ESTIMATING CONSUMER EXPOSURE TO PFOS AND PFOA

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Introduction

Perfluorinated compounds (PFCs) are ubiquitous in the environment[1]. The two most commonly investigated classes of PFCs are perfluorinated sulfonates (PFSAs) and perfluorinated carboxylates (PFCAs), with perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) as the two most often measured compounds. PFOS as well as PFOA are moderately toxic to mammals[2,3], bioaccumulative[1] and very persistent to degradation[2,3]. They have been measured in outdoor and indoor air[4], water[5], house dust[4], animal tissue[6], human blood serum and human breast milk[7]. Current concentrations of PFOA in drinking water in New Jersey, for example, are already close to a guidance value of 0.04 ppb derived from toxicity studies[8].

Perfluorinated compounds (PFCs) have been in use since 50 years[9] as process aids mainly in the production of fluorochemicals (used in, e.g., non-sticking cookware), as surfactants in products such as fire fighting foams and as surface protection for, e.g., textiles[10]. The largest producer of PFOS ceased production in 2001[1], which led to a considerable decrease in the use of PFOS in consumer products. In contrast, PFOA is still used and may be found as residual in consumer products.

This study estimates in a comprehensive way the total consumer exposure to PFOS and PFOA, taking into account all relevant pathways that may lead to the uptake of these compounds into the human body[11].

Methods

Scenario-Based Risk Assessment (SceBRA)

In this study we model the uptake doses of PFOS and PFOA to consumers in North America and Europe by using a scenario-based approach that consists of the following steps: (1) analysis of the chemical's life cycle, (2) definition of exposure pathways and scenarios representing all relevant situations leading to human exposure, (3) calculation of uptake doses, (4) comparison of calculated doses with results from human screening studies, and (5) evaluation of risk. Steps one to four have been conducted in this study while step five has been omitted due to the lack of suitable toxicity data.

In this study we focus on consumer exposure whereas occupational exposure to PFCs is not investigated. Consumer exposure occurs via the consumption of contaminated food, the use of contaminated products (e.g., sprays) and the intake of contaminated residential (e.g., house dust) or environmental media (e.g., ambient air).

As oral pathways of exposure to PFOS and PFOA we considered the intake of contaminated food and house dust, the hand-to-mouth contact with treated clothes and upholstery, the mouthing of treated clothes, carpet, and upholstery (infants and toddlers only), and the intake of food contaminated by PFOA migrating from non-stick cookware. As inhalation pathways we modelled contact to contaminated indoor and ambient air and to impregnation spray droplets. As dermal pathways we included the contact to treated clothes, carpets, and upholstery, deposition of spray droplets on the skin during spraying and deposition of contaminated dust on the skin. For each exposure pathway three scenarios (low exposure based on 5th percentile values, intermediate based on median values, and high exposure based on 95th percentile values) were modelled. The total uptake dose was calculated by adding up doses resulting from all low-exposure, intermediate, and high-exposure scenarios, respectively.

Seven groups of consumers are considered in the assessment: infants (0-12 months), toddlers (1-4 years), children (5-11 years), female and male teenagers (12-20 years) and female and male adults (> 20 years). Exposure can occur via continuous or intermittent long-term contact and via three routes of exposure (oral, dermal, and inhalation). The resulting uptake doses are calculated on a per-day basis and given in units of ng/(kg_{bw}·day). Wherever possible, two data sets have been compiled to reflect the different exposure situations in North America and Europe. Due to the lack of data, the same data sets have been used for the occurrence of PFOS and PFOA in consumer products, environmental media and food (except for fish and drinking water). The

total uptake doses based on the SceBRA approach are compared to doses derived from human screening studies by applying a one-compartment pharmacokinetic model (PK model) and assuming steady state conditions for concentrations measured in human blood serum.

