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# Environmental Occurrence of PAHs, Their FTIR-Derived Functional Groups, and Human Health Risks of Deposited Dust from Hospitals in Northwestern Nigeria

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## Abstract

# Background

The study investigated the occurrence of polycyclic aromatic hydrocarbons (PAHs), associated functional groups, and potential human health risks in deposited dust from selected teaching hospitals in Northwestern Nigeria.

# Methods

Deposited dust samples were collected from indoor, outdoor, and junction areas of five teaching hospitals and analyzed for 16 PAHs using gas chromatography–flame ionization detection (GC-FID). Fourier Transform Infrared (FTIR) spectroscopy was used to identify functional groups in the dust matrix. Hierarchical cluster analysis of PAH concentrations was conducted in Minitab 22 to assess spatial similarities among sampling environments. Human health risks were evaluated using the U.S. EPA model by estimating chronic daily intake for adults and children via ingestion, inhalation, and dermal contact. Non-carcinogenic risk was expressed as the Hazard Index, while carcinogenic risk was assessed using incremental lifetime cancer risk based on benzo(a)pyrene equivalency and interpreted against the U.S. EPA acceptable range ($10^{-6}$–$10^{-4}$).

# Results

Total PAH concentrations (Σ16PAHs) were detected in all deposited dust samples. Molecular weight distribution indicated dominance of high-molecular-weight PAHs (4–6 rings), including benzo(a)pyrene, benzo(b)fluoranthene, chrysene, and indeno(1,2,3-cd)pyrene. FTIR spectra consistently showed absorption bands at 3420 $cm^{-1}$ (O–H), 3050 $cm^{-1}$ (aromatic C–H), 2920–2850 $cm^{-1}$ (aliphatic C–H), 1705 $cm^{-1}$ (C = O), and 1600 $cm^{-1}$ (aromatic C = C), confirming PAH-related functional groups. Cluster analysis demonstrated distinct grouping of sampling environments, with indoor samples separating from outdoor and junction locations. Toxicological classification identified carcinogenic, genotoxic, and teratogenic PAHs. Non-carcinogenic risk was low, with Hazard Index values (0.0043–0.0084) below the U.S. EPA threshold (HI < 1). Incremental lifetime cancer risk was higher in children than adults and largely within the U.S. EPA acceptable range ($10^{-6}$–$10^{-4}$).

# Conclusions

The study revealed the occurrence of PAHs and associated functional groups in deposited dust from hospital environments. Health risk assessment showed that non-carcinogenic risks were within acceptable limits, while cancer risks, particularly for children, were of potential concern. These findings

show the need for improved dust management, ventilation, and pollution control strategies in healthcare facilities to reduce chronic PAH exposure.

# Introduction

Polycyclic aromatic hydrocarbons (PAHs) are a class of ubiquitous organic contaminants composed of two or more fused aromatic rings, primarily generated through incomplete combustion of organic materials such as fossil fuels, biomass, and waste [1]. Due to their hydrophobicity, chemical persistence, and semi-volatile nature, PAHs are widely distributed across environmental compartments, including air, soil, water, and indoor environments [2]. Several PAHs have been classified as carcinogenic, mutagenic, genotoxic and teratogenic [1, 3], raising substantial public health concerns worldwide. Human exposure to PAHs occurs through multiple pathways, notably inhalation of contaminated air, ingestion of dust and food, and dermal contact with polluted surfaces. In indoor environments, particularly those characterized by high human activity, limited ventilation, and diverse emission sources, PAHs can accumulate in settled or deposited dust, serving as both a sink and a secondary source of exposure [4].

Deposited dust acts as an effective carrier of PAHs due to its high surface area and affinity for hydrophobic compounds [5]. Once deposited on floors, windowsills, furniture, and ventilation systems, dust can be resuspended through human movement, cleaning activities, or airflow, thereby increasing inhalation exposure. Additionally, dust ingestion, particularly through hand-to-mouth behavior, represents a significant exposure pathway, while dermal contact may contribute to cumulative PAH intake [6]. These exposure routes are especially relevant in hospital environments where prolonged occupancy and repeated contact with indoor surfaces are common.

Beyond quantifying PAHs, understanding the broader chemical composition of deposited dust is essential for elucidating pollutant sources, transformation processes, and potential interactions that influence toxicity and bioavailability. Fourier Transform Infrared Spectroscopy (FTIR) has emerged as a powerful, non-destructive analytical technique for characterizing functional groups in complex environmental matrices [7]. FTIR enables the identification of key organic and inorganic functional groups such as aliphatic and aromatic C–H, carbonyl (C = O), hydroxyl (O–H), nitro (N–O), sulfate, and silicate groups, which collectively provide information on dust composition, source contributions, and chemical ageing [8].

Integrating FTIR functional group analysis with PAHs assessment offers a more comprehensive chemical characterization of deposited dust than either approach alone. While chromatographic techniques, FTIR contributes complementary information on the overall chemical matrix, enabling source inference and interpretation of pollutant behavior within dust [7]. This integrated approach is particularly valuable in resource-limited settings, where advanced analytical infrastructure may be constrained, yet robust chemical insights are still required to inform environmental health decision-making.

Human health risk assessment is a critical component of environmental contamination studies, translating chemical concentrations into meaningful indicators of potential adverse effects. For PAHs,

risk assessment commonly focuses on both non-carcinogenic and carcinogenic risks through established exposure pathways, including ingestion, inhalation, and dermal contact. Benzo[a]pyrene and other high-molecular-weight PAHs are of particular concern due to their strong carcinogenic potential [9]. Estimating incremental lifetime cancer risk (ILCR) associated with PAHs exposure in hospital environments is essential for evaluating occupational and public health implications. However, few studies have assessed PAH-related health risks in hospital dust, and such assessments are absent from Nigerian literature.

Existing studies in Nigeria and other African countries have predominantly focused on heavy metals in foods, indoor and outdoor dust, with comparatively little attention given to organic contaminants such as PAHs [10, 11, 12, 13, 14]. Where PAHs have been investigated, studies have largely targeted outdoor air, soil, or road dust [15, 16, 17], leaving indoor healthcare environments understudied. Furthermore, few investigations have employed integrated analytical approaches that combine pollutant quantification, functional group characterization, and health risk assessment. This knowledge gap limits the ability to fully understand the chemical complexity of hospital dust and its implications for human exposure and health.

Against this backdrop, the present study investigated the environmental occurrence of PAHs in deposited dust collected from teaching hospitals in Northwestern Nigeria, characterized functional groups using FTIR spectroscopy, and evaluated potential human health risks associated with PAHs exposure. The findings are expected to contribute baseline data for hospital indoor, outdoor and junction environments in Nigeria, support evidence-based risk management strategies, and advance the application of integrated chemical assessment frameworks in environmental health research.

## Experimental Methods

# Study Area and Sampling Locations

The study was conducted in Kano, Katsina and Kaduna. The five teaching hospitals in Kano, Katsina and Kaduna were purposively selected for the study. Deposited dust samples were collected from indoor, outdoor, and junction environments within the hospital premises. Indoor locations included offices, wards, and corridors, while outdoor sites comprised open spaces around hospital buildings. Junction area represented a junction on the road network close to the hospital. These locations were selected to capture spatial variations in deposited dust composition and potential pollutant sources within the hospital environment. Information on the sample collection codes and dates are presented in Table 1.

Table 1
Information on sample collection in the studied teaching hospitals

| S/N | Study location | Code | Date sample was collected |
|---|---|---|---|
| 1 | Amina Kano Teaching Hospital Kano Indoor | AKTH I | 12/08/2024 |
| 2 | Amina Kano Teaching Hospital Kano Outdoor | AKTH O | 12/08/2024 |
| 3 | Junction close to Amina Kano Teaching Hospital | AKTH J | 12/08/2024 |
| 4 | Muhammad Abdullahi Wase Teaching Hospital Indoor | KNSTH I | 15/08/2024 |
| 5 | Muhammad Abdullahi Wase Teaching Hospital Outdoor | KNSTH O | 15/08/2024 |
| 6 | Junction close to Muhammad Abdullahi Wase Teaching Hospital | KNSTH J | 15/08/2024 |
| 7 | Ahmadu Bello University Teaching Hospital Kaduna Indoor | ABUTH I | 16/08/2024 |
| 8 | Ahmadu Bello University Teaching Hospital Kaduna Outdoor | ABUTH O | 16/08/2024 |
| 9 | Junction close to Ahmadu Bello Teaching Kaduna Hospital | ABUTH J | 16/08/2024 |
| 10 | Barau Dikko Teaching Hospital Kaduna Indoor | KDSTH I | 19/08/2024 |
| 11 | Barau Dikko Teaching Hospital Kaduna Outdoor | KDSTH O | 19/08/2024 |
| 12 | Junction close to Barau Dikko Teaching Hospital Kaduna | KDSTH J | 19/08/2024 |
| 13 | Federal Teaching Hospital, Katsina Indoor | KTSTH I | 13/08/2024 |
| 14 | Federal Teaching Hospital, Katsina Outdoor | KTSTH O | 13/08/2024 |
| 15 | Junction close to Federal Teaching Hospital Katsina | KTSTH J | 13/08/2024 |

## Deposited Dust Sample Collection

Deposited dust samples were collected using a manual brushing method, which is widely applied for dust studies [18, 19, 20, 11]. Clean, soft-bristle brushes were used to gently sweep dust from smooth surfaces such as window sills, shelves, ledges, floors, and outdoor pavements to form composite samples for indoor, outdoor, and junction samples, respectively. Prior to sampling, brushes and collection containers were thoroughly cleaned to avoid cross-contamination. Dust from each sampling point was carefully transferred into pre-labelled, airtight polyethylene sample containers. Separate

samples were collected for indoor, outdoor, and junction environments. The collected samples were transported to the laboratory and stored in a clean, dry environment at room temperature until analysis.

## GC FID Sample Preparation

In the laboratory, dust samples were air-dried at ambient temperature to remove residual moisture. Visible debris, such as stones, fibres, or plant fragments, was manually removed. The dried samples were gently homogenized using a clean agate mortar and pestle to obtain a fine and uniform particle size suitable for spectroscopic analysis. Prepared samples were stored in desiccators prior to GC-FID analysis and FTIR analysis.

