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Polycyclic Aromatic Hydrocarbons (PAHs) in Waters and Sediments of Oil Impacted Communities in Ogbia, Bayelsa State: Concentrations, Health and Ecological Risks Assessment

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Abstract: Polycyclic aromatic hydrocarbons (PAHs) in water and sediments from oil-impacted communities in Ogbia Local Government Area, Bayelsa State, Nigeria, were investigated to determine their concentrations, spatial variability, source characteristics, and associated ecological and human health risks. Water and sediment samples were extracted using liquid–liquid and Soxhlet extraction methods, respectively, with dichloromethane (DCM) and analyzed by gas chromatography–mass spectrometry (GC–MS). Total concentrations of the sixteen priority PAHs (∑16PAHs) in water ranged from 91.40 to 191.30 µg/L (mean: 128.44 ± 40.81 µg/L), while sediment concentrations ranged from 15,572.35 to 46,043.75 µg/kg (mean: 27,258.72 ± 12,137.95 µg/kg). High-molecular-weight PAHs dominated over low-molecular-weight compounds in both matrices, with dibenzo[a,h]anthracene, benzo[a]pyrene, benzo[k]fluoranthene, benzo[b]fluoranthene, and indeno[1,2,3-cd]pyrene being the most abundant, indicating aged petroleum-related contamination. Statistical analysis revealed high spatial variability, with coefficients of variation of 33.4% and 44.5% for water and sediments, respectively, reflecting heterogeneous anthropogenic inputs. A strong positive correlation ($r > 0.90$) was observed between total PAH concentrations and total toxicity equivalence quotients (∑TEQs) in sediments, confirming that elevated PAH burdens directly translated into increased toxic potency. Sediment ∑16PAHs values largely fell between effects range-low and effects range-median guidelines, suggesting a possible-effect range where adverse ecological effects may occasionally occur. Non-carcinogenic risk assessment showed hazard indices below unity for both dermal contact and ingestion pathways, indicating negligible non-cancer risks. However, incremental lifetime cancer risk values for both adults and children exceeded the 1.0×10^{-6} benchmark, implying elevated carcinogenic risk, particularly from dermal exposure. The persistence, variability, and strong toxicity–concentration relationship observed in sediments highlight their role as long-term secondary sources of contamination and underscore the urgent need for remediation and strengthened environmental management in the affected communities.

Keywords: *petroleum, hazard index, cancer risks, sediment, water quality, reference dose.*

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1.0.Introduction

Polycyclic aromatic hydrocarbons (PAHs) are a class of persistent organic pollutants that have attracted increasing scientific attention due to their adverse effects on human health and the environment. Several PAHs compounds including benzo[a]pyrene (BaP) and benzo[a]anthracene (BaA) are known carcinogens; others, such as: benzo[b]fluoranthene (BbF) are precursors to carcinogenic daughter compounds (Zheng *et al*., 2014; Bjelić *et al*., 2022). The toxicity, mutagenicity, teratogenicity, and immunotoxicity of PAHs to several organisms have been well documented (Abdel-Shafy and Mansour, 2016; Varjani *et al*., 2017; Oliveira *et al*., 2019; Patel *et al*., 2020; Chokor & Achugwo, 2022). Their aromatic structure, coupled with hydrophobicity and thermal stability, makes PAHs resistant to degradation, enabling them to persist in the environment and exert long-term toxic effects on wildlife and humans (Sun *et al*., 2021; Caumo *et al*., 2022; Chokor, 2024). PAHs in the environment could originate from either natural or anthropogenic sources. Natural sources include: the synthesis of PAHs by biological species such as micro-organisms, phytoplankton, algae, and plants and the transformation of natural organic precursors by diagenic processes (Mojiri *et al.,* 2019; Chokor, 2022a); while anthropogenic sources come from: combustion processes, coal tar processing wastes, and petroleum-related activities (Bjelić *et al*., 2022; Chokor & Ediagbonya, 2024). Although natural processes contribute to the presence of PAHs in the environment, elevated concentrations in contaminated ecosystems are predominantly associated with anthropogenic activities (Chokor, 2021). PAHs enter water bodies through different pathways including atmospheric fallout, urban runoff, industrial and municipal waste water discharges, commercial shipping activities, oil spill, indiscriminate dumping of petroleum products on water ways, etc. (Essien *et al*., 2011; Chokor, 2021). PAHs being hydrophobic are readily absorbed by suspended particles coated in a complex matrix of organic matter in the water column, over time, particles settled down as sediment. Thus, the sediments serve as the major sink for PAHs in water bodies (streams, lakes, estuaries, and oceans). Under perturbation, these same sediments could also serve as an intrinsic source of PAHs to the water column. Thus, an equilibrium relationship often exists between PAHs in sediments and those in the overlying water column. Elevated concentrations of PAHs in aquatic ecosystems pose significant threats to aquatic organisms and present serious public health concerns due to bioaccumulation and potential human exposure. Though numerous PAHs exist in the environment, the United States Environmental Protection Agency listed 16 of these PAHs as priority pollutants to be routinely analyzed, monitored, and controlled; viz: naphthalene (Nap), acenaphthylene (Acy), acenaphthene (Acp), fluorene (Flr), anthracene (Ant), phenantrene (Phe), fluoranthene (Flt), pyrene (Pyr), benzo[a]anthracene (BaA), chrysene (Chr), benzo[b]fluoranthene (BbF), benzo[k]fluoranthene (BkF), benzo[a]pyrene (BaP), dibenzo[a,h]anthracene (DhA), indeno[1,2,3-cd]pyrene (IcP), and benzo[g,h,i]perylene (BgP) (USEPA, 2011). While several studies have investigated polycyclic aromatic hydrocarbons in different regions, data for Ogbia Local Government Area remain scarce despite its ecological and economic importance within the Niger Delta. Furthermore, existing studies in the region largely focus on either surface water or sediments in isolation, with limited attention to integrated water–sediment assessment and associated human health and ecological risks.

This study, therefore, aims to concurrently assess the concentrations, distribution patterns, and composition of priority PAHs in water and sediment samples from oil-impacted communities in Ogbia LGA, Bayelsa State, and to evaluate their potential ecological and human health risks. The findings are expected to provide critical baseline information for environmental monitoring, risk management, and remediation planning in oil-producing communities of the Niger Delta.