Results and discussion

Fig. 1 shows the total uptake doses of PFOS for North American and European consumer groups. The ranges are similar for North America and Europe spanning roughly two orders of magnitude from 3.2 ng/(kg_{bw}·day) to 220 ng/(kg_{bw}·day). Young consumers tend to receive the highest doses due to typical behaviour patterns like extensive hand-to-mouth contact with treated carpets and due to greater intake of food relative to their body weight. The low-exposure and intermediate scenarios are dominated by intake of contaminated food in all consumer groups (at least 97% contribution). The high-exposure scenarios, in contrast, are beside intake of contaminated food (between 29% and 82%) also influenced by contact with treated carpets (between 5% and 64%) and inadvertent ingestion of contaminated house dust (between 6% and 16%). All other pathways together contribute less than 1% to the total doses in the high-exposure scenario. The influence of dust ingestion on the total doses to teenagers and adults may be underestimated in the low-exposure and intermediate scenarios because their amounts of ingested dust spans several orders of magnitude with comparably low 5th percentile and median values. On the other hand, the importance of food intake may be overestimated in the low-exposure and underestimated in the high-exposure scenarios because we used only one data set in all scenarios to characterize food intake and did not take into account possible food intake patterns expressed in correlations between the amounts of food products consumed (e.g., somebody eats a lot of fish but no meat).
A sensitivity analysis performed for all input parameters reveals that the most influential parameters are all related to the food pathway. This is of no surprise, as the food pathway dominates the low-exposure and intermediate scenarios and still contributes between 29% and 82% to the uptake in the high-exposure scenarios.

Fig. 2 shows the total uptake doses of PFOA for all consumer groups in North America and Europe revealing the similarity in exposure to PFOA in both regions. The doses are lower than those of PFOS spanning a range of 0.4 ng/(kg_{bw}·day) to 130 ng/(kg_{bw}·day). The trend that younger consumers receive higher doses than older consumers is less accentuated than in the case of PFOS. The low-exposure and intermediate scenarios are dominated by the intake of contaminated food (at least 91%). The high-exposure scenarios are mainly influenced by the intake of food (between 14% and 26%), inadvertent ingestion of dust (between 8% and 31%), and by product-related pathways, namely hand-to-mouth contact with treated carpets (between 2% and 59%), ingestion of food that has been contaminated by PFC-treated food contact materials (between 2% and 16%), and inhalation of aerosol droplets during the use of PFOA containing impregnation sprays (between 4% and 61%).
A sensitivity analysis shows that all influential input parameters are related to the food intake pathway. Although the product-related exposure is more important in the case of PFOA, the dominating influence of the food pathway remains.

Our analysis shows that consumers in North America and Europe experience chronic exposure to PFOS and PFOA in similar ranges. Exposure results mainly from food intake; consumer products tend to contribute less to consumer exposure to these chemicals. The tendency of higher uptake doses of PFOS to consumers in North America and Europe might be surprising, as the main producer phased out the production of PFOS in 2001 whereas PFOA is still used in the manufacture of products. However, due to the much larger amounts of PFOS produced and its persistent nature, the concentrations measured in foods, such as fish, are still higher than concentrations of PFOA. Additionally, other producers (for instance in China and India) might continue to produce and use PFOS, leading to ongoing emissions into the environment. We also assume that carpets may still contain PFOS, leading to comparably higher doses of PFOS than of PFOA in this pathway.

The modelled dose levels are compared to total uptake doses derived from blood serum levels by applying a one-compartment PK model. This PK model was developed for adults so that a comparison is made for doses of PFCs to adults only. The variables used in the model, such as half-lives of the chemicals in the human body, are rather uncertain. Fig. 3 shows that the general tendency of higher uptake doses of PFOS is also reflected in the doses derived with the PK model. The uptake doses for PFOS based on SceBRA lie clearly within the dose

ranges calculated with the PK model. This may indicate that all relevant exposure pathways are covered by our modelling approach and that the scenarios tend to accurately represent human exposure to PFOS.
A comparison of the total uptake doses of PFOA based on scenarios, with the values derived from the PK model shows that both approaches result in similar dose ranges. However, the mean tendencies of the scenario-based approach are slightly lower than those derived with the PK model. This could indicate that our scenarios do not accurately represent all aspects of human exposure to PFOA or that our data sets are not representative of actual exposure conditions.

