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Relative Contribution of Dermal Contact with Indoor Dust to Overall Human Exposure to Perfluoroalkyl Substances

Oddný Ragnarsdóttir [1,2,*], Mohamed Abou-Elwafa Abdallah [1] and Stuart Harrad [1]

[1] School of Geography, Earth & Environmental Sciences, University of Birmingham, Birmingham B15 2TT, UK
[2] Department of Pharmacology and Toxicology, University of Iceland, Hagi, Hofsvallagata 53, 107 Reykjavik, Iceland
* Correspondence: oragnarsdottir@hi.is

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Abstract: Perfluoroalkyl substances (PFAS) have been quantified in household dust samples all over the world. This presents an exposure hazard via ingestion and dermal contact. Based on previously-reported bioaccessibility and bioavailability data, human dermal exposure to PFAS via dermal contact with indoor dust was estimated for the following six PFAS: C_5-C_8-perfluorocarboxylic acids (PFCAs), plus C_4 and C_7-perfluorosulfonic acids (PFSAs). Exposure for people from five countries (Sweden, Japan, Canada, Australia and Norway) was estimated and compared for two age groups (adults and toddlers) under two exposure scenarios (summer and winter). S_6PFAS exposure via dermal uptake from dust during summer ranged from 25–767 pg/kg bw/week and 91–2761 pg/kg bw/week for adults and toddlers, respectively. In general, dermal uptake of PFAS from dust was 10 times lower in winter than in summertime. Perfluorohexanoic acid (PFHxA) made the highest contribution to dermal exposure to S_6PFAS in all countries except Norway, where PFOA > PFHxA. Dermal exposure to S_6PFAS via dermal contact with dust was compared to published exposure estimates via inhalation, indoor dust ingestion, and diet for a cohort of Norwegian adults for which temporally-consistent exposure data were available. Of our target PFAS, dermal exposure of this cohort via contact with dust was greatest for PFOA (2.1 pg/kg bw/day in summer), followed by PFHxA (1.4 pg/kg bw/day in summer). In general, dermal contact with dust did not contribute substantially to overall human exposure to PFSAs, compared to PFCAs. For PFCAs, dermal exposure is a potentially important pathway, with dermal exposure to dust alone contributing to similar levels of exposure as indoor air inhalation for certain compounds. Consequently, this pathway should not be dismissed in future exposure assessments, especially if other sources of dermal exposure to PFAS, e.g., personal care products and fabrics, are considered.

Keywords: PFAS; dermal absorption; human exposure; dust

1. Introduction

Perfluoroalkyl substances (PFAS) are a class of synthetic organofluorine compounds. This group comprises thousands of compounds, which are chemically and thermally stable as well as having hydrophobic and lipophobic properties. These properties have made the use of PFAS common in various industries (e.g., in plastic production and the electronic industry), since the 1940s, as well as in a broad range of consumer products (i.e., textiles and cosmetics) [1,2]. Ever since the presence of PFAS in human sera was highlighted in 2001, their effects on human health have been a concern [3,4]. Studies have linked human exposure to PFAS to a range of health effects, such as lowered immune response to vaccination [5,6], impaired liver function [7,8], in addition to decreased birth weight and higher risk of preterm birth [9,10]. This led the European Food Safety Authority (EFSA) to set a

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recommended tolerable weekly intake of 4.4 ng/kg bw/week for the sum of four PFAS (perfluorooctanoic acid (PFOA), perfluoronoananoic acid (PFNA), perfluorohexane sulfonic acid (PFHxS), and perfluorooctane sulfonic acid (PFOS)) [3].

Human exposure to PFAS can happen through various pathways. Diet is widely considered the main pathway of exposure to PFAS, with seafood being a major contributor to the total dietary exposure [11–13]. Drinking water is also considered a potentially important source of PFAS exposure [12,14]. Other, less significant exposure pathways are indoor air inhalation and dust ingestion, although these exposure pathways may be more significant for toddlers [13,15–17].

By comparison, current knowledge on the dermal pathway compared to other pathways of human exposure to PFAS is lacking [16]. This is due to the expectation that most PFAS will be in the ionised form at skin surface pH, leading to the assumption that dermal uptake would be less likely. However, recent in vivo and in vitro studies have shown that the transdermal permeation of PFAS is possible under normal physiological conditions [18–20]. A study involving one human volunteer exposed to mass labelled PFOA fortified sunscreen found that 1.6% of the applied mass was absorbed through the skin. The permeation appeared to be slow, with the peak PFOA plasma levels being detected 22 days after exposure [18]. Another study using male Sprague-Dawley rats exposed to 15 PFAS, found that fractions of both PFCAs and PFSAs were able to penetrate the rat skin. Moreover, they reported a peak of PFAS in the systemic circulation at 8–72 h, which is significantly later than that reported following oral administration (1–24 h) [19]. This corroborates what was observed in the human study, i.e., that dermal exposure can be long-lasting and contribute to total human body burdens of PFAS. Most recently, an in vitro study exposing 3D-human skin equivalent (3D-HSE) models to 10 PFCAs (C_5–C_{14}) and 6 PFSAs (C_4–C_9) in methanol for 36 h investigated the dermal permeation of PFAS. The study found that lower chain compounds relatively readily permeated, with permeation decreasing with increasing chain length. The highest permeation was seen for PFPeA (59%) and PFBS (49%). The longer chain compounds included, ≥C_{10} PFCAs and perfluorononanoic acid (PFNA) were not directly absorbed. They were however detected within the skin tissues themselves with C_{11} and C_9 PFCAs being detected at the highest concentrations within the skin (67% and 68% respectively), which could indicate very slow permeation of the longer chain compounds [20]. It is important to note that the dose applied at the start of exposure experiments can impact the fraction that permeates through the skin [21]. Consequently, the in vitro study provided permeation coefficients ($\mathbf{P_{app}}$) for eight of the studied PFAS which had the highest permeation. These coefficients are independent of the applied dose and can thus be used in estimations of human exposure via dermal contact over a range of doses under various exposure scenarios [20].