This investigation is necessary because communities in Ogbia Local Government Area, Bayelsa State, are continuously exposed to petroleum-related activities that are known sources of polycyclic aromatic hydrocarbons (PAHs), yet reliable and comprehensive data on the levels and risks of these pollutants in their aquatic environment are lacking. The absence of up-to-date, location-specific information on PAHs in both water and sediments limits the ability of regulatory agencies and public health authorities to accurately evaluate environmental quality, potential human exposure, and ecological threats. Given the reliance of local populations on creeks and surface waters for domestic use, fishing, and other livelihood activities, it is essential to investigate the occurrence, distribution, and possible health and ecological risks of PAHs before further degradation occurs. Conducting this study is therefore crucial for early identification of contamination hotspots, informed decision-making, and proactive environmental management and protection in the oil-impacted communities of Ogbia LGA.

2.0. Materials and Methods

2.1. Study area

The study, Ogbia Local Government Area (LGA), Bayelsa State, is located in the heart of the Niger Delta, in the south-southern region of Nigeria, between latitudes 4°39′ N and 5°02′ N and longitudes 6°16′ E and 6°35′ E, covering approximately 695 km² with a population of over 179,926 (NPC, 2006). The area is characterized by low-lying, swampy terrain with a dense network of rivers and creeks that support diverse ecosystems and sustain the local economy. Major occupations include fishing, farming, and trade, with agriculture playing a key economic role. As an oil-producing region, Ogbia LGA also hosts numerous oil and gas operations, which have contributed to economic growth but also pose environmental and social challenges.

2. 2. Sample collection

Composite samples were collected from the surface water and surface sediments (in the bottom) of some oil impacted communities in Ogbia, viz: A (Epebu: N04º39’09.7’’ E06º13’00.5’’), B (Imiringi: N04º51’08.7’’ E06º22’28.1’’), C (Kolo: N04º53’15.9’’ E06º22’25.6’’), D (Amorikeni: N04º45’27.2’’ E06º20’42.7’’), and E (Akipelai: N04º37’55.9’’ E06º20’37.8’’). Water samples were taken with 1-liter clean glass amber bottles, pre-rinsed with surrounding water, and taken below the surface (between 0 and 0.5 m). Sediment samples were taken from the bottom surface (5– 10cm) with a stainless steel Van Veen grab sampler and kept in pre cleaned wide-mouth amber bottles. The bottles were tightly covered with Teflon-lined caps to prevent any sort of contamination. The bottles containing water and sediments samples were carefully labelled and kept in ice chest at temperature below 4 °C for onward transportation to the laboratory for analysis (Ikpe *et al*., 2016; Chokor, 2024).

2. 3. PAHs Extraction

Water samples were extracted using liquid-liquid extraction with dichloromethane (DCM) after spiking with 1 mL of 10 µg/mL ortho-terphenyl and 2-fluorobiphenyl surrogate standards. Sediment samples were air-dried in the dark for five days, homogenized, and sieved through a 0.5 mm mesh. Ten grams of

sediment were mixed with ~5 g anhydrous sodium sulphate (Na_2SO_4) to remove moisture, spiked with surrogate standards, and extracted with 200 mL DCM for 17 h. Extracts were dried over anhydrous Na_2SO_4 and concentrated under reduced pressure using a rotary evaporator before column clean-up

2. 4. Sample clean-up and separation

Sample clean-up was performed using open glass columns (10 mm i.d. × 30 cm) packed with 2 g glass wool at the bottom, 10 g activated silica gel in the middle, and anhydrous Na_2SO_4 at the top. Concentrated extracts were loaded onto the column; the aliphatic fraction was first eluted with 30 mL n-hexane, followed by PAHs elution with 30 mL DCM. The aromatic fraction was concentrated to ~2 mL using a rotary evaporator at 30 °C. Aliquots (1.5 mL) were transferred into vials and stored at 4 °C until GC-MS analysis. Blank samples were processed similarly for quality assurance (Iyang *et al*., 2018; Adeniji *et al*., 2019; Chokor, 2022a,b)

2. 5. Gas chromatography analysis

A gas chromatography-mass spectrometry (Agilent 6890N (GC) coupled to an Agilent 5975B mass selective detector (Agilent Technologies, Santa Clara, USA) was employed in the analyses of the Polycyclic aromatic hydrocarbons (PAHs). A 30m × 0.32 mm × 0.25 µm dimension capillary column coated with DB-5 was used for the separation. The samples were injected into the GC via a pulsed split-less mode with an injection volume of 1µL. Pure helium gas at a flow velocity of 1 mL/min was used as the carrier gas. Column temperature was initially held at 70 °C for 20 min., followed by an increase to 150 °C at rate of 25 °C min^{-1}. The temperature was further raised to 200 °C at 3 °C min^{-1}, and finally to 300 °C at 2 °C min^{-1}. The injection port, ion source, quadrupole and transfer line temperatures were maintained at 250, 230, 150 and 280 °C, respectively.

2. 6. Identification and Quantification

Sample quantification and the quantifications of procedural recovery involved the use of Deuterated PAH internal standard solutions (naphthalene-d8, acenaphthene-10, phenanthrene-d10, chrysene-d12, and perylene-d12) and surrogate standard solutions (2-fluorobiphenyl and 4-terphenyl-d14). PAHs identification was done by comparing their retention time with those of corresponding standards, and quantification was performed using response factors related to the respective internal standards based on a five-point calibration curve for the individual PAH.

2. 7. Determination of Organic Carbon

Organic carbon contents were determined using Standard methods (Nelson and Sommers, 1982).

