LEVELS AND HUMAN HEALTH RISK OF POLYCYCLIC AROMATIC HYDROCARBONS (PAHs) IN INDOOR DUST IN HANOI, VIETNAM

HÀM LƯỢNG VÀ ĐÁNH GIÁ RỦI RO SỨC KHOẺ TỚI CON NGƯỜI CỦA HYDROCARBON THƠM ĐA VÒNG (PAHs) TRONG BỤI TRONG NHÀ TẠI HÀ NỘI

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DOI: https://doi.org/10.57001/huih5804.2023.231

ABSTRACT

Polycyclic aromatic hydrocarbons (PAHs) were studied in 15 indoor dust samples collected from 15 dwellings in urban Hanoi and estimated health risk assessment to the local population via dust ingestion and dermal contact. Sixteen PAHs were detected with a detection frequency of 80 - 100%, and a range of concentrations of 22 - 1490ng/g. Benz (j&b) fluoranthene (BbF), fluoranthene (Flt), and chrysene&triphenylen (Chr) were the most dominant compounds with the mean concentration of 1490; 984; 806, and 714ng/g, respectively. The contribution of 3 and 4-ring PAHs dominated the profile of PAHs significantly in the indoor dust. Results calculated benzo(a)pyrene equivalent carcinogenic power (BaPE) showed that 5- and 6-aromatic rings PAHs were major toxicity threats in the carcinogenic index of PAHs in indoor dust. Incremental lifetime cancer risk (ILCR) for adults and young calculated using ingestion and dermal contact was within the limits set by USEPA using the total concentration of 7 PAHs. The estimated ILCR for adults and children are $3.9x10^{-3}$ and $4.2x10^{-3}$, respectively. These results are much higher than the recommended safety limit by EPA being $1x10^{-4}$ which showed that PAHs in indoor dust has a significant long-term effect on adults and young children in urban of Hanoi.

***Keywords*:** *PAHs, indoor dust, health risk assessment, Hanoi*

TÓM TẮT

Trong nghiên cứu này, hydrocacbon thơm đa vòng (PAHs) đã được nghiên cứu trong 15 mẫu bụi trong nhà thu thập từ 15 căn hộ và nhà ở tại nội thành Hà Nội. Và rủi ro sức khỏe tới con người từ phơi nhiễm PAHs đã được đánh giá thông qua việc nuốt phải bụi và tiếp xúc qua da. Mười sáu PAH đã được phát hiện với tần suất phát hiện là 80 - 100% và khoảng nồng độ là 22 - 1490ng/g. Benz (j&b) fluoranthene (BbF), fluoranthene (Flt) và chrysene&triphenylen (Chr) là những hợp chất chiếm ưu thế nhất với nồng độ trung bình tương ứng là 1490; 984; 806 và 714ng/g. Nồng độ của PAH 3 và 4 vòng chiếm đáng kể trong hàm lượng của PAH trong bụi trong nhà. Kết quả tính toán khả năng gây ung thư của benzo(a)pyrene (BaPE) cho thấy PAHs 5 và 6 vòng thơm có khả năng là nguy cơ gây độc chính trong bụi trong nhà. Nguy cơ ung thư suốt đời gia tăng (ILCR) đối với người lớn và trẻ nhỏ được tính toán khi nuốt phải và tiếp xúc với da đều nằm trong giới hạn do USEPA đặt ra khi sử dụng tổng nồng độ 7 PAH. Chỉ số ILCR ước tính cho người lớn và trẻ em lần lượt là $3,9x10^{-3}$ và $4,2x10^{-3}$. Kết quả này cao hơn nhiều so với giới hạn an toàn khuyến cáo của EPA là $1x10^{-4}$, cho thấy PAHs trong bụi trong nhà có ảnh hưởng lâu dài rõ rệt đối với người lớn và trẻ nhỏ ở nội thành Hà Nội.

