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ABSTRACT

Very few studies have reported the presence of the organophosphate esters (OPEs) in African indoor microenvironments. We therefore document here, the concentrations, profiles, and human exposure to eight organophosphate esters (OPEs) for the first time in indoor dust from various microenvironments in Nigeria, specifically: cars/buses (n = 10), homes (n = 20), offices (n = 20), and medical centres (n = 14). The concentrations of OPEs in these indoor dust samples were among the lowest reported internationally. Concentrations of $\sum_8$OPEs varied substantially between individual samples and the predominant OPEs were: tris(2-butoxyethyl) phosphate (TBOEP) (detection frequency (DF) = 90–100%), tris(1-chloro-2-propyl) phosphate (TCIPP) (DF = 100%), and 2-ethylhexyl-diphenyl phosphate (EHDPP) (DF = 100%). There were no significant differences (P>0.05) between $\sum_8$OPEs concentrations in dust samples from cars/buses (average = 295 ng/g), offices (231 ng/g), homes (277 ng/g), and medical centres (127 ng/g). Concentrations of chlorinated OPEs: tris(2-chloroethyl) phosphate (TCEP), TCIPP, and tris(1,3-dichloro-2-propyl) phosphate (TDCIPP) were significantly correlated with those of triphenyl phosphate (TPhP), EHDPP, and TBOEP. Estimated daily intakes (EDI) of target OPEs via indoor dust ingestion and dermal absorption were lower than the corresponding reference dose (RfD) values, indicating that exposure to the studied OPEs in the indoor environment does not pose a significant health risk for the general population in Nigeria, even under a high-end exposure scenario.

1. Introduction

Due to concerns about the toxicity and bioaccumulation potential of brominated flame retardants (BFRs), that have led to bans and restrictions on the manufacture and new use of polybrominated diphenyl ethers (PBDEs) and hexabromocyclododecane (HBCDD); production and use of organophosphate esters (OPEs) as alternative flame retardants (FRs) has grown [1]. OPEs represent a heterogeneous class of phosphoric acid esters in which the hydrogen in the phosphate group is replaced by an alkyl, aryl, or chlorinated group [2]. As of 2016, OPEs represented the second most produced flame retardants (FRs) reaching 18% of the total global market [3], with a substantial increase reported in global consumption of OPEs from 680,000 t in 2015 to 2,800,000 t in 2018 [4–6]. As a result of their release from commercial and industrial products, OPEs have been detected in diverse indoor and outdoor environmental matrices, such as: air and dust [7–11], drinking water and sediments [5,12–15], soil [16,17], food [18–23], as well as handwipes and wristbands [24]. Furthermore, human biomonitoring studies have detected OPEs in human tissues or biological fluids and other non-invasive matrices [25,26].

Moreover, several toxicological studies have shown that some OPEs are neurotoxic, carcinogenic, and endocrine disruptors. For instance, tris(2-chloroethyl) phosphate (TCEP) and tris(1,3-dichloro-2-propyl) phosphate (TDCIPP) were reported to exhibit

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neurotoxic effects on PC12 cells [27]. TDCIPP also elicited developmental abnormalities and neurotoxicity in embryonic zebrafish [28], while TCEP has been shown to be carcinogenic to both rats and mice and classified as a Category 2 carcinogen by the European Union [29,30]. In addition, tri-n-butyl phosphate (TNBP) and triphenyl phosphate (TPhP) have been reported to be neurotoxic to zebrafish larvae and rats [31,32].

Indoor dust serves as a sink for many organic pollutants and its ingestion has been identified as a major human exposure pathway to emerging pollutants such as OPEs [11,33]. However, despite the mounting evidence of human exposure to and consequent adverse effects of OPEs, there remains scarce information on their presence in indoor environments in developing countries. Several studies have reported a significant correlation between estimated external exposure to OPEs via indoor dust ingestion and estimated internal exposure derived from urinary metabolite measurements [34–36].

Although only a few studies have reported human exposure to ∑OPEs from domestic and non-domestic microenvironments in Europe and Eastern Mediterranean countries [10,37–39], there is to date a paucity of data in sub-Saharan African countries (e.g. Ref. [9]) on the presence of and estimates of human exposure to these compounds in indoor microenvironments, such as offices, cars, homes, and medical centres. This study thus constitutes the first report of concentrations of OPEs in indoor dust from domestic and non-domestic microenvironments (offices, cars, medical centres, and homes) in Nigeria. The aims of the present study are to: (i) investigate the concentrations and profiles of target OPEs in indoor dust from Nigeria, (ii) compare the concentration profiles of OPEs in different microenvironments (cars, offices, medical centres, and homes), (iii) evaluate possible sources of OPEs in Nigerian indoor dust, and (iv) derive estimates of human exposure to OPEs via indoor dust in Nigeria and its potential human health implications.

2. Materials and methods

2.1. Standards and reagents

Eight OPEs, comprising: TCEP, TCIPP, TDCIPP, TBOEP, TPHP, EHDPP, tri-n-butyl phosphate (TnBP), and tri-*m*-tolyl phosphate (TMTP); as well as two isotopically labelled internal (or surrogate) standards (TnBP-d_{27} and TPHP-d_{15}) and 2,3,4,6-tetrachlorobiphenyl (PCB-62) used as a recovery determination standard (RDS) (or syringe standard) were purchased from Wellington Laboratories, (Guelph, ON, Canada). The purity of all analytical standards was > 98% except for TBOEP (> 94%). The physicochemical properties of our target OPEs are provided in Table S1 (Supporting Information). HPLC-grade n-hexane (HEX, 95%), ethyl acetate (ETAC, 99.8%), dichloromethane (DCM, 99.8%), iso-octane (ISOC, 99.5%) and acetone (ACE, 99.8%) were purchased from Fisher Scientific (Loughborough, UK) and Sigma-Aldrich (St Louis, MO, USA). Indoor dust standard reference material SRM 2585 was purchased from the US National Institute of Standards and Technology (NIST, Gaithersburg, MD, USA). Hypersep Florisil® SPE cartridges were purchased from Thermo Scientific (Rockwood, USA), and the nitrogen gas used for solvent evaporation was purchased from BOC gases, United Kingdom.

