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Exposure to organophosphate esters in elderly people: Relationships of OPE body burdens with indoor air and dust concentrations and food consumption

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ABSTRACT

Human exposure to OPEs is pervasive and should be of great concern due to associations with adverse health effects, especially in susceptible populations. In this study, body burdens and exposure pathways of OPEs were investigated for 76 healthy elderly people in Jinan, China based on the measured OPE and metabolite concentrations in human bio-samples (whole blood and urine) and paired environmental matrices (air and dust), as well as food frequency questionnaire. Eight of 16 OPEs and 5 of 11 metabolites were detected in > 50% of whole blood and urine samples, respectively. Tri(1-chloro-2-propyl) phosphate (TCIPP), tris(2-chloroethyl) phosphate (TCEP), tri-phenyl phosphate (TPHP), and 2-ethylhexyl di-phenyl phosphate (EHDPP) were frequently detected and abundant in whole blood, while their corresponding metabolites were detected at low frequencies or levels in urine. The reduced metabolic and/or excretory capacity of elderly people may be an important reason, implying a higher health risk to them. Fourteen OPEs had over 50% detection frequencies in indoor air and dust, while 6 di-esters in indoor dust. Tris(2-ethylhexyl) phosphate (TEHP) in indoor dust and tri-n-butyl phosphate (TnBP) in indoor air were positively correlated with paired levels in blood but not with their metabolites (BEHP and DnBP) in urine. Combined with the direct intakes of BEHP and DBP from dust, blood is indicated as more suitable biomarker for TEHP and TnBP exposure. High consumption frequencies of several foods were associated with higher blood concentrations of three OPEs and urinary levels of four di-OPEs, indicating the importance of dietary exposure pathway. Estimated daily total intakes of OPEs *via* inhalation, dust ingestion, and dermal absorption ranged from 2.78 to 42.0 ng/kg bw/day, which were far less than the reference dosage values. Further studies were warranted to explore the potential health effects of OPE exposure in the elderly populations.

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1. Introduction

Organophosphate esters (OPEs) are substitutes for polybrominated diphenyl ethers (PBDEs) and are widely used as flame retardants in plastics, textiles, and polyurethane foams as well as plasticizers in resins and synthetic rubbers (van der Veen and de Boer, 2012; Wei et al., 2015). Some OPEs are also used for other applications such as hydraulic fluids, food packaging materials, floor waxes, and nail polishes (Larsson et al., 2018; Mendelsohn et al., 2016; van der Veen and de Boer, 2012). Global use of OPEs was approximately 500,000 tonnes in 2011 and has increased annually by 7.9% (Zhang et al., 2018b). In China, production of OPEs was 100,000 tonnes in 2011 with the demand expected to increase 15% annually (Ali et al., 2017).

Since OPEs are often physically added to but not covalently bound to various products (Wang et al., 2017; Wei et al., 2015), they can be easily released by volatilization, abrasion, and leaching (Wei et al., 2015), which has led to ubiquitous detection in various surrounding microenvironments (e.g., homes and office) globally (Cequier et al., 2014; Li et al., 2019b; Shoeib et al., 2019; Tao et al., 2019). OPEs have been frequently found in multiple environmental matrices (e.g., air, dust, food, etc.) (Cequier et al., 2014; Li et al., 2019a; Meng et al., 2020; Vykoukalova et al., 2017), and numerous adverse health outcomes have been reported, including carcinogenicity (van der Veen and de Boer, 2012; Wei et al., 2015), endocrine disruption (Zhang et al., 2016; Zhang et al., 2014), neurotoxicity (Dishaw et al., 2011) and reproductive toxicity (Zhang et al., 2018c), raising great concerns about human exposure. Results of both *in vitro* and *in vivo* studies have indicated that OPEs are readily metabolized to their respective diesters (di-OPEs) and/or hydroxylated metabolites (OH-OPEs) (Hou et al., 2018; Su et al., 2015; Van den Eede et al., 2013; Wang et al., 2016). Therefore, evaluation of human OPE exposure has previously mainly focused on measurement of these OPE metabolites (m-OPEs) in urine (He et al., 2018b; Hoffman et al., 2017; Sun et al., 2018; Wang et al., 2019; Zhang et al., 2018b), while few studies focused on parent OPEs in blood (Hou et al., 2020a; Wang et al., 2020a; Ya et al., 2019; Zhao et al., 2016). Human blood could be used as a more proximal measure of accumulated OPEs and internal doses. Additionally, to the best of our knowledge, no studies have investigated temporal variability of OPEs in human blood.

Because of deteriorations in physiological functions, such as decreases in organ functions, metabolic processes, hepatic and renal clearances, and cellular defense mechanisms, the elderly people ($\geq$60 years of age) are particularly susceptible to environmental chemical exposures (Choi et al., 2017; Hong, 2013; Tuttle et al., 2013). Moreover, the behavior of the elderly populations (e.g., spending more time at home due to retirement and declining physical functioning) (Tuttle et al., 2013) and their physiological changes might result in greater exposure to OPEs and differential distribution and metabolic characteristics compared with younger individuals. However, body burdens and potential health risks of OPEs in these susceptible elderly populations remained unclear.

Individuals can be exposed to OPEs *via* inhalation, ingestion, and dermal absorption. Although previous studies have investigated pathways of exposure to OPEs, no consistent conclusions had been reached. Also, most previous studies had only focused on OPEs in environmental matrices to evaluate external exposures (Cequier et al., 2014; Kim et al., 2019; Schreder et al., 2016; Zhao et al., 2020) or di-OPEs in urine to monitor the internal doses with the assumption that di-OPEs in human body were the result of OPE metabolism. Only a few studies have investigated the associations between external exposures and internal doses for specific populations (e.g., children and adults) and have been in a limited spectrum of exposure pathways (Dodson et al., 2014; Larsson et al., 2018; Phillips et al., 2018; Xu et al., 2019a; Xu et al., 2016). Additionally, di-OPEs have been detected in settled dust and food products (He et al., 2018c; Hu et al., 2020; Li et al., 2020b; Tan et al., 2019), and OH-OPEs (e.g., 3-hydroxyphenyl diphenyl phosphate (meta-OH-TPHP)) have been found in sediment samples as impurities of some OPEs (Ye et al., 2021), indicating possible direct human exposure to them. Thus, whether di-OPEs in urine are derived from direct exposure or from OPE metabolism needs to be elucidated, which will help us to understand whether and to what extent direct exposure interferes with urinary di-OPEs as biomarkers for assessing OPE exposure.

In the present study, OPEs and their metabolites (Table S1) were measured in whole blood and urine collected from healthy elderly people together with their paired environmental samples (air and dust). The objectives were to (1) monitor the internal doses of OPEs and their metabolites and characterizing the metabolism of OPEs in elderly people; (2) explore the relationships between OPEs/di-OPEs in whole blood/urine from elderly people with air, and dust concentrations of corresponding OPEs, as well as food consumption; and (3) estimate exposure to OPEs *via* three potential exposure pathways and to di-OPEs *via* dust ingestion on the basis of the measured levels in environmental matrices and determining the contributions of different exposure pathways.

2. Materials and methods

2.1. Study design

This study was conducted as part of the Biomarkers of Air Pollutants Exposure in the Chinese aged 60–69 (China BAPE) study (Fang et al., 2020; Guo et al., 2021; Koelmel et al., 2020; Zhou et al., 2020) in collaboration with the Ankang Community Hospital. Study participants were recruited from the Dianliu Community (28,025 residents) in Jinan, Shandong Province, China, which is a major city in northern China with over 8 million population. Healthy elderly individuals, meeting the following criteria were included in this panel study: (1) age between 60 and 69; (2) healthy without any acute or chronic diseases; (3) not smoking or abusing alcohol or use of medication; and (4) no plans to travel during the survey. Ultimately, a total of 76 participants from 69 homes (including 7 couples) were included. These participants were equally distributed between male and female (50% male). Fifty nine percent of participants were older than 65. Their mean BMI was 24.8 with a range of 17.1–29.5. Mean (SD) period at home per day was 18.2 $\pm$ 1.86 h (Table S2). All participants provided a written informed consent, and the study was approved by the Ethical Commissions of the National Institute of Environmental Health (NIEH), China CDC (No. 201816).

2.2. Onsite investigation and collection of bio-samples

Participants completed questionnaires and medical examinations at the Ankang Community Hospital once a month for 5 continuous months between September 2018 and January 2019. Information such as demographics, residential characteristics, and frequencies of food consumption were collected. During each physical examination, fasting whole blood and morning urine samples (7:00 AM) were collected for each participant by a medical professional. Samples of urine were collected in standard polypropylene specimen containers. Whole blood was collected into anticoagulant vacutainer and then transferred 2 mL into a vial (CryoKING, Biologix, USA). Nineteen of the participants missed 1–3 samplings. Thus, a total of 705 bio-samples (352 whole blood and 353 urine) were collected. These samples were immediately placed in dry ice and were subsequently transported to the laboratory in Beijing by a cold-chain shipment. All samples were stored at −80 °C until analysis.

2.3. Collection of dust, air, and frequencies of food consumption

Dust and air samples were collected along with the fourth and fifth onsite investigation and bio-samples collection. Specifically, settled dust was collected from the living room of each participant's residence (n = 64) twice in December 2018 (the day before the fourth collection of bio-samples) and January 2019 (the day before the fifth collection of bio-

samples), respectively, using a domestic vacuum cleaner (Midea C3-L143C, China) with separate home-made nylon socks inserted into the nozzle. Meanwhile, outdoor dust was collected from multiple locations on the roof of the community hospital and mixed into a composite sample. Indoor air samples (n = 63) were collected in participant's residences for 30 days during the period of two sampling campaigns for dust samples by use of double-bowl passive air samplers, containing a sorbent (finely ground XAD-4 resin (Supelco, Bellefonte, PA, USA))-impregnated polyurethane (SIP) foam disk. At the same time, outdoor air samples (n = 6) were collected from approximately 1.5 m above the roof on top of the community hospital. Following each sampling period, all samples were wrapped in aluminum foil and sealed in ziplock bags for transportation to the laboratory. Dust samples from each household were combined into a composite sample. Samples were then sieved through a precleaned 500 μm mesh sieve, homogenized thoroughly, and stored at −18 °C until analysis. SIP air samples were extracted as soon as they were transported to the laboratory. More details of dust and air sampling are described in the Supporting Information (SI).

During each onsite investigation, information on the participants' consumption frequencies of 24 different foods or drinks (e.g., water, rice, meat, milk, and egg) in the past month were collected *via* questionnaires. The frequencies were aggregated into two- or three-category variables, such as <3 days a month, 1–3 days a week, and >4 days a week (Table S3). Additionally, the community hospital provided all the participants with three meals (breakfast, lunch, and dinner) for five consecutive days before the collection of bio-samples. Each meal was the same for all participants and was provided with a fixed amount of food, including the staple food, vegetables, meat, egg, fruits, and porridge. Information on whether the participants drank tea or intook other diets in addition to the meal provided during the 3 days before bio-sample collection were collected. The amount of water participants drank daily during the 3 days before bio-samples collection were also recorded (Table S3).

2.4. Quality assurance and quality control

Procedural blanks, field blanks, and matrix spiked recoveries were used to examine potential background contamination and to ensure the quality of the generated data. The concentrations of analytes were corrected with blanks (OPEs: <method detection limit (MDL)-0.163 ng/mL for whole blood and urine; <MDL-0.237 ng/m^3 for air; <MDL-6.25 ng/g for dust; metabolites: <MDL-0.0780 ng/mL for urine and < MDL-2.29 ng/g for dust, respectively) (Tables S4 and S5). Additionally, standard reference materials-SRM2585 and SRM3673 (NIST, USA) were used as quality controls in the analysis of OPEs in dust and di-OPEs in urine (Tables S6 and S7), respectively. More detailed information about QA/QC are described in the SI.

2.5. Statistical analysis

Statistical analyses were performed by using SPSS 19 (IBM, New York, US) and were performed only for analytes with a detection frequency (DF) > 50%. A value of MDL/2 was used as a surrogate to estimate concentrations less than the MDLs. Concentrations of target analytes in urine were corrected by specific gravity (SG) (1.015–1.030) as creatinine (Cr) had a significant sex difference ($p < 0.001$) in this study (SI). Because data were not normally distributed, nonparametric statistical tests were applied. Spearman correlation analyses (r_s) were used to determine the correlations between analytes, between sample types for a target analyte, between analyte levels in whole blood or urine and demographics or food intake frequencies of participants, and between OPE concentrations in indoor air or dust and residential characteristics. The demographics that showed significant correlations in the bivariable analyses were further included in a linear regression model to evaluate their relationships with analyte concentrations in bio-samples. Additionally, linear regression analyses were also performed to assess the associations between analytes in bio-samples with OPEs in environmental matrices and the different food intake frequencies. The models were adjusted for age, sex, and BMI of participants. Nonparametric paired tests (Wilcoxon test) were used for comparisons of analyte compositions among matched air and dust samples. Pearson correlation (r_p) was used to determine the correlations between median values of log concentration ratios of dust to air for OPEs and their logK_{oa} (Table S1). Statistical significance was set at $p = 0.05$. Intraclass correlation coefficients (ICCs) and their 95% confidence intervals (CIs) were used to assess the time variability in urinary di-OPE levels and blood OPE concentrations using mixed random-effects models. Estimated daily intakes (EDIs) of OPEs or di-OPEs for the elderly were calculated based on the concentrations in air and dust, on the blood OPE concentrations, and on the urinary di-OPE levels, respectively. The detailed estimation equations and parameters are displayed in the SI.

3. Results and discussion

3.1. OPEs and m-OPEs in human bio-samples

The detection frequencies, concentration ranges, medians, and geometric means for OPEs and m-OPEs in whole blood and urine samples are summarized in Table 1 and Fig. 1c, d.

3.1.1. OPEs in whole blood

Concentrations of $\sum_{16}$OPEs in whole blood samples (n = 352) ranged from 0.818 to 21.6 ng/mL (median 4.96 ng/mL). TCIPP, TPHP, and EHDPP were detected in 77–78% of samples, followed by TnBP, TCEP, TEP, TiBP and TEHP with frequencies from 51% to 59%. The DFs of the other measured OPEs were <50%. Among the 16 OPEs measured, TCIPP (median: 0.743 ng/mL) and TPHP (0.400 ng/mL) were the predominant compounds, contributing 28% and 15% of $\sum_{16}$OPEs, respectively (Fig. 1c, f), which is in line with a study conducted in Hengshui City, China (Wang et al., 2020a). However, in human blood collected from Beijing (Hou et al., 2020a) and four cities in Jiangsu province, China (Ya et al., 2019), EHDPP was found to be the most abundant OPE, while TnBP predominated in human blood from Shenzhen, China (Zhao et al., 2016). The concentrations of TPHP in this study were similar to those observed in several previous studies in China (median range: 0.35–0.46 ng/mL) (Table S10). TCIPP concentrations were comparable to those observed in Shenzhen (0.71 ng/mL) (Zhao et al., 2016), but greater than those measured in Jiangsu (0.05 ng/mL) (Ya et al., 2019), Hengshui (0.36 ng/mL) (Wang et al., 2020a), and Beijing (ND) (Hou et al., 2020a). Such differences might be due to differences in blood matrices used (e.g., whole blood or serum/plasma), environmental factors (e.g., residential characteristics, lifestyles, or diet), or the metabolism of various population in these studies. Significant positive correlations were found among several OPEs in whole blood, such as TnBP and TiBP ($r_s = 0.553$, $p < 0.01$), TEHP and EHDPP ($r_s = 0.301$, $p < 0.01$), and TCEP and TCIPP ($r_s = 0.289$, $p < 0.01$) (Table S11).

To date, the temporal variability of OPEs in whole blood has not been considered, which is important for understanding whether a spot sample is representative of an individual longer-term exposure. In this study, moderate variations across the five-month period were found for TEP (ICC: 0.41, 95% CI: 0.13–0.62) and TCIPP (0.40, 95% CI: 0.11–0.62), while the other OPEs (TnBP, TiBP, TEHP, TCEP, TPHP, and EHDPP) exhibited relatively great variabilities (ICC: <0.27) (Table S12), which might be due to the rapid metabolism of OPEs in human body or different exposures per month. Associations between OPE concentrations in whole blood and demographic factors were analyzed (Tables S13 and S14). The concentrations of TCIPP ($n = 76$, $r_s = 0.241$, $p = 0.036$), TnBP ($n = 76$, $r_s = 0.310$, $p = 0.007$), and TiBP ($n = 76$, $r_s = 0.354$, $p = 0.002$) were significantly increased with age. Male blood concentrations of $\sum_{16}$OPEs were significantly higher than those of female (1.17 times, 95 %CI: 1.04–1.31, $p = 0.012$). However, such a relationship was not observed for individual OPE.

Table 1
Concentrations of OPEs and m-OPEs in whole blood (n = 352) and urine (n = 352) from 76 elderly people.

Chemicals	Whole blood (ng/mL)				Chemicals	Urine (ng/mL)[c]			
	DF[a]	Range	Median	Geomean		DF	Range	Median	Geomean
TMP	16	<MDL-6.25	-[b]	-[b]	TEP	43	<MDL-2.83	-[b]	-[b]
TEP	54	<MDL-18.4	0.136	0.148	TPrP	68	<MDL-3.04	0.0640	0.0510
TPrP	0.90	<MDL-0.250	-[b]	-[b]	TnBP	15	<MDL-1.20	-[b]	-[b]
TnBP	59	<MDL-5.14	0.250	0.245	TiBP	29	<MDL-1.08	-[b]	-[b]
TiBP	51	<MDL-6.13	0.229	0.295	TBOEP	17	<MDL-3.86	-[b]	-[b]
TEHP	51	<MDL-3.46	0.0910	0.132	TCEP	17	<MDL-2.40	-[b]	-[b]
TBOEP	41	<MDL-6.17	-[b]	-[b]	TCIPP	40	<MDL-1.47	-[b]	-[b]
TPHP	78	<MDL-7.52	0.400	0.408	TDCIPP	48	<MDL-1.61	-[b]	-[b]
EHDPP	77	<MDL-3.57	0.209	0.132	DBP	69	<MDL −0.661	0.0520	0.0510
CDPP	7	<MDL-1.10	-[b]	-[b]	BEHP	77	<MDL-1.91	0.146	0.147
TMPP	33	<MDL-0.162	-[b]	-[b]	BBOEP	13	<MDL-16.7	-[b]	-[b]
BABP	47	<MDL-0.0690	-[b]	-[b]	BBOEHEP	3.0	<MDL-0.202	-[b]	-[b]
RDP	21	<MDL-0.370	-[b]	-[b]	DPHP	79	<MDL-6.82	0.0890	0.0830
TCEP	56	<MDL-5.06	0.298	0.307	4-OH-DPHP	21	<MDL-0.242	-[b]	-[b]
TCIPP	77	<MDL-4.45	0.743	0.508	5-OH-EHDPP	13	<MDL-0.205	-[b]	-[b]
TDCIPP	34	<MDL-2.57	-[b]	-[b]	BMPP	65	<MDL-0.159	0.0140	0.0130
$\sum_{16}$OPEs	100	0.818–21.6	4.96	5.00	BCEP	20	<MDL-8.53	-[b]	-[b]
					BCIPP	28	<MDL-1.30	-[b]	-[b]
					BDCIPP	76	<MDL-4.78	0.121	0.106

[a]DF: Detection frequency; [b]Median and geomean values were not calculated due to low DFs; [c]SG-adjusted urinary concentrations. The SG of one participant's urine was not available, so the number of urine samples was 352. The unadjusted urinary concentrations were showed in Table S15. The DFs of TEHP, TPHP, EHDPP, TMPP, CDPP, BABP, and RDP in urine samples were lower than 5%.

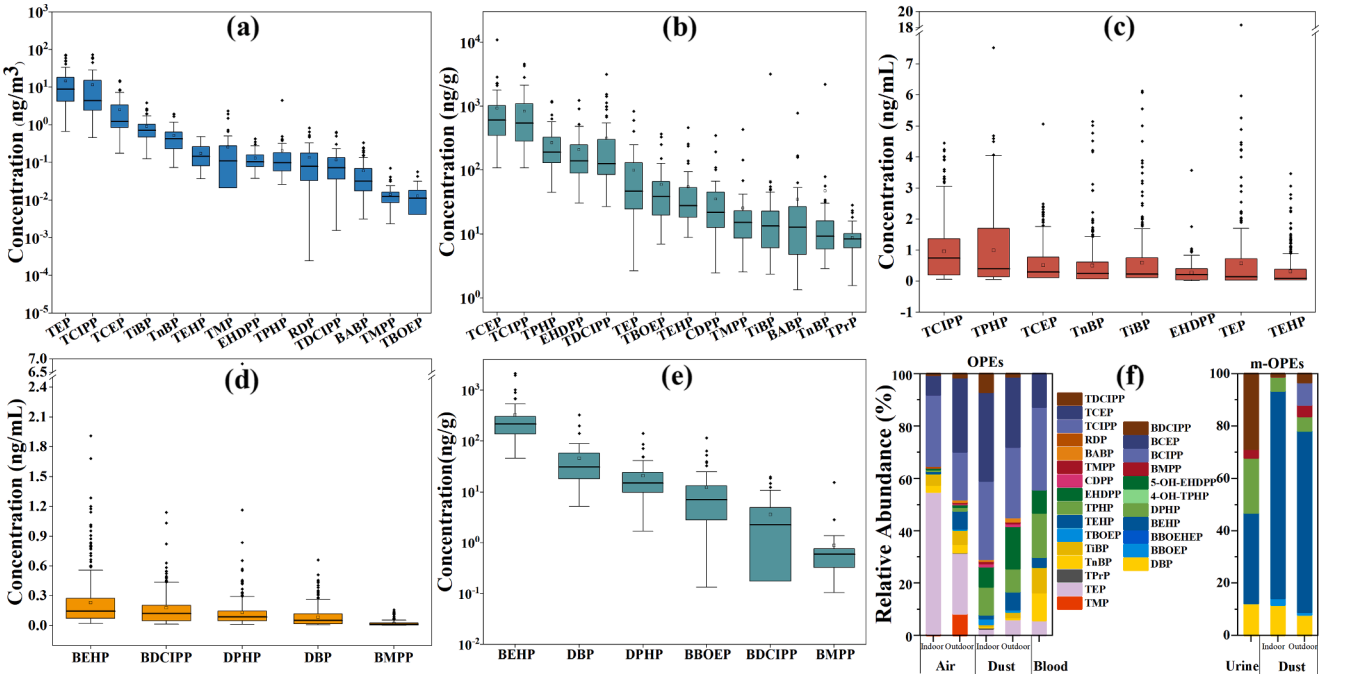

Fig. 1. Concentrations of OPEs in indoor air samples (n = 63) (a), indoor dust samples (n = 64) (b), and whole blood samples (n = 352) (c). Concentrations of di-OPEs in urine samples (n = 352) (d) and indoor dust samples (n = 61) (e). Composition profile of OPEs and m-OPEs in various environmental and human matrices (f). Only analytes with DF > 50% were showed. Horizontal lines on the boxplots represent the 25th, 50th and 75th percentiles; the whiskers represent ± 1.5 interquartile range (IQR); and dots represent outliers.

