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Compositional Pattern and Human Exposure Risk of Polycyclic Aromatic Hydrocarbons (PAHs) in Fine Dust from Indoor Environments in Port Harcourt, Nigeria

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ABSTRACT: Indoor fine dusts in residential buildings, public buildings and vehicles from Port Harcourt, Nigeria were assessed for their polycyclic aromatic hydrocarbons (PAHs) concentrations, compositional patterns, and human exposure risks. The PAHs in the dust samples were quantified by gas chromatograph equipped with mass spectrometry (GC-MS) after extraction by ultra-sonication with hexane/dichloromethane/acetone and cleaned up on a silica gel/alumina column. The Σ16 PAH concentrations in these indoor fine dusts ranged from 3136 µg kg^{-1} to 44332 µg kg^{-1}, 2580 µg kg^{-1} to 15372 µg kg^{-1} and 7248 µg kg^{-1} to 11246 µg kg^{-1} for residential buildings, public buildings and vehicles respectively. The compositional pattern of the PAHs in the indoor fine dusts from the residential buildings was in the order: 5>3>6>4>2 rings, while in the public buildings and vehicles, the compositional patterns followed the order: 3>5>6>4>2 rings, and 5>6>3>4>2 rings respectively. The BaP_{TEQ} for all the dust samples ranged from 356 to 42321 µg kg^{-1} while the BaP_{MEQ} values varied from 338 to 41209 µg kg^{-1}. The hazard index (HI) values for PAHs in indoor fine dust from these indoor environments were generally <1 and suggests there is no adverse non-carcinogenic risk associated with human exposure to these indoor fine dusts. The total cancer risk values obtained for both children and adults in these indoor fine dusts were greater than the acceptable risk value of 1×10^{-6} except for inhalation pathway and indicated a high potential carcinogenic risk from PAHs exposure in these indoor environments.

Keywords: Port Harcourt, Dust, Residential, Public, Vehicles, PAHs

Introduction

Port Harcourt and environ has always been associated with particulate air pollution due to flared gases from oil and gas facilities and other industrial installation (Osuji and Avwiri, 2005; Antai *et al.*, 2019); however, particulate pollution levels in Port Harcourt metropolis took a worsening turn since 2016 due to plumes of soot (BC) in the environment. The BC which was said to be generated by artisanal refining and other sundry activities, migrates from the outdoor environment to indoors through crevices on buildings, open windows, and doors. The US EPA defined particulate matter (PM) indoors to include particles of outdoor origin that migrate indoors and particles that originate from indoor sources, which are majorly combustion sources (tobacco smoke, firewood smoke, lamp smoke, fireplace, and cooking appliances), commonly used household products such as cleaning products, paints, insecticides, and degraded building materials such as wood and asbestos fibers. The World Health Organization (WHO) in 2020 reported that due to crude cooking and poor lighting practices, about 4 million people died prematurely due to household air pollution. The report indicated that close to half of the

deaths due to pneumonia amongst children under five years of age were caused by the inhalation of BC polluted air in households. Exposure to such particles can affect both the lungs and the heart. Several research works have linked particulate matter exposure to premature death with persons with heart and lung diseases, non-fatal heart attacks, irregular heartbeat, aggravated asthma, decreased lung function, increased respiratory symptoms such as irritation of the airways, coughing or difficulty in breathing (Paulin and Hansel, 2016; Bouazza *et al*., 2017; Liu *et al*., 2018).

PM (soot, dust, etc.) that migrate indoors from outdoors, settle as fine dust after mixing with indoor particles and contaminants. The fine particles which are usually 2.5 µm and below are not visible to the naked eyes but settles on household installations at heights and appliances, fans, as well as being absorbed by air conditioner filters in homes where they are deployed for indoor air conditioning. Many compounds in dust such as PAHs are known to be associated with hormone disruption, cancer, and reproductive damage according to human epidemiological cell studies (Zhang *et al*., 2016; Vondracek *et al*., 2018; Ramesh *et al*., 2022; Ravanbakhsh *et al*., 2023). These compounds in dust can be absorbed through skin, inhaled, or ingested when humans put dusty hands into their mouths. PAHs are constituents of indoor dust due to the presence of BC and household chemicals that contain organic substances. PAHs are a group of persistent organic pollutants (POPs) produced primarily due to incomplete combustion of fossil fuels, biofuels and vegetation fumes. They are lipophilic and therefore are rapidly distributed and ubiquitous. They can remain in the environment for a long time. PAHs are a group of carbon-based fused rings that have two to six aromatic compounds (Tesi *et al*., 2016). There are several PAHs compounds but for the purposes of their impact on man and the environment, sixteen (16) out of the lot have been identified by US EPA as priority environmental pollutants. These 16 PAHs compounds comprise of two to six aromatic rings with molecular weight range of 128 to 278 g/mol (Chukwuogene *et al*., 2021). These compounds are identified as priority pollutants as they are known to be persistent, non-biodegradable and have the capacity to cause deleterious environmental and health challenges. They are found to be carcinogenic, mutagenic, and genotoxic. They are ranked based on the benzene ring available, as low molecular weight (LMW) or high molecular weight (HMW). LMW PAHs have either two or three fused rings (e.g. naphthalene, acenaphthylene, fluorene, phenanthrene, anthracene and acenaphthene) while HMW PAHs have four to six fused rings (e.g. fluoranthene, chrysene, pyrene, benzo (a) anthracene, benzo (b) fluoranthene, benzo (k) fluoranthene, dibenzo (a,h) anthracene, indenol (1,2,3-cd) pyrene and benzo (g,h,i) perylene. PAHs can be from petrogenic or pyrogenic sources. Petrogenic PAHs are commonly LMW compounds while pyrogenic PAHs are made up of HMW compounds. The mass ratio of LMW/HMW has been generally utilized as the standard for distinguishing petrogenic from pyrogenic sources (Soclo, *et al*., 2000; De Luca *et al*., 2005). The ratios of LMW/HMW higher than 1 specifies petrogenic source (fossil fuel emissions) while the ratios of LMW/HMW lower than 1 indicates pyrogenic source (fossil fuel combustion).

The International Agency for Research on Cancer (IARC) classified most of the HMW PAHs as more carcinogenic than the LMW PAHs except for naphthalene. The IARC classified Benzo (a) Pyrene as group 1 (carcinogenic) and Dibenzo (a, h) Anthracene as group 2A (probably carcinogenic) while Benzo(a) Anthracene, Chrysene, Benzo (b) Fluoranthene, Benzo (k) Fluoranthene, Indeno (1,2,3- c,d) Pyrene and Naphthalene as group 2B (possibly carcinogenic) (Straif *et al*., 2005). Since the advent of BC pollution in Port Harcourt, the human exposure risk of PAHs in the outdoor environment has been greatly studied. Several studies have linked exposure to PAHs to different types of cancer (Okona-Mensah *et al*., 2005; Alhamdow *et al*., 2019; Gamboa-Loira *et al*., 2022). A study on the incremental lifetime cancer risk (ILCR) of PAHs of Port Harcourt BC using the three pathways of exposure- ingestion, inhalation, and dermal contact indicated that people living in the research area where artisanal refining activities take place were more exposed to high carcinogenic risk with the likelihood of one incidence of cancer in every thirty (30) persons in the wet season and one (1) incidence of cancer in eight hundred and seventy (807) persons in the dry season (Oloyede *et al*., 2020). Kanee *et al* (2020) in a study carried out in Port Harcourt established using Wister Rats that the effect of PAHs was more prevalent in the outdoor environment than indoors, as there were more incidences of dead rats in the outdoor during the experiment. This implies that residents were more pre-disposed to PAHs morbidity and mortality outside than inside. They observed oxidative stress and tissue toxicity in the rats beyond the threshold and immune depression caused by oxidative stress and changes in DNA methylation and expression of specific genes, as absolute care was taken to prevent physical stress and trauma to the rats before the experiment. The study observed that these findings could explain hematologist sudden notice of myelofibrosis in some young residents of Port Harcourt engaged in artisanal refining. These clinician observations negate empirical evidence of myelofibrosis found to be common in older folks over sixty years. It is a fact that humans spend most of their time indoors than outdoors, however, most of the research on Port Harcourt BC had dwelt on the outdoor environment. Consequently, it became expedient to conduct a study to determine the exposure effect of PAH in indoor fine dust on humans. Thus, the objectives of this study were to determine the compositional pattern and risks of PAHs in indoor fine dust from different indoor environment in Port Harcourt, Nigeria.

