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External PFAS Exposure from Food and Drinking Water compared with Internal Exposure

A Comparative Analysis using Kinetic Models

RIVM letter report 2026-0112
E.J.P. Hermans et al.

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Synopsis

External PFAS Exposure from Food and Drinking Water compared with Internal Exposure

A Comparative Analysis using Kinetic Models

RIVM is investigating ways to reduce PFAS exposure in the Netherlands. To do so, there needs to be clarity about the sources of people's exposure to these substances. Earlier research by RIVM showed that food and drinking water are important sources. The latest research examined whether these are indeed the main sources of total PFAS exposure.

RIVM compared the predicted amounts of PFAS that people in the Netherlands ingest through food and drinking water with the amounts measured in blood. This was done using kinetic models. These are calculation models that show how the amount of a substance in the body changes over time. RIVM used the quantities it had previously calculated for six PFAS.

The observed amounts in blood (internal exposure) differ little from the predicted amounts ingested through food and drinking water (external exposure). This indicates that food and drinking water are major sources of PFAS exposure. However, RIVM cannot conclude with certainty that food and drinking water are the main sources of PFAS. There are too many uncertainties to determine the exact extent to which food and drinking water contribute to total exposure. This is partly because observed PFAS levels in blood and information on food consumption was from different groups of people.

To better understand the contribution of other sources, RIVM recommends conducting more measurements of PFAS in the Netherlands. There are currently few data on sources other than food and drinking water. Another recommendation is to measure PFAS in blood and record what the same individuals eat and drink.

RIVM carried out this research on behalf of the ministries of Infrastructure and Water Management (IenW), Health, Welfare and Sport (VWS) and Agriculture, Fisheries, Food Security and Nature (LVVN) within the PFAS research programme.

Keywords: PFAS, human biomonitoring, food, drinking water, PBK modelling

Publiekssamenvatting

Externe blootstelling aan PFAS uit voedsel en drinkwater vergeleken met de interne blootstelling

Een analyse met kinetische modellen

Het RIVM onderzoekt mogelijkheden om de blootstelling aan PFAS in Nederland te verminderen. Daarvoor moet duidelijk zijn via welke bronnen mensen aan deze stoffen blootstaan. Eerder onderzoek van het RIVM liet zien dat voedsel en drinkwater belangrijke bronnen zijn. Nu is onderzocht of zij de belangrijkste bronnen voor de totale blootstelling aan PFAS zijn.

Het RIVM heeft hiervoor de berekende hoeveelheden PFAS die mensen in Nederland binnenkrijgen via voedsel en drinkwater vergeleken met de gemeten hoeveelheden in bloed. Dit is gedaan met zogeheten kinetische modellen. Deze rekenmodellen laten zien hoe de hoeveelheid van een stof in het lichaam door de tijd heen verandert. Het RIVM werkte hiervoor met de hoeveelheden die het eerder voor zes PFAS berekende.

De gemeten hoeveelheden in bloed (interne blootstelling) blijken weinig te verschillen van de berekende hoeveelheden vanuit voedsel en drinkwater (externe blootstelling). Dat geeft aan dat voedsel en drinkwater belangrijke bronnen van PFAS blootstelling zijn. Maar het RIVM kan niet met zekerheid concluderen of voedsel en drinkwater de *belangrijkste* PFAS-bronnen zijn. Er zijn te veel onzekerheden om te kunnen zeggen hoeveel voedsel en drinkwater precies bijdragen aan de totale blootstelling. Dit komt onder andere doordat de hoeveelheid PFAS in bloed en informatie over voedsel bij verschillende groepen mensen is gemeten en verzameld.

Om duidelijker te krijgen wat de bijdrage is van andere bronnen, raadt het RIVM onder andere aan om in Nederland meer te gaan meten. Over andere bronnen dan voedsel en drinkwater zijn nu nog weinig data. Een andere aanbeveling is om bij dezelfde mensen PFAS in bloed te meten en in kaart te brengen wat ze eten en drinken.

Het RIVM deed dit onderzoek in opdracht van de ministeries van Infrastructuur en Waterstaat (IenW), Volksgezondheid, Welzijn en Sport (VWS) en Landbouw, Visserij, Voedselzekerheid en Natuur (LVVN) binnen het PFAS-onderzoeksprogramma.

Kernwoorden: PFAS, humane biomonitoring, voedsel, drinkwater, PBK-model

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Samenvatting

Inleiding en doelstelling

Per- en polyfluoralkylstoffen (PFAS) zijn persistente synthetische chemische stoffen die in veel producten en processen worden gebruikt. PFAS worden onder andere aangetroffen in drinkwater, voedsel, lucht, bodem en consumentenproducten, wat leidt tot een wijdverspreide blootstelling. In Nederland wordt de bijdrage van inname via voedsel drie keer hoger geschat dan de bijdrage via drinkwater (Schepens et al., 2023). Humane biomonitoring (HBM)-studies hebben aangetoond dat inwoners van Nederland meerdere PFAS in hun bloed hebben, waarbij PFOS in de hoogste concentraties voorkomt (Bil et al. 2025; Sprong et al., 2026).

Dit project richt zich op de vergelijking tussen de interne en externe blootstelling aan zes PFAS (PFOA, PFOS, PFNA, PFHxS, PFDA, PFUnDA). Omdat in de beschikbare studies over bronnen anders dan voedsel en drinkwater weinig Nederlandse gegevens beschikbaar zijn, de beschikbare informatie over deze bronnen beperkt en niet homogeen is en er verschillende methoden voor het schatten van de blootstelling worden gebruikt richt deze studie zich alleen op voedsel en drinkwater. Met behulp van toxicokinetische modellen wordt de geschatte externe blootstelling aan PFAS via voedsel en drinkwater omgerekend naar serumconcentraties. Vervolgens worden die berekende serumconcentraties vergeleken met de gemeten PFAS-concentraties in bloedserum om na te gaan of voedsel en drinkwater de belangrijkste bronnen van blootstelling zijn.

Methode

Berekening van externe blootstelling
Data van Schepens et al. (2023) zijn gebruikt om de externe blootstelling van PFOA, PFOS, PFNA, PFHxS, PFDA, en PFUnDA via voedsel en drinkwater te berekenen. In aanvulling op Schepens et al. zijn de berekeningen in dit rapport voor de individuele stoffen zonder toepassing van relatieve potentiefactoren gegeven en is aanvullend het vijfde percentiel (P05) berekend. PFAS-concentraties waren beschikbaar voor een breed scala aan voedingsmiddelen en voor drinkwater (afkomstig van grondwater (GW) en oppervlakte-/infiltratiewater (SI)), van monsters die verzameld zijn in 2021–2022. Deze concentraties zijn gecombineerd met consumptiegegevens uit de Nederlandse Voedselconsumptiepeiling (2012–2016; volwassenen 18–79 jaar). PFAS-concentraties onder de detectie- of kwantificeringslimiet (LOQ) zijn verwerkt in een ‘lower bound’ (LB) en ‘upper bound’ (UB) scenario, waarbij deze waarden respectievelijk als nul en als LOQ zijn behandeld. Langdurige dagelijkse innames (ng/kg lichaamsgewicht/dag) voor de volwassen algemene populatie zijn berekend met het Observed Individual Mean (OIM) model, geïmplementeerd in de Monte Carlo Risk Assessment (MCRA) software, en uitgedrukt als 5e, 50e en 95e percentielen (P05, P50, P95) voor elke PFAS, drinkwaterbron (GW vs SI) en LOD/LOQ substitutiescenario (LB vs UB).

Gemeten interne blootstelling
Voor de interne blootstelling zijn gewogen populatiepercentielen (P50, P95) van serumconcentraties van de 6 PFAS gemeten in 967 volwassen bloeddonors (monsters uit 2025) uit Sprong et al. (2026) gebruikt, met een LOQ van 0,1 µg/L voor alle PFAS. PFAS-concentraties onder deze LOQ zijn verwerkt in een LB- en UB-scenario. In aanvulling op Sprong et al. is het P05 van de gewogen serumconcentraties bepaald.

Vergelijking van gemeten en voorspelde concentraties
De gegevens over de externe blootstelling aan PFAS via voedsel en drinkwater zijn gebruikt om met behulp van de kinetische modellen serumconcentraties (interne blootstelling) te voorspellen. Er zijn verschillende kinetische modellen toegepast: een fysiologisch gebaseerd kinetisch (PBK) model voor PFOA (Pachoulide et al. 2026); PBK-modellen van EFSA (2020) voor PFOA, PFOS, PFNA en PFHxS; één-compartiment toxicokinetische (TK) modellen van ATSDR (2021) voor PFOA, PFOS, PFNA en PFHxS; en één-compartiment TK-modellen gebaseerd op Abraham et al. (2024) voor alle zes PFAS.
In de simulaties is uitgegaan van een constante dagelijkse blootstelling, en geen verschil tussen PFAS-concentraties in plasma- en serum. De voorspelde serumconcentraties van volwassenen bij blootstelling via voedsel en drinkwater zijn vergeleken met de gemeten concentraties bij bloeddonors.

Resultaten

Vergelijking van de gemeten en voorspelde serumconcentraties
De voorspelde serumconcentraties, berekend op basis van blootstelling aan PFAS via voedsel en drinkwater, waren lager dan de gemeten serumconcentraties voor de laagste (LB) scenario's, met uitzondering van PFUnDA. UB-scenario's leidden over het algemeen tot voorspelde serumconcentraties die hoger waren dan de gemeten concentraties, met uitzondering van PFOS en PFHxS.

Voor PFOS lagen de voorspelde serumconcentraties voor alle scenario's lager dan de gemeten concentraties. Voor PFUnDA waren de voorspelde waarden vaak hoger dan de gemeten P95 waarden.

De verschillen in voorspelde PFAS-concentraties tussen de verschillende kinetische modellen waren kleiner dan het verschil tussen de voorspelde en gemeten concentraties. Dus de modelkeuze lijkt niet de belangrijkste oorzaak te zijn voor de verschillen tussen de voorspelde en gemeten concentraties.

