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Environmental Protection Agency

Contribution of different exposure pathways to the total human exposure to PFAS

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Report Title: Contribution of different exposure pathways to the human exposure to PFAS

Purpose of the Report:
This report covers one of seven knowledge building projects, which were launched in 2024 following the report of the Danish Knowledge Taskforce for PFAS on "Begrænsning af menneskers og miljøets eksponering for PFAS i Danmark – Del 1: Identifikation af vidanshull-er" (Baun et al., 2024). In the report, the Knowledge Taskforce for PFAS identified knowledge gaps within the PFAS area and proposed twelve knowledge-building projects that address some of these knowledge gaps. It was decided to initiate seven of these projects in 2024, where this is Project 6.

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Preface

This report covers one of seven knowledge building projects, which were launched in 2024 following the report of the Danish Knowledge Taskforce for PFAS on "Begrænsning af menneskers og miljøets eksponering for PFAS i Danmark – Del 1: Identifikation af videnshuller" (Baun et al., 2024). In the report, the Knowledge Taskforce for PFAS identified knowledge gaps within the PFAS area and proposed twelve knowledge building projects that address some of these knowledge gaps. It was decided to initiate seven of these projects in 2024. An overview is shown below.

Original project number and project title

Project 3: PFAS in residual products for agricultural use

Project 4: Screening of different types of food and feed for PFAS content

Project 5: Plan for biomonitoring for PFAS in the Danish population

Project 6: Contribution of different exposure pathways to the total human exposure to PFAS

Project 8: Further development of analytical methods for PFAS monitoring of environmental, food and human samples

Project 9: Conceptual model for transport and fate of PFAS at contaminated sites

Project 10: Diffuse pollution and pre-existing concentrations of PFAS

The Danish Knowledge Taskforce for PFAS was set up in August 2023 with the aim of collecting the existing national and international knowledge on PFAS. Based on the available knowledge, the expertise of the Knowledge Task Force and the results from the described knowledge building projects, the Knowledge Taskforce has in their concluding report suggested a series of actions, which will form the basis for the authorities' future focus and efforts against PFAS pollution.

The Knowledge Taskforce for PFAS is an independent expert group with the Danish Environmental Protection Agency as secretariat. The Knowledge Taskforce has the following members: Professor Anders Baun, Technical University of Denmark (chairperson); Chief physician Ann Lyngberg, Department of Occupational and Social Medicine, Holbæk Hospital; Professor Anne Marie Vinggaard, Technical University of Denmark; Associate Professor Bjarne W. Strobel, University of Copenhagen; Deputy Head of Department John Jensen, Aarhus University; Professor Katrin Vorkamp, Aarhus University; Professor Poul L. Bjerg, Technical University of Denmark; Professor Tina Kold Jensen, University of Southern Denmark; Associate Professor Xenia Trier, University of Copenhagen.

The present project “Contribution of different exposure pathways to the total human exposure to PFAS” is described as project no. 6 in Baun et al. (2024). The project was carried out in the period 19 April 2024 – 31 December 2024.

Summary

This report evaluates human exposure to individual per- and polyfluoroalkyl substances (PFAS) across the following key media: food and drinking water, food contact materials, indoor air and dust, outdoor dust, consumer products, and occupational settings, the latter with no systematic or exhaustive approach. It considered exposure through ingestion, inhalation and dermal contact. The study identified the most frequently reported compounds, estimated their uptake doses and quantified their contributions to the overall uptake. Uptake doses included the direct exposure to a given PFAS and an indirect exposure component via the biotransformation of a precursor. These precursors comprise a variety of less persistent PFAS some of which can be converted to persistent PFAS such as perfluorooctanoic acid (PFOA) or other legacy PFAS within the human body or in the environment.

Methodology:

A comprehensive literature review identified PFAS in relevant exposure media. The top five PFAS that were reported most frequently for each exposure medium were selected for further quantitative analysis, i.e. the calculation of uptake doses. Additionally, relevant precursors, such as fluorotelomer alcohols (FTOHs) and perfluoroalkyl phosphate diesters (diPAPs), were included to assess the indirect exposure to the selected compounds via biotransformation. It should be noted that the five most frequently reported PFAS are influenced by the selection of PFAS for each study, carrying a risk of overlooking less commonly analysed PFAS.

Daily uptake doses were calculated for ingestion, inhalation, and dermal exposure, depending on which exposure pathway was considered relevant for the different source categories. Calculations incorporated age-specific factors (e.g. body weight, intake rates) and distinguished between direct exposure of a given PFAS and its indirect exposure (via the biotransformation of a precursor). Biotransformation factors for precursors were retrieved from the literature.

Two exposure scenarios were considered: A low exposure (LE) and a high exposure scenario (HE). In LE the lowest median or mean concentrations in the different media were used together with the lowest values of the parameters included in the uptake dose calculations. Likewise, in HE the highest values were used.

Key results:

In total, 57 different PFAS were reported in food, 55 in drinking water, 70 in food contact materials, 60 in indoor dust/air, 43 in outdoor dust/air, 189 in consumer products and 158 in occupational settings. PFOA, perfluorooctane sulfonate (PFOS), perfluorononanoic acid (PFNA), perfluorohexanoic acid (PFHxA) and perfluoroheptanoic acid (PFHpA) were key compounds across media, while long-chain PFCAs (such as perfluoroundecanoic acid, PFUnDA) were characteristic for food (in particular animal-based food and seafood), and perfluorobutane sulfonate (PFBS) was detected in drinking water. FTOHs and diPAPs were main precursor groups identified in multiple media except food, however, uptake rates could not be calculated for diPAPs because of missing data.

In the HE scenario, nearly all PFAS exposure was related to food ingestion. However, the food data included fish and seafood from China and the USA as well as from sites with suspected local emission sources and might exceed typical Danish food exposure. Dermal uptake from consumer products also contributed with approximately 5-6% under this scenario, while all other exposure sources and pathways were insignificant.

In the LE scenario, the main PFAS exposure (> 89%) was from dermal contact with consumer products. The second highest contribution was from indoor air with 3-10%, mainly via FTOHs in the gas phase. Food contributed <1% under this scenario, which was likely related to very low levels of PFAS in milk, dairy products, alcoholic beverages and game birds used in this scenario, potentially underestimating the typical Danish food exposure. Exposure from indoor dust was similar to food in the LE scenario, also with significant direct and indirect exposure contributions from precursors. The concept of high vs. low exposure scenarios as well as relative and absolute contributions is illustrated in Figure 0.

Children generally had higher uptake doses than adults, related to lower body weight and, in some cases, higher uptake rates (e.g. of dust). Regarding PFAS exposure from consumer products, ingestion was considered for

children (hand-to-mouth activity; licking), while inhalation (from sprays) was only considered for adults. Neither exposure route resulted in a significant contribution.Occupational exposure has not been in the main scope of this study. However, specific occupations (e.g., firefighting, manufacturing) show important localized contributions, involving both direct and indirect exposure.Conclusions and outlook:

While several methodological limitations have to be considered, the study has shown

- that the number of different PFAS in exposure media, in particular consumer products, is substantial and probably not sufficiently covered in typical analyses,
- that some PFAS are linked to specific exposure media, such as PFUnDA in food and PFBS in water. The long-chain PFCAs might be underrated in the current focus of only four PFAS in food,
- that food is the relevant exposure pathway in HE scenarios. However, market basket analyses of a typical Danish diet will give more precise and accurate results than the HE and LE approaches of this study,
- that dermal uptake from consumer products can be important under LE scenarios and need more attention, also considering the variety of different PFAS in consumer products,
- that precursors should be considered in exposure assessments, in particular FTOHs and diPAPs, both for their direct exposure and their biotransformation to persistent PFAS.

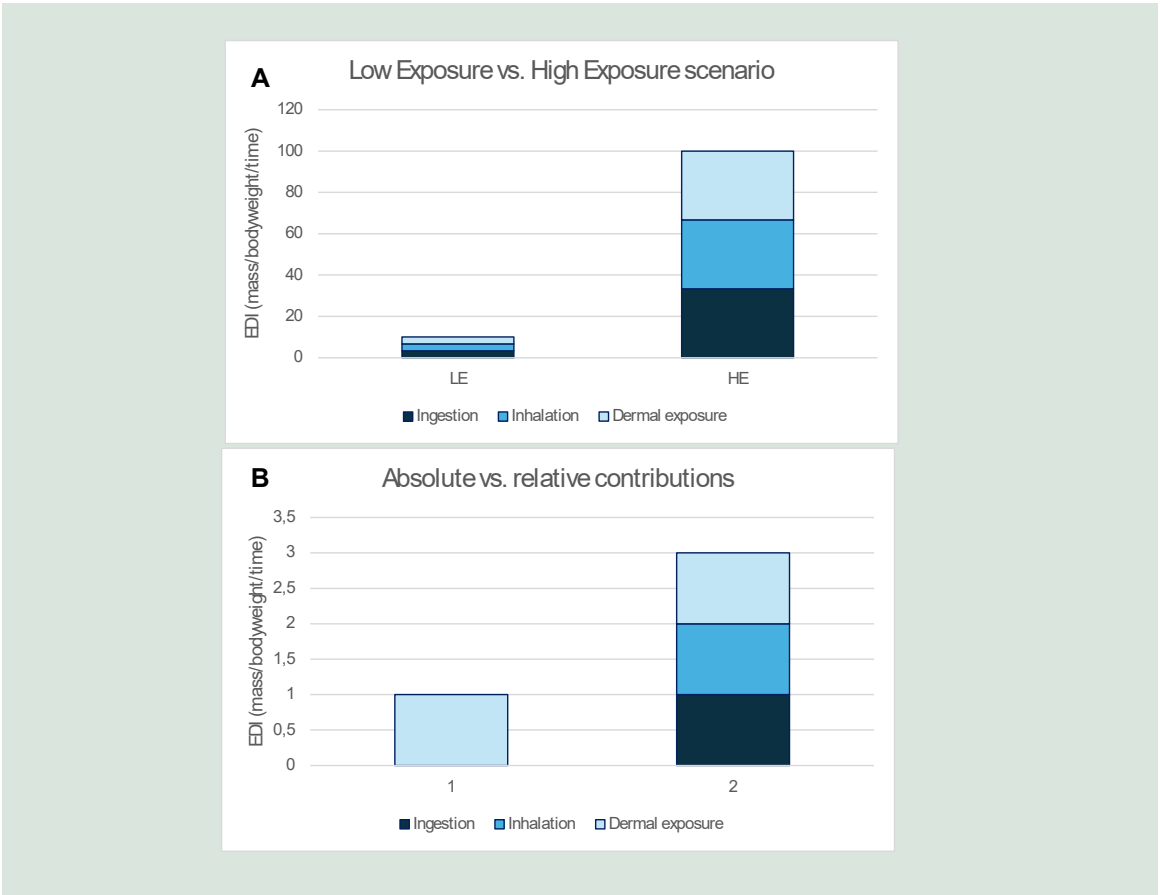

FIGURE 0. A: Illustration of Low Exposure (LE) vs. High Exposure (HE) scenario, using arbitrary numbers of 10 and 100 for Estimated Daily Intakes (EDI) in mass/bodyweight/time, for example ng/kg bw/day. In both cases, the three exposure pathways contribute equally with 33.3%, but the absolute exposure levels differ. B: Illustration of relative vs. absolute exposure contributions to the EDI. In both cases, dermal exposure is given an arbitrary number of 1. Dermal exposure has relative contributions of 100% and 33.3% in case A and case B, respectively, although the absolute exposure is identical. Note that these are schematic illustrations with arbitrary numbers, no real uptake doses.

1. Introduction

Per- and polyfluoroalkyl substances (PFAS) are a group of several thousand chemicals widely used in industrial processes and applications and in consumer products. An expert group under the Organization for Economic Co-operation and Development (OECD) proposed a definition according to which PFAS are fluorinated substances with at least one fully fluorinated methyl or methylene carbon atom (OECD, 2021). Some of the most frequently studied compounds include perfluorooctanoic acid (PFOA) and perfluorooctane sulfonic acid (PFOS), which represent perfluorocarboxylic acids (PFCAs) and perfluorosulfonic acids (PFSAs), respectively. Together with other acids, they are summarized as perfluoroalkylic acids (PFAAs). The wide definition proposed by the expert group under OECD also includes other compounds, commonly described as precursors (to more stable PFAAs).

While PFAS are generally considered persistent, due to the stable carbon-fluorine bond, precursors are typically less persistent and can be transformed to more stable PFAS, for example fluorotelomer alcohols (FTOHs) can be oxidized to PFCAs. Considering this transformation potential, together with the wide PFAS definition, not all PFAS are persistent. In this study we also consider the human exposure to precursors which can be transformed in the environment or in the body to the more frequently studied and stable PFAAs.

PFAS are used for their unique properties, including water, oil, and stain repellence, and their resistance to heat. Among other applications, PFAS are found in textiles (e.g., waterproof clothing and upholstery), food packaging, non-stick cookware, firefighting foams, cosmetics, medical devices, and electronics. They also play a role in metal plating, where they provide surface protection, and in construction for insulation materials (ECHA, 2023; Brunn et al., 2023; Podder et al., 2021). The PFAS annual tonnages that are placed on the market are summarized inTable 1, indicated as volume ranges.

Table 1: PFAS uses in the EU based on data from 2020 (ECHA, 2023).

Application	Tonnage range	Tonnage (t/year)
Applications of fluorinated gases	5	> 10,000
Textiles, upholstery, leather, apparel and carpets	5	> 10,000
Medical devices	5	> 10,000
Manufacture	5	> 10,000
Food contact materials and packaging	5	> 10,000
Transport	5	> 10,000
Construction products	4	1000-10,000
Electronics and semiconductors	4	1000-10,000
Lubricants	4	1000-10,000
Petroleum and mining	4	1000-10,000
Energy sector	4	1000-10,000
Metal plating and manufacture of metal products	3	100-1000
Cosmetics	2	10-100
Consumer mixtures	2	10-100
Ski wax	1	0-10

PFAS emissions to the environment have been divided into direct and indirect sources, with direct sources resulting from the manufacture and use of PFAS, while indirect sources are those where PFAS are present as chemical reaction impurities or where precursors may degrade to form PFAS (OECD, 2015). However, strictly speaking, this primarily applies to PFAA sources. Since precursors are also PFAS, based on the updated proposal for a

PFAS definition (OECD, 2021), their emission to the environment is a direct PFAS emission. Thus, considering the broad definition of PFAS, the distinction between direct and indirect sources might become obsolete. However, it is important to note that persistent PFAS, such as PFAAs, can have direct and indirect sources, the latter via their precursors. PFAS can be released to different environmental compartments, i.e. to the aquatic environment, for example from industrial and municipal wastewater, to the terrestrial environment, for example from the disposal of PFAS-containing products, and to the atmosphere with airborne emissions.

In the atmosphere, volatile PFAS such as FTOHs can travel long distances before being deposited via rain and snow. PFAS are also detected in remote regions like the Arctic, following transport by long-range atmospheric pathways and ocean currents or local emissions (Lohmann et al., 2024). Water is an important transport pathway, as ionic PFAS are highly water soluble, making them prone to contaminating rivers, lakes, and groundwater. In soil, long-chain compounds such as PFOA might accumulate over time and only migrate to groundwater gradually (ECHA, 2023; Brunn et al., 2023). However, short-chain PFAS compounds such as perfluorobutane sulfonate (PFBS) are more water-soluble and can leach into drinking water sources more easily. While being less bioaccumulative than the long-chain PFAAs, they pose significant environmental risks due to their persistence and mobility, and their long-term effects remain poorly studied (Brendel et al., 2018). Their growing use as alternatives to bioaccumulative PFAS, such as PFOS and PFOA, emphasizes the need for further investigation (Kjølholt et al., 2015).

Humans can be exposed to PFAS through ingestion, inhalation, and dermal contact:

- **Ingestion:** Contaminated drinking water and food, such as fish from polluted waters and/or high in the food chain, are the primary routes of exposure. PFOA, PFOS, perfluorononanoic acid (PFNA) and other long-chain PFCAs accumulate in the food chain.
- **Inhalation:** Airborne particles, especially in indoor environments with PFAS-treated products like carpets and textiles, can lead to exposure. Volatile FTOHs are present in the gas phase (Morales-McDevitt et al., 2021).
- **Dermal uptake:** PFAS exposure through skin contact can occur from the use of consumer products such as waterproof clothing, cosmetics, and cleaning products, although this is considered a less significant pathway compared to ingestion (ECHA, 2023; Brunn et al., 2023).

Ingestion of food and water is generally found to be the most important one for human exposure due to the PFAS potential for bioaccumulation and their presence in some water bodies. Drinking water can represent a significant source if it is contaminated with mobile PFAS compounds such as PFBS. Inhalation, although generally considered less significant than ingestion, can be important in indoor environments where PFAS-treated consumer products are prevalent. Dermal exposure, while expected to be minor, can still contribute to overall exposure, particularly for workers handling PFAS-containing materials. However, this project primarily considers exposure of the general population in contrast to exposure from specific workplaces where PFAS might be used.

Although several studies have investigated sources of PFAS exposure, significant knowledge gaps remain that need to be addressed to provide a more comprehensive basis for preventing harmful levels of PFAS exposure (Baun et al., 2024). This report focuses on the general population, with an emphasis on Denmark, while also incorporating findings from the Nordic countries, Europe, and globally. Based on existing data, the objective of the report is to contribute new qualitative and quantitative insights into the following topics:
A) Which PFAS are primarily present in exposure-relevant media?
B) Which exposure pathways are most relevant for different individual compounds?
C) How much does each source contribute to the total human exposure to various PFAS?
D) How do the contributions of individual sources compare to each other?
E) Are there obvious differences across different age groups?

2. Methods

2.1. Source categories

Based on the exposure characterization by De Silva et al. (2021) (Figure 1), the following exposure sources were studied:

- Food, drinking water and food contact materials
- Indoor air and dust
- Outdoor air and dust
- Consumer products, cosmetics, clothes, carpet, furniture, electronics etc.
- Work(place)/occupational

Occupational exposure at the workplace was not studied systematically. It had been discussed and agreed on with the advisory group of the project that occupational exposure would not be in the scope of the study, since it is not the core expertise of the project group. Information on occupational exposure will be included to the extent that it is found in the general literature search, but it is not a focus area.

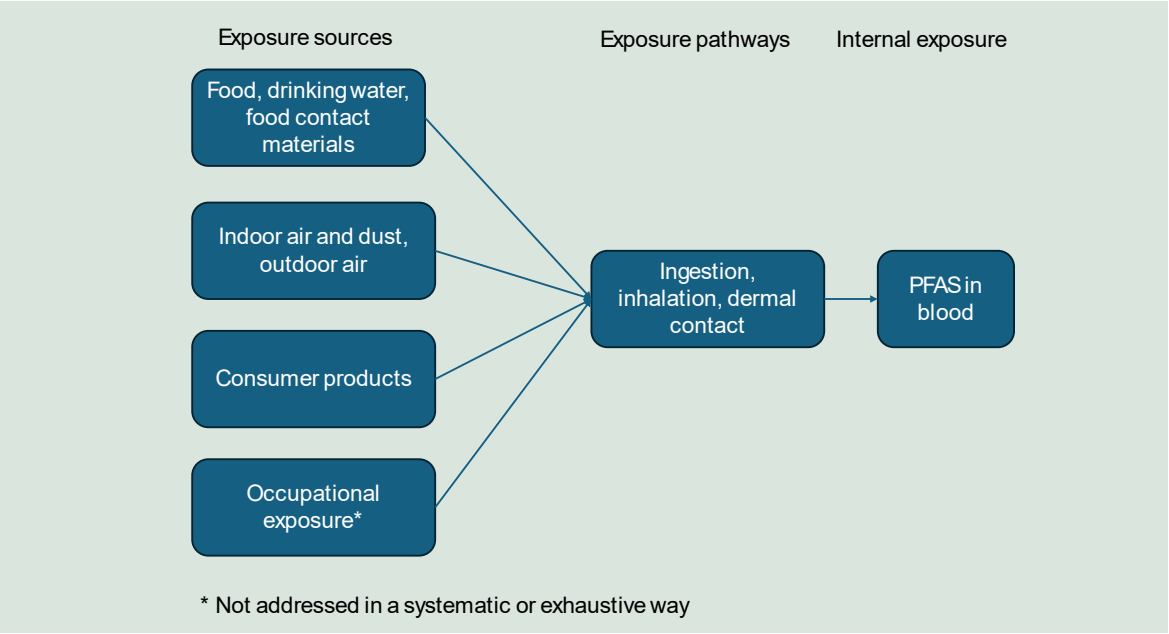

FIGURE 1. Schematic representation of PFAS exposure. Redrawn from De Silva et al. (2021).

2.2. Search criteria

A literature search in Web of Science (WoS) was performed with the search string shown in Figure 2, including references until June 2024. This search resulted in 496 references. A similar search for Scandinavian literature was done in Danish in Google Scholar (Figure 3), which gave 75 references.

TS=(PFAS OR PFCA OR PFSA OR PFOA OR PFOS OR FTOH)

AND

TS=(monomer OR "long-chain" OR "short-chain")

AND

TS=(food OR fish OR packaging OR "drinking water" OR groundwater OR indoor OR outdoor OR dust OR particles OR "consumer product" OR pharmaceutical OR air OR cosmetic OR clothes)

AND

TS=("exposure pathway" OR human OR Denmark OR work OR concentration)

FIGURE 2. Search string for literature review in Web of Science.

(PFAS OR PFCA OR PFSA OR PFOA OR PFOS OR FTOH)

AND

(monomer OR langkæde OR enkeltstof OR kortkæde)

AND

(eksponering OR kilde OR human OR transport OR Danmark OR bidrag OR befolkning OR arbejdsmiljø OR lokal OR alder OR geografi OR koncentration OR optag OR medie)

AND

(fødevare OR fisk OR emballage OR drikkevand OR grundvand OR indeklima OR indendørs OR udendørs OR støv OR partikler OR forbrugerprodukter OR lægemidler OR vand OR luft OR kosmetik OR tøj)

FIGURE 3. Search string for literature review in Google Scholar.

