

## Household dust: loadings and PM<sub>10</sub>-bound plasticizers and polycyclic aromatic hydrocarbons

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**Abstract:** Residential dust is recognized as a major source of environmental contaminants, including polycyclic aromatic hydrocarbons (PAHs) and phthalic acid esters (PAEs). To characterize the thoracic dust fraction (PM<sub>10</sub>), a sampling campaign was carried out with an in-situ resuspension chamber in three rooms (kitchen, living room and bedroom) of four Spanish houses. Two samples per room were collected with, at least, a one-week interval. The PM<sub>10</sub> samples were analyzed for their carbonaceous content by a thermo-optical technique and, after solvent extraction, for 20 PAHs, 8 phthalate plasticizers (PAEs) and one non-phthalate plasticizer (DEHA) by gas chromatography-mass spectrometry. In general, higher dust loads were observed for parquet flooring compared with tile. The highest dust loads were obtained for rugs. Total carbon accounted for 9.3 to 51%wt of the PM<sub>10</sub> mass. Plasticizer mass fractions varied from 5 µg g<sup>-1</sup> to 17 mg g<sup>-1</sup> PM<sub>10</sub>, whereas lower contributions were registered for PAHs (0.98–116 µg g<sup>-1</sup>). The plasticizer and PAH daily intakes for children and adults via dust ingestion were estimated to be 3–4 orders of magnitude higher than those via inhalation and dermal contact. Potential carcinogenic and negligible non-carcinogenic risks arising from exposure to PAHs were found.

**Keywords:** resuspension; household dust; PM<sub>10</sub>; organic and elemental carbon; phthalic acid esters; polycyclic aromatic hydrocarbons

## 1. Introduction

In industrialized nations, people spend most of their time in closed environments, especially at home [1]. Walking induced particle resuspension has been reported to be an important source of indoor particulate matter [2–4]. Several factors affect resuspension of particles, including relative humidity, flooring type and dust loadings [3]. Household dust is a complex mixture of particles of both indoor and outdoor origin, including organic, inorganic and biological components, many of which are toxicants, carcinogens and/or allergens [5,6]. Its composition depends on numerous conditions, such as environmental and seasonal factors, ventilation and air filtration, homeowner activities, and in- and outdoor sources [7]. Several studies have shown that inhalation of dust particles is linked to an increased risk of a range of health hazards, spanning from asthma symptoms in susceptible adults and children [8–11] to cancer and fertility problems [12].

Residential dust is recognized as a major source of environmental contaminants, including polycyclic aromatic hydrocarbons (PAHs) and phthalic acid esters (PAEs) [13–15]. PAHs are primarily byproducts of incomplete combustion of fossil fuels and biomass and pyrosynthesis of organic materials [16]. Dust, especially in the carpeted floor, can be a permanent reservoir for these chemicals, which may be inhaled through resuspension into air, ingested accidentally by children or absorbed through the skin [13,17,18]. Because of their widespread sources and strong carcinogenicity, cytotoxicity, mutagenicity, endocrine disrupting and other hazardous properties, PAHs have been the focus of extensive attention by scientists and governmental organizations [7,18–24]. Benzo[a]pyrene (BaP), the most extensively studied carcinogenic PAH, is classified by the International Agency for Research on Cancer (IARC) as a Group 1 or known human carcinogen [25]. Four of the top ten priority pollutants, nominated by the Agency for Toxic Substances and Disease Registry (ATSDR) in 2011, are single PAHs or PAH mixtures (PAHs, BaP, benzo[b]fluoranthene, and dibenzo[a,h]anthracene) [26].

PAEs (phthalates) are used as plasticizers in several consumer products, commodities, and building materials [27]. Therefore, phthalates are ubiquitous in residential and occupational environments, where they are present in high concentrations, both in air and in dust [14,27–41]. Comparisons of mass fractions reported in different studies indicate that, for measurements conducted over the past decade, the levels of phthalates in indoor dust tend to be 3–5 orders of magnitude higher than those of PAHs [14]. Recent toxicological studies have proven the potential of phthalates to disturb the human hormonal system and human sexual development and reproduction [42,43]. Moreover, phthalates are suspected to trigger asthma and dermal diseases in children [44; and references therein]. An EU risk assessment classified bis(2-ethylhexyl)phthalate (DEHP), dibutyl phthalate (DBP), and benzyl butyl-phthalate (BBP) as hazardous substances in 2005, and has issued a Directive to ban these materials from products, particularly toys and cosmetics [45].

Indoor aerosol sources may significantly contribute to the daily dose of particles deposited into the human respiratory system [46]. Since indoor dust contributes to human exposure, its resuspension rates and chemical composition should be evaluated. The selection of appropriate techniques to assess household dust loadings and composition is a major challenge since several methodologies have been

employed, including passive (dust settling) and active techniques (surface wiping, press sampling, sweeping, or vacuuming) [6,47].

Studies on the concentration of contaminants in household dust have focused on the analysis of the total mass or of sieved fractions, where the smallest particle size obtained is generally 38  $\mu\text{m}$  [48]. Considering the possibility that household dust is resuspendable and can become airborne, most methodologies have drawbacks when assessing human inhalation exposure. Such an approach requires measurements of the contaminant concentration in smaller particle sizes. The main goal of the present study was to determine household dust loadings and the respective PAH and plasticizer levels in the thoracic fraction ( $< 10 \mu\text{m}$ ) of resuspendable material on the floor, which deposit anywhere within the lung airways. The in-situ resuspension chamber was previously devised and successfully applied to collect the deposited  $\text{PM}_{10}$  fraction from road pavements [49], but it was the first time this active sampling methodology was used to collect settled thoracic particles directly from the floor.

## 2. Methodologies

To determine and characterize dust loadings, a sampling campaign was carried out in four different houses located in the Spanish city of León (Table 1). In each housing unit, three rooms were investigated, including the kitchen, the living room and a bedroom. In each room, two samples were collected with, at least, a one-week interval. Samples in each house were performed one or two days before weekly cleaning. For dust collection, an in-situ resuspension chamber operating at an air flow rate of  $25 \text{ L min}^{-1}$  was used [49]. After vacuuming,  $\text{PM}_{10}$  was separated from the total dust through a Negretti stainless steel elutriation filter and collected onto 47 mm quartz fiber filters (Pallflex®), while particles with aerodynamic diameter  $> 10 \mu\text{m}$  were deposited in the methacrylate chamber and along the elutriation filter. Sampling was performed in surface areas of  $1 \text{ m}^2$  for 30 minutes. Two to three different square meters were sampled using the same filter in order to ensure enough particulate mass for the subsequent gravimetric and chemical analyzes. Since some compounds may derive from the resuspension chamber itself or from ambient air that enters the system and passes through the filter during dust collection from the floor, air samples were vacuumed through the same system after sampling in each room. Background air filters were sampled for 30 minutes, as were  $\text{PM}_{10}$  floor dust samples.

**Table 1.** Characteristics of houses where floor dust ( $\text{PM}_{10}$ ) was sampled.

House	Characteristics	Room	Flooring
1		Kitchen	Tile

	Suburban two-story house with well-ventilated kitchen. Two occupants.	Bedroom Living room Living room rug Bedroom rug	Parquet Parquet Cut pile carpet/rug Long threads shag rug
2	Single story apartment located in the city center. Two occupants.	Kitchen Bedroom Living room	Tile Parquet Parquet
3	Rural two-story house with open fireplace in the living room. Two occupants.	Kitchen Bedroom Living room	Tile Parquet Tile
4	Single story apartment with small kitchen open to the living room. One occupant.	Kitchen Bedroom Living room	Tile Tile Tile

Due to the loss of a small filter fragment, the kitchen sample from house 2 obtained in the 2<sup>nd</sup> week of sampling was discarded. After gravimetric determination, two punches (9 mm) of each filter were analyzed by a thermo-optical transmission technique to obtain the PM<sub>10</sub> carbonaceous content (organic and elemental carbon, OC and EC). This method is based on the quantification of the CO<sub>2</sub> released from the volatilization and oxidation of different carbon fractions under controlled heating by a non-dispersive infrared (NDIR) analyzer. The blackening of the filter is monitored using a laser beam and a photodetector, which enables separating the EC formed by pyrolysis [50]. The remaining portion of each filter was extracted by sonication for 15 minutes with three aliquots (25 mL each) of dichloromethane. After filtration, the solvent was concentrated in a TurboVap system from Biotage and evaporated to dryness by a gentle nitrogen stream. All the extracts were analyzed by gas chromatography-mass spectrometry (GC-MS) in a Shimadzu QP5050A equipped with a TRB-5MS 30 m × 0.25 mm × 0.25 μm column. The quantitative analysis was performed by single ion monitoring (SIM). Background air filters were analyzed in the same way as the samples to obtain blank-corrected results. Data were acquired in the electron impact (EI) mode (70 eV). The oven temperature program was as follows: 60 °C (1 min); 60–150 °C (10 °C min<sup>-1</sup>), 150–290 °C (5 °C min<sup>-1</sup>), 290 °C (30 min) and using helium as carrier gas at 1.2 mL min<sup>-1</sup>. For the quantification of PAHs, a mixture of deuterated internal standards (IS) was used: 1,4-dichlorobenzene-d4, naphthalene-d8, acenaphthene-d10, phenanthrene-d10, chrysene-d12 and perylene-d12 (Supelco). In the case of plasticizers, deuterated diethyl phthalate-3,4,5,6-d4 and bis(2-ethylhexyl)phthalate-3,4,5,6-d4 (Supelco) were used as IS. Calibrations were performed with authentic standards (Sigma-Aldrich) in eight different concentration levels.

