Consolidated feedback from Queensland Government representatives

Senate Foreign Affairs, Defence and Trade References Committee Public Hearing

Inquiry into Contamination of Australia's Defence Force facilities and other Commonwealth, State and Territory sites in Australia

Oakey

9 March, 2016.

Provision of material offered by representatives at the Hearing

- Mr Connor committed to providing the Committee with information about the consultation process undertaken to develop the draft policy for managing firefighting foams, including a list of organisations consulted.
- **Attachment 1** provides an outline of the consultation process undertaken and the organisations involved.

Additional information in support of comments made by Dr Young

- During the Oakey public hearing concerning "*Contamination caused by firefighting foams at RAAF Base Williamtown and other sites*" Dr Young presented data on PFOS and PFOA levels reported from testing done in 2002-2003. The Chair asked if there had been subsequent studies.
- While Dr Young was not aware of any at that time, following the Oakey hearing she was advised of some studies done since 2002-2003. Dr Young would like one of these studies, which relates to the decline in PFOS and PFOA levels in an Australian population from 2002 to 2011, provided to the Committee. Accordingly, a copy is at **Attachment 2**.
- In addition, Dr Young reported that there was work being undertaken at a national level which would provide a single source of advice relating to perfluorinated chemicals. That advice was not endorsed at the time of the Oakey hearing but is now available and is provided at **Attachments 3-4** for the information of the Committee.

ATTACHMENT 1
Department of Environment and Heritage Protection Draft Policy on Environmental Management of Firefighting Foam
Key Stakeholder Consultation Program 2015

Background & Scope

As part of the development of the draft Policy on Environmental Management of Firefighting Foam (the Policy) a wide range of firefighting foam key stakeholders were identified on the basis of the scale of their potential use and the extent of the risks posed by potential releases from their day-to-day or emergency activities. Included in this were industry groups and the relevant state and commonwealth regulatory agencies and organisations who would be called-on to set standards and provide advice on response, compliance and remediation issues for those end-users.

The primary foam end-users and relevant regulators/advisors stakeholders included:

- Ports and related activities e.g. from fuel transfer wharfs, storage tanks and firefighting tugs.
- Petroleum facilities (refineries and bulk storage) (BP, Caltex, AIP).
- Offshore petroleum and gas production platforms and onshore receiving facilities (Chevron Australia, Conoco-Phillips, Woodside Energy).
- Fire brigades (Qld, WA, Vic, NSW, Tas, SA, NZ, NT).
- State environmental regulatory agencies (WA, Vic, NSW, NT, Tas, SA, NZ).
- Commonwealth environmental and chemical regulators (National Industrial Chemicals Notification and Assessment Scheme (NICNAS), Department of Environment (DoE), National Measurement Institute (NMI)).
- Defence estate managers, related consultants & firefighters.
- Airport managers & firefighters (Perth, Brisbane, AirServices Australia).
- Environmental consultants.
- Waste management industry.
- Professional associations (Australian Institute of Dangerous Goods Consultants (AIDGC), Australian Land and Groundwater Association (ALGA)).
- Shipping management (Maritime Safety Queensland (MSQ)).
- Firefighting foam suppliers.

Discussion with each group generally included an overview of the current state-of-knowledge and international directions regarding environmental issues, risks and liabilities associated with the different types of firefighting foam with clarification of any misinformation or misconceptions held. Discussions then explored and considered potential industry-specific or site-specific concerns or issues.

The total face-to-face consultation audience so far has been more than 365 persons from the above stakeholder groups. In addition to this there have been telephone and email consultation with about another 20-30 stakeholders on particular issues.

Table 1 (below) contains details of stakeholder consultation events with respect to the 2nd Draft of the Policy.

Table 2 (below) lists the organisations on the mailing list that were sent the 2nd draft of the Policy for consultation purposes.

Table 1. Firefighting Foam – Stakeholder Consultation Diary (Second draft Policy)

Date/Location	Stakeholder(s)
30 Oct 2014 Gold Coast	**Ecoforum Conference** Persistent Organic Pollutants theme. About 80 attendees.
31 Oct 2014 Narangba	**Toxfree waste destruction facility** 3 staff.
23 Apr 2015 Brisbane	**Australian Land and Groundwater Association (Consultants)** 70 attendees. Broad range of consultants and industry.
03 June 2015 Brisbane	**Environment and Heritage Protection (EHP) (internal)** **Maritime Safety Queensland**
04 June 2015 Gallipoli Barracks	**DEFENCE.** Managers, environmental officers, firefighters, contractors and consultants. 31 attendees. Department of Defence, Jacobs, AECOM, Coffey, RAAF, Aurecon, GMD, ERM, Defence Support Organisations, GHD, Golder, CH2M.
04 June 2015 Cannon Hill	**Qld Fire & Emergency Services (QFES)** HazMat / Scientific Services 5 pers.
05 June 2015 Whyte Island	**Qld Fire & Emergency Services (QFES)** Fire training facility. 5 attendees.
05 June 2015 Brisbane	**Brisbane Airport Corporation** Airport manager & legal counsel.
09 June 2015 Townsville	**Townsville Airport** RAAF, AirServices firefighters, AECOM, Spotless, Townsville Airport, Defence Services Environment, Golder Associates, Defence. 10 attendees.
09 June 2015 Townsville	**Port of Townsville** Port manager.
11 June 2015 Melbourne	**Vic Metro Fire Brigade (MFB) & Country Fire Authority (CFA)** 12 attendees. MFB 6, CFA 4, Solberg Foam 2.
12 June 2015 Melbourne	**Petroleum industry** BP, Caltex, Esso, Australian Institute of Petroleum, Viva Energy. 8 attendees.
16 June 2015 Sydney	**HazMat 2015 SDS theme** Presentation on the inadequacies of safety data sheets in respect to all chemicals. N Holmes. About 50 attendees.
17 June 2015 Sydney	**Australian Institute of Dangerous Goods Consultants** 28 attendees. DG consultants, Shine Lawyers, TPG, Tyco Fire, Fire Protection Association/Firefighting Foam Coalition, BP, FM Global, Golder, WSP Group, ACOR, Haztech, Fire Services, & others.
18 Jun 2015 Sydney	**EHP Sydney Firefighting Foam Seminar** 59 attendees. Commonwealth Department of Environment, NICNAS, National Measurement Institute, NZ Fire, NSW Fire, Tas Fire, NZ Environmental Protection Agency (EPA), Tas EPA, WA Department of Environment and Regulation, NSW EPA, SA EPA, Qld EHP, CRC-CARE, AirServices, ALS Laboratories, Solberg Foam, Sandvik Foam, Jacobs, ERM, AECOM, GHD, Golder, Shine Lawyers, Brisbane Airport Corp, Elide Fire, FM Global, Scott Safety, Viva Energy, Esso, BP, Caltex, UTC Fire/foam, Fire Protection Technologies, M Willson, Tyco Fire.
19 Jun 2015 Sydney	**NICNAS Headquarters.** 29 attendees of department heads and senior officers.
19 Jun 2015 Sydney	**Victorian Parliamentary Enquiry into Fiskville Fire Training Facility** Bronwyn Halfpenny MP, Tim McCurdy MP, Vicki Ward MP, Dr Kelly Butler Research Officer, +1.

