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Organophosphate Flame Retardants in Indoor Dust from Egypt: Implications for Human Exposure

Mohamed Abou-Elwafa Abdallah[†,‡,*] and Adrian Covaci[§]

[†]Division of Environmental Health and Risk Management, School of Geography, Earth and Environmental Sciences, University of Birmingham, Birmingham B15 2TT, United Kingdom

[‡]Department of Analytical Chemistry, Faculty of Pharmacy, Assiut University, 71526 Assiut, Egypt

[§]Toxicological Center, University of Antwerp, Universiteitsplein 1 2610 Wilrijk, Belgium

Ⓢ *Supporting Information*

ABSTRACT: Organophosphate flame retardants (PFRs) have been proposed as alternatives for the phased out PBDE formulations. However, there exists no information on indoor dust contamination with PFRs in Africa. In this study, we report—*for the first time*—on levels and profiles of PFRs in dust samples from Egyptian houses ($n = 20$), offices ($n = 20$), cars ($n = 20$), and public microenvironments (PMEs; $n = 11$). Results revealed that PFR levels in Egyptian indoor dust are among the lowest reported worldwide. This may be attributed to less strict fire-safety standards and lack of regulatory actions against PBDEs. Triphenylphosphate was the only PFR detected in all samples with highest average concentration (386 ng g^{-1}). While tris-2-chloroethyl phosphate, tris(1-chloro-2-propyl)phosphate and tris-1,3-dichloropropylphosphate showed higher detection frequency (DF = 69%, 57%, and 56%; average = 233, 229, and 144 ng g^{-1}, respectively), tri(2-butoxyexthyl)phosphate (37%; 294 ng g^{-1}) displayed the second highest average concentration. Statistical analysis revealed significantly ($P < 0.05$) higher concentrations of ΣPFRs in cars (average = 1011 ng g^{-1}) and PMEs (2167 ng g^{-1}) than in houses (310 ng g^{-1}) and offices (450 ng g^{-1}). Estimated exposures of adults and toddlers to PFRs via dust ingestion were much lower than the reported reference doses, indicating no immediate health risk to the Egyptian population.
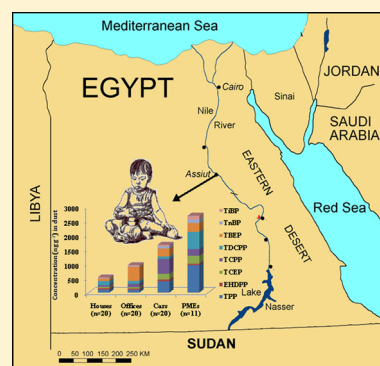
■ INTRODUCTION

The persistent, bioaccumulative, and toxic properties of polybrominated diphenyl ethers (PBDEs) have led to various bans on their production and usage as additive flame retardants (FRs) worldwide.[1,2] As a result, PBDE consumption in Europe has decreased from 51 150 to 45 000 tons between 2004 and 2006.[3] In 2009, PBDE congeners associated with penta- and octa-BDE commercial formulations were listed as persistent organic pollutants (POPs) under Annex A of the UNEP Stockholm Convention on POPs,[4] which prohibits further production and usage of these chemicals and provides guidance within a specified time frame for the recycling and use/reuse of articles that may contain prohibited PBDEs. These restrictions on the production and usage of PBDEs have led to the application of alternative FRs to meet the fire safety regulations for various consumer products. Among those alternatives, organophosphate flame retardants (PFRs) have been associated recently with a variety of applications in a wide range of products.[5] In contrast to PBDEs, the EU market demand for PFRs has increased from 83 700 tons in 2004 to 91 000 tons in 2006 accounting for 20% of the European FR market consumption in 2006.[3] PFRs can be applied to a wide range of polymers due to their compatibility with other processing chemicals and ease of use.[6] Tris-2-chloroethyl phosphate (TCEP), tris(1-chloro-2-propyl) phosphate (TCIPP) and tris-1,3-dichloropropyl phosphate (TDCPP) are used as flame retardants in flexible and rigid in polyurethane foams (PUFs) deployed in furniture, car upholstery and related products.[7] Triphenyl phosphate (TPHP) can be used as both FR and plasticizer in PVC, cellulosic polymers, thermoplastics and synthetic rubber in addition to its use in Firemaster 550 applied to flame retard PUF products.[8] Nonhalogenated alkyl phosphates (e.g., tri-*n*-butyl phosphate (TnBP) and tri-*iso*-butyl phosphate (TiBP)) are mainly applied as plasticizers in polyester resins, PVC, acrylonitrile-butadiene-styrene (ABS) and synthetic rubber. Tri (2-butoxyethyl) phosphate (TBOEP) is often used in floor polish and rubber stoppers, while 2-ethylhexyldiphenyl phosphate (EHDPP) is applied to hydraulic fluids, PVC, and food packaging.[7]