2.8 Human health risks assessment of PAHs in water

Non-carcinogenic and carcinogenic health risks from ingestion and dermal exposure to PAHs in water were assessed using average daily dose (ADD), chronic daily intake (CDI), hazard quotient (HQ), and incremental lifetime cancer risk (ILCR) indices (equations 1–9).

$$ADDi = \frac{(C * IR * ED * EF)}{(Bw * ATnc)} \quad (1)$$

$$ADDd = \frac{(C * Kp * SA * ET * ED * EF * CF)}{(Bw * ATnc)} \quad (2)$$

$$CDIi = \frac{(C * IR * ED * EF)}{(Bw * ATc)} \quad (3)$$

$$CDId = {(C * Kp * SA * ET * ED * EF * CF)}/{(Bw * ATc)} \quad (4)$$

where ADD_i and ADD_d refer to average daily doses due to ingestions and dermal contacts respectively; while, CDI_i and CDI_d represent the chronic daily intakes due to ingestions and dermal contacts respectively. C is the concentrations of PAHs or its BaP equivalent concentration (mg/L), Kp is the skin permeability coefficient of the PAHs (cm/h), SA represent the body surface areas (cm^2), ED is the exposure duration (Years), Bw is the body weight (Kg), ATnc and ATc are the averaging time for non-cancer and cancer risks (days) (Table 1).

Table 1: Input data used to calculate non-carcinogenic and carcinogenic human health risk due to PAHs exposure through ingestions and dermal contact.

		Adults	Children
Body surface area (SA)	cm^2	18000	6600
Average ingestion of water (IR)	L/day	2.5	0.78
Exposure duration (ED)	Years	30	6
Average body weight (Bw)	Kg	70	15
Average time for non-cancer risk (ATnc)	days	10950	2190
Average time for cancer risk (ATc)	days	25550	25550
Dermal permeability coefficient (Kp)	cm/h	1.2(for BaP)	1.2 (for BaP)
Exposure time of shower & bathing (ET)	h/day	0.58	1.0
Exposure frequency (EF)	days/yr	350	350
Conversion factor (CF)	L/1000cm	-	-

The hazard quotient (HQ) is the ratio between the calculated ADD of PAH and the reference dose (RfD, mg/kg.day) that connotes the daily exposure to which human populations could be exposed to continually over an average life time without incidence of negative health impacts. The reference doses were abstracted from USEPA (2007)

$$HQ = {ADD}/{RfD} \quad (5)$$

$$HI = \sum_{i=1}^{n} HQ \quad (6)$$

The exposed populations is saved from non-carcinogenic harms if the sum of the hazard quotients - ∑HQs (hazard index, HI) - is less than one; whereas, HI > 1 debits the possibility of non-carcinogenic health risks to the local populations (Bandowe *et al*., 2014; USEPA, 2014; Chokor & Ediagbonya, 2024).

The incremental lifetime cancer risks (ILCR) for each PAH were calculated from equations 7 – 9.

$$ILCR = SFi * CDIi \quad (7)$$

$$ILCRd = SFd * CDId \quad (8)$$

$$ILCRt = ILCRi * ILCRd \quad (9)$$

where: ILCRi and ILCRd are the incremental lifetime cancer risks due to ingestions and

dermal contacts, ILCRt represent the sum of both, while SFi and SFd are the slope factors for ingestions and dermal contacts with values of 7.3 and 12 mg/kg/day, respectively, for BaP. Values for other PAHs were extrapolated by multiplying the SF for BaP by the toxic factor of individual PAHs.

2.9. Ecological risks of PAHs in sediments

Ecological risks of PAHs in sediments were evaluated using sediment quality guidelines, including effect range-low (ERL) and effect range-median (ERM) values (Johnson *et al*., 2006; Baumard *et al*., 1998; Long *et al*., 1995; McGrath *et al*., 2019), as well as toxic equivalence factors (TEFs) (Nisbet and LaGoy, 1992; USEPA, 2003; Lee & Vu, 2010).

2.10. Statistical analysis

The analytical results were compiled to form multi-element data base. Excel was used as statistical software.

3. 0. Result and Discussion

3.1. Concentrations and distributions of PAHs in water

Table 2 shows the concentration profiles of the 16 priority PAHs in water columns at the different communities. Individual PAH concentrations in the water column ranged from 0.10 – 46.30µg/L; while the sum of 16 priority PAHs (∑16PAHs) was from 91.40 – 191.30 µg/L with a mean of 128.44µg/L. The most dominant PAH was dibenzo(a)anthracene (DhA) with values ranging from 3.35 – 46.30 µg/L (mean: 26.24µg/L). The concentrations of benzo(a)pyrene (BaP) – a major carcinogenic PAH were from 8.10 – 46.45 µg/L with a mean value of 21.08 µg/L, representing 16.41% of the total PAHs. Other abundant PAHs listed in decreasing order are BkF, BgP, IcP, and BbF with mean values of: 19.59, 15.67, 15.24, and 13.87µg/L and representing 15.25, 12.20, 11.87, and 10.80% of the total PAHs, respectively. The lower molecular weight PAHs (LMWP) tended to have lower concentrations in the water columns than the high molecular weight ones (HMWP).The lowest concentrations were observed for naphthalene (Nap) and acenaphthylene (Acy), both with mean values of 1.00µg/L. This may be attributed to the higher volatility and greater biodegradability of LMW PAHs compared to HMW PAHs, leading to their lower concentrations in water (Sun *et al*., 2021).The order of total PAHs concentrations in the various communities was: Kolo (C) >Akipelai (E) >Imiringi (B) >Epebu (A) >Amorikeni (D). The range of total PAHs (∑16PAHs) obtained in this study, is comparable to the 14.91 - 206µg/L obtained for the Buffalo River water by Adeniji *et al* (2019), and the 24.39 – 283.6 (mean: 176.1) µg/L reported by Okoro (2008) for Ekpan Creek, Warri, Nigeria; but were much lower than the 6024 - 29252 µg/L recorded for the Woji Creek, Nigeria (Ihunwo *et al*., 2019). However, these concentrations were considerably higher than those reported for the Urban River Network of Shanghai (China), which ranged from 0.072 – 0.461 µg/L in winter and 0.047 – 0.222 µg/L in summer (Liu *et al*., 2016). The results were also higher than the range of values (33.2 – 84.5 and 4.5 – 13.94µg/L) observed for the surface water of Tema Harbour, Ghana, and bore-hole water from the vicinity of an unlined dumpsite in Akwa, Nigeria, respectively (Gorleku *et al*, 2014; Aralu et al., 2023).