## Solvent Extraction of PAHs (EPA Method 3540C) for GC FID

PAHs were extracted from the solid samples using solvent extraction following EPA Method 3540C with slight modifications. Briefly, 10 g of each homogenized sample was accurately weighed into a clean glass extraction container. To remove residual moisture, 1–2 g of anhydrous sodium sulfate ($Na_2SO_4$) was thoroughly mixed with the sample until a free-flowing, dry consistency was achieved. A volume of 20–40 mL of extraction solvent (dichloromethane or n-hexane) was added to completely immerse the sample matrix. The mixture was placed in an ultrasonic bath and sonicated for 15–30 minutes at 40 kHz, facilitating efficient desorption of PAHs from the solid matrix into the solvent phase. After sonication, the extract was allowed to settle and subsequently filtered using Whatman No. 1 filter paper and a glass funnel into a clean, pre-labelled glass container.

## Clean-Up of Extracts (Silica Gel Clean-Up, EPA Method 3630C) for GC FID

To remove co-extracted interferences such as lipids, pigments, and other polar organic compounds, the filtered extracts were subjected to silica gel column clean-up following EPA Method 3630C. A glass chromatography column (10–15 cm length, 1 cm internal diameter) was packed with 5 g of activated silica gel (60–100 mesh) and topped with a 1 cm layer of anhydrous sodium sulfate to prevent moisture interference. The column was preconditioned with 10 mL of the extraction solvent (dichloromethane or n-hexane). The filtered extract was carefully loaded onto the column using a glass pipette and allowed to percolate at a controlled flow rate. Elution was carried out with 20 mL of the extraction solvent, and the eluate was collected into a clean glass container. The cleaned extract was then concentrated by allowing the solvent to evaporate under ambient conditions in a fume hood or under a gentle stream of high-purity nitrogen gas. The final volume was reduced to 1 mL, after which the concentrate was transferred into a pre-labelled GC vial fitted with a Teflon-lined cap for chromatographic analysis.

# GC-FID Analysis of PAHs (EPA Method 8100)

Quantitative determination of PAHs was performed using Gas Chromatography coupled with a Flame Ionization Detector (GC-FID) in accordance with EPA Method 8100. Analysis was carried out using a Buck M910 Scientific Gas Chromatograph equipped with an FID, which provides high sensitivity for trace-level organic contaminants. Separation was achieved on a VF-5 capillary column (30 m + 10 m EZ-Guard column × 0.25 mm internal diameter, 0.25 µm film thickness). The injector and detector temperatures were maintained at 250°C and 280°C, respectively. The oven temperature program was set as follows: initial temperature of 120°C held for 4 min, ramped at 10°C $min^{-1}$ to 180°C and held for 2 min, followed by a final ramp at 5°C $min^{-1}$ to 300°C. Helium was used as the carrier gas at a constant flow rate of 1.0 mL $min^{-1}$, with a detector make-up gas flow of 29 mL $min^{-1}$. A 1.0 µL aliquot of the extract was injected for analysis. This method was similar to the methods used by Di Fiore et al. [21] and Olatunji et al. [22].

## Quality Control, Calibration, and Quantification

Quantification of PAHs was carried out using the external standard method. A certified PAH standard mixture (EPA 610 PAH Mix) obtained from Supelco was used for calibration. The standard mixture contained 16 priority PAHs, including naphthalene (Nap), acenaphthylene (Acy), acenaphthene (Ace), fluorene (Flu), phenanthrene (Phen), anthracene (Anth), fluoranthene (Fla), pyrene (Pyr), benzo(a)anthracene (BaA), chrysene (Chy), benzo(b)fluoranthene (BbF), benzo(k)fluoranthene (BkF), benzo(a)pyrene (BaP), dibenz(a,h)anthracene (DbA), benzo(ghi)perylene (BghiP), and indeno(1,2,3-cd)pyrene (IP). Five-point calibration curves were generated by plotting peak areas against corresponding standard concentrations, yielding excellent linearity ($R^2 > 0.99$). Identification of PAHs in samples was based on comparison of retention times with those of the injected standards. Quantification was achieved by extrapolating sample peak areas from the calibration curves within the linear detector response range. The method detection limits (LOD) ranged from 0.0007 to 0.016 µg $kg^{-1}$, while limits of quantification (LOQ) ranged between $1.8 \times 10^{-7}$ and $4.1 \times 10^{-5}$ µg $kg^{-1}$. Method recovery was assessed by analysing filters spiked with known concentrations of PAH standards, with recoveries generally ranging between 70–80%. Field and laboratory blanks were routinely analysed to ensure data quality, and blank levels were typically very low or below detection limits.

## Fourier Transform Infrared (FTIR) Analysis

Functional group characterization of the deposited dust samples was carried out using Fourier Transform Infrared (FTIR) spectroscopy [23]. The analysis was performed with a Thermo Fisher Scientific FTIR spectrometer under standard operating conditions. Spectra were recorded in the mid-infrared region ranging from 4000 to 400 $cm^{-1}$, with a spectral resolution of 4 $cm^{-1}$. For each sample, 16 scans were collected and averaged to improve the signal-to-noise ratio, while 16 background scans were recorded prior to sample analysis. The instrument parameters included a sample gain of 1.0, an optical velocity of 0.4747, and an aperture setting of 100. Samples were analyzed directly without chemical treatment, and spectra were recorded in % transmittance mode.

# Spectral Interpretation and Functional Group Assignment

FTIR spectra were visualized and interpreted using Python (Matplotlib and NumPy). Major absorption bands were identified based on peak positions and compared with standard reference spectra and published literature to assign corresponding functional groups. Identified functional groups were used to infer the presence of mineral components, inorganic salts, silicates, and possible organic or anthropogenic contaminants in the deposited dust.

# Exposure Assessment and Chronic Daily Intake (CDI)

Human exposure to PAHs in deposited dust was assessed for adults and children through ingestion, dermal contact, and inhalation pathways following U.S. EPA human health risk assessment guidelines [24, 25].

Chronic daily intake (CDI) was calculated as:

$$CDI_{ing} = (C \times IR \times EF \times ED) / (BW \times AT)$$

$$CDI_{derm} = (C \times SA \times AF \times ABS \times EF \times ED) / (BW \times AT)$$

$$CDI_{inh} = (C \times IR_{air} \times EF \times ED) / (PEF \times BW \times AT)$$

Where C is PAH concentration, IR is ingestion rate, SA is skin surface area, AF is adherence factor, ABS is dermal absorption factor, IR_air is inhalation rate, PEF is particle emission factor, EF is exposure frequency, ED is exposure duration, BW is body weight, and AT is averaging time. The values for these parameters are in the supplementary document S1.

# Non-Carcinogenic Risk Assessment

Hazard Quotient (HQ) was calculated as HQ = CDI / RfD. The Hazard Index (HI) was obtained by summing HQ values across pathways. HI > 1 indicates a potential for adverse non-carcinogenic health effects.

# Carcinogenic Risk Assessment

Carcinogenic risk was assessed using Incremental Lifetime Cancer Risk (ILCR). PAH concentrations were converted to benzo(a)pyrene equivalent concentrations (BaP_eq) using toxicity equivalency factors.

$$BaP_{eq} = \Sigma (Ci \times TEFi)$$

$$ILCR = CDI \times CSF$$

Total incremental lifetime cancer risk (ILCR) was estimated by summing the cancer risks from ingestion, dermal contact, and inhalation exposure pathways. The resulting combined risk values were evaluated

against the U.S. Environmental Protection Agency's benchmark acceptable risk range, defined as $1\times10^{-6}$ to $1\times10^{-4}$ for carcinogenic effects [26].

# Data Analysis

All statistical analyses were performed using Minitab (version 22). The PAH concentration data obtained from laboratory analyses were initially screened for completeness and consistency. Descriptive statistics, including mean values, were calculated for individual PAHs to summarize their concentration levels across the hospital sites. Boxplots were constructed to visualize data distribution, central tendency, variability, and potential outliers among the different sites. All graphical outputs were produced using Minitab. In addition, cluster analysis was employed to classify sampling locations and PAHs based on similarities in their concentration profiles.

# Compliance with Guidelines and Regulations

All sampling procedures, laboratory analyses, and human health risk assessment methods used in this study were performed in accordance with relevant international guidelines and regulatory frameworks, including the United States Environmental Protection Agency (U.S. EPA) Exposure Factors Handbook, the United States Environmental Protection Agency (U.S. EPA) Risk Assessment Guidance for Superfund (RAGS), Volume I: Human Health Evaluation Manual, and applicable United States Environmental Protection Agency (U.S. EPA) Test Methods for Evaluating Solid Waste (SW-846) protocols governing PAH extraction, clean-up, and GC-FID analysis. Fourier Transform Infrared (FTIR) measurements and spectral interpretation were conducted following established spectroscopic principles and band assignment conventions described in the Spectrometric Identification of Organic Compounds and the Interpretation of Infrared Spectra: A Practical Approach.

# Ethics Committee

### Ethical approval

for this study was obtained from the following committees:

1. Rivers State Health Research Ethics Committee
2. Ahmadu Bello University Teaching Hospital Health Research Ethics Committee
3. Federal Teaching Hospital Katsina Health Research Ethical Review Committee
4. Kaduna State Health Research Ethics Committee

# Results and Discussions

The present study revealed the ubiquitous occurrence of PAHs in deposited dust collected from hospital environments in Northwestern Nigeria, confirming that healthcare settings are not exempt from contamination by organic pollutants. Detectable levels of all 16 PAHs across indoor, outdoor, and junction environments highlight deposited dust as an important sink and secondary exposure medium.

Similar observations have been reported in indoor and bar settings, where dust acts as a reservoir for semi-volatile organic compounds, including PAHs [27, 28].

The spatial distribution patterns (Figs. 1 and 2) demonstrated relatively higher PAH burdens in outdoor and junction environments compared with indoor locations. This gradient reflects the influence of outdoor emission sources such as vehicular traffic, diesel-powered generators, refuse burning, and surrounding urban activities, which are characteristic of Nigerian settings [29, 30]. Junction areas, representing interfaces between indoor and outdoor spaces, showed elevated PAH concentrations due to particle infiltration and resuspension, a phenomenon widely documented in indoor air–dust exchange studies [31, 32].

FTIR spectroscopy provided complementary chemical evidence (Figs. 3–7 and Table 2) supporting the chromatographic identification of PAHs in deposited dust. The consistent detection of absorption bands at 3050 $cm^{-1}$, 1600 $cm^{-1}$, and 800 $cm^{-1}$ corresponds to aromatic C–H stretching, aromatic C = C stretching, and aromatic C–H out-of-plane bending vibrations, respectively. The observed FTIR absorption bands fall within standard functional-group regions reported in the literature (Silverstein et al., 2014; Smith, 2011; Socrates, 2004; Coates, 2000). These bands are widely recognized as diagnostic features of aromatic hydrocarbon structures, which are the chemical backbone of PAHs [33].