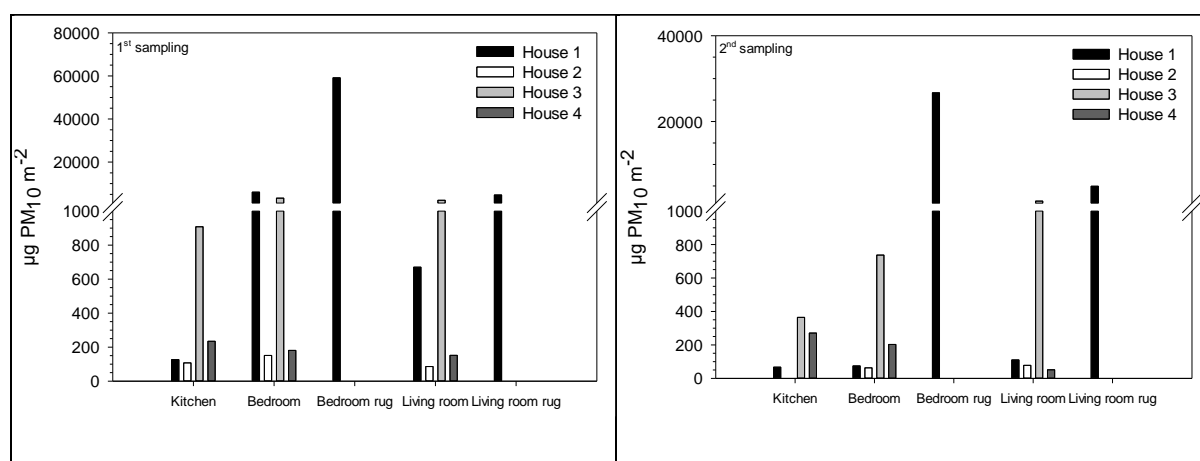
To assess recoveries during extraction, pre-baked blank filters were spiked with known amounts of standards, covering the concentration range commonly reported in the literature for household dust. Six pre-baked blank filters were also extracted and analyzed. The filters were subjected to the same methodology of extraction and analysis used for the samples. Four distinct concentrations were tested, in triplicate, and each extract was injected 3 times. The following overall recoveries (%) were obtained: diethyl phthalate  $38.4\pm 8.05$ , di-n-butyl phthalate  $113\pm 17.4$ , benzyl butyl phthalate  $70.2\pm 12.7$ , bis(2-ethylhexyl) adipate  $70.4\pm 12.9$ , bis(2-ethylhexyl) phthalate  $108\pm 10.9$ , phenanthrene  $53.0\pm 10.6$ , anthracene  $67.1\pm 11.5$ , fluoranthene  $84.7\pm 11.2$ , pyrene  $81.9\pm 11.0$ , benzo[a]anthracene  $97.2\pm 11.4$ , chrysene  $81.9\pm 9.7$ , benzo[b]fluoranthene  $106\pm 10.0$ , benzo[k]fluoranthene  $108\pm 10.6$ , benzo[a]pyrene  $93.4\pm 13.3$ , indeno[1,2,3-cd]pyrene  $90.6\pm 15.8$ , dibenzo[a,h]anthracene  $87.2\pm 16.7$ , and benzo[g,h,i]perylene  $84.7\pm 16.5$ . The concentrations shown throughout the manuscript were not adjusted with these percent recoveries. Due to its high volatility and concentration variability in both dust and background air samples, naphthalene was excluded from quantification.

### 3. Results and Discussion

#### 3.1. Dust loadings

Huge differences in dust loadings between rugs and hard floorings were registered (Figure 1). Rugs represented the surface with highest dust loadings. In general, higher dust loadings were observed for parquet flooring compared with tile. Among the four dwellings, the highest dust loadings were obtained in the living room of a suburban family house with an open fireplace. Several factors can affect the surface dust loadings, such as walking, cleaning frequency, household materials and indoor particle sources. Thus, the presence or absence of occupants can have a marked effect on resuspension levels. In house 1, for example, a very significant decrease in dust loadings from 1<sup>st</sup> to 2<sup>nd</sup> week were registered, possibly due to the absence of owners for a few days. Using a mechanical resuspension device, Tian et al. [3] characterized walking-induced particle resuspension as a function of flooring type. Results showed that for particles at  $0.4\text{--}3.0\ \mu\text{m}$ , the difference in resuspension fraction between carpets and hard floorings was not significant. It was also found that for fine particles ( $0.4\text{--}3.0\ \mu\text{m}$ ), the difference in resuspension caused by flooring type is negligible, while for coarse particles ( $3.0\text{--}10\ \mu\text{m}$ ) carpets are associated with 2–4 times higher resuspended concentration in comparison with hard floorings. In fact, carpeted floors may contribute to significantly higher surface dust loadings and allergen concentrations than hard floors [51–55]. Roberts et al. [56] used a high-volume surface sampler to measure surface dust in carpets. Dust loadings ranged from  $0.32$  to  $14.4\ \text{g m}^{-2}$ . Adgate et al. [57] collected bare floor and carpet dust samples in 216 Jersey City, New Jersey, homes using quantitative wipe and vacuum sampling techniques. Dust loadings varied from  $0.05$  and  $7.0\ \text{g m}^{-2}$  and from  $0.3$  and  $99\ \text{g m}^{-2}$  in the

wipe floor and vacuum samples, respectively. Indoor settled harmattan dusts at seven sampling stations from the University of Agriculture Makurdi (Nigeria) were collected for five months using the wipe sampling methodology [58]. Dust loadings within the institution were in the range from 4.5 to 6.5 g m<sup>-2</sup>. Masses of indoor settled dust were expectedly higher along high traffic, untarred roads and construction sites, as well as in older buildings. It should be borne in mind that dissimilar indoor dust sampling strategies (e.g., wipe versus vacuum methods) are used to measure loadings and amounts of toxicants per unit area, which renders comparisons between studies difficult. Liroy et al. [59] found that while loadings were substantially greater with wipe sampling, metal concentrations within the dust samples were similar for both methods of sampling. Vacuum cleaner sampling has its own series of problems, especially the variability in design and efficiency, and likely will not retain particles below 10 µm [47]. Bai et al. [60] evaluated five methods of sampling lead-contaminated dust on carpets: (i) wipe, (ii) adhesive label, (iii) C18 sheet, (iv) vacuum, and (v) hand rinse. The wipe and vacuum methods showed the best reproducibility and correlation with other sampling techniques. The authors concluded that surface wipe sampling was the best method to measure accessible lead from carpets for exposure assessment, while vacuum sampling was most effective for providing information on total lead accumulation (long-term concentrations). In their review paper, Liroy et al. [6] state that although we have come a long way in determining the uses of house dust to identify sources of indoor contamination and to provide improved estimates of residential human exposure, one of the challenges still lies in the reliability of sampling techniques.

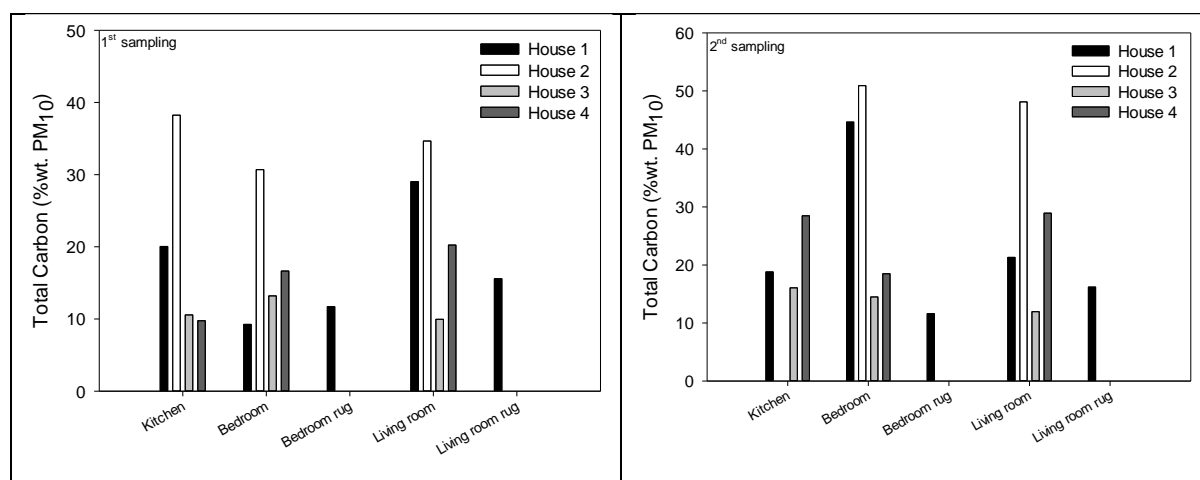


**Figure 1.** Dust loadings in the different rooms of the various houses for the two sampling campaigns.

### 3.2. Carbonaceous, plasticizer and PAH particulate mass fractions

Total carbon accounted for 9.3 to 51% wt of PM<sub>10</sub> with the highest mass fractions recorded in dust samples collected in the city center apartment (Figure 2). An overwhelming proportion, always higher than 80%, of the total carbonaceous matter was composed of OC, whereas in many samples the EC was

too low or undetectable. The highest percentages of EC (10 to 20% of TC) were observed in kitchens, where there are sources of combustion.



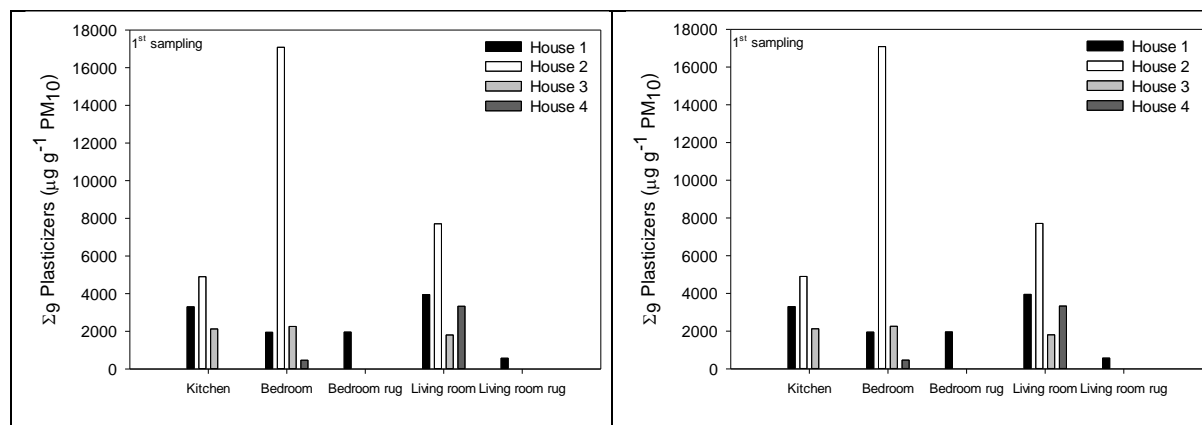
**Figure 2.** Total carbon (TC = OC + EC) mass fraction in resuspendable PM<sub>10</sub> from household dust.

