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Environmental Distribution and Occupational Exposure for Aromatic Polycyclic Hydrocarbons within Industrial Regions of Central India: Implications for Human Health Risk

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Abstract

The accelerated pace of industrialization in Central India has resulted in heightened emissions of polycyclic aromatic hydrocarbons, thereby eliciting substantial environmental and public health apprehensions. This investigation assessed the prevalence, spatial variability, and interlinkages between environmental and biological PAH levels in soil, plant, and blood sample collected from prominent industrial locales across Central India with a focus on evaluating potential exposure among industrial workers. In aggregate, 250 samples were subjected to analysis through liquid chromatography and conventional gas chromatography accompanied by tandem mass spectrometry techniques. Soil samples exhibited the highest concentrations of PAHs (0.11–1.58 ppm; mean 0.63 ppm), followed by plant samples (0.05–2.59 ppm; mean 0.77 ppm), whereas blood samples exhibited detectable concentrations (51.81–29712.66 ppb; mean 1059.74 ppb), indicating potential human exposure. Spatial analysis identified the Siltara Area and Korba Industrial Area as the primary contamination hotspots. Correlation analysis revealed a weak negative relationship between soil and plant samples ($r = -0.28$) and a stronger negative association between plant and blood samples ($r = -0.73$), suggesting complex exposure pathways. Risk evaluation utilizing toxic equivalency concentrations, incremental

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lifetime cancer risk, and hazard index demonstrated that non-carcinogenic risks were confined to acceptable thresholds; however, marginally elevated carcinogenic risks were identified in the Siltara region. The detection of low-molecular-weight PAHs in blood samples may suggest prospective occupational exposure among industrial workers, aligning with occupational safety concerns. These observations emphasize the necessity for sustained environmental monitoring and occupational health surveillance across industrial areas in Central India.

Introduction

Aromatic polycyclic compounds constitute persistent the formation of organic contaminants primarily during Unfinished combustion of matter that is organic.[1] Major anthropogenic sources include coal-fired thermal power plants, sponge iron industries, vehicular emissions, petroleum refining units and various metallurgical processes.[1–3] Due to their hydrophobic and lipophilic nature, PAHs strongly adsorb onto soil particles and plant surfaces, facilitating accumulation in terrestrial ecosystems and entry into the human body via inhalation, ingestion and cutaneous exposure pathways.[4]

Several Polycyclic aromatic hydrocarbons exhibit mutagenic, teratogenic and carcinogenic properties, raising serious environmental and public health concerns.[5–9] Industrial regions of Central India, particularly Chhattisgarh, are characterized by intensive coal-based thermal power generation and heavy industrial operations, contributing substantially to atmospheric PAH emissions.[10–12] However, comprehensive information regarding simultaneous assessment of PAHs in environmental and biological matrices from this region remains limited.

Therefore, the present study intended to quantify concentrations of PAHs present in soil, plant, blood samples obtained from major industrial areas of Central India, examine their spatial distribution, and explore potential relationships between environmental contamination and biological exposure and occupational exposure among industrial workers.

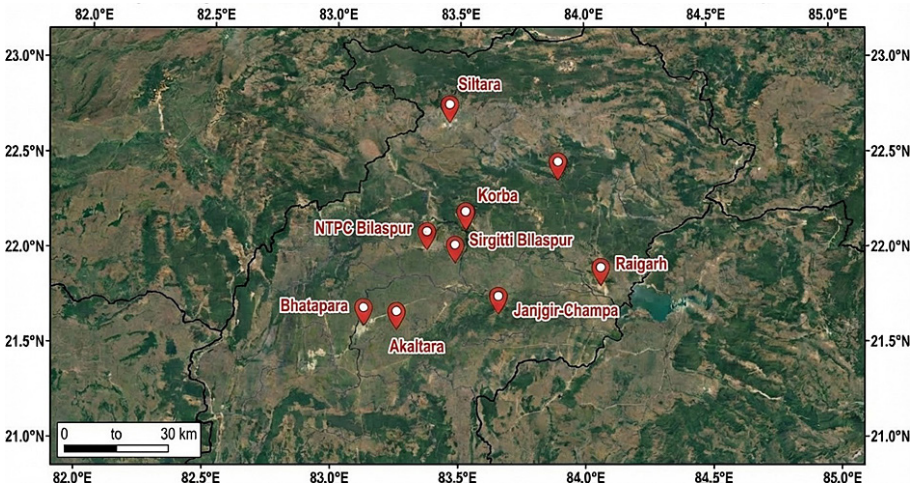

Fig. 1: Map showing the location of selected industrial sampling sites in Central India.