The presence of aliphatic C–H stretching bands (2920–2850 $cm^{-1}$) suggests co-existing alkanes and petroleum-derived residues that often accompany PAHs in combustion emissions. Similarly, the carbonyl (C = O) band at 1705 $cm^{-1}$ and hydroxyl (O–H) band at 3420 $cm^{-1}$ indicate oxygenated functional groups, which may arise from atmospheric aging, photo-oxidation, and the formation of oxy-PAHs [34]. The detection of C–H bending and N–O stretching bands around 1450 $cm^{-1}$ further suggests the possible presence of nitro-PAHs, which are formed during high-temperature combustion and secondary atmospheric reactions. Nitro-PAHs are of particular concern due to their strong mutagenic and genotoxic properties [35, 36].

Table 2
FTIR Absorption Bands and Observed Functional Groups in Deposited Dust Samples Linked to PAHs Identified in Deposited Dust

| Wavenumber Range ($cm^{-1}$) | Observed Peak ($cm^{-1}$) | Functional Group Assignment | Link to PAHs | Probable Source / Interpretation |
|---|---|---|---|---|
| 3600–3200 [37, 38] | 3420 | O–H stretching (hydroxyl) | Directly associated with aromatic ring structures of PAHs | Moisture, alcohols, phenols, clay minerals |
| 3000–2850 [37, 38] | 2920, 2850 | Aliphatic C–H stretching (–$CH_2$, –$CH_3$) | Indicative of condensed aromatic systems typical of high-MW PAHs | Alkanes, petroleum residues, organic matter |
| 3100–3000 [37, 38] | 3050 | Aromatic C–H stretching | Fingerprint region for substituted PAHs | Aromatic hydrocarbons (PAHs) |
| 1750–1650 [37, 38] | 1705 | C = O stretching (carbonyl) | Associated with alkylated PAHs and co-emitted hydrocarbons | Ketones, aldehydes, carboxylic acids |
| 1650–1580 [37, 38] | 1600 | C = C stretching (aromatic ring) | Reflects oxygenated PAH derivatives (oxy-PAHs) | Aromatic compounds, soot-related carbon |
| 1550–1350 [37, 38] | 1450 | C–H bending / N–O stretching | Associated with nitro-PAHs and organic combustion residues | Organic matter, nitro compounds |
| 900–700 [37, 38] | 800 | Aromatic C–H out-of-plane bending | Link to PAHs | Substituted aromatic rings (PAHs) |

Cluster analysis (Fig. 8) revealed grouping patterns that reflect similarities in PAHs composition among hospitals and environments. Indoor samples tended to cluster separately from outdoor and junction samples, reinforcing the influence of external emission sources on PAH profiles. Similar clustering trends have been reported in urban dust studies where traffic density and fuel combustion intensity drive spatial variability [39]. The clustering of junction samples with outdoor samples further confirms the role of dust transport and resuspension at building interfaces.

Table 3 and Fig. 9 highlight the molecular characteristics and carcinogenic classifications of the detected PAHs, providing a toxicological context for interpreting exposure risks. The predominance of PAHs containing 4–6 aromatic rings aligns with the observed dominance of high-molecular-weight compounds, which are known to be more persistent, less volatile, and more strongly adsorbed to particulate matter than low-molecular-weight PAHs [3]. These properties increase their environmental persistence and potential for chronic human exposure. Of particular concern is the consistent detection of benzo(a)pyrene, which is classified as Group 1 (carcinogenic to humans) [40, 41]. Benzo(a)pyrene is widely used as an indicator compound for PAH-related carcinogenic risk due to its strong mutagenic properties and well-established cancer potency [42]. The presence of several Group 2A and Group 2B

PAHs, including dibenzo(a,h)anthracene, benzo(b)fluoranthene, and indeno(1,2,3-cd)pyrene, further suggests the potential for cumulative carcinogenic effects arising from combined exposure to multiple PAHs.

Although several low-molecular-weight PAHs detected in this study are classified as Group 3 (not classifiable as to carcinogenicity), their environmental relevance should not be overlooked. These compounds can serve as precursors to more toxic derivatives through atmospheric transformation processes and may contribute indirectly to overall health risk [43]. The toxicological profile presented in Table 3, therefore emphasizes that deposited dust in hospital environments contains a chemically diverse mixture of PAHs with varying degrees of health significance.

Table 3
PAHs analyzed, number of aromatic rings in the chemical structure, molecular weight (MW), and their IARC classifications

| PAH | Molecular Weight (g/mol) | Number of Aromatic Rings | IARC Classification (IARC, 2010; IARC, 2012) |
|---|---|---|---|
| Naphthalene | 128 | 2 | Group 2B |
| Acenaphthylene | 152 | 3 | Group 3 |
| Acenaphthene | 154 | 3 | Group 3 |
| Fluorene | 166 | 3 | Group 3 |
| Phenanthrene | 178 | 3 | Group 3 |
| Anthracene | 178 | 3 | Group 3 |
| Fluoranthene | 202 | 4 | Group 3 |
| Pyrene | 202 | 4 | Group 3 |
| Benzo(a)anthracene | 228 | 4 | Group 2B |
| Chrysene | 228 | 4 | Group 2B |
| Benzo(e)anthracene | 228 | 4 | Group 3 |
| Benzo(b)fluoranthene | 252 | 5 | Group 2B |
| Benzo(k)fluoranthene | 252 | 5 | Group 2B |
| Benzo(a)pyrene | 252 | 5 | Group 1 |
| Dibenzo(a,h)anthracene | 278 | 5 | Group 2A |
| Benzo(g,h,i)perylene | 276 | 6 | Group 3 |
| Indeno(1,2,3-c,d)pyrene | 276 | 6 | Group 2B |

*Group 1 “carcinogenic to humans,” Group 2A “probably carcinogenic to humans,” and Group 2B “possibly carcinogenic to humans”, Group 3 “Not classifiable as to its carcinogenicity to humans*

Comparable dominance of HMW PAHs has been reported in road dust, urban dust, and indoor dust from developing countries, where fossil fuel combustion remains a major energy source [44, 45, 46, 47, 48]. The relatively lower contribution of low-molecular-weight PAHs may reflect volatilization losses under tropical conditions and the preferential adsorption of HMW PAHs onto fine dust particles.

In the present study, deposited dust from hospital environments was dominated by HMW PAHs. This pattern is characteristic of pyrogenic sources, particularly high-temperature combustion processes such as vehicular exhaust and diesel generator emissions, which are prevalent around Nigerian hospitals. This HMW dominance contrasts with the findings of Liu et al. [27] in Chinese household dust, where LMW PAHs (2–3 rings) accounted for over 85% of total PAHs. That distribution was attributed primarily to indoor cooking with coal and biomass fuels. The divergence highlights how activity type and fuel usage strongly influence PAH molecular profiles, with domestic cooking favoring LMW PAHs and traffic-related emissions favoring HMW PAHs. Despite these differences, both studies confirm that dust acts as a long-term sink for PAHs regardless of molecular class.

Findings from Nigerian indoor dust studies further support the dominance of medium- to high-ring PAHs. Offor and Nduka [45] reported that indoor dust from residential environments was a major sink for 3–5 ring PAHs, while Iwegbue et al. [48] observed dominance of 3- and 5-ring PAHs in electronic repair workshop dusts. Similarly, Kanchana-at et al. [49] identified elevated concentrations of HMW PAHs such as chrysene and benzo[a]anthracene in fine particulate matter from religious spaces, driven by incense burning. These studies, together with the present findings, indicate that HMW PAHs are consistently enriched in the environment.

The toxicological classification (Fig. 10) revealed the presence of carcinogenic, genotoxic, and teratogenic PAHs in deposited dust. Benzo(a)pyrene, classified as Group 1 (carcinogenic to humans) by IARC, was detected across multiple locations. Other PAHs classified as Group 2A and 2B were also present, indicating potential cumulative health risks [40, 41].

The co-occurrence of multiple toxic PAHs is particularly concerning in hospital environments, where vulnerable populations such as children, the elderly, and immunocompromised patients may experience prolonged exposure. Similar toxicological profiles have been reported in dust studies in Asia, emphasizing the global relevance of PAHs contamination [50, 6]. The non-carcinogenic health risk assessment indicated that Hazard Index values for both adults and children were below the U.S. EPA threshold of concern (HI < 1) (Fig. 11a,b). These results suggest that short-term or chronic non-cancer health effects are unlikely under current exposure conditions. Similar low HI values have been reported in indoor dust studies from urban environments with comparable PAH concentrations (27, 45].

However, children consistently exhibited higher HI values than adults, reflecting their lower body weight, higher dust ingestion rates, and increased hand-to-mouth behavior. This pattern aligns with established exposure models and reinforces children as a sensitive subpopulation in environmental risk assessments [17].

CDI is a fundamental metric in human health risk assessment, as it integrates contaminant concentration with exposure frequency, duration, and population-specific physiological parameters to estimate long-term daily exposure. In the present study, CDI was evaluated for adults and children through ingestion, dermal contact, and inhalation pathways, providing insight into dominant exposure routes and population vulnerability in hospital environments. The results (Fig. 12a) indicate that dust ingestion was the predominant contributor to total CDI for both adults and children, a trend widely reported in indoor and urban dust studies. This dominance reflects the strong sorption of PAHs HMW compounds onto fine dust particles, which are readily transferred to the mouth via hand-to-mouth behavior. Similar findings have been documented in household and public indoor environments. Liu et al. [27] reported that ingestion accounted for more than 80% of total PAH CDI in residential dust, while Offor and Nduka [45] observed ingestion as the primary exposure pathway in Nigerian indoor dust. Studies in schools, bars, and occupational settings have also consistently identified ingestion as the most influential route governing PAH exposure [28, 48]. In hospital environments, where dust accumulates on floors, furniture, and high-contact surfaces, ingestion-related exposure is particularly relevant for children accompanying patients or caregivers. The present findings align strongly with these evidences, confirming that ingestion-driven CDI is a characteristic of PAH exposure in dust-dominated environments.