Several potential sources of human dermal exposure to PFAS have been identified [16]. One possibly important source of exposure is personal care products. PFAS have been identified in cosmetics from all over the world, i.e., sunscreen, foundations, moisturisers, and hand sanitisers [2,22–24]. These products can contain PFAS in quite high concentrations, for example, cosmetics and sunscreens from Japan were found to contain PFCAs in concentrations up to 5.9 μg/g in cosmetics and 19 μg/g respectively [22]. Another possible source of dermal exposure is fabrics treated with PFAS. Studies have identified PFAS, e.g., in durable water repellent clothing as well as children's school uniforms and car seats [25–28]. Finally, PFAS have been quantified in indoor dust from all over the world, which can be a source of dermal exposure to PFAS [29,30].

It is important to understand that there are two fundamental processes involved in human dermal uptake of chemicals from a source material. Bioaccessibility refers to the fraction of chemicals that are released from a matrix (e.g., dust) into the body's fluids (a mixture of sweat and sebum). Bioavailability however, is defined as the fraction of a chemical that reaches the systemic circulation [31–33]. A combination of data on bioaccessibility and subsequent percutaneous penetration is necessary to fully understand the extent of human dermal uptake of chemicals (e.g., PFAS) [32,34,35]. In essence, a chemical must first transfer from a source matrix like dust into skin fluids (become bioaccessible) and it is that only that fraction present in skin fluid that is then available for transfer across the skin into the circulation.

Against this backdrop, the current study, for the first time, presents dermal uptake data based on a combination of the dermal bioaccessibility of PFAS present in indoor dust as well as the skin permeation coefficients ($\mathbf{P_{app}}$) previously reported by our research group [20,36]. Using these data, more accurate estimations can be made of human internal exposure to PFAS via contact with indoor dust using different scenarios for various age groups. These results are then compared to other exposure pathways. While only one of many possible sources of dermal exposure to PFAS is considered here (i.e., dermal contact with indoor dust), the significance of the dermal pathway to human PFAS exposure and its contribution to total human body burdens is highlighted.

2. Methods

PFAS concentrations in dust from multiple countries have been published previously [29,37]. Concentrations of those PFAS identified in dust in two selected studies and for which dermal bioaccessibility and dermal penetration values are available [20,36] were used here to estimate human exposure to PFAS via dermal uptake from dust for five countries (Table 1). Where median values were below the limit of detection (LOD), half of the LOD values for each compound were used.

Table 1. Median dust concentrations (ng/g) from various countries used for dermal uptake estimates. Concentrations from [28] except Norwegian concentrations from [15].

	Sweden (ng/g)	Japan (ng/g)	Canada (ng/g)	Australia (ng/g)	Norway (ng/g)
PFPeA	<0.36	4.5	1.62	0.55	NR [a]
PFHxA	7.05	12	7.44	4.77	0.40
PFHpA	1.46	6.54	4.75	2.18	<0.1
PFOA	14.4	25.5	21	13.5	3.60
PFBS	1.24	1.33	3.44	1.29	<DF [b]
PFHxS	0.15	0.76	3.75	4.32	0.06

[a] not reported; [b] compound detected in less than 45% of samples and thus not included in exposure estimations.

Estimations of Human Dermal Uptake of PFAS from Dust

Uptakes of individual PFAS from dermal contact with dust were estimated based on exposure parameters obtained from the USEPA exposure factors handbook [38]. A summary of these parameters can be found in Table 2.

When estimating dermal uptake, it is essential to account for both the bioaccessibility and bioavailability of a chemical. For compounds bound to particulate matter such as indoor dust, their release into the skin's surface fluids (bioaccessibility) can be a limiting factor [32,39,40]. Once bioaccessible, that fraction of a chemical can then pass through the outermost layer of the skin (the stratum corneum*)* where it could either be metabolised in situ or enter the systemic circulation (i.e., becomes bioavailable) through the viable epidermis and dermis layers [16]. Thus both bioaccessibility and bioavailability of the selected PFAS was considered in this study.

Table 2. Parameters used in dermal exposure assessment of target PFAS: (a) Dust adhered to skin and skin surface area calculated for summer and winter as described in the main text [38]; (b) Permeation coefficients (P_{app}, [20]), bioaccessible fraction ($F_{bioaccessible}$, [35], t = time spent indoors and body weight (BW, [38]).

