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Assessing per- and polyfluoroalkyl substance (PFAS) exposure from cell phone contact

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Per- and polyfluoroalkyl substances (PFAS) are found throughout the environment and can adversely affect human health. In this study, we monitored PFAS on cell phones to understand whether cell phones contribute to human PFAS exposure through dermal adsorption and hand-to-mouth exposure. Cell phone ($n = 118$) and hand wipes ($n = 50$) were collected in Ontario, Canada and each sample was paired with a participant lifestyle survey. Wipes were analyzed for 25 PFAS using liquid chromatography tandem mass spectrometry (LC-MS/MS). The analytes included perfluorinated carboxylic acids (PFCAs), perfluorinated sulfonic acids (PFSAs) and polyfluorinated phosphate esters (PAPs). PFAS were detected on 99.2% of cell phones and 100% of hand wipes. The 6 : 2 disubstituted polyfluorinated phosphate ester (6 : 2 diPAP) was detected most frequently on both cell phone and hand wipes. The range of ∑PFAS was <MDL to 65.5 ng on cell phones and 0.1 to 259 ng on hands. The median estimated dermal adsorption was 15.3 and 3.1 ng per day from hands and cell phones, respectively. The median estimated hand-to-mouth exposure was 0.93 and 0.49 ng per day from hands and cell phones, respectively. While cell phone wipes may offer supplementary information, the findings suggest that hand wipes remain the preferred matrix for accurate exposure assessment. Cell phones were demonstrated to be an additional source of exposure to perfluorohexanoic acid (PFHxA), perfluorooctanoic acid (PFOA), 8 : 2 monoPAP, 6 : 2 diPAP, and 6 : 2/8 : 2 diPAP. PFAS exposure was also correlated with age (14–65+), education, race, and continent of birth, based on lifestyle survey findings. Our results point towards diverse and multi-factor exposure pathways for the examined PFAS.

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Environmental significance

Per- and polyfluoroalkyl substances (PFAS) are pervasive in modern environments, and frequent contact with cell phones may contribute to personal exposure. This study evaluated whether PFAS present on cell phones contribute to hand-to-mouth and dermal PFAS exposure. PFAS were detected on 99.2% of cell phones and 100% of hand wipes, but differences in concentrations and chemical profiles indicate that cell phone wipes are not a suitable proxy for hand wipes in exposure assessment. While analytical and survey data offer some guidance for individuals seeking to reduce exposure, the findings reinforce the need for strong regulatory action and systemic change to effectively mitigate PFAS exposure.

1 Introduction

Per- and polyfluoroalkyl substances (PFAS) are a class of over 15 000 human-made chemicals.[1] They are used in many commercial products and industrial processes for their water and oil repellent properties and their chemical and thermal stability. Human exposure to PFAS can occur through drinking water, food, and indoor dust from various sources of PFAS, including consumer products (*e.g.*, cosmetics, non-stick cookware, water- and stain-resistant textiles and food packaging) that can result in exposure *via* multiple routes.[2–6] An extensive body of research demonstrates their persistence, environmental mobility, accumulation and harm to organisms across trophic levels and to humans.[7–10]

In Canada, perfluorooctane sulfonic acid (PFOS), perfluorooctanoic acid (PFOA), long-chain perfluorinated carboxylic acids (PFCAs) containing nine or more carbons, their salts and precursors have been banned from manufacture, use, sale and import since 2009 (PFOS) and 2016 (PFOA and PFCAs).[11] The government has recently proposed to designate most PFAS as toxic under the Canadian Environmental Protection Act and discussions to phase out use of these substances in Canada are underway.[1] Currently, PFOA and other PFCAs are still found in Canadian drinking water, dust and a range of food and commercial products.[2,3,12,13]

Previous research has shown adverse outcomes from dermal exposure to PFCAs using a mouse model.[14] However, gaps in PFAS research still exist surrounding their dermal bioavailability and the contribution of dermal adsorption to body

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burden.[15] There have also been a few studies modeling dermal bioavailability of neutral and ionic PFAS.[16–18] Of the two studies modeling ionic PFAS bioavailability, the first studied PFOA exclusively and observed 48% of the applied dose penetrated the epidermis.[18] The second determined dermal bioavailability for a range of PFCAs and perfluorinated sulfonic acids (PFSAs).[17]

While dermal uptake of PFAS has often been considered negligible,[19,20] several recent studies suggest it may contribute to overall exposure. For example, Poothong *et al.* measured PFAS on hands as a representative indicator of dermal and hand-to-mouth exposure.[21] Authors estimated a median dermal absorption of 1.0×10^{-2} ng kg^{-1} body weight per day and median hand-to-mouth intake of 1.7×10^{-2} ng kg^{-1} day^{-1} from ten PFAS.[21] In addition, PFAS in breast milk has been positively associated with cosmetic and personal care product use, indicating a potential role for dermal pathways.[22] Dermal exposure to PFAS may be more or less significant, depending on the exposure route to which it is compared. Inhalation of ionic PFAS has been estimated at $\sim 1.8 \times 10^{-3}$ ng kg^{-1} day^{-1}, and ingestion from water and dust at $\sim$0.36 ng kg^{-1} day^{-1},[23] while ingestion from packaged food and a Canadian diet study span a wide range (0.35–103.5 ng kg^{-1} day^{-1}).[24–26] Together, these findings demonstrate that dermal uptake, while historically underappreciated, is increasingly recognized as a potentially important exposure pathway. Our study builds on this growing body of research by directly quantifying PFAS on hands, providing data to inform future exposure and risk assessments.

