

COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

COT statement on the potential risks from perfluorooctane sulfonate (PFOS) in the infant diet

Background

1. The Scientific Advisory Committee on Nutrition (SACN) is undertaking a review of scientific evidence that bears on the Government's dietary recommendations for infants and young children. The review will identify new evidence that has emerged since the Government's current recommendations were formulated, and will appraise that evidence to determine whether the advice should be revised. The recommendations cover diet from birth to age five years, but will be considered in two stages, focussing first on infants aged 0 – 12 months, and then on advice for children aged 1 to 5 years. SACN is examining the nutritional basis of the advice, and has asked that evidence on possible adverse effects of diet should be considered by other advisory committees with relevant expertise. SACN asked COT to review the risks of toxicity from chemicals in the infant diet.

2. This statement gives an overview of the potential risks from perfluorooctane sulfonate (PFOS) in the infant diet. None of Government's current dietary recommendations for infants and young children relates to PFOS.

3. PFOS belongs to a large class of chemicals known as perfluorinated alkyl compounds. It has surfactant properties and has been used extensively in the manufacture of plastics, electronic equipment and textiles. PFOS is widely distributed in the environment and since 2009 has been designated as a Persistent Organic Pollutant under the Stockholm Convention (UNEP, 2009). This designation requires that usage must be phased out, but some uses have been allowed to continue until suitable alternative products are available.

4. Within the EU, all uses other than in certain metal (chromium) plating processes are being phased out. In the UK, PFOS-containing stockpiles for use in non-decorative hard chrome plating have been notified to the relevant authorities. In 2012, a total 3,654 kg of PFOS-containing material, equating to 88 kg of PFOS, was notified by four companies. Information from the manufacturer suggests that these quantities will diminish in the future since alternatives are now being used and products reformulated (Personal communication, Defra 2013).

5. Because it is widely distributed in the environment, PFOS occurs in food (FSA, 2009; EFSA, 2008). Fish and fish products seem to be an important source of human exposure to PFOS (EFSA, 2008).

6. PFOS has the potential to cause a range of adverse health effects, including hepatotoxicity, developmental toxicity, neurobehavioral toxicity, immunotoxicity, reproductive toxicity, lung toxicity, hormonal effects and carcinogenicity. Evaluations of PFOS in food have been conducted by the COT¹ and the European Food Safety Authority (EFSA) (EFSA, 2008). This statement draws on information from those reviews and in addition provides estimates of the exposures of infants to PFOS from breast milk, infant formula, complementary foods and non-dietary sources.

Previous evaluations by COT and EFSA

COT

7. In a 2006 statement, the COT concluded that PFOS has the potential to cause a range of adverse health effects. Given the bioaccumulative properties of the chemical, the Committee considered that health-based guidance values would ideally be set in relation to body burden, but knowledge of the toxicokinetics of PFOS did not allow adequate estimation of the body burden.

8. As an alternative, the COT proposed a provisional tolerable daily intake (TDI) of 300 ng/kg bw/day for PFOS, based on a study by Seacat *et al.* (2002), in which PFOS was administered by gavage to cynomolgus monkeys at 0, 0.03, 0.15, or 0.75 mg/kg bw/day for 182 days. Treatment-related effects at the highest dose were increased liver weights and decreased haemoglobin levels. In addition, changes in serum thyroid hormone levels were reported at 0.15 and 0.75 mg/kg bw/day. The Committee considered that the no observed adverse effect level (NOAEL) in this study was 0.03 mg/kg bw/day, based on the totality of the data from the analysis of thyroid hormone levels at higher doses. The Committee noted that pharmacokinetic data indicated an elimination half-life of between 110 and 180 days, and therefore tissue levels in the monkeys would have reached approximately half their steady state by the end of the study. However, taking into account that the study was in primates and the effects were mild, the COT concluded that it was not necessary to apply an additional uncertainty factor to allow for the incomplete attainment of steady state. The Committee therefore applied the usual uncertainty factor of 100 to allow for inter- and intra-species variation to the NOAEL of 0.03 mg/kg bw/day to derive the TDI of 300 ng/kg bw/day. This value was considered provisional and to be reviewed as new information became available.