3.1.2. OPEs and m-OPEs in urine

OPEs and their 8 diester and 3 hydroxylated metabolites were measured in urine (n = 353) (Table 1). All OPEs, except TPrP (68%), had DFs<50%, indicating that urinary excretion in the form of triesters is not the main elimination route of OPEs from the human body, especially with higher molecular weight and hydrophobic compounds. Both *in vitro* and *in vivo* studies have shown that several OPEs such as TPHP, EHDPP and TBOEP can be metabolized to their corresponding hydroxylated metabolites (Hou et al., 2018; Su et al., 2014; Van den Eede et al., 2013). However, in this study, three hydroxylated metabolites, bis(2-butoxyethyl) hydroxyethyl phosphate (BBOEHEP/desbutyl-TBOEP), 4-hydroxyphenyl diphenyl phosphate (4-OH-DPHP), and 2-ethyl-5-hydroxyhexyl diphenyl phosphate (5-OH-EHDPP), were detected in 3.0–21% of urine samples, which were similar to the results from adolescents in Hangzhou, China (Ding et al., 2019) and adults in Ontario, Canada (Siddique et al., 2020), but different with those from adults in Shenzhen, China (Zhao et al., 2019a) and children in Australia (He et al., 2018b) and Sapporo, Japan (Araki et al., 2018), where 5-OH-EHDPP, 4-OH-DPHP or

BBOEHEP were detected in most of urine samples. These observations may be related to the differences in exposure characteristics or metabolisms of the various populations.

Among the eight diester metabolites, DBP (DnBP + DiBP), BEHP, BDCIPP, DPHP, and BMPP (DoCP + DpCP) were detected in 65–79% of urine samples, whereas low DFs were observed for BBOEP, BCEP, and BCIPP (<28%). Strong correlations were found between unadjusted and SG-adjusted urinary concentrations (r_s = 0.863–0.920, p < 0.0001) (Table S15). Therefore, SG-adjusted concentrations of di-OPEs were used in further analyses. In this study, BEHP exhibited the greatest median concentration (0.146 ng/mL), which was lower than that observed in Beijing (6.76 ng/mL) (Hou et al., 2020a), but higher than those found in Shanghai, China (0.0864 ng/mL) (Sun et al., 2018), Australia (not detected) (He et al., 2018a), and New York, USA (0.015 ng/mL) (Wang et al., 2019). BDCIPP (0.121 ng/mL) was the second most abundant di-OPE in urine, followed by DPHP (0.0890 ng/mL), DBP (0.0520 ng/mL), and BMPP (0.0140 ng/mL) (Fig. 1d). Concentrations of BDCIPP were comparable to those reported for adults and children from several Chinese cities (0.05–0.291 ng/mL) (Chen et al., 2018; Chen et al., 2019; Hou et al., 2020a; Zhang et al., 2018a; Zhang et al., 2018b), but significantly lower than those of adolescents from Hangzhou, China (6.17 ng/mL) (Ding et al., 2019) and people from the USA (Butt et al., 2014; Hoffman et al., 2014; Petropoulou et al., 2016; Thomas et al., 2017; Wang et al., 2019) and Australia (He et al., 2018a; He et al., 2018b; Van den Eede et al., 2015). Concentrations of DPHP and DBP detected here were lower compared to most other studies (Table S16). The observed low DFs and concentrations of OPE metabolites in urine might be due to the low sensitivity of BCEP (MDL: 0.625 ng/mL), the lower OPE concentrations found in homes of participants than other studies (Tables S21 and S24), and the reduced metabolic capacity and renal clearance of the elderly (Choi et al., 2017; Tuttle et al., 2013). This is evidenced by a previous study showing that urinary levels of DPHP, BDCIPP, and 1-hydroxy-2-propyl bis(1-chloro-2-propyl) phosphate (BCIPHIPP) in people older than 60 were significantly lower than those in younger people (Van den Eede et al., 2015). Correlation analyses within di-OPEs with DFs > 50% in urine showed that BDCIPP, DBP, DPHP, and BMPP are significantly positively correlated with each other (r_s = 0.292–0.438, p < 0.001), but not with BEHP (Table S17), suggesting that DBP, BDCIPP, DPHP and BMPP have common sources and similar toxicokinetic processes in the human body.

Previous studies have reported moderate to strong diurnal and inter-day reliabilities in urinary di-OPE concentrations (Cequier et al., 2015; Hoffman et al., 2014; Hoffman et al., 2015; Meeker et al., 2013; Romano et al., 2017). Similarly, moderate reliabilities across the study's longitudinal sampling period were observed for SG-adjusted concentrations of DPHP, BMPP and BDCIPP (ICCs: 0.41–0.64). Relatively lower ICC values (<0.29) were observed for DBP and BEHP, which indicated their strong variabilities in concentrations over the five-month period (Table S12). No significant relationships were found between concentrations of di-OPEs in urine and demographic characteristics of study participants (Tables S18).

3.1.3. Associations between whole blood and urine

No significant correlations were observed between OPEs and their respective di-OPEs in matched samples of whole blood and urine for compounds with DFs > 50% (TBP-DBP, TEHP-BEHP, and TPHP/EHDPP-DPHP) (Table S19). This might result from direct exposure of people to metabolites because that DBP, BEHP, and DPHP were found as the main di-OPEs in indoor dust during this study (Fig. 1e, f; Table S21) and in indoor dust and food samples in several previous studies (He et al., 2018c; Hu et al., 2020; Tan et al., 2019). Results of *in vitro* studies have demonstrated that other aryl-OPEs, except TPHP and EHDPP, could also metabolized to DPHP (Ballesteros-Gomez et al., 2015b; Phillips et al., 2020; Van den Eede et al., 2013), which might be another reason for the lack of correlations between TPHP/EHDPP and DPHP. In addition, differences in metabolisms and excretions among individuals are likely to weaken the observed correlations.

3.2. OPEs and di-OPEs in environmental matrices

Presence of OPEs in the air and dust samples (indoor and outdoor) (Fig. 1a, b and Table S20) as well as di-OPEs in dust samples (Fig. 1e and Table S21) were investigated to characterize external exposure to OPEs/di-OPEs for elderly people.

3.2.1. OPEs in air and dust

Eleven OPEs were detected in most indoor air samples (>97%) except TMP, TPrP, TBOEP, RDP and CDPP (Table S20). Median concentrations of TEP (8.85 ng/m^3), TCIPP (4.42 ng/m^3), and TCEP (1.23 ng/m^3) were 1–2 orders of magnitude greater in indoor air than those of other OPEs (0.0110–0.175 ng/m^3), accounting for 44%, 32%, and 11% of $\sum_{16}$OPEs, respectively (Fig. 1a, f). Fourteen OPEs were detected in almost all indoor dust samples (83–100%) except TMP and RDP. Like indoor air, TCEP (610 ng/g) and TCIPP (544 ng/g) were the most abundant OPEs in indoor dust, accounting for 28% and 27% of $\sum_{16}$OPEs, respectively (Fig. 1b, f). These findings support the extensive use of TCIPP and TCEP in Chinese commercial products. The concentration of $\sum_{16}$OPEs in outdoor air (2.78 ng/m^3) was approximately 10-fold less than that in indoor air (21.4 ng/m^3). $\sum_{16}$OPE concentration in outdoor dust (725 ng/g) was approximately three times less than that in indoor dust (2340 ng/g). Detailed discussion of air and dust OPE concentrations and correlations with building characteristics are presented in the SI (Text S8-S9).

3.2.2. Di-OPEs in dust and associations with corresponding parents

BEHP, DBP, DPHP, BBOEP, and BMPP were detected in almost all indoor dust samples (≥98%). BDCIPP was detected in 62% of the samples, whereas BCEP and BCIPP had lesser DFs (37%) (Table S21). Among the 8 di-OPEs measured, median concentrations of BEHP (216 ng/g), DBP (31.0 ng/g), and DPHP (14.9 ng/g) were 1–3 orders of magnitude greater than those of other di-OPEs (<MDL-7.10 ng/g) (Fig. 1e), which might be associated with their direct commercial applications (Bjornsdotter et al., 2018; Quintana et al., 2006). Like indoor dust, BEHP was also the most abundant di-OPE in outdoor dust (45.8 ng/g), followed by BCIPP (5.63 ng/g), DBP (5.22 ng/g) and DPHP (3.69 ng/g). To explore sources of di-OPEs in indoor dust, correlations between di-OPEs and OPEs were examined and concentration ratios were calculated for pairs of di-OPEs and OPEs (Fig. S2, Table S21). The median concentration ratios of BEHP and DBP to their respective tri-OPEs (TEHP and TBP) were 7.49 and 1.35, respectively. No correlations were observed for these two pairs, suggesting that BEHP and DBP in indoor dust may mainly originate from their direct applications in consumer products (Wang et al., 2020b). DPHP was significantly correlated with TPHP (r_s = 0.401, p = 0.001), EHDPP (r_s = 0.299, p = 0.020), CDPP (r_s = 0.356, p = 0.005) and the combination of several aryl-OPEs (including TPHP, EHDPP, CDPP, RDP, and BABP) (r_s = 0.412, p = 0.001). The concentration ratios of DPHP to these aryl-OPEs were all lower than 1 (median range: 0.0290–0.649), suggesting that there are three possible sources of DPHP in indoor dust: (1) the degradation of aryl-OPEs under environmental condition; (2) DPHP as an impurity in commercial aryl-OPEs formulas; (3) the application of DPHP in consumer products where aryl-OPEs are added (Wang et al., 2020b; Bjornsdotter et al., 2018). The di-/tri-OPEs ratios for other di-OPEs were close to zero, indicating the lack of commercial applications of these di-OPEs or the weak degradability of their parents. However, the possible degradation of OPEs in materials during industrial manufacturing procedures should not be ignored (Xu et al., 2019b), which needs to be explored in further studies.

3.3. Metabolic characteristics of OPEs in elderly people

Composition profiles of OPEs and di-OPEs in environmental and human matrices were compared to understand the distribution and metabolic characteristics of OPEs in elderly people (Fig. 1). TCIPP and TCEP were the dominant OPEs found in indoor air, dust, and whole blood, accounting for 35% (sum of TCIPP and TCEP), 64%, and 44% of the total OPE concentrations, respectively, whereas their respective di-ester metabolites (BCIPP and BCEP) were detected in few urine samples (DFs: 28% and 20%). This phenomenon may be due to three factors. Firstly, the low clearances of TCEP (7.0% and 19%) and TCIPP (33% and 28%) by human liver microsomes (HLM) and S9 fractions, respectively, have been reported in *in vitro* studies (Van den Eede et al., 2013). Additionally, chlorinated OPEs were detected as the most accumulated OPEs in mice following chronic inhalation exposure (Chen et al., 2020). The estimated hepatic clearances of chlorinated OPEs in humans (20.6–53.6 mL/kg/day) were much lower than those of aryl-OPEs (166 mL/kg/day) and alkyl-OPEs (330 mL/kg/day) (Wang et al., 2020a). All these findings suggest that both TCIPP and TCEP may be resistant to metabolism in living organisms. Secondly, although BCIPP and BCEP were the major metabolites of TCIPP and TCEP, respectively, their fractions in all metabolites from their individual parents were calculated to be only 0.27 ± 0.12 and 0.42 ± 0.08, respectively (Wang et al., 2020a). Some previous studies reported that BCIPHIPP, another main metabolite of TCIPP (Van den Eede et al., 2013), is more highly correlated with TCIPP in handwipes or wristbands than BCIPP (Hammel et al., 2020; Hammel et al., 2016), suggesting that BCIPHIPP, rather than BCIPP, is a better biomarker for exposure to TCIPP. However, in our study, BCIPHIPP was not monitored due to the absence of reference standard. Additionally, the relatively high detection limit of BCEP (0.625 ng/mL) may also be responsible for the low DF of BCEP in urine, although its median levels in urine from other studies in China were usually>0.625 ng/mL (median range: 0.520–2.57 ng/mL, Table S16). Thirdly, the decreased metabolic and/or excretory capacities of elderly people may be another important reason, which is consistent with the significant increase of TCIPP in whole blood with age in our study. It has been reported that human hepatic blood flow, activity of hepatic microsomal enzymes, and renal function would decrease with age (Eldesoky, 2007). Previous studies conducted in Australia (Van den Eede et al., 2015), USA (Hoffman et al., 2015), Norway (Xu et al., 2019a), and China (Lu et al., 2017; Sun et al., 2018) have found that urinary concentrations of several OPE metabolites, including BCIPP, BCIPHIPP, BCEP, BDCIPP, DPHP, and DnBP, exhibited significant nagative correlations with participants'age, with ranges of 0–75, 19–67, 20–65, 0.4–87, and 25–90 years, respectively. Therefore, the age related metabolic characteristic of OPEs should be emphasized, which means that some OPEs may have greater levels and longer presence in whole blood in elderly individuals, posing greater health risks to them compared to younger adults. Similar situations were observed for TPHP and EHDPP. Both of them were the dominant OPEs detected in dust and whole blood samples, while their hydroxylated metabolites, 4-OH-DPHP and 5-OH-EHDPP, were found to have rather small DFs (<21%) in urine. Additionally, urinary DPHP levels (0.0890 ng/mL) were lower compared with those in children and adults in most other Chinese studies (median range: 0.066–0.42 ng/mL), although several aryl-OPEs, including TPHP, EHDPP, CDPP, RDP, and BABP (Ballesteros-Gomez et al., 2015a; Ballesteros-Gomez et al., 2015b; Van den Eede et al., 2013) that could be metabolized to DPHP were frequently detected in air and dust samples, and DPHP was the third most abundant di-OPE in indoor dust. In addition to the reduced metabolic or excretory capacities of the elderly, the low formations of DPHP from TPHP (22%) and EHDPP (4%) in HLM may explain the low urinary levels of DPHP (Ballesteros-Gomez et al., 2015a). In contrast with the four OPEs discussed above, TDCIPP had low proportions in air and dust samples and was detected in 34% of whole blood samples, while its metabolite, BDCIPP, was detected in 76% of urine samples and was the second most abundant di-OPE in urine. This is consistent with the great clearances of TDCIPP (46%, 68%, >70%, and > 95% for HLM, S9 fractions, polar bear microsomes, and rat liver microsomes, respectively) in *in vitro* studies (Chu et al., 2011; Strobel et al., 2018; Van den Eede et al., 2013). However, Wang et al., (2020a) have estimated that TDCIPP has a lower hepatic clearance in humans (20.6 mL/kg/day) compared to TCEP (35.3 mL/kg/day) and TCIPP (53.6 mL/kg/day) due to its high plasma protein binding affinity. Therefore, we speculated that the high detection and levels of BDCIPP in urine may be due to the higher yield of BDCIPP from its parent (TDCIPP) (0.69 ± 0.13) compared to BCEP (0.42 ± 0.07) and BCIPP (0.27 ± 0.12) (Wang et al., 2020a). Additionally, as reported by He et al. (2018c), direct intake of BDCIPP from food may be also an important source of BDCIPP in urine. For TEP, a low proportion was found in whole blood (9.1%) although it was the most abundant OPE in air samples. This may be because it is easily eliminated *via* urine due to its great hydrophilicity or it is rapidly metabolized in the body as DEP was detected as the most abundant di-OPE in urine from adults in New York of USA (Wang et al., 2019). Further studies are needed to investigate the metabolic characteristics of different OPEs in humans, especially the possible perturbations in pharmacokinetics of OPEs in elderly people due to their organ function changes.

3.4. Relationships between whole blood/urine with air and dust

Average of OPE/di-OPE levels in whole blood/urine collected from the fourth and fifth months were used to examine their correlations with OPEs in indoor air and dust because that the environmental samples were only collected during the fourth and fifth sampling campaigns, as described in Section 2.3 (Table S19). Concentrations of TEHP in indoor dust were positively correlated with those in whole blood (n = 55, r_s = 0.306, p = 0.023). In addition, a significant positive correlation was observed for TnBP between indoor air and whole blood (r_s = 0.392, p = 0.003). These relationships were further evaluated by use of linear regression analyses adjusting for age, sex, and BMI of participants. Clear positive relationships were observed between TEHP or TnBP in whole blood and their tertile categories in indoor dust and air, respectively (Fig. 2). Such modest correlations could be due to that other sources (e.g., diet) also contributed to the blood concentrations or differences in metabolisms between individuals. But the observations still suggest that indoor air and dust may be a primary source of the participants' exposure to TnBP and TEHP, respectively. This could be further supported by the exposure assessment results in Section 3.6, showing that inhalation accounted for 79.6% of total human exposure to TnBP and dust ingestion accounted for 50.9% of total TEHP intake. However, these correlations were not observed for BEHP or DBP in urine. This might result from the direct human exposure to these two di-OPEs because they have been reported as plasticizers and used for metal extraction (He et al., 2018b) and have been observed as the main di-OPEs in home dust in our study (Table S21). In this study, the estimated daily intakes (EDIs) of BEHP and DBP *via* dust contributed to 1.09–107% (median: 12.5%) and 0.230–37.5% (2.30%) of their urinary levels, respectively (Table S33), which were described in more detail below. Additionally, BEHP and DBP have also been frequently detected in food products in Queensland, Australia (He et al., 2018c). These findings indicated that whole blood may be a better monitor for TnBP and TEHP exposure in elderly people than urine. For other OPEs, no correlations were found between indoor air or dust and bio-samples. Similar results have been reported in several previous studies (Carignan et al., 2013; Castorina et al., 2017; Dodson et al., 2014; Hammel et al., 2020; Hoffman et al., 2015; Phillips et al., 2018; Tao et al., 2018). However, TDCIPP in floor dust has been found to be significantly associated with BDCIPP in urine of women in Canada (Yang et al., 2019), adults in USA (Meeker et al., 2013), adults and children in Oslo, Norway (Cequier et al., 2015; Xu et al., 2019a). In addition, significant correlations have also been found between TPHP in floor dust and DPHP in urine of children and adults in Oslo, Norway (Cequier et al., 2015; Xu et al., 2019a). One reason for the absence of

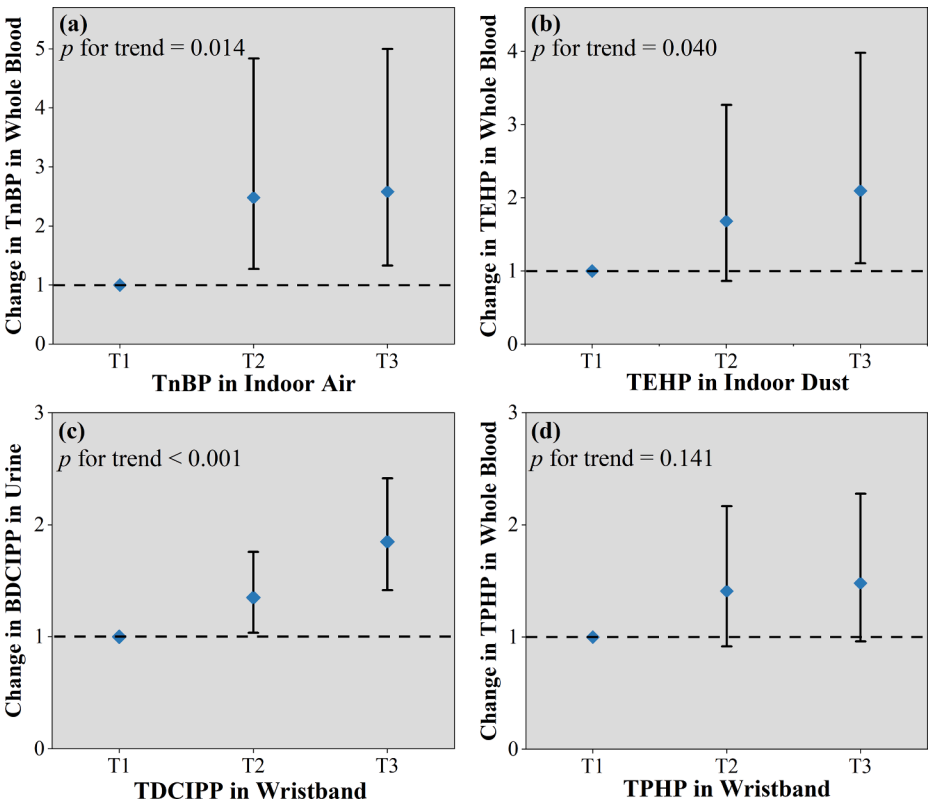

Fig. 2. Relationships (a) between TnBP in whole blood and indoor air, (b) between TEHP in whole blood and indoor settled dust, (c) between BDCIPP in urine and TDCIPP in wristband, and (d) between TPHP in whole blood and wristband, adjusting for the elderly's sex, age, and BMI. Tertile 1 (T1), the first tertile; T2, the second tertile; T3, the third tertile. *P*-values for trend were derived using a continuous variable with the median value of each tertile. Average of TnBP/TEHP levels in whole blood collected during the fourth and fifth months were used for regression analyses because air and dust samples were collected during this period. The DFs of TnBP and TEHP in whole blood were 84% and 64%, respectively. The measurement data from seven couples were removed from the regression analyses for TnBP and TEHP (n = 55) to ensure the environmental measurements were independent.

these relationships in our study might be due to much lower concentrations of TDCIPP (median: 126 ng/g) and TPHP (192 ng/g) in indoor dust compared to levels in Oslo, Norway (median range of TDCIPP and TPHP: 397–1130 and 722–1230 ng/g, respectively) (Cequier et al., 2015; Xu et al., 2019a), USA (TDCIPP: 1620 ng/g) (Meeker et al., 2013), and Canada (TDCIPP: 1380 ng/g) (Yang et al., 2019). In addition, other factors, such as other sources, interindividual variations in absorption, metabolism, and activity pattern, or existence of di-OPEs in environmental matrices, may also affect these correlations. For examples, hand wipes and wristband samples have been reported to have better correlations with OPE metabolites in urine (e.g., BDCIPP, DPHP, BCIPP, BCIPHIPP) of children and adults in USA than dust or air samples since they integrated exposures from multiple sources and accounted for individual's activity pattern (Hammel et al., 2020; Hoffman et al., 2015; Phillips et al., 2018; Tao et al., 2018). As another part of the China BAPE study, wristband samples (n = 293) were also collected from the participants to assess their personal exposures to 76 airborne chemicals, including 3 OPEs (TCIPP, TDCIPP, and TPHP), during 5 months (Guo et al., 2021; Koelmel et al., 2020). The detailed detection and concentrations of these three OPEs in wristband samples were described in another paper (Guo et al., 2021). Therefore, in our study, correlation analyses were conducted between OPEs/di-OPEs in whole blood/urine and OPEs in wristband. The results showed that TDCIPP in wristband was significantly correlated with BDCIPP in urine of the elderly (n = 292, $r_s = 0.301$, $p < 0.001$), while a weak positive correlation was found for TPHP between wristband and whole blood ($r_s = 0.111$, $p < 0.057$). Further regression analyses revealed a significant dose–response relationship for TDCIPP, but not for TPHP (Fig. 2). The results suggest that wristband is a better indicator of TDCIPP exposure in elderly people than indoor air and dust.