Discussie en aanbevelingen

De analyse suggereert dat de huidige schattingen van blootstelling via voedsel en drinkwater de gemeten interne PFAS-concentraties bij volwassenen in Nederland niet volledig kunnen verklaren, vooral voor PFOS. Dit kan verschillende oorzaken hebben. Door het grote aantal meetwaarden onder de LOD/LOQ kan de blootstelling via voedsel en drinkwater onder- of overschat zijn door gebruik van respectievelijk het LB- en UB-scenario. Ook de incomplete dekking van voedingsmiddelen en de gebruikte modelaannames kunnen een rol spelen. Daarnaast zijn er onzekerheden in de kinetische modellen, met name in

parameterwaarden zoals eliminatiehalfwaardetijden en verdelingsvolumes, en door het gebruik van vaste (punt)schattingen voor deze parameters en voor lichaamsgewicht in plaats van populatieverdelingen. Als laatste is de historische blootstelling niet meegenomen bij het voorspellen van serumconcentraties.

Verdere verfijning van de blootstellingsschatting, met name voor niet-voedingsroutes en historische blootstelling, is noodzakelijk om een volledig en geïntegreerd beeld van PFAS-blootstelling te verkrijgen. Niet-voedingsbronnen, zoals textiel, persoonlijke verzorgingsproducten, sprays, huisstof en binnenlucht, kunnen voor sommige personen substantieel bijdragen aan de totale blootstelling (Affourtit et al. 2026). Het wordt daarom aanbevolen om gegevens over PFAS-concentraties in consumentenproducten, huisstof en binnenlucht te verbeteren en te actualiseren, bij voorkeur gebruikmakend van methoden met lagere detectielimieten. Een andere aanbeveling is om bij dezelfde mensen PFAS in bloed te meten en in kaart te brengen wat ze eten en drinken om stevigere conclusies te trekken over de bijdrage van voedsel en drinkwater in relatie tot andere bronnen van PFAS-blootstelling. Een nationaal humaan biomonitoringsprogramma kan hierin een belangrijke rol spelen door zowel biomonitoringsgegevens als gedetailleerde blootstellingsinformatie systematisch te verzamelen bij een representatieve steekproef van de bevolking.

Conclusie

De resultaten van dit project bevestigen dat blootstelling via voedsel en drinkwater een belangrijke route is voor PFAS-blootstelling bij de volwassen Nederlandse bevolking. De huidige schattingen van blootstelling via voedsel en drinkwater kunnen echter de gemeten interne PFAS-concentraties bij volwassenen in Nederland niet volledig verklaren, vooral niet voor PFOS. Over het algemeen lagen de voorspelde serumconcentraties in dezelfde orde van grootte als de gemeten concentraties. Tegelijkertijd laten de bevindingen geen harde, kwantitatieve conclusie toe dat voedsel en drinkwater de belangrijkste bronnen van blootstelling zijn. Het wegnemen van de onzekerheden in de analyse en toekomstig onderzoek met gedetailleerde deelnemersinformatie en HBM-data is nodig om de rol van voedsel en drinkwater ten opzichte van andere PFAS-bronnen beter te bepalen.

1 Introduction

PFAS (per- and polyfluoroalkyl substances) are a large group of synthetic chemicals that do not occur naturally in the environment. The Dutch general population is exposed to PFAS through various sources and routes of exposure. PFAS are used in a range of products and industrial processes due to their useful properties, for example in fluorinated gases, textiles, medical devices and personal care products (Affourtit et al., 2026). Because of their widespread use and persistence in the environment, PFAS are present in various environmental media, including groundwater (GW) and surface/infiltration (SI) water used for drinking water production. Moreover, bioaccumulation in aquatic and terrestrial food chains causes food to be contaminated with PFAS. Another source of PFAS in food is transfer from PFAS-containing food contact materials (Bokkers et al., 2019).

Given the potential impact of diet and drinking water as exposure routes, PFAS exposure via these pathways has been characterised for the Dutch population. Specifically, an investigation into PFAS exposure in the Netherlands via food and drinking water showed the amount of PFAS that people ingest via this route is above the health-based guidance value. The contribution of food was three times higher than the contribution of drinking water (Schepens et al., 2023). PFUnDA, PFOS and PFDA contributed most to PFAS exposure via food and drinking water. The exposure was calculated using PFAS concentrations as measured in a broad range of food items and water samples from all ten Dutch drinking water companies. These data were combined with Dutch food consumption data to calculate dietary exposure levels.

To investigate the relationship between these external exposure levels and internal concentrations, human biomonitoring (HBM) can be a particularly valuable tool. A study by Bil et al. (2025), using serum samples from 2016-2017, showed that all people aged 0 to 87 years in the Netherlands have multiple PFAS in their blood. Nearly all serum samples contained quantifiable levels of PFOA, PFNA, PFDA, PFUnDA, PFHxS, PFHpS and PFOS, with PFOS found in the highest quantities. Concentrations in blood of children and women of childbearing age were slightly lower compared to the general population. A more recent HBM study among blood donors (aged 18-77 years) in the Netherlands measured PFAS in serum samples retrieved in 2025 (Sprong et al., 2026). PFOA, PFHxS, and PFOS were found in virtually all participants, with PFOS in the highest quantities. Moreover, other PFAS were found in 91% (PFNA), 37% (PFDA), 19% (PFHpS) and 12% (PFUnDA) of the adult population respectively. Other PFAS were only found in ≤2% of the population (PFBA, PFHpA, PFBS, PFDoDA) or were not found above the detection limit at all (PFPeA, PFHxA, PFTrDA, PFTeDA, PFDS, HFPO-DA). The general population had slightly higher PFAS concentrations in their blood compared to women of childbearing age.

Epidemiological studies in humans have linked long-term exposure to PFAS to adverse effects, such as liver damage, reduced birth weight, and a decreased immune response (EFSA, 2020). The cumulative

internal exposure[1] to PFAS exceeds the health-based guidance value in a large part of the Dutch population (Bil et al., 2025; Sprong et al., 2026). Above this guidance value, adverse effects on the immune system cannot be ruled out. Given that cumulative internal exposure exceeds the health-based guidance value in almost the entire Dutch population, understanding the contribution of different exposure pathways has become particularly important (Sprong et al., 2026).

On behalf of the ministries of Infrastructure and Water Management (IenW), Health, Welfare and Sport (VWS), and Agriculture, Fisheries, Food Security and Nature (LVVN), the RIVM is investigating how the exposure of the Dutch population to PFAS through food, drinking water and the environment can be reduced, in addition to the measures already foreseen (Bulder et al., 2024; https://www.rivm.nl/pfas/onderzoeksprogramma). As part of this research programme, the sources of exposure to PFAS are characterised, and the relationship between these external sources and internal exposure is quantitatively assessed.

One of the aims of the research programme is to relate the exposure to PFAS via food, drinking water and other sources to internal exposure. Comparing HBM data with the predicted exposure can provide an indication of the extent to which all exposure sources have been identified (Bulder et al., 2024). Affourtit et al. (2026) studied the exposure to house dust, indoor air, water-repellent outdoor clothing, impregnation sprays and personal care products. They report that, due to limited and heterogeneous data for non-food sources, conservative exposure estimates and the different methods for exposure assessments, no realistic quantitative estimate of total PFAS exposure could be made. Furthermore, data on non-food sources are often not specific for the Dutch context, which makes comparison with Dutch blood levels less relevant. Therefore, the aim of the current study was limited to evaluating whether exposure via food and drinking water is the main source of PFAS exposure. To do this, we used kinetic models to compare the most recent studies on external dietary exposure from food and drinking water (Schepens et al., 2023) and internal exposure to PFAS in the adult Dutch population (Sprong et al., 2026). The focus is on PFOA, PFOS, PFNA, PFHxS, PFDA, and PFUnDA as these PFAS had the highest detection frequencies in the HBM blood donor study (Sprong et al., 2026) and had the highest contribution to dietary exposure from food and drinking water (Schepens et al., 2023). Furthermore, these PFAS have at least one existing toxicokinetic model in the public literature to convert external dietary exposure to serum levels.

[1] Internal exposure refers to the amount of PFAS that is taken up by the body from external sources and that is systemically available. Internal exposure is reflected by human biomonitoring, which includes measuring the concentration of chemicals in biological matrices such as blood, urine, saliva, or hair.

2 Material and Methods

2.1 External exposure from dietary sources

Data from Schepens et al. (2023) were used to calculate the external dietary exposure to six PFAS (PFOA, PFOS, PFNA, PFHxS, PFDA, and PFUnDA) in the Dutch population. In addition to Schepens et al. (2023), these dietary exposure estimates were calculated for single PFAS instead of mixtures. Furthermore, the estimates are applied without conversion using relative potency factors, in order to allow direct comparison between external and internal exposure estimates for the individual PFAS. Also, the 5th percentile (P05) of the exposure distribution was calculated alongside the median (P50) and 95th percentile (P95).

An extensive description of the methods can be found in Schepens et al. (2023). A brief description is given below.

2.1.1 Food consumption data

Food consumption data were derived from the Dutch National Food Consumption Survey (DNFCS) of 2012-2016 (van Rossum et al., 2020). In the DNFCS food consumption data were obtained from 2147 participants aged 18-79 years, by means of two non-consecutive 24-hour dietary recalls in accordance with the EFSA Guidance for the collection of national food consumption data (EFSA, 2009).

2.1.2 Concentration data

Food

Concentration data in food and drinking water were derived for food and drinking water collected in 2021-2022. Food products available on the Dutch market were sampled and analysed for PFAS. Food products were selected based on the consumption patterns of the whole Dutch population, as reported in the DNFCS of 2012-2016 (van Rossum et al., 2020). The reported concentrations represent the linear isomers, except for PFOS, of which the sum of linear and branched isomers is reported. The limit of detection (LOD) in the food samples varied from 0.05 to 90 pg/g and the limit of quantification (LOQ) from 0.1 to 180 pg/g, depending on the PFAS and the specific food products (Schepens et al., 2023).