Cell phones are high-contact personal items frequently handled throughout the day. Some cell phone manufacturers, such as Apple Inc., have disclosed the use of PFAS in cell phones.[27] While the company committed to phasing out PFAS from their manufacturing in 2022, no timeline was given and details on which PFAS are used in which components were not disclosed.[27] It is possible that certain PFAS (*e.g.*, fluoropolymers) may be present in multiple components, including wiring, circuit boards, semiconductors, and screen coatings.[28] In combination, we hypothesize that cell phones are a possible route of dermal exposure, given the average person touches their phone over 2600 times per day with heavy users averaging over 5400.[29] Yang *et al.* observed organophosphate ester (OPE) flame retardants and plasticizers on cell phones were positively correlated to OPE levels on hands and OPE metabolites in urine.[30] To our knowledge, similar research to assess PFAS on cell phones and impacts on dermal PFAS exposure has not been conducted.

In this study, we aim to characterize the types and amounts of 25 PFAS on cell phones, which may come from manufacturing and/or contact with hands and other surfaces, and estimate exposure through dermal contact. We then compare our findings to hand-to-mouth exposure and dermal absorption of PFAS on hands. We also investigate the impact of individual demographic, socioeconomic and lifestyle factors such as age, education and diet, on PFAS levels on hands and cell phones. The PFAS analyzed in this study include PFCAs, their PAP precursors and PFSAs, chosen based on their previous inclusion in exposure modeling and biomonitoring work.[21,23,31,32] This selection captures legacy and precursor PFAS. While neutral PFAS such as fluorotelomer alcohols (FTOHs) may also act as PFCA precursors, they were not included due to methodological constraints related to their volatility and detectability.

2 Materials & methods

2.1 Materials

Sterile gauze pads (3 inch^2, 12 ply) were purchased from Dukal corporation *via* Amazon.*ca.* Methanol (99.9% pure, OmniSolv LC-MS grade), acetonitrile (99.9% pure, OmniSolv LC-MS grade) and 2-propanol (99.9% pure, LiChrosolv LC-MS grade) were purchased from EMD Millipore Corp (Burlington, MA). Ammonium acetate (>98% pure, reagent grade) was purchased from Bioshop (Burlington, ON) and ammonium hydroxide (25%) from Ricca (Ottawa, ON). The following native and mass labeled standards were purchased from Wellington laboratories (Guleph, ON): MPFAC-MXA (mass labeled PFCAs and PFSAs), PFAC-MXB (native PFCAs and PFSAs), 6 : 2PAP, 6 : 2diPAP, 8 : 2PAP, 8 : 2diPAP, 6 : 2/8 : 2diPAP, M2-6 : 2PAP, M4-6 : 2diPAP, M2-8 : 2PAP and M4-8 : 2diPAP.

2.2 Sample collection

Participants ($n = 118$) aged 14+ were recruited in the Ottawa and Toronto regions of Ontario, Canada. Participants 18 and older gave informed consent and participants aged 14–17 gave informed and parental consent to participate in accordance and approval from the Carleton University Research Ethics Board (CUREB-B #119952). Wipe samples were prepared and collected based on methods described in Poothong *et al.*[21] Each cell phone wipe sample was prepared using a gauze pad soaked in 3 mL isopropyl alcohol and stored in a 50 mL polypropylene Falcon tube. Each wipe was taken from participants by wiping the entire outer surface area of their cell phone (*i.e.* the exterior of any case or screen protector, where their hands would regularly contact).[30] The researcher collecting the samples wore clean nitrile gloves. For approximately half of participants aged 18+ ($n = 50$), a hand wipe was simultaneously collected. These samples were prepared the same way as cell phone wipes and were collected by the researchers by wiping a gauze pad over the interior of both hands, from wrist to the fingertips, twice. Participants providing a hand wipe sample declared their hands were unwashed at least 60 min before collection. After collection, samples were stored at 4 °C until extraction. Four unused wipes were extracted and analyzed in the same manner as the samples, as procedural blanks to pre-screen for background PFAS levels. These field blanks ($n = 4$) were taken by gently waving the soaked wipes in the air for ten seconds at different sampling locations and then storing and extracting them in the same manner as the samples.

2.3 Participant surveys

To delineate potential factors leading to increased PFAS exposure, PFAS levels on hand and cell phone wipes were compared to information from a survey completed by participants. The survey comprised information on participant demographics

(age, sex, ethnicity and country of birth) socioeconomic status (education and occupation) and lifestyle factors (diet, use of non-stick cookware, take-out containers, water-proof textiles, cosmetics, personal care products, frequency of phone washing, phone brand and material of phone case).

2.4 Sample extraction

Samples were analyzed for perfluorinated alkyl acids (PFAAs) and PAPs based on traditional PFAS monitoring studies and previous work showing high dermal exposure to PAPs.[3,21,31,32] Mass labeled internal standards (3 ng each, Wellington MPFAC-MXA, M2-6 : 2PAP, M4-6 : 2diPAP, M2-8 : 2PAP, M4-8 : 2diPAP) were applied to each wipe. To extract these PFAS, approximately 20 mL of methanol was added to the tube containing the wipe sample. The samples were sonicated for 30 min, and the methanol decanted into clean 50 mL polypropylene tubes. Another 20 mL of methanol was added, the sonication was repeated, and the two supernatants combined. The combined fractions were evaporated to dryness in a warm water bath (50 to 60 °C) and under a gentle stream of N_2. The samples were reconstituted using 500 μL of methanol and stored at −20 °C until LC-MS/MS analysis.

