<0.25	0.04-211	1.0	0.006-18.2
Other vegetables	<0.25	0.004-1.54 <sup>a</sup>	0.003-0.004	0.013-0.54
Livestock meat	<0.25	0.003-1.74	0.076	0.008-3.3
Root vegetables	<0.25	1.2	Not detected	0.031
Sausages	<0.25	0.08-16.5	Not detected	Not detected
Shellfish	<0.25	0.02-2.9	Not detected	0.03-0.98

<sup>a</sup> PFOS range for "other vegetables" also includes wild edible fungi.

The frequency of detection of PFHxS in Europe was considerably lower than PFOS. For example from 927 samples of fish meat reported there was only a single detection. The lower detection frequency in the EFSA monitoring, and the tendency for concentration ranges when detected to be below the New Zealand LOR, indicates the sensitivity of the LOR likely would have to increase to establish the true baseline ranges.

The general absence of PFAS in the diet suggests that the current LORs can be used as the baseline to identify any dietary elevation. However, a site investigation may need to establish a localised baseline to attribute any elevation to an individual source with a region.

## 5.2 PFHXA

### 5.2.1 Context of finding and potential sources

Food from the general supply in New Zealand has not previously been tested for PFHxA, and hence there is not a baseline against which the findings of the current study can be evaluated.

The 2012 UK Total Diet Study tested composite food samples for PFHxA (as part of an assay for the levels of 11 PFAS congeners) (Fernandes *et al.*, 2012). The reported PFHxA concentrations were 0.79  $\mu\text{g}/\text{kg}$  in the offal composite sample (85 individual samples) and 0.5  $\mu\text{g}/\text{kg}$  in the meat products composite sample (123 individual samples). Levels were below reporting limits in the carcass meat composite samples (51 individual samples). In contrast, PFHxA was detected in green vegetables at 2.8  $\mu\text{g}/\text{kg}$  for the composite (23 individual samples), the highest in the survey.

EFSA's review of dietary levels of various PFAS congeners across Europe also included results for PFHxA (EFSA, 2012). Only 1/183 pooled livestock meat samples analysed for PFHxA had a reported result (0.3  $\mu\text{g}/\text{kg}$ ). PFHxA was more prevalent in farmed animal offal samples (6%), with a reported results range of 0.3-3.4  $\mu\text{g}/\text{kg}$ . PFHxA was also not detected in a study of PFAS in beef muscle (176 samples) and liver (117 samples) in Xinjiang, China, although 12% of liver samples had detectable concentrations (mean: 0.016  $\mu\text{g}/\text{kg}$ , maximum: 0.25  $\mu\text{g}/\text{kg}$ ; Wang *et al.*, 2017). Finally, a recent study of PFAS concentration in food purchase in Taipei City detected PFHxA in the ten beef samples tested, with a reported geometric mean of 1.16  $\mu\text{g}/\text{kg}$  (Chen *et al.*, 2018).

The reported concentration of PFHxA in beef steak in the New Zealand survey is consistent with the concentration range reported for this congener in overseas reports. Unfortunately, the analysis of a composite of four individual samples makes identification of the source of the PFHxA difficult.

PFHxA is more rapidly eliminated than PFOS or PFOA, with similar clearance mechanisms amongst mammalian species (Russell *et al.*, 2013). The absence of any other PFAS congeners, in particular the more environmentally and biologically persistent compounds such as PFOS, suggest the source was unlikely to be from an environmental contamination. This is supported by detection, in the

absence of PFHxA (<0.5 µg/L all samples), of other PFAS congeners including PFOA (mean: 0.24 µg/L), PFDA (mean: 9.7 µg/L), PFHxS (mean: 52 µg/L) and PFOS (mean 509 µg/L) in sera from cattle (15 samples) inside the contamination plume from a spill of PFAS containing material around Australia's Army Aviation Centre Oahey (Bräunig *et al.*, 2017).

It is possible that the PFHxA was from a non-agricultural source. A study in Japan comparing PFAS between livestock and domestic pet species (Guruge *et al.*, 2008) showed that farmed animals only rarely had PFHxA in liver or sera, whereas the sera of dogs was always positive. Potential sources allowing migration could be waxes or polymers used in the abattoir, fluorinated coatings on utensils or surfaces used in the butchering, coatings in the packaging, or cooking utensils used in the laboratory preparation.

PFHxA can be formed through the metabolism or decomposition of other fluorinated chemicals, most notably 6:2 fluorotelomer alcohol (6:2 FTOH) and 6:2 dipolyfluoroalkyl diesterphosphate (6:2 diPAP). The former migrates to foods from both non-stick cookware and from packaging (Sinclair *et al.*, 2007; Yuan *et al.*, 2016); the latter also from packaging (Gebbinck *et al.*, 2013, 2015).

### 5.2.2 Hazard characterisation

Health based guidance values for PFHxA are not currently available in New Zealand. An alternative approach of estimating a margin of exposure, the magnitude of the difference between the estimated exposure in the diet and a toxicological point of departure obtained from an animal study, is therefore required to carry out a risk assessment of for the occurrence of PFHxA.

