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Working globally for a toxic free future

The Persistence and Toxicity of Perfluorinated Compounds in Australia

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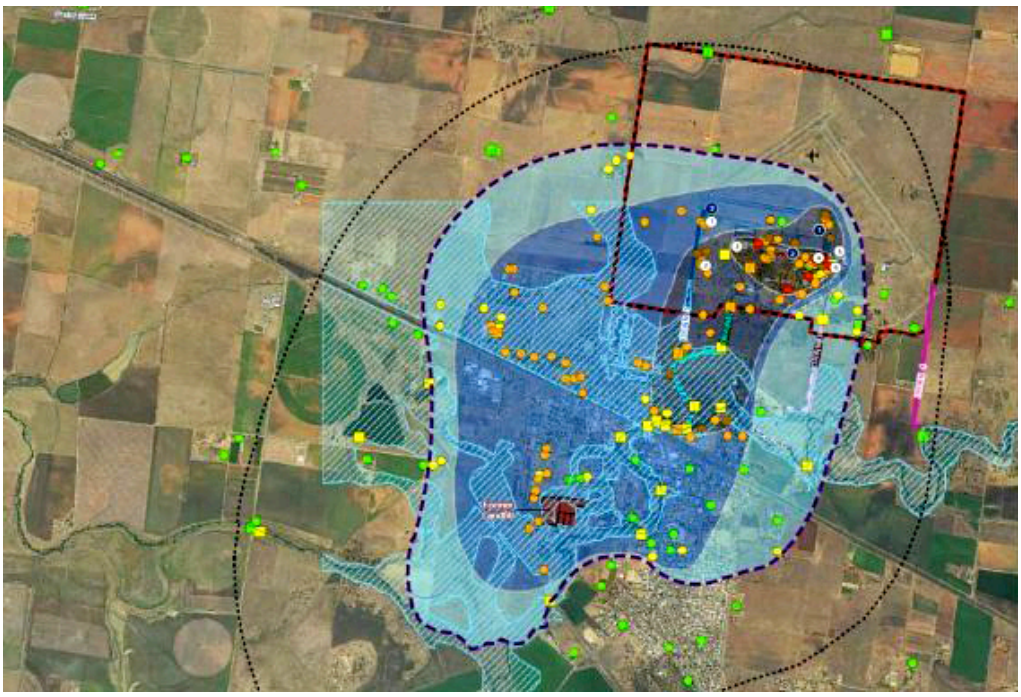
This report is intended to be a living document and will be updates as new important information is released.

Contents



Contaminated Groundwater Plume, Oakey Queensland

1 Executive Summary

"Given the inherent properties of PFOS, together with demonstrated or potential environmental concentrations that may exceed the effect levels for certain higher trophic level biota such as piscivorous birds and mammals; and given the widespread occurrence of PFOS in biota, including in remote areas; and given that PFOS precursors may contribute to the overall presence of PFOS in the environment, it is concluded that PFOS is likely, as a result of its long-range environmental transport, to lead to significant adverse human health and environmental effects, such that global action is warranted." PERFLUOROOCTANE SULFONATE RISK PROFILE

November 2006

"Based on the persistence, bioaccumulation, toxicity in mammals including humans and widespread occurrence in environmental compartments, it is concluded that PFOA, its salts and related compounds are likely, as a result of their long-range environmental transport, to lead to significant adverse human health and environmental effects such that global action is warranted."

PENTADECAFLUOROOCTANOIC ACID (PFOA, PERFLUOROOCTANOIC ACID), ITS SALTS AND PFOA-RELATED COMPOUNDS RISK PROFILE

October 2016

These are the findings of the POPs Review Committee; the United Nations' expert committee for the *Stockholm Convention on Persistent Organic Pollutants* 2001. [1]

Manufactured fluorinated compounds have been widely used in a variety of consumer goods from non-stick kitchenware to waterproof clothing and even cosmetics, as well as many industrial applications. Produced commercially since the 1950s, two of the most persistent; perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) have been shown to be toxic and bioaccumulative, posing a global contamination threat to the environment and all its inhabitants.

PFOS and PFOA have been found in the blood, urine and breast milk of Australians. Recent research suggests that PFOS concentrations at current population levels may already be causing adverse health impacts, in particular thyroid disease, endocrine impacts in women and immunotoxicity.[2, 3, 4, 5] As PFOS and PFOA do not break down, they are passed from one generation to the next via breast milk and *in utero*, and have in some cases demonstrated changes in gene expression at very low levels. It is possible that like lead and mercury, there may be no safe level of exposure to PFOS and /or PFOA. Due to their long half-life in human beings, there is an increasing risk over time that the exposure will cause adverse effects as both PFOA and PFOS are linked to an increased risk of cancer, endocrine disruption and reproductive harm.[6]

These chemicals are not manufactured in Australia but are found in imported products and in current stockpiles of old, but still used, fire-fighting foams. Currently, there are investigations into environmental contamination with perfluorinated compounds (PFCs)[7] at 18 priority defence sites around Australia affecting over 1200 households. These include defence bases in New South Wales, Queensland, South Australia, West Australia and Northern Territory. Environmental contamination with PFOS at and around the Fiskville Country Fire Authority's (CFA) training college in Victoria resulted in its permanent closure.