Materials and methods

Study area: The study area (Figure 1), Port Harcourt lies along the Bonny River, and it is located on longitude 4º 49′ 27″ N and latitude 7º 2′ 1″ E. It is the capital city of River State, one of the oil producing states of the Niger Delta region of Nigeria.

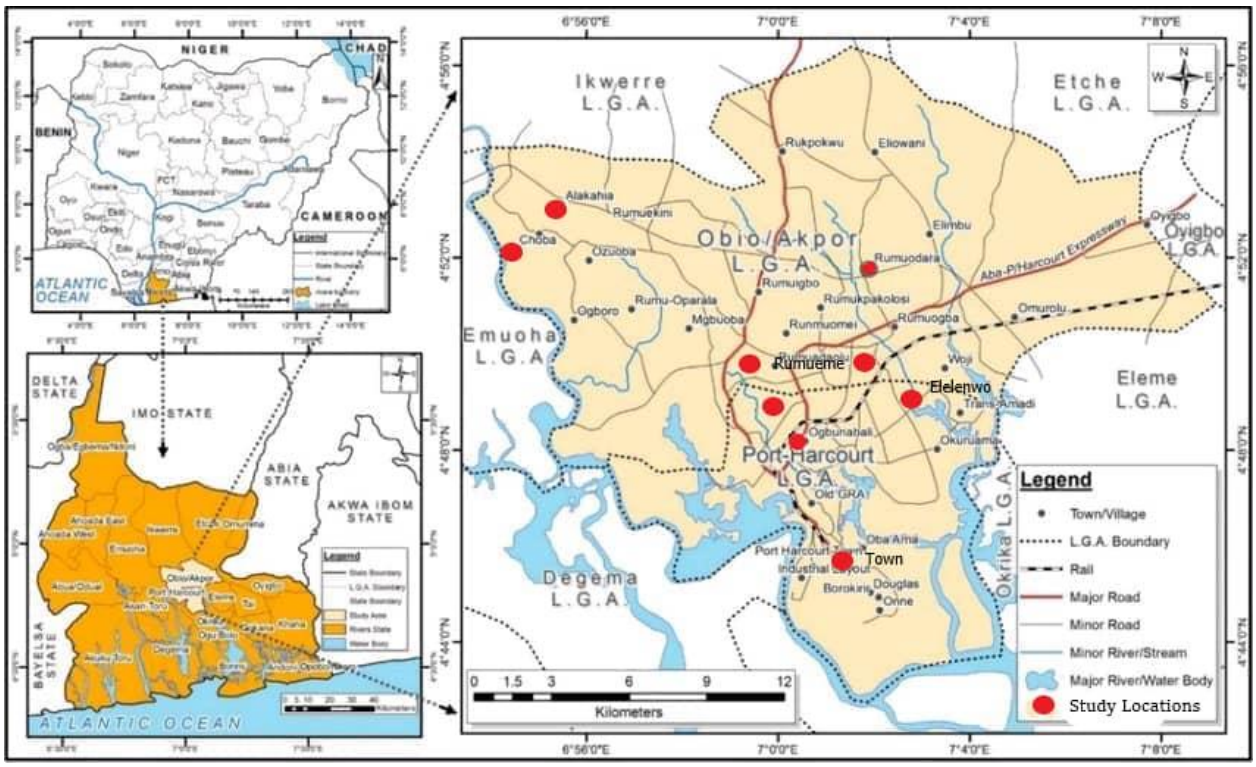

Figure 1: Map of Port Harcourt and its environs showing the sampling points of the study

The city which is the fourth largest city in Nigeria after Lagos, Kano and Ibadan is cosmopolitan in nature. Port Harcourt metropolis is majorly comprised of two local government areas (Obio-Akpor and Port Harcourt) out of the twenty-three (23) local government areas of the State. It has a 2021 United Nations estimated population of 3, 17, 076 inhabitants as against the 1,382,592 recorded in the 2006 census conducted in Nigeria. The city which is surrounded by riverine and upland communities has a lot of adjoining creeks, tributaries, estuaries, and mangroves. The riverine area is majorly part of the old Port Harcourt Township, which spans from Old GRA, Trans-Amadi industrial layout, Abuloma and Borokiri while places like Rumueme, Rukpoku, Rumuokoro, Rumuodamaya, Choba, etc. are upland and part of Obio-Akpor local government area. The areas for this study were carefully selected based on the population, ongoing activities, and the environment. Abuloma, Diobu and Town in Port Harcourt local government area were chosen due to their huge population and their closeness to illegal artisanal refining sites as well as slaughterhouses while Choba in Obio-Akpor local government area, aside from its large population, also hosted asphalt production factories.

Sample collection: The indoor fine dust samples were collected from Port Harcourt metropolis (Abuloma, Elekahia, Diobu, Rumueme, Choba, Elelenwo, Runuodara and Runuodomaya). A total number of thirty (30) samples were collected from the study areas, comprising 20 samples from residential apartments, 7 samples from public buildings and 3 samples from vehicles. The accumulated dust samples were collected by brushing them off, standing/ceiling fans, window blinds, wall brackets, air-conditioners' filters, etc. while the samples from the vehicles were collected only from the air-conditioners' filters. Samples were stored in a zip-lock bag, labelled properly, and transported to the laboratory for analysis. As a quality control measure, a new brush was used for each sample collection.

Chemicals and reagents: Acetone, n-hexane, and dichloromethane (high performance liquid chromatography grade) (Sigma-Aldrich, St Louis, MO, USA); silica gel and alumina (analytical grade) (Merck, Darmstadt, Germany); and anhydrous Na_2SO_4 (analytical grade) (Honeywell Fluka, Seelze, Germany) were used as received. A mixed standard solution containing the US EPA 16 PAHs was obtained from AccuStandard Inc. (New Haven, CT, USA). A surrogate standard solution containing six deuterium-labelled PAHs, namely, Nap-d8, Ace-d10, Phen-d10, Chry-d12, DahA-d12, and perylene-d12 (Pery-d12) was purchased from Cambridge Isotope Laboratories Inc.

Sample preparation procedure: PAHs in the samples were extracted by ultrasonication (US EPA Method 3550C). A mass of 5.0 g of dust was homogenized with the same mass of anhydrous Na_2SO_4. A 30 mL aliquot of a 1:1:1 dichloromethane (DCM)/n-hexane/Acetone mixture was added to the homogenate, and the mixture was ultrasonicated for 2 hours at 35 °C. The extracts were then filtered through a 0.45 µm glass microfiber filter, concentrated to 1 mL by rotary evaporation and cleaned-up in a silica gel (4.0 g)/alumina (2.0 g) packed column. A volume of 10 mL of a 1:1:1 DCM/n-hexane/Acetone mixture was then used to elute the PAHs from the column and the eluate was concentrated to 1 mL final volume.