PFAS concentrations reported below the LOD/LOQ were substituted using a lower bound (LB) and upper bound (UB) scenario, in which these values were treated as zero or LOQ, respectively. The estimated LB and UB exposures show the possible range of dietary exposure to PFAS, considering the analytical uncertainties around the PFAS concentration. It is anticipated that the LB and UB exposures will, respectively, underestimate and overestimate the actual dietary exposure to PFAS (Schepens et al., 2023).

Drinking water

Samples of drinking water sourced from GW and SI were collected from all ten Dutch drinking water companies, ensuring nationwide coverage, and analysed for PFAS concentrations. The concentrations of the linear

isomers were reported for all PFAS analysed. For PFOS, PFOA and PFHxS, the sum of linear and branched isomers was reported. The LOQ in the drinking water samples ranged from 0.1 to 5 ng/L, depending on the PFAS and the drinking water type (Schepens et al., 2023). To ensure uniformity between the different laboratories, no LODs were reported by Schepens et al. (2023). Concentrations for each individual PFAS were imputed according to the LB and UB scenario, and a mean concentration of each PFAS was calculated for drinking water sourced from GW and SI separately.

2.1.3 Allocating PFAS concentrations to consumed foods and drinking water

The measured PFAS concentrations in the food products and drinking water were combined with the DNFCS. The Dutch food conversion model (van Dooren et al., 1995) was applied to estimate PFAS concentrations in composite foods and in processed variants of the sampled food products. PFAS concentrations measured in drinking water were allocated to beverages containing drinking water as an ingredient. Two different scenarios were run for drinking water exposure: one with drinking water from GW and the other one from SI.

2.1.4 Exposure calculation

Chronic exposure to each PFAS was estimated using the Observed Individual Mean (OIM) model implemented in the Monte Carlo Risk Assessment (MCRA) software (version 10.2). Schepens et al. (2023) note that other models are available which estimate high exposure more realistically. However, the data were not fit for this purpose and therefore the OIM model was applied. For each of the participants in the DNFCS, daily consumption amounts of foods and drinking water were multiplied by the mean of measured PFAS concentrations in the respective foods and drinking water, and the resulting values were summed across all foods and drinking water for each day. Per individual, the daily exposure levels were averaged over the two consumption days, and divided by the participants' body weight (bw). This procedure resulted in distributions of the estimated daily dietary exposure (ng/kg bw/day) per PFAS in Dutch adults (Schepens et al., 2023).

To quantify sampling uncertainty in food consumption and concentration data caused by a limited sampling size the bootstrapping approach was used in Schepens et al. (2023). In the kinetic models only the median value was used.

The 5^{th}, 50^{th} and 95^{th} percentile of these distributions were used as input for toxicokinetic modelling (further delineated in section 2.3 below), representing the low, average, and highly exposed consumers respectively.

2.2 Internal exposure concentrations

The internal exposure of Dutch adults was assessed by Sprong et al. (2026) using measured PFAS concentrations in serum obtained from healthy blood donors retrieved in 2025 (Sprong et al., 2026). Additionally, the P05 of the exposure distribution was extracted alongside the P50 and P95. The LOQ of the analytical method used in this study equalled the LOD and was 0.1 µg/L. The reported

concentrations represent the linear isomers, except for PFOA, PFHxS and PFOS, of which the sum of linear and branched isomers is reported. In total, 967 blood serum samples were available for statistical analysis. To obtain a nationally representative picture of internal PFAS exposure, the data were weighted to correct for overrepresentation of participants from Dordrecht and the Western Scheldt areas and for underrepresentation of sociodemographic groups. Weighting used CBS census distributions (1 January 2017) for age, sex, education (8-category scale recoded to three census levels), province and degree of urbanisation. More information on the methods can be found in Sprong et al. (2026).

2.3 Kinetic models

2.3.1 Changes in bodyweight over life

As the internal exposure of Dutch adults is also the result of dietary exposure during childhood and adolescence, we simulated exposure from birth up to age 79. With the daily dietary exposure expressed in ng/kg bw/day, we applied a mathematical model (equation 1) in all of the kinetic models to describe the changes in bodyweight over life for the Dutch population (te Biesebeek et al., 2014).

$$bw = a(c_1 - (c_1 - 1)e^{-\left(\frac{age}{b_1}\right)^{d_1}})(c_2 - (c_2 - 1)e^{-\left(\frac{age}{b_2}\right)^{d_2}}) \qquad \text{(equation 1)}$$

The mathematical model described in equation 1 was fitted separately for males and females (te Biesebeek et al., 2014), yielding two sets of parameter values. The resulting sets of parameter values represent an average male and an average female, and are reported in Table 1.

Table 1 Overview of parameter values (rounded to 3 significant digits) used to model the change in bodyweight over life for the average Dutch male and female as reported according to te Biesebeek et al. (2014)

Parameter	Male	Female
a	2.24	2.64
b_1	4.62	5.44
b_2	14.2	12.3
c_1	11.4	10.1
c_2	3.48	2.74
d_1	0.571	0.643
d_2	2.98	3.30

Model simulations were done separately for the average male and the average female, from birth up to age 79. We combined both simulations to calculate the average concentrations for the average Dutch adult.

2.3.2 Selection of PBK models

The PBK models to perform the analysis were selected based on their use in regulatory contexts, availability for the PFAS of interest and the accessibility of the model and the associated parameter data to the investigators. Several PBK models are currently available, especially for PFOS and PFOA, including both multi-compartment and single-compartment models. For PFDA and PFUnDA only the toxicokinetic model based on Abraham et al. (2024) was available. Highly complex

multi-compartment models were not strictly required in order to predict blood concentrations. However, multiple models were included whenever available, in order to evaluate the consistency of model predictions and identify any divergent results.

2.3.3 *PBK model for PFOA (Pachoulide et al., 2026)*

Previously, a mechanistic whole-body human PBK model was developed for PFOA, fully relying on *in vitro* and *in silico* input data (Pachoulide et al., 2026). This model includes parameters for active transport in the intestine, kidney and liver, of which parameter values were extrapolated from *in vitro* to *in vivo*. The tissue-to-plasma partition coefficients were estimated based on the work from Utsey et al. (2020). Other parameters, such as organ weights and blood flows, are based on human physiology of an average person. The model differentiates between male and female subjects, takes into account age-dependent changes in glomerular filtration rate and includes menstrual clearance for females between the age of 12 and 51. For the current report, the model as published by Pachoulide et al. (2026) was implemented in R (version 4.5.1) and was adapted to incorporate the change in bodyweight over life for the Dutch population, as described in Section 2.3.1.

For the intake, we applied the external exposure estimates as explained in Section 2.1.4 and assumed that the daily exposure (expressed in ng/kg bw/day) was constant throughout life. We also assumed that the concentrations of PFOA in plasma equals those in serum (Ehresman et al., 2007; Poothong et al., 2017).

2.3.4 *PBK models for PFOA, PFOS, PFNA and PFHxS (EFSA, 2020)*

In their scientific opinion, the EFSA CONTAM panel applied a whole-body human PBK model that was developed for PFOA and PFOS to simulate the kinetics of PFOA, PFOS, PFNA and PFHxS over lifetime (EFSA, 2020). These models were based on those of Loccisano et al. (2011), but some modifications were implemented by EFSA (EFSA, 2018). These modifications include the use of the tissue/plasma partition coefficients rather than the tissue/blood partition coefficients for PFOS, adjusted values for renal resorption to represent the correct unit (µg instead of mg), a half-life of 2.3 years for PFOA instead of 3.8 years, a cardiac output fraction to the liver of 0.069 instead of 0.25, administration of the daily exposure in 24h instead of 0.6h and the implementation of a growth equation. Due to the lack of PBK models for PFNA and PFHxS, the PBK model for PFOA was applied to PFNA and the PBK model for PFOS was applied to PFHxS, assuming identical kinetics for PFOA and PFNA, and for PFOS and PFHxS. The PBK models for PFOA and PFOS included fitted parameters for renal clearance, resulting in half-lives of 2.3 and 5.4 years, respectively. For the derivation of tissue-to-plasma partition coefficients, *in vivo* animal data were used. Other parameters, such as organ weights and blood flows, are based on human physiology of an average person.

For the current report, the model as published in EFSA (2020) was implemented in R (version 4.5.1) and adapted to incorporate the change in bodyweight over life for the Dutch population, as described in Section 2.3.1.

The EFSA model considers exposure during the first year of life only by means of breastfeeding. For the first year of life we applied the same breast milk concentrations as estimated by EFSA. Thereafter, we applied the external dietary exposure estimates as explained in Section 2.1.4 and assumed that the daily exposure (expressed in ng/kg bw/day) was constant throughout life. We also assumed that the concentrations of the PFAS in plasma are equal to those in serum (Ehresman et al., 2007; Poothong et al., 2017).

2.3.5 *Toxicokinetic models for PFOA, PFOS, PFHxS and PFNA (ATSDR, 2021)*

The ATSDR (2021) presented a one-compartment model for PFOA, PFOS, PFHxS and PFNA (Figure 1), for which model parameters were derived based on various human and animal studies. In the model, the concentration in serum is determined by the amount in serum, divided by the volume of distribution (V_d), and the elimination from serum, determined by an elimination rate constant (k_e). For all four PFAS, ATSDR (2021) reported a gastrointestinal absorption fraction of 1.

The volume of distribution is dependent on the bodyweight, for which we applied the change in bodyweight over life, as described in Section 2.3.1 (Equation 1). The model was implemented in R (version 4.5.1) and the parameter values used in the models are presented in Table 2.

Figure 1 Graphical representation of the one-compartment model as presented by ATSDR (2021). V_d, volume of distribution; k_e, serum elimination rate constant.