Date/Location	Stakeholder(s)
22 Jun 2015 Perth	**Chevron Australia** Emergency management, Occupational Health and Safety, environment senior officer. 5 attendees.
22 Jun 2015 Perth	**WA Department of Environment Regulation (DER)** DER managers and compliance officers. 8 attendees.
23 Jun 2015 Perth	**WA Department of Environment Regulation (DER)** Ken Raine & Stuart Cowie, (Executive Director Compliance and Enforcement)
23 Jun 2015 Perth	**Woodside Energy** Environmental engineering group & Workplace Health and Safety. 9 attendees.
24 Jun 2015 Perth	**Conoco-Phillips** Emergency management specialist & emergency response coordinator.
24 Jun 2015 Perth	**Perth Airport Corporation** Environment & Sustainability Manager and Airport Environment Officer.
02 Jul 2015 Adelaide	**SA Metro Fire Brigade (MFB) & Country Fire Authority (CFA)** Scientific section, Senior MFB & CFA officers, MFB firefighters. WH&S. 15 Attendees.
03 Aug 2015 Adelaide	**SA Metro Fire Brigade (MFB) & Country Fire Authority (CFA)** Scientific section, Senior MFB & CFA officers, MFB firefighters & union rep. 8 Attendees.
20 Aug 2015 Melbourne	**Victorian Environmental Protection Agency (EPA)** 24 attendees. Vic EPA officers & Executive Director, Land & Water, Health Dept. CFA.
Key industries lead-up consultation	
19 Feb 2014 Mackay **05 Dec 2014** Brisbane	**Port Corporations** Harbour masters, port managers, shipping berth and site operators, tug operators. Mackay 16, Brisbane 3 attendees.
30 Apr 2014 Brisbane	**Adani Coal Project** General Manager Environment and Sustainability, procurement managers, Sandvik Mining Supplies.

Table 2. Second Draft Firefighting Foam Policy Stakeholder Engagement List

*Note: ** denotes that more than one individual within the respective organisation was provided with a copy of the Second Draft of the Policy. Personal information has been removed from the list.*

Organisation	
AECOM	National Fire Industry Association Western Australia
Air Services Australia**	National Offshore Petroleum Safety and Environmental Management Authority (NOPSEMA)
Angus Fire UK	North Queensland Bulk Ports Corporation
Australasian Institute of Dangerous Goods Consultants	NRG Gladstone Power Station
Australian Fire and Emergency Services Authorities Council**	Origin Energy
Australian Institute of Petroleum Ltd	Perth Airport Pty Ltd
Australian Marine Oil Spill Centre	Port of Brisbane Pty Ltd
Australian Maritime Safety Authority**	Port of Townsville Limited
Australian Shipowners Association	Ports North**
Beca Consulting	PT Hydraulics Australia
BlueSphere Environmental	QGC
BP Australia Pty Ltd**	QLD Parks and Wildlife Service
Brisbane Airport Corporation Pty Ltd	QLD Transport and Main Roads
Brisbane City Council	Queensland Emergency Services
BRT Fire and Rescue Supplies	Queensland Ports Association
Caltex Australia Ltd	Queensland Resources Council
Caltex Australia Petroleum Pty Ltd	Roma Firefighting Equipment
Caltex Refineries Qld Pty Ltd	SA Metropolitan Fire Service
Chamber of Commerce and Industry of Western Australia	Sandvik Fire Suppression
Chamber of Commerce and Industry Queensland	SANTOS, GLNG Operations Pty Ltd**
Civil Aviation Safety Authority**	Solberg Asia Pacific Pty Ltd**
ConocoPhillips Australia Pacific LNG Downstream Project	Solberg Foam**
CRC CARE Pty Ltd (Cooperative Research Centre for Contamination Assessment and Remediation of the Environment)	Stirling Group
Defence Support – Queensland	Surface Mining - Australasia

Organisation	
Delta Fire & Safety (QLD) Pty Ltd	SVITZER Australia Pty Ltd
Department of Defence**	Tasmania Fire Service
Dynax Corporation	The Australian Petroleum Production & Exploration Assoc.**
eurofins (laboratories)	The Chamber of Minerals and Energy of WA
Fire Engineering Solutions Pty Ltd	The Shell Company of Australia Limited
Fire Protection Association Australia**	Thiess Mining Australia
Fire Rescue Safety Australia**	Tyco Fire & Security Australia (Wormald)
Firefighting Foam Coalition	Tyco Fire Protection Products
Hemming Fire UK	UK CAA (ex)
Insurance Council of Australia	UTC Building & Industrial Systems (Chubb & Kidde)
Kidde	Victorian Metro Fire Brigade
Kwinana Industries Council	WA Department of Fire and Emergency Services
Local Government Association of Queensland Ltd	WA Department of Mines and Petroleum
Maritime Safety Queensland	WA Health
MMG Century (Kurumba)	WA Local Government Association
Moreton Bay Regional Council	Waste, Recycling Industry Association (QLD) Inc**
National Fire Industry Association	Williams Fire & Hazard Control | Tyco Fire Protection Products
National Fire Industry Association Queensland	Willson Consulting

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Decline in perfluorooctane sulfonate and perfluorooctanoate serum concentrations in an Australian population from 2002 to 2011

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ABSTRACT

Some perfluoroalkyl and polyfluoroalkyl substances (PFASs) have become widespread pollutants detected in human and wildlife samples worldwide. The main objective of this study was to assess temporal trends of PFAS concentrations in human blood in Australia over the last decade (2002–2011), taking into consideration age and sex trends.

Pooled human sera from 2002/03 (n = 26); 2008/09 (n = 24) and 2010/11 (n = 24) from South East Queensland, Australia were obtained from de-identified surplus pathology samples and compared with samples collected previously from 2006/07 (n = 84). A total of 9775 samples in 158 pools were available for an assessment of PFASs. Stratification criteria included sex and age: <16 years (2002/03 only); 0–4 (2006/07, 2008/09, 2010/11); 5–15 (2006/07, 2008/09, 2010/11); 16–30; 31–45; 46–60; and >60 years (all collection periods). Sera were analyzed using on-line solid-phase extraction coupled to high-performance liquid chromatography–isotope dilution-tandem mass spectrometry.

Perfluorooctane sulfonate (PFOS) was detected in the highest concentrations ranging from 5.3–19.2 ng/ml (2008/09) to 4.4–17.4 ng/ml (2010/11). Perfluorooctanoate (PFOA) was detected in the next highest concentration ranging from 2.8–7.3 ng/ml (2008/09) to 3.1–6.5 ng/ml (2010/11). All other measured PFASs were detected at concentrations <1 ng/ml with the exception of perfluorohexane sulfonate which ranged from 1.2–5.7 ng/ml (08/09) and 1.4–5.4 ng/ml (10/11). The mean concentrations of both PFOS and PFOA in the 2010/11 period compared to 2002/03 were lower for all adult age groups by 56%. For 5–15 year olds, the decrease was 66% (PFOS) and 63% (PFOA) from 2002/03 to 2010/11. For 0–4 year olds the decrease from 2006/07 (when data were first available for this age group) was 50% (PFOS) and 22% (PFOA).