Urgent regulatory action is needed to ensure Australian citizens are protected from ongoing exposures to perfluorinated compounds both via consumer products and environmental releases. Special consideration must be given to ensuring contaminated sites are cleaned up and fire-fighter's and other relevant worker's health is monitored. The Australian government must immediately ratify the listing of PFOS on the Stockholm Convention on Persistent Organic Pollutant (POPs) and undertake an urgent recall of all old stocks of PFOS/PFOA contaminated fire-fighting foams.

2 PFOA and PFOS: the dangerous sister chemicals recognised globally

In 2015, the Madrid Statement on Poly- and Perfluoroalkyl Substances (PFASs) was signed by scientists and environmental health specialists from across the globe calling for urgent action of perfluorinated compounds *(PFCs).* [8]

In 2009, PFOS, the well known ingredient of 3M's Scotchguard products was listed on the United Nations' *Stockholm Convention on Persistent Organic Pollutants* 2001, a convention to eliminate some of the world's most dangerous chemicals.

PFOS is extremely persistent and does not break down. As a persistent organic pollutant (POP), it travels the world in water and air currents, contaminating ecosystems and their inhabitants. PFOA is infamous as the basis for the manufacture of Teflon, the polymer used in non-stick coatings yet in October 2015, the POPs Review Committee, concluded that PFOA met all criteria (eg toxicity, bioaccumulation, persistency and long range transport) for further evaluation as a POP; a decision that started its journey to listing on the convention and global elimination. On PFOA's adverse effects, in a consensus decision experts agreed there was *"epidemiological evidence*[9] *for kidney and testicular cancer, disruption of thyroid function and endocrine disruption in women."* [10] In addition, they concluded PFOA was highly persistent, and does not undergo any degradation under environmental conditions.

The Committee acknowledged that PFOA was detected at sites remote from any known point sources indicating that it undergoes long-range transport via ocean currents and via atmospheric transport of volatile precursors of PFOA. They confirmed that it biomagnifies in animals, threatening the food chain and noted that mothers excrete PFOA via breast milk, transferring PFOA to their infants. After giving birth and at the end of breast feeding PFOA then re-accumulates in maternal blood.

The next stage of assessment, the Risk Profile was completed in September 2016. PFOA related products that eventually breakdown to PFOA, e.g., fluorotelomer alchols (FTOHs) and fluoropolymers were identified as sources of PFOA with studies suggesting that some breakdown to up to 30% PFOA in the natural environment.[11] The Risk Profile concluded that '*based on its persistence, bioaccumulation, toxicity in mammals including humans and widespread occurrence in environmental compartments, that PFOA, its salts and related compounds were likely, to lead to significant adverse human health and environmental effects and that global action was warranted.'*

3 How toxic are perfluorinated compounds?

People and animals are exposed to PFCs via food, drinking water, direct contact with products and exposure to indoor and ambient air contaminated with PFCs. PFOS, PFOA, perfluorohexanesulfonate (PFHxS) are found in human blood, urine, breast-milk and babies umbilical cord blood. They remain in the human body for many years, accumulating primarily in the blood, kidneys and liver. Due to their long half-life in human beings (PFOS 5.4 years and PFOA 3.8 years approx.) there is an increasing risk over time that the exposure will cause adverse effects. The toxic effects of PFCs were evident by the late 1970s, based on studies with laboratory animals.[12] Internal documents from one manufacturer, Dupont[13] released through the US courts have shown that by 1989, the company was aware of elevated rates of certain cancers in workers, including kidney cancer and had known that exposed workers suffered more frequently from endocrine disorders. The company did not disclose the results of its in-house studies that had found birth defects among its workers' children and in 2005, were fined $16.5 million for failing to report the birth-defect findings and other data to the US EPA.