Chromatographic analysis: The PAH concentrations in the sample extracts were determined by gas chromatography-mass spectrometry with an Agilent 6890 gas chromatograph (GC) interfaced with an Agilent 5973 mass selective detector (Agilent Technologies, Santa Clara, USA). A Varian ms-5 capillary column (30 m length ×0.25 µm film thickness × 0.25 mm i.d.) was used for separation, and pure helium gas at a flow velocity of 1mL/min was used as the carrier gas. The gas chromatographic column had an initial temperature of 50 °C for PAHs, which was held for 5mins for PAHs and was then increased at 15°C min^{-1} to 150 °C; it was further raised to 200 °C at 3 °C min^{-1}, and finally increased to 300 °C at 2 °C min^{-1}. The temperature of the injection port, ion source, quadrupole and transfer line were 250, 230,150 and 280 °C respectively. The sample was injected into the GC via a pulsed splitless mode with an injection volume of 3µL for PAHs.

Quality assurance/quality control: The analytical procedure was validated by quantifying an external calibration method. Linear regression coefficient (r^2) values of 0.9991 to 0.9999 were obtained for the PAH calibration lines. As to validate the functionality of the GC, a standard was run, and the results obtained were found to correspond with the previous standard used to calibrate the equipment. Dichloromethane (DCM) was run as solvent blank for the method to be analysed, duplicate sample analysis was carried out to ascertain repeatability and the raw data were corrected with reagent blank. The required temperature for the equipment for the method was attained before the analysis**.** The limits of detection (LOD) and quantification (LOQs) were determined as three and ten times the signal-to-noise ratio of the blanks respectively. The LODs and LOQs for the PAHs ranged from 0.01 to 0.05 µg/kg and 0.03 and 0.15 µg/kg respectively. Glassware was rinsed by n-hexane for three times and then washed in the ultrasonic cleaner for 30 min, baked at 400 °C for five hours in the muffle furnace before use.

Statistical analysis: Analysis of variance (ANOVA) was used to determine if there was significant difference in PAHs concentrations among the different indoor environments.

Human health risk assessment: The human health risks from PAHs exposure in these indoor fine dusts were assessed using *BaP toxicity and mutagenic potencies*, hazard index and total cancer risk models adopted from the United States Environmental Protection Agency (USEPA).

BaP toxicity and mutagenic potencies: The BaP carcinogenic (BaP_{TEQ}) and mutagenic (BaP_{MEQ}) potency due exposure to PAHs in indoor dusts from Port Harcourt were evaluated by comparing the carcinogenic and mutagenic toxicity of the individual PAHs with that of BaP as the reference compound. The equations 1 and 2 were used to calculate the BaP_{TEQ} and BaP_{MEQ} of the PAHs respectively.

$$BaP_{TEQ} = \Sigma Ci \times BaP_{TEF} \quad (1)$$

$$BaP_{MEQ} = \Sigma Ci \times BaP_{MEF} \quad (2)$$

Ci is the concentrations of the individual PAHs and BaP_{TEF} and BaP_{MEF} represent the carcinogenic and mutagenic potency factors for the PAHs. The BaP_{TEF} (USEPA, 2001) and BaP_{MEF} (Durant, 1996) values are provided in Ossai *et al*. (2021) and Iwegbue *et al*. (2018).

Non-carcinogenic and carcinogenic risk assessment: The hazard index (HI) is a measure of non-carcinogenic risk and defined as the sum of the hazard quotients related to human interface with dusts from Port Harcourt through accidental oral ingestion, contact with exposed skin surface and nasal inhalation of dust particles. For non-carcinogenic risk assessment, only the concentrations of the following PAHs; Nap, Acy, Ace, Flu, Phen and Flt were considered, while Nap, BaA, Chry, BbF, BkF, BaP, IndP and DahA concentrations were considered in the evaluation of lifetime cancer risk. The CDI for three exposure routes were computed using the following equations (USEPA, 1989; 2001, 2009).

$$CDI_{ing\text{-}nc} = \frac{C \times IngR \times EF \times ED}{BW \times AT_{nc}} \times 10^{-6} \quad (3)$$

$$CDI_{dermal\text{-}nc} = \frac{C \times SA \times AF \times ABS \times EF \times ED}{BW \times AT_{nc}} \times 10^{-6} \quad (4)$$

$$CDI_{inh\text{-}nc} = \frac{C \times InhR \times EF \times ET \times ED}{PEF \times 24 \times AT_{nc}} \quad (5)$$

$$HQ = \frac{CDI_{nc}}{RfD} \quad (6)$$

$$\text{Hazard index (HI)} = \sum HQ = HQ_{ing} + HQ_{inh} + HQ_{dermal} \quad (7)$$

The cancer risk values associated with exposure to PAHs in dusts through ingestion, inhalation and dermal contact pathway were estimated using equations 8-11.

$$Risk_{ing} = \frac{C \times IngR \times EF \times ED \times CF \times SFO}{BW \times ATca} \quad (8)$$

$$Risk_{inh} = \frac{C \times Inh \times EF \times ED \times IUR}{PEF \times ATca} \quad (9)$$

$$Ris_{derm} = \frac{C \times SA \times AF \times ABS \times EF \times ED \times CF \times SFO \times GIABS}{BW \times ATca} \quad (10)$$

$$ILCR = \Sigma Risk = Risk_{ing} + Risk_{inh} + Risk_{dermal} \quad (11)$$

The definition of terms and values for all variables can be found in Ossai *et al.* (2021) and Iwegbue *et al.* (2018). Usually, HI value above 1 show that there is potential non-cancer risk while total cancer risk of 1×10^{-6} shows there is no cancer risk (USEPA, 2022).

Results

The summary results of the PAHs concentrations in the indoor fine dust from the three indoor environments displayed in Table 1, ANOVA result to find out if there was significant difference in the PAHs concentrations among the indoor environments is shown in Table 2 while the compositional pattern of PAHs in the indoor fine dust is displayed in Figure 2. Tables 3, 4 and 5 showed the computed BaP_{TEQ} and BaP_{MEQ} values, HI and TCR values respectively.

Table 1: Summary statistics of PAHs concentrations in indoor fine dust from Port Harcourt

Table 1 showed that PAH concentrations in the fine dust samples in residential buildings ranged from 3136 µg kg^{-1} to 44332 µg kg^{-1}, public buildings ranged from 2580 µg kg^{-1} to 15372 µg kg^{-1} and vehicles ranged from 7248 µg kg^{-1} to 11246 µg kg^{-1}. The mean ± SD concentrations were 9464 ± 9763µg kg^{-1}, 7907 ± 5015µg kg^{-1} and 9255 µg kg^{-1} ± 1999 µg kg^{-1} for residential buildings, public buildings, and vehicles respectively.

