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Estimating Consumer Exposure to PFOS and PFOA

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Perfluorinated compounds have been used for more than 50 years as process aids, surfactants, and for surface protection. This study is a comprehensive assessment of consumer exposure to perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA) from a variety of environmental and product-related sources. To identify relevant pathways leading to consumer exposure to PFOS and PFOA a scenario-based approach has been applied. Scenarios represent realistic situations where age- and gender-specific exposure occurs in the everyday life of consumers. We find that North American and European consumers are likely to experience ubiquitous and long-term uptake doses of PFOS and PFOA in the range of 3 to 220 ng per kg body weight per day (ng/kg$_{bw}$/day) and 1 to 130 ng/kg$_{bw}$/day, respectively. The greatest portion of the chronic exposure to PFOS and PFOA is likely to result from the intake of contaminated foods, including drinking water. Consumer products cause a minor portion of the consumer exposure to PFOS and PFOA. Of these, it is mainly impregnation sprays, treated carpets in homes, and coated food contact materials that may lead to consumer exposure to PFOS and PFOA. Children tend to experience higher total uptake doses (on a body weight basis) than teenagers and adults because of higher relative uptake via food consumption and hand-to-mouth transfer of chemical from treated carpets and ingestion of dust. The uptake estimates based on scenarios are within the range of values derived from blood serum data by applying a one-compartment pharmacokinetic model.

KEY WORDS: Consumer exposure; consumer products; exposure analysis; perfluorinated chemicals; PFOA; PFOS

1. INTRODUCTION

Perfluorinated compounds (PFCs) occur globally in wildlife and humans.[1] The most commonly studied PFC classes are the perfluorinated sulfonates (PFSAs) and the perfluorinated carboxylates (PFCAs) and the most commonly measured compounds in these classes are perfluorooctane sulfonate (PFOS) and perfluorooctanoic acid (PFOA). Both compounds are moderately toxic to mammals,[2,3] bioaccumulative,[1] and very persistent to abiotic and biotic degradation.[2,3] It has been proposed to classify PFOS as a persistent organic pollutant (POP) under the Stockholm Convention.[4]

Both PFCAs and PFSAs have been produced for more than 50 years,[5] but have become of interest to researchers since the late 1990s. The increasing interest in these compounds is due to the recent advances in analytical methodology that has enabled their widespread detection in the environment and humans at trace levels. PFCs have been found in outdoor and indoor air,[6–9] surface and drinking water,[10–12] house dust,[8,13–15] animal tissue,[16,17] human blood serum, and human breast milk.[18,19] Because of the high persistence of PFOS and PFOA, the two compounds

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accumulate in the environment and concentrations in humans and environmental media may eventually be close to levels of concern. Current concentrations of PFOA measured in drinking water in New Jersey,[20] for example, are already close to a guidance value of 0.04 ppb that has been derived from toxicity studies in animals.[21] In several species, PFOS and PFOA have been found to exhibit acute and subchronic toxicity with the liver as a primary target organ;[22,23] in addition, developmental toxicity has been observed for both chemicals.[23]

PFCAs are primarily used as process aids in the manufacture of fluoropolymers, but have also been used historically in a variety of commercial products.[24] Fluoropolymers such as polytetrafluoroethylene (PTFE) are films (e.g., on nonstick cookware) or membranes (e.g., in outerwear) and are characterized by a fluorocarbon chain within the polymer backbone. Residual PFCA may be present in fluoropolymer films and membranes used in manufacturing certain consumer articles.[25] PFCAs are also present as reaction impurities in consumer products containing fluorinated polymers, which are added to products to make them stain, soil, water, and grease resistant.[25] Fluorinated polymers comprise a hydrocarbon backbone (e.g., polyesters, polyurethanes, polyethers) with perfluorinated side-chains. Consumer products treated with fluorinated polymers include clothes and textiles, carpets, leather, paper, and cardboard.[26,27] PFSAs and related compounds have been primarily contained in fluorinated polymers used in protective coatings for carpets and apparel, paper coatings approved for food contact, insecticide formulations, and surfactants, as, for example, in fire-fighting foams.[28,29] By 2001, the principal manufacturer of PFOS and related compounds with a chain length of eight carbon atoms had phased out manufacture leaving only small producers in Europe and Asia.

Consumers are exposed to PFOS and PFOA from the use of various PFC-containing products and the intake of contaminated food, environmental media, and house dust. However, a comprehensive assessment of consumer exposure to PFOS and PFOA including all relevant pathways is missing. Previous studies focused on selected pathways such as consumer products,[25] drinking water and fish,[30] and outdoor air.[6] The occurrence of PFCs in humans has been investigated in several screening studies,[18,31–37] suggesting that concentrations of PFOS and PFOA in blood samples tend to be slightly higher in North America than in Europe.

The primary goal of this study is to comprehensively assess the total daily consumer exposure to PFOS and PFOA in North America and Europe occurring via various pathways and to identify the major pathways. Scenarios have been generated to represent relevant situations where consumer exposure to PFOS and PFOA occurs by contact with consumer products, foods, or contaminated environmental media. The most recently published data have been used to characterize the occurrence of PFOS and PFOA in consumer products, food, air, and other media.

2. METHODS

2.1. Scenario-Based Risk Assessment (SceBRA)

The SceBRA approach is a method to assess in a broad perspective human exposure to multiuse chemicals such as solvents[38] or plasticizers.[39] The approach consists of the following steps: (1) analysis of a chemical's life cycle, that is, distribution pathways and applications; (2) definition of exposure pathways and scenarios representing all relevant situations leading to human exposure; (3) calculation of uptake dose; (4) comparison of calculated doses with human screening data; (5) evaluation of risk. High emphasis is put on research into the conditions during relevant situations of exposure. The usefulness of the approach in the assessment of occupational exposure to chlorinated solvents[40,41] and consumer exposure to plasticizers and fragrance materials[39,42] has been demonstrated. In all these case studies, the approach yielded realistic levels of human uptake of chemicals.

2.1.1. System Boundaries

PFCs are used in a wide variety of applications and products for industrial and consumer use. Here, we focus on consumer exposure; occupational exposure to PFOS and PFOA is not investigated. Consumer exposure includes all exposures occurring via the use of consumer products, consumption of different types of food, and intake of environmental and residential media such as air, water or dust. We investigate chronic exposure of consumers caused by continuous or intermittent long-term contact with PFC-containing media. Exposure is calculated on a per day basis; resulting uptake doses refer to the amount of chemical entering the human body, and are normalized to the body weight of consumers. Thus, the uptake doses calculated here reflect internal, time-weighted averages with the unit $ng/kg_{bw}/day$.

Exposure to PFOS and PFOA is investigated separately for North American and European

consumers. The consumption and product-use habits of consumers are described in two separate data sets for North America and Europe. Due to the limited availability of screening studies, the same data sets on the occurrence of PFOS and PFOA in consumer products, environmental media, and foods (except for fish and tap water) are used for North America and Europe. The timeframe of the data collected ranges from 1999 to 2007.

2.1.2. Calculation of Uptake Doses

We differentiate between pathways of direct and indirect consumer exposure. Direct exposure to PFOS and PFOA is caused by a clearly defined source in the form of a consumer product such as impregnation spray. Indirect exposure is caused by sources that cannot be allocated to a specific product, that is, the contact media has been contaminated by multiple or unknown sources. (The terms direct and indirect as used here do not correspond to direct and indirect sources as used by Prevedouros *et al.*[24]) To reflect different behavior patterns and physiological parameters within the population, seven consumer groups are distinguished: infants (0–1 years), toddlers (1–4 years), children (5–11 years), female and male teenagers (12–20 years), and female and male adults (>20 years) (adapted from Health Canada[43]).