The current understanding of the toxicological effects of PFRs is far from sufficient. Few studies have reported on adverse effects of PFRs, including liver toxicity, neurotoxicity, and interference with normal growth upon long-term exposure in laboratory animals.[7] Reported health effects in humans include Sick Building Syndrome from TnBP,[9] contact dermatitis, reduction of sperm count and inhibition of androgen receptors from TPHP[10,11] and reduced thyroid

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hormone levels from TDCPP.[11] Furthermore, TCEP, TCIPP, and TDCPP were subject to an EU risk assessment process under an Existing Substances Regulation (EEC 793/93).[12] They were classified as persistent in the aquatic environment and reported to fulfill PBT criteria.[13] In addition, several studies have reported them to display adverse effects including reproductive toxicity and carcinogenic effects on lab animals.[7,13] Hence, TCEP is classified by the EU as a "potential human carcinogen" (carcinogen category 3),[12] while TDCPP is classified under regulation EC 1272/2008 as a category 2 carcinogen with hazard statement H351 "suspected of causing cancer".[14]

PFRs are used as additives (i.e., not chemically bonded) to the polymer matrix. Therefore, they are likely to leach out from treated products by abrasion and/or volatilization to contaminate the surrounding environment in a similar scenario to PBDEs and other additive FRs.[15] PFRs have recently been detected in both indoor and outdoor environments.[7,15] Several studies have reported on levels of various PFRs in water, soil, sediment, and air.[7,15−17] Moreover, PFRs were recently reported in biota and human breast milk, indicating their bioavailability to humans and wildlife.[18−20]

Despite the mounting evidence on the importance of indoor dust ingestion as a pathway of human exposure to organic pollutants, still little is known about contamination of indoor environments with PFRs. While indoor dust ingestion was reported as the major exposure pathway of American adults and toddlers to PBDEs, to the best of our knowledge, there exists no data on the relative contribution of different exposure pathways (e.g., dietary intake, inhalation, dust ingestion, dermal uptake, and so forth) to the overall human exposure to PFRs. Few studies have detected PFRs at higher levels than PBDEs in indoor dust from various countries in the $\mu g\ g^{-1}$ level.[21] While available data indicate that concentrations of PFRs in indoor dust from U.S.A. and Japan are higher than those reported from Europe,[8,22−24] information about the indoor contamination with PFRs in developing countries is scarce.[25,26] To the authors' knowledge, there are hitherto no data available on indoor levels of PFRs and human exposure to these compounds in the whole continent of Africa and the East Mediterranean region. Although few studies have previously estimated human exposure to ΣPFRs via ingestion of household dust,[7] there is—to date—a paucity of data on human exposure to PFRs via dust ingestion that considers intake from both domestic and nondomestic microenvironments, such as cars, offices, and public microenvironments (PMEs), including restaurants, coffee shops, and supermarkets.

Therefore, the aims of the current study are (a) to report—for the first time—on the levels and profiles of eight PFRs in indoor dust of different microenvironments from Egypt (Africa); (b) to compare the levels and profiles of PFRs in dust collected from 4 different microenvironment categories (homes, offices, cars, and PMEs); and (c) to estimate human exposure to PFRs via ingestion of indoor dust in Egypt using different exposure scenarios to assess the related potential risk.

■ MATERIALS AND METHODS

Sampling Strategy. Dust samples were collected between September 2012 and January 2013 from 71 microenvironments within Assiut, Egypt. A microenvironment is defined as a small, specific area, distinguished from its immediate surroundings by such factors as the amount of light, the degree of moisture, the range of temperatures, and the specific contents.[27] All sampled

microenvironments comprised a convenience sample of acquaintances of the authors. Samples were taken under normal room use conditions to reflect actual human exposure. The following microenvironment categories were selected for study: homes (living rooms, $n = 20$), offices (private and public sector, $n = 20$), PMEs (public microenvironments represented by 4 coffee shops, 4 restaurants, and 3 supermarkets) and cars ($n = 20$). Further information on the sampled microenvironments is provided in Supporting Information, SI, Table SI-1.