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<http://cot.food.gov.uk/cotstatements/cotstatementsyrs/cotstatements2006/cotstatementpfos200609>

9. In 2006, the COT noted that the results of a Food Standards Agency (FSA) analysis of composite food group samples from the 2004 Total Diet Study (TDS) indicated that high level consumers aged 1.5-6 years might exceed the TDI. However, there were major uncertainties in the estimates of dietary exposure because of the conservative approach adopted to samples that did not contain detectable levels of PFOS. Thus these potential exceedances were judged not to present an immediate toxicological concern.

10. The COT statement made no specific reference to infants. No exposure assessment was performed for infants and no occurrence data were reported for levels of PFOS in breast milk.

EFSA

11. EFSA (2008) concluded that it was not possible to assess fully the relative contribution of different foodstuffs to human exposure to PFOS because data were insufficient. However based on the information that was available, one important source seemed to be fish and fish products.

12. Using occurrence data for PFOS in fish, fish products and drinking water, and consumption data from four Member States, the EFSA derived indicative exposures of 60 and 200 ng/kg bw/day for average and high level consumers of fish and fish products. However, the occurrence data that underpinned these estimates may have over-represented fish from more polluted areas. Non-food sources of PFOS were estimated to contribute approximately 2% of average dietary exposure, and drinking water less than 0.5%.

13. PFOS blood and tissue levels measured in humans were considered not necessarily to reflect exposure to PFOS from food and non-food sources, since there were also a number of potentially important precursors that could be transformed into PFOS in the body. However, there was no information on human exposure to such precursors, on their rate of transformation in the body, or on their occurrence in food.

14. Like the COT, EFSA identified a NOAEL of 0.03 mg/kg bw/day from the study by Seacat *et al.* (2002) in cynomolgus monkeys as the point of departure for their assessment of risk. An uncertainty factor of 100 was applied to cover possible inter- and intra-species differences, and an additional factor of 2 “to compensate for uncertainties in connection to the relatively short duration of the key study and the internal dose kinetics”. Hence EFSA established a TDI for PFOS of 150 ng/kg bw/day (EFSA, 2008). EFSA noted that the indicative dietary exposure of 60 ng/kg bw/day was below the TDI, but that the most highly exposed people in the general population might slightly exceed this TDI.

15. EFSA also noted that the margins between serum levels in monkeys at the NOAEL and serum levels in the general population of European countries were between 200 and 3,000. Given these margins, EFSA considered it

unlikely that adverse effects of PFOS were occurring in the general population.

16. Occurrence data for PFOS in breast milk (0.060-0.470 (mean, 0.201) ng/mL) in the area of Uppsala, Sweden (Kärman *et al.*, 2007) were used to estimate PFOS exposures of infants from breast milk (approximately 9.6-75 (mean, 32) ng/kg bw/day), but with recognition that this might not be representative of exposures in other regions (EFSA, 2008).

Differences between the TDIs set by COT and EFSA

17. The TDIs proposed by COT and EFSA were both based on a NOAEL of 0.03 mg/kg bw/day in the study by Seacat *et al.* (2002), which showed changes in serum thyroid hormone levels at higher doses. The difference in TDI values arose from an additional uncertainty factor of 2 applied by EFSA. The EFSA report noted that this additional uncertainty factor was “to compensate for uncertainties in connection to the relatively short duration of the key study and the internal dose kinetics”, but gave no further explanation.

18. In 2009, as part of a review of perfluorooctanoic acid (PFOA) in drinking water, the COT confirmed its TDI of 300 ng/kg bw/day for PFOS².

