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Exposure to per- and polyfluoroalkyl substances (PFAS) in North Carolina homes: results from the indoor PFAS assessment (IPA) campaign†

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Per and polyfluoroalkyl substances (PFAS) are ubiquitous in the indoor environment, resulting in indoor exposure. However, a dearth of concurrent indoor multi-compartment PFAS measurements, including air, has limited our understanding of the contributions of each exposure pathway to residential PFAS exposure. As part of the Indoor PFAS Assessment (IPA) Campaign, we measured 35 neutral and ionic PFAS in air, settled dust, drinking water, clothing, and on surfaces in 11 North Carolina homes. Ionic and neutral PFAS measurements reported previously and ionic PFAS measurements reported herein for drinking water (1.4–34.1 ng L^{-1}), dust (202–1036 ng g^{-1}), and surfaces (4.1×10^{-4}–1.7×10^{-2} ng cm^{-2}) were used to conduct a residential indoor PFAS exposure assessment. We considered inhalation of air, ingestion of drinking water and dust, mouthing of clothing (children only), and transdermal uptake from contact with dust, air, and surfaces. Average intake rates were estimated to be 3.6 ng kg^{-1} per day (adults) and 12.4 ng kg^{-1} per day (2 year-old), with neutral PFAS contributing over 80% total PFAS intake. Excluding dietary ingestion, which was not measured, inhalation contributed over 65% of PFAS intake and was dominated by neutral PFAS because fluorotelomer alcohol (FTOH) concentrations in air were several orders of magnitude greater than ionic PFAS concentrations. Perfluorooctanoic acid (PFOA) intake was 6.1×10^{-2} ng kg^{-1} per day (adults) and 1.5×10^{-1} ng kg^{-1} per day (2 year-old), and biotransformation of 8 : 2 FTOH to PFOA increased this PFOA body burden by 14% (adults) and 17% (2 year-old), suggesting inhalation may also be a meaningful contributor to ionic PFAS exposure through biotransformation.

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Environmental significance

Per- and polyfluoroalkyl substances (PFAS) are prevalent in the indoor environment, and several PFAS compounds are associated with adverse health effects. Limited measurements of PFAS from indoor environmental media (*i.e.*, air, clothing, and surfaces) have hindered our ability to conduct data-informed exposure assessments. Using measurements of indoor air (particles and/or gas phase), settled dust, drinking water, surfaces, and clothing from the Indoor PFAS Assessment Campaign in 11 North Carolina homes, we found that inhalation was the major non-dietary residential exposure pathway for the sum of 35 measured PFAS for both adults and children. We highlight the need for more measurements of PFAS in diet and indoor air, as well as improved characterization of the biotransformation and toxicokinetics of PFAS.

Introduction

Per- and polyfluoroalkyl substances (PFAS), which consist of thousands of fluorinated compounds across several compound classes,[1] are ubiquitous in the indoor environment[2,3] because they are present in many consumer products due to their water- and oil-repellency and surfactant-like properties.[4] PFAS have been detected in cookware,[5] carpets,[6,7] clothing,[2,8,9] paints,[10] and in paper/packaging materials.[11,12] Several studies have found that indoor concentrations of PFAS are higher than outdoor concentrations,[13–15] suggesting that while PFAS have been detected in outdoor air[16–18] and residential soil,[19] indoor concentrations are largely driven by emissions from consumer products and materials, as well as occupant activities, particularly for homes away from major sources of environmental contamination.[15] Some PFAS have been associated with negative health effects such as kidney and testicular cancer, immunotoxicity, increased cholesterol, and hepatotoxicity.[3] The most commonly studied PFAS are the perfluoroalkyl acids, or PFAAs,

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which include perfluorooctanoic acid (PFOA) and perfluorooctanesulfonic acid (PFOS). Due to increased concern regarding their toxicity, bioaccumulation, and resistance to degradation, major fluorochemical manufacturers began phasing out PFOA and PFOS in the early 2000s.[20] However, they remain prevalent outdoors[17,18,21] and indoors,[22–24] in part due to precursor compounds that can degrade to form terminal PFAAs.[25] Precursor compounds such as the fluorotelomer alcohols (FTOHs), perfluorooctane sulfonamides (FOSAs), perfluorooctane sulfonamidoethanols (FOSEs), and polyfluoroalkyl phosphate esters (PAPs) are also prevalent in the indoor environment, often at elevated concentrations compared to the PFAAs.[2,23,26–29] The FTOHs and FOSEs/FOSAs are also referred to as neutral PFAS and are more volatile[30] than both the PFAAs and PAPs, which are collectively referred to as ionic PFAS.

Although indoor measurements remain limited, PFAS (neutral and/or ionic) have been detected in dust,[23,27,31–33] air (gas[2,26] and particle-phase[15,34]), surface films,[22,35] cloth,[2,8,36] dryer lint,[24] and drinking water[37–39] collected from homes globally and in North America. The major pathways of exposure to PFAS are generally considered to be ingestion *via* diet,[3,40] settled dust,[23,41] and contaminated drinking water,[42,43] with additional contributions from inhalation of air[44,45] and dermal exposure from contact with dust and clothing.[2,40,46,47] However, most studies use measurements from the literature collected at differing times and locations to conduct exposure assessments,[40,46,48–52] investigate a limited number of environmental compartments,[23,26,41] and/or assess exposure to a limited number of PFAS, typically neglecting the precursor compounds (*e.g.*, FTOHs and PAPs).[48] This hinders our understanding of individual contributions to exposure. Having measurements from multiple environmental compartments from the same homes provides valuable insight into the contribution to exposure from various pathways. Likewise, characterizing exposure to different types of PFAS, particularly PFAA precursors, can improve our understanding of the body burden for these compounds.

There are several challenges with PFAS exposure assessments, including the large number of PFAS compounds, a lack of information on their occurrence/levels in the residential environment, and limited availability of PFAS physicochemical properties and toxicokinetics, particularly for inhalation. Indoor air measurements, in particular, have largely focused on volatile precursors such as the FTOHs, FOSEs, and FOSAs,[2,53–55] with fewer studies analyzing for PFAAs and PAPs.[26,34,56,57] Settled dust[58–61] and water[39,62–65] are commonly analyzed for PFAAs, with a recent study finding that PFAS concentrations in water sources for 6 million people in the United States (US) exceeded the US Environmental Protection Agency's (EPA) lifetime health advisory of 70 ng L^{-1} for PFOA and PFOS.[66] PFAS have been detected in water throughout the US,[39,43,66–68] including the Cape Fear River watershed and other waters in North Carolina (NC).[65,67,69–72] Communities near contaminated water sources tend to have higher blood sera levels of PFAAs compared to the general US population.[20,62] There has been an increased focus on reducing PFAS exposure by reducing ingestion of PFAS in drinking water and diet,[73] with some companies pledging to reduce the use of PFAS in food packaging materials and stricter regulations on allowable levels of PFAS in drinking water.[74] With increasing regulation of PFAS in food and water, other pathways, such as inhalation and dermal exposure, which are less represented in the literature,[46,75] may become more important. Thus, there is a need to characterize their contributions to residential PFAS exposure since humans tend to spend ~90% of their time indoors and ~70% of their time in homes.[76]

Most exposure assessments focus on PFAAs[77] because several PFAAs have well established toxicity data. However, concentrations in some indoor compartments (*i.e.*, air[26] and dust[23,44,56]) are higher for neutral PFAS than for PFAAs. Additionally, some precursors, such as the FTOHs, can biotransform to PFAAs and PAPs with known negative health effects,[78,79] but few studies have assessed the contribution of biotransformed precursor compounds to PFAAs.[25,52] The contributions to total exposure, and subsequent health impacts, for ionic and neutral PFAS are likely to vary depending on the exposure pathway because they have different physicochemical properties (*i.e.*, volatility). Exposure pathways also vary for adults and children due to different physiologic functions and behaviors.[80] Young children spend extended periods of time on or near the floor, close to dust, and also exhibit hand-to-mouth behavior[80] that is less common in older children and adults. Because of their proximity to the floor and settled dust, as well as their propensity to mouth objects and their hands, ingestion of settled dust and chemical residues on surfaces may be a more significant pathway of exposure for children.[49,80,81]

In this study, we present results from the Indoor PFAS Assessment (IPA) Campaign, which was conducted in 11 homes in North Carolina, and provide insights into the contributions of different exposure routes and pathways to PFAS exposure for simulated adults and 2 year-old children using neutral and ionic PFAS measurements. The IPA Campaign is a unique study, in terms of the number of concurrent measurements made in homes across multiple environmental media and analyzed for neutral and/or ionic PFAS. IPA Campaign measurements of PFAS in air,[2] quartz fiber filter (QFF)-collected $PM_{2.5}$,[15] clothing,[2] and neutral PFAS in settled dust (Eichler *et al.*, 2024)[82] have been reported previously. Herein we report (for the first time) concentrations of ionic PFAS measured concurrently in settled dust, on glass slabs, and in drinking water. Few studies have conducted exposure assessments for PFAS considering more than three pathways;[46,50,52] we provide estimates of daily intake (DI) using measured concentrations of ionic and/or neutral PFAS from inhalation (gas and particles), ingestion (settled dust, drinking water, hand-to-mouth transfer after surface contact, and mouthing of clothing), as well as dermal uptake (settled dust, contact with surfaces, and air-to-skin uptake) to characterize the contributions of each exposure pathway to total PFAS exposure in NC homes.