2.5 LC/MS analysis

A Waters Acquity UPLC I-Class PLUS system coupled to a Waters Xevo TQS-Micro MS/MS was used for analysis. A Waters BEH C18 column (50 mm, 2.1 mm, 1.7 μm) was used at 40 °C for PFAS separation, with an injection volume of 5 μL. Two mobile phase gradients were used to target different analytes. PFCAs and PFSAs had A: 95/5 water/methanol with 2 mM ammonium acetate and B: 75/20/5 methanol/acetonitrile/water with 2 mM ammonium acetate. The flow was 0.2 mL min^{-1} starting at 90 : 10 A : B, holding for 0.63 min, ramping to 20 : 80 at 6.3 min and 5 : 95 at 6.4 min, holding until 10 min, then ramping to 90 : 10 at 10.63 min and holding until 12.5 min. For PAPs analysis, mobile phases were A: 0.1% ammonium hydroxide in water and B: 100% methanol. The flow was 0.3 mL min^{-1} starting at 80 : 20 A : B, ramping to 50 : 50 at 2 min, 20 : 80 at 3 min, 5 : 95 at 7 min, 100 B at 7.25 min holding until 8 min, then ramping to 80 : 20 at 9 min and holding until 10 min.

Mass transitions and collision energies are provided in the SI (Table S1).

2.6 Quality assurance and control

A spike and recovery test was performed to validate the extraction method. Three wipes prepared with isopropyl alcohol were spiked with 5 ng of each standard then extracted as described above, while another three wipes were extracted then spiked with 5 ng of each standard for comparison. Absolute recoveries ranged from 88.2–99.5% for PFCAs and PFSAs with standard deviations of 1.7–9.8% (Fig. S1). For PAPs, absolute recoveries ranged from 67.2–105.8% with standard deviations of 1.2–16.4% (Fig. S2). Sample results were not corrected for recovery. Method detection and quantitation limits (MDLs and MQLs respectively) were calculated using procedural blanks or the standard curve if the analyte was not detected in the procedural blanks (eqn (S1) and (S2), results in Table S2). All sample results for PFOA—the only analyte detected in field blank—were blank subtracted using the average of four field blanks. A cell phone wipe efficiency test ($n = 1$) was also performed to quantify the extent of PFAS removed by the isopropyl alcohol wipe; 5 ng of mass-labeled standards (Wellington MPFAC-MXA, M2-6 : 2PAP, M4-6 : 2diPAP, M2-8 : 2PAP, M4-8 : 2diPAP) were pipetted over the exterior of a cell phone. The test phone was dried overnight at room temperature, then a wipe was performed and extracted as described above. Overnight evaporation of the tested PFAS was assumed to be low based on their vapour pressures (2.55×10^{-3} to 251 Pa at 25 °C)[33,34] and previous research by Weed *et al.*[35] Wipe efficiency of most PFAS ranged from 28.8–51.6% except for MPFBA (19.1%) and M4-6 : 2diPAP (90.5%) (Fig. S3). Because wipe-efficiency testing was conducted with a single replicate per compound, these values should be interpreted with caution as they may not fully capture variability in recovery. Nonetheless, cell phone sample concentrations were corrected using these efficiency estimates to reduce potential bias. Where a mass-labeled standard was unavailable for efficiency testing, the standard with the most similar structure was used for the correction. We acknowledge the limitation of not considering hand wipe efficiency; however, it was not feasible to experimentally determine wipe efficiency for hands because of the ethical considerations of intentionally applying PFAS to human skin.

2.7 Scanning electron microscopy (SEM)

To understand differences in PFAS abundance on hand *versus* cell phone wipes, particle size and particle element abundance were assessed. Scanning electron microscopy (SEM) was performed by the Carleton University Nano Imaging Facility using a Tescan Vega-II XMU Scanning Electron Microscope on five cell phone wipes, five hand wipes and two blanks. Results are available in the SI.

2.8 Statistical analysis

GraphPad Prism 10 (version 10.4.2) was used for statistical analysis. Spearman Rho Rank correlation was used for comparing different PFAS amounts within and between cell phones and hands. Values below MDL were replaced with one half the MDL. Analytical data were determined to be non-parametric by a Shapiro–Wilk test ($p < 0.0001$). A Mann–Whitney U test or a Kruskal–Wallis test was used to compare PFAS amounts and survey results for two or three or more groups respectively. Significance was determined with a p-value ≤ 0.05. Compounds detected in less than 50% of samples were excluded from statistical analysis.