Here we differentiate between exposure, where a chemical is in contact with the outer human body, and uptake dose occurring after the absorption of a chemical into the body. We calculate the total daily uptake dose (D_{total}) using Equation (1). D_{total} (ng/kg$_{\text{bw}}$/day) is the sum of exposure estimates of all pathways (n) relevant for a specific consumer group.

$$D_{\text{total}} = \sum_{1}^{n} D_n \tag{1}$$

To reflect the variability and uncertainty in the input parameters, low-exposure, intermediate, and high-exposure scenarios are calculated. These generic scenarios are intended to cover a reasonable range of exposure that can be expected for each consumer group. In general, the 5th percentiles of input parameter values are combined to obtain a low-exposure scenario; median parameter values are combined for an intermediate scenario; and 95th percentiles of parameter values are combined for a high-exposure scenario. If a data set contains measurements below the limit of detection, the values are set to zero for the low-exposure scenarios, to one-half of the detection limit for the intermediate scenarios, and to the detection limit for the high-exposure scenarios. However, if the limit of detection is higher than the highest concentration measured in any other measurement of the same medium, the data are excluded from further analysis (this was the case for some measurements of PFOS and PFOA in foods). It is not possible to use a more sophisticated approach for the estimation of censored data[44] because too few measurements above the limit of detection are available. To better understand the influence of uncertainty and variability of input parameters, the contribution to variance is calculated for each input parameter.[45]

Finally, the results of this study are compared to doses derived from concentrations measured in human blood.[18,31,33–36,46] For this purpose an existing one-compartment pharmacokinetic model is used.[25] This model links the chemical concentration in blood serum to the uptake dose by setting up a mass balance of the chemical in one compartment. We use this simple model because it requires only two input parameters, the elimination rate constant of the chemical and the volume of distribution. A more detailed model of the fate of PFOS and PFOA in the human body is currently not available and development of such a model would be beyond the scope of this work. Equation (2) shows the mass balance with C_s as concentration of PFOS or PFOA in blood serum, D_{total} as the total dose, k as elimination rate constant ($k = \ln 2/t_{1/2}$), and V_D as fractional volume of distribution (normalized to body weight).

$$V_D \frac{dC_s}{dt} = D_{\text{total}} - k \cdot V_D \cdot C_s \tag{2}$$

At steady state, $dC_s/dt = 0$, and

$$D_{\text{total}} = k \cdot V_D \cdot C_s. \tag{3}$$

The volume of distribution is taken from two subchronic monkey studies.[47,48] Half-lives for PFOS and PFOA in humans are taken from Reference 2 (for range of values see Table X).

2.2. Pathways of Oral Exposure

We use gastrointestinal uptake fractions for PFOS and PFOA to transform external exposure in the intestine into uptake doses. Gastrointestinal uptake of PFOA has been studied in four rodent species and large differences exist in the absorption and excretion of PFOA in different species and even

between sexes.[49] Here the fractions of an oral dose of PFOA recovered in urine, expired air, and tissue are added up to estimate the human gastrointestinal uptake fraction of PFOA. This uptake fraction is applied to all consumer groups and equals 66%, 80%, and 91% in the low-exposure, intermediate, and high-exposure scenarios (5th, 50th, and 95th percentiles of cases mentioned in Reference 49). Because PFOS showed similar behavior as PFOA in uptake studies[2] we use the same uptake fractions as for PFOA in the calculations.

2.2.1. Food Consumption

This pathway involves the consumption of different types of food and the concentrations of PFOS and PFOA in these foods (for equation, see Table I). Population-wide average quantities of daily intake of different types of food have been determined in North America[43,50] and Europe.[51–54] The average intake amounts shown in Table II represent the entire population, that is, the fraction of nonconsumers of specific foods (such as vegetarians) is included. The average values are used for all three scenarios.

PFOS and PFOA have been detected in various types of food including edibles of animal origin[17,55–62] and of plant origin,[55,56,61,62] in snacks,[57] in tap and surface water,[11,12,63] and in mother's milk.[19,37] Except for fish (Europe:[61,62,64], North America:[17,56,58,65]) and tap water (Europe:[12], North America:[11,63]), the data on concentrations of PFCs in food are summarized in one set used for North America and Europe because there are too few studies (14 in total) to develop different data sets (Table III).

2.2.2. Ingestion of House Dust

This pathway involves amounts of house dust inadvertently ingested by consumers and concentrations of PFOS and PFOA in dust (for equation, see Table I). Available studies on the ingestion of dust and soil use concentrations of trace elements in dust/soil, food, and feces to estimate the daily intake. Wide ranges of possible intake rates of soil and dust have been reported.[66–70] We derive the range of daily dust ingestion from available data using the limiting tracer method.[69] Amounts of dust typically ingested by infants and toddlers are taken from Calabrese *et al.*[70] (Table IV). The amounts of dust ingested by children, teenagers, and adults are calculated from data representing the total quantity of daily ingested soil and dust,[67] by multiplying the values for soil ingestion with the ratio of concentrations of tracer elements in soil and dust (Table IV). The quantities of daily-ingested dust are adjusted to the time spent indoors. We assume that consumers stay inside of buildings 94% to 98% of the day in Europe[71–73] and 85% to 94% of the day in North America.[74,75]

Concentrations of PFOS and PFOA in total house dust samples, which originate most likely from indoor sources, such as use, wear, and abrasion of consumer products,[13] have been determined in the United States and Japan.[8,13–15]

2.2.3. Hand-to-Mouth Transfer from Treated Carpets

This pathway describes the transfer of PFOS and PFOA to the hand after contact with carpets treated with products containing PFCs and subsequent ingestion of PFOS and PFOA after hand-to-mouth contact (for equation, see Table I). We assume that the hand is replenished with chemical before each contact with the mouth. Data required for these scenarios include the extractable concentrations of PFOS and PFOA in carpets, the market fraction of carpets treated with PFCs, the time people are exposed to carpets, the fraction of compounds transferred from carpet to the skin, the frequency of hand-to-mouth contact, the fraction of compounds transferred from the hand to saliva, and the skin surface area in contact with the mouth.

Former commercial products for carpet protection contained fluoroalkyl polymers based on PFOS chemistry and likely contained traces of PFOS or related substances with the potential to degrade to PFOS.[2,29,76,77] Since the phase-out of PFOS production, only fluorotelomer-based products are used for carpet protection.[25,76,77] These products may contain traces of PFOA.[25] Extractable concentrations of PFOS in mill-treated carpets have not been measured directly. Here, they are calculated from the content of PFCs in the carpet treatment solutions,[29,76] the fractions of PFOS-related residuals, and PFOS in the polymers,[76] amounts of carpet treatment solution used per surface area of carpet (calculated from Reference 25), the fraction of PFC treatment remaining in the carpet after several years of usage (based on 3M cited in Reference 29), and the ratio of applied concentrations of PFOA[25] and PFOS.

Extractable concentrations of PFOA in mill-treated carpets are given by Washburn et al.[25] To refresh the protection, carpets can be treated at home by professional carpet cleaners.[29] Extractable

Table I. Equations for Uptake Doses (ng/kg$_{bw}$/day) for All Pathways Modeled Within This Study

Exposure Pathway	No.	Equation	Required Parameters and Units
Oral			
Food	1	$\text{NA}: D_{\text{food}} = \sum_{i=1}^{n} (C_{\text{food}_i} \cdot q_{\text{food}_i}) \cdot F_{\text{uptake}}$ $\quad \text{EU}: D_{\text{food}} = \frac{\sum_{i=1}^{n} (C_{\text{food}_i} \cdot q_{\text{food}_i})}{m_{\text{bw}}} \cdot F_{\text{uptake}}$	C_{food}: concentration of PFOS or PFOA in food type i (ng/g$_{ww}$), q$_{\text{food}}$: amount food type i consumed daily (NA: g/(kg $*$ day); EU: g/day), m_{bw}: body weight (kg), F_{uptake}: uptake fraction of compounds via the gastrointestinal tract (GIT), (NA = North America, EU = Europe)
Dust	2	$D_{\text{dust\_ing}} = \frac{C_{\text{dust}} \cdot q_{\text{dust}} \cdot F_{\text{time\_in}}}{m_{\text{bw}}} \cdot F_{\text{uptake}}$	C_{dust}: concentration of PFOS and PFOA in dust (ng/mg), q_{dust}: quantity of dust ingested daily (mg/day), $F_{\text{time\_in}}$: fraction of time spent indoors, m_{bw}: body weight (kg), F_{uptake}: uptake fraction of compounds via the GIT
Hand-to-mouth (carpets & upholstery)	3	$D_{\text{carpets\_htm}} = \frac{C_{\text{product}} \cdot MF_{\text{product}} \cdot t_{\text{exp}} \cdot TF_{\text{product-hand}} \cdot f_{\text{htm}} \cdot TF_{\text{hand-mouth}} \cdot A_{\text{skin}}}{m_{\text{bw}}} \cdot F_{\text{uptake}}$	C_{product}: concentration of PFOS and PFOA in carpets and upholstery (products) (ng/cm^2), MF_{product}: market fraction of products treated with PFCs, t_{exp}: time exposed to the products (hour/day), $TF_{\text{product-hand}}$: fraction of compounds transferred from product to hand, f_{htm}: frequency of hand-to-mouth contact events (hour^{-1}), $TF_{\text{hand-mouth}}$: fraction transferred from hand-to-mouth, A_{skin}: skin area in contact with the mouth (cm^2), m_{bw}: body weight (kg), F_{uptake}: uptake fraction of compounds via the GIT
Food contact material (paper & cardboard)	4	$D_{\text{fcm}} = \frac{C_{\text{pc}} \cdot r_{\text{migr}} \cdot MF_{\text{pc}} \cdot f_{\text{food\_pc}} \cdot A_{\text{contact}} \cdot t_{\text{contact}}}{m_{\text{bw}}} \cdot F_{\text{uptake}}$	C_{pc}: concentration of PFOA in food contact material (ng/cm^2), r_{migr}: migration rate of PFOA from food contact material into food (hour^{-1}), MF_{pc}: market fraction of food contact material treated with PFCs, $f_{\text{food\_pc}}$: contact frequency of food with treated materials (day^{-1}), A_{contact}: contact area of food with contact material (cm^2), t_{contact}: contact time of food with contact material (hour), m_{bw}: body weight (kg), F_{uptake}: uptake fraction of PFOA via the GIT
Inhalation			
Indoor & outdoor air	5	$D_{\text{air}} = \frac{C_{\text{air\_in}} \cdot \sum_{i=1}^{n} (V_{\text{inh\_act}_i} \cdot t_{\text{act}_i}) + C_{\text{air\_out}} \cdot \sum_{j=1}^{m} (V_{\text{inh\_act}_j} \cdot t_{\text{act}_j})}{m_{\text{bw}}} \cdot F_{\text{uptake}}$	$C_{\text{air\_in}}$: concentration of PFOS and PFOA in indoor air (ng/m^3), $V_{\text{inh\_act}_{i,j}}$: activity-dependent inhalation rate (m^3/hour), $t_{\text{acti,j}}$: time spent on different activities (hour/day), $C_{\text{air\_out}}$: concentration of PFOS and PFOA in outdoor air, m_{bw}: body weight (kg), F_{uptake}: uptake fraction of compounds via the lungs
Spray droplets	6	$D_{\text{spray\_inhal}} = \frac{C_{\text{spray}} \cdot MF_{\text{spray}} \cdot f_{\text{spray}} \cdot t_{\text{spray}} \cdot r_{\text{spray}} \cdot F_{\text{resp}} \cdot V_{\text{inh}}}{m_{\text{bw}}} \cdot \left(\frac{t_{\text{spray\_NF}}}{V_{\text{NF}}} + \frac{t_{\text{spray\_FF}}}{V_{\text{FF}}} \right) \cdot F_{\text{uptake}}$	C_{spray}: concentration of PFOA in impregnation sprays (ng/g), MF_{spray}: market fraction of impregnation sprays containing PFCs, f_{exp}: frequency exposed to impregnation sprays (day^{-1}), t_{spray}: time spent spraying (min), r_{spray}: rate of spray emission (g/minute), F_{resp}: fraction of respirable aerosols generated, V_{inh}: inhalation rate (m^3/hour), $t_{\text{spray\_NF}}$: time exposed to aerosols in the near field (hour), V_{NF}: near-field volume around consumer (m^3), $t_{\text{spray\_FF}}$: time exposed to aerosols in the far field (hour), V_{FF}: far-field volume around consumer (m^3), m_{bw}: body weight (kg), F_{uptake}: uptake fraction of PFOA via the lungs