Sampling Methods. Dust samples were collected using a Black & Decker Pivot Dustbuster 1600 W vacuum cleaner. Sampling was conducted according to a clearly defined standard protocol by one of the research team.[28] In offices, PMEs, and homes, 1 m² of carpet was vacuumed for 2 min and, in case of bare floors, 4 m² for 4 min. In cars, only the surface of the seats with which occupants would have direct contact (i.e., not including seat backs) and the front panel was sampled for 4 min. Samples were collected using nylon sample socks (25 μm pore size) that were mounted in the furniture attachment tube of the vacuum cleaner. After sampling, socks were closed with a twist tie, sealed in a plastic bag and stored at −20 °C. Before and after sampling, the furniture attachment was cleaned thoroughly and wiped using an isopropanol-impregnated disposable wipe.

Analytical Protocols. *Sample Preparation and Extraction.* Dust samples were sieved through a 500 μm mesh size sieve, visible human hairs, carpet fibers, or food remains were removed by clean tweezers. Accurately weighed aliquots (~50 mg) of the homogeneous dust sample were extracted according to the method described by Van den Eede et al.[29] Briefly, dust samples were spiked with internal (surrogate) standard mixture containing 50 ng of isotopically labeled TCEP-d_{12}, TBOEP-d_6, TDCPP-d_{15}, and TPHP-d_{15} prior to exhaustive ultrasonic extraction with hexane/acetone (3:1) mixture. The crude extract was evaporated under a gentle stream of N_2 and resolubilized in hexane. The resolubilized extract was loaded onto a preconditioned Florisil cartridge and washed with 8 mL of hexane prior to elution of the target PFRs with 10 mL of ethyl acetate. The clean eluate was evaporated to dryness and resolubilized in 80 μL of isooctane containing 200 pg μL^{-1} of BDE-77 used as a recovery determination (syringe) standard (RDS) for QA/QC purposes. Further details are provided in the SI.

Instrumental Analysis. Quantification of target PFRs was performed using a FOCUS GC coupled to a DSQII mass spectrometer (Thermo Fisher Scientific, Austin, TX, U.S.A.) operated in electron ionization (EI) mode according to the previously described method.[29] Separation of target PFRs was performed on Agilent DB-5 capillary column (30m × 0.25 mm; 0.25 μm) using helium as the carrier gas. The mass spectrometer was run in selected ion monitoring (SIM) with ion source, quadrupole and mass transfer line temperatures set at 230 °C, 150 °C, and 300 °C, respectively. Further details of the GC-EI/MS method are provided in the SI.

QA/QC. Good recoveries (69−112%) were obtained for the deuterated IS. Six method blanks (sample is omitted) and 6 field blanks (0.2 g of sodium sulfate treated as a dust sample) were analyzed alongside the samples. While the concentrations of target compounds, when detected, in method blanks were <0.5% of sample values, TCIPP, TnBP, and TiBP were detected in all field blanks at concentrations >1 ng g^{-1} (SI Table SI-2). Therefore, results for all analytes were blank corrected by subtraction of mean blank values from the raw

**Table 1. Statistical Summary of OPFR Concentrations (ng g^{-1}) in the Studied Microenvironments**

Microenvironment		TPHP	EHDPP	TCEP	TCIPP	TDCPP	TBOEP	TnBP	TiBP	ΣPFRs
Houses (n=20)	DF (%)*	100	55	55	45	65	25	40	25	
	Average	101	52	49	53	147	86	17	25	310
	SD**	86	32	49	45	164	125	4	4	276
	Median	67	42	22	28	72	18	17	23	189
	Min	8	<LOQ	<LOQ	<LOQ	<LOQ	<LOQ	<LOQ	<LOQ	38
	Max	289	102	132	123	557	305	26	31	962
Offices (n=20)	DF (%)	100	60	55	55	70	30	40	30	
	Average	94	43	61	119	99	263	27	28	550
	SD	90	21	42	196	137	515	11	4	532
	Median	73	48	31	80	49	143	23	28	221
	Min	11	<LOQ	<LOQ	<LOQ	<LOQ	<LOQ	<LOQ	<LOQ	38
	Max	337	74	125	700	490	1244	46	34	1514
Cars (n=20)	DF (%)	100	50	50	55	50	30	55	60	
	Average	392	66	198	513	87	284	74	54	1011
	SD	584	53	195	475	76	274	70	24	914
	Median	135	54	127	291	61	190	59	51	556
	Min	26	<LOQ	<LOQ	<LOQ	<LOQ	<LOQ	<LOQ	<LOQ	199
	Max	1872	203	572	1425	283	778	258	103	3367
PMEs (n=11)	DF (%)	100	36	64	73	91	64	45	36	
	Average	959	49	277	232	601	311	112	139	2167
	SD	812	19	189	178	572	425	89	78	1533
	Median	629	44	234	232	416	68	73	133	1470
	Min	116	<LOQ	<LOQ	<LOQ	<LOQ	<LOQ	<LOQ	<LOQ	937
	Max	2357	74	538	465	1616	1029	261	225	5235

