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Comprehensive Study of Human External Exposure to Organophosphate Flame Retardants via Air, Dust, and Hand Wipes: The Importance of Sampling and Assessment Strategy

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Supporting Information

ABSTRACT: We compared the human exposure to organophosphate flame retardants (PFRs) via inhalation, dust ingestion, and dermal absorption using different sampling and assessment strategies. Air (indoor stationary air and personal ambient air), dust (floor dust and surface dust), and hand wipes were sampled from 61 participants and their houses. We found that stationary air contains higher levels of ΣPFRs (median = 163 ng/m^3, IQR = 161 ng/m^3) than personal air (median = 44 ng/m^3, IQR = 55 ng/m^3), suggesting that the stationary air sample could generate a larger bias for inhalation exposure assessment. Tris(chloropropyl) phosphate isomers (ΣTCPP) accounted for over 80% of ΣPFRs in both stationary and personal air. PFRs were frequently detected in both surface dust (ΣPFRs median = 33 100 ng/g, IQR = 62 300 ng/g) and floor dust (ΣPFRs median = 20 500 ng/g, IQR = 30 300 ng/g). Tris(2-butoxylethyl) phosphate (TBOEP) accounted for 40% and 60% of ΣPFRs in surface and floor dust, respectively, followed by ΣTCPP (30% and 20%, respectively). TBOEP (median = 46 ng, IQR = 69 ng) and ΣTCPP (median = 37 ng, IQR = 49 ng) were also frequently detected in hand wipe samples. For the first time, a comprehensive assessment of human exposure to PFRs via inhalation, dust ingestion, and dermal absorption was conducted with individual personal data rather than reference factors of the general population. Inhalation seems to be the major exposure pathway for ΣTCPP and tris(2-chloroethyl) phosphate (TCEP), while participants had higher exposure to TBOEP and triphenyl phosphate (TPHP) via dust ingestion. Estimated exposure to ΣPFRs was the highest with stationary air inhalation (median =34 ng·kg bw^{-1}·day^{-1}, IQR = 38 ng·kg bw^{-1}·day^{-1}), followed by surface dust ingestion (median = 13 ng·kg bw^{-1}·day^{-1}, IQR = 28 ng·kg bw^{-1}·day^{-1}), floor dust ingestion and personal air inhalation. The median dermal exposure on hand wipes was 0.32 ng·kg bw^{-1}·day^{-1} (IQR = 0.58 ng·kg bw^{-1}·day^{-1}) for ΣTCPP. The selection of sampling and assessment strategies could significantly affect the results of exposure assessment.

INTRODUCTION

Flame retardants (FRs) are commonly added to construction materials and consumer products to fulfill fire safety criteria and regulations of different countries.[1−3] Polybrominated diphenyl ethers (PBDEs) were well-known and widely used FRs. Due to their persistency and toxicity, PBDEs are phased out gradually.[2,4] Stockholm Convention has listed Penta-BDE and Octa-BDE commercial mixtures in Annex A as substances of elimination,[5,6] while the proposal of listing Deca-BDE is under review. Recently, many alternative FRs, including dechlorane plus (DP), emerging brominated flame retardants (EBFRs), and organophosphate flame retardants (PFRs), are replacing the market-share of PBDEs. PFRs are widely used as FRs in textiles, plastics, foams, lubricants, paints, and so forth.[1] They accounted for about 11.5% of world consumption of FRs (200 000 tones, 800 million USD by value), while, in the EU, PFRs accounted for 20% of total FR consumption in 2006.[1,7] Some PFRs, like triphenyl phosphate (TPHP), are also used as plasticizers and in nail polish, while tris(2-butoxyethyl) phosphate (TBOEP) has been used in floor wax.[1,8] 2-Ethylhexyl diphenyl phosphate could be found in PVC, rubber, photo films, paints, pigment dispersions, adhesives, textile, cable coatings, and food packaging.[9] As FRs are usually used as additives in commercial products, they can be emitted from the treated products, contaminating indoor and outdoor environment.[10−12] PFRs have been reported in air, dust, water, sediment, and soil. In the indoor environment, the PFR contamination levels are comparable, or even higher, to PBDEs.[3,4,11−16] Recently, low levels of PFRs have also been found in food and biota samples,[17−20] which raise serious concerns of PFR pollution in environment.