3.2 Health risks assessment of PAHs in water

The calculated hazard quotients due to dermal contact with the waters (HQs dermal) for the individual PAH in the water column were all less than one (1) (Table S1). The sums of HQs dermal ($\sum HQ_d$) for the different communities which ranged from 3.12×10^{-6} – 6.72×10^{-6} with a mean of 4.67×10^{-6} for adults, and 9.20×10^{-5} – 1.44×10^{-4} (mean: 1.38×10^{-4}) for children were also less than one (Table 3). This implies that there would be no visible non-cancer risks as a result of having dermal contact

with the waters through regular bathing or recreation activities.

The hazard quotients due to ingestions (HQ_i) though much higher than those resulting from dermal contacts; also had its HQs for individual (Table S1); and its total HQs (HI) less than one (1) (Table 3). The ∑HI (sum of ∑HQs from dermal and ingestion) of the water were still less than one, indicating the safety of the water from non-carcinogenic harms.

The calculated incremental lifetime cancer risks (ILCR) due to dermal and ingestion for the sum of seven (7) carcinogenic PAHs ($\sum_7$ILCRt) were, however, greater than the 1.0 × 10^{-6} recommended standard. The calculated incremental lifetime cancer risks due to dermal contacts for the seven carcinogenic PAHs ($\sum_7$ILCRd) ranged from 5.0 X10^{-3} – 1.19 X 10^{-2}; and 2.2 × 10^{-2} – 9.57 × 10^{-2} for adults and childre respectively.

Table 2: mean concentrations (µg/L) of PAHs in the waters

compound	Ring No	Concentrations (µg/L H_2O)					
		A	B	C	D	E	Ave
Nap	2	1.30	0.65	0.85	0.25	1.95	1.00
Acy	3	0.95	1.10	0.35	1.50	1.10	1.00
Acp	3	0.75	2.55	1.85	1.55	1.95	1.73
Flr	3	1.75	2.00	2.15	1.75	2.20	1.97
Ant	3	2.55	0.40	1.20	1.25	1.70	1.42
Phe	3	2.90	1.20	0.30	0.50	0.85	1.15
Flt	4	0.70	1.20	2.90	0.35	0.15	1.06
Pyr	4	0.75	1.00	0.20	0.10	3.80	1.17
BaA	4	1.50	1.95	4.70	2.50	2.60	2.65
Chr	4	1.65	2.25	10.65	1.95	1.50	3.60
BbF	5	14.40	8.50	10.85	9.00	26.60	13.87
BkF	5	31.75	2.60	26.40	15.30	21.90	19.59
BaP	5	11.20	27.90	46.45	11.75	8.10	21.08
DhA	5	3.35	34.90	36.05	10.60	46.30	26.24
IcP	6	15.10	8.00	13.95	22.70	16.45	15.24
BgP	6	9.25	17.55	32.45	10.35	8.75	15.67
∑PAHs		99.85	113.75	191.30	91.40	145.90	128.44

*A: Epebu; B: Imiringi; C: Kolo; D: Amorikeni; E: Akipelai;Nap: naphthalene; Acy: acenaphthylene; Acp: acenaphthene; Flr: fluorine; Ant: anthracene; Phe: phenantrene; Flt: fluoranthene; Pyr: pyrene; BaA: benzo[a]anthracene; Chr: chrysene; BbF: benzo[b]fluoranthene; BkF: benzo[k]fluoranthene; BaP: benzo[a]pyrene; DhA: dibenzo[a,h]anthracene; IcP: indeno[1,2,3-cd]pyrene; and BgP: benzo[g,h,i]perylene.**

The mean $\sum_7$ILCRd values for adults and children were 1.02 × 10^{-2} and 5.69 X 10^{-2} respectively. The total Incremental life time cancer risk ($\sum_7$ILCRi) due to ingestions for adult for the different stations ranged from 2.3 × 10^{-3} – 9.7 × 10^{-3} (mean: 5.8 X 10^{-3}); while that due to ingestion by children was from 6.7 × 10^{-4} – 2.8 × 10^{-3} (mean: 1.68 × 10^{-3}). Individual values for ILCRd and ILCRi for the various PAHs in the different communities are as shown in Table S2. The ILCRd – cancer risks due to dermal contact for children were

higher than those for adults. Probable reasons for this might be: (1) less thick and more permeable skin of children in comparison to adults, (2) increased exposure consequence of large skin surface area relative to body weight in children compared to adults, and (3) a greater susceptibility arising from the organs and immune system of children still at the developmental stage. The reverse was, however, observed for cancer risks due to ingestions (ILCRi). Greater consumptions, cumulative risk due to longer exposure times, and differences in metabolic pathways in adults compared to children are probable reasons for this observation. Dermal risks were also higher than those due to ingestions for both adults and children; probably because dermal exposures and its slop factors (SF) are much higher than those due to ingestions. The overall risk due to both dermal and ingestions was higher in children than in adults. A lot of factors, such as the developing immune system and pica activities of children, may increase the vulnerability of children to cancer more than in adults. According to the United States Environmental Protection Agency (USEPA, 2007), incremental lifetime cancer risk (ILCR) values less than one in a million ($\leq 10^{-6}$) connote virtual safety, values in the range of $10^{-6} - 10^{-4}$ represent potential risks, whereas values larger than 10^{-4} implicate potentially high risks. The values obtained in this report implicate potentially high risk for both adults and children developing cancer in their lifetime due to dermal contact and ingestion of these waters. As the water may not often be ingested, the major risk would be exposure of the populations to skin cancers.