(a)						
Dust adhered to skin (mg/cm^2)			**Surface Area (cm^2)**			
			Adults		**Toddler**	
	Adults	**Toddler**	**Male**	**Female**	**Male**	**Female**
Head	0.024	0.054	1360	1140	615.5	592.8
Forearms	0.038	0.048	1460	1090	425.6	418
Hands	0.160	0.170	1070	870	364.8	372.4
Thighs	0.019 [a]	0.051 [a]	4113	3560	1140	1185.6
Lower legs			2710	2300	782.8	790.4
Feet	0.140	0.200	1380	1210	494	478.8
(b)						
	P_{app}	**$F_{bioaccessible}$**	**T (h)**		**BW (kg)**	
	(cm/h)	**(unitless)**	**Adult**	**Toddler**	**Adult**	**Toddler**
PFPeA	4.33×10^{-2}	0.91	4	6	80	15
PFHxA	1.80×10^{-2}	0.88	4	6	80	15
PFHpA	8.10×10^{-3}	0.75	4	6	80	15
PFOA	3.82×10^{-3}	0.70	4	6	80	15
PFBS	1.41×10^{-2}	0.74	4	6	80	15
PFHxS	7.50×10^{-3}	0.59	4	6	80	15

[a] Dust adhered to skin of legs.

Two exposure scenarios were applied for each of the following two age groups (toddlers and adults) [41]:

i. **Summer:** assuming head, forearms, hands, thighs, lower legs, and feet were exposed to dust (i.e., person wearing shorts and t-shirt).

ii. **Winter:** Assuming head and hands exposed to dust (i.e., person wearing full-length trousers, long-sleeve top, and socks).

based on the following equations. Firstly, the dose of PFAS (ng/cm^2) was determined (Equation (1))

$$Dose = C_{dust} * DAS * f_{bioaccessible} \quad (1)$$

where C_{dust} = PFAS concentration in dust (ng/g), DAS = dust adhering to skin (g/cm^2), and $f_{bioaccessible}$ = bioaccessible fraction (fraction of PFAS available for dermal uptake (unitless)). These values were based on values from Ragnarsdóttir et al., 2023 [35].

From there, the flux (ng/cm^2·h) could be determined (Equation (2))

$$Flux = P_{app} * Dose \quad (2)$$

where $\mathbf{P_{app}}$ = Permeation coefficient (values from [19] (cm/h))

Finally, the mass permeated per day (pg/day) and the resulting daily exposure (pg/kg bw/day) were estimated (Equations (3) and (4))

$$Mass\ permeated = Flux * t * BSA \quad (3)$$

$$Daily\ exposure = \frac{mass\ permeated}{BW} \quad (4)$$

where t = time of exposure (h), BSA = body surface area exposed (cm^2), and BW = body weight (kg).

3. Results and Discussion

3.1. Dermal Uptake of PFAS from Indoor Dust

Considering both the dermal bioaccessibility and bioavailability of PFAS, human exposure to PFAS via dermal contact with dust was estimated for two age groups of residents of four countries (Swede, Japan, Canada and Australia). Out of the four countries considered here, PFAS concentrations in dust were highest in Japan followed by Canada which resulted in the highest dermal exposures for the residents of those countries. Our estimates (presented in Table 3) of weekly exposure to PFAS highlight the magnitude of dermal exposure as an exposure pathway, especially in summer. Total PFAS exposure via dermal uptake from dust during summer ranged from 25–767 pg/kg bw/week and 91–2761 pg/kg bw/week for adults and toddlers, respectively. In general, dermal uptake of PFAS from dust was 10 times lower in winter compared to summer, ranging from 2–70 pg/kg bw/week for adults and 10–300 pg/kg bw/week for toddlers. This is unsurprising as less skin is typically exposed in winter leading to a smaller skin surface area exposed. Dermal uptake from dust for the Norwegian population was noticeably lower compared to the estimates derived for the other countries considered here. This is due to the dust concentrations from Norway being significantly lower compared to the other countries.

While PFOA was the target PFAS detected at the highest concentrations in dust samples from all countries considered; the greater dermal bioaccessibility and bioavailability of PFHxA meant that exposure via dermal contact with dust was highest for PFHxA for all countries except Norway, ranging from 10–296 pg/kg bw/week for adults and 35–1064 pg/kg bw/week for toddlers in summer across all countries. The second highest dermal exposures were for PFOA (highest for Norway) and PFPeA; ranging from 15–106 pg/kg bw/week and 0–277 pg/kg bw/week respectively for adults and 54–380 pg/kg bw/week and 0–998 pg/kg bw/week respectively for toddlers.

When comparing our estimates of dermal exposure from dust to the EFSA recommended tolerable weekly intake of 4.4 ng/kg bw/week for the sum of four PFAS (PFOA, PFNA, PFHxS, and PFOS = EFSA 4), dermal exposure from dust to adults during summer can be up to 0.1 ng (EFSA 4)/kg bw/week (Canada and Japan). Compared to other sources of dermal exposure to PFAS (e.g., clothing, cosmetics etc.), it is likely that exposure via dermal contact with dust is a minor pathway of human dermal exposure to PFAS [16]. For toddlers however, dermal uptake via contact with dust is a more substantial source of exposure with Canadian and Japanese toddlers being exposed to 0.4 ng (EFSA 4)/kg bw/week. This difference can be explained by the fact that toddlers have more dermal contact with dust compared to adults as well as a higher ratio of skin surface area to body weight.