By May 2016, after many years of assessment, the United States Environmental Protection Authourity (USEPA) concluded:

***"For PFOS,** oral animal studies of short-term and subchronic duration are available in multiple species including monkeys, rats and mice. These studies report developmental effects (decreased body weight, survival, and increased serum glucose levels and insulin resistance in adult offspring), reproductive (mating behavior), liver toxicity (liver weight co-occurring with decreased cholesterol, hepatic steatosis), developmental neurotoxicity (altered spatial learning and memory), immune effects, and cancer (thyroid and liver). Overall, the toxicity studies available for PFOS demonstrate that the developing fetus is particularly sensitive to PFOS induced toxicity.*

***Human epidemiology data** report associations between PFOS exposure and high cholesterol, thyroid disease, immune suppression, and some reproductive and developmental parameters, including reduced fertility and fecundity. Although some human studies suggest an association with bladder, colon, and prostate cancer, the literature is inconsistent and some studies are confounded by failure to control for risk factors such as smoking."*[14]

***"For PFOA**, oral animal studies of short-term, subchronic, and chronic duration are available in multiple species including monkeys, rats and mice. These studies report developmental effects (survival, body weight changes, reduced ossification, delays in eye opening, altered puberty, and retarded mammary gland development), liver toxicity (hypertrophy, necrosis, and effects on the metabolism and deposition of dietary lipids), kidney toxicity (weight), immune effects, and cancer (liver, testicular, and pancreatic). Overall, the toxicity studies available for PFOA demonstrate that the developing fetus is particularly sensitive to PFOA-induced toxicity.*

***Human epidemiology data** report 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)."*[15]

In May 2016, after carefully examining the epidemiological literature, the German Human Biomonitoring Commission (HBM Commission) came to similar conclusions rating the detrimental effects in the following areas as well proven, relevant, and significantly associated with exposure to PFOA and/or PFOS:

- Fertility and pregnancy such as increased time to wanted pregnancy, destosis and gestational diabetes, reduced weight of newborns at birth
- Lipid metabolism
- Immunity after vaccination, immunological development
- Hormonal development, age at puberty / menarche
- Thyroid metabolism
- Onset of menopause. [16]

3.1 Exposure to Perfluorinated Mixtures

Mixtures of PFCs have been shown to interfere with the functioning of hormones. At least five PFCs have been shown to be endocrine disrupting compounds (EDCs), affecting sex hormones like the estrogen and androgen receptor. In some cases, the mixture effect of exposure to multiple PFCs is more than just additive, which emphasizes the importance of considering the combined action of PFCs when assessing health risks.[17] Unfortunately, other than PFOA and PFOS, there is little information on the toxicology and health impacts of the suite of fluorochemicals to which people are exposed.[18]

A US study analysed PFC levels and the menopausal status of 2,732 women between the ages of 20 and 65 and found that women with higher levels of PFCs had earlier menopause compared to women with the lowest levels. Women with the higher levels of PFOA and PFOS were also slightly more likely to have had a hysterectomy. [19] Higher serum PFOA and PFOS levels have also been found in patients with polycystic ovary syndrome.[20]

Other studies have demonstrated a relationship between prenatal exposure to PFCs and adiposity (obesity) in children born to women who lived downstream from a fluoropolymer manufacturing plant. Higher prenatal serum PFOA concentrations were associated with greater obesity at 8 years and a more rapid increase in the body mass index (BMI) between 2-8 years.[21] A study of 815 children from the National Health and Nutrition Examination Survey 1999–2008 found an association between serum PFOA and PFOS levels and dyslipidemia (abnormal amount of cholesterol and/or fat in the blood). Dyslipidemia in children is associated with accelerated disease of the arteries and earlier onset of cardiovascular disease. A significant association was found in adolescents, even at the lower "background" exposure levels of the US general population.[22]

Based on the data of 3,974 adults sampled in the US National Health and Nutrition Examination Survey (NHANES), researchers concluded that higher concentrations of serum PFOA and PFOS are associated with current thyroid disease in the general adult population.[23]

3.2 Reproductive and Developmental Toxicity

The developing fetus is particularly sensitive to both PFOS and PFOA induced toxicity. In animal studies, PFOS has caused reproductive and developmental impacts, including developmental neurotoxicity and immunotoxicity.[24] In animal studies, PFOA caused increased mortality in rat pups and as a developmental toxicant, prenatal exposure caused significant delays in mammary developmental in the female offspring.[25] In humans, there is an association between PFOS exposure and reduced fertility and in Europe, PFOA is classified as reproductive toxin, requiring to be labelled, *"May damage the unborn child"*. The USEPA review also concluded that

PFOA poses a risk for childbearing women; the estimated exposure range for humans, based on rat studies, having already overlapped with what the US EPA deem as unacceptable for toxic substances.[26]

A study released in 2016, tested 223 Taiwanese mothers and their infants for PFOA and other perfluorocarboxylic acids (PFCAs). Based on a range of development and growth factors measured at approximately 2, 5, 8, and 11 years, the study showed that prenatal exposure to long-chain PFCAs can interfere with fetal and childhood growth in girls, and childhood growth in boys.[27]