Table 3: Calculated values for hazard index (HI) and total incremental lifetime cancer risks(ILCRt) for waters in the different communities

Group	Parameters	A	B	C	D	E	Mean
Adult	$\sum_7$HQd	4.87E-06	4.69E-06	3.97E-06	3.12E-06	6.72E-06	4.67E-06
	$\sum_7$HQi	9.72E-06	9.36E-03	7.92E-03	6.22E-03	1.34E-02	7.38E-03
	HI	1.46E-05	9.36E-03	7.92E-03	6.22E-03	1.34E-02	7.39E-03
Child	$\sum_7$HQd	1.44E-04	1.38E-04	1.17E-04	9.20E-05	1.98E-04	1.38E-04
	$\sum_7$HQi	1.41E-02	1.36E-02	1.15E-02	9.06E-03	1.95E-02	1.36E-02
	HI	1.42E-02	1.37E-02	1.16E-02	9.15E-03	1.97E-02	1.37E-02
Adult	$\sum_7$ILCRd	1.19E-02	1.62E-02	5.00E-03	8.00E-03	9.70E-03	1.02E-02
	$\sum_7$ILCRi	2.30E-03	7.10E-03	9.70E-03	3.00E-03	6.70E-03	5.76E-03
	TCR	1.42E-02	2.33E-02	1.47E-02	1.10E-02	1.64E-02	1.59E-02
Child	$\sum_7$ILCRd	2.26E-02	7.04E-02	9.57E-02	2.96E-02	6.64E-02	5.69E-02
	$\sum_7$ILCRi	6.70E-04	2.08E-03	2.83E-03	8.80E-04	1.96E-03	1.68E-03
	ILCRt	2.33E-02	7.25E-02	9.85E-02	3.05E-02	6.84E-02	5.86E-02

*A: Epebu; B: Imiringi; C: Kolo; D: Amorikeni; E: Akipelai

3.3 Concentrations and distributions of PAHs in sediments

Table 4 shows the concentrations of individual PAH and total PAHs ($\sum$16PAHs) in sediments. The values of PAH in sediment ranged from 70.02 – 14818.71µg/kg. The proportions of 5 – 6 ring PAHs were much higher in the sediments compared to 4 rings, and 2- 3 rings PAHs (Fig. 1). The mean proportions of 2-3 rings, 4 rings, and 5 -6 rings PAHs in the sampled communities were 13.4, 12.8, and 73.7%, respectively. The $\sum$16PAHs values (µg/kg) ranged from 15,572.35 in Amorikeni to 46,043.75 in Kolo; values in other communities

are: Imiringi (29,914.61), Akiplai (27,137.3), and Epebu (17,625.37). The mean value for all communities was 27,258.72µg/kg.

The range and mean obtained in this study are comparable to those recorded in sediments by Howard *et al* (2001) and Kuang,*et al* (2026), but much higher than those obtained by Okoro (2008), Da Silva *et al* (2007), Chokor and Achugwo (2022), Edokpayi *et al* (2016), and Oyo-Ita *et al* (2013, 2017) (Table 5). The values were, however, much lower than those reported for sediments by Gorleku *et al* (2014), Kafilzadeh et al (2011), and Wang *et al* (2001) (Table 5).

Table 4. Mean PAHs concentrations (µg/Kg) in the Sediments of the different communities: A: Epebu; B: Imiringi; C: Kolo; D: Amorikeni; E: Akipelai

PAH	Ring No	Concentrations (µg/kg Sediment)							
		A	B	C	D	E	Ave	ERL	ERM
Nap	2	70.02^{x}	289.39^{y}	270.02^{y}	330.05^{y}	400.11^{y}	271.92^{y}	160	2100
Acy	3	480.15^{y}	160.77^{y}	140.01^{y}	340.05^{y}	760.2^{y}	376.24^{y}	44	640
Acp	3	220.07^{y}	342.99^{y}	1620.13^{z}	70.01^{y}	1990.54^{z}	848.75^{z}	16	500
Flr	3	590.18^{z}	943.21^{z}	680.06^{z}	480.07^{x}	2880.77^{z}	1114.86^{z}	19	540
Ant	3	240.07^{x}	203.65^{x}	1030.08^{y}	330.05^{x}	1180.32^{z}	596.83^{x}	843	1100
Phe	3	440.13^{y}	75.03^{x}	330.03^{y}	340.05^{y}	340.09^{y}	305.07^{y}	240	1500
Flt	4	80.02^{x}	439.45^{x}	690.06^{y}	200.03^{x}	1090.29^{y}	499.97^{x}	600	5100
Pyr	4	620.19^{x}	546.63^{x}	2530.21^{y}	240.04^{x}	340.09^{x}	855.43^{y}	665	2600
BaA	4	1520.46^{y}	1168.3^{y}	3740.3^{y}	350.05^{y}	2170.58^{z}	1789.94^{z}	261	1600
Chr	4	730.22^{y}	450.17^{y}	1020.08^{y}	260.04^{x}	360.1^{x}	564.12^{z}	384	2800
BbF	5	2220.68^{x}	14818.71^{x}	5370.44^{x}	1670.25^{x}	1300.35^{x}	5076.09^{x}	NA	NA
BkF	5	2060.53^{x}	2604.55^{x}	10170.83^{x}	2330.35^{x}	2030.55^{x}	3839.36^{x}	NA	NA
BaP	5	4201.28^{y}	535.92^{y}	4870.4^{z}	2650.4^{z}	2150.58^{z}	2881.72^{z}	430	1600
DhA	5	970.3^{z}	3376.27^{z}	7350.6^{z}	3590.54^{z}	2560.69^{z}	3569.68^{z}	63.4	260
IcP	6	1120.34^{x}	1286.2^{x}	3330.27^{x}	1640.25^{x}	4101.1^{x}	2295.63^{x}	NA	NA
BgP	6	2060.63^{x}	2679.58^{x}	2900.24^{x}	750.11^{x}	3480.94^{x}	2374.30^{x}	NA	NA
∑PAHs		17625.37^{y}	29914.81^{y}	46043.75^{z}	15572.35^{y}	27137.3^{y}	27258.72^{y}	4022	44792

*x, y, and z represent concentrations: < ERL; between ERL - ERM; and > ERM respectively; ERL: Effects Range Low; ERM: Effects Range Median; Nap: naphthalene; Acy: acenaphthylene; Acp: acenaphthene; Flr: fluorine; Ant: anthracene; Phe: phenantrene; Flt: fluoranthene; Pyr: pyrene; BaA: benzo[a]anthracene; Chr: chrysene; BbF: benzo[b]fluoranthene; BkF: benzo[k]fluoranthene; BaP: benzo[a]pyrene; DhA: dibenzo[a,h]anthracene; IcP: indeno[1,2,3-cd]pyrene; and BgP: benzo[g,h,i]perylene.**