2.2. Sampling methods

A total of 64 indoor dust samples were collected from offices (n = 20), cars and buses (n = 10), homes (n = 20) and medical centres (n = 14) between July 2021 to September 2021 in Ijebu-Ode in Ogun State and Lagos State in Southwest Nigeria. The medical centres examined comprise a range of hospitals providing both surgical and non-surgical care. The hospital room contained some electronic and surgical instruments (CT scan, x-rays, Stethoscope, TVs at the waiting section), an infusion pump, as well as several items of furniture containing foam and fabrics. In each home, two samples were collected within 1 m distance of the electronics shelf containing TVs, radios, speakers, and other electronic gadgets). Other characteristics of the selected microenvironments are presented in Table S2. At the time of sampling the daily temperature was between 24 and 32 °C and information on potential sources of OPEs in each sampled indoor environment was recorded (Table S2). Surface dust from offices, homes, and medical centres were collected using paintbrushes (prewashed with hexane and dichloromethane) from elevated surfaces such as tables and shelves as well as from air conditioning and electronic control panels (TVs and radios) with which the occupants have daily contact. For cars and buses, indoor dust was collected from the interior according to a modified version of the protocol reported by Ref. [40]. Following collection, dust samples were wrapped in aluminium foil [41], transferred to individual zip lock bags, stored at −20 °C and carefully conveyed to the University of Birmingham, UK for extraction and GC-MS analysis. At the University of Birmingham, dust samples were passed through a 500 μm mesh sieve and homogenised thoroughly prior to extraction and analysis.

2.3. Sample extraction and clean-up

Accurately weighed aliquots (~ 100 mg) of the homogenised dust samples were extracted according to the method described by Ref. [42]. Samples were spiked with internal (surrogate) standard mixture containing 50 ng of isotopically labelled TnBP-d_{27} and TPhP-d_{15} and extracted using 4 mL of hexane: acetone (3:1 v/v) by a combination of vortexing (2 min) and ultrasonication extraction (5 min) in three cycles. After each extraction cycle, dust extracts were centrifuged at 3500 rpm for 5 min and the supernatants collected and transferred into clean glass tubes. Pooled supernatants were evaporated to incipient dryness under a gentle nitrogen flow and resolubilised in 1 mL of hexane. These concentrates were fractionated into two fractions using Hypersep Florisil® cartridges conditioned with 6 mL of hexane. Extracts were quantitatively transferred to the cartridge, and eluted with 8 mL hexane (F1, discarded) followed by 10 mL ethyl acetate (ETAC) (F2) which contained the target OPEs. The second fraction (F2) was evaporated to incipient dryness and resolubilised in 100 μL of 500 pg/μL of PCB-62 in isooctane as recovery determination or (syringe) standard. These final extracts were transferred into glass vials and analysed using GC-EI/MS. Detailed information on the instrumental analysis is provided in supplementary information section 1.

2.4. QA/QC

Quality assurance and control checks were carried out using indoor dust standard reference material (SRM 2585). Two procedural blanks (without dust samples) and one indoor dust standard reference material (SRM 2585) were analysed for each batch of ten dust samples to evaluate the accuracy and precision of the method. In addition, five field blanks (comprising ~ 0.5 g of sodium sulfate treated as a dust sample following the same sampling method) were analysed to check for background contamination from the materials used for sampling and storage. Among the OPEs analysed, only TCEP was detected in the blanks at between 5 and 20% of the concentration detected in samples. Thus, the mean concentration of TCEP detected in the blanks from the same batch was subtracted from the raw TCEP concentration in the samples for each batch. Average recoveries of 79 ± 1.2% and 93 ± 3.0% were obtained for TBP-d_{27} and TPHP-d_{15} respectively (Table S4). OPE recoveries from anhydrous sodium sulfate spiked at 50 ng of each native target OPE range from 87 to 102.3% with a relative standard deviation < 20%

(n = 5). A 5-point calibration plot was constructed with OPE standard solutions in the concentration range 50 pg/μL − 750 pg/μL, with excellent linear response coefficients ($r^2 > 0.99$). Moreover, the relative standard deviations of the relative response factors (RRFs) in the five calibration standards were below 6%. For every ten samples, a 500-pg/μL OPE standard mixture was injected to check instrumental stability and calibration. Instrumental limits of detection (iLODs) and limits of quantification (iLOQs) were calculated as the amount of analyte that give a signal to noise ratio of 3 and 10, respectively (Table S4). For target OPEs not detected in the blanks, sample LOQs were calculated based on a signal-to-noise ratio of 10:1. For TCEP which was detected in blanks, the LOQ was calculated as the three times the standard deviation of the blank value divided by the mass of dust typically used for analysis (~ 100 mg) [10]. A further quality control check was achieved via replicate analysis (n = 7) of NIST SRM 2585 (organic contaminants in house dust). Results showed good accuracy and precision of the method compared to concentrations of our target OPEs reported previously for NIST SRM 2585 [10,39,43–46] (Table S5).