Materials and Methods

Research Region and Data Acquisition

Specimen were acquired from several major industrial zones in Chhattisgarh, including Siltara, Korba, NTPC Bilaspur, Janjgir–Champa, Akaltara, Raigarh, and Bhatapara. These areas are largely dominated by industrial activities such as coal-based thermal power generation, sponge iron production, and steel

manufacturing units, which are known to contribute significantly to regional emissions of pollutants.[13]

Surface soil samples were collected using pre-cleaned stainless-steel augers to avoid contamination during sampling. Plant samples were collected from vegetation naturally growing near the selected industrial locations. Blood samples were obtained from volunteers who either resided in these areas or were occupationally exposed to industrial environments. All blood sample collection procedures were carried out after securing informed consent from participants and approval from Institutional Ethics Committee. In total, 250 samples comprising soil, plant, and blood matrices were collected for analysis For analysis, 250 samples totalling soil, plant, and blood matrices were gathered. (plant: n = 50, blood: n = 80, soil: n = 120).Blood samples separated into two groups for biological evaluation according to occupational and residential characteristics: non-industrial (n = 40) which represented background exposure conditions, and industrial (n = 40) which represented comparatively higher potential exposure. Blood samples were taken from adult volunteers (mean age: 39.29 years; age range: 17–80 years), comprising 52 females (65%) and 28 men (35%) who represented both vocational and residential populations. It is acknowledged as a limitation that the current study did not include information on potential confounding factors, such as dietary intake, smoking behaviours, and other lifestyle characteristics.

Chemicals and Standards

A PAH calibration mixture containing The USEPA has identified sixteen significance PAHs:, acenaphthylene, acenaphthene, fluorene, pyrene, phenanthrene, anthracene, fluoranthene naphthalene, benzo[a]anthracene, chrysene, benzo[b]fluoranthene, benzo[k]fluoranthene, and benzo[a]pyrene, (TraceCERT®, Supelco; CRM47940, 1 mL, 10 µg/mL each component in acetonitrile) was used for calibration and quantification during analysis. Deuterated compounds including naphthalene-d_8, acenaphthene-d_{10}, phenanthrene-d_{10}, fluoranthene-d_{10}, pyrene-d_{10}, chrysene-d_{12}, benzo[a]pyrene-d_{12}, perylene-d_{12} served as internal standards to monitor extraction efficiency and analytical accuracy.

Analytical- and HPLC-grade solvents, namely hexane, dichloromethane, acetone, and acetonitrile, were employed for extraction and chromatographic analysis. HPLC-grade reagents, including sodium chloride, primary secondary amine, activated silica gel, neutral alumina, anhydrous sodium sulphate, and ultrapure water, were utilized for phase separation, moisture removal, and cleanup of sample extracts prior to instrumental analysis. High-purity acetone and acetonitrile, suitable for high-performance liquid chromatography, were sourced from Chem Super Supply.

Extraction-Purification

Soils and plant exemplars underwent Soxhlet extraction using an n-hexane: dichloromethane solvent mixture for 16 h. The extracts were subsequently concentrated and purified by silica gel column chromatography.[14-16] Blood samples were processed via liquid–liquid extraction, centrifugation, and solvent concentration.[5,9,17]

Analysis of Tandem Mass Spectrometry and Gas Chromatography

PAH quantification is conducted employing a Shimadzu GCMS-TQ2030 triple quadrupole gas chromatograph–tandem mass spectrometer employing electron ionization (70 eV). Chromatographic. The process of segregation occurred on Rxi-5Sil MS open-tubular section, section (coating width: 30 m. × 0.25 mm. × 0.25 µm.). 99.999 percent pure helium measured employed as a transport gas (1.0 mL per minute). Temperature program for oven began according to 60 degree Celsius, increased up to 280 degree Celsius at a pace of 10 degree Celsius per minute and maintained isothermally for 10 minutes.[17–19] Analyte identification relied on retention time matching and comparison against the NIST spectral library. Quantitation was performed using external calibration curves, with quality control ensured through analysis of blanks, duplicates, and calibration verification standards.

High-Performance Liquid Chromatography Analysis

Serum samples underwent quantitative analysis via an ultra- HPLC system integrated coupled with a photodiode array detector. The system comprised solvent delivery pumps, an autosampler,

a column oven, the detector, and a data acquisition unit. Separation using chromatography was accomplished using a Hypersil Gold C18 column employing an isocratic acetonitrile: water mobile phase, thereby enabling sensitive analyte detection at 254 nm.[20,21]

Statistical Analyses

Statistical analyses were conducted to examine relationships between PAH concentrations and clinical biomarkers. Summary statistics, comprising means, standard deviations, and ranges, were computed for all variables. Pearson's correlation coefficients were employed to assess associations with soil and plant PAHs, as well as between blood PAH levels and renal, hepatic, and haematological parameters. Linear regression analysis was utilised to assess associations among variables; however, the findings were interpreted with caution, and no causal relationships were deduced. The value of coefficient of determination(R^2),was utilized to describe The percentage of variance that the model can account for, rather than implying causation.[9,22,23] The analysis of principal components was used in order to identify source profiles along with clustering patterns across testing sites and biomarkers. Health risk indicators—such as toxic equivalent concentrations, incremental lifetime cancer risks, and hazard indices—were computed according to USEPA guidelines.[6,9,24] All analyses were conducted using R statistical software within RStudio.