The results were reviewed and non-relevant articles were removed from the list. Additional references were included from the articles reviewed in the first step. For food, the focus was on new studies that have been published since the risk assessment by the European Food Safety Authority (EFSA), resulting in tolerable weekly intakes for the sum of PFOS, PFOA, PFNA and perfluorohexane sulfonate (PFHxS) (EFSA, 2020). The final numbers of references for the project are summarized in Table 2.

Table 2: Summary of the literature search.

Exposure sources	Number of references
Food, drinking water and food contact materials	89
Indoor air and dust	28
Outdoor air, particles	12
Consumer products	51
Occupational exposure*	10
Relevant articles with general information	13

*not addressed in a systematic or exhaustive way

2.3. Data compilation for qualitative and quantitative analysis

Information on the detected PFAS was extracted from the selected references and compiled in a spreadsheet (Table 3). A separate table was prepared for each of the five categories in Section 2.1. The qualitative analysis focused on the occurrence of PFAS in the relevant exposure media, i.e. the identity of the compound (without considering the concentration), the location and year of detection. The table included specific information on the different exposure sources and an initial assessment of relevant exposure pathways (i.e. inhalation, ingestion, dermal uptake). Each row in the table included one individual PFAS per exposure source and study. Years were pooled, i.e. detections of a PFAS in a sample in several years only counted as one.

In a second step, compounds were selected for which uptake doses were calculated, i.e. an Estimated Daily Intake (EDI). This selection for the quantitative part of the study was based on the following steps and criteria:

1. The number of entries (rows) were counted for each PFAS and each exposure source, e.g. PFOS in fish.
2. The five PFAS with most counts were prioritized for the quantitative analysis, i.e. the calculation of uptake doses.
3. In addition, relevant compounds not included in the top five were added according to the following criteria:
    a. Precursors that are associated with the top five compounds
    b. Compounds that are associated with several exposure media, e.g. consumer products and dust

This means that the selection of compounds was primarily based on the frequency of detection and thus strongly determined by the availability of information. Thus, the selection of specific compounds for the calculations was not based on risk or hazard, neither were new nor rarely studied compounds prioritized. It has to be noted that this selection is likely to include a research bias as only those compounds are reported that are included in the chemical analysis. Often, a standard set of PFAS are included in the analysis rather than a matrix-specifically optimized set of PFAS. Consequently, the results are dominated by frequently studied PFAS such as PFOS and PFOA. Furthermore, the effects of bans or substitutions will not be reflected in the findings. All compounds beyond the five selected ones can, however, be also found in the full list in Appendix A.

Following the selection of the top five and potentially additional compounds, the references were revisited, and the reported concentrations in the respective matrices were added to the table. Median and mean values were the first and second choice, respectively. If only concentrations below the limit of quantification (LoQ) were available, the value 0.5*LoQ was used, if LoQ was given in the reference. Concentrations below the limit of detection (LoD) were omitted.

Table 3: Structure of the data compilation.

Matrix										Exposure pathways			
Food and drinking water	Indoor air and dust	Outdoor air and dust	Consumer products	Workplaces	Compound	Location	Year	Precursor relevant	Median conc. (unit)	Inhalation	Ingestion	Dermal	Ref.

2.4. Exposure scenarios

Two exposure scenarios were calculated, referred to as “High Exposure (HE)” and “Low Exposure (LE)” and roughly representing “worst case” and “best case” situations (Figure 4). Throughout the report results are given for two exposure scenarios that use different values for the quantitative assessment of uptake doses:

- The High Exposure (HE) scenario represents the highest uptake dose for a given compound, medium and exposure pathway. This scenario is based on the highest median (or mean) concentration for each PFAS in each of the studied media. It also included the highest values of the parameters that are entered in the equations for uptake dose calculations in section 2.5.
- The Low Exposure scenario (LE) represents the lowest uptake dose for a given compound, medium and exposure pathway. Contrasting the HE, this scenario used the lowest median (or mean) concentrations for a given PFAS in the different media, combined with lowest values of the parameters that are entered in the equations for uptake dose calculations in section2.5.

The highest (or lowest) median concentration typically represents one study. If a study included several comparable samples, for which a median (or mean) was reported, this was included in a ranking of median concentrations. The lowest one was used for the LE scenario, while the highest one was used for the HE scenario. For food in particular, it should be noted that the selection of the highest or lowest values for food exposure does not represent a realistic diet which consists of many components with different PFAS concentrations. The calculations were not based on typical dietary habits or frequently consumed food products, as it was not possible within the frame for the project to collect this type of data. As will be discussed in section 3, the concentration of PFAS in different food items varies substantially, with extremely low and high values determining the LE and HE scenarios for food.

It should be also noted that the equations include a biotransformation factor that quantifies the extent to which a precursor is transformed to a more persistent PFAS. A high biotransformation factor results in a high formation of the persistent PFAS from the precursors.

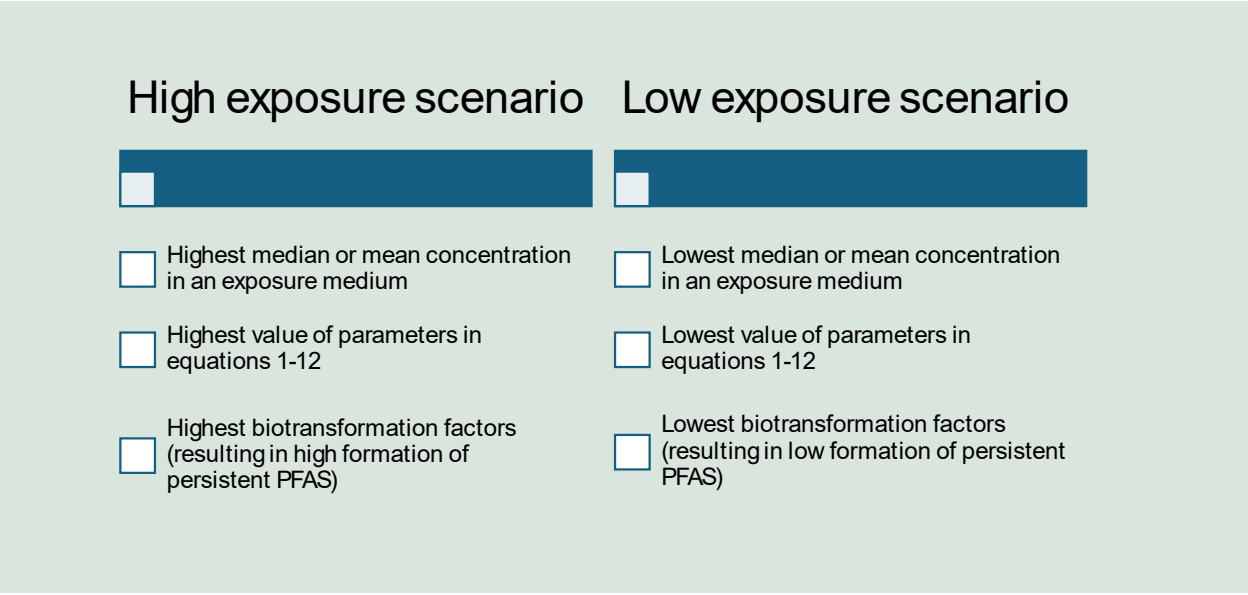

FIGURE 4. Summary of high exposure (HE) and low exposure (LE) scenarios.

2.5. Calculation of uptake dose

The concentrations in the respective matrices were used to calculate the uptake doses for each of the selected compounds and the three exposure pathways (inhalation, ingestion, dermal). The uptake calculations consider direct exposure to a persistent PFAS as well as the indirect exposure to this persistent PFAS via the uptake of a

precursor and its biotransformation. In addition, direct exposure to the precursor is considered where relevant. It is important to note that measures of internal exposure, i.e. PFAS in blood, do not usually yield the precursors due to their transformability. Thus, measurements of internal exposure do not directly reflect the external exposure situation.

To derive body-internal doses of the persistent PFAS from precursors, knowledge regarding the biotransformation yield is needed (FEA, 2014). It is assumed that elimination of the metabolite (i.e. the persistent PFAS) is much slower than the transformation of the precursor compound. Thus, the indirect exposure to a persistent PFAS can be calculated by multiplying the internal exposure of the precursor compound with a biotransformation factor ($F_{biotransf}$) (FEA, 2014; Gebbink et al., 2015). The biotransformation factors are given as ranges inTable 4. As described in section 2.4, the lowest and highest biotransformation factors are used to represent the LE and HE scenarios, respectively. While this reflects the exposure to the persistent PFAS (i.e. a high transformation of the precursor results in a high formation of the metabolite), it affects the direct exposure to the precursor in the opposite way.

In the calculation of uptake doses for the precursors, i.e. the direct exposure to the precursor, the concentration of the persistent PFAS = 0. The precursor concentration is multiplied with (1- $\Sigma F_{biotransf}$) according to the equations below. This concentration represents the fraction of the precursor that does not biotransform into the respective PFAS in the body.

Uptake doses have been calculated for adults and for children.

2.5.1. Food, drinking water and food contact materials

The uptake doses have been calculated as EDIs (ng/kg bw/d) via ingestion of food and drinking water from Trudel et al. (2008), Poothong et al. (2020) and Gebbink et al. (2015):

$$\mathrm{EDI}_{\mathrm{ingestion,food}} = \left(\frac{\mathrm{Q_{food}}}{\mathrm{BW}} \cdot \mathrm{F_{uptake,GIT}}\right) \cdot \left(\mathrm{C_{PFAS,food}} \cdot \mathrm{C_{precurs,food}} \cdot \mathrm{F_{biotransf}}\right) \quad \textbf{(Eq.1)}$$

$$\mathrm{EDI}\ \ ingestion, drinking\_water = \left(\frac{Q_{drinking\_water}}{BW} \cdot F_{uptake,GIT}\right) \cdot \left(C_{PFAS,drinking\_water} \cdot C_{precurs,drinking\_water} \cdot F_{biotransf}\right) \quad \textbf{(Eq.2)}$$

$C_{PFAS,food}$ and $C_{PFAS,drinking\_water}$ are the concentration of the persistent PFAS in food and drinking water, respectively (ng/g). $C_{precurs,food}$ and $C_{precurs,drinking\_water}$ are the concentration of the precursor in food and drinking water, respectively (ng/g). Q_{food} and $Q_{drinking\_water}$ are the amount of food and drinking water, respectively, consumed daily (g/d). BW is the average body weight (kg). $F_{uptake,GIT}$ is the uptake fraction of compound via the gastrointestinal tract (GIT) (unitless). $F_{biotransf}$ is the biotransformation factor in the body (unitless), see Table 4.

The uptake doses via ingestion of food in contact with food materials were calculated as EDI rates (ng/kg bw/d) from Trudel et al. (2008):

$$\mathrm{EDI}_{\mathrm{ingenstion,food,cont,mat}} = \left(\frac{\mathrm{Q_{food}} \cdot \mathrm{r_{migr}} \cdot \mathrm{MF_{PFAS}} \cdot \mathrm{f_{food_{mat}}} \cdot \mathrm{A_{contact}} \cdot \mathrm{f_{contact}}}{\mathrm{BW}} \cdot \mathrm{F_{uptake,GIT}}\right) \cdot \left(\mathrm{C_{PFAS,food_{cont_{mat}}}} \cdot \mathrm{C_{precurs,food_{cont_{mat}}}} \cdot \mathrm{F_{biotransf}}\right)$$

(Eq. 3)

$C_{PFAS,food\_cont\_mat}$ is the concentration of the persistent PFAS in food contact material (ng/dm^2). $C_{precurs,food\_cont\_mat}$ is the concentration of the precursor in food contact material (ng/dm2). Q_{food} is food consumption per day (kg), r_{migr} is the migration rate of PFAS incl. precursor from food contact material into food (h^{-1}). MF_{PFAS} is the market fraction of food contact material treated with PFAS (incl. precursors) (unitless). f_{food_mat} is the contact frequency of food with treated material (d^{-1}). $A_{contact}$ is the contact area of food with contact material (6 dm^2/kg). $f_{contact}$ is the

contact time of food with the contact material (h). BW is the average body weight (kg). $F_{uptake,GIT}$ is the uptake fraction of PFAS incl. precursor via the GIT (unitless). $F_{biotransf}$ is the biotransformation factor in the body (unitless), see Table 4.

2.5.2. Air and dust

The uptake doses from inhalation of indoor air were calculated as EDIs (ng/kg bw/d) from Trudel et al. (2008), Gebbink et al. (2015), Poothong et al. (2020) and Morales-McDewitt et al. (2021):

$$EDI_{inhalation,air} = \left(\frac{InhR \cdot F_{time,in}}{BW} \cdot F_{uptake,lung}\right) \cdot \left(C_{PFAS.air,in} \cdot C_{precurs,air,in} \cdot F_{biotransf}\right) \quad \textbf{(Eq. 4)}$$

$C_{PFAS,air,in}$ is the concentration of the persistent PFAS in indoor air (ng/g). $C_{precurs,air,in}$ is the concentration of the precursor in indoor air (ng/g). $F_{time,in}$ is the fraction of time spent indoor in a day (unitless). BW is the average body weight (kg). $F_{uptake,lung}$ is the uptake fraction of compound via the lungs (unitless). $F_{biotransf}$ is the biotransformation factor in the body (unitless). InhR is the inhalation rate (m^3/d).

Correspondingly, the EDIs (ng/kg bw/d) of chemicals on dust via ingestion and dermal exposure with dust have been calculated from Gebbink et al. (2015), Winkens et al. (2018), Wu et al. (2020) and Zhu et al. (2023) :

$$EDI_{ingestion,dust} = \left(\frac{InhR \cdot F_{time,}}{BW} \cdot F_{uptake,GIT}\right) \cdot \left(C_{PFAS.dust} \cdot C_{precurs,dust} \cdot F_{biotransf}\right) \quad \textbf{(Eq. 5)}$$

$$EDI_{dermal,dust} = \left(\frac{BSA \cdot DAS \cdot F_{uptake,skin} \cdot t}{BW}\right) \cdot \left(C_{PFAS.dust} \cdot C_{precurs,dust} \cdot F_{biotransf}\right) \quad \textbf{(Eq. 6)}$$

$C_{PFAS,dust}$ is the concentration of the persistent PFAS in dust (ng/g). $C_{precurs,dust}$ is the concentration of the precursor in dust (ng/g). IngR describes the ingestion rate of dust (g/d). F_{time} is the fraction of time spent in the specific exposure situation in a day (unitless). BW is the average body weight (kg). $F_{uptake,GIT}$ is the uptake fraction of compound via the GIT (unitless). $F_{biotransf}$ is the biotransformation factor in the body (unitless). BSA is the exposed body surface area (cm^2). DAS is dust adhered to skin (mg/cm^2). $F_{uptake,skin}$ is the fraction of compound absorbed by skin (unitless). T is the time spent in the exposed environment (h/d).

Due to considerably lower concentrations in outdoor air, exposure to PFAS was considered negligible. For this reason, no EDIs were calculated for outdoor air. Outdoor dust was calculated according to Eq. 5.

2.5.3. Consumer products

PFAS concentrations in textiles, upholstery, leather, apparel, carpets, electronics, cosmetics and consumer mixtures are summarized in Appendix A. A consumer in direct contact with PFAS-containing clothes and other products can be directly exposed via dermal exposure. A less frequent exposure pathway is a situation where children lick the clothing/textiles, or a hand-to-mouth transfer of compounds that are on the children's hands. Other exposure pathways are inhalation of evaporated volatile compounds in indoor air and inhalation of textile dust. These are emphasized when the textiles are impregnated. Use of impregnation products to shoes, curtains, tablecloths, carpets, bed linen and furniture can lead to both inhalation and dermal exposure of PFAS (Lassen et al., 2015).

The EDIs (ng/kg bw/d) of chemicals in consumer products were calculated for:

- Dermal uptake via contact with textiles, i.e. clothing, furniture, carpets and masks (FEA, 2014, Poulsen et al., 2018; 2021)

$$EDI_{dermal,textiles} = \left(\frac{TF_{PFAS,skin} \cdot A_{skin} \cdot F_{uptake,skin}}{BW}\right) \cdot \left(C_{PFAS.textiles} \cdot C_{precurs,textiles} \cdot F_{biotransf}\right) \quad \textbf{(Eq. 7)}$$

$C_{PFAS,textiles}$ and $C_{precurs,textiles}$ are the concentration of the persistent PFAS and precursors, respectively, in textiles (µg/m^2). $TF_{PFAS,skin}$ is the fraction of compound transferred from textiles to skin (unitless). A_{skin} is the body surface area that is in contact with clothing, textiles and carpets (cm^2). BW is the average body weight (kg). $F_{uptake,skin}$ is the uptake rate of compound through skin (h^{-1}).

FEA (2014) assumed that the skin area in contact with clothes (A_{skin}) comprises the whole body except head and feet. This assumption of body surface in contact with PFAS-treated textiles probably presents an overestimation of the actual contact area since jackets are typically worn as a second or third layer without direct skin contact. In this report the fraction of the body surface that is in contact with the clothes, typically jackets, constitutes 5% (LE) and 50% (HE).

The fraction of the human body in contact with carpets depends largely on the individual's activity and age. For young children, particularly toddlers who crawl, play, or sit on the floor, 15-25% of their body surface area can be estimated to come into direct contact with carpets. This estimate is based on their typical behaviour, as they often spend extended periods of time on carpets, with their hands, knees, and legs being the primary body parts in contact with the carpet. However, the rest of their body, such as their torso and head, is not in constant contact with the carpet (US EPA, 2002, 2011). The contact time and surface area relative to body size lead to higher dermal exposure risk in children compared to adults. Adults' contact is usually limited to the feet (while barefoot) or hands (during cleaning or sitting on the floor). This would likely involve less than 5% of their total body surface area (US EPA, 2002, 2011).
The uptake of PFCAs and FTOHs via the skin ($F_{uptake,skin}$) was derived from a study by Fasano et al. (2005) who estimated the absorption of PFOA through human skin. A cumulative fraction of chemical absorbed over 48 h was found to be 0.048. Dividing the cumulative fraction by the duration of the test (48 h) leads to a fraction of chemical absorbed per hour of 0.01 h^{-1}. $F_{biotransf}$ is the biotransformation factor in the body (unitless), see Table 4.

$C_{precurs,textiles}$ is given in ng/g for fabric masks (Poulsen et al., 2021). The concentration in $\mu g/m^2$ is found from:

$$C_{precurs,textiles}\left(\mu g/m^2\right) = \frac{C_{precurs,textiles}\left(ng/g\right)\cdot}{\text{Surface area mask } (m^2)\cdot 1000\left(ng/\mu g\right)} \quad \textbf{(Eq. 8)}$$

According to the assumptions in Poulsen et al. (2021) the contact area between mask and skin (A_{skin}) is set to the total area of the mask, as a worst-case approach. Furthermore, children are assumed to use adult masks and therefore have the same contact area.

- Ingestion via hand-to-mouth contact with various consumer products, for children (Trudel et al., 2008, FEA, 2014):

$$EDI_{\text{hand-to-mouth}} = \left(\frac{TF_{PFAS,skin}\cdot TF_{skin,saliva}\cdot f_{htm}\cdot t_{exp,prod}\cdot A_{skin\text{-}mouth}\cdot F_{uptake,GIT}}{BW}\right)\cdot\left(C_{PFAS,product}\cdot C_{precurs,product}\cdot F_{biotransf}\right) \quad \textbf{(Eq. 9)}$$

$C_{PFAS,product}$ and $C_{precurs,product}$ are the concentration of the persistent PFAS and precursors, respectively, in the product ($\mu g/m^2$). $TF_{skin,saliva}$ is the fraction of compound transferred from skin to saliva (unitless). f_{htm} is the frequency of hand-to-mouth events (h^{-1}). $t_{exp,prod}$ is the time exposed to the product (h/d). $A_{skin\text{-}mouth}$ is the skin surface area that is in contact with the mouth (cm^2). BW is the average body weight (kg). $F_{uptake,GIT}$ is the uptake of compound through the GIT (unitless). $F_{biotransf}$ is the biotransformation factor in the body (unitless), see Table 4.

- Inhalation and dermal contact from use of impregnation sprays (Trudel et al., 2008, FEA, 2014):

$$EDI_{\text{inhalation,spray}} = \left(\frac{f_{imp}\cdot t_{imp}\cdot r_{spray}\cdot F_{resp}\cdot V_{inhal}\cdot MF_{imp}\cdot F_{uptake,lung}}{BW}\right)\cdot\left(\frac{t_{spray,NF}}{V_{NF}}+\frac{t_{spray,FF}}{V_{FF}}\right)\cdot\left(C_{PFAS,spray}\cdot C_{precurs,spray}\cdot F_{biotransf}\right) \quad \textbf{(Eq. 10)}$$

$$EDI_{\text{dermal,spray}} = \left(\frac{f_{imp}\cdot t_{imp}\cdot q_{spray}\cdot t_{esp}\cdot MF_{imp}\cdot F_{uptake,skin}}{BW}\right)\cdot\left(C_{PFAS,spray}\cdot C_{precurs,spray}\cdot F_{biotransf}\right) \quad \textbf{(Eq. 11)}$$

$C_{PFAS,spray}$ and $C_{precurs,spray}$ are the concentration of the persistent PFAS and precursors, respectively, in the spray (ng/g). f_{imp} is the frequency of impregnation spray use (d^{-1}). t_{imp} is the duration of impregnation spray use (min). r_{spray} is the spray emission rate (g/min). F_{resp} is the fraction of respirable spray droplets (unitless). V_{inhal} is the inhalation volume during spray use (m^3/h). MF_{imp} is the market fraction of impregnation sprays containing the compound (unitless). VNF is the near-field volume around the consumer (m^3). VFF is the far-field volume around the consumer (m^3). $t_{spray,NF}$ is the time exposed to spray droplets, near-field (h). $t_{spray,FF}$ is the time exposed to spray droplets, far-field (h). q_{spray} is the quantity of spray deposited on skin (g/min). t_{exp} is the time before skin is washed (h). BW is the average body weight (kg). $F_{uptake,lung}$ is the uptake of compound through the lungs (unitless). $F_{uptake,skin}$ is the uptake of compound through the skin (h^{-1}). $F_{biotransf}$ is the biotransformation factor in the body (unitless), see Table 4.