2.5. Data analysis

Descriptive statistics (mean, median, maximum, minimum, standard deviation, and the 95th percentile concentrations) were calculated using Microsoft 365 Excel, with IBM SPSS statistics version 28 used for: analysis of variance (ANOVA), correlational analysis (CA), principal component analysis (PCA), and hierarchical cluster analysis (HCA). The statistical distribution of the OPEs concentrations was evaluated in SPSS using the Shapiro-Wilk and Kolmogorov-Smirnov normality tests and found to be normally distributed (Table S7). PCA was performed with Kaiser-Meyer Olkin (KMO) test for sampling adequacy test and Bartlett's test of sphericity that was found to be significant ($p < 0.05$). Potential linear relationships between the variables were investigated using Pearson's rank correlation. The statistical differences between or within studied microenvironments were conducted using ANOVA. P-values lower than 0.05 ($p < 0.05$) were accepted as statistically significant. Hierarchical cluster analysis (HCA) was used for the identification and classification of variables with similar characteristics to a new group using the average linkage method. GraphPad prism was used for the box and whisker plots. Where the concentrations of a given OPE with detection frequency (DF > 50%) were below the LOQ, concentrations were assigned as half of the LOQ for that compound (<LOQ = 0.5 * LOQ). Then when the detection frequency (DF) of the target OPE was <50%, concentrations below LOQ were reported as *f* * LOQ, where *f* is the fraction of samples above LOQ. This was done to reduce the influence of non-detects on the estimated average concentrations and for other statistical analysis [10,47,48].

2.6. Estimated daily intake and exposure risk assessment

Concentrations of OPEs were used to determine human exposure via indoor dust ingestion in Nigeria for toddlers and adults using two exposure scenarios. These were: (i) average, where it is assumed that humans ingest dust contaminated at the average concentration at an average ingestion rate of 50 and 20 mg day^{-1} for toddlers and adults respectively, and (ii) a high exposure scenario where it is assumed that humans ingest dust contaminated at the 95th percentile concentration and at a high-end ingestion rate of 200 and 50 mg day^{-1} for toddlers and adults respectively [10,49,50]. We also assumed 100% absorption of intake in the absence of experimental data for this parameter [10,51]. As a result of a lack of comprehensive data on time - activity patterns for the Nigerian population, we assumed that dust ingestion occurs pro rata to typical time activity patterns previously reported (i.e., for adults: 63.8% homes; 22.3% offices; 4.1% cars; 5.1% medical centres, and 4.7% outdoors; and for toddlers: 86.1%, 5.1%, 4.1%, and 4.7% for homes, medical centres, cars, and outdoors [10,44,52,53]. The estimated daily intake (EDI) of OPEs via indoor dust ingestion and dermal uptake for toddlers and adults were calculated using equations (1) and (2) [44,45,54].

Table 1
Statistical summary of OPE concentrations (ng/g) and detection frequencies (DF, %) in indoor dust from Lagos, Nigeria.

Microenvironment	Statistical Parameter	TCEP	TCIPP	TDCIPP	TPHP	EHDPP	TBOEP	TnBP	TMTP	∑OPEs
Cars/Buses (n = 10)	Mean	8.2	71	18	19	37	99	26	17	295
	Median	6.3	73	13	16	37	80	24	<LOQ	250
	Standard deviation	5.0	18	19	8.6	6.8	45	7.7	30	140
	Minimum	3.8	38	6.7	12	30	63	19	<LOQ	173
	Maximum	21	103	69	42	53	199	40	83	612
	95th percentile	16	96	48	32	48	176	39	70	449
	DF (%)	100	100	100	100	100	100	100	30	
Offices (n = 20)	Mean	40	34	20	29	32	66	8.6	0.7	231
	Median	5.7	19	13	18	22	54	8	<LOQ	139
	SD	78	34	34	31	35	52	5	2	271
	Min.	1.9	5.1	1.8	2.6	2.6	1.4	1.4	<LOQ	17
	Max.	259	126	159	124	129	257	20	7.9	1082
	95th percentile	216	97	43	77	102	111	16	4.4	573
	DF (%)	100	100	100	100	100	100	100	15	
Homes (n = 20)	Mean	20	81	26	30	25	88	<LOQ	7.6	277
	Median	13	85	18	18	20	78	<LOQ	<LOQ	231
	SD	15	43	26	60	17	57	<LOQ	18	235
	Min.	3.5	12	2.6	0.015	5.8	0.49	<LOQ	<LOQ	24
	Max.	59	148	124	282	73	200	<LOQ	70	956
	95th percentile	44	141	53	51	62	177	<LOQ	39	457
	DF (%)	100	100	100	95	100	90	0	30	
Medical centres (n = 14)	Mean	3.5	13	6.2	5.4	23	53	14	9.1	127
	Median	3.3	13	4.7	4.6	9.7	49	13	13	110
	SD	1.1	2.7	5.4	2.8	47	13	2.7	8.8	83
	Min.	2.5	9.2	1.8	3.8	6.3	41	11	<LOQ	76
	Max.	6.7	19	24	15	184	93	20	25	386
	95th percentile	5.2	17	14	9.2	81	73	18	22	220
	DF (%)	100	100	100	100	100	100	100	57	