	Residential Buildings						Public Buildings						Vehicles					
	MEAN	SD	MEDIAN	MIN	MAX	CV%	MEAN	SD	MEDIAN	MIN	MAX	CV%	MEAN	SD	MEDIAN	MIN	MAX	CV%
Nap	70	168	25	10	774	240	32	28	18	14	88	87	70	0	70	70	70	0
Acy	74	58	58	8	252	79	44	45	22	4	122	103	193	20	204	170	204	10
Ace	258	253	162	4	802	98	243	254	108	48	632	104	364	139	284	284	524	38
Flu	1669	1582	1041	56	6004	95	2889	2618	2006	522	6680	91	203	231	70	70	470	114
Phen	624	1900	103	12	8616	304	567	1133	146	24	3116	200	315	136	268	208	468	43
Ant	526	441	419	32	1644	84	663	395	450	304	1392	60	267	133	200	180	420	50
Flt	66	43	61	2	168	64	62	41	70	6	114	65	259	115	192	192	392	45
Pyr	63	39	65	10	142	61	43	32	34	10	92	75	221	110	168	148	348	50
BaA	142	95	125	18	306	67	122	54	144	18	162	44	209	92	156	156	316	44
Chry	210	209	151	0	958	100	131	77	118	48	240	59	315	209	196	192	556	66
BbF	394	356	295	96	1464	90	407	438	190	102	1174	108	633	46	606	606	686	7
BkF	569	330	499	144	1226	58	386	250	390	120	806	65	696	35	716	656	716	5
BaP	2660	8963	437	112	40492	337	428	472	264	52	1436	110	349	129	296	256	496	37
DahA	913	753	554	236	2580	82	973	736	702	190	1898	76	2274	471	2546	1730	2546	21
IndP	619	382	512	56	1776	62	576	283	478	328	1140	49	1669	479	1946	1116	1946	29
BghiP	608	813	400	0	3808	134	341	257	260	52	798	75	1219	133	1174	1114	1368	11
Total	9464	9763	6028	3136	44332	103	7907	5015	7120	2580	15732	63	9255	1999	9272	7248	11246	22
2R	70	168	25	10	774	240	32	28	18	14	88	87	70	0	70	70	70	0
3R	3151	3108	1889	356	12238	99	4406	4016	2758	952	11510	91	1341	647	1026	912	2086	48
4R	482	288	386	172	1374	60	358	140	334	184	544	39	1004	527	708	692	1612	52
5R	4535	9089	1901	1152	42508	200	2194	1225	2390	692	3980	56	3952	550	4164	3328	4364	14
6R	1227	1135	817	294	5584	93	917	389	1090	424	1368	42	2888	578	3120	2230	3314	20
7C	5506	9197	2710	1646	43606	167	3023	1481	3130	1104	4954	49	6145	1183	6662	4792	6982	19

Table 2: Analysis of variance (ANOVA) result comparing PAHs in dusts from the three indoor environments

Source of Variation	SS	df	MS	F	P-value	F crit
Between Groups	89392.63	2	44696.31	9.100189	0.0204867	3.204317
Within Groups	20075298	45	446117.7			
Total	20164691	47				

From Table 2, the F-calculated was 9.100189 and was greater than the F-critical of 3.204317 while the p-value of 0.0204867 was lower than 0.05. These indicate that there was significant difference in the concentrations of PAHs in the three indoor environments.

Figure 2: Compositional pattern of PAHs in the indoor fine dust

As shown in Figure 2, the PAHs compositional pattern and profiles in the indoor fine dusts from the residential buildings was in the order: 5 > 3 > 6 > 4 >2 rings, while in the public buildings and vehicles, the occurrence patterns followed the order: 3 > 5 > 6 > 4 > 2 rings, and 5 > 6 > 3 > 4 >2 rings respectively.

Table 3: BaP_{TEQ} and BaP_{MEQ} concentrations (µg kg^{-1}) in the indoor dusts

	BaA	Chry	BbF	BkF	BaP	IndP	DahA	BapTEQ	BaA	Chry	BbF	BkF	BaP	IndP	DahA	BapMEQ
ABL1	26.0	0.07	17.4	12.3	722	67.0	258	**1103**	21.3	1.16	43.5	135	722	208	74.8	**1205**
ABL2	9.40	0.08	31.0	3.3	308	72.4	582	**1006**	7.71	1.39	77.5	36.7	308	224	169	**825**
RUM1	30.6	0.40	146	11.5	550	178	2446	**3363**	25.1	6.77	366	127	550	551	709	**2335**
RUM2	28.0	0.00	101	4.3	392	60.2	484	**1070**	23.0	0.00	254	47.1	392	187	140	**1043**
CHB1	10.0	0.07	31.0	6.0	1036	29.4	1850	**2963**	8.20	1.26	77.5	66.4	1036	91	537	**1817**
CHB2	16.2	0.96	29.6	7.7	1054	121	2580	**3809**	13.3	16.29	74.0	85.1	1054	374	748	**2365**
DBU1	29.6	0.28	34.4	6.5	166	38.8	1666	**1942**	24.3	4.69	86.0	71.7	166	120	483	**956**
DBU2	29.6	0.37	13.0	1.4	40492	42.8	1742	**42321**	24.3	6.36	32.5	15.8	40492	133	505	**41209**
DBU3	11.8	0.07	12.2	10.5	4844	104	1440	**6423**	9.68	1.22	30.5	116	4844	322	418	**5741**
ELK1	1.80	0.12	23.2	1.4	704	43.6	236	**1010**	1.48	2.07	58.0	15.8	704	135	68.4	**985**
ELK2	16.0	0.14	19.0	8.1	112	41.4	354	**551**	13.1	2.31	47.5	88.7	112	128	103	**495**
ELW1	5.60	0.06	100	2.7	222	74.8	498	**903**	4.59	1.09	250	29.9	222	232	144	**884**
ELW2	5.40	0.13	9.6	9.5	200	5.60	602	**832**	4.43	2.18	24.0	105	200	17.4	175	**527**
RDR1	6.40	0.17	18.8	3.2	160	22.0	526	**737**	5.25	2.82	47.0	35.4	160	68.2	153	**471**
RDR2	9.20	0.22	14.8	4.8	208	45.4	746	**1028**	7.54	3.77	37.0	53.2	208	141	216	**667**
RMD1	13.8	0.32	38.2	5.6	270	51.2	324	**703**	11.3	5.37	95.5	61.8	270	159	94	**697**
RMD2	13.8	0.32	28.2	2.8	270	51.2	324	**690**	11.3	5.37	70.5	30.4	270	159	94	**640**
TWN1	13.2	0.09	40.2	2.2	482	40.4	850	**1428**	10.8	1.46	101	24.2	482	125	247	**991**
TWN2	4.20	0.17	29.4	5.1	504	74.2	370	**987**	3.44	2.82	73.5	56.5	504	230	107	**978**
TWN3	4.20	0.17	50.8	4.5	504	74.2	372	**1010**	3.44	2.82	127	49.9	504	230	108	**1025**
ABL3	15.4	0.23	10.2	3.9	1436	72.8	1426	**2965**	12.6	3.91	25.5	42.9	1436	226	414	**2160**
RUM3	16.2	0.24	117	6.1	344	57.2	1854	**2395**	13.3	4.08	294	66.9	344	177	538	**1437**
CHB3	14.4	0.12	10.8	1.2	264	47.8	1898	**2236**	11.8	2.01	27.0	13.2	264	148	550	**1017**
ELK3	16.0	0.14	19.0	8.1	52	41.4	354	**491**	13.1	2.31	47.5	88.7	52	128	103	**435**
ELW3	7.80	0.08	89.6	4.0	544	114	702	**1461**	6.40	1.36	224	44.0	544	353	204	**1377**
RDR3	13.8	0.05	20.8	1.6	242	37.2	388	**703**	11.3	0.82	52.0	17.6	242	115	113	**552**
RMD3	1.80	0.07	17.0	2.2	112	32.8	190	**356**	1.48	1.12	42.5	24.2	112	102	55.1	**338**
PHV1	15.6	0.20	60.6	7.2	496	195	2546	**3320**	12.8	3.33	152	78.8	496	603	738	**2084**
PHV2	31.6	0.56	60.6	7.2	296	195	2546	**3137**	25.9	9.45	152	78.8	296	603	738	**1903**
PHV3	15.6	0.19	68.6	6.6	256	112	1730	**2189**	12.8	3.26	172	72.2	256	346	502	**1363**
MEAN	**14.4**	**0.20**	**42.1**	**5.39**	**1908**	**71.4**	**1063**	**3104**	**11.8**	**3.43**	**105**	**59**	**1908**	**221**	**308**	**2617**
MIN	**1.80**	**0.00**	**9.60**	**1.20**	**52.0**	**5.60**	**190**	**356**	**1.48**	**0.00**	**24.0**	**13.2**	**52.0**	**17.4**	**55.1**	**338**
MAX	**31.6**	**0.96**	**146**	**12.3**	**40492**	**195**	**2580**	**42321**	**25.9**	**16.3**	**366**	**135**	**40492**	**603**	**748**	**41209**