Materials and methods

Indoor PFAS assessment (IPA) campaign

As part of the IPA Campaign (UNC-Chapel Hill IRB# 20-2771), staggered sampling in 11 non-smoking, single-family detached homes located in the Chapel Hill and Durham, NC, region took

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place from July 2021 to May 2022. Eleven homes participated in the study with ten homes completing the study (6–9 months study participation); one home (Home 82) left after one month. Several environmental matrices were sampled multiple times for neutral and/or ionic PFAS; indoor air (gas[2] and particle phase[15]), surface wipes of windows and mounted glass slabs, clothing,[2] dryer lint (Eichler *et al.*, 2024),[82] settled dust from the main living area,[82] heating and air conditioning (HAC) filters,[82] and drinking water. Active air sampling took place three times over 6 days in the main living area of each home. Concentrations of carbon dioxide (CO_2) were logged over the 6 day periods to estimate air change rates (ACH); indoor temperature (T) and indoor relative humidity (RH) were logged throughout the entire campaign in each home. Additional details concerning the study design, surveys, home characteristics, and auxiliary measurements are provided elsewhere.[2]

Chemicals and reagents

IPA Campaign samples were analyzed for the nine neutral PFAS and 26 ionic PFAS listed in Table S1.† Specifically, we measured three FTOHs, two FTACs, two FOSEs, two FOSAs, 13 perfluoroalkyl carboxylic acids (PFCAs), eight perfluoroalkanesulfonic acids (PFSAs), GenX (hexafluoropropylene oxide-dimer acid; HFPO-DA), and four PAPs. These were selected due to their previous detection in an indoor compartment,[23,26,32,83] or their detection in North Carolina environments,[18,65] and the availability of authentic standards. Native and mass-labelled PFAS standards (Wellington Laboratories, Guelph, Ontario, Canada) were used to quantify concentrations (Table S1†). All other solvents and reagents were HPLC grade and purchased from Thermo Fisher Scientific (Waltham, MA, USA).

Sample collection

Drinking water. Drinking water was collected from the kitchen faucet in each home at the 1 month and 6 month visits. One field blank was prepared the day before collecting a drinking water sample. For samples and field blanks, 1 g of HPLC-grade ammonium acetate (97% purity, Fisher Chemical, Fair Lawn, NJ) was added to each 1 L polypropylene (PP) sample bottle to convert free chlorine to chloramine, per EPA Method 533.[84] Field blank bottles were then filled with 1 L of MilliQ water and all bottles were stored in the fridge until transport to the field in a cooler at 4 °C. At the home, kitchen faucets were allowed to run (cold) for 2 min prior to collecting 1 L of tap water. For the three homes that used a water filter, drinking water was collected directly from the filter (*e.g.*, pitcher filter). Both the collected sample and field blank were placed in a cooler, transported back to lab, and then stored at −80 °C until extraction. To assess the effect of storage time on PFAS recovery from water samples, three storage blanks were created by spiking 1 ng of analyte PFAS mix into 1 L of MilliQ water before storing at −80 °C for around 1 year. Storage blanks were handled and processed the same way as samples and other blanks. In total, 10 samples and 10 field blanks were collected. Drinking water from Home 82 was not analyzed because they left the study after 1 month.

Glass slabs. At the first visit, pre-cleaned (three times each with MilliQ water, methanol, and hexane; see section S2†) glass slabs (GS) were mounted vertically on a wall in the living area of each home, away from direct sunlight, except for Home 78, where no glass slabs were installed because the walls had been recently painted. In total, six GS were mounted between 1.2 to 2.3 m above the floor and spaced between 1.3 to 5.1 cm apart. Once mounted, GS were cleaned by wiping with a methanol-wetted Kimwipe at least three times and until there was no visible discoloration on the Kimwipe. Background (t0) wipes were collected after the mounted GS were cleaned. For sampling, GS were wiped three times in a pattern (Fig. S1†) using two methanol-wetted Kimwipes after exposure to room air for ∼6 months. Field blanks were collected in each home by wetting two Kimwipes with methanol, as we did for sample collection, waving them in the air for 2 min, and then storing them in a 50 mL PP tube. Samples and blanks were transported at 4 °C in a cooler to the lab and stored at −80 °C until extraction. In total 31 field blanks and 18 6-month GS samples were collected. GS results will be discussed in more depth in a future paper.

Dust. Settled dust was collected in sampling socks (1–5 socks per sampling event; nylon, 25 μm mesh, 1.75″ × 5″, Dulytek) located in the wand behind the vacuum head. Each main living area was sampled twice: once during the first sampling visit (t0) and again after 6 months (t6), except for Home 82, which was sampled at t0 only. A field blank was collected by installing a sampling sock into the vacuum, removing it, sealing it in pre-baked aluminum foil within a PP zipper bag, and transporting it back to the lab in a cooler. Prior to sampling, participants were asked to refrain from vacuuming for 6 days. In total, 21 dust samples and 22 field blanks were collected. Samples and field blanks were stored at −80 °C until further processing and extraction. Additional details about the dust collection and relevant home characteristics are provided in Table S2† and by Eichler *et al.*, (2024).[82]

Air. Air was collected in the main living area of each home 1 to 3 times. Air sampling for neutral species is detailed in Eichler *et al.* (2023),[2] while details regarding sampling for ionic PFAS is provided in Chang *et al.* (2024).[15] Samples used herein are described in brief here. Air (gas and particle phase) samples for neutral PFAS were collected using polyurethane foam (PUF)-XAD2-PUF sandwich cartridges (ORBO 1500 Precleaned Small PUF/Amberlite XAD-2/PUF Cartridge, Supelco, Bellefonte, PA) for ∼72 h (∼5 L min^{-1}, 21.2 m^3). Air samples (gas and particle phase) for ionic PFAS were collected onto two quartz fiber filters (QFFs) in series downstream of a 2.5 μm impactor in the main living area of each home over 6 days (∼10.5 L min^{-1}, 91.1 m^3). PUF-XAD2-PUF field blanks ($n = 13$) were brought to the field wrapped in pre-baked aluminum foil and sealed in PP zip-lock bags. They were then left in the home near the sampling equipment for the 3 day sampling period. For additional details, see Eichler *et al.* (2023).[2] Details regarding QFF field blanks are described in Chang *et al.* (2024).[15] In brief, field blanks ($n = 10$) were loaded into filter cassettes and inlet heads, removed, and stored in a zip-lock bag along with the loaded inlet heads prior

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to deployment. At the homes, field blanks were exposed to home air for 2 minutes before being placed back into the sealed zip-lock bag and left near the sampling equipment for the entire 6 day sampling period. The QFFs collect particles ($PM_{2.5}$) with >99% collection efficiency; QFFs also adsorb some gases due to their large surface area.[85] Since ionic PFAS are present in both gases and particles,[86,87] the front QFF provides an upper-bound for ionic PFAS in $PM_{2.5}$, and the front plus backup QFF provides a lower-bound for ionic PFAS in both gases and particles. We used the sum of front plus backup QFFs in the exposure assessment.