Note: Sources for body weights: North America[104] and Europe.[105]

Table II. Amounts of Food Consumed Daily (Europe: (g/day); North America (g/kg_{bw}/day))

	Infant		Toddler		Child		Female Teen		Male Teen		Female Adult		Male Adult	
Food Category	EU	NA	EU	NA	EU	NA	EU	NA	EU	NA	EU	NA	EU	NA
Cereal products	17	2.5	92	3.6	148	2.5	133	1.4	176	1.7	128	1.2	181	1.3
Cereals	21	4.3	37	6.2	54	3.7	67	2.3	76	1.9	73	1.6	92	1.5
Dairy products	41	0.2	47	0.9	54	0.8	39	0.5	45	0.5	47	0.5	46	0.5
Eggs	3	0.8	8	0.7	9	0.4	9	0.2	12	0.2	16	0.2	22	0.2
Fats and oils	1	0.5	6	0.2	6	0.2	6	0.2	8	0.2	9	0.2	15	0.2
Fish and shellfish	3	0.8	10	0.4	15	0.2	15	0.2	18	0.2	31	0.2	31	0.2
Fruits	29	15	50	10	65	5.0	51	2.2	43	2.2	103	2.0	87	2.0
Human milk	77	41	0	0	0	0	0	0	0	0	0	0	0	0
Meat	14	2.9	42	4.1	60	3.0	70	2.1	108	2.1	84	1.8	138	1.8
Milk	478	63	288	23	208	12	138	5.8	215	5.8	200	2.9	226	2.9
Potatoes	14	1.1	48	2.2	85	1.7	105	1.2	129	1.2	93	0.9	117	0.9
Poultry	4	0.8	10	1.1	22	0.9	37	0.6	45	0.6	46	0.5	62	0.5
Snacks	0.3	0.3	13	0.5	25	0.3	28	0.2	39	0.2	15	0.1	24	0.1
Sweets	2	0.4	25	1.1	35	0.9	35	0.5	46	0.5	22	0.4	32	0.4
Tap water	0	44	306	47	83	32	117	18	125	18	314	20	239	20
Vegetables	48	5.7	39	5.3	61	3.8	81	2.6	83	2.6	132	2.7	137	2.7
Water-based drinks	20	1.1	452	6.8	737	3.9	803	3.0	1010	2.7	1340	7.5	1870	6.3

concentrations of PFOA in home-treated carpets are taken from Reference 25 (Table V).

The fraction of mill-treated carpets available on the market is assumed to be 1%, 10%, and 50% for the low-exposure, intermediate, and high-exposure scenarios. Corresponding fractions for home-treated carpets are 1%, 5%, and 20% (Table V). The time of direct contact with carpets is defined as the active time spent indoors in North America[50,74] and Europe[71–73] combined with the market fraction of carpet used as flooring material.[78,79] The fraction of compound transferred from carpet to skin depends on the duration and intensity of contact and the sweatiness of the skin. Default values for chemical transfer are provided by the U.S. EPA's Office of Pesticide Programs (OPP)[80] and have been adopted by Washburn

Table III. Concentration of PFOS and PFOA in Food (ng/g_{ww}) for Low-Exposure (Low), Intermediate (Int), and High-Exposure (High) Scenarios

	PFOS NA				PFOS EU				PFOA NA				PFOA EU			
Food Category	Low	Int	High	n(N)[a]	Low	Int	High	n(N)[a]	Low	Int	High	n(N)[a]	Low	Int	high	n(N)[a]
Cereal products	0	0	0	37 (2)	0	0	0	37 (2)	0	0.3	0.5	36 (1)	0	0.3	0.5	36 (1)
Cereals	0	0	0	3 (2)	0	0	0	3 (2)	0	0	0	3 (2)	0	0	0	3 (2)
Dairy products	0.04	0.06	0.08	3 (2)	0.04	0.06	0.08	3 (2)	0	0	0	3 (2)	0	0	0	3 (2)
Eggs	0.08	0.3	0.5	43 (3)	0.08	0.3	0.5	43 (3)	0	0	0	43 (3)	0	0	0	43 (3)
Fats and oils	0	0	0	1 (1)	0	0	0	1 (1)	0	0	0	1 (1)	0	0	0	1 (1)
Fish and shellfish	19	39	56	130 (5)	0.2	10	60	72 (3)	0	0.3	2	83 (3)	0	0	2	72 (3)
Fruits	0	0	0	38 (2)	0	0	0	38 (2)	0	0.1	0.3	38 (2)	0	0.1	0.3	38 (2)
Human milk	0.05	0.1	0.4	31 (2)	0.05	0.1	0.4	31 (2)	0.05	0.1	0.3	31 (2)	0.05	0.1	0.3	31 (2)
Meat	0.03	0.3	0.5	128 (3)	0.03	0.3	0.5	128 (3)	0	0.2	1	131 (4)	0	0.2	1	131 (4)
Milk	0	0.3	0.5	41 (3)	0	0.3	0.5	41 (3)	0	0	0	41 (3)	0	0	0	41 (3)
Potatoes	4	6	8	13 (2)	4	6	8	13 (2)	0.4	1	2	13 (2)	0.4	1	2	13 (2)
Poultry	0	0	0	39 (3)	0	0	0	39 (3)	0	0	0	39 (3)	0	0	0	39 (3)
Snacks	0.05	0.5	0.9	2 (1)	0.05	0.5	0.9	2 (1)	0.9	2	3	2 (1)	0.9	2	3	2 (1)
Sweets	0.8	1	1.2	1 (1)	0.8	1	1.2	1 (1)	0	0	0	1 (1)	0	0	0	1 (1)
Tap water	0.002	0.003	0.006	102 (2)	0	0.003	0.01	28 (1)	0.009	0.01	0.02	102 (2)	0	0.04	0.2	28 (1)
Vegetables	0	0.3	0.5	39 (3)	0	0.3	0.5	39 (3)	0	0.1	0.3	38 (2)	0	0.1	0.3	39 (2)
Water-based drinks	0	0	0	1 (1)	0	0	0	1 (1)	0	0	0	1 (1)	0	0	0	1 (1)

[a] n = total number of observations used to derive values for scenarios (composite samples count as one observation), N = number of studies (all equally weighted).