New data

19. Since the previous COT statement and EFSA opinion were published further toxicokinetic, toxicological and epidemiological studies have been carried out. EFSA has commissioned a review of the new data, and will consider whether there is a need to revisit its previous evaluation when the review is received.

Sources of PFOS exposure

20. Due to the persistence of PFOS in the environment, humans can be exposed directly via food, dust, air and water. In addition, indirect exposure to PFOS can occur through exposure to PFOS precursors (PreFOS), if these are then converted to PFOS in the body.

PFOS precursors

21. The Organisation for Economic Co-operation and Development (OECD) lists 165 PFOS-related substances (OECD, 2007). PreFOS are higher molecular weight derivatives of PFOS with the potential to degrade to the latter. They are either manufactured intentionally, or occur as residual contaminants in manufactured products (Table 1) (Martin *et al.*, 2010).

² <http://cot.food.gov.uk/cotstatements/cotstatementsyrs/cotstatements2009/cot200902>

22. Much of the environmental burden of PFOS and a proportion of the human body burden derives from the degradation of PreFOS compounds. Paul *et al.* (2009) estimated that the worldwide maximum direct emissions of PFOS to the environment during 1970-2002 were 450-2700 tonnes compared to 6800-45250 tonnes for PreFOS. PreFOS compounds can be converted to PFOS by both biotic and abiotic mechanisms. Quantification of the metabolism of PreFOS to PFOS within the body is complicated by the large number of Pre-FOS compounds that may be involved. Studies *in vivo* and *in vitro* have demonstrated the formation of PFOS from PreFOS, but the pharmacokinetics of PreFOS at low doses remains an area of much uncertainty (Martin *et al.*, 2010). It has been reported that in adults, daily intakes of PreFOS were slightly higher than for PFOS, with intake of PFOS intake being dominated by the diet, while for PreFOS, indoor air, house dust and diet were all important contributors (Fromme *et al.*, 2009).

Table 1. Some of the PFOS precursors known to have been intentionally manufactured or present as residuals in manufactured products (as listed in Martin *et al.*, 2010).

Precursor name	acronym
Perfluorooctanesulfonyl fluoride	POSF
Perfluorooctanesulfinate	PFOSI
Perfluorooctanesulfonamido alkyl amine oxide salts	
N-ethyl perfluorooctanesulfonamide	NEtFOSA
N-ethyl perfluorooctanesulfonamidoethanol	NEtFOSE
N-methyl perfluorooctanesulfonamidoacetate	NMeFOSAA
Perfluorooctanesulfonamidoacetate	FOSAA
di-NEtFOSE phosphate [mono- and tri- also manufactured]	
NEtFOSE acrylate	
NEtFOSE methacrylate	
Urethane-linked NMeFOSE	
Perfluorooctane sulphonamide	PFOSA
Perfluorooctanesulfonamido propanimium salts	
N-methyl perfluorooctanesulfonamide	NMeFOSA
N-methyl perfluorooctanesulfonamidoethanol	NMeFOSE
Perfluorooctanesulfonamidoethanol	FOSE
N-ethyl perfluorooctanesulfonamidoacetate	NEtFOSAA
di-NMeFOSE phosphate	
NMeFOSE acrylate	
NMeFOSE methacrylate	
Ester-linked NMeFOSE methacrylate	

Dust and soil

23. PFOS was measured in indoor dust collected by vacuum cleaning in cars (n=20), classrooms (n=42), homes (n=45) and offices (n=20) in Birmingham, UK, between 2007 and 2009. PFOS was detected at concentrations of 20-1500 (mean, 260; median 97), 22-3700 (mean, 980; median 840), 3.5-7400 (mean, 450; median 140) and 20-1000 (mean, 370; median 230) ng/g in cars, classrooms, homes and offices, respectively. (Goosey and Harrad, 2011). The mean levels detected in cars, homes and offices were similar to those described in reports evaluated by EFSA that related to homes in Japan and Canada (EFSA, 2008). However mean PFOS levels in classrooms were higher than those reported in homes in the EFSA opinion.

