



Research article

Phase distribution and probabilistic risk assessment of polycyclic aromatic hydrocarbons in indoor air of coffee shops at Zahedan, Iran

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ABSTRACT

Polycyclic aromatic hydrocarbons (PAHs) are a class of hydrocarbons, some of which are established human carcinogens. Human exposure to these chemicals is complex and originates from both indoor and outdoor sources. This study measured the concentration of PAHs in the gaseous and particulate phases during the cold months of 2022 using XAD-2 sorbent tubes and Polytetrafluoroethylene (PTFE) filters in the indoor air of coffee shops in Zahedan, Iran (n = 23). The average concentrations of particulate-bound PAHs and gaseous PAHs were $13,411.86 \pm 6517.24$ ng/m³ and 6432.76 ± 4311.72 ng/m³, respectively. Source apportionment analyses indicated that the primary sources of PAHs in coffee shops were fossil fuel combustion and environmental tobacco smoke (ETS), commonly referred to as second and third-hand smoke. The lifetime cancer risk (LTCR) of inhaled PAHs was calculated using the Monte Carlo simulation method. The mean LTCR for adults and children from inhaling these substances were $9.43 \times 10^{-6} \pm 5.06 \times 10^{-6}$ and $5.34 \times 10^{-6} \pm 2.87 \times 10^{-6}$, respectively. The hazard quotient (HQ) of PAHs exceeded 1. These findings highlight the need to reduce PAHs exposure in public spaces through proper health warning labels and regulated indoor smoking policies.

1. Introduction

Indoor air quality (IAQ) significantly impacts public health. In modern societies, individuals spend over 90 % of their time indoors,

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including in homes, offices, schools, transit hubs, and public spaces [1,2]. Recent studies suggest that the health effects of indoor air pollution may surpass those associated with ambient air pollution [3–6].

As crowded public spaces, Coffee shops and restaurants host various age groups with diverse health conditions and susceptibilities. These spaces often lack clear smoking regulations, and the use of cigarettes and hookahs is commonplace. Customers and staff are also exposed to cooking-related fumes and particles, including polycyclic aromatic hydrocarbons (PAHs). Non-smoking patrons may be inadvertently exposed to second-hand smoke, third-hand smoke, and cooking byproducts in their breathing zones [7–11].

PAHs are chemical compounds consisting of fused aromatic rings of hydrogen and carbon atoms, exhibiting linear, clustered, and angular configurations with diverse complexity and lipophilic properties [12,13]. Particulate-phase PAHs are toxic organic substances with low volatility that can travel long distances, potentially causing genotoxic effects when inhaled [14,15]. PAH emissions arise from various sources, such as road traffic (e.g., automobile engines) and the incomplete combustion of fuel in industrial processes, cooking, and biomass burning [16,17]. The United States Environmental Protection Agency (USEPA) has labeled several PAH species as priority pollutants due to their mutagenic and carcinogenic properties [18,19]. Benzo(a)pyrene (BaP) is extensively used in cancer risk assessments as a surrogate for all PAHs due to its well-documented carcinogenic effects [20,21].

Current environmental epidemiology research and air quality guidelines in various countries emphasize the health effects of particulate matter-bound PAHs. Numerous studies have been conducted in this field, examining outdoor air [17,22–27], indoor and outdoor air in school classrooms [28], rural and urban residential houses [6,29,30], urban slums, and rural areas [31]. Additional