Table IV. Amounts of Dust Ingested per Day (mg/day) for Low-Exposure (Low), Intermediate (Int), and High-Exposure (High) Scenarios

Media	Scenario	Infants	Toddlers	Children	Female Teens	Male Teens	Female Adults	Male Adults
Dust	Low	0.8	0.8	7.3	0.2	$2 \cdot 10^{-4}$	0.2	$2 \cdot 10^{-4}$
	Int	9.0	9.0	26	5.2	0.2	5.2	0.2
	High	106	106	95	138	197	138	197

Table V. Concentration of PFOS and PFOA in Consumer Products and Environmental Media: Market Fraction (MF) of Products for Low-Exposure (Low), Intermediate (Int), and High-Exposure (High) Scenarios

		PFOS			PFOA			MF		
Consumer Products	Unit	Low	Int	High	Low	Int	High	Low	Int	High
Carpet, mill-treated	ng/cm^2	0.1	1.3	73	0.1	7	23	0.01	0.1	0.5
Carpet, home-treated	ng/cm^2	–	–	–	28	36	50	0.01	0.05	0.2
Upholstery	ng/cm^2	–	–	–	0.4	2.2	4	0.01	0.05	0.1
Food contact material	ng/cm^2	–	–	–	1.7	15	53	0.1	0.5	1
Impregnation sprays	ng/g	–	–	–	$5 \cdot 10^3$	$2 \cdot 10^4$	$4 \cdot 10^4$	0.5	0.5	0.5
Clothes	ng/cm^2	$3 \cdot 10^{-4}$	$2 \cdot 10^{-3}$	0.2	0.01	0.3	1.1	0.01	0.1	0.5
PTFE-covered cookware	ng/cm^2	–	–	–	0.07	0.7	1.3	0.5	0.5	0.5
		PFOS			PFOA					
Environmental Media	Unit	Low	Int	High	Low	Int	High			
House dust	ng/mg	0.01	0.4	1.2	0.02	0.1	1.2			
Indoor air	ng/m^3	$4 \cdot 10^{-6}$	$4 \cdot 10^{-3}$	0.02	$4 \cdot 10^{-5}$	$3 \cdot 10^{-4}$	0.02			
Outdoor air	ng/m^3	$6 \cdot 10^{-5}$	$6 \cdot 10^{-3}$	0.02	0.2	0.3	0.4			

et al.[25] to be applied to PFOS and PFOA. The higher values for infants, toddlers, and children are based on the assumption that they are likely to have sweatier skin than teenagers and adults. The OPP default values for the frequency of hand-to-mouth contact events in infants and toddlers is 9.5 contacts per hour.[80] This frequency decreases with increasing age (five, two, and one contact per hour for children, teenagers, and adults, respectively;[80] see Table VI). The fraction of chemical transferred from hand to saliva is based on information on transfer of soil and pesticides from hand to mouth.[80,81] It is assumed that the skin surface area in contact with the mouth is three fingers of one hand for infants, toddlers, and children (corresponding to 5% of the surface area of both hands),[80] and three fingertips of one hand for teenagers and adults (corresponding to 1.7% of the surface area of both hands). The hands' surface areas are calculated from skin surface area data given in the U.S. EPA *Exposure Factors Handbook*.[50]

Table VI. Frequencies of Performing Tasks and Behavioral Patterns for Low-Exposure (Low), Intermediate (Int), and High-Exposure (High) Scenarios

	Impregnating Clothes and Other Textiles (day^{-1})			Consuming Food in Contact with Paper or Cardboard (day^{-1})			Consuming Food in Contact with Paper or Cardboard (day^{-1})			Hand-to-Mouth Contact Events (hour^{-1})		
				North America			Europe					
	Low	Int	High	Low	Int	High	Low	Int	High	Low	Int	High
Infants	0	0	0.01	0	0.02	0.03	0	0.02	0.03	9.5	9.5	9.5
Toddlers	0	0	0.01	0.03	0.1	0.5	0.03	0.07	0.3	9.5	9.5	9.5
Children	0	0	0.01	0.03	0.1	0.5	0.03	0.07	0.3	5	5	5
Female teens	0	0	0.01	0.03	0.3	1	0.03	0.07	0.4	2	2	2
Male teens	0	0	0.01	0.03	0.3	1	0.03	0.07	0.4	2	2	2
Female adults	0	0	0.01	0.03	0.3	1	0.03	0.1	0.4	1	1	1
Male adults	0	0	0.01	0.03	0.3	1	0.03	0.1	0.4	1	1	1

2.2.4. *Migration from Paper and Cardboard into Food*

This pathway is modeled for PFOA only. Required data include concentrations of PFOA in food contact materials such as wrapping paper, cardboard plates and packaging, and baking parchment, the migration rate of PFOA from food contact materials into the food, the market fraction of PFC-treated materials, the frequency of contact of food with treated materials, the area of material in contact with food, and the duration of contact (for equation, see Table I).

Concentrations of PFCs in treated food contact materials are between 0.5% and 3.6% on a per-weight basis,[82] which can be transformed into PFOA concentrations by applying the concentration of PFOA residuals in the PFC treatment solution.[27] Weight-based concentrations are converted into per-area concentrations by assuming a paper weight of 40 g/m^2 (low-exposure), 53 g/m^2 (intermediate), and 100 g/m^2 (high-exposure), corresponding to the top layer (0.25 mm) of treated cardboard (Table V). Experiments have demonstrated that fluorotelomers may migrate from food contact materials into food with a rate of approximately $5 \cdot 10^{-4}$ min^{-1}.[27] This rate is adopted here for PFOA and extrapolated to one hour. The fraction of PFC-containing food contact materials on the market is assumed to be 10%, 50%, and 100% in the low-exposure, intermediate, and high-exposure scenarios (Table V). The contact frequency of food with treated materials is assumed to be once per month in the low-exposure scenarios, reflecting the use of such materials in various applications such as baking parchment. In the intermediate and high-exposure scenarios, the frequency of contact is set equal to the frequency of consuming fast food in North America[83–86] and Europe[83,87,88] (Table VI), as the use of fast food wrapping and take-away paper plates is assumed to dominate these scenarios. The contact time of food with treated materials is set to 0.25 hour, 0.5 hour, and 1 hour. The area of materials in contact with food is obtained by multiplying the apparent contact area and the fraction of material actually contacting the food due to the food's irregular surface. The values are assumed to be 10%, 20%, and 50% in the low-exposure, intermediate, and high-exposure scenarios.

2.3. Pathways of Inhalation Exposure

PFOS and PFOA have a low volatility[2,3] and their airborne fractions are expected to occur mainly bound to aerosol particles. Our scenarios only consider the inhalation of particles small enough to reach the alveoli ($< 4\ \mu$m). Larger particles can be inhaled but are deposited on mucosa and transported into the intestine. Therefore, they are considered in the pathway "ingestion of dust." Uptake fractions for the lungs are applied to transform inhaled concentrations into internal exposures. So far, exposure to PFOS and PFOA via inhalation has not been quantified. Animal studies suggest that PFOA is easily absorbed via the lungs.[22] The uptake fractions of PFOS and PFOA are set to 1 for all consumer groups as a conservative assumption.

2.3.1. *Inhalation of Indoor and Ambient Air*

Data for this pathway include the concentrations of PFOS and PFOA in indoor and ambient air, the activity-dependent inhalation rates, the time spent on different activities, and the location of these activities (for equation, see Table I).

The concentration of particle-bound PFOS and PFOA has been examined in outdoor air in Japan and the United Kingdom.[6,89,90] The concentrations of PFOS and PFOA in indoor air are estimated from concentrations of PFOS and PFOA in house dust[13–15] (Table V), the concentration of respirable particulate matter in indoor air,[91,92] and the fraction of airborne particles with a possible origin in house dust.[91] Concentrations of PFOS and PFOA recently measured in indoor air are similar to the ones obtained with our procedure.[9]

American studies on consumers' daily activities investigated the time spent at different levels of physical activity (and associated ventilation) rather than the duration of single activities.[50,75] The volumes of indoor and ambient air inhaled daily are obtained by multiplying the durations spent at different levels of physical activities by the associated ventilation rates and the fraction of a day spent indoors or outdoors (Table VII). Surveys in Europe have investigated daily time-location activity patterns.[71–73] Each activity is assigned a location and an activity-dependent inhalation volume[74,93] (see Tables VIII and IX). Summing up the inhalation volumes of the activities multiplied by the time spent for each activity and considering the location of the activity yields the total volume of indoor and outdoor air inhaled per day.