24. No data were available on measured levels of PFOS in soil in the UK.

Food contact materials

25. In a study by Jogsten *et al.* (2009) a higher PFOS concentration was detected (mean and standard deviation (SD)) ng/g fresh weight) in packaged lettuce (0.034, SD 0.012) than in unpackaged lettuce samples (0.010, SD 0.007). Similarly, the PFOS level in packaged marinated salmon (0.054, SD 0.055) was higher than that found in home-produced marinated salmon (0.026, SD 0.015) (Jogsten *et al.*, 2009). These studies suggest that food packaging could be a source of PFOS exposure, but no studies were found that directly measured the levels of PFOS migration from food packaging into food.

Drinking water

26. Drinking water is not routinely monitored for PFOS, but the Drinking Water Inspectorate (DWI) specifies that water companies should ensure that PFOS is adequately addressed in their risk assessments, and that if appropriate, they should consider initiating monitoring for PFOS at their works. The DWI has established a tiered approach for monitoring levels of PFOS in drinking water. Guidance levels are set for water companies to take increasing action at PFOS levels >0.3 µg/L, >1.0 µg/L and >9.0 µg/L (DWI, 2009). The value of 0.3 µg/L is based on allocation of 10% of the COT TDI to 1 litre of drinking-water consumed daily by a one-year-old child weighing 10 kg.

27. In a survey of PFOS in drinking water at 20 UK sites, including 15 sites deemed to have a greater likelihood of elevated PFOS levels (for example, because of proximity to airfields; semi-conductor industries; carpet or textile manufacturers; and chrome (VI) plating industries) and five control sites (rural areas, with no perceived PFOS sources nearby). PFOS was not present at the limit of detection (LOD) of 0.011 µg/L at the control sites. Among the 15 sites at which higher levels of PFOS might be expected, PFOS was detected at only four (at concentrations of 0.012-0.208 µg/L). Concentrations were

similar in repeated samples and showed no clear relationship to potential source of PFOS, water treatment process, or the season (Atkinson *et al.*, 2008).

Air

28. Chaemfa *et al.* (2010) measured levels of PFOS in the air at 22 locations in the UK in 2006/2007. PFOS was below the LOD of 0.6 pg/m³ at 13 of the locations. The range of PFOS measurements at the remaining nine sites was 1.0 to 111 pg/m³ (Chaemfa *et al.*, 2010). PFOS was measured in 10 different outdoor locations within a 1.5 km radius of the University of Birmingham campus in March 2009. PFOS concentrations were <1.0-6.1 (mean, 2.3; median, 1.5) pg/m³ (Goosey and Harrad, 2012).

29. Barber *et al.* (2007) measured PFOS in outdoor air at two locations in the UK in November 2005–February 2006, as part of a study analysing per- and polyfluorinated alkyl substances in air samples from Northwest Europe. Air (500-1800 m³) was sampled over 3-14 days using high volume samplers, and analysed by liquid chromatography-time of flight mass spectrometry. The arithmetic mean concentrations of PFOS in the air were 1.6 and 7.1 pg/m³ at Hazelrigg (Lancaster) and Manchester, respectively (Barber *et al.*, 2007).

Dietary occurrence of PFOS

Breast milk

30. Levels of PFOS have been measured in human milk in a number of studies, of which three (Barbarossa *et al.*, 2013; Kadar *et al.*, 2011; Croes *et al.*, 2012), which analysed samples from European countries in the last six years (2008 or later), were considered most relevant (Table 2). A median value of 74 ng/L was obtained in the study by Kadar *et al.* (2011) and the highest concentration of 288 ng/L was measured in the study by Barabrossa *et al.* (2013). No data were available on PFOS levels in breast milk of women from the UK.