This study provides strong evidence for decreasing serum PFOS and PFOA concentrations in an Australian population from 2002 through 2011. Age trends were variable and concentrations were higher in males than in females. Global use has been in decline since around 2002 and hence primary exposure levels are expected to be decreasing. Further biomonitoring will allow assessment of PFAS exposures to confirm trends in exposure as primary and eventually secondary sources are depleted.

1. Introduction

Perfluoroalkyl and polyfluoroalkyl substances (PFASs) have entered the environment since the 1950s from fluoropolymer manufacturing processes and disposal of products containing fluorochemicals, such as carpet and apparel, pharmaceuticals, fire fighting foams, lubricants, adhesives, cosmetics, paper coatings, leather, pesticides and insecticides (Key et al., 1997; Paul et al., 2009; Prevedouros et al., 2006). Directly emitted PFASs are globally distributed and transported long distances via oceanic transport (Armitage et al., 2009; Wania, 2007). Perfluorinated alkylated acids, one type of PFASs, including perfluorooctane sulfonate (PFOS) and perfluorooctanoate (PFOA), are also distributed through wet and dry deposition as a result of oxidative degradation processes in the atmosphere of volatile precursors, such as fluorotelomer alcohols, perfluorinated sulfonamide alcohols, fluorotelomer acrylates and fluorotelomer olefins (Ellis et al., 2003; Young and Mabury, 2010).

In recent years, PFOS and PFOA have been studied extensively due to their high resistance to both chemical and biological degradation as well as potential for bioaccumulation (Lau et al., 2007). As a consequence of

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their persistence and widespread usage, ubiquitous distribution in both environmental and human samples exists (e.g. Calafat et al., 2007a,b; Giesy and Kannan, 2001; Kannan et al., 2004; Kärrman et al., 2007). Because of its characteristics of toxicity, persistence, bioaccumulation and long range transport, PFOS was listed under the Stockholm Convention on Persistent Organic Pollutants in 2009 (Stockholm Convention on POPs, 2010). Definitive health risks associated with PFAS exposure in humans have not been reported, with studies of persons occupationally exposed to relatively high concentrations showing varying results (Olsen et al., 2003; Wang et al., 2012). Similarly, epidemiological studies of PFASs and various endpoints have also shown varying results. Several authors have reported associations between maternal PFAS concentrations and negative effects with regard to fetal development. Fei et al. (2007) reported PFOA levels were inversely associated with birth weight; Apelberg et al. (2007) found negative associations between both PFOS and PFOA concentrations and birth weight and size; and Darrow et al. (2013) also found a negative association with PFOS and birth weight. Some studies have suggested associations between PFOS and PFOA serum concentrations and thyroid disease (Melzer et al., 2010); and alterations to lipid metabolism (Steenland et al., 2009). Associations have been observed for perfluorohexane sulfonate (PFHxS) but not PFOA and PFOS with an elevated odds of high cholesterol, total cholesterol, low-density lipoprotein cholesterol, total cholesterol/high density lipoprotein (HDL) cholesterol ratio and non-HDL cholesterol (Fisher et al., 2013). An assessment of potentially exposed persons in West Virginia, USA found among other results, probable links between PFOA exposure and diseases such as kidney and testicular cancer (Barry et al., 2013), thyroid disease (Winquist and Steenland, 2014) high cholesterol, ulcerative colitis and pregnancy-induced hypertension (C8 Science Panel, 2014).

Human exposure to PFASs occurs via point sources such as manufacturing plants (Oliaei et al., 2013), food (Clarke et al., 2010; Egeghy and Lorber, 2011; Fromme et al., 2009) and its packaging (Begley et al., 2005), drinking water (Eriksson et al., 2013), and household dust (Fraser et al., 2013; Goosey and Harrad, 2011). Both direct and indirect exposures occur because PFOS and PFOA are stable degradation products/metabolites of other PFASs (Kato et al., 2011).

PFASs were first detected in Australian human blood serum collected in 2002–03 at concentrations equal to or higher than reported in Europe and Asia but lower than in the USA (Kärrman et al., 2006). This finding was unexpected as concentrations of "traditional" persistent organic pollutants such as dioxins and polychlorinated biphenyls have been relatively low in Australia (Harden et al., 2007).

The main objective of this study is to assess temporal trends of PFAS concentrations in Australia over the last decade (2002–2011). Assessment of any temporal trends allows a gauge of the success of the increased regulatory scrutiny in recent years of PFAS in Australia. It can also reflect changes in the pattern and extent of exposure in the Australian population following a global decrease in manufacture and emission of certain PFASs and potential increase in others due to shifts in production. In this study existing data on PFASs in blood from Australians in 2006/07 (Toms et al., 2009), archived samples from 2002/03, and newly collected samples from 2008/09 and 2010/11 will be used to evaluate whether global changes in PFAS usage have affected human exposure to these chemicals in Australia.

2. Materials and methods

2.1. Sample collection

We used archived pooled human sera from 2002/03 (n = 26) and samples collected in 2008/09 (n = 24 pools, 2400 individual samples) and 2010/11 (n = 24 pools, 2400 individual samples) from South East Queensland, Australia. PFAS pooled serum concentrations from 2006/07 (n = 84) were reported previously (Toms et al., 2009). All samples were obtained in collaboration with Sullivan Nicolaides Pathology from de-identified surplus pathology samples. That is, samples were collected from individuals in the community setting for assessment of biochemical parameters; the serum remaining after these assessments was surplus to requirement by the pathology clinic and made available for research purposes. Stratification criteria included age: <16 years (02/03 only); 0–4 (08/09, 10/11); 5–15 (08/09, 10/11); 16–30; 31–45; 46–60; and >60 years (all collection periods). As reported and discussed in detail in Toms et al. (2009), the 2006/07 samples were stratified as follows: 0–0.5; 0.6–1; 1.1–1.5; 1.6–2; 2.1–2.5; 2.6–3; 3.1–3.5; 3.6–4; 4.1–6; 6.1–9; 9.1–12; 12.1–15 years. For comparative purposes, in this study these age brackets will be combined into 0–4 years or 5–15 years as appropriate for comparison with age groups from other collection periods. Both males and females were included. Each pool consisted of up to 100 samples (see Harden et al., 2007 for details), with the exception of the 2006/07 pools that consisted of approximately 30 samples (see Toms et al., 2009 for details). It was not possible to determine if any one donor contributed to more than one collection period. Ethics approval for this study was granted by the University of Queensland Medical Research Ethics Committee. The involvement of investigators at the Centers for Disease Control and Prevention (CDC) was determined not to constitute engagement in human subjects research [45 CFR 46.101(d)].

2.2. Measurement of PFASs in serum

All samples were analyzed at the Division of Laboratory Sciences, National Center for Environmental Health, CDC, Atlanta, USA by a modification of the Kuklenyik et al. (2005) approach, involving on-line solid-phase extraction coupled to high-performance liquid chromatography–isotope dilution-tandem mass spectrometry (Calafat et al., 2007a,b). Limits of detection (LOD) were 0.2 ng/ml (2-(N-ethyl-perfluorooctanesulfonamido) acetate [Et-PFOSA-AcOH], 2-(N-methyl-perfluorooctanesulfonamido) acetate [Me-PFOSA-AcOH], perfluorodecanoate [PFDeA]) and 0.1 ng/ml (PFHxS, perfluorononanoate [PFNA], PFOA, PFOS, perfluorooctanesulfonamide [PFOSA]) (Calafat et al., 2007a,b). Quality control/quality assurance (QC/QA) included sampling replication of pools for a given strata and analysis of blank samples. CDC researchers received coded samples and were blind to the pools' characteristics. Analytical standards, low- and high-concentration QC samples (prepared from spiked calf serum) and reagent blank samples were included in each analytical batch along with the study samples (Kuklenyik et al., 2005).