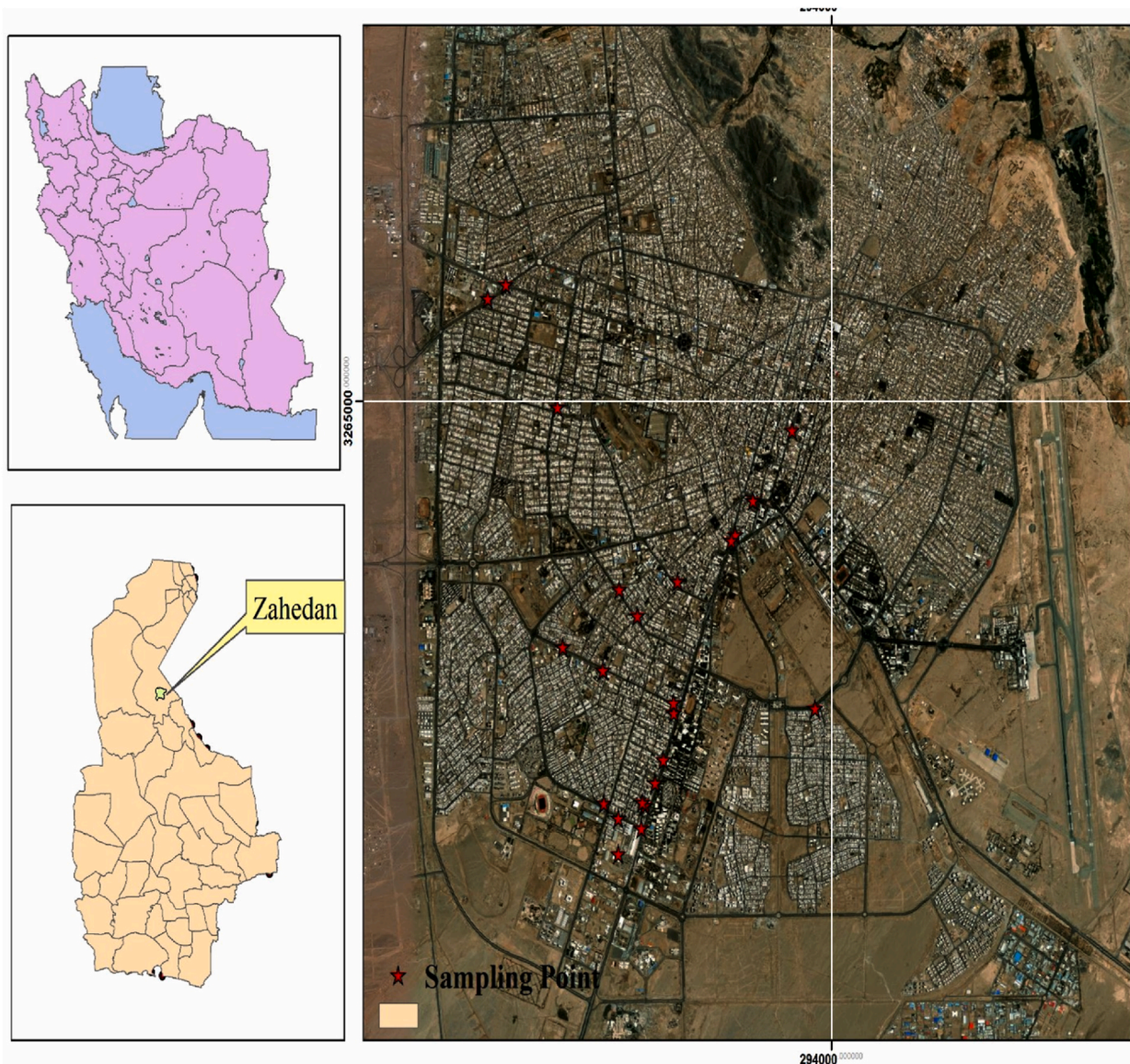


Fig. 1. Map of the study area and sampling points.

studies have focused on indoor air in school classes [32–34], public bars [35], dormitory rooms [36], water pipe cafes [37], and residential houses in urban areas and rural areas [38–40].

Despite this extensive research, limited studies have quantified the levels of particulate-bound PAHs and gaseous PAHs in the indoor air of kitchens using various fuels [41] or solely in the gaseous phase of rural house indoor air [42]. Therefore, measuring the concentrations and toxicity of particulate-bound and gaseous PAHs in public environments such as restaurants and coffee shops is essential. Furthermore, it is crucial to determine the concentration of these substances in public spaces and advocate for appropriate, sustainable control plans to manage or reduce such emissions.

This study aimed to quantify customers' exposure to gaseous and particulate PAHs and particulate matter (PM) in coffee shops. We also examined the influence of various environmental factors, including ventilation systems, relative humidity, surface area of the coffee shop, location, indoor temperature, and floor number, on the observed PAH concentrations. Additionally, we determined the health risk posed by PAHs exposure using a probabilistic risk assessment approach based on Monte Carlo simulation.

2. Material and methods

2.1. Study area

This research was conducted in Zahedan, located in the Sistan and Baluchistan provinces of Iran. According to the 2016 census, Zahedan has a population of approximately 587,730 and covers an area of around 55.7 km². Situated in a hot and dry region (coordinates 29°07' N, 60°35' to 61°22' E, altitude: 1352 m), Zahedan experiences an average annual rainfall of 61.94 mm and an average relative humidity of 17 %. The predominant wind direction is from the north to the east. The highest recorded temperature is 42 °C, while the lowest is –7.2 °C [43]. The study area and sampling locations are illustrated in Fig. 1. To gather data on the coffee shops' ventilation systems, relative humidity, indoor and outdoor temperatures, floor number, surface area, and location, we employed a questionnaire. Observations at each site were recorded using this questionnaire, as detailed in Tables S1 and S2 of the Supplementary Material.

2.2. Air sampling procedure

Air samples were collected following the NIOSH Manual of Analytical Methods Approach 5515 [44], with some modifications. For indoor air collecting, we used dichotomous sampling media with SKC STANDARD sampler pumps (224-44TX, United Kingdom) operating at a flow rate of 2 L/min for 5 h, sampling total 600 L of air. PTFE filters (2.0 µm, 37 mm diameter) were employed for collecting particulate-bound PAHs, while XAD-2 adsorbent tubes were used for gas-phase PAHs [44]. Prior to sampling, the pumps were calibrated utilizing a primary flow meter (Bios International Dry Cal DC-Lite, United States). Samples were taken at the customers' breathing zone, approximately 150 cm above ground level. Sampling was conducted in the evening (5–10 p.m.) during February and March 2022.

After sampling, filters were put in filter holders, and each filter and sorbent tube was wrapped in foil separately. The samples were then stored at –20 °C for 72 h before analysis. The mass concentrations of total suspended particles (TSP) were determined gravimetrically by comparing the filter weights before and after collecting, using a microbalance with a 0.0001g sensitivity (Sartorius BL210S Analytical Balance, United States).

Using the NIOSH sampling method No. 5515, samples collected on filters and sorbent were extracted with an appropriate solvent [44,45]. In brief, the PTFE filters, along with the front and back sections of the XAD-2 sorbent, were placed in separate glass jars with screw-on lids. Each jar contained 5 mL of dichloromethane, and the contents were subjected to ultrasonic agitation for 30 min. The extracts were then condensed to less than 1 mL by carefully applying a gentle stream of dry nitrogen. Dichloromethane was used to adjust the final volume to precisely 1 mL.

The extracted PAHs were analyzed using gas chromatography (GC) with a 5975C mass detector and a split/splitless injector (Agilent 7890A, Agilent Technologies, United States). Helium carrier gas with a purity of 99.999 % was maintained at a consistent flow rate of 1.3 mL/min. The GC oven temperature program was as follows: initial temperature of 80 °C for 1 min, ramping up at 25 °C/min to 200 °C for 1 min, followed by an 8 °C/min ramp to 325 °C. The ion source, quadrupole mass analyzer, injection port, and transfer line were maintained at 320 °C, 150 °C, 320 °C, and 320 °C, respectively. The injection volume was 2 µL, with a split ratio of 1:10 at the injector [46].