The study of dermal permeation of PFAS used in this evaluation was limited by the survival of the skin cells (36 h) [20]. Because of this, permeation coefficients ($\mathbf{P_{app}}$) for PFOS and PFNA could not be derived, thereby preventing us estimating dermal exposure for these PFAS, even though they were identified in dust samples from all countries [29]. It is important to stress that while their direct permeation was negligible after 36 h, both compounds were identified within the skin itself at the end of exposure which may indicate that dermal permeation occurs but over a longer period of time [20]. This is consistent with a study by Abraham and Monien where a

single human subject was exposed to $^{13}C_4$-PFOA mixed into sunscreen. This study found that peak plasma levels were reached 22 days after application to the skin [18].

Table 3. Weekly human exposure to PFAS via dermal contact with dust based on age group, country, and season (pg/kg bw/week). Sum of the EFSA 4 is provided for comparison with EFSA TWI of 4400 pg/kg bw/week.

a. Summer	**Adults**					**Toddlers**				
	Sweden	**Japan**	**Canada**	**Australia**	**Norway**	**Sweden**	**Japan**	**Canada**	**Australia**	**Norway**
PFPeA	11	277	100	34	- [a]	42	998	359	122	- [a]
PFHxA	174	296	183	117	10	625	1064	659	423	35
PFHpA	14	62	45	21	0	50	223	162	74	2
PFOA	60	106	87	56	15	215	380	313	201	54
PFBS	20	22	56	21	- [a]	73	78	201	75	- [a]
PFHxS	1	5	26	30	0	4	19	93	107	1
∑$_6$PFAS	**268**	**767**	**497**	**279**	**25**	**966**	**2761**	**1788**	**1003**	**92**
EFSA 4 [b]	**61**	**111**	**113**	**86**	**15**	**218**	**399**	**406**	**308**	**55**
b. Winter	**Adults**					**Toddlers**				
	Sweden	**Japan**	**Canada**	**Australia**	**Norway**	**Sweden**	**Japan**	**Canada**	**Australia**	**Norway**
PFPeA	0	25	9	3	- [a]	7	109	39	13	- [a]
PFHxA	16	27	17	11	1	68	116	72	46	4
PFHpA	1	6	4	2	0	5	24	18	8	0
PFOA	5	10	8	5	1	23	41	34	22	6
PFBS	2	2	5	2	- [a]	8	8	22	8	- [a]
PFHxS	0	0	2	3	0	0	2	10	12	0
∑$_6$PFAS	**25**	**70**	**46**	**26**	**2**	**105**	**300**	**194**	**109**	**10**
EFSA 4 [b]	**6**	**10**	**10**	**8**	**1**	**24**	**43**	**44**	**34**	**6**

[a] Not detected in a sufficient proportion of dust samples; [b] EFSA 4: PFOA, PFNA, PFHxS, and PFOS [3].

3.2. Dermal Exposure to PFAS in Norway via Dermal Contact with Dust; Comparison to Other Exposure Pathways

While data on exposure via non-dermal pathways was not available for Australia, Canada, Japan, and Sweden, contemporaneous data on exposure of a cohort (n = 60) of Norwegian adults via diet, indoor air, and indoor dust ingestion were available [15]. We therefore compared these data with our estimated exposures to PFAS via dermal contact with dust for adults in Norway. The median exposures reported by Poothong et al. (2020) [15] were compared to our results as median dust concentrations were used for the dermal uptake estimates. The comparison of the different pathways is presented in Figure 1. Diet is the main exposure pathway for all compounds. However, while a minor pathway by comparison to diet, dermal exposure via contact with dust during summer did contribute discernibly to overall exposure to PFCAs. Dermal exposure from dust was less prominent in the winter for PFCAs. Exposure via dermal exposure to dust in summer was greatest for PFOA (2.1 pg/kg bw/day), followed by PFHxA (1.4 pg/kg bw/day). In general, exposure via dermal contact with dust did not contribute substantially to human exposure to C_4 and C_7 PFSAs. However, we caution that dermal exposure is a potentially important pathway of non-dietary exposure to C_5–C_8 PFCAs, which should not be dismissed in future exposure assessments, especially considering other sources of dermal exposure to PFAS, i.e., personal care products. In particular, we stress that only dermal exposure to PFAS via contact with dust is considered here. This is likely only one of many sources of dermal exposure to PFAS, which if source materials such as fabrics and cosmetics contain higher PFAS concentrations than in indoor dust, would likely result in higher dermal exposure values than shown here [16]. Recent studies have shown positive correlations between the use of personal care products and serum PFAS levels indicating them as an important source of dermal exposure to PFAS [42,43].

In general, human exposure via dermal contact with indoor dust was lower for PFSAs than PFCAs. This is because of the lower concentrations of PFSAs in dust samples, as well as the lower dermal permeation of PFSAs compared to PFCAs of the same chain length. Similar patterns are seen for other sources of dermal exposure to PFAS with fabric samples containing higher concentrations of PFCAs than PFSAs [27,28,44]. Similarly, cosmetics from both European and North American markets had higher concentrations of PFCAs than PFSAs, with no PFSAs being detected above the limit of detection in European cosmetic samples [23,24,45,46].