3.3 Threats to the Immune System

Both PFOA and PFOS have been shown to suppress immune responses in adult mice and exposed humans including changes in immune and inflammatory responses.[28] Elevated exposures to PFCs including PFOA and PFOS were associated with reduced immune response to routine childhood immunizations in children aged 5 and 7 years [29] and a reduction of the early immune response to booster vaccination in healthy adults.[30] In 2016, the U.S. National Institute of Environmental Health Sciences' National Toxicology Program (NTP) released their review of PFOS or PFOA immunotoxicity for humans. Based on 33 human studies, 93 animal studies, and 27 in vitro/mechanistic studies NTP concluded both PFOS and PFOA were immune hazards to humans. They based this conclusion on evidence of suppression of the antibody response and increased hypersensitivity, as well as additional evidence that is primarily from epidemiological studies that PFOA reduced infectious disease resistance and increased autoimmune disease. [31]

3.4 Carcinogenicity

In animal studies, PFOS has caused testicular and pancreatic tumours. Some human studies have also linked PFOS to cancer of the bladder, colon and prostate while human population studies report associations between PFOA exposure and testicular and kidney cancer. Following the class action between DuPont (a manufacturer of Teflon which contains PFOA) and US residents affected by Dupont's contamination, the jointly established *C8 Science Panel* concluded that PFOA can cause kidney cancer and testicular cancer.[32] The association between PFOA exposure and increased risk of testicular and/or kidney cancers is supported by a number of researchers. [33]

PFOA has also been shown to affect the expression of genes[34,35], while other researchers have shown that PFOA has 'genotoxic' effects[36] on human liver cells.[37] Genotoxic chemicals damage the genetic information within a cell, which can cause mutations and lead to cancer. These chemicals may have no safe level of exposure. The US EPA's expert committee recommended that PFOA be considered 'likely to be carcinogenic to humans,[38] while the International Agency for Research on Cancer (IARC) has classified PFOA as "possibly carcinogenic to humans" (Group 2B), based on limited evidence in humans that it can cause testicular and kidney cancer.

In October 2015, an Ohio woman was awarded $1.6 million in compensation after a jury ruled that PFOA from US DuPont plant contaminated drinking water and contributed to the development of kidney cancer.[39] In 2016 DuPont was also found responsible for a man's testicular cancer and was ordered to pay $5.1 million in associated damages.[40]

While evidence of adverse health effects of both PFOS and PFOA is significant and increasing, exposure to all PFCs needs to be assessed. More research on the toxicity, bioaccumulation and exposure pathways for the other approximately one thousand PFAS is urgently needed. [41]

4 Perfluorinated compounds in the Australian population

Most Australians have accumulated PFCs including PFOS and PFOA in their bodies. These can remain for many years, accumulating primarily in the blood, kidneys and liver. When compared to monitoring results from elsewhere the world, concentrations of PFOS and PFOA in the Australian population in 2010-2011 were similar or higher than overseas counterparts. Concentrations in Australian women of child-bearing age were almost twice that found in pregnant women from Germany while PFOS and PFOA concentrations are reported to be 1.5 and twice those found in adults from the USA.[42]

Nevertheless, PFOS and PFOA concentrations have been decreasing in Australian adults [43], most likely due to the decline in global use of the chemicals since 2002. Average Australian PFOS serum levels in 2008/09 ranged from 5.3–19.2 ng/ml, declining to 4.4–17.4 ng/ml in 2010/11. PFOA was the next highest concentration at 2.8–7.3 ng/ml (2008/09) and 3.1–6.5 ng/ml (2010/11). All other measured PFCs were detected at concentrations <1 ng/ml with the exception of perfluorohexane sulfonate, which in 2010-11 was detected at 1.4–5.4 ng/ml. [44]

In Queensland, of the 75 Oakey residents tested, the level of PFOS and PFOA in their blood was found to be, on average, three times higher than other Australians and in some cases as high as 18 times above average. Oakey male residents were also reported [45] to be 17 times above the German 'safe' level of 5 nanograms per millilitre (ng/mL).

4.1 Perfluorinated compounds in Australian firefighters

A 2014 study of 149 Queensland firefighters detected multiple PFCs in their serum. The three most prevalent and detected in all samples were PFOS, perfluorohexanesulfonic acid (PFHxS) and PFOA. Their serum levels of PFOS were approximately six to ten times higher than those found in the general population in Australia. The median/mean level in firefighters was 66/74 ng/mL compared to 12 ng/mL (mean) and 6.8 (median) ng/mL in the general population in Australia. The serum levels of other PFCs like PFHxS in firefighters were approximately 10 to 15 times higher compared to the general population levels in Australia. Even ten years after the phase out of 3M AFFF Industrial Fire Fighting Foam, PFOS serum levels remained above 100 ng/mL and 200 ng/mL in 27% and 3% of the participating firefighters, respectively.[46]