Table 3 showed that the BaP_{TEQ} for dusts from the three indoor environments varied from 356 to 42321 µg kg^{-1} with an average of 3104 µg kg^{-1} while the BaP_{MEQ} values varied from 338 to 41209 µg kg^{-1} with an average of µg kg^{-1}.

Table 4: Hazard index of PAHs in the dust from the three indoor environments

		CHILD				ADULT			
		HQING	**HQINH**	**HQDERM**	**HI**	**HQING**	**HQINH**	**HQDERM**	**HI**
Residential	ABL1	8.29E-01	1.91E-04	2.36E-03	**8.32E-01**	7.77E-02	7.06E-05	3.15E-04	**7.81E-02**
	ABL2	1.52E-01	7.68E-05	4.36E-04	**1.53E-01**	1.43E-02	2.59E-05	5.83E-05	**1.44E-02**
	RUM1	3.96E+00	1.78E-03	1.37E-02	**3.97**	3.71E-01	7.39E-04	1.82E-03	**3.74E-01**
	RUM2	2.41E+00	3.03E-04	6.91E-03	**2.41**	2.26E-01	1.19E-04	9.23E-04	**2.27E-01**
	CHB1	2.10E+00	3.72E-04	6.22E-03	**2.11**	1.97E-01	1.38E-04	8.31E-04	**1.98E-01**
	CHB2	1.90E+00	4.23E-04	5.45E-03	**1.9**	1.78E-01	1.58E-04	7.28E-04	**1.79E-01**
	DBU1	4.57E-01	1.45E-04	1.32E-03	**4.59E-01**	4.29E-02	5.32E-05	1.76E-04	**4.31E-02**
	DBU2	1.11E-01	5.49E-05	3.30E-04	**1.11E-01**	1.04E-02	1.84E-05	4.41E-05	**1.04E-02**
	DBU3	4.23E-01	1.00E-04	1.20E-03	**4.24E-01**	3.96E-02	3.50E-05	1.61E-04	**3.98E-02**
	ELK1	1.07E+00	1.62E-04	3.00E-03	**1.07**	1.00E-01	6.19E-05	4.01E-04	**1.01E-01**
	ELK2	8.83E-01	1.29E-04	2.52E-03	**8.86E-01**	8.28E-02	5.13E-05	3.36E-04	**8.32E-02**
	ELW1	3.16E-01	8.94E-05	8.98E-04	**3.17E-01**	2.97E-02	3.35E-05	1.20E-04	**2.98E-02**
	ELW2	2.80E-01	1.19E-04	7.92E-04	**2.81E-01**	2.63E-02	4.32E-05	1.06E-04	**2.64E-02**
	RDR1	4.26E-01	9.00E-05	1.20E-03	**4.27E-01**	3.99E-02	3.34E-05	1.61E-04	**4.01E-02**
	RDR2	3.72E-01	7.91E-05	1.07E-03	**3.73E-01**	3.49E-02	2.94E-05	1.42E-04	**3.51E-02**
	RMD1	3.15E-01	5.66E-05	8.86E-04	**3.16E-01**	2.95E-02	1.98E-05	1.18E-04	**2.97E-02**
	RMD2	2.59E-01	5.04E-05	7.31E-04	**2.60E-01**	2.43E-02	1.76E-05	9.76E-05	**2.44E-02**
	TWN1	5.33E-01	8.21E-05	1.53E-03	**5.35E-01**	5.00E-02	3.08E-05	2.05E-04	**5.02E-02**
	TWN2	7.66E-01	1.37E-04	2.26E-03	**7.68E-01**	7.18E-02	5.31E-05	3.03E-04	**7.22E-02**
	TWN3	8.19E-01	1.43E-04	2.41E-03	**8.22E-01**	7.68E-02	5.55E-05	3.22E-04	**7.72E-02**
Public	ABL3	5.83E-01	1.04E-04	1.76E-03	**5.84E-01**	5.46E-02	3.99E-05	2.35E-04	**5.49E-02**
	RUM3	1.17E+00	2.10E-04	3.35E-03	**1.18**	1.10E-01	8.08E-05	4.47E-04	**1.11E-01**
	CHB3	3.35E+00	5.62E-04	1.03E-02	**3.36**	3.14E-01	2.35E-04	1.38E-03	**3.16E-01**
	ELK3	7.68E-01	1.16E-04	2.19E-03	**7.70E-01**	7.20E-02	4.50E-05	2.93E-04	**7.23E-02**
	ELW3	2.37E+00	2.84E-04	6.65E-03	**2.38**	2.22E-01	1.08E-04	8.88E-04	**2.23E-01**
	RDR3	2.37E-01	5.77E-05	6.75E-04	**2.38E-01**	2.22E-02	2.05E-05	9.01E-05	**2.23E-02**
	RMD3	2.86E-01	7.22E-05	8.10E-04	**2.87E-01**	2.68E-02	2.75E-05	1.08E-04	**2.69E-02**
Vehicles	PHV1	3.90E-01	1.85E-04	1.17E-03	**3.91E-01**	3.65E-02	6.47E-05	1.56E-04	**3.68E-02**
	PHV2	7.91E-01	2.32E-04	2.35E-03	**7.94E-01**	7.42E-02	8.72E-05	3.15E-04	**7.46E-02**
	PHV3	3.71E-01	1.78E-04	1.10E-03	**3.72E-01**	3.48E-02	6.31E-05	1.47E-04	**3.50E-02**
	MEAN	9.57E-01	2.19E-04	2.85E-03	**9.59E-01**	8.97E-02	8.53E-05	3.81E-04	**9.02E-02**
	MIN	1.11E-01	5.04E-05	3.30E-04	**1.11E-01**	1.04E-02	1.76E-05	4.41E-05	**1.04E-02**
	MAX	3.96	1.78E-03	1.37E-02	**3.97**	3.71E-01	7.39E-04	1.82E-03	**3.74E-01**

Table 4 showed that the HQ values arising from human exposure to PAHs in the indoor fine dust from these indoor environments followed the order: $HQ_{ing} > HQ_{derm} > HQ_{inh}$ while the HI values ranged from 1.11×10^{-1} to 3.97 and 1.04×10^{-1} to 3.74×10^{-1} for children and adults respectively.