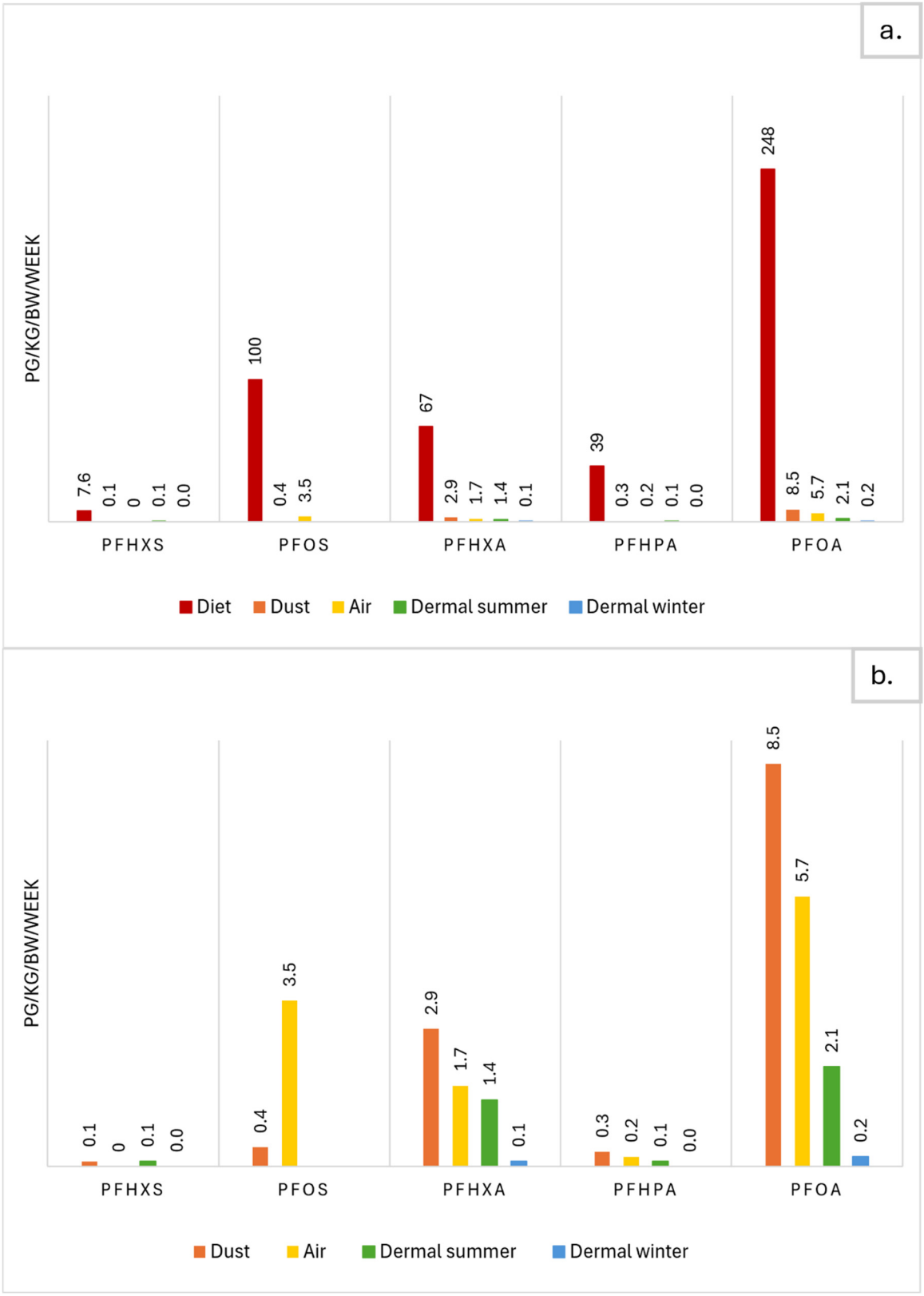

Figure 1. Human exposure to PFAS via dermal contact to dust compared to other known exposure pathways including (**a**) and excluding (**b**) diet.

4. Conclusions and Future Research

This study highlights the importance of human dermal uptake as an exposure pathway to PFAS. Even though only dust was considered here as a possible source of dermal exposure to PFAS, it did contribute to overall human exposure to perfluoroalkyl acids (PFAAs), albeit less than other exposure pathways. Other sources of dermal

exposure to PFAS (e.g., fabrics and/or cosmetics) would likely result in discernibly higher exposure. Thus, it is important for future exposure assessments not to dismiss the dermal exposure pathway.

Moreover, precursor compounds to both PFCAs and PFSAs have been identified in both fabric and cosmetic samples [27,45,46]. In cosmetics, polyfluoroalkyl phosphates (PAPs) and fluorotelomer sulfonic acids (FTSAs) have been detected in high concentrations while PAPs, fluorotelomer alcohols (FTOHs), and fluorotelomer acrylates and methacrylates (FTAcs/FTMAcs) have been identified in fabric samples [26]. The studies of the dermal permeation potential of such precursor ions are limited. However, an in vivo rat study found that both 6:2 and 8:2 diPAPs were able to permeate through the skin with 7.8% and 7.2% of the applied dose having permeated respectively after 144 h [19]. Future studies to better understand both the direct permeation of the precursor ions as well as the possible dermal metabolism potential of those compounds, would further aid understanding of the contribution of the dermal pathway to overall human exposure to PFAS.