2.3.2. *Inhalation of Impregnation Spray Aerosols*

This pathway is modeled for PFOA only and reflects the exposure of consumers using sprays to

Table VII. Time-Activity Patterns and Corresponding Inhalation Rates for North America

North America Location	Activity	Duration of Levels of Activities (hour/day)					Inhalation Rates (m^3/hour)						
		Infants	Toddlers	Children	Teens	Adults	Infants	Toddlers	Children	Female Teens	Male Teens	Female Adults	Male Adults
Indoor	Active	9.4	n.a.	n.a.	n.a.	n.a.	0.1	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
	Inactive	11.1	n.a.	n.a.	n.a.	n.a.	0.3	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
	Resting	n.a.	9.9	10.7	10.5	10.8	n.a.	0.2	0.2	0.3	0.4	0.3	0.7
	Light	n.a.	9.9	10.7	10.5	10.8	n.a.	0.3	0.5	0.7	0.8	0.5	0.8
	Moderate	n.a.	0.7	0.8	0.7	0.8	n.a.	0.7	0.9	1.3	1.5	1.6	2.5
	Heavy	n.a.	0.09	0.1	0.1	0.1	n.a.	1.7	2.3	3.2	3.8	2.9	4.8
Outdoor	Active	1.6	n.a.	n.a.	n.a.	n.a.	0.1	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
	Inactive	1.9	n.a.	n.a.	n.a.	n.a.	0.3	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
	Resting	n.a.	1.0	0.5	0.6	0.4	n.a.	0.2	0.2	0.3	0.4	0.3	0.7
	Light	n.a.	1.0	0.5	0.6	0.4	n.a.	0.3	0.5	0.7	0.8	0.5	0.8
	Moderate	n.a.	1.3	0.7	0.8	0.5	n.a.	0.7	0.9	1.3	1.5	1.6	2.5
	Heavy	n.a.	0.2	0.1	0.1	0.1	n.a.	1.7	2.3	3.2	3.8	2.9	4.8

impregnate textiles (teenagers and adults) and of consumers staying in the same room during and after spraying (all consumer groups) (for equation, see Table I). To this end, a two-compartment model including a near field (close vicinity of the source during use) and a far field (room where spraying takes place) is used to calculate exposure to spray-derived aerosols. Required data include the concentration of PFOA in impregnation sprays, the market fraction of impregnation sprays containing PFCs, the frequency of using impregnation sprays, the duration of use, the emission rate of impregnation sprays, the fraction of respirable aerosols generated, the inhalation rate during and after spray use, the time exposed to aerosols in the near field and far field, and the near field and far field volume.

Specific values for the concentration of PFOA in impregnation sprays were only found in Washburn *et al.*[25] where the concentration ranges from below LOD (1 ppm) to 50 ppm. The market fraction of impregnation sprays containing PFCs is estimated from the Swiss product register and set to 50%.[94] Only 20% of consumers use impregnation sprays[95] and, therefore, only the high-exposure scenarios are modeled for this pathway. The frequency of using impregnation sprays is set to 4.4/year (calculated 95th percentile from U.S. EPA data[50]). The duration of spray use is set to half of the values provided by U.S. EPA,[50] because we assume that the spray is discharged only during 50% of the total spraying time.[96] The rate of spray emission is calculated from data given by Glensvig *et al.*[97] The fraction of respirable particles generated is set to 0.25%, assuming that 55% of generated overspray is small enough to reach the alveoli ($< 4\ \mu$m).[96,98,99] After spraying, consumers stay in the same room up to four hours.[50] During and after spraying the consumers are expected to perform light-to-moderate activities with inhalation rates of 1 to 1.6 m^3/hour.[50] The volumes of the near-field and far-field zones are set to 1 m^3 and 50 m^3, respectively.[98,100]

2.4. Other Pathways

Besides the pathways presented above, the following pathways have also been modeled: oral exposure from hand-to-mouth contact with clothes and upholstery, from mouthing of clothes, carpet, and upholstery (only infants and toddlers), from migration into food prepared with PTFE-coated cookware, dermal exposure from wearing of treated clothes, from deposition of spray droplets on skin while impregnating, from skin contact with treated carpet and with upholstery, and from deposition of dust on skin.

Detailed analysis of these pathways showed that none of them causes significant exposure to PFOS or PFOA under the assumption of reasonable conditions during exposure, that is, the contribution of each pathway to the total uptake dose is less than 1% in any of the scenarios.

3. RESULTS

3.1. Exposure to PFOS

Fig. 1 shows the long-term average of the total daily uptake dose of PFOS in North America and Europe for different consumer groups. The levels

Table VIII. Time-Activity Patterns and Corresponding Inhalation Rates for Europe

Europe Activity	Duration Spent on Activities Each Day (minute/day)						Location		Activity Level	Activity-Dependent Inhalation Rates (L/Minute)					
	Infants	Toddlers	Children	Female Teens	Male Teens	Female Adults	Male Adults	Indoor	Outdoor		Infants	Toddlers	Children	Female Teens	Male Teens	Female Adults	Male Adults
Sleeping	673	732	613	550	550	515	501	100%		Lying	2.6	4.4	7.5	7.1	8.9	7.1	8.9
Napping, resting	251	156	0	0	0	21	22	100%		Lying, sitting	2.6	4.4	7.5	7.4	9.1	7.4	9.1
Eating	79	88	67	72	72	84	81	100%		Sitting	4.4	7.0	7.3	7.7	9.3	7.7	9.3
Personal care	0	0	48	50	50	56	54	100%		Sitting, standing	4.4	7.0	7.9	8.0	10.0	8.0	10.0
Employment	0	0	5	181	181	155	244	100%		Sitting, standing, walking	4.4	7.0	10.5	12.8	16.5	12.8	16.5
Study	0	0	231	84	84			100%		Sitting	4.4	7.0	7.3	7.7	9.3	7.7	9.3
Housework, shopping	0	0	45	74	74	212	97	100%		Standing, walking	n.a.	7.0	12.2	15.3	20.1	15.3	20.1
Childcare	0	0	1	14	14	31	12	100%		Sitting, standing	4.4	7.0	7.9	8.0	10.0	8.0	10.0
Voluntary work	0	0	9	9	9	13	13	100%		Sitting, standing, walking	4.4	7.0	10.5	12.8	16.5	12.8	16.5
Social life	0	0	52	91	91	56	50	100%		Sitting, standing	4.4	7.0	7.9	8.0	10.0	8.0	10.0
Entertainment	0	0	9	8	8	6	6	100%		Sitting, standing	4.4	7.0	7.9	8.0	10.0	8.0	10.0
Sport, outdoor activity	29	80	29	20	20	53	59		100%	Walking, running	n.a.	7.0	23.8	35.2	45.0	35.2	45.0
Hobbies	0	0	95	26	26	18	44	50%	50%	Sitting, standing, walking, running	4.4	7.0	15.8	21.6	27.5	21.6	27.5
Reading	0	0	10	10	10	31	31	100%		Lying, sitting	3.5	5.7	7.4	7.4	9.1	7.4	9.1
Watching TV, video	0	0	140	142	142	120	138	100%		Sitting	4.4	7.0	7.3	7.7	9.3	7.7	9.3
Listening to music	0	0	8	11	11			100%		Lying, sitting	3.5	5.7	7.4	7.4	9.1	7.4	9.1
Commuting, travel	0	0	77	97	97	60	77	90%	10%	Sitting, walking	4.4	7.0	11.6	15.0	19.4	15.0	19.4
Other	408	385	0	0	0	10	12	100%		Sitting, standing, walking	4.4	7.0	10.5	12.8	16.5	12.8	16.5

Table IX. Inhalation Rates Per Activity Level Used for Europeans (L/Minute)

Consumer Group	Lying	Sitting	Standing	Walking	Running
Infants	2.6	4.4	n.a.	n.a.	n.a.
Toddlers	4.4	7.0	7.0	7.0	n.a.
Children	7.5	7.3	8.5	15.8	31.8
Female teens	7.1	7.7	8.4	22.3	48.2
Male teens	8.9	9.3	10.7	29.5	60.5
Female adults	7.1	7.7	8.4	22.3	48.2
Male adults	8.9	9.3	10.7	29.5	60.5

obtained for North America and Europe are in the same range. The results suggest that consumers receive chronic doses of PFOS in North America and Europe; young consumers tend to receive the highest dose of PFOS. The range between the lowest and highest doses exceeds one order of magnitude in all consumer groups. The highest calculated dose is found for North American infants with 219 ng/kg_{bw}/day in the high-exposure scenario. The lowest calculated dose is found for European female adults with 3.2 ng/kg_{bw}/day in the low-exposure scenario.