Dermal contact represented the second most important pathway contributing to CDI for both populations. This pathway reflects direct skin contact with contaminated dust deposited on floors, bed rails, furniture, and other surfaces common in hospital settings. Although dermal absorption efficiency of PAHs is lower than ingestion, prolonged and repeated contact increases cumulative exposure. Comparable results have been reported in urban dust and occupational studies. Chen et al. [51] demonstrated that dermal contact contributed between 10–30% of total PAH CDI in indoor environments, while Jakovljević et al. [52] reported dermal exposure as a significant secondary pathway in public buildings. Nigerian studies in workshops and public spaces similarly identified dermal contact as a meaningful contributor to overall PAH intake [48].

The relevance of dermal exposure is further enhanced by the dominance of HMW PAHs observed in this study, as these compounds exhibit strong particle affinity and persistence, increasing skin contact potential. Thus, the present results corroborate existing literature that dermal contact is a non-negligible exposure route, particularly in environments with chronic dust deposition.

Inhalation consistently contributed the lowest CDI values for both adults and children. This pattern is widely reported in PAH risk assessments involving dust matrices and reflects the low volatility of HMW PAHs and limited resuspension of coarse dust particles under typical indoor conditions. Several studies have similarly reported inhalation as the least significant pathway. Liu et al. [27] and Santijitpakdee et al. [6] found inhalation contributions to total CDI to be below 5% in indoor environments, while Offor and Nduka [45] reported minimal inhalation exposure in Nigerian residential dust. Even in high-activity environments such as bars and workshops, inhalation has been shown to play a secondary role relative to ingestion and dermal contact [28].

Nonetheless, inhalation exposure may increase transiently in outdoor and junction areas due to dust resuspension from vehicular movement and foot traffic, as observed in the present study. Although quantitatively small, inhalation remains environmentally relevant, especially under chronic exposure scenarios.

Across all exposure pathways, children show higher CDI values than adults, a pattern consistently reported in PAH exposure studies. This disparity is primarily attributable to children's lower body weight, higher dust ingestion rates, greater skin surface area-to-body weight ratio, and more frequent hand-to-mouth behavior [53]. Similar population-based trends have been reported in the literature. Yang et al. [17] and Chen et al. [51] showed that children's CDI values were 2–5 times higher than adults in indoor dust environments. Nigerian studies have also reported elevated CDI and associated cancer risks for children compared with adults [45, 48]. The higher CDI values observed for children in this study directly explain the elevated HI and ILCR values reported, despite overall risks remaining within U.S. EPA acceptable limits. This reinforces children as a sensitive and priority subpopulation in hospital exposure assessments.

While numerous studies have examined CDI of PAHs in residential, occupational, and public indoor environments, data on hospital dust remain scarce, particularly in sub-Saharan Africa. The present study fills this gap by demonstrating that CDI patterns in hospitals mirror those reported in other indoor environments, with ingestion > dermal contact > inhalation and higher exposure in children.

Although non-carcinogenic risks were low, ILCR estimates (Fig. 12b) indicated potential long-term cancer risks, particularly for children. ILCR values generally fell within the U.S. EPA acceptable risk range ($10^{-6}$–$10^{-4}$), but their proximity to the upper bound in some locations warrants concern. Comparable ILCR levels have been reported in urban dust and indoor environments in developing countries, where combustion sources are prevalent [52].

In the present study, non-carcinogenic risk values (HI) for both adults and children were below the U.S. EPA threshold of concern, indicating limited potential for adverse non-cancer effects. However, ILCR values were consistently higher for children, reflecting greater vulnerability due to higher dust ingestion rates and lower body weight. This risk pattern is consistent with Liu et al. [27], who reported low overall cancer risks in household dust but demonstrated that 4–6 ring PAHs contributed more than 96% of total ILCR, with benzo(a)pyrene and dibenzo(a,h)anthracene as dominant risk drivers. Similarly, Chen et al. [51] showed that carcinogenic PAHs, expressed as benzo[a]pyrene equivalents, accounted for over 95% of total carcinogenic potential in ambient, indoor, and personal exposure, despite lower personal exposure concentrations. These findings align strongly with the present study, showing that toxic potency, rather than concentration alone, governs cancer risk.

Studies in Nigerian occupational and high-activity environments reported comparatively higher risks. Iwegbue et al. [48] found cancer risks from ingestion and dermal contact with electronic workshop dust to exceed acceptable limits, while Adesina et al. [28] reported inhalation-based ILCR values approaching regulatory thresholds in tobacco-smoke-impacted public bars. In contrast, the present hospital study

shows lower overall cancer risk magnitudes, reflecting reduced emission intensity. Nonetheless, the presence of Group 1 and Group 2 carcinogenic PAHs indicates that chronic exposure remains a concern, particularly in sensitive populations. The findings of Kanchana-at et al. [49] further illustrate how sustained indoor combustion can elevate both carcinogenic and non-carcinogenic risks beyond regulatory limits. Compared with these environments, hospitals appear less hazardous; however, the present study demonstrates that even indirect accumulation of HMW PAHs in dust can result in measurable cancer risks.

When compared with other studies, the PAHs profiles observed in Nigerian hospital dust are broadly consistent with findings from other environments in Asia, where combustion-related sources dominate PAH contamination. Studies conducted in schools and hospitals in Thailand and Ecuador have reported similar dominance of high-molecular-weight PAHs, particularly benzo(a)pyrene, chrysene, and fluoranthene, with higher concentrations in outdoor and entrance areas than in indoor wards [6, 54, 55].

However, the Nigerian context presents unique exposure considerations. The frequent reliance on diesel-powered generators due to unstable electricity supply, combined with high traffic density and limited buffer zones around hospital premises, likely amplifies PAH inputs relative to some high-income settings. Additionally, dust resuspension under dry climatic conditions may further enhance exposure potential. Despite these differences, the observed Hazard Index values in this study are comparable to those reported globally, while ILCR estimates fall within internationally accepted risk ranges, suggesting that Nigerian hospital environments exhibit similar long-term cancer risk profiles to those reported elsewhere, albeit under distinct socio-environmental conditions.

A major strength of this study is the integrated use of GC-FID, FTIR spectroscopy, and human health risk assessment, providing a comprehensive chemical and toxicological characterization of hospital dust. However, limitations include the absence of seasonal variation analysis and the lack of particle size–specific PAH measurements, which should be addressed in future studies.

# Conclusions

This study provided an integrated assessment of PAHs, associated functional groups, and potential human health risks in deposited dust from teaching hospitals in Northwestern Nigeria. The findings confirmed that dust deposited in hospital environments contains a complex mixture of PAHs, dominated by high-molecular-weight compounds. FTIR functional group analysis corroborated chromatographic results by revealing characteristic aromatic, aliphatic, carbonyl, hydroxyl, and nitro-related bands, indicating the presence of both parent PAHs and their transformed derivatives within the dust matrix. Human health risk assessment showed that non-carcinogenic risks for both adults and children were within acceptable limits; however, incremental lifetime cancer risk values were consistently higher for children and approached established regulatory thresholds in some locations. This highlights the potential for long-term health concerns arising from chronic exposure to PAH-contaminated dust in healthcare settings. The study established baseline data on hospital dust contamination in Nigeria,

demonstrated the value of integrating GC-FID with FTIR for comprehensive chemical characterisation, and showed the need for routine monitoring, improved ventilation, and effective dust management strategies to reduce PAHs exposure in hospital environments.

# Abbreviations

**AOAC** Association of Official Analytical Chemists

**BaP** Benzo(a)pyrene

**CDI** Chronic Daily Intake

**CSF** Cancer Slope Factor

**FTIR** Fourier Transform Infrared Spectroscopy

**GC-FID** Gas Chromatography–Flame Ionization Detection

**HHRA** Human Health Risk Assessment

**HI** Hazard Index

**HMW** High Molecular Weight

**IARC** International Agency for Research on Cancer

**ILCR** Incremental Lifetime Cancer Risk

**LMW** Low Molecular Weight

**LOD** Limit of Detection

**LOQ** Limit of Quantification

**MW** Molecular Weight

**PAHs** Polycyclic Aromatic Hydrocarbons

**RfD** Reference Dose

**TEQ** Toxicity Equivalent

**U.S. EPA** United States Environmental Protection Agency

# Declarations

### Ethics approval

Not applicable

**Consent to participate**

Not applicable

**Consent for publication**

Not applicable

# Competing interests

The authors declare no competing interests.

# Funding

None

# Author Contribution

KU developed the study methodology, conducted deposited dust sampling, performed data analysis and predictive modelling, drafted the manuscript, and obtained ethical approvals. BN conceived and designed the study and provided overall scientific direction. BN, KLJ, and KAI supervised the research as PhD supervisors, contributing technical expertise, critical review, and continuous mentorship throughout the study. All authors read and approved the final manuscript.

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# Data Availability

The datasets generated and/or analysed during the current study are attached as supplementary documents.