Eight phthalate plasticizers (PAEs) and one non-phthalate plasticizer [bis(2-ethylhexyl) adipate, DEHA] were quantified in PM<sub>10</sub> from settled house dust. PAEs included dimethyl phthalate (DMP), dimethylpropyl phthalate (DMPP), diethyl phthalate (DEP), diisobutyl phthalate (DIBP), di-n-butyl phthalate (DBP), di-n-hexylphthalate (DNHP), benzyl butyl phthalate (BBP), dicyclohexyl phthalate (DCHP), bis(2-ethylhexyl) phthalate (DEHP), di-n-octyl phthalate (DNOP), di-isononylphthalate (DINP) and di-isodecylphthalate (DIDP). The total mass fractions varied from 5  $\mu\text{g g}^{-1}$  to 17  $\text{mg g}^{-1}$  PM<sub>10</sub> (Figure 3). As observed with dust loads, huge differences were observed from week to week and from home to home. The highest values were registered in the bedroom of the city center apartment and in the living room of the rural house. In addition to variability in sources, the concentrations of these compounds depend on the same factors already mentioned for the dust loads (e.g. ventilation, domestic routines, cleaning activities). Widely scattered concentration levels were also documented in many previous works. For example, Kubwabo et al. [33] analyzed 17 phthalate esters in 126 Canadian household dust samples, evidencing the huge variability in spatial and temporal distribution of these compounds across different areas of the home and, thus, the difficulty in predicting potential household exposures.

Most likely due to its high volatility, DMP was the compound with the lowest mass fractions (Table 2). On the other hand, DEHP, DNOP and DBP were the major phthalates in household dust. While high DBP values were observed in all parquet floor bedrooms, only samples from two living rooms showed detectable masses. Bamai et al. [61] also associated higher DBP levels in floor dust with compressed wooden floor. This type of flooring is usually composed of thin pieces, which are glued together and covered with wax, paint, and sometimes flame retardants. The surface applied products (gloss agents, plastic additives, paint, and varnish) contain DBP [61]. From quantitative and qualitative emission data



on phthalates from different materials, Afshari et al. [62] reported that polyolefin covered with wax for floor polishing increased DBP concentration in chamber air by two-fold. DBP is also employed as a coalescing aid in latex adhesives, as well as a plasticizer in cellulose plastics and a solvent for dyes [61]. Furthermore, DBP has been reported to be largely present in cosmetic and personal care products [63].



**Figure 3.** Plasticizer mass fractions in PM<sub>10</sub> from resuspended household dust.

Despite the concentration of DNOP in floor dust has been reported in only a very limited number of publications, the mass fractions of the present study are higher than those described in the literature. There were no appreciable differences between the amounts found in PM<sub>10</sub> of the various rooms of the houses. DNOP is used in carpet back coating, floor tile, and adhesives. It is also employed in cosmetics and pesticides. DEHP was present at higher concentrations in the bedroom and living room samples. Although DEHP has been consistently described as one of the most abundant phthalates in settled dust, the levels have decreased over time, reflecting its phase-out in the EU. In Europe, the use of DEHP decreased drastically in 2001 and has to a large extent been replaced by DINP and DIDP, with their longer chains and lower volatility [64]. DEHP has been used in numerous consumer products, children toys, medical devices and building materials (e.g., vinyl flooring, furniture, paints, cables, wires, wall coverings, packaging materials) [65]. Bamai et al. [61] and Bornehag et al. [34] associated polyvinyl chloride (PVC) flooring with DEHP levels in house dust. Kolarik et al. [66] documented higher concentration of BBP, DNOP and DEHP in indoor dust in homes where polishing agents were employed compared to homes where such products were infrequently used or not at all. As PVC flooring was not present in any of the houses of the present study, other emission sources may have contributed to the DEHP levels. It has been shown that source characteristics (surface area and material phase concentration of DEHP), as well as the external mass-transfer coefficient and ventilation rate, are important variables that influence the steady-state DEHP concentration and the resulting exposure [67]. DEHP and other phthalates are strongly sorbed to surfaces. A relatively small gas-phase concentration, such as 0.1 ppb, is enough for significant vapor transport of a PAE and its subsequent partitioning between the gas phase and indoor surfaces, including airborne particles and settled dust [68].



BBP was present in all samples at relatively close concentrations regardless of room type. The values fall within the range reported for household dust from other regions. BBP is commonly employed as a plasticizer for vinyl foams, which are often used as floor tiles. Other uses are in artificial leather, paints and adhesives. BBP was classified as toxic by the European Chemical Bureau and therefore its use has decayed rapidly in the last decade.

No appreciable differences were found between DINP concentrations in samples from the various rooms of the houses. DIDP, instead, was present in higher amounts in living rooms and at lower levels in kitchens. The values observed in the present study for these two compounds seem to be lower than those documented by Santillo et al. [69] for dust samples of houses in several European cities, although the ranges reported by these researchers are very wide. Based on risk assessment, in 2013, the European Chemicals Agency (ECHA) concluded that there is no evidence that would justify a re-examination of the existing restriction on DINP and DIDP in toys and childcare articles which can be placed in the mouth by children [70]. About 95% of DINP is used in PVC applications. The other 5% is employed in non-PVC applications such as rubbers, adhesives, sealants, paints and lacquers and lubricants. For DIDP, non-PVC applications are comparatively small, but comprise use in anti-corrosion and anti-fouling paints, sealing compounds and textile inks [70; and references therein].

**Table 2.** Comparison of mass fractions of plasticizers ( $\mu\text{g g}^{-1}$ ) in house dust.

Sampling information	Statistics	DMP	DMPP	DEP	DIBP	DBP	DNHP	BBP	DEHA	DCHP	DEHP	DNOP	DINP	DIDP	Ref.
Kitchens	Mean	0.27		142		562		6.3	17		271	927	10.2	6.6	This study
	Median	< 0.1		< 0.1		< 0.1		6.4	< 0.1		< 0.1	407	9.0	< 0.1	
Bedrooms	Mean	0.33		45		2353		10.4	36		897	969	19.9	35.3	This study
	Median	0.19		< 0.1		964		7.0	7.4		442	316	7.3	3.9	
Living rooms	Mean	0.93		139		1181		11.1	19		1352	751	28.2	66.4	This study
	Median	< 0.1		16		< 0.1		9.3	< 0.1		218	473	8.2	16.6	
Living room rug	Mean	0.60		21		893		8.54	23.4		1997	807	9.8	23.8	This study
Bedroom rug	Mean	1.31		5.8		139		3.44	3.7		92	119	5.3	9.1	This study
Dust samples from houses in several regions of China collected with brushes by gentle sweep	Mean	0.693		0.187	17.1	26.4	0.003	0.040		0.015	105	0.342			[39]
	Median	0.181		0.116	9.33	12.9	0.001	0.014		0.007	25.6	0.130			
30 household dust samples (< 63 $\mu\text{m}$ ) from vacuum cleaner	Median					87.4		15.2			604		129	33.6	[32]
Dust from 30 apartments in bags of vacuum cleaner	Mean	10.8	54.6	44.6		55.6		86.1			776				[71]
	Median	1.5	37.5	6.1		47.0		29.7			703				
Dust from 11 houses, 3 labs and 1 hospital in vacuum cleaning bags, Qatar	Mean	0.89		7.9		12.5	1.6	2.6	10.7		288	5.8	106	11.4	[35]
	Median	0.98		7.0		17.0	0.12	1.95	9.6		395	3.0	101	11.0	
Dust samples from homes in Kuwait from vacuum cleaning bags	Mean	0.01		1.5		51	0.2	6.4		0.3	1700	14			[72]
	Median	0.03		1.8		45	0.39	8.6		2.9	2256	14			
House dust from China collected by sweeping the floor and wiping the top of furniture (< 2 mm)	Median	0.2		0.4	17.2	20.1	nd	0.2		nd	228	0.2			[40]
Dust from Albany, USA, from vacuum cleaner bags of several homes (< 2 mm)	Median	0.08		2.0	3.8	13.1	0.6	21.1		nd	304	0.4			[40]
Saudi floor dust from vacuum cleaner bags	Mean	1.4		4.2	33.6	80.2		1.5			1140	102.4			[37]
	Median	0.6		1.4	22.1	33.3		0.8			1020	26.8			
Kuwaiti floor dust from vacuum cleaner bags	Mean	0.2		5.1	20.0	4.0		1.3			220	2.4			[37]
	Median	0.1		2.7	17.2	1.6		0.8			240	2.8			
	Mean	260		350		7850		320			960	250			[66]

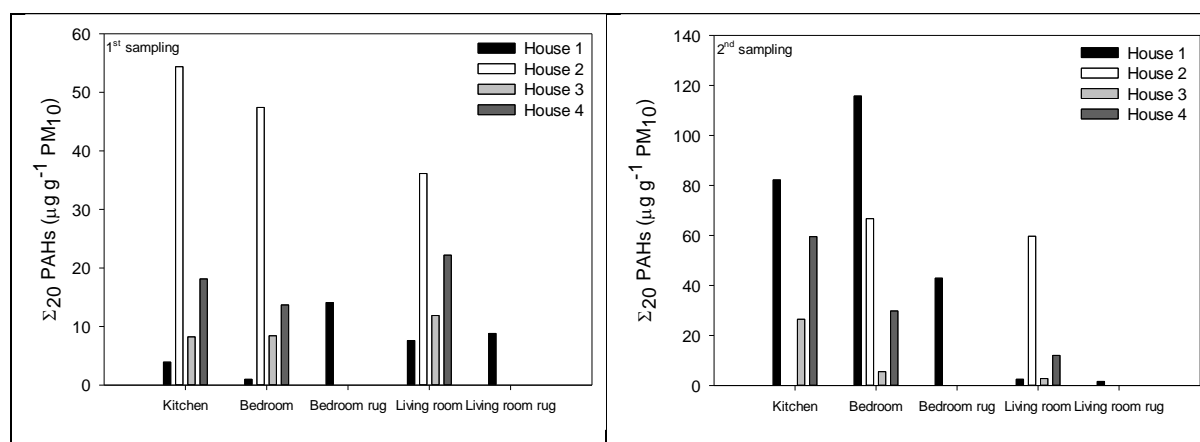
Settled dust collected in the child's room, above the floor level, in Bulgarian homes by a vacuum cleaner	Median	280		340		9930		340		1050	300			
Dust from rooms in Germany collected with vacuum cleaner (< 2 mm)	Mean	1.32		80.7	107.1	384		85.8		1026		114	13.5	[69]
	Median	1.42		12.9	36.5	44.1		82.2		996		113	< 0.1	
Dust from rooms in Spain (Madrid) collected with vacuum cleaner (< 2 mm)	Mean	0.14		14.0	265	131		6.55		464		69.5	10.2	[69]
	Median	< 0.1		7.88	201	145		5.3		370		< 0.1	< 0.1	
Dust from rooms in France (Paris) collected with vacuum cleaner (< 2 mm)	Mean	< 0.1		9.88	91.4	174		227		111		122	48.2	[69]
	Median	< 0.1		8.72	86.1	65.8		200		356		115	< 0.1	
Dust from rooms in Italy (Rome) collected with vacuum cleaner (< 2 mm)	Mean	0.30		9.60	221	36.6		89.0		503		76.0	142	[69]
	Median	< 0.1		6.78	180	42.8		23.6		434		< 0.1	< 0.1	
Dust from rooms in the UK collected with vacuum cleaner (< 2 mm)	Mean	0.12		12.2	52	50.2		56.5		192		48.5	20.8	[69]
	Median	< 0.1		3.5	43.2	52.8		24.5		195		< 0.1	< 0.1	
Settled dust from apartment buildings in Stockholm from vacuum cleaner	Median	0.47		14	104	103		16		449	0.00	106	56	[64]
Floor dust collected from the floor surface and from objects within 35 cm above the floor with hand-held vacuum cleaner in Japanese dwellings	Median	< 0.5		< 0.5	3.1	16.6		2.0	8.0	1110		139		[61]
Dust samples collected with vacuum cleaner from the floor of living rooms in 41 dwellings, Sapporo (Japan)	Median	< 0.2		0.33	2.9	19.8		4.2	6.5	880		126		[73]
Dust samples from household vacuum cleaner bags collected in French dwellings (<100 µm)	Mean	0.26		10.6	111	10.5		25.8		441		158		[17]
	Median	0.25		4.9	20	9.1		6.1		462		139		
House dust from urban dwellings in Nanjing, China, collected with vacuum cleaner	Mean	0.4		0.9		52.3		2.9		462	1.6			[36]
	Median	0.1		0.2		23.7		1.6		183	0.1			
Dust from homes across the USA collected with vacuum cleaner	Mean	0.05		2.34	6.24	4.17		214	77.1	97.2	144			[29]
	Median	0.04		0.49	4.08	3.54		51.2	21.8	73.1	74.9			