2.9 Estimated human exposure from hand-to-mouth contact and dermal absorption

Daily intake of $\sum$PFAS from hand-to-mouth contact was estimated from amounts on wipe samples using the following equation:

$$\mathrm{EDI} = Q \times \mathrm{TF} \times \mathrm{CA} \times f \times t \times U$$

The EDI (estimated daily intake, ng) was calculated for ∑PFAS using: Q (mass of ∑PFAS, ng), TF (transfer fraction) estimated at 50% directly from hands and 25% from cell phones to hand-to-mouth,[36] CA (hand-to-mouth contact area) estimated at 5%, f (frequency of hand-to-mouth contact, h^{-1}) estimated at 2 times per hour, t (transfer time, h) estimated at 16 hours awake per day, and U (uptake efficiency by gastrointestinal tract) estimated at 100%.[21] It was assumed that total PFAS mass on hands and cell phones was constant. Estimations were based on previous hand-to-mouth studies.[21,36,37]

Daily dermal absorption of ∑PFAS was estimated using:

$$\mathrm{EDA} = \Sigma(Q_n \times \mathrm{DA}_n) \times t$$

where Q_n is the mass (ng) of each PFAS on the wipe, and DA_n is the compound-specific dermal absorption factor. For PFAAs, human *in vitro* absorbed fractions from Ragnarsdóttir *et al.* (2024) were applied. For PFBA and 6 : 2 and 8 : 2 diPAPs, compounds not included in Ragnarsdóttir *et al.*, absorbed fractions from a rat dermal absorption model were used, with the limitation that rat skin generally underestimates PFAA relative to human models.[38] For monoPAPs and 6 : 2/8 : 2 diPAP, 6 : 2 diPAP was used as a proxy, consistent with the approach used by Poothong *et al.* The dermal transfer time (t) was set to 24 h for hand wipes and estimated at 4.6 h for cell phones. Total PFAS mass on hands and phones was assumed to remain constant over the transfer interval.

The transfer time for cell phones is based on the 2022 Canadian average daily use, which aligns with 2023 American average daily cell phone use, but is less than 2024 American average daily use.[39,40] For this study, 4.6 hours may also be an underestimation as it does not account for the time people spend holding their phone, but not using it.

3 Results & discussion

3.1 Levels and composition of PFAS on cell phones

From the cell phone wipes collected, 99.2% had detectable PFAS, which included mono- and diPAPs, short-chain and some long-chain PFCAs and PFSAs (Fig. 1). A median of three compounds were detected on each phone ranging up to eight. The maximum amount of ∑PFAS was 96.2 ng (Table S3).

The most frequently detected analyte was 6 : 2 diPAP, present in 94% of samples. Compared to other PFAS measured, it had the greatest median and maximum values: 0.44 ng and 53.4 ng, respectively. Other PAPs were also detected; the 6 : 2/8 : 2 diPAP and 8 : 2 diPAP had detection frequencies of 24 and 7.6%, and 6 : 2, 8 : 2, and 10 : 2 monoPAP had detection frequencies of 0.8, 20 and 5.9%, respectively. The 6 : 2 diPAP accounted for 48% of the ∑PFAS on cell phones wipes and ∑PAPs accounted for 64%.

PFAAs above the MDLs had detection frequencies ranging from 0.8 to 49%. The PFCAs were more frequently detected than PFSAs. Of the PFCAs, PFHxA was detected most frequently (50%) followed by PFOA (37%), then PFPeA (5.9%). PFOA had the highest mean (1.80 ng, median <MDL, 0.03 ng) and maximum (36.5 ng), 5.3- and 8.5-fold higher than PFHxA, respectively. Long-chain PFCAs with 9–12 carbons were rarely detected, 0.8 to 3.4%, and those with 13–18 carbons were not detected. Comparing the PFSAs, PFOS was detected on 8.4% of cell phones with most below the MDL (0.14 ng) up to 1.40 ng. Other PFSAs were rarely detected, including PFBS, PFHxS and PFDS (n = 1–2). While low, these PFAS are still worth reporting to avoid selective bias toward more frequently detected analytes, ensuring that all potentially relevant PFAS exposures are captured. This in turn helps identify emerging sources and compare studies. Similarly, low detection frequencies (1–17%) of PFAAs have also been reported in recent (2021) analyses of

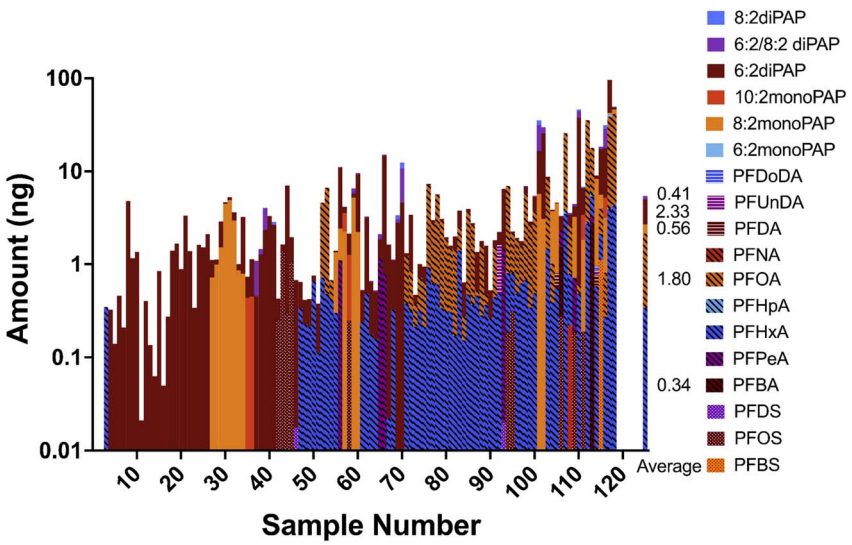

Fig. 1 PFAS across cell phone samples (n = 118). Samples are ordered by increasing number of compounds detected to facilitate comparison, rather than by sample ID. Apparent absence of PFHxA in the first ~45 samples reflects this ordering, not a true absence from lower-numbered samples. Concentrations are plotted on a log scale, and averages of PFAS with ≥ 10% detection frequency are shown in the right panel.

food packaging,[26] and these data may be useful when tracking temporal and spatial differences in PFAS-containing sources.