Moreover, for the 2006/07, 2002/03 and 2008/09 pools, two, two and one samples of calf serum (Sigma Aldrich B8655), respectively, known to have concentrations of the target PFASs below the LOD, were aliquoted, pooled, stored, shipped and analyzed using identical procedures to human blood sera. No PFASs were detected in these blank samples. Sample replication between pools of the same strata (i.e., two pools which were obtained for the same age and sex) was assessed using the normalized difference ($([a–b]/([a + b]/2)) \times 100\%$) (where a is the value from Pool 1 and b is the value from Pool 2) and the average described as the mean normalized difference (MND) for all age groups and both sexes combined. In 2008/09, the MNDs for PFOS and PFOA were 13% and 10%, respectively and in 2010/11, the MNDs for PFOS and PFOA were 18% and 16%, respectively. This agreement between replicates and absence of PFASs in the blank serum suggests that the pooling procedures were uniform and contamination during sampling or analysis was unlikely.

2.3. Statistical analysis

Statistical analysis was mainly descriptive to estimate average concentrations (means or medians as appropriate) and standard deviations or ranges. ANOVA models with the Tukey's post test carried out using GraphPad Prism 5. The conventional 5% cut-off was used to report

results as statistically significant. Concentrations <LOD were imputed a value of zero in the statistical analysis.

3. Results and discussion

PFASs were detected in all pools of human blood sera from all four collection periods.

The PFASs detected in the highest concentrations were PFOS (total PFOS — linear plus branched structural isomers) which ranged from 5.3–19.2 ng/ml (2008/09) to 4.4–17.4 ng/ml (2010/11). PFOA was detected in the next highest concentration ranging from 2.8–7.3 ng/ml (2008/09) to 3.1–6.5 ng/ml (2010/11). All other measured PFASs were detected at concentrations <1 ng/ml with the exception of PFHxS which ranged from 1.2–5.7 ng/ml (2008/09) to 1.4–5.4 ng/ml (2010/11). All results for 2002/02, 2008/09 and 2010 are available in the Supporting information, Table S1, those for 2006/07 are available in Toms et al. (2009). Temporal and international trends as well as age and sex trends are discussed below.

3.1. Temporal trends

Concentrations of PFASs determined in pools of human blood serum collected in Australia in 2002/03, 2006/07, 2008/09 and 2010/11 were compared to assess changes over time (Figs. 1–2). From the data obtained in this study, decreasing temporal trends were apparent from 2002/03 to 2010/11. The mean concentrations of both PFOS and PFOA in the 2010/11 period compared to 2002/03 were lower for all adult age groups by 56%. For 5–15 year olds the decrease was 66% and 63% for PFOS and PFOA, respectively, from 2002/03 to 2010/11. For 0–4 year olds the decrease from 2006/07 (when data were first available for this age group) was 50% and 22% for PFOS and PFOA, respectively (Table 1). It is expected that exposure as evident from the concentrations in the 2010/11 pools, will be lower than in the early 2000s and will likely continue to decrease slowly. As exposure continues to decrease and elimination occurs, although at differing rates considering the varying human half-lives for PFASs (e.g., 4.8 years [PFOS], 3.5 years [PFOA], (Olsen et al., 2007)), current PFAS serum concentrations will reflect the recent decreased exposure due to global changes in use.

Trends in concentrations of the other PFAS should be interpreted with caution due to low detection rates and low concentrations. Et-PFOSA-AcOH and Me-PFOSA-AcOH decreased 75% and 63% from 2002/03 to 2010/11 for 5–15 and >16 year olds, respectively. Concentrations of PFHxS increased 5% in the >16 years group but decreased 48% for the 5–15 year olds. PFNA concentrations increased 51% and 19% for 5–15 and >16 year olds, respectively. PFDeA concentrations increased from below the limit of detection (0.1 ng/ml) in 2002/03 to 0.3 and 0.4 ng/ml for 5–15 and >16 year olds, respectively. PFOSA was detected in only a few pools in 2002/03 and then not at all in 2010/11.

The ANOVA test results show the mean concentration difference is statistically significant between years of collection for PFOS (Fig. 1) for 0–4 year olds ($p = 0.002$), 5–15 year olds ($p < 0.0001$) and >16 year olds ($p < 0.0001$). Tukey's post test showed statistically significant differences between mean concentrations across collection periods ($p < 0.05$) with the exception of 2008/09 and 2010/11 for all three age groupings. For PFOA (Fig. 2), the mean concentration difference was statistically significant between year of collection for 0–4 year olds ($p = 0.004$), 5–15 year olds ($p < 0.0001$) and >16 years olds ($p < 0.0001$) with the Tukey's post test reaching significance for all collection periods except 2008/09 and 2010/11 for 0–4 year olds and 5–15 year olds and 2006/07 and 2008/09 for >16 year olds.

These overall decreasing levels are in accordance with the voluntary phase out of perfluorooctanesulfonyl fluoride (PFOSF) based compounds by the 3M Company in the USA, which was completed in 2002 (3M, 2000). In Australia, PFOS and PFOA have been imported and used as, among others, mist suppressants in the metal plating industry, hydraulic fluid in the aviation industry, surfactants in the photography industry and as fire-fighting foams. While alternatives to PFOS are available for mist suppressants in the metal plating industry and for fire-fighting foams, some of these are still fluorinated. Accordingly, PFAS-based chemicals with no known suitable and less hazardous alternatives are still used mainly as mist suppressants in the metal plating industry, hydraulic fluid in the aviation industry, surfactants in the photography industry and as fire-fighting foams. While importation of PFOS increased between 2006 and 2008, this was mostly for uses for which alternatives are not readily available and overall from 2006 to 2008 PFOS stocks in Australia had decreased (NICNAS, 2013). Furthermore, PFAS use has been discouraged by the National Industrial Chemical Notification and Assessment Scheme (NICNAS) in Australia with voluntary phase out agreements by Australian industries since 2000 resulting in a rapid decrease in the use of PFOS-related chemicals (NICNAS, 2013). However, old stock of PFOS- and PFAS-based products could still be found in Australia or be held by consumers and industrial users, although import of PFOA-containing polymers virtually ceased after NICNAS and industry co-regulatory activity (NICNAS, 2013).

PFOS is still produced in at least three countries, namely Germany, Italy and China (Oliaei et al., 2013). As a result, PFOS levels have fallen in many parts of the world, but have increased in others—most notably in China (Oliaei et al., 2013). Also of importance is that both PFOS and PFOA are also distributed through wet and dry deposition as a result of oxidative degradation processes in the atmosphere of volatile precursors, such as fluorotelomer alcohols, perfluorinated sulfonamide alcohols, fluorotelomer acrylates and fluorotelomer olefins (Ellis et al., 2003; Young and Mabury, 2010). These changes are likely the reasons for decreasing concentrations of PFASs in human blood serum, but exposure to PFAS likely continues.