- Dermal contact and inhalation of chemicals in cosmetics, such as creams and lotions, via dermal application, and sprays (Brinch et al., 2017):

$$EDI_{dermal,cosmetics} = \left(\frac{Appl_{cosmetics} \cdot F_{uptake,skin,cosm}}{BW}\right) \cdot \left(C_{PFAS,cosm} \cdot C_{precurs,cosm} \cdot F_{biotransf}\right) \quad \textbf{(Eq. 12)}$$

$C_{PFAS,cosm}$ and $C_{precurs,cosm}$ are the concentration of the persistent PFAS and precursors, respectively, in the cosmetic product (ng/g). $Appl_{cosmetics}$ is the daily application rate of the cosmetic product (g/d). $F_{uptake,skin,cosm}$ is the uptake fraction of compound through the skin (unitless). $F_{biotransf}$ is the biotransformation factor in the body (unitless), see Table 4.

Dermal contact and inhalation of cosmetic sprays can be estimated from the equations for impregnation sprays (Eq. 10 and 11).

2.5.4. Biotransformation factors

The biotransformation factor $F_{biotransf}$ is used to estimate the amount of persistent PFAS, i.e. PFCAs and PFSAs, that is formed by the transformation of a precursor in the body. Table 4 shows values for $F_{biotransf}$ from the literature for selected PFCAs, PFSAs and precursors as % conversion of the precursor to the PFAS in mammals.

The lower and the higher values are used to represent the bioconcentration factors in the LE and HE scenario, respectively. The scenarios are further determined by the range of exposure concentrations, as described in section 2.4.

Table 4: Selected PFCAs and PFSAs and their precursors. Biotransformation factors ($F_{biotransf}$) in mammals from the literature are shown as % conversion of precursors to the respective PFAS. Where no values are given ("-"), the biotransformation process was not considered relevant.

	FTOH	diPAP	MeFOSE	EtFOSE	N-MeFOSA	N-EtFOSA	N-MeFBSA	N-EtFBSA	FBSA	FBSE
PFOA[a,d,e,f]	8:2 FTOH (1-10%) 10:2 FTOH (3-5%)	8:2 diPAP (1-5%)	-	-	-	-	-	-	-	-
PFOS[h,i,j,k]	-	-	MeFOSE (10-40%)	EtFOSE (15-40%)	N-MeFOSA (2-10%)	N-EtFOSA (5-15%)	-	-	-	-
PFHxA[a,b,c]	6:2 FTOH (10-20%)	6:2 diPAP (2-5%)	-	-	-	-	-	-	-	-
PFHpA[b,g]	7:3 FTOH (2-5%)	-	-	-	-	-	-	-	-	-
PFNA[d,e,f]	8:2 FTOH (2-5%) 10:2 FTOH (3-5%)	8:2 diPAP (1-3%)	-	-	-	-	-	-	-	-
PFDA[f,g]	10:2 FTOH (5-20%)	-	-	-	-	-	-	-	-	-
PFBS[i,j,k,l]	-	-	-	-	-	-	N-MeFBSA (10-20%)	N-EtFBSA (10-20%)	FBSA (<10%)	FBSE (10-15%)
PFPeA[b,c]	6:2 FTOH (2-5%)	6:2 diPAP (1-2%)	-	-	-	-	-	-	-	-
PFBA[a,b]	6:2 FTOH (1-3%)	6:2 diPAP (<2%)	-	-	-	-	-	-	-	-
PFHpS[j,k]	-	-	-	-	-	-	N-MeFBSA (5-20%)	N-EtFBSA (5-15%)		-
PFUnA[f,g]	10:2 FTOH (3-5%) 9:1 FTOH (2-5%)	-	-	-	-	-	-	-	-	-
PFDoA[f,g]	10:2 FTOH (5-10%) 9:1 FTOH (3-5%)	-	-	-	-	-	-	-	-	-
PFUnDA[g,i,j]	-	-	-	-	-	-	N-MeFBSA (5-20%)	N-EtFBSA (5-15%)	-	-
PFDoDA[i,j]	-	-	-	-	-	-	N-MeFBSA (5-20%)	N-EtFBSA (5-15%)	-	-

a Fasano et al. (2006); b Nilsson et al. (2013); c D'Eon et al. (2011); d Martin et al. (2009); e Lee et al. (2010); f Rosenmai et al. (2016); g Butt et al. (2014); h Tomy et al. (2004); i Lieder et al. (2009); j Olsen et al. (2007); k Benskin et al. (2009)

3. Results

3.1. Occurrence of PFAS in exposure media

The literature review showed that a large number of different PFAS had been detected in the various exposure media (Figure 5). In total, 57 PFAS had been reported in food, 55 PFAS in drinking water, 70 PFAS in food contact materials, 60 PFAS in indoor dust/air, 43 PFAS in outdoor dust/air, 189 PFAS in consumer products and 158 PFAS in occupational exposure settings, the latter not representing a systematic or exhaustive review. A full list with detailed information is included in Appendix A. Among all reported PFAS in different exposure matrices, we chose the five PFAS with most reports in the literature in each matrix for the quantitative analysis, with some additions as described in section 2.3.

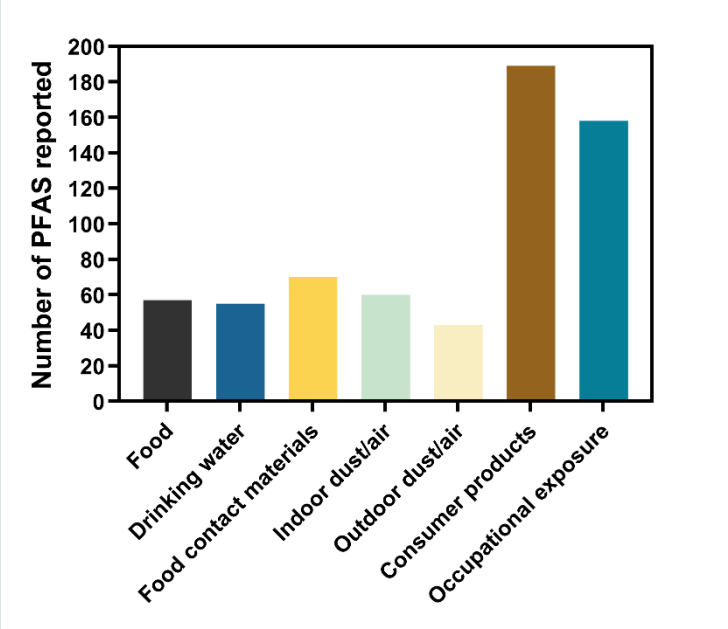

FIGURE 5. Total number of different PFAS in each exposure-relevant matrix extracted from the reviewed literature. Each PFAS is counted once in each matrix.

3.1.1. Food, drinking water and food contact materials

3.1.1.1. Food and drinking water

EFSA (2020) summarized PFAS levels in food and drinking water based on the literature from 2007 to 2018. The dataset in the EFSA report is mainly from European countries, primarily from Norway, Germany, and France, but also includes food products originating from North and South America, Africa, Asia and Australia that are imported into the European market. Our literature research built on the EFSA report and expanded the dataset on the occurrence of PFAS in food, drinking water, and other sources worldwide with additional literature from 2018 to 2024.

A total of 57 PFAS were reported in food samples in the reviewed literature, including the data from EFSA (2020). The top five PFAS, reported most frequently in food, were PFOS, PFOA, PFNA, perfluorodecanoic acid (PFDA), and perfluoroundecanoic acid (PFUnDA) (Table 5). These are all PFAS with carbon chains ≥ 8. As discussed in

section 2.3, their findings might reflect a selection bias as they are commonly analysed and expected to be present in the samples.

PFAS have been detected in a wide range of food categories, including both animal and plant-based products, as well as raw and processed foods. PFAS can enter food chains through various pathways, including environmental contamination from contaminated soil, water and air, and migration from food packaging materials. Animal-derived foods, such as meat, dairy, eggs and seafood, often show higher PFAS concentrations, likely due to bioaccumulation and biomagnification in aquatic and terrestrial food chains. However, plant-based foods are not exempt from contamination, as PFAS can be absorbed from contaminated water and soils. Processed and packaged foods may also contain PFAS introduced through contact with treated food packaging materials, as further discussed in section 3.1.1.2.

A targeted seafood survey conducted in the United States in 2022 highlighted the widespread presence of PFAS in seafood, which is a significant source of dietary exposure. According to the survey[1], 74% of tested seafood samples—including clams, cod, crab, pollock, salmon, shrimp, tilapia, and tuna—contained detectable levels of PFAS. This finding agrees with other studies, including the review by EFSA (2000), indicating that seafood often exhibits some of the highest PFAS concentrations among food groups due to bioaccumulation and biomagnification in aquatic ecosystems.

Table 5: Results of the literature search on the occurrence of PFAS in food, including the five most frequently reported compounds, their number of entries and country of the study. An extended table with concentrations and indications of relevant exposure pathways can be found in Appendix A.

Compound	Count	Food category	Country	Reference
PFOS	162	Eggs, seafoods, fish and fish products, fish roe, grains grain based products, vegetables, meat and meat products, milk and milk products, nuts, oilseeds, fruits, edible offal, pastes, pâtés and terrines, animal and vegetable fats and oils, sugar and confectionary, fruit and vegetable juices, alcoholic beverages, herbs, spices and condiments, food for infants and small children, composite food (including frozen products), snacks, desserts, and other foods	Belgium, USA, Sweden, China, Vietnam, Norway, Italy, Denmark, Greece, East Africa, Germany, Austria, Belgium, Cyprus, Czech Republic, Spain, Finland, UK, France, Ireland, Malta, Slovenia	Lasters et al. (2024), Young et al. (2022), Miranda et al. (2023), Augustsson et al. (2021), Cara et al. (2022), Chen et al. (2022), Feng et al. (2021), Diao et al. (2022), Hoa et al. (2022), Li et al. (2023), Munoz et al. (2022), De Silva et al. (2021), Zhang et al. (2023), Zhang et al. (2022), Mazzoni et al. (2019), Meng et al. (2019), Zheng et al. (2021), Fair et al. (2019), Sonne et al. (2019), Kedikoglou et al. (2019), Bao et al. (2019), Arinaitwe et al. (2020), Ruffle et al. (2020), Goodrow et al. (2020), Rüdel et al. (2022), EFSA (2020)

[1] https://www.fda.gov/food/process-contaminants-food/questions-and-answers-pfas-food

Compound	Count	Food category	Country	Reference
PFOA	150	Eggs, seafoods, fish and fish products, fish roe, grains grain based products, vegetables, meat and meat products, milk and milk products, nuts, oilseeds, fruits, edible offal, pastes, pâtés and terrines, animal and vegetable fats and oils, sugar and confectionary, fruit and vegetable juices, alcoholic beverages, herbs, spices and condiments, food for infants and small children, composite food (including frozen products), snacks, desserts, and other foods	Belgium, Italy, USA, China, Vietnam, Canada, Germany, Japan, Norway, Korea, Finland, Ireland, Denmark, Greece, Austria, Belgium, Cyprus, Czech Republic, Spain, UK, France, Malta, Slovenia	Lasters et al. (2024), Young et al. (2022), Miranda et al. (2023), Cara et al. (2022), Chen et al. (2022), Feng et al. (2021), Diao et al. (2022), Hoa et al. (2022), Li et al. (2023), Munoz et al. (2022), De Silva et al. (2021), Zhang et al. (2023), Zhang et al. (2022), Mazzoni et al. (2019), Meng et al. (2019), Zheng et al. (2021), Sonne et al. (2019), Kedikoglou et al. (2019), Bao et al. (2019), Ruffle et al. (2020), Goodrow et al. (2020), Rüdel et al. (2022), EFSA (2020), Stecconi et al. (2024)
PFNA	137	Eggs, seafoods, fish and fish products, fish roe, grains grain based products, vegetables, meat and meat products, milk and milk products, nuts, oilseeds, fruits, edible offal, pastes, pâtés and terrines, animal and vegetable fats and oils, sugar and confectionary, fruit and vegetable juices, alcoholic beverages, herbs, spices and condiments, food for infants and small children, composite food (including frozen products), snacks, desserts, and other foods	Belgium, Italy, USA, China, Vietnam, Canada, Korea, Finland, Norway, Germany, Austria, Belgium, Cyprus, Czech Republic, Denmark, Spain, UK, France, Ireland, Malta, Slovenia	Lasters et al. (2024), Young et al. (2022), Miranda et al. (2023), Cara et al. (2022), Chen et al. (2022), Feng et al. (2021), Diao et al. (2022), Hoa et al. (2022), Li et al. (2023), Munoz et al. (2022), De Silva et al. (2021), Zhang et al. (2023), Zhang et al. (2022), Mazzoni et al. (2019), Meng et al. (2019), Zheng et al. (2021), Fair et al. (2019), Bao et al. (2019), Ruffle et al. (2020), Goodrow et al. (2020), Rüdel et al. (2022), EFSA (2020), Stecconi et al. (2024), Kowalcyzyk et al. (2020)
PFDA	131	Eggs, seafoods, fish and fish products, fish roe, grains grain based products, vegetables, meat and meat products, milk and milk products, nuts, oilseeds, fruits, edible offal, pastes, pâtés and terrines, animal and vegetable fats and oils, sugar and confectionary, fruit and vegetable juices, alcoholic beverages, herbs, spices and condiments, food for infants and small children, composite food (including frozen products), snacks, desserts, and other foods	Belgium, Italy, USA, China, Vietnam, Canada, Norway, East Africa, Germany, Austria, Cyprus, Czech Republic, Denmark, Spain, Finland, UK, France, Ireland, Malta, Slovenia	Lasters et al. (2024), Young et al. (2022), Miranda et al. (2023), Cara et al. (2022), Chen et al. (2022), Feng et al. (2021), Diao et al. (2022), Hoa et al. (2022), Li et al. (2023), Munoz et al. (2022), De Silva et al. (2021), Zhang et al. (2022), Mazzoni et al. (2019), Meng et al. (2019), Zheng et al. (2021), Fair et al. (2019), Bao et al. (2019), Arinaitwe et al. (2020), Ruffle et al. (2020), Goodrow et al. (2020), Rüdel et al. (2022), EFSA (2020), Stecconi et al. (2024), Kowalcyzyk et al. (2020)

Compound	Count	Food category	Country	Reference
PFUnDA	130	Eggs, seafoods, fish and fish products, fish roe, grains grain based products, vegetables, meat and meat products, milk and milk products, nuts, oilseeds, fruits, edible offal, pastes, pâtés and terrines, animal and vegetable fats and oils, sugar and confectionary, fruit and vegetable juices, alcoholic beverages, herbs, spices and condiments, food for infants and small children, composite food (including frozen products), snacks, desserts, and other foods	Belgium, Italy, USA, China, Vietnam, Canada, Norway, Denmark, East Africa, Germany, Austria, Cyprus, Czech Republic, Germany, Spain, Finland, UK, France, Ireland, Malta, Slovenia	Lasters et al. (2024), Young et al. (2022), Miranda et al. (2023), Cara et al. (2022), Chen et al. (2022), Feng et al. (2021), Diao et al. (2022), Hoa et al. (2022), Li et al. (2023), Munoz et al. (2022), De Silva et al. (2021), Zhang et al. (2022), Meng et al. (2019), Zheng et al. (2021), Fair et al. (2019), Sonne et al. (2019), Arinaitwe et al. (2020), Ruffle et al. (2020), Goodrow et al. (2020), Rüdel et al. (2022), EFSA (2020), Stecconi et al. (2024), Kowalcyzyk et al. (2020)

Compared to the extensive research on PFAS in food, less literature is available on PFAS in drinking water. However, the studies still reported the presence of 55 different, PFAS in drinking water, including bottled water as well as natural water sources.

The five PFAS in drinking water reported most frequently our literature review were PFOA, PFNA, PFOS, PFBS, and perfluoroheptanoic acid (PFHpA) (Table 6). Unlike PFAS in food, which are often dominated by long-chain compounds (≥ C8), the PFAS most frequently detected in drinking water and related sources tend to have shorter carbon chains (≤ C9). This difference is likely attributed to the higher water solubility and mobility of shorter-chain PFAS, which makes them more likely to occur in aquatic environments and contaminate water supplies. However, selection biases have to be born in mind, as mentioned before, as study designs might use the same list of frequently measured PFAS, without much adaptation to specific matrices. These approaches might overlook PFAS that are specific to a given matrix.

PFAS can enter water sources through industrial waste, sewage treatment plants, and leaking landfills, as well as through diffuse pollution. Contamination of groundwater and surface water may lead to PFAS presence in drinking water supplies. Long-range transport through air can result in PFAS contamination of soils and surface water far from the original source.

Table 6: Results of the literature search on the occurrence of PFAS in drinking water and sources, including the five most frequently reported compounds, their number of entries and country of the study. An extended table with concentrations and indications of relevant exposure pathways can be found in Appendix A.

Compound	Count	Drinking waters and sources	Country	Reference
PFOA	21	Tap water, bottled water, surface water, ground water	Pakistan, USA, China, Czech Republic, Germany, Ireland, Canada, Burkina Faso, Chile, Ivory Coast, Japan, Mexico, Netherlands	Khan et al. (2024), Pelch et al. (2023), Jiao et al. (2022), Dvorakova et al. (2023), Xu et al. (2023), Brandsma et al. (2019), Babayev et al. (2022), Neuwald et al. (2022), Feng et al. (2021), Wang et al. (2023), Harrad et al. (2019), Meng et al. (2019), EFSA (2020), Chow et al. (2021), Wang et al. (2022), Kaboré et al. (2018)

PFNA	19	Tap water, bottled water, surface water, ground water	Pakistan, USA, China, Czech Republic, Germany, Ireland Canada, Burkina Faso, Chile, Ivory Coast, Japan, Mexico	Khan et al. (2024), Pelch et al. (2023), Jiao et al. (2022), Dvorakova et al. (2023), Xu et al. (2023), Brandsma et al. (2019), Babayev et al. (2022), Neuwald et al. (2022), Feng et al. (2021), Wang et al. (2023), Harrad et al. (2019), Meng et al. (2019), EFSA (2020), Chow et al. (2021), Wang et al. (2022), Kaboré et al. (2018), Meng et al. (2019)
PFOS	19	Tap water, bottled water, surface water, ground water	Pakistan, USA, China, Netherlands, Germany, Ireland, Canada, Burkina Faso, Chile, EU, Ivory Coast, Japan, Mexico	Khan et al. (2024), Pelch et al. (2023), Jiao et al. (2022), Dvorakova et al. (2023), Xu et al. (2023), Brandsma et al. (2019), Babayev et al. (2022), Neuwald et al. (2022), Feng et al. (2021), Wang et al. (2023), Harrad et al. (2019), Meng et al. (2019), EFSA (2020), Chow et al. (2021), Wang et al. (2022), Kaboré et al. (2018), Meng et al. (2019)
PFBS	18	Tap water, bottled water, surface water, ground water	Pakistan, USA, China, Czech Republic, Netherlands, Germany, Ireland, Canada, Burkina Faso, Chile, EU, Ivory Coast, Japan, Mexico	Khan et al. (2024), Pelch et al. (2023), Jiao et al. (2022), Dvorakova et al. (2023), Xu et al. (2023), Brandsma et al. (2019), Babayev et al. (2022), Neuwald et al. (2022), Wang et al. (2023), Harrad et al. (2019), Meng et al. (2019), EFSA (2020), Chow et al. (2021), Wang et al. (2022), Kaboré et al. (2018)
PFHpA	18	Tap water, bottled water, surface water, ground water	Pakistan, USA, China, Czech Republic, Netherlands, Germany, Ireland, Canada, Burkina Faso, Chile, Ivory Coast, Japan	Khan et al. (2024), Pelch et al. (2023), Jiao et al. (2022), Dvorakova et al. (2023), Xu et al. (2023), Brandsma et al. (2019), Babayev et al. (2022), Neuwald et al. (2022), Feng et al. (2021), Wang et al. (2023), Meng et al. (2019), EFSA (2020), Chow et al. (2021), Wang et al. (2022), Kaboré et al. (2018), Meng et al. (2019)

3.1.1.2. Food contact materials

PFAS are extensively used in food contact materials due to their unique chemical properties, including resistance to oil, grease, water, and heat (Schaider et al., 2017). These characteristics make PFAS highly effective in a variety of packaging applications, particularly in the food industry. Their widespread use and potential for environmental and health impacts have made them a focal point of scientific and regulatory attention. In Denmark, PFAS have been phased out from use in food contact materials.

A major use of PFAS in food contact materials is paper and board substrates for fast food wrapping, but PFAS can also occur in greaseproof paper, baking paper, heat resistant packaging and coatings of cans. Currently there are three main types of PFAS used in packaging (ECHA, 2023):

- Short-chain fluorotelomer sidechain (C6) polymeric PFASs, with high molecular weight acrylic polymers that contain fluorotelomer functionality to provide repellent performance
- Perfluoropolyether (PFPE) based products
- Fluoroplastics: Fluorinated ethylene propylene (FEP), perfluoroalkoxy ethanes (PFA) and fluorocarbon-based fluoroelastomer materials

A significant concern with PFAS in food contact materials is their potential to leach into food. This migration process is influenced by several factors, such as type of food, contact time, temperature, and material composition. When PFAS migrate into food or beverages, human exposure occurs primarily through ingestion. Otherwise, the dermal contact when handling the material could also be a potential source of exposure, although it was not indicated in the reviewed literature.