31. Sundstrom *et al.* (2011) reported temporal trends in levels of PFOS in pooled samples of breast milk from 1972 to 2008. PFOS levels increased from 1972 (23 ng/L) through to the late 1990s (234 ng/L in 1999). Levels then remained similar until 2001, after which they decreased through to 2008 (75 ng/L). The PFOS levels measured in this study are consistent with those measured at similar times in other European countries (Kärroman *et al.*, 2007; Völkel *et al.*, 2008; Fromme *et al.*, 2010; Kärroman *et al.*, 2010).

Table 2. Concentrations of PFOS in breast milk in recent EU studies

Region, country	Year of sampling	No. of samples	Mean (SEM) (ng/L)	Median (ng/L)	Range (ng/L)	Reference
Barcelona, Spain	2009	20	116 (42)	84	< LOQ-865	Llorca <i>et al.</i> , 2010
France	2010	30	78	74	24-171	Kadar <i>et al.</i> , 2011
Belgium	2009- 2010	40 (P & M)	130	NR	NR	Croes <i>et al.</i> , 2012
Bologna, Italy	2010	21 (P)	57 (13)	NR	<15-288	Barbarossa <i>et al.</i> , 2013
		16 (M)	36 (7)	NR	<15-116	

P – primiparous; M – multiparous; SEM – standard error of the mean; NR – not reported

Infant formulae

32. Only three studies were found in which PFOS was measured in infant formulae (Table 3). PFOS was not found above the limit of quantitation (LOQ) of 10 ng/L in any of the samples from the study by Fromme (2010), and only one sample had a detectable level (11.3 ng/L) in the study by Tao (2008) (LOQ 11.0 ng/L). Higher concentrations were reported by Llorca *et al.* (2009), who noted that because of the scarcity of available data it was difficult to explain the reason for the differences.

Table 3. Concentrations of PFOS in infant formulae

Region, country	Year of sampling	No. samples	Mean PFOS (ng/L)	Range (ng/L)	Reference
Munich, Germany	2007-2009	4 Not detected in any samples	< 10	< 10 (reconstituted)	Fromme <i>et al.</i> , 2010
Washington D.C, U.S.A.	2007	21 only detected in 1 sample	<11	<11-11.3 (reconstituted)	Tao <i>et al.</i> , 2008
Barcelona, Spain	2009	3	577 (86*)	229-1098 (34-165*)	Llorca <i>et al.</i> , 2010

*Calculated from data for powdered formula in the original paper, with the assumption that the powder accounted for 15% of the total volume of reconstituted formula.

Complementary foods³

33. Llorca *et al.* (2010) reported PFOS concentrations of 0.162 and 0.458 µg/kg in two samples of non-reconstituted baby food cereal purchased in Spain in 2009.

³ Solid foods introduced into the infant diet to complement the milk feed, which remains the predominant part of the infant diet for most of the first year of life.

34. In a TDS of perfluorinated chemicals in retail samples of foods on sale in the UK (Rose and Robinson, 2012), PFOS was detected in all food groups at concentrations ranging from 0.02 µg/kg to 2.66 µg/kg. The highest concentrations were in offal and fish samples (Table 4).

Table 4. Concentrations of PFOS in composite UK food samples

Product	Number of samples	PFOS (µg/kg)
Bread	29	0.1
Cereals	40	0.09
Carcass meat	51	0.22
Offal	85	2.66
Meat products	123	0.17
Poultry	51	0.16
Fish	140	0.96
Fats and oils	84	0.15
Eggs	34	0.31
Sugars and preserves	30	0.08
Green vegetables	23	0.1
Potatoes	23	0.05
Other vegetables	40	0.04
Canned vegetables	15	0.03
Fresh fruit	23	0.07
Fruit products	15	0.02
Milk	44	0.05
Milk and dairy	102	0.06
Nuts	34	0.1

Exposure to PFOS

35. The assessments of exposure from air, soil and dust, and the diet that are presented in this section relate to external exposure. Bodyweight data were taken from the UK Dietary and Nutrition Survey of Infants and Young Children (DNSIYC) (DH, 2013), in which the average bodyweights were 7.8, 8.7 and 9.6 kg for infants aged 4.0-<6.0, 6.0-<9.0 and 9.0-<12.0 months old, respectively. Since DNSIYC did not include infants younger than 4 months, in this statement a value of 5.9 kg for infants aged 0-3 months, from an earlier survey (DH, 1994), is assumed for infants aged 0-4 months.