In Fig. 2, the high-exposure scenarios with the portion of direct and indirect exposure and the contributions of pathways are shown for PFOS in North American consumers. In the high-exposure scenario, the contribution of consumer products to the total daily uptake of PFOS can be considerable. For toddlers and children, products may cause more than half of the uptake of PFOS. In general, however, the consumption of contaminated food is the dominant pathway leading to consumer exposure in the high-exposure scenarios, contributing between 29% and 82%. Hand-to-mouth contact with mill-treated carpets is the most important product-related pathway, contributing between 5% and 64% to the North American uptake of PFOS in the high-exposure scenarios. The ingestion of house dust can contribute between 6% and 16% to the total uptake in these scenarios. A similar exposure pattern can be observed in Europe, where direct exposure to PFOS from the use of products is, however, less important than in North America; it accounts for a maximum of 43% (toddlers and children) of the calculated uptake doses. Indirect exposure clearly dominates in the low-exposure and intermediate scenarios in Europe and North America (not shown in Fig. 2). In these scenarios, contaminated food is the major source of consumer exposure to PFOS. All other pathways considered here contribute together less than 3% to the total daily

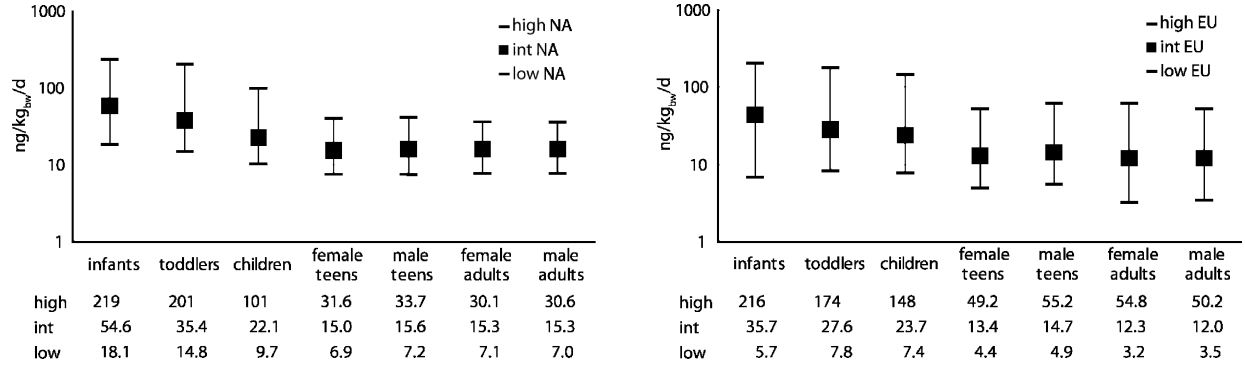

Fig. 1. Uptake doses of PFOS in North America (NA), left, and Europe (EU), right, in ng per kg bodyweight per day for low-exposure (low), intermediate (int), and high-exposure (high) scenario.

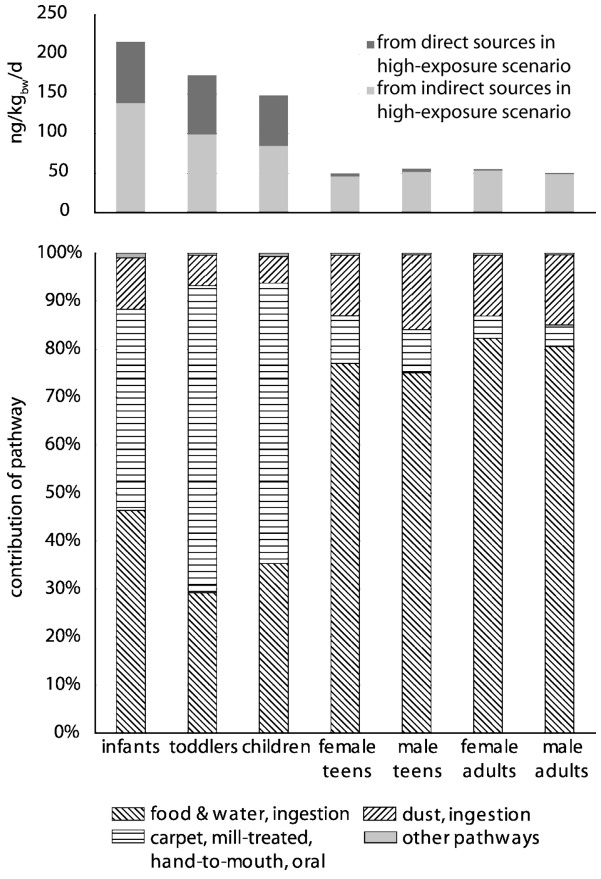

Fig. 2. Direct versus indirect exposure to PFOS (top) and contribution of pathways in North America (bottom) for the high-exposure scenario.

doses in the low-exposure and intermediate scenarios.

We also investigated the importance of each input parameter for the total daily uptake doses by taking their uncertainty and variability into account. Fig. 3 shows the input parameters with the highest contributions to variance of doses of infants in North America and Europe. All input parameters notably influencing the modeled exposure are part of indirect pathways, and all parameters with a contribution to variance of at least 5% belong to the pathway "food consumption." This pattern is found for all consumer groups and is in line with the finding that the consumption of contaminated foods is an important pathway of exposure to PFOS for all consumers. The most influential input parameters (contribution to variance greater than 5%) for uptake of PFOS by infants are the concentrations measured in milk and fish, the gastrointestinal uptake fractions, and the body weight (Europe only). In the other consumer groups, the concentrations measured in meat and potatoes are also influential.

3.2. Exposure to PFOA

Fig. 4 shows the calculated long-term average of the total daily uptake doses of PFOA in North America and Europe for all seven consumer groups. The calculated uptake is in the same range in North America and Europe. As in the case of PFOS, the results suggest that consumers are ubiquitously exposed to PFOA in North America and Europe. There is a tendency that young consumers receive higher uptake doses than older consumers. The range between the lowest and highest doses is approximately two orders of magnitude in all consumer groups. The highest dose is obtained for North American toddlers (128 ng/kg$_{bw}$/day in the high-exposure scenario). The lowest dose is found for North American

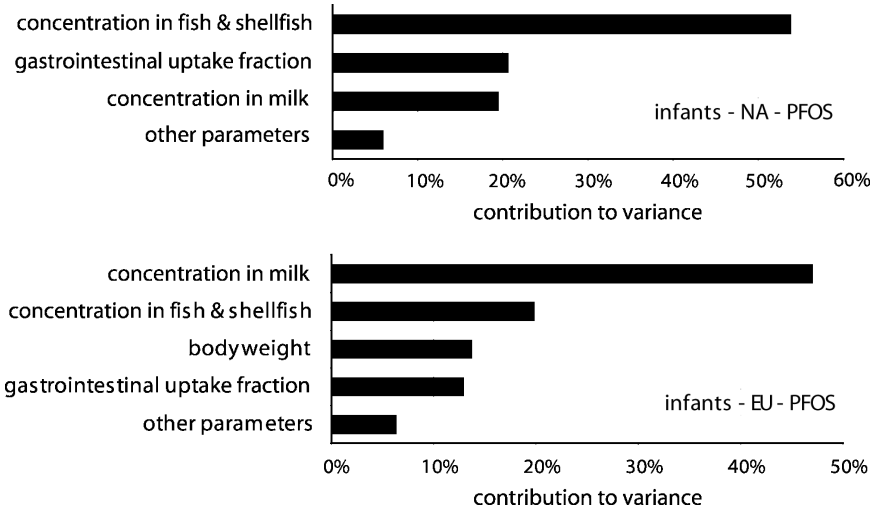

Fig. 3. Input parameters with highest contribution to variance of uptake of PFOS; results for infants in North America (NA) and Europe (EU).

female and male adults (0.4 ng/kg$_{bw}$/day in the low-exposure scenarios). There is a general tendency that the uptake in the intermediate scenarios exceeds the low-exposure scenarios by a factor of five, whereas the high-exposure scenarios exceed the intermediate scenarios by more than one order of magnitude.

In Fig. 5 (top panels), the portion of direct and indirect exposure of North American consumers to PFOA and the contribution of pathways in all scenarios is shown. The high-exposure scenarios are dominated by exposure to PFOA from products (see below). Indirect exposure via contaminated food and house dust dominates the uptake in the low-exposure scenarios. In the intermediate scenarios, food and house dust still account for at least 92% of the total uptake doses.

The picture for exposure of Europeans to PFOA is similar to that for North American consumers. The low-exposure scenarios are dominated by contaminated food and house dust. The most important pathways in the intermediate scenarios are also food and house dust, contributing at least 94% to the total uptake doses. For the high-exposure scenarios the direct, product-related exposure is dominant, with contributions ranging from 53% in the case of infants to 72% in the case of female teens.

For North America, the graph at the bottom of Fig. 5 depicts pathways with a contribution exceeding 1% of the total uptake; the other pathways are summarized under "Other pathways." In the high-exposure scenarios the contribution of the food pathway does not exceed 24% (Europe 26%). Ingestion of house dust, hand-to-mouth contact with

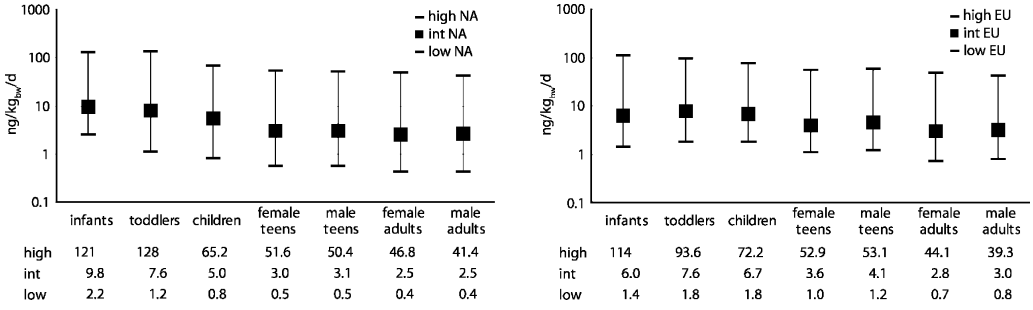

Fig. 4. Uptake doses of PFOA in North America (NA), left, and Europe (EU), right, in ng per kg bodyweight per day for low-exposure (low), intermediate (int), and high-exposure (high) scenario.