In total, 70 PFAS were reported in food contact materials in our literature review, indicating a larger diversity than that of PFAS reported in food and drinking water. As shown in Table 7, the five PFAS reported most frequently in food contact materials are all PFCAs, i.e. PFOA, perfluorohexanoic acid (PFHxA), PFHpA, PFDA, and PFNA with 46, 44, 37, 37 and 36 reports, respectively. In addition, the perfluoroalkyl phosphate diesters (diPAPs) 6:2 diPAP and 8:2 diPAP were the most frequently reported non-PFCAs with 22 and 21 reports, respectively. As shown in Table 4, 6:2 diPAP and 8:2 diPAP are precursors of PFOA, PFNA, and PFHxA. As these compounds are also among the top five PFAS present in food contact materials, exposure to PFOA, PFNA and PFHxA can happen directly from contact with or uptake of these PFCAs as well as indirectly via biotransformation of the PFAS precursors.

Table 7: Results of the literature search on the occurrence of PFAS in food contact materials including the five most frequently reported compounds, their number of entries and country of study. An extended table with concentrations and indications of relevant exposure pathways can be found in Appendix A.

Compound	Count	Food contact materials	Country	Reference
PFOA	46	Popcorn bags, non-stick baking paper, French fries box, sandwich wrapper, hamburger box, paper straw, paper and board (take away hot use), paper analogues (hot use), pizzas and sandwiches, non-stick baking cups, paper bowl, paper tableware, cupcake cup, paper cup, food packaging material from fast-food restaurants, PTFE coated pans, beverage and ice cream cups, aluminum foil bags and wrappers, breakfast bags, roasting bags, paper bags for pet food packaging, grease-proof paper, muffin cup, ice cream tub, red plate, plastic (milk bottle, muffin cup, precooked food wrapper, cup of coffee), instant food cups, dessert container	USA, Vietnam, Australia, Belgium, Brazil, Egypt, China Czech Republic, Denmark, France, Germany, Greece, Hungary, India, Italy, Mexico, Netherland, Poland, Portugal, Spain, Sweden, Thailand, UK	Liu et al. (2014), Hoang et al. (2023), Whitehead et al. (2023), Di Mario et al. (2024), Zabaleta et al. (2017), Shoeib et al. (2016), Dueñas-Mas et al. (2023), Zefeiraki et al. (2014), Yuan et al. (2016), Vázquez Loureiro et al. (2024), Kotthoff et al. (2015), Surma et al. (2015), Elizalde et al. (2018), Gebbink et al. (2013), Zabaleta et al. (2020), Martinez-Moral et al. (2012), Xu et al. (2013), Robel et al. (2017), Chinthakindi et al. (2021), Moreta et al. (2013), Zabaleta et al. (2016), Sapozhnikova et al. (2023), Schaider et al. (2017), Schlummer et al. (2015), Ritter et al. (2017), Dolman et al. (2011), Phelps et al. (2024), Poothong et al. (2012), Brenes et al. (2019)

Compound	Count	Food contact materials	Country	Reference
PFHxA	44	Paper straws, paper and board (take away cold use), paper and board (take away hot use), paper analogues (cold use), paper analogues (hot use), popcorn bags, sandwich wrapping papers, cardboard box for French fries, pizzas and sandwiches, non-stick baking cups, paper bowl, paper tableware, cupcake cup, food packaging material from fast-food restaurants, PTFE coated pans, beverage and ice cream cups, aluminum foil bags and wrappers, breakfast bags, roasting bags, paper bags for pet food packaging, muffin cup, plate and cup cardboard, burger wrapper, baking paper, Plastic (milk bottle, muffin cup, pre-cooked food wrapper, cup of coffee), Mochi paper tray	USA, Vietnam, Belgium Brazil, Egypt, China, Czech Republic, Denmark, France, Germany, Greece, Hungary, Italy Mexico, Netherland, Poland, Portugal, Spain, Sweden, UK	Liu et al. (2014), Hoang et al. (2023), Whitehead et al. (2023), Di Mario et al. (2024), Zabaleta et al. (2017), Shoeib et al. (2016), Dueñas-Mas et al. (2023), Zefeiraki et al. (2014), Yuan et al. (2016), Vázquez Loureiro et al. (2024), Kotthoff et al. (2015), Surma et al. (2015), Elizalde et al. (2018), Gebbink et al. (2013), Zabaleta et al. (2020), Martinez-Moral et al. (2012), Xu et al. (2013), Robel et al. (2017), Chinthakindi et al. (2021), Moreta et al. (2013), Zabaleta et al. (2016), Sapozhnikova et al. (2023), Schaider et al. (2017), Schlummer et al. (2015), Ritter et al. (2017)
PFHpA	37	Paper straws, paper and board (take away cold use), paper and board (take away hot use), paper analogues (cold use), paper analogues (hot use), popcorn bags, sandwich wrapping papers, cardboard box for French fries, pizzas and sandwiches, non-stick baking cups, paper bowl, paper tableware, cupcake cup, food packaging material from fast-food restaurants, PTFE coated pans, beverage and ice cream cups, aluminum foil bags and wrappers, breakfast bags, roasting bags, paper bags for pet food packaging, muffin cup, plate and cup cardboard, burger wrapper, baking paper, plastic (milk bottle, muffin cup, pre-cooked food wrapper, cup of coffee), Mochi paper tray, ice cream tub	USA, Vietnam, Belgium, Brazil, Egypt, China, Denmark, France, Germany, Greece, Italy, Netherland, Poland, Portugal, Spain, Sweden, UK	Liu et al. (2014), Hoang et al. (2023), Whitehead et al. (2023), Di Mario et al. (2024), Zabaleta et al. (2017), Shoeib et al. (2016), Dueñas-Mas et al. (2023), Zefeiraki et al. (2014), Yuan et al. (2016), Vázquez Loureiro et al. (2024), Kotthoff et al. (2015), Surma et al. (2015), Elizalde et al. (2018), Gebbink et al. (2013), Zabaleta et al. (2020), Martinez-Moral et al. (2012), Xu et al. (2013), Robel et al. (2017), Chinthakindi et al. (2021), Moreta et al. (2013), Zabaleta et al. (2016), Sapozhnikova et al. (2023), Schaider et al. (2017), Schlummer et al. (2015)

Compound	Count	Food contact materials	Country	Reference
PFDA	37	Paper straws, paper analogues (hot use), popcorn bags, sandwich wrapping papers, cardboard box for French fries, pizzas and sandwiches, non-stick baking cups, soup cups, paper bowl, paper tableware, cupcake cup, paper box, paper bag, baking and sandwich papers, PTFE coated pans, beverage and ice cream cups, fast food wrappers, aluminum foil bags and wrappers, breakfast bags, roasting bags, paper bags for pet food packaging, muffin cup, plate and cup cardboard, burger wrapper, Ice cream tub, plastic (milk bottle, muffin cup, pre-cooked food wrapper, cup of coffee), roasting bags, Mochi paper tray, coconut water box, potato snack box, instant noodle cup, cookies wrapper	USA, Vietnam, Belgium, Brazil, Egypt, China, Denmark, France, Germany, Greece, Hungary, India, Italy, Mexico, Poland, Portugal, Spain Sweden, UK	Liu et al. (2014), Hoang et al. (2023), Whitehead et al. (2023), Di Mario et al. (2024), Zabaleta et al. (2017), Shoeib et al. (2016), Dueñas-Mas et al. (2023), Zefeiraki et al. (2014), Yuan et al. (2016), Vázquez Loureiro et al. (2024), Kotthoff et al. (2015), Surma et al. (2015), Elizalde et al. (2018), Gebbink et al. (2013), Zabaleta et al. (2020), Martinez-Moral et al. (2012), Xu et al. (2013), Robel et al. (2017), Chinthakindi et al. (2021), Moreta et al. (2013), Zabaleta et al. (2016), Sapozhnikova et al. (2023), Schaider et al. (2017), Schlummer et al. (2015),
PFNA	36	Paper straws, paper analogues (hot use), popcorn bags, sandwich wrapping papers, cardboard box for French fries, pizzas and sandwiches, non-stick baking cups, soup cups, paper bowl, paper tableware, cupcake cup, paper box, paper bag, baking and sandwich papers, PTFE coated pans, beverage and ice cream cups, fast food wrappers, aluminum foil bags and wrappers, breakfast bags, roasting bags, paper bags for pet food packaging, muffin cup, burger wrapper, red plate for birthday, burger box, burger grease proof paper, fluorinated containers, Mochi paper tray, coconut water box, soymilk box, peanut box, Instant noodle cup, cookies wrapper	USA, Vietnam, Belgium, Brazil, Egypt, China, Denmark, France, Germany, Greece, Hungary, Ireland, Italy, Mexico, Netherland, Poland, Portugal, Spain, Sweden, UK	Liu et al. (2014), Hoang et al. (2023), Whitehead et al. (2023), Di Mario et al. (2024), Zabaleta et al. (2017), Shoeib et al. (2016), Dueñas-Mas et al. (2023), Zefeiraki et al. (2014), Yuan et al. (2016), Vázquez Loureiro et al. (2024), Kotthoff et al. (2015), Surma et al. (2015), Elizalde et al. (2018), Gebbink et al. (2013), Zabaleta et al. (2020), Martinez-Moral et al. (2012), Xu et al. (2013), Robel et al. (2017), Chinthakindi et al. (2021), Schlummer et al. (2015),
6:2 diPAP	22	Pizza grease proof paper, board cup, fries box, burger box, burger grease proof paper, popcorn bags, food packaging material from fast-food restaurants, baking paper, muffin cup, plate and cup cardboard, burger wrapper, grease proof boxes, French fries baking paper, muffin cup, plate and cup cardboard, burger wrapper, grease proof boxes, French fries wrappers, burger clams, grease proof bag, cardboards, pizza boxes, pet food bag, plastic (milk bottle, muffin cup, pre-cooked food wrapper, cup of coffee)	USA, Belgium, Brazil, China, Czech Republic, France, Germany, Hungary, Ireland, Italy, Mexico, Netherland, Portugal, Spain, Sweden, UK	Gebbink et al. (2013), Zabaleta et al. (2017), Dueñas-Mas et al. (2023), Zabaleta et al. (2020), Robel et al. (2017), Zabaleta et al. (2016), Sapozhnikova et al. (2023)

Compound	Count	Food contact materials	Country	Reference
8:2 diPAP	21	Popcorn bag, pizza grease proof paper, board cup, fries box, burger box, burger grease proof paper, food packaging material from fast-food restaurants, baking paper, muffin cup, plate and cup cardboard, burger wrapper, French fries baking paper, burger clams, pizza boxes, pet food bag, Plastic (milk bottle, muffin cup, pre-cooked food wrapper, cup of coffee)	USA, Brazil, China, Czech Republic, France, Germany, Hungary, Ireland, Italy, Mexico, Netherland, Portugal, Spain, Sweden, UK	Gebbink et al. (2013), Zabaleta et al. (2017), Dueñas-Mas et al. (2023), Zabaleta et al. (2020), Robel et al. (2017)

3.1.2. Air and dust

3.1.2.1. Indoor air and dust

Exposure to chemicals in indoor dust mainly happens through ingestion (which is particularly important for toddlers), while inhalation is the main route of exposure to PFAS in indoor air, which might contain PFAS in gaseous form and particle-bound PFAS, including suspended dust. Investigation of indoor exposure to PFAS should consider the variety of physical–chemical properties for PFAS and the complexity of precursors and polymers.

Semivolatile organic compounds will tend to partition between the vapor phase, suspended particulate, dust, and indoor surfaces (including skin and clothing), depending in part on their octanol–air partition coefficients. Some PFAS, such as FTOHs and perfluorooctane sulfonamidoethanol (EtFOSE), are relatively volatile and are found in the vapor phase indoors (Table 8), whereas both neutral and ionic PFAS are more likely to be present in dust (Table 9). The concentration of neutral PFAS that are present in the gas phase can be up to 1000 times higher than the concentration of persistent PFAAs, which are mainly bound to particles. Once inhaled, the precursor will be metabolized to e.g. PFOA and PFNA. 6:2 FTOH and 8:2 FTOH are frequently found in the gas phase.

Concentrations of PFAS in indoor environments are usually measured through filtration or adsorption to a solid phase (filters or sorbents) using either active air pumping or passive samples. In indoor air, concentrations can be an order of magnitude higher (ng/m^3 levels) than outdoor environments.

In recent years, ultrashort- and short-chain PFAAs have received increasing attention. They have been detected in human matrices, including levels of trifluoroacetic acid (TFA) in a study from China that were comparable to those of several long-chain PFAAs in human blood. A recent study from China also found ultrashort- and short-chain PFAAs in indoor and outdoor dust, with TFA as the most abundant compound (Wang, 2022).

Table 8: Results of the literature search on the occurrence of PFAS in indoor air (gas phase), resulting in two main compounds.

Compound name	Count	Indoor air	Country	Reference
6:2 FTOH	9	Residential homes, children's bedrooms, childcare centers,	Canada, China, Germany, Japan, Sweden, Finland, Norway	Shoeib et al. (2011), Yao et al. (2018), Fromme et al. (2015), Liu et al. (2013), Winkens et al. (2019), Padilla-Sanchez et al. (2017),
8:2 FTOH	9	Residential homes, children's bedrooms, childcare centers,	Canada, China, Germany, Japan, Sweden, Finland, Norway	Shoeib et al. (2011), Yao et al. (2018), Fromme et al. (2015), Liu et al. (2013), Winkens et al. (2019), Padilla-Sanchez et al. (2017),

Zheng et al. (2023) applied a toxicokinetic model to 47 PFAAs and their precursors in paired samples of dust and drinking water collected from residential homes in the USA, and in blood and urine samples collected from the residents of these homes. The results of the model showed that consumption of drinking water and dust intake

contributed only ~20% to the total PFAA levels in blood, suggesting other exposure pathways for these compounds. However, high levels of several PFAA-precursors in dust and a strong relationship found between the dust levels of some precursors, such as polyfluoroalkyl phosphate esters (PAPs), with those of TFA and perfluoropropanoic acid (PFPrA) in blood indicate common sources and suggest that biotransformation of PAPs could be a potential indirect source of the ultrashort-chain PFAAs in humans.

A worldwide study published by Eriksson et al. (2015) found high concentrations of mono-, di- and tri-PAPs in dust samples revealing a ubiquitous spread in private households from diverse geographic areas, with significant differences between countries. Mono-, di- and tri-PAPs are precursors of some persistent PFAS (Table 4).

Table 9: Results of the literature search on the occurrence of PFAS in indoor dust, including the most frequently reported compounds and relevant precursors.

Compound	Count	Indoor dust	Country	Reference
PFNA	20	Residential homes, Fire stations, clothing shops, children's bedrooms, childcare centers, hotels, shops, cinemas	USA, India, China, Australia, Canada, Sweden, Japan, Finland, Greece, Nepal, Spain, Faroe Islands, Ireland	Zheng et al. (2023), Eriksson et al. (2015), Feng et al. (2021), Gustafsson et al. (2022), Hall et al. (2020), Liu et al. (2011), Wu et al. (2019), Winkens et al. (2019), Zheng et al. (2020), Yao et al. (2019), Wang et al. (2021), Shin et al. (2020), Su et al. (2016), Yamazaki et al. (2023), Ao et al. (2019), Harrad et al. (2019)
PFOA	21	Residential homes, Fire stations, clothing shops, children's bedrooms, childcare centers, hotels, shops, cinemas	USA, India, China, Australia, Canada, Sweden, Japan, Finland, Greece, Nepal, Spain, Faroe Islands, Ireland	Zheng et al. (2023), Eriksson et al. (2015), Feng et al. (2021), Gustafsson et al. (2022), Hall et al. (2020), Liu et al. (2011), Wu et al. (2019), Winkens et al. (2019), Zheng et al. (2020), Yao et al. (2019), Wang et al. (2021), Shin et al. (2020), Su et al. (2016), Yamazaki et al. (2023), Ao et al. (2019), Harrad et al. (2019)
PFHpA	21	Residential homes, Fire stations, clothing shops, children's bedrooms, childcare centers, hotels, shops, cinemas	USA, India, China, Australia, Canada, Sweden, Japan, Finland, Greece, Nepal, Spain, Faroe Islands, Ireland	Zheng et al. (2023), Eriksson et al. (2015), Feng et al. (2021), Gustafsson et al. (2022), Hall et al. (2020), Liu et al. (2011), Wu et al. (2019), Winkens et al. (2019), Zheng et al. (2020), Yao et al. (2019), Wang et al. (2021), Shin et al. (2020), Su et al. (2016), Yamazaki et al. (2023), Ao et al. (2019), Harrad et al. (2019)
PFOS	19	Residential homes, Fire stations, clothing shops, children's bedrooms, childcare centers, hotels, shops, cinemas	USA, India, China, Australia, Canada, Sweden, Japan, Finland, Greece, Nepal, Spain, Faroe Islands, Ireland	Zheng et al. (2023), Eriksson et al. (2015), Feng et al. (2021), Gustafsson et al. (2022), Hall et al. (2020), Liu et al. (2011), Wu et al. (2019), Winkens et al. (2019), Zheng et al. (2020), Yao et al. (2019), Wang et al. (2021), Shin et al. (2020), Su et al. (2016), Yamazaki et al. (2023), Ao et al. (2019), Harrad et al. (2019)

Compound	Count	Indoor dust	Country	Reference
PFDA	18	Residential homes, Fire stations, clothing shops, children's bedrooms, childcare centers, hotels, shops, cinemas	USA, India, China, Australia, Canada, Sweden, Japan, Finland, Greece, Nepal, Spain, Faroe Islands, Ireland	Zheng et al. (2023), Eriksson et al. (2015), Feng et al. (2021), Gustafsson et al. (2022), Hall et al. (2020), Liu et al. (2011), Wu et al. (2019), Winkens et al. (2019), Zheng et al. (2020), Yao et al. (2019), Wang et al. (2021), Shin et al. (2020), Su et al. (2016), Yamazaki et al. (2023), Ao et al. (2019), Harrad et al. (2019)
PFHxA	20	Residential homes, Fire stations, clothing shops, children's bedrooms, childcare centers, hotels, shops, cinemas	USA, India, China, Australia, Canada, Sweden, Japan, Finland, Greece, Nepal, Spain, Faroe Islands, Ireland	Zheng et al. (2023), Eriksson et al. (2015), Feng et al. (2021), Gustafsson et al. (2022), Hall et al. (2020), Liu et al. (2011), Wu et al. (2019), Winkens et al. (2019), Zheng et al. (2020), Yao et al. (2019), Wang et al. (2021), Shin et al. (2020), Su et al. (2016), Yamazaki et al. (2023), Ao et al. (2019), Harrad et al. (2019)
6:2 FTOH	5	Residential homes, Fire stations, clothing shops, children's bedrooms, childcare centers, hotels, shops, cinemas	USA, India, China, Australia, Canada, Sweden, Japan, Finland, Greece, Nepal, Spain, Faroe Islands, Ireland	Zheng et al. (2023), Eriksson et al. (2015), Feng et al. (2021), Gustafsson et al. (2022), Hall et al. (2020), Liu et al. (2011), Wu et al. (2019), Winkens et al. (2019), Zheng et al. (2020), Yao et al. (2019), Wang et al. (2021), Shin et al. (2020), Su et al. (2016), Yamazaki et al. (2023), Ao et al. (2019), Harrad et al. (2019)
8:2 FTOH	7	Residential homes, Fire stations, clothing shops, children's bedrooms, childcare centers, hotels, shops, cinemas	USA, India, China, Australia, Canada, Sweden, Japan, Finland, Greece, Nepal, Spain, Faroe Islands, Ireland	Zheng et al. (2023), Eriksson et al. (2015), Feng et al. (2021), Gustafsson et al. (2022), Hall et al. (2020), Liu et al. (2011), Wu et al. (2019), Winkens et al. (2019), Zheng et al. (2020), Yao et al. (2019), Wang et al. (2021), Shin et al. (2020), Su et al. (2016), Yamazaki et al. (2023), Ao et al. (2019), Harrad et al. (2019)
6:2 diPAP	**5**	Residential homes, Fire stations, clothing shops, children's bedrooms, childcare centers, hotels, shops, cinemas	USA, India, China, Australia, Canada, Sweden, Japan, Finland, Greece, Nepal, Spain, Faroe Islands, Ireland	Zheng et al. (2023), Eriksson et al. (2015), Feng et al. (2021), Gustafsson et al. (2022), Hall et al. (2020), Liu et al. (2011), Wu et al. (2019), Winkens et al. (2019), Zheng et al. (2020), Yao et al. (2019), Wang et al. (2021), Shin et al. (2020), Su et al. (2016), Yamazaki et al. (2023), Ao et al. (2019), Harrad et al. (2019)

Compound	Count	Indoor dust	Country	Reference
8:2 diPAP	**5**	Residential homes, Fire stations, clothing shops, children's bedrooms, childcare centers, hotels, shops, cinemas	USA, India, China, Australia, Canada, Sweden, Japan, Finland, Greece, Nepal, Spain, Faroe Islands, Ireland	Zheng et al. (2023), Eriksson et al. (2015), Feng et al. (2021), Gustafsson et al. (2022), Hall et al. (2020), Liu et al. (2011), Wu et al. (2019), Winkens et al. (2019), Zheng et al. (2020), Yao et al. (2019), Wang et al. (2021), Shin et al. (2020), Su et al. (2016), Yamazaki et al. (2023), Ao et al. (2019), Harrad et al. (2019)

3.1.2.2. Outdoor air and dust

While volatile neutral PFAS are common in the gas phase, most airborne PFAAs are more likely to adsorb to particulate matter as the main carrier. The concentration distribution of PFASs in the atmosphere is usually associated with the level of anthropogenic activities, such as the industrialization and the population density. Liu et al (2019) investigated human inhalation exposure to PFAS in outdoor PM10. Compared with ingestion via daily diet, the inhalation of PM10 exhibited an insignificant contribution to the estimated average daily intakes of PFAS by different age groups.

Table 10: Results of the literature search on the occurrence of PFAS in outdoor dust, including the most frequently reported compounds and relevant precursors.