*DF = detection frequency; SD = standard deviation.

PFR values in the samples. Method limits of quantification (LOQ) were calculated as three times the standard deviation of blank values and divided by the amount of dust typically used for analysis (50 mg). For analytes not detected in the blanks, the LOQ was calculated based on a signal-to-noise ratio of 10:1 (SI Table SI-3). Further quality control was achieved via replicate analysis ($n = 5$) of NIST SRM 2585 (organics in house dust). Our results compared favorably with those reported previously[23,30] indicating good accuracy and precision of the applied method.

Statistical Analysis. Statistical analysis of the data was conducted using Excel (Microsoft Office 2010) and SPSS version 15.0. As a first step, the statistical distribution of concentrations within each data set was evaluated using both *Shapiro−Wilk* and *Kolmogorov−Smirnov* goodness of fit tests. The results, combined with visual inspection of frequency diagrams, revealed concentrations in all data sets to be log-normally distributed. Hence, further analysis of variance (ANOVA) and *t* tests were performed on log-transformed

concentrations. Statistical differences between two strata were tested in the logged database with a *t* test for equality of means and with ANOVA when testing more than two strata. Results were considered significant with *P* values less than 0.05. Where concentrations were below the LOQ, concentrations were assumed to equal half the LOQ when the detection frequency of the studied compound was >50%. However, when the detection frequency of the target compound was <50%, concentrations below LOQ were reported as *f* * LOQ with *f* being the fraction of samples above LOQ. This approach was adopted to minimize the effect of nondetects on the estimated averages and further statistical tests.[31,32] Potential associations of the target compounds and their potential sources were explored using scatter plots, box plots, Spearman's correlation, single and multiple linear regression analyses. For adjusted multiple linear regression models, source data were categorized as ordinal variables.
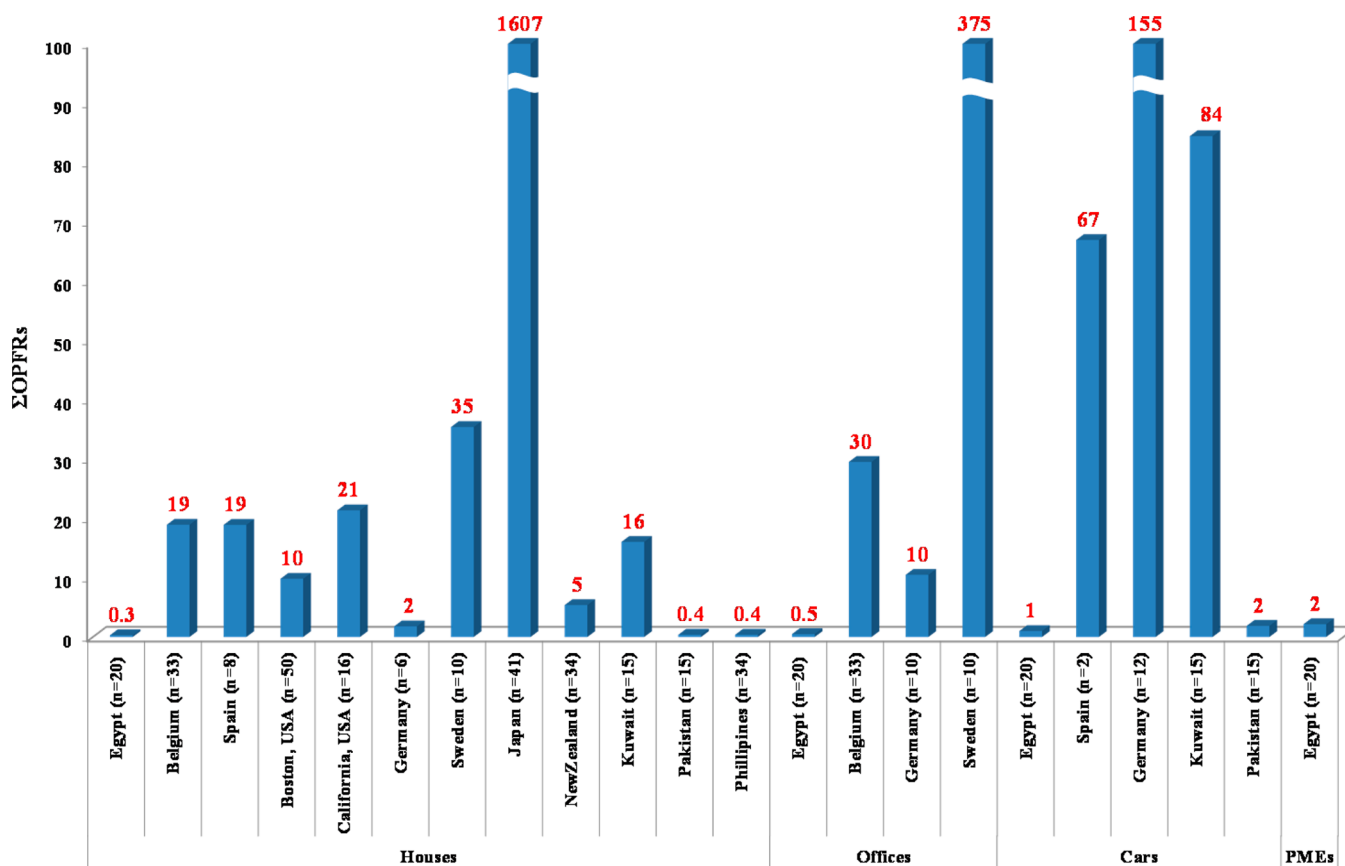
Figure 1. International comparison between average concentrations of ΣPFRs (μg g^{-1}) reported in indoor dust from different microenvironments. [The reported sampling years for the analyzed dust were as follows: Egypt (2012−2013), Belgium (2010),[23] Spain (2006),[48] Boston U.S.A. (2002−2007),[8] California U.S.A. (2011),[37] Germany (2010−2011),[24] Sweden (2010),[34] Japan (2006−2007),[9] New Zealand (2006),[46] Kuwait (2011),[25] Pakistan (2011),[25] Phillipines (2008).][26]