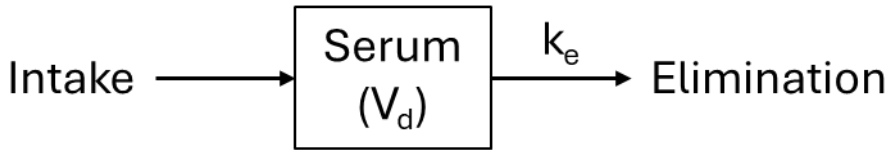


Table 2 Overview of model parameters for PFOA, PFOS, PFNA and PFHxS as reported by ATSDR (2021)

Parameter	PFOA	PFOS	PFNA	PFHxS
Serum elimination rat constant, k_e (day^{-1})	0.000495	0.000347	0.000759	0.000223
Apparent volume of distribution, V_d (L/kg)	0.2	0.2	0.2	0.287

For the intake, we applied the external dietary exposure estimates as explained in Section 2.1.4 and assumed that the daily exposure (expressed in ng/kg bw/day) was constant throughout life.

2.3.6 *Toxicokinetic models for PFOA, PFOS, PFNA, PFHxS, PFDA, PFUnDA (Abraham et al., 2024)*

Abraham et al. (2024) presented two-compartment models for a wide range of PFAS, including for PFOA, PFOS, PFHxS, PFNA, PFDA and PFUnDA. The second compartment was included to better describe the initial distribution of the PFAS during the first days after bolus exposure and led to better estimation of the terminal elimination rates and

volumes of distribution when compared to a one-compartment model. Unfortunately, the kinetic parameters for the distribution to the second compartment are not presented by Abraham et al. (2024). However, as we are simulating lifetime exposure rather than a single bolus exposure, the initial distribution of the PFAS is not of interest. The second compartment is therefore not required. As a result, the structure of the model based on the data of Abraham is the same as the model presented by ATSDR (Figure 1). The model parameters were fitted to the experimental data of the study and are presented in Table 3. Abraham et al. (2024) also presented values for the serum elimination rate constant in the supplemental material when the data were fitted to a one-compartment model. However, the visual inspection of the fits showed a clear overestimation of the elimination rate and no values were provided for the volume of distribution. Therefore, we decided not to include these parameter values in our current assessment. Based on Table 2 in Abraham et al. (2024) the dose unabsorbed was 0% for PFHxS and PFOS, ≤0.05% for PFOA, PFNA, and PFDA, and 0.2% for PFUnDA. The unabsorbed fraction was considered negligible and, therefore, a gastrointestinal absorption fraction of 1 was assumed for these PFAS.

Table 3 Overview of model parameters for PFOA, PFOS, PFNA, PFHxS, PFDA and PFUnDA as reported by Abraham et al., 2024

Parameter	PFOA	PFOS	PFNA	PFHxS	PFDA	PFUnDA
Serum elimination rate constant, k_e (day^{-1})	0.000345	0.000573	0.000531	0.000424	0.000804	0.00119
Apparent volume of distribution, V_d (L/kg)	0.121	0.152	0.124	0.125	0.184	0.241

For the intake, we applied the external dietary exposure estimates as explained in Section 2.1.4 and assumed that the daily exposure (expressed in ng/kg bw/day) was constant throughout life. We also assumed that the concentrations of the PFAS in plasma equal those in serum (Ehresman et al., 2007; Poothong et al., 2017). Here, we also applied the change in bodyweight over life, as described in Section 2.3.1 (Equation 1).

2.4 Statistical analysis

To identify any deviations between predicted and observed concentrations, we calculated the ratio of observed to predicted concentrations for the P05, P50 and P95 of the lower and upper bound measured concentrations across all exposure scenarios. The goal was to identify the difference between predicted and observed concentrations, independent of the direction of difference. To prevent values above and below 1 cancelling each other out, the higher value was always divided by the lower one. If the observed concentration was 0 (i.e. below the LOQ in the lower-bound scenario), no ratio was calculated. For each

PFAS, the ratios per scenario and the mean ratio over all scenarios are reported.

To assess uncertainty in the kinetic models, we similarly calculated ratios between predicted concentrations: for each PFAS, the ratios between the predicted P05, P50 and P95 concentrations of each model to the predicted P05, P50 and P95 concentrations of the other models were calculated. Again, the higher value was always divided by the lower value. Since for PFDA and PFUnDA only one model was used, no model uncertainty was determined for these PFAS.

3 Results

3.1 External exposure estimates

Below, the daily dietary exposure estimates of PFOA, PFNA, PFDA, PFUnDA, PFHxS, and PFOS for the adult Dutch population are provided (Table 4). These daily estimates were based on the data used in Schepens et al. (2023) and served as modelling input to simulate the predicted PFAS concentrations in serum upon chronic exposure, whereby it was assumed that the daily exposure was constant throughout life.

Table 4 Daily exposure estimates of six PFAS in the Dutch population (ages 18-79) resulting from diet (including drinking water) for the low (P05), average (P50), and highly exposed consumer (P95) (ng/kg bw/day), stratified by drinking water source for the lower and upper bound scenario respectively. Only the median value after bootstrapping was used in the kinetic models, and therefore no uncertainty is presented here.

PFAS	Drinking water source	Treatment of left censored data	Daily exposure estimate from diet (ng/kg bw/day)		
			P05	P50	P95
PFOA	GW	LB	0.0076	0.018	0.043
	GW	UB	0.13	0.23	0.42
	SI	LB	0.029	0.06	0.13
	SI	UB	0.15	0.26	0.47
PFNA	GW	LB	0.00050	0.0020	0.0056
	GW	UB	0.031	0.056	0.14
	SI	LB	0.0019	0.0042	0.0086
	SI	UB	0.025	0.046	0.12
PFDA	GW	LB	0.0015	0.0039	0.036
	GW	UB	0.029	0.054	0.10
	SI	LB	0.0015	0.004	0.037
	SI	UB	0.022	0.040	0.081
PFUnDA	GW	LB	0.015	0.058	0.17
	GW	UB	0.055	0.12	0.26
	SI	LB	0.015	0.058	0.16
	SI	UB	0.047	0.11	0.25
PFHxS	GW	LB	0.0013	0.0026	0.0058
	GW	UB	0.021	0.040	0.072
	SI	LB	0.0059	0.013	0.026
	SI	UB	0.020	0.037	0.065
PFOS	GW	LB	0.015	0.041	0.16
	GW	UB	0.042	0.077	0.20
	SI	LB	0.026	0.054	0.17
	SI	UB	0.042	0.077	0.20

GW, groundwater; SI, surface and infiltration water; LB, lower bound; UB, upper bound

3.2 Observed PFAS serum concentrations in the blood donor study

In Table 5, the weighted serum concentrations of the different PFAS are shown, derived from Sprong et al. (2026), apart from the P05 that was calculated for the purpose of this report. These observed serum

concentration data in the blood donor study were used to compare to the predicted serum concentrations resulting from dietary exposure (see Section 3.3 below).

Table 5 Overview of the percentage of measurements above the limit of quantification (LOQ), the weighted 5th percentile (P05), median (P50), and 95th percentile (P95) for PFAS in blood serum (µg/L) for the Dutch adult population (N=967), for both the lower bound and upper bound treatment of left censored data.

PFAS	LOD	LOQ	% > LOQ[a]	Lower bound (µg/L)			Upper bound (µg/L)		
				P05	P50	P95	P05	P50	P95
T-PFOA[b]	0.1[e]	0.1[e]	100[f]	0.36	0.85	2.0	0.46	0.95	2.1
PFNA	0.1	0.1	91	0	0.18	0.40	0.10	0.18	0.40
PFDA	0.1	0.1	37	0	0	0.21	0.10	0.10	0.21
PFUnDA	0.1	0.1	12	0	0	0.11	0.10	0.10	0.11
T-PFHxS[c]	0.1[e]	0.1[e]	99[f]	0.15	0.39	1.1	0.25	0.49	1.2
T-PFOS[d]	0.1[e]	0.1[e]	100	0.8	1.7	4.7	0.8	1.7	4.7

LOD: Limit of detection, LOQ: Limit of quantification, PFAS: per- and polyfluoroalkyl substances
[a] The percentage of serum samples above the LOQ of 0.1 ng/mL
[b] T-PFOA concentrations were obtained by summing branched and linear PFOA concentrations
[c] T-PFHxS concentrations were obtained by summing branched and linear PFHxS concentrations
[d] T-PFOS concentrations were obtained by summing branched and linear PFOS concentrations
[e] The LOD and LOQ for both the branched and linear isomer was 0.1 ng/mL
[f] The presented percentage is of the linear isomer, which was more often detected than the branched isomer

3.3 Comparison of observed and predicted PFAS serum concentrations

PFAS concentrations in serum were predicted from daily dietary exposure estimates and kinetic modelling. These predicted concentrations are compared to those observed in the blood donor study.