Some PFRs, such as tris(2-chloroethyl) phosphate (TCEP), tris (1,3-dichloro-2-propyl) phosphate (TDCIPP) and TPHP,

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are suspected to be carcinogenic, mutagenic, or neurotoxic.[1,21] TDCIPP was found to weaken the fecundity and development of *Daphnia magna*.[22] Tricresyl phosphate (TMPP) could cause the organophosphate-induced delayed neuropathy.[23] Moreover, PFR levels in house dust were associated with the altered hormone levels and decrease of semen quality.[24] Recently, PFRs have been found in breast milk,[25,26] implying a potential health threat to newborns. Also, significant correlations have been reported between air/dust samples and human hair.[27] PFRs are less persistent and have lower log K_{ow} than PBDEs, thus, are easier metabolized and further excreted via urine.[28−30] Some PFR metabolites in urine were associated with their parent compounds in indoor dust or hand wipes,[2,29−31] indicating a common exposure of the general public to PFRs

Stationary sampling of indoor air has been commonly used to study human inhalation exposure to FRs.[3,13,32] However, the accuracy of this technique for exposure assessment through inhalation has seldom been evaluated. Carignan et al.[32] collected stationary air samples in a gym and found up to six-fold higher levels of PBDEs in the air collected near foam pits than in the air collected at the opposite side of the room. This indicates that the location of the stationary pump has a large impact on the measured concentrations. Moreover, human daily activities are not limited to these stationary sampling sites, but occur in several different microenvironments, which in most cases are not sampled. Therefore, the accuracy in the exposure assessment through inhalation using the stationary air sampling technique could be criticized.

Besides the traditional indoor air sampling with stationary pumps, half of our participants also provided synchronized personal ambient air (personal air) samples by carrying portable air samplers for 24 h in order to mimic real-life inhalation. To our knowledge, this is the first time that sampling of personal ambient air and stationary air have been compared to study human exposure to PFRs through inhalation. Inhalation and dust ingestion are considered as two important pathways of human exposure to PFRs.[3,13,28,33,34] The presence of FRs on hands could be linked to exposure via dermal absorption and hand-to-mouth transmission,[2,28,35,36] but information about transport of on-skin PFR is still limited. So far, it is still unclear which pathway is more important for PFR exposure. In this study, we present a comprehensive assessment of human exposure to PFRs through the indoor environment using air, dust, and hand wipes. To compare PFR profiles and levels, we have sampled both floor and surface dust.

MATERIALS AND METHODS

Sample Collection. Details about the sampling campaign of the A-TEAM cohort are described in Papadoupolou et al.[37] In brief, all samples were collected in Oslo (Norway) during weekdays. Sixty-one participants (all adults) were recruited to assess their exposure to PFRs, EBFRs, phthalates, and per- and polyfluoroalkyl substances. Each participant was asked to provide a set of samples during a 24 h surveillance, including personal and stationary air, indoor dust (from floor, surface, and vacuum cleaner bags), and hand wipes. Information about personal physical condition, home environment, and other lifestyle characteristics were collected via questionnaires.

The air sampling procedure was slightly modified from a validated method for analyzing PFRs and phthalates in air.[38] Stationary air samples were collected from the living room of each participant for 24 h with a SKC Legacy Low volume Pump (SKC Inc., Eight Four, PA, U.S.) connected with four ENV+ SPE cartridges (200 mg, 6 mL, Biotage, Uppsala, Sweden) in parallel (Figure SI-1 of the Supporting Information, SI). A portable SKC 224-PCMTX4 pump (SKC Inc., Eight Four, PA, U.S.) was carried by the participants for 24 h to mimic his/her inhalation of ambient air. An ENV+ SPE cartridge (1 g, 25 mL, Biotage Uppsala, Sweden), connected with portable pump, was fixed above the chest of the participant with about 30 cm distance from his/her nose (Figure SI-2). All cartridges were precleaned with acetone and sealed with foil before use. The airflow, for both personal and stationary air collection, was set to 1−1.2 L/min for each cartridge. The exact starting and finishing time of sampling, as well as the airflow, were recorded for the calculation of the sampled volume of air.

Floor dust was sampled from the participants' living rooms using an industrial vacuum cleaner (GM 80P, Nilfisk, Penith, U.K.) connected with a dust-sampling filter (KTM AB, Bålsta, Sweden), while surface dust was collected from the surface of furniture and decoration items in the same rooms that were at least 0.5 m above the floor. The living room was not vacuumed for 2−3 weeks before dust collection. Once in the lab, hair, food crumbs, stones, and other large particles were carefully removed from the dust samples. All dust was further aliquoted into four parts for different analyses.

Four hand wipes (3 × 3 in^2 Sterile Gauze Pads, Swift First Aid Inc., Valencia, CA, U.S.) were collected throughout the day for each participant, and the noontime sample was assigned for PFR analysis. Participants were asked to not wash hands for at least 1 h before the hand wipe collection. Both hands of the participant were thoroughly wiped using two isopropanol-infused gauze pads (one on each hand). After collection, all samples were properly packed, aliquoted, sealed, and then stored in −20 °C until analysis. Information about sample treatment and analysis is described in SI-Section 2.