Clothing. Clothing samples were articles of clothing, such as a t-shirt, made of 100% cotton that had been laundered and stored in participants' homes for at least three months. Clothing was stored in either a drawer or closet and left untouched before being collected for analysis. Household clothing items donated by participants were of differing thicknesses, and an appropriate substrate to use as a field blank was difficult to ascertain. We determined MDLs for clothing items based on the instrument detection limits and we corrected for recoveries. In addition, we analyzed precleaned, standard thickness, 100% cotton cloth strips that had been transported to the field, exposed to home air for 1–2 minutes, and then transported, stored and analyzed with samples to provide an assessment of the potential for clothing sample contamination in transit and storage. Concentrations of the precleaned cloth strips were mostly below detection limits, with the exception of 6 : 2 FTOH (detection frequency; DF = 67%) for which concentrations were still low (0.014 ± 0.015 ng cm^{-2}). Additional details regarding clothing sample collection are provided elsewhere (Eichler *et al.*, 2023).[2]

Sample processing

Drinking water and glass slabs were analyzed for ionic PFAS only, clothing was analyzed only for neutral PFAS, and air and settled dust were analyzed for both. Limitations in extraction and analytical methods meant that not all types of samples could be analyzed for both types of PFAS. Details regarding ionic PFAS sample processing for drinking water, GS, and settled dust are provided below and in section S2.† Sample processing for ionic PFAS on QFFs (Chang *et al.*, 2024)[15] and neutral PFAS in air,[2] clothing,[2] and settled dust (Eichler *et al.*, 2024)[82] are provided elsewhere.

Ionic PFAS. Drinking water samples were extracted (for ionic PFAS only) *via* solid phase extraction (SPE) using Oasis WAX Plus cartridges (60 μm, 225 mg, Waters) following the US EPA method D-EMMD-PHCB-043-SOP-03),[88] with a few differences described in section S2.† Briefly, samples and blanks were spiked with 1 ng of mass-labelled PFAS standards and loaded onto conditioned WAX SPE cartridges. Targeted PFAS were eluted with a 0.1% basic methanol solution and concentrated under the gentle flow of nitrogen to ~25 μL. Around 75 μL of MilliQ water was added to the extract to match the initial mobile phase composition of 75 : 25 (v/v) Milli-Q water and HPLC-grade methanol. A total of 10 samples, 10 field blanks, and 10 lab blanks were analyzed. Lab blanks consisted of 1 L of MilliQ water and remained in the lab.

Glass slabs and QFFs were analyzed for ionic PFAS only. Glass slabs were extracted for ionic PFAS following the method detailed in Zhou *et al.* (2022)[89] with a few modifications. Each GS sample and blank was spiked with 1 ng of a mass-labelled PFAS internal standard mix before extraction in 50 mL PP centrifuge tubes with 20 mL of methanol three times. For QFFs, each filter was spiked with 1 ng of mass-labelled PFAS internal standard and then extracted in 3 mL of methanol *via* 15 min of sonication three times. The supernatants for each sample were combined and then concentrated under the gentle flow of nitrogen (Airgas, Radnor, PA, USA) to ~3–5 mL, filtered (nylon membrane, 13 mm diameter, 0.22 μm pore size, VWR, Radnor, PA), and then further evaporated to 150–300 μL. Extracts were transferred to pre-weighed vials and evaporated to ~25 μL. They were then brought to a final volume of ~100 μL by adding ~75 μL of Milli-Q water.

For dust, large particles such as leaves and hair were removed from dust samples using methanol-cleaned forceps and then the dust was sieved to <500 μm. For ionic PFAS analysis, ~100 mg of sieved dust was placed into a 15 mL PP centrifuge tube and extracted three times in 2 mL of methanol by sonicating for 15 min each time. Approximately 50 mg of ENVI-Carb (Supelclean ENVI-Carb SPE Bulk Packing, Supelco, Bellefonte, PA) was added for cleanup before evaporation to 3–5 mL. Extracts were then centrifuged (9 min, 4500 rpm) and filtered (nylon membrane, 13 mm diameter, 0.2 μm pore size, VWR, Radnor, PA) before concentrating further to 1 mL. Just before analysis, a 25 μL aliquot of extract was combined with 75 μL of MilliQ water to match the initial mobile phase composition. Lab blanks consisted of extracted nylon sampling socks that remained in the lab. Method blanks were handled and processed the same way as other samples and blanks, but without sample media.

Neutral PFAS. Clothing, PUF-XAD2-PUF cartridges, and settled dust samples were spiked with mass-labelled standards and extracted in a 3 : 1 (v/v) hexane/methanol solvent mixture. Extracts were combined and cleaned using ENVI-Carb before being concentrated under nitrogen to ~1000 μL for PUF-XAD2-PUF extracts and ~300–500 μL for clothing and dust extracts. Additional details regarding clothing and PUF-XAD2-PUF sandwiches can be found in Eichler *et al.* (2023)[2] and in Eichler *et al.* (2024)[82] for settled dust.

Analysis and QA/QC

Ionic PFAS analysis and QA/QC. An AB SCIEX Triple Quad™ 6500 ultra-high performance liquid chromatography-electrospray ionization tandem mass spectrometry (UHPLC-ESI-MS/MS) operated in the negative mode with multiple reaction monitoring was used to analyze extracted samples for the 26 ionic PFAS. Samples and blanks were quantified using a 5-point calibration curve (0.2 ng mL^{-1} to 20 ng mL^{-1}). All Teflon tubing within the SCIEX Triple Quad had been previously replaced with PEEK tubing to minimize contamination and a delay column (Zorbax RR Eclipse Plus, C18, 4.6 × 50 mm, 3.5 μm) was installed between the pump and injector. See Zhou *et al.* (2021)[18] and Chang *et al.* (2024)[15] for additional method parameters and operating details. Drinking water, glass slabs,

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settled dust, and QFFs were analyzed for ionic PFAS. Several studies have shown that mimics, like saturated oxo-fatty acids (SOFAs)[90,91] can interfere with peak identification of short-chain PFAAs (*e.g.*, PFBA). However, our method, which uses a 75 : 25 gradient has been shown to improve separation between mimics and targeted analytes,[90] and all peaks were manually verified and the analyte's retention time was checked against the matching internal standard.

Quality assurance and quality control (QA/QC) measures are provided in Chang *et al.* (2024)[15] for QFFs and in section S3† for the other sample types. The analytical detection limit (ADL) ranged from 0.01 to 0.54 ng g^{-1} for dust, 2.1×10^{-3} to 8.0×10^{-2} pg cm^{-2} for surface wipes, 0.002 to 0.074 ng L^{-1} for drinking water, and 0.02 to 0.81 pg m^{-3} for QFFs. Method detection limits (MDLs) ranged from 2×10^{-3} to 0.72 ng L^{-1} for drinking water, 2.1×10^{-3} to 0.85 pg cm^{-2} for glass slabs, from 0.03 to 11.1 ng g^{-1} except for PFOS (37.1 ng g^{-1}), 6 : 2 diPAP (293 ng g^{-1}), 8 : 2 diPAP (79.3 ng g^{-1}), and 6 : 2 monoPAP (69 ng g^{-1}) for dust, and from 0.04 to 0.77 pg m^{-3} for QFFs except for PFHpS and PAPs (0.65–2.49 pg m^{-3}).[15] Analytical precision (expressed as the pooled coefficient of variation of duplicate sample analyses) was within 41% for dust, drinking water, GS, and QFF samples for all targeted ionic PFAS, with the exception of the PAPs. Precision for 6 : 2 diPAP, 8 : 2 diPAP, 6 : 2 monoPAP, and 8 : 2 monoPAP was 70%, 46%, 13%, and 11% for dust, respectively, and from 85% and 204% for 6 : 2 and 8 : 2 diPAPs, respectively on QFFs (monoPAPs were not detected on QFFs).[15] For GS, analytical precision was 43% for 6 : 2 diPAP, 76% for 8 : 2 diPAP, 3% for 6 : 2 monoPAP, and 38% for 8 : 2 monoPAP.

Average recoveries for ionic PFAS in dust were between 52% and 125%, except for 8 : 2 diPAP (172%) and 8 : 2 monoPAP (150%). QFF recoveries were between 78% to 126% except for PFODA (25%) and the PAPs (83–177%). Results for 6 : 2 monoPAP and 8 : 2 diPAP should be interpreted with caution. Drinking water recoveries ranged from 38% (PFODA) to 99% (PFHxS), with higher recoveries for shorter chain-length compounds. Drinking water storage recoveries ranged from 40% (PFDA) to 85% (PFHxS), with the exception of PFNS, PFUnA, PFDS, PFDoA, PFTrA, PFDoS, PFTA, and PFODA, which were below 35% and should be treated with caution. PAPs were excluded from drinking water analysis. Drinking water samples and blanks were corrected for recovery but not for storage loss. Surface wipe recoveries ranged from 41% to 86% for C_4–C_{10} PFCAs and < C_9 PFSAs and were higher for 8 : 2 diPAP (222%). In general, surface wipe recoveries were lower (7% to 66%) for longer chain PFCAs (C_{11}–C_{14}), PFSAs (C_9–C_{12}), as well as the remaining PAPs, and thus should be interpreted with caution. Surface wipe, dust, and QFF concentrations were corrected for recoveries.

