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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

1 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
Adopted by the Persistent Organic Pollutants Review Committee at its second meeting 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 DRAFT RISK PROFILE March 2016

Manufactured fluorinated chemicals are 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 groups of perfluorinated compounds (PFCs), the perfluoroalkyl sulfonates (PFSAs) and the perfluorocarboxylic acids (PFCAs) have raised alarm bells on a global scale. In particular, perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) have been shown to be toxic and very persistent, posing a global contamination problem.

Australian citizens have both PFOS and PFOA in their blood, urine and breast milk. 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.[1, 2, 3, 4] 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.

Due to their long half-life in human beings, there is an increasing risk over time that the exposure will cause adverse effects. Both PFOA and PFOS are linked to an increased risk of cancer, endocrine disruption and reproductive harm[5].

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 PFC environmental contamination at 18 priority defence sites around Australia affecting 1200 households. These include the Williamstown Air Base in New South Wales, the Army Aviation Centre near the rural town of Oakey in Queensland and at other Royal Australian Air Force Base (RAAF) bases in South Australia, West Australian and Queensland. As well, environmental contamination at and around the Fiskville Country Fire Authority's (CFA) training college in Victoria has resulted in its 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.

The 2015 *Madrid Statement* on Poly- and Perfluoroalkyl Substances (PFASs) signed by scientists and environmental health specialists from across the globe has called for urgent action of PFCs. [6]

2 PFOA and PFOS: the dangerous sister chemicals

In 2009, PFOS, the well known ingredient of 3M's Scotchguard products, after a 3 year international assessment, was listed on the United Nation's *Stockholm Convention on Persistent Organic Pollutants* (POPs), a convention to eliminate some of the world's most dangerous chemicals. PFOS is extremely persistent and does not break down. It travels the world in water and air currents, contaminating ecosystems and their inhabitants. In Australia in 2007, there was 160,000 litres of PFOS Fire fighting foam in stock.[7] Information on its management or use is simply not available.

PFOA is infamous as the basis for the manufacture of Teflon, the polymer used in non-stick coatings. PFOA is a very toxic chemical, which has been nominated for listing on the United Nation's *Stockholm Convention on Persistent Organic Pollutants* (POPs), a convention that seeks to eliminate some of the world's most dangerous chemicals.

At the October 2015 meeting of the UN POP Review Committee, the expert group of the Stockholm Convention, committee members concluded that PFOA met all criteria (eg toxicity, bioaccumulation, persistency and long range transport) for further evaluation as a POP; a decision that starts its journey to global elimination. On PFOA's adverse effects, in a consensus decision, the experts agreed there was "*epidemiological evidence[8] for kidney and testicular cancer, disruption of thyroid function and endocrine disruption in women."* [9] In addition, the committee concluded PFOA was highly persistent, and does not undergo any degradation under environmental conditions.

The Committee noted that PFOA was detected at sites remote from 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, posing a threat to the mother itself.

For the next stage of the Stockholm Convention assessment, the Risk Profile, the committee has called for information on the range of PFOA related products that will eventually breakdown to PFOA. For example, fluorotelomer alcohols (FTOHs) have been identified as potential sources of PFOA with studies suggesting that some convert to up to 30% PFOA in the natural environment.[10]

PFOS and PFOA are not manufactured in Australia but are found in many imported products and in current stockpiles of old, but still used, fire-fighting foams. The use of these fire-fighting foams has led to extensive contamination of groundwater and soil with PFOS and PFOA affecting rural and regional communities across Australia. Currently there are investigations into environmental contamination with PFOS and PFOA at a range of defence bases and fire fighting training grounds around Australia.

3 How toxic are perfluorinated compounds?

People and animals are exposed to PFCs via food, drinking water, direct contact with products and exposure from indoor and ambient air. PFCs like PFOS, PFOA, perfluorohexanesulfonate (PFHxS) are found in human blood, urine, breast-milk and babies umbilical cord blood. PFCs remain in the human body for many years, accumulating primarily in the blood, kidneys and liver and 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 already evident by the late 1970s, based on studies with laboratory animals. [11]

By May 2016, the US EPA 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."[12]

"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)."[13]

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 additive, which emphasizes the importance of considering the combined action of PFCs when assessing health risks.[14] Unfortunately, other than PFOA and PFOS, there is little information on the toxicology and health impacts of the suite of fluorochemicals people are exposed to.[15]

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 higher levels of PFOA and PFOS were also slightly more likely to have had a hysterectomy. [16]