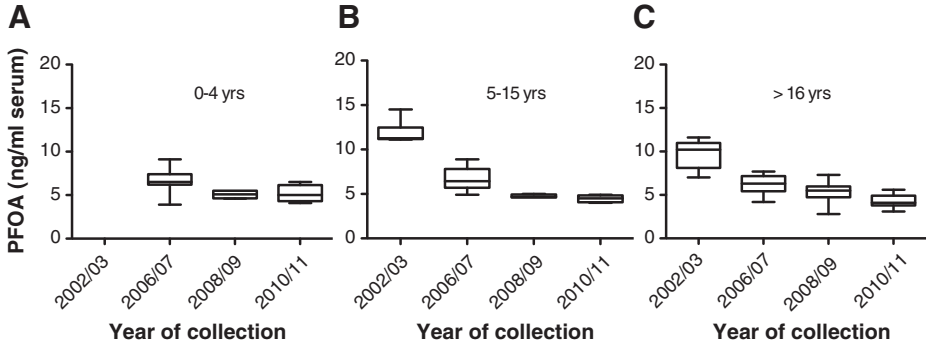

Fig. 1. Box and whisker plots with median, minimum, maximum, 25th and 75th percentile data for PFOA combined by sex and age (0–4 years A; <16 (5–15) years B; and >16 years C) by collection date. Note: 0–4 years not available from 2002/03.

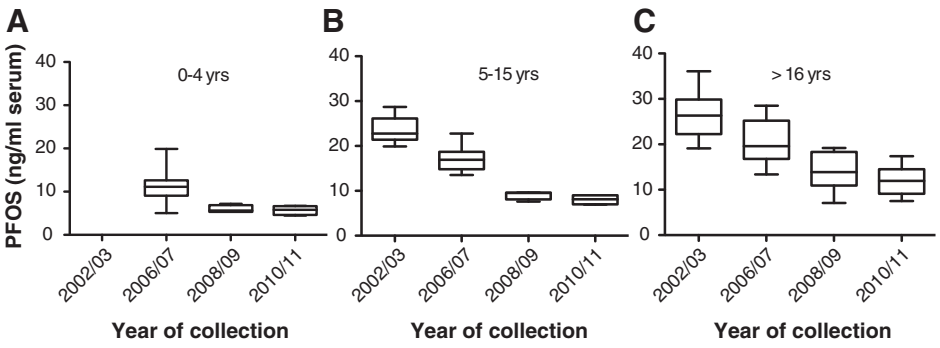

Fig. 2. Box and whisker plots with median, minimum, maximum, 25th and 75th percentile data for PFOS combined by sex and age (0–4 years A; <16 (5–15) years B; and >16 years C) by collection date. Note: 0–4 years not available from 2002/03.

In addition to overall concentrations, detection frequency changed over the 9 year sampling period but only for Et-PFOSA-AcOH, Me-PFOSA-AcOH, PFDeA and PFOSA. Et-PFOSA-ACOH and PFOSA decreased in detection frequency at 100%, 1%, 0% and 0% and 19%, 24%, 0% and 0% from 2002/03, 2006/07, 2008/09 and 2010/11, respectively (Table 2). Me-PFOSA-AcOH, PFHxS, PFNA, PFOA and PFOS were detected consistently at or close to 100% across all periods. The detection frequency of PFDeA increased from 0%, 90%, 88% and 100% from 2002/03, 2006/07, 2008/09 to 2010/11, respectively, but mean concentrations from 2006/07 through 2010/11 remained the same. This change in exposure scenario may be influenced by an onset of production of different homologues and relocation of manufacturing activities resulting in different exposure to the Australian population. Analytical reasons are unlikely as the LODs did not change and the 2002/03 pools were analyzed at the same time as the 2008/09 pools.

3.2. Age and sex trends

Concentrations of PFOS, PFOA PFNA and PFHxS appeared to be higher in males than in females across all adult ages. In the younger age groups, in particular, 0–4 and 5–15 years in 2008/09, concentrations were higher in females compared to males and then a shift occurred around 16 years and concentrations in males were higher than in females. The greatest difference was seen in 30–45 and 46–60 year old persons then a stabilization occurred in the >60 year adults, with similar concentrations in both males and females. Sex differences in concentrations of PFDeA, Me-PFOSA-AcOH and Et-PFOSA-AcOH were not obvious, although this may be attributable to the low detection of these chemicals. Differences in exposure and/or pharmacokinetic reasons have been suggested for sex differences in PFAS concentrations (Calafat et al., 2007a,b) although these are yet to be completely elucidated. Lactation and pregnancy (Fei et al., 2007; Kärrman et al., 2007; So et al., 2006; Tao et al., 2008) result in the reduction of adult female PFAS body burden and menstruation has been investigated as an elimination route for pre-menopausal females (Harada et al., 2005; Knox et al., 2011; Taylor et al., 2014). Thompson et al. (2010) hypothesized that if blood loss via menstruation was the predominant reason for the observed differences in male and female serum PFAS concentrations, then the concentrations in males who are regular blood donors should be similar to those in typical pre-menopausal females. In fact, PFOS and PFOA concentrations in Australian males who regularly donated blood were almost half the concentration in males from the general population in Australia and much closer to concentrations in females (Thompson et al., 2010), thus supporting the hypothesis of blood loss contributing to lower PFAS concentrations in human serum.

There were varying patterns of PFAS concentrations by age in these Australian pools (Fig. S1, Supporting information). PFAS concentrations appeared to increase from birth with the maximum concentrations of all PFASs detected in children <15 years with the exception of PFOS where concentrations increased with age peaking at >60 years. Interestingly, a comparison of the data from samples collected in 2002/03 with those collected in 2008/09 and 2010/11 showed some differences in the age trend. For example, for PFOS no clear age trend was observed in the 2002/03 samples whereas the concentrations clearly increased with age in the 2010/11 samples. For PFOA, we observed a decrease from the youngest age toward older age groups in the 2002/03 data whereas no decrease was observable in the recently collected samples. It could be that these differences likely reflect a more rapid response to changing exposure concentrations in younger age groups which results in changing age trends. The age trend in the most recently collected samples may reflect that older persons were more exposed than younger people when PFOS/PFOA were used/manufactured/imported and what is seen now is driven mainly by the elimination half-lives of these compounds. In 2002/03, in addition to elimination, concurrent exposure occurred and an age trend was harder to observe.

3.3. International comparisons and temporal trends in human samples

When compared to results from elsewhere, concentrations of PFOS and PFOA from 2010/11 in Australia are similar or higher. Concentrations are more than 6 and 2 times higher for PFOS and PFOA, respectively, than found in adults from Henan, an agricultural province in China (Fu et al., 2014), but similar to slightly higher in pregnant women from Tianjin, China (Jiang et al., 2014). Concentrations in Australian females of child-bearing age (16–30 and 31–45 years) are

Table 1
Mean concentrations (ng/ml serum) of PFOS and PFOA by age group (years) and year of collection with percent difference to 2002/03 in brackets.