PAH congeners, including Benzo[a]pyrene (BaP), Benzo[e]pyrene (BeP), Naphthalene (Nap), Benzo[b]fluoranthene (BbF), Benzo[k]fluoranthene (BkF), Acenaphthene (AcP), Acenaphthylene (AcPy), Phenanthrene (PhA), Anthracene (AnT), Fluorene (Flu), Fluoranthene (FluA), Benzo[a]anthracene (BaA), Pyrene (Pyr), Dibenzo[a,h]anthracene (DbA), Benzo[ghi]perylene (BghiP), Indeno[1,2,3-cd]pyrene (InP), and Chrysene (Chr), were detected and quantified. Relative humidity and temperature in the coffee shops during sampling were measured using a hygrometer and thermometer (STANDARD ST-625, Hong Kong). Analytical-grade materials were used for all compounds and reagents. Microsoft Excel was employed for data analysis and to achieve the research objectives.

2.3. Validation of data and quality assurance

Blank samples were included with each set of filters to assess potential contamination during sampling and analysis. For calculating instrument detection limits (IDLs) and method detection limits (MDLs), one field blank sample and one laboratory blank sample were used for every ten samples [47]. In the blank samples evaluated, the concentration of any chemical did not exceed the lowest point on

the calibration curve. The method's accuracy, based on the average error of values from a typical analytical solution (three replicates), was estimated to be approximately 98 % ($R^2 > 0.98$) [48].

Phenanthrene d10 and each target PAH were spiked onto the filters and adsorbent tubes to measure the relative response factors (RRFs), comparing the relative sensitivity in the two procedures [49,50]. The recovery rates for species on the filter and XAD-2 tube samples exceeded 75 % and 92 %, respectively. IDLs ranged from 0.08 $\mu\text{g}/\text{m}^3$ for acenaphthylene to 0.1 $\mu\text{g}/\text{m}^3$ for Benzo[a]pyrene. The IDLs and MDLs values are provided in Table S3.

2.4. Statistical analysis

Data were analyzed using IBM® SPSS Statistics version 25.0, with a significance level of 0.05 for all tests. Statistical tests, including the normality test, *t*-test, Pearson rank correlation, and linear regression, were employed to investigate relationships between PAHs and environmental parameters (temperature, relative humidity, ventilation system, surface area, location, and floor number).

2.5. Health risk assessment

The health risk posed by PAHs was calculated using the lifetime cancer risk (LTCR) of inhaled compounds. Equation (1) was used to determine LTCR. Given the age-specific nature of the health risk assessment, the population was split into two groups: adults and children. Table 1 provides the data required for the health risk assessment.

$$LTCR = CDI \times CSF \quad (1)$$

where CDI represents the chronic daily intake ($\text{mg}/\text{kg}\cdot\text{day}$), and CSF denotes the cancer slope factor ($\text{mg}/\text{kg}\cdot\text{day}$)⁻¹. CDI was calculated using Equation (2) [51].

$$CDI = \frac{CS \times IR \times EF \times ED \times CF}{AT \times BW} \quad (2)$$

Here, CS represents the concentration of pollutants in indoor air ($\mu\text{g}/\text{m}^3$), IR is the human inhalation rate (m^3/day), and CF is the conversion factor ($\text{mg}/\mu\text{g}$). ED denotes exposure duration (years), AT is the average lifetime (days), BW is body weight (kg), and EF is exposure frequency (days/year) [51].

The concentration of the Benzo[a]pyrene total potency equivalency (BaP TEF) was applied to determine the CS. According to Gope et al. (2018) [52], the BaP TEF for PAHs was determined using Equation (3):

$$CS = \sum \text{BaP}_{\text{eq}} = \sum_{i=1}^n (C_i \times \text{TEF}_i) \quad (3)$$

where TEF is the individual toxic equivalency factor for PAHs, and C is the concentration of PAHs [53]. The TEF values of each component were listed in Table S4 of the supplementary file.

The hazard quotient (HQ) parameter was used to assess the risk of non-carcinogenic target components, obtained from Equation (4) [51].

$$HQ = \frac{CDI}{\text{RfC}} \quad (4)$$

HQ values below or equal to one indicate an acceptable risk level, while HQ values above one suggest a potentially significant risk [54,55]. The chronic daily consumption was calculated using the mean PAH concentrations [55–57].

2.6. Monte Carlo simulation to assess health risks

Nicholas Metropolis first proposed the Monte Carlo simulation (MCS) approach for health risk simulation [51]. MCS is a critical

Table 1

Risk parameters applied for calculation of HQ and LTCR for PAHs in indoor air of coffee shops.

Parameters	Adult	Child	Reference
Inhalation rate (m^3/day)	15.73	11.41	[47,48]
Body weight (kg)	71.05	29.70	[51]
Exposure duration (year)	26	8.5	[51]
Exposure frequency (day/year)	Questionnaire (2.1)	Questionnaire (2.1)	[50]
^a Averaging time (day)	25550	25550	[50]
Inhalation reference concentration of BaP (^b RfC)($\text{mg}/\text{kg}\cdot\text{day}$)	4.42×10^{-7}	7.68×10^{-7}	[51,53]
Cancer slope factor($\text{mg}/\text{kg}\cdot\text{day}$) ⁻¹	12	12	[52]

^a Averaging exposure time (days) for carcinogens = (70 years) \times 365 days per year.