Analytical Method Validation and Quality Control

The analytical technique was comprehensively validated to verify its reliability, accuracy, and precision for PAH quantification.[6,25] Multi-level calibration curves were generated using external standards over a suitable concentration range, exhibiting strong linearity with correlation coefficients ($r > 0.995$) across all target PAHs. LOD as well as LOQ both were established determined using signal noise ratios of 10:1 and 3:1, yielding LOD values between 0.0005–0.002 ppm and LOQ values of 0.002–0.005 ppm, compound-dependent. Recovery experiments involved matrix spiking of blank soil, plant, and blood samples with known PAH levels, producing mean recoveries of 82–108% that verified extraction efficacy.[8,9,26,27] Precision was assessed via replicate analyses, with relative standard deviations <10%, attesting to excellent reproducibility. Quality control incorporated procedural blanks, duplicates, and routine calibration verification to mitigate contamination and instrumental drift.

Health Hazard Assessment of PAHs

The human health risks arising from exposure to soil-bound PAHs were assessed using toxic equivalent concentrations,[28–30] incremental lifetime cancer risks,[31–33] and hazard index methods.10 These established approaches are routinely employed to evaluate carcinogenic and non-carcinogenic risks linked to PAH contamination in environmental media.

Toxic Equivalent Concentration

The carcinogenic potential of individual PAHs was quantified as Equivalent amounts of benzo[a]pyrene through this application out of toxic equivalency factors, Risk assessment calculations were performed under standard exposure assumptions, and the results were interpreted cautiously without implying direct causality. in accordance with recommendations from the *World Health Organization and US Environmental Protection Agency.*

$$TEQ=\sum(C_{-j}\times TEF_{-j})$$

where Ci denotes the measured number of individual PAHs as well as TEFi represents the toxic equivalence factor that corresponds to it.

Lifetime Incremental Cancer Risk (ILCR)

In compliance with USEPA risk assessment procedures, the ILCR was calculated to quantify the lifetime likelihood of acquiring cancer from chronic PAH exposure via contaminated soil29,34. In accordance with USEPA risk assessment guidelines, where CDI stands for chronic daily intake and together with CSF for the slope factor for cancer for carcinogenic PAHs, ILCR, or incremental lifetime cancer risk, is calculated in order to assess potential lifetime probability of cancer development associated with chronic exposure to PAHs through contaminated environmental media. Due to the

lack of site-specific individual exposure data, the parameters utilized for CDI estimation— body weight, average time, frequency and length of exposure, and ingestion rate—were taken from standard USEPA exposure factors. CSF values were derived from pertinent literature and well-established USEPA databases.[29,34]

Incremental Lifetime Cancer Risk (ILCR)=CDI×CSF

The ILCR provides a prediction of the risk over a lifetime that a person will get cancer due to exposure. Interpretation of ILCR values followed standard regulatory criteria:

- ILCR < 10^{-6}→ negligible risk
- 10^{-6}– 10^{-4}→ acceptable risk range
- ILCR > 10^{-4}→ elevated cancer risk

Non-Carcinogenic-Risk-Assessment

Non-carcinogenic risks were assessed using hazard quotient and hazard index metrics.

$$\text{Hazard Quotient } (HQ) = \frac{C_{measured}}{C_{reference}}$$

where $C_{measured}$ represents PAH concentration obtained from soil or biomonitoring analysis and $C_{referencede}$ notes the corresponding permissible or screening value.

Regulatory Benchmark Comparison

Derived TEQ, ILCR, and HI values were compared with regulatory thresholds and acceptable risk levels established by agencies such as USEPA, WHO, OSHA, and IARC to evaluate potential health risks associated with PAH exposure, without implying direct causality.

Results and Discussion

Spatial Patterns of PAHs in Soil

Soil samples, as detailed in Table 1, displayed markedly higher PAH concentrations compared to those in plant and blood matrices,[35] thereby establishing soil as the principal environmental reservoir.[36] As illustrated in Figure 2, elevated levels of high molecular weight PAHs were particularly evident in Siltara and Korba, attributable to intensive industrial activities associated with combustion processes.[24] Furthermore, the predominance of low molecular weight PAHs in these areas suggests possible recent inputs from incomplete combustion sources, however source attribution cannot be conclusively established based solely on concentration data in line with previous studies on industrial emissions.[37]

Table 1: Mean levels of Aromatic Polycyclic hydrocarbons in soil samples among selected industrial regions of Central India (in ppm; µg kg-¹).