Dimethyl phthalate (DMP), dimethylpropyl phthalate (DMPP), diethyl phthalate (DEP), diisobutyl phthalate (DIBP), di-n-butyl phthalate (DBP), di-n-hexylphthalate (DNHP), benzyl butyl phthalate (BBP), bis(2-ethylhexyl) adipate (DEHA), dicyclohexyl phthalate (DCHP), bis(2-ethylhexyl) phthalate (DEHP), di-n-octyl phthalate (DNOP), di-isononylphthalate (DINP), di-isodecylphthalate (DIDP), nd - not detected

1  
2 Compared to plasticizer compounds, PAHs accounted for a much smaller mass of household dust  
3 ( $\Sigma_{20}\text{PAHs}$  0.98–116  $\mu\text{g g}^{-1}$   $\text{PM}_{10}$ ). Given the small number of samples and the variability in  
4 concentrations, it is difficult to infer patterns between house or room typologies (Figure 4). The values  
5 obtained in this study fall into the broad range of values reported in the literature. Wang et al. [22]  
6 analyzed 15 PAHs in settled house dust of urban dwellings with preschool-aged children in Nanjing,  
7 China.  $\Sigma_{15}\text{PAHs}$  ranged from 1.2 to 280.4  $\mu\text{g g}^{-1}$ , averaging 11.1  $\mu\text{g g}^{-1}$ . Yadav et al. [74] investigated  
8 the contamination level of EPA's priority PAHs in indoor dust from residential, educational,  
9 commercial, public places and office premises in four major cities of Nepal. Concentrations of  $\Sigma_{16}\text{PAHs}$   
10 ranged from 747 to 4910  $\text{ng g}^{-1}$  (median 1320  $\text{ng g}^{-1}$ ). The median concentration of  $\Sigma_{16}\text{PAHs}$  quantified  
11 by Mahfouz et al. [75] in dust retained in air-conditioning unit filters from 13 households in Greater  
12 Doha, Qatar, was 218.0  $\text{ng g}^{-1}$ , but a wide range of variation was registered (1.6–477.3  $\text{ng g}^{-1}$ ). In  
13 Palermo, Italy, Mannino and Orecchio [76] collected indoor dust samples by brushing from surfaces at  
14 a height of 1.5–2.0 m above the ground level in bedrooms, living rooms, kitchens, laboratories, offices,  
15 in a market and in a car.  $\Sigma_{16}\text{PAH}$  concentrations were within a broad interval (36–34,453  $\mu\text{g g}^{-1}$ ), with  
16 an average of 5111  $\mu\text{g g}^{-1}$ , indicating heterogeneous levels of contamination in the investigated  
17 microenvironments. Organic extracts of sieved vacuum cleaner dust from 51 homes in Canada were  
18 examined for the presence of 13 PAHs [77]. Total concentrations varied between 1.5 and 325  $\mu\text{g g}^{-1}$   
19 with a geometric mean of 12.9  $\mu\text{g g}^{-1}$ . These values were found to be comparable to those documented  
20 in a previous review in which the total PAHs in samples collected from urban, rural, and suburban  
21 homes ranged between 0.4–544  $\mu\text{g g}^{-1}$  with a geometric mean of 4.5  $\mu\text{g g}^{-1}$  [7]. High concentrations of  
22  $\Sigma_{16}\text{PAHs}$  were also observed in indoor dust samples collected across China from 45 private domiciles  
23 and 36 public buildings (1.00–470  $\mu\text{g g}^{-1}$ , mean value of 30.9  $\mu\text{g g}^{-1}$ ) [78]. PAH concentrations in  
24 electronic repair workshop dusts were reported to range between 205 and 2963  $\mu\text{g kg}^{-1}$ , with dominance  
25 of 5- and 3-ring homologues [79]. Liu et al. [24] collected and sieved indoor dust from office and public  
26 microenvironments (laboratories, classroom, lobby, hotel and supermarket) in Nanjing, China, into 6  
27 size fractions (<43  $\mu\text{m}$ , 43–63  $\mu\text{m}$ , 63–100  $\mu\text{m}$ , 100–150  $\mu\text{m}$ , 150–200  $\mu\text{m}$ , and 200–2000  $\mu\text{m}$ ). Total  
28 PAH concentrations varied from 10.6 to 42.2  $\mu\text{g g}^{-1}$ . Higher concentrations, and consequently more  
29 health risks, were observed for the smallest particles (< 43  $\mu\text{m}$ ). It must be borne in mind, once again,  
30 that comparability between results of various works should be made with caution, as they concern  
31 different surfaces, particle sizes, sampling and analytical methodologies, and list of compounds. A  
32 summary of average PAH concentrations in settled house dust by country and year (data from 35  
33 studies) can be found in a recent review article [80]. A major issue encountered when comparing these  
34 studies was the variability in both the sampling methods employed and the dust particle size fractions  
35 subjected to analysis. In 21 out of the 35 studies reviewed, the particle size cut-off points were either  
36 150  $\mu\text{m}$  or 63  $\mu\text{m}$ . It has been suggested that particles > 150  $\mu\text{m}$  do not easily and efficiently adhere to

37 hands or skin. Therefore, these sizes are less relevant when evaluating exposure via ingestion or dermal  
 38 pathways [81].

39



40

41 **Figure 4.** PAH mass fractions in PM<sub>10</sub> dust samples collected in two different sampling periods.

42

43 Low molecular weight-PAHs (LMW, two and three rings) were less abundant than high molecular  
 44 weight-PAHs (HMW, four and six rings), suggesting the dominance of pyrogenic sources. Regardless  
 45 of the microenvironment, the median LMW/HMW ratios were always in the range from 0.3 to 0.5. The  
 46 main PAHs in the thoracic fraction of resuspended dust were pyrene, retene and indeno[1,2,3-  
 47 cd]pyrene, reaching concentrations up to 27, 21 and 13 µg g<sup>-1</sup>, respectively (Table 3). While the medians  
 48 of the latter two compounds were higher in the PM<sub>10</sub> sampled in the living rooms, pyrene showed higher  
 49 levels in the kitchens. PAH levels and speciation are highly dependent on the cooking methods [82],  
 50 biomass burning appliances and operating conditions [83], traffic fleet and meteorology in the outdoor  
 51 surrounding environment [84], among other factors.

52

**Table 3.** Comparison of PAH levels ( $\mu\text{g g}^{-1}$ ) in household dust from different countries

PAHs	This study								[85]	[74]	[77]	[78]	[86]					
	Kitchens		Living Rooms		Bedrooms		Rug bed-room	Rug living room	House vacuum samples Brisbane	Indoor dust from distinct buildings Nepal	Settled Dust from homes in Ottawa		Dust samples from private domiciles and public buildings across China		Saudi household floor dust		Kuwaiti household floor dust	
	Mean	Median	Mean	Median	Mean	Median	Mean	Mean	Mean	Median	Mean	Median	Mean	Median	Mean	Median	Mean	Median
ACY	1.75	0.432	3.22	1.15	4.70	2.04	0.988	< 0.001	6.92	0.031	0.039	0.005	0.173	0.055	0.160	0.080	0.090	0.060
ACE	0.344	< 0.001	0.74	0.215	0.246	0.063	0.067	0.136	202	0.018			0.093	0.052	0.110	0.105	0.065	0.060
FLU	0.073	< 0.001	0.036	< 0.001	0.017	< 0.001	0.036	0.012	12.6	0.048	0.170	0.093	0.464	0.200	0.290	0.170	0.165	0.080
PHE	0.213	< 0.001	0.260	0.154	0.306	0.177	0.169	0.275	8.77	0.173	2.78	1.48	4.19	1.67	0.425	0.160	0.510	0.410
ANT	1.56	< 0.001	1.07	< 0.001	< 0.001	< 0.001	0.002	1.52	3.61	0.062	0.485	0.196	0.418	0.196	0.105	0.050	0.032	0.025
FLUA	1.56	0.277	1.12	< 0.001	1.60	0.045	< 0.001	0.197	53.6	0.128			4.26	1.77	0.425	0.175	0.220	0.140
PYR	4.46	2.17	3.67	1.51	4.65	0.604	0.003	1.06	77.3	0.107			2.89	1.18	0.385	0.160	0.035	0.017
CHR	0.921	0.404	0.695	0.506	0.610	0.23	0.120	0.053	5.42	0.157	3.29	1.19	1.79	0.717	0.095	0.085	0.055	0.040
PER	4.29	< 0.001	3.73	1.70	3.26	1.25	0.301	0.602										
CAR	1.08	< 0.001	1.83	0.440	1.64	< 0.001	0.001	0.141										
TER	< 0.001	< 0.001	0.135	< 0.001	0.021	< 0.001	0.028	< 0.001										
RET	3.15	0.737	5.02	2.20	5.28	0.966	< 0.001	2.09										
BaA	1.55	1.78	0.516	0.136	0.333	< 0.001	0.040	1.40	6.39	0.078	2.38	0.696	2.37	0.569	0.090	0.080	0.090	0.055
BbF	1.91	1.63	2.10	1.31	1.50	0.124	0.304	1.07	11.3	0.018	4.87	1.66	6.23	1.52	0.700	0.575	0.245	0.210
BkF	1.10	< 0.001	1.02	< 0.001	0.960	< 0.001	< 0.001	0.710	3.48	0.075	1.60	0.582	0.763	0.248	0.110	0.105	0.070	0.040
BeP	0.206	< 0.001	0.844	< 0.001	< 0.001	< 0.001	< 0.001	0.746										
BaP	1.53	1.63	1.23	0.564	1.11	0.219	0.061	1.43	2.97	0.094	2.91	0.803	1.71	0.310	0.550	0.290	0.140	0.080
IcdP	3.48	1.75	3.11	2.85	2.76	2.32	2.61	0.565	3.34	0.115	3.07	0.911	1.97	0.356	0.015	< 0.001	0.055	< 0.001
DahA	0.289	< 0.001	0.790	0.314	0.395	0.155	0.162	0.071	20.1	0.046	0.549	0.185	0.510	0.090	0.016	< 0.001	0.015	< 0.001