Neutral PFAS analysis and QA/QC. Nine neutral PFAS, specifically three FTOHs (6 : 2, 8 : 2, and 10 : 2 FTOH), two fluorotelomer acrylates (8 : 2 and 10 : 2 FTAC), MeFOSA and EtFOSA, as well as MeFOSE and EtFOSE were analyzed for in clothing,[2] air,[2] and settled dust (Eichler *et al.*, 2024).[82] All extracts were quantified using a seven-point calibration curve (0.001 ng μL^{-1} to 1.0 ng uL^{-1}) using selected ion monitoring (SIM) mode using an Agilent 8890 gas chromatograph (GC) with an Agilent DB-WAX column (30 m, 0.25 mm ID, 0.25 μm film thickness) and an Agilent 5977B electron impact (EI) mass-spectrometry detector. Additional details are provided elsewhere.[2,82]

Details regarding QA/QC for neutral species can be found in Eichler *et al.* (2023)[2] for cloth and PUF-XAD2-PUF sandwiches and in Eichler *et al.* (2024)[82] for settled dust. In brief, average recoveries ranged from 84% to 121% for dust, from 74% to 108% for air, and from 30% to 66% for clothing. Precision was better than 23% for all dust, air, and clothing samples, except for EtFOSA (65%) for clothing only.

Exposure assessment

Daily intake rates (ng kg^{-1} per day) were estimated for: inhalation of air (neutral and ionic PFAS), ingestion of settled dust (neutral and ionic PFAS) and drinking water (ionic PFAS), hand-to-mouth behavior after touching surfaces (ionic PFAS) and mouthing of clothing (neutral PFAS) for children, from dermal uptake *via* direct contact with surfaces (ionic PFAS) and settled dust (neutral and ionic PFAS), as well as air-to-skin uptake (neutral PFAS). Airborne ionic PFAS concentrations are the sum of the mean field blank-subtracted front and backup QFF concentrations and are a lower bound estimate for total air concentrations. For air-to-skin uptake, only the FOSEs/FOSAs were considered because PFAA and FTOH uptake through the skin is expected to be minimal.[47] Dermal uptake from clothing was not included because of high uncertainty and unknown parameters.

Equations and parameters such as ingestion and inhalation rates, skin surface area, and hand-to-mouth behavior were sourced from the US EPA's Exposure Factor's Handbook[92] (Table S8†). Dietary intake was not measured in the IPA Campaign. While there is uncertainty around the exposure parameters from mouthing of clothing (*i.e.*, saliva extractability), we provide an estimate of the intake rate through this pathway for children because it is a common behavior, and has been shown to contribute significantly to total exposure for semivolatile (SVOCs) and volatile organic compounds (VOCs) such as phthalates[93] and methamphetamine[94] for this age group.[80] The actual extractability of the neutral PFAS from clothing by saliva is unknown, but because chlorpyrifos has a similar water solubility (3.5×10^{-6} mol L^{-1})[95,96] to the estimated value for 6 : 2 FTOH (4.8×10^{-5} mol L^{-1}),[97] using the same saliva extraction coefficient seems reasonable. An estimate of the biotransformation of 8 : 2 FTOHs to PFOA was calculated using a biotransformation factor of 0.5%, which was measured for 8 : 2 FTOH transformation to PFOA for Sprague-Dawley rats.[79] Biotransformation of 4 : 2 and 6 : 2 FTOH was not considered in this paper because biotransformation factors for these two compounds to ionic PFAS have not been published.

Data processing and statistical analysis

All data processing was conducted in Excel and RStudio 2022.07.02 Build 576 (RStudio, PBC).

Results and discussion

Fig. 1 presents IPA Campaign concentration distributions for sum of ionic PFAS and sum of neutral PFAS (*i.e.*, Σ(ionic PFAS)

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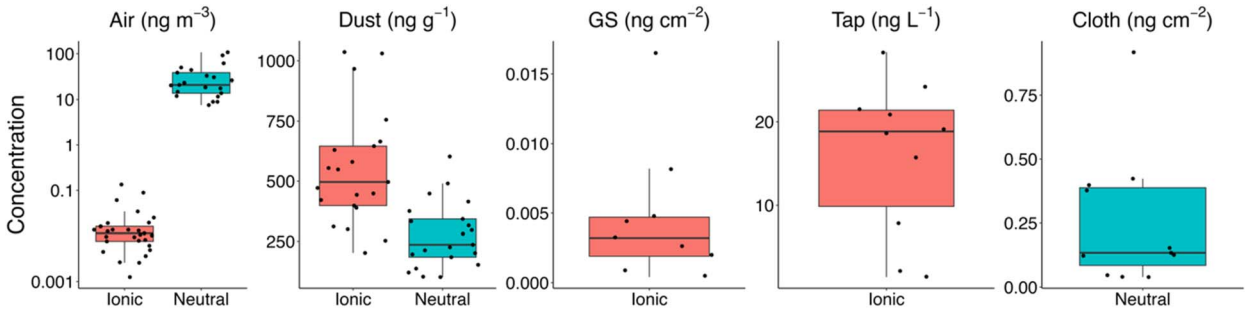

Fig. 1 Concentrations of Σ(ionic PFAS) and Σ(neutral PFAS) for air, dust, tap water, glass slabs (GS), and clothing from IPA Campaign homes. Note that only air concentrations (ng m^{-3}) are plotted on a log10-scale to better represent the wide range in airborne concentrations of neutral and ionic PFAS in homes. The black dots represent individually measured concentrations from homes. Air samples were collected between 1 to 3 times in each home, dust was collected twice in each home, while tap water, 6 month glass slabs, and clothing were collected once in each home.

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and Σ(neutral PFAS)) for air,[2,15] dust,[82] surfaces (glass slabs), drinking water, and/or clothing[2] used in the exposure assessment. Below, we present ionic PFAS results for drinking water, dust, and non-porous surface measurements in more detail because they have not been reported before. Then we report the exposure assessment results.

Neutral PFAS concentrations (Σ(neutral PFAS) = 7.5 to 108 ng m^{-3}) in air samples[2] were 2 to 5 orders of magnitude greater than ionic PFAS concentrations (Σ(ionic PFAS) = 1.3 × 10^{-3} to 0.14 ng m^{-3}; Chang *et al.*, 2024),[15] which is consistent with findings from other studies.[26,34] Eichler *et al.* (2024)[9] and others[13,86,87] found that the FTOHs were largely present in the gas-phase rather than particle-phase and that the FOSEs were found in both phases. In contrast, Chang *et al.* (2024)[15] and others[86,87] have found that PFAAs are found in both phases, with the gas-particle partitioning of PFAAs varying based on compound chain-length and functional group. The large difference in airborne concentrations between neutral and ionic species may be due to the preference of these neutral PFAS for partitioning to the gas-phase.[26,98] However, Vestergren *et al.*, (2015)[99] found that FTOH concentrations were around 2 to 3 orders of magnitude greater than the concentrations of targeted PFCAs in Norwegian consumer products themselves, suggesting that the higher concentrations of neutral, compared to ionic PFAS, may be explained by the higher concentrations in household items themselves.

Dust concentrations were more similar for the two subclasses, ranging from 102 to 602 ng g^{-1} for Σ(neutral PFAS) (Eichler *et al.*, 2024)[82] and from 202 to 1036 ng g^{-1} for Σ(ionic PFAS). Σ(ionic PFAS) ranged from 1.4 to 34.1 ng L^{-1} for drinking water and 4.1 × 10^{-4} to 1.7 × 10^{-2} ng cm^{-2} for glass slabs. Concentrations of Σ(neutral PFAS) in clothing samples[2] ranged from 0.039 to 0.92 ng cm^{-2}.

Drinking water

Mean (median) concentrations of PFOS, 2.95 (2.75) ng L^{-1}, and PFOA, 3.6 (3.8) ng L^{-1}, were highest, followed by PFBS, 2.7 (2.7) ng L^{-1}, and PFBA, 1.5 (1.3) ng L^{-1}. PFBA, PFBS, PFHpA, PFHpS, and PFOA were detected in all kitchen drinking water samples (Fig. 2 and Table S9†). PFDA, PFHxA, PFHxS, PFNA, and PFTA were the next most frequently detected (DF = 90%) in drinking water samples. Because of poor recovery (Table S6†) of the four PAP compounds, they were excluded from this analysis. Homes that used a drinking water filter (Homes 18, 30, and 50) had lower concentrations of PFAS (Fig. 2) in their drinking water. Home 18 used a reverse osmosis (RO) system, Home 30 used a gravity-fed water filter that contained activated carbon and an ion exchange resin, and Home 50 used a granular activated charcoal (GAC) water pitcher purifier.