5 Is there a 'safe' level for perfluorinated compounds?

In 2006, the Biomonitoring Commission of the German Federal Environmental Agency established preliminary reference values for PFOA and PFOS in plasma of children and adults. They recommended a maximum permissible serum level for PFOA of ten micrograms per litre (10 µg/l) for all groups.[1] For PFOS, they recommended 10 µg/l for children at school beginner age, 15 µg/l for adult females and 25 µg/l for adult males.[47,48] In May 2016, Germany's Commission on Human Biomonitoring significantly reduced these to five nanograms per millilitre (5ng/mL) for PFOS and 2 ng PFOA/mL blood plasma.[49]

[1] Note : Nanogram (ng) / millilitre (mL) = parts per billion (ppb)
Micrograms (µg) / litre (l) = parts per billion
Milligrams (mg) / litre = parts per million (ppm)

Population studies suggests that PFOS concentrations at current population levels may be causing adverse health impacts.[50, 51] A recent study [52] also demonstrated that a serum level of PFOS of 14.1 ng/mL was associated with impacts on DNA methylation, suggesting that PFOS may be epigenetically active. Epigenetics refers to heritable changes in gene expression (active versus inactive genes) that does not involve changes to the underlying DNA sequence.[53] Methylation modifies the function of the DNA, typically acting to suppress gene transcription, which in turn may be associated with the development of cancer.[54]

In May 2016, the U.S. EPA's Office of Water issued lifetime drinking water health advisory (HA) for both PFOS and PFOA of 0.07 µg/l, individually or combined.[55] While a significant reduction on the previous advisory level of 0.4ppb, it is still criticised by some researchers as not protective enough of human health, particularly infant and neonatal exposure. The U.S. states of New Jersey and Vermont set their own advisory level for PFOA in drinking water of 0.04 ppb and 0.02 ppb respectively.

Researchers, Grandjean and Clapp recommend a PFOA limit in drinking water of 0.001 ppb based on a serum concentration of 0.1 ng/mL.[56] They argue that the experimental studies the regulatory agencies have relied upon so far have been superseded with more recent studies. Using the data from a recent study of immunotoxicity in children, they calculated a reference dose serum concentration of about or below 0.1 ng/mL.[57] As PFOS and PFOA do not break down, are passed from one generation to the next via breast milk and *in utero*, and have in some cases demonstrated changes in gene expression at very low levels, it is possible that like lead and mercury, there may be no safe level of exposure to PFOS and /or PFOA.

5.1 Australian Interim Health Reference Values: Inadequate and Outdated

An Australian government 'invitation only' workshop tasked with reviewing overseas PFC/ PFAS standards and draft Australian human health toxicity reference values for PFOS and PFOA was held in early 2016. Rather than following the lead of the US EPA and the German Human Biomonitoring Commission, the assembled 'experts' (including consultants working for the Defence Department, the polluter) decided to adopt the out-of-date 2008 European Food Safety Authority's (EFSA) derivation of Tolerable Daily Intake (TDI) values for PFOS and PFOA as appropriate interim national guidance for use in contaminated site investigations in Australia. [58]

EFSA TDI are 0.15 microgram per kilogram per day (µg/kg/day)[2] for PFOS and PFOA at 1.5 µg/kg/day compared to the US EPA set values of 0.02 ug/kg/day (USEPA Reference dose) for both PFOS and PFOA based on cancer and non cancer effects including developmental toxicity. The drinking water guidelines recommended by Australian experts were 0.5 µg/l for PFOS and PFHxS and 5 µg/l for PFOA. Combined these are 78 times higher then those set out by the USEPA of 0.07µg/l combined.

Unlike their US and German counterparts, Australian government claim *"that there is currently no consistent evidence that exposure to PFOS or PFOA causes adverse human health outcomes in pregnant women or their babies ..and that in humans, there is no conclusive evidence that PFASs cause any specific illnesses, including cancer."* [59] This statement is in direct opposition to the findings of the UN POPs Review committee. The Committee unanimously agreed *"adverse health effects such as elevated cholesterol levels, altered reproductive/developmental effects, endocrine disruption, impaired neurodevelopment, as well as increased risk of cancer associated with PFOA exposure in humans …and that scientific data have demonstrated PFOA-mediated immunotoxicity, primarily suppression of antibody response, in humans."* [60]

[2] microgram per kilogram per day (µg/kg/day) = parts per billion per day

6 Perfluorinated compounds in the environment

PFCs are released into the air and water from waste sites, manufacturing facilities, sewerage treatment works and fire-fighting operations. They also migrate out of consumer products like all-weather clothing, carpets and camping gear finding their way into household air and dust, soil, ground and surface water and food. Additionally many PFOA-related substances (eg fluoropolymers) can degrade to PFOA under certain conditions. Researchers in 2015 [61] concluded that emissions from consumer products imported from China were responsible for 1.5% of PFOA discharges to wastewater.