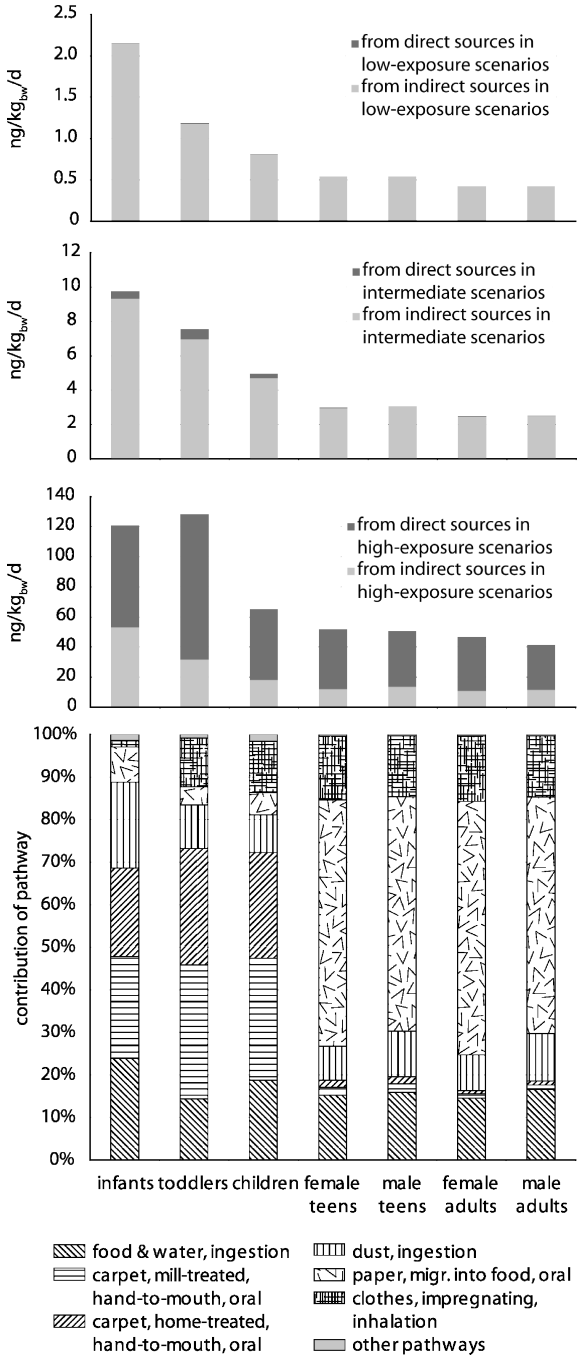

Fig. 5. Direct versus indirect exposure to PFOA in North America for all three scenarios (top panels) and contribution of different pathways to results for high-exposure scenarios (bottom).

contaminated carpets, migration of PFOA from treated paper or cardboard into food, and inhalation of contaminated aerosols while impregnating clothes cause the major portion of the exposure. The pattern is similar for Europe.

To explore how uncertainty and variability of all input parameters in combination influence the variability in calculated uptake doses, contributions to variance have been calculated and results for infants are shown in Fig. 6. The most influential input parameters (contribution to variance greater than 5%) for infants are concentrations measured in potatoes, human milk, vegetables, meat, and fruit, gastrointestinal uptake fraction, and body weight (Europe only). For the other consumer groups the situation is similar but instead of the concentration measured in human milk, the concentrations of PFOA in tap water, snacks, and cereal products such as bread or biscuits become important.

3.3. Comparison with Results from Human Screening Studies

To compare our results to uptake doses derived from concentrations of PFOS and PFOA in human blood, we use the pharmacokinetic model of Washburn *et al.*[25] As the model has been developed for adults, only dose estimates from adults are compared. Fig. 7 shows that the uptake doses derived from human blood samples cover approximately two orders of magnitude. This is due to the variability of the concentration measured in blood samples and the high uncertainty of the volume of distribution and half-lives of PFOS and PFOA in the human body. The uptake doses obtained with the SceBRA approach have a smaller range and all but one (low-exposure scenario for PFOA in North America) are within the ranges of the blood serum values. The uptake doses derived with the pharmacokinetic model for North America are slightly higher than for Europe, and the intermediate results for uptake doses of PFOA are higher than the SceBRA results by factors of five (North America) and three (Europe). For PFOS the results of the intermediate scenarios calculated with SceBRA and the intermediate results from the pharmacokinetic model are very close.

4. DISCUSSION

The present modeling study makes it possible to analyze and compare the contribution of many pathways to exposure of consumers in North America and Europe to PFOS and PFOA. The consumption of contaminated food is the most important pathway causing exposure to PFOS and PFOA, followed by the ingestion of dust and inhalation of air. Consumer products are probably a less important source

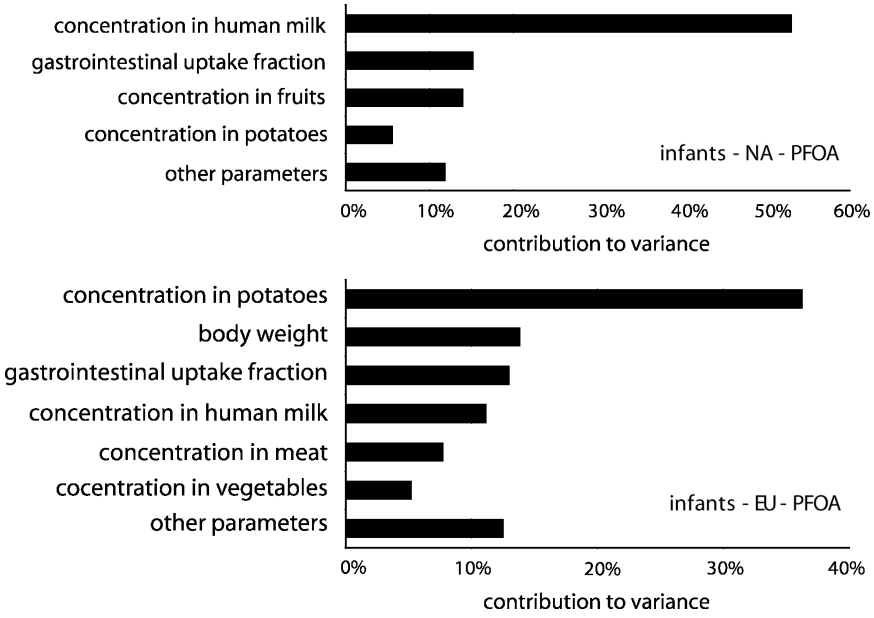

Fig. 6. Input parameters with highest contribution to variance of uptake of PFOA; results for infants in North America (NA) and Europe (EU).

of exposure for the majority of consumers, which is in accordance with the findings of Washburn *et al.*[25] However, product-related pathways do contribute to the exposure of those consumers who regularly use PFC-containing products such as impregnation sprays or who have mill-treated carpets in their homes, which is reflected in the high-exposure scenarios in our study. Product-related exposure tends to be more important for PFOA, most likely because PFOS is no longer used in consumer products. Nevertheless, the total uptake doses of PFOS are still greater than doses of PFOA by approximately one order of magnitude in the low-exposure scenarios, by a factor of five in the intermediate scenarios, and by a factor of two in the high-exposure scenarios (for teenagers and adults the uptake of PFOS is almost similar or even lower than the uptake of PFOA in the high-exposure scenarios). This difference in uptake is due to the dominating influence of the food pathway that reflects the past use of PFOS in various consumer products and its emission into the environment from production plants, and subsequent bioaccumulation leading to contaminated foods. That the difference between uptake of PFOS and of PFOA decreases from low-exposure to high-exposure scenarios is in line with the declining importance of the food pathway from low-exposure to high-exposure scenarios.

It is important to note that the uptake dose estimates in this study are based on steady-state assumptions. Hence, the results represent a snapshot of the time between 1999 and 2007. The production of PFOS was phased out in 2001[1] and emission reductions for PFOA are being implemented by the PFOA producing industry.[24] Because half-lives of PFOS[2] and PFOA[3] in the human body are long (Table X), levels of PFOS and PFOA in human blood would probably reflect higher exposure from the late 1990s and earlier. Accordingly, one should expect that the uptake dose estimates based on pharmacokinetic modeling should be higher than the SceBRA estimates.

