Concerns about the safety of traditional Māori food in south Taranaki, New Zealand have been prompted by recent reports of residues of perfluorooctanesulfonic acid (PFOS; Figure 1) derived from firefighting foam in south Taranaki (specifically in the environs of a fire training facility near to the Oaonui Stream) groundwater (0.36µg/L; not used for drinking water), water cress (1.4µg/kg; very low level) and eels (410µg/kg) harvested from the Oaonui Stream in the same region. Monitoring focused on the Oaonui Stream because it flows past a fire training facility where PFOS-containing firefighting foams might have been used. PFOS residues were not detected in mussels from the coastal region near to the Oaonui Stream outlet. These results are included in a data report provided by the Taranaki Regional Council. They refer to samples (n=6) taken on 10 May 2018 (water samples) and 18 July 2018 (eel and watercress samples) as part of the Council’s monitoring programme.

Rationale for risk assessment

In this risk assessment, the highest PFOS level in eels will be used to reflect a worst-case exposure scenario. PFOS exposure will be calculated based on the eel residue level and a Māori eel consumption estimate, because eel is a traditional Māori food. Eel-consuming Māori PFOS exposure levels will be compared with exposure levels in rat carcinogenicity studies as a means of assessing worst-case cancer risk.

Chemical properties, food residues and biological half-life of PFOS

PFOS is very hydrophobic (estimated LogK_{ow} = 4.49, water solubility = 3.2x10^{-3}mg/L at 25°C) and so would be absorbed by the lipid-based cell membranes of microorganisms, which form the basis of the aquatic food chain. Concentration up the food chain (bioaccumulation) leads to high levels in top or near top creatures (eg, eels). Eels’ longevity means that they inhabit contaminated environments for a long time and constantly accumulate hydrophobic chemicals such as PFOS—this is the worst-case food residues scenario.

The hydrophobicity of PFOS means that it binds to lipoproteins in blood and resides in biological membranes, which likely accounts for its long serum half-life of >5 years and its incredibly long biological half-life of approximately 90 years in humans.

Is PFOS carcinogenic?

There is conjecture about whether or not PFOS is carcinogenic. Studies in rats have shown a statistically significant increase in hepatocellular adenoma at a 20ppm (mg/kg) in diet PFOS dose. Nakayama et al (2005) linked PFOS workplace exposure to prostate and bladder cancer in humans, but these human carcinogenicity findings were later refuted. However, an extensive and comprehensive review of occupational and environmental PFOS exposure concluded that there is no causal link between PFOS and cancer in humans. Despite this, the United States Environmental Protection Agency (USEPA) in a comprehensive PFOS human health risk assessment acknowledged the uncertainties relating to human carcinogenicity, but concluded that there is “suggestive evidence of carcinogenic potential for PFOS”.

Interestingly, the molecular structure of PFOS (Figure 1) and a consideration of its chemical reactivity does not suggest that there is a potential covalent reaction pathway between PFOS and nucleic acids because perfluorocarbons are generally chemically inert due to their strong C-F bonds. This means that PFOS is unlikely to be a genotoxic carcinogen, which is supported
by genotoxicity studies being uniformly negative.\textsuperscript{8} This suggests that if PFOS is carcinogenic then it is via a non-genotoxic mechanism, and again this is supported by negative genotoxicity studies,\textsuperscript{8} or that a carcinogenic impurity is present in technical PFOS (akin to carcinogenic dioxin contaminants in Agent Orange)\textsuperscript{9}. Indeed, the PFOS preparation used by Butenhoff et al was technical grade (86.9\% pure)\textsuperscript{5} and so might have contained carcinogenic impurities.

From the point of view of direct human exposure to technical PFOS (eg, occupational exposure) whether PFOS per se or an impurity results in cancer is irrelevant. However, the situation is quite different for a food exposure scenario where PFOS and the putative carcinogenic contaminant might be separated during transfer through environmental systems (eg, aquatic environment) prior to forming residues in food species (eg, long fin eel) inhabiting the contaminated environment. This might mean that PFOS residues in food species do not mirror cancer risk because the putative carcinogen residues might be separated from PFOS residues.

However, the conflicting evidence about PFOS’s carcinogenicity supports implementation of the precautionary principle in a human risk assessment context. To assess human cancer risk, intake and a trigger level are needed. In this assessment, we will use the rat hepatocellular adenoma dose as the ‘carcinogenicity’ trigger level (this is an extreme worst case because PFOS results in hepatocellular adenoma, which is not malignant) and intake via eel consumption.

Assessing Māori exposure to PFOS from eel consumption

Assessing human PFOS exposure levels via eel consumption is difficult because there are no published data on eel consumption rates in Māori. Informal discussions with some of the whanau of Muruwi marae in Opape and Punawhakareia marae in Rotoiti suggest that the consumption of eels in this group is approximately 1kg/year. Consumption varies throughout the year, increasing in times when eels are traditionally caught and eaten. Since PFOS is very hydrophobic it is likely to accumulate in the body and thus its effects are likely to be cumulative.

Māori PFOS exposure cancer risk assessment

The maximum longfin eel PFOS residue level found was 410µg/kg,\textsuperscript{1} which means that approximately 410µg would be ingested per person per year based on our estimated eel consumption. Assuming an average human body weight of 70kg, this equals a dose of 5.8µg/kg body weight/year (approx. 0.016µg/kg body weight/day). This equates to an approximate two-year cumulative dose of 11.6µg/kg body weight. The dose that resulted in hepatocellular adenoma in the rat studies is 20mg/kg in diet.\textsuperscript{5} Assuming that a 200g rat (ie, approx. weight of rats in study) consumes 150g food/day,\textsuperscript{10} the PFOS intake in the rat study was 15mg/kg body weight/day. Therefore, the PFOS dose that caused hepatocellular adenoma in rats was 15mg/kg body weight/day and the estimated Māori PFOS dose from eel consumption is approximately 0.016µg (0.000016mg)/kg body weight/day (averaged from yearly intake); therefore, the Māori dose is approximately 0.0001\% of the ‘carcinogenic’ dose in rats. Consumption of the yearly eel intake in a single meal would lead to a PFOS dose of 3µg/kg body weight/day, which is approximately 0.02\% of the ‘carcinogenic’ rat dose—these very low PFOS intakes suggests that the cancer risk to Māori consuming 1kg eels per year with PFOS residues akin to those found in the south Taranaki region is negligible.
Food Standards Australia New Zealand PFOS Tolerable Daily Intake

Food Standards Australia New Zealand (FSANZ) set a tolerable daily intake (TDI) for PFOS of 20ng/kg body weight/day based on decreased parental and offspring weight in rat reproductive toxicity studies.11 FSANZ acknowledge that this TDI is very conservative and was set as an interim measure to protect human health. Our calculated PFOS average daily intake (0.016µg (16ng)/kg body weight/day) is marginally below the FZANZ TDI whereas the ‘single meal’ PFOS intake (3µg (3,000ng)/kg body weight/day) exceeds the FSANZ TDI by at least two orders of magnitude. The TDI is the amount of a compound that can be consumed daily for a lifetime with no ill effects. Assuming Māori consume 1kg eels per year, the best estimation of daily exposure is our calculated average daily intake, which is just below the TDI.

In conclusion, PFOS residues in eels are likely to be of no concern from a carcinogenicity perspective, but are close to the highly conservative FSANZ TDI when PFOS intake is averaged across a year, but this is of questionable toxicological significance.

Competing interests:
Nil.

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