Age group (years)	2002/03		2006/07**		2008/09		2010/11	
	PFOS	PFOA	PFOS	PFOA	PFOS	PFOA	PFOS	PFOA
0–4			11.4	6.7	6	5.1	5.7 (50%*)	5.2 (22%*)
5–15 (<16 — 2002/03)	23.6	12	17 (28%)	6.6 (45%)	9.1 (61%)	4.8 (60%)	8 (66%)	4.5 (63%)
>16	27	9.7	20.5 (24%)	6.2 (36%)	14.1 (48%)	5.3 (45%)	12 (56%)	4.3 (56%)

* 2010/11 compared to 2006/07.
** Data from Toms et al. (2009).

Table 2
Summary results of PFAS concentrations (ng/ml) from 2002/03 (26 pools), 2006/07 (84 pools), 2008/09 (24 pools) and 2010/11 (24 pools) of human blood serum, all ages and both sexes combined.

Collection period	PFC	Frequency of detection[a]	Range	Mean	Standard deviation	Median
2002/03	Et-PFOSA-AcOH	100%	0.3–0.8	0.5	0.1	0.5
2006/07		1%	<LOD–0.2	n/a	n/a	n/a
2008/09		0%	n/a	n/a	n/a	n/a
2010/11		0%	n/a	n/a	n/a	n/a
2002/03	Me-PFOSA-AcOH	100%	0.5–1.6	0.9	0.3	1.2
2006/07		94%	<LOD–2	0.7	0.4	0.6
2008/09		83%	<LOD–0.5	0.3	0.2	0.3
2010/11		100%	0.1–0.5	0.3	0.1	0.2
2002/03	PFDeA	0%	n/a	n/a	n/a	n/a
2006/07		90%	<LOD–0.8	0.3	0.1	0.3
2008/09		88%	<LOD–0.4	0.3	0.1	0.3
2010/11		100%	0.2–0.4	0.3	0.1	0.3
2002/03	PFHxS	100%	2–12.8	4.3	2.7	3.6
2006/07		95%	<LOD–11.3	3.1	2	2.9
2008/09		100%	1.2–5.7	2.9	1	3
2010/11		100%	1.4–5.4	3.3	1	3.3
2002/03	PFNA	100%	0.4–0.7	0.5	0.09	0.5
2006/07		100%	0.1–1.4	0.8	0.3	0.8
2008/09		100%	0.9–1.6	1.2	0.2	1.2
2010/11		100%	0.6–0.9	0.7	0.1	0.8
2002/03	PFOA	100%	7–14.5	10.2	1.7	10.6
2006/07		100%	0.8–9.1	6.4	1.5	6.4
2008/09		100%	2.8–7.3	5.2	1	5.1
2010/11		100%	3.1–6.5	4.5	0.8	4.3
2002/03	PFOS	100%	19.1–36.1	25.9	4.7	25.4
2006/07		100%	5–28.5	15.2	4.9	14.8
2008/09		100%	5.3–19.2	11.9	4.6	11
2010/11		100%	4.4–17.4	10.2	3.7	9.4
2002/03	PFOSA	19%	<LOD–0.5	<LOD	n/a	n/a
2006/07		24%	<LOD–0.5	0.4	0.1	0.1
2008/09		0%	n/a	n/a	n/a	n/a
2010/11		0%	n/a	n/a	n/a	n/a

[a] The limits of detection (LOD) were 0.2 ng/ml (Et-PFOSA-AcOH — 2-(N-ethyl-perfluorooctane sulfonamido) acetate; Me-PFOSA-AcOH — 2-(N-methyl-perfluorooctane sulfonamido) acetate; PFDeA — perfluorodecanoate) and 0.1 ng/ml (PFHxS — perfluorohexane sulfonate; PFNA — perfluorononanoate; PFOA — perfluorooctanoate; PFOS — perfluorooctane sulfonate; and PFOSA — perfluorooctane sulfonamide).

more than and almost twice (PFOS and PFOA, respectively) than that found in pregnant women from Germany (Fromme et al., 2010). PFOS and PFOA concentrations are 1.5 and twice those found in adults from the USA (Olsen et al., 2012).

A number of studies have been published examining temporal trends in human serum. Archived samples from Korea showed little variation in PFOS concentrations measured at three time points (1994, 2000, 2008) in Busan, and two points in Seoul (1994, 2007). However, PFOA concentrations appeared to increase in Seoul (Harada et al., 2010). The same study reported a significant decrease in PFOA concentrations in Osaka, Japan, when measured in 2004 and 2008. However no significant differences were seen in two other Japanese cities. Temporal trends from 2003 to 2011 were observed in serum from pregnant women in Hokkaido, Japan with a decline in PFOS and PFOA at 8.4%/year and 3.1%/year, respectively, while concentrations of PFNA and perfluorodecanoate (PFDA) increased 4.7%/year and 2.4%/year, respectively (Okada et al., 2013). In the USA, comparison of blood samples from adults collected in 2000–2001 with samples collected in 2010 show a 76% and 48% decrease in PFOS and PFOA concentrations, respectively (Olsen et al., 2012). The more rapid decrease in PFOS was suggested as resulting from the 3M phaseout of PFOSF production (Olsen et al., 2008). In 2006, the US Environment Protection Agency, along with eight major companies launched a PFOA Stewardship Program, in which companies committed to reduce global facility emissions and product content of PFOA and related chemicals by 95% by 2010, and to work toward eliminating emissions and product content by 2015 (USEPA, 2013). Similarly comparing results from the National Health and Nutrition Examination Survey, an investigation of the health of about 5000 people every year, over several year points to an overall decrease in the US general population exposure to some PFASs (Calafat et al., 2006, 2007a,b). Recently, Kato et al. (2011) reported decreasing trends for PFOS (1999/2000 to 2008) and PFHxS (1999 to 2006), but PFNA had an increasing trend and PFOA remained stable from 2003 to 2008. Concentrations of PFOS and PFOA decreased 26% and 23%, respectively from 2001 to 2007 while PFHxS increased from 1979 to 2001 but not between 2001 and 2007 in males in Norway. Concentrations of PFNA and PFDA increased from 1979 to 2007 (Nøst et al., 2014). In Germany, trends were assessed from 1982 to 2010 with overall downward trend for PFOS, PFOA and PFHxS with no trends observable for PFNA (Schroter-Kermani et al., 2013).

Both the human and environmental studies present mixed conclusions on temporal trends of PFAS concentrations, and clearly point to a situation whereby local concentrations are influenced by production sources nearby. In some cases the increase in PFOA and other PFASs may reflect the increased use of telomeric compounds relative to PFOSF based ones. Worldwide, human and environmental concentrations of PFOS are decreasing in response to a decreased global production, however, exposure will continue via PFAS-containing products that will remain in circulation long after actual manufacture ceases.

4. Conclusions

This study provides strong evidence for decreasing serum PFOS and PFOA concentrations in an Australian general population from 2002 through 2011. By extension this suggests background levels of these compounds in Australia are also decreasing. Taken together, these findings may be reflective of the recent global production changes, as well as manufacturers' and regulatory bodies' efforts to limit emissions to the environment. Further monitoring of the Australian population's concentrations of PFAS will allow assessment of PFAS exposures as primary and secondary stocks are depleted and exposure decreases further.