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.[17] 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.[18]

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.[19]

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.[20] In humans, there is an association between PFOS exposure and reduced fertility. 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 of mice.[21] In Europe, PFOA is classified as reproductive toxin and is required to be labelled, "May damage the unborn child". The US EPA review 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.[22]

A study released in 2016, tested 223 Taiwanese mothers and their term infants for PFOA and 4 long-chain Perfluorocarboxylic Acids (PFCAs, ng/mL) in third trimester maternal serum; infant weight (kg), length and head circumference (cm) at birth; and childhood weight and height at approximately 2, 5, 8, and 11 years of age were measured. The study showed that prenatal exposure to long-chain PFCAs can interfere with fetal and childhood growth in girls, and childhood growth in boys.[23]

3.3 Immunotoxicity

Both PFOA and PFOS have been shown to suppress immune responses in adult mice and exposed humans. Elevated levels of PFOA and PFOS have also been associated with changes in immune and inflammatory responses.[24] 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.[25] A reduction of the early immune response to booster vaccination in healthy adults has also been observed supporting the previous findings of PFCs immunosuppression in humans.[26] Mounting evidence suggests immune suppression occurs at serum concentrations below, within, or just above the reported range for humans and wildlife, which is a potential risk for altered disease resistance. The risk of immune effects for humans and wildlife may be even more significant when bioaccumulation and exposure to multiple PFCs from multiple sources are considered.

3.4 Carcinogenicity

In animals studies, PFOS has caused testicular and pancreatic tumours. Some human studies have linked PFOS to cancer of the bladder, colon and prostate while human population studies report associations between PFOA exposure and testicular and kidney cancer. PFOA has been shown to affect the expression of genes[27,28], while other researchers have demonstrated that PFOA has 'genotoxic' effects[29] on human liver cells.[30] 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,[31] 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.

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, testicular cancer, ulcerative colitis, thyroid disease, pregnancy-induced hypertension and medically diagnosed high cholesterol in humans.[32]

While evidence of adverse health effects of both PFOS and PFOA is significant and increasing, to assess the health risks from environmental contamination with PFCs, 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. [33]

4 Perfluorinated compounds in the Australian population

Most Australians have accumulated PFCs including PFOS and PFOA in their bodies. These can remain there 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 are similar or higher than our overseas counterparts. Concentrations in Australian women of child-bearing age are almost twice that found in pregnant women from Germany while PFOS and PFOA concentrations are 1.5 and twice those found in adults from the USA.[34]

PFOS and PFOA concentrations have been decreasing in Australian adults[35], most likely due to the decline in global use of the chemicals since 2002. PFOS serum levels in 2008/09 ranged from 5.3–19.2 ng/ml and declined 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. [36]

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 to be 17 times above the German 'safe' level of 5 nanograms per millilitre (ng/mL). [37]

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.[38]

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 10 µg/l for all groups[39]. 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.[40,41] It is reported that in May 2016, Germany's Commission on Human Biomonitoring significantly reduced these to a PFOS 'risk-free' blood level of five nanograms per millilitre (ng/mL).[42]

Population studies suggests that PFOS concentrations at current population levels may be causing adverse health impacts.[43, 44] A recent study[45] demonstrated that the mean serum levels of PFOS of 14.1 ng/mL was associated with impacts on DNA methylation, suggesting that PFOS may be epigenetically active.[46] Methylation modifies the function of the DNA, typically acting to suppress gene transcription, which in turn may be associated with the development of cancer.[47]

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 micrograms per litre (µg/L), individually or combined. [48]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 2015 recommend a PFOA limit in drinking water of 0.001 ppb (based on a serum concentration of 0.1 ng/mL).[49] 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 and assuming a linear dose-dependence of the effects, the Benchmark Dose Level (BMDL) is calculated to be approximately 1.3 ng/mL for PFOS and 0.3 ng/mL for PFOA in terms of the serum concentration. Applying an uncertainty factor of ten to take into account individual susceptibility, the BMDLs would therefore result in a reference dose serum concentration of about or below 0.1 ng/mL.[50]

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.

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 migrate out of consumer products such as all-weather clothing, carpets and camping gear into the air, household dust, food, soil, as well as ground and surface water. PFOS and PFOA have shown no evidence of degradation in the environment. [51, 52] Additionally many PFOA-related substances (e.g. fluoropolymers) can degrade to PFOA under environmentally relevant conditions. The issue of human exposure via consumer products was examined by researchers in 2015, [53] who concluded that emissions from consumer products imported from China were responsible for 1.5% of PFOA discharges to wastewater.