Author Contributions

O.R.: Writing—Original Draft, Investigation, Formal Analysis, Data Curation, Conceptualization. M.A.-E.A.: Writing—Review & Editing, Conceptualization, Methodology, Funding Acquisition. S.H.: Writing—Review & Editing, Conceptualization, Supervision, Funding Acquisition. All authors have read and agreed to the published version of the manuscript.

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Data Availability Statement

Data are available on request.

Conflicts of Interest

The authors declare no conflict of interest. Given the role as the Editor-in-Chief, Stuart Harrad had no involvement in the peer review of this paper and had no access to information regarding its peer-review process. Full responsibility for the editorial process of this paper was delegated to another editor of the journal.

References

1. Buck, R.C.; Franklin, J.; Berger, U.; et al. Perfluoroalkyl and Polyfluoroalkyl Substances in the Environment: Terminology, Classification, and Origins. *Integr. Environ. Assess. Manag.* **2011**, *7*, 513–541. https://doi.org/10.1002/ieam.258.
2. Glüge, J.; Scheringer, M.; Cousins, I.T.; et al. An Overview of the Uses of Per- and Polyfluoroalkyl Substances (PFAS). *Environ. Sci. Process. Impacts* **2020**, *22*, 2345–2373. https://doi.org/10.1039/D0EM00291G.
3. EFSA CONTAM Panel (EFSA Panel on Contaminants in the Food Chain). Risk to Human Health Related to the Presence of Perfluoroalkyl Substances in Food. *EFSA J.* **2020**, *18*, 6223. https://doi.org/10.2903/j.efsa.2020.6223.
4. Hansen, K.J.; Clemen, L.A.; Ellefson, M.E.; et al. Compound-Specific, Quantitative Characterization of Organic Fluorochemicals in Biological Matrices. *Environ. Sci. Technol.* **2001**, *35*, 766–770. https://doi.org/10.1021/es001489z.
5. DeWitt, J.C.; Blossom, S.J.; Schaider, L.A. Exposure to Per-Fluoroalkyl and Polyfluoroalkyl Substances Leads to Immunotoxicity: Epidemiological and Toxicological Evidence. *J. Expo. Sci. Environ. Epidemiol.* **2019**, *29*, 148–156. https://doi.org/10.1038/s41370-018-0097-y.
6. Grandjean, P.; Heilmann, C.; Weihe, P.; et al. Estimated Exposures to Perfluorinated Compounds in Infancy Predict Attenuated Vaccine Antibody Concentrations at Age 5-Years. *J. Immunotoxicol.* **2017**, *14*, 188–195. https://doi.org/10.1080/1547691X.2017.1360968.
7. Salihovic, S.; Stubleski, J.; Kärrman, A.; et al. Changes in Markers of Liver Function in Relation to Changes in Perfluoroalkyl Substances—A Longitudinal Study. *Environ. Int.* **2018**, *117*, 196–203. https://doi.org/10.1016/j.envint.2018.04.052.
8. Sen, P.; Qadri, S.; Luukkonen, P.K.; et al. Exposure to Environmental Contaminants Is Associated with Altered Hepatic Lipid Metabolism in Non-Alcoholic Fatty Liver Disease. *J. Hepatol.* **2022**, *76*, 283–293. https://doi.org/10.1016/j.jhep.2021.09.039.
9. Meng, Q.; Inoue, K.; Ritz, B.; et al. Prenatal Exposure to Perfluoroalkyl Substances and Birth Outcomes; An Updated Analysis from the Danish National Birth Cohort. *Int. J. Environ. Res. Public. Health* **2018**, *15*, 1832. https://doi.org/10.3390/ijerph15091832.