Homes 1, 18, and 59 were serviced by the same water utility (Orange Water and Sewer Authority; OWASA) and the PFAS profiles for Homes 1 and 59 are similar (Home 18 used a RO water filtration system). Similarly, the PFAS profiles for Homes 10, 35, 43, and 78 were similar and they all share a common water utility (City of Durham); Homes 30 and 50 also share the same water utility but filtered their water. Home 65 received water from the Town of Pittsboro and their PFAS profile was quite different from the other homes. For Home 65, concentrations of PFOS were much lower (0.37 ng L^{-1}) than in other homes without water filters (1.0 to 6.5 ng L^{-1}), but concentrations of PFPeA (6.5 ng L^{-1}) and PFHxA (4.3 ng L^{-1}) were higher than in other homes (n.d. to 3.1 ng L^{-1} and 0.4–3.8 ng L^{-1}, respectively). GenX was detected above DL (>0.047 ng L^{-1}) in home 65 only, at 0.06 ng L^{-1}. Mean and median drinking water concentrations of PFOA and PFOS were below EPA's 2024 Maximum Contaminant Level (MCL) concentrations (4 ng L^{-1}) in all homes.

Measured drinking water concentrations from IPA Campaign homes agree well with reported values from their respective water systems. The North Carolina Department of Environmental Quality (NC DEQ) reported concentrations of GenX, PFOA, and PFOS in drinking water[100] in NC counties in 2022. In Orange County, where water is sourced from University Lake and Cane Creek Reservoir, GenX was not detected, PFOA concentrations ranged from 1.37 to 20.2 ng L^{-1}, and PFOS concentrations ranged from 2.53 to 18.8 ng L^{-1}. In Durham County, where water is sourced from Lake Michie and Little River Reservoir, GenX was not detected, PFOA ranged from 3.57 to 4.28 ng L^{-1} and PFOS ranged from 6.54 to 8.2 ng L^{-1}.[100] The Haw River, situated in the Haw River Watershed, is the source of water for the Town of Pittsboro where concentrations for GenX

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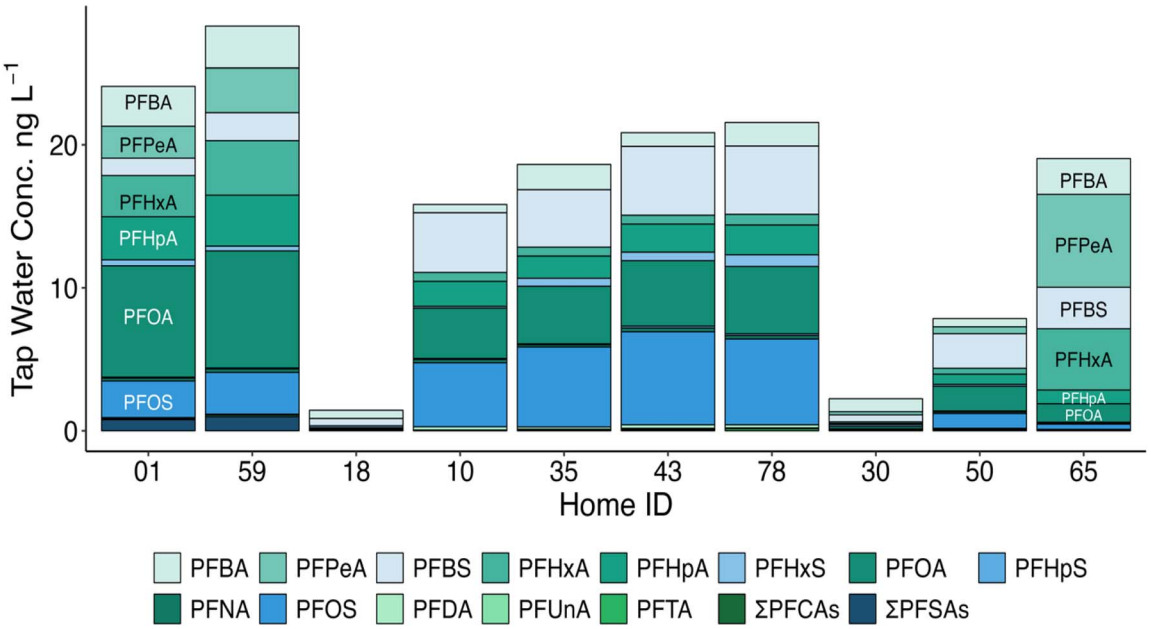

Fig. 2 PFAA profiles and concentrations in kitchen tap water samples. Compounds with DF > 50% are shown while those with DF < 50% were summed into their respective subclass (*i.e.,* ΣPFCAs or ΣPFSAs). All carboxylic acids (PFCAs) are in shades of green and the sulfonic acids (PFSAs) are in shades of blue. Each bar is plotted such that PFBA is on top, followed by PFPeA and PFBS, with ΣPFCAs and ΣPFSAs last. Concentrations are mean field blank subtracted and corrected for recoveries.

ranged from n.d. to 0.262 ng L^{-1}, from n.d. to 11.2 ng L^{-1} for PFOA, and n.d. to 14.6 ng L^{-1} for PFOS.[100] Several studies have shown elevated PFAS levels from tap water collected in Pittsboro.[101,102] Treated wastewater from other cities and towns that discharge into the Haw River or its tributaries[103] may contribute to differences in profiles between Home 65 (Pittsboro, Chatham, Co.) and homes in Orange and Durham Counties. Herkert *et al.*, (2020)[101] also sampled in 61 NC homes between May 2018 and March 2019 located in Chatham, Durham, Orange, and Wake counties. The median ΣPFAA concentration was 43 ng L^{-1} and ranged from 6 ng L^{-1} to 759 ng L^{-1}, with Pittsboro having the highest ΣPFAA concentrations, followed by OWASA, and then Durham County. Additionally, Herkert *et al.* (2020)[101] found that reverse osmosis and two-stage filters removed over 88% of PFCAs and PFSAs, with pitcher filters having lower removal efficiencies, from 36% (PFBA) to 71% (PFOS). This is in agreement with our findings that homes using a water filter had lower PFAS concentrations.

6 month glass slabs

PFAAs and PAPs were detected on glass slabs sampled after 6 months of exposure to room air in all homes (Table S10,† Fig. 3; see Fig. S2† for PAP profiles/concentrations). Median concentrations of PFOA (0.63 pg cm^{-2}) were highest, followed by PFHpA, PFOS, PFDA (0.15 pg cm^{-2}), PFHxA (0.14 pg cm^{-2}), and PFNA (0.14 pg cm^{-2}). PFHxA, PFHpA, PFOA, PFHpS, and PFDA were detected in 100% of homes, while PFOS (89%), PFBA (78%), PFNA (67%), PFUnA (67%), PFDoA (56%), 6 : 2 diPAP (67%), and 8 : 2 diPAP (67%) were detected in over 50% of homes. Note, however, that the recoveries for PFOS (38%), PFDA, PFUnA, PFDoA, and 6 : 2 diPAP were below 40% and should thus be interpreted with caution.

Indoor surfaces are a major reservoir for organic compounds in homes because of the large surface area to air volume ratios and because surfaces are covered with grime (surface films). Particle- and gas-phase deposition results in the development of highly complex indoor surface films that facilitate the absorptive partitioning of volatile and semivolatile organic gases and water vapor.[104–106] Chang *et al.* (2024)[15] and others[26,34,41,107] have documented the presence of ionic PFAS in residential indoor air, including in the gas phase. The presence of ionic PFAS across the volatility range on these glass slab samples suggests that PFAS on non-porous, vertically mounted residential surfaces originate from both particle deposition and gas-phase sorptive partitioning.