Từ khóa: *PAHs, bụi trong nhà, đánh giá rủi ro sức khỏe, Hà Nội.*

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Received: 06/9/2023
Revised: 29/10/2023
Accepted: 25/11/2023

1. INTRODUCTION

Polyaromatic hydrocarbons (PAHs) refer to a class of persistent organic compounds that consists of two or more aromatic rings. These chemical compounds exist as the by-product of incomplete combustion of fossil fuels, biomass, and the pyrolysis of organic material (i.e., wood,

tobacco,…) [1-4]. Beside the natural sources (i.e., volcanic activity and forest fire), PAHs are mainly generated from the anthropogenic sources associated with energy consumption activities such as industrial emissions, vehicular emissions,… [1-7]. Once released to the atmosphere, these compounds redistribute between vapor and airborne particles and can be absorbed in settled dust [4, 7, 8]. Generally, PAHs with 2-3 rings are primarily released in the gas phase and deposit directly to the surface by dry gaseous or wet deposition due to their high vapor pressure; while the PAHs with 5-6 rings tend to bound to particles before deposition [9]. As a consequence of deposition, PAHs are ubiquitously found in soil, sediment, and dust [1, 4, 7, 9, 10]. PAHs are classified as carcinogenic by International Agency for Research on Cancer and found to have mutagenic effects as they tend to bioaccumulate in lipid tissue in plants and animals through the food chain [11-13]. Sixteen PAHs have been listed as priority pollutants by the United States Environmental Protection Agency (USEPA) [14].

Indoor dust is found to be a reservoir of organic pollutants including PAHs due to limited ventilation and lack of sunlight [4, 8]. Indoor sources of PAHs can be listed as smoking, burning wood, coal for heating, cooking,…[1, 3, 5, 15]. Since people spend more than 90% of their time indoors, they are highly exposed to PAHs via inhalation, dust ingestion and dermal contact in which oral intake and inhalation are the main pathways, especially for a toddler since their hand-to-mouth habits. Hence, it is necessary to provide information on the health risks of PAHs exposure.

In terms of Vietnam, there were few studies on PAHs in indoor dust have been conducted. Hoang et al. [16] analyzed 16 PAHs in settled house dust and road dust in urban Hanoi and found that the concentration was around 830 - 3500ng/g and 1400 - 4700ng/g, respectively. The BaP-EQs, an index in risk assessment to estimate the carcinogenicity equivalent to Benzo(a)pyrene, ranged from 81 to 850ng/g with the main contributor of dibenz[a,h]anthracene. High-molecular-weight PAHs had a higher proportion in almost all the samples, suggesting that the combustion process was the dominant source rather than the petrogenic source. The occurrence of 15 PAHs were investigated in 32 indoor and outdoor PM2.5 samples collected from 16 nursery schools in Hanoi, the results showed mean concentrations of total 15 PAHs in indoor and outdoor PM2.5 were 267.1ng/m^3 and 843.4ng/m^3, respectively [17]. However, the data on the occurrence of PAHs in indoor dust still rare.

For these reasons, the objectives of this research are: (1) to determine the concentration and profile of PAHs in indoor dust in urban Hanoi; (2) to distinguish the sources of PAHs in indoor dust; (3) to assess the health risk via ingestion and dermal contact based on the estimation of benzo(a)pyrene equivalent carcinogenic power (BaPE) and incremental lifetime cancer risk (ILCR).

2. MATERIALS AND METHODS

2.1. Chemicals

Sixteen PAHs including naphthalene (Naph), acenaphthylene (Acy), acenaphthene (Ace), fluorene (Flu), phenanthrene (Phe), anthracene (Ant), fluoranthene (Flt), pyrene (Pyr), benzo (a) anthracene (BaA), chrysene & triphenylene (Chr), benzo (j&b) fluoranthene (BbF), benzo (k) fluoranthene (BkF), benzo (a) pyrene (BaP), indeno (I, 2, 3-c,d) pyrene (IcdP), dibenzo (a, h) anthracene (DahA), benzo (g,h,i) perylene (BghiP)) in 2000 µg/mL stock standard solution was purchased from Supelco, Bellefonte, PA (USA). Six internal standards (IS) with 1,4-dichlorobenzene-d4; naphthalene-d8; acenaphlene-d10; anthracene-d10; chrysene-d12; perylene-d12 were supplied by Wellington Laboratories (Canada), Sigma–Aldrich Japan K.K. (Japan) and Restek (USA).

Solvents such as acetone, dichloromethane, methanol, and n–hexane were obtained from Kanto Chemical Co. (Japan). Sodium sulfate (Na_2SO_4) dry at the grade of 99% was obtained from Kanto Chemical Co (Japan).

2.2. Sample collection and analysis

Fifteen indoor dust samples were taken in 15 dwellings (8 story houses and 7 apartments) in urban Hanoi in May and June 2018. Indoor dust sample was collected from the floor of living room, kitchen and bedrooms of a dwelling location using a vacuum cleaner [18]. Then, the collected dust sample was wrapped with aluminum foil, putted in a polyethylene bag and transport to the lab within 4 hours. In the lab, the samples were sieved via a 250µm mesh sieve before preserved at -20°C.