QA/QC and Data Analysis. To avoid contamination, glassware was baked at 400 °C overnight before use. Results were blank subtracted if necessary. Method limit of quantifications (MLQs) were presented in Table 1. Air sampling cartridges was tested for 6, 12, and 24 h, respectively, and no breakthrough of PFRs was observed (Table SI-2). ENV+ cartridges (air samplers) were selected according to literature.[38] Spiking tests on ENV+ cartridges also achieved sufficiently elution and high recoveries for PFRs with the air extraction method. Standard reference material - SRM2585 (NIST, U.S.) was used as a quality control for dust analysis (n = 5, Table SI-3), and results were within 15% of the assigned or indicative values. Hand wipes were spiked with standards (n = 3), obtaining 70−130% recoveries. More information about QA/QC is provided in SI-Section 3. Statistical analysis was performed with Excel and JMP Pro 11. For PFR levels <MLQ, a value equal to 1/2 × MLQ was used for exposure assessment. Further statistical analysis (principal component analysis and Spearman correlation) was performed only for PFRs with detection frequency (DF) > 50%.

RESULTS AND DISCUSSION

Most target PFRs, except TNBP, were detected in at least three out of the five matrices (Table 1). The median levels of ΣPFRs in personal air, stationary air, floor dust, surface dust, and hand wipes were 44 ng/m^3, 163 ng/m^3, 20 500 ng/g, 33 100 ng/g, and 192 ng, respectively. ΣTCPP was frequently detected in all matrices (DF > 85%), indicating its ubiquitous presence in indoor environment and its wide application in commercial products. Tris(1-chloro-2-propyl) phosphate (TCIPP) was the major TCPP isomer detected. TBOEP was commonly

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Table 1. PFR Levels, Detection Frequencies and MLQ in Air, Dust, and Hand Wipe Samples[a,b]

		TEHP	TNBP	EHDPHP	TCEP	TBOEP	TPHP	ΣTMPP	TDCIPP	ΣTCPP	ΣPFRs
personal ambient air (ng/m^3)	range	<MLQ-22	<MLQ-28	<MLQ-1.3	<MLQ-8.1	<MLQ-26	<MLQ-5.6	<MLQ	<MLQ-12	10–172	12–183
	median	<MLQ	<MLQ	<MLQ	3	<MLQ	1	<MLQ	<MLQ	28	44
	MLQ	5.4	2.8	1.2	1.0	6.9	0.3	1.5	1.0	1.4	
	DF (n = 31)	19%	35%	3%	77%	42%	74%	0%	16%	100%	
indoor stationary air (ng/m^3)	range	<MLQ-42	<MLQ-119	<MLQ-8	<MLQ-76	<MLQ-16	<MLQ-9	<MLQ	<MLQ-31	<MLQ-987	28–1018
	median	<MLQ	14	<MLQ	3	<MLQ	1	<MLQ	<MLQ	128	163
	MLQ	4.6	2.7	0.6	0.9	5.6	0.3	1.5	0.9	1.2	
	DF (n = 58)	10%	98%	19%	93%	3%	88%	0%	2%	98%	
floor dust (ng/g)	range	<MLQ-3980	<MLQ-2950	83–12500	<MLQ-350 000	727–311000	155–276000	<MLQ-7520	<MLQ-6000	<MLQ-145 000	3662–505 000
	median	401	<MLQ	420	435	8146	722	179	397	1997	20 500
	MLQ	140	190	3	170	90	5	20	55	55	
	DF (n = 61)	82%	23%	100%	77%	100%	100%	95%	98%	95%	
surface dust (ng/g)	range	<MLQ-450 000	<MLQ-3030	212–30200	<MLQ-15 200	<MLQ-540 000	326–956 000	<MLQ-30 100	<MLQ-366 000	<MLQ-498 000	5800–1490 000
	median	710	<MLQ	617	455	6796	1228	334	1130	5241	33 100
	MLQ	140	190	3	170	90	5	20	55	55	
	DF (n = 61)	93%	21%	100%	75%	98%	100%	97%	98%	98%	
hand wipe (ng)	range	<MLD-191	<MLD-76	<MLD-65	<MLD-76	<MLD-921	<MLD	<MLD-14 100	<MLD-432	<MLD-261	20–14 100
	median	<MLD	<MLD	11	<MLD	46	<MLD	<MLD	<MLD	37	192
	MLQ	8	11	4	4	8	100	1	34	15	
	DF (n = 55)	47%	13%	75%	49%	78%	0%	42%	29%	87%	

[a]DF: detection frequency. [b]MLQ: method limit of quantification.

detected in high concentrations in dust and hand wipes, but not in air.

PFRs in Floor and Surface Dust. Most PFRs, except TNBP, were detected in both floor and surface dust with DF > 70% (Table 1), indicating that dust is an ideal reservoir and potential indicator for indoor organic contaminants. TBOEP and ΣTCPP were the major PFRs in dust, accounting for approximately 90% of ΣPFRs in both dust types (Figure 1).