References

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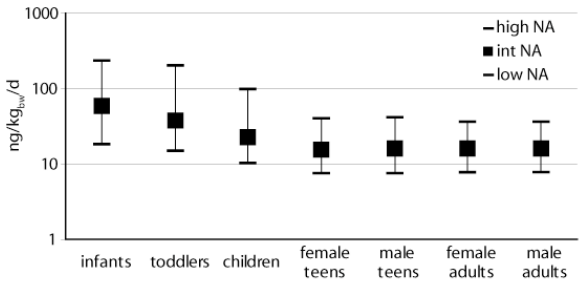
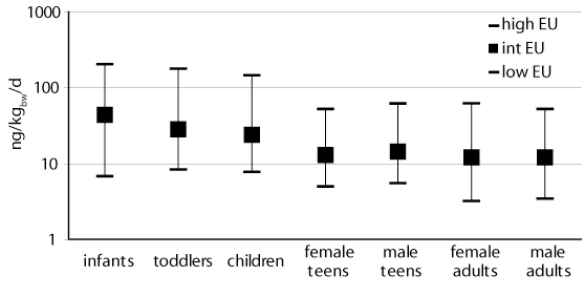
Figure 1: Total uptake doses of PFOS in North America (NA) and Europe (EU)

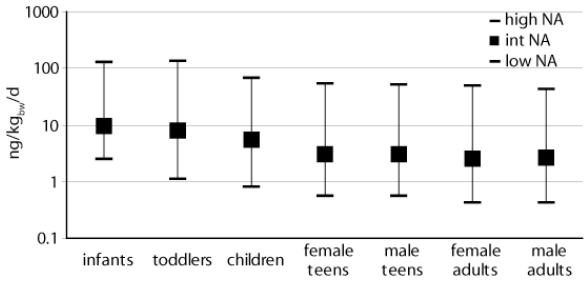


	infants	toddlers	children	female teens	male teens	female adults	male adults
high	220	200	100	32	34	30	30
int	55	35	22	15	16	15	15
low	18	15	10	7.9	7.2	7.1	7.0

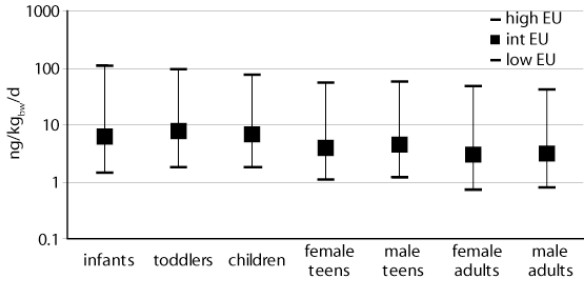


	infants	toddlers	children	female teens	male teens	female adults	male adults
high	220	170	150	49	55	55	50
int	36	28	24	13	15	12	12
low	5.7	7.8	7.4	4.4	4.9	3.2	3.5

Figure 2: Total uptake doses of PFOA in North America (NA) and Europe (EU)



	infants	toddlers	children	female teens	male teens	female adults	male adults
high	120	130	65	52	50	47	41
int	9.8	7.6	5.0	3.0	3.1	2.5	2.5
low	2.2	1.2	0.8	0.5	0.5	0.4	0.4



	infants	toddlers	children	female teens	male teens	female adults	male adults
high	110	94	72	53	53	44	39
int	6.0	7.6	6.7	3.6	4.1	2.8	3.0
low	1.4	1.8	1.8	1.0	1.2	0.7	0.8

Figure 3: Comparison of doses derived from pharamcokinetic model and doses based on SceBRA calculations