The indoor dust samples were extracted according to the method described by Hanh et al. [19] with some modifications: 1g indoor dust samples were homogenized with 10mL hexane in a brown centrifuge tube. 20mL dichloromethane was added to centrifuge tube and put it in the ultrasonic bath at a temperature of 25 - 28°C, ultrasonically agitated for 20min in the dark. Then centrifuged samples at 3000rpm for 10min. The supernatant was decanted into a pre-cleaned 50mL evaporating flask. This extraction was repeated twice with 15mL of dichloromethane for each extraction. The extraction solutions were combined and concentrated by using a rotary evaporator to nearly 1mL, 5mL hexane was added, and concentrated under the flow of a gentle stream of nitrogen to 1mL. Na_2SO_4 column was used to remove water. Then 1µg of internal standards (phenanthrene -D12 and chrysene D12, 4µg/mL) was added and then it was picked up to 1 mL by hexane to analyze 16 PAHs by GC-MS.

Determination of 16 PAHs in samples by Shimadzu GCMS-QP 2010 Plus with capillary column J&W DB-5 ms (5% phenyl - 95% methyl silicone) fused silica capillary column (30m x 0.25mm i.d., 0.25m film) and with selected ion monitoring (SIM). The temperatures of the injector, transfer line and ion sources were 250°C, 300°C, and 200°C, respectively. The injection volume was 1µL at splitless mode

with 1 min for purge-off time. Helium was used as carrier gas at 1.5mL.min^{-1}. The column temperature was maintained for 2min at 40°C, increased to 310°C at a rate of 8°C.min^{-1}, and kept this temperature for 5min. To monitor for PAHs using ion m/z 128, 136, 152, 154, 164, 166, 178, 188, 202, 228, 240, 256, 258, 264, 276, 278, and 288. The method detection limits (MDL) are shown in Table 1.

To check contaminants of instrumentation, blank filter samples (n = 3) were analyzed prior to carrying out indoor dust samples. The laboratory blank samples (n = 3), indoor dust samples, and indoor dust standard reference materials (SRMs) from the National Institute of Standards & Technology SRM 2585 (N = 2) were analyzed with a similar analytical method to evaluate method accuracy. The results of the relative standard deviation of the values of PAHs in SRM 2585 were smaller than 30%.

2.3. Human health risk assessment

PAHs were listed as carcinogenic compounds by US Environmental Protection Agency (US EPA), United States Agency for Toxic Substances and Disease Registry (ATSDR) and International Agency for Research on Cancer (IARC). Among PAHs, Benzo (a) pyrene (BaP) is well toxicologically characterized. Besides, the high molecular-mass PAHs such as BaA, BbF, BkF, IcdP, and DahA have high carcinogenic potential (US EPA, 1993). Their toxic potency is used as a reference to express the toxic potency of PAHs [3, 20, 21] by the concept of BaP equivalent carcinogenic power (BaPE) and calculate by Qi et al. [22].

$$BaPE = BaA \times 0.06 + (BbF + BkF) \times 0.07 + BaP + DahA \times 0.6 + IcdP \times 0.08 \quad (1)$$

For a better understanding of the contribution of each PAHs to the carcinogenic index BaPE, the BaPeq as TEQ estimated based on the toxic equivalency factor (TEF) using the USEPA equation as follows:

$$BaPeq\ as\ TEQ = \Sigma C_n \times TEF \quad (2)$$

The health risk assessment on PAHs was estimated based on the following indices: chronic daily intake for each route (CDI) including ingestion and dermal exposure, hazard quotient (HQ), and hazard index (HI) [23]. The calculation for each index is described as follows:

$$CDI_{Ingestion\text{-}nca} = C_n \times [(R_{ing} \times EF \times ED)/(BW \times AT_{nca})] \times CF \quad (3)$$

$$CDI_{dermal\ contact\text{-}nca} = C_n \times [(Sa \times SL \times ABSd \times EF \times ED) / (BW \times AT_{nca})] \times CF \quad (4)$$

$$HQ = (CDI)_{nca} / RfD \quad (5)$$

$$HI = (HQ)_{inhalation} + (HQ)_{dermal\ contact} \quad (6)$$

The carcinogenic risk exposure (CDI) for each exposure route and incremental lifetime cancer risk (ILCR) were also estimated by the following equations:

$$CDI_{Ingestion\text{-}ca} = C_n \times [(IR \times EF) / AT_{ca}] \times CF \quad (7)$$

$$CDI_{Dermal\ contact\text{-}ca} = C_n \times [(ABS_d \times EF \times DFS_{adj})/AT_{ca}] \times CF \quad (8)$$

$$ILRC = (CDI_{Ingestion\text{-}ca} \times SF_{ingestion}) + (CDI_{Dermal\ contact\text{-}ca} \times SF_{dermal}) \quad (9)$$