Two studies have measured PFAAs and/or PAPs on glass surfaces in homes: in Toronto, Canada in 2007 (ref. 22) and in ten cities in Asia in 2018.[35] A 1 month wipe sample was collected from seven windows in downtown ($n = 2$), suburban ($n = 3$), and rural ($n = 2$) homes in or near Toronto, Canada and analyzed for 11 PFAAs and 3 unsaturated fluorotelomer acids (FTUCAs).[22] Twenty paired indoor/outdoor residential window wipe samples from cities in Asia with accumulation times between 2–3 days and 17 months were analyzed for 29 PFAAs.[35] Despite differences in accumulation time and targeted PFAS, concentration ranges for all three studies were similar in order of magnitude. Indoor concentrations of total PFAS (ΣPFAS) in the Canadian[22] study ranged from 0.45 to 20 pg cm^{-2} and from 0.76 to 33 pg cm^{-2} in Asian cities,[35] whereas concentrations of Σ(ionic PFAS) (including the PAPs) in the IPA Campaign ranged from 0.41 to 16.6 pg cm^{-2}. Meaningful comparison with these studies is

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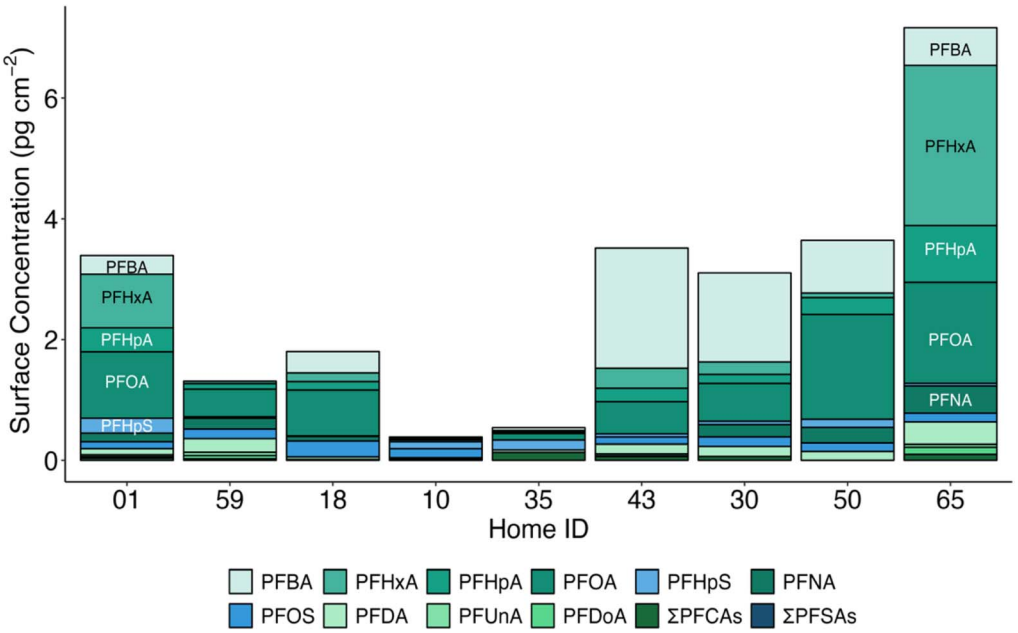

Fig. 3 6 month glass slab PFAA profiles. PFCAs are shades of green and PFSAs are shades of blue. PFAS with DF > 50% are shown individually and those with lower DFs as ΣPFCAs or ΣPFSAs. PAPs are shown in Fig. S2.† Note that no GS were mounted in home 78.

difficult because the sampling times, targeted PFAS, sample collection procedure, and even sampled surfaces are different. In contrast to this study, the two other studies sampled windows, which are exposed to direct sunlight[108,109] and more variable temperature gradients,[35] which may affect chemistry, partitioning, and/or deposition on surfaces.[110–112]

Ionic PFAS in dust

Dust concentrations are dominated by the PAPs and PFOS (Fig. 4), suggesting that sources of these compounds are important in most homes. The median concentration of ionic PFAS in dust was highest for PFOS, whereas the maximum concentration was highest for 6 : 2 diPAP. Maximum concentrations were 735 ng g^{-1} for 6 : 2 diPAP, 402 ng g^{-1} for PFOS, and 284 ng g^{-1} for PFBA. Note that MDLs were high for PAPs, reducing detection frequencies. PFOA was detected in 81% of samples at a median concentration of 8.3 ng g^{-1}, PFOS (median 80.8 ng g^{-1}) and PFBA (median 9.2 ng g^{-1}) were detected in 76% of dust samples (t0 and t6), while PFHxA (median 7.2 ng g^{-1}) was detected in 62% of samples (Table S11†). Within-home species concentration profiles across different sampling periods were similar for some homes, *i.e.*, Home 65, Home 18, Home 78, and Home 01. PFAS in dust may be from abrasion of products and textiles (*i.e.*, couches and carpets),[41] outdoor-to-indoor tracking of PFAS-contaminated soil,[59] and from partitioning from air to dust.[23,41] The significant presence of lower volatility PFAS like the PAPs and PFOS in dust samples suggests that dust may be an important reservoir for these PFAS in homes, and dust may be a potential route of exposure to these compounds, in agreement with other studies and discussed further below.[27,31,113]

Globally, PFAAs and PAPs have been measured in indoor dust at varying concentrations (Table S12†). Median concentrations from selected studies ranged from 0.42 ng g^{-1} (Ireland; 2016–2017)[45] to 741 ng g^{-1} (Finland; 2014–2015)[23] for PFOA and from 0.96 ng g^{-1} (Ireland)[45] to 1890 ng g^{-1} (Finland)[23] for PFOS. Of the handful of studies that targeted the PAPs, concentrations ranged from <0.48 ng g^{-1} (NC)[31] to 687 ng g^{-1} (Canada).[113] Median concentrations of targeted PFAS in dust in the IPA Campaign generally fell within the range reported in the literature, except for PFHxS (DF = 10%; MDL = 0.06 ng g^{-1}), PFDA (DF = 38%; MDL = 0.03 ng g^{-1}), and PFNA (n.d.; MDL = 0.03 ng g^{-1}), for which detection frequencies in IPA homes were too low to determine medians. Dust (<500 μm) was collected from NC homes (n = 184) between 2014 and 2016 as part of the Toddlers' Exposure to Semivolatile organic contaminants in Indoor Environments (TESIE) study.[31] In the TESIE study, PFOA (DF = 100%) was detected at median concentrations[31] of 7.9 ng g^{-1}, which is similar to the median concentration of PFOA (8.3 ng g^{-1}; DF = 81%) detected in IPA Campaign homes. PFOS was detected at a median concentration of 4.4 ng g^{-1} in the TESIE study but at 80.8 ng g^{-1} in IPA Campaign homes. Although PFNA was detected (median 3.3 ng g^{-1}) in 95% of samples in TESIE, no dust samples collected for the IPA campaign contained detectable concentrations of this compound.

Residential exposure to PFAS

For the routes and pathways evaluated in this study, average exposure to ΣPFAS was dominated by neutral PFAS for children and adults. For adults, ΣPFAS exposure was 3.6 ng kg^{-1} per day, of which neutral PFAS contributed 3.2 ng kg^{-1} per day (Fig. 5 and Table S14†). For a 2 year-old child (Table S15†), average

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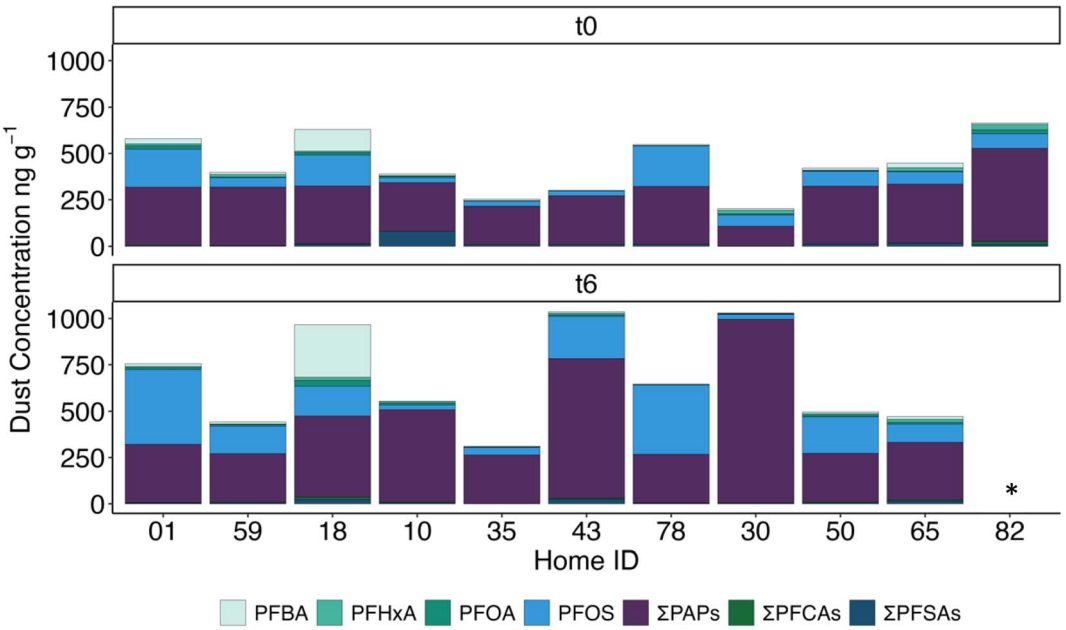