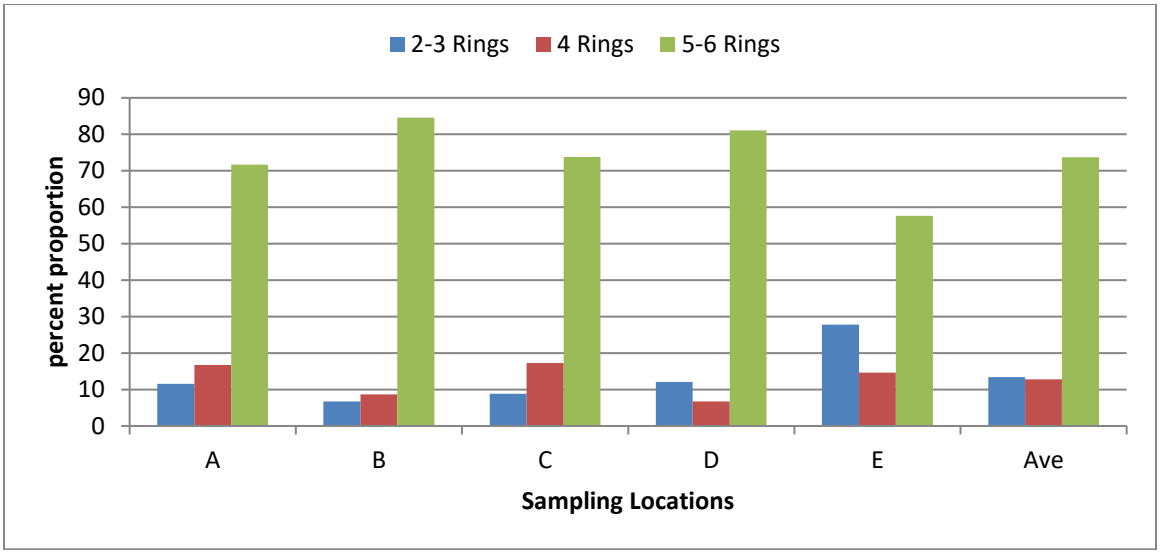

Fig. 1: Percent proportions of 2 - 3 rings, 4 rings, and 5 - 6 rings in the different sediments
*** A: Epebu; B: Imiringi; C: Kolo; D: Amorikeni; E: Akipelai; Ave: Average**

Table 5: Comparison of PAHs levels in sediments in this study with those of others

Region	ΣPAHs (µg/kg)	References
Ekpan Creek, Warri, Nigeria	5583.0 – 9326.5	Okoro, 2008
Guanabara Bay, Brazil	77 - 7751	Da Silva et al., 2007
Aba River, Nigeria	481.95 – 2,562.55 (1122.64±839)	Chokor and Achugwo, 2022
Mvudi and Nzhelele River, Vhembe District, South-Africa	440 – 21,600 206 – 13,710	Edokpayi et al., 2016
Calabar River, Nigeria	1,670 – 20,100 (4,796)	Oyo-Ita et al., 2013
Imo River, Nigeria	409.43 – 41.198 (9,370)	Oyo-Ita et al., 2017
River bed sediment from Artisanal Crude refining Area, Niger-Delta, Nigeria	23,461 – 89,886	Howard et al., 2021
River Taihu Lake, China	5,736.2 – 69,362.8	Dong et al., 2021
Jema Habour, Ghana	28,600 – 190,300	Gorleku et al., 2014
Kor River, Iran	167,400 – 530,300	Kafilzadeh et al., 2011
Boston Habour, USA.	7,300 – 358,000	Wang et al., 2001

*** mean in Brackets**

3.4 Assessment of PAHs eco-toxic potentials in the sediments

The potential risks of PAHs in sediments to aquatic eco-system were assessed using sediment quality guidelines – Effect range low

(ERL) and Effect range median (ERM) target values (Table 4). Concentrations of PAHs in sediments that are lower than ERL and larger than ERM connotes the probability of adverse effects being less than 10% and higher than 50%, respectively. This means that concentrations below ERL represent a minimal-effect range in which negative effects are rarely observed, while concentrations above ERM indicate a probable-effect range in which negative health effects are frequently observed. The possible-effect range in which adverse effects would occasionally occur lies between the ERL and ERM (MacDonald *et al*., 1996; McGrath *et al*., 2019; Chokor, 2022b). The comparison of PAHs concentrations with ERL and ERM shown in Table 4; reveal that individual PAH in most communities, except for anthracene (Ant) and pyrene (Pyr) were higher than the value for ERL but lower than that ERM; implicating a possible-effect range. The values for BaP and DhA were, however higher than ERM in most communities; BaA was also higher than the ERM in Kolo (C) and Amorikeni (D) communities, while Acy and Acp had values higher than the ERM only in Akipelai (E) community. This tends to implicate high probability of more frequent negative health effects for these individual PAHs in these communities. The overall mean total PAHs for all communities and the total PAHs for individual community except in Kolo (which was slightly higher), however lie within ERL and ERM. This implies that generally, PAHs in these study communities will show a possible-effect range in which adverse effects will be occasionally observed. Johnson et al (2006) stipulated sediment quality guideline of 1000 µg/Kg dw as benchmark to protect estuarine fish against several important health effects. The results of this study were all higher than this guideline. Baumard *et al* (1998) also classified pollutant PAHs levels in sediments into: low (0 – 100µg/Kg), moderate (100 – 1,000µg/Kg), high (1,000 – 5,000µg/Kg), and very high (>5,000µg/Kg). The sedimentary PAH levels of this study can thus, be characterized based on the above classification as very high.

The toxicity of PAHs was also assessed using the toxic equivalence factors (TEFs). Benzo(a)pyrene – one of the most carcinogenic PAHs with a documented history of toxic potency – was used as an exposure marker for the risk assessments (Nisbet and LaGoy, 1992; USEPA, 2003; Lee and Vu, 2010). The Benzo(a)pyrene toxic equivalence quotients (TEQs) was calculated using equation. 10.

$$\sum TEQi = \sum Ci\ X\ TEFi \qquad (10)$$

where TEQ = toxic equivalent quotient for individual PAH, C_i = concentration of individual PAH, TEF_i toxic equivalent relative to benzo(a)pyrene. The observed values for total TEQs (∑C16TEQs) ranged from 5729.54 – 14537.95µg/kg dry wt. with an average value of 7791.12 µg/kg dry wt (Table 6). The range for the seven carcinogenic PAHs was from 5675.13 – 14492.38 µg/kg dry wt. (mean: 7757.14 µg/kg dry wt.) representing about 99.56% of the mean total TEQs (∑C16TEQs).