PFOS and PFOA have shown no evidence of degradation in the environment. [62, 63] These extremely persistent PFCs travel the globe via air and water currents, as well as in wildlife. In the air, volatile PFCs (eg polyfluorinated fluorotelomer alcohol (FTOH) and sulfonates) can travel thousands of kilometres [64] while others are carried in particulate matter, which eventually washes out being deposited in rain and snow. PFCs contaminate every ecosystem in the world from the remote Arctic to the tropics. In recent sampling of snow in remote locations and water from mountain lakes, PFCs were present in nearly all the samples. [65] These included short chain PFCs, which industry is increasingly using, arguing that they are less harmful than long chain PFCs like PFOS. [66] Unfortunately, there is little information on the toxic effects of short chain PFCs but concern over their detection in remote places is growing. There are some indications that some of the new PFCs are as hazardous as their predecessors. PFCs in the environment are taken up by wildlife in their food and water; bioaccumulating in mammals, birds and fish with concentrations increasing further up the food chain.

6.1 PFCs in Australian Water

PFCs have been found in drinking water collected from 34 locations including capital cities and regional centres in Australia,. PFOS and PFOA were the most commonly detected; 49% and 44% of all samples respectively. While the maximum concentration in any sample was PFOS at 16 ng /l, the second highest maximums were for PFHxS and PFOA measured at 13 and 9.7 ng/l.[67] Discharges from wastewater treatment plants (WWTPs) can be contaminated with PFCs, representing a hazard to the aquatic environment. In Australian water reclamation and recycling plants, PFOS, PFOA, PFHxS and perfluorohexanoic acid (PFHxA) were the most frequently detected PFCs. Only those recycling plants using reverse osmosis (RO) technology were shown to reduce PFC concentrations to below detection and reporting limits (0.4–1.5 ng/l). [68] In an Australian study of leachate from landfills, evaporation and aeration ponds, PFOA was found in every sample. (0.5-0.88ug/l) with 6 samples returning measurements of PFOA greater than 0.5ug/l [69].

6.2 Environmental Contamination with PFCs

The use of PFOS and PFOA in fire-fighting foams has been linked to environmental contamination of groundwater in Germany, Sweden, the US and Australia. In 2007 broad scale contamination of groundwater with PFOS from firefighting foams was reported in in Düsseldorf, Germany. In Sauerland, Germany in 2006, contaminated biosolids applied to land resulted in PFOS contamination of water, pasture, forage and animal products. In 2004, DuPont settled a class-action covering 80,000 people affected by PFOA contamination of their drinking water. In Italy in 2013, surface, groundwater and tap water were found to be contaminated with PFOS, downstream from a PFC/PFAS production plant. As PFOS and PFOA do not degrade, once released to aquifers they are transported along with the groundwater, [70] with the concentrations decreasing only due to diffusion and dispersion.

DuPont is reported to have released approximately 1,136,364 kg of PFOA into the air and water around its West Virginia plant between 1951 and 2003.[71] The U.S. military is currently assessing 664 sites where the military has conducted fire or crash training using PFC based fire fighting foams. As PFOS and PFOA do not degrade, once released to aquifers they are transported along with the groundwater, [72] with the concentrations decreasing only due to diffusion and dispersion.

6.3 Contamination from Fire Fighting Foam in Australia

PFOS is the active ingredient (fluorosurfactant) in Aqueous Film Forming foams (AFFF™) and Alcohol-Type Concentrate (ATC™)) produced by 3M. In 2007, industry reported [73] approximately 160,000 litres of class B fire fighting foam products containing PFOS held in stock, roughly translating to a stockpile of 7.6 tonnes of PFOS. PFOS fire-fighting foam products are designated for emergency use only yet there is evidence that some fire authorities are still using them.[74] PFOA has also been used to produce firefighting foams.

High levels of PFC contamination at the Fiskville Country Fire Authority's (CFA) training college in Victoria resulted in its closure. The results of 550 tests showed PFOS contamination of the college's water supply and high levels of the toxic chemical in the fire training area and others areas at the site. PFOS levels in water were reported to be 50 ug/L above international guidelines. A farmer adjacent to the site was forced to cease selling animal produce after PFOS was found in the soil and sheep. Concerning levels of PFOS were found in the blood of the farmer and his children.[75]

Some of Australia's airports, where AFFF has been used are being investigated. PFC contamination has been reported at the Gold Coast Airport and Cairns Airport in Queensland,[76] as well as Sydney Airport and its surrounds.

6.4 Contamination at Australian Defence Bases

Currently, there are ongoing investigations into PFC environmental contamination at 18 priority defence sites around Australia affecting over 1200 households. These include the Williamstown Royal Australian Air Force Base (RAAF) in New South Wales, the Army Aviation Centre near the rural town of Oakey in Queensland [77] and other defence bases in South Australia, West Australian, Northern Territory and Queensland.