Site	Nap.	Acy.	Flu.	Phe.	Ant.	Flt.	Pyr.	BbF.	BkF.	BjF.	BeP.	Btri.	BbFlu.
Sil	75.91	34.11	16.14	58.56	46.93	29.57	79.38	18.51	4.29	17.35	18.14	23.89	-
Kor	395.52	10.43	46.59	35.12	1016.97	45.88	27.15	-	-	-	13.01	-	40.52
NTPC	26.25	16.26	-	39.23	-	-	39.17	-	-	-	-	-	-
Sir	352.26	-	-	17.53	-	-	26.77	-	-	-	-	-	-
Aka	-	-	-	-	2056.92	-	-	-	-	-	-	-	-
J-C	390.02	-	-	-	-	-	1.84	-	-	-	-	-	-
Bha	66.67	-	-	2.71	-	35.92	7.35	-	-	-	-	-	-
Rai	-	-	-	-	-	-	1.38	-	-	-	-	-	-

Abbreviation

Nap: Naphthalene; Acy: Acenaphthylene; Flu: Fluorene; BkF: Benzo[k]fluoranthene; Phe: Phenanthrene; Ant: Anthracene; Flt: Fluoranthene; Pyr: Pyrene; BbF: Benzo[b]fluoranthene; BjF: Benzo[j]fluoranthene; BeP: Benzo[e]pyrene; Btri: Benzo[a] triphenylene; BbFlu: 11H-Benzofluorene; (-): Not detected SIL – Siltara; KOR – Korba; NTPC – NTPC Bilaspur; SIR – Sirgitti; AKA – Akaltara; JC – Janjgir–Champa; BHA – Bhatapara; RAI – Raigarh

PAHs in Plant Samples

PAH concentrations in plant samples, as presented in Table 2, were substantially lower than those recorded in the corresponding soil samples, in accordance with contaminant transfer processes mediated by root uptake and atmospheric deposition.[38] High-molecular-weight PAHs showed restricted accumulation within plant tissues owing to limited translocation, as evidenced in prior research.[38] In contrast, low-molecular-weight PAHs—most notably

naphthalene—prevailed, which may attributable to their greater mobility and uptake proficiency in plant matrices.[39] The spatial distribution patterns across industrial sites, as illustrated in Figure 3, Elevated anthracene concentrations were observed in soil samples from Akaltara Jangir–Champa, according to a comparison of soil and plant data. Plant samples also demonstrated a noticeable rise exclusively in Akaltara–Jangir area. This implies that transmission and accumulation might differ depending on the compound. Siltara, on the other hand, regularly displayed the presence of several PAH compounds in both soil and plant matrices, suggesting a more comprehensive contamination profile in contrast to the predominance of a single molecule. Underscoring a possible association and linkage between soil contamination and PAH bioaccumulation in vegetation at targeted locations.

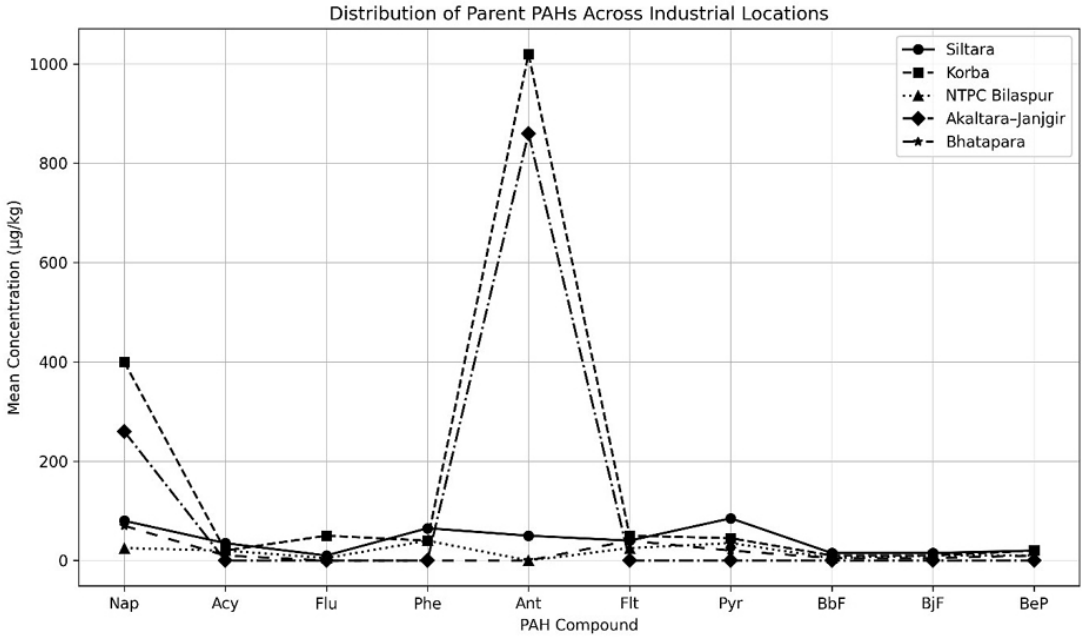

Fig. 2: Spatial Patterns of total PAHs in soil samples from selected industrial sites of Central India (expressed in µg kg-¹), showing variability across locations.