Canadian regulatory prohibitions on the manufacture, use and sale, as well as drinking water quality guidelines, may explain why PFOS, PFHxS and long-chain PFCAs were infrequently detected.[11] However, these regulations also apply to PFOA. Two possible explanations are: (1) the direct contamination of the environment and consumer products with PFOA and (2) the indirect production of PFOA from the biotransformation of PFOA-precursors, including PAPs that have been found in diverse consumer-products such as cosmetics, food packaging and sanitary products (diapers, pads and toilet paper).[2–4,41,42]

3.2 Levels and composition of PFAS on hand wipes

All 50 hand wipes had detectable levels of PFAS (Table S4). These results align with hand wipe results from Poothong *et al.*, which also had a 100% detection rate.[21] Poothong *et al.* is, to our knowledge, the only other study examining PFAS on hand wipes from the general population; one additional investigation about PFAS on hand wipes was conducted from occupationally exposed populations.[21,43] In our study, the amount of each analyte and mixture of analytes on each wipe varied between individuals (Fig. 2). A median of two compounds were detected on each hand wipe, ranging from one to nine. The maximum level of ∑PFAS on hands was 259 ng.

Like the cell phone wipes, the most frequently detected analyte on hands was 6 : 2 diPAP (96% detection rate) and it had the greatest average (4.26 ng). However, unlike the cell phone wipes, the maximum 6 : 2 diPAP on hand wipes (93 ng) was surpassed by 6 : 2/8 : 2 diPAP which had a maximum of 104 ng (34% detection frequency). The 6 : 2 diPAP accounted for 67% of the PFAS on hand wipes and ∑PAPs accounted for 90%. A moderate positive correlations was observed between 6 : 2 diPAP with the ∑PFAS ($r = 0.695$, $p < 0.001$).

The 6 : 2 diPAP detection rate was similar to Poothong *et al.*, at 98%.[21] The previous study did not analyze for 6 : 2/8 : 2 diPAP, however 8 : 2 mono- and diPAP both had 100% detection rates.[21] This differs from our rates of 24% and 8% respectively, which suggests either geographical or temporal differences in PFAS use and exposure; Poothong *et al.* collected samples in Norway 10 years prior (2013–2014).[21]

The PFAAs above the MDLs had detection frequencies ranging from 2 to 24%. Like the cell phone wipes, the PFCAs were more frequently detected than PFSAs. Of the PFCAs, PFHxA was detected most frequently (24%), followed by PFOA (10%). PFOA had the highest mean (0.16 ng, median <MDL, 0.03 ng) and maximum amount (3.97 ng), 3.2- and 8.1-fold higher than PFHxA. Other PFCAs, including PFBA, PFHpA, and C9–13 PFCAs had detection frequencies at approximately 2%. PFPeA and C14–18 PFCAs were not detected. Comparing the PFSAs, PFOS was detected on only 8% of hands with an average below the MDL (0.14 ng) and maximum level of 0.95 ng. For other PFSAs, PFBS was detected once and PFHxS and PFDS were not detected.

Comparing our PFAA results to those from Poothong *et al.*, we noted differences in detection frequencies. The only PFAA with a higher detection frequency in the current study was PFHxA (7 *vs.* 24%).[21] In contrast, PFOA was detected more frequently on the Poothong *et al.* hand wipes, at 80%, although we note similarities between the average and maximum level (0.18 and 2.8 ng, respectively).[21] The authors also observed higher detection frequencies of PFSAs (25–98%) and long-chain (C9–12) PFCAs (10–47%).[21] Like the PAPs, this may show variation in exposure from different countries or regulatory shifts for commercial use over the past 10 years.

3.3 Cross-matrix comparison of PFAS profiles

Comparing hand and cell phone wipe results, hand wipes contained higher overall PFAS loads (up to 259 ng *vs.* 65.6 ng on phones), higher diversity of PAPs, and less consistent detection

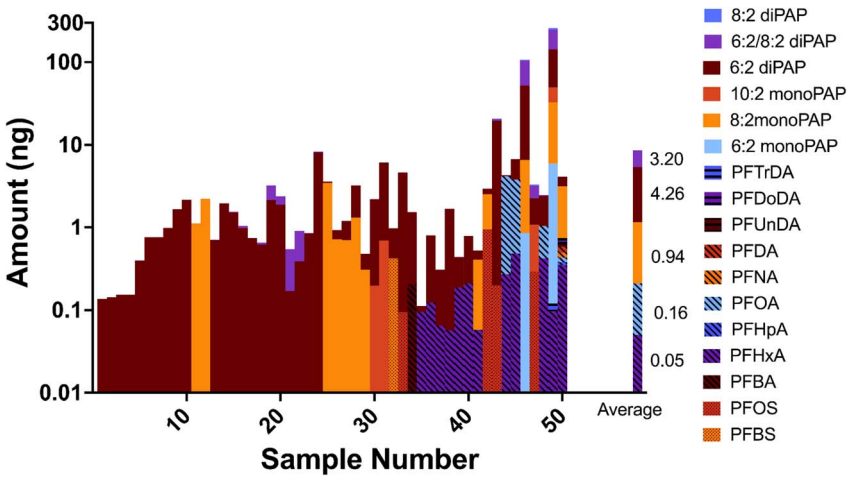

Fig. 2 PFAS across hand wipe samples ($n = 50$). Samples are ordered by increasing number of compounds detected to facilitate comparison, rather than by sample ID. Apparent absence of PFHxA in the first ~35 samples reflects this ordering, not a true absence from lower-numbered samples. Concentrations of are plotted on a log scale, and averages of PFAS with ≥ 10% detection frequency are shown in the right panel.