PFCs are extremely persistent in the environment and travel the globe via air and water currents. In the air, volatile PFCs (eg polyfluorinated fluorotelomer alcohol (FTOH) and sulfonates) are transported thousands of kilometres [54] while others are carried by suspended particulate matter, which is eventually washed out and deposited in rain and snow. PFCs now 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.[55] These data include short chain PFCs, which industry is increasingly using, arguing that they are less harmful than long chain PFCs like PFOS.[56] Unfortunately, like many of the PFCs, there is little information on toxic effects of short chain PFCs, however, their detection in remote places is of concern. There are 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.

In Australia, PFCs have been found in drinking water collected from 34 locations including capital cities and regional centres. PFOS and PFOA were the most commonly detected; 49% and 44% of all samples respectively. While the maximum concentration in any sample was for PFOS with a concentration of 16 ng /l, the second highest maximums were for PFHxS and PFOA measured at 13 and 9.7 ng/l.[57]

Discharges from wastewater treatment plants (WWTPs) are point sources for PFCs to the aquatic environment. In Australian water reclamation and recycling plants PFOS, PFOA, perfluorohexanesulfonate (PFHxS) and perfluorohexanoic acid (PFHxA) are the most frequently detected PFCs and only those recycling plants using reverse osmosis (RO) technology have been shown to reduce PFC concentrations to below detection and reporting limits (0.4–1.5 ng/l). [58] 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 [59].

6.1 Environmental PFC Contamination

The use of PFCs, particularly in fire-fighting foams has been linked to environmental contamination of groundwater in Germany, Sweden, the US [60] and Australia. In 2006 in Sauerland, Germany PFOS contamination of water, pasture, forage and animal products occurred from industrially contaminated biosolids applied to land, while in 2007 there was broad scale contamination of groundwater with PFOS from firefighting foams in Düsseldorf, Germany. In 2013 in Italy, surface, groundwater and tap water were found to be contaminated with PFOS, downstream from a PFAS production plant. Since biodegradation and adsorption of PFOS and PFOA are not known to occur in the aquifers and soils, these are transported at nearly the same rate as groundwater, [61] with the concentrations decreasing only with distance from the source due to diffusion and dispersion.

In the USA in October 2015, an Ohio woman was awarded $1.6 million in compensation after a jury ruled that PFOA from US company DuPont plant contaminated drinking water and contributed to her development of kidney cancer.[62] 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.[63] This case was just the first of many against Dupont, who is facing litigation from an additional 3,500 residents near its plant in West Virginia. The U.S. military is currently assessing 664 sites where the military has conducted fire or crash training using PFC based fire fighting foams.

6.2 Contamination from Fire Fighting Foam

PFOS is the active ingredient (fluorosurfactant) in Aqueous Film Forming (AFFF™) and Alcohol-Type Concentrate (ATC™)) produced by 3M. In 2007, Australian industry reported [64] approximately 160,000 litres of class B fire fighting foam products containing PFOS held in stock, translating roughly 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.[65]

Currently, there are investigations into environmental contamination with PFOS and PFOA at the Fiskville Country Fire Authority's (CFA) training college in Victoria. In 2015, the CFA training base was shut down permanently after tests showed PFOS contamination of the base's water supply. Tests have also showed high levels of the toxic chemical in the fire training area and others areas at the site. The results of 550 tests showed PFOS levels in water as high as 50 micrograms per litre 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. High levels were also found in the farmer's blood and that of his children. The Victorian government inquiry concluded *"That epidemiological evidence*

suggests that the contamination at Fiskville is likely to have caused cancer and other illnesses." [66]

Fire fighting foams are reported to have contaminated the Gold Coast Airport in Queensland.

6.3 Contamination at Australian Defence Bases

Currently, there are investigations into PFC environmental contamination at 18 priority defence sites around Australia affecting at least 1200 households. These include the Williamstown Air Base in New South Wales, the Army Aviation Centre near the rural town of Oakey in Queensland, Royal Australian Air Force Base (RAAF) base at Edinburgh in South Australia, RAAF Bases in Townsville and Amberley, near Ipswich and at other RAAF bases in West Australia as well as in Darwin and Tindal, and Robertson Barracks in Northern Territory.