Table 5: Total cancer risk of PAHs in the dust from the three indoor environments

		CHILD				ADULT			
		ILCRING	**ILCRINH**	**ILCRDERM**	**Total Cancer Risk**	**ILCRING**	**ILCRINH**	**ILCRDERM**	**Total Cancer Risk**
Residential	ABL1	1.03E-01	2.88E-08	3.75E-02	**1.40E-01**	3.51E-03	2.09E-08	1.82E-03	**5.33E-03**
	ABL2	9.39E-02	2.54E-08	3.42E-02	**1.28E-01**	3.20E-03	1.84E-08	1.66E-03	**4.86E-03**
	RUM1	3.14E-01	8.59E-08	1.14E-01	**4.28E-01**	1.07E-02	6.25E-08	5.55E-03	**1.62E-02**
	RUM2	9.99E-02	2.68E-08	3.63E-02	**1.36E-01**	3.40E-03	1.95E-08	1.77E-03	**5.17E-03**
	CHB1	2.77E-01	7.41E-08	1.01E-01	**3.77E-01**	9.43E-03	5.39E-08	4.89E-03	**1.43E-02**
	CHB2	3.56E-01	9.59E-08	1.29E-01	**4.85E-01**	1.21E-02	6.98E-08	6.29E-03	**1.84E-02**
	DBU1	1.81E-01	5.01E-08	6.60E-02	**2.47E-01**	6.18E-03	3.65E-08	3.20E-03	**9.38E-03**
	DBU2	3.95E+00	9.89E-07	1.44E+00	**5.39E+00**	1.35E-01	7.19E-07	6.98E-02	**2.05E-01**
	DBU3	5.99E-01	1.55E-07	2.18E-01	**8.18E-01**	2.04E-02	1.13E-07	1.06E-02	**3.10E-02**
	ELK1	9.43E-02	2.43E-08	3.43E-02	**1.29E-01**	3.21E-03	1.77E-08	1.67E-03	**4.88E-03**
	ELK2	5.14E-02	1.53E-08	1.87E-02	**7.01E-02**	1.75E-03	1.11E-08	9.09E-04	**2.66E-03**
	ELW1	8.43E-02	2.27E-08	3.07E-02	**1.15E-01**	2.87E-03	1.65E-08	1.49E-03	**4.36E-03**
	ELW2	7.77E-02	2.27E-08	2.83E-02	**1.06E-01**	2.65E-03	1.65E-08	1.37E-03	**4.02E-03**

		CHILD				ADULT			
		ILCRING	ILCRINH	ILCRDERM	Total Cancer Risk	ILCRING	ILCRINH	ILCRDERM	Total Cancer Risk
	RDR1	6.87E-02	1.90E-08	2.50E-02	**9.38E-02**	2.34E-03	1.38E-08	1.22E-03	**3.56E-03**
	RDR2	9.60E-02	2.66E-08	3.49E-02	**1.31E-01**	3.27E-03	1.93E-08	1.70E-03	**4.97E-03**
Residential	RMD1	6.56E-02	1.83E-08	2.39E-02	**8.95E-02**	2.24E-03	1.33E-08	1.16E-03	**3.40E-03**
	RMD2	6.44E-02	1.74E-08	2.35E-02	**8.79E-02**	2.20E-03	1.26E-08	1.14E-03	**3.34E-03**
	TWN1	1.33E-01	3.55E-08	4.85E-02	**1.82E-01**	4.54E-03	2.58E-08	2.36E-03	**6.90E-03**
	TWN2	9.21E-02	2.49E-08	3.35E-02	**1.26E-01**	3.14E-03	1.81E-08	1.63E-03	**4.77E-03**
	TWN3	9.43E-02	2.53E-08	3.43E-02	**1.29E-01**	3.21E-03	1.84E-08	1.67E-03	**4.88E-03**
Public	ABL3	2.77E-01	7.29E-08	1.01E-01	**3.77E-01**	9.43E-03	5.30E-08	4.89E-03	**1.43E-02**
	RUM3	2.24E-01	6.10E-08	8.14E-02	**3.05E-01**	7.62E-03	4.43E-08	3.95E-03	**1.16E-02**
	CHB3	2.09E-01	5.63E-08	7.60E-02	**2.85E-01**	7.12E-03	4.10E-08	3.69E-03	**1.08E-02**
	ELK3	4.58E-02	1.39E-08	1.67E-02	**6.25E-02**	1.56E-03	1.01E-08	8.10E-04	**2.37E-03**
	ELW3	1.36E-01	3.63E-08	4.97E-02	**1.86E-01**	4.65E-03	2.64E-08	2.41E-03	**7.06E-03**
	RDR3	6.57E-02	1.75E-08	2.39E-02	**8.96E-02**	2.24E-03	1.28E-08	1.16E-03	**3.40E-03**
	RMD3	3.32E-02	9.16E-09	1.21E-02	**4.53E-02**	1.13E-03	6.66E-09	5.87E-04	**1.72E-03**
Vehicles	PHV1	3.10E-01	8.42E-08	1.13E-01	**4.23E-01**	1.06E-02	6.12E-08	5.48E-03	**1.60E-02**
	PHV2	2.93E-01	8.00E-08	1.07E-01	**3.99E-01**	9.98E-03	5.82E-08	5.18E-03	**1.52E-02**
	PHV3	2.04E-01	5.60E-08	7.44E-02	**2.79E-01**	6.96E-03	4.07E-08	3.61E-03	**1.06E-02**
	MEAN	2.90E-01	7.57E-08	1.06E-01	**3.95E-01**	9.89E-03	5.50E-08	5.12E-03	**1.50E-02**
	MIN	3.32E-02	9.16E-09	1.21E-02	**4.53E-02**	1.13E-03	6.66E-09	5.87E-04	**1.72E-03**
	MAX	3.95E+00	9.89E-07	1.44E+00	**5.39E+00**	1.35E-01	7.19E-07	6.98E-02	**2.05E-01**

Table 5 showed that the total cancer risk values ranged from 4.53×10^{-2} to 5.39 for children and 1.72×10^{-3} to 2.05×10^{-1} for adults.

Table 6: Comparison of PAH concentrations in indoor dust with those reported in other parts of the world*

Country	Location	Sampling Year	Sampler	No. of Samples	Particle Size	No. of PAHs	∑PAH (µg/kg)	BaP_{TEQ} (µg/kg)	Reference
Nigeria	Port Harcourt	2023	HB	30	Fine dust	16	2580-44332	3104	This Study
Nigeria	Port-Harcourt	2021	HB	20	<63 µm	16	276-9128	-	Ossai *et al*. (2021)
Nigeria	Warri	2016	HB	20	<63 µm	15	4531-111914	3288	Iwegbue *et al*. (2018)
Australia	Brisbane	2003	NA	11	<1 mm	14	7440	106	Ayoko *et al*. (2005)
Brazil	-	2008	HB	9	NA	16	4091	288	Coronas *et al*. (2013)
Canada	Ottawa	2002-2003	VC	51	<150 µm	13	29,300	4724	Maertens *et al*. (2008)
China	Shanghai	2005	HB	27	NA	16	20,674	4393	Ren *et al*. (2006)
China	Hong Kong	[b]	VC	55	<100 µm	16	6070	635	Kang *et al*. (2010)
China	Pearl Delta	2010	VC	55	<100 µm	16	5910	345	Kang *et al*. (2011)
China	Shanghai	2010	HB	22	NA	16	11,575	829	Peng *et al*. (2012)
China	Guangzhou	2010	VC	20	<100 µm	16	5916	788	Wang *et al*. (2013a)
China	Guangzhou	2011-2012	VC	70	<100 µm	16	8130	843	Wang *et al*. (2013b)
China	Qingyang	2011-2012	VC	70	<100 µm	16	34,800	3446	Wang *et al*. (2013b)
China	Changchun	2014-2015	HB	31	NA	16	21,800-329,600	NA	Wang *et al*. (2016)
China	23 Cities	2010	HB	81	NA	16	1000-470,000	NA	Qi *et al*. (2014)
China	Xinxiang	2012	PC	20	NA	16	1470-21,800	NA	Yang ZY *et al*. (2015)
China	Guizhou	2012	HB	88	80 µm	18	2180-14,200	NA	Yang Q *et al*. (2015)
Germany	Berlin	1997-98, 2000[c]	VC	123	Fine dust	18	6140	485	Fromme *et al*. (2004)
Ghana	Cape coast/KEEA	2012	TS	10	NA	15	ND-3240	0.25-218	Essumang *et al*. (2014)
Italy	Palermo	2006	HB	45	NA	16	5111	262	Mannino and Orecchio (2008)
Nigeria	Lagos	2016	HB		NA	16	904-7677	41-719	Oluseyi *et al*. (2016)
Poland	Warsaw	2003-2004	VC	48	<150 µm	16	35,030	2389	Tatur *et al*. (2009)
United Kingdom	Cambridge	2010	VC	1	<63 µm	15	5095	345	Anders *et al*. (2012)
USA	Ohio	1992-1993	HVS3	24	<150 µm	19	115,817	15,530	Chuang *et al*. (1993); Chuang *et al*. (1995)
USA	WA	1992-1993	HVS3	9	<150 µm	16	10,249	1235	Chuang *et al*. (1994)