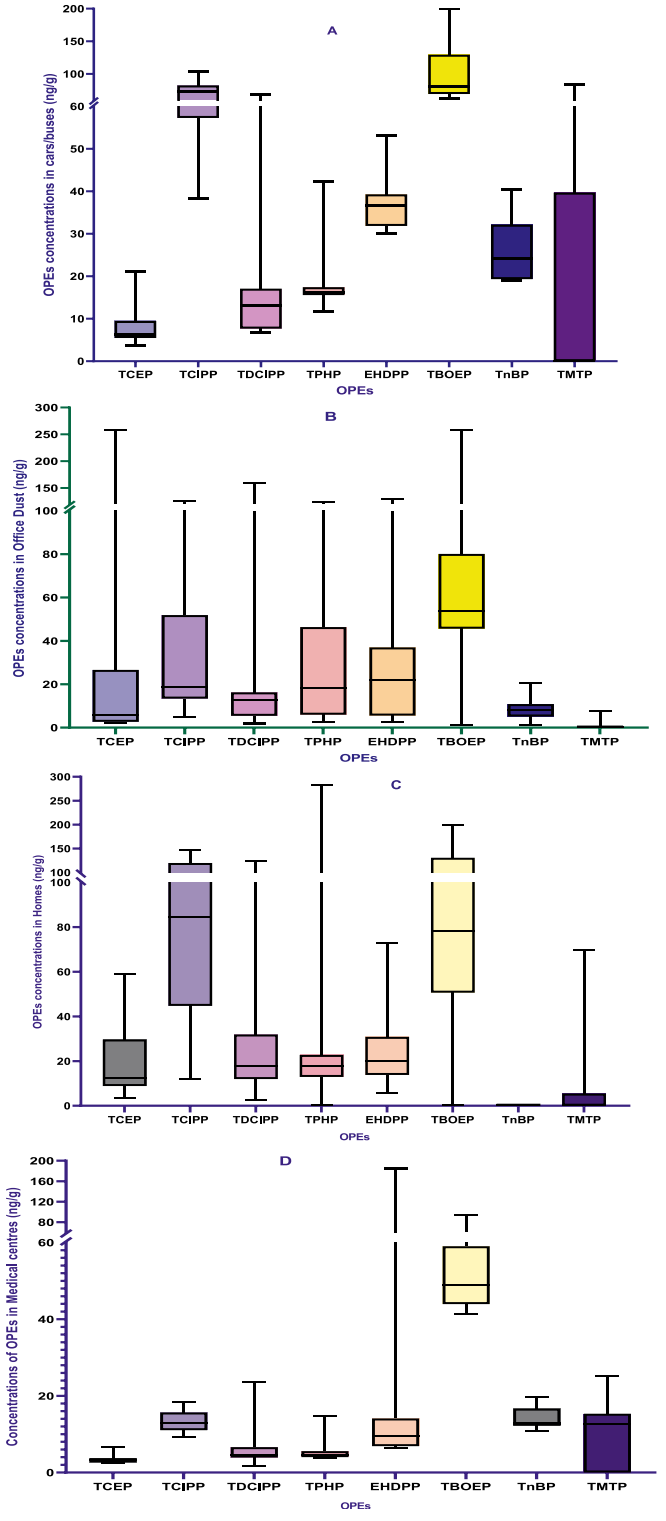

Fig. 1. Box plots of OPE concentrations in indoor dust from four microenvironments in Nigeria: (A) Cars/Buses; (B) Offices (C) Homes (D) Medical centres from Nigeria [The concentrations of OPEs at 25th and 75th percentile is what the central box represents, the middle bold line represents the median OPEs concentrations. The bottom and the top whiskers represent the maximum and minimum OPEs concentrations].

$$EDI_{ingestion} = \frac{C_{dust} x\ IngR_{dust} x AF_{gastro} x\ ITAP}{BW} \quad (1)$$

$$EDI_{dermal} = \frac{C_{dust} x\ DAS\ x\ ESSA\ x\ FA_{dermal}\ x\ ITAP}{BW} \quad (2)$$

In the algorithms above, C_{dust} is the concentration of OPEs in indoor dust (average and 95th percentile concentrations, ng/g); $IngR_{dust}$ is the indoor dust ingestion rate (average = 50 and 20 mg day^{-1}; high-end = 200 and 50 mg day^{-1} for toddlers and adults respectively) [10,51]; AF_{gastro} is the gastrointestinal absorption fraction (100% for all OPEs, as there were no experimental data) [44,45,55], ITAP is the indoor time activity pattern given above and BW is the body weight (14 kg for toddlers and 80 kg for adults) [44]. DAS represents the dust adhered to skin rate which is 0.04 mg cm^{-2} and 0.01 mg cm^{-2} for toddlers and adults respectively [51,52]; ESSA is the exposed skin surface area for which the values assumed were: 2564 cm^2 and 4615 cm^2 for toddlers and adults respectively [8,54], and FA_{dermal} is the fraction of OPEs sorbed by the skin (TCIPP: 0.25; TCEP: 0.28; TDCIPP: 0.13 and 0.17 for TPHP, EHDP, TNBP and TBOEP [40,56].