3.5. Relationships between whole blood/urine with food consumption

Firstly, we investigated whether the water, tea, and additional diets consumed by the participants in several days before bio-samples collection affected the analyte concentrations in bio-samples by spearman correlation analyses (Tables S29 and S30). The results showed that the amount of water elderly people drank per day in the three days before bio-samples collection was positively correlated with TEP concentrations in whole blood. Participants who ingested other diet besides those provided by us in the three days before bio-samples collection had significantly higher blood levels of TEP and urinary levels of DPHP and DBP (Fig. 3). We recorded that the participants' supplementary diets consisted mainly of yogurt, milk, fruits, melon seeds, walnut, and peanuts. These findings are in line with previous studies in China reporting that TEP was the dominant OPE detected in tap water, filtered drinking water (Ding et al., 2015), dairy products, fruits, nuts, and vegetables (Zhao et al., 2019b). TPHP and TnBP have also been frequently detected in dairy products, fruits, walnut, and melon seeds in China (Zhao et al., 2019b). Moreover, DPHP and DBP have been detected as the dominant di-OPEs in various food categories in Australia (e.g., fruits, dairy products, and vegetables) (He et al., 2018c) and in tap water in Anhui, China (Li et al., 2020a). In addition, the higher levels of DPHP and DBP in urine from elderly people who consumed other diets in several days before bio-samples collection indicated that these two di-OPEs or their parents in bio-samples tended to reflect the participants' exposure in recent days, which are consistent with the short half-lives of TPHP (9.68 days) and TBP (4.76 days) in humans estimated by Wang et al. (2020a). Additionally, we explored the associations between OPE/di-OPE levels in whole blood/urine with the monthly consumption frequencies of 24 different foods (e.g., water, rice, meat, bean products, and fruits) of participants. Several correlations were observed (Tables S29 and S30). Consumption of red meat, nuts, and tea presented positive correlations with DPHP in urine. Bean products intake was associated with higher blood levels of TCEP and higher urinary levels of BMPP. Consumption of sodas was related to higher levels of BEHP in urine.

Further, food items correlated with analyte concentrations in blood or urine in the bivariable analysis ($p < 0.200$) were included in multivariable linear regression models of log transformed OPE/di-OPE

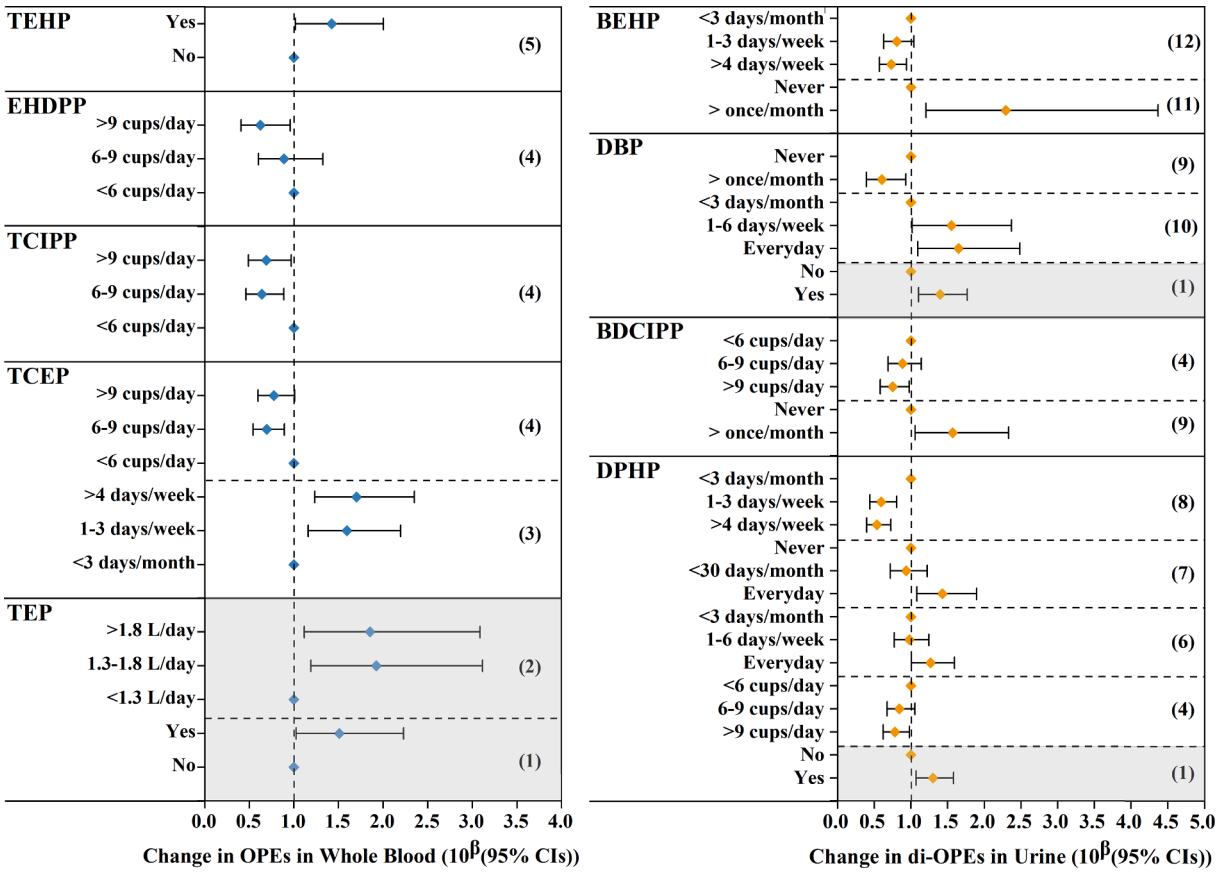

Fig. 3. Multivariate linear regression analyses of different food intake frequencies with blood levels of OPEs or urinary levels of di-OPEs, including (1) other diet intake besides food provided by us in the three days before bio-sample collection; (2) daily water intake in the three days before bio-sample collection; (3) bean product intake; (4) daily water intake within a month; (5) alcohol drinking within a month; (6) dairy product intake; (7) tea intake; (8) rice intake; (9) fresh fruit and vegetable juice intake; (10) other staple food intake; (11) soda intake; and (12) nut intake. All models were adjusted for sex, age, and BMI of the participants. The areas with gray background represent information on diets that elderly people consumed in several days before bio-samples collection, while the other areas are the monthly consumption frequencies of different food.

concentrations (Table S31, Fig. 3). For TEP and TCEP in blood, the associations with water, additional diet or bean products from the bivariate analyses remained in the multivariate models. For di-OPEs in urine, DPHP was no longer significantly correlated with the consumption of red meat and nuts, while was positively correlated with dairy products intake in the model. Several significant correlations, such as BDCIPP with fresh fruit and vegetable juice, DBP with other staple food (e.g., corn and millet) and additional diets, were observed in the models although not in the spearman rank analyses. The positive association between BEHP and sodas remained, while that between BMPP and bean products not. These observed relationships suggest that diet may be an important source of elderly people exposure to several OPEs (e.g., TEP, TCEP, and TPHP) or a direct source of human intake of di-OPEs (e.g., DPHP). He et al. (2018) has reported that DPHP in urine of Australian adults may be due to direct intake from food. Only Cequier et al. (2015) have investigated the associations between BDCIPP and DPHP in urine of mother and child and the amount of different foods ingested during 24 h, showing a weak correlation between DPHP concentrations and vegetables consumed. The different findings indicated that the primary pathways of human exposure to OPEs may be differed between compounds, regions, and populations and depend on individual exposure factors such as behavioral patterns, dietary habits, and the concentrations of OPEs in food, dust, and air they encountered. Additionally, several negative associations were found in both bivariable analyses and the multivariate models (Tables S29, S30, and S31). For example, high water intake was associated with a decrease in blood EHDPP, TCEP and TCIPP levels and urinary DPHP and BDCIPP levels. We recorded that the higher water consumption participants drank mostly tap water, barreled water or tap water through a purifier. The positive correlation between water consumption and TEP concentrations in blood was consistent with the high abundance of TEP in tap water and filtered drinking water in eastern China (Ding et al., 2015), suggesting that water ingestion is an important exposure pathway for TEP. However, the opposite situations found for these OPEs/di-OPEs indicate that drinking water may play a greater role in promoting the metabolism or excretion of these chemicals than does the intake of them.

3.6. *Exposure assessment*

We used di-OPE levels in dust to estimate EDIs to di-OPEs *via* dust ingestion for the elderly and to evaluate its potential contributions to urinary di-OPE levels, assuming 100% excretion rates of di-OPEs (Table S33). The highest EDI was found for BEHP, with a median value of 0.306 ng/kg bw/day, which was six times greater than that of its parent (TEHP). Results showed that 1.09–107% of urinary BEHP (median: 12.5%) may be due to direct intake from dust. For other di-OPEs, the median EDIs *via* dust ingestion ranged from 0.001 to 0.0470 ng/kg bw/day with relatively small contributions to their urinary levels (median range: 0.120–2.42%). Considering that there are other exposure pathways of di-OPEs (e.g., diet) (He et al., 2018c), the interference of di-OPEs in environmental matrices to OPE exposure estimates should be concerned when using urinary di-OPEs as exposure biomarkers.

In addition, based on the OPE concentrations in air and dust and the personal information of the participants (e.g., gender, body weight, and times spending at home and outdoor), we estimated the daily intakes of OPEs *via* inhalation, dust ingestion, and dermal absorption from dust (Table S32 and Fig. S3). The EDIs of $\sum_{16}$OPEs *via* the three pathways were 0.366–22.9, 1.00–25.5, and 0.444–11.4 ng/kg bw/day for the elderly, respectively. Inhalation was the predominant pathway for TMP, TEP, TnBP, TiBP, and RDP, with the contributions of 94%, 94%, 80%, 82%, and 99% of total intakes, respectively, while dust ingestion was the dominant pathway for the other ten OPEs (51–75%). Additionally, TCIPP had similar intakes from inhalation (42%) and dust ingestion (39%). Zhao et al. (2019b) have estimated the daily dietary intakes of 9 OPEs in Chinese adults (mean values: from 0.200 ng/kg bw/day for TMPP to 14.3 ng/kg bw/day for TCEP), which were one order of magnitude higher than the total intakes of individual OPE *via* the three pathways in our study (median values: from 0.0310 ng/kg bw/day for TMPP to 1.71 ng/kg bw/day for TEP). The high dietary intakes of OPEs reported in previous study (Zhao et al., 2019b), together with the associations between food intake frequencies and analyte levels in bio-samples found in our study, suggest that diet might be an important source of human exposure to some OPEs. Therefore, further studies are needed to focus on the presence of OPEs and di-OPEs in food samples and to elucidate the dietary exposure to OPEs among different populations.

Furthermore, human daily intakes of several OPEs were also derived from OPE concentrations in whole blood (EDI_{blood}) and corresponding di-OPE levels in urine (EDI_{urine}), respectively (Table S32). The EDIs of individual OPE calculated from these two methods were far higher than those *via* air and dust, which was probably due to the existence of other exposure sources (e.g., diet or direct contact with emission sources) (Yang et al., 2020; Zhao et al., 2019b). Additionally, some of the di-OPEs in urine may derived from direct intake rather than OPE metabolism. Moreover, the OPEs detected in whole blood were cumulative concentrations of prolonged exposure. Both these facts might lead to an overestimation of daily OPE exposure. In addition, uncertainties might be introduced in the estimations due to the assumptions of two key parameters, f (the molar fraction of urine-excreted metabolite with respect to its parent) and CL_h (*in vivo* hepatic clearance rate of OPEs) (Equations S6 and S7). The f values were assumed to have a range of 0.1 to 0.9 based on the known urinary excretion fractions of DBP (0.18) and BDCIPP (0.63) in *in vivo* rat studies (Lynn et al., 1981; Suzuki et al., 1984; Wang et al., 2019). The CL_h values of TPHP, TCEP, TnBP, and TCIPP were estimated from *in vitro* incubation system in a previous study (Wang et al., 2020a). Additionally, the CL_h values of EHDPP, TiBP, and TEHP were assumed to be the same as those of TPHP, TnBP, and TBOEP, respectively, due to their similar structures and metabolic characteristics (Ballesteros-Gomez et al., 2015a; Hou et al., 2020b). Further relevant studies are needed for accurate exposure assessments based on analyte levels in bio-samples. Overall, whether based on the OPE concentrations in air and dust, OPE levels in whole blood, or the di-OPE levels in urine samples, the estimated daily exposures of each OPE were far less than their respective RfD values (<1–5 orders of magnitude) (Table S32), implying a minimus risk of OPEs to health of elderly people.

In summary, this is the first study to focus on longitudinal OPE exposures in the healthy elderly populations and to comprehensively explore their body burdens and exposure pathways by questionnaire and collecting multiple paired samples (whole blood, urine, air, and dust). There are several limitations of the present study. First, as study participants were all elderly people and their physiological features and lifestyles are different from other populations, our findings may not be generalized to the overall population. Second, only information on food intake frequencies, not duplicated diet samples, were collected, which limited the accurate assessments of participants' dietary exposure to OPEs or di-OPEs. Moreover, the participants were provided unified diets for five days before human bio-samples collection, which may somewhat introduce the participants' exposure to some OPEs. Bio-samples were collected from the participants only once a month, thus, these facts may weaken the correlations analyses between analyte levels in human bio-samples and food intake frequencies, leading to a potential bias. Third, di-OPEs were not measured in air and whole blood samples due to the limited amount of samples, which would otherwise be more indicative of elderly people's ability to metabolize OPEs and better reflect elderly individuals' exposure to di-OPEs *via* inhalation. Fourth, the sampling rates of passive air sampler were not validated using an active sampler, which may introduce uncertainties of compound concentrations in air but would not affect our main conclusions.

CRediT authorship contribution statement

Minmin Hou: Conceptualization, Data curation, Investigation, Methodology, Formal analysis, Validation, Visualization, Writing - original draft. **Jianlong Fang:** Investigation, Data curation, Project administration. **Yali Shi:** Conceptualization, Data curation, Funding acquisition, Investigation, Methodology, Project administration, Resources, Writing - review & editing. **Song Tang:** Conceptualization, Data curation, Funding acquisition, Investigation, Methodology, Project administration, Resources, Writing - review & editing. **Haoran Dong:** Investigation, Data curation. **Yuanyuan Liu:** Investigation, Data curation. **Fuchang Deng:** Investigation, Data curation. **John P. Giesy:** Supervision, Visualization, Writing - review & editing. **Krystal J. Godri Pollitt:** Supervision, Visualization, Writing - review & editing. **Yaqi Cai:** Conceptualization, Data curation, Funding acquisition, Investigation, Methodology, Project administration, Resources, Writing - review & editing. **Xiaoming Shi:** Conceptualization, Data curation, Funding acquisition, Investigation, Methodology, Project administration, Resources, Writing - review & editing.

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 material

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2021.106803.

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Supporting Information

Exposure to Organophosphate Esters in Elderly People: Relationships of OPE Body Burdens with Indoor Air and Dust concentrations and Food Consumption

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This Supporting Information contains 48 pages, 33 tables, and 4 figures.

Contents

Materials and Methods

Materials

TMP, TEP, TPrP, TnBP, TiBP, TEHP, TBOEP, TCEP, TCIPP, TDCIPP, TPHP, TMPP, EHDPP, and CDPP standards were purchased from Dr. Ehrenstorfer GmbH (Augsburg, Germany). TMP-d9, TEP-d15 and TPrP-d21 were purchased from C/D/N Isotopes Inc. (Quebec, Canada). TnBP-d27 and TPHP-d15 were purchased from Cambridge Isotope Laboratories (Andover, MA, USA). RDP, BABP, TCIPP-d18, DnBP, DiBP, BEHP, BBOEP, BCEP, BCIPP, BDCIPP, DPHP, DoCP, DpCP, DnBP-d27, BEHP-d34, BBOEP-d8, BCEP-d8, BCIPP-d12, BDCIPP-d10, DPHP-d10, DoCP-d14, and DpCP-d14 were purchased from Toronto Research Chemicals Inc. (Toronto, Canada). BBOEHEP, 4-OH-DPHP, 5-OH-EHDPP were synthesized. High-performance liquid chromatography (HPLC)-grade methanol (Meth), dichloromethane (DCM), acetonitrile (ACE), and n-hexane (HEX) were purchased from Fisher Chemical (USA) and Merck (Darmstadt, Germany). β-glucuronidase/aryl sulfatase enzyme (2 mL) was purchased from Merck (Darmstadt, Germany). Sodium acetate was purchased from Sigma-Aldrich (St. Louis, MO, USA). Triethyl amine was purchased from Alfa Aesar (Thermol Fisher Scientific, USA).

Synthesis of 5-OH-EHDPP

Triethylamine (1 mL) was added to 1 mmol of 2-ethylhexane-1,5-diol in tetrahydrofuran (10 mL) for synthesis of 5-OH-EHDPP. 5 mL of diphenyl phosphorochloridate (1.2 mmol) was then added dropwise to the mixed solution. The reaction solution was stirred for 3 h at 0 °C and then ice water was added. The mixture was extracted with 40 mL of tetrahydrofuran for 3 times. The organic layers were combined, dried over anhydrous sodium sulfate, and evaporated to dryness. The extract was loaded on a silica gel column, eluted with methanol/dichloromethane (v/v) 1:20 and concentrated. The products with the purity of > 99% were obtained.

Sample Treatment

Air samples (SIPs) were spiked with 10 ng internal standards mixture (TMP-d9, TEP-d15, TPrP-d21, TnBP-d27, TPHP-d15, TCIPP-d18, and TCEP-d12), and then extracted twice with hexane/dichloromethane (1:1, v/v) using accelerated solvent extraction (Dionex, ASE 350). The extract temperature and static time were set at 100 °C and 5 min, respectively. Extraction of OPEs and di-OPEs from dust followed the same procedures as described in Tan et al..[1] Briefly, approximately 200 mg of sieved dust was transferred to a 10 mL glass tube, spiked with 10 ng internal standards and extracted with 5 mL of a mixture of hexane and dichloromethane (HEX: DCM, 1:1, v/v) under sonication. The extraction was repeated three cycles (30 min each) and the supernatants were combined. Extraction of OPEs from whole blood followed the same methods as described in a previous study.[3] Briefly, 0.4 mL whole blood was transferred into a 10 mL glass centrifuge tube and spiked with internal standards. After mixing and aging for 30 min, samples were extracted three times with acetonitrile (in the order of 10, 2, and 2 mL), and the organic layers were combined. All the extract was concentrated to near dryness under a gentle stream of nitrogen and then diluted to approximately 30 mL with ultrapure water.