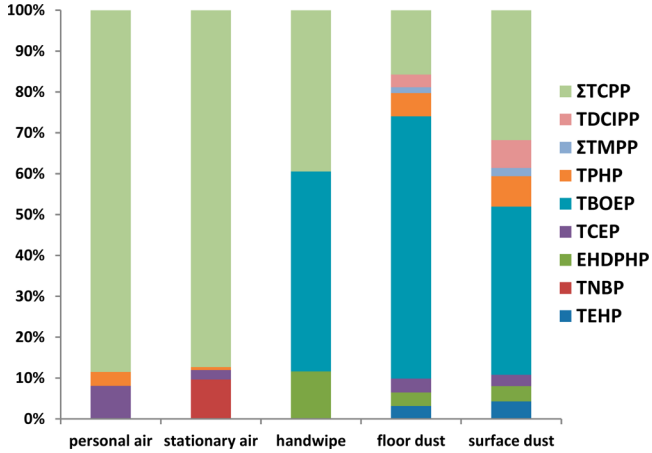

Figure 1. Composition of individual PFRs in personal air, stationary air, surface dust, floor dust and hand wipes from Norwegian households/participants. The proportions were calculated with the median levels of individual compounds in the five types of samples.

A similar PFR profile with slightly higher levels was reported by Cequier et al.[3] TBOEP had the highest level in both surface (median 6800 ng/g, IQR 11 600 ng/g) and floor dust (median 8100 ng/g, IQR 13 300 ng/g), respectively, while ΣTCPP accounted for approximately 30% of ΣPFRs in surface dust (median 5240 ng/g, IQR 16 400 ng/g) and 20% in floor dust (median 2000 ng/g; IQR 2530 ng/g), respectively. High levels of TBOEP and ΣTCPP in dust suggest their common usage in commercial products. ΣPFR levels in floor and surface dust ranged from 3660–505 000 ng/g and 5820–1 490 000 ng/g, respectively, and were higher than in Belgium house dust (median 13 000 ng/g), Kuwait (median 6555 ng/g) and Pakistan (median 575 ng/g),[15,39] and were similar or higher than the indoor dust from e-waste recycling workshops (and houses nearby e-waste recycling sites) in China.[11] Because all PBDEs (including Deca-BDE) have been banned in Norway since 2008,[3] it is not unexpected to see more alternative FRs in Norwegian indoor dust.

Floor dust had significant lower PFR levels than surface dust (Table SI-5, Wilcoxon signed rank, n = 61), with the exception of TBOEP and TCEP, which showed no significant difference between two types of dust. This could be due to the higher content of sand, dirt and other large particles (like food residue and floc) in floor dust, which may dilute the PFR levels. Moreover, surface dust has finer particle size, which have higher capability of adsorbing organic contaminants from ambient environment due to a larger surface area.[40−42] The migration pathways of FRs to dust are possibly: (1) tearing or abrasion (2) volatilization-adsorption.[41] Since TBOEP and TCEP have the lowest and highest vapor pressure, respectively (Table SI-1), TBOEP possibly migrates to dust through the process (1), while TCEP are more likely to migrate via the second process (2). Further studies should test this hypothesis. Significant

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Table 2. Spearman's Rank Correlations between Floor Dust and Surface Dust, Floor Dust, and Stationary Air, Surface Dust and Stationary Air, and Personal Air and Stationary Air[a,b,c]

	floor dust vs surface dust (n = 61 pairs)		floor dust vs stationary air (n = 58 pairs)		surface dust vs stationary air (n = 58 pairs)		personal air vs stationary air (n = 29 pairs)	
	Rho	p	Rho	p	Rho	p	Rho	p
TEHP	0.4	**0.002**	NA	NA	NA	NA	NA	NA
TNBP	NA	NA	NA	NA	NA	NA	NA	NA
EHDPHP	0.55	**0.0001**	NA	NA	NA	NA	NA	NA
TCEP	0.65	**0.0001**	0.51	**0.0001**	0.62	**0.0001**	0.3774	**0.0454**
TBOEP	0.37	**0.004**	NA	NA	NA	NA	NA	NA
TPHP	0.37	**0.0045**	0.09	0.49	0.16	0.23	0.0371	0.8701
ΣTMPP	0.37	**0.0045**	NA	NA	NA	NA	NA	NA
TDCIPP	0.049	**0.0001**	NA	NA	NA	NA	NA	NA
ΣTCPP	0.62	**0.0001**	0.44	**0.0005**	0.3	**0.022**	0.2987	0.1157

[a]No correlations of personal air vs surface and personal air vs floor dust were found (p > 0.1 for all compounds). [b]NA, not available due to low detection frequency (DF < 50%). [c]p < 0.05 indicates significant correlation between two data set.

underestimation would be seen for individual PFRs if the estimation of human exposure is based only on floor dust, especially for adults who are more likely to be exposed to surface dust than floor dust.

Significant differences were observed between the two types of dust for TBOEP and ΣTCPP, but not for the other PFRs (Figure 1 and Table SI-5). Also Cequier et al.[3] have reported no differences in the ΣPFR levels between surface and floor dust. Due to its dominance in stationary air, TCPP may have a higher absorption on finer particles that contributed to its increasing proportion in surface dust. As TCPP and TBOEP are the major components in the two types of dust, accounting for over 70% of ΣPFR, selection of dust sampling would lead to different exposure profiles for these PFRs. We would suggest selecting different dusts for the exposure assessment of different populations, e.g., floor dust for toddlers and surface dust for adults. For all frequently detected PFRs, positive and significant correlations were observed between floor and surface dust samples collected from the same houses (Table 2), which suggests that the two types of dust refer to similar contamination sources.

