Environment International 37 (2011) 687–693

Contents lists available at ScienceDirect

Environment International

journal homepage: www.elsevier.com/locate/envint

Characterisation of human exposure pathways to perfluorinated compounds — Comparing exposure estimates with biomarkers of exposure ☆

Line S. Haug [a,*], Sandra Huber [b], Georg Becher [a,c], Cathrine Thomsen [a]

[a] Norwegian Institute of Public Health, P.O. Box 4404 Nydalen, NO-0403 Oslo, Norway
[b] Norwegian Institute for Air Research (NILU), FRAM Centre, Hjalmar Johansens gate 14, NO-9296 Tromsø, Norway
[c] Department of Chemistry, University of Oslo, P.O. Box 1033 Blindern, NO-0315 Oslo, Norway

ARTICLE INFO

Article history:
Received 25 November 2010
Accepted 21 January 2011
Available online 18 February 2011

Keywords:
Perfluorinated compounds
Exposure pathways
Diet
Indoor environment
Intakes
Biomonitoring

ABSTRACT

Commercially used per- and polyfluorinated compounds (PFCs) have been widely detected in humans, but the sources of human exposure are not fully characterized. The objectives of this study were to assess the relative importance of different exposure pathways of PFCs in a group of Norwegians and compare estimated intakes with internal doses obtained through biomonitoring. Individual PFC intakes from multiple exposure sources for a study group of 41 Norwegian women were estimated using measured PFC concentrations in indoor air and house dust as well as information from food frequency questionnaires and PFC concentrations in Norwegian food. Food was generally the major exposure source, representing 67–84% of the median total intake for PFOA and 88–99% for PFOS using different dust ingestion rates and biotransformation factors of 'precursor' compounds. However, on an individual basis, the indoor environment accounted for up to around 50% of the total intake for several women. Significant positive associations between concentrations of PFCs in house dust and the corresponding serum concentrations underline the importance of indoor environment as an exposure pathway for PFCs. For breast-fed infants, breast milk was calculated to be the single most important source to PFCs by far. The estimated intakes were confirmed by comparing serum concentrations of PFOA and PFOS calculated using PK models, with the corresponding concentrations measured in serum. Even though food in general is the major source of exposure for PFCs, the indoor environment may be an important contributor to human exposure. This study provides valuable knowledge for risk assessment of PFCs and control strategies.

1. Introduction

Per- and polyfluorinated compounds (PFCs) have been used during the last 50 years in many commercial applications including surfactants, lubricants, paints, polishes, paper and textile coatings, food packaging and fire-retarding foams (Kissa, 2001). PFCs comprise a diverse class of chemicals consisting of an alkyl chain which is partly (poly) or fully (per) fluorinated and have different functional groups attached. Among the perfluorinated compounds are the perfluoroalkyl carboxylic acids (PFCAs), perfluoroalkyl sulfonic acids (PFSAs), perfluoroalkyl sulfonamides (FOSAs) and perfluoroalkyl sulfonamidoethanols (FOSEs), while the polyfluorinated compounds comprise e.g. fluorotelomer alcohols (FTOHs).

Concerns about the persistence and bioaccumulative properties of PFCs were raised when the widely used surfactant perfluorooctyl sulfonic acid (PFOS) was found to be ubiquitously distributed in wildlife and human populations worldwide (Houde et al., 2006; Lau et al., 2007). Long elimination half-lives have been observed for perfluorooctanoic acid (PFOA), perfluorohexane sulfonic acid (PFHxS) and PFOS in humans (Bartell et al., 2009; Hölzer et al., 2009; Olsen et al., 2007; Seals et al., 2010) and two recent studies indicated long elimination half-lives for perfluorononanoic acid (PFNA), perfluorodecanoic acid (PFDA) and perfluoroundecanoic acid (PFUnDA) as well (Freberg et al., 2010; Nilsson et al., 2010). Animal studies have demonstrated hepatotoxicity, developmental toxicity, immunotoxicity and hormonal effects (Lau et al., 2007). Thus, PFOS fulfills the