The final extract for OPEs was cleaned through an ENVI-18 cartridge (6 mL, 500 mg; Supelco), which was preconditioned with 5 mL of acetonitrile and 5 mL of ultrapure water sequentially. After the sample was loaded, the cartridge was washed with 10 mL of water, dried for 30 min, and finally eluted with 6 mL of 25% DCM in acetonitrile. The eluent was concentrated to near dryness under a gentle nitrogen stream, reconstituted with 1 mL of methanol and filtered through a 0.22 μm nylon filter for instrumental analysis.

The final extract for di-OPEs was cleaned through an HLB cartridge (6 mL, 200 mg; Waters)

conditioned with 5 mL of methanol and 5 mL of ultrapure water sequentially. After the samples were loaded, the cartridges were rinsed with 10 mL of ultrapure water, dried for 30 min, and finally eluted with 6 mL of methanol. The eluent was concentrated to 1 mL of methanol and filtered through a 0.22 μm nylon filter for instrumental analysis.

The analysis of OPEs and their metabolites in urine samples followed the same procedures as described in He et al..[4] Briefly, 2 mL urine was spiked with mixed internal standards (10 ng each), buffered with 0.7 mL sodium acetate (pH = 5, 1 M), and digested overnight with 100μL of enzyme solution (1000 units per mL, β-glucuronidase/aryl sulfatase enzyme) at 37 °C. Samples were then extracted using STRATA-X-AW cartridge (3 cm^3, 60 mg; Phenomenex Inc., Torrance, CA, U.S.) conditioned with 2 mL acetonitrile and 2 mL water. After loading the sample, cartridges were rinsed with 2 mL water and the target chemicals eluted with 2 mL of 5% triethyl amine in acetonitrile. The eluent was concentrated to near dryness under a gentle nitrogen stream, reconstituted with 1 mL of methanol and filtered through a 0.22 μm nylon filter for instrumental analysis.

Instrumental Analysis

The separation of OPEs was achieved using an Acquity UPLC BEH C18 column (2.1 mm × 100 mm, 1.7 μm particle size, Waters, USA) preceded by an Acquity UPLC BEH C18 guard column (2.1 mm × 5 mm). 5 mM ammonium acetate in water (A) and methanol (B) were used as the mobile phases, the column oven was 25°C, the injection volume was 5μL, and the flow rate was 400 μL/min. The gradient was set as follows: the initial 10% B and increased linearly to 40% in 1 min; followed by an increase to 90% B in 3 min; then increased to 100% B in 0.1 min and held for 4.9 min. Finally, the gradient was returned to the initial conditions of 10% B in 0.1 min and held for 3.9 min. The MS was operated in positive ion multiple reaction-monitoring mode (MRM).

The separation of di-OPEs was achieved using an Acquity UPLC BEH C18 column (2.1 mm × 100 mm, 1.7 μm particle size, Waters, USA). 5 mM ammonium acetate in water (A) and methanol (B) were used as the mobile phases, the column oven was 25°C, the injection volume was 5μL, and the flow rate was 400 μL/min. The gradient was set as follows: the initial 20% B and hold for 0.5 min, increased to 40% in 0.5 min; followed by an increase to 90% in 3 min, hold for 3.5 min; then decreased to 20% B in 0.1 min and hold for 4.4 min to allow for equilibration. The MS was operated in negative ion multiple reaction-monitoring mode (MRM). DoCP and DpCP, DnBP and DiBP could not be completely separated from each other. Therefore, they were referred to as BMPP and DBP, respectively.

Quality Assurance and Quality Control

The field blanks consist of pre-cleaned SIPs and anhydrous sodium sulfate for air (n = 2) and dust samples (n = 2), respectively. The field blanks for whole blood (n = 20) and urine (n = 20) were prepared by transferring ultrapure water of the same volume as the samples into sampling containers, followed by storing and passing through the entire analytical procedure. A procedural blank was analyzed in each batch of 23 samples. The final concentrations of target analytes were blank-corrected (Tables S4 and S5). Specifically, the concentrations of most analytes in field blanks were consistent with those in procedural blanks except TCIPP that showed higher concentrations in field blanks for air. Therefore, the concentrations of TCIPP in air samples were calculated by subtracting the average field blanks while those of other analytes were obtained by subtracting the average blank concentrations (including field blanks and procedural blanks). Standard reference material-SRM2585 and SRM3673 (NIST, U.S.) were used as quality control for analysis of OPEs in dust and di-OPEs in urine, respectively (n = 6, Tables S6 and S7). The results were comparable with the values from previous studies (within 15% of the assigned or indicative values). SIP, dust, whole blood, and urine samples

were spiked with standards (n = 3), obtaining 74-108%, 63-125%, 57-114%, and 35-112% recoveries for OPEs, respectively (Table S4). The recoveries of di-OPEs in dust and urine samples ranged from 73-116% and 85-108%, respectively (Table S5). Recoveries of the internal standards in all samples ranged from 32% to 92.7% for OPEs and 48.5-135% for di-OPEs. The method detection limits (MDLs) were calculated as the mean blank + 3 times the standard deviation of the blanks normalized to the sample volume for air and dust samples, and as the 3 times the standard deviation of the blanks normalized to the sample volume for whole blood and urine samples. For target compounds not present in the blanks, MDLs were calculated according to a signal/noise ratio of 3. The MDLs of OPEs were 0.0044-0.220 ng/mL, 0.0026-0.221 ng/mL, 0.0005-0.442 ng/m^3, and 0.04-11.5 ng/g for whole blood, urine, home air, and dust samples, respectively (Table S4). The MDLs of m-OPEs were 0.0038-0.625 ng/mL and 0.044-14.7 ng/g for urine and dust samples, respectively (Table S5).

Dust and Passive Air Sampling

Indoor dust was collected using a vacuum cleaner made for domestic applications (Midea C3-L143C, China) with separate nylon socks inserted in the suction nozzle. The nylon socks were purchased online, made by ourself and were cleaned with water and then soaked in methanol for 2 times before use. Respective sampling socks were used for each sample. Dust samples were collected by the investigator from floor and the elevated surface in living room of the participants.

Before sampling, PUF disks (14 cm diameter × 1.35 cm thick, surface area 365 cm^2, mass 4.40 g, volume 207 cm^3; Tisch Environmental, Cleves, OH, U.S.A.) were pre-washed with hexane/dichloromethane (1/1, v/v), by use of accelerated solvent extraction (ASE 350, Dionex Corporation, California, U.S.A.), then impregnated with finely ground XAD-4 resin (Supelco, Bellefonte, PA, U.S.A.), by use of a previously reported method.[15] Rates of sampling (m^3/day) for OPEs were a uniform 0.82 m^3/day indoors and 3.5 m^3/day outdoors, respectively.

To our knowledge, only two studies have evaluated the OPE-specific PAS sampling rates in ambient air using the air sampler (double-bowl fitted with a SIP disk) deployed in this study. Liu et al.[16] calculated the specific sampling rates in SIP-PAS for six OPEs (TCEP, TCIPP, TDCIPP, TPHP, CDPP, and TMPP), while Abdollahi et al.[17] obtained the sampling rates for 3 OPEs congeners (TCIPP, TDCIPP, and TPHP). Due to the lack of sampling rate for each target compound, in this study, the sampling rates for all OPEs in outdoor air were assumed to be the average sampling rate (3.5 m^3/d) obtained in the study of Liu et al..[16]

For indoor environment, Vykoukalová et al.[18] evaluated the sampling rates for 6 OPEs using double-bowl shaped passive sampler containing a polyurethane foam disk (PUF-PAS) and obtained an average sampling rate for OPEs in indoor air (0.82 m^3/d). Considering that the average sampling rate of PUF and SIP sampler for OPEs was comparable in the study of Liu et al.,[16] 0.82 m^3/d was used as the sampling rate for all OPEs in the indoor environment in this study. Given the difficulty of calculating accurate and meaningful sampling rates, this approach seems reasonable.

Methods for Measuring Specific Gravity and Creatinine and for Correcting Urinary Levels

The urinary creatinine and specific gravity were measured at community hospitals. Creatinine of urine was measured by the picric acid spectrophotometry using a spectrophotometer (Xinyue-T6, Persee Analytics, Beijing, China). SG was measured using a urine analyzer (URIT-180, Guilin, China).

1. For SG adjustment, the following formula was used:

$$U_{SG}=U\times\frac{SG_m-1}{SG-1}$$

where U_{SG} is the SG-adjusted concentrations of analytes (ng/mL), U is the measured urinary concentrations of analytes (ng/mL), and SG_m is the median SG level in the study population.

2. For Cr adjustment, the following formula was used:

$$U_{Cr} = \frac{U}{Cr} \times 1000$$

where U_{Cr} is the Cr-adjusted concentrations of analytes (ng/g), U is the measured urinary concentrations of analytes (ng/mL), and Cr is the creatinine level in the study population (g/L).

Exposure Assessment

1. The estimated daily intakes of OPEs via inhalation, dust ingestion, and dermal absorption based on OPE concentrations in home and outdoor air and dust ($EDI_{external}$) were calculated using the following equation; The calculation methods and parameters were referred from literatures.[5,6]

$$(1)\ EDI_{external} = EDI_{inh} + EDI_{ing} + EDI_{der}$$

$$(2)\ EDI_{inh} = \frac{C_{in\text{-}a} \times IR \times t_{in} + C_{out\text{-}a} \times IR \times t_{out}}{BW}$$

Where EDI_{inh} is the estimated daily intakes of OPEs via inhalation (ng/kg bw/day); $C_{in\text{-}a}$ is the OPE concentrations in home air (ng/m^3); $C_{out\text{-}a}$ is the OPE concentrations in outdoor air (ng/m^3); IR is the mean daily inhalation rate for 60-70 age group (m^3/d) at 14.26 m^3/d for male and 11.21 m^3/d for female;[7] t_{in} is the time participant spent in home per day (h/day), and t_{out} is the time the participant spent outdoor per day (h/day). The information was collected from the participants.

$$(3)\ EDI_{ing} = \frac{C_{in\text{-}d} \times DI \times t_{in} + C_{out\text{-}d} \times DI \times t_{out}}{BW}$$

Where EDI_{ing} is the estimated daily intakes of OPEs via dust ingestion (ng/kg bw/day); $C_{in\text{-}d}$ is the OPE concentrations in indoor dust (ng/g); $C_{out\text{-}d}$ is the OPE concentrations in outdoor dust (ng/g); DI is daily dust intake (mg/day) at 100 mg/day take into account the lifestyle and behavior patterns of the elderly in this study.

$$(4)\ EDI_{der} = \frac{(C_{in\text{-}d} \times t_{in} + C_{out\text{-}d} \times t_{out}) \times ESA \times DA \times AF}{BW}$$

Where EDI_{der} is the estimated daily intakes of OPEs via dermal absorption (ng/kg bw/day); ESA is exposed skin area (for hands only, cm^2) at 1070 cm^2 for male and 890 cm^2 for female; DA is dust adherence at 0.19 mg/cm^2 for hand;[8] AF is the absorption factor at 0.28 for TCEP, 0.25 for TCIPP, 0.13 for TDCIPP and an average value (0.22) for other OPEs.[9]

2. Estimated daily intakes of di-OPEs via ingestion of dust ingestion based on di-OPE concentrations in home and outdoor dust ($EDI_{dust\text{-}m}$) were calculated using the following equation:

$$(5)\ EDI_{dust\text{-}m} = \frac{C_{in\text{-}d\text{-}m} \times DI \times t_{in} + C_{out\text{-}d\text{-}m} \times DI \times t_{out}}{BW}$$

Where $C_{in\text{-}d\text{-}m}$ is the di-OPE concentrations in home dust (ng/g); $C_{out\text{-}d\text{-}m}$ is the di-OPE concentrations in outdoor dust (ng/g).

3. The estimated daily intakes of OPEs based on di-OPE concentrations in urine (EDI_{urine}) were calculated using the following equation:[10]

$$(6)\ EDI_{urine}\ (ng/kg\ bw/day) = \frac{C_{m\text{-}urine} \times V_{urine}}{f \times BW} \times \frac{MW_P}{MW_m}$$

Where $C_{m\text{-}urine}$ is the unadjusted urinary concentration of di-OPEs (ng/mL); V_{urine} is the daily urinary excretion (mL/day) at 1200 mL/day for female and 1400 mL/day for male;[11, 12] f is the molar fraction of the urine-excreted metabolite with respect to its parents, which is related to the excretion fraction of di-OPEs in urine and the fraction of the metabolite formed from its parent; However, the information on toxicokinetic and metabolism of OPEs in human body are

limited, thus the f value of 0.1 and 0.9 were assumed to represent low and high transformation of parent to its metabolites, respectively, in this study based on the known urinary excretion fractions of DBP (0.18) and BDCIPP (0.63) in *in vivo* rat studies.[13,14] BW is the body weight of the participants (kg); and MW_p and MW_m are the molecular weights of OPEs and the corresponding metabolites (see Table S1).

4. The estimated daily intakes of OPEs based on OPE concentrations in whole blood (EDI_{blood}) were calculated using the following equation:[15,16]

$$(7)\quad EDI_{blood} = \frac{0.693}{t_{1/2}} \times V_d \times C_s = CL_h \times C_b$$

Where $t_{1/2}$ is the half-life of serum elimination of OPEs; V_d is the volume distribution (mL/kg bw); CL_h is the in vivo hepatic clearance rate of OPEs. Wang et al. estimated the CL_h values of TPHP, TDCIPP, TCEP, TnBP, TBOEP, and TCIPP at 166, 20.6, 35.3, 330, 330, and 53.6 mL/kg bw/d, respectively.[16] The CL_h values of TiBP, TEHP, and EHDPP were set as 330, 330, and 166 mL/kg bw/d in this study. C_b is the concentrations of OPEs in whole blood (ng/mL). Due to the low DFs of TDCIPP and TBOEP in whole blood, the estimations were not conducted for these two compounds in this study.

Concentration Comparison, Correlation, and Influence Factor Analysis of OPEs in Air and Dust

Few studies have investigated the levels of TEP in home air. The TEP concentrations determined in home air of this study (8.85 ng/m^3) were at the same level of those measured in Japan (2.4 ng/m^3) and Stockholm, Sweden (7.3 ng/m^3), but higher than that in Beijing, China (0.39 ng/m^3). The TCIPP and TCEP levels in this study were lower than those detected in home air from the US, Canada, Czech Republic, Australian, Sweden, and Norway, but higher than those in Japan, Nepal, and Beijing, China (Table S23).

The observed concentrations of OPEs in home dust in this study were lower than those in other Chinese cities (Beijing, Shanghai, Guangzhou, Harbin), especially for TCIPP, TCEP, TnBP, TBOEP and TEHP (Table S26). This could be due to the investigated populations usually lived in much older houses with less decoration. Unlike our study, TBOEP was reported as the most abundant OPE in home dust in Germany, Sweden, Norway, Spain, Australian, Japan and Brazil, with median concentrations ranged from 2.64-82 μg/g, which were 2-3 orders of magnitude higher than that in our study (38.4 ng/g). This difference was likely due to the different usage pattern of OPEs in these regions. Additionally, cement or tile floor that generally not be treated with polish or wax was used in the elderly's houses in this study. TDCIPP or TPHP was detected as the dominant OPEs in home dust from Netherlands, US, and Canada, and their concentrations in these regions were also much higher than those found in our study (Table S26).

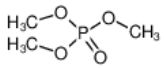
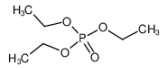
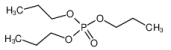
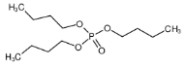
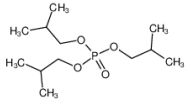
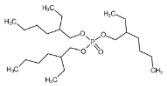
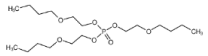
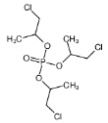
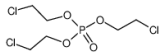
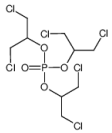
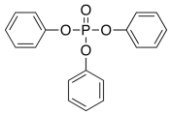
Tables S24 and S27 show results of correlation analyses between OPE concentrations in air and dust samples, respectively. Significantly positive correlations were found between TnBP and TiBP levels in both air and dust samples ($0.796 < r_s < 0.821$, $p < 0.001$), and also for TMPP, TPHP and EHDPP ($0.414 < r_s < 0.768$, $p < 0.001$). In addition, chlorinated OPEs (TCIPP, TCEP, and TDCIPP), TEP, and TPHP were moderate to highly correlated with each other in both indoor air and dust samples ($0.262 < r_s < 0.645$, $p < 0.05$), except TEP and TDCIPP in air and TCEP and TPHP in dust. Considering that many OPEs are used in a wide range of commercial products and several OPEs are added to products together, these significant correlations were not unexpected. For example, chlorinated OPEs and TEP are usually used as flame retardants in polyurethane foam in furniture, while TEP, TCEP, TPHP, TMPP, TEHP and EHDPP were added in PVC as plasticizers.[21,22] In addition, some OPEs, such as TCEP, TDCIPP, TnBP, TPHP, and TBOEP are also used in lacquer, paint, glue, or textile.[22,23] Significant correlations were

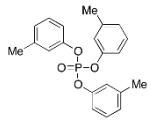
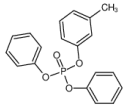
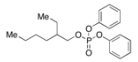
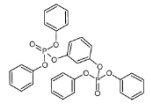
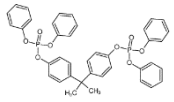
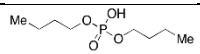
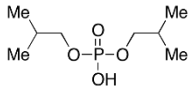
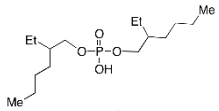
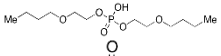
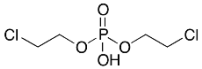
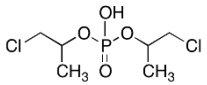
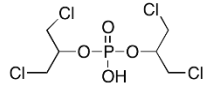
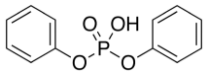
also observed between TBOEP, TPHP and TDCIPP ($0.335 < r_s < 0.458$, $p < 0.01$) in dust as well as between TEHP and EHDPP ($r_s = 0.290$, $p < 0.05$). RDP is used as a substitute for chlorinated OPEs and TPHP.[22,24] Therefore, significant correlations were found between RDP and TPHP, as well as chlorinated OPEs in home air ($0.367 < r_s < 0.459$, $p < 0.01$).

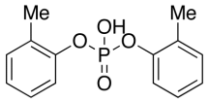
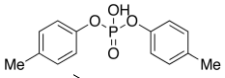
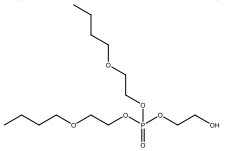
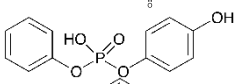
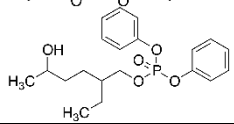
In this study, significant positive correlations were observed for $\sum_{16}$OPEs ($r_s = 0.387$, $p < 0.01$), TEP ($r_s = 0.256$, $p < 0.05$), TCIPP ($r_s = 0.392$, $p < 0.01$), TCEP ($r_s = 0.434$, $p < 0.001$), and TDCIPP ($r_s = 0.288$, $p < 0.05$) between matched air and dust samples. In addition, median values of $\log K_{dust\text{-}air}$ ($K_{dust\text{-}air} = C_{dust}/C_{air}$, where C_{dust} and C_{air} are the OPE concentrations in dust and air, respectively) for OPEs were significantly correlated with their $\log K_{oa}$ (6.63-12, Table S1) ($r_p = 0.823$, $p < 0.01$) (Figure S1), suggesting that equilibrium conditions were reached between the two phases for OPEs with $\log K_{oa} < 12$. Similar partitioning behaviors have also been reported in previous studies.[5,20]

The participants in this study answered a questionnaire containing several factors that may affect the indoor concentrations of OPEs. Spearman correlations of OPE concentrations in air and dust versus building characteristics were carried out (Tables S25 and S28). Negative correlations were obtained between TPHP and EHDPP in air with the building construction years, suggesting that the newer the houses, the higher the concentrations of these OPEs. However, these correlations were not found in home dust. Renovation of the house in the last 10 years was positively correlated with TPHP and $\sum_{16}$OPEs in dust, which may be due to more OPEs were emitted from the decoration materials. These results reflect OPEs, as alternatives for PBDEs, may have become more common recently. The size of the apartment was positively correlated with TEP in home air and TEP, TPHP, and TDCIPP in home dust. In addition, Concentrations of TEHP and TMPP in home air and CDPP in home dust decreased with the increase of distance from house to the road, which may be associated with the addition of these OPEs in lubricating oil or hydraulic oil[23] that were usually used in vehicle and mechanical equipment in outdoor environment. Significantly negative correlation was found between TMPP concentrations in home dust and floors. Some other correlations were also observed. However, given the relatively small sample size (n = 63) and some factors, such as contents of soft furnishings and number of electronic equipment, were not included in this study, these correlations may be accidental.