PFRs in Personal and Stationary Air. We compared the stationary air sampling with the personal air sampling. Personal air was already used to assess the inhalation exposure to volatile PFASs of professional ski waxers.[43] It was also used to study the personal inhalation exposure to PBDEs and chlorinated PFRs.[33,44] Personal pumps have constant flow, which might not completely mimic the variation of human breathing, but they lead to more accurate inhalation exposure assessment than the stationary sampling.

Due to some sampling limitations (cartridge backpressure, noise, four parallels cartridges per site, etc.), we had to use lower airflow (1.2 L/min air through each cartridge, 4.8 L/min total flow) for the stationary air sampling. Similar to personal air, no replicates were collected, since the portable pump only allowed a total flow of 1.2 L/min, and samples from only 30 individuals were collected. Therefore, the MLQs for air samples were higher than that in Cequier et al.,[3] who used a 12 L/min flow for stationary air sampling. Cequier et al.[3] used PUF as sampler, which gives lower backpressure than SPE cartridges, allowing higher sampling flows. However, the extraction of PUF samplers was more time-consuming and laborious.

Significantly higher PFR concentrations (Table 1 and Table SI-5) were found in stationary air (median = 163 ng/m^3, IQR = 161 ng/m^3) than personal air (median = 44 ng/m^3, IQR = 55 ng/m^3). ΣTCPP, TPHP, and TCEP were frequently detected in both types of air samples (DF range: 74%−100%), while ΣTCPP was the predominant PFR (Figure 1). Median level of ΣTCPP in stationary air (median = 128 ng/m^3, IQR = 175 ng/m^3) was five folds higher than in personal air. TNBP and TCEP were the second dominating PFRs in stationary air (median = 14.1 ng/m^3, IQR = 9.0 ng/m^3) and personal air (median = 2.6 ng/m^3, IQR = 2.0 ng/m^3), respectively. Slightly lower levels of PFRs have been reported in household stationary air from Norway (median levels of TCIPP, TCEP, and TPHP are 42, 5, and 0.3 ng/m^3, respectively),[3] while higher ΣPFR levels were found in Swedish indoor air from offices (mean = 3700 ng/m^3) and daycare centers (mean = 2000 ng/m^3).[13] ΣTMPP and EHDPHP had low DF, which may partly due to their lower volatilities. No significant difference was found between personal and stationary air for TPHP in our study.

Stationary and personal air samples from a participant were collected simultaneously (24 h) during weekdays, so most of our participants probably spent fewer hours in living rooms, but more time at work and other environments (e.g., outdoor and bedroom). PFR levels and profiles in personal air might thus be different from stationary air. Levels of TNBP, TCEP, and ΣTCPP were higher in stationary air than in personal air. The other PFRs had also higher DFs and maximum levels in stationary air (Table 1), except for TBOEP. Differences in the concentrations of various pollutants and particulate matter between personal air and stationary air sampling have been reported for exposure assessments in several studies.[45−47] Obviously, the use of stationary air sampling may generate significant bias during exposure assessment as compared to personal air sampling. Usually, public places, like offices and cinemas, have more strict fire safety code and more intensive FR usage, which should lead to higher FR levels in air. Allen et al.[44] reported higher level of PBDEs in personal air than stationary air in at home, which is different from what we observed for PFRs. As all participants worked in old buildings, a possible explanation would be that PFRs are not intensively applied in their working environmental comparing to their homes. Since Norway was one of the first countries to phase out PBDEs,[3] more PFRs might have been applied in recently purchased products; homes usually have more new products. Such hypothesis will possibly be confirmed in a parallel study on EBFRs in dust, air, and hand wipes by a partner of the A-TEAM project.

Further statistical analysis was performed for three compounds with DF > 50% and for participants who provide both types of air samples. Principal components analysis (PCA)

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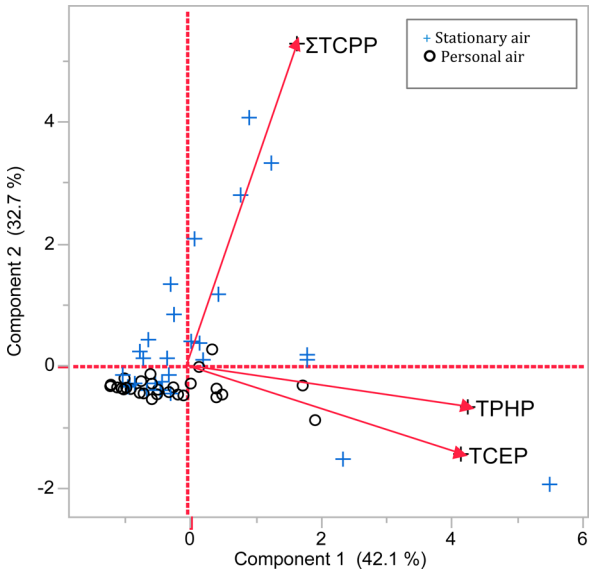

Figure 2. Principal components analysis for three PFRs (ΣTCPP, TCEP, and TPHP) in personal ambient air and indoor stationary air samples.