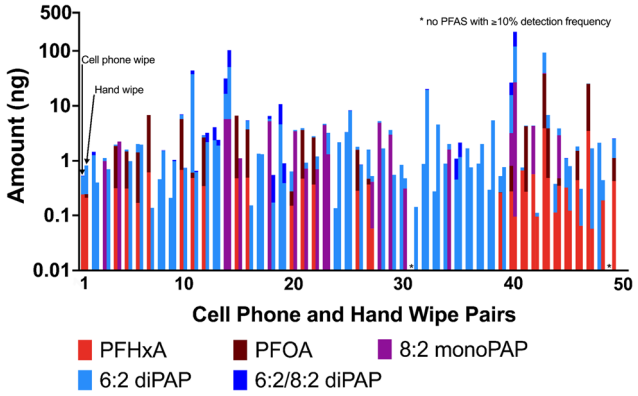

Fig. 3 Comparison of PFAS levels on paired cell phone and hand wipe samples. PFAS with ≥ 10% detection frequency across paired cell phone (left) and hand wipe (right) samples ($n = 50$). Cell phone sample numbers 31 and 50 (marked with an asterisk) did not contain any PFAS above the ≥ 10% detection frequency threshold, resulting in a single hand wipe bar.

of PFHxA and PFOA. Notably, the hand wipe sample with the greatest PFAS load was from a different participant than the cell phone wipe with the highest PFAS load (Fig. 3). A significant weak correlation was observed for 6 : 2 diPAP ($r = 0.28$, $p < 0.05$); however no correlation was observed between the $\sum$PFAS in each matrix.

Overall, the results show that cell phone wipes are not a proxy for hand wipes and should be considered a separate matrix in PFAS monitoring. While some PFAS overlapped between matrices, hands consistently showed higher concentrations for several compounds, different detection frequencies, and moderate or weak correlations with phone wipes. Previous research demonstrated that particle adherence is highly matrix-dependent.[44,45] Tan *et al.* showed that dust adhesion varies widely depending on surface chemistry, surface roughness, and electrostatic behavior, which helps explain why different surfaces retain particles to different extents.[44] Human skin has unique characteristics, including natural oils, micro-topography, and cellular membrane proteins,[46] which differ substantially from the smooth glass, metal or plastic typical of phones and are therefore likely to influence particle retention. Although we did not observe significant differences in particle abundance or composition between our cell phone and hand wipes (SI SEM Results, Fig. S4), PFAS-containing particles may nevertheless adhere more readily to skin than to phone surfaces, even if the total number of particle load appears similar.

3.4 Survey results

Statistical analysis of survey results in relation to analytical values of compounds detected in at least 50% of samples was completed to determine potential sources of exposure. While many factors were evaluated, such as phone brand, case type, cleaning phones *etc.*, only statistically significant results are discussed (Table 1).

Age was significantly associated with increased PFHxA levels on cell phones ($p = 0.008$, Fig. 4a). Increased PFAA exposure has

Table 1 Descriptive statistics of cell phone and hand wipe study participants

	Cell phone wipe ($N = 118$)		Hand wipe ($N = 50$)	
	Number of participants (N)	Percentage of participants[b] (%)	Number of participants (N)	Percentage of participants[b] (%)
Age				
14–17	10	8.5	—[a]	—[a]
18–25	53	45	24	48
26–40	15	13	8	16
41–64	22	19	10	20
65+	10	8.5	3	6
Continent of birth				
North America	78	66	29	58
Asia	22	19	11	22
Africa	8	6.8	7	14
Europe	4	3.4	2	4

[a] Participants under the age of 18 were not included in hand wipe sampling. [b] Percentage may not reach 100 where participants did not respond to a particular survey question.

previously been associated with age using blood serum tests.[31,32] While PFHxA was not included in this previous observation, possibly due to its shorter biological half-life, increased PFOA, PFNA, PFDA, PFHxS, PFOS levels were all correlated to age.[31,32]

Significant differences in 6 : 2 diPAP exposure was observed by continent of birth ($p = 0.022$, Fig. 4b). Participants born in North America had the highest maximum and average 6 : 2 diPAP on hand wipes while Asian-born participants had the lowest levels. The reasons for these disparities remain unclear, as all participants resided in Ontario at the time of sampling. While PFHxA and PAPs were not measured, previous research in the U.S. observed race-based differences in exposure to PFOA, PFNA, PFHxS and PFOS, despite participants residing in the same city or state.[47,48] The authors suggested the exposure differences may be related to racial residential segregation.[47]

Among dietary factors analyzed, only meat consumption showed significant associations with PFHxA exposure (Fig. S5). A non-linear relationship peaking at a consumption frequency of one to two times per month was seen for PFHxA on phone wipes with both bacon and pork ($p = 0.015$ and 0.0008). While PFHxA has been detected in food and food packaging, evidence of their presence specifically in pork, bacon and hamburger packaging is lacking.[4,24]