385 **Table S1.** Names, abbreviations, formulas, structure, and properties of the OPEs and m-OPEs standards involved in the present study

Name	Abbreviation	Formula	Molecular weight	Structure	CAS number	LogK$_{ow}$	logK$_{oa}$	Solubility (mg/L) (25^{o}C)
Trimethyl phosphate	TMP	$C_3H_9O_4P$	140.08		512-56-1	-0.65	5.88	-
Triethyl phosphate	TEP	$C_6H_{15}O_4P$	182.16		78-40-0	0.80	6.63	5.00×10^5
Tripropyl phosphate	TPrP	$C_9H_{21}O_4P$	224.23		513-08-06	1.87	6.42	827
Tri-n-butyl phosphate	TnBP	$C_{12}H_{27}O_4P$	266.31		126-73-8	4.00	7.70	280
Tri-iso-butyl phosphate	TiBP	$C_{12}H_{27}O_4P$	266.31		126-71-6	3.60	7.48	3.72
Tris(2-ethylhexyl) phosphate	TEHP	$C_{24}H_{51}O_4P$	434.63		78-42-2	9.49	11.9	0.6
Tri(2-butoxyethyl) phosphate	TBOEP	$C_{18}H_{39}O_7P$	398.47		78-51-3	3.75	11.6	1.2×10^3
Tri(1-chloro-2-propyl) phosphate	TCIPP	$C_9H_{18}Cl_3O_4P$	327.57		13674-84-5	2.59	8.5	1.6×10^3
Tri(2-chloroethyl) phosphate	TCEP	$C_6H_{12}Cl_3O_4P$	285.49		115-96-8	1.44	7.6	7×10^3
Tri(1,3-dichloro-2-propyl) phosphate	TDCIPP	$C_9H_{15}Cl_6O_4P$	430.90		13674-87-8	3.65	10.6	1.5
Tri-phenyl phosphate	TPHP	$C_{18}H_{15}O_4P$	326.28		115-86-6	4.59	10.5	1.9

Trimethylphenyl phosphate	TMPP	$C_{21}H_{21}O_4P$	368.36		563-04-2/1330-78-5	5.11	12	0.36
Cresyl diphenyl phosphate	CDPP	C19H17O4P	340.31		26444-49-5	4.51	10.9	0.24
2-Ethylhexyl di-phenyl phosphate	EHDPP	C20H27O4P	362.41		1241-94-7	6.64	11.3	1.9
Resorcinol bis(diphenyl phosphate)	RDP	$C_{30}H_{24}O_8P_2$	574.45		57583-54-7	7.41	18.33	1.1×10^{-4}
Bisphenol-A bis(diphenyl phosphate)	BABP	$C_{39}H_{34}O_8P_2$	692.63		5945-33-5	10.02	21.74	1.09×10^{-7}
Di-n-butyl phosphate	DnBP	$C_8H_{19}O_4P$	210.208		107-66-4	2.29	9.049	430.1
Di-iso-butyl phosphate	DiBP	$C_8H_{19}O_4P$	210.208		6303-30-6	2.14	8.899	574.3
Bis(2-ethylhexyl) phosphate	BEHP	$C_{16}H_{35}O_4P$	322.421		298-07-7	6.07	11.845	0.05926
Bis(2-butoxyethyl) phosphate	BBOEP	$C_{12}H_{27}O_6P$	298.313		14260-97-0	1.74	12.116	410.1
Bis(2-chloroethyl) phosphate	BCEP	$C_4H_9Cl_2O_4P$	222.992		3040-56-0	0.83	8.988	6456
Bis(1-chloro-2-propyl) phosphate	BCIPP	$C_6H_{13}Cl_2O_4P$	251.045		789440-10-4	1.67	9.581	878.8
Bis(1,3-dichloro-2-propyl) phosphate	BDCIPP	$C_6H_{11}Cl_4O_4P$	319.935		72236-72-7	2.18	10.998	130
Di-phenyl phosphate	DPHP	$C_{12}H_{11}O_4P$	250.187		838-85-7	2.88	11.243	82.38

Di-o-tolyl-phosphate	DoCP	$C_{14}H_{15}O_4P$	278.240		35787-74-7	3.97	12.245	6.652
Di-p-tolyl-phosphate	DpCP	$C_{14}H_{15}O_4P$	278.24		843-24-3	3.97	12.245	6.652
bis(2-butoxyethyl) hydroxyethyl phosphate	BBOEHEP	$C_{14}H_{31}O_7P$	342.37		1477494-86-2	0.82	13.004	4422
4-hydroxyphenyl diphenyl phosphate	4-OH-DPHP	$C_{12}H_{11}O_5P$	266.19		114527-61-6	2.4	14.743	655.2
2-ethyl-5-hydroxyhexyl diphenyl phosphate	5-OH-EHDPP	$C_{20}H_{27}O_5P$	378.4		2173149-33-0	4.76	14.192	1.161

The formulae, molecular weight, CAS number, $logK_{ow}$, and solubility (mg/L) (25 ºC) of OPEs were compiled from previous studies;[22,23] $logK_{oa}$ of OPEs were obtained from previous studies[20] and the US Environmental Protection Agency's EPI Suite™ (https://www.epa.gov/tscascreening-tools/download-epi-suitetm-estimation-program-interface-v411); $logK_{ow}$, $logK_{oa}$ and solubility of di-OPEs were obtained from supplementary information of Wang et al, 2019[10] and the EPI Suite™.

Table S2. Demographic characteristics of the study population (n = 76).

Variables	Number (percent)
Sex	
Male	38 (50)
Female	38 (50)
Age	
< 65	31 (40.8)
≥ 65	45 (59.2)
Body mass index (BMI)	
≤ 23.95	26 (34.2)
23.95-26.90	34 (44.7)
> 26.90	16 (21.1)
	Mean (SD)
Age (years)	65.1 (2.73)
Body mass index	24.8 (2.62)
Weight (kg)	65.9 (9.12)
Waist circumference (cm)	81.2 (4.95)
Average hours/day spent at home (h)	18.2 (1.86)

Table S3. Information on participants' monthly consumption frequencies of 24 different food (n = 352-353)[a]

Food		Frequency	Number	Food		Frequency	Number
Tea	1[b]	Never	138	Nut	1	<3 days/month	120
	2	<30 days/month	63		2	1-3 days/week	109
	3	Everyday	152		3	>4 days/week	124
Water-1month	1	<6 cups/day	120	Liquor	1	Never	266
	2	6-9 cups/day	122		2	>1 time/month	87
	3	>9 cups/day	111	Fresh vegetables	1	<7 days/week	6
Rice	1	<3 days/month	45		2	Everyday	347
	2	1-3 days/week	151	Pickled vegetable	1	Never	163
	3	>4 days/week	157		2	<3 days/month	64
Cooked wheaten food	1	<3 days/month	6		3	>1 day/week	126
	2	1-6 days/week	127	Fried food	1	Never	152
	3	Everyday	220		2	<3 days/month	128
Other staple food (corn, millet)	1	<3 days/month	31		3	>1 day/week	73
	2	1-6 days/week	136	Spicy food	1	Never	228
	3	Everyday	186		2	<3 days/month	52
Bean product	1	<3 days/month	49		3	>1 day/week	73
	2	1-3 days/week	156	Fresh fruit and vegetable juice	1	Never	327
	3	>4 days/week	148		2	>1 time/month	26
Red meat (Beef, Pork, Mutton)	1	<3 days/month	31	Juice	1	Never	347
	2	1-6 days/week	149		2	>1 time/month	6
	3	Everyday	173	Coffee	1	Never	333
White meat (Chicken, duck)	1	<3 days/month	198		2	>1 time/month	20
	2	1-6 days/week	152	Soda	1	Never	344
	3	Everyday	3		2	>1 time/month	9
Fish and marine product	1	<3 days/month	147	Other drink	1	Never	346
	2	1-3 days/week	184		2	>1 time/month	6
	3	>4 days/week	22	Barbecue	1	Never	345
Fruits	1	<3 days/month	15		2	>1 time/month	7
	2	1-6 days/week	49	Tea-3 days[c]	1	No	151
	3	Everyday	289		2	Yes	202
Dairy products (milk, yogurt)	1	<3 days/month	112	Water-3 days[d]	1	<1300mL/day	124
	2	1-6 day/week	112		2	1300-1800mL/day	110
	3	Everyday	129		3	>1800mL/day	119
Egg	1	<7 days/week	30	Other diet-3 days[e]	1	No	122
	2	Everyday	323		2	Yes	223

[a]The information were collected once a month for 5 months via questionnaire. [b]The number was used in correlation analyses. [c]Did the participants drink tea in the three days before bio-samples collection; [d]The amount of water participants drank each day in the three days before bio-samples collected; The average amount of water the participants drank over the three days were used. [e]Did the participants intake other diet besides food provided by us in the three days before bio-samples collection.

Table S4. Method detection limits (MDLs), blank concentrations, and matrix spike recoveries (%) of OPEs in whole blood, air, dust, and urine samples.

Analytes	Internal Standard	Whole Blood			Indoor Air			Dust			Urine		
		Blanks (ng/mL)	MDL (ng/mL)	Recovery [a] (%, RSD)	Blanks (ng/m^3)	MDL (ng/m^3)	Recovery [b] (%, RSD)	Blanks (ng/g)	MDL (ng/g)	Recovery (%, RSD) [c]	Blanks (ng/mL)	MDL (ng/mL)	Recovery [d] (%, RSD)
TMP	TMP-d9	-[e]	0.146	92 (15)	0.018	0.042	89 (4.2)	-	1.39	86 (0.7)	-	0.073	84 (4.1)
TEP	TEP-d15	0.083	0.055	97 (5.1)	0.167	0.259	93 (3.0)	2.89	5.35	97 (0.7)	0.087	0.096	99 (6.2)
TPrP	TPrP-d21	-	0.017	98 (5.3)	0.003	0.004	82 (2.7)	-	0.06	111 (4.0)	0.0169	0.022	109 (13)
TnBP	TnBP-d27	0.093	0.145	91 (13)	0.026	0.031	78 (2.3)	1.00	2.60	88 (8.9)	-	0.0062	82 (7.8)
TiBP	TnBP-d27	0.127	0.220	88 (7.6)	0.020	0.049	83 (2.1)	1.62	4.73	93 (4.8)	-	0.0091	80 (2.2)
TEHP	TPHP-d15	0.042	0.086	67 (21)	0.024	0.025	79 (2.8)	1.52	7.07	94 (5.2)	-	0.0052	71 (10)
TBOEP	TPHP-d15	0.031	0.052	85 (4.7)	0.003	0.008	93 (6.8)	1.32	2.63	115 (19)	-	0.0026	112 (6.0)
TCIPP	TCIPP-d18	0.109	0.130	91 (12)	0.237	0.442	76 (2.1)	6.25	11.5	98 (0.9)	0.049	0.0385	90 (3.1)
TCEP	TCEP-d12	0.163	0.220	104 (5.6)	0.050	0.129	82 (7.2)	1.86	3.96	102 (5.5)	-	0.0095	104 (8.3)
TDCIPP	TPHP-d15	0.035	0.214	70 (0.92)	-	0.003	74 (4.2	0.855	2.33	125 (14)	-	0.0263	88 (9.4)
TPHP	TPHP-d15	0.069	0.110	100 (1.8)	0.005	0.006	86 (0.8)	0.367	1.31	112 (5.2)	-	0.0326	104 (2.2)
TMPP	TPHP-d15	-	0.0222	57 (0.3)	0.003	0.001	85 (3.5)	0.093	0.29	82 (4.5)	0.011	0.009	72 (3.1)
CDPP	TPHP-d15	-	0.166	114 (4.2)	-	0.018	108 (2.6)	0.443	0.87	85 (7.7)	-	0.221	77 (2.1)
EHDPP	TPHP-d15	0.099	0.026	87 (5.1)	-	0.001	85 (10.4)	1.15	3.49	86 (4.7)	0.0204	0.0179	80 (2.3)
RDP	TPHP-d15	-	0.0219	88 (3.6)	-	0.0005	94 (4.1)	-	0.04	70 (2.2)	-	0.0075	35 (2.7)
BABP	TPHP-d15	-	0.0044	81 (4.6)	0.001	0.002	88 (5.1)	0.032	0.07	63 (1.8)	0.0172	0.0214	87 (5.7)

[a] Matrix Spike Recovery (%, n=3) of OPEs in whole blood (4ng/mL); [b] Matrix Spike Recovery (%, n=3) of OPEs in air (10ng/SIP); [c] Blank spike Recovery (%, n=3) of OPEs in dust (50 ng/g); [d] Matrix Spike Recovery (%, n=3) of OPEs in urine (2ng/mL); [e] The compound was not detected in blank samples.

Table S5. Method detection limits (MDLs), blank concentrations, and matrix spike recoveries (%) of m-OPEs in urine and dust samples.

Analytes	Internal standard	Urine			Dust		
		Blanks (ng/mL)	MDL (ng/mL)	Recovery [a] (%, RSD, n=3)	Blanks (ng/g)	MDL (ng/g)	Recovery (%, RSD, n=3) [c]
DBP	DnBP-d18	0.017	0.013	85 (3.5)	0.13	0.26	109 (1.1)
BBOEP	BBOEP-d8	0.021	0.032	104 (3.5)	-	0.133	91 (12)
BEHP	BEHP-d34	0.078	0.069	103 (10.4)	2.29	14.7	87 (11)
BCEP	BCEP-d8	-[b]	0.625	102 (2.9)	-	10.3	73 (10)
BCIPP	BCIPP-d12	-	0.042	96 (0.9)	-	0.415	109 (8.3)
BDCIPP	BDCIPP-d10	0.034	0.048	95 (1.4)	-	0.349	116 (2.6)
DPHP	DPHP-d10	0.026	0.038	101 (6.5)	-	0.380	104 (9.9)
BMPP	BMPP-d14	0.003	0.004	94 (4.7)	-	0.044	91 (7.5)
BBOEHEP	TCIPP-d18	-	0.0211	101 (5.2)	-	-	-
4-OH-DPHP	TPHP-d15	-	0.022	91 (5.7)	-	-	-
5-OH-EHDPP	TPHP-d15	-	0.0038	119 (2.6)	-	-	-

[a] Matrix Spike Recovery (%, n=3) of m-OPEs in urine (5 ng/mL for di-OPEs; 2ng/mL for hydroxylated OPEs); [b] The compound was not detected in blank samples. [c] Blank spike Recovery (%, n=3) of di-OPEs in dust (10 ng/g).

Table S6. Comparison of concentrations (μg/g) of OPEs in dust NIST Standard Reference Material (SRM) 2585 across different studies. Our values based on six replicates

Chemicals	This study	Shoeib et al 2019[26]	Van den Eede et al 2011[27]	Bergh et al 2012[28]	Brandsma et al 2013[29b]	Fan et al 2014[30]	Vykoukalová et al 2017[20] [c]	
TnBP	0.16 (0.04)	-[a]	0.18 (0.02)	0.19 (0.02)	0.269	0.24 (0.04)	0.690 (0.03)	0.572 (0.05)
TCEP	0.97 (0.15)	0.91(0.02)	0.70 (0.17)	0.84(0.06)	0.792	0.88 (6.9)	0.80(0.07)	1.86(0.08)
TCPP	0.86 (0.08)	0.64(0.02)	0.82 (0.10)	0.88 (0.14)	0.944	1.0 (0.15)	1.77(0.07)	0.99(0.07)
TDCIPP	2.58 (0.23)	2.22(0.03)	2.0 (0.26)	2.3 (0.28)	1.556	2.3 (0.16)	2.07(0.26)	3.73(0.18)
TBOEP	96.9 (16.2)	73.4(7.2)	49 (9.6)	82 (6.5)	73.464	83 (6.9)	-	-
TPHP	0.84 (0.06)	1.34(0.07)	0.99(0.07)	1.1(0.10)	1.104	0.92(0.13)	0.94(0.03)	1.52(0.16)
TMPP	0.75 (0.06)	-	1.07(0.11)	0.74(0.11)	NA	0.71(0.08)	-	-
EHDPP	1.2 (0.18)	-	-	1.3 (0.12)	0.963	1.3 (0.25)	4.7 (5.2)	1.8 (0.10)
TEHP	0.93 (0.02)	0.4(0.06)	-	0.37 (0.33)	0.265	-	0.14(0.07)	0.98(0.07)

The values were showed as mean (SD). [a] This compound was not measured in this study. [b] The values from Brandsma et al., 2013 indicating mean values from 14 laboratories in 10 different countries. [c] Values in the first column are from RECETOX, second column are from Indiana University (both values are from Vykoukalová et al., 2017).

Table S7. Comparison of concentrations (ng/mL) of di-OPEs in urine NIST Standard Reference Material (SRM) 3673 across different studies. Our values based on seven replicates

Chemicals	This study[a]	Bastiaensen et al 2019[31] [b]
BCEP	< MDLs	<LOQ-1.68
BCIPP	0.090 (0.002)	<LOQ-0.601
BDCIPP	1.680 (0.224)	0.848-1.50
DPHP	0.752 (0.187)	0.387-0.763
BBOEP	0.286 (0.027)	-

[a]The concentrations were showed as mean values (SD); [b]The values from Bastiaensen et al., 2019 indicating mean ranges from 9 laboratories.

Table S8. MS/MS parameters used for OPEs analysis.

Native chemicals	Quantitative transition		DP (V)	CE (V)	CXP (V)	Qualitative transition		DP (V)	CE (V)	CXP (V)	RT (Min)
TMP	141.1	109.1	60	22	10	141.1	79.0	60	29	6	2.50
TEP	183.0	99.0	54	24	7	183.0	81.0	60	50	8	5.10
TPrP	225.4	99.0	60	22	7	225.4	141	60	24	10	6.41
TnBP	267.4	99.0	60	20	10	267.4	155	60	12	10	7.06
TiBP	267.4	99.0	60	20	10	267.4	155	60	12	10	7.01
TEHP	435.3	99.0	140	22	9	435.3	113	120	16	8	8.74
TBOEP	399.3	299.3	95	19	10	399.3	199.0	95	21	10	7.12
TCIPP	327.0	99.0	70	30	10	329.1	99.0	70	28	10	6.41
TCEP	285.0	63.0	80	42	10	285.0	99.2	75	30	10	5.54
TDCIPP	431.1	98.9	85	35	9	431.1	208.9	84	20	8	6.80
TPHP	327.1	77.1	130	65	7	327.1	152.0	130	42	11	6.85
TMPP	369.2	166.1	147	37	11	369.2	90.9	147	61	8	7.30
CDPP	341.1	152.1	135	40	10	341.1	165.1	135	40	10	7.01
EHDPP	363.2	251.0	72	12	9	363.2	76.9	70	71	7	7.43
RDP	575.3	419.2	190	46	15	597.2	481.1	183	46	15	7.15
BABP	693.2	367.0	200	45	15	693.2	693.3	200	12	15	7.51
d9-TMP	150.1	83.1	90	31	7						2.40
d15-TEP	198.1	101.9	65	27	8						5.05
d21-TPRP	246.4	102	120	25	9						6.36
d27-TnBP	294.4	101.9	140	25	10						7.01
d15-TPHP	342.3	160	135	47	10						6.80
d12-TCEP	299.1	102	75	30	6						5.51
d18-TCIPP	345.1	101.9	75	30	8						6.38

Table S9. MS/MS parameters used for m-OPEs analysis.

Native chemicals	Quantitative transition		DP (V)	CE (V)	CXP (V)	Qualitative transition		DP (V)	CE (V)	CXP (V)	RT (Min)
DBP(DnBP+DiBP)	209	78.9	-70	-25	-7	209	152.9	-70	-19	-8	5.27
BBOEP	297	78.8	-100	-28	-6	297	197.0	-100	-25	-10	5.64
BEHP	321	78.9	-110	-70	-7	321.3	209.0	-110	-33	-8	6.77
BCEP	221	35.1	-15	-22	-10	222.7	36.9	-15	-23	-10	1.70
BCIPP	248.8	34.9	-30	-24	-9	250.9	37.0	-30	-26	-10	4.36
BDCIPP	316.9	35.0	-35	-37	-5	318.9	37.0	-35	-39	-9	5.46
DPHP	248.9	92.9	-80	-30	-6	248.9	155	-80	-28	-10	5.08
BMPP(DoCP+DpCP)	277	107	-95	-32	-8	277	169.0	-95	-30	-6	5.67
BBOEHEP	343.2	243.1	72	15	16	343.2	101.1	70	19	10	6.33
4-OH-DPHP	343.1	141.1	119	36	5	343.1	215.1	130	36	8	6.54
5-OH-EHDPP	379.1	251.0	70	18	12	379.1	153.1	130	46	7	6.78
d18-DnBP	227.1	78.9	-70	-27	-7	227	163	-20	-22	-8	5.24
d34-BEHP	355.3	78.9	-130	-85	-6	355.3	227.1	-130	-34	-8	6.73
d8-BBOEP	305.0	78.9	-52	-61	-7	-	-	-	-	-	5.61
d8-BCEP	229.0	35.0	-22	-27	-9	-	-	-	-	-	1.65
d12-BCIPP	260.6	35	-20	-23	-9	263.0	37.0	-20	-26	-10	4.30
d10-BDCIPP	326.8	35	-40	-38	-9	328.7	35	-40	-38	-9	5.42
d10-DPHP	258.9	98	-90	-35	-8	258.9	158.9	-90	-30	-8	5.02
d14-DoCP	291	114	-100	-37	-9	291	174.9	-100	-33	-12	5.63
d14-DpCP	291	114	-100	-37	-9	291	174.9	-100	-33	-12	5.63

Table S10. Comparison of median concentration of OPEs (ng/mL) in blood with relevant studies available across the world

Country	Number	Date	Age (years)	Types	TMP	TEP	TnBP	TiBP	TEHP	TBOEP	TCIPP	TCEP	TDCIPP	TPHP	EHDPP	TMPP	References
Jinan, China	352	2018	60-70	Whole blood	ND	0.136	0.250	0.229	0.091	ND	**0.743**	0.298	0.107	**0.400**	0.209	ND	This study
Shenzhen, China	255	2012	20-50	Whole blood	-	0.49	**37.8**	-	0.04	0.54	0.71	ND	ND	0.43	1.22	0.09	[32]
Four cities in Jiangsu Province, China	99	2013	18-87	Plasma/ serum	-	0.15	ND	-	ND	0.05	0.05	0.1	ND	0.35	**0.85**	-	[33]
Hengshui, China	30	2017	20-50	Plasma	-	-	ND	-	-	ND	**0.36**	0.18	ND	**0.46**	-	-	[16]
Beijing, China	57	2018	17-87	Whole blood	ND	0.432	0.176	0.532	ND	0.164	ND	ND	ND	0.366	**1.10**	ND	[3]
Gran Canaria, Spain [a]	20	2016	-	Serum	-	ND	64.8	47.7	0.4	56.4	93.9	3.69	ND	22.7	**425.8**	ND	[34]
Bohai Bay, North China [a]	89	2018	22-88	Serum	-	ND	ND	ND	-	-	ND	**214**	-	ND	7.2	ND	[35]

[a]Concentration unit is ng/g lipid weight.