In NSW, the Australian Defence Force has informed the NSW government that the Williamtown site contains high concentrations of PFOS and other PFCs.[67] The chemicals contaminate not only the RAAF site but also the groundwater under the site and adjacent land and may represent a serious risk to Tomago sands, an important drinking water catchment site situated close to the Williamtown base. Surface water samples have also been found to contain PFOS while ground water and fish from the local creeks were tested for PFOS and found to contain high levels resulting in an indefinite ban on commercial fishing.[68]

The 'Inquiry into firefighting foam contamination Part A - RAAF Base Williamtown' have made 8 recommendations including for Defence to arrange and fund a program of blood tests for residents in the investigation area on an annual basis. The Australian Government at first[69] 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."* However, the 2016 Federal Elections saw both the government and opposition commit to voluntary blood testing for affected residents.

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 have been told that of the 112 bores tested by Defence in the Oakey area, 42 bores had levels, which 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 estimated to be more than 40 times the national average. The ADF has not provided compensation for loss of income or other losses due to the contamination to Oakey business owners/operators. [70]

The Department of Defence (DOD) review of DOD sites based on the known prevalence of the use of Aqueous Film Forming Foam and environmental factors such as groundwater use and hydrogeological conditions saw the RAAF Base Pearce in West Australia, RAAF Base East Sale in Victoria and HMAS Albatross in NSW prioritised for PFOS/PFOA contamination investigations beginning in March 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. It is expected that significant levels of PFOS and PFOA will be found to contaminate soils at RAAF Pearce where fire-fighting practice was undertaken and waste water stored. The degree and extent to which underlying groundwater has been contaminated will be determined during investigations in 2016. RAAF Pearce is situated on 1000 hectares in a semi-rural

area 35 kilometres north northeast of Perth next to the town of Bullsbrook with 4,300 residents.

7 The Alternatives

While an immediate call in 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 draft consolidated guidance on alternatives to PFOS and related chemicals[71], 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. They found replacement products were based on short-chain PFAS[72] and various fluorinated telomers, eg C6-fluorotelomers such as perfluorohexane ethyl sulfonyl betaine. The potential degradation product of short chain fluorotelomer-based surfactants is the terminal degradation product, Perfluorohexanoic acid (PFHxA), which is extremely persistent and is now increasingly being detected in the environment with high concentrations found in several European rivers.

The POPs Review Committee working group expressed concerns that PFHxA could bioconcentrate, bioaccumulate or biomagnify. They identified that for 3M's alternative Dodecafluoro-2-methylpentan-3-one, the information gaps were major although a variety of liver effects were already noted and its probable persistency and volatility of concern. [73]

Other common alternatives are perfluorohexylsulfonyl (PFHxS)-based polymers. PFHxS is another terminal degradation product and hence, very persistent. PFHxS is found in seawater, animals, and humans in the Arctic. There is a growing body of evidence of its toxic effects in humans and animal studies and can affect the thyroid hormone (TH) pathway and inhibit 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 much more liver toxic than PFBS and PFOS.[74] 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. PFHxS has also been found in fish from Japan and gulls from the Norwegian Arctic.

Substitution with shorter chain substances based on perfluorobutane sulfonate or perfluorobutane sulfonic acid (PFBS) has seen increasing detection of PFBS in the dolphin liver samples[75] despite claims by Australia's regulatory agencies that PFBS is not bioaccumulative. 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. It has been detected in the Arctic and is also found in indoor dust from homes and offices. In a Germany study, PFBS was found in 33% of the children. It has demonstrated developmental neurotoxicity[76] and like PFOS and PFOA can affect the production of estrogen in placental cells.

8 International regulatory responses

The Organisation for Economic Cooperation and Development (OECD) undertook a hazard assessment on PFOS and its salts In 2000, concluding that the persistence of PFOS in the environment, its toxicity and bioaccumulation potential indicated cause for concern for the environment and human health.[77] In the same year, the United States Environmental Protection Agency (US EPA) severely restricts the use of PFOS and other perfluoroalkyl substances (PFASs) to uses where no safer alternative is available[78] and 3M announced it would cease production.

Canada has prohibited the manufacture, use, sale, offer for sale and import of PFOS and related substances and in 2006, the European Union adopted a resolution restricting the marketing and use of PFOS and related substances.[79]

In 2009, PFOS was formally listed on the United Nation's *Stockholm Convention on Persistent Organic Pollutants*. Its listing on Annex B permitted some limited ongoing uses however, work by the conventions scientific and technical committee continues to assess and phase out the remaining uses. For instance, 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). 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

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 a national integrated approach needs to be developed. 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 (C6) 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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