Relative importance of PFOS and PFOS precursors

36. Vestergren et al. (2008) used a scenario-based risk assessment approach to model exposures to PFOS and PreFOS from various different pathways, and to estimate daily doses resulting from uptake into the human body. They illustrated the range of exposures to PFOS and PreFOS and resulting internal doses of PFOS by generating low, intermediate and high exposure scenarios. These took the fifth percentile, median and 95th percentile value, respectively, for each individual input parameter. In the low, intermediate and high exposure scenarios, biotransformation factors for PreFOS to PFOS were set at 0.002, 0.005 and 0.017 for one PreFOS

compound, and at 0.01, 0.2 and 1.0 for others, reflecting the large uncertainty in this parameter. In the low exposure scenario, direct exposures to PFOS accounted for virtually 100% of the dose in both infant and toddler groups. In the intermediate scenario, these groups received up to 4% of their PFOS dose from precursors. However, in the high exposure scenario, the contribution of PreFOS increased to 78% for infants and 68% for toddlers. The overall conclusion of the study was that precursor compounds made only a minor contribution to the daily dose of PFOS. This, however, remains uncertain. Moreover, it refers to the population as whole and not to individuals at the upper extreme of the distribution. The topic continues to be an active area of research. Vestergren et al. (2008) reported that food and drinking water were the dominant pathways for exposure to PFOS (and by implication its precursors).

Soil and dust

37. Potential exposures of infants to PFOS through ingestion of soil/dust were calculated assuming ingestion of 100 mg dust/day (WHO, 2007) containing PFOS at the mean concentration measured in UK homes of 450 ng/g. Calculations were for an infant aged 9.0-12.0 months (with an assumed bodyweight of 9.6 kg (DH, 2013)), since infants are likely to consume more dust at this age than when they are younger and less able to move around. The estimated exposure from ingestion of soil/dust, based on the mean dust concentration, was 4.69 ng/kg bw/day.

Air

38. Potential exposures of UK infants to PFOS in air were calculated assuming a ventilation rate of 3 m³/day (US EPA, 1989), and airborne PFOS concentrations of 0.23-111 pg/m³ as measured in the UK in 2006/7. The resultant exposure estimates ranged from 0.000072 to 0.056 ng/kg bw/day. (Table 5).

Table 5. Estimated UK infant exposure to PFOS (ng/kg bw/day) from the air

PFOS concentration (pg/m ³)	Age (months)			
	0-<4.0	4.0-<6.0	6.0-<9.0	9.0-12.0
0.23	0.00016	0.000089	0.000079	0.000072
111	0.056	0.043	0.038	0.035

Diet

Breast milk

39. Based on the highest reported median and maximum PFOS levels in breast milk sampled over the past 5 years from European women (74 and 288

ng/L, respectively) (Table 2), PFOS exposures were estimated for exclusively breastfed infants consuming average (800 mL) and high (1200 mL) volumes of breast milk daily (Table 6). Estimated levels of exposure for exclusively breastfed infants ranged from 7.6 to 59 ng/kg bw/day.