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Disclaimer: The findings and conclusions in this report are those of the authors and do not necessarily represent the official position of the CDC or the views of the Australian Department of the Environment.

Appendix A. Supplementary data

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.envint.2014.05.019.

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enHEALTH
Environmental Health Standing Committee (enHealth) of the
Australian Health Protection Principal Committee

enHealth Guidance Statements on Perfluorinated Chemicals

Background and context:

Perfluorinated chemicals (PFCs) are a class of manufactured chemicals that have been used since the 1950s to make products that resist heat, stains, grease and water. Products that may contain PFCs include furniture and carpets treated for stain resistance, foams used for firefighting, fast food or packaged food containers, make up and personal care products and cleaning products. Other chemicals used in these applications may be precursors to PFCs, and the PFCs are formed when these chemicals are released into the environment.

PFCs are of concern around the world because they are not broken down in the environment and so can persist for a long time. Their widespread use and persistence means that many PFCs are ubiquitous global contaminants.

The PFCs of most concern are perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). Many countries have phased out, or are in the process of phasing out the use of PFOS and PFOA due to concerns about their persistence, bioaccumulation and toxicity.

Because of their widespread use, most people in Australia will have some PFOS and PFOA in their body. PFOS and PFOA are readily absorbed through the gut, and once these chemicals are in a person's body it takes about two to nine years, depending on the study, before those levels go down by half, even if no more is taken in.

The Australian Government has been working since 2002 to reduce the importation of some PFCs. In Australia and internationally where the use of PFCs has become restricted a general trend towards lower PFCs levels in a person's body has been observed.

Outside of the occupational setting, exposure to PFCs can occur from the air, indoor dust, food, water and various consumer products. For most people food is expected to be the primary source of exposure to PFOS and PFOA. Human breast milk may contribute to exposure in infants since PFCs have been detected in human breast milk.

For some communities near facilities where PFOS and PFOA have been extensively used, higher levels may be found in the surrounding environment and exposure may occur through other means, including drinking water supplied from groundwater.

In chronic exposure studies on laboratory animals, research into PFOS and PFOA has shown adverse effects on the liver, gastrointestinal tract and thyroid hormones. However, the applicability of these studies to humans is not well established.

In humans, research has not conclusively demonstrated that PFCs are related to specific illnesses, even under conditions of occupational exposure. Recent studies have found possible associations to some health problems, although more research is required before definitive statements can be made on causality or risk.

Because the human body is slow to rid itself of PFOS and PFOA, continued exposure to these chemicals can result in accumulation in the body. Due to the potential for accumulation, and while uncertainty around their potential to cause human adverse health effects remains, it is prudent to reduce exposure to PFCs as far as is practicable. This means that action needs to be taken to address the exposure source or possible routes of exposure. Determination of exposure is best achieved through a full human health risk assessment that examines all routes of exposure.

It is understandable that communities living in PFC affected areas may want to know what their level of exposure to PFCs is and what this means for their health and the health of their families. The lack of certainty around the potential for health effects can compound concerns.

A blood test can measure the level of PFOS and PFOA in a person's blood and can tell a person concerned about exposure to PFCs how their blood PFOS and PFOA levels compare with the levels seen in the general Australian population. However, these tests are not routine and there is at present insufficient scientific evidence for a medical practitioner to be able to tell a person whether their blood level will make them sick now or later in life, or if any current health problems are related to the PFC levels found in their blood.

As such, blood tests have no diagnostic or prognostic value and are not recommended for the purpose of determining whether an individual's medical condition is attributable to exposure to PFOS or PFOA.

In the absence of any test, including a blood test, being definitive in informing individual risk and clinical management, exposure reduction is the key measure to reduce any possible risks posed by PFCs.

At a population level, blood tests can inform a community that they have been exposed to PFCs at a level above that of the general population. The monitoring of pooled community blood samples over time may help determine the success of exposure reduction measures.

Recognising the difficulty in assessing and communicating the risks posed by PFCs to the community, enHealth has developed these guidance statements on key health issues to support jurisdictional responses to incidents of environmental PFC contamination.

Guidance statements:

1. Health impacts from exposure to PFOS and PFOA

There is currently no consistent evidence that exposure to PFOS and PFOA causes adverse human health effects.

Because these chemicals persist in humans and the environment, enHealth recommends that human exposure to these chemicals is minimised as a precaution.

2. Major human exposure pathways

For the general community, enHealth considers ingestion of food contaminated with PFOS and PFOA is the major human exposure pathway.

In sites contaminated by PFOS and PFOA, drinking water and specific foods may be important exposure pathways.

3. Reference values for PFOS and PFOA

In early 2016, enHealth will convene an expert group to provide advice to the Australian Health Protection Principal Committee on the development of an Australian interim health reference value for PFOS and PFOA for consistent use in the undertaking of human health risk assessments.

The interim health reference value will consider relevant international guidelines, as well as contemporary scientific and technical issues.

4. Breast feeding

The significant health benefits of breast feeding are well established and far outweigh any potential health risks to an infant from any PFOS or PFOA transferred through breast milk.

enHealth does not recommend that mothers living in or around sites contaminated with PFOS or PFOA cease breast feeding.

5. Pregnancy

There is currently no consistent evidence that exposure to PFOS or PFOA causes adverse human health outcomes in pregnant women or their babies.

Nonetheless, enHealth recommends that pregnant women should be considered a potentially sensitive population when investigating PFOS and PFOA contaminated sites, with a view to minimising their exposure to PFOS and PFOA.

6. Blood tests

There is currently no accepted clinical treatment to reduce levels of PFCs in the human body.

Given the uncertainty that PFCs are directly linked to adverse health outcomes, blood tests cannot determine if the PFC levels in a person's blood will make them sick now or later in life.

Therefore, blood tests are not recommended to determine whether any medical condition is attributable to exposure to PFOS or PFOA and have no current value in informing clinical management, including diagnosis, treatment or prognosis in terms of increased risk of particular conditions over time.

It is noted that various organisations around the world have collected blood samples from people as part of ongoing investigations into PFC contamination of soil and water. The purpose of these tests was either as part of a defined research program, or to determine how much of these chemicals may be entering a person's body. The value of blood testing is limited to assessing exposure, such as monitoring over time, which may help determine the success of exposure reduction measures. However, given the long biological half-life of PFCs, frequent blood monitoring is of no value.

enHealth recommends that:

- blood testing has no current value in informing clinical management; and
- the monitoring of pooled community blood samples over time may help determine the success of exposure reduction measures.

Australian Health Protection Principal Committee

Perfluorinated Chemicals (PFCs) FactSheet

What are perfluorinated chemicals?

Perfluorinated chemicals, also known as “PFCs”, are a group of manufactured chemicals that have been used since the 1950s in a range of common household products and specialty applications, including in the manufacture of non-stick cookware; fabric, furniture and carpet stain protection applications; food packaging; some industrial processes; and in some types of fire-fighting foam.

There are many types of PFCs. The best known examples are:

- perfluorooctane sulfonate, also known as “PFOS”; and
- perfluorooctanoic acid, also known as “PFOA”.

Are these chemicals manufactured or used in Australia?