3. Results and discussion

3.1. Total concentrations and profiles of OPEs in indoor dust from different microenvironments

Descriptive statistics of OPE concentrations in the four microenvironments studied are summarised in Table 1. Most target OPEs were detected in all microenvironments with a detection frequency (DF) of 100%. However, TnBP was not detected in any samples from homes and its DF varies from 10 to 100% for medical centres, offices, and cars/buses respectively. In addition, the DF of TMTP varies from 15 to 57% in the studied microenvironments. Among the OPEs considered in this study, TBOEP displays the highest average concentrations with a DF of 100% in all four microenvironments. The range and average concentrations of TBOEP were: cars/buses (mean = 99; range: = 63–199 ng/g), offices (mean = 66; range = 1.4–257 ng/g), homes (mean = 88; range = 0.49–200 ng/g), and medical centres (mean = 53; range = 41–93 ng/g), representing between 28 and 41% of the total OPE concentrations in the 4 microenvironments (Table 1; Fig. 2). The high concentrations of TBOEP obtained in this present study are similar to those reported in previous studies in house dust from Japan and the Netherlands (median = 5.08 × 10^5 and 22,000 ng/g) [57,58] and office dust in Sweden (average = 2.5 × 10^5 ng/g) [59]. This predominance of TBOEP may be attributed to its widespread use in floor finish and wax, paint, and glue. The mean and range of concentrations of $\sum_8$OPEs in the microenvironments were in the order: cars (mean: 295; range: 173–612 ng/g) > homes (mean: 277; range: 24–956 ng/g) > offices (mean: 231; range: 173–612 ng/g) > medical centres (mean: 127; range: 76–386 ng/g) respectively. The highest TBOEP concentrations (mean: 99 ng/g; 63–199 ng/g) were obtained in car dust, followed by house dust (mean: 88 ng/g; 0.49–200 ng/g), office dust (66 ng/g; 1.4–257 ng/g) and the lowest in medical centres (mean: 53 ng/g; 41–93 ng/g). The three Cl-OPEs represented between 3 and 24%, 8–17%, 7–29% and 2–10% of the mean concentration of $\sum_8$OPEs for car/buses, offices, homes, and medical centres respectively (Table 1). The highest concentrations of the chlorinated OPEs: TCEP (DF = 100%) (mean: 40; range: 1.9–269 ng/g), TCIPP (DF = 100%) (mean: 34; range: 5.1–126 ng/g) and TDCIPP (DF = 100%) (mean: 20; range: 1.8–159 ng/g) were found in offices, followed by car/buses, homes with the lowest seen in medical centres (Table 1).

The ubiquitous presence of Cl-OPEs in dust in this study, implies wide application of these compounds in various consumer and industrial products in Nigeria [10,52]. The observed high detection

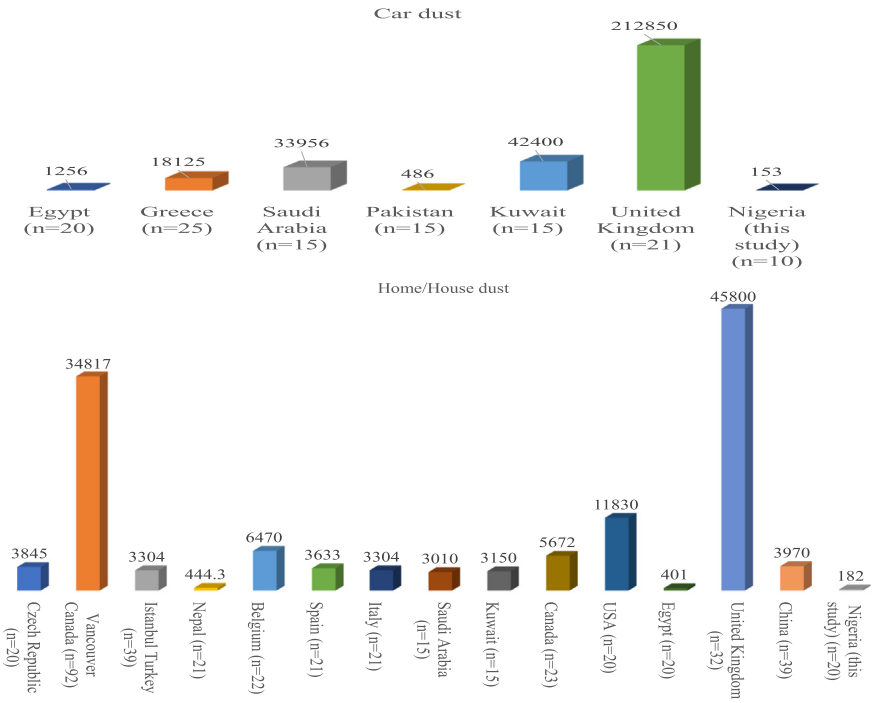

Fig. 2. Global comparison between average concentrations of Σ_5OPEs (ng/g) reported from car dust and home/house dust
[Reported sampling years for the analysed dust were: Egypt (2012–2013) [10], UK (2011–2012) [11], China (2015) [8,70]; Czech Republic, Canada, and the USA (2013) [69]; Vancouver, Canada, and Istanbul, Turkey (2007–2008) [94], Nepal (2014, 2015) [33,67]; Belgium, Spain, and Italy (2016–2017) [45]; Saudi Arabia (2014) [71]; Kuwait and Pakistan (2011) [61]; Greece (2018) [37]*. *[NB: For Greece, the publication year was assumed as the sampling year].

Table 2
Principal component analysis (after varimax rotation) showing contribution of statistically significant variables (in bold).