Table 6. PFOS exposure (ng/kg bw/day) from exclusive breastfeeding of infants estimated for average and high level consumption of breast milk

PFOS concentration in breast milk (ng/L)	Age in months (consumption volume per day)			
	0-4.0 (800 mL)	0-4.0 (1200 mL)	>4.0-6.0 (800 mL)	>4.0-6.0 (1200 mL)
84 (median from Llorca <i>et al.</i> , 2010)	11	17	8.6	13
288 (maximum from Barbarossa <i>et al.</i> , 2013)	39	59	30	44

Infant formulae

40. Mean and maximum PFOS concentrations in reconstituted infant formulae of 86 and 165 ng/L, calculated from the study by Llorca *et al.* (2010), were used to estimate exposures in exclusively formula-fed infants (Table 7). These estimates did not include any PFOS present in the drinking water used for reconstitution. Basing the estimates on the data from Llorca *et al.* (2010) is likely to be highly conservative, since other studies have reported much lower concentrations of PFOS in infant formulae.

Table 7. PFOS exposure (ng/kg bw/day) from exclusive feeding of infant formulae, estimated for average and high level consumption of milk, but excluding contribution from water used in reconstitution.

PFOS concentration in reconstituted infant formula (ng/L)	Age in months (consumption volume per day)			
	0-<4.0 (800 mL)	0-<4.0 (1200 mL)	4.0-<6.0 (800 mL)	4.0-<6.0 (1200 mL)
Mean – 86	11.7	17.5	8.82	13.2
Maximum - 165	22.4	33.6	16.9	25.4

41. In addition, infants could be exposed to PFOS through drinking water used to reconstitute infant formula. Exposure values from water were estimated using the minimum and maximum concentrations recorded in water sampled in the UK (paragraph 27). Calculated additional exposures from PFOS in water ranged from 0.96 to 36.0 ng/kg bw/day (Table 8), compared to the 8.82 to 33.6 calculated for the formula powder. Thus water used in reconstituting infant formula has the potential to more than double the PFOS exposure of formula-fed infants, resulting in a total of up to 70 ng/kg bw/day. However, since the water with the maximum reported level of PFOS was from a site selected because it was thought more likely to have elevated PFOS

levels, and PFOS was not detected at a number of other sites (including some that also were expected to have higher levels), exposure from water would be a minor contributor in most cases.

Table 8. Possible additional PFOS exposure of exclusively formula fed infants through use of drinking water to reconstitute the formula (ng/kg bw/day).

PFOS concentration in drinking water (ng/L)	Age in months (consumption volume per day)			
	0-<4.0 (800 mL)	0-<4.0 (1200 mL)	4.0-<6.0 (800 mL)	4.0-<6.0 (1200 mL)
Control level (< 11)	< 1.27	< 1.9	< 0.96	< 1.44
Maximum level from water sampled near a PFOS source (208)	24.0	36.0	18.1	27.2

The exposure is calculated assuming that water accounts for 85% of the total volume of reconstituted formula.

Complementary foods

42. Data on consumption from the DNSIYC together with those on occurrence of perfluorinated chemicals from the UK TDS (Table 4) were used to estimate dietary exposures of infants to PFOS from complementary foods (Table 9). Mean and 97.5th percentile exposures ranged from 0.03 ng/kg bw/d to 0.84 ng/kg bw/d and from 0.13 ng/kg bw/d to 5.84 ng/kg bw/d respectively. Consumption of dairy products, fish and offals resulted in the highest exposures. The overall mean and 97.5th percentile exposures from consumption of all foods combined were 3.95 ng/kg bw/d and 9.34 ng/kg bw/d respectively. These estimated exposures are for foods prepared in the home and not for commercially produced infant-specific foods.

Table 9. Estimated exposures of UK infants to PFOS from complementary foods (ng/kg bw/day).

Food Group	Exposure	
	Mean	97.5 th percentile
Dairy products	0.84	5.84
Fish	1.45	4.78
Offals	1.72	4.52
Milk	1.07	3.40
Eggs	0.44	1.54
Carcase meat	0.36	1.50
Misc cereals	0.39	1.35
Fresh fruit	0.41	1.31
Meat products	0.28	0.96
Poultry	0.22	0.76
Bread	0.21	0.66
Green vegetables	0.17	0.65
Potatoes	0.17	0.57
Other vegetables	0.12	0.40
Canned vegetables	0.07	0.29
Fruit products	0.04	0.23
Sugars	0.05	0.19
Fats and oils	0.03	0.13
Nuts	0.03	0.13
Overall exposure from all groups*	3.95	9.34

* Whereas the overall exposure is based on all survey respondents who consumed food in each group, the exposure from a particular food group is based only on individuals who ate the foods represented by the group. The number of survey respondents who ate a food may differ by food group. Therefore, the sum of the means or 95th percentile exposure for each individual food group will not equal the estimated mean for overall exposure.