In Queensland from 1970 to 2005, the Australian Defence Force (ADF) regularly conducted fire-fighting drills at the Oakey Airbase using PFC based fire-fighting foams. Following an environmental audit of the area in 2010, further tests were conducted in 2014, which confirmed that the contamination had spread beyond the base into water bores used by local land holders and into Oakey Creek. PFOS and PFOA contaminated groundwater has been detected several kilometres to the west and southwest of the base. Residents were told that 42 out of 112 bores tested by Defence in the Oakey area, had levels that exceeded the health advisory drinking limit. The ADF has advised local residents not to drink their bore water or creek water. Queensland Health is understood to be advising residents not to eat eggs or drink milk from animals raised within the contamination zone, as well as avoiding fish caught in nearby creeks. Blood tests of Oakey residents have returned very high PFC levels well above the national average. The ADF has not provided any compensation for loss of income or other losses due to the contamination to Oakey business owners/operators. [78]

In NSW, the Australian Defence Force informed the NSW government that they had found high concentrations of PFOS and other PFCs at their Williamtown RAAF site.[79] The chemicals contaminate the, base and adjacent land, as well as the associated groundwater, which is a serious risk to the Tomago sands, an important drinking water catchment site situated close to the Williamtown base. Surface water samples have also been found to contain PFOS and tests on fish from the local creeks found high levels of PFOS resulting in a ban on commercial fishing.[80]

The Senate Inquiry into firefighting foam contamination at the Williamtown RAAF Base made 8 recommendations including funding a program of blood tests for residents in the investigation area on an annual basis, however the Australian Government[81] rejected this, stating that the Environmental Health Standing Committee (enHealth), comprising of representatives of state and commonwealth health departments, *"advises against blood testing of individuals for PFOS and PFOA."*

The Department of Defence (DOD) has initiated a review of DOD sites around the country based on their use of AFFF and environmental factors such as groundwater and hydrogeological conditions. As a result RAAF Base Pearce in West Australia, RAAF Base East Sale in Victoria and HMAS Albatross in NSW are now prioritised for PFOS/PFOA contamination investigations beginning in 2016. RAAF Pearce has already been the subject of trials to treat PFOS/PFOA contaminated wastewater in collaboration with the Cooperative Research Centre for Contamination Assessment and Remediation of the Environment (CRC CARE) since 2011. The remediation program is no longer operational and contamination remains.

7 The Alternatives

While an immediate government call in and collection of all PFOA /PFOS based fire fighting foams is well overdue, an urgent review of what has replaced them is also needed. The POPs Review Committee in their guidance on alternatives to PFOS and related chemicals[82], noted that there is little independent and reliable information available on the toxicology and ecotoxicology, persistence or degradation products of the fluorinated alternatives to PFOS and PFOA based products.

Much of the information is claimed as commercial secrets, including the specific identity of active ingredients of AFFF. Yet a range of replacement products based on short-chain PFAS[83] and various fluorinated telomers have been identified including perfluorohexane ethyl sulfonyl betaine, perfluorobutane sulfonic acid (PFBS) and PFHxS and /or Perfluorohexanoic acid (PFHxA) based polymers and reportedly, Ammonium 2333-Tetrafluoro-2 Propanoate.

The potential degradation products of some of the fluorinated replacements are terminal degradation products like PFHxA and PFHxS. These extremely persistent chemicals are increasingly being detected in the environment with high concentrations of PFHxA being found in several European rivers. PFHxS has been found in seawater, birds, animals, and humans in the Arctic. Measurement in fire fighters show equal levels of PFHxS and PFOS, which suggest the use of PFHxS in fire fighting foam.[84] PFHxS has been detected in the Australian population as well as increasing levels in Australian firefighters. A study of 300 children in the US from birth to 12 years of age showed that PFHxS was present in >92% with increasing concentrations by age.

There is a growing evidence of the toxic effects of PFHxS in humans and animal studies including affects on the thyroid hormone (TH) pathway and inhibiting intercellular communication. A recent study of attention deficit / hyperactivity disorder (ADHD) in children, showed that increasing PFHxS levels were associated with increasing prevalence of ADHD (adjusted odds ratio of 1.59). PFHxS is more liver toxic than either PFBS and PFOS.[85] PFHxS is currently under evaluation as a PBT (persistent, bioaccumulative, toxic) substance under REACH in Europe.[86]

Substitution with substances based on perfluorobutane sulfonate or perfluorobutane sulfonic acid (PFBS) may be responsible for the increasing detection of PFBS in marine mammals like dolphins.[87] PFBS is the principal terminal degradation product of PFBS-based products and is extremely persistent. It has been widely detected in water as well as municipal landfill leachates, in indoor dust from homes and offices and has been found in the Arctic. In a Germany study, PFBS was found in 33% of the children. It has demonstrated developmental neurotoxicity[88] and like PFOS and PFOA can affect the production of estrogen in placental cells.