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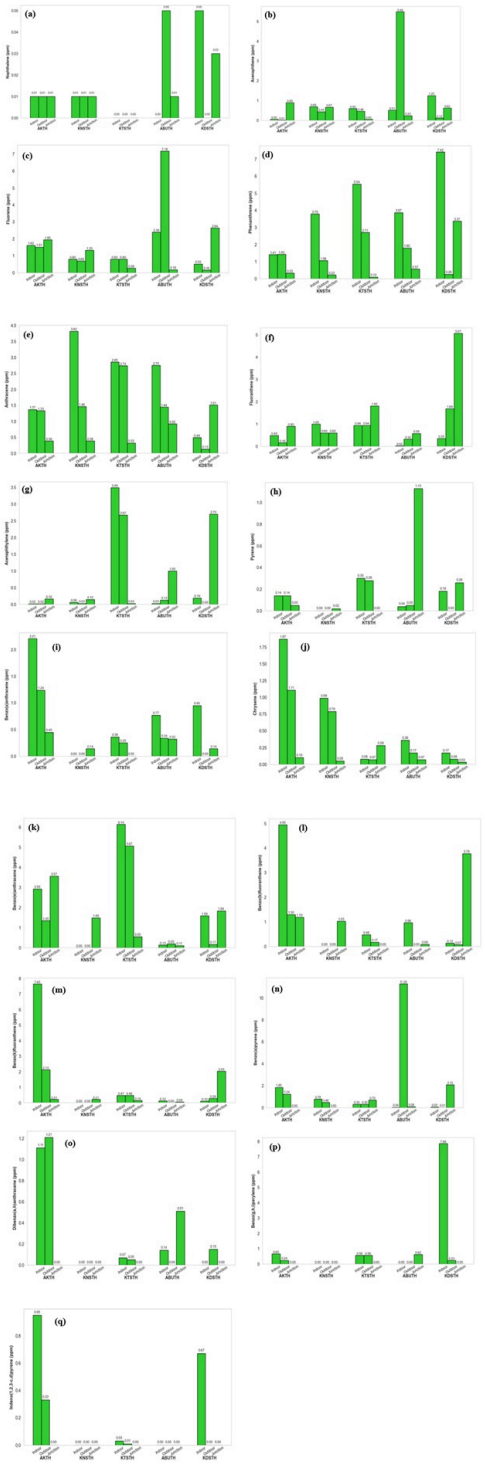
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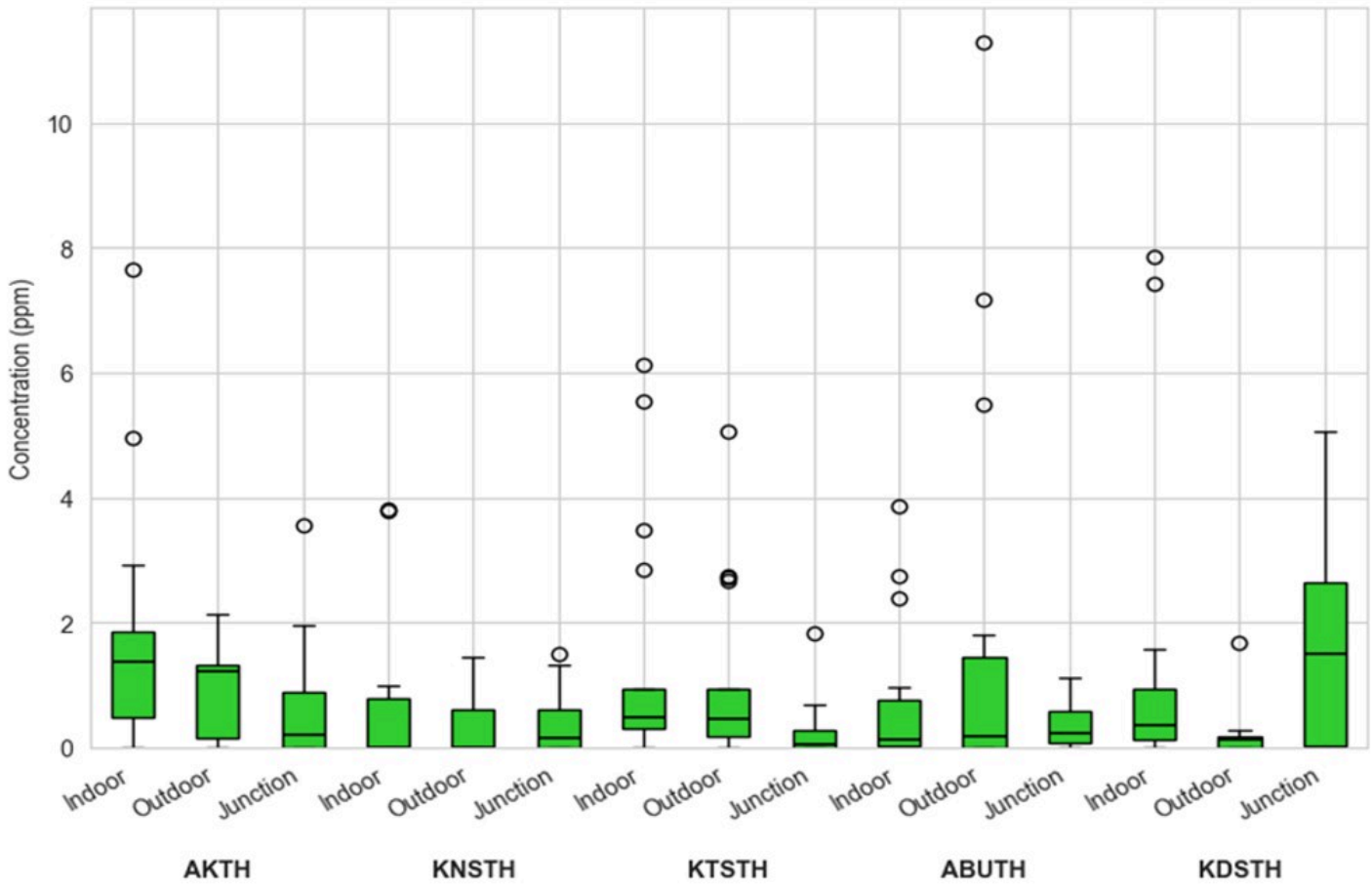
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# Figures



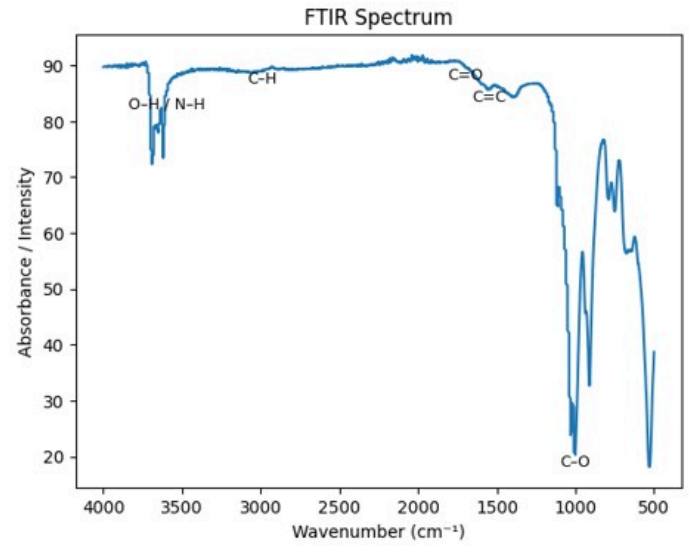
## Figure 1

## Concentrations of the different PAHs across the studied hospitals

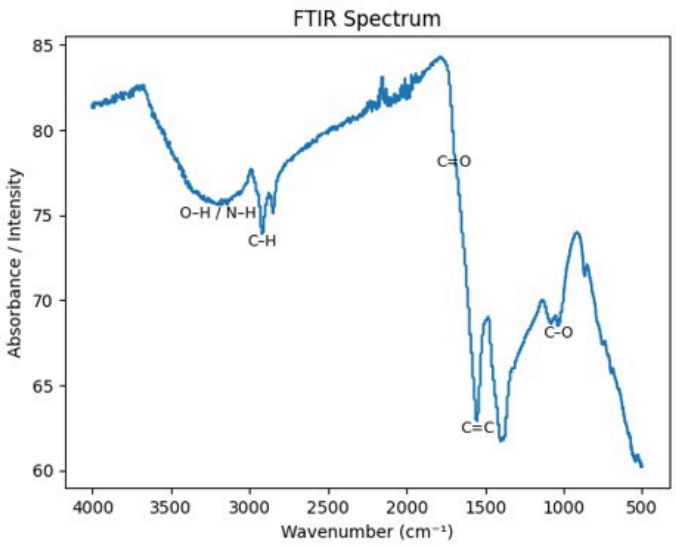



## Figure 2

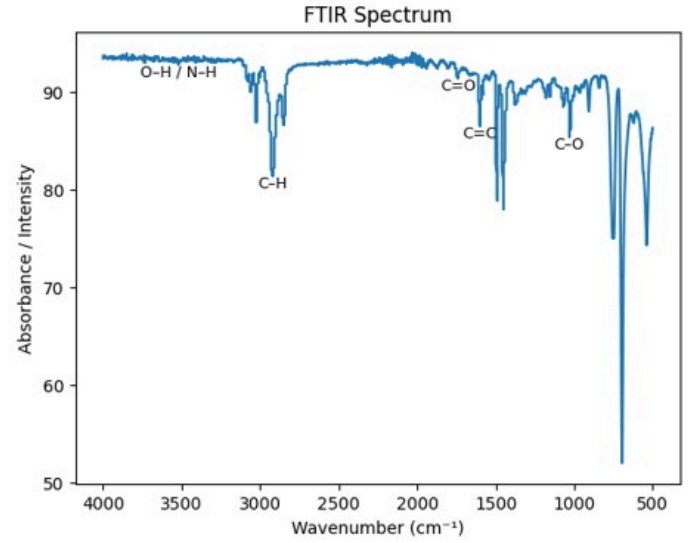
## Distribution of PAHs across the studied hospitals