^b RfC with the unit $\text{mg}/\text{kg}\cdot\text{day}$ = (inhalation reference concentration ($2 \times 10^{-6} \text{ mg}/\text{m}^3$) \times Assumed inhalation rate (m^3/day) \times 1/BW (kg).

numerical computing method that uses random sampling guided by probability theory [58]. The probabilistic risk of exposure to PAHs was evaluated using the Monte Carlo simulation in Crystal Ball 11.1.2 software. The risk parameters used in the Monte Carlo simulation are detailed in Table S5 of the supplementary material.

3. Results and discussion

3.1. Mass concentrations of PM

Table 2 presents a summary of the descriptive statistics of PM mass concentrations, which ranged from 150 to 2420 $\mu\text{g}/\text{m}^3$. Direct comparison of total PM levels to national PM_{2.5} and PM₁₀ guidelines in Iran, the WHO, and the EPA was not feasible. However, the size of inhaled particles significantly impacts human health, and thus, their levels were evaluated in most of the research literature. For this purpose, comparisons were made with previous studies on PM₁, PM_{2.5}, and PM₁₀ concentrations.

Levy et al. evaluated PM_{2.5} concentrations in select coffee shops in Boston and Massachusetts, reporting mean PM_{2.5} concentrations 1 $\mu\text{g}/\text{m}^3$ lower than those in this study [59]. In a study by Lung et al., PM_{2.5} concentrations in two 24-h coffee shops in Taichung, Taiwan, had a mean value of 84.7 $\mu\text{g}/\text{m}^3$, ranging from 71.8 to 97.5 $\mu\text{g}/\text{m}^3$, which is lower than the concentrations reported in this study [60]. Research on restaurants, bars, discos, and coffee shops indicated PM_{2.5} concentrations ranging from 178 to 808 $\mu\text{g}/\text{m}^3$, higher than the values reported here [61]. In Toronto and Windsor, a study found the mean PM concentration in 15 coffee shops to be 182.3 $\mu\text{g}/\text{m}^3$, with data ranging from 128.8 to 235.8 $\mu\text{g}/\text{m}^3$, which is lower than the findings of this study [62]. Additionally, Ścibor et al. reported a mean PM_{2.5} level of 23.1 $\mu\text{g}/\text{m}^3$ in residential areas in Kraków, Poland, significantly lower than the levels found in this study [63].

3.2. PAHs concentration phase distribution

The statistical analysis of particulate-bound and gaseous PAH levels is presented in Table 2. The sum of individual PAHs (17 PAHs) ranged from 109,357.00 to 228,001.55 ng/m^3 across the gaseous and particulate phases. In coffee shops, the mean PAH concentrations in the air (i.e., the total concentrations of PAHs in both the particulate and gas phases) was $19,844.62 \pm 10,604.94 \text{ ng}/\text{m}^3$, with 17 PAHs ranging from 555.56 to 40,369.00 ng/m^3 . Specifically, the mean values were $6432.76 \pm 4311.72 \text{ ng}/\text{m}^3$ in the gaseous phase and $13,411.86 \pm 6517.24 \text{ ng}/\text{m}^3$ in the particulate phase.

Nap and Flu were the most prevalent indoor particulate-bound PAHs in coffee shops, with mean concentrations of $1094.40 \pm 713.51 \text{ ng}/\text{m}^3$ and $1016.10 \pm 888.95 \text{ ng}/\text{m}^3$, respectively. These compounds constituted approximately 11.12 % and 10.32 % of the particulate phase PAHs. Other PAHs with high concentrations in the particle phase included AcPy (7.89 %), AcP (7.38 %), FluA (7.28 %), and Pyr (7.11 %). BghiP had the lowest concentration of particulate-bound PAHs, constituting less than 0.5 %. In the gas phase, DbA, BghiP, and InP were not detected. Nap was found in the highest quantity, with a mean concentration of $660.78 \pm 322.18 \text{ ng}/\text{m}^3$, accounting for 13.69 % of gaseous PAHs. The relative abundances of other PAHs in the gaseous phase were Pyr (10.2 %), Flu (9.67 %), AnT (8.74 %), and AcPy (8.43 %) (Fig. 2). Overall, 67.09 % of PAHs were present in the particle phase, while 32.91 % were present in

Table 2

Mean concentrations of PAHs in particulate phase and gas phase indoor air of coffee shops.

	Units	Particulate phase				Gaseous phase				
		Min	Max	Mean	SD	Min	Max	Mean	SD	
1	PM	$\mu\text{g}/\text{m}^3$	150.00	2420.00	1047.00	786.79	–	–	–	–
2	Nap	ng/m^3	423.73	3645.83	1094.40	713.51	223.21	1346.15	660.78	322.18
3	AcPy	ng/m^3	338.98	1777.78	776.92	415.47	ND	1277.78	407.22	383.06
4	AcP	ng/m^3	ND	3020.83	726.64	688.32	ND	1770.83	348.03	445.57
5	Flu	ng/m^3	ND	3555.56	1016.10	888.95	ND	1444.44	466.73	396.16
6	PhA	ng/m^3	ND	1222.22	676.18	321.62	ND	1120.69	349.57	374.70
7	AnT	ng/m^3	ND	1465.52	618.09	488.36	ND	1333.33	421.92	425.32
8	FluA	ng/m^3	ND	1777.78	716.82	508.64	ND	775.86	189.93	224.66
9	Pyr	ng/m^3	ND	1979.17	699.88	441.39	ND	1777.78	492.35	461.68
10	BaA	ng/m^3	ND	1458.33	517.46	374.58	ND	888.89	204.65	275.01
11	BbF	ng/m^3	ND	1336.21	591.94	409.05	ND	1077.59	333.25	380.66
12	BkF	ng/m^3	ND	1206.90	581.53	391.15	ND	1034.48	268.71	342.75
13	BaP	ng/m^3	ND	1637.93	622.87	476.68	ND	974.58	276.66	299.46
14	BeP	ng/m^3	ND	1979.17	349.56	551.41	ND	1145.83	83.03	256.38
15	DbA	ng/m^3	ND	1041.67	90.03	259.73	ND	ND	ND	ND
16	BghiP	ng/m^3	ND	555.56	24.15	115.84	ND	ND	ND	ND
17	InP	ng/m^3	ND	1666.67	236.56	444.07	ND	ND	ND	ND
18	Chr	ng/m^3	ND	1458.33	460.66	461.37	ND	1060.61	365.19	323.25
19	ΣPAHs	ng/m^3	555.56	25,171.12	13,411.86	6517.24	ND	1,5197.88	6432.76	4311.72
20	ΣCarPAHs ^a	ng/m^3	2070.70	14,326.00	18,482.87	4804.41	ND	7664.66	7676.83	3120.71
21	ΣCOM PAHs ^b	ng/m^3	555.56	16,486.76	11,666.71	5259.28	ND	11,324.02	5155.52	3557.38