Where: C_n is the average concentration of PAHs (µg/g) in indoor dust. TEF (ng/g) is the toxic equivalence factor of each individual PAH (Table 2) [3, 24]. Ingestion rate (Ring) (mg/day) for children and adults were 200 and 100, respectively [3]; Exposure frequency (EF) (day/year) was 350 [25]; Duration of exposure (ED) (year) for children and adults were 2 and 30, respectively [15]; Exposed skin are (SA) (cm^3) for children and adults were 1600 and 6700, respectively [15]; Dust to skin adherence factor (SL) (mg/cm^2) was 0.5 for both children and adults [15]; Dermal absorption factor (ABSd) for children and adults were 0.03 and 0.001, respectively [20]; Body weight (BW) (kg) for children and adults were 15 and 70, respectively [3]; Lifetime (LT) (year) was 70 for both children and adults [3]; Conversion factor (CF) was 1×10^{-6} for both children and adults [25]; Dust dermal contact factor-age-adjusted (DFSadj) (mg.year/kg.day) was 362.4 for both children and adults [25]; Dust ingestion rate age-adjusted (IR) (mg year/kg day) was 113 for both children and adults [25]; Average non-carcinogenic exposure time (ATnca) was ED×365 for both children and adults [23]; Average carcinogenic exposure time (ATca) was LT×365 for both children and adults [16]. Cancer slope factor (SF) (mg/kg.day) for ingestion ($SF_{ingestion}$) and dermal contact (SF_{dermal})were 7.3, 3.85, and 25, respectively [26]. Reference dose (RfD) for each PAHs were listed as follows: Naph = 0,02; Acy = 0,06; Ace = 0,06; Flu = 0,04; Phe = 0,04; Ant = 0,03; Flt = 0,04; Pyr = 0,03 [26].

3. RESULTS AND DISCUSSION

3.1. The levels of PAHs in indoor dust in Hanoi

Sixteen PAHs were detected among 15 indoor dust samples with the range of detection frequency (DF) and the range of average concentration were 80-100% and 22 - 1490ng/g (average concentration 466ng/g), respectively (Table 1). The total concentration of 16 PAHs (Σ_{16}PAHs) was 1320 - 54000ng/g (average Σ_{16}PAHs 7460ng/g). Many previous studies report the existence of PAHs in indoor dust [3, 4, 7, 22, 27]. The average concentration of PAHs recorded in this study was much lower than in other areas such as 66 times lower than in China, 50 times in Texas, and 22 times in Canada (Ottawa) [22, 28, 29]. This difference is the result of living habits such as burning materials in cooking, heating (coal, oil, gas, or wood), smoking tobacco, using furniture, home fragrance products, etc. In comparison with a previous study on the occurrence of PAHs in 2016 in road dust (1400 - 4700ng/g) and house dust (830 - 3500ng/g) in Hanoi [16], the observed concentration of PAHs in this study were higher, which may imply an increasing in the emissions of PAHs in indoor environment Hanoi, Vietnam during recent years.

Among 16 PAHs, BbF was the most dominant with the average concentration of 1490ng/g, (accounted for 20% of Σ_{16}PAHs in indoor dust samples), followed by Flt at 984ng/g (13%) and Chr at 806ng/g (11%). Ace was found to be the least PAH at 22ng/g (about 0,3%) due to its high volatility. The previous study in Kuwaiti about PAHs in household floor