While there were significant differences between the above groups, there is not a clear trend between consumption frequency and PFHxA exposure. The relationships were non-linear, peaking at a consumption frequency of one to two times per month. We must recognize the potential impact of reporting inaccuracies from social desirability bias, a known phenomenon in self-reporting surveys.[48] This was directly observed in our study as 12.7% of participants who reported washing their phones at least monthly were unable to recall when they last did so. Multiple linear regression analysis was

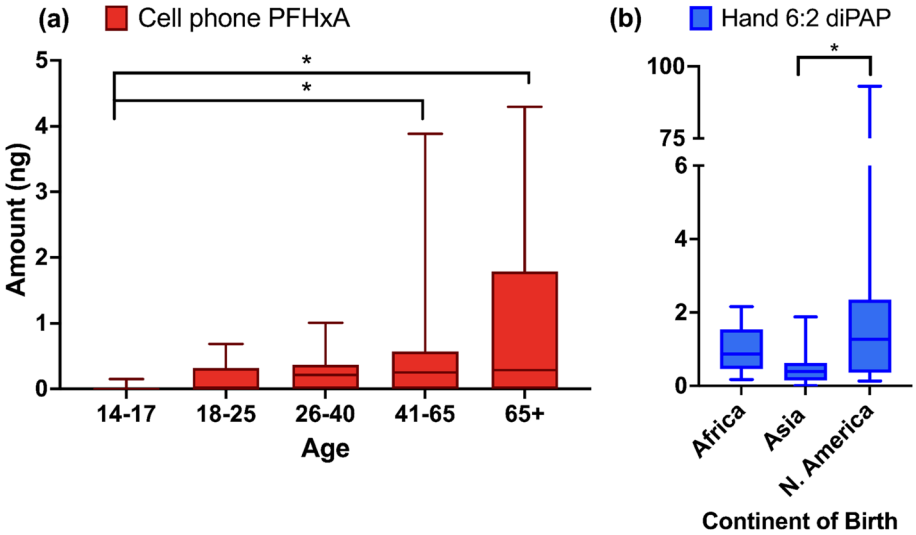

Fig. 4 (a) PFHxA on cell phone wipes by age groups and (b) 6 : 2 diPAP on hand wipes by continent of birth. Bottom and top bars show minimum and maximum values, respectively, with mean shown by middle bar. Statistical significance was determined by Kruskal–Wallis test, $*p < 0.05$.

not effective due to the non-parametric nature of the data; however, the significant effect of demographic (age and country of birth) and dietary habits (particularly pork and bacon consumption) on PFHxA and 6 : 2 diPAP suggests PFAS exposure pathways are complex and influenced by multiple sources.

3.5 Estimated daily exposure

EDA and EDI give valuable information for understanding the risk associated with PFAS exposure. EDA and EDI ranges were calculated based on the minimum and maximum ∑PFAS amounts found on cell phone and hand wipes (Table 2). Our median EDA and EDI values from hand wipes (1.7×10^{-3} and 1.3×10^{-2} ng kg^{-1} day^{-1}, respectively) were similar to those reported by Poothong *et al.* As in Poothong *et al.*, we observed substantial inter-individual variability. Importantly, Poothong *et al.* relied on a single absorbed fraction for PFOA (48%),[21] whereas our estimates incorporate more recent absorption factors for multiple PFAAs (29.2–76.2%) from human *in vitro* skin models and rat-derived values for 6 : 2 and 8 : 2 diPAP (respectively 7.8 and 7.2%).[17,38] Since PAPs, particularly 6 : 2 and 6 : 2/8 : 2 diPAPs, were dominant by mass, their lower absorbed fractions substantially impacts EDA. Given that rat models underestimate PFAA absorption relative to human skin, our EDA values for PAP-rich profiles are likely conservative. This uncertainty reinforces ongoing research on dermal uptake, highlighted by Ragnarsdóttir *et al.*, for systematic measurements of dermal bioavailability for environmentally relevant PAPs.[15]

Dietary intake, contaminated drinking water, and settled dust are well-recognized contributors to PAP and PFAA exposure. Tittlemier *et al.* (2007) estimated an adult dietary intake of ~250 ng day^{-1} in the Canadian population, using PFCA and PFOS concentrations in food composites sampled in 2004. Although dated, to our knowledge this remains the only Canadian dietary estimate.[24] In the U.S., more recent work based on food contact paper found a wide range of intakes: 103.5 ng kg^{-1} day^{-1} (sum of PAPs, PFAAs and FTOHs) from samples collected in 2008–2013, and 0.35 ng kg^{-1} day^{-1} (sum of PAPs and PFAAs) from samples collected in 2021.[25,26] In Europe, dietary estimates also vary considerably, from 103 ng kg^{-1} day^{-1} (sum PFAAs) from Norwegian samples collected in 2008–2009 to 2.6 ng kg^{-1} day^{-1} (sum of PAPs, PFAAs and PFOS precursors) in Swedish samples collected in 2010.[49,50] By comparison, our calculated EDAs and EDIs are several orders of magnitude lower than dietary estimates and 27- to 205-fold lower than the recent estimates from food contact paper. We note that dietary exposure literature is extensive, with many additional studies beyond those summarized here. Our intention was not to provide a comprehensive dietary review, but rather to situate our hand and phone wipe estimates within a representative range of reported dietary intakes.