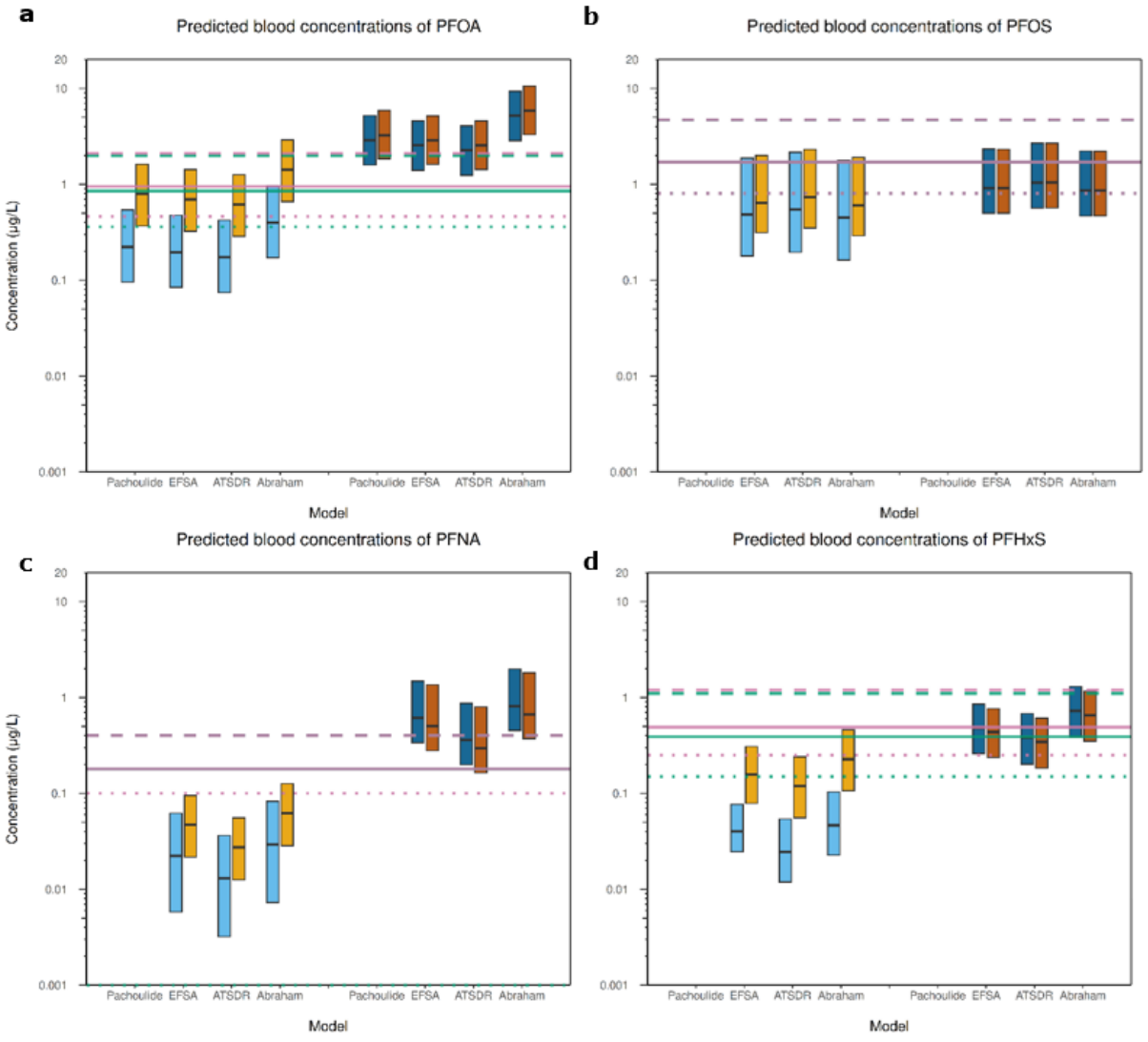
Figure 2 shows the observed and predicted PFAS concentrations for PFOA, PFOS, PFNA, PFHxS, PFDA and PFUnDA. Generally, the predicted PFAS serum concentrations for the LB scenarios are lower than the observed serum concentrations (Figure 2, Appendix 1 and Appendix 2). For PFOA, PFNA and PFHxS, the predicted serum concentrations based on the P50 exposure from food and drinking water in the GW-LB scenario were up to 20 times lower than the measured P50. The difference was smaller for the predicted serum concentrations in the SI-LB scenario, but the predicted values also remained below the measured serum concentrations. For the UB scenarios, the predicted serum concentrations are generally higher than the observed serum concentrations, except for PFOS and PFHxS. For PFOA, PFNA, PFDA and PFUnDA, the predicted serum concentrations based on the P50 exposure from food and drinking water in the UB scenarios were up to 7 times higher than the measured P50. For PFOS, the predicted P95 values are close to the observed P50 values. For PFHxS, instead of higher predicted concentrations, the predictions for the UB scenarios generally lie between the P05 and P95 of the observed serum concentrations. For PFDA and PFUnDA, many measured concentrations were equal to or below the LOQ. For PFUnDA, the predictions are generally higher than the observed concentrations, including the UB P95 observed.

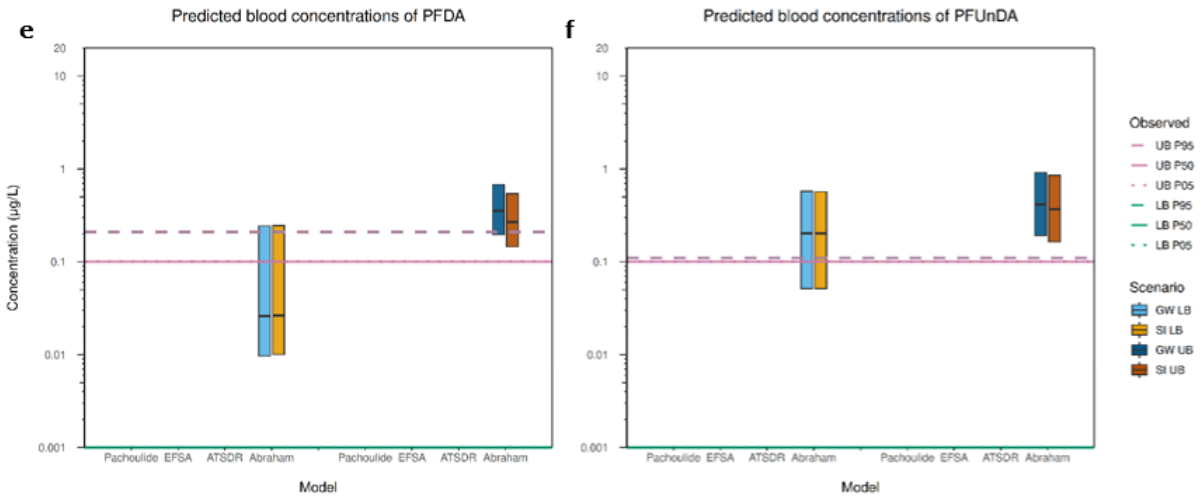
The relative variability across models is PFAS-specific. For PFOS, the predictions are relatively similar, whereas the differences between models are larger for PFOA, PFNA and PFHxS (Appendix 3C). In general, the kinetic models of Abraham et al. (2024) resulted in the highest predicted serum concentrations, whereas the kinetic models of ATSDR (2021) resulted in the lowest predicted serum concentrations (Figure 2, Appendix 1).

Regarding the scenarios, the SI scenario results in higher predicted serum concentrations than the GW scenario in the LB scenario, mainly for PFOA, PFOS, PFNA and PFHxS. On the other hand, in the UB scenario the opposite is seen for PFNA, PFHxS, PFDA and PFUnDA, indicating that for these four PFAS the number of values below the LOQ is higher in the LB scenario compared to the UB scenario. The differences between the GW and SI scenario are smaller in the UB scenario compared to the LB scenario.

The difference between the models is smaller than the difference between the predicted and the observed concentration (Table 6, Appendix 3). Thus, the model choice does not seem to be the driving factor behind the discrepancy between the model predictions and the observations.

Figure 2 Observed (lines) and predicted (boxplots) serum concentrations (in µg/L) of a) PFOA, b) PFOS, c) PFNA, d) PFHxS, e) PFDA and f) PFUnDA, based on four exposure scenarios and up to four kinetic models. The boxplots represent the P05, P50 and P95 of the predicted average serum concentrations between 18 and 79 years of age, derived from daily external dietary exposure estimates. The black line inside the box represents the median. The exposure scenarios distinguish between lower bound (LB, light boxplots, left) and upper bound (UB, dark boxplots, right) estimates, and between drinking water derived from groundwater (GW, blue boxplots) or surface and infiltration (SI, orange boxplots) water. The four kinetic models are represented on the horizontal axis for the LB and UB scenarios separately. Observed PFAS serum concentrations from the blood donor study are shown as lines, with solid lines for P50, dashed lines for P95 and dotted lines for P05. For each percentile, UB values are depicted in red and LB values in green. In some cases, the UB and LB lines overlap, making them visually indistinguishable.




e
Predicted blood concentrations of PFDA
f
Predicted blood concentrations of PFUnDA
Concentration (µg/L)
20
10
1
0.1
0.01
0.001
Pachoulide EFSA ATSDR Abraham Pachoulide EFSA ATSDR Abraham
Model
Observed
UB P95
UB P50
UB P05
LB P95
LB P50
LB P05
Scenario
GW LB
SI LB
GW UB
SI UB

4 Discussion

The aim of this project was to compare external dietary exposure and internal exposure to PFAS in the adult Dutch population and to evaluate whether dietary exposure is the main source of exposure to PFAS. The results indicate that the predicted serum concentrations, relating to dietary sources, are generally lower than the observed internal serum concentrations for the LB scenarios, whereas they are generally higher for the UB scenarios, except for PFOS and PFHxS. However, in general, predicted serum concentrations were of the same order of magnitude as the observed concentrations. Based on the current analysis, it can be concluded that dietary exposure via food and drinking water is an important pathway for exposure in the adult Dutch population. However, food and drinking water cannot explain the total internal PFAS exposure, especially for PFOS. There are several aspects in the analysis that complicate drawing a firm, quantitative conclusion that food and drinking water are the main sources of exposure. These are discussed in more detail below.

4.1 External exposure estimations

The data, method and resulting uncertainties as described by Schepens et al. (2023) to calculate the external dietary exposure also apply to this study. These include uncertainties in the concentration data, allocation of PFAS concentrations to consumed foods, model used to calculate the exposure, effect of processing and food consumption data.

In the food and drinking water samples, there was a large number of non-detects (< LOD) and non-quantified concentrations (< LOQ) that impacted calculation of the daily dietary exposure to PFAS (Schepens et al., 2023). Therefore, both a LB and UB scenario were used to estimate external dietary exposure, where the LB scenario is expected to be an underestimation of the actual exposure and the UB scenario an overestimation. Because of the high number of non-detects, the difference between the LB and UB scenarios is substantial in these cases.