Country	Location	Sampling Year	Sampler	No. of Samples	Particle Size	No. of PAHs	∑PAH (µg/kg)	BaP_{TEQ} (µg/kg)	Reference
USA	NC	1994	HVS3	24	<150 µm	19	4200	439	Chuang *et al*. (1999)
USA	NC	1995	HVS3	4	<150 µm	19	3936	421	Chuang *et al*. (1997a)
USA	MD	1995-1996	HVS3	126	<150 µm	11	81,190	12,169	Egeghy *et al*. (2005); USEPA (2011)
USA	KY	1995-1996	HVS3	3	<150 µm	19	3034	327	Chuang *et al*. (1996)
USA	NC	1996	HVS3	13	<150 µm	19	3230	286	Chuang *et al*. (1997b)
USA	NC	1996	HVS3	25	<150 µm	10	20,100	3268	Lewis *et al*. (1999)
USA	NC	1997	HVS3	10	<150 µm	19	2729	351	Wilson *et al*. (2001)
USA	NC	1997	HVS3	13	<150 µm	19	2180	267	Wilson *et al*. (2003)
USA	MI/IA/CA/WA	1998-2000	VC	616	<150 µm	7	8570	2103	Camann *et al*. (2002)
USA	MA	1999	VC	6	<150 µm	2	5810	3191	Rudel *et al*. (2003)
USA	MA	1999-2001	VC	120	<150 µm	4	5761	1680	Fromme *et al*. (2004)
USA	TX	2008	HVS3	23	<150 µm	16	73,521	7449	Mahler *et al*. (2010)
USA	CO	2010	VC	3	<63 µm	15	3358	126	Anders *et al*. (2012)

HVS3 = high volume small surface sampler; VC = vacuum cleaner; HB = hand brushing; PC = pre-cleaned cotton; TS = Teflon sheet; NA -Not Available; NR = Not Reported
*Adopted from Iwegbue *et al*. (2018) with some modifications.

Discussion

Concentrations and compositional pattern of PAHs in the indoor fine dust: The concentrations and compositional patterns of PAHs in the indoor fine dusts from the three indoor environments varied significantly (Tables 1 and 2). The differences in the concentrations and compositions may have been influenced by variations in indoor activities, nature of the buildings, proximity to high traffic density routes and artisanal refining activities. The highest concentrations of ∑16 PAHs was found in DBU2 while the lowest ∑16 PAHs concentration was found in RMD3. On the average, the PAH concentrations in the indoor dusts followed the sequence: residential buildings > vehicles > public buildings. A comparison of the PAH concentrations in indoor dusts of this study with those previously reported for a wide variety of indoor environments, but possibly determined following different extraction and chromatographic techniques is shown in Table 6. The PAH concentrations in these samples fall into the same range previously reported for indoor dusts (Table 6). The high molecular weight PAHs (HMW) showed dominance over the low molecular weight PAHs (LMW) in these indoor dusts, which may be related to the fact that LMW tend to be associated with gas-phase partitioning, whereas HMW tend to be associated with particulate phases because of their lipophilic properties (Orrechio, 2011, Tesi *et al*., 2016).

The concentrations of the sum of 2-ring PAH (Nap) ranged from 10 to 774 µg/kg in all the dust samples and accounted for 0.0 to 3.1 % of the ∑16 PAHs concentrations. The concentrations of the sum of 3-ring PAHs (i.e. Acy + Ace + Flu + Phe + Ant) ranged from 356 to 12238 µg/kg in all the dust samples and accounted for 1.3 to 73.2 % of the total PAHs concentrations. The maximum and minimum concentrations of the sum of 3-ring PAH were found at RUM1 and ABL2 respectively. On the average, the 3-rings PAHs were in the order of Flu > Phen > Ant > Ace > Acy. The concentrations of Acy ranged from 4.0 to 252 µg/kg in all the dust samples and makes up 0.03 to 2.4 % of the total PAHs.

Acenapthene ranged from 4.0 to 802 µg/kg in all the dust samples and constituted 0.05 to 6.9 % of the total PAHs. Flu was detected at concentrations ranged from 56 to 6680 µg/kg in all the dust samples and comprised 0.13 to 53.5 % of the total PAHs. The concentrations of Phen and Ant ranged from 12 to 8616 µg/kg and 32 to 1644 µg/kg in all the dust samples and constituted 0.15 to 34.2 % and 0.29 to 14.3 % respectively of the total PAHs. The concentration of the sum of 4ring PAHs (Flt + Pyr + BaA + Chry) ranged from 172 to 1612 µg/kg in all the dust samples and makes up 1.7 to 16.6 % of the total PAHs. On the average, the 4-rings PAHs were in the order of Chry > BaA > Flt > Pyr. Fluoranthene was detected at concentrations which ranged from 2 to 392 µg/kg in all the dust samples and consisted of 0.06 to 3.5 % of the total PAHs concentration. Pyrene was detected at concentrations which ranged from 10 to 348 µg/kg in all the dust samples and makes up 0.02 to 3.1 % of the total PAHs concentration. Benzo(a)anthracene was detected at concentrations which ranged from 18 to 316 µg/kg in all the dust samples. Benzo(a)anthracene constituted 0.29 to 5.3 % of the PAHs concentrations in these dusts. Chrysene concentrations ranged from not detected to 958 µg/kg in all the dust samples and constituted 0.0 to 10.1 % of the total PAHs concentrations. The concentration of the sum of the five ring PAHs (BbF + BkF +

BaP + DahA) ranged from 692 to 42508 µg/kg in all the dust samples. The sum of the five ring PAHs constituted 15.2 to 95.9 % of the total PAH concentrations respectively. On the average, the 5-rings PAHs were in the order of BaP > DahA > BkF > BbF. The concentrations of Benzo(b)fluoranthene and Benzo(k)fluoranthene ranged from 96 to 1464 µg/kg and 120 to 1226 µg/kg respectively in all the dust samples. They make up 0.29 to 23.1 % and 0.32 to 26.4 % of the total PAHs concentrations. The concentrations of BaP ranged from 52 to 40492 µg/kg in all the dust samples, while that of dibenzo(a,h)anthracene ranged from 190 to 2580 µg/kg in all the dust samples. They both constituted 1.03 to 91.3 % and 3.30 to 27.5 % of the total PAHs respectively. The concentration of the sum of the six ring PAHs (IndP+BghiP) ranged from 294 to 5584 µg/kg in all the dust samples and constituted 1.1 to 38.9 % of the total PAHs. On the average, the 6-ring PAHs were in the order IndP>BghiP. The concentration of IndP ranged from 56 to 1946 µg/kg in all the dust samples and makes up 0.97 to 21.0 % of the total PAHs concentrations while the concentration of BghiP ranged from 0 to 3808 µg/kg in all the dust samples and makes up 0.00 to 19.0 % of the total PAHs concentrations.