Fig. 4 Dust samples were collected at t0 and t6 from 11 homes in the IPA Campaign. Individual PFAS with DF > 50% are shown, while those with DF < 50% are summed up as ΣPFCA, ΣPFSA, and ΣPAP. GenX (PFECA) was not detected in any samples. *Note that Home 82 left the study after 1 month and no t6 sample was collected. PAP concentrations below the MDL were replaced with MDL/$\sqrt{2}$.

exposure to ΣPFAS was 12.4 ng kg^{-1} per day, with neutral PFAS contributing 10 ng kg^{-1} per day or 81%. Of the considered exposure routes and pathways, inhalation of indoor air dominated exposure to ΣPFAS and Σ(neutral PFAS), for adults, but not for Σ(ionic PFAS). Inhalation exposure contributed 87% to total PFAS exposure, followed by ingestion of drinking water (7%), and ingestion of dust (6%). For Σ(ionic PFAS) alone, ingestion of drinking water (65%) and ingestion of dust (35%) contributed to the majority of exposure, with minimal contribution from inhalation (<1%). For Σ(neutral PFAS), inhalation (98%) was the dominant exposure route, followed by ingestion of dust (2%), which had a much smaller contribution to the neutral PFAS burden. Dermal uptake was a negligible contribution to the intake of ΣPFAS for adults.

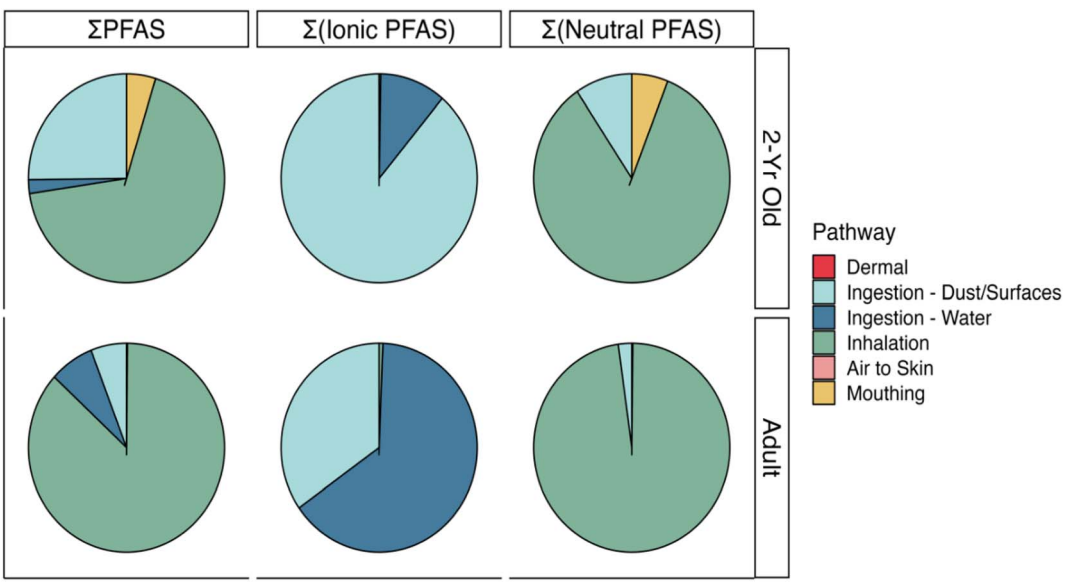

Fig. 5 Relative contributions to daily exposure to ΣPFAS, Σ(ionic PFAS), and Σ(neutral PFAS) for a simulated adult and 2 year-old child from inhalation, ingestion of dust, hand-to-mouth behavior after surface contact, ingestion of tap water, dermal uptake from direct contact with dust and surfaces, as well as air-to-skin uptake for the FOSE/FOSAs only. Mouthing of clothing was only included for neutral PFAS for children. Note that dietary ingestion was not considered.

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The contributors to residential PFAS exposure for a 2 year-old child were quite different. Inhalation (68%) plus ingestion of dust and hand-to-mouth ingestion after touching surfaces (25%) contributed the most to ΣPFAS exposure, followed by mouthing of clothing (5%), ingestion of water (2%), and dermal uptake (<1%). For ionic PFAS, over 89% of exposure was attributable to ingestion of dust and hand-to-mouth behavior after contact with surfaces, 11% originated from ingestion of water, and the remaining <1% was from inhalation. For neutral PFAS, inhalation contributed 85% of total PFAS exposure, while ingestion of dust and mouthing of clothing contributed 9% and 6%, respectively (see Table S15†).

The large contribution to exposure *via* inhalation is largely driven by the high concentrations of neutral PFAS in IPA Campaign homes. It is likely that exposure to ionic PFAS *via* inhalation is underestimated here because we estimate a lower-bound on total (gas + particle) ionic PFAS concentrations in air by using both front and backup QFFs. However, in our estimation, a more accurate measurement of total (gas + particle) ionic PFAS rather than our lower-bound estimate would not alter the conclusions. For example, we estimate a mean total (gas + particle) PFOA concentration of 1.73 pg m^{-3} using the PFOA partitioning coefficient derived using QFF measurements ($\log K_p = -0.77$ m^3 μg^{-1}) from Ahrens *et al.* (2012),[87] mean front filter PFOA from this study ($C_{PFOA} = 0.8$ pg m^{-3}), and mean IPA Campaign $PM_{2.5}$ mass ($m = 5.04$ μg m^{-3}) Eichler *et al.* (2024).[82] This is around 57% larger than the mean front + backup QFF PFOA (1.1 pg m^{-3}), which we used in the exposure assessment. In contrast, neutral PFAS concentrations are 2 to 5 orders of magnitude higher than ionic PFAS concentrations in air. Thus, while inhalation exposures to ionic PFAS could be somewhat higher, the conclusions drawn herein are robust.

Daily intake rates from dermal exposure were estimated using an absorption fraction of 0.048%, which was determined for PFOA on human skin by Fasano *et al.* (2005).[114] This was around 30 times less (1.44 ± 1.13%) than the absorption fraction using a rat model in the same study.[114] A more recent study by Chen *et al.*, (2022)[115] determined absorption fractions for 15 PFAAs and PAPs using a rat model, ranging from 4.1–18% and 5.3–15.1% in the low and high dose conditions. Chen *et al.* (2022)[115] determined an absorption fraction for PFOA of 8.2 ± 1.7% and 7.2 ± 1.7% in the low and high dose groups, respectively, around 5–6 times higher than determined by Fasano *et al.* (2005).[114] The highest absorption fraction Chen *et al.* (2022)[115] determined was 18% for PFHxA and PFBS, roughly 12.5 times higher than the rat absorption fraction value determined by Fasano *et al.* (2005).[114] However, because estimates of dermal exposure contribution to total ionic PFAS exposure, in this study, were below 0.05%, this increase in absorption fraction is unlikely to significantly change this result. Similar to inhalation, it is likely that we have underestimated the dermal exposure to ionic PFAS, but our conclusion that dermal uptake is not a significant route of exposure remains robust.

The relative contributions of various PFAS subclasses (*i.e.*, ΣPFCAs, ΣPFSAs, and ΣFTOHs) to ΣPFAS exposure (Fig. 6) *via* ingestion of water and inhalation were similar for adults and 2 year-olds. However, exposure *via* dermal uptake (a modest contributor) and non-drinking water ingestion were different. For adults, nearly 99% of dermal exposure from all pathways (dust and air-to-skin uptake) was from neutral species (air-to-skin uptake of FOSEs and FOSAs), while only around 65% of total exposure *via* this pathway was attributable to neutral species for children due to lower exposed body surface area and increased contact with dust and surfaces. Neutral species also contributed to around 50% of non-drinking water ingestion for 2 year-old children due to inclusion of mouthing of clothing (neutral PFAS only) and hand-to-mouth behavior after touching surfaces (ionic PFAS only), but to only 33% of non-drinking water ingestion exposure for adults. Young children are likely to exhibit hand-to-mouth behavior, touching surfaces and objects and then touching their mouths. This behavior is less common in adults and was therefore not included for that age group. The different non-drinking water ingestion exposure composition for children was largely due to their increased propensity to ingest dust, which had higher concentrations of ionic PFAS than air. Hand-to-mouth behavior after touching surfaces also contributed but note that only ionic species were measured on non-porous surfaces.