P-ISSN 1859-3585 | E-ISSN 2615-9619

Table 1. The concentration of 16 PAHs in indoor dust

No.	Analyte	MDL (ng/g)	Average (ng/g)	StDev (ng/g)	Median (ng/g)	Min (ng/g)	Max (ng/g)	Detection frequency (%)
1	Naph	5.61	651	1840	113	53	7250	100
2	Acy	8.20	38	59	24	7	249	100
3	Ace	5.57	22	44	11	5	181	100
4	Flu	10.5	48	91	26	11	375	100
5	Phe	20.1	665	1250	362	158	5160	100
6	Ant	0.612	63	105	38	17	441	100
7	Flt	5.82	984	1920	526	179	7890	100
8	Pyr	3.12	714	1330	354	148	5470	100
9	BaA	0.322	247	432	105	36	1760	100
10	Chr	0.981	806	1390	426	121	5680	100
11	BbF	0.382	1490	2150	905	203	8920	100
12	BkF	0.322	279	417	167	0	1680	80
13	BaP	0.211	197	323	115	42	1350	100
14	IcdP	0.112	533	687	421	77	2900	100
15	DahA	0.112	78	114	52	16	479	100
16	BghiP	0.115	642	899	438	57	3740	100

dust also reported BbF and Phe were the dominant compounds [4].

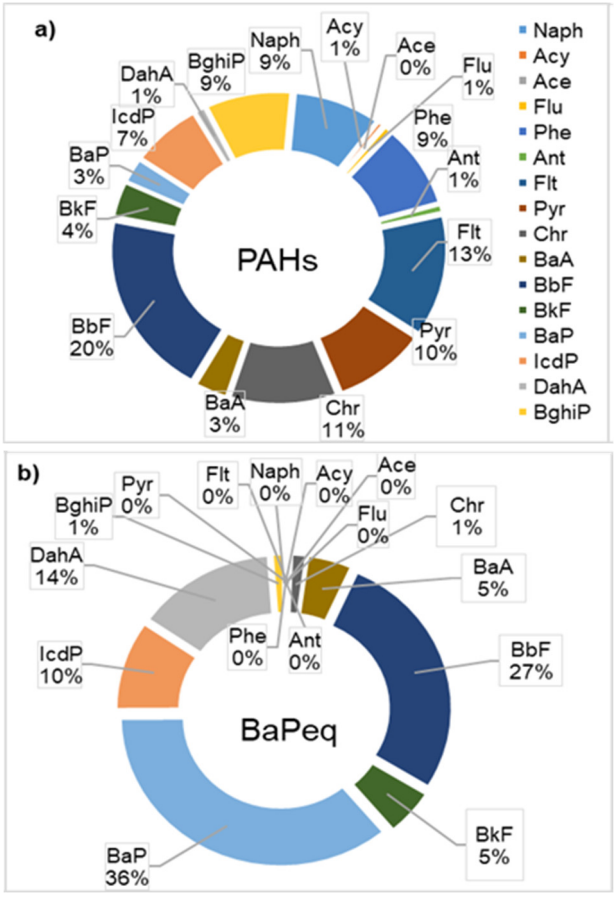

Figure 1. Profile of PAHs in indoor dust (a), Ccontribution of each PAH in BaPeq as TEQ (b)

The profile of the analyzed PAHs was described in Figure 1a. The low-molecular-weight PAHs (LMW-PAHs) that consist of 3-4 carbon rings (Naph, Acy, Ace, Flu, Phe, Ant, Flt, Pyr, and Chr) take the dominance with the contribution of 54% of average Σ_{16}PAHs in indoor dust samples, which is consistent with previous studies [3, 4]. The high-molecular-weight PAHs (HMW-PAHs) which contain 5-6 carbon rings (BaA, BbF, BkF, BaP, IcdP, DahA, and BghiP) shows an even contribution 46% of average Σ16PAHs in indoor dust samples, with the proportions of BbF, BgiP, IcdP, BaP, BaA, and DahA are 20%, 9%, 7%, 3%, 3% and 1% of average Σ_{16}PAHs in indoor dust samples, respectively. The LMW-PAHs were reported to have a high concentration in gaseous samples due to their high volatility, while HMW-PAHs, the more toxic and persistent compounds, were mainly found in settled dust. Hence, indoor dust is an important source for both HMW and LMW-PAHs to enter the human body via digestion and dermal contact [3, 4].

3.2. Source apportionment

Composition of PAHs were products from varies emission sources which the most important sources were petrogenic and pyrogenic sources [1, 22]. The LMW-PAHs are emitted from the combustion of crude oil and petroleum products, being the petrogenic source. Whilst, HMW-PAHs are mainly released from the combustion of coal, fossil, fuels, natural gas, and diesel, which is categorized as a pyrogenic source [1, 15].