RESULTS AND DISCUSSION

Levels and Profiles of PFRs in Egyptian Dust. Target PFRs were detected in all the analyzed dust samples with a detection frequency up to 100% for TPHP. A statistical summary of the concentrations of 8 studied PFRs in the analyzed dust is provided in Table 1. Interestingly, the levels of ΣPFRs in house dust from Egypt are among the lowest reported worldwide (Figure 1). In this study, TPHP was the only target PFR detected in all samples and showed the highest detection frequency and highest average concentration of target PFRs in all the studied microenvironments (average = 386 ng g^{-1}, Table 1). This is in agreement with the results of previous studies from Sweden[33] and U.S.A.[8] While only a moderate correlation (Spearman r = 0.4) was observed between the concentrations of TPHP, TBB, and TBPH (main components of the Firemaster 550 commercial formulation) in 50 dust samples from U.S.A., the high levels of TPHP were explained by its wider application profile as both FR and plasticizer in a variety of consumer products.[8]

While the chlorinated alkyl phosphates, TDCPP, TCIPP, and TCEP, showed higher detection frequency (DF = 69%, 57%, and 56%; average = 233, 229, and 144 ng g^{-1}, respectively), TBOEP (DF = 37%; average = 294 ng g^{-1}) displayed the second highest average concentration (following TPHP) in the studied dust samples. Chlorinated alkyl phosphates were reported as the main PFRs in Kuwaiti house and car dust samples, where the release from a wide variety of consumer products containing flame-retarded PUFs was suggested as

their main source.[25] TBOEP was reported at very high concentrations in Japanese house dust (average = 1570 μg g^{-1})[16] and Swedish office dust (average = 250 μg g^{-1}),[34] yet no clear explanation was provided for such elevated concentrations. Few studies have reported on the levels of EHDPP in indoor dust. However, the concentrations of EHDPP in Egyptian dust (DF = 49%; average = 50 ng g^{-1}) are lower than those reported from other countries (SI Table SI-4).