Table S11. Spearman correlation coefficients between concentrations of OPEs in whole blood (n = 352)

Name	TEP	TnBP	TiBP	TEHP	TPHP	EHDPP	TCEP	TCIPP
TEP								
TnBP	-0.027							
TiBP	-0.051	**0.553****						
TEHP	-0.209**	**0.135***	0.050					
TPHP	0.013	**0.200****	0.037	0.027				
EHDPP	-0.118*	**0.228****	0.068	**0.301****	-0.174**			
TCEP	**0.106***	**0.131***	**0.179****	**0.119***	-0.003	0.085		
TCIPP	0.088	**0.125***	**0.157****	-0.036	-0.046	**0.229****	**0.289****	

**p<0.01 (Two tails); *p<0.05 (Two tails). Correlation analyses were not performed for other OPEs due to their low DFs (<50%) in whole blood samples.

Table S12. ICC (95% CIs) for OPE concentrations in whole blood and di-OPE concentrations in urine

OPEs in whole blood[a]	ICC (95% CIs)	Urinary metabolites[b]	Unadjusted	SG-adjusted
TEP	**0.41 (0.13-0.62)**	DBP	**0.45 (0.19-0.65)**	0.29 (-0.06-0.54)
TnBP	0.08 (-0.37-0.41)	BEHP	0.22 (-0.14-0.50)	-0.09 (-0.61-0.30)
TiBP	-0.04 (-0.53-0.34)	DPHP	**0.74 (0.61-0.83)**	**0.64 (0.46-0.77)**
TEHP	-0.001 (-0.48-0.36)	BMPP	**0.64 (0.46-0.77)**	**0.64 (0.46-0.77)**
TCEP	0.27 (-0.07-0.53)	BDCIPP	**0.51 (0.27-0.68)**	**0.41 (0.13-0.62)**
TCIPP	**0.40 (0.11-0.62)**			
TPHP	0.27 (-0.09-0.53)			
EHDPP	0.13 (-0.28-0.44)			

The concentrations of OPEs and di-OPEs were $\log_{10}$ transformed

[a] n = 352 whole blood samples collected every month for 5 months from 76 elderly people

[b] n = 353 urine samples collected every month for 5 months from 76 elderly people

Table S13. Spearman correlation coefficients between concentrations of OPEs in whole blood and demographic parameters of participants (n = 76)[a]

Category	TEP	TnBP	TiBP	TEHP	TPHP	EHDPP	TCEP	TCIPP	∑ 16OPEs
Sex	-0.182	-0.174	0.041	-0.082	-0.079	-0.005	-0.049	-0.135	**-0.283**
	0.115	0.133	0.723	0.484	0.497	0.967	0.673	0.245	**0.013**
Age	0.164	**0.310**	**0.354**	-0.024	0.035	-0.042	0.082	**0.241**	0.207
	0.156	**0.007**	**0.002**	0.839	0.761	0.722	0.480	**0.036**	0.073
BMI	0.017	-0.137	-0.135	-0.153	0.022	-0.163	0.108	-0.092	-0.178
	0.886	0.239	0.245	0.187	0.850	0.160	0.354	0.429	0.123
Weight	0.122	-0.051	-0.104	-0.088	0.115	-0.195	0.016	-0.086	-0.010
	0.292	0.662	0.370	0.449	0.325	0.092	0.890	0.458	0.931
Waist circumference	0.104	-0.003	-0.071	-0.107	0.009	-0.218	0.140	-0.034	-0.013
	0.372	0.977	0.540	0.357	0.940	0.058	0.228	0.771	0.909
Hours[b]	-0.136	0.050	0.063	0.057	0.184	-0.115	-0.001	-0.097	0.016
	0.242	0.667	0.588	0.626	0.111	0.322	0.991	0.404	0.888

[a]Average concentrations of OPEs in whole blood collected five times were used. [b]Average hours per day spent in home of the participants.

Table S14. Results of regression analyses for predicting concentrations of several OPEs in whole blood

Predictor	TnBP		TiBP		TCIPP		∑ 16OPEs	
	10^{β} (95% CI)	*p*	10^{β} (95% CI)	*p*	10^{β} (95% CI)	*p*	10^{β} (95% CI)	*p*
Sex								
Female	Reference		Reference		Reference		Reference	
Male	1.22(0.914-1.62)	0.177	0.953(0.688-1.32)	0.766	1.28(0.929-1.75)	0.129	**1.17(1.04-1.31)**	**0.012**
Age(years)	**1.06(1.01-1.12)**	**0.017**	**1.09(1.04-1.16)**	**0.002**	1.04(0.988-1.11)	0.113	1.02(0.995-1.04)	0.101

Table S15. Distribution of unadjusted and specific gravity (SG)-adjusted concentrations of OPEs and their metabolites in urine collected every 1 month for 5 months from 76 elderly people

Chemicals	Unadjusted (ng/mL) (n=353)				SG-adjusted (ng/mL) (n=352)			Correlation[a]
	DF (%)	Range	Median	Geomean	Range	Median	Geomean	r_s
DBP	69	<0.026-0.568	0.049	0.0448	ND-0.661	0.052	0.051	0.920**
BEHP	77	<0.069-1.03	0.149	0.129	ND-1.91	0.146	0.147	0.906**
BBOEP	13	<0.003-16.7			ND-16.7	-	-	-
BCEP	20	<0.625-6.40			ND-8.53	-	-	-
BCIPP	28	<0.042-1.30			ND-1.30	-	-	-
BDCIPP	76	<0.016-3.81	0.108	0.0933	ND-4.78	0.121	0.106	0.890**
DPHP	79	<0.038-3.41	0.084	0.0736	ND-6.82	0.089	0.083	0.863**
BMPP	65	<0.008-0.185	0.013	0.0116	ND-0.159	0.014	0.013	0.894**
BBOEHEP	3	<0.021-0.202			ND-0.202	-	-	-
DPHP-OH	21	<0.022-0.226			ND-0.242	-	-	-
EHDPP-OH	13	<0.003-0.103			ND-0.205	-	-	-
TEP	43	<0.011-2.28			ND-2.83	-	-	-
TPrP	68	<0.022-0.760	0.065	0.0513	ND-3.04	0.064	0.051	-
TnBP	15	<0.005-0.902			ND-1.20	-	-	-
TiBP	29	<0.006-0.868			ND-1.08	-	-	-
TBOEP	17	<0.003-3.86			ND-3.86	-	-	-
TCEP	17	<0.039-1.05			ND-2.40	-	-	-
TCIPP	40	<0.010-1.22			ND-1.47	-	-	-
TDCIPP	48	<0.026-1.02			ND-1.61	-	-	-

The DFs of TMP, TEHP, TPHP, EHDPP, TMPP, CDPP, BABP, and RDP in urine samples were lower than 5%. [a]The spearman correlation coefficients between unadjusted urinary concentrations and SG-adjusted concentrations.

Table S16. Comparison of median concentration of di-OPEs (ng/mL) in urine with relevant studies available across the world

Countries	Population	n	DBP	BEHP	BBOEP	BCIPP	BCEP	BDCIPP	DPHP	BMPP	Reference
Jinan, China	The elderly [a]	353	0.052	0.146	ND	ND	ND	0.121	0.089	0.014	This study
Beijing, China	Adults [b]	52	0.230	6.76	2.65	0.150	2.57	0.291	0.177	0.013	3
Shanghai, China,	Adults and Children [a]	180	0.0031	0.0864	0.0975	ND	ND	ND	0.066	ND	36
Nanchang, China	Children [b]	227	0.06	-	0.04	0.69	0.85	0.08	0.27	0.004	37
Guangzhou, China	Children [a]	199	0.12	-	0.05	0.14	0.52	0.06	0.13	0.03	38
Shenzhen, China	Children [a]	212	0.13	-	0.05	0.16	2.11	0.05	0.35	0.02	
13 cities, China	[b]	323	0.29	-	0.026	0.30	0.68	0.15	0.30	0.015	12
Shenzhen, China	Primiparas [a]	84	0.18	-	0.06	0.14	1.32	0.25	0.26	0.07	39
Hangzhou, China	Adolescents [b]	306	2.54	-	0.11	0.18	1.0	6.17	0.42	-	40
New Jersey, US	Children	22	-	-	-	(ND) [c]	-	(2.4)	(1.9)	-	41
	Mothers	26	-	-	-	(ND)	-	(5.6)	(3.0)	-	
US	Pregnant women	8	-	-	-	-	-	(1.3)	(1.9)	-	42
California, US	Children	33	-	-	-	(ND)	-	(10.9)	(2.9)	-	43
	Mothers	28	-	-	-	(ND)	-	(3.3)	(1.2)	-	
California, US	Adults [b]	13	-	-	-	0.3	1.3	2.4	1.5	-	44
USA	Toddlers	41	-	-	-	-	-	(6.81)	(3.37)	-	45
New York, US	Adults [a]	213	0.018	0.015	0.029	0.033	0.302	0.359	0.919	ND	10
California, US	Adults [b]	16	0.11	-	ND	ND	0.63	0.09	0.44	-	46
Australia	Population [b]	28	(ND)	-	(ND)	-	-	(1)	(24.4)	-	47
Australia	Children [b]	400	(0.18)	(ND)	(0.32)	(0.85)	(ND)	(2.6)	(25)	(0.024)	4
Australia	Young children [b]	-	0.15	ND	0.10	0.68	ND	3.3	1	0.015	48
Canada	Pregnant women [b]	24	-	-	ND	0.46	0.46	0.26	2.94	0.69	49
Germany	Children [b]	312	0.2	-	2.0	ND	0.2	-	0.8	ND	50
Norway	Children [a]	112	ND	-	ND	-	-	0.23	1.1	-	51
	Mothers [a]	244	ND	-	ND	-	-	0.08	0.63	-	

[a] Specific gravity adjusted concentrations. [b] unadjusted concentrations. [c] geometric mean (in parentheses).

Table S17. Spearman correlation coefficients between concentrations of di-OPEs in urine

Chemicals	Unadjusted (n = 353)					SG-adjusted (n = 352)				
	DPHP	**BDCIPP**	**DBP**	**BMPP**	**BEHP**	**DPHP**	**BDCIPP**	**DBP**	**BMPP**	**BEHP**
DPHP	1.000					1.000				
	.					.				
BDCIPP	**0.299****	1.000				**0.292****	1.000			
	0.000	.				**0.000**	.			
DBP	**0.385****	**0.145****	1.000			**0.413****	**0.209****	1.000		
	0.000	**0.006**	.			**0.000**	**0.000**	.		
BMPP	**0.485****	**0.306****	**0.329****	1.000		**0.438****	**0.302****	**0.350****	1.000	
	0.000	**0.000**	**0.000**	.		**0.000**	**0.000**	**0.000**	.	
BEHP	-0.042	-0.023	-0.161**	-0.144**	1.000	-0.046	0.028	-0.048	-0.115*	1.000
	0.426	0.670	0.002	0.007	.	0.392	0.600	0.373	0.031	.

**p<0.01 (Two tails); *p<0.05 (Two tails). Correlation analyses were not performed for BCEP and BCIPP due to their low DFs (<50%) in urine.

Table S18. Spearman correlation coefficients between concentrations of di-OPEs in urine and demographic parameters of participants (n=76)[a]

Category	Unadjusted					SG-adjusted				
	DPHP	**BDCIPP**	**DBP**	**BMPP**	**BEHP**	**DPHP**	**BDCIPP**	**DBP**	**BMPP**	**BEHP**
Sex	-.113	-.154	.006	-.180	.041	-.064	-.088	.061	-.139	.100
	.329	.184	.959	.120	.726	.585	.0.452	.603	.230	.389
Age (years)	.083	-.082	-.128	-.091	-.203	.053	0.055	-.100	-.012	-.154
	.475	.481	.272	.435	.079	.650	.639	.391	.920	.183
Weight (kg)	.054	.199	.134	.179	.012	-.006	.106	.099	.154	-.044
	.643	.085	.250	.122	.915	.958	.362	.397	.185	.704
Waist circumference (cm)	-.013	.204	.090	.163	-.079	-.114	.143	.020	.158	-.143
	.908	.076	.440	.160	.498	.328	.219	.866	.172	.217
BMI	-.010	.081	.153	.077	-.057	-.094	.059	.136	.079	-.073
	.929	.486	.186	.508	.627	.419	.612	.241	.496	.534
Hours (h)[b]	.123	-.047	-.147	-.006	-.008	.067	.083	-.142	.017	.090
	.288	.685	.204	.957	.946	.567	.478	.221	.882	.441

[a]Average concentrations of di-OPEs in urine collected five times were used. [b]Average hours per day spent in home of the participants.

Table S19. Associations between OPEs in whole blood/di-OPEs in urine with OPEs in indoor dust or air, along with the associations between urine and indoor dust for di-OPEs, and between di-OPEs in urine and OPEs in whole blood

	OPEs in whole blood[a]			**di-OPEs in urine[b]**				
	vs. home air	**vs. home dust**		**vs. home air**	**vs. home dust**	**vs. OPEs in whole blood[c]**		**vs. di-OPEs in home dust**
	n = 55	**n = 55**		**n = 63**	**n = 63**	**n = 76**		**n = 50**
TEP	-0.041[d]	0.023	DBP-TBP	-0.059	0.071	-0.174	DBP	0.063
	0.768[e]	0.866		0.645	0.582	0.133		0.662
TnBP	**0.392****	0.082	BEHP-TEHP	-0.205	-0.117	-0.011	BEHP	-0.015
	0.003	0.550		0.106	0.362	0.928		0.917
TiBP	0.230	-0.069	DPHP-TPHP	0.032	0.094	0.178	DPHP	-0.104
	0.092	0.618		0.801	0.463	0.125		0.470
TEHP	0.111	**0.306***	DPHP-EHDPP	-0.028	-0.065	-0.042		-
	0.419	**0.023**		0.825	0.611	0.719		-
TPHP	-0.136	-0.089	DPHP-BABP	0.094	-0.143	-		-
	0.323	0.517		0.462	0.265	-		-
EHDPP	0.131	-0.034	BMPP-TMPP	-0.241	-0.028	-	BMPP	-0.013
	0.339	0.805		0.057	0.825	-		0.930
TMPP	0.107	-0.060	BDCIPP-TDCIPP	-0.111	-0.064	-	BDCIPP	-0.098
	0.435	0.663		0.388	0.617	-		0.498
TCEP	-0.061	0.172						
	0.659	0.209						
TCIPP	-0.064	0.110						
	0.643	0.425						

Associations were not analyzed for other OPEs/di-OPEs due to their low detection frequencies; [a]The average concentrations of OPEs in whole blood collected in the fourth and fifth sampling campaign were used; The measurements from seven couples were removed from the correlation analyses. The DFs of TEP, TnBP, TiBP TEHP, TPHP, EHDPP, TMPP, TCEP, and TCIPP in whole blood from the fourth and fifth sampling campaign were 82%, 84%, 82%, 64%, 98%, 95%, 58%, 82%, and 89%, respectively. [b]The average SG-adjusted concentrations of di-OPEs in urine collected in the fourth and fifth sampling campaign were used; [c]The average concentrations of OPEs/di-OPEs in whole blood/urine collected from the five months were used; [d]Spearman correlation coefficient; [e]*p* value;

Table S20. Concentrations of OPEs in air and dust samples and spearman's rank correlations (r_s) between indoor air and dust samples. The font is bold if significance was found.

Chemicals	Indoor air (ng/m^3) (n=63)				Outdoor air (ng/m^3) (n=6)	Indoor dust (ng/g) (n=64)				Outdoor dust (ng/g) (n=1)	Correlations between indoor air and dust (n=62)	
	DF[a]	Range	Median	Geomean	Median	DF	Range	Median	Geomean		r_s [b]	*p*
TMP	62	<0.042-2.354	0.111	0.099	0.215	25	<1.39-9.00	-	-	<1.39	- [c]	-
TEP	100	0.666-72.3	8.85	8.42	0.600	98	2.68-834	47.0	52.3	44.5	**0.256***	**0.044**
TPrP	27	<0.004-0.036	-	-	0.003	100	1.56-28.3	8.34	7.84	<0.0633	-	-
TnBP	100	0.073-1.95	0.424	0.398	0.084	100	2.89-2192	9.20	10.7	6.28	0.152	0.239
TiBP	100	0.126-3.86	0.715	0.700	0.144	83	2.36-3201	13.4	12.2	14.4	0.169	0.188
TEHP	100	0.037-0.488	0.145	0.145	0.177	100	8.83-464	27.8	34.6	49.3	-0.135	0.297
TBOEP	60	<0.008-0.057	0.011	0.010	0.012	100	6.95-369	38.4	38.4	6.93	-0.107	0.409
TPHP	100	0.026-4.49	0.098	0.113	0.036	100	44.7-1199	192	205	63.4	0.147	0.253
EHDPP	100	0.038-0.429	0.104	0.112	0.024	100	30.7-1228	140	151	119	0.228	0.074
CDPP	48	<0.018-0.102	-	-	0.011	100	2.46-347	21.9	21.7	7.01	-	-
TMPP	100	0.002-0.071	0.012	0.012	0.014	100	2.54-435	15.3	14.9	5.57	-0.075	0.560
BABP	100	0.003-0.338	0.032	0.036	0.023	100	1.35-784	12.8	12.8	11.29	-0.108	0.404
RDP	79	<0.0002-0.829	0.080	0.033	-	28	<0.04-94.3	-	-	<0.04	-	-
TCEP	100	0.174-15.0	1.23	1.50	0.731	100	108-10950	610	560	195	**0.434****	**0.0004**
TCIPP	100	0.461-72.5	4.42	5.51	0.474	100	109-4581	544	556	194	**0.392****	**0.002**
TDCIPP	97	<0.004-0.635	0.072	0.072	0.036	100	26.8-3166	126	165	7.74	**0.288***	**0.023**
$\sum_{16}$OPEs		2.10-137	21.4	21.3	2.78		789-16840	2340	2412	725	**0.387****	**0.002**

[a] Detection frequency; [b] Spearman correlation coefficient; [c] Correlation analyses were not performed for compounds with DFs < 50% in indoor air or indoor dust.

Table S21. Concentrations of di-OPEs in indoor dust (n = 61; ng/g) and correlations and concentration ratios for di-/tri-OPEs pairs

	Indoor dust							Outdoor dust [c]	
	DF (%)	Range	Median	Geomean	Di/tri-OPEs pairs	Correlations	Concentration Ratios	Concentration	Concentration Ratio
BEHP	100	45.8-2112	216	232	BEHP/TEHP	0.148	7.49 (0.498-124) [b]	45.8	0.929
DBP	100	5.22-326	31.8	33.1	DBP/TBP	-0.168	1.35 (0.174-37.0)	5.22	0.252
DPHP	100	1.70-141	14.9	14.9	DPHP/TPHP	**0.401****	0.065 (0.011-1.47)	3.69	0.0583
					DPHP/EHDPP	**0.299***	0.103 (0.010-0.993)		0.0312
					DPHP/CDPP	**0.356****	0.649 (0.046-22.3)		0.527
					DPHP/BABP	-0.089	0.046 (0.0002-11.1)		0.327
BDCIPP	62	<0.349-19.7	2.29	1.27	BDCIPP/TDCIPP	**0.516****	0.008 (0.0004-0.371)	2.17	0.280
BCIPP	37	<0.415-48.4	-	-	BCIPP/TCIPP	- [a]	0.001 (0.00005-0.077)	5.63	0.0289
BCEP	37	<10.3-794	-	-	BCEP/TCEP	-	0.042 (0.004-0.504)	ND	-
BBOEP	98	<0.133-116	7.10	6.77	BBOEP/TBOEP	**0.662****	0.200 (0.012-0.942)	0.655	0.0945
BMPP	100	0.104-15.5	0.591	0.527	BMPP/TMPP	0.207	0.032 (0.005-0.334)	2.86	0.514

**p<0.01 (Two tails); *p<0.05 (Two tails).

[a] Correlation analyses were not performed for BCIPP/TCIPP and BCEP/TCEP pairs due to the low DFs of BCIPP and BCEP in indoor dust.

[b] Concentration ratios are expressed as the median values and ranges (in parentheses).

[c] One value was obtained as we only collected a mixed outdoor dust sample.

Table S22. Spearman correlation coefficients between concentrations of di-OPEs in home dust samples (n = 61)

	BEHP	DPHP	DBP	BDCIPP	BBOEP	BMPP
BEHP						
DPHP	**0.612****					
DBP	**0.590****	**0.754****				
BDCIPP	0.227	**0.382****	**0.428****			
BBOEP	**0.576****	**0.489****	**0.663****	0.236		
BMPP	**0.428****	**0.454****	**0.576****	**0.356****	**0.375****	

**p<0.01 (Two tails); *p<0.05 (Two tails).

Correlation analyses were not performed for BCEP and BCIPP due to their low DFs (<50%) in indoor dust.