Table 2: Mean levels of Aromatic polycyclic hydrocarbons in plant samples among selected industrial regions of Central India (in ppm; µg kg-¹).

Site	Nap.	Acy.	Flu.	Phe.	Ant.	Flt.	Pyr.	BbF.	BkF.	BjF.	BeP.	Btri.	BbFlu.
Sil	12.52	96.72	44.94	-	258.08	-	50.23	-	-	33.33	24.94	-	-
Kor	255.79	160.83	8.10	38.77	-	27.59	-	-	-	-	-	-	-
NTPC	65.01	-	-	114.79	-	-	24.50	-	-	-	-	-	-
Aka–Jc	16.52	-	-	-	2586.62	-	32.67	-	-	-	-	-	-
Bha	-	-	-	9.95	-	-	-	-	-	-	-	-	-

Abbreviation
Nap: Naphthalene; Acy: Acenaphthylene; Flu: Fluorene; Ant: Anthracene; Flt: Fluoranthene; BbF: Benzo[b]fluoranthene; Pyr: Pyrene; BkF: Benzo[k]fluoranthene; BjF: Benzo[j]fluoranthene; Phe: Phenanthrene; BeP: Benzo[e]pyrene; Btri: Benzo[ghi]triphenylene; BbFlu: 11H-Benzo[fluorene]; (-): Not detected SIL – Siltara; KOR – Korba; NTPC – NTPC Bilaspur;AKA–JC – Akaltara–Janjgir; BHA – Bhatapara.

Soil–Plant Correlation

Pearson correlation analysis revealed strong positive relationships between soil and plant PAH concentrations for naphthalene (r=0.93) and pyrene (r=0.91), reflecting association from soil to vegetation. By contrast, anthracene showed

a weak negative correlation (r=-0.28), Moderate positive correlations were too detected in the case of fluoranthene (r =0.73) and phenanthrene (r = 0.39), indicating variable relationships across different PAH compounds.

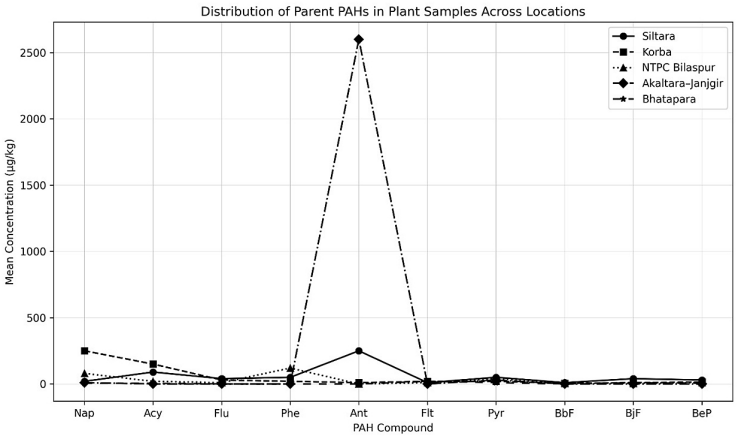

Fig. 3: Spatial Patterns of total PAHs in plants samples from selected industrial sites of Central India (expressed in µg kg^{-1}), showing variable concentrations across sites.

Table 3: Pearson correlation coefficients between polycyclic aromatic hydrocarbon concentrations in soil and plant samples across the investigated industrial sites.

Compound	Correlation Coefficient (r)
Naphthalene	0.93
Phenanthrene	0.39
Anthracene	−0.2
Fluoranthene	0.73
Pyrene	0.91

These findings indicate that the distribution of PAHs between soil and plant matrices may vary depending on compound-specific physicochemical properties; however, direct transfer mechanisms cannot be conclusively established based solely on correlation analysis. These observations are consistent with patterns reported in previous studies[40–43]

Blood PAHs and Clinical Parameters

Detectable levels of PAHs were identified in blood samples from industrial workers in the exposed group, indicating Potential occupational exposure.[44] Quantifiable concentrations of low-molecular-weight PAHs, including naphthalene (1059.74 ± 4624.31), acenaphthylene (95.33 ± 354.6),, and phenanthrene (70.72 ± 11.2), were exclusively observed among exposed workers, while PAH levels in the non-exposed group remained below the limit of detection; no high-molecular-weight PAHs were detected, consistent with findings reported by elsewhere.[45–47] Comparative analysis of biomarkers between non-exposed and exposed workers, as presented in Table 4, revealed primarily weak associations between PAH levels and clinical parameters, with the notable exception of haemoglobin as indicated by the statistical analysis,[48,49] However, these associations do not establish causality and should be interpreted with caution. Nevertheless, the detection of PAHs in blood confirms persistent low-level exposure although background exposure cannot be ruled out.