(Figure 2) shows distinct profiles of the two types of air samples: personal air data are less scattered than the stationary air data. Since people have different preferences to decorate and furnish their homes, it is not unexpected to see such variations in the stationary air data. However, personal air highly depends on personal activities. Its less scattered PCA profile comparing to stationary air suggests that our participants were exposed to some similar contamination sources for these three compounds. There are some hypothetical explanations for this: (1) since our participants were recruited from the same organization, they might have similar exposures during working hours; (2) participants might be surrounded by some common products during daily life, such as bed mattresses, office products and cars, which may have similar PFR emissions. ΣTCPP was the major factor that influenced the PFR profiles in stationary air, while personal air profiles were under the coinfluence of TCEP and TPHP. PCA results may also imply that a personal air sampling technique might better represent the PFR inhalation exposure from a certain occupation/work environment, while the stationary air could be used for mapping air profiles among different environments.

A significant Spearman's correlation was found between two types of air for TCEP only (Rho = 0.35, p = 0.045; Table 2). Figure SI-6 shows the linear regression of logTCEP levels between the two types of air, implying that the TCEP variation in personal air samples may relate to its level in living room air. However, no such correlation was found for other PFRs between the two types of air. Furthermore, correlations between dust and indoor stationary air were also tested for TPHP, TCEP, and ΣTCPP (Table 2). TCEP and ΣTCPP had positive and significant correlations between stationary air and floor dust, as well as between stationary air and surface dust, but no air-dust correlation was found for TPHP. Similar air−dust correlations have also been reported by other researchers.[3,13] It seems that chlorinated PFRs have more significant air−dust correlations than TPHP, maybe due to their higher volatilities than TPHP. No significant correlations (p > 0.1 for all PFRs) were found between indoor dust and personal air (n = 31).

The impact of house size and family size to indoor PFR contamination were also studied. For families with two children or more, the ΣPFR level in the stationary air has a clear positive correlation with house size (Figure SI-8). No clear correlation was found for families with one or no child. Participants from larger families (with two children or more) fall into the similar age group (middle-age) and possibly have similar marriage and economical status, which reduce the statistical influence of these factors. Participants from smaller families cover a wider age range and marriage status and possibly have different lifestyles which would introduce more variation to the data set. No clear correlation between house size or family size with ΣPFR levels in dust was observed.

PFR in Hand Wipes and Correlations with Other Matrices. Hand wipe extracts were fractionated on APS cartridges to remove the lipid interferences, achieving a better cleanup than Florisil cartridges according to our in-house comparison. All target compounds could be detected in hand wipes, while only two to three PFRs were reported in other studies.[2,36] ΣPFR levels found in hand wipes ranged from 20 to 14 100 ng (IQR = 252 ng). Three compounds, namely ΣTCPP, TBOEP, and EHDPHP, had DFs > 60%. Similar to dust, TBOEP was found to have the highest level in hand wipes (median = 46 ng, IQR = 69 ng), followed by ΣTCPP (median 35 ng, IQR = 49 ng) and EHDPHP (median = 11 ng, IQR = 17 ng). TEHP, TCEP, and TMPP were detected in 42−49% of the samples, while the rest of the PFRs had DF < 30%. Due to the rather high TPHP background in the blank samples, its information in hand wipes was not usable.

Hand wipes have been considered a good indicator for indoor contamination.[32,36] Hoffman et al.[2] reported <MLQ−547 ng of TDCIPP in their hand wipes, while Stapleton et al.[36] found <MLQ−530 ng of TDCIPP in hand wipes. While different from our results, TDCIPP had higher levels and DFs than TCPP in hand wipes collected from U.S. individuals, while TDCIPP was found to have similar or higher levels than TCPP in air and dust.[2,4,32,36] It is possible that TDCIPP has a lower application rate in Norway than in U.S. The use of TCPP in similar applications as a cheaper alternative to TDCIPP might me another reason.

Interestingly, EHDPHP was the third most frequently detected PFRs in hand wipes from the present study (median 11 ng, DF = 75%, IQR = 17 ng), but neither air, nor dust samples contained high levels of EHDPHP. Since our hand wipes were collected at noon, when most of the participants were at work, this might be caused by higher EHDPHP contamination in offices or migration from common office products. Unfortunately, this hypothesis could not be tested, since neither dust, nor product wipe was specifically collected from the offices. Also, the lower MLQ of EHDPHP for hand wipes comparing to other PFRs could also contribute to its higher DF.