^a Carcinogenic PAHs includes BaA, Chr, BbF, BkF, BaP, DbA, and InP.

^b Combustion PAHs includes FluA, Pyr, BaA, Chr, BbF, BkF, BaP, BghiP, and InP.

the gas phase.

BaA, Chr, BbF, BkF, BaP, DbA, and InP were identified as probable carcinogens among the 17 PAHs [64,65]. Fig. 3 illustrates the average concentrations of carcinogenic PAHs in both particulate-bound and gaseous phases. The data indicate that BaP and DbA had the highest concentrations in both phases. BaP is the most hazardous and prevalent PAHs, with a carcinogenic potency approximately ten times greater than that of DbA. Following these, Chr and BaA were the next most significant carcinogens in the particulate-bound PAHs. In the gaseous phase, BbF and BkF were the most prominent carcinogens. The mean concentration of carcinogenic PAHs in coffee shops' indoor air was $14,948.40 \pm 7848.75 \text{ ng/m}^3$. The distribution of gaseous and particulate-bound carcinogenic PAHs showed slight differences, with 32.42 % in the particulate phase and 28.08 % in the gas phase. The predominance of carcinogenic PAHs in particles is consistent with previous studies.

The combustion-related PAHs identified in this study included FluA, Pyr, BaA, Chr, BbF, BkF, BaP, BghiP, and InP. As shown in Table 2, the mean levels of these combustion compounds in the particulate and gaseous phases were $11,666.71 \pm 5259.28 \text{ ng/m}^3$ and $5155.52 \pm 3557.38 \text{ ng/m}^3$, respectively. These values represent approximately 46.05 % and 42.42 % of the total PAHs studied, indicating that combustion PAHs significantly contribute to the overall PAH emissions in the indoor air of coffee shops. These substantial values suggest that combustion processes are likely a major source of PAHs in this environment.

The values reported in this study were higher compared to those from studies by Levy et al. (2002) [59], Lung et al. (2004) [60], Bolte et al. (2008) [61], Zhang et al. (2010) [62], Castro et al. (2011) [66], and Krugly et al. (2014) [67]. This indicates that the indoor air of these public spaces is significantly polluted. To safeguard public health, it is essential to develop and implement strategies to reduce exposure to PAHs (both gaseous and particulate), with particular emphasis on carcinogenic PAHs [68].

3.3. The correlations among PAHs species and regression analysis of PAHs and PM levels with environmental factors

The relationships between PAH species in the particulate phase are summarized in Table 3. No significant correlations were observed between PAH species. Krugly et al. (2014) [67] suggested that high correlations between PAH compounds indicate similar emission sources. However, in this study, the correlation coefficients (r) for PAH compounds were generally low. The minimum correlation coefficients ($r = 0.42$, $P\text{-value} = 0.01$) were observed between Nap and PM, Flu and FluA, Flu and BaP, and BaA and BbF. These data suggest multiple sources for PAH emissions. Internal sources of PAH emissions include cooking, burning fossil fuels, and smoking, with ETS contributing as both second-hand and third-hand smoke. Additionally, heating, insect repellents, building materials, and the penetration of PAHs from external sources influenced indoor PAH levels [69–71].

Indoor PM concentration showed a positive correlation with relative humidity across all coffee shops (Table 4). Conversely, it was