Table 6: TEFs and TEQs values for the sixteen priority PAHs in the sediments

PAHs	TEFs	TEQs (µg/Kg dw)						
		A	B	C	D	E	Ave	%contr.
Nap	0.001	0.07	0.29	0.27	0.33	0.40	0.27	0.00
Acy	0.001	0.48	0.16	0.14	0.34	0.76	0.38	0.00
Acp	0.001	0.22	0.34	1.62	0.07	1.99	0.85	0.01
Flr	0.001	0.59	0.94	0.68	0.48	2.88	1.11	0.01
Ant	0.010	2.40	2.04	10.30	3.30	11.80	5.97	0.08

Phe	0.001	0.44	0.08	0.33	0.34	0.34	0.31	0.00
Flt	0.001	0.08	0.44	0.69	0.20	1.09	0.50	0.01
Pyr	0.001	0.62	0.55	2.53	0.24	0.34	0.86	0.01
BaA	0.100	152.05	116.83	374.03	35.01	217.06	178.99	2.30
Chr	0.010	7.30	4.50	10.20	2.60	3.60	5.64	0.07
BbF	0.100	222.07	1481.87	537.04	167.03	130.04	507.61	6.52
BkF	0.100	206.05	260.46	1017.08	233.04	203.06	383.94	4.93
BaP	1.000	4201.28	535.92	4870.40	2650.40	2150.58	2881.72	36.99
DhA	1.000	970.30	3376.27	7350.60	3590.54	2560.69	3569.68	45.82
IcP	0.100	112.03	128.62	333.03	164.03	410.11	229.56	2.95
BgP	0.010	20.61	26.80	29.00	7.50	34.81	23.74	0.30
∑C7TEQs		5871.08	5904.47	14492.38	6842.63	5675.13	7757.14	99.56
∑C16TEQs		5896.59	5936.10	14537.95	6855.43	5729.54	7791.12	100.00

ΣC7TEQs: Toxic equivalent quotient for the seven carcinogenic PAHs, ΣC16TEQs: TEQs value for the 16 PAHsA: Epebu; B: Imiringi; C: Kolo; D: Amorikeni; E: Akipelai, Nap: naphthalene; Acy: acenaphthylene; Acp: acenaphthene; Flr: fluorine; Ant: anthracene; Phe: phenantrene; Flt: fluoranthene; Pyr: pyrene; BaA: benzo[a]anthracene; Chr: chrysene; BbF: benzo[b]fluoranthene; BkF: benzo[k]fluoranthene; BaP: benzo[a]pyrene; DhA: dibenzo[a,h]anthracene; IcP: indeno[1,2,3-cd]pyrene; and BgP: benzo[g,h,i]perylene.

The values obtained in this report were much higher than the recommended toxic level of 137 µg/kg ∑C7TEQs by the United States Environmental Agency for the clean-up level for a mixture of carcinogenic PAHs in sediments. The major contributors to the sediments' toxicities were: DhA (45.82%) and BaP (36.99%). Other significant but much less contributors were: BbF (6.52%), BkF (4.93%), and IcP (2.95%). Contributions to total TEQs by the other PAHs were infinitesimally small. The order of total PAHs (∑16PAHs) concentrations in the communities, which was: C > B > E > A >D was different from that due to total TEQs (∑16TEQs) concentrations which was: C > D > B > A > E, evidencing the impacts of individual PAH profiles on the total PAHs toxicity at the different communities. The toxicities of PAHs in sediments are impacted by the amount of organic carbon (OC) available in the sediment matrices. The more the OC in sediments, the more PAHs are bonded to it, rendering them less bioavailable to impact toxicity to aquatic life. Thus, to factor in the amount of organic carbon in sediments, it is usual to normalized PAHs at 1% of OC concentrations. The organic carbon (OC) concentrations for the different stations, viz.: A (3.45%), B (3.75%), C (4.93%), D (3.20%), and E (4.75%) were used to normalized TEQs values at 1% OC. The values obtained for the normalized total TEQs (∑C16TEQs) (in µg/kg dw.OC) for the stations in order of decreasing magnitudes were: C (2.949×10^5), D (2.142×10^5), A (1.709×10^5), B (1.583×10^5), and E (1.206×10^5). The mean total TEQs value was 1.918×10^5. Again, this order was different from the un-normalized ∑TEQ, emphasizing the impacts of organic carbon (OC) content on the bio-availabilities and consequent toxicities of PAHs. The toxicities of PAHs in sediments can be highly cushioned by the abundance of organic carbon in the sediments (Chokor and Achugwo, 2022) it is noted that in all modes of evaluations, Kolo (C) community always appeared to be the most polluted. This is perhaps due to oil-related activities that are more pronounced in the Kolo community than in most other communities.

3.5 Further Statistical Analysis

Further statistical analyses were conducted to strengthen the interpretation of the spatial distribution, variability, and toxicological relevance of polycyclic aromatic hydrocarbons (PAHs) measured in water and sediment matrices across the studied communities. Descriptive statistics and correlation analysis were applied to experimentally determine PAH concentrations in order to quantitatively assess heterogeneity, source influence, and the relationship between total PAH burden and associated toxic potency.

The degree of spatial variability of PAHs in water and sediments was evaluated using the coefficient of variation (CV), which provides a normalized measure of dispersion relative to the mean concentration. The coefficient of variation was calculated using equation 11

$$CV(\%) = \frac{\sigma}{\mu} \times \frac{100}{1} \qquad (11)$$

where σ is the standard deviation of the measured PAH concentrations and μ is the arithmetic mean concentration. The results of the variability analysis are presented in Table 7.