(a) Indoor FTIR Spectrum of Dust Samples from ABUTH



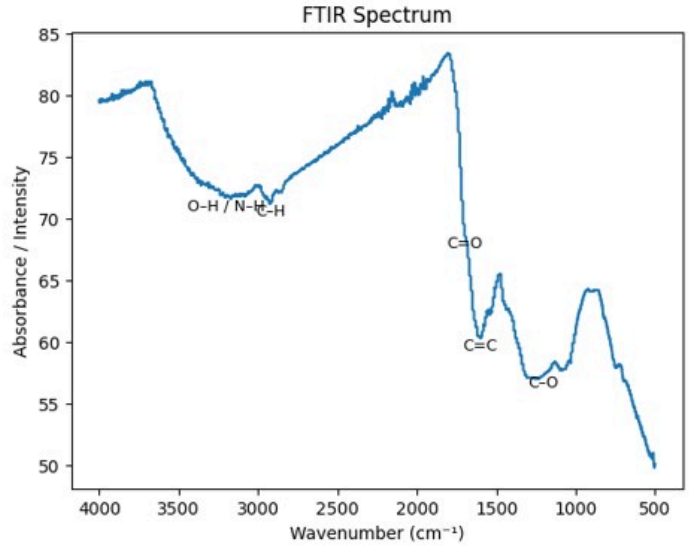

(b) Outdoor FTIR Spectrum of Dust Samples from ABUTH



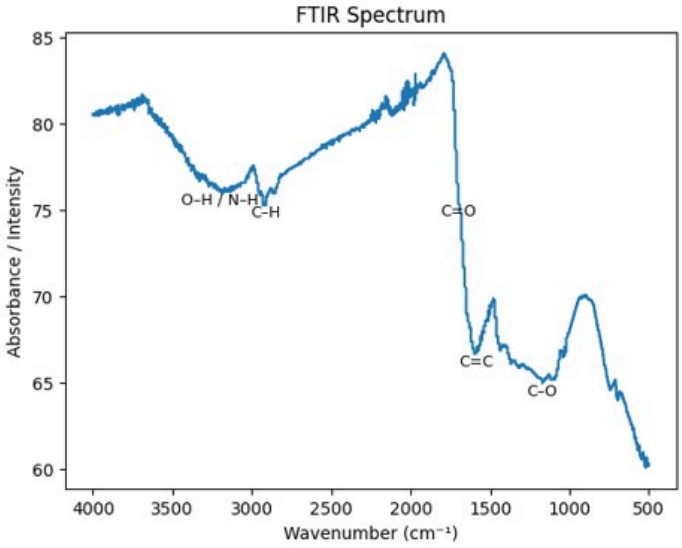

(c) Junction FTIR Spectrum of Dust Samples from ABUTH

## Figure 3

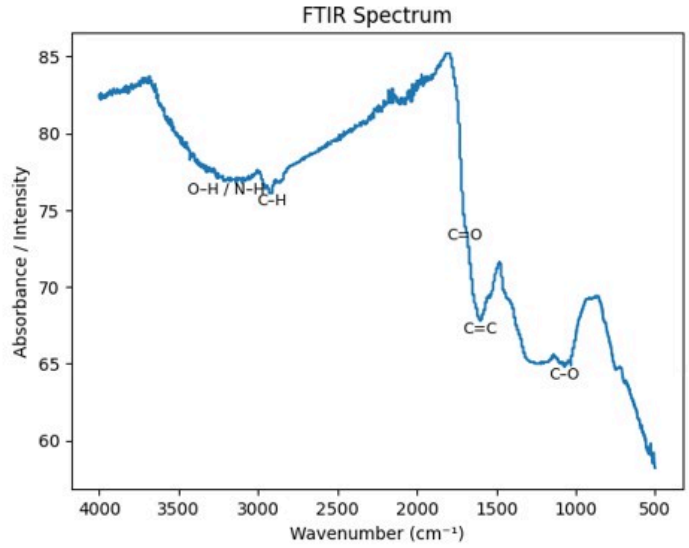
## FTIR spectra of deposited dust samples from ABUTH




**(a) Indoor FTIR Spectrum of Dust Samples from AKTH**



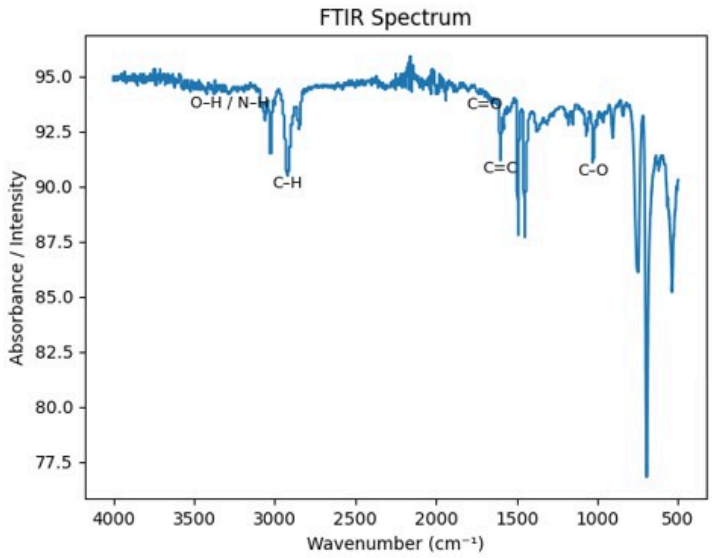

**(b) Outdoor FTIR Spectrum of Dust Samples from AKTH**



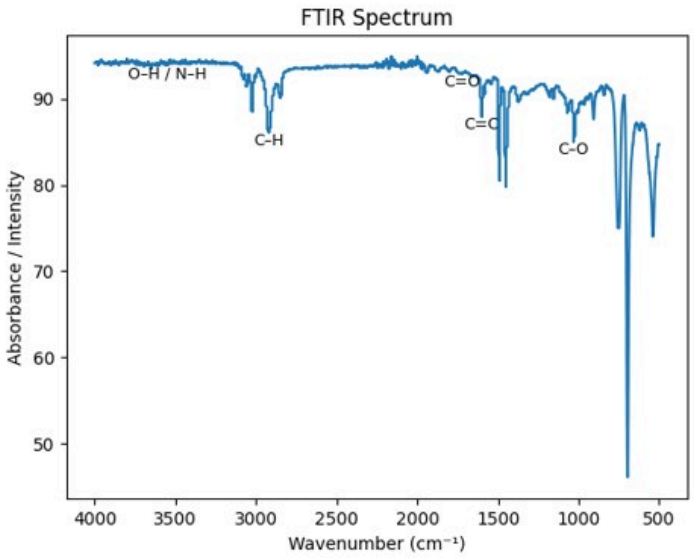

**(c) Junction FTIR Spectrum of Dust Samples from AKTH**

## Figure 4

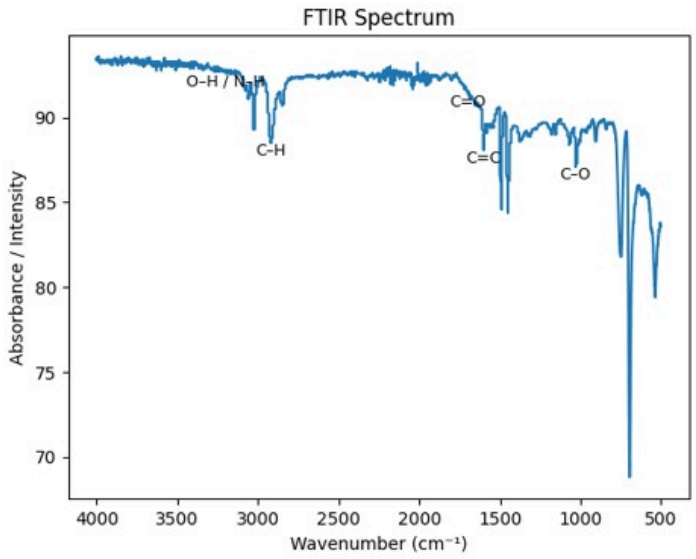
## FTIR spectra of deposited dust samples from AKTH




**(a) Indoor FTIR Spectrum of Dust Samples from KDSTH**



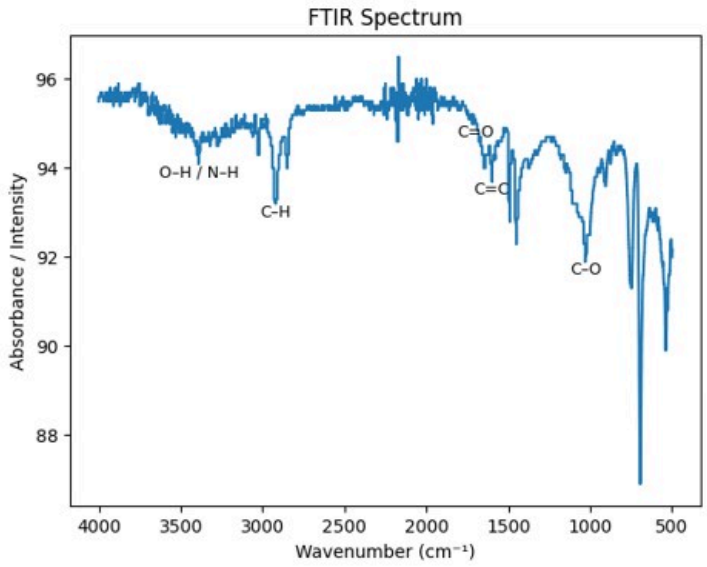

**(b) Outdoor FTIR Spectrum of Dust Samples from KDSTH**



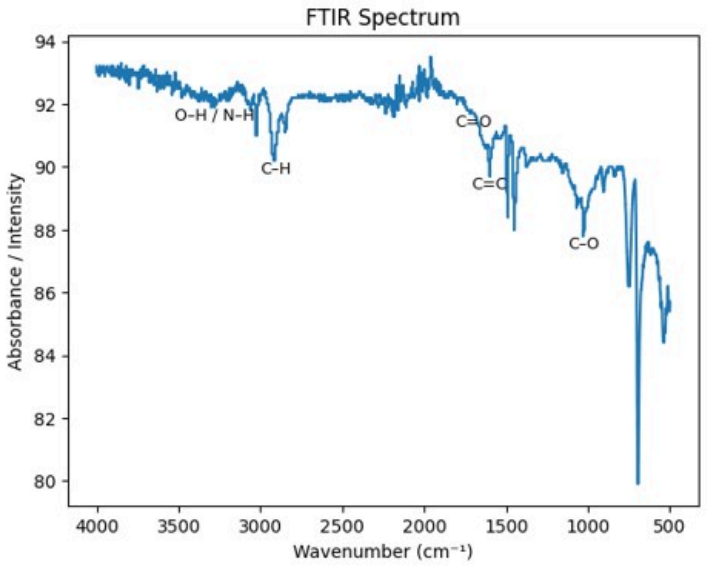

**(c) Junction FTIR Spectrum of Dust Samples from KDSTH**

## Figure 5

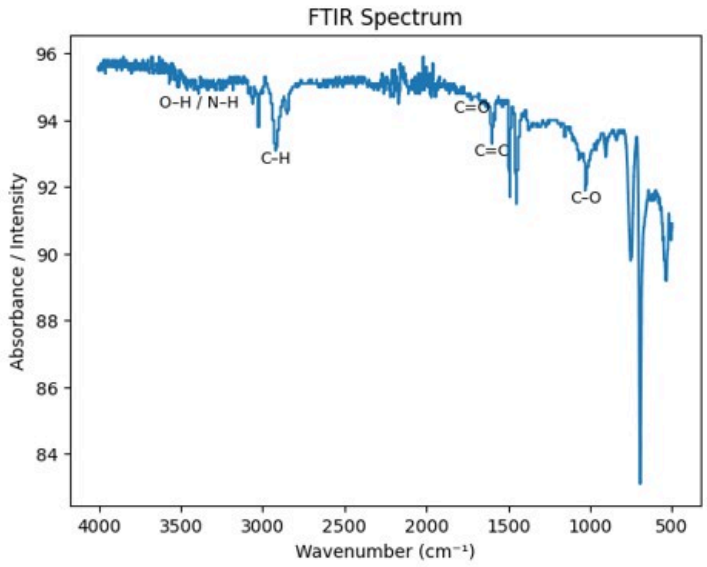
## FTIR spectra of deposited dust samples from KDSTH