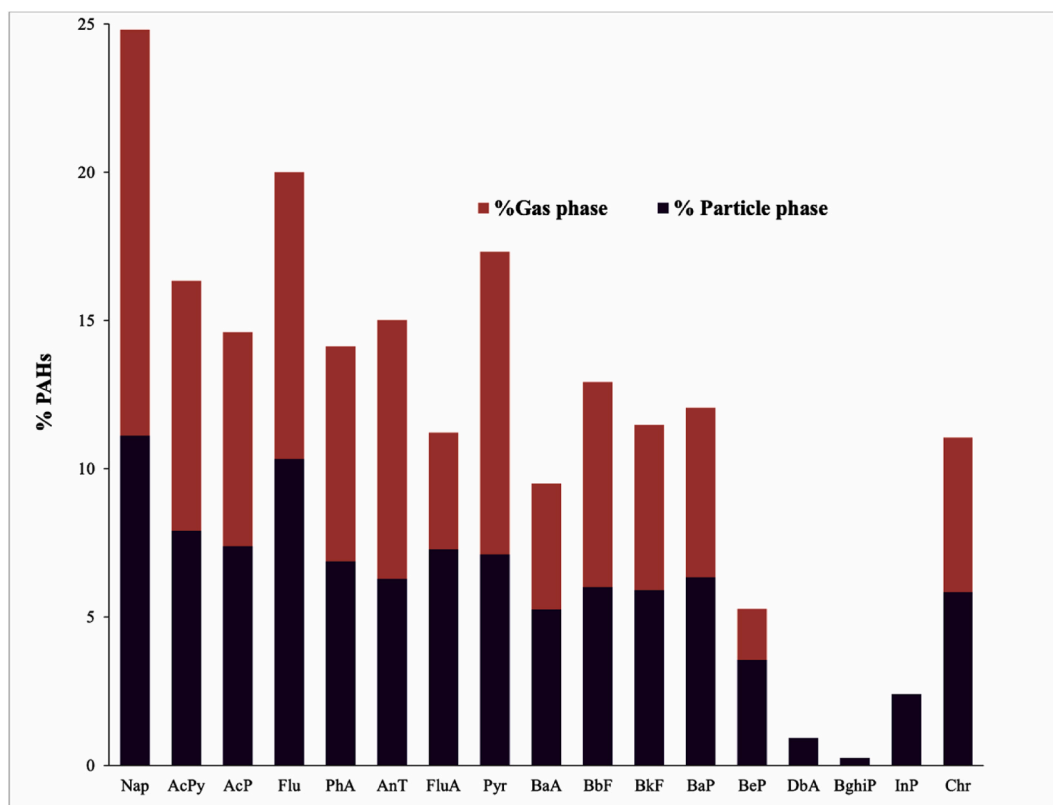


Fig. 2. %PAHs in gaseous and particulate phases.

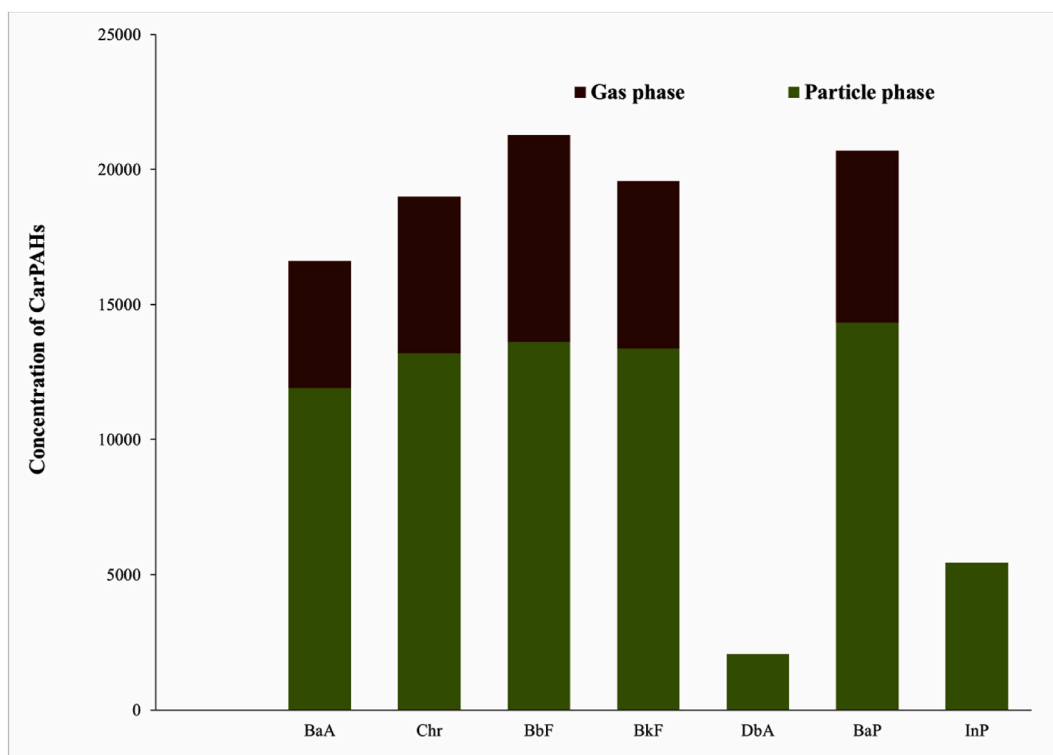


Fig. 3. Carcinogenic %PAHs in gaseous and particulate phases.

negatively correlated with ventilation and the floor number (ground floor, basement, or upper floors). Regression analysis indicated that the coffee shop area significantly affected PM concentration. Larger coffee shops, with wider doors and large windows that facilitate air exchange through natural ventilation, had reduced levels of suspended particles [37]. The study found notable differences in PM concentrations between ground-floor and basement coffee shops. Among the sampled locations, 19 coffee shops were on the ground floor or first floor, and four were in the basement. The findings indicated that ground-floor coffee shops had lower PM levels compared to those in basements [37]. Other factors, such as indoor temperature, outdoor temperature, and distance from the main street, did not significantly affect indoor PM levels.

According to Table 4, the concentration of some internal PAH compounds correlated only with relative humidity, indoor temperature, and the location of the coffee shops (distance from the main street). Other factors, such as the number of floors, ventilation, area, and outdoor temperature, did not have a significant effect [37,59].

Ultimately, the differences in ventilation systems or specific configuration patterns in coffee shops could explain the observed variations in PAH concentrations. Additionally, these factors may vary between coffee shops. It is important to note that the micro-environmental evaluations were conducted solely during the winter and over a brief period, limiting the generalizability of the findings to other seasons and larger sample sizes [59].

3.4. Health risk assessment

In order to determine the LTCR probability estimation of PAHs regarding the indoor air of coffee shops in Zahedan City for adults and children, the Monte Carlo simulation method was employed (Fig. 4). To assess the risk and the LTCR, 100 thousand trials were conducted and the total BaP_{eq} PAHs were used, respectively. The calculated mean LTCRs were $9.43 \times 10^{-6} \pm 5.06 \times 10^{-6}$ and $5.34 \times 10^{-6} \pm 2.87 \times 10^{-6}$ for adults and children, respectively. These results represent an increased risk in comparison with the appropriate baseline level of 1×10^{-6} . Considering the international regulatory standards, LTCR scores of lower than 1×10^{-6} show an acceptable safety level, scores within the range of $1 \times 10^{-6} - 1 \times 10^{-4}$ represent a probable risk and values higher than 1×10^{-4} signify an increased level of risk [72].