Table 4: Comparison of biochemical parameters between exposed and non-exposed groups

Parameter	Exposed (Mean ± SD)	Non-exposed (Mean ± SD)	t-value	p-value
Haemoglobin (g/dL)	11.92 ± 2.42	8.67 ± 3.91	3.21	0.002
Lymphocyte (%)	28.74 ± 8.11	27.84 ± 8.94	0.54	0.590
Eosinophils (%)	3.79 ± 3.44	3.96 ± 3.58	−0.27	0.785
Serum Creatinine (mg/dL)	1.33 ± 1.21	1.25 ± 1.08	0.34	0.736
Blood Urea Nitrogen (mg/dL)	11.35 ± 6.81	12.48 ± 8.67	−0.71	0.481
Alkaline Phosphatase (U/L)	113.22 ± 84.71	87.66 ± 60.31	1.62	0.109
AST (U/L)	35.42 ± 51.83	42.73 ± 63.94	−0.56	0.578
ALT (U/L)	28.16 ± 33.24	33.45 ± 41.87	−0.63	0.530
Bilirubin (mg/dL)	0.73 ± 0.52	0.68 ± 0.48	0.48	0.634

Correlation between Polycyclic Aromatic Hydrocarbons and Biochemical Health Indicators

Figure 4 presents the results of the correlation analysis, which elucidates diverse associations between polycyclic aromatic hydrocarbon compounds and biochemical markers pertaining to hepatic, renal, and haematological functions. Pearson correlation coefficients (r) were used to assess linear relationships, and the strength of associations was interpreted based on standard correlation ranges.

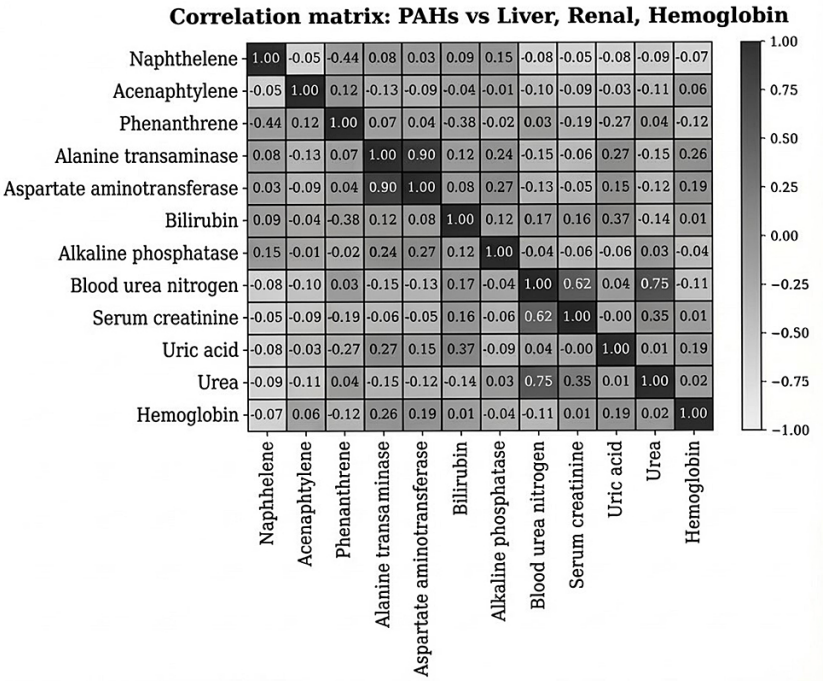

Fig. 4: Pearson Correlation Heatmap Showing Relationships Between Blood Pahs and Renal, Hepatic And Haematological Biomarkers.

Naphthalene exhibited weak correlations with the majority of biochemical parameters. Modest positive correlations were evident with alanine transaminase (ALT) (r = 0.08), bilirubin (r = 0.09), and alkaline phosphatase (ALP) (r = 0.15), whereas blood urea nitrogen (BUN) (r = −0.08), serum creatinine (r =

−0.05), uric acid (r = −0.08), urea (r = −0.09), and haemoglobin (r = −0.07). Showed weak negative correlations. These observations indicate limited associations rather than definitive relationships between naphthalene levels and biochemical parameters.

Similarly, acenaphthylene demonstrated generally weak correlations with biochemical indicators. A small positive relationship was observed with phenanthrene (r = 0.12) and haemoglobin (r = 0.06), whereas negative correlations were identified with ALT (r = −0.13), AST (r = −0.09), bilirubin (r = −0.04), BUN (r = −0.10), serum creatinine (r = −0.09), as well as uric acid (r = −0.03), and urea (r = −0.11). These findings indicate that acenaphthylene does not exhibit a strong association with the evaluated biochemical parameters.