(a) Indoor FTIR Spectrum of Dust Samples from KSTH



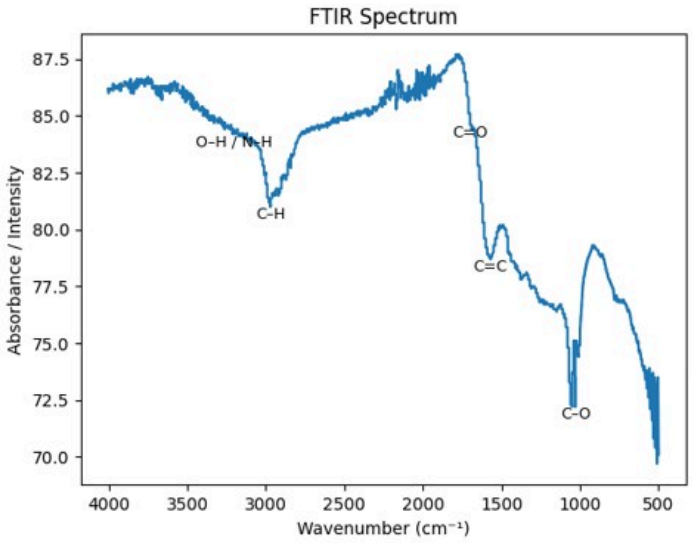

(b) Outdoor FTIR Spectrum of Dust Samples from KSTH



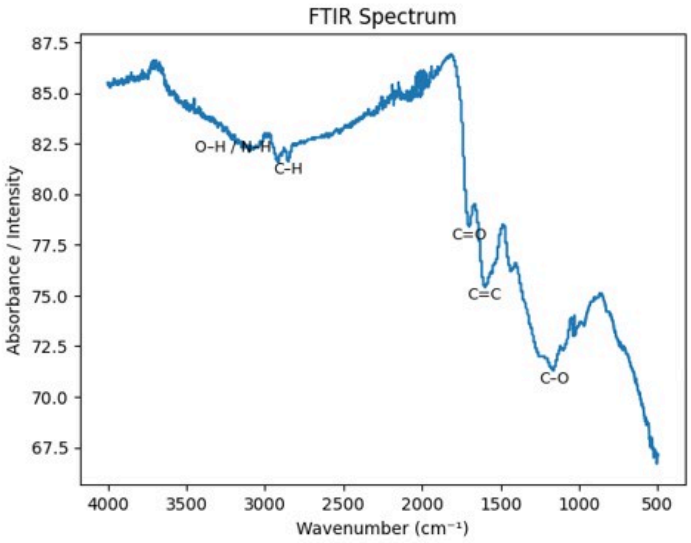

(c) Junction FTIR Spectrum of Dust Samples from KSTH

## Figure 6

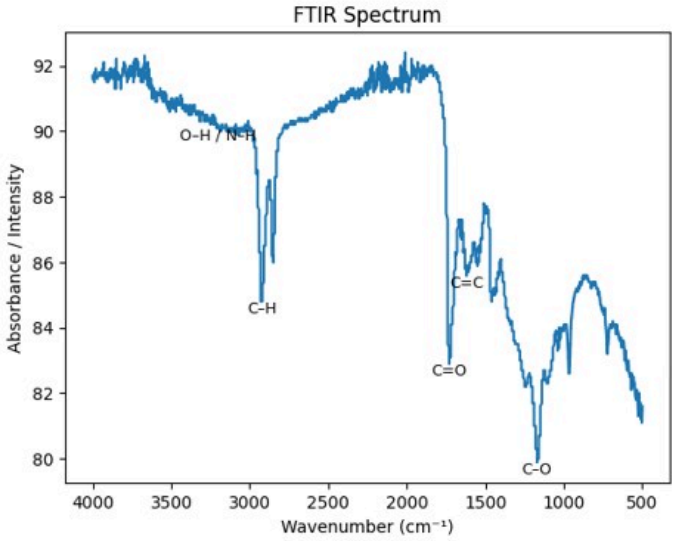
## FTIR spectra of deposited dust samples from KSTH




(a) Indoor FTIR Spectrum of Dust Samples from KTSTH



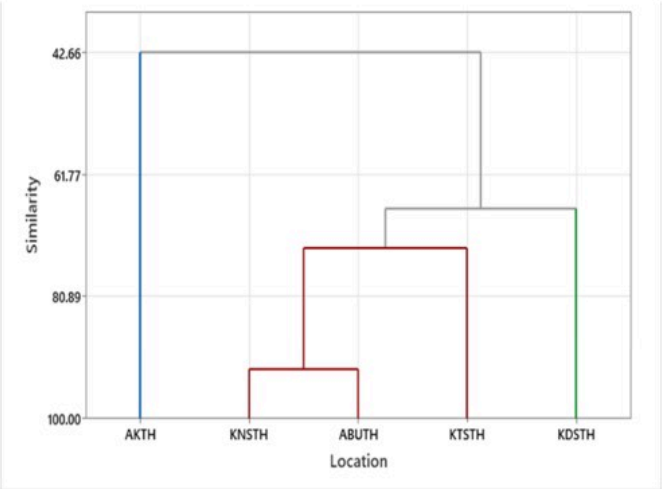

(b) Outdoor FTIR Spectrum of Dust Samples from KTSTH



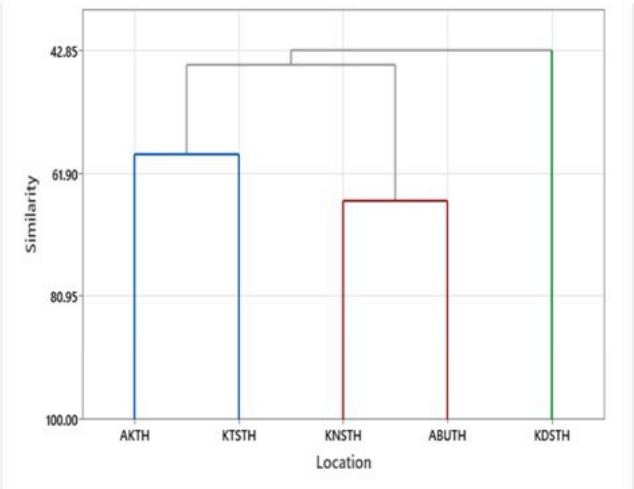

(c) Junction FTIR Spectrum of Dust Samples from KTSTH

## Figure 7

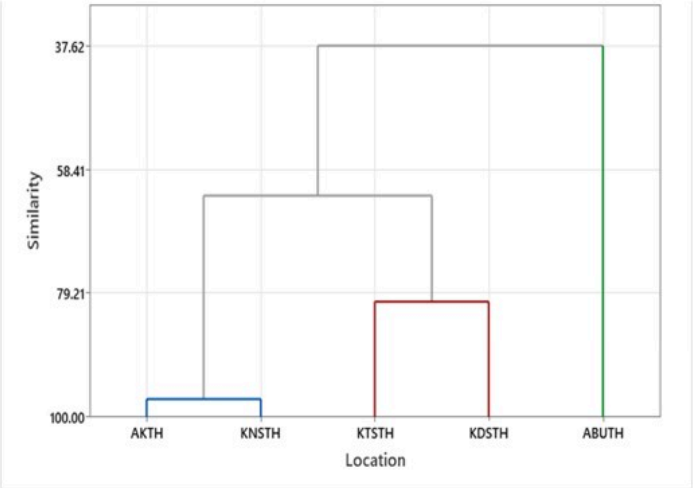
## FTIR spectra of deposited dust samples from KTSTH