Also, not all food products that possibly contained PFAS were analysed. In that case PFAS concentrations were assigned based on food product similarity (e.g. oranges to citrus fruit to fruit). Additionally, the Dutch food conversion model was used to estimate PFAS concentrations in composite foods and processed counterparts of the food products (Schepens et al., 2023). Thereby, more than 99% of the consumed amount reported in the DNFCS was considered in the exposure assessment. Although under- or overestimation of PFAS concentrations in the non-sampled products cannot be ruled out, Schepens et al. expected that this uncertainty is minimal due to the diverse range of non-sampled food products.

Another uncertainty in the external exposure estimation could arise from the Observed Individual Mean (OIM), which was used to calculate long-term exposure to PFAS. Due to the limited duration of the dietary survey

(i.e. two days), the OIM model tends to overestimate the high (P95) exposure, while the mean and median exposure are considered to be representative of the actual exposure (Boon and Van der Voet, 2015).

Limited and inconsistent data on processing mean that changes in PFAS concentrations during food preparation and processing may have resulted in either over- or underestimation of exposure (Schepens et al., 2023). Similarly, potential PFAS migration from food contact materials was not included in the exposure assessment. However, EFSA considers the contribution of migration to overall exposure to be small (EFSA, 2020).

The Dutch food conversion data from 2012-2016 were used for the conversion instead of the more recent data available from 2019-2021. The more recent data reflect the dietary changes that have occurred since 2016, e.g. people are eating more fruits and vegetables, and unsalted nuts, but less fish, and less red and processed meat. To determine the effect of these changes in the diet on exposure to PFAS, a new exposure estimation is needed.

Schepens et al. (2023) conclude that given the uncertainties and assumptions of the assessment, the LB scenario is an underestimation of the real exposure, but closer to the actual exposure than the UB scenario.

4.2 Predicted serum concentrations resulting from dietary exposure

Across the different kinetic models, predicted serum concentrations varied slightly, with predicted median values varying up to 2.3-fold. The difference in model outcomes was marginal for PFOS. For PFOA, PFNA and PFHxS, the kinetic model based on Abraham et al. (2024) simulated higher serum concentrations compared to the other one-compartment model by ATSDR (2021), and the two PBK models by Pachoulide et al. (2026) and EFSA (2020). For PFDA and PFUnDA, only the kinetic model based on Abraham et al. (2024) was available.

The elimination half-life and the volume of distribution are important parameters to predict serum concentrations with a one-compartment kinetic model, and therefore comparison of this parameter input is critical for interpreting the differences in predictions between the one-compartment kinetic models based on Abraham et al. (2024) and ATSDR (2021). The information in Abraham et al. (2024) is derived from a single individual, a 67-year-old male subject, whereas in ATSDR (2021) the elimination half-lives were obtained in different populations (Table 2). For PFOA and PFNA, the elimination half-lives reported by Abraham et al. (2024) were longer than those observed in a mixed group of fluorochemical production workers (n = 26, 2 females) (Olsen et al., 2007) and in a group of women of reproductive age (Zheng et al., 2013), respectively. Conversely, the elimination half-lives for PFOS and PFHxS were substantially lower in Abraham et al. (2024) compared to those used by the ATSDR (2021) (Table 2). With the use of the serum elimination rate parameter fitted for the one-compartment model in Abraham et al. (2024) the elimination half-lives for PFOA and PFNA are more comparable (1076 and 892 days, respectively) to those used by

the ATSDR (2021). However, for PFOS and PFHxS, the estimated elimination half-lives become even lower (909 and 654 days, respectively) and deviate more from those used by the ATSDR (2021). Note that the visual inspection of the fits for the one-compartment model in Abraham et al. (2024) showed a clear overestimation of the elimination rate and thus an underestimation of the elimination half-lives.

The volume of distribution, which also has a large impact on serum concentration simulations, was generally smaller in Abraham et al. (2024) than in ATSDR (2021) (Table 2). A larger volume of distribution generally results in lower serum concentrations due to more extensive distribution to peripheral tissues. For PFHxS, ATSDR (2021) used a substantially larger volume of distribution than that reported in Abraham et al. (2024). Despite the longer half-life for PFHxS used by ATSDR (2021), the predicted serum concentrations were lower in the one-compartment model of ATSDR (2021) than in the model based on Abraham et al. (2024), consistent with the expectation that a larger volume of distribution results in lower serum concentrations.

Table 2 Elimination half-lives and volumes of distribution that served as input for TK modelling.

PFAS	Model	$T_{1/2}$ (mean and 95% confidence interval) (days)	Population	Reference	Vd (mL/kg bw)	Species, population	Reference
PFOA	Abraham et al. (2024)	2011 (1466-3206)	Male (n = 1), age 67	Abraham et al. (2024)	121	Male (n = 1), age 67	Abraham et al. (2024)
	ATSDR (2021)	1400 (1130-1610)	Fluorochemical production workers (24 males, 2 females)	Olsen et al. (2007)	200	Female non-human primates	Butenhoff et al. (2004)
PFOS	Abraham et al. (2024)	1211 (927-NA)	Male (n = 1), age 67	Abraham et al. (2024)	152	Male (n = 1), age 67	Abraham et al. (2024)
	ATSDR (2021)	2000 (1420-2520)	Fluorochemical production workers (24 males, 2 females)	Olsen et al. (2007)	200	Male non-human primates	Chang et al. (2012)
PFNA	Abraham et al. (2024)	1305 (1054-1716)	Male (n = 1), age 67	Abraham et al. (2024)	124	Male (n = 1), age 67	Abraham et al. (2024)
	ATSDR (2021)	900 (140-2810)*	Young females (age ≤50 years, n=20)	Zhang et al. (2013)	200	Assumed similar to PFOA	-
PFHxS	Abraham et al. (2024)	1634 (1166-2733)	Male (n = 1), age 67	Abraham et al. (2024)	125	Male (n = 1), age 67	Abraham et al. (2024)
	ATSDR (2021)	3100 (2340-3870)	Fluorochemical production workers (24 males, 2 females)	Olsen et al. (2007)	287	Male Cynomolgus monkeys	Sundström et al. (2012)

* Values in between brackets represent the minimum and maximum values rather than the 95% confidence interval

The kinetic models of Pachoulide et al. (2026) and EFSA (2020) did not produce predictions that substantially differed from the simpler one-compartment toxicokinetic model predictions of ATSDR (2021). This illustrates that, for the purpose of predicting serum concentrations of the selected PFAS upon chronic exposure in the adult population, one-compartment models can function equally well as whole-body PBK models, whereas the latter require substantially more information for model parametrization. However, it should be noted that *in vivo* data were necessary for the model parameters of all kinetic models, except the mechanistic kinetic model of Pachoulide et al. (2026), of which the parameters are solely based on *in vitro* and *in silico* data. Furthermore, for other purposes, such as modelling prenatal exposure, modelling the elimination through breastfeeding or modelling specific target tissue concentrations or subpopulations, the added value of more elaborate models is quite evident.

Although EFSA used the kinetic models for PFOA and PFOS to model the concentrations of PFNA and PFHxS, respectively (EFSA, 2020), the predicted serum concentrations were similar to those obtained with the kinetic models of Abraham et al. (2024) and ATSDR (2021). Therefore, the assumption of equal kinetics between PFNA and PFOA, and between PFHxS and PFOS, seems to be valid and yields predictions that are comparable to those obtained using substance-specific toxicokinetic information.

It should be noted that the kinetic models included in the current report used point estimates as parameter input for the model predictions. The toxicokinetic information that serves to parametrize these models is commonly variable, provided that the sample size allows quantification of this variability. For instance, while the arithmetic mean (AM) elimination half-life for PFOA was set at ~1400 days (equal to 3.8 years) in the one-compartment model of the ATSDR (2021), the 95% confidence interval of this parameter was in fact 3.1-4.4 years (Olsen et al., 2007). Furthermore, we simulated the change in bodyweight over life by means of a function that represents the average adult, whereas the variability in bodyweights for the entire population is evident. Such uncertainties and variabilities are ideally taken into account when predicting serum concentrations in the Dutch general adult population resulting from dietary exposure.

In addition to uncertainties in the kinetic models, PFAS precursors were not measured in food and drinking water. PFAS precursors are PFAS that may transform through metabolic or environmental processes into more persistent PFAS, such as the PFAS in this study. Although a recent RIVM report concluded that there is insufficient knowledge about the conversion of precursors in humans to set relative potency factors, literature suggests conversion and conjugation does occur (Bokkers et al., 2026). In addition, PFAS-precursors have been observed in blood of the Dutch population (Bil et al., 2025). Therefore, part of the difference observed between the predicted and observed PFAS concentrations, might be caused by the presence of precursors in food.

4.3 Observed serum concentrations

Blood serum concentrations used in this study were derived from a study in blood donors in the Netherlands (Sprong et al., 2026), and therefore the uncertainties in the serum concentrations as described by Sprong et al. also apply here.

For PFDA and PFUnDA, the detection frequency in the blood of the test subjects was much lower than for the other PFAS (37% and 12%, respectively). However, the contribution of these PFAS to the exposure through food and drinking water is large (Schepens et al., 2023), resulting in a higher predicted serum concentration than observed in some cases. Applying a more sensitive analytical method with a lower LOD and LOQ would likely have resulted in a smaller difference between the LB and UB scenarios for these two substances, thereby facilitating the interpretation of these results in the context of the other uncertainties.

Participants of the blood donor study came from all regions in the country and were between 18 and 77 years of age. However, the blood donor study was not designed to draw a representative sample of the Dutch population. Although the data were weighted for some factors that were overrepresented, such as higher educational level, Dutch origin, having a healthier lifestyle and being healthier in general (Atsma et al., 2011; Timmer et al., 2019), the potential impact of selection bias on the study outcomes cannot be ruled out (Sprong et al., 2026).

Lastly, while the impact is expected to be limited, donating whole blood is an elimination route of PFAS from the human body (Gasiorowski et al., 2022). As a consequence, the PFAS concentrations observed in the blood donor study may have been an underestimation of the PFAS concentrations in a representative Dutch population.