Consideration of dietary exposure

We did not collect food diaries to assess the types of food and frequency of consumption from participants in the IPA Campaign, nor did we analyze any food items for PFAS. Thus, we are not able to characterize dietary exposure to PFAS in our study directly. There is a general dearth of data regarding dietary exposure to PFAS, especially in the US,[40,116–119] which hampers our ability to fully evaluate the importance of dietary exposure relative to the routes we investigated. However,

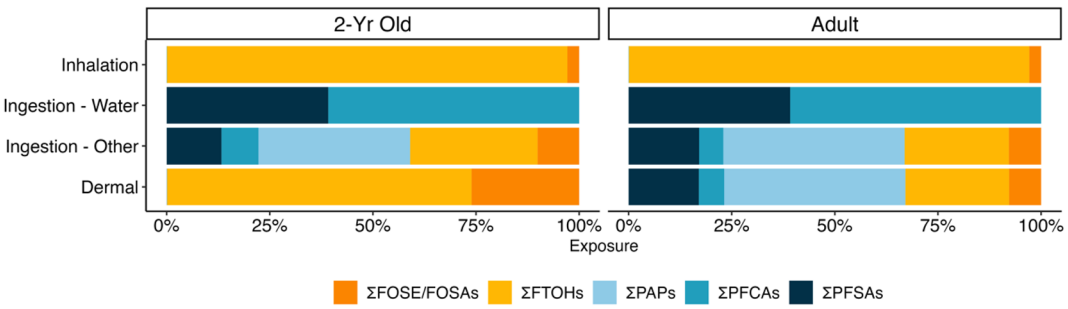

Fig. 6 Contributions of PFAS subclasses to total PFAS exposure for a simulated adult and 2 year-old child.

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Tittlemier *et al.*, 2007 (ref. 120) estimated an average daily intake of 250 ng per day of PFHpA, PFOA, PFNA, PFDA, PFUnA, PFDoA, PFTA, and PFOS for Canadians (≥12 years) in 2007. By dividing this value by body weights (dietary intake for adults: 3.5 ng kg^{-1} per day; children: 18.9 ng kg^{-1} per day) given in Table S8† and comparing to the average non-dietary intake estimated herein, we predict that inclusion of dietary PFAS would increase ΣPFAS exposure by a factor two for adults and a factor of 2.5 for 2 year-olds (7 ng kg^{-1} per day for adults; 30 ng kg^{-1} per day for 2 year-old; Table S16 and Fig. S3†). Using this approach, diet would account for roughly half of ΣPFAS exposure and roughly 90% of ΣPFCA exposure for adults. For children, diet would account for roughly 60% of ΣPFAS exposure and 90% of ΣPFCA exposure. However, children tend to consume more fruit and milk products per body weight and do not necessarily eat the same types of foods as adults.[80] As such, these projections do not account for differences in the dietary behavior of children and adults, and may be overly simplistic. In agreement with other studies,[40,46,77,119,121] we conclude that dietary exposure contributes a significant portion of ΣPFAS exposure for both adults and children and further dietary PFAS measurements are warranted. We further highlight the relevance of other exposure routes, particularly inhalation of indoor air, which was estimated to contribute to 44% and 27% of ΣPFAS intake for adults and children, respectively. For children, hand-to-mouth behavior and mouthing of clothing was estimated to contribute to 12% of ΣPFAS intake. There is a need for additional research into PFAS uptake through these pathways as well as toxicity studies, particularly for neutral PFAS like the FTOHs.

Relevance of high exposure to neutral PFAS

An additional potential source of exposure to PFAAs is from biotransformation of precursors that have been ingested, inhaled, or dermally absorbed into the body. FTOHs have been shown to biotransform to terminal PFAS such as PFHxA, PFHpA, and PFOA,[122] and thus may contribute to ΣPFCA concentrations in the body. Considering all exposure pathways, conversion of 8 : 2 FTOH to PFOA increases the body burden of PFOA by 14% for adults and 17% for children (Table S17 and Fig. S4†). Considering only inhalation, conversion of 8 : 2 FTOH results in a 700-fold increase in the body burden of PFOA due to inhalation for adults and children. The larger impact on inhalation exposures is driven by the fact that ΣFTOH concentrations were 2 to 5 orders of magnitude higher than ΣPFCA concentrations in air. Biotransformation had a negligible impact (<3%) on PFOA and Σ(ionic PFAS) concentrations through non-dietary ingestion and dermal uptake of PFAA precursors for both age groups.

Limitations

There are several limitations to this study. First, the homes recruited to participate in the IPA Campaign were a small convenience sample located in NC, and thus, are not representative of the general US or global population. Second, the recoveries of several PFAS were well below or above 100% and varied by sample type. In general, PFAAs with greater than nine carbons had lower recoveries for the glass slabs, dust, and tap water and the analytical precision for the PAPs (excluded from tap water analysis) was large. Thus, reported concentrations for these analytes should be interpreted with caution due to analytical limits. Extraction and analytical methods are being optimized for future studies. Third, we did not collect data regarding dietary exposure to PFAS, and dietary ingestion is considered one of the main pathways of exposure. The estimate from Tittlemier *et al.*, (2007)[120] is specific to Canadians 12 years and older and was considered a conservative estimate of dietary exposure because it represented only a portion of the average Canadian's diet. At this point, their study is also somewhat outdated; especially because several changes in the production and use of PFAS have occurred since 2007.[73,123,124] Furthermore, the dietary behavior of children is different from adults, which is not accounted for in this study.[120]

A challenge with exposure assessments is the lack of data on absorption fractions and permeability coefficients.[48] We assumed absorption fractions ranging between 0.048% (dermal)[114] to 90% (gut) for all PFAS. However, permeability coefficients and absorption fractions will likely be different for each PFAS, with greater differences between subclasses (*i.e.*, PFCAs *vs.* PFSAs *vs.* FTOHs). More research is needed to characterize fundamental properties of PFAS to better understand their dynamics in the environment as well as to conduct more accurate exposure assessments. It should be noted that biotransformation rates of FTOHs to PFCAs for humans and the biotransformation factors for 4 : 2 and 6 : 2 FTOHs to PFCAs are not available. Human pharmacokinetics can vary greatly from the values determined from rats.[125] As such, the actual percent increase in Σ(ionic PFAS) and ΣPFCAs intake due to biotransformation should be interpreted with caution. However, the large increase in intake *via* inhalation is noteworthy and thus warrants further investigation. Because FTOHs are present in indoor environments at elevated concentrations,[2,26] more research is needed to better characterize their uptake, toxicity, biotransformation, and fate in the human body. Neutral PFAS are often neglected in exposure assessments, but growing evidence of their biotransformation to metabolites and terminal PFAS[78,79,126] highlight the importance of including them. We also estimated the contribution of biotransformed precursors to the body burden of ionic PFAS concentration from these pathway, which few studies[52,127] have done.

Additionally, in this study, we targeted 35 neutral and ionic PFAS even though hundreds more have been measured in environmental media using nontargeted mass spectrometry methods[128,129] and not all sample types were analyzed for both neutral and ionic PFAS. It is now estimated that over 7 million PFAS exist and the number is likely to continue to grow.[130] Surfaces and drinking water were analyzed for ionic species only, while clothing was analyzed for neutral species only. We also did not include exposure *via* dermal uptake from clothing nor textiles (*e.g.*, carpets and furniture). Air measurements analyzed for ionic species were made using a pair of QFFs, which likely did not collect all gas-phase ionic PFAS. We also did not include inhalation exposure resulting from emissions of

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PFAS from worn clothing, which might be significant.[2,131] Object-to-mouth behavior (other than mouthing clothing) was also neglected, but may be an important pathway for young children. We also did not collect any biomonitoring data, which could be used to constrain total intake.[118]

Despite these limitations, we demonstrate the importance of inhalation as an exposure pathway for certain populations. Elevated levels of neutral PFAS in air contribute to the body burden of ionic PFAS due to biotransformation, and mouthing of clothing may be an important exposure pathway for children. Future exposure assessments should include neutral PFAS, their biotransformation, diet, and mouthing of clothing to better characterize the extent of exposure to PFAS and the contributions from each pathway to total exposure.

Data availability

The data supporting this article have been included as part of the ESI.†

Conflicts of interest

There are no conflicts to declare.

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