43. EFSA (2012) estimated the exposures of infants to PFOS using consumption data from dietary surveys in Bulgaria and Italy (the latter included very few participants) and occurrence data submitted by 13 European countries on foods sampled during 2006-2011. The lower bound to upper bound⁴ range of exposures calculated at the mean was 0.29-11 ng/kg PFOS bw/day and at the 95th percentile was 0.7-12 ng/kg PFOS bw/day. The highest contributors to dietary PFOS exposure across all age classes were 'Fish and other seafood' (50 to 80 %) followed by 'Fruits and fruit products' (8 to 27 %) and 'Meat and meat products' (5 to 8 %).

Risk characterisation

44. In this assessment, the highest estimated exposures of infants to PFOS before introduction of complementary feeding were 59 and 70 ng/kg

⁴ Lower bound assigns the value of zero to non-quantified data, upper bound assigns the value of the LOD/LOQ.

bw/day from breast milk and infant formula (including water used in reconstitution), respectively. An estimated additional contribution of 0.056 ng/kg bw/day from air was negligible. For older infants, the highest estimated exposures were 4.69, 0.038 and 9.34 ng/kg bw/day from soil/dust, air and food, respectively. Precursors of PFOS are likely to make a minor contribution to exposure in most infants. The estimates of exposure were based on limited data sets, but indicate that total exposure of infants to PFOS is likely to be well below the TDI of 300 ng/kg bw/day set by the COT and also below the TDI of 150 ng/kg bw/day set by EFSA. Thus, they do not suggest a concern for infants' health.

Conclusions

45. PFOS was evaluated by the COT (in 2006) and EFSA (in 2008) who proposed TDIs of 300 and 150 ng/kg bw/day, respectively. The difference between these two values resulted from differences in the uncertainty factors applied to the same toxicological data. The COT confirmed its TDI in 2009. Since these evaluations were performed, new toxicokinetic, toxicological and epidemiological data have been published, which EFSA is reviewing, and which might lead to a change in its TDI.

46. PFOS precursors are higher molecular weight PFOS derivatives which have the potential to degrade to PFOS. Although present in the environment and in food, the exposure to PFOS precursors has been estimated to make a minor contribution to the daily dose of PFOS.

47. Although based on limited data, particularly for the UK, estimates of infant dietary exposures to PFOS are below the current COT and EFSA TDIs for the compound, and even when allowance is made for additional exposure to PFOS precursors, they do not indicate a need for formulation of dietary recommendations to protect the health of infants.

48. The COT conclusions on PFOS in the infant diet may need to be reconsidered in the future if the EFSA review results in a lowering of its TDI.

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Abbreviations

COT	Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment
Defra	Department for the Environment, Food and Rural Affairs
DNSIYC	Diet and Nutrition Survey of Infants and Young Children
DWI	Drinking Water Inspectorate
EFSA	European Food Safety Authority
FSA	Food Standards Agency
LOD	Limit of detection
LOQ	Limit of quantitation
NOAEL	No observed adverse effect level
OECD	Organisation for Economic Co-operation and Development
PFOA	Perfluorooctanoic acid
PFOS	Perfluorooctane sulfonic acid
PreFOS	PFOS precursors
SACN	Scientific Advisory Committee on Nutrition
SD	Standard deviation
SEM	Standard error of the mean
TDI	Tolerable Daily Intake
TDS	Total Diet Study
WHO	World Health Organization