Compound	Count	Outdoor dust	Country	Reference
PFNA	5	Road dust in the vicinity of a fluorochemical plant	India, China	Yao et al. (2016), Yamazaki et al. (2023), Wang et al. (2021)
PFOA	4	Road dust in the vicinity of a fluorochemical plant	India, China	Yao et al. (2016), Yamazaki et al. (2023), Wang et al. (2021)
PFHpA	3	Road dust in the vicinity of a fluorochemical plant	India, China	Yao et al. (2016), Yamazaki et al. (2023), Wang et al. (2021)
PFOS	5	Road dust in the vicinity of a fluorochemical plant	India, China	Yao et al. (2016), Yamazaki et al. (2023), Wang et al. (2021)
PFDA	5	Road dust in the vicinity of a fluorochemical plant	India, China	Yao et al. (2016), Yamazaki et al. (2023), Wang et al. (2021)
PFHxA	3	Road dust in the vicinity of a fluorochemical plant	India, China	Yao et al. (2016), Yamazaki et al. (2023), Wang et al. (2021)
6:2 FTOH	2	Road dust in the vicinity of a fluorochemical plant	India, China	Yao et al. (2016), Yamazaki et al. (2023), Wang et al. (2021)
8:2 FTOH	2	Road dust in the vicinity of a fluorochemical plant	India, China	Yao et al. (2016), Yamazaki et al. (2023), Wang et al. (2021)
6:2 diPAP	2	Road dust in the vicinity of a fluorochemical plant	India, China	Yao et al. (2016), Yamazaki et al. (2023), Wang et al. (2021)
8:2 diPAP	2	Road dust in the vicinity of a fluorochemical plant	India, China	Yao et al. (2016), Yamazaki et al. (2023), Wang et al. (2021)

Most of the literature on outdoor measurements of PFAS has focused on general levels in a certain area and diurnal and/or seasonal trends. Two papers have been published that focus on outdoor concentrations in relation to human exposure. In the study from India, road dust has been analysed (Yamazaki et al., 2023), and in the study from China both road dust and air were analysed (Yao et al., 2016) (Table 10). The study from Wang et al. (2021) investigated atmospheric diffusion of PFAAs emitted from fluorochemical industry and its associated health risks. Apart from studies relating to specific sources, the outdoor concentrations of particle-bound and gaseous PFAS are generally very low (pg/m3) and thus less relevant for human exposure compared to the indoor concentrations.

3.1.3. Consumer products

Using the application terminology by ECHA (2023), consumer products are

- Textiles, upholstery, leather, apparel and carpets

- Cosmetics
- Electronics
- Consumer mixtures

The latter includes cleaners for glass, metal, ceramic, carpet and upholstery; waxes and polishes for e.g. furniture, floors and cars; floor polish removers; drycleaning products; dishwashing products as rinse aid; windscreen treatments for automobiles and windscreen wiper fluids; car care products; rain-repellent fluids in the aviation industry; anti-fog agents; Teflon spray for lubrication of doors, locks, bike chains, motorcycles etc. (ECHA, 2023).

Table 11 shows an extract from the literature where the most frequently detected PFAS in consumer products are given together with the number of entries and countries where the studies were conducted. The top five compounds reported in the literature were PFOA, perfluorobutanoic acid (PFBA), PFHxA, PFHpA and PFDA. Table 4 indicates that 6:2 FTOH, 8:2 FTOH,10:2 FTOH, 7:3 FTOH, 6:2 diPAP and 8:2 diPAP are relevant precursors to these five PFAS. Of these it is only 6:2 FTOH, 8:2 FTOH and 10:2 FTOH that have more than two entries and are included in the quantitative analysis of EDI calculations in section 3.2. As stated earlier, it has to be kept in mind that the frequency of detection will be affected by a selection bias of those PFAS that are expected to be in the samples or that are just commonly measured. Due to this selection bias, other PFAS can be present that were not included in the analytical programme of the respective study.

Table 11: Results of the literature search on the occurrence of PFAS in consumer products including the five most frequently detected compounds and three related precursors, together with number of entries and countries where the studies were conducted. An extended table with concentrations and indications of relevant exposure pathways can be found in Appendix A.

Compound	Counts	Consumer products	Country	Reference
PFOA	188	Bicycle helmets/straps, childrens clothing, jacket, children's jackets, children's rain trousers, jackets, poncho for children, trousers and winterclothing for children, Teflon cloth, body lotion, concealer, creme/lotion, foundation, highlighter, eyeshadow, sunscreen, carpets for children, solar cells and computers and mobile phones and digital home appliance, pre-treated carpeting, commercial carpet/fabric-care liquids, household carpet/fabric-care liquids and foams, treated apparel, treated home textile and upholstery, treated nonwoven medical garments, treated floor waxes and stone/wood sealants, membranes for apparel, thread-sealant tapes and pastes, sanitary pads, panty liners, menstrual cups, paper diapers, sprays for fabrics and textile, rust inhibitors, hygiene paper	Denmark, China, Indonesia, Vietnam, Ukraine, Norway, Japan, USA, Nordic	Poulsen et al., (2018), Lassen et al., (2015), Greenpeace (2012 & 2013), SFT (2006), Norges Naturvernforbund (2006), Brinch et al. (2017), Fujii et al. (2013), Herzke et al. (2012), Liu et al. (2014), Klinke et al. (2016), Wang et al. (2019), Zhou et al. (2023), Ye et al. (2015), Chen et al. (2023)

Compound	Counts	Consumer products	Country	Reference
PFBA	122	Jackets, children's jackets, children's rain trousers, poncho for children, trousers and winter clothing for children, Teflon cloth, facescrub, BB cream, body lotion, CC cream, concealer, creme/lotion, foundation, highlighter, powder, eyeshadow, carpets for children, commercial carpet/fabric-care liquids, household carpet/fabric-care liquids and foams, treated apparel, treated home textile and upholstery, treated non-woven medical garments, treated floor waxes and stone/wood sealants, membranes for apparel, thread-sealant tapes and pastes, bacterial liquids, stationery paper	Denmark, China, Indonesia, Vietnam, Ukraine, Norway, USA	Greenpeace (2012 & 2013), SFT (2006), Norges Naturvernforbund (2006), Brinch et al. (2017), Liu et al. (2014), Klinke et al. (2016), Zhou et al. (2023), Chen et al. (2023)
PFHxA	120	Bicycle helmets/straps, children's clothing, , children's jackets, jackets, trousers and winterclothing for children, Teflon cloth, facescrub, BB cream, body lotion, CC cream, concealer, creme/lotion, foundation, highlighter, powder, eyeshadow, sunscreen, carpets for children, pre-treated carpeting, commercial carpet/fabric-care liquids, household carpet/fabric-care liquids and foams, treated apparel, treated home textile and upholstery, treated non-woven medical garments, treated floor waxes and stone/wood sealants, membranes for apparel, thread-sealant tapes and pastes, hygiene paper	Denmark, China, Indonesia, Norway, Japan, USA	Poulsen et al. (2018), Lassen et al. (2015), Greenpeace (2012 & 2013), SFT (2006), Norges Naturvernforbund (2006), Brinch et al. (2017), Fujii et al. (2013), Herzke et al. (2012), Liu et al. (2014), Klinke et al. (2016), Chen et al. (2023)
PFHpA	115	Children's clothing, children's jackets, children's rain trousers, jackets, trousers and winterclothing for children, Teflon cloth, facescrub, body lotion, CC cream, concealer, creme/lotion, foundation, highlighter, powder, eyeshadow, sunscreen, carpets for children, pre-treated carpeting, commercial carpet/fabric-care liquids, household carpet/fabric-care liquids and foams, treated apparel, treated home textile and upholstery, treated non-woven medical garments, treated floor waxes and stone/wood sealants, membranes for apparel, thread-sealant tapes and pastes	Denmark, China, Indonesia, Norway, Japan, USA	Lassen et al. (2015), Greenpeace (2012 & 2013), SFT (2006), Norges Naturvernforbund (2006), Brinch et al., (2017), Fujii et al. (2013), Herzke et al. (2012), Liu et al. (2014), Klinke et al. (2016)

Compound	Counts	Consumer products	Country	Reference
PFDA	105	Childrens clothing, childrens jackets, childrens rain trousers, jackets, poncho for children, trousers and winterclothing for children, Teflon cloth, body lotion, concealer, creme/lotion, foundation, highlighter, eyeshadow, sunscreen, carpets for children, pretreated carpeting, commercial carpet/fabric-care liquids, household carpet/fabric-care liquids and foams, treated apparel, treated home textile and upholstery, treated non-woven medical garments, treated floor waxes and stone/wood sealants, membranes for apparel, thread-sealant tapes and pastes, panty liners, sprays for fabrics and textile, rust inhibitors	Denmark, Indonesia, China, Vietnam, Norway, Japan, USA	Lassen et al. (2015), Greenpeace (2012 & 2013), SFT (2006), Norges Naturvernforbund (2006), Brinch et al. (2017), Fujii et al. (2013), Liu et al. (2014), Zhou et al., (2023), Ye et al. (2015)
10:2 FTOH	50	Childrens clothing, Childrens jackets, jackets, jackets, trousers and winter clothing for children, Teflon cloth, carpets for children, other functions	Denmark, China, Norway	Lassen et al. (2015), Greenpeace (2012), SFT (2006), Norges Naturvernforbund (2006), Herzke et al. (2012), Nicolajsen and Tsitonaki (2016)
8:2 FTOH	51	Childrens clothing/jackets, jackets, trousers and winter clothing for children, carpets for children, Teflon cloth	Denmark, China, Norway	Lassen et a. (2015), Greenpeace (2012), SFT (2006), Norges Naturvernforbund (2006), Herzke et al. (2012), Nicolajsen and Tsitonaki (2016)
6:2 FTOH	49	Childrens clothing, jackets, poncho for children, carpets for children, masks of fabric	Denmark, China, Norway	Lassen et a. (2015), Greenpeace (2012), Norges Naturvernforbund (2006), Herzke et al. (2012), Poulsen et al. (2021)

3.1.3.1 Textiles, upholstery, leather, apparel and carpets

PFAS have commonly been used in multiple textiles, upholstery, leather, apparel and carpet products, as well as in mixtures for re-impregnation of different products (Table 12). The key function that PFAS provide in these applications are water and oil repellence, stain resistance and thermal stability.

Table 12: Overview of different textiles, upholstery, leather, apparel and carpets categories (ECHA, 2023).

Major use category	Subcategory uses with examples	Technical function of PFASs indicated by stakeholders
Home textiles	Carpets and rugs	Water repellence, oil repellence
	Curtains and blinds	Water repellence, oil repellence
	Textile based coverings (e.g. fabrics for soft-furnishings, tablecloths, bedding)	Water repellence, oil repellence
Consumer apparel and accessories	Indoor and outdoor wear	Water repellence
	Sportswear	Water repellence, oil repellence
	Footwear	Water repellence, oil repellence
	Accessories (e.g. umbrellas, bags, wallets)	Water repellence
Professional apparel	Professional sportswear and footwear	Water repellence, oil repellence
	PPE for industrial and professional use (other than sportswear)	Water repellence, oil repellence, stain-resistance, soil protection
Technical textiles*	Outdoor technical textiles (e.g. canvas, awnings, tarps, tents, sails, rope)	Water repellence, oil repellence, stain-resistance, soil protection
	Medical applications (e.g. surgical drapes, gowns, curtains)	Water repellence, oil repellence, stain-resistance

Major use category	Subcategory uses with examples	Technical function of PFASs indicated by stakeholders
	High performance membranes (e.g. automotive and medical)	Water repellence, oil repellence, stain-resistance, thermal stability
Leather applications	Leather based goods (e.g. leather bags, wallets, belts)	Water repellence, oil repellence
	Indoor and outdoor wear	Water repellence, oil repellence
	Footwear	Water repellence, oil repellence
	Professional sportswear and footwear	Water repellence, oil repellence
Other	E.g. home fabric treatments (sprays) for leather/textiles	Water repellence, oil repellence, stain-resistance, soil protection

* Textile product manufactured for non-aesthetic purposes, where function is the primary criterion.

ECHA (2023) estimated that the annual amount of PFAS used in this sector was between 41,000 and 143,000 tons. Over 75% of PFAS are fluoropolymers, with Teflon (polytetrafluoroethylene, PTFE) being the most common one, alongside polyvinylidene fluoride (PVDF), PFPE, FEP, and PFA. The most abundant PFAS types are C2-C3, C6, and side-chain fluorinated polymers. Many of the textiles on the European market are imported, mainly from Asia, making textiles, upholstery, leather, apparel and carpets potentially important PFAS sources for human exposure and environmental emissions.

3.1.3.2. Cosmetics

PFAS serve numerous purposes in a wide range of cosmetic products, including use as emulsifiers, antistatic agents, stabilizers, skin conditioners, binders, and viscosity regulators. The most commonly identified functions of PFAS in cosmetics are conditioning and film formation, besides PFAS acting as solvents and surfactants. ECHA (2023) compiled the most frequently used PFAS in cosmetics and their properties (Table 13), showing that C9-15 fluoroalcohol phosphate and PTFE are the most frequently used PFAS in cosmetic products.

Table 13: Main PFAS and identified properties in cosmetics (ECHA, 2023).

PFAS	PFAS category	Identified properties
PTFE	Polymeric PFAS	Bulking
C9-15 fluoroalcohol phosphate	PFAA and PFAA precursors	Skin conditioning
Perfluorodecalin	Fluorocarbon	Detangling Skin conditioning Solvent
Perfluorooctyl triethoxysilane	PFAA and PFAA precursors, containing Si	Binding
Perfluorononyl dimethicone	Polymeric PFAS containing Si	Skin conditioning
Polyperfluoromethylisopropyl ether	Polymeric PFAS	Skin conditioning
Octafluoropentyl methacrylate	PFAA and PFAA precursors	Binding
Acetyl trifluoromethylphenyl valyl-glycine	Fluorinated amino acid	Skin conditioning
Methyl perfluorobutyl ether	PFAA and PFAA precursors	Solvent Viscosity controlling

Brinch et al. (2017) primarily used Kemiluppen[2], a portal of the Danish consumer protection organization Tænk[3] to identify PFAS in cosmetic products and to select 18 products for further chemical analysis. The data extraction from Kemiluppen by Brinch et al. (2017) is from 2017, and changes can be expected to have occurred since then. We have extracted updated information as of 2024 for this report, but have used the concentrations obtained in Brinch et al. (2017) for the quantitative part of this project in section 3.2.

[2] https://taenk.dk/kemi/plejeprodukter-og-kosmetik/kemiluppen-tjek-din-personlige-pleje-uoensket-kemi
[3] https://taenk.dk/

The portal Vidensbank.mst.dk[4] is maintained by the Danish Environmental Protection Agency (EPA) and includes concentrations of individual compounds in consumer products such as textiles, carpets and cosmetics. The data sources are the Danish EPA's own investigations. Data search requires entries of specific compound names or CAS numbers. In this study, searches in the database used the compound names that had been identified in the literature, which may result in incomplete data returns of the PFAS in the database.

3.1.3.3. Electronics

PFAS compounds are used in electronics for their stability, insulating properties and resistance to heat. These include (ECHA, 2023):

- Fluoropolymers: PTFE, PFA, PVDF and FEP.
- Non-polymeric PFAS: Compounds such as PFBS are used as surfactants in electronics.

Considering consumer uses, main products with relevance for human exposure include:

- Electronic devices (e.g., smartphones, computers): PFAS are used in wires, cables, and coatings on printed circuit boards for insulation and fire resistance.
- Household appliances: PFAS are found in electrical components, such as cables and circuits, due to their durability and resistance to wear.
- Telecommunication equipment: Devices like routers and antennas use PFAS in circuit boards and wiring insulation to maintain performance over time.

Their use might lead to the following PFAS exposure routes for consumers:

- Dermal contact: Prolonged or frequent handling of electronics containing PFAS-coated components may lead to skin exposure.
- Ingestion: PFAS can migrate from electronics to household dust, which can be ingested inadvertently, especially by children who are more likely to come into contact with dust while playing.
- Indoor air inhalation: PFAS may slowly diffuse from electronics and other household appliances, contributing to low-level indoor air contamination. However, considering that most PFAS are not volatile, this is considered a minor pathway.

While exposure levels for consumers are generally considered low, PFAS in electronics may contribute to long-term, indirect environmental and household contamination (ECHA, 2023).

3.1.3.4. Consumer mixtures

As described above, consumer mixtures include cleaning products, waxes and polishes, car care products, rain-repellent fluids in the aviation industry etc. The main PFAS found in these consumer mixtures include various FTOHs, fluorotelomer ethoxylates (FTEOs), and PFCAs. These substances are present in a range of everyday products, leading to potential human exposure. The products and their related exposure pathways include:

- Cleaners (e.g., for glass, metal, and upholstery): These products often contain PFAS compounds such as FTOHs and ethoxylates.
- Waxes and polishes (e.g., for furniture, floors, and cars): Car waxes and floor polishes commonly contain PFAS compounds such as perfluoroalkyl phosphonic acids (PFPAs).
- Dishwashing products: PFAS are used as rinse aids, contributing to exposure through dishware use.
- Textile treatments: Waterproofing agents for textiles can contain PFAS that can leach into the environment and/or come into contact with skin.
- Car care products: PFAS can be present in windscreen treatments and other car maintenance products.
- Lubricants (e.g., for bike chains and locks): PTFE spray is commonly used, containing fluoropolymers.

The human exposure routes related to the use of these products include:

- Dermal contact: Skin contact with PFAS-treated products, such as cleaning or polishing surfaces, or when handling waterproofed textiles.

[4] https://vidensbank.mst.dk/

- Ingestion: Indirect exposure can happen through residues left on dishware or eating utensils treated with PFAS-containing products
  Inhalation: This occurs when PFAS are aerosolized during the application of sprays (e.g., cleaning products, car treatments).

3.1.4. Occupational exposure – snapshots

Occupational exposure to PFAS was not studied systematically in this project. For this reason, this section only provides snapshots of information found in the literature search and does not intend to present an exhaustive description of occupationally caused PFAS exposure. As summarized by ECHA (2023), the occupational use of PFAS involves various industries, including metal plating, firefighting, textiles, petroleum, and electronics, where PFAS are used for their chemical resistance, water repellence, and heat stability:

- Metal plating: PFAS are used as wetting agents and mist suppressants. Exposure occurs during electroplating, where PFAS mists or aerosols are released.
- Firefighting: PFOS was a key component in Aqueous Film-Forming Foams (AFFFs) used for extinguishing fuel fires. Other PFAS, such as FTOHs have been used since PFOS was banned in AFFFs, but are subject to phase-outs at the European level. Firefighters can be exposed when using AFFFs during fire suppression or training.
- Textiles: PFAS are applied to fabrics for stain and water resistance, especially in protective clothing. Workers may encounter PFAS exposure during fabric coating and treatment processes.
- Petroleum and mining: PFAS are used in drilling fluids and as anti-corrosion agents, with non-polymeric PFAS such as PFCAs having a crucial role. Drilling operations may expose workers to PFAS in fluids and surfactants used for extraction.
- Electronics: PFAS compounds such as fluoropolymers are employed as insulators and coatings in semiconductors and other electronics.
- Ski waxes can be a notable source of PFAS exposure. Ski waxes were shown to include PFOA, PFNA, PFDA and PFUnDA as well as PFSAs, present either as additives or impurities (Freberg et al., 2010, ECHA, 2023). During application, ski waxes are heated to 120–180°C, releasing aerosolized PFAS.

Considering these applications, the following exposure routes may occur for workers:

- Dermal Contact: Handling PFAS-containing materials, such as fabrics, drilling equipment or ski waxes, can lead to dermal absorption of PFAS.
- Ingestion: Workers might inadvertently ingest PFAS through hand-to-mouth actions after contact with contaminated surfaces or materials.
  Inhalation: Workers can inhale PFAS-containing mist or dust, especially in metal plating and firefighting activities where PFAS carrying aerosols are prevalent (ECHA, 2023). Inhalation of PFAS from ski waxes is the main exposure pathway for professionals in this field in poorly ventilated spaces (Freberg et al., 2010).

Nicolajsen and Tsitonaki (2016) aimed to identify industries and businesses in Denmark that have used or are using PFAS, which may serve as potential sources of human exposure through soil and groundwater contamination. Although PFAS have never been produced in Denmark, they have been imported and used in various industrial and consumer products. Certain industrial sites that have used PFAS could be contaminated to levels that pose a risk to human health through exposure to polluted soil, groundwater, or surface water, besides accumulation of PFAS in food chains. The Danish Regions are tasked with identifying such sites, producing an overview of PFAS use in Denmark essential for addressing human exposure risks.

The project run by the Danish Regions examined the use of PFAS in Denmark by conducting an in-depth review of data from the Product Register, detailing both historical and current industrial applications of PFAS. However, the information is limited, as reporting is only required for substances used in quantities over 100 kg or containing more than 1% of a hazardous substances or substances under the Classification, Labelling and Packaging Regulation (CLP) of the European Union.

Wang et al. (2019) found that most of the available information suggests that there is a greater risk for exposure for workers than end-consumers, and that releases to the environment occur during the manufacturing phase. However, this does not exclude the potential exposure of the general population to these substances via diffuse pollution since environmental releases during the manufacturing phase can also lead to the contamination of different environmental media such as drinking water or agricultural land.

The information compiled by Nicolajsen and Tsitonaki (2016) is included in Appendix A. It is notable that the list of compounds includes a relatively large number of PFAS that are not included in environmental monitoring campaigns or surveys of food and consumer products.

3.2. Uptake doses

The daily uptake doses of PFAS were estimated for each of the source matrices and low and high exposure scenarios (Section 2.4). For persistent PFAS, e.g. PFCAs and PFSAs, for which relevant precursors have been detected, the uptake consists of the direct uptake of the persistent PFAS and an indirect contribution via the precursor. For relevant precursors a direct uptake was calculated, represented by a precursor concentration that is not biotransformed into a persistent PFAS.

This section presents the LE and HE median, or mean concentrations, stated for the top five PFAS and relevant precursors for each exposure source and the resulting inhalation, ingestion and dermal uptake doses based on direct and indirect exposure.