In this research, diagnostic indexes and different relative distributions are applied to evaluate the source of PAHs. The diagnostic index is based on the similarity in structure of the analyzed PAHs. Five groups of analytes were categorized

based on the resemblance in the number of carbon rings and molecular weight, namely: (Flu, Pyr), (Phe, Ant), (BaA, Chr), (BghiP, BaP), and (IcdP, BghiP). As a result, the chemicals in each category have similarities in physicochemical characteristics, fate in the environment, and even emission sources. The diagnostic indexes are affected by several factors such as the absorption of dust, transportation, and the dissociation of PAHs in the atmosphere [27, 28]. However, this is still a reliable method to estimate the source of PAHs.

The result calculated showed that average ratios of Phe/Ant and Ant/(Ant+Phe) ranged between 6.72 - 12.3 (average 9.82) and 0.08 - 0.13 (average 0.09), respectively. The ratio of Phe/Ant > 10 indicates the petroleum sources, while Phe/Ant < 10 suggests the combustion of wood, diesel, and gasoline [32]. In the same manner, Ant/(Ant+Phe) <0.1 indicates a petrogenic source, otherwise, combustion is the main source [33]. As HMW-PAHs are not identified in all samples, the source appointment for these compounds was negligible.

The average ratio of Flu/(Flu+Pyr) was in the range of 0.03 - 0.1 (average 0.07). The ratio of Flu/(Flu+Pyr) < 0.5 indicates gasoline and fuel oil sources, while the ratio of Flu/(Flu+Pyr) > 0.5 suggests emission from coal and wood combustion [30]. This result confirms the combustion of gasoline and fuel oil was the cause of Flu and Pyr (Figure 2).

The average ratio of BaA/(BaA+Chr), IcdP/(IcdP+BghiP), and BghiP/BaP ranged from 0.1 - 0.32 (average 0.23), 0.39 - 0.77 (average 0.47) and 0.98 - 5.23 (average 3.33), respectively. It was reported that BaA/(BaA+Chr) < 0.2 indicates the petroleum source, otherwise, diesel and gasoline (Figure 2) [4, 16, 34, 35]. This demonstrated that both sources contributed to the emission of these PAHs. These results are consistent with previous studies in studies of Saudi, Kuwait [4].

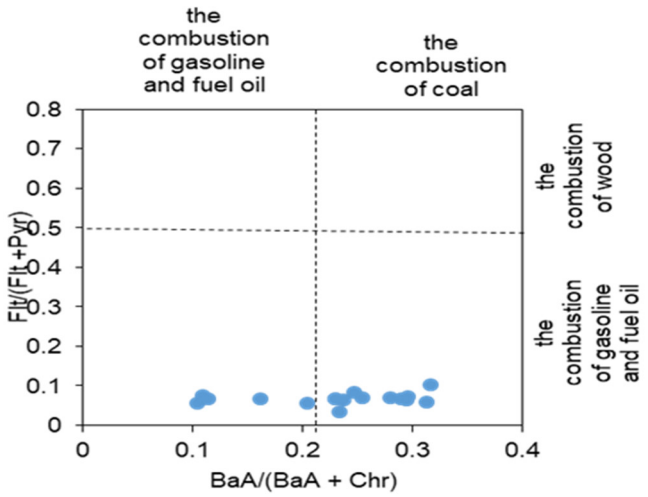

Figure 2. BaA/(BaA+Chr) ratio and Flu/(Flu+Pyr) in study area with thresholds of emission origin

The diagnostic index of LMW-PAHs indicates the petrogenic source and the pyrogenic source [1, 22, 36]. Other factors that might affect these ratios were not considered in this study. PAHs sources in indoor dust may vary from cooking, smoking tobacco, and cross-ventilation... which were different in each household based on their living habit, construction, etc. Therefore, a large-scale study is needed to have insight into the PAHs source.

3.3. Health risk assessment

The BaPE was calculated for indoor dust samples ranged 75 - 2700ng/g (mean: 425ng/g, median: 261ng/g), these values were greatly different between studies sites. This indicates a population in urban Hanoi has a risk of exposure to carcinogenic PAHs in indoor dust. A similar result was shown in the study in Saudi [4].