Rotated Component Matrix[a]				Communalities
	Component			
	1	2	3	
TDCIPP	**.900**	.031	.002	0.810
TPHP	**.824**	−.203	−.110	0.733
TCEP	**.748**	.108	.085	0.579
EHDPP	**.698**	.360	−.132	0.635
TCIPP	**.592**	−.388	.431	0.687
TBOEP	**.455**	.366	.427	0.523
TnBP	.020	**.879**	.128	0.790
TMTP	−.154	.101	**.855**	0.765
Eigen-value	3.151	1.352	1.019	
% of variance explained	48.980	15.650	14.397	
Cumulative (%)	48.980	64.630	79.027	

frequency (DF = 100%) of the Cl-OPEs in this study is similar to reports from: Egypt [10], Iraq [44], China [8,50], Canada and Turkey [39], the UK [11,60], Greece [37], Kuwait and Pakistan [61], and Spain [7]. High concentrations of Cl-OPEs in house and car dust from Kuwait were attributed to their use in polyurethane foam furniture filling and as plasticisers [61]. Similar studies conducted in Iraq and Spain found TCIPP was the most abundant OPE [44,62], while in the US, TDCIPP and TCIPP were the most abundant OPEs [63,64]. Broadly in line with our study, in Egypt, the dominant OPEs were TDCIPP and TBOEP in house and office dust respectively [10]. In this study, the mean concentrations obtained for TPhP and EHDPP were comparable to those reported for office dust and house dust in Egypt (73 and 48 ng/g) [10] and Nepal (48 and 25.9 ng/g) [33]. We found no statistically significant difference ($p > 0.05$) between the TPhP and EHDPP concentrations observed in dust from the four microenvironments studied. However, the concentrations we detected of TPhP and EHDPP in Nigerian indoor dust were at least an order of magnitude lower than those reported for western countries (Table S3; Fig. 2). The higher concentrations of aryl-OPEs reported in most western countries compared to those we detected in Nigeria, may be due to international differences in their use patterns [39,65,66].

Interestingly, concentrations of $\sum_5$OPEs (TCEP, TCIPP, TDCIPP, TPHP, and EHDPP) (DF = 100%) observed in this study (averages - offices:155 ng/g; homes: 182 ng/kg; cars: 153 ng/g and medical centres: 51.1 ng/g) were the lowest reported worldwide (Fig. 1), although comparable to the values reported in Nepal (247 ng/g) [67] and in Egypt (401 ng/g) [10]. The average concentration of $\sum_5$OPEs in this study for medical centres (51.1 ng/g) was below those reported for hospital wards in Sweden (10,200 ng/g) [68] and public microenvironments (PMEs) in Egypt (2118 ng/g) [10]. Concentrations of TPhP and EHDPP have been reported in different

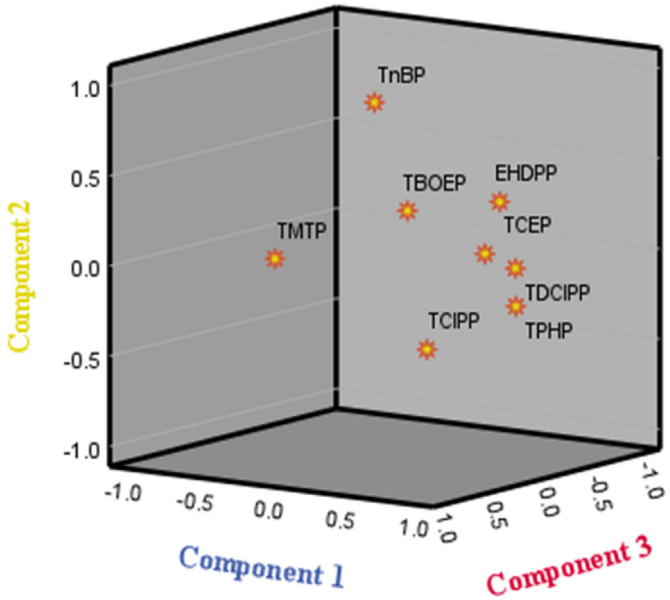

Fig. 3. Graphical representation of principal components PC-1 (48.98%), PC-2 (15.65%) and PC-3 (14.40%).

microenvironments from various countries, however, those obtained in this study were lower than those previously reported (Table S6). The highest concentrations of TPhP (30 ng/g) and EHDPP (37 ng/g) detected in this study were found in house and car dust respectively. The median concentrations of TPhP and EHDPP in dust from offices (18 and 22 ng/g), homes (18 and 20 ng/g), and cars (16 and 37 ng/kg) in this study were between 4-239 and 2–265 times lower than those reported in indoor dust from the same microenvironments in Egypt [10], China [8], and the UK [11]. Our mean concentrations for TnBP in: cars = 26 ng/g; offices = 8.6 ng/g; medical centres = 14 ng/g, and homes = <LOQ were below those reported in Egypt (cars: 74 ng/g; offices: 27 ng/g) [10], China (offices: 57 ng/g; homes: 38 ng/g) [8], Germany (cars: 110 ng/g; homes: 130 ng/g; and offices: 220 ng/g), and US, Canada and Czech Republic (homes: 114, 63, and 51.6 ng/g) [69] (Table S6). As observed by Ref. [10], the low concentrations of OPEs detected in house dust from Africa and some parts of Asia and South America may be attributed to factors such as: fewer fire safety regulations, combined with the tropical nature of the climate in such regions which leads to increased ventilation of indoor environments e.g., via air-conditioning.