3.2.1. Food, drinking water and food contact materials

The estimated daily intakes of PFAS via food and drinking water were calculated based on Eq. 1 and 2 considering LE and HE scenarios for children and adults. The results are summarized in Table 14 and Table 15 for children and adults, respectively. For the exposure from food and drinking water, ingestion was considered as the only relevant exposure pathway. For this reason, inhalation and dermal uptake are not included in Table 14 and Table 15. Furthermore, PFAS precursors were rarely reported in foods and drinking waters since these precursors are likely metabolized or degraded in foods, food processing and drinking water treatment. Therefore, no indirect exposure to PFAS precursors is included in the calculation of EDIs in foods and drinking waters.

Table 14: Estimated Daily Intake (in ng/kg bw/day) for children based on median concentrations of the most frequently detected PFAS in food and drinking water in the literature. LE: Low exposure. HE: High exposure. Only direct exposure was considered, i.e. no exposure via precursors. "Total" is the sum of the rows above.

Compound	Exposure medium	Median concentration, (ng/g)		Ingestion, (ng/kg bw/d)	
		LE	HE	LE	HE
PFOS	Food	3.0E-04	528	0.0141	24845
PFOA	Food	0.001	305	0.0471	14352
PFNA	Food	0.001	42.2	0.0471	1985
PFDA	Food	0.001	20.7	0.0471	973.1
PFUnDA	Food	8.0E-04	16.0	0.0376	753.8
PFOA	Drinking water	6.4E-05	0.0295	0.0029	1.342
PFNA	Drinking water	1.89E-05	0.004	0.0009	0.1840
PFBS	Drinking water	5.0E-05	0.0137	0.0023	0.6241
PFHpA	Drinking water	7.0E-05	0.007	0.0032	0.3189
PFOS	Drinking water	2.6E-05	0.0149	0.0012	0.6788
Total	Food+Drinking water			0.2035	42912

Since 1 January 2023, maximum levels for PFAS have applied to certain food items (Table 16). Maximum levels were set for the first time for PFOS, PFOA, PFNA and PFHxS and for the sum of these four PFAS for eggs, fish, fishery products, bivalve molluscs, crustaceans, meat, game and offal (EU, 2023). However, PFDA and PFUnDA, which were identified as frequently detected PFAS in food in our study, were not considered in this approach.

The concentrations in food items varied by several orders of magnitude between the LE and HE scenario, ranging from pg/g to ng/g in the LE and HE scenario, respectively The LE concentrations of the top five PFAS originate from data for milk and milk-based drinks, cheese, game birds and alcoholic drinks, ranging from 0.3 to 1 pg/g. The LE concentrations of milk and milk-based products are probably more relevant for the daily exposure of PFAS of both children and adults, while alcoholic drinks are only consumed by adults and game birds are not consumed frequently. The HE concentration of PFOS found in food is 528 ng/g, followed by PFOA (305 ng/g), PFNA (42.2 ng/g), PFDA (20.7 ng/g), and PFUnDA (16.0 ng/g). However, these HE concentrations are mostly from contaminated areas including industrial areas and some areas outside of Europe.

Table 15: Estimated Daily Intakes (in ng/kg bw/day) for adults based on median concentrations of the most frequently detected PFAS in food and drinking water in the literature. LE: Low Exposure. HE: High Exposure. Only direct exposure was considered, i.e. no exposure via precursors. "Total" is the sum of the rows above.

Compound	Exposure medium	Median concentration, (ng/g)		Ingestion, (ng/kg bw/d)	
		LE	HE	LE	HE
PFOS	Food	3.0E-4	528	0.0047	8335
PFOA	Food	0.001	305	0.0158	4815
PFNA	Food	0.001	42.2	0.0158	666
PFDA	Food	0.001	20.7	0.0158	326.4
PFUnDA	Food	0.0008	16.0	0.0158	252.9
PFOA	Drinking water	6.4E-05	0.0295	0.0017	0.7918
PFNA	Drinking water	1.89E-05	0.004	0.0005	0.1086
PFBS	Drinking water	5.0E-05	0.0137	4.52E-06	0.3683
PFHpA	Drinking water	7.0E-05	0.007	0.0019	0.1882
PFOS	Drinking water	2.6E-05	0.0149	0.0007	0.4006
Total	Food+Drinking water	-	-	0.0727	14433

Table 16: Maximum Levels of PFAS in foodstuffs (ng/g) (ECHA, 2023).

Foodstuffs	PFOS	PFOA	PFNA	PFHxS	Sum of four PFAS
Meat and editable offal	0.3-50	0.2-25	0.2-45	0.2-3.0	1.3-50
Fish products	2.0-35	0.2-8.0	0.5-8.0	0.2-1.5	2.0-45
Crustaceans and bivalve molluscs	3.0	0.7	1.0	1.5	5
Eggs	1.0	0.3	0.7	0.3	1.7

The European Union's recast Drinking Water Directive limits PFAS Total (measured as Extractable Organic Fluorine) in drinking water to 0.5 µg/L and the sum of 20 individual PFAS to 0.1 µg/L and member states are required to comply with these standards by 2026 (EU, 2020). It should be noted that PFAS Total refers to a different analytical method, which aims at determining the total content of (extractable) organic fluorine in a sample. This parameter is likely to exceed any sum of PFAS determined from individual target analyses.

The HE concentration of PFOA identified in drinking water in this study is 0.0295 µg/L, followed by PFOS with 0.0149 µg/L and PFBS with 0.0137 µg/L. In their sum, these values will be below the EU limit for the sum of 20 individual PFAS (0.1 µg/L), however, other PFAS might contribute to the sum that were not considered here.

In 2020, EFSA significantly reduced the tolerable weekly intake (TWI) for PFAS to 4.4 ng/kg body weight (EFSA, 2020). The total PFAS exposure (sum of the top five PFAS in food and drinking water) via ingestion of foods and drinking waters for children were 0.204 ng/kg bw/d (1.423 ng/kg bw/week) in the LE scenario and 42.9 µg/kg bw/d (300 µg/kg bw/week) in the HE scenario. For adults, the corresponding estimates were 0.0727 ng/kg bw/d (0.509 ng/kg bw/week) in the LE scenario and 14.4 µg/kg bw/d (101 µg/kg bw/week) in the HE scenario. The estimated exposure values in the LE scenario did not exceed the TWI for PFAS exposure set by EFSA, while the estimated exposure values in the HE scenario were considerably higher than the TWI values.

The PFAS exposure resulting from food contact materials is summarized in Table 17 and Table 18 for children and adults, respectively. The same reasoning as for food and drinking water applies here, i.e. ingestion was considered as the only relevant exposure pathway, and no indirect exposure via precursors was included. It can be discussed whether the contact with PFAS in food contact materials could also lead to dermal uptake, but this was not indicated in the reviewed literature. Considering the longer contact time with the food itself, the migration into food and exposure via food ingestion is probably more relevant than dermal contact to the food contact material.

Table 17: Estimated Daily Intake (in ng/kg bw/day) for children based on median concentrations of the most frequently detected PFAS in food contact materials. LE: Low Exposure. HE: High Exposure. Only direct exposure was considered, i.e. no exposure via precursors.

Compound	Exposure medium	Median concentration, (ng/dm^2)		Ingestion, (ng/kg bw/d)	
		LE	HE	LE	HE
PFOA	Food contact materials	4.55	15.8	5.63E-05	0.07821
PFHxA	Food contact materials	0.4	3.9	4.95E-06	0.01931
PFHpA	Food contact materials	0.45	2.1	5.57E-06	0.01040
PFDA	Food contact materials	0.45	11.8	5.57E-06	0.05841
PFNA	Food contact materials	0.02	2.6	2.48E-07	0.01287

Table 18: Estimated Daily Intake (in ng/kg bw/day) for adults based on median concentrations of the most frequently detected PFAS in food contact materials. LE: Low Exposure. HE: High Exposure. Only direct exposure was considered, i.e. no exposure via precursors.

Compound	Exposure medium	Median concentration (ng/dm^2)		Ingestion, (ng/kg bw/d)	
		LE	HE	LE	HE
PFOA	Food contact materials	4.55	15.8	3.06E-05	5.67E-02
PFHxA	Food contact materials	0.4	3.9	2.69E-06	1.40E-02
PFHpA	Food contact materials	0.45	2.1	3.03E-06	7.53E-03
PFDA	Food contact materials	0.45	11.8	3.03E-06	4.23E-02
PFNA	Food contact materials	0.02	2.6	1.35E-07	9.33E-03

In the EU, estimated exposures to chemicals in food contact materials are based on the assumption that a person weighs 60 kg and daily consumes 1 kg of packaged food in contact with 6 dm^2 of food contact material (Muncke, 2009). Different units are used in the literature for data describing the migration from food packaging into food, either µg/kg or ng/dm^2. The latter refers to the surface of the packaging in contact with the food material and can be directly used in the estimation of human exposure to PFAS. Although an increasing number of stud-

ies have reported occurrence of PFAS in food contact materials in recent years, there are very limited quantitative data on the PFAS migration for the top five PFAS and no available data for their precursors. Therefore, 6:2 diPAP and 8:2 diPAP were not included in the calculation although they had been identified as important precursors in the qualitative analysis in section 3.1 (Table 7).

The estimated exposure to PFAS from food contact materials through ingestion is several orders of magnitude lower than the exposure via ingestion of food and drinking water. However, as shown in Figure 5, the types of PFAS in food contact materials are more diverse compared to the PFAS spectrum in food and drinking water, which might result in human exposure to unknown and unidentified PFAS via food contact materials. It is striking that two PFAS (6:2 diPAP and 8:2 diPAP) were identified as important for human exposure from food contact materials, but their EDI could not be calculated because of lack of data. However, the marketing of food contact material that contains PFAS has been banned in Denmark since 2020. Consequently, this contribution to the total PFAS exposure can be expected to be further decreasing in Denmark.

3.2.2. Air and dust

PFAS concentrations in outdoor air are in the pg/m^3 range. Compared to the concentrations in indoor environments, where the concentrations can be in the ng/m^3 range, outdoor air is assessed not to be a relevant exposure pathway for human exposure and is therefore not included in the EDI calculations. For indoor air we have calculated the direct exposure to precursors and indirect exposure of PFAS exposure via precursors considering biotransformation within the body. The two compounds found at highest concentration in indoor air are 6:2 FTOH and 8:2 FTOH. Since these two compounds are volatile and present in the gas phase, they are directly inhaled and transferred to the lungs. Thus, we have calculated the exposure through inhalation only for the two FTOHs as well as the indirect exposure to the persistent PFAS that can be formed from biotransformation of 6:2 FTOH and 8:2 FTOH. The results for children and adults are summarized in Table 19 and Table 20, respectively.

Table 19: Estimated Daily Intakes (in ng/kg bw/day) for children based on median FTOH concentrations in indoor air (gas phase). LE: Low Exposure. HE: High Exposure. For the persistent PFAS, only indirect exposure from the biotransformation of FTOHs is considered. For cells with no data, no relevant exposure was identified.

Compound	Exposure medium	Median concentration, (ng/m^3)		Inhalation (ng/kg bw/d)			
		LE	HE	LE (direct)	LE (indirect)	HE (direct)	HE (indirect)
PFHxA	Indoor air (gas phase)	-	-	-	3.41E-03	-	2.93E+00
PFOA	Indoor air (gas phase)	-	-	-	2.11E-02	-	2.40E+00
PFNA	Indoor air (gas phase)	-	-	-	4.23E-02	-	1.20E+00
6:2 FTOH	Indoor air (gas phase)	0.042	18.0	3.41E-02	-	1.46E+01	-
8:2 FTOH	Indoor air (gas phase)	2.6	29.5	2.11E+00	-	2.40E+01	-

Table 20: Estimated Daily Intakes (in ng/kg bw/day) for adults based on median FTOH concentrations in indoor air (gas phase). LE: Low Exposure. HE: High Exposure. For the persistent PFAS, only indirect exposure from the biotransformation of FTOHs is considered. For cells with no data, no relevant exposure was identified.

Compound	Exposure medium	Median concentration, (ng/m^3)		Inhalation (ng/kg bw/d)			
		LE	HE	LE (direct)	LE (indirect)	HE (direct)	HE (indirect)
PFHxA	Indoor air (gas phase)	-	-	-	7.39E-04	-	6.33E-01

Compound	Exposure medium	Median concentration, (ng/m^3)		Inhalation (ng/kg bw/d)			
PFOA	Indoor air (gas phase)	-	-	-	4.58E-03	-	5.19E-01
PFNA	Indoor air (gas phase)	-	-	-	9.15E-03	-	2.60E-01
6:2 FTOH	Indoor air (gas phase)	0.042	18.0	7.39E-03	-	3.17E+00	-
8:2 FTOH	Indoor air (gas phase)	2.6	29.5	4.58E-01	-	5.19E+00	-

Dust exposure has been calculated as ingestion and not as inhalation based on the assumption that inhaled dust particles are transferred from the respiratory system to the mouth and then ingested. Exposure to indoor dust for children and adults for the relevant compounds are summarized in Table 21 and Table 22, respectively. Exposure to outdoor dust has been calculated on the basis of the data from only two studies from India and China, respectively, both conducted in the vicinity of a fluorochemical plant. Consequently, these calculations are likely less representative of the exposure of the general Danish population to PFAS from outdoor dust sources. The calculations represent a situation of a local emission source in the vicinity of the sampling site. Exposure data for outdoor dust, based on these studies from India and China, are summarized in Table 23 and Table 24 for children and adults, respectively.

Table 21: Estimated Daily Intakes (in ng/kg bw/day) for children based on median concentrations of the most frequently detected PFAS and relevant precursors in indoor dust. LE: Low Exposure. HE: High Exposure. Direct: Exposure to the compound in question. Indirect: Exposure through relevant precursors. For cells with no data, no relevant exposure was identified.

		Median concentration, (ng/g)		Ingestion (ng/kg bw/d)				Dermal contact (ng/kg bw/d)			
Compound	Exposure medium	LE	HE	LE (direct)	LE (indirect)	HE (direct)	HE (indirect)	LE (direct)	LE (indirect)	HE (direct)	HE (indirect)
PFHxA	Indoor dust	0.16	147.0	3.06E-04	2.35E-03	2.81E-01	316E-01	4.09E-05	314E-04	3.76E-02	4.23E-02
PFHpA	Indoor dust	0.61	92.6	1.17E-03	-	1.77E-01	-	1.56E-04	-	2.37E-02	-
PFOA	Indoor dust	0.42	852.0	8.03E-04	8.98E-05	1.63E+00	2.84E-01	1.07E-04	1.20E-05	2.18E-01	3.79E-02
PFNA	Indoor dust	0.63	38.1	1.20E-03	1.24E-04	7.28E-02	1.43E-01	1.61E-04	1.66E-05	9.73E-03	1.91E-02
PFDA	Indoor dust	0.32	22.5	6.12E-04	-	4.30E-02	-	8.18E-05	-	5.75E-03	-
PFOS	Indoor dust	0.87	185.0	1.66E-03	-	3.54E-01	-	2.22E-04	-	4.73E-02	-
6:2 diPAP	Indoor dust	50.40	287.0	9.44E-02	-	5.21E-01	-	1.26E-02	-	6.97E-02	-
8:2 diPAP	Indoor dust	2.90	99.3	5.43E-03	-	1.75E-01	-	7.26E-04	-	2.33E-02	-
6:2 FTOH	Indoor dust	2.20	756.0	3.78E-03	-	1.16E+00	-	5.06E-04	-	1.55E-01	-
8:2 FTOH	Indoor dust	1.80	1435.0	3.34E-03	-	2.33E+00	-	4.46E-04	-	3.12E-01	-

Table 22: Estimated Daily Intakes (in ng/kg bw/day) for adults based on median concentrations of the most frequently detected PFAS and relevant precursors in indoor dust. LE: Low Exposure. HE: High Exposure. Direct: Exposure to the compound in question. Indirect: Exposure through relevant precursors. For cells with no data, no relevant exposure was identified.

		Median concentration,(ng/g)		Ingestion, (ng/kg bw/d)				Dermal contact, (ng/kg bw/d)			
Compound	**Exposure medium**	**LE**	**HE**	**LE (direct)**	**LE (indirect)**	**HE (direct)**	**HE (indirect)**	**LE (direct)**	**LE (indirect)**	**HE (direct)**	**HE (indirect)**
PFHxA	Indoor dust	0.16	147.0	8.59E-05	6.60E-04	7.90E-02	8.89E-02	1.78E-05	1.37E-04	1.64E-02	1.85E-02
PFHpA	Indoor dust	0.61	92.6	3.28E-04	-	4.97E-02	-	6.80E-05	-	1.03E-02	-
PFOA	Indoor dust	0.42	852.0	2.26E-04	2.52E-05	4.58E-01	7.97E-02	4.68E-05	5.24E-06	9.50E-02	1.66E-02
PFNA	Indoor dust	0.63	38.1	3.38E-04	3.49E-05	2.05E-02	4.01E-02	7.03E-05	7.25E-06	4.25E-03	8.33E-03
PFDA	Indoor dust	0.32	22.5	1.72E-04	-	1.21E-02	-	3.57E-05	-	2.51E-03	-
PFOS	Indoor dust	0.87	185.0	4.67E-04	-	9.94E-02	-	9.70E-05	-	2.06E-02	-
6:2 diPAP	Indoor dust	50.40	287.0	2.65E-02	-	1.46E-01	-	5.51E-03	-	3.04E-02	-
8:2 diPAP	Indoor dust	2.90	99.3	1.53E-03	-	4.91E-02	-	3.17E-04	-	1.02E-02	-
6:2 FTOH	Indoor dust	2.20	756.0	1.06E-03	-	3.25E-01	-	2.21E-04	-	6.74E-02	-
8:2 FTOH	Indoor dust	1.80	1435.0	9.38E-04	-	6.55E-01	-	1.95E-04	-	1.36E-01	-

Table 23: Estimated Daily Intake (in ng/kg bw/day) for children based on median concentrations of the most frequently detected PFAS and relevant precursors in outdoor dust. LE: Low Exposure. HE: High Exposure. Direct: Exposure to the compound in question. Indirect: Exposure through relevant precursors. For cells with no data, no relevant exposure was identified. It should be noted that the data are from the vicinity of fluorochemical plants in India and China (see Table 10).

		Median concentration, (ng/g)		Ingestion (ng/kg bw/d)				Dermal contact (ng/kg bw/d)			
Compound	**Exposure medium**	**LE**	**HE**	**LE (direct)**	**LE (indirect)**	**HE (direct)**	**HE (indirect)**	**LE (direct)**	**LE (indirect)**	**HE (direct)**	**HE (indirect)**
PFHxA	Outdoor dust	0.02	0.3	6.48E-06	1.86E-07	9.94E-05	2.14E-06	8.66E-07	2.48E-08	1.33E-05	2.86E-07
PFHpA	Outdoor dust	0.05	0.4	1.51E-05	-	1.10E-04	-	2.02E-06	-	1.47E-05	-
PFOA	Outdoor dust	0.09	2.1	2.92E-05	1.30E-06	6.50E-04	4.65E-05	3.90E-06	1.74E-07	8.69E-05	6.22E-06
PFNA	Outdoor dust	0.3	0.4	9.35E-05	1.30E-06	1.21E-04	2.79E-05	1.25E-05	1.74E-07	1.62E-05	3.73E-06
PFDA	Outdoor dust	0.03	7.4	7.99E-06	-	2.30E-03	-	1.07E-06	-	3.08E-04	-
PFOS	Outdoor dust	0.01	2.0	2.81E-06	-	6.22E-04	-	3.75E-07	-	8.32E-05	-
6:2 diPAP	Outdoor dust	0.03	0.1	9.10E-06	-	4.06E-05	-	1.22E-06	-	5.43E-06	-
8:2 diPAP	Outdoor dust	0.4	3.0	1.28E-04	-	8.56E-04	-	1.71E-05	-	1.14E-04	-

Table 24: Estimated Daily Intake (in ng/kg bw/day) for adults based on median concentrations of the most frequently detected PFAS and relevant precursors in outdoor dust. LE: Low Exposure. HE: High Exposure. Direct: Exposure to the compound in question. Indirect: Exposure through relevant precursors. For cells with no data, no relevant exposure was identified. It should be noted that the data are from the vicinity of fluorochemical plants in India and China (see Table 10).

		Median concentration, (ng/g)		Ingestion, (ng/kg bw/d)				Dermal contact(ng/kg bw/d)			
Compound	Exposure medium	LE	HE	LE (direct)	LE (indirect)	HE (direct)	HE (indirect)	LE (direct)	LE (indirect)	HE (direct)	HE (indirect)
PFHxA	Outdoor dust	0.02	0.3	7.14E-07	2.05E-08	1.10E-05	2.36E-07	1.48E-07	4.25E-09	2.27E-06	4.89E-08
PFHpA	Outdoor dust	0.05	0.4	1.67E-06	-	1.21E-05	-	3.46E-07	-	2.51E-06	-
PFOA	Outdoor dust	0.09	2.1	3.21E-06	1.44E-07	7.16E-05	5.13E-06	6.67E-07	2.99E-08	1.49E-05	1.06E-06
PFNA	Outdoor dust	0.3	0.4	1.03E-05	1.44E-07	1.34E-05	3.08E-06	2.14E-06	2.99E-08	2.78E-06	6.38E-07
PFDA	Outdoor dust	0.03	7.4	8.81E-07	-	2.54E-04	-	1.83E-07	-	5.27E-05	-
PFOS	Outdoor dust	0.01	2.0	3.10E-07	-	6.86E-05	-	6.43E-08	-	1.42E-05	-
6:2 diPAP	Outdoor dust	0.03	0.1	1.00E-06	-	4.48E-06	-	2.10E-07	-	8.81E-07	-
8:2 diPAP	Outdoor dust	0.4	3.0	1.41E-05	-	9.43E-05	-	2.87E-06	-	1.92E-05	-

The uptake doses by indoor dust ingestion are in the same order of magnitude for the six persistent PFAS in the LE scenario, while they are significantly higher for PFOA compared to the other compounds in the HE scenario of indoor dust ingestion. Given the high concentrations of FTOHs in indoor dust, they contribute substantially to the indirect uptake of e.g. PFOA. However, the indirect exposure to PFOA from FTOHs in the gas phase is even higher than the indirect exposure from dust ingestion. The two exposure pathways become comparable when direct and indirect exposure from dust ingestion are combined.