Correlations between hand wipes and air have been reported for PBDEs,[48] but not yet for PFRs. A significant Spearman correlation was found between personal air and hand wipe levels of ΣTCPP (Rho = 0.45, p = 0.021). Figure SI-7 also shows the linear regression for logTCPP levels between these two types of samples. This significant correlation indicates that it might be possible to predict dermal accumulation of ΣTCPP, maybe even for other PFRs, using its level in the personal air. Since only ΣTCPP was frequently detected in both hand wipes and personal air, we did not perform statistics for the other PFRs.

Comparing Human PFR Exposure Using Different Assessment Strategies. Figure 3 shows the human PFR exposure estimated based on different sampling procedures and using data from each individual participant (body weight, gender

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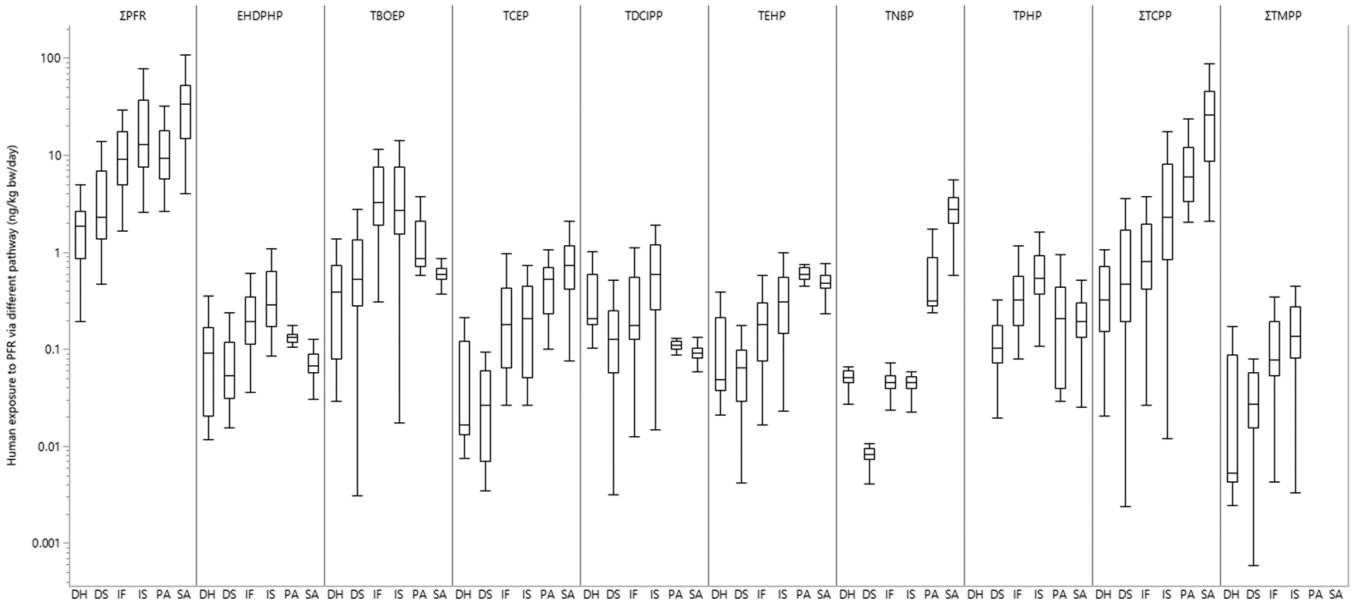

Figure 3. Estimated adults exposure to individual PFRs via different pathways (unit: ng·kg bw^{-1}·day^{-1}), including ingestion of floor dust (IF) and surface dust (IS), inhalation via personal air (PA) and stationary air (SA), and dermal absorption based on hand wipe (DH) and based on surface dust attachment (DS). The assessment was calculated based on physical data from each individual participant (body weight, gender, age, etc., Table SI-5). Since dermal accessibility rates (under sweat/sebum 1:1 condition) are only available for TCEP (10.4%), TCPP (17.4%) and TDCIPP (18.6%), dermal exposure for other PFRs were estimated with the average dermal accessibility rate (15.4%) of TCEP, TCPP, and TDCIPP. Estimated exposures using this value are only for the purpose of comparing assessment strategy. For more details, see SI.

factor, age factor, etc., Tables SI-6 and SI-7). This is also the first study performing a comprehensive external human exposure assessment to PFR using personalized data. Details of assessments could be found in the SI.

Estimated inhalation exposure to ΣPFR based on stationary air (SA) has the highest median value among all pathways (median =34 ng·kg bw^{-1}·day^{-1}, IQR = 38 ng·kg bw^{-1}·day^{-1}), followed by surface dust ingestion (IS, median =13 ng·kg bw^{-1}·day^{-1}, IQR = 28 ng·kg bw^{-1}·day^{-1}), floor dust ingestion (IF), inhalation of personal air (PA), dermal absorption via surface dust deposited on hands (DS), and dermal absorption assessed with hand wipes (DH). Since hand wipes were collected at noon, it probably reflected mostly the exposure at work. The median of SA was about 4-fold higher the PA median, indicating that stationary air sampling could generate a bias in the exposure assessment when compared to personal air sampling. Moreover, individual PFRs have different major pathways. Our findings show that for the heavier PFRs humans are mainly exposed via dust ingestion, such as TBOEP, TPHP, and TMPP, while inhalation is the major exposure pathway for volatile compounds, like TCEP and TCPP.