Abbreviations: AMAP, Arctic Monitoring and Assessment Programme; EFSA, European Food Safety Authority; FFQ, food frequency questionnaire; FOSA, perfluoroalkyl sulfonamide; FOSE, perfluoroalkyl sulfonamidoethanol; FTOH, fluorotelomer alcohol; LOQ, limit of quantification; MLR, multiple linear regression; POP, persistent organic pollutant; PFCA, perfluoroalkyl carboxylic acid; PFCs, per- and polyfluorinated compounds; PFOA, perfluorooctanoic acid; PFNA, perfluorononanoic acid; PFDA, perfluorodecanoic acid; PFUnDA, perfluoroundecanoic acid; PFHxS, perfluorohexane sulfonic acid; PFHpS, perfluoroheptane sulfonic acid; PFOS, perfluorooctane sulfonic acid; PFSA, perfluoroalkyl sulfonic acid; PK model, pharmacokinetic model; TDI, tolerable daily intake.

☆ Competing interest declaration: The authors declare they have no competing financial interests.

* Corresponding author at: Division of Environmental Medicine, Norwegian Institute of Public Health P.O. Box 4404 Nydalen, NO-0403 Oslo, Norway. Tel.: +47 21076549; fax: +47 21076686.

E-mail addresses: line.smastuen.haug@fhi.no (L.S. Haug), sandra.huber@nilu.no (S. Huber), georg.becher@fhi.no (G. Becher), cathrine.thomsen@fhi.no (C. Thomsen).

0160-4120/$ – see front matter © 2011 Elsevier Ltd. All rights reserved.
doi:10.1016/j.envint.2011.01.011

criteria of the Stockholm Convention on persistent organic pollutants (POPs) and has been included in the list of restricted chemicals in 2009 (Stockholm Convention on Persistent Organic Pollutants, 2009).

The large historical production volumes and widespread applications of PFCs also in consumer products represent a potential for contamination of the indoor as well as the outdoor environment and thereby also food and drinking water. Dietary exposure has been suggested to be the main exposure route of PFCs in adult general populations (Fromme et al., 2009; Trudel et al., 2008; Vestergren and Cousins, 2009), and we have previously reported significant associations between estimated dietary intakes of PFOA, PFOS and PFUnDA and the corresponding serum concentrations (Haug et al., 2010). In certain cases also contaminated drinking water has been shown to be a major source of exposure (Egeghy and Lorber, 2010; Emmett et al., 2006; Vestergren and Cousins, 2009). Further, the contribution from dust ingestion was estimated to be nearly as great as from food ingestion for 2 years old children in USA (Egeghy and Lorber, 2010). A recent review by Harrad et al. (2010) emphasized the importance of evaluating exposure from ingestion of house dust and inhalation of indoor air. Further, it has been demonstrated that FTOHs can be biodegraded to PFCAs (Nabb et al., 2007) and FOSA/FOSEs can biodegrade to PFSAs (Tomy et al., 2004). Thus, exposure to PFCAs and PFSAs can also occur through degradation of 'precursors' such as FTOHs and FOSA/FOSEs. The knowledge on the relative impact of precursors to the total exposure of PFCs in humans is limited. Vestergren et al. (2008) estimated the relative contribution of precursor compounds to be 2–8% of the total exposure for PFOA and PFOS in an intermediate scenario, but as high as 28–80% in a high-exposure scenario. To our knowledge, no studies have so far considered multiple exposure sources including dust, air and diet enabling comparison of different exposure pathways on an individual basis.

For the youngest children, consumption of breast milk might be an additional source of PFC exposure. Even though the concentrations of PFCs in breast milk are considered to be low compared to blood (Kärrman et al., 2007), Thomsen et al. (2010) showed that the intake of PFCs through breast milk is similar to the dietary intake for Norwegian adults.

The aims of this study were to estimate and compare individual intakes of PFCs from food, drinking water, dust ingestion and inhalation of precursors in indoor air in a group of Norwegian women, and explore relationships between the estimated intakes and the measured serum concentrations. Further, we wanted to calculate the PFC exposure of infants through consumption of breast milk as well as inhalation of indoor air and ingestion of house dust.