BghiP	2.43	0.922	1.10	0.275	1.46	< 0.001	< 0.001	0.263	6.08	0.068	2.79	0.793	2.14	0.398	0.105	0.075	0.070	< 0.001
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ACY – acenaphthylene, ACE – acenaphthene, FLU – fluorene, PHE – phenanthrene, ANT – anthracene, FLUA – fluoranthene, PYR – pyrene, CHR – chrysene, PER – perylene, CAR – carbazole, TER – p-terphenyl, RET – retene, BaA – benzo[a]anthracene, BbF – benzo[b]fluoranthene, BkF – benzo[k]fluoranthene, BeP – benzo[e]pyrene, BaP – benzo[a]pyrene, IcdP – indeno[1,2,3-cd]pyrene, DahA – dibenzo[a,h]anthracene, BghiP – benzo[ghi]perylene



### 3.3. Human exposure to plasticizers and PAHs in resuspended dust

Human exposure to plasticizers and PAHs occurs via ingestion, inhalation and dermal contact. The daily intake (DI) through different pathways were estimated by the following equations [39; and references therein], where  $DI_{ing}$ ,  $DI_{inh}$  and  $DI_{der}$  are the daily intakes ( $ng\ kg^{-1}\ day^{-1}$ ) via dust ingestion, inhalation and dermal contact, respectively:

$$DI_{ing} = \frac{C_{dust} \times IngR \times EF \times ED \times CF}{BW \times AT} \quad (\text{eq. 1})$$

$$DI_{inh} = \frac{C_{dust} \times InhR \times EF \times ED}{BW \times AT \times PEF} \quad (\text{eq. 2})$$

$$DI_{der} = \frac{C_{dust} \times SA \times AF_{dust} \times ABS \times EF \times ED \times CF}{BW \times AT} \quad (\text{eq. 3})$$

$C_{dust}$  represent the mean concentrations in dust ( $ng\ g^{-1}$ );  $IngR$  is the ingestion rate of indoor dust (200  $mg\ day^{-1}$  for children, 100  $mg\ day^{-1}$  for adults);  $EF$  is the exposure frequency (180 days per year for both children and adults);  $ED$  is the exposure duration (6 year for children, 24 years for adults);  $CF$  is an unit conversion factor ( $10^{-3}\ g\ mg^{-1}$ );  $BW$  is the body weight (15 kg for children, 70 kg for adults);  $AT$  is the average time (2190 days for children, 8760 days for adults);  $InhR$  is the inhalation rate (7.6  $m^3\ day^{-1}$  for children, 12.8  $m^3\ day^{-1}$  for adults);  $PEF$  is the particulate emission factor  $1.36 \times 10^6\ m^3\ g^{-1}$ ;  $SA$  is the dermal exposure area (1150  $cm^2$  for children, 2145  $cm^2$  for adults);  $AF_{dust}$  is the dust adherence factor (0.2  $mg\ cm^{-2}\ day^{-1}$  for children, 0.07  $mg\ cm^{-2}\ day^{-1}$  for adults); and  $ABS$  is the dermal adsorption fraction (0.001 for both children and adults, dimensionless).

Ingestion is the main pathway for intake of plasticizers from dust (Table 4). Regardless of the route, household residents are exposed to higher intakes in the bedrooms, whereas the lowest doses are experienced in the kitchens. The daily intakes for children and adults (13–29 and 1.4–3.1  $\mu g\ kg^{-1}\ day^{-1}$ , respectively) via dust ingestion were 3–4 orders of magnitude higher than those via inhalation (0.357–0.802 and 0.129–0.289  $ng\ kg^{-1}\ day^{-1}$ , respectively) and dermal contact (14.9–33.0 and 2.05–4.62  $ng\ kg^{-1}\ day^{-1}$ , respectively). Children are at higher risk of exposure to plasticizers than adults. The total  $DI_{ing}$ ,  $DI_{inh}$  and  $DI_{der}$  for children were about 9.3, 2.8 and 7.1 times higher than those estimated for adults, respectively. DBP, DNOP and DEHP contributed the most to daily intakes. The dust ingestion intakes for these compounds were lower than the U.S. EPA maximum acceptable oral doses of 0.8, 0.01 and 0.02  $mg\ kg^{-1}\ day^{-1}$ , respectively. However, the daily intakes of plasticizers through dust ingestion, inhalation and dermal contact of the present study are higher than those estimated for indoor dust from houses of several Chinese regions [39], and childcare facilities, salons, and homes across the USA [29]. Albar et al. [37] assessed human exposure to phthalates via dust ingestion for the worst-case scenario

(with 95<sup>th</sup> percentile levels) for Saudi and Kuwaiti toddlers and adults. The exposure to DEHP, which is cardiotoxic and endocrine disruptor, for Saudi toddlers was estimated to be 37,630 ng kg<sup>-1</sup> day<sup>-1</sup>, while for Kuwaiti toddlers it was 6722 ng kg<sup>-1</sup> day<sup>-1</sup>. Similarly, exposure estimates to other PAEs, such as DBP, DIBP, and DNOP, was also higher for Saudi toddlers. In the case of Saudi and Kuwaiti adults, dust ingestion intakes for DEHP were estimated at 1613 and 288 ng kg<sup>-1</sup> day<sup>-1</sup>, respectively.

**Table 4.** Mean daily intakes (ng kg<sup>-1</sup> day<sup>-1</sup>) of plasticizers via dust ingestion, inhalation and dermal contact for children and adults.

	DMP	DEP	DBP	BBP	DEHA	DEHP	DNOP	DINP	DIDP	Σ
DI <sub>ing</sub> – CHILDREN										
Kitchens	1.78	934	3695	41.4	112	1782	6095	67.1	43.4	12,772
Bedrooms	2.17	296	15,472	68.4	237	5898	6372	131	232	28,707
Living rooms	6.12	914	7765	73.0	125	8890	4938	185	437	23,333
DI <sub>inh</sub> – CHILDREN										
Kitchens	0.000	0.0261	0.103	0.003	0.001	0.050	0.170	0.002	0.001	0.357
Bedrooms	0.000	0.0083	0.432	0.007	0.002	0.165	0.178	0.004	0.006	0.802
Living rooms	0.000	0.0255	0.217	0.003	0.002	0.248	0.138	0.005	0.012	0.652
DI <sub>der</sub> – CHILDREN										
Kitchens	0.002	1.07	4.25	0.129	0.048	2.05	7.01	0.077	0.050	14.7
Bedrooms	0.003	0.340	17.8	0.272	0.079	6.78	7.33	0.150	0.267	33.0
Living rooms	0.007	1.05	8.93	0.144	0.084	10.2	5.68	0.213	0.502	26.8
DI <sub>ing</sub> – ADULTS										
Kitchens	0.190	100	396	12.0	4.44	191	653	7.19	4.65	1368
Bedrooms	0.232	31.7	1658	25.4	7.33	632	683	14.0	24.9	3076
Living rooms	0.655	97.9	832	13.4	7.82	952	529	19.9	46.8	2500
DI <sub>inh</sub> – ADULTS										
Kitchens	0.000	0.009	0.037	0.001	0.000	0.018	0.062	0.001	0.000	0.129
Bedrooms	0.000	0.003	0.156	0.002	0.001	0.059	0.064	0.001	0.002	0.289
Living rooms	0.000	0.009	0.078	0.001	0.001	0.090	0.050	0.002	0.004	0.235
DI <sub>der</sub> – ADULTS										
Kitchens	0.000	0.150	0.595	0.018	0.0067	0.287	0.981	0.011	0.007	2.05
Bedrooms	0.000	0.048	2.49	0.038	0.0110	0.949	1.03	0.021	0.037	4.62
Living rooms	0.001	0.147	1.25	0.020	0.0117	1.43	0.794	0.030	0.070	3.75

Like plasticizers, PAHs can enter the body through ingestion (swallowing), inhalation (breathing) and skin contact. The carcinogenic potency of PAHs in indoor dust can be evaluated by comparing the carcinogenic potency of each PAH to that of BaP. The BaP carcinogenic equivalent concentration (BaP<sub>TEQ</sub>) is defined as follows:

$$\text{BaP}_{\text{TEQ}} = \sum C_i \times \text{BaP}_{\text{TEF}} \quad (\text{eq. 4})$$

where  $C_i$  is the concentration of each individual PAH and  $\text{BaP}_{\text{TEF}}$  are toxic equivalency factors [24]. BaP, DahA and IcdP were the compounds that most contributed to the carcinogenic potency, accounting for 45-57%, 11-29% and 11-13% of the total BaP<sub>TEQ</sub>, respectively. The incremental lifetime cancer risk