4.4 Comparison of predicted and observed serum concentrations

For the prediction of the serum concentrations resulting from dietary PFAS exposure, a distinction was made between the use of GW and SI as drinking water. For the blood donor study a distinction on the type of drinking water the participants were mainly exposed was not made. Therefore, no distinction by drinking water type was made in the measured serum concentrations. Including this distinction would improve the comparison between the predicted and observed PFAS concentrations.

The blood donor study was conducted in 2025, and therefore is a recent reflection of internal exposure. The study from Schepens et al. (2023) is based on food samples from 2021 and 2022. As the regulatory landscape for PFAS moves so quickly, several measures were taken between 2022-2025 that could have influenced predicted versus observed serum concentrations between this period. For instance, in 2023, maximum limits for PFOA, PFNA, PFHxS, and PFOS came into force for several food categories that substantially contribute to the daily exposure estimates as presented in Table 3, such as fish, meat, and eggs. However, levels of these PFAS in the Netherlands were already

below these limits. Therefore, these regulations likely did not affect PFAS concentrations in these products.

Since the beginning of the 21st century, various measures have been taken to reduce the use of different PFAS and their emission to the environment. This has led for example to lower blood concentrations of PFOS, as observed in both a French (Frenoy et al., 2024) and a German study (Göckener et al., 2010), indicating that the exposure to PFOS was higher in the past than it is now. Although it is unclear whether this reduction is predominantly caused by a reduction of PFOS in foods and/or drinking water, it is likely that dietary PFOS exposure is also reduced.

The predicted serum concentrations represent estimated lifetime exposure to PFAS. For all kinetic models it is assumed that the daily exposure was constant throughout life, and the same as measured in 2021-2022 in the food and drinking water samples. However, we know that historically internal PFAS exposure was higher in Europe as no restrictions were in place yet (Dassuncao et al., 2018). The PFAS included in this study have a long elimination half-life and are bioaccumulative. Therefore, the predicted lifetime serum concentrations resulting from dietary exposure are likely underestimated, as they do not account for exposure that occurred prior to 2021-2025. This could partially explain why the predicted PFAS serum concentrations are generally lower than the observed serum concentrations for the LB scenarios.

An important limitation of the current study is that no information on dietary exposure from the participants in the blood donor study was available and, therefore, no comparison between their external dietary exposure and internal exposure could be obtained. Therefore, it was not possible to assess the individual contribution of food and drinking water to overall internal exposure. Instead, the comparison was made at the population level.

Other sources of exposure
Another likely explanation for the discrepancies between the external dietary and internal PFAS exposure are exposure sources other than food and drinking water. Affourtit et al. (2026) mapped and prioritized PFAS sources other than food and drinking water, although not including exposure through work or industrial emissions. Their overview and qualitative assessment indicated textiles, impregnation or lubricant sprays, personal care products, house dust and indoor air as potentially important sources of PFAS exposure. However, it was concluded that further steps are required to obtain a comprehensive overview of PFAS exposure from these sources. The main limitation at this stage is the absence of realistic quantitative exposure estimates from non-dietary sources, which means that these sources could not be included in the current predictions of PFAS blood concentrations. Hence, the relative contributions of these various sources could not be taken into account.

Other studies, performed outside of the Netherlands, also show that people are exposed to PFAS from sources other than food and drinking water. Although, on average, dietary exposure seems to have the

highest contribution to PFAS exposure, large individual differences have been observed (Poothong et al., 2020; Husøy et al., 2023; Feng et al., 2021; Wang et al., 2021). These differences were most pronounced for the individuals with the highest estimated PFAS exposure (Poothong et al., 2020). Poothong et al. (2020) reported that, in some individuals, house dust and indoor air contributed more to PFAS exposure than food and drinks, whereas Husøy et al. (2023) showed that, for some individuals, PFOA exposure from personal care products can exceed exposure from diet.

5 Recommendations

We recommend to improve and update PFAS concentration data in consumer products, dust and indoor air. In addition, more information is needed about the exposure to these sources, for example the use of consumer products. Further refinement of the exposure assessment, particularly regarding the inclusion of non-dietary pathways and historical baseline exposure, is needed to achieve a comprehensive overview of aggregate PFAS exposure and to identify the main contributing sources of exposure.

Additionally, future HBM studies should collect more detailed information on participants (e.g. body weight, diet and other relevant lifestyle factors), enabling more specific external exposure analyses, in combination with HBM measurements in the same individuals. Ideally, PFAS levels are measured in the consumed food items. Another option would be that participants maintain a food diary so their consumption is known. Such information is needed to validate (existing) kinetic models. In addition, these data could facilitate developing new kinetic models for specific PFAS which currently have no kinetic model yet, or extend existing models with other (e.g. inhalation and dermal) routes of exposure. Therefore, we recommend the development of new PBK models for PFAS with multiple routes of exposure to better represent PFAS aggregate exposure.

A national human biomonitoring (HBM) programme could play an important role in this effort by systematically collecting both biomonitoring data and detailed exposure-related information from a representative sample of the population.

6 Conclusion

The findings of this project support the conclusion that dietary exposure via food and drinking water is an important pathway for PFAS exposure in the adult Dutch population. However, food and drinking water cannot explain the total internal PFAS exposure, especially for PFOS. In general, predicted serum concentrations were of the same order of magnitude as the observed concentrations, which is consistent with previous European work indicating diet as a key contributor to PFAS body burdens. At the same time, the results do not allow a firm, quantitative conclusion that food and drinking water are the main sources of exposure.

The main limitations hampering such a conclusion include the concentration data for food and drinking water, differences and assumptions in kinetic models (especially half-lives and historical exposure), analytical constraints and selection bias in biomonitoring data, and the absence of quantitative estimates for non-dietary sources. Addressing these limitations and conducting future studies combining detailed participant information with HBM data, ideally including individual dietary records and measurements of PFAS in consumed foods, would enable more robust conclusions on the contribution of food and drinking water in relation to other sources of PFAS exposure.

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Appendix 1 Observed and Predicted Serum PFAS Concentrations for Multiple Exposure Scenarios and Kinetic Models

Table A1.1 Observed and predicted PFAS concentrations in serum (µg/L) based on 4 different exposure scenarios and a maximum of 4 different kinetic models.

PFAS	**Scenario**	**Model**	**Observed concentration (µg/L)**						**Predicted concentration (µg/L)**		
			P05 LB	**P50 LB**	**P95 LB**	**P05 UB**	**P50 UB**	**P95 UB**	**P05**	**P50**	**P95**
PFDA	GW LB	Pachoulide	0	0	0.21	0.10	0.10	0.21	N.A.	N.A.	N.A.
PFDA	GW LB	EFSA							N.A.	N.A.	N.A.
PFDA	GW LB	ATSDR							N.A.	N.A.	N.A.
PFDA	GW LB	Abraham							0.010	0.026	0.24
PFDA	GW UB	Pachoulide							N.A.	N.A.	N.A.
PFDA	GW UB	EFSA							N.A.	N.A.	N.A.
PFDA	GW UB	ATSDR							N.A.	N.A.	N.A.
PFDA	GW UB	Abraham							0.20	0.36	0.68
PFDA	SI LB	Pachoulide							N.A.	N.A.	N.A.
PFDA	SI LB	EFSA							N.A.	N.A.	N.A.
PFDA	SI LB	ATSDR							N.A.	N.A.	N.A.
PFDA	SI LB	Abraham							0.010	0.026	0.25
PFDA	SI UB	Pachoulide							N.A.	N.A.	N.A.
PFDA	SI UB	EFSA							N.A.	N.A.	N.A.
PFDA	SI UB	ATSDR							N.A.	N.A.	N.A.
PFDA	SI UB	Abraham							0.15	0.27	0.54
PFHxS	GW LB	Pachoulide	0.15	0.39	1.1	0.25	0.49	1.2	N.A.	N.A.	N.A.
PFHxS	GW LB	EFSA							0.025	0.040	0.077
PFHxS	GW LB	ATSDR							0.012	0.024	0.054
PFHxS	GW LB	Abraham							0.023	0.047	0.10

PFAS	Scenario	Model	Observed concentration (µg/L)						Predicted concentration (µg/L)		
			P05 LB	P50 LB	P95 LB	P05 UB	P50 UB	P95 UB	P05	P50	P95
PFHxS	GW UB	Pachoulide							N.A.	N.A.	N.A.
PFHxS	GW UB	EFSA							0.26	0.48	0.85
PFHxS	GW UB	ATSDR							0.20	0.38	0.68
PFHxS	GW UB	Abraham							0.39	0.73	1.3
PFHxS	SI LB	Pachoulide							N.A.	N.A.	N.A.
PFHxS	SI LB	EFSA							0.079	0.16	0.31
PFHxS	SI LB	ATSDR							0.056	0.12	0.24
PFHxS	SI LB	Abraham							0.11	0.23	0.46
PFHxS	SI UB	Pachoulide							N.A.	N.A.	N.A.
PFHxS	SI UB	EFSA							0.24	0.44	0.76
PFHxS	SI UB	ATSDR							0.18	0.34	0.61
PFHxS	SI UB	Abraham							0.35	0.66	1.2
PFNA	GW LB	Pachoulide							N.A.	N.A.	N.A.
PFNA	GW LB	EFSA							0.006	0.022	0.062
PFNA	GW LB	ATSDR							0.003	0.013	0.036
PFNA	GW LB	Abraham							0.007	0.029	0.083
PFNA	GW UB	Pachoulide							N.A.	N.A.	N.A.
PFNA	GW UB	EFSA							0.34	0.61	1.5
PFNA	GW UB	ATSDR							0.20	0.36	0.87
PFNA	GW UB	Abraham	0	0.18	0.40	0.10	0.18	0.40	0.45	0.82	2.0
PFNA	SI LB	Pachoulide							N.A.	N.A.	N.A.
PFNA	SI LB	EFSA							0.022	0.047	0.095
PFNA	SI LB	ATSDR							0.013	0.027	0.055
PFNA	SI LB	Abraham							0.028	0.062	0.13
PFNA	SI UB	Pachoulide							N.A.	N.A.	N.A.
PFNA	SI UB	EFSA							0.28	0.50	1.4
PFNA	SI UB	ATSDR							0.16	0.29	0.80