For 3M's alternative Dodecafluoro-2-methylpentan-3-one, the information gaps were major although a variety of liver effects were noted and its probable persistency and volatility is of concern. [89] With little toxicology or ecotoxicology data available on the alternatives and evidence of persistency, bioaccumulation and long range transport we may be at risk of repeating past mistakes, replacing one POPs with another potential POP.

8 International Regulatory Responses

Australia has not banned PFOS despite OECD (Organisation for Economic Cooperation and Development) concluding in 2000 that the persistency of PFOS in the environment, its toxicity and bioaccumulation all indicated cause for concern for both the environment and human health.[90] In 2002, under pressure from the USEPA, the major manufacturer of PFOS and its precursors, 3M, ceased production. The USEPA has severely restricted the use of PFOS and other perfluoroalkyl substances (PFASs) to uses where no safer alternative is available.[91]

Canada has also prohibited the manufacture, use, sale, offer for sale and import of PFOS and related substances and in 2006, while the European Union adopted a resolution restricting the marketing and use of PFOS and related substances.[92] In 2009, PFOS was listed on Annex B of the Stockholm Convention. This permitted some limited ongoing uses however, the POPs Review Committee continues to assess and recommend phase out of some of the remaining uses. In May 2015, the Stockholm Convention's conference of parties removed a further six of the previously permitted uses. Despite being a signatory to the Convention, Australia has not yet ratified the listing of PFOS.

In 2006, the US EPA established the PFOA product stewardship program, a voluntary initiative to the phase-out the manufacture and use of PFOA by 2015. It includes eight major manufacturers of PFOA, its salts and PFOA-related compounds (Arkema, Asahi, BASF, Clariant, Daikin, 3M/Dyneon, Dupont, Solvay Solexis) but did not include the main producers of PFOA in China. In 2006, the Government of Canada published a Notice of Action Plan for the assessment and management of perfluorocarboxylic acids and their precursors and included measures to prevent the introduction of new substances into Canada that would contribute to the level of PFCAs and address sources of PFCAs already in Canadian commerce. A voluntary Environmental Performance Agreement was signed in 2010 with signatories agreeing to eliminate PFOA and long-chain perfluorocarboxylic acids by 2015.

In 2013, Europe identified both PFOA and the ammonium salt of PFOA (ammonium perfluorooctanoic acid; APFO) as Substances of Very High Concern (SVHC) based on their persistent, bioaccumulative and toxic properites and included them in the REACH-Candidate List. This means that articles may not contain more than 0.1% of PFOA. In 2014, the Norwegian Environment Agency effectively banned the use of PFOA in consumer products and textiles. In October 2015, PFOA was nominated for listing on the United Nation's *Stockholm Convention on Persistent Organic Pollutants*.

9 Conclusions

When considering information regarding contamination with toxic chemicals such as PFOS and PFOA it needs to be acknowledged that governments and regulatory bodies can be influenced by their own and other vested interest. Remediating the Australian wide PFC contamination will be expensive and take time. There are other issues that may result and assist in government inaction such as scientific studies can be inconsistent, risk assessment choices conflicting and complete causation may be hard to prove (ie tobacco, asbestos, climate chaos). The recognition of endocrine disruption has also resulted in a paradigm shift in toxicology towards the acceptance of non-linear dose responses. Unfortunately not all toxicologist have caught up or agree to these latest developments. Additionally regulators have only slowly began to recognize that individual safety levels must include multiplier effects and that timing of exposure (window of vulnerability) is crucial in assessing the potential effect on an individual.

Nonetheless urgent regulatory action is needed to ensure Australian citizens are protected from ongoing exposures to perfluorinated compounds in consumer products, in food and drinking water. This requires:

- Priority phase out of perfluorinated compounds in consumer products and immediate cessation of the import and use of PFOS and PFOA and those products that degrade to PFOS and PFOA.

- During the phase out period perfluorinated compounds should be labelled to inform consumers, users and waste managers.

- Particular attention should be given to the waste phase of perfluorinated compounds with national regulators ensuring access to non-combustion destruction technologies and sound waste management practices for PFC treatment.

- Investigation and remediation of environmental contamination is long overdue and regulatory agencies should ensure contaminated sites are cleaned up and fair and equitable compensation processes for affected communities are initiated.

- Special consideration must be given to the health and well-being of firefighter's and other affected workers. An immediate recall of PFOS based fire-fighting foams should be undertaken and an inventory of all PFC based foams commenced.

- A review of the current replacement fire-fighting foams based on short chain fluorotelomer-based surfactants with a commitment to support fluorine free foams.

- Australia must immediately ratify the listing of PFOS.

10 Endnotes

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