The contribution of individual PAH in the profile of BaPeq as TEQ is described in Figure 1b, in which HMW-PAHs showed their high impact with 98% in the profile of BaPeq as TEQ, and BaP is dominant with the contribution of 36%, followed by BbF (27%) and Dah (14%). Although BaP and DahA only accounted for 3% and 1% Σ16PAHs, respectively in the profile of PAHs (Figure 1a), the total of both BaP and DahA contributed 50% to the BaPeq as TEQ (Figure 1b). The obtained values for calculated BaPeq as TEQ revealed a major toxicity threat associated with PAHs in all indoor dust from HMW-PAHs with 5 and 6 aromatic rings. This observes also reported in the lite in the literature of ATSDR (2009) and USEPA (1993).

Table 2. Concentration (ng/g) of BaPeq as TEQ

No.	Analyte	Toxic Equival Factors TEF [29]	BaPeq as TEQ			
			Average	Median	Min	Max
1	Naph	0.001	1	0.11	0.05	7.3
2	Acy	0.001	0	0.02	0.01	0.2
3	Ace	0.001	0	0.01	0.01	0.2
4	Flu	0.001	0	0.03	0.01	0.4
5	Phe	0.001	1	0.36	0.16	5.2
6	Ant	0.01	1	0.38	0.17	4.4
7	Flt	0.001	1	0.53	0.18	7.9
8	Pyr	0.001	1	0.35	0.15	5.5
9	BaA	0.100	25	11	3.6	176
10	Chr	0.010	8	4.3	1.2	57
11	BbF	0.100	149	91	20	892
12	BkF	0.100	28	17	0	168
13	BaP	1.000	197	115	42	1350
14	IcdP	0.100	53	42	8	290
15	DahA	1.000	78	52	16	479
16	BghiP	0.010	6	4.4	0.57	37

The estimated hazard quotient (HQ), hazard index (HI), and incremental lifetime cancer risk (ILCR) were calculated for 7 PAHs (Ace, Flu, Phe, Ant, Flt, Pyr, and BaP) and described in Table 3. BaP had the HI values of 1.9 and 18 (ng/kg-bw/day) for adults and children, respectively, which were

much higher than its reference dose (RfD) ($1.4x10^{-4}$ ng/kg-bw/day). These results indicated significant high carcinogenic risk when being exposed to BaP for adults and children [3, 31]. In addition, the HI values of Pyr ($3.3x10^{-2}$ and $3.1x10^{-1}$ ng/kg-bw/day for adults and children, respectively), Phe ($2.8x10^{-1}$ ng/kg-bw/day for children) and Flt ($2.8x10^{-1}$ ng/kg-bw/day for children) were also higher than their corresponding RfDs, which suggested the carcinogenic risk for children and/or adults when being exposed to Pyr, Phe and Flt [3, 31]. The other PAHs had HI values less than their corresponding RfDs, which indicates low carcinogenic risk when being exposed to these chemicals.

Table 3. The estimated hazard quotient (HQ), hazard index (HI), chronic daily intake (CDI), and incremental lifetime cancer risk (ILCR) were calculated for adults and children