10. Sagiv, S.K.; Rifas-Shiman, S.L.; Fleisch, A.F.; et al. Early-Pregnancy Plasma Concentrations of Perfluoroalkyl Substances and Birth Outcomes in Project Viva: Confounded by Pregnancy Hemodynamics? *Am. J. Epidemiol.* **2018**, *187*, 793–802. https://doi.org/10.1093/aje/kwx332.
11. Gebbink, W.A.; Berger, U.; Cousins, I.T. Estimating Human Exposure to PFOS Isomers and PFCA Homologues: The Relative Importance of Direct and Indirect (Precursor) Exposure. *Environ. Int.* **2015**, *74*, 160–169. https://doi.org/10.1016/j.envint.2014.10.013.
12. Sunderland, E.M.; Hu, X.C.; Dassuncao, C.; et al. A Review of the Pathways of Human Exposure to Poly- and Perfluoroalkyl Substances (PFASs) and Present Understanding of Health Effects. *J. Expo. Sci. Environ. Epidemiol.* **2019**, *29*, 131–147. https://doi.org/10.1038/s41370-018-0094-1.
13. Trudel, D.; Horowitz, L.; Wormuth, M.; et al. Estimating Consumer Exposure to PFOS and PFOA. *Risk Anal.* **2008**, *28*, 251–269. https://doi.org/10.1111/j.1539-6924.2008.01017.x.
14. European Union. Directive (EU) 2020/2184 of the European Parliament and of the Council of 16 December 2020 on the Quality of Water Intended for Human Consumption; 2020/2184; 2020. Available online: https://eur-lex.europa.eu/eli/dir/2020/2184/oj (accessed on 11 September 2025).
15. Poothong, S.; Papadopoulou, E.; Padilla-Sánchez, J.A.; et al. Multiple Pathways of Human Exposure to Poly- and Perfluoroalkyl Substances (PFASs): From External Exposure to Human Blood. *Environ. Int.* **2020**, *134*, 105244. https://doi.org/10.1016/j.envint.2019.105244.
16. Ragnarsdóttir, O.; Abdallah, M.A.-E.; Harrad, S. Dermal Uptake: An Important Pathway of Human Exposure to Perfluoroalkyl Substances? *Environ. Pollut.* **2022**, *307*, 119478. https://doi.org/10.1016/j.envpol.2022.119478.
17. Wu, Y.; Romanak, K.; Bruton, T.; et al. Per- and polyfluoroalkyl substances in paired dust and carpets from childcare centers. *Chemosphere* **2020**, *251*, 126771. https://doi.org/10.1016/j.chemosphere.2020.126771.
18. Abraham, K.; Monien, B.H. Transdermal Absorption of ^{13}C4-Perfluorooctanoic Acid (13C4-PFOA) from a Sunscreen in a Male Volunteer—What Could Be the Contribution of Cosmetics to the Internal Exposure of Perfluoroalkyl Substances (PFAS)? *Environ. Int.* **2022**, *169*, 107549. https://doi.org/10.1016/j.envint.2022.107549.
19. Chen, Q.; Yi, S.; Ye, Q.; et al. Insights into the Dermal Absorption, Deposition, and Elimination of Poly- and Perfluoroalkyl Substances in Rats: The Importance of Skin Exposure. *Environ. Sci. Technol.* **2022**, *56*, 16975–16984. https://doi.org/10.1021/acs.est.2c03181.
20. Ragnarsdóttir, O.; Abou-Elwafa Abdallah, M.; Harrad, S. Dermal Bioavailability of Perfluoroalkyl Substances Using in Vitro 3D Human Skin Equivalent Models. *Environ. Int.* **2024**, *188*, 108772. https://doi.org/10.1016/j.envint.2024.108772.
21. Kissel, J.C. The Mismeasure of Dermal Absorption. *J. Expo. Sci. Environ. Epidemiol.* **2011**, *21*, 302–309. https://doi.org/10.1038/jes.2010.22.
22. Fujii, Y.; Harada, K.H.; Koizumi, A. Occurrence of Perfluorinated Carboxylic Acids (PFCAs) in Personal Care Products and Compounding Agents. *Chemosphere* **2013**, *93*, 538–544. https://doi.org/10.1016/j.chemosphere.2013.06.049.
23. Schultes, L.; Vestergren, R.; Volkova, K.; et al. Per- and Polyfluoroalkyl Substances and Fluorine Mass Balance in Cosmetic Products from the Swedish Market: Implications for Environmental Emissions and Human Exposure. *Environ. Sci. Process. Impacts* **2018**, *20*, 1680–1690. https://doi.org/10.1039/C8EM00368H.
24. Whitehead, H.D.; Venier, M.; Wu, Y.; et al. Fluorinated Compounds in North American Cosmetics. *Environ. Sci. Technol. Lett.* **2021**, *8*, 538–544. https://doi.org/10.1021/acs.estlett.1c00240.
25. van der Veen, I.; Hanning, A.-C.; Stare, A.; et al. The Effect of Weathering on Per- and Polyfluoroalkyl Substances (PFASs) from Durable Water Repellent (DWR) Clothing. *Chemosphere* **2020**, *249*, 126100. https://doi.org/10.1016/j.chemosphere.2020.126100.
26. Wu, Y.; Miller, G.Z.; Gearhart, J.; et al. Side-Chain Fluorotelomer-Based Polymers in Children Car Seats. *Environ. Pollut.* **2021**, *268*, 115477. https://doi.org/10.1016/j.envpol.2020.115477.
27. Xia, C.; Diamond, M.L.; Peaslee, G.F.; et al. Per- and Polyfluoroalkyl Substances in North American School Uniforms. *Environ. Sci. Technol.* **2022**, *56*, 13845–13857. https://doi.org/10.1021/acs.est.2c02111.
28. Zhu, H.; Kannan, K. Total Oxidizable Precursor Assay in the Determination of Perfluoroalkyl Acids in Textiles Collected from the United States. *Environ. Pollut.* **2020**, *265*, 114940. https://doi.org/10.1016/j.envpol.2020.114940.
29. Eriksson, U.; Kärrman, A. World-Wide Indoor Exposure to Polyfluoroalkyl Phosphate Esters (PAPs) and Other PFASs in Household Dust. *Environ. Sci. Technol.* **2015**, *49*, 14503–14511. https://doi.org/10.1021/acs.est.5b00679.
30. Harrad, S.; Wemken, N.; Drage, D.S.; et al. Perfluoroalkyl Substances in Drinking Water, Indoor Air and Dust from Ireland: Implications for Human Exposure. *Environ. Sci. Technol.* **2019**, *53*, 13449–13457. https://doi.org/10.1021/acs.est.9b04604.
31. Ertl, H.; Butte, W. Bioaccessibility of Pesticides and Polychlorinated Biphenyls from House Dust: In-Vitro Methods and Human Exposure Assessment. *J. Expo. Sci. Environ. Epidemiol.* **2012**, *22*, 574–583. https://doi.org/10.1038/jes.2012.50.