In contrast to PFAS congeners of regulatory interest where a large disparity is seen between humans and laboratory animal species, the elimination kinetics of PFHxA are considered to be similar between mammalian species. However, published studies indicate that the bioaccumulation potential of PFHxA is less than that of the longer-chain PFAS compounds (Rice, 2015). This is supported by the absence of detected PFHxA in a New Zealand serum monitoring study in which the longer-chain PFAS congeners (such as PFOS) were detected (t' Mannetje *et al.*, 2013). The comparable elimination of PFHxA likely reflects a similar mechanism of elimination and a reduced influence of reabsorption transporters (Rice, 2015).

The likely comparable toxicokinetic profile of PFHxA across species provides more certainty for directly comparing the points of departure in laboratory animal studies. A two year study where male rats were orally dosed in groups with either 0, 2.5, 15 and 100 mg/kg/day PFHxA; and female rats with 0, 5, 30 and 200 mg/kg/day PFHxA (Klaunig *et al.*, 2015) was identified as the critical study for establishing a point of departure of PFHxA. Effects were reported only in the highest dose group for both sexes. Male rats had lower urinary pH and reduced serum triglyceride and free fatty acids. Female rats had pathological changes in the kidneys, and occasional decreases in erythrocyte count and haemoglobin and increase in reticulocyte counts. Carcinogenicity and dose-related neurotoxicity were not reported in any dosing group. The reported "no observed adverse effect levels" (NOAELs) were 15 mg/kg/day for males and 30 mg/kg/day for females.

Based on the NOAEL in male rats, a point of departure of 15 mg/kg/ bw/day was selected to use in the risk characterisation for PFHxA.

An alternative, more conservative approach compared the exposure values against the TDI for PFOA. There is no indication PFHxA shares the slow elimination of PFOA, unlike the comparable elimination profiles of PFHxS and PFOS. However, it was concluded that the mechanism of toxicity was sufficiently similar to deem the PFOA TDI a conservative characterisation of the potential hazard. While the considerably more rapid elimination profile of PFHxA suggests a reduced potency in comparison to PFOA, equivalence factors have not been considered to date.

### 5.2.3 Exposure assessment

Average exposure over time is most important for a hazard with chronic human health effects. The detection of PFHxA above the LOR in just one of the eight samples of beef rump steak tested suggests that it is unlikely that every steak consumed by an individual over time would contain PFHxA at a concentration of 0.42 µg/kg.

However, PFHxA may be present in steak and other foods at levels below the LOR and an estimate of exposure using the LB and UB means was carried out (Table 8). The mean exposure for all the population cohorts was calculated and applied to the foods in which PFHxA was not detected above the LOR (Table 8).

Both exposure calculations use the intake values for each population cohort from the simulated diets in the 2016 NZTDS (Pearson *et al.*, 2018)

Table 8: Estimated lower-bound and upper-bound mean dietary exposure ranges (in ng/kg bw/day) for PFHxA.

Scenario	Adult females	Adult males	Teenage boys	Teenage girls	Children	Toddler	Infant
Beef steak only	0.01-0.03	0.01-0.04	0.01-0.07	0.01-0.06	0.01-0.07	0.02-0.08	0.01-0.06
All tested foods	0.01-0.42	0.01-0.50	0.01-0.76	0.01-0.64	0.01-0.89	0.02-1.30	0.01-1.12

#### 5.2.4 Risk characterisation

A margin of exposure was calculated by comparing the estimated exposures to PFHxA in the diet through consumption of beef rump steak to the point of departure. The margin of exposure between the two values was at least 100 million for all population cohorts, reflecting an exposure eight orders of magnitude below the dose causing toxicity in animals. Similarly, an assumption that PFHxA is present in all other foods tested at a level equalling the LOR (0.25 µg/kg) still results in the margin of exposure of at least 10 million for all population cohorts.

A margin of exposure of PFHxA greater than 100 for a compound without suspected or demonstrated carcinogenicity, deems its presence in the foods tested in this survey to be a negligible dietary risk. In addition, the observation that none of the estimated exposures exceeded 1% of the PFOA TDI (160 ng/kg bw/day), gives further confidence of negligible dietary risk.

## 6 Conclusion

A survey of 12 food types was undertaken to establish current baselines of PFAS in the New Zealand diet. Testing for 29 PFAS congeners was undertaken on 96 composite food samples purchased from retail and prepared for consumption as would be expected by the general population.

PFAS congeners were detected above the LOR in one of 2698 reported results, specifically PFHxA at 0.42 µg/kg in a beef rump steak sample. PFAS congeners related to current environmental investigations in New Zealand (PFOS, PFHxS and PFOA) were not detected above the LOR of 0.1-0.25 µg/kg.

PFOS and PFOA have been detected overseas above 0.25 µg/kg and this indicates that the LOR are sufficiently sensitive to derive a New Zealand baseline. Despite the absence of PFOS, PFHxS and PFOA, a risk assessment was undertaken to establish theoretical ranges of dietary exposure in New Zealand. Characterisation of the theoretical exposure ranges against the FSANZ TDI values for PFOS+ PFHxS and PFOA indicates that even worst-case baseline exposure would not exceed 12% of the TDI.

PFHxA from the beef steak most likely originated from its packaging or cooking utensils. As health based guidance values for PFHxA are not available a margin of exposure was calculated from the critical endpoint in an animal toxicity trial. PFHxA appears to be rapidly eliminated in humans and the disparity between excretion rates from laboratory animals to humans reported for other PFAS compounds was not evident. As a result, characterising the risk in terms of effects in laboratory animals is justified.

The exposure assessment for PFHxA gave a margin of exposure of 10-100 million, which indicates that the reported detection does not present a dietary risk.

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