Exposure from air inhalation and dust ingestion is generally higher for children than for adults. Exposure through ingestion of dust is far higher than dermal exposure for dust both indoor and outdoor. Indoor dust exposure is also far higher than outdoor dust exposure although the median concentrations in Table 23 and Table 24 are assumed to present values that relate to a local emission point source in India and China and might thus not be representative of the exposure situation of the general population in Denmark. Considering the persistent PFAS such as PFOA, the indirect exposure from gas phase precursors is important, followed by the accidental ingestion of indoor dust as a direct and indirect exposure source.

3.2.3. Consumer products

Uptake rates of PFAS from consumer products have been calculated for dermal contact as well as ingestion, the latter referring to hand-to-mouth contact and licking of e.g. textiles, which is only assumed relevant for children. The resulting EDI values are summarized in Table 25 and Table 26 for children and adults, respectively, in both cases considering LE and HE scenarios for the five most frequently reported persistent PFAS and three relevant precursors in consumer products.

Table 25: Estimated Daily Intake (in ng/kg bw/day) for children based on median PFAS concentrations in consumer products. Five frequently detected compounds and relevant precursors are included. LE: Low Exposure. HE: High Exposure. Direct: Exposure to a specific compound (persistent PFAS or precursor). Indirect: Exposure to persistent PFAS via its precursor. n.e.: not estimated. o.v: only one value available. Inhalation is not included as it will mainly result from sprays. This exposure pathway is not considered relevant for children. Blank cells ("-") indicate pathways that were considered irrelevant.

		Median concentration, (ng/g (red) or µg/m²)		Ingestion[a] (ng/kg bw/d)				Dermal contact, (ng/kg bw/d)			
Compound	**Products**	**LE**	**HE**	**LE (direct)**	**LE (indirect)**	**HE (direct)**	**HE (indirect)**	**LE (direct)**	**LE (indirect)**	**HE (direct)**	**HE (indirect)**
PFOA	Clothing, textiles	4.50E-01	2.31E+00	2.87E-04	2.29E-04	5.21E-02	3.41E-01	1.27E-01	2.48E-01	1.03E+01	1.05E+02
PFOA	Carpets	2.00E-01	1.67E+00	1.28E-04	n.e.	3.77E-02	3.50E-01	1.70E-01	n.e.	3.74E+00	5.39E+01
PFOA	Sanitary pads, paper diapers	2.01E+00	1.17E+01	n.e.	n.e.	n.e.	n.e.	2.10E+00 [b]	n.e.	4.40E+00 [b]	n.e.
PFOA	Body lotion, sunscreen	1.36E+01	2.85E+03	n.e.	n.e.	n.e.	n.e.	3.35E-05	n.e.	2.46E-01	n.e.
PFBA	Clothing, textiles	2.20E-01	2.26E+00	1.40E-04	2.17E-06	5.10E-02	7.67E-03	6.22E-02	2.35E-03	1.01E+01	2.36E+00
PFBA	Carpets	o.v.	5.10E-01	n.e.	n.e.	1.15E-02	5.17E-02	n.e.	n.e.	1.14E+00	7.96E+00
PFBA	Body lotion	o.v.	4.10E+00	n.e.	n.e.	n.e.	n.e.	n.e.	n.e.	3.54E-04	n.e.
PFHxA	Clothing, textiles	1.60E-01	1.45E+00	1.02E-04	2.17E-05	3.27E-02	5.11E-02	4.52E-02	2.35E-02	6.49E+00	1.58E+01
PFHxA	Carpets	9.60E-01	1.11E+00	6.12E-04	n.e.	2.50E-02	3.44E-01	8.14E-01	n.e.	2.49E+00	5.31E+01
PFHxA	Hygiene paper	o.v.	7.70E-01	n.e.	n.e.	n.e.	n.e.	n.e.	n.e.	n.e.	n.e.
PFHxA	Body lotion, sunscreen	1.43E+01	3.34E+03	n.e.	n.e.	n.e.	n.e.	3.51E-05	n.e.	2.88E-01	n.e.
PFHpA	Clothing, textiles	3.10E-02	8.40E-01	1.98E-05	n.e.	1.89E-02	n.e.	8.77E-03	n.e.	3.76E+00	n.e.
PFHpA	Carpets	5.10E-01	1.10E+00	3.25E-04	n.e.	2.48E-02	n.e.	4.33E-01	n.e.	2.46E+00	n.e.
PFHpA	Body lotion, sunscreen	4.95E+00	3.62E+02	n.e.	n.e.	n.e.	n.e.	1.22E-05	n.e.	3.12E-02	n.e.
PFDA	Clothing, textiles	2.00E-01	1.02E+00	1.28E-04	1.79E-04	2.30E-02	5.49E-01	5.66E-02	1.94E-01	4.57E+00	1.69E+02
PFDA	Body lotion, sunscreen	6.90E+00	1.45E+03	n.e.	n.e.	n.e.	n.e.	1.70E-05	n.e.	1.25E-01	n.e.
10:2 FTOH	Clothing, textiles	1.37E+01	1.89E+02	3.29E-03	-	2.06E+00	-	3.56E+00	-	6.35E+02	-
10:2 FTOH	Carpets	o.v.	9.14E+01	n.e.	-	9.96E-01	-	n.e.	-	1.53E+02	-
8:2 FTOH	Clothing, textiles	4.66E+01	1.40E+02	1.20E-02	-	1.83E+00	-	1.30E+01	-	5.64E+02	-
8:2 FTOH	Carpets	o.v.	1.95E+02	n.e.	-	2.55E+00	-	n.e.	-	3.93E+02	-
6:2 FTOH	Clothing, textiles	8.30E-01	1.76E+01	1.93E-04	-	1.97E-01	-	2.09E-01	-	6.07E+01	-
6:2 FTOH	Carpets	o.v.	1.19E+02	n.e.	-	1.33E+00	-	n.e.	-	2.04E+02	-
6:2 FTOH	Fabric masks	o.v.	6.78E-04	n.e.	-	n.e.	-	n.e.	-	3.30E-07	-

[a] Ingestion is based on hand-to-mouth and licking

[b] Dermal uptake doses from personal hygiene products are calculated in Zhou et al. (2023). The LE values correspond to calculated mean values in Zhou et al. (2023)

Table 26: Estimated Daily Intake (in ng/kg bw/day) for adults based on median PFAS concentrations in consumer products. Five frequently detected compounds and relevant precursors are included. LE: Low Exposure. HE: High Exposure. Direct: Exposure to a specific compound (persistent PFAS or precursor). Indirect: Exposure to persistent PFAS via its precursor. n.e.: not estimated. o.v: Only one value available. Ingestion is not included as it will mainly result from licking products and hand-to-mouth-activity. This exposure pathway is not considered relevant for adults. Blank cells ("-") indicate pathways that were considered irrelevant.

		Median concentration (ng/g (red) or µg/m2)		Inhalation, (ng/kg bw/d)				Dermal contact, (ng/kg bw/d)			
Compound	Products	LE	HE	LE (direct)	LE (indirect)	HE (direct)	HE (indirect)	LE (direct)	LE (indirect)	HE (direct)	HE (indirect)
PFOA	Clothing, textiles	2.00E-01	1.26E+01	-	-	-	-	3.96E-02	1.84E-01	3.94E+01	5.04E+01
PFOA	Carpets	o.v.	1.67E+00	-	-	-	-	n.e.	n.e.	5.23E-01	7.54E+00
PFOA	Spray for impregnation	o.v.	3.60E+01	n.e.	n.e.	1-31E-06	n.e.	n.e.	n.e.	4.94E-04	n.e.
PFOA	Sanitary pads, panty liners, menstrual cups, paper diapers	1.91E+00	2.01E+00	-	-	-	-	8.50E-03 a	n.e.	4.50E+00 a	n.e.
PFOA	Cosmetics, body lotion, sunscreen	2.10E+00	2.85E+03	-	-	-	-	2.49E-07	n.e.	2.46E-01	n.e.
PFBA	Clothing, textiles	2.00E-01	2.62E+00	-	-	-	-	3.96E-02	1.52E-02	8.21E+00	8.04E+00
PFBA	Cosmetics, body lotion	2.50E+00	1.80E+02	-	-	-	-	2.97E-07	n.e.	1.55E-02	n.e.
PFHxA	Clothing, textiles	8.00E-02	5.45E+00	-	-	-	-	1.58E-02	1.52E-01	1.71E+01	5.36E+01
PFHxA	Carpets	o.v.	1.11E+00	-	-	-	-	n.e.	n.e.	3.48E-01	7.42E+00
PFHxA	Hygiene paper	o.v.	7.70E-01	-	-	-	-	n.e.	n.e.	n.e.	n.e.
PFHxA	Cosmetics, body lotion, sunscreen	2.05E+00	3.34E+03	-	-	-	-	2.43E-07	n.e.	2.88E-01	n.e.
PFHpA	Clothing, textiles	8.00E-02	3.28E+00	-	-	-	-	1.58E-02	n.e.	1.03E+01	n.e.
PFHpA	Carpets	o.v.	5.10E-01	-	-	-	-	n.e.	n.e.	1.60E-01	n.e.
PFHpA	Menstrual cups	o.v.	4.80E-01	-	-	-	-	1.90E-01 [a]	n.e.	7.20E-01a	n.e.
PFHpA	Cosmetics, body lotion, sunscreen	1.25E+00	3.62E+02	-	-	-	-	1.48E-07	n.e.	3.12E-02	n.e.
PFDA	Clothing, textiles	8.00E-02	1.14E+01	-	-	-	-	1.58E-02	2.08E-01	3.57E+01	2.98E+00

		Median concentration (ng/g (red) or µg/m2)		Inhalation, (ng/kg bw/d)				Dermal contact, (ng/kg bw/d)			
PFDA	Spray for impregnation	o.v.	2.80E+01	n.e.	n.e.	1.02E-06	n.e.	n.e.	n.e.	3.84E-04	n.e.
PFDA	Panty liners	o.v.	4.20E-02	-	-	-	-	n.e.	n.e.	1.00E-02 [a]	n.e.
PFDA	Cosmetics, body lotion, sunscreen	3.00E+00	1.45E+03	-	-	-	-	3.56E-07	n.e.	1.25E-01	n.e.
10:2 FTOH	Clothing, textiles	2.10E+01	4.75E+01	-	-	-	-	3.82E+00	-	1.12E+02	-
10:2 FTOH	Carpets	o.v.	9.14E+01	-	-	-	-	n.e.	-	-	-
8:2 FTOH	Clothing, textiles	3.00E+01	1.37E+02	-	-	-	-	5.88E+00	-	3.86E+02	-
8:2 FTOH	Carpets	o.v.	1.95E+02	-	-	-	-	n.e.	-	-	-
6:2 FTOH	Clothing, textiles	7.70E+00	8.55E+01	-	-	-	-	1.36E+00	-	2.06E+02	-
6:2 FTOH	Carpets	o.v.	1.19E+02	-	-	-	-	n.e.	-	-	-
6:2 FTOH	Fabric masks	o.v.	6.78E-04	n.e.	-	n.e.	-	n.e.	-	8.50E-08	-

[a] Dermal uptake doses from personal hygiene products are calculated in Zhou et al. (2023). The LE values correspond to calculated mean values in Zhou et al. (2023)

The contributions from the different exposure pathways to the exposure of the five most frequently reported PFAS and the precursors, respectively, are summarized in Table 27, only considering consumer products.

The main source category for uptake of the five persistent PFAS and three precursors is clothing, textiles and carpets. For children and adults this source category contributes 90% (LE) and >99% (HE) for children and >98% (LE, HE) for adults, of the total uptake for all consumer product categories. The main exposure pathway for the compounds studied here is through the skin, i.e. >99% of the total uptake for all exposure pathways concerning consumer products, for both LE and HE and both children and adults. Direct uptake via the skin of the five PFAS and three precursors account for 97.7% (LE) and 83.1% (HE) of the total uptake for all pathways of PFAS exposure from consumer products for children (Table 27). The numbers are similar for adults, with 95.3% (LE) and 86.4% (HE), respectively (Table 27).

The direct exposure to the three FTOHs account for 79.6% (LE) and 81.1% (HE) of the total uptake from PFAS in consumer products, i.e. of all exposure pathways, for children, and 92.6% (LE) and 74.0% (HE) for adults, respectively. This direct uptake of FTOHs is nearly exclusively through dermal uptake. The indirect uptake of the five PFAS through dermal uptake of precursors is considerably smaller than the direct exposure to the precursors. It constitutes 2.2% (LE) and 16.4% (HE) of the total uptake, i.e. of all exposure pathways, for children, with similar numbers for adults (Table 27).

Table 27: Contribution in % of exposure pathways to the uptake doses from consumer products, for the top five persistent PFAS and the relevant precursors and for children and adults, respectively. LE: Low Exposure. HE: High Exposure. d: direct. i: indirect. The green cells indicate the predominant exposure pathways, and the orange cells indicate significant exposure pathways for consumer products. Blank cells ("-") were not included in the calculations because they were considered irrelevant.

	Inhalation				Ingestion				Dermal			
	LE (d)	LE (i)	HE (d)	HE (i)	LE (d)	LE (i)	HE (d)	HE (i)	LE (d)	LE (i)	HE (d)	HE (i)
Children: five PFAS	-	-	-	-	<0,01%	<0,01%	<0.01%	0.07%	18,1%	2,2%	2.0%	16.4%
Children: precursors	-	-	-	-	0,07%	-	0.36%	-	79.6%	-	81.1%	-
Adults: five PFAS	0%	0%	<0.01%	0%	-	-	-	-	2.7%	4,7%	12,4%	13,7%
Adults: precursors	0%	0%	0%	0%	-	-	-	-	92,6%	-	74.0%	-

In summary, both for children and adults, dermal exposure to the precursors 10:2 FTOH, 8:2 FTOH and 6:2 FTOH from clothing, textiles and carpets is the predominant pathway to PFAS uptake from consumer products (green cells in Table 27). In the HE scenario, direct and indirect dermal uptake of the five PFAS from clothing, textiles and carpets, are significant pathways to PFAS uptake from consumer products (orange cells in Table 27). It has to be noted that these are relative numbers within the group of consumer products that do not contain information about the absolute level of exposure. This information is given in the Table 25 and Table 26.

3.2.4. Combining results from different exposure media

Comparing the calculated EDIs across the different exposure matrices, the following points emerge:

- For food, drinking water and food contact materials, ingestion is the only relevant exposure pathway. Direct uptake of the five most frequently reported PFAS is the only uptake, as no relevant precursors were identified in the literature review. However, studies on these media do not usually include precursors, leading to a risk of a selection bias in the top five PFAS for which uptake doses were calculated.
- For indoor and outdoor dust, ingestion is approx. a factor of 8 and 5 higher than dermal uptake for children and adults, respectively.

- For indoor air, inhalation of two precursors, i.e. 6:2 FTOH and 8:2 FTOH, is the main exposure situation, leading to indirect exposure of PFOA, PFNA and PFHxA.
- Dermal exposure of the precursors 10:2 FTOH, 8:2 FTOH and 6:2 FTOH from clothing, textiles and carpets, is the predominant pathway to PFAS uptake from consumer products.
- In the HE scenario direct and indirect dermal uptake of the five PFAS from clothing, textiles and carpets (i.e. as persistent PFAS plus a contribution from their precursors), are significant pathways to PFAS uptake from consumer products.

It has to be noted that these points refer to relative contributions within each exposure medium, in order to identify the most relevant compounds and exposure pathways in the source categories that were studied in this project (see Section 2.1).

PFOA is a top five compound in all exposure matrices, except indoor air (gas phase), and can therefore be used in a comparison across the exposure sources (Figure 6). Although it was not identified as a frequently reported compound in indoor air (since it is not volatile and thus not likely to be present in the gas phase), it was included in the calculations for exposure from indoor air in relation to indirect exposure from the uptake of FTOHs (Table 19 and Table 20). It is therefore possible to compare EDIs for PFOA across all source categories and exposure pathways.

A comparison of relative uptake doses for children reveals that in the LE scenario, food and consumer products contribute 1.7% and approx. 97% of the total uptake dose of PFOA, respectively. The opposite is the case in the HE scenario, where food and consumer products contribute approx. 99% and 1.2% of the total uptake dose of PFOA, respectively. This variation is caused by a significant variation (factor of 10^6) in lowest and highest median PFOA concentrations in food. The variation for consumer products is significantly lower (factor of 10^2). The uptake doses for adults show the same tendency but are generally slightly lower than the corresponding values for children (Figure 6).

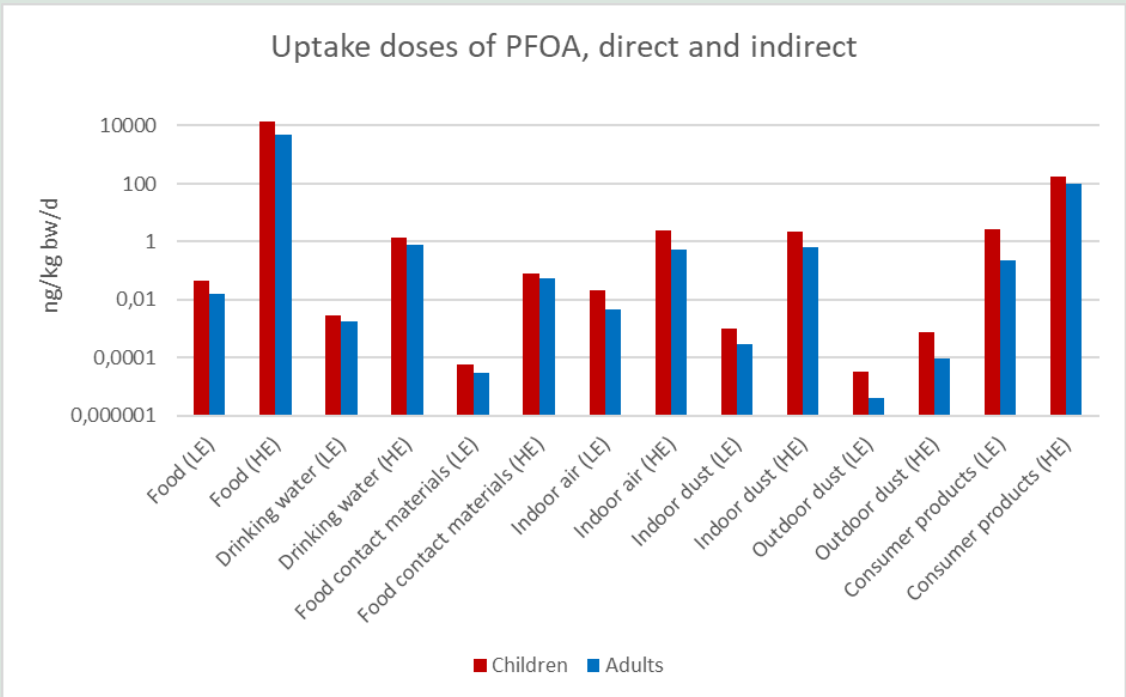

FIGURE 6. Uptake doses (ng/kg bw/d) of PFOA for both children and adults and different source categories, considering direct and indirect exposure. LE: Low exposure. HE: High exposure. Note that the y-axis has a logarithmic scale. The large variation in uptake rates for food is caused by large concentration ranges for PFAS in food items in the literature and is discussed further in the text.

It is important to note that this high contribution from consumer products is a relative one in the LE scenario. At this level, dermal uptake from PFAS in consumer products can contribute significantly, based on the data in the literature review, but it remains a low exposure level. Furthermore, the relative contribution (in percentage) would become lower if the lowest levels in food were higher. However, the absolute uptake dose (combining all source categories) would be higher (in cases of higher levels in food, all other absolute contributions remaining unchanged).

In both the LE and HE scenario, the relative exposure from indoor air contributes < 2%, drinking water contributes < 1%, and indoor dust contributes < 0.5% of the PFOA uptake for adults. The numbers are similar for children. Outdoor dust and food contact materials contribute insignificant amounts, both in absolute values (Figure 6) and with their relative contributions.

The calculated uptake doses of the other compounds can be used to quantify their relative significance for each of the source matrices (Figure 7). PFOA and PFOS contribute the most to the PFAS uptake of food and outdoor dust (HE), while PFOA contributes most to drinking water (HE) and food contact materials (LE). The FTOH precursors constitute the predominant PFAS uptake for consumer products, indoor dust and indoor air. As discussed previously, the compounds identified as primary ones in a certain matrix are likely affected by a research bias, as they are limited by the spectrum of compounds originally selected for the chemical analysis in each study of the literature review. All numbers can be found in Appendix B.