Until recently, assessments of human exposure to FRs seldom included dermal exposure as possible pathway, but rather focused on the hand-mouth-contact pathways.[28,36,49] Recently, several publications have raised attention to dermal exposure to FRs.[35,50−52] Pawar et al.[50] reported the dermal accessibility through dust using in vitro skin models for TCEP, TDCIPP, and TCPP, finding dermal exposure to PFR via dust to be lower than, but still considerable, exposure via dust ingestion, which is in accordance with our results. However, they estimated the exposure only for skin-adhered dust, while the hand/skin washing frequency was not considered. In our assessment (Figure 3 and SI-Section 4), for the first time, we compared dermal absorption via surface dust (considering a fix amount of dust deposited onto skin) with adsorption assessed via individual hand wipes. Different from other studies,[50,51] we only consider the skin area of the hands, as it is more likely to be in contact with ambient environment than body skin, but a four-time daily hand-wash-frequency[53] was included. Considering only dermal exposure from the hand may lead to an underestimation, as dermal exposure may also occur through contact with textiles.[54] Since body wipes were not collected in this study and since exposure from clothing may have a completely different PFR profile than hand wipes, we have decided not to extend the exposure assessment to the entire body.

So far, dermal accessibility information (sweat/sebum 1:1) are only available for TCEP (10.4%), TCPP (17.4%), and TDCIPP (18.6%), dermal exposure for other PFRs were estimated with the average dermal accessibility rate (15.4%). The estimated exposures using this average value are, thus, only for the purpose of comparing assessment strategies. The median DH for ΣTCPP and TCEP were 0.32 ng·kg bw^{-1}·day^{-1} (IQR = 0.58 ng·kg bw^{-1}·day^{-1}) and 0.02 ng·kg bw^{-1}·day^{-1} (IQR = 0.11 ng·kg bw^{-1}·day^{-1}), respectively, which is lower than those for DS. Deductively, DS might add a larger bias to dermal exposure assessment than DH, because it assumes a fixed amount of dust attached on skin (0.01 mg/cm^2). Therefore, hand wipes could be a valuable tool for estimating dermal absorption of PFRs, since it represents the real scenario of skin contamination and it is not influenced by hand sizes.

In the assessment (Figure SI-9 and Table SI-8), personal physical data (like body weight) from individual participants and relevant exposure factors (inhalation rate for different age-groups, hand-size for different genders; Table SI-6) were introduced to compare with traditional assessment strategy—applying general population factors for estimation (e.g., all body weight = 70 kg,[11,39,55] all inhalation rate = 16 m^3/day[53]). For our participant, the assessment performed with personalized data/factors was significantly lower than that performed with general population data for DS, PA, and, especially, SA, which was 20% less

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with personalized estimation. No significant differences were observed between the two strategies for IS, IF, and DH (Table SI-9). Apparently, the mean body weight for the participants was close to 70 kg, so it would not have an impact in the statistics. For IS, IF, and DH, no other personalized data were applied for assessment beside body weight, so their estimated exposure would not be different from using general population data.

In contrast, inhalation rates for individual genders and age groups were applied for PA and SA in the personalized assessment, while hand-sizes for genders were introduced for DS. For specific participant groups or small populations, the use of personal factors during assessment, which might not be normally distributed, might lead to statistically significant differences in the results using general population factors. Although personalized exposure assessment could generate exposure assessment for individuals, it requires large amount of extra work during sampling and data analysis; while the traditional assessment strategy does not require this, since it only provides estimation for the general population. For PFR exposure via dust ingestion and dermal exposure assessed with hand wipes, it might not be necessary to apply personalized assessment strategy. For inhalation exposure or dermal absorption via dust attachment, personalized assessment strategy might reduce the bias of exposure estimation.

This study has found differences in the exposure pathways for the various PFRs. The selection of dust sampling strategy should be based on the target population groups. Personal air sampling is likely to result in a more accurate inhalation exposure than using stationary air measurements. Taking into account that different results were obtained when using personalized data compared to general population data, personalized data are recommended for exposure assessment. Current studies are focusing on the exposure to PFRs through diet, as well as on the assessment of internal exposure using samples of urine and blood on the same population. Along with the results of this study, a complete and comprehensive assessment of human external and internal exposure to PFRs will be possible by using a modeling approach.

■ ASSOCIATED CONTENT

Supporting Information

The Supporting Information is available free of charge on the ACS Publications website at DOI: 10.1021/acs.est.6b00246.

Details about the (1) air sampling devices and target compounds, (2) sample preparation and analysis, (3) QA/QC, and (4) human exposure assessment (PDF)

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Notes

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