Fig. 4 shows the probability density curves illustrating the LTCR aggregated value of PAHs among adults and children. The risk distribution of PAHs for both age groups was expressed using predefined percentiles of the Monte Carlo simulation (5th, 30th, 50th, and 95th). The probability distributions for both age groups were skewed to the right, indicating a bias toward low-risk values. Although the USEPA's suggested admissible threshold for exposure is 1×10^{-6} , the fifth percentile indicated an exposure of about 1×10^{-8} for adults and children. In 95 % of instances, the cancer risk followed by inhaling 17 PAHs was greater than 1×10^{-7} for both groups. The 15th percentile score for both adults and children indicated no risk of cancer, as it was lower than 1×10^{-6} [73].

Table 3

Pearson correlation test between individual PAHs concentrations (particulate phase).

	Nap	AcPy	AcP	Flu	PhA	AnT	FluA	Pyr	BaA	BbF	BkF	BaP	Chr	PM
Nap	1	0.44*	0.81**	0.65**	0.56**	0.51*		0.62**	0.70**	0.50*		0.69**	0.45*	0.42*
AcPy		1		0.60**	0.72**	0.58**			0.46*		0.44*			0.57**
AcP			1	0.55**	0.54**			0.63**	0.50*	0.48*		0.45*		
Flu				1	0.70**	0.46*	0.42*					0.42*		
PhA					1	0.51*						0.45*		
AnT						1			0.60**	0.58**		0.54**		0.77**
FluA							1							
Pyr								1						
BaA									1	0.42*		0.74**		0.43*
BbF										1				0.64**
BkF											1			
BaP												1		0.51*
Chr													1	
PM														1

Single stated correlations are significant at $P = 0.05$ while double stated correlation coefficients are significant at $p = 0.01$.

∞

Table 4
Mixed-effects regression analysis PM and PAHs levels in the indoor air of coffee shops and other variables.

Independent variable	dependent variable	Regression model	Unstandardized Coefficients		Standardized Coefficients	P-value
			B	Std. Error	Beta	
Relative humidity	PM	Linear	0.292	0.100	0.539	0.001
	Nap	Linear	0.154	0.067	0.448	0.003
	AcP	Linear	0.364	0.092	0.718	0.002
	PhA	Linear	0.123	0.032	0.653	0.003
	AnT	Linear	0.134	0.050	0.533	0.001
	BaP	Linear	0.835	0.038	0.492	0.004
	BbF	Linear	0.002	0.001	0.445	0.034
	FluA	Linear	-0.002	0.001	-0.516	0.012
	Surface-area Location	PM	Linear	-0.441	0.204	0.426
PhA		Linear	0.000	0.000	-0.404	0.046
AnT		Linear	0.000	0.000	-0.478	0.021
Floor number	FluA	Linear	0.000	0.000	0.490	0.018
	PM	Linear	-1.679	0.630	-0.503	0.001
	AnT	Linear	-0.760	0.318	-0.491	0.003
Ventilation	PM	Linear	-0.570	0.197	-0.533	0.000
Indoor temperature	BbF	Linear	0.001	0.001	0.423	0.044