3.2. Potential sources of OPEs in the studied indoor microenvironment in Nigeria

Concentrations of all OPEs were normally distributed using the Shapiro-Wilk and Kolmogorov-Smirnov tests (Table S7), thus Pearson rank correlations were performed to evaluate the possible common exposure sources and ascertain whether their presence in the consumer products from different countries are similar with this study. The results showed a significant correlation between the Cl-OPEs ($r = 0.346–0.586$; $p < 0.01$) (Table S8). In addition, TCEP showed significant correlation with TPhP, EHDPP, and TBOEP ($r = 0.403–0.470$; $p < 0.01$) (Table S8), TDCIPP showed a significant positive correlation with TPhP, EHDPP and TBOEP ($r = 0.421–0.791$; $p < 0.01$) while for TCIPP, the correlation was significant with TPhP ($r = 0.451$; $p < 0.01$) (Table S8). Moreover, TPhP and EHDPP were significantly correlated ($r = 0.453$; $p < 0.01$) as were EHDPP vs TBOEP ($r = 0.278$; $p < 0.05$) (Table S8). Those OPEs displaying significant positive correlations likely share a common source or sources. These findings are largely corroborated by the results of the principal component analysis (PCA) where the initial dimension reduction produced three components that explained 79% of the total variation (Table 2). The first principal component (PC-1) explained 48.98% of the total variation and was primarily driven in a positive direction by the three Cl-OPEs: TDCIPP (0.90), TCEP (0.75), TCIPP (0.59) and two aryl-OPEs: TPHP (0.82) and EHDPP (0.70) (Table 2). This supports the hypothesis that these OPEs have similar sources. The second and third principal components (PC-2 and PC-3) respectively explained 15.65% and 14.40% of the total variance and were driven primarily by TnBP (PC-2 = 0.879) and TMTP (PC-3 = 0.855) (Table 2; Fig. 3). These two compounds have been reported to have applications distinct from those of the other OPEs measured. Hierarchical cluster analysis (HCA) was also

Table 3
Estimated daily intakes (EDIs, ng/kg bw/day) of OPEs via indoor dust ingestion and dermal absorption for toddlers and adults in Nigeria (numbers in parentheses represent EDIs as a percentage of the corresponding reference dose value).

OPE	RfD (ng/kg bw/day)	Dust ingestion (ng/kg body weight/day)						Dermal absorption (ng/kg body weight/day)					
		Toddlers			Adults			Toddlers			Adults		
		Mean	Median	95th Percentile	Mean	Median	95th Percentile	Mean	Median	95th Percentile	Mean	Median	95th Percentile
TCEP	7000[a]	0.22 (0.003)	0.07 (0.001)	3.7 (0.05)	0.006 (<0.001)	0.003 (<0.001)	0.05 (<0.001)	0.12 (0.002)	0.04 (<0.001)	0.54 (0.008)	0.004 (<0.001)	0.002 (<0.001)	0.013 (<0.001)
TCIPP	10,000[a]	0.48 (0.005)	0.44 (0.004)	3.7 (0.04)	0.02 (<0.001)	0.02 (<0.001)	0.09 (<0.001)	0.25 (0.003)	0.22 (0.002)	0.47 (0.005)	0.013 (<0.001)	0.013 (<0.001)	0.022 (<0.001)
TDCIPP	20,000[a]	0.18 (0.001)	0.12 (0.001)	1.5 (0.008)	0.007 (<0.001)	0.005 (<0.001)	0.04 (<0.001)	0.05 (<0.001)	0.03 (<0.001)	0.10 (<0.001)	0.002 (<0.001)	0.001 (<0.001)	0.005 (<0.001)
TPHP	7000[b]	0.23 (0.003)	0.15 (0.002)	1.9 (0.03)	0.008 (<0.001)	0.006 (<0.001)	0.04 (<0.001)	0.08 (0.001)	0.05 (<0.001)	0.17 (0.002)	0.003 (<0.001)	0.002 (<0.001)	0.006 (<0.001)
EHDPP	600[c]	0.25 (0.04)	0.19 (0.03)	2.6 (0.43)	0.01 (0.002)	0.008 (0.001)	0.05 (0.008)	0.09 (0.02)	0.07 (0.01)	0.22 (0.04)	0.004 (0.001)	0.003 (0.001)	0.008 (0.001)
TBOEP	1500[b]	0.66 (0.04)	0.56 (0.04)	4.8 (0.3)	0.03 (0.002)	0.02 (0.001)	0.13 (0.009)	0.23 (0.02)	0.19 (0.01)	0.42 (0.03)	0.011 (0.001)	0.010 (0.001)	0.021 (0.001)
TnBP	10,000[a]	0.07 (0.001)	0.07 (0.001)	0.48 (0.005)	0.003 (<0.001)	0.003 (<0.001)	0.01 (<0.001)	0.02 (0.002)	0.02 (<0.001)	0.04 (<0.001)	0.001 (<0.001)	0.001 (<0.001)	0.002 (<0.001)
TMTP	–	0.05	0.002	0.97	0.003	0.0002	0.04	0.02	0.00	0.1	0.001	0.0001	0.005
$\sum_8$OPEs	–	2.1	1.6	19.8	0.09	0.07	0.45	0.86	0.63	2.05	0.04	0.03	0.08

[a] Reference dose (RfD) values of [75].
[b] [50].
[c] [76].

performed on the data to further establish the similarity between concentrations of OPEs using the average linkage method. This revealed two distinctive clusters with varied degree of similarities between the variables, as shown by the dendrogram provided in Fig. S2. The first cluster (Cluster-I) was comprised of: TDCIPP, TPhP, and EHDPP with major similarities with TCEP and TCIPP and a minor similarity with TBOEP through the subclusters (Fig. S2), that suggests similar sources of these OPEs. Cluster-II consisted of TnBP and TMTP (Fig. S2). Thus, the HCA findings are consistent with both our correlation analyses and PCA.