Table 2 Estimated dermal absorption (EDA) and estimated daily intake (EDI) by hand-to-mouth exposure for ∑PFAS (range and median) calculated from actual wipes and median for a theoretical 70 kg adult

	∑PFAS EDA		∑PFAS EDI	
Sample type	Range (median) ng day^{-1}	Median ng kg^{-1} day^{-1}	Range (median) ng day^{-1}	Median ng kg^{-1} day^{-1}
Cell phone wipes	<MDL to 52.5 (0.90)	1.3×10^{-2}	<MDL to 38.5 (0.78)	1.1×10^{-2}
Hand wipes	0.01 to 40.5 (0.12)	1.7×10^{-3}	0.09 to 207 (0.93)	1.3×10^{-2}

A recent study estimated adult ingestion of PAPs and PFAAs from settled dust at 0.13 ng kg^{-1} day^{-1} in U.S. homes (2021–2022), with drinking water contributing 0.23 ng kg^{-1} day^{-1}.[23] Similar dust-derived estimates were reported in Canada (0.12 ng kg^{-1} day^{-1}; 2013–2014).[51] Our calculated EDAs and EDIs are roughly 10-fold lower.

Inhalation, by contrast, appears to contribute only a small fraction of ionic PFAS exposure.[23,52,53] Indoor measurements from North Carolina homes estimated ionic PFAS (PAPs and PFAAs) inhalation at 1.8×10^{-3} ng kg^{-1} day^{-1}, while an outdoor study in China reported 2.4×10^{-3} ng kg^{-1} day^{-1} for PFBA, PFOA and PFOS.[23,54] These values are approximately seven-fold lower than our median EDAs and EDI estimates. Notably, inhalation is a more prominent pathway for neutral PFAS, which dominate indoor air concentrations and were not measured in this study.[23,52,53]

All studies discussed above measured different PFAS classes using a variety of sampling approaches (*e.g.*, PM2.5 collected on quartz fiber filters *vs.* total suspended particulate),[23,54] and had diverse sampling locations and time periods. Therefore, calculated exposure estimates are not directly comparable. Instead, they collectively provide a framework for interpreting where dermal and hand-to-mouth exposures fall within the broader PFAS exposure landscape.

Taken together, available evidence indicates that dermal and hand-to-mouth pathways for ionic PFAS are generally lower than dietary intake and typically lower than exposure from settled dust or drinking water, consistent with previous conclusions (*e.g.*, Poothong *et al.*). However, they are not negligible. Our results indicate that these pathways can approach or exceed ionic PFAS inhalation exposures and may be particularly relevant in scenarios of elevated surface contamination, such as environments where children frequently contact surfaces, in occupational settings, or where personal electronics accumulate high PFAS residues. Thus, while diet remains the dominant pathway of exposure, dermal and hand-to-mouth routes merit inclusion in exposure modeling to capture the full range of human PFAS contact.

3.6 Limitations

Several methodological and exposure-modelling uncertainties should be considered when interpreting these results. First, while field blanks indicated minimal contamination (low-level PFOA only), the small number ($n = 4$) limits the ability to assess variability and detect sporadic contamination events. Similarly, the precision of cell-phone-specific corrections is also limited by only having one wipe efficiency test. Second, uncertainties are also associated with the exposure factors used. The dermal absorbed fractions for PAPs were unavailable for human skin, requiring use of surrogate rat-derived values, which are known to underestimate absorption relative to human skin and therefore likely make our PAP-rich EDA estimates conservative. In addition, our assumed cell phone exposure time (4.6 h day^{-1}) accounts for active device use and does not account for passive or intermittent handling. The hand-to-mouth contact frequency (2 contacts per hour) is also at the low end of exposure estimates, further contributing to conservative intake values. Thirdly, substitution of non-detects with half MDL values limits statistical analysis of sample variability. Finally, because this study focused on ionic PFAS, we did not capture neutral PFAS, which dominate indoor air and may contribute substantially to total PFAS exposure.

4 Conclusions and implications

Cell phone wipes are not suitable surrogates for hand wipes in PFAS exposure assessment, but they still demonstrate the importance of personal electronics as an exposure pathway, especially to dermal absorption. Demographic data points towards increased PFHxA exposure with age as seen for other PFAS in previous studies.[31] Some dietary habits showed significant but non-linear effects on PFHxA exposure. The study is limited in terms of the geographic region, as the sampling only occurred in eastern Ontario, and demographics of the population sampled. The high prevalence of PAPs, currently unmonitored in national surveys (*e.g.* Canadian Health Measures Survey), calls for expanded regulatory focus and continued biomonitoring to assess the impact of Canada's phaseout of PFAS.[1]

Author contributions

Sierra T. Peskett: conceptualization, methodology, investigation, formal analysis, project administration, validation, visualization, writing – original draft. Salma A. Abu Odeh: investigation. Amy A. Rand: funding acquisition, resources, supervision, writing – review and editing.

Conflicts of interest

There are no conflicts to declare.

Data availability

Data for this article, including survey, analytical and SEM results are available at Open Science Framework at https://osf.io/mzjvn/?view_only=59153f7d4c124371a37c1315fbec8cce.

Supplementary information (SI) is available. See DOI: https://doi.org/10.1039/d5va00197h.

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