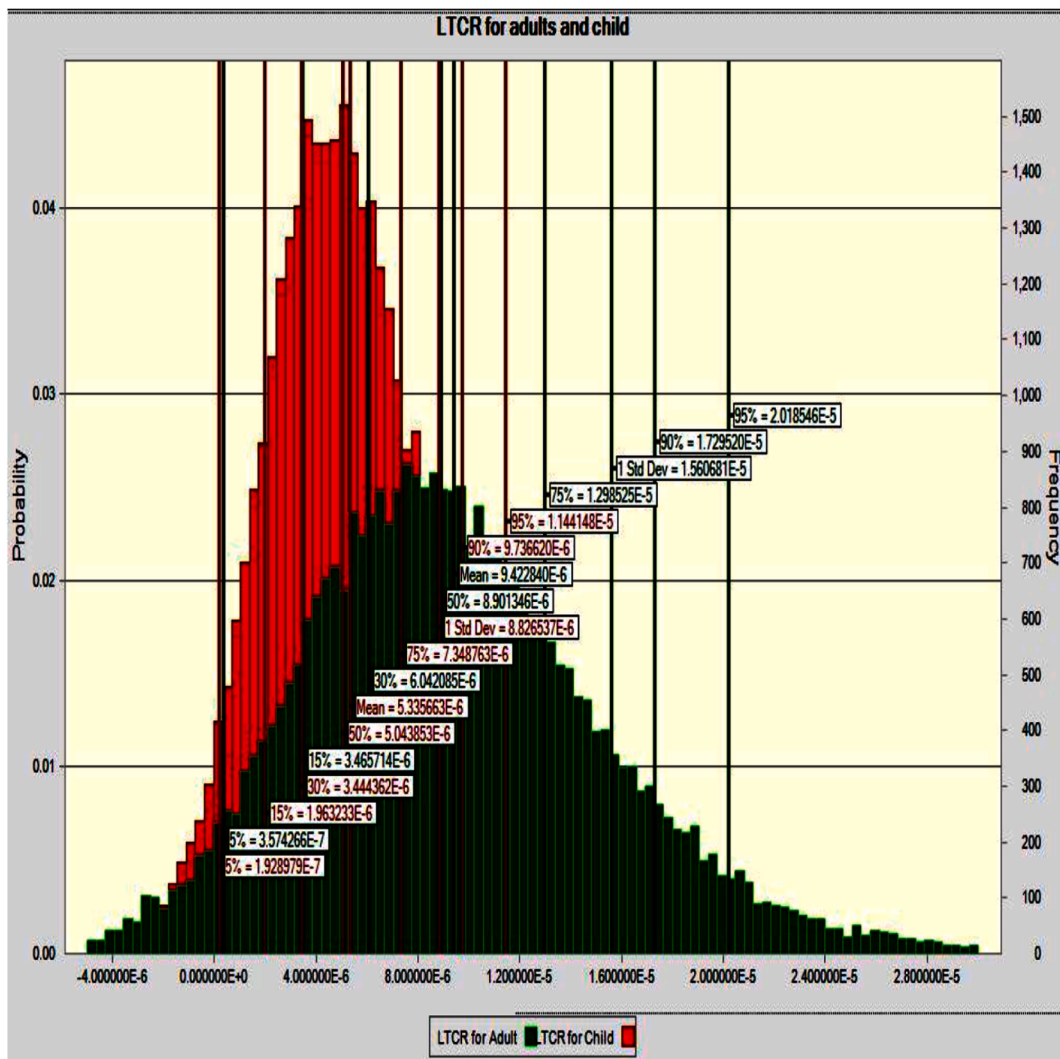


Fig. 4. Predicted probability density functions of PAHs exposure risks in the indoor air of Zahedan coffee shops for children and adults.

In Zahedan, adults are more likely to develop cancer from indoor air PAH exposure than children. The 30th and 50th percentile values of the total cancer risk for children were approximately 3.4 and 5.02 times higher than the acceptable value (1×10^{-6}), whereas, for adults, these values were around 6.05 and 8.87 times greater. The 75th and 95th percentile values demonstrated that the risk for adults would increase by 12.97 and 20.45 times, respectively, while the risk for children would increase by 7.36 and 11.52 times. Although indoor air PAHs may pose a cancer risk, the current investigation revealed no evidence of a substantial risk to either group, as all values were below 1×10^{-4} .

The results showed that the mean PAHs HQ for adults and children were 1.70 ± 2.57 and 0.56 ± 2.53 , respectively. In this vein, the HQ scores greater than one show that the level of exposure was high, which is related to an increased level of risk for the chronic non-cancerous diseases in different parts of human body [74,75].

To ensure safety, concentrations of PAHs should be reduced to safe and healthy levels by significantly increasing the interior air exchange rate. Given the considerable non-carcinogenic dangers associated with PAHs, proper written health alert notices and regulations addressing indoor smoking should be administered to mitigate health risks in these areas.

3.5. Suggested solutions to control PM and PAHs

Indoor air quality has become increasingly important, especially with the advent of COVID-19. Ventilation is probably the most popular method for reducing indoor air pollution concentrations. However, when outdoor air is more contaminated, or ventilation is impractical, additional strategies such as source management and pollutant extraction must be employed. Air cleaning technologies, a rapidly advancing field in IAQ, offer effective solutions. Several air treatment technologies can be utilized to manage pollutants, including physicochemical methods such as filtration, adsorption, UV-photocatalytic oxidation, ultraviolet disinfection, and ionization. Biological methods, such as using certain plants that can absorb and metabolize pollutants, can also be effective. Implementing these technologies and strategies can significantly reduce concentrations of PM and PAHs, leading to a safer and healthier indoor environment [76].

Among the mentioned methods, phytoremediation is regarded as a cost-effective solution with low maintenance and implementation costs compared to other technologies. Phytoremediation involves the use of plants to absorb air pollutants. Numerous studies on indoor air purification have demonstrated that plants are effective in eliminating dangerous contaminants. This technique can be implemented through active filtration utilizing green walls or plant filters or passive filtration with potted plants [77,78].

4. Limitation

One of the limitations of this research was the restricted number of samples. The inability to collect samples in different months and seasons due to the non-cooperation of coffee shop owners or changes in the use of these spaces impacted the study. Consequently, this limitation somewhat affected the ability to correlate pollutant levels with environmental factors accurately.

5. Conclusion

Indoor air pollution significantly affects human health in both developed and underdeveloped countries. This study identified coffee shops in Zahedan City as a significant source of exposure to carcinogenic substances. The proposed sources of PAH emissions included the burning of fossil fuels (cooking), the entry of PAHs from outside sources, cigarette smoke, and ETS, also known as second-hand and third-hand smoke. The LTCR of inhaling PAHs was calculated using the Monte Carlo simulation method. Since all values were less than 10^{-4} , the current investigation showed no substantial possible health risk for either children or adults. However, HQ values were more than one, indicating unacceptably high exposure levels with a high risk of chronic non-cancerous diseases affecting target organs in the human body.

Compliance with ethical standards

This article does not contain any studies involving human or animal subjects.

Data availability

Data will be made available on request.

CRedit authorship contribution statement

Shahnaz Sargazi: Writing – review & editing, Writing – original draft, Software, Methodology, Formal analysis, Data curation, Conceptualization. **Seyed Mehdi Tabatabaei:** Writing – review & editing, Resources, Conceptualization. **Mohammad Hassan Ehrampoush:** Writing – review & editing, Conceptualization. **Ramin Saravani:** Writing – review & editing, Formal analysis, Conceptualization. **Mohammad Javad Zare Sakhvidi:** Writing – review & editing, Data curation, Conceptualization. **Hossein Fallahzadeh:** Writing – review & editing, Software, Formal analysis, Data curation, Conceptualization. **Ali Asghar Ebrahimi:** Writing – review & editing, Formal analysis, Conceptualization.

Declaration of competing interest

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

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.heliyon.2024.e36291>.

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