(ILCR) is generally used to quantitatively estimate the exposure risks from the three exposure routes[24,87]:

$$ILCR_{ing} = \frac{CS \times \left( CSF_{ing} \times \sqrt[3]{\frac{BW}{70}} \right) \times IngR \times EF \times ED \times CF}{BW \times AT} \quad (\text{eq. 5})$$

$$ILCR_{inh} = \frac{CS \times \left( CSF_{inh} \times \sqrt[3]{\frac{BW}{70}} \right) \times InhR \times EF \times ED}{BW \times AT \times PEF} \quad (\text{eq. 6})$$

$$ILCR_{der} = \frac{CS \times \left( CSF_{der} \times \sqrt[3]{\frac{BW}{70}} \right) \times SA \times AF_{dust} \times ABS \times EF \times ED \times CF}{BW \times AT} \quad (\text{eq. 7})$$

where CS is the BaP<sub>TEQ</sub> concentration (mg kg<sup>-1</sup>); CSF<sub>ing</sub>, CSF<sub>inh</sub>, and CSF<sub>der</sub> are carcinogenic slope factors of 7.3, 3.85 and 25 (mg kg<sup>-1</sup> day<sup>-1</sup>)<sup>-1</sup>, respectively. As shown in Table 5, the total cancer risk could be attributed almost entirely to ingestion and did not vary much with microenvironment. Therefore, inhalation of resuspended particles through the mouth and nose or via dermal contact was almost negligible when compared with the ingestion route. Under most regulatory programmes, an ILCR between 10<sup>-6</sup> and 10<sup>-4</sup> indicates potential risk, an ILCR of 10<sup>-6</sup> or less is considered insignificant and an ILCR ≥ 10<sup>-4</sup> is taken as high risk. In the present study, the total risk of adult and children exposure to PAHs in dust via the three pathways ranged from 7.2 × 10<sup>-6</sup> to 1.4 × 10<sup>-5</sup>. This means that the resuspendable thoracic fraction of household dust can contribute to an estimated excess of 7.2 to 14 per million people new cancer cases. One cancer case per million people is usually used as a baseline level of acceptable risk.

**Table 5.** Incremental lifetime cancer risk from human exposure to PAHs in PM<sub>10</sub> resuspended from household dust via ingestion, inhalation and dermal absorption.

		Kitchens	Bedrooms	Living rooms
ILCR <sub>ing</sub>	children	9.2×10 <sup>-6</sup>	7.2×10 <sup>-6</sup>	9.4×10 <sup>-6</sup>
	Adults	1.4×10 <sup>-5</sup>	1.1×10 <sup>-5</sup>	1.4×10 <sup>-5</sup>
ILCR <sub>inh</sub>	children	1.4×10 <sup>-10</sup>	1.1×10 <sup>-10</sup>	1.4×10 <sup>-10</sup>
	Adults	6.9×10 <sup>-10</sup>	5.4×10 <sup>-10</sup>	9.0×10 <sup>-10</sup>
ILCR <sub>der</sub>	children	3.6×10 <sup>-8</sup>	2.8×10 <sup>-8</sup>	3.7×10 <sup>-8</sup>
	Adults	7.1×10 <sup>-8</sup>	5.6×10 <sup>-8</sup>	7.3×10 <sup>-8</sup>
Total ILCR	Children	9.3×10 <sup>-6</sup>	7.2×10 <sup>-6</sup>	9.4×10 <sup>-6</sup>
	Adults	1.4×10 <sup>-5</sup>	1.1×10 <sup>-5</sup>	1.4×10 <sup>-5</sup>

To assess the potential health risk of foliar dust in Nanjing, China, Zha et al. [87] analyzed the contents of 16 priority PAHs. Total concentrations in dust ranged from 1.77 to 19.02  $\mu\text{g g}^{-1}$ , with an average value of 6.98  $\mu\text{g g}^{-1}$ . The cancer risk levels via dermal contact and ingestion oscillated from  $10^{-8}$  to  $10^{-6}$  in all the dust samples, while the mean cancer risk via inhalation was  $10^{-10}$  to  $10^{-12}$ , about  $10^4$  to  $10^7$  times lower than through ingestion and dermal contact. ILCR values due to human exposure to PAHs in indoor dust in city, town, village and orefield of Guizhou province, China, were  $6.14 \times 10^{-6}$ ,  $5.00 \times 10^{-6}$ ,  $3.08 \times 10^{-6}$ ,  $6.02 \times 10^{-6}$  for children and  $5.92 \times 10^{-6}$ ,  $4.83 \times 10^{-6}$ ,  $2.97 \times 10^{-6}$ ,  $5.81 \times 10^{-6}$  for adults, respectively [16]. As noted in our study, inhalation of resuspended particles through mouth and nose was very small, when compared with the ingestion pathway. Maertens et al. [77] estimated higher excess lifetime cancer risks, ranging from  $1 \times 10^{-6}$  and  $1 \times 10^{-4}$ , associated with nondietary ingestion of PAHs in settled dust from homes in Ottawa, Canada, during preschool years. However, the researchers observed that the level of risk varies substantially according to the ingestion rates adopted to perform the estimates.

Since ingestion was found to be the dominant exposure pathway, the risk associated with seven non-carcinogenic PAHs (ACY, ACE, FLU, PHE, ANT, FLUA and PYR) in household  $\text{PM}_{10}$  dust was estimated through the hazard quotient (HQ):

$$\text{HQ} = \text{DI}_{\text{nc}}/\text{R}_f\text{D} \quad (\text{eq. 8})$$

where  $\text{DI}_{\text{nc}}$  is the daily intake via ingestion of non-carcinogenic PAHs, obtained by equation 1, and  $\text{R}_f\text{D}$  is the reference dose. The oral ingestion  $\text{R}_f\text{D}$  values were taken from Iwegbue et al. [79]. Under most programmes, if the HQ value is greater than 1, the exposed population is likely to experience considerable non-carcinogenic effects. The highest daily intakes were obtained for PYR (24–31  $\text{ng kg}^{-1} \text{day}^{-1}$  for children, 2.6–3.3  $\text{ng kg}^{-1} \text{day}^{-1}$  for adults) and ACY (12–31  $\text{ng kg}^{-1} \text{day}^{-1}$  for children, 1.2–3.3  $\text{ng kg}^{-1} \text{day}^{-1}$  for adults). These values are higher than those documented for dust samples collected from three different microenvironments (cars, air conditioner filters and household floor dust) of Jeddah, Saudi Arabia, and Kuwait [86]. Nevertheless, the HQ values of the present study were less than 1, indicating that there was no considerable non-carcinogenic risk arising from ingestion of PAHs in resuspended  $\text{PM}_{10}$ . Although the risk remains very low for both age groups, higher HQ values for children ( $1.51 \times 10^{-3}$ – $1.89 \times 10^{-3}$ ) than those obtained for adults ( $1.32 \times 10^{-4}$ – $2.03 \times 10^{-4}$ ), demonstrate greater susceptibility of the younger ones. It should be noted that HQ values are underestimated as naphthalene was not included in the quantifications. Despite its volatility, naphthalene is normally quite abundant in the particulate phase. Higher non-carcinogenic risks due to exposure to PAHs have been reported for some occupational environments. For example,  $\text{HQ}_{\text{ing}}$  from  $1.38 \times 10^{-2}$  to  $1.09 \times 10^{-1}$  were estimated for indoor dusts from electronic repair workshops in southern Nigeria [79].

#### 4. Conclusions

This preliminary study provides a first insight on the occurrence of plasticizers and PAHs in PM<sub>10</sub> from resuspended dust samples from Spanish households and adds to the growing evidence that non-dietary exposure contributes to the total body burden. Considering that people spend most of their time indoors, exposure to these pollutants might lead to an increased human health risk. Although no appreciable differences between plasticizer and PAH levels in resuspended dust from the various residential microenvironments were observed, it was concluded that exposure through the ingestion route poses much higher risks compared to inhalation and dermal contact. This is of particular concern for infants due to their higher dust intake via frequent hand-to-mouth activities. Because of the small number of samples analyzed in this study, it should be noted that exposure estimates are only an indication of the likely range for children and adults within the studied population. More assessments with wider spatial and temporal coverage are needed to better understand the dynamics and possible effects of these pollutants in different indoor microenvironments. As observed in other works, this study on exposure to organic pollutants suggests that measurements only in food and outdoor environments can substantially underestimate exposures to chemicals. Comparisons of the results of this study with those reported in the literature revealed huge amplitudes in the numbers and great difficulty in generalizing conclusions, mainly due to the heterogeneity of applied methodologies. Thus, the scientific community and international agencies should discuss and establish standardized protocols.

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## References

1. Schweizer, C.; Edwards, R.D.; Bayer-Oglesby, L.; Gauderman, W.J.; Ilacqua, V.; Juhani Jantunen, M.; Lai, H.K.; Nieuwenhuijsen, M.; Künzli, N. Indoor time–microenvironment–activity patterns in seven regions of Europe. *J. Expo. Sci. Environ. Epidemiol.* 2007, 17, 170–181.
2. Qian, J.; Peccia, J.; Ferro, A.R. Walking-induced particle resuspension in indoor environments. *Atmos. Environ.* 2014, 89, 464–481.
3. Tian, Y.; Sul, K.; Qian, J.; Mondal, S.; Ferro, A.R. A comparative study of walking-induced dust resuspension using a consistent test mechanism. *Indoor Air* 2014, 24, 592–603.
4. Bo, M.; Salizzoni, P.; Clerico, M.; Buccolieri, R. Assessment of indoor-outdoor particulate matter air pollution: A review. *Atmosphere (Basel)*. 2017, 8.
5. Naspinski, C.; Lingenfelter, R.; He, L.Y.; Cizmas, L.; Naufal, Z.; Islamzadeh, A.; Li, Z.; Li, Z.; Donnelly, K.C.; McDonald, T. A comparison of concentrations of polycyclic aromatic compounds detected in dust samples from various regions of the world. *Environ. Int.* 2008, 34, 988–993.
6. Lioy, P.J.; Freeman, N.C.G.; Millette, J.R. Review Dust : A Metric for Use in Residential and Building Exposure Assessment and. *Environ. Health Perspect.* 2002, 110, 969–983.
7. Maertens, R.M.; Bailey, J.; White, P.A. The mutagenic hazards of settled house dust: a review. *Mutat. Res.* 2004, 567, 401–25.
8. Liu, H.Y.; Dunea, D.; Iordache, S.; Pohoata, A. A review of airborne particulate matter effects on young children’s respiratory symptoms and diseases. *Atmosphere (Basel)*. 2018, 9.
9. Cipriani, F.; Calamelli, E.; Ricci, G. Allergen avoidance in allergic asthma. *Front. Pediatr.* 2017, 5.
10. Ahluwalia, S.K.; Matsui, E.C. The indoor environment and its effects on childhood asthma. *Curr. Opin. Allergy Clin. Immunol.* 2011, 11, 137–143.
11. McCormack, M.C.; Breyse, P.N.; Matsui, E.C.; Hansel, N.N.; Peng, R.D.; Curtin-Brosnan, J.; Williams, D.L.; Wills-Karp, M.; Diette, G.B.; Center for Childhood Asthma in the Urban Environment Indoor particulate matter increases asthma morbidity in children with non-atopic and atopic asthma. *Ann. Allergy. Asthma Immunol.* 2011, 106, 308–15.
12. Mitro, S.D.; Dodson, R.E.; Singla, V.; Adamkiewicz, G.; Elmi, A.F.; Tilly, M.K.; Zota, A.R. Consumer Product Chemicals in Indoor Dust: A Quantitative Meta-analysis of U.S. Studies. *Environ. Sci. Technol.* 2016, 50, 10661–10672.
13. Roberts, J.W.; Wallace, L.A.; Camann, D.E.; Dickey, P.; Gilbert, S.G.; Lewis, R.G.; Takaro, T.K. Monitoring and Reducing Exposure of Infants to Pollutants in House Dust. In *Reviews of environmental contamination and toxicology*; 2009; Vol. 201, pp. 1–39.
14. Langer, S.; Weschler, C.J.; Fischer, A.; Bekö, G.; Toftum, J.; Clausen, G. Phthalate and PAH concentrations in dust collected from Danish homes and daycare centers. *Atmos. Environ.* 2010, 44, 2294–2301.
15. Dodson, R.E.; Camann, D.E.; Morello-Frosch, R.; Brody, J.G.; Rudel, R.A. Semivolatile organic compounds in homes: strategies for efficient and systematic exposure measurement based on empirical and theoretical factors. *Environ. Sci. Technol.* 2015, 49, 113–22.
16. Yang, Q.; Chen, H.; Li, B. Polycyclic aromatic hydrocarbons (PAHs) in indoor dusts of Guizhou, southwest of China: status, sources and potential human health risk. *PLoS One* 2015,