While the concentrations of ΣPFRs in Egyptian house dust (average = 310 ng g^{-1}; n = 20) are generally in line with those reported from Pakistan[25] (average = 394 ng g^{-1}; n = 15) and the Philippines (average = 387 ng g^{-1}; n = 34),[26] they are at least an order of magnitude lower than those reported from western industrial countries.[7] Except for the use of air-conditioning rather than central heating systems, the general features of a typical Egyptian house are not substantially different from a standard home in Europe (See SI Table SI-1 for details of the sampled homes). Hence, the low levels of PFRs in the sampled house dust may be attributed to less strict fire-safety regulations in Egypt, which is still not a member of the UNEP Stockholm Convention so far.[35] Therefore, the production and usage of PBDEs is not banned under the current national regulations.[36] While there are no registered facilities for production of PBDEs in Egypt,[36] the ongoing use of PBDEs as FRs may account for the low concentrations of PFRs applied mainly as substituents for commercial PBDE formulations.[37]

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Although the production and usage of PFRs in Egypt is unknown,[7] no statistically significant correlation ($P > 0.05$) could be established between the levels of PFRs in dust and the number of electronic equipment or foam-containing furniture in the studied microenvironments using both single and multiple linear regression models (MLRM). Attempts to use adjusted MLRM by including other factors (e.g., type of ventilation, age of building/manufacture, and/or carpeted floors) for identification of significant potential sources of PFRs in the studied microenvironments have also not lead to significant correlations. This is in agreement with previous reports, which could not establish any significant correlations between the levels of PFRs in dust and the contents of the sampled microenvironments.[21,23,37] The relatively small number of dust samples analyzed in this study (although as mentioned earlier, similar results were reported elsewhere) may be partly responsible for the lack of statistically significant correlations between PFR levels in dust and the contents of the sampled microenvironments. Therefore, more detailed studies and further integrated statistical models are required to improve the current understanding of the mechanism of PFRs release from treated products to the indoor environment.

Levels and Profiles of PFRs in Dust from Different Microenvironments. Statistical analysis of ΣPFR concentrations reported from different types of indoor microenvironments in this study (Table 1) revealed significantly ($P < 0.05$) higher concentrations of ΣPFRs in cars (average = 1011 ng g^{-1}) and PMEs (average = 2167 ng g^{-1}) than in houses (average = 310 ng g^{-1}) and offices (average = 550 ng g^{-1}). No significant differences ($P > 0.05$) were observed between PFR levels in houses and offices or between ΣPFRs in cars and PMEs.

While this study is the first to report on PFR levels in public microenvironments (e.g., coffee shops, supermarkets, and restaurants), a previous study has reported on significantly higher concentrations of PFRs in car dust (average = 155 μg g^{-1}) than in house (average = 2 μg g^{-1}) and office (average = 10 μg g^{-1}) dust samples collected from Germany.[24] Recently, Ali et al.[25] found significantly higher PFR levels in cars than in house dust samples from Kuwait and Pakistan. While the high levels of ΣPFRs in German cars were attributed to a substantially high contribution of TDCPP,[24] profiles of ΣPFRs in car dust from Egypt-like those from Kuwait[25]-show a higher contribution of TCIPP (Figure 2). Given that TCIPP and TDCPP are both used in flexible and rigid PUFs deployed in car upholstery, we hypothesize that the chlorinated PFRs are released from the car upholstery via abrasion of the fabric during the normal use of the car. To test this hypothesis, we studied the correlation between target PFR concentrations and the age of the sampled cars. While ΣPFRs in the studied cars were not significantly correlated with the car age or year of manufacture, a significant correlation ($P = 0.042$) could be established only between TCIPP levels and the age of the cars adjusted to the mileage completed by each car at the time of sampling. This is generally in agreement with the results of Brommer et al., who reported a positive linear correlation ($P = 0.05$) between TDCPP concentrations in car dust and the estimated mileage completed by 7 cars from Germany.[24] In general, the higher levels of PFRs in cars than in houses and offices may be attributed to the application of ABS and polypropylene polymers in the molded plastic panels, electronics, and PUF upholstery in the interior of the vehicles.
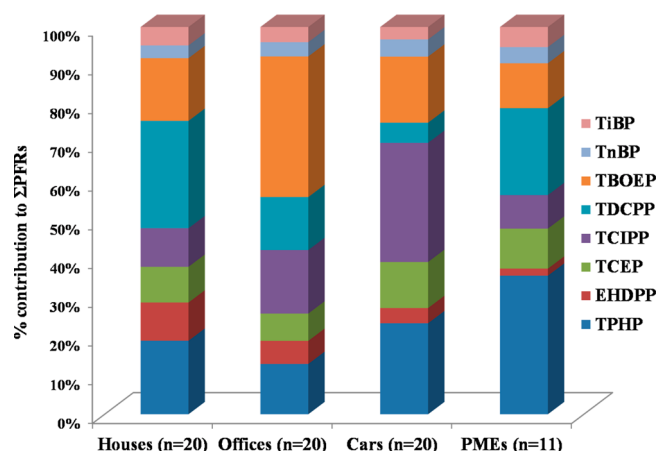
Figure 2. PFR profiles showing average % contribution of target analytes to ΣPFRs in the sampled microenvironments.