32. Pawar, G.; Abdallah, M.A.-E.; de Sáa, E.V.; et al. Dermal Bioaccessibility of Flame Retardants from Indoor Dust and the Influence of Topically Applied Cosmetics. *J. Expo. Sci. Environ. Epidemiol.* **2017**, *27*, 100–105. https://doi.org/10.1038/jes.2015.84.
33. Ruby, M.V.; Davis, A.; Schoof, R.; et al. Estimation of Lead and Arsenic Bioavailability Using a Physiologically Based Extraction Test. *Environ. Sci. Technol.* **1996**, *30*, 422–430. https://doi.org/10.1021/es950057z.
34. Abdallah, M.A.-E.; Pawar, G.; Harrad, S. Evaluation of 3D-Human Skin Equivalents for Assessment of Human Dermal Absorption of Some Brominated Flame Retardants. *Environ. Int.* **2015**, *84*, 64–70. https://doi.org/10.1016/j.envint.2015.07.015.
35. Abdallah, M.A.-E.; Tilston, E.; Harrad, S.; et al. In Vitro Assessment of the Bioaccessibility of Brominated Flame Retardants in Indoor Dust Using a Colon Extended Model of the Human Gastrointestinal Tract. *J. Environ. Monit.* **2012**, *14*, 3276. https://doi.org/10.1039/c2em30690e.
36. Ragnarsdóttir, O.; Abdallah, M.A.-E.; Harrad, S. Dermal Bioaccessibility of Perfluoroalkyl Substances from Household Dust; Influence of Topically Applied Cosmetics. *Environ. Res.* **2023**, *238*, 117093. https://doi.org/10.1016/j.envres.2023.117093.
37. Padilla-Sánchez, J.A.; Haug, L.S. A Fast and Sensitive Method for the Simultaneous Analysis of a Wide Range of Per- and Polyfluoroalkyl Substances in Indoor Dust Using on-Line Solid Phase Extraction-Ultrahigh Performance Liquid Chromatography-Time-of-Flight-Mass Spectrometry. *J. Chromatogr. A* **2016**, *1445*, 36–45. https://doi.org/10.1016/j.chroma.2016.03.058.
38. U.S. EPA. *Exposure Factors Handbook*; U.S. EPA: Los Angeles, CA, USA, 2011.
39. Luo, K.; Zeng, D.; Kang, Y.; et al. Dermal Bioaccessibility and Absorption of Polycyclic Aromatic Hydrocarbons (PAHs) in Indoor Dust and Its Implication in Risk Assessment. *Environ. Pollut.* **2020**, *264*, 114829. https://doi.org/10.1016/j.envpol.2020.114829.
40. Zeng, D.; Kang, Y.; Chen, J.; et al. Dermal Bioaccessibility of Plasticizers in Indoor Dust and Clothing. *Sci. Total Environ.* **2019**, *672*, 798–805. https://doi.org/10.1016/j.scitotenv.2019.04.028.
41. Abdallah, M.A.-E.; Harrad, S. Dermal Contact with Furniture Fabrics Is a Significant Pathway of Human Exposure to Brominated Flame Retardants. *Environ. Int.* **2018**, *118*, 26–33. https://doi.org/10.1016/j.envint.2018.05.027.
42. Thépaut, E.; Dirven, H.A.A.M.; Haug, L.S.; et al. Per- and Polyfluoroalkyl Substances in Serum and Associations with Food Consumption and Use of Personal Care Products in the Norwegian Biomonitoring Study from the EU Project EuroMix. *Environ. Res.* **2021**, *195*, 110795. https://doi.org/10.1016/j.envres.2021.110795.
43. Serrano, L.; Iribarne-Durán, L.M.; Suárez, B.; et al. Concentrations of Perfluoroalkyl Substances in Donor Breast Milk in Southern Spain and Their Potential Determinants. *Int. J. Hyg. Environ. Health* **2021**, *236*, 113796. https://doi.org/10.1016/j.ijheh.2021.113796.
44. Greenpeace. Leaving Traces. The Hidden Hazardous Chemicals in Outdoor Gear. Greanpeace Product Test 2016; 2016. Available online: https://www.greenpeace.de/sites/www.greenpeace.de/files/publications/21_01_16_greenpeace_outdoor_report.pdf (accessed on 12 May 2025).
45. Pütz, K.W.; Namazkar, S.; Plassmann, M.; et al. Are Cosmetics a Significant Source of PFAS in Europe? Product Inventories, Chemical Characterization and Emission Estimates. *Environ. Sci. Process. Impacts* **2022**, *24*, 1697–1707. https://doi.org/10.1039/D2EM00123C.
46. Harris, K.J.; Munoz, G.; Woo, V.; et al. Targeted and Suspect Screening of Per- and Polyfluoroalkyl Substances in Cosmetics and Personal Care Products. *Environ. Sci. Technol.* **2022**, *56*, 14594–14604. https://doi.org/10.1021/acs.est.2c02660.