3.3. Human exposure to OPEs via indoor dust ingestion and dermal absorption

Concentrations of OPEs in indoor dust in this study were used to assess the exposure via dust ingestion and dermal absorption from dust of Nigerian toddlers and adults. Estimated daily intakes (EDIs) of our target OPEs via indoor dust ingestion and dermal absorption in Nigeria are summarised in Table 3. The mean and high exposure doses (based on the mean and 95th percentile concentrations respectively) are presented in Table 3. The same table also expresses EDIs for each OPE as a percentage of the corresponding reference dose (RfD) (provided in parentheses in Table 3). The results show that the median exposure doses of $\sum_8$OPEs via indoor dust ingestion and dermal absorption from the microenvironments were (1.6 and 0.07 ng/kg bw/day) and (0.63 and 0.03 ng/kg bw/day) for toddlers and adults respectively (Table 3; Table S10; Fig. S3). As a result of their lower body weight, more frequent hand-to-mouth contact [1], and greater proximity to the floor; toddlers are more exposed to OPEs than adults. Specifically, the median EDI value for ΣOPEs obtained for toddlers in this study was about 21–23 times higher than that for adults via the two exposure pathways considered (Table 3; Table S10; Fig. S3). The median estimated EDIs for toddlers and adults in this study via indoor dust ingestion and dermal absorption were comparable to those reported in China [54,72], Nepal [67] and Egypt [73]; below the value reported in the UK [11], Germany [38], Pakistan and Kuwait [61], Saudi Arabia [71], Egypt [10], the USA, Canada, and the Czech Republic [69], Iraq [44], Canada, Turkey, and Egypt [39]; China [8,50], South Africa [9] US [74], and Norway [75]. However, the median and high-end EDIs obtained for TCEP (4.3×10^{-5} and 1.6×10^{-4} ng/kg bw/day), TCIPP (2×10^{-4} and 5×10^{-4} ng/kg bw/day), and TDCIPP (6×10^{-5} and 4.5×10^{-4} ng/kg bw/day) for adults in medical centres in this study, were below the value reported in hospital wards in Sweden [68], in indoor dust from Egypt [10], China [8], and Canada [39] (Table S11). In the same vein, our median EDI value for six OPEs (TCEP, TCIPP, TDCIPP, TPHP, EHDPP and TnBP) combined for dermal absorption from dust for adults (0.02 ng/kg bw/day) was lower than the value reported for dermal absorption in Nepal (5.58 ng/kg bw/day) [67]. In this study, our median and high exposure EDIs via dust ingestion for toddlers in offices (0.50 and 9.5 ng/kg bw/day) and house dust (0.71 and 7.0 ng/kg bw/day) were significantly higher ($p < 0.05$) than those obtained for cars/buses (0.37 and 3.1 ng/kg bw/day) and medical centres (0.02 and 0.17 ng/kg bw/day) respectively (Table 3; Table S10; Fig. S3). The same trends were observed for dermal absorption from dust for adults and toddlers (Table 3; Table S10; Fig. S3).

In addition, the overall mean, median, and high exposure scenario EDIs for each OPE for both toddlers and adults were compared with their respective reference doses (RfDs) obtained from Refs. [55,76,77] (value in parenthesis in Table 3) to ascertain the human health risk via the two exposure pathways (Table 3). The results showed that TBOEP poses the greatest exposure risk of our target OPEs, with OPE risk arising from both pathways combined falling in the order: TBOEP > EHDPP > TPHP > TCIPP > TCEP > TDCIPP > TnBP for adults and toddlers (Table 3). There is no RfD value for TMTP currently with which the TMTP EDI value can be compared. In summary, the levels of exposure for OPEs were several orders of magnitude lower than their respective RfD values except for some compounds such as TBOEP and EHDPP where the EDI value under the high exposure scenario for toddlers via indoor dust ingestion was about 0.3% and 0.4% of their respective RfD value (Table 3; Table S10). Similar to what was reported for indoor dust in Greece by Ref. [37], the exposure risk obtained in this study via dust ingestion exceeded that from dermal absorption (Table S10). Our data for the sum of the median EDIs for all the microenvironments studied for dust ingestion for toddlers (1.6 ng Σ_8OPEs/kg bw/day) and dermal absorption (0.63 ng Σ_8OPEs/kg bw/day) showed that dust ingestion contributed about 72% while dermal absorption comprised 28% of the total exposure (Table 3). For adults, dust ingestion contributed about 70% with the remaining 30% arising from dermal absorption (Table S10). Therefore, for both toddlers and adults, dust ingestion is the main human exposure route to OPEs present in indoor dust.

4. Conclusions

This present study reports the concentrations of OPEs in indoor dust from four microenvironments (cars/buses, offices, homes, and medical centres) in Nigeria for the first time. Concentrations of OPEs detected in this study were comparable to those reported in other developing African and Asian countries and lower than the values reported in most western countries. Our findings add to the increasing evidence of indoor contamination with OPEs. Correlation, principal component, and hierarchical cluster analyses revealed similar sources for most target OPEs, but distinct sources of TnBP and TMTP. Assessment of exposure to OPEs of Nigerians via indoor dust ingestion and absorption from dermal contact with indoor dust and comparison with the corresponding health-based limit values, do not reveal any significant health risks associated with such exposure. Exposure via dust ingestion exceeds that via dermal absorption from contact with dust. However, further studies are needed to fully determine overall human exposure to OPEs of the Nigerian population via other exposure routes including air inhalation [78], dietary ingestion [23], water ingestion [12], and dermal uptake via contact with OPE-containing products like furniture [79].

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.emcon.2023.100208.

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