- 10, e0118141.
17. Mercier, F.; Glorennec, P.; Thomas, O.; Bot, B. Le Organic contamination of settled house dust, a review for exposure assessment purposes. *Environ. Sci. Technol.* 2011, 45, 6716–6727.
  18. DellaValle, C.T.; Deziel, N.C.; Jones, R.R.; Colt, J.S.; De Roos, A.J.; Cerhan, J.R.; Cozen, W.; Severson, R.K.; Flory, A.R.; Morton, L.M.; et al. Polycyclic aromatic hydrocarbons: determinants of residential carpet dust levels and risk of non-Hodgkin lymphoma. *Cancer Causes Control* 2016, 27, 1–13.
  19. Boström, C.E.; Gerde, P.; Hanberg, A.; Jernström, B.; Johansson, C.; Kyrklund, T.; Rannug, A.; Törnqvist, M.; Victorin, K.; Westerholm, R. Cancer risk assessment, indicators, and guidelines for polycyclic aromatic hydrocarbons in the ambient air. *Environ. Health Perspect.* 2002, 110, 451–488.
  20. Kim, K.H.; Jahan, S.A.; Kabir, E.; Brown, R.J.C. A review of airborne polycyclic aromatic hydrocarbons (PAHs) and their human health effects. *Environ. Int.* 2013, 60, 71–80.
  21. Deziel, N.C.; Rull, R.P.; Colt, J.S.; Reynolds, P.; Whitehead, T.P.; Gunier, R.B.; Month, S.R.; Taggart, D.R.; Buffler, P.; Ward, M.H.; et al. Polycyclic aromatic hydrocarbons in residential dust and risk of childhood acute lymphoblastic leukemia. *Environ. Res.* 2014, 133, 388–395.
  22. Wang, B.L.; Pang, S.T.; Zhang, X.L.; Li, X.L.; Sun, Y.G.; Lu, X.M.; Zhang, Q.; Zhang, Z.D. Levels and neurodevelopmental effects of polycyclic aromatic hydrocarbons in settled house dust of urban dwellings on preschool-aged children in Nanjing, China. *Atmos. Pollut. Res.* 2014, 5, 292–302.
  23. Kang, Y.; Cheung, K.C.; Wong, M.H. Polycyclic aromatic hydrocarbons (PAHs) in different indoor dusts and their potential cytotoxicity based on two human cell lines. *Environ. Int.* 2010, 36, 542–7.
  24. Liu, R.; He, R.; Cui, X.; Ma, L.Q. Impact of particle size on distribution, bioaccessibility, and cytotoxicity of polycyclic aromatic hydrocarbons in indoor dust. *J. Hazard. Mater.* 2018, 357, 341–347.
  25. IARC Some non-heterocyclic polycyclic aromatic hydrocarbons and some related exposures. Monographs on the evaluation of carcinogenic risks to humans. Lyon, France, 2010.
  26. ATSDR Priority list of hazardous substances Available online: <http://www.atsdr.cdc.gov/SPL/resources/>.
  27. Ma, W.-L.; Subedi, B.; Kannan, K. The Occurrence of Bisphenol A, Phthalates, Parabens and Other Environmental Phenolic Compounds in House Dust: A Review. *Curr. Org. Chem.* 2014, 18, 2182–2199.
  28. Larsson, K.; Lindh, C.H.; Jönsson, B.A.; Giovanoulis, G.; Bibi, M.; Bottai, M.; Bergström, A.; Berglund, M. Phthalates, non-phthalate plasticizers and bisphenols in Swedish preschool dust in relation to children’s exposure. *Environ. Int.* 2017, 102, 114–124.
  29. Subedi, B.; Sullivan, K.D.; Dhungana, B. Phthalate and non-phthalate plasticizers in indoor dust from childcare facilities, salons, and homes across the USA. *Environ. Pollut.* 2017, 230, 701–708.
  30. Kang, Y.; Man, Y.B.; Cheung, K.C.; Wong, M.H. Risk assessment of human exposure to bioaccessible phthalate esters via indoor dust around the Pearl River Delta. *Environ. Sci. Technol.* 2012, 46, 8422–30.
  31. Kweon, D.J.; Kim, M.K.; Zoh, K.D. Distribution of brominated flame retardants and phthalate esters in house dust in Korea. *Environ. Eng. Res.* 2018, 23, 354–363.
  32. Abb, M.; Heinrich, T.; Sorkau, E.; Lorenz, W. Phthalates in house dust. *Environ. Int.* 2009, 35, 965–970.



33. Kubwabo, C.; Rasmussen, P.E.; Fan, X.; Kosarac, I.; Wu, F.; Zidek, A.; Kuchta, S.L. Analysis of selected phthalates in Canadian indoor dust collected using household vacuum and standardized sampling techniques. *Indoor Air* 2013, 23, 506–14.
34. Bornehag, C.G.; Lundgren, B.; Weschler, C.J.; Sigsgaard, T.; Hagerhed-Engman, L.; Sundell, J. Phthalates in indoor dust and their association with building characteristics. *Environ. Health Perspect.* 2005, 113, 1399–1404.
35. Al Qasbi, N.N.; Al-Thaiban, H.; Helaleh, M.I.H. Indoor phthalates from household dust in Qatar: implications for non-dietary human exposure. *Environ. Sci. Pollut. Res. Int.* 2019, 26, 421–430.
36. Zhang, Q.; Lu, X.M.; Zhang, X.L.; Sun, Y.G.; Zhu, D.M.; Wang, B.L.; Zhao, R.Z.; Zhang, Z.D. Levels of phthalate esters in settled house dust from urban dwellings with young children in Nanjing, China. *Atmos. Environ.* 2013, 69, 258–264.
37. Albar, H.M.S.A.; Ali, N.; Shahzad, K.; Ismail, I.M.I.; Rashid, M.I.; Wang, W.; Ali, L.N.; Eqani, S.A.M.A.S. Phthalate esters in settled dust of different indoor microenvironments; source of non-dietary human exposure. *Microchem. J.* 2017, 132, 227–232.
38. Kashyap, D.; Agarwal, T. Concentration and factors affecting the distribution of phthalates in the air and dust: A global scenario. *Sci. Total Environ.* 2018, 635, 817–827.
39. Zhu, Q.; Jia, J.; Zhang, K.; Zhang, H.; Liao, C.; Jiang, G. Phthalate esters in indoor dust from several regions, China and their implications for human exposure. *Sci. Total Environ.* 2019, 652, 1187–1194.
40. Guo, Y.; Kannan, K. Comparative assessment of human exposure to phthalate esters from house dust in China and the United States. *Environ. Sci. Technol.* 2011, 45, 3788–94.
41. Orecchio, S.; Indelicato, R.; Barreca, S. The distribution of phthalate esters in indoor dust of Palermo (Italy). *Environ. Geochem. Health* 2013, 35, 613–624.
42. Kay, V.R.; Bloom, M.S.; Foster, W.G. Reproductive and developmental effects of phthalate diesters in males. *Crit. Rev. Toxicol.* 2014, 44, 467–98.
43. Kay, V.R.; Chambers, C.; Foster, W.G. Reproductive and developmental effects of phthalate diesters in females. *Crit. Rev. Toxicol.* 2013, 43, 200–19.
44. Wormuth, M.; Scheringer, M.; Vollenweider, M.; Hungerbühler, K. What are the sources of exposure to eight frequently used phthalic acid esters in Europeans? *Risk Anal.* 2006, 26, 803–24.
45. Directive 2005/84/EC of the European Parliament and of the Council of 14 December 2005 amending for the 22nd time Council Directive 76/769/EEC on the approximation of the laws, regulations and administrative provisions of the Member States relating to restrictions on the marketing and use of certain dangerous substances and preparations (phthalates in toys and childcare articles).
46. Manigrasso, M.; Guerriero, E.; Avino, P. Ultrafine particles in residential indoors and doses deposited in the human respiratory system. *Atmosphere (Basel)*. 2015, 6, 1444–1461.
47. *Indoor Environment. Airborne Particles and Settled Dust*; Morawska, L., Salthammer, T., Eds.; Wiley-VCH GmbH & Co. KGaA, 2003; ISBN 9783527305254.
48. Kitsa, V.; Liroy, P.J.; Chow, J.C.; Watson, J.G.; Shupack, S.; Howell, T.; Sanders, P. Particle-size distribution of chromium: Total and hexavalent chromium in inspirable, thoracic, and respirable soil particles from contaminated sites in New Jersey. *Aerosol Sci. Technol.* 1992, 17, 213–229.
49. Amato, F.; Pandolfi, M.; Viana, M.; Querol, X.; Alastuey, A.; Moreno, T. Spatial and chemical patterns of PM10 in road dust deposited in urban environment. *Atmos. Environ.* 2009, 43,