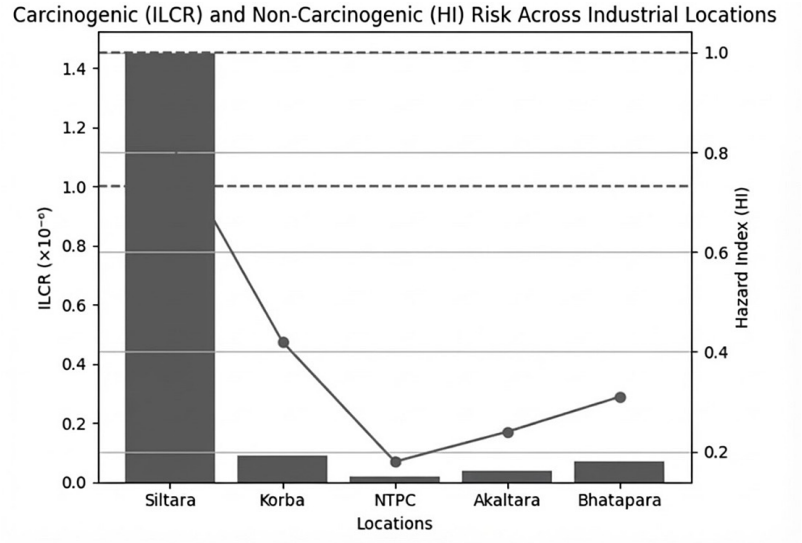

Fig. 5: Carcinogenic (ILCR) and non-carcinogenic (HI) health risk assessment across industrial locations. The dashed horizontal lines represent the acceptable cancer risk level (10-⁶) and the safety threshold for non-carcinogenic risk (HI = 1). Siltara exhibited comparatively higher carcinogenic risk values, whereas NTPC showed the lowest risk.

In contrast, phenanthrene displayed slightly stronger relationships with some biochemical markers. A moderate negative correlation was observed with naphthalene (r = −0.44) and bilirubin (r = −0.38), and weak inverse associations have been also observed with serum creatinine (r = −0.19) and uric acid (r = −0.27). Weak positive correlations were noted with ALT (r = 0.07) and AST (r= 0.04). In summary, the associations between PAHs and biochemical indicators were predominantly weak, signifying indicating limited and non-significant relationships between PAH concentrations and perturbations in hepatic, renal, and haematological parameters within the investigated cohort. However, these findings do not establish causal relationships and should be interpreted cautiously. Other studies report significant correlations between PAH exposure and adverse health effects, including altered lung function, oxidative stress markers, and inflammatory responses.[50]

Health Implications and Comparison with Regulatory Exposure Standards

The health risk assessment outcomes, as summarized in the accompanying table, demonstrate that exposure to soil-bound high-molecular-weight PAHs at investigated industrial regions poses varying levels of environmental and human health risks. Siltara recorded the highest toxic equivalent concentration of 1.99 ppm, driven by elevated contributions from carcinogenic PAHs such as benzo-[b]-fluoranthene along with benzo-[a]-pyrene. These Aromatic compounds, being characteristically linked to combustion-derived industrial emissions,

encompassing thermal power production.[10,51] The calculated incremental lifetime cancer risk for Siltara was 1.45 × 10^{-6}, which slightly exceeds the lower acceptable risk threshold recommended via the USEPA.52 According to USEPA guidelines, acceptable cancer risk levels generally fall within the range of 10^{-6} to 10^{-4},10 indicating that possible exposure to PAH-contaminated soil at Siltara could confer a modest increment in lifetime cancer risk. Conversely, Korba exhibited a substantially lower ILCR value of 0.09 × 10^{-6}, while NTPC Bilaspur, Akaltara–Janjgir, and Bhatapara displayed negligible incremental lifetime cancer risk values, signifying minimal carcinogenic concerns in risk under current exposure assumptions.

Evaluation of non-carcinogenic health risks through the hazard index demonstrated that all estimated values fell below the acceptable threshold of (HI < 1). The maximum HI was recorded at Siltara(0.82), followed by Korba (0.41), while NTPC Bilaspur (0.18), Akaltara–Janjgir(0.22), and Bhatapara(0.29) exhibited substantially lower values. USEPA risk assessment guidelines posit that HI values under 1 indicate negligible likelihood of adverse non-carcinogenic effects under prevailing exposure scenarios. Regulatory bodies, as recommended by USEPA classify numerous PAHs as an hazardous pollutants owing to their carcinogenic and mutagenic potential during chronic exposure.[53,54]

Table 5: Location-wise toxicity equivalency (TEQ), ILCR, and non-carcinogenic HI results estimated in industrial regions. The calculated ILCR values were compared with the acceptable cancer risk range recommended by the USEPA (10^{-6}–10^{-4}) to evaluate potential carcinogenic risk, whereas the HI is used to assess non-carcinogenic health effects.