PFAS	Scenario	Model	Observed concentration (µg/L)						Predicted concentration (µg/L)		
			P05 LB	P50 LB	P95 LB	P05 UB	P50 UB	P95 UB	P05	P50	P95
PFNA	SI UB	Abraham							0.37	0.67	1.8
PFOA	GW LB	Pachoulide	0.36	0.85	2.0	0.46	0.95	2.1	0.095	0.22	0.54
PFOA	GW LB	EFSA							0.084	0.20	0.48
PFOA	GW LB	ATSDR							0.074	0.17	0.42
PFOA	GW LB	Abraham							0.17	0.40	0.97
PFOA	GW UB	Pachoulide							1.6	2.9	5.2
PFOA	GW UB	EFSA							1.4	2.5	4.6
PFOA	GW UB	ATSDR							1.2	2.3	4.1
PFOA	GW UB	Abraham							2.9	5.2	9.4
PFOA	SI LB	Pachoulide							0.37	0.79	1.6
PFOA	SI LB	EFSA							0.32	0.70	1.4
PFOA	SI LB	ATSDR							0.29	0.62	1.3
PFOA	SI LB	Abraham							0.66	1.4	2.9
PFOA	SI UB	Pachoulide							1.8	3.3	5.9
PFOA	SI UB	EFSA							1.6	2.9	5.2
PFOA	SI UB	ATSDR							1.4	2.5	4.6
PFOA	SI UB	Abraham							3.3	5.8	10.6
PFOS	GW LB	Pachoulide	0.8	1.7	4.7	0.8	1.7	4.7	N.A.	N.A.	N.A.
PFOS	GW LB	EFSA							0.18	0.48	1.9
PFOS	GW LB	ATSDR							0.20	0.55	2.2
PFOS	GW LB	Abraham							0.16	0.45	1.8
PFOS	GW UB	Pachoulide							N.A.	N.A.	N.A.
PFOS	GW UB	EFSA							0.50	0.91	2.3
PFOS	GW UB	ATSDR							0.57	1.0	2.7
PFOS	GW UB	Abraham							0.47	0.86	2.2
PFOS	SI LB	Pachoulide							N.A.	N.A.	N.A.
PFOS	SI LB	EFSA							0.31	0.64	2.0

PFAS	Scenario	Model	Observed concentration (µg/L)						Predicted concentration (µg/L)		
			P05 LB	P50 LB	P95 LB	P05 UB	P50 UB	P95 UB	P05	P50	P95
PFOS	SI LB	ATSDR							0.35	0.73	2.3
PFOS	SI LB	Abraham							0.29	0.61	1.9
PFOS	SI UB	Pachoulide							N.A.	N.A.	N.A.
PFOS	SI UB	EFSA							0.50	0.91	2.3
PFOS	SI UB	ATSDR							0.57	1.0	2.7
PFOS	SI UB	Abraham							0.47	0.86	2.2
PFUnDA	GW LB	Pachoulide							N.A.	N.A.	N.A.
PFUnDA	GW LB	EFSA							N.A.	N.A.	N.A.
PFUnDA	GW LB	ATSDR							N.A.	N.A.	N.A.
PFUnDA	GW LB	Abraham							0.051	0.20	0.57
PFUnDA	GW UB	Pachoulide							N.A.	N.A.	N.A.
PFUnDA	GW UB	EFSA							N.A.	N.A.	N.A.
PFUnDA	GW UB	ATSDR							N.A.	N.A.	N.A.
PFUnDA	GW UB	Abraham	0	0	0.11	0.10	0.10	0.11	0.19	0.41	0.91
PFUnDA	SI LB	Pachoulide							N.A.	N.A.	N.A.
PFUnDA	SI LB	EFSA							N.A.	N.A.	N.A.
PFUnDA	SI LB	ATSDR							N.A.	N.A.	N.A.
PFUnDA	SI LB	Abraham							0.051	0.20	0.57
PFUnDA	SI UB	Pachoulide							N.A.	N.A.	N.A.
PFUnDA	SI UB	EFSA							N.A.	N.A.	N.A.
PFUnDA	SI UB	ATSDR							N.A.	N.A.	N.A.
PFUnDA	SI UB	Abraham							0.16	0.37	0.85

Appendix 2 Ratio between predicted and observed concentrations

Table A2.1 Ratio predicted vs observed concentrations. Values <1 indicate that predicted concentrations were lower than the observed concentrations. Values >1 indicate that predicted concentrations were higher than the observed concentrations

PFAS	Scenario	Ratio predicted vs observed LB	Ratio predicted vs observed UB	Average ratio predicted vs observed (LB and UB)
PFOA	GW LB	0.3	0.3	0.3
	SI LB	1.0	0.9	1.0
	GW UB	3.9	3.3	3.6
	SI UB	4.4	3.8	4.1
	All	2.4	2.1	2.2
PFOS	GW LB	0.3	0.3	0.3
	SI LB	0.4	0.4	0.4
	GW UB	0.6	0.6	0.6
	SI UB	0.6	0.6	0.6
	All	0.5	0.5	0.5
PFNA	GW LB	0.1	0.1	0.1
	SI LB	0.2	0.2	0.2
	GW UB	3.5	3.4	3.4
	SI UB	3.0	2.9	3.0
	All	1.7	1.7	1.7
PFHxS	GW LB	0.1	0.1	0.1
	SI LB	0.4	0.3	0.4
	GW UB	1.4	1.0	1.2
	SI UB	1.2	0.9	1.1
	All	0.8	0.6	0.7
PFDA	GW LB	1.2	0.5	0.8
	SI LB	1.2	0.5	0.8
	GW UB	3.2	2.9	3.1
	SI UB	2.6	2.2	2.4
	All	2.0	1.5	1.7
PFUnDA	GW LB	5.2	2.6	3.9
	SI LB	5.2	2.6	3.9
	GW UB	8.3	4.8	6.5
	SI UB	7.7	4.4	6.1
	All	6.6	3.6	4.3

Appendix 3 Difference between the predicted and observed concentrations and between models

Table A3.1 Ratio of the difference between predicted and observed concentrations. The ratio of observed to predicted concentrations was calculated for the P05, P50 and P95 of the lower and upper bound measured concentrations across all exposure scenarios. The goal was to identify the difference between predicted and observed concentrations, independent of the direction of difference. To prevent values above and below 1 cancelling each other out, the higher value was always divided by the lower one. If the observed concentration was 0 (i.e. below the LOQ in the lower-bound scenario), no ratio was calculated.

PFAS	Scenario	Ratio predicted vs observed LB	Ratio predicted vs observed UB	Ratio predicted vs observed LB and UB
PFOA	GW LB	3.7	4.3	4.0
	SI LB	1.4	1.4	1.4
	GW UB	3.9	3.3	3.6
	SI UB	4.4	3.8	4.1
	All	3.3	3.2	3.3
PFOS	GW LB	3.5	3.5	3.5
	SI LB	2.5	2.5	2.5
	GW UB	1.8	1.8	1.8
	SI UB	1.8	1.8	1.8
	All	2.4	2.4	2.4
PFNA	GW LB	8.4	12.5	10.4
	SI LB	4.7	4.9	4.8
	GW UB	3.5	3.4	3.4
	SI UB	3.0	2.9	3.0
	All	4.9	5.9	5.5
PFHxS	GW LB	11.6	15.0	13.3
	SI LB	2.7	3.4	3.1
	GW UB	1.5	1.3	1.4
	SI UB	1.5	1.4	1.4
	All	4.3	5.3	4.8
PFDA	GW LB	1.2	5.1	3.1
	SI LB	1.2	5.0	3.1
	GW UB	3.2	2.9	3.1
	SI UB	2.6	2.2	2.4
	All	2.0	3.8	3.4
PFUnDA	GW LB	5.2	3.1	4.1
	SI LB	5.2	3.0	4.1
	GW UB	8.3	4.8	6.5
	SI UB	7.7	4.4	6.1
	All	6.6	3.8	4.5

Table A3.2 Ratio of the difference between models. To assess uncertainty in the kinetic models for each PFAS, the ratios between the predicted P05, P50 and P95 concentrations of each model to the predicted P05, P50 and P95 concentrations of the other models were calculated. The higher value was always divided by the lower value to prevent values above and below 1 cancelling each other out. Since for PFDA and PFUnDA only one model was used, no model uncertainty was determined for these PFAS.

PFAS	Scenario	Pachoulide vs EFSA	Pachoulide vs ATSDR	Pachoulide vs Abrahams	EFSA vs ATSDR	EFSA vs Abrahams	ATSDR vs Abrahams	All models
PFOA	GW LB	1.1	1.3	1.8	1.1	2.0	2.3	1.6
	SI LB	1.1	1.3	1.8	1.1	2.0	2.3	1.6
	GW UB	1.1	1.3	1.8	1.1	2.0	2.3	1.6
	SI UB	1.1	1.3	1.8	1.1	2.0	2.3	1.6
	All	1.1	1.3	1.8	1.1	2.0	2.3	1.6
PFOS	GW LB				1.1	1.1	1.2	1.1
	SI LB				1.1	1.1	1.2	1.1
	GW UB				1.1	1.1	1.2	1.1
	SI UB				1.1	1.1	1.2	1.1
	All				1.1	1.1	1.2	1.1
PFNA	GW LB				1.7	1.3	2.3	1.8
	SI LB				1.7	1.3	2.3	1.8
	GW UB				1.7	1.3	2.3	1.8
	SI UB				1.7	1.3	2.3	1.8
	All				1.7	1.3	2.3	1.8
PFHxS	GW LB				1.7	1.2	1.9	1.6
	SI LB				1.3	1.4	1.9	1.6
	GW UB				1.3	1.5	1.9	1.6
	SI UB				1.3	1.5	1.9	1.6
	All				1.4	1.4	1.9	1.6

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