- 1650–1659.
50. Alves, C.A.; Vicente, A.; Monteiro, C.; Gonçalves, C.; Evtyugina, M.; Pio, C. Emission of trace gases and organic components in smoke particles from a wildfire in a mixed-evergreen forest in Portugal. *Sci. Total Environ.* 2011, 409, 1466–1475.
  51. Lewis, R.D.; Ong, K.H.; Emo, B.; Kennedy, J.; Kesavan, J.; Elliot, M. Resuspension of house dust and allergens during walking and vacuum cleaning. *J. Occup. Environ. Hyg.* 2018, 15, 235–245.
  52. Becher, R.; Øvrevik, J.; Schwarze, P.E.; Nilsen, S.; Hongslo, J.K.; Bakke, J.V. Do carpets impair indoor air quality and cause adverse health outcomes: A review. *Int. J. Environ. Res. Public Health* 2018, 15.
  53. Foarde, K.; Berry, M. Comparison of biocontaminant levels associated with hard vs. carpet floors in nonproblem schools: results of a year long study. *J. Expo. Anal. Environ. Epidemiol.* 2004, 14 Suppl 1, S41–8.
  54. Tranter, D.C. Indoor allergens in settled school dust: a review of findings and significant factors. *Clin. Exp. Allergy* 2005, 35, 126–36.
  55. Causer, S.; Shorter, C.; Sercombe, J. Effect of floorcovering construction on content and vertical distribution of house dust mite allergen, Der p I. *J. Occup. Environ. Hyg.* 2006, 3, 161–168.
  56. Roberts, J.W.; Clifford, W.S.; Glass, G.; Hummer, P.G. Reducing dust, lead, dust mites, bacteria, and fungi in carpets by vacuuming. *Arch. Environ. Contam. Toxicol.* 1999, 36, 477–484.
  57. Adgate, J.L.; Weisel, C.; Wang, Y.; Rhoads, G.G.; Lioy, P.J. Lead in house dust: Relationships between exposure metrics. *Environ. Res.* 1995, 70, 134–147.
  58. Eneji, I.S.; Adams, I.U.; Julius, K.A. Assessment of Heavy Metals in Indoor Settled Harmattan Dust from the University of Agriculture Makurdi, Nigeria. *Niger. Open J. Air Pollut.* 2015, 4, 198–207.
  59. Lioy, P.J.; Freeman, N.C.; Wainman, T.; Stern, A.H.; Boesch, R.; Howell, T.; Shupack, S.I. Microenvironmental analysis of residential exposure to chromium-laden wastes in and around New Jersey homes. *Risk Anal.* 1992, 12, 287–99.
  60. Bai, Z.; Yiin, L.M.; Rich, D.Q.; Adgate, J.L.; Ashley, P.J.; Lioy, P.J.; Rhoads, G.G.; Zhang, J. Field evaluation and comparison of five methods of sampling lead dust on carpets. *Am. Ind. Hyg. Assoc. J.* 2003, 64, 528–532.
  61. Bamai, Y.A.; Araki, A.; Kawai, T.; Tsuboi, T.; Saito, I.; Yoshioka, E.; Kanazawa, A.; Tajima, S.; Shi, C.; Tamakoshi, A.; et al. Associations of phthalate concentrations in floor dust and multi-surface dust with the interior materials in Japanese dwellings. *Sci. Total Environ.* 2014, 468-469, 147–157.
  62. Afshari, A.; Gunnarsen, L.; Clausen, P.A.; Hansen, V. Emission of phthalates from PVC and other materials. *Indoor Air* 2004, 14, 120–128.
  63. Koniecki, D.; Wang, R.; Moody, R.P.; Zhu, J. Phthalates in cosmetic and personal care products: Concentrations and possible dermal exposure. *Environ. Res.* 2011, 111, 329–336.
  64. Luongo, G.; Östman, C. Organophosphate and phthalate esters in settled dust from apartment buildings in Stockholm. *Indoor Air* 2016, 26, 414–25.
  65. Brouwere, K.; Standaert, A.; Torfs, R. Integrated Exposure for Risk Assessment in Indoor Environment (INTERA). Final Report. Boeretang, Belgium, 2012.
  66. Kolarik, B.; Bornehag, C.G.; Naydenov, K.; Sundell, J.; Stavova, P.; Nielsen, O.F. The concentrations of phthalates in settled dust in Bulgarian homes in relation to building

- characteristic and cleaning habits in the family. *Atmos. Environ.* 2008, 42, 8553–8559.
67. Xu, Y.; Cohen Hubal, E.A.; Little, J.C. Predicting residential exposure to phthalate plasticizer emitted from vinyl flooring: sensitivity, uncertainty, and implications for biomonitoring. *Environ. Health Perspect.* 2010, 118, 253–8.
  68. Weschler, C.J. Indoor/outdoor connections exemplified by processes that depend on an organic compound's saturation vapor pressure. In *Proceedings of the Atmospheric Environment*; Elsevier Ltd, 2003; Vol. 37, pp. 5455–5465.
  69. Santillo, D.; Labunska, I.; Davidson, H.; Johnston, P.; Strutt, M.; Knowles, O. Consuming Chemicals–Hazardous Chemicals in House Dust as an Indicator of Chemical Exposure in the Home Greenpeace Research Laboratories; 2003, GRL-TN-01-2003.
  70. European Chemicals Agency Evaluation of new scientific evidence concerning DINP and DIDP in relation to entry 52 of Annex XVII to REACH Regulation (EC) No 1907/2006; Final review report. ECHA-13-R-07-EN. Helsinki, Finland, 2013.
  71. Fromme, H.; Lahrz, T.; Piloty, M.; Gebhart, H.; Oddoy, A.; Rüden, H. Occurrence of phthalates and musk fragrances in indoor air and dust from apartments and kindergartens in Berlin (Germany). *Indoor Air* 2004, 14, 188–95.
  72. Gevao, B.; Al-Ghadban, A.N.; Bahloul, M.; Uddin, S.; Zafar, J. Phthalates in indoor dust in Kuwait: implications for non-dietary human exposure. *Indoor Air* 2013, 23, 126–33.
  73. Kanazawa, A.; Saito, I.; Araki, A.; Takeda, M.; Ma, M.; Saijo, Y.; Kishi, R. Association between indoor exposure to semi-volatile organic compounds and building-related symptoms among the occupants of residential dwellings. *Indoor Air* 2010, 20, 72–84.
  74. Yadav, I.C.; Devi, N.L.; Li, J.; Zhang, G. Polycyclic aromatic hydrocarbons in house dust and surface soil in major urban regions of Nepal: Implication on source apportionment and toxicological effect. *Sci. Total Environ.* 2018, 616-617, 223–235.
  75. Mahfouz, M.M.; Hassan, H.M.; Elobaid, E.A.; Yigiterhan, O.; Alfoldy, B. PAH concentrations and exposure assessment from house dust retained in air-conditioning filters collected from Greater Doha, Qatar. *Environ. Geochem. Health* 2019, 1–13.
  76. Mannino, M.R.; Orecchio, S. Polycyclic aromatic hydrocarbons (PAHs) in indoor dust matter of Palermo (Italy) area: Extraction, GC-MS analysis, distribution and sources. *Atmos. Environ.* 2008, 42, 1801–1817.
  77. Maertens, R.M.; Yang, X.; Zhu, J.; Gagne, R.W.; Douglas, G.R.; White, P.A. Mutagenic and carcinogenic hazards of settled house dust I: Polycyclic aromatic hydrocarbon content and excess lifetime cancer risk from preschool exposure. *Environ. Sci. Technol.* 2008, 42, 1747–1753.
  78. Qi, H.; Li, W.L.; Zhu, N.Z.; Ma, W.L.; Liu, L.Y.; Zhang, F.; Li, Y.F. Concentrations and sources of polycyclic aromatic hydrocarbons in indoor dust in China. *Sci. Total Environ.* 2014, 491-492, 100–107.
  79. Iwegbue, C.M.A.; Obi, G.; Uzoekwe, S.A.; Egobueze, F.E.; Odali, E.W.; Tesi, G.O.; Nwajei, G.E.; Martincigh, B.S. Distribution, sources and risk of exposure to polycyclic aromatic hydrocarbons in indoor dusts from electronic repair workshops in southern Nigeria. *Emerging Contaminants*, 2019, 5, 23–30.
  80. Ma, Y.; Harrad, S. Spatiotemporal analysis and human exposure assessment on polycyclic aromatic hydrocarbons in indoor air, settled house dust, and diet: A review. *Environ. Int.* 2015, 84, 7–16.
  81. Driver, J.H.; Konz, J.J.; Whitmyre, G.K. Soil adherence to human skin. *Bull. Environ. Contam. Toxicol.* 1989, 43, 814–820.

82. Abdullahi, K.L.; Delgado-Saborit, J.M.; Harrison, R.M. Emissions and indoor concentrations of particulate matter and its specific chemical components from cooking: A review. *Atmos. Environ.* 2013, 71, 260–294.
83. Vicente, E.D.; Alves, C.A. An overview of particulate emissions from residential biomass combustion. *Atmos. Res.* 2018, 199, 159–185.
84. Cheruyiot, N.K.; Lee, W.J.; Mwangi, J.K.; Wang, L.C.; Lin, N.H.; Lin, Y.C.; Cao, J.; Zhang, R.; Chang-Chien, G.P. An overview: Polycyclic aromatic hydrocarbon emissions from the stationary and mobile sources and in the ambient air. *Aerosol Air Qual. Res.* 2015, 15, 2730–2762.
85. Ong, S.; Ayoko, G.; Kokot, S.; Morawska, L. Polycyclic aromatic hydrocarbons in house dust samples: Source identification and apportionment. In *Proceedings of the 14th International IUAPPA World Congress*; Brisbane, Australia, 2007.
86. Ali, N.; Ismail, I.M.I.; Khoder, M.; Shamy, M.; Alghamdi, M.; Costa, M.; Ali, L.N.; Wang, W.; Eqani, S.A.M.A.S. Polycyclic aromatic hydrocarbons (PAHs) in indoor dust samples from Cities of Jeddah and Kuwait: Levels, sources and non-dietary human exposure. *Sci. Total Environ.* 2016, 573, 1607–1614.
87. Zha, Y.; Zhang, Y.L.; Tang, J.; Sun, K. Status, sources, and human health risk assessment of PAHs via foliar dust from different functional areas in Nanjing, China. *J. Environ. Sci. Heal. - Part A Toxic/Hazardous Subst. Environ. Eng.* 2018, 53, 571–582.