**(a) Indoor dendogram for the spatial distribution of PAHs**



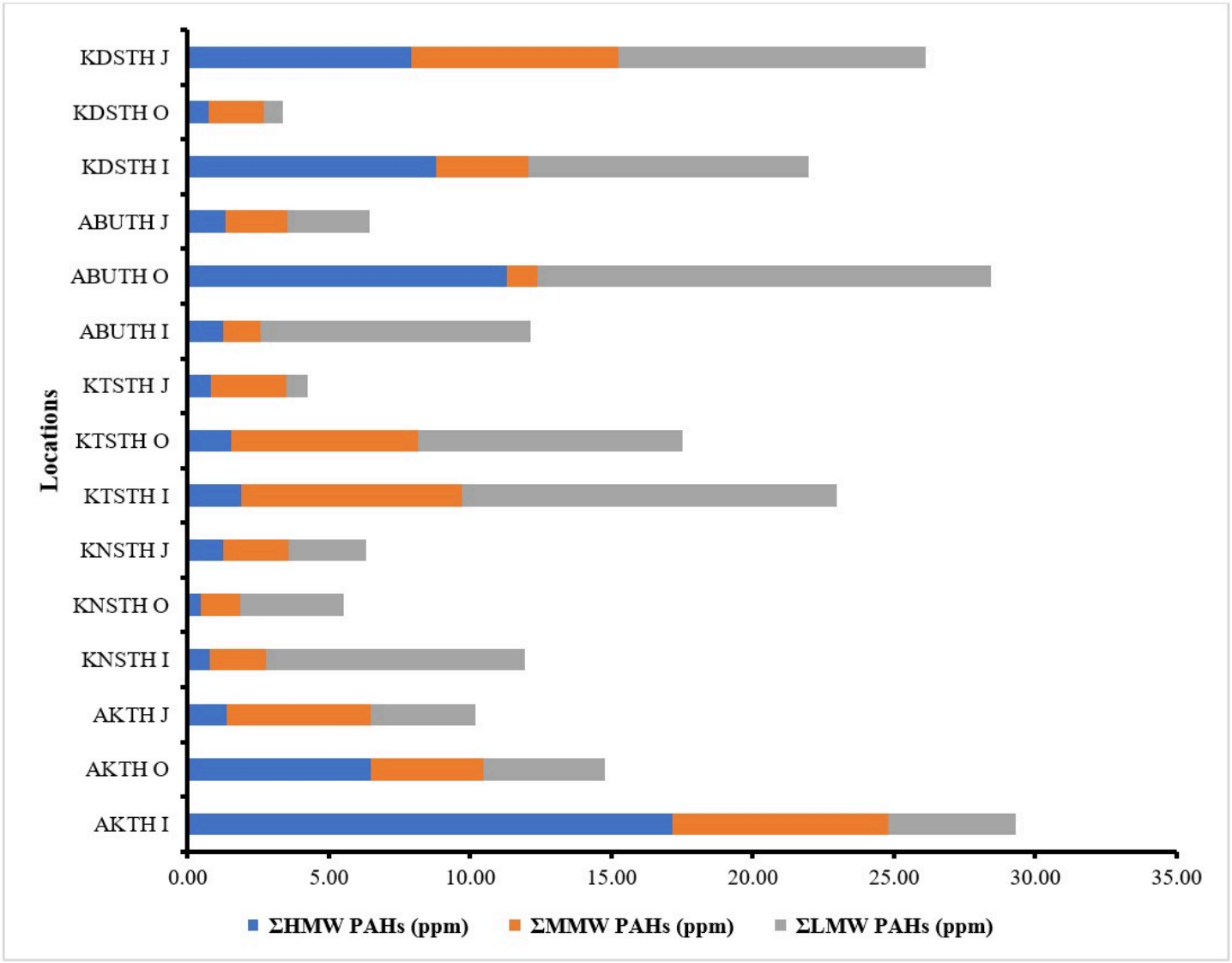

**(b) Outdoor dendogram for the spatial distribution of PAHs**




**(c) Junction dendogram for the spatial distribution of PAHs**

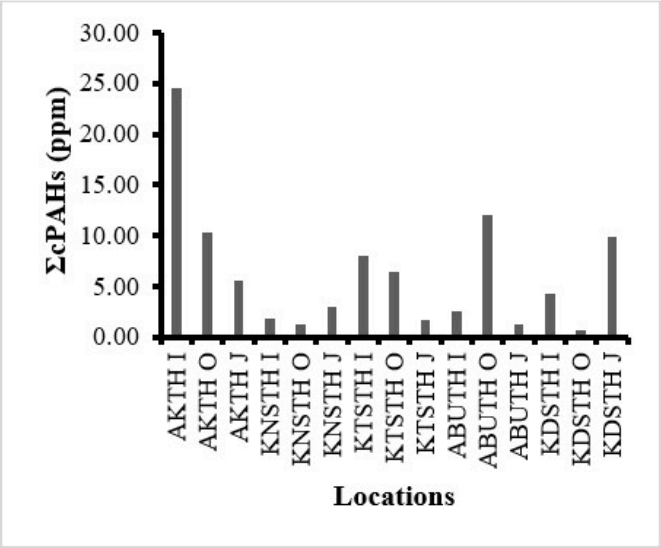
## Figure 8

## Cluster analysis showing the distribution of PAHs in the locations of the studied hospitals

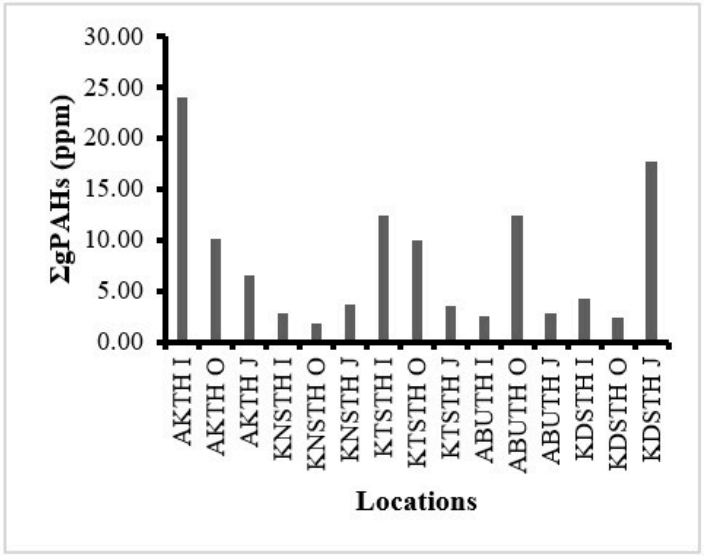



## Figure 9

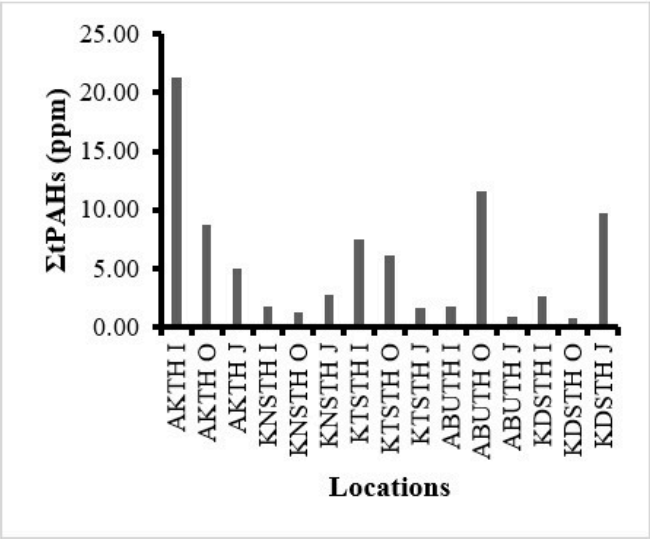
## PAH Concentrations by Molecular Weight Class




(a) Location Levels of Carcinogenic PAHs (ΣcPAHs)



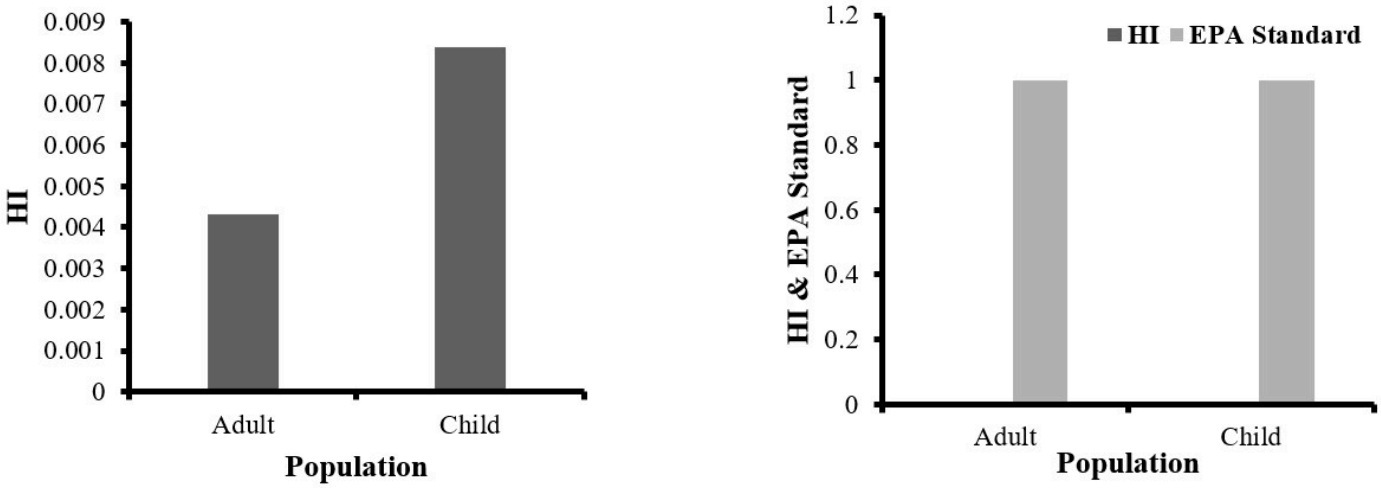

(b) Location levels of genotoxic PAHs ΣgPAHs




(c) Location levels of teratogenic PAHs ΣgPAHs

## Figure 10

## Toxicological classification of detected PAHs

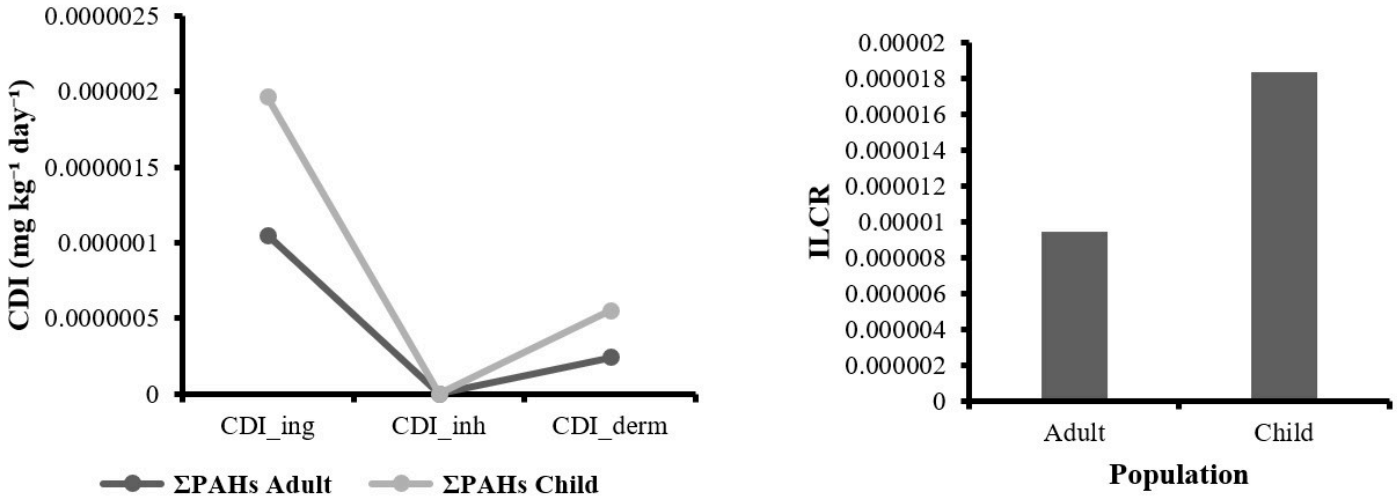



**(a) Adult and Child Hazard Index (HI) Comparison** **(b) Population Hazard Index Comparison**

**HI is unitless, with values below 1 indicating acceptable non-carcinogenic risk according to U.S. EPA guidelines.*

Figure 11

Population-based non-carcinogenic health risk (Hazard Index) due to PAH




**(a) Chronic Daily Intake (CDI) Comparison** **(b) Chronic Daily Intake (CDI) Comparison**

**ILCR values are unitless and interpreted relative to the US EPA acceptable risk range ($10^{-6}$–$10^{-4}$)*

Figure 12

Population-Based Chronic Daily Intake and Incremental Lifetime Cancer Risk