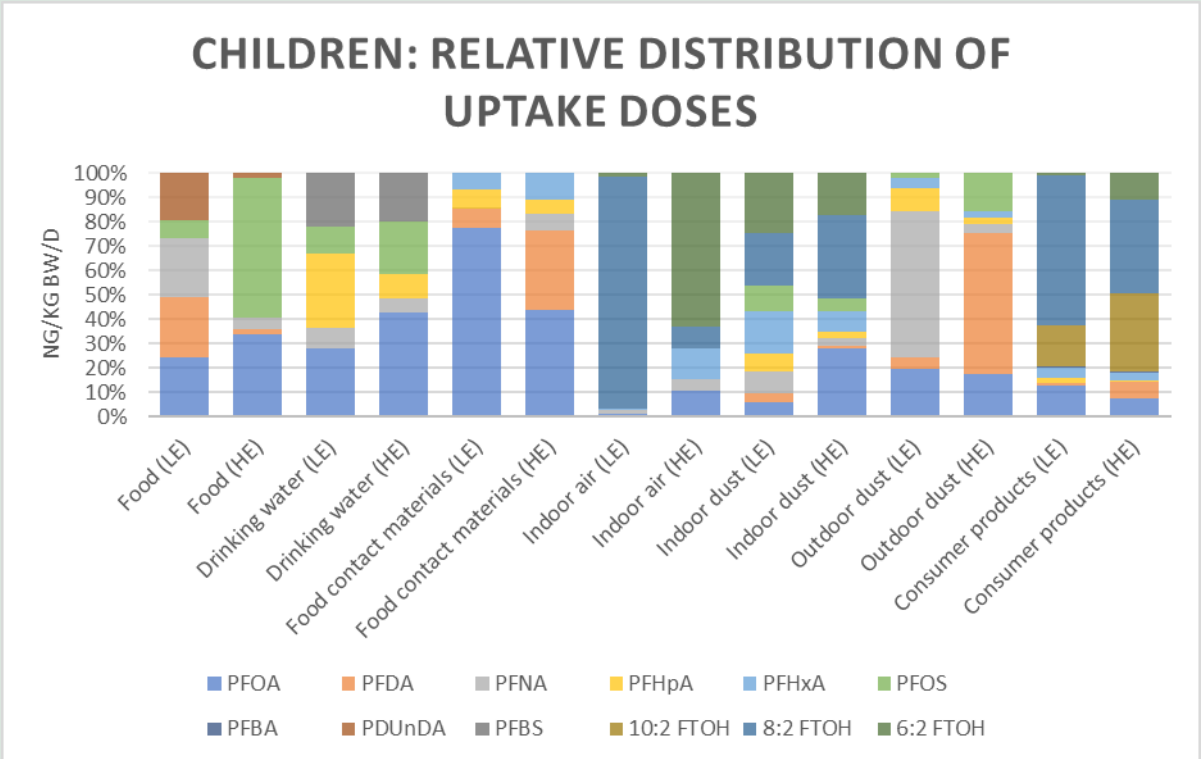

FIGURE 7. Relative contribution of the five most frequently reported PFAS and relevant precursors to the total uptake dose in children for each source matrix. LE: Low exposure. HE: High exposure. The distribution is similar for adults.

4. Discussion

4.1. Exposure pathways

Five persistent PFAS and relevant precursors were selected based on the number of detections in the source matrices (Section2.5). This selection includes the risk of a research bias, since the reported compounds are determined by the original selection for the chemical analysis. This often includes a standard set of PFAS and is not necessarily tailored to specific matrices. This means that other PFAS might have been overlooked. In addition, the detection frequency is not equivalent to a high concentration. It is important to note that the methodology has implications for the results. Alternatively, or in future more comprehensive assessment of PFAS exposure, compounds could be selected based on hazard or human risk, or new and emerging compounds could be included, for example identified in non-target screening approaches. However, concentration data will be needed for calculations of uptake doses.

Considering these potential caveats, the results of this study indicate that precursors are relevant for PFAS exposure from indoor air and dust, consumer products and food contact materials, causing direct and indirect exposure. However, the overall results indicate that indoor air and dust as well as food contact materials contribute insignificantly to the total exposure, whereas consumer products are suggested to contribute in the LE scenario, i.e. at a generally low exposure level.

The most frequently reported PFAS in food and drinking water were PFCAs and PFSAs, including PFOA, PFDA, PFNA, PFUnDA, PFHpA as well as PFOS and PFBS. PFOA, PFNA, PFDA, PFOS, and PFHpA are consistently reported as the top PFAS in multiple matrices investigated in the present study, which confirms their widespread presence, but does not exclude that other PFAS that were not included in the study might be equally important. PFUnDA is uniquely prominent in food, particularly in fatty animal-based foods and seafood. This pattern likely results from its long-chain structure, which facilitates bioaccumulation in animal tissues and aquatic ecosystems. Its high prevalence in food highlights the importance of dietary exposure in PFAS risk assessments.

PFBS is notably more prevalent in drinking water and water sources, reflecting its increased use as a replacement for longer-chain PFAS such as PFOS. Its shorter carbon chain makes it more water-soluble, allowing it to persist in aquatic systems and contaminate drinking water supplies. While it is less bioaccumulative than long-chain PFAS, its environmental persistence and mobility in aquatic environment remain a concern (Brendel et al., 2018). In general, little information is available on health effects of these shorter-chain PFAS.

PFCAs were found to be predominant in food contact materials. However, a wide range of other PFAS compounds have been identified in food contact materials in the literature, including precursors of more persistent PFAS, which highlights the complexity of PFAS exposure from this source. While PFAS have been phased out in food contact materials in Denmark, they are likely to be used in similar products elsewhere. The results indicate that the actual migration of PFAS into food is limited compared to the environmental levels of food itself. However, due to lack of occurrence data of PFAS precursors in food contact materials, related to indirect exposure, exposure to PFAS migration from these materials is likely underestimated. In addition, data gaps remain on the extent and actual migration process from PFAS in packaging into food.

Food obviously accounts for the majority of PFOA, PFDA, PFNA, PFOS and PFUnDA in HE scenarios, with contributions exceeding 85%. Even in LE scenarios, food contributes with a notable percentage, often around 0.5–2%, reflecting its significance even under highly conservative assumptions. In our calculations, the LE scenario via food ingestion was defined by the lowest concentrations of PFAS in milk and milk-based products, alcoholic drinks and game birds, whereas the HE scenario was defined by the highest PFAS concentrations in seafood and eggs. However, the human daily diet includes different food types. Therefore, the variation of EDIs via food ingestion will likely be smaller if the calculations were based on actual food consumption patterns and market basket studies. In addition, average exposure scenarios will likely also be closer to the real exposure situation than the HE and LE scenarios calculated in this study.

In both scenarios, children consistently showed higher PFAS exposure level than adults via ingestion of food and drinking water. This is related to their consumption patterns as well as lower body weight. A significant part of the data on PFAS exposure from consumer products is associated with exposure to children, e.g. from contact with clothing and carpets. Therefore, the assessment of uptake doses will give a good indication of the exposure of this sensitive age group.

Some values exceeded the TWI thresholds (4.4 ng/kg body weight per week) (EFSA, 2020), raising concerns about cumulative health risks. Drinking water had a relatively low contribution, typically <1% in both LE and HE scenarios for both children and adults. However, this situation might be different at contaminated sites where drinking water has higher PFAS concentrations (Domingo and Nadal, 2019).

Regarding the PFAS exposure from indoor air and dust, Trudel et al. (2008) considered the inhalation of particles small enough to reach the alveoli (< 4 µm). Larger particles can be inhaled but are likely deposited on mucosa and transported into the intestine. Therefore, they were considered in the pathway “ingestion of dust.” Uptake fractions for the lungs are applied to transform inhaled concentrations into uptake doses.

Dust ingestion considers the accidental ingestion of dust from e.g. hand-to-mouth activity as well as the inhalation of larger particles, which will be coughed up and transported in the intestine. The samples analysed in the literature are primarily comprised of settled dust, which are larger particles. Smaller particles, i.e. PM2.5, can be transported deeper into the lungs and have the potential to settle in the lungs and release sorbed compounds. Data on PM2.5 were therefore included in exposure scenario for inhalation of air.

The number of PFAS reported in consumer products and in occupational settings are higher than for the other source matrices. The relatively high uptake dose from dermal exposure, i.e. with consumer products such as clothing, textiles and carpets, was unexpected although it is still order of magnitudes below the highest intake from food. However, there are large uncertainties associated with the estimation of PFAS uptake from these consumer products. The study on children's carpets by Klinke et al. (2017) reported that no specific data on the skin absorption of fluorinated compounds was identified in the literature. Given their relatively high molecular weight, it was assumed that skin absorption is minimal. Moreover, since these substances are highly absorbable through oral intake, any potential contribution from dermal exposure was considered insignificant compared to oral exposure.

Also, the assumption of fractional uptake from dermal exposure was questioned by Kissel (2011). Fractional absorption depends on surface load and cannot be easily generalized across different conditions. High loads can artificially lower fractional absorption, leading to underestimation of skin permeability, while low loads can exaggerate efficiency. This metric also overlooks depletion effects and the influence of the experimental design. Kissel (2011) recommends that fractional absorption should only be used under consistent, well-defined loading and experimental conditions to ensure reliability.

FTOHs in consumer products such as clothing, textiles and carpets were discussed by Herzke et al. (2012). They reported that there was little knowledge of PFAS contents in consumer products and as a consequence, there was limited knowledge about possible emissions of PFAS from consumer products. In general, a number of fluorinated precursors are used to treat the surface of the consumer product material or they are chemically bound to polymers. Herzke et al. (2012) furthermore reported that there was limited knowledge about potential transformation routes to stable PFAS. There are estimates that 85% of indirect emissions of precursors transformed to stable PFCAs and PFSAs are a result of losses from consumer products during use and disposal (e.g. from carpets, clothing, paper and packaging, etc.). The remaining 15% are associated with manufacturing releases from secondary applications.

These challenges related to estimating uptake doses from consumer products have partly been overcome by new data, which have quantified the content of a large number of PFAS concentrations, including precursors, in a wide range of consumer products. Regarding the use of fractional uptake, the approach to use median or mean concentrations ensures that no extreme values are used in the calculation of dermal uptake. In this way a more reliable comparison was achieved between dermal uptake from different consumer products and e.g. from dust, although many uncertainties remain.

4.2. Comparison with exposure uptake values from other studies

EFSA (2020) calculated the human exposure to the sum of PFOA, PFNA, PFHxS and PFOS in food and drinking water for different age groups. The median exposure in adolescents, adults, elderly and very elderly ranged from 0.42 to 3.1 ng/kg bw/d for a low exposure situation and from 11.4 to 41.5 ng/kg bw/d for a high exposure situation. Toddlers and other children had approximately two-fold higher mean intake than older age groups, ranging from 0.84 to 6.5 ng/kg bw/d in a low exposure situation and from 38.5 to 112 ng/kg bw/d in a high exposure situation. In infants, the mean exposure ranges were 2.4–12.2 ng/kg bw/d and 42.8–115 ng/kg bw/d for a low and high exposure situation, respectively.

The total estimated daily intakes of the top five PFAS in food and drinking water for children (0.203 ng/kg bw/day) and adults (0.07 ng/kg bw/d) calculated for a low exposure scenario in our study were lower than the corresponding values in the EFSA report. However, the HE exposure to PFAS in food and drinking water for both children (42.9 µg/kg bw/d) and adults (14.4 µg/kg bw/d) exceeded the values calculated by EFSA by several orders of magnitude. This underlines that the LE and HE results of our study represent more extreme values than what will be representative for the Danish population. For example, we have included studies which investigated foods in high contaminated areas, such as industrial areas, as well as studies from China and USA where the contamination situation might be different. The highest concentrations of the top five PFAS in food in the present study were reported for garden eggs near a flurochemical plant (Lasters et al., 2024).

FEA (2014) calculated uptake doses of PFAS from durable water repellent jackets and found exposure levels for the general population of 0.054 ng/kg bw/d for PFOA, and up to 2.6 ng/kg bw/d for FTOHs in occupational scenarios. These were lower than dietary intake (3-10 times less for PFOA). However, occupational exposure in outdoor clothing stores could exceed dietary sources. In the present study uptake doses were approximately a factor of 10 higher in the LE scenario for consumer products. FEA (2014) considered a weighted approach considering new and washed clothes, which could explain some of the differences in findings. FEA (2014) also found that emissions during wearing and washing of jackets included air release of volatile precursors and complete leaching of extractable PFOA into washing water, i.e. emissions contributing to air and water. Furthermore, exposure from durable water repellent jackets was modelled against other sources. While dietary intake remained the dominant pathway for most populations, the contribution of consumer products became more significant in occupational settings.

Trudel et al. (2008) performed an assessment of consumer exposure to PFOS and PFOA from a variety of environmental and product-related sources. They found that consumers experience ubiquitous and long-term uptake doses of PFOS and PFOA in the range of 3-220 ng/kg bw/d and 1-130 ng/kg bw/d, respectively. In the present study the lower values correspond with the uptake doses for LE, while the higher values are significantly lower than the uptake doses for HE, which is caused by high values in food.

Trudel et al. (2008) also reported that the greatest exposure was likely to result from the intake of contaminated foods, including drinking water. Consumer products caused a minor portion of the total exposure to PFOS and PFOA. Of these, it was mainly impregnation sprays, treated carpets in homes, and coated food contact materials that contributed to PFAS exposure. Children tended to experience higher total uptake doses than teenagers and adults because of higher relative uptake via food consumption and hand-to-mouth transfer of compounds from treated carpets and ingestion of dust.

Gebbink et al. (2014) estimated direct and indirect uptake doses from ingestion of dust, food, drinking water, and inhalation of air and reported values that corresponded to or were a factor of up to 10 lower than LE values found in the present study for PFOS, PFBA, PFHxA, PFOA and PFDA. However, Gebbink et al. (2014) did not consider consumer products, which are significant for low exposure to PFBA, PFHxA, PFOA and PFDA, according to this study.

4.3. Data gaps

As previously discussed, the study did not consider a typical market basket that represents an average Danish diet, but used highest and lowest values in the literature. Some of these are likely different from exposure situation in Denmark. More precise exposure estimates can be achieved if food consumption was characterized more precisely

All calculations of uptake rates (Eq. 1-12) included fractions or rates that might not be available at all or not with the desired precision for specific compounds. For example, these include the fraction of a compound transferred from textiles to skin, the fraction transferred from skin to saliva and the uptake rate of a compound through skin, the gastrointestinal tract and the lungs. These data are largely missing and generate uncertainties in the calculations.

Most of the available literature on PFAS in food contact materials reports concentrations in the unit of mass/mass, typically nanograms per gram (ng/g). This unit focuses on the amount of PFAS present in the material itself, according to the chemical composition of the packaging or coating. While valuable for understanding the presence of PFAS in these materials, this unit is not suitable for the calculation of EDIs under the framework of EU regulations where the contact area with the food is important. In addition, there are no useful data for PFAS precursors in these materials to calculate direct exposure to precursors and indirect exposure when precursors are transformed. Therefore, the estimation of human exposure to migration of PFAS from food contact materials is likely underestimated. On the other hand, it can be assumed that the Danish population is exposed to PFAS from food contact materials to a minor extent because PFAS have been phased out from these materials.

Insufficient data for some of the precursors which were found to be relevant for human exposure prevented calculation of uptake doses for these, including uptake doses for the persistent PFAAs they can be transformed this. While some calculations were possible for FTOHs, we did not include diPAPs in the quantitative part of estimating uptake doses. However, diPAPs were found to be potentially important with regard to exposure from food contact materials, dust and consumer products. In addition, the large number of PFAS identified in consumer products without information of exposure and uptake rates might also present a significant data gap.

5. Conclusions

Some methodological aspects are important to note for the interpretation of the results of this study:

- Based on a literature review, we have calculated uptake doses for the most frequently reported persistent PFAS and relevant precursors. The frequency of reporting most likely includes a research bias as it depends on the compounds selected for the chemical analysis. This carries the risk that less frequently studied compounds are overlooked. In addition, the ranking according to frequency of reporting does not consider the exposure concentrations or the hazard of a compound.
- The literature review includes some studies from e.g. China and the USA as well as studies from locations that might be affected by local emission sources of PFAS. These are included in the exposure calculations and will potentially lead to estimates of uptake doses that might not be representative of the general exposure situation in Denmark.
- The uptake doses have been calculated for a low and a high exposure scenario, representing the lowest and highest median or mean values in the respective media, and the lowest and highest parameter values that are used in the equations, respectively. These do not represent an average exposure situation, but rather a worst case scenario and best case scenario.
- It is also important for the interpretation of the results to distinguish between relative and absolute values. The relative contribution of one source category depends on the relative contribution of the others. Given the low contribution of food in the LE scenario, the other source categories can have high relative contributions.
- Several data gaps and uncertainties exist, with some examples given in Section 4.3.

The project aimed at answering the following questions (Section 1):

A) Which PFAS are primarily present in exposure-relevant media?
   - Key compounds identified across media include PFOA, PFOS, PFNA, PFHxA and PFHpA. However, their identification might be affected by a selection bias.
   - DiPAPs are frequently reported for food contact materials, indoor dust and consumer products, but uptake rates could not be calculated because of missing data. Thus, their contribution to the total PFAS exposure might be underestimated.
   - Long-chain compounds are often reported in food. Besides PFOS and PFOA, PFNA, PFDA and PFUnDA were important in food.
   - PFOA, PFBS and PFHpA were important compounds in drinking water (besides PFOS).
   - The precursors 6:2 FTOH, 8:2 FTOH and 10:2 FTOH were identified in many consumer products. PFBA, PFHxA and PFHpA were also identified as relevant PFAS in consumer products. Given the high number of PFAS in consumer products, other PFAS might be important for which data are missing.
   - FTOHs and diPAPs are also important in indoor air and dust. However, they contribute less than e.g. food and consumer products to the total PFAS exposure under different scenarios.
   - Precursors in indoor air, dust, and consumer products give rise to exposure through biotransformation into more persistent PFAS such as PFOA. More specifically, indirect dermal uptake of FTOHs in consumer products account for approx. 2% of the total exposure, predominantly of PFOA and PFDA.

B) Which exposure pathways are most relevant for different individual compounds?
   - In the HE scenario, ingestion of food is the most significant pathway, contributing approx. 98% of the total exposure for long-chain PFAS including PFOA, PFOS, PFNA, PFDA and PFUnDA.
   - In the LE scenario, dermal uptake contributes >90% of the total exposure, particularly from consumer products, such as treated textiles, and includes a substantial contribution from FTOH precursors. The uptake rates for food ingestion are lower under the LE scenario than the uptake rates from dermal contact, influenced by the large range of PFAS concentrations in food.

- Inhalation contributes <2% in either exposure scenario, primarily from indoor air containing volatile precursors, primarily FTOHs.

C) How much does each source contribute to the total human exposure to various PFAS?
- Food is the dominant source of exposure under the HE scenario, contributing approx. 98% to the total PFAS exposure, mainly from fatty animal-based food and seafood. However, the contribution of food is small under the LE scenario (<1%), owing to the large variation in food exposure concentrations. As discussed above, this might not be representative of the exposure situation from a conventional mixed diet.
- The contribution of drinking water is relatively small and accounts for <1% in both HE and LE scenarios, here primarily for PFOA, PFBS and PFHpA. The situation can be different in areas with contaminated drinking water.
- The contribution from food contact materials was generally negligible. However, the research bias may play a role here, causing a risk that not frequently analysed PFAS may be overlooked. Critical data gaps exist for the quantification of this exposure pathway.
- The contribution from the indoor environment (dust and air) is < 1% under the HE scenario and 4-10% under the LE scenario. The main factor is the exposure to precursors, in particular FTOHs. Outdoor dust was found to be negligible in both scenario calculations.
- Consumer products contribute with approximately 5-6% to the total PFAS exposure under the HE scenario, but > 89% under the LE scenario. This high relative contribution is influenced by the very low food concentrations in the LE scenario. The main compounds were FTOHs, but also PFOA and PFDA. Given the high number of PFAS compiled in consumer products, there is a risk that important PFAS are overlooked in exposure assessments. Critical data gaps exist in the quantification of the uptake doses.

D) How do the contributions of individual sources compare to each other?
- HE scenario: Ingestion of food is a primary source of PFAS exposure (> 93%), with additional contributions from consumer products (5-6%). The remaining media each contribute with minor percentages.
- LE scenario: Dermal uptake of precursors in consumer products account for the main part (>89%) of the total PFAS uptake. Ingestion contributes with 1.7% for children and 6.2% for adults, across all different source categories. The main ingestion is from food, the second highest contribution is from indoor dust. Inhalation of indoor air contributes to the total uptake of PFOA and PFNA via the exposure to precursors.

E) Are there obvious differences across different age groups?
- Most uptake doses are higher for children than for adults, which is mainly related to the lower body weight in the calculations.
- Children experience higher dermal exposure and dust ingestion from hand-to-mouth behaviour. For consumer products, ingestion was only considered for children (resulting from hand-to-mouth and licking behaviour), whereas inhalation (from spray products) was only considered for adults. However, both pathways contribute insignificantly to the total PFAS exposure.

In this study uptake doses were generally higher than what had been reported in previous studies. This could be due to the fact that more source matrices have been included here, and that concentrations in food that are used here, in some cases are significantly higher than in the previous studies.

The lowest food concentrations in the LE scenario were defined by milk and milk-based drinks, alcoholic drinks and game birds in current calculations, which resulted in low EDIs via food ingestion in the LE scenario. Considering human diet is a mix of different foodstuffs, these values might be uncharacteristically low, with implications for relative contributions from other source categories.

In summary, while ingestion of food is the largest source of PFAS exposure, our calculations indicate a contribution from consumer products via dermal pathways for a low exposure scenario, which is influenced by low PFAS concentrations in food. Precursors like FTOHs and diPAPs play an important role, contributing to direct and indirect exposure through transformation into persistent PFAS, but missing data complicate the uptake dose calculations and increase uncertainties. However, they should not be disregarded in total exposure assessments.

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Appendix A: Literature review

The data compiled in the literature review are available in a separate spreadsheet.

Appendix B: Uptake doses

The calculations of uptake doses are available in a separate spreadsheet.

Summary:
The presence of PFAS in products, food and the environment can lead to PFAS exposure from multiple sources and through various pathways. The objective of this project was to study their contribution to the total PFAS exposure.
A literature review identified 189 different PFAS in consumer products, 57 in food items, 55 in drinking water, 77 in food contact materials and 60 in indoor air and dust. This list might be biased as it reflects the PFAS typically selected for analysis. Daily intakes were estimated for frequently re-ported PFAS and associated precursors, considering direct and indirect exposure. For example, PFOA can be taken up directly with food or result from uptake of precursors transformed in the body.
In a high exposure scenario using the highest values in the literature, nearly all PFAS exposure was through food, with perfluorooctane sulfonate (PFOS) and long-chain perfluorocarboxylic acids (PFCAs) as the main compounds. In a low exposure scenario, dermal uptake of PFAS from consumer products had an important relative contribution. This was related to small contributions from food, increasing the relative significance of other exposure sources. PFAS in consumer products included PFCAs of different chain lengths, fluorotelomer alcohols (FTOHs) and less-studied PFAS such as polyfluoroalkyl phosphoric acid diesters (di-PAPs). Exposure from indoor air and dust was generally < 10%.
The project indicates that other exposure pathways than food can have relative significance if the PFAS content in food is low. The project also underlines the complexity of many different compounds in exposure-related media.

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