The congener profile of PFRs varied substantially between different types of studied microenvironments (Figure 2). While TDCPP was the predominant PFR in the analyzed house dust (contributing 28% to the average ΣPFRs), TBOEP, TCIPP, and TPHP showed the highest contribution to average ΣPFRs (36, 31, and 36%) in office, car, and PMEs dust, respectively. This is generally in agreement with the findings of previous studies[7] and can be explained by the different sources and variety of applications of target PFRs in various types of microenvironments. The concentrations and profiles of target PFRs in the studied microenvironment categories were compared using Kruskal−Wallis test and multiple pairwise comparisons. Intracategory comparison revealed statistically significant differences ($P = 0.02$) between PFR levels in the studied offices, while no significant differences ($P > 0.05$) in PFR concentrations were observed between the sampled houses, cars, or PMEs. Intercategory comparison revealed significant differences between the concentrations of PFRs in the studied microenvironment categories. Although statistical analysis revealed the levels of TPHP in the studied PMEs were significantly higher than those in house and office dust, no significant differences were observed between PFR congener profiles in the studied microenvironment categories (Figure 2).

Chlorinated alkyl phosphates (i.e., TDCPP, TCIPP, and TCEP) dominated the PFR congener profile in all the studied microenvironments contributing 52−80% to ΣPFRs in dust. This may indicate the release from PUF objects as a major source of indoor dust contamination with PFRs. Further detailed studies are required to identify possible transfer mechanisms of chlorinated alkyl phosphates from treated PUF objects to indoor dust in different types of microenvironments. Bearing in mind the carcinogenic potential of TCEP and TDCPP,[14] such studies are warranted in the near future.

Human Exposure to PFRs via Indoor Dust Ingestion. The measured concentrations of PFRs were used to estimate the exposure of adults and toddlers to the target compounds via indoor dust ingestion in Egypt. To do so, we have assumed (in the absence of experimental data) 100% absorption of intake, average adult and toddler dust ingestion figures of 20 and 50 mg day^{-1}, and high dust ingestion figures for adults and toddlers of 50 and 200 mg day^{-1}.[40] To our knowledge, there exists no comprehensive data set that describes time-activity patterns for the Egyptian population. Hence, overall exposure estimates are based on the assumption that dust ingestion

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occurs pro-rata to typical activity patterns reported previously (i.e., for adults 63.8% home, 22.3% office, 5.1% PMEs, 4.1% car, and 4.7% outdoors; for toddlers 86.1% home, 5.1% PMEs, 4.1% car, and 4.7% outdoors).[41,42] We have then estimated plausible dust ingestion exposure scenarios, using median and average concentrations in the dust samples reported in this study (Table 2). The algorithm given below[43] was used to estimate

Table 2. Summary of Egyptian Adult and Toddler Exposure to Target PFRs (ng day^{-1}) via Indoor Dust Ingestion Using Different Exposure Scenarios

PFR	Reference dose*[46] Adult	Reference dose*[46] Toddler	Exposure scenario	Adult Average	Adult Median	Toddler Average	Toddler Median
TPHP	490000	140000	Mean#	3.0	1.9	7.5	4.8
			High$	7.5	4.8	30.0	19.3
EHDPP	n.a	n.a	Mean	1.0	0.8	2.4	2.1
			High	2.4	2.1	9.6	8.4
TCEP	154000	44000	Mean	1.3	0.8	3.2	1.9
			High	3.2	1.9	13.0	7.6
TCIPP	560000	160000	Mean	1.9	1.2	4.7	3.0
			High	4.7	3.0	18.6	11.9
TDCPP	105000	30000	Mean	3.0	1.6	7.5	4.0
			High	7.5	4.0	30.0	16.1
TBOEP	105000	30000	Mean	3.8	2.0	9.6	5.0
			High	9.6	5.0	38.5	20.0
TnBP	168000	48000	Mean	0.5	0.4	1.3	1.1
			High	1.3	1.1	5.2	4.4
TiBP	n.a	n.a	Mean	0.6	0.6	1.6	1.5
			High	1.6	1.5	6.3	5.9
ΣPFRs	n.a	n.a	Mean	9.0	5.2	22.5	13.0
			High	22.5	13.0	90.0	51.9