In summary, the most contaminated location in the study was found in Diobu (DBU-2) while the least was in Elelenwo (ELW-2). The contamination in Diobu may be attributable to the urban nature, high population density, household cooking methods and high vehicular emissions in the area. Diobu, though an urban setting is dominated by low-income houses with single rooms (face-me-I-face-you). Due to poverty, most households in Diobu, use crude cooking implements such as stoves, charcoal and in some extreme cases firewood. These cooking methods, in addition to the use of power generating sets as well as BC from illegal refining sites at riverine creeks close to the area have probably made the indoor fine dusts in these environments to be laden with HMW PAHs. However, Elelenwo which is also located in an urban setting, has better housing types-bungalows, flats, and duplexes. The area is inhabited by a mixture of not so poor and middle-class residents that can afford better cooking (gas cookers) and lighting methods. It is also worthy to note that the vehicular traffic in the area is also lighter than the Diobu area and it is also far removed from areas where artisanal refining activities in Port Harcourt occur. Samples collected from public buildings in the study contained higher LMW PAHs (48%) as against 38% and 15% for residential and vehicles respectively. The reason may be because public buildings such as hotels are cleaned/dusted regularly and thoroughly on daily basis compared to residential buildings, whose occupants may vacuum/sweep floors without dusting certain areas for a long time. The study also revealed that vehicles in the study had higher concentrations of HMW PAHs (85%) as against 62% and 52% for residential and public buildings respectively. The fine dust collected from air conditioner filters of vehicles were dark in colour and resemble pure BC. These dust particles may have entered vehicles when driven in BC/dust charged environments with unwound windows; the particles are then resuspended and absorbed by the air conditioner filters when the vehicle air conditioner is turned on. It is a known fact that highest concentrations of atmospheric PAHs are found in urban environments with high vehicular emissions and scarce dispersion of atmospheric pollutants (Ravindra et al., 2008 and Honedar, *et al*., 2011). The HMW PAHs is an indication of the atmospheric condition of the environment; the results from the vehicles showed that particulates/dust in the atmosphere of Port Harcourt are charged with BC.

Human health risks: The carcinogenic and mutagenic potency of PAHs in the dusts from the indoor environments expressed in terms of BaP_{TEQ} and BaP_{MEQ} are shown in Table 3. The BaP_{TEQ} and BaP_{MEQ} were computed for the seven carcinogenic PAHs (PAH_{7C}). The BaP_{TEQ} and BaP_{MEQ} values for the dusts followed the order: residential building > vehicles > public building. The order of contribution of the seven carcinogenic PAHs (PAH_{7C}) to the BaP_{TEQ} and BaP_{MEQ} values of the indoor dusts followed the order: BaP > DahA > IndP > BbF > BaA > BkF > Chry. The result showed that BaP and DahA are the main contributors to the carcinogenic and mutagenic potencies of these indoor dusts. The BaP_{TEQ} values of the investigated indoor fine dusts for these indoor environments from Port Harcourt correspond to the global trends reported for indoor environments in the literature (Table 6).

The non-cancer risk was expressed in terms of the hazard index (HI), which is the sum of the hazard quotient (HQ) values for the non-dietary ingestion (HQ_{ing}), dermal contact (HQ_{derm}) and inhalation (HQ_{inh}) exposure pathways. The evaluation of the HQ and HI values for the three exposure pathways was based on the concentrations of the eight non-carcinogenic PAHs (NaP, Acy, Ace, Flu, Phen, Ant, Flt and Pyr). The HQ values arising from human exposure to PAHs in the indoor dust from these indoor environments followed the order: $HQ_{ing} > HQ_{derm} > HQ_{inh}$ (Table 4). The HQs and HI for children were found to be higher than for adults. This could be due to high physical contact with dust from peer play times and a smaller body weight of children (Tesi *et al*., 2016; Iwegbue *et al*., 2018). The HI values for PAHs in dust from these indoor environments were generally < 1, which suggests there is no adverse non-carcinogenic risk associated with human exposure to these dusts. However, HI values from five residential buildings (RUM1, RUM2, CHB1, CHB2 and ELK1) and three public buildings (RUM3, CHB3 and ELK3) for children exposure were > 1 indicating that there is considerable non-carcinogenic risk associated with children exposure to the dust particles in these eight buildings.

The total cancer risk reflects the age-specific potential cancer risk associated with human exposure to environmental PAH sources via non-dietary ingestion, inhalation of dust particles and dermal contact routes.

The total cancer risks associated with exposure of children and adults to PAHs from these indoor environments are shown in Table 5. The incremental lifetime cancer risk (ILCR) cancer risks associated with ingestion were of a higher magnitude than those of dermal and inhalation routes which suggests that the ingestion pathway contributes significantly to the cancer risks while inhalation contributed less. Cancer risk in the three sampling points were in the order, residential>vehicles>public. Public buildings (hotels) had the least cancer risk due to domination of LMW PAHs in the fine dust samples. Even though, the risk of contracting cancer in vehicles was higher than those of public buildings, this risk is insignificant because the dust from the vehicles were collected from air-conditioner filters and the only plausible way occupants of vehicles would be affected is by inhalation pathway and not by ingestion or dermal contact. However, cancer risk by inhalation in this study was within the acceptable US EPA risk criteria. Like the HQ and HI, the ILCR and TCR for children were higher than that of adults. This may also be related to the hand-to-mouth habits of children. In addition, the PAH intake by children is greater than that of adults because of their smaller body weights. The total cancer risk values obtained for both children and adults in these indoor environments were greater than the acceptable risk value of 1×10^{-6}, which indicates a high potential carcinogenic risk in these indoor environments. The cancer risk values obtained in this study were like those previously reported from Port Harcourt (Ossai *et al*., 2021) and Warri (Iwegbue *et al*., 2018). The result affirms the need for remedial actions in these indoor environments to reduce the risk of exposure to PAHs. The highest cancer risk for children and adults was obtained in a residential building in Diobu (DBU-2) while the lowest for children was obtained in a public building in Elelenwo (ELW-3) and adults in Rumuodomaya (RMD-3).

Conclusion

This study has investigated the PAHs concentrations, compositional pattern and human exposure risks in indoor fine dusts in residential buildings, public buildings and vehicles from Port Harcourt, Nigeria. The result showed that the PAHs in these indoor fine dusts were comparable to the range reported for indoor dust from around the globe. The compositional pattern of the PAHs in the indoor fine dusts from the residential buildings was in the order: 5 > 3 > 6 > 4 >2 rings, while in the public buildings and vehicles, the compositional patterns followed the order: 3 > 5 > 6 > 4 > 2 rings, and 5 > 6 > 3 > 4 >2 rings respectively. The hazard index (HI) values for PAHs in indoor fine dust from these indoor environments suggested there is no adverse non-carcinogenic risk associated with human exposure to these indoor fine dusts except for 5 residential and 3 public buildings investigated. The cancer risk values obtained indicated a high potential carcinogenic risk from PAHs exposure in these indoor environments for ingestion and dermal pathways of exposure for both children and adults.

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