Table S23. Comparison of median concentration of OPEs (ng/m^3) in home air with relevant studies available across the world. Compounds with orange background were the dominant OPEs in home air in the literatures.

Country	Number	TMP	TEP	TnBP	TiBP	TEHP	TBOEP	TCIPP	TCEP	TDCIPP	TPHP	EHDPP	TMPP	Method	References
Jinan, China	63	0.111	8.85	0.424	0.715	0.145	0.011	4.42	1.23	0.072	0.098	0.104	0.012	SIP disks	This study
Beijing, China	15	ND	0.39	0.17	0.15	ND	0.0027	3.8	0.17	ND	0.034	0.0084	ND	PUF disks	[52]
Haerbin, China	25	-	-	1.27	1.82	0.066	0.401	ND	ND	ND	0.284	0.125	-	PUF disks	[53]
Bloomington, IN, US	20	-	-	6.07	-	0.376	-	26.3	6.81	0.372	0.799	0.739	0.142		
Toronto, ON, Canada	23	-	-	6.2	-	0.042	-	73.6	6.35	0.525	0.723	1.71	0.005	PUF disks	[20]
Brno, Czech Republic	20	-	-	2.34	-	0.037	-	16.4	2.96	0.311	0.592	0.375	0.001		
Oslo, Norway	61	-	-	14	-	ND	ND	128	3	ND	1	ND	ND	SPE cartridges	[6]
Oslo, Norway	47	-	-	5.09	-	-	0.598	42.3	2.25	0.084	0.258	0.119	ND	Active sampling	[5]
Australia	40	-	-	1.8	-	0.014	0.14	16	3.2	0.054	0.37	0.10	0.018	PUF-GFF	[54]
Nepal	34	-	-	0.21	-	0.79	-	0.63	0.33	0.07	0.23	0.41	2.56	PUF disks	[55]
Japan	18	ND	2.4	4.0	-	-	1.8	1.9	1.3	ND	ND	-	-	Active sampling	[56]
Germany	7	-	ND	0.62	4.05	ND	ND	4.15	ND	ND	-	-	-	Active sampling	[57]
Stockholm, Sweden	10	-	7.3	9.1	13	ND	ND	5.6	4.8	ND	ND	-	-	SPE cartridges	[58]

Table S24. Spearman correlation coefficients between concentrations of OPEs in home air samples (n = 63)

	TMP	**TEP**	**TnBP**	**TiBP**	**TBOEP**	**TEHP**	**TPHP**	**EHDPP**	**TMPP**	**BABP**	**RDP**	**TCIPP**	**TCEP**	**TDCIPP**
TMP														
TEP	0.240													
TnBP	-0.141	**0.328****												
TiBP	-0.044	**0.391****	**0.821****											
TBOEP	0.064	0.024	0.135	0.130										
TEHP	0.000	-0.231	0.015	0.090	-0.001									
TPHP	0.176	**0.289***	0.214	**0.294***	0.231	0.219								
EHDPP	**0.273***	**0.364****	**0.299***	**0.389****	**0.335****	0.182	**0.768****							
TMPP	0.117	**0.254***	0.119	0.210	0.165	0.238	**0.590****	**0.625****						
BABP	0.153	0.216	0.182	**0.268***	0.186	-0.011	**0.443****	**0.554****	**0.352****					
RDP	-0.072	0.226	0.241	0.198	-0.045	0.132	**0.371****	**0.306***	0.233	0.113				
TCIPP	0.196	**0.566****	**0.312***	**0.321****	0.031	0.082	**0.494****	**0.443****	**0.289***	0.196	**0.459****			
TCEP	0.160	**0.308***	0.125	0.165	0.210	0.147	**0.491****	**0.460****	**0.381****	0.211	**0.367****	**0.470****		
TDCIPP	**0.286***	0.218	0.130	0.126	0.130	0.246	**0.285***	**0.270***	0.201	0.071	**0.415****	**0.375****	**0.645****	

***p*<0.01 (Two tails); **p*<0.05 (Two tails). Correlation analyses were not performed for TPrP and CDPP due to their low DFs (<50%) in indoor air

Table S25. Spearman's rank correlations between concentrations of OPEs in home air and household factors

n=63		TMP	TEP	TNBP	TIBP	TBOEP	TEHP	TPHP	EHDPP	TMPP	BABP	RDP	TCPP	TCEP	TDCIPP	SUM
T (°C) [a]	R	0.154	-0.024	-0.214	-0.220	0.134	-0.504	-0.070	0.002	0.103	0.013	-0.295	-0.168	-0.044	-0.109	-0.071
	p	0.227	0.854	0.092	0.083	0.297	0.000	0.585	0.986	0.421	0.920	0.019	0.188	0.734	0.396	0.580
H (%) [b]	R	0.070	0.185	0.061	0.079	0.074	-0.148	0.055	-0.045	0.026	-0.145	0.078	0.264	0.337	0.408	0.287
	p	0.584	0.147	0.633	0.541	0.566	0.247	0.667	0.729	0.838	0.255	0.543	0.037	0.007	0.001	0.022
Building constr. year (year) [c]	R	-.107	-.066	-.188	-.030	-.065	-.013	-.255	-.249	-.155	.079	.002	-.139	-.043	-.227	-.149
	p	.404	.609	.141	.814	.610	.920	.044	.049	.224	.538	.990	.277	.740	.074	.244
Years lived in the household [d]	R	.072	-.033	-.101	.007	-.219	.145	-.096	-.012	-.036	-.065	.282	-.081	.082	.040	-.084
	p	.573	.796	.431	.956	.084	.258	.453	.929	.777	.611	.025	.530	.524	.753	.512
Number of inhabitants [e]	R	.077	.156	.326	.193	.022	.014	.041	-.010	.065	.005	-.088	-.009	-.188	-.158	.100
	p	.546	.222	.009	.130	.863	.912	.749	.939	.612	.967	.494	.945	.140	.216	.436
Size of apartment (m^2) [f]	R	-.042	.348	.113	.138	.023	-.095	-.029	-.032	-.010	.146	-.046	.051	-.078	-.078	.222
	p	.744	.005	.380	.281	.855	.458	.823	.802	.940	.254	.723	.694	.545	.545	.081
Floor [g]	R	.125	.030	.063	.139	.085	-.098	.039	.053	-.165	.007	-.083	.109	.146	.162	.102
	p	.330	.815	.622	.276	.505	.447	.764	.681	.195	.954	.519	.397	.255	.204	.425
Annual household Income (Yuan) [h]	R	-.136	.062	.070	.037	-.175	-.156	-.205	-.313	-.187	-.018	-.122	-.058	-.126	-.121	-.012
	p	.289	.629	.586	.775	.170	.223	.106	.013	.142	.886	.341	.653	.326	.343	.924
Dry sweep or vacuum cleaning/3 days [i]	R	.136	.199	-.224	-.148	-.149	-.242	.046	.071	-.079	.004	-.019	-.041	-.056	-.025	.139
	p	.288	.117	.078	.246	.242	.056	.721	.578	.536	.978	.879	.747	.660	.848	.276
Window opening time/day [j]	R	.185	-.019	.107	.122	-.036	.215	.133	.078	-.075	-.091	-.067	.060	-.016	.156	.032
	p	.147	.882	.406	.342	.777	.091	.298	.543	.558	.477	.603	.641	.900	.222	.801
Window opening time/day [k]	R	-.118	.091	.246	.173	.117	.153	.151	.176	-.119	.057	.135	.047	-.041	.195	.092
	p	.357	.479	.052	.175	.362	.233	.238	.167	.352	.655	.293	.716	.747	.126	.475
Doors and windows remold (yes/no) [l]	R	-.104	.002	.043	.066	-.061	-.102	-.004	.053	.013	-.079	-.050	.057	.058	.074	.058
	p	.416	.989	.739	.605	.633	.426	.973	.678	.919	.536	.697	.659	.654	.563	.654
Renovation of the house(yes/no) [m]	R	.164	-.124	-.246	-.228	-.017	-.140	-.055	-.057	-.043	.102	.080	.072	.112	.165	-.077
	p	.198	.334	.052	.073	.893	.275	.667	.657	.737	.427	.532	.575	.380	.196	.547
Fresh air system (yes/no) [n]	R	-.038	-.209	-.033	-.014	.104	-.069	-.008	-.058	.100	-.276	-.191	-.191	.124	.111	-.161
	p	.765	.099	.799	.911	.418	.591	.950	.654	.435	.029	.134	.134	.333	.388	.208
Distance to main road [o]	R	-.016	.082	-.145	-.112	.014	-.256	-.201	-.071	-.269	-.064	.042	-.143	-.102	.001	-.047
	p	.902	.525	.257	.383	.913	.042	.114	.581	.033	.616	.746	.265	.424	.992	.717

Values with orange background means significant correlations ($p < 0.05$). Values with gray background was $p < 0.10$.

Fifteen factors were evaluated in this study, including: [a] Temperature; [b] Humidity; [c] Building construction year (< 20 years; ≥20 years); [d] Years

lived in the household (<10 years; ≥10 years); [e] Number of inhabitants in the household (≤ 3; >3); [f] Size of the apartment in m^2 (≤ 73 m^2; >73 m^2); [g] Floor (≤ 4; >4); [h] Annual household Income (≤ 87000yuan; >87000 yuan); [i] Number of dry sweeping and vacuum cleaning per 3 days (≤ 2; >2); [j] The daily window opening time of the living room (≤1h; >1h); [k] The daily window opening time of the bedroom (≤2h; >2h); [l] Doors and windows remold (1: yes; 2: no); [m] Renovation of the house in the last 10 years (1: yes; 2: no); [n] Use of fresh air system (1: yes; 2: no); [o] Distance to main road (<50m; 50-200m; >200m).

Table S26. Comparison of median concentration of OPEs (ng/g) in indoor dust with relevant studies available across the world. Compounds with orange background were the dominant OPEs in home dust in the literatures.

Country	Num	TEP	TPrP	TnBP	TiBP	TEHP	TBOEP	TCIPP	TCEP	TDCIP	TPHP	EHDPP	TMPP	CDPP	BABP	RDP	References
Jinan, China	64	47.	8.34	9.20	13.4	27.8	38.4	544	610	126	192	140	15.3	21.9	12.8	ND	This study
Beijing, China	21	37	ND	122	50	-	2259	2048	4992	479	376	-	448	-	-	-	59
Beijing, China	39	170	ND	30	13	280	110	1400	790	120	400	250	38	89	-	-	52
Shanghai, China	15	50	-	200	-	1200	1600	1500	1200	600	900	900	300	-	-	-	60
Guangzhou,	51	20	-	70	-	740	290	600	380	2800	530	830	190	80	530	60	24
Guangzhou,	11	110	-	80	-	140	320	750	3780	130	150	360	ND	-	-	-	61
Qingyuan, China	25	60	-	140	-	190	200	1220	1930	150	1090	310	ND	-	-	-	
Haerbin, China	25	-	-	115	263	352	ND	1780	754	196	374	230	-	-	-	-	53
Germany	15	ND	-	250	380	ND	4300	4200	1100	ND	1200	-	ND	-	-	-	62
Stockholm,	62	190	-	5600	530	ND	11000	11000	4000	2000	4300	-	2700	-	-	-	63
Oslo, Norway	61	-	-	ND	-	401	8146	1997	435	397	722	420	179	-	-	-	6
Oslo, Norway	48	-	-	55	-	-	13400	2680	414	501	981	617	307	-	-	-	5
Barcelona, Spain	11	-	-	-	-	115	2635	-	96	221	369	289	52	-	-	-	64
Belgian	33	ND	-	130	299	-	2030	1380	230	360	500	-	240	-	-	-	27
Portugal	28	-	2.4	28		1700	-	-	17	22	660	620	97	-	-	-	65
Australia	40	-	-	ND		ND	10000	6400	660	920	740	880	120	-	-	-	54
Japan	10	-	-	130	ND	ND	82000	1700	2700	2200	820	200	1200	-	-	-	66
Brazil	10	-	-	12.3	30.7	397	15900	771	230	1370	3900	1590		-	-	-	67
The Netherlands	21	-	-	-	-	-	-	815	157	1051	404	-	58	-	58	-	68
US	20	-	-	114	-	1360	-	2790	1440	3680	3040	889	82.5	-	-	-	
Canada	23	-	-	63	-	101	-	1470	181	917	2350	754	6.18	-	-	-	20
Czech Republic	20	-	-	51.6	-	153	-	1860	155	183	811	836	201	-	-	-	
Nepal	28	-	-	22.2	-	41.8	-	61.7	15.7	22 . 1	71.3	76.7	420	-	-	-	55
Assiut, Egypt	20	-	-	23	17	-	18	28	22	72	67	42	-	-	-	-	69

Table S27. Spearman correlation coefficients between concentrations of OPEs in home dust samples (n = 64)

	TEP	**TPrP**	**TnBP**	**TiBP**	**TBOEP**	**TEHP**	**TPHP**	**EHDPP**	**CDPP**	**TMPP**	**BABP**	**TCIPP**	**TCEP**	**TDCIPP**
TEP														
TPrP	0.002													
TnBP	0.126	-0.025												
TiBP	0.140	0.042	**0.796****											
TBOEP	**0.251***	0.115	-0.008	-0.095										
TEHP	0.020	0.077	-0.127	-0.137	0.233									
TPHP	**0.308***	0.222	0.133	0.136	**0.335****	0.096								
EHDPP	0.085	0.075	-0.063	0.031	0.110	**0.290***	**0.560****							
CDPP	**0.335****	0.189	0.125	0.153	**0.387****	0.102	**0.496****	**0.346****						
TMPP	**0.319***	0.180	**0.248***	0.173	**0.433****	0.173	**0.502****	**0.414****	**0.571****					
BABP	0.053	-0.083	0.004	0.000	0.156	**0.367****	0.027	-0.090	0.134	0.139				
TCIPP	**0.417****	-0.046	0.132	0.053	0.245	0.136	**0.262***	0.108	**0.281***	**0.299***	**0.307***			
TCEP	**0.321****	0.195	0.070	0.037	0.242	**0.343****	0.189	0.140	0.099	0.149	0.236	**0.374****		
TDCIPP	**0.338****	**0.347****	-0.011	0.079	**0.438****	**0.294***	**0.458****	0.226	**0.445****	**0.464****	0.097	**0.371****	**0.293***	

**p<0.01 (Two tails); *p<0.05 (Two tails). Correlation analyses were not performed for TMP and RDP due to their low DFs (<50%) in indoor dust

Table S28. Spearman's rank correlations between concentrations of OPEs in indoor dust and household factors

n=63		TEP	TPRP	TNBP	TIBP	TBOEP	TEHP	TPHP	EHDPP	CDPP	TMPP	BABP	TCPP	TCEP	TDCIPP	SUM
T (°C) [a]	R	-0.174	0.054	0.120	0.125	-0.138	0.145	-0.031	0.196	-0.257	-0.138	0.009	-0.244	0.292	-0.034	0.045
	p	0.172	0.675	0.348	0.331	0.282	0.258	0.808	0.124	0.042	0.282	0.946	0.054	0.020	0.790	0.724
H (%) [b]	R	0.046	-0.043	-0.240	-0.324	0.263	0.167	-0.075	-0.069	-0.020	0.263	0.062	0.154	0.161	0.103	0.219
	p	0.722	0.738	0.058	0.009	0.038	0.192	0.559	0.592	0.874	0.038	0.628	0.229	0.208	0.420	0.084
Building constr. year (year) [c]	R	-.227	.106	.014	.146	.092	.147	-.138	-.208	-.032	.057	.114	-.125	-.103	-.071	-.103
	p	.073	.409	.916	.254	.474	.252	.280	.102	.804	.655	.372	.331	.422	.579	.422
Years lived in the household [d]	R	-.030	-.012	-.093	.016	.044	.313	-.148	.057	.023	.087	.066	-.132	-.101	.077	-.078
	p	.817	.926	.470	.901	.733	.013	.247	.659	.859	.498	.607	.301	.431	.548	.544
Number of inhabitants [e]	R	.095	.054	.114	.102	.216	-.155	.141	-.018	.155	.046	.023	.012	-.205	.070	.123
	p	.461	.677	.373	.426	.089	.224	.271	.890	.224	.718	.857	.923	.107	.588	.336
Size of apartment (m^2) [f]	R	.389	.004	.127	.123	.166	-.186	.283	-.086	-.019	-.022	.002	.210	.051	.257	.190
	p	.002	.973	.319	.339	.194	.145	.025	.505	.881	.865	.989	.099	.694	.042	.135
Floor [g]	R	.049	-.042	.071	.025	-.014	-.067	.018	.057	-.048	-.341	-.147	.088	.239	.067	.007
	p	.700	.745	.581	.847	.916	.601	.888	.656	.708	.006	.252	.495	.059	.603	.958
Annual household Income (Yuan) [h]	R	.250	.003	-.051	-.107	.372	-.038	.222	.017	.133	-.076	.061	.093	.101	.218	.343
	p	.048	.978	.693	.404	.003	.765	.080	.892	.299	.554	.634	.470	.429	.085	.006
Dry sweep or vacuum cleaning/3 days [i]	R	.229	-.026	.037	.099	.023	.035	.284	.289	.118	-.100	.019	-.035	.168	.037	.011
	p	.071	.837	.773	.440	.858	.784	.024	.021	.356	.437	.880	.784	.189	.773	.934
Window opening time/ day [j]	R	.236	-.167	.186	.210	-.110	.023	.118	-.042	.011	-.076	.202	.221	-.013	-.013	-.105
	p	.063	.190	.144	.099	.389	.859	.357	.745	.929	.553	.113	.082	.918	.918	.415
Window opening time/ day [k]		.157	-.157	.083	.074	-.093	.036	.039	.032	-.039	.029	.034	.164	-.123	.025	-.270
		.219	.219	.518	.563	.466	.778	.763	.805	.763	.821	.794	.199	.335	.848	.032
Doors and windows remold (yes/no) [l]	R	.033	.149	.225	.098	.129	.119	.168	-.040	.087	.039	.073	.175	.300	.145	.197
	p	.796	.243	.076	.445	.313	.354	.189	.755	.496	.760	.568	.171	.017	.257	.121
Renovation of the house (yes/no) [m]	R	-.211	.147	-.118	-.190	-.104	.077	-.248	-.146	-.054	-.180	-.202	-.202	.135	-.021	-.290
	p	.097	.251	.358	.135	.419	.547	.050	.254	.672	.158	.112	.112	.291	.870	.021
Fresh air system (yes/no) [n]	R	-.070	.146	.046	-.034	.084	-.131	-.200	-.130	-.020	.001	.189	-.167	.048	-.094	.097
	p	.584	.254	.718	.790	.514	.305	.115	.310	.877	.995	.138	.191	.708	.463	.448
Distance to main road [o]	R	-.109	.140	-.114	.041	-.208	.061	-.071	.057	-.315	-.079	-.111	-.103	-.016	-.123	-.164
	p	.397	.273	.374	.753	.102	.636	.579	.658	.012	.540	.385	.422	.899	.336	.199

Values with orange background means significant correlations ($p < 0.05$). Values with gray background was $p < 0.10$.

Fifteen factors were evaluated in this study, including: [a] Temperature; [b] Humidity; [c] Building construction year (< 20 years; ≥20 years); [d] Years

lived in the household (<10 years; ≥10 years); [e] Number of inhabitants in the household (≤ 3; >3); [f] Size of the apartment in m^2 (≤ 73 m^2; >73 m^2); [g] Floor (≤ 4; >4); [h] Annual household Income (≤ 87000yuan; >87000 yuan); [i] Number of dry sweeping and vacuum cleaning per 3 days (≤ 2; >2); [j] The daily window opening time of the living room (≤1h; >1h); [k] The daily window opening time of the bedroom (≤2h; >2h); [l] Doors and windows remold (1: yes; 2: no); [m] Renovation of the house in the last 10 years (1: yes; 2: no); [n] Use of fresh air system (1: yes; 2: no); [o] Distance to main road (<50m; 50-200m; >200m).

Table S29. Spearman's rank correlations between blood concentrations of OPEs and frequencies of food consumption (n = 352).