*Assuming average adult and toddler body weights of 70 and 20 kg,[47] respectively. #Assuming mean dust ingestion rate of 20 and 50 mg day^{-1} for adults and toddlers, respectively.[40] $Assuming high dust ingestion rate of 50 and 200 mg day^{-1} for adults and toddlers, respectively.[40]

both adult and toddler exposure to target PFRs via indoor dust ingestion.

$$\Sigma_{(evdi)} = [(C_H F_H) + (C_O F_O) + (C_C F_C) + (C_{PME} F_{PME})]RR$$

where $\Sigma_{(evdi)}$ is the total daily human exposure to the studied PFRs via dust ingestion (ng day^{-1}); C_H, C_O, C_C, or C_{PME} is the PFR concentration (ng g^{-1}) at the respective exposure scenario in homes, offices, cars or public microenvironments, respectively. $F_H/F_O/C_C/F_{PME}$ is the respective average fraction of time spent in each microenvironment, and RR is the daily dust ingestion rate (mg day^{-1}). It is stressed that the range of exposure estimates via dust ingestion thus derived is only an indication of the likely range within the population. This is due to the highly uncertain nature of the ingestion rates used here (and in other studies) as they are based on a small number of studies involving primary data collection.[42,44,45]

Results revealed that human exposure estimates to PFRs via accidental indoor dust ingestion in Egypt (median exposure to ΣPFRs = 5 and 13 ng day^{-1} for adults and toddlers, respectively) are generally in line with those reported from Pakistan (median exposure to ΣPFRs = 12 and 32 ng day^{-1} for adults and toddlers, respectively),[25] and the Philippines (median exposure to ΣPFRs = 6 and 17 ng day^{-1} for adults and toddlers, respectively).[26] However, our exposure estimates (Table 2) are at least an order of magnitude lower than those reported from Europe, Japan, and the U.S.A.[7] The maximum adult and toddler exposures to TBOEP (25 and 100 ng day^{-1}, respectively) in this study (assuming a high dust ingestion rate and maximum contamination of the reported concentrations)

are several orders of magnitude lower than reported reference dose for this compound, which has the lowest reference dose among the studied PFRs based on liver toxicity (γ-glutamyl transferase and vacuolization).[46] A similar case was observed for the other target PFRs where maximum exposure estimates for adults and toddlers via indoor dust ingestion were much lower than the reported reference doses[46] (Table 2). Given the significantly higher concentrations of the studied PFRs in cars than in offices, we estimated the occupational exposure of taxi driver working 8 h day^{-1} in his car. Results revealed maximum overall exposure of 29 and 74 ng day^{-1} using the mean and high dust exposure scenarios, respectively. While the estimated exposure of a taxi driver to PFRs was higher than that of an average adult working in an office (Table 2), such exposures were still orders of magnitude lower than the toxicological reference doses.

Collectively, the results of this study indicate that current human exposure to PFRs via accidental ingestion of indoor dust does not pose an immediate health risk to the Egyptian population based on the current state of knowledge about the toxicological properties of PFRs. However, further studies are required to fully characterize the overall human exposure to PFRs via other exposure pathways including inhalation, diet, and dermal exposure. Moreover, further understanding of the potential adverse effects resulting from human exposure to a mixture of PFRs within a "cocktail" of other anthropogenic chemicals via indoor dust (e.g., PCBs, PBDEs, and so forth) is required to improve our understanding of the potential risk arising from the concomitant exposure to these various chemicals.

ASSOCIATED CONTENT

Ⓢ Supporting Information
Further details of the sampled microenvironments, analytical methodology, quality assurance/quality control parameters and international levels of target PFRs in indoor dust. This material is available free of charge via the Internet at http://pubs.acs.org/.

AUTHOR INFORMATION

Corresponding Author
*Phone: +44 121 414 7297; fax: +44 121 414 3078; e-mail: mae_abdallah@yahoo.co.uk.

Notes
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