Location	TEQ (ppm BaP-eq)	ILCR (×10^{-6})	USEPA Acceptable Cancer Risk Range	Hazard Index (HI)
Siltara	1.99	1.45	10^{-6} – 10^{-4}	0.82
Korba	0.13	0.09	10^{-6} – 10^{-4}	0.41
NTPC Bilaspur	0	0	10^{-6} – 10^{-4}	0.18
Akaltara–Janjgir	0	0	10^{-6} – 10^{-4}	0.22
Bhatapara	0	0	10^{-6} – 10^{-4}	0.29

Pursuant to Occupational Safety and Health Administration guidelines, occupational exposure to polycyclic aromatic hydrocarbon mixtures, such as coal tar pitch volatiles, must remain below 0.2 mg m^{-3} over an 8-hour time-weighted average, as exceedances are linked to heightened carcinogenic risks. However, direct comparison with these standards is limited, as the present study is based on environmental and biomonitoring data rather than personal exposure measurements. In this study, the elevated toxic equivalent concentrations and incremental lifetime cancer risk values at Siltara indicate a substantially greater PAH burden in this industrial locale. Furthermore, the presence of low molecular weight PAHs in blood samples of exposed individuals indicates occupational exposure to combustion-derived PAHs among industrial workers via inhalation or dermal contact routes.[4,53] This biomonitoring evidence corroborates the potential exceedance of recommended environmental safety thresholds, aligning with observations from international health agencies.[55,56] Furthermore, the International-Agency for Research on Cancer, categorizes several aromatic-polycyclic-hydro-carbons, particularly benzo-[a]-pyrene, as Group-1 carcinogens, emphasizing dangers associated with prolonged exposure.[57,58]

Overall, these results suggest that while most sites meet regulatory thresholds, industrial areas like Siltara exhibit heightened occupational exposure risks, calling for ongoing environmental monitoring, worker health surveillance, and robust emission mitigation measures to avert long-term health threats.

Conclusion

This study examined the environmental distribution, bioaccumulation, and prospective health risks of

polycyclic aromatic hydrocarbons across principal industrial zones in Central India. Analyses of soil, plant, and blood samples unveiled the pervasive occurrence of PAHs in industrial milieus, attributable to anthropogenic sources including coal-fired thermal power production, metallurgical processes, and fossil fuel combustion. Siltara and Korba were discerned as primary contamination foci, registering elevated PAH concentrations in environmental compartments.

Soil samples exhibited the paramount PAH accumulation, affirming soil's function as a principal repository for these recalcitrant contaminants. PAHs in plant tissues evince transfer via atmospheric deposition and rhizospheric uptake. Critically, low-molecular-weight PAHs detected in blood of exposed subjects denote occupational or ambient exposure among workers in proximate industrial vicinities. This biomonitoring substantiates continual worker exposure to pyrogenic PAHs through inhalational and dermal conduits.

Health risk appraisals utilizing toxic equivalency, incremental lifetime cancer risk, and hazard index metrics disclosed that non-carcinogenic risks at most sites abided by acceptable benchmarks. Nonetheless, Siltara displayed augmented TEQ and ILCR, intimating a modestly heightened carcinogenic these values remained within the acceptable risk range under standard regulatory guidelines.

These findings suggest that industrial emissions are a significant regional vector of long-term exposure to PAHs, which could lead to increasing health problems in susceptible populations. PAHs in abiotic and biotic matrices suggest occupational absorption as a potential contributor to increasing neoplastic risks and comorbidities in the workforce, despite the need for additional epidemiological data to be interpreted cautiously.

In summation, the study underscores the exigency for perpetual environmental oversight, refined industrial emission abatement, and systematic occupational health monitoring in industrialized precincts. Deployment of assiduous remediation tactics and worker safeguards is pivotal to attenuate protracted contamination and attenuate PAH-attendant health hazards.

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The authors do not have any conflict of interest.

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All relevant data generated or analysed during this study are included in this published article. Additional data are available from the corresponding author upon reasonable request.

Ethics Statement
The study involving human participants was conducted in accordance with the ethical standards of the Institutional Ethics Committee. Ethical approval was obtained from the Institutional Ethics Committee of Guru Ghasidas Vishwavidyalaya, Bilaspur, Chhattisgarh (Approval No.: GGV/IEC/2022/02/014).

Informed Consent Statement
Written informed consent was obtained from all participants prior to sample collection. Participation was voluntary, and confidentiality of personal data was strictly maintained throughout the study.

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Not Applicable

Author Contributions

- **Aayushi Pathak:** Sample Collection, Laboratory Analysis, Data Interpretation, Writing – Original Draft.
- **Neelabh Kashyap:** Methodology Development, Instrumental Analysis, Quality Assurance, Writing – Review & Editing.
- **Tandesh Lal Chandra:** Sample Processing, Data Validation, Visualization.
- **Pranjal Yadav:** Methodology Development, Instrumental Analysis, Quality Assurance, Writing – Review & Editing.
- **Sudhir Yadav:** Conceptualization, Supervision, Writing–Review & Editing, Project Administration.

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