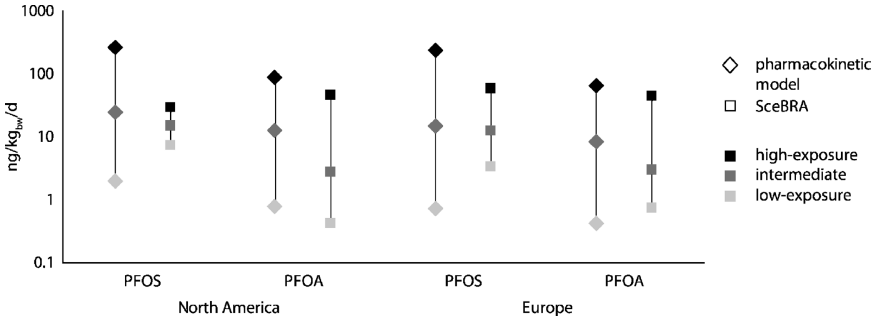

Fig. 7. Comparison of total uptake doses from pharmacokinetic model (diamonds) and SceBRA (squares).

For PFOS, however, such a discrepancy is not visible in Fig. 7. Possible reasons might be that we used measurements for the SceBRA calculations from before and after the phase-out of PFOS, leveling out this effect, or that some values used for input parameters overestimate the concentrations of PFOS in contact media such as potatoes. The uptake of PFOA, on the other hand, shows lower estimates for the intermediate scenarios calculated with SceBRA compared to the pharmacokinetic model values. This leads to the question of whether the SceBRA estimates already reflect the recent emission reductions.[24] Unfortunately, because time series of PFC concentrations in exposure media and blood are still short, it is currently not possible to attempt a dynamic modeling of human exposure.

On the other hand, it is not clear whether the one-box model correctly reflects the retention of PFOS and PFOA in the human body. The model uses the high half-lives of PFOS and PFOA in the human body reported in the literature[2,3] in combination with an uncertainty range of a factor of nine between highest and lowest value. In addition, the uncertainty of the volume of distribution (factor of five between highest and lowest value) contributes to the large overall uncertainty of the uptake doses derived from the one-compartment model. Nevertheless, the model is useful here because, while using only a few input parameters, it provides points of reference for the results obtained with the SceBRA method. For a more detailed PBPK model, much more comprehensive and detailed information about the fate of PFOS and PFOA in the human body is required.

The modeled uptake doses in seven consumer groups indicate a general trend of higher exposure in infants and decreasing exposure with increasing age. This is because of the higher intake of food per kilogram body weight of young consumers and age-specific behavior such as mouthing of objects and hands. In the high-exposure scenarios the dominating pathways are product- and age-dependent: hand-to-mouth contact with carpets for infants, toddlers, and children; uptake from food contact material for teenagers and adults.

This study proposes that the intake of contaminated food is the major pathway leading to exposure to PFOS and PFOA in the general public. Few studies have investigated the occurrence of perfluorinated chemicals in different types of food. Thus, the values describing the occurrence of PFOS and PFOA in the most important contact media are still fraught with considerable uncertainty. It is striking that all parameters with a high contribution to variance describe indirect pathways, and the most influential ones are all part of the food intake pathway (see Figs. 3 and 6). This is due to the uncertain and relatively high concentrations measured in contaminated foods, the continuous consumption of these foods in relatively large amounts, and the high gastrointestinal uptake fractions into the human body. Therefore, comprehensive food surveys and studies on gastrointestinal uptake are urgently required for a better understanding of the contribution of the food pathway to consumer exposure to PFCs. In future studies, different homologues of PFCAs and PFSAs should be measured in food samples so that homologue patterns in food and human blood can be compared. In blood PFOA is known to be the dominant PFCA homologue,[1] but the dominant homologue in total food samples is unknown.

In the case of PFOS, the main producer phased out production so that recent concentrations in food are likely to mainly reflect its past use in products and emission from production plants. In contrast, PFOA is still present in many consumer products, although only as a contaminant, and is further released into the environment, contaminating food and other environmental media. PFOA is also found in snacks such as pizza and microwave popcorn, which are found to cause notable exposure in this study. It is likely that these types of food are contaminated by contact with grease-repelling food contact materials. Because the exact source of PFC contamination of snacks is unknown, we included them in the food

Table X. Parameter Values Used for One-Compartment Pharmacokinetic Model for Low-Exposure (Low), Intermediate (Int), and High-Exposure (High) Scenarios

		Measured Blood Levels (ng/mL)			Volume of Distribution (mL/kg $_{bw}$)			Half-Lives (day)		
		Low	Int	High	Low	Int	High	Low	Int	High
North America	PFOS	17.7	31.2	52.3	1,300	3,600	6,000	800	3,200	7,800
	PFOA	4.3	8.1	12.0	1,300	3,600	6,000	500	1,600	4,900
Europe	PFOS	6.5	18.7	48.0	1,300	3,600	6,000	800	3,200	7,800
	PFOA	2.4	5.3	8.7	1,300	3,600	6,000	500	1,600	4,900

pathway representing indirect consumer exposure to PFCs.

Consumer exposure to PFOS and PFOA occurring via the inadvertent intake of contaminated house dust is also considered as indirect exposure. A significant positive correlation between the percentage of carpeting inside a house and the measured concentrations of PFCs in dust has been reported.[15] It may therefore be assumed that PFC-containing materials such as carpets, upholstery, and clothes in the household contribute considerably to the contamination of dust. In infants, toddlers, and children, the ingestion of dust contributes notably to the uptake of PFCs in the intermediate and high-exposure scenarios. This indicates that contaminated house dust may be a common source of exposure in these consumer groups, although less important than food. Under the current assumptions, ingestion of dust contributes notably to the uptake of PFOS and PFOA by teenagers and adults only in the high-exposure scenarios. This is because available studies on soil and dust ingestion show a large variability and low median amounts of ingested dust for teenagers and adults, resulting in low contributions for this pathway in the intermediate scenarios. If a dust intake rate of 50 mg/day is used for teenagers and adults (as suggested by the U.S. EPA[50]), the total uptake dose increases by 3% at most in the intermediate scenarios. If a dust intake rate of 100 mg/day is used for infants, toddlers, and children (as suggested by U.S. EPA[74]), the increase in total uptake is between 3% (children) and 19% (infants) in the intermediate scenarios. Hence the uptake by infants, toddlers, and children might be slightly underestimated by our results. More accurate data on the inadvertent ingestion of house dust are needed to clarify the importance of this pathway to consumer exposure.

Carpets may not only lead to PFC contamination of house dust but they also cause direct consumer exposure to PFOS and PFOA via hand-to-mouth contact. This pathway is important mainly for the exposure of infants, toddlers, and children. The age-specific behavior in these groups leads to more frequent contact with carpets and subsequent contact of the hands with the mouth, leading to oral uptake of chemical.[80,101] We assume that only older mill-treated carpets contain PFOS. However, because carpets have a relatively long lifetime we assume that consumers are still exposed to PFOS from carpets to some extent. In contrast, PFOA may be contained as a contaminant in older and in new products. Therefore, consumer exposure to PFOA may occur via mill-treated as well as home-treated carpets.

Consumers treating their clothes with impregnation sprays may be exposed substantially to PFOA contaminating the products. Users are exposed to significantly higher doses than consumers staying in the vicinity of the use location (results from two-box model with near and far field). Regular users of impregnation sprays may experience the greatest portion of their exposure from the use of such sprays (see high-exposure scenarios). This is still the case after translating the peak exposures during use into long-term averaged exposures.

Coated paper and cardboard used as food contact material may cause remarkable consumer exposure to PFOA. Current knowledge supports the assumption that PFCs can migrate from package materials or baking parchment into food.[27] Uncertainty exists, however, about the rates of migration and the fraction of PFOA in the materials prone to migration. Future research should try to clarify to what extent PFOA may be released from packages.

Our finding that other indirect and direct pathways (such as exposure to contaminated upholstery) contribute less to the uptake of PFOS and PFOA by consumers does not necessarily mean that these pathways can be fully neglected in further exposure assessments investigating PFCs. Our study indicates that these additional pathways, under the present assumptions for input parameter values, are less likely to cause significant uptakes of PFOS and PFOA than the pathways described in more detail in Sections 2.2 and 2.3. However, because high uncertainty is associated with input parameters such as concentrations in foods, future assessments should focus on specifying the range of possible input values more accurately.

This study does not consider precursor compounds that could be taken up and converted to PFOS or PFOA within the human body. It is likely that N-alkylperfluorooctanesulfonamides are converted to PFOS[102] in humans because they are known to be metabolically converted in rats, and that fluorotelomer alcohols taken up by humans are metabolically converted to PFOA.[103] In addition, there are many other potential precursor compounds that could contribute to exposure. Therefore, future studies should take into account precursors and their effect on consumer exposure to PFOS and PFOA.

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On page 253, Equation 1 should read

$$D_{\text{total}} = \sum_{j=1}^{n} D_j \text{ and not } D_{\text{total}} = \sum_{1}^{n} D_n.$$

On page 263, there is an error in the lowest panel of Figure 5. The legends for "paper, migr. into food, oral" and for "clothes, impregnating, inhalation" should be exchanged.

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