2. Materials and methods

2.1. Study subjects

A study group of 41 female volunteers from the Oslo area, Norway was established. (Characteristics are given in Supplemental Material Table S1). Informed consent was obtained from all the participants and the project was approved by the Regional Committee for Medical Research Ethics (S-0711a, 2.2007.260). Samples of house dust as well as indoor air from the women's residences were collected between February and May 2008. Details on the sampling procedures as well as the measured concentrations of PFCs are described in Haug et al. (submitted for publication). The women also donated a serum sample and completed a questionnaire covering demographic information, different life style factors as well as dietary habits. In addition, about half of the women provided a sample of breast milk ($n=19$).

2.2. Data collection

Blood serum (3.5–13 mL) was collected either by general practitioners or a medical laboratory technician at the Norwegian Institute of Public Health, between August 2007 and May 2008. Breast milk ($n=19$) was collected by the women themselves between August 2007 and September 2008. The mothers were provided pre-cleaned screw cap bottles (PE) and the breast milk was obtained by manual expression to avoid potential contamination by breast pumps. The mothers were free to collect the breast milk whenever they liked during the day and sampling could occur on consecutive days as long as the breast milk was frozen between each sampling. Between 30 and 100 mL breast milk was obtained from the women. The samples were stored at below −18 °C until analyses.

Based on the knowledge of decreasing PFC concentrations in breast milk during the lactation period, serum and breast milk samples should ideally have been collected within a short period of time for each woman. Due to practical reasons this was not possible for many of the mothers. As an experiment we back-calculated either the serum or the breast milk concentration as if the samples were collected within a two week period using the models for depuration obtained by Thomsen et al. (2010). Further, the adjusted concentrations were used in the calculations, but only minor changes were observed, both in the linear curves and in the correlation coefficients compared to the curves obtained using the measured concentrations. Thus, the measured concentrations have been used throughout the paper.

2.3. Chemicals analysis of serum and breast milk

The nineteen PFCs, twelve isotope labeled internal standards and all other chemicals used are described elsewhere (Haug et al., 2009b). The serum samples were analyzed according to a previously described method (Haug et al., 2009b), while the breast milk samples were prepared and analyzed using the method by Thomsen et al. (2010). The procedural blanks (serum; $n=6$ and breast milk; $n=3$) analyzed together with the samples, did not contain any of the PFCs above limit of quantification (LOQ). For quantification of PFOS, the total area of the linear and branched isomers was integrated. The relative amount of the branched PFOS isomers was also calculated.

High quality of the serum determinations was assured by analyzing three replicates of three different in-house quality control samples ($n=3\times3$) as well as human serum samples from an interlaboratory comparison study organized by Institute national de santé publique du Québec (Canada) for the Arctic Monitoring and Assessment Programme (AMAP) ($n=3$) (AMAP, 2008). All results from the AMAP interlaboratory comparison were within ±1 SD of the consensus concentrations. The variations in the three replicates of each in-house quality control sample were less than 12% (RSD).

For the breast milk samples high quality of the determinations was assured by analyzing an in-house quality control sample ($n=6$). The relative standard deviations of the six replicates were 6.1% for PFOA and 3.1% for PFOS, the only two PFCs detected in this sample. In addition, the laboratory participated in a proficiency test on determination of PFCs in breast milk and obtained concentrations within ±1 SD of the consensus value for all PFCs found above the LOQ (B. van Bavel, personal communication).

2.4. Calculation of PFC intakes

Due to limited data on concentrations of PFCs in Norwegian food, estimation of dietary intakes were possible only for PFOA, PFUnDA and PFOS, and as PFUnDA was not observed above LOQ in any of the dust samples, intake estimates are presented for PFOS and PFOA only. Information regarding calculation of the intakes is given in the Supplemental Material (text and Table S2). For the 41 women, individual intakes were estimated based on consumption of food