PAHs	RfD	Adults			Children		
	[26]	$HQ_{Ingestion}$	HQ_{Dermal}	HI	$HQ_{Ingestion}$	HQ_{Dermal}	HI
Ace	$6x10^{-2}$	$5.1x10^{-4}$	$1.0x10^{-6}$	$5.1x10^{-4}$	$4.8x10^{-3}$	$3.4x10^{-5}$	$4.8x10^{-3}$
Flu	$4x10^{-2}$	$1.7x10^{-3}$	$2.2x10^{-6}$	$1.7x10^{-3}$	$1.5x10^{-2}$	$7.4x10^{-5}$	$1.6x10^{-2}$
Phe	$4x10^{-2}$	$2.3x10^{-2}$	$3.1x10^{-5}$	$2.3x10^{-2}$	$2.8x10^{-1}$	$1.0x10^{-3}$	$2.8x10^{-1}$
Ant	$3x10^{-1}$	$2.9x10^{-4}$	$2.9x10^{-6}$	$2.9x10^{-4}$	$2.7x10^{-2}$	$9.7x10^{-5}$	$2.7x10^{-2}$
Flt	$4x10^{-2}$	$3.4x10^{-2}$	$4.5x10^{-5}$	$3.4x10^{-2}$	$3.1x10^{-1}$	$1.5x10^{-3}$	$3.2x10^{-1}$
Pyr	$3x10^{-2}$	$3.3x10^{-2}$	$3.3x10^{-5}$	$3.3x10^{-2}$	$3.0x10^{-1}$	$1.1x10^{-3}$	$3.1x10^{-1}$
BaP	$1.4x10^{-4}$	1.9	$9.0x10^{-6}$	1.9	$1.8x10^{1}$	$3.0x10^{-4}$	$1.8x10^{1}$
PAHs		$CDI_{Digestion}$	CDI_{Dermal}	ILRC	$CDI_{Digestion}$	CDI_{Dermal}	ILRC
Ace		$3.5x10^{-5}$	$1.1x10^{-7}$	$3.5x10^{-5}$	$3.5x10^{-5}$	$3.3x10^{-6}$	$3.8x10^{-5}$
Flu		$7.5x10^{-5}$	$2.4x10^{-7}$	$7.5x10^{-5}$	$7.5x10^{-5}$	$7.2x10^{-6}$	$8.2x10^{-5}$
Phe		$1.0x10^{-3}$	$3.3x10^{-6}$	$1.0x10^{-3}$	$1.0x10^{-3}$	$9.9x10^{-5}$	$1.1x10^{-3}$
Ant		$9.7x10^{-5}$	$3.1x10^{-7}$	$9.8x10^{-5}$	$9.7x10^{-5}$	$9.4x10^{-6}$	$1.1x10^{-4}$
Flt		$1.5x10^{-3}$	$4.9x10^{-6}$	$1.5x10^{-3}$	$1.5x10^{-3}$	$1.5x10^{-4}$	$1.7x10^{-3}$
Pyr		$1.1x10^{-3}$	$3.5x10^{-6}$	$1.1x10^{-3}$	$1.1x10^{-3}$	$1.1x10^{-4}$	$1.2x10^{-3}$
BaP		$3.1x10^{-4}$	$9.8x10^{-7}$	$3.1x10^{-4}$	$3.1x10^{-4}$	$2.9x10^{-5}$	$3.3x10^{-4}$
Σ7PAHs		$3.9x10^{-3}$	$1.2x10^{-5}$	$3.9x10^{-3}$	$3.9x10^{-3}$	$3.7x10^{-4}$	$4.2x10^{-3}$

RfD: The reference doses (ng/kg-bw/day) were suggested by US EPA [26]

The chronic daily intake (CDI) via digestion was the highest, followed by dermal which indicates that oral exposure to 7 PAHs (Ace, Flu, Phe, Ant, Flt, Pyr, and BaP) is the dominant pathway causing cancer risk for both adults and children (Table 3). The estimated ILCR for total PAHs for adults and children are $3.9x10^{-3}$ and $4.2x10^{-3}$, respectively. These results are much higher than the recommended safety limit by EPA being $1x10^{-4}$ which showed that PAHs in indoor dust have a significant long-term effect on adults and young children.

Although, this study with limited sample size, this is a study about monitoring organic pollutants which might impact human health in indoor environments. The levels of PAHs in indoor dust in urban Hanoi are an indication of potential risk for the health of the population from PAHs in indoor dust via ingestion and demand dust. However, there is also a need for more studies with a larger number of samples and assessments of ventilation levels (structures in apartments), and living habits to have more detailed assessments, along with more studies on PAHs in indoor air for a more uniform assessment.

4. CONCLUSION

In this research, sixteen PAHs were found in indoor dust collected from the different apartments in urban Hanoi and each of found PAHs was analyzed for concentrations, source, and risk assessment. The total concentration of sixteen PAHs was 1320 - 54000ng/g with an average of 7460ng/g. Among 16 PAHs, BbF showed the highest contribution with a mean concentration of 1490ng/g (accounted for 20% of the total concentration of 16 PAHs), followed by Flt and Chr with a concentration were 984ng/g (13%) and 806 (11%), respectively, and Ace was the least PAHs at 22ng/g (equivalent to 0,3%). The LMW-PAHs were dominant in the profile of PAHs, however, HMW-PAHs accounted for 98% in BaPeq as TEQ profile, emphasizing the significant health effects of these compounds. Based on the diagnostic analysis, it was indicated that the LMW-PAHs derived from both petrogenic and pyrogenic except for Flu and Pyr, which are found to come from gasoline and petroleum source. HMW-PAHs claimed their high impacts on the toxicity with the contribution of 98% in the BaPeq as TEQ profile. The estimated ILCR for both adults and children was higher than the recommended safety level, which confirmed the long-term cancer risk when exposed to these chemicals. Digestion of indoor dust was the dominant exposure pathway of PAHs for the population in urban Hanoi.

ACKNOWLEDGMENTS

The Vietnam Academy of Science and Technology under grant number "TĐPCCC.05/21-23" supports this research.

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