		TEP	TnBP	TiBP	TEHP	TPHP	EHDPP	TCEP	TCIPP
Rice	R	-.009	-.032	.009	-.026	-.080	.039	.048	.061
	p	.861	.548	.861	.623	.132	.468	.367	.254
Cooked wheaten food	R	.002	.012	-.029	.034	.054	-.046	-.010	-.007
	p	.972	.827	.590	.522	.316	.390	.855	.892
Other staple food (corn, millet)	R	-.090	.007	-.008	.068	.010	.078	.030	.016
	p	.092	.898	.886	.202	.859	.147	.569	.765
Red meat (beef, pork, mutton)	R	-.077	.034	-.059	.049	-.081	.091	.023	.073
	p	.152	.527	.273	.356	.130	.089	.661	.169
White meat (chicken, duck)	R	.042	.005	.035	.021	.078	.026	-.011	.025
	p	.431	.932	.514	.688	.147	.623	.839	.640
Fish and marine product	R	.091	-.056	-.035	.074	.024	.046	-.052	.055
	p	.087	.291	.508	.168	.660	.390	.335	.302
Egg	R	-.017	.078	.089	.048	.026	.050	-.016	-.042
	p	.755	.142	.097	.374	.627	.346	.760	.437
Fresh vegetables	R	-.030	-.018	.025	.101	.009	.101	-.005	.084
	p	.580	.738	.638	.058	.866	.059	.923	.117
Bean products	R	-.027	-.064	-.018	-.018	.056	.047	**-.132***	-.066
	p	.618	.234	.738	.737	.296	.378	**.013**	.220
Fruits	R	-.051	-.038	-.051	**.112***	-.098	.089	.063	.044
	p	.338	.475	.338	**.036**	.067	.095	.240	.414
Dairy products (milk and yogurt)	R	-.092	.094	**.106***	.042	-.053	.072	.028	.062
	p	.085	.079	**.046**	.436	.325	.179	.596	.245
Nuts	R	-.075	.015	.009	.056	-.027	.102	.032	.010
	p	.163	.786	.868	.298	.609	.057	.544	.850
Pickled vegetable	R	-.047	**.117****	.060	-.031	-.072	.090	.086	**.177****
	p	.381	**.028**	.260	.567	.181	.092	.106	**.001**
Fried food	R	.065	-.025	.007	.006	-.063	.043	-.046	-.031
	p	.224	.642	.892	.907	.239	.417	.386	.557
Barbecue	R	.023	-.048	.009	-.065	-.038	-.037	.005	-.047
	p	.664	.370	.859	.223	.478	.483	.931	.380
Spicy food	R	-.007	.030	.079	-.014	-.043	.095	.018	.082
	p	.889	.576	.139	.789	.422	.074	.743	.126
Fresh fruit and vegetable juice	R	-.076	.040	.037	-.002	-.054	.050	.009	.014
	p	.153	.455	.485	.968	.312	.350	.862	.791
Juice	R	-.014	-.005	-.004	.021	-.028	-.017	.036	-.045
	p	.798	.932	.945	.693	.596	.748	.505	.395
Coffee	R	.007	-.023	-.017	.050	.010	.070	-.032	-.033
	p	.889	.668	.757	.348	.851	.187	.551	.537
Soda	R	.042	**-.111***	-.100	-.093	-.063	-.012	.032	-.052
	p	.433	**.037**	.060	.083	.238	.816	.551	.331
Other drink	R	.061	.021	.040	-.026	-.007	-.069	.028	-.041
	p	.256	.688	.450	.628	.892	.197	.605	.446
Liquor	R	.034	-.095	-.065	-.096	.008	.004	-.058	.019
	p	.526	.074	.227	.072	.888	.946	.280	.727
Water	R	.089	**-.105****	-.098	-.104	.047	**-.135***	-.088	**-.142****
	p	.094	**.048**	.067	.052	.384	**.011**	.101	**.007**

Tea	R	-.009	.002	.037	-.013	-.003	.057	.047	.010
	p	.864	.973	.489	.811	.955	.282	.380	.859
Tea-3 days	R	-.016	.021	.032	.039	.003	.054	-.023	-.032
	p	.770	.700	.552	.466	.952	.316	.666	.546
Additional diet-3 days	R	**-.112***	-.019	.006	.061	-.079	.040	.003	.071
	p	**.036**	.723	.910	.253	.137	.450	.958	.186
Water-3 days	R	**.177****	-.068	**-.117***	-.022	-.018	**-.129***	-.025	-.008
	p	**.001**	.200	**.028**	.687	.739	**.016**	.646	.884

Value with gray background means $p < 0.20$; Value with orange background means $p < 0.05$. Food intake frequencies were treated as rank variables and assigned increasing numbers, which were displayed in Table S9.

Table S30. Spearman's rank correlations between urinary concentrations of di-OPEs and frequencies of food consumption (n = 352).

		DPHP	BDCIPP	DBP	BMPP	BEHP
Rice	R	.095	.076	.008	.092	.024
	p	.074	.157	.881	.086	.651
Cooked wheaten food	R	-.093	-.010	-.029	-.083	.048
	p	.082	.852	.587	.120	.373
Other staple food (corn, millet)	R	-.067	.024	-.103	.052	.009
	p	.208	.651	.054	.330	.868
Red meat (beef, pork, mutton)	R	**-.111***	-.027	-.056	-.102	-.032
	p	**.037**	.609	.291	.057	.545
White meat (chicken, duck)	R	-.019	-.022	.005	-.031	.065
	p	.717	.687	.919	.558	.223
Fish and marine product	R	-.039	.047	.091	.051	.035
	p	.464	.379	.089	.339	.518
Egg	R	-.046	-.010	.015	-.064	.008
	p	.385	.856	.773	.234	.880
Fresh vegetables	R	-.063	-.018	-.082	-.032	-.002
	p	.239	.734	.127	.545	.973
Bean products	R	-.058	.020	.010	**-.175****	.074
	p	.274	.704	.854	**.001**	.165
Fruits	R	-.016	.079	-.057	.022	-.025
	p	.762	.141	.283	.675	.635
Dairy products (milk and yogurt)	R	-.098	-.048	-.060	-.036	-.001
	p	.065	.370	.262	.499	.980
Nuts	R	**-.118***	.019	-.018	-.072	**.129***
	p	**.027**	.718	.734	.180	**.015**
Pickled vegetable	R	.023	.052	-.056	-.037	-.019
	p	.665	.330	.291	.487	.724
Fried food	R	-.021	-.054	-.030	.020	-.023
	p	.693	.315	.570	.705	.661
Barbecue	R	-.027	-.021	.060	-.037	-.031
	p	.608	.689	.260	.494	.558
Spicy food	R	-.009	-.039	.011	-.074	.018
	p	.860	.461	.835	.167	.733
Fresh fruit and vegetable juice	R	-.021	-.096	**.114***	-.026	-.003
	p	.700	.072	**.032**	.621	.955
Juice	R	-.028	-.066	.052	-.023	.078
	p	.595	.215	.331	.672	.145
Coffee	R	-.082	.101	.009	-.015	-.017
	p	.123	.059	.873	.777	.752
Soda	R	.088	-.104	.028	-.043	**-.120***
	p	.098	.051	.596	.423	**.025**
Other drink	R	.026	-.016	.027	-.057	-.026
	p	.626	.760	.615	.282	.625
Liquor	R	.011	.002	-.014	.039	.004
	p	.837	.966	.797	.462	.946
Water	R	-.087	**-.121***	.036	-.083	.009

	p	.104	**.024**	.503	.119	.869
Tea	R	**-.157****	-.020	.017	.004	.043
	p	**.003**	.710	.756	.934	.419
Tea-3 days	R	**-.135***	-.078	-.068	-.001	.066
	p	**.011**	.146	.204	.978	.216
Additional diet-3 days	R	**-.134***	.030	-.092	-.032	.024
	p	**.012**	.577	.084	.555	.650
Water-3 days	R	-.011	-.017	.103	-.041	.008
	p	.831	.753	.053	.440	.876

Value with gray background means $p < 0.20$; Value with orange background means $p < 0.05$. Food intake frequencies were treated as rank variables and assigned increasing numbers, which were displayed in Table S9.

Table S31. Adjusted linear regression associations between the consumption frequencies of different food and measured levels of OPEs in whole blood or di-OPEs in urine.

Compound	Diet	Frequencies	10^{β}	95% CI	Sig
TEP	Other diet-3 days	No	1		
		Yes	1.51	1.02, 2.23	0.037
	Water-3 days	<1.3 L/day	1		
		1.3-1.8 L/day	1.92	1.19, 3.11	0.008
		>1.8 L/day	1.85	1.11, 3.09	0.018
TCEP	Bean products	<3 days/month	1		
		1-3 days/week	1.60	1.16, 2.20	0.004
		>4 days/week	1.70	1.23, 2.35	0.001
	Water	< 6 cups/day	1		
		6-9 cups/day	0.692	0.538, 0.891	0.004
		>9 cups/day	0.774	0.595, 1.01	0.056
TCIPP	Water	< 6 cups/day	1		
		6-9 cups/day	0.638	0.459, 0.886	0.008
		>9 cups/day	0.687	0.487, 0.969	0.033
EHDPP	Water	< 6 cups/day	1		
		6-9 cups/day	0.891	0.598, 1.33	0.567
		>9 cups/day	0.622	0.403, 0.959	0.032
TEHP	Alcohol drinking	No	1		
		Yes	1.43	1.02, 2.00	0.041
DPHP	Other diet-3 days	No	1		
		Yes	1.30	1.07, 1.58	0.008
	Dairy products	<3 days/month	1		
		1-6 days/week	0.98	0.77, 1.24	0.869
		Everyday	1.27	1.01, 1.59	0.044
	Tea	Never	1		
		<30 days/month	0.94	0.72, 1.22	0.626
		Everyday	1.43	1.08, 1.89	0.013
	Water	< 6 cups/day	1		
		6-9 cups/day	0.843	0.674, 1.05	0.133
		>9 cups/day	0.781	0.622, 0.979	0.032
BDCIPP	Rice	<3 days/month	1		
		1-3 days/week	0.594	0.439, 0.804	<0.001
		>4 days/week	0.536	0.396, 0.725	0.001
	Water	< 6 cups/day	1		
		6-9 cups/day	0.886	0.688, 1.14	0.349
		>9 cups/day	0.753	0.582, 0.974	0.031
DBP	Fresh fruit and vegetable juice	Never	1		
		>once/month	1.57	1.06, 2.33	0.025
	Other diet-3 days	No	1		
		Yes	1.40	1.10, 1.77	0.005
	Other staple food	<3 days/month	1		
		1-6 days/week	1.55	1.02, 2.37	0.008
		Everyday	1.65	1.09, 2.48	0.039
BEHP	Fresh fruit and vegetable juice	Never	1		
		>once/month	0.605	0.394, 0.929	0.022
	Soda	Never	1		
		>once/month	2.29	1.21, 4.37	0.012
	Nut	<3 days/month	1		
		1-3 days/week	0.807	0.626, 1.04	0.097
		>4 days/week	0.731	0.570, 0.938	0.014

Only the significant associations were displayed in the table.

Table S32. Estimated daily intakes (ng/kg bw/day) of OPEs calculated from OPE concentrations in environmental matrices, OPE concentrations in whole blood, and di-OPE levels in urine.

	EDI_{Inh}		EDI_{ing}		EDI_{der}		$EDI_{external}$		RfD[a]
	range	median	range	median	range	median	range	median	
TMP	0.006-0.394	0.024	-[c]	-	-	-	0.008-0.395	0.027	-
TEP	0.107-13.0	1.41	0.011-1.19	0.068	0.005-0.444	0.027	0.157-13.4	1.78	125000
TPrP	-	-	0.002-0.037	0.011	0.001-0.017	0.005	0.003-0.055	0.017	-
TnBP	0.012-0.382	0.068	0.005-2.56	0.013	0.002-1.14	0.005	0.026-4.00	0.092	2400
TiBP	0.023-0.547	0.126	0.004-3.74	0.020	0.002-1.67	0.008	0.042-5.65	0.160	-
TBOEP	0.001-0.011	0.002	0.009-0.407	0.053	0.004-0.169	0.020	0.015-0.560	0.072	1500
TEHP	0.010-0.082	0.028	0.020-0.692	0.049	0.008-0.257	0.019	0.055-0.972	0.106	35000
TPHP	0.005-0.955	0.018	0.063-1.46	0.287	0.028-0.651	0.111	0.102-2.11	0.415	7000
EHDPP	0.006-0.064	0.019	0.059-1.83	0.199	0.026-0.680	0.084	0.099-2.54	0.301	600
CDPP	-	-	0.005-0.369	0.030	0.002-0.165	0.012	0.008-0.551	0.044	-
TMPP	0.001-0.015	0.002	0.003-0.707	0.020	0.002-0.263	0.009	0.008-0.973	0.031	1300
BABP	0.001-0.050	0.006	0.002-1.10	0.017	0.001-0.407	0.007	0.007-1.50	0.036	-
RDP	0.000-0.156	0.012	-	-	-	-	0.00008-0.233	0.022	-
TCIPP	0.093-11.9	0.719	0.173-8.16	0.675	0.079-3.45	0.320	0.390-19.8	1.75	3600
TCEP	0.044-3.05	0.225	0.189-16.4	0.764	0.105-9.36	0.404	0.453-28.9	1.63	2200
TDCIPP	0.001-0.102	0.013	0.031-4.75	0.171	0.008-1.26	0.044	0.045-6.04	0.219	1500[b]
$\sum_{16}$OPEs	0.369-22.9	3.92	1.00-25.5	3.10	0.444-11.4	1.31	2.78-42.0	7.58	-

	EDI_{blood}	
	range	median
TnBP	23.9-934	113
TiBP	36.3-1029	168
TEHP	14.2-256	35.1
TPHP	9.12-682	249
EHDPP	2.19-305	25.4
TCIPP	3.47-156	46.4
TCEP	3.88-60.6	14.0

	EDI_{urine}			
	f = 0.1		**f = 0.9**	
	range	median	range	median
TBP	5.55-84.4	25.2	0.62-9.38	2.80
TEHP	7.39-138	34.0	0.82-15.3	3.77
TPHP	4.58-399	26.4	0.51-44.4	2.94
TMPP	3.41-2170	3.96	0.10-2.47	0.44
TDCIPP	5.50-280	29.6	0.61-31.1	3.29

[a] Reference does, obtained from Zhao et al.;[2]

[b] The RfD of TDCIPP was obtained from He et al.;[70]

[c] The value was not calculated due to the low DFs of compounds.

Table S33. Estimated daily intakes (ng/kg bw/day) of di-OPEs via dust ingestion and their contributions to urinary levels

Chemicals	EDI_{dust-m}		Contribution (%) [a]	
	range	median	range	median
DBP	0.008-0.406	0.047	0.23-37.5	2.34
BEHP	0.062-2.44	0.306	1.09-107	12.5
BBOEP	0.0003-0.172	0.009	0.002-48.2	2.42
DPHP	0.003-0.175	0.020	0.02-13.5	1.01
BMPP	0.001-0.026	0.001	0.08-6.15	0.49
BDCIPP	0.0004-0.030	0.003	0.003-4.88	0.12
BCEP	-[b]	-	-	-
BCIPP	-	-	-	-

[a] The contribution of human direct exposure to di-OPEs via dust ingestion to their urinary levels;
[b] The value was not calculated due to the low DFs of compounds.

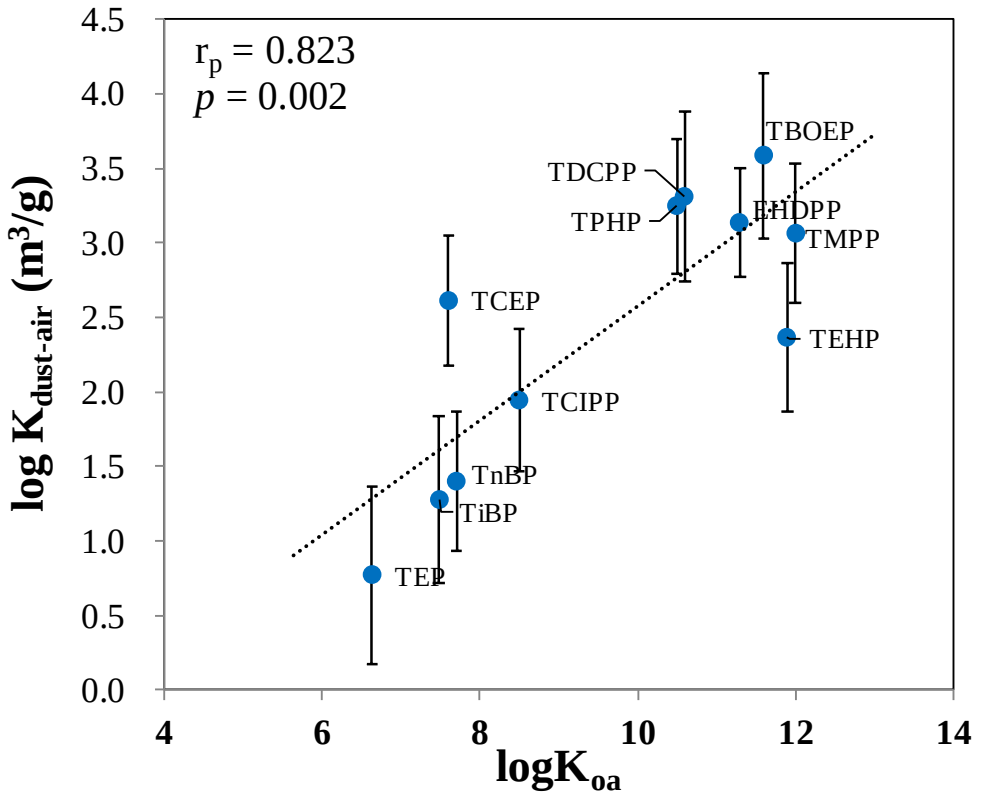


Figure S1. Pearson correlation between the median log $K_{dust-air}$ (log C_{dust}/C_{air}) and $logK_{oa}$ value of OPEs. Blue points represent median values; error bars represent standard deviations.

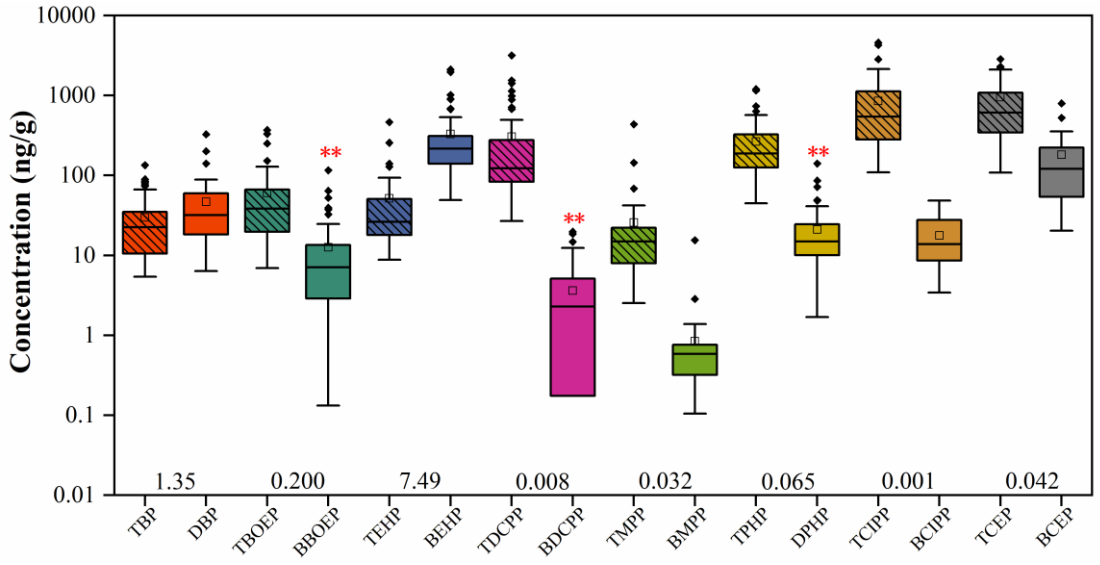


Figure S2. Comparisons of the concentrations (ng/g) of OPEs and di-OPEs in indoor dust. Hatched boxes represent OPE data. Horizontal lines on the box plots represent the 25th, 50th and 75th percentiles; the whiskers represent ± 1.5 interquartile range (IQR); and dots represent outliers. Asterisks represent significant correlations between OPEs and their corresponding di-OPEs. The number above the abscissa represent the median concentration ratios of di-OPEs/OPEs. It should be noted that the detection frequencies of BCIPP and BCEP were 37% and 37%, respectively. ***p*<0.01 for the correlation analyses.

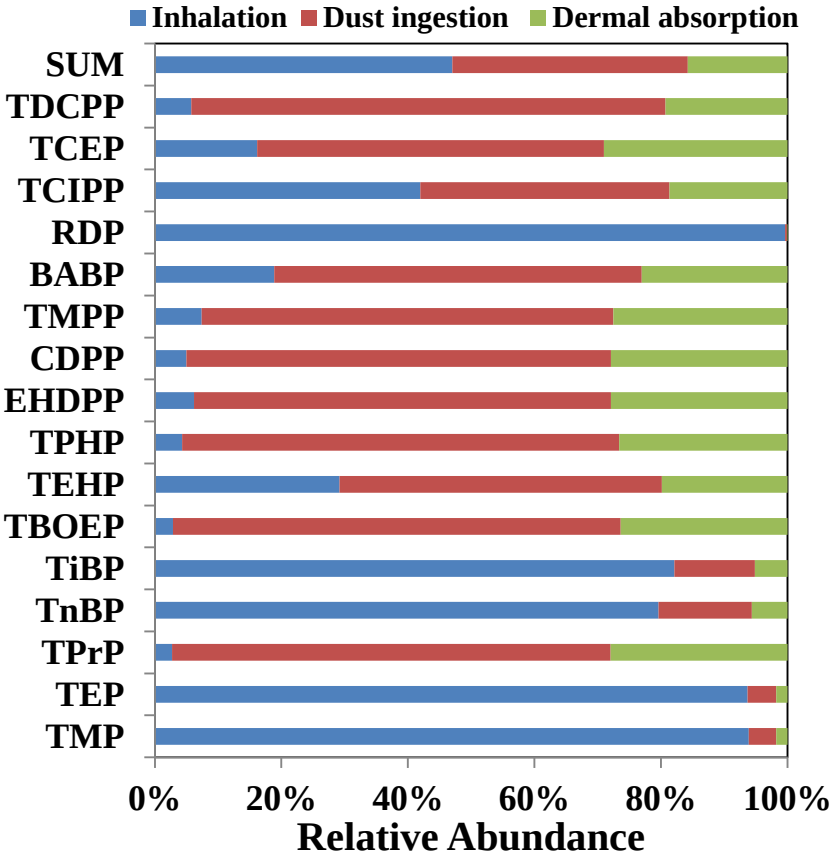


Figure S3. Comparison of EDIs (ng/kg bw/day) of OPEs *via* different exposure pathways, including inhalation, dust ingestion, and dermal absorption from dust.

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