The National Industrial Chemicals Notification and Assessment Scheme (NICNAS) has monitored PFOS and PFOA use in Australia through four national surveys, which show that these chemicals are not manufactured in Australia.

PFOS and related compounds are currently imported into Australia, mainly for use as mist suppressants in the metal plating industry, hydraulic fluid in the aviation industry and surfactants in the photography industry.

PFOA and related chemicals were previously imported into Australia and used in the local manufacture of non-stick cookware. These chemicals are not present in the finished cookware.

Until recently, PFOS and PFOA were added to some types of fire-fighting foam to improve the foam’s ability to smother fires. There are believed to be stockpiles of fire-fighting foams containing PFCs still in use.

PFOS and PFOA may be present in a range of imported consumer products, although many countries have phased out, or are progressively phasing out the use of PFOS and PFOA due to concerns about their persistence, bioaccumulation and environmental toxicity.

NICNAS has recommended since 2002 that Australian industries should actively seek alternatives to PFCs and PFC-related substances. The alternative chemicals should be less toxic and not persist in the environment.

Have PFOS and PFOA contaminated sites in Australia?

Currently there are investigations into environmental contamination with PFOS and PFOA at a number of sites around Australia. These include the Country Fire Authority training facility at Fiskville, Victoria; the RAAF Base at Williamtown, NSW; and the Army Aviation Centre at Oakey, Queensland.

The historic use of PFC-containing fire-fighting foams has resulted in areas within these sites becoming contaminated with PFOS and PFOA. Over the past decades, these chemicals have worked their way through the soil to contaminate surface and ground water, and have also migrated into adjoining land areas.

There are potentially other contaminated sites around Australia at which PFC-containing fire-fighting foams have been used, which are being investigated.

How do PFCs enter the environment?

In addition to contamination from the use of fire-fighting foams, PFCs can be released into the environment from landfill sites where products and materials that contain these chemicals are sent for disposal, and into ground and surface water through sewer discharges.

Manufacturing facilities that handle PFCs are also sources of PFC release into the environment.

The biggest environmental concern about PFOS and PFOA is that they do not break down in the environment and can travel long distances in water and air currents. They have been shown to be widespread global contaminants and many countries are now monitoring and restricting their use.

PFOS and PFOA have been shown to be toxic to some animals, and because they don't break down they can bioaccumulate and biomagnify in some wildlife, including fish. This means that fish and animals higher in the food chain may accumulate high concentrations of PFOS and PFOA in their bodies.

The toxicity, mobility, persistence and bioaccumulation potential of PFOS and PFOA pose potential concerns for the environment and for human health.

How could I be exposed to PFCs?

The general public are exposed to small amounts of PFOS or PFOA in everyday life through exposure to dust, indoor and outdoor air, food, water and contact with consumer products that contain these chemicals.

For most people, food is thought to be the most important source of exposure. Treated carpets and floors treated with waxes and sealants that contain PFCs can be an important source of exposure for babies and infants.

PFOS and PFOA are readily absorbed through the gut and are not metabolised or broken down in the body. These chemicals are only very slowly eliminated from the body. Studies have shown that Australians have small amounts of PFOS and PFOA in their blood. PFOS and PFOA can also be found in urine and breast milk.

People who work in industries that use PFOS and PFOA, or use products containing these chemicals, may be exposed to higher levels than the general public.

Where larger quantities of PFOS and PFOA have been released into the environment, communities located near those sites may be exposed to higher levels than the general public. It is important to understand how people living near contaminated areas may come into contact with PFOS and PFOA so that exposure may be minimised. This could include by examining in detail the pathways through which people could be exposed to these chemicals.

How do PFCs affect human health?

Whether PFOS or PFOA cause health problems in humans is currently unknown, but on current evidence from studies in animals the potential for adverse health effects cannot be excluded. Because the elimination of PFCs from the human body is slow there is a risk that continued exposure to PFOS and PFOA could cause adverse health effects.

Adverse health effects have been demonstrated in animal studies, but at higher levels than are found in people. As well, the applicability of the effects in animals to humans is not well established.

Much of the research on humans has been done with people who were exposed to relatively high levels of PFCs through their work. Workers involved in the manufacture or use of PFCs usually have higher blood PFC levels than the general public. Studies on PFC workers have looked for effects on cholesterol levels, male hormones, heart disease, liver changes and other effects, including cancer. These studies have not consistently shown that PFC exposure is linked to health problems.

As a precaution, people living in or near an area that has been identified as having been contaminated with PFOS or PFOA should take steps to limit their exposure to these chemicals. Your state or territory health department can provide you with advice on how to limit your exposure to PFOS and PFOA specific to your location and circumstances.

Can PFOS or PFOA cause human cancers?

In humans, there is no conclusive evidence that PFCs cause any specific illnesses, including cancer.

Studies in laboratory animals suggest that PFOS and PFOA may cause some cancers in those animals following prolonged exposure to relatively high levels. However, no existing studies have found a causal link between exposure to PFOS and PFOA and cancer in humans.

Studies of workers involved in the manufacture or use of PFOS and PFOA have looked at whether there is any link between these chemicals and the development of prostate, bladder and liver cancer in humans. There have been no consistent findings in these studies.

The International Agency for Cancer Research (IARC) has classified PFOA as possibly causing some cancers. Other studies have concluded that the evidence does not support an association between human cancer and either PFOS or PFOA exposure.

Does exposure to PFCs during pregnancy pose an increased health risk?

PFOS and PFOA are not known to cause adverse health effects on unborn babies. However, as a precaution, pregnant women living in or near an area that has been identified as having been contaminated with PFOS or PFOA should take steps to limit their exposure to these chemicals.

Your state or territory health department can provide you with advice regarding PFOS and PFOA specific to your location and circumstances.

Should I breastfeed if I have been exposed to PFCs?

Although there is evidence that PFOS occurs in breast milk, it is unclear what, if any, the risks to the baby may be from PFOS or PFOA exposure through breast milk.

The significant health benefits of breast feeding are well established and far outweigh any potential health risks to an infant from any PFOS or PFOA transferred through breast milk.

Breast feeding of babies should not be discontinued due to concerns about PFOS and PFOA exposure.

Should I get a blood test if I think I have been exposed to PFOS or PFOA?

Blood tests are not recommended to determine whether any medical condition is attributable to exposure to PFOS or PFOA and have no current value in informing clinical management, including diagnosis, treatment or prognosis in terms of increased risk of particular conditions over time.

The value of blood testing is limited to assessing exposure at a population level, such as monitoring over time, which may help determine the success of exposure reduction measures. However, given the long biological half-life of PFCs, frequent blood monitoring is of no value.

If you think you have been exposed to PFOS or PFOA and you have any health concerns, please consult your general practitioner.

Are blood tests useful at a population level?

Various organisations around the world have collected blood samples from people as part of ongoing investigations into PFC contamination of soil and water. The purpose of these tests was either as part of a defined research program, or to determine how much of these chemicals may be entering a person's body.

A blood test can tell a person if they have PFOS or PFOA in their blood and at what levels. These levels can be compared with the levels seen in the general Australian population.

Blood tests can also inform a community if they have been exposed to PFCs at a level above or below that of the general population.

The monitoring of pooled community blood samples over time may help determine the success of exposure reduction measures.