Table 7: Descriptive statistics and coefficient of variation of total PAHs in water and sediments

Matrix	Mean ∑PAHs	Coefficient of Variation (%)
Water	128.84 μg/L	31.77
Sediment	27,258.72 μg/kg	44.53

The coefficient of variation for total PAHs in water (31.77%) indicates moderate spatial variability across the studied communities, suggesting that although PAHs are widespread, their concentrations are influenced by localized inputs and hydrodynamic mixing processes within the aquatic system. In contrast, the substantially higher coefficient of variation observed for sediments (44.53%) reflects pronounced spatial heterogeneity. This finding confirms that sediments serve as a long-term sink for PAHs, accumulating contaminants differentially depending on proximity to pollution sources, sediment composition, and depositional conditions.

These statistical outcomes are consistent with the experimental findings of markedly higher PAH concentrations in sediments compared to the overlying water column, particularly in communities such as Kolo and Imiringi. Similar patterns of elevated variability in sediments relative to water have been reported for oil-impacted environments in the Niger Delta and other petroleum-producing regions, where sediments retain historical contamination and respond more slowly to environmental flushing and degradation processes.

To further examine the toxicological relevance of the measured PAHs, Pearson's correlation analysis was applied to assess the relationship between total PAH concentrations (∑PAHs) and their corresponding toxic equivalency quotients (∑TEQs) in sediments. The Pearson correlation coefficient was calculated using equation 12

$$r = \frac{\sum(x_i - \bar{x})\,(y_i - \bar{y})}{\sum(x_i - \bar{x})^2\, 2\sum(y_i - \bar{y})^2} \qquad (12)$$

where x_i and y_i represent individual values of ∑PAHs and ∑TEQs respectively, and $\bar{x}$ and $\bar{y}$ are their mean values.The correlation matrix obtained from this analysis is presented in Table 8.

Table 8: Pearson correlation matrix between total PAHs and toxic equivalency quotients in sediments

Parameter	∑PAHs	∑TEQs
∑PAHs	1.00	0.82
∑TEQs	0.82	1.00

A strong positive correlation (r = 0.82) was observed between total PAH concentrations and toxic equivalency quotients in sediments. This indicates that increases in overall PAH burden are accompanied by corresponding

increases in toxic potency, largely driven by carcinogenic high-molecular-weight PAHs. This relationship demonstrates that toxicity in the study area is not merely a function of total concentration but is strongly influenced by PAH composition.

The strong PAH–TEQ association corroborates the experimental health risk assessment results, which showed elevated incremental lifetime cancer risks (ILCRs), particularly via dermal exposure pathways. Sites with higher sediment PAH concentrations also exhibited disproportionately high TEQ values, explaining the observed exceedance of USEPA cancer risk thresholds despite moderate non-carcinogenic hazard indices. Comparable correlations between PAH concentrations and TEQs have been documented in contaminated sediments from coastal and estuarine systems subjected to chronic petroleum inputs.

Overall, the statistical analyses provide quantitative support for the experimental findings, confirming that sediment-associated PAHs dominate the contamination profile and are the primary contributors to carcinogenic risk in the study area. The pronounced spatial heterogeneity and strong linkage between concentration and toxicity underscore the need for site-specific risk management strategies that prioritize sediment remediation and reduction of dermal exposure, particularly for vulnerable populations such as children.

5.0 Conclusion

This study comprehensively assessed the concentrations, spatial distribution, ecological implications, and human health risks of polycyclic aromatic hydrocarbons (PAHs) in water and sediments from oil-impacted communities in Ogbia Local Government Area. The mean ∑16PAHs concentrations of 128.44 ± 40.81 µg/L in water and 27,258.72 ± 12,137.95 µg/kg in sediments clearly demonstrate substantial PAH contamination, with the large standard deviations and high coefficients of variation reflecting strong spatial heterogeneity and the predominance of localized anthropogenic inputs, notably petroleum-related activities. High-molecular-weight PAHs, particularly dibenzo[a,h]anthracene (DhA), benzo[a]pyrene (BaP), benzo[k]fluoranthene (BkF), benzo[b]fluoranthene (BbF), and indeno[1,2,3-cd]pyrene (IcP), dominated both environmental matrices, indicating a strong pyrogenic and petrogenic influence. Statistical analyses further confirmed moderate variability in water (CV ≈ 32%) and pronounced heterogeneity in sediments (CV ≈ 45%), underscoring the role of sediments as long-term sinks and secondary sources of PAHs. The strong positive correlation between total PAHs and toxic equivalency quotients in sediments highlights that increasing PAH burden is directly associated with elevated carcinogenic potency.

Health risk assessment showed that non-carcinogenic risks from both dermal contact and ingestion of water were below threshold levels for adults and children. However, incremental lifetime cancer risk values exceeded recommended limits, particularly through dermal exposure, indicating significant carcinogenic risk potential for exposed populations. Although PAH concentrations in the water column were comparatively lower, the substantial accumulation in sediments suggests that future disturbances could remobilize contaminants, leading to episodic increases in waterborne PAHs and sustained exposure risks.

Ecological risk evaluation revealed that sediment ∑16PAHs concentrations largely fell between the effects range-low (ERL) and effects range-median (ERM), signifying a possible-effects range in which adverse biological effects may occur intermittently. Furthermore, the total toxic equivalency quotients for all communities exceeded established sediment clean-up benchmarks for mixtures of carcinogenic PAHs, clearly

indicating the need for remediation. Overall, the findings demonstrate that sediments represent the primary reservoir and risk driver of PAHs in the study area, necessitating targeted management and remediation strategies to mitigate long-term ecological degradation and human health risks.

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Declaration

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Competing Statement

There are no competing financial interests in this research work.

Ethical considerations

Not applicable

Data availability

The microcontroller source code and any other information can be obtained from the corresponding author via email.

Authors' Contribution

Augustine Avwerosuo Chokor conceptualized the research idea and methodology. Thomas Ohwofasa Ikpesu, Thompson Faraday Ediagbonya, and Chimezie Nathaniel Achugwo contributed to field work, sample analysis, data acquisition, and interpretations of results. All parties were involved in manuscript development. Augustine Avwerosuo Chokor wrote the original draft, which was reviewed and edited by all parties.

SUPPLEMENTAL MATERIALS

Table S1: Harzard Quotients (HQs) and Index (Hi) for the different communities

PAH	Dermal HQs for Adults					
	A	B	C	D	E	mean
Nap	1.12E-06	5.58E-07	7.29E-07	2.15E-07	1.67E-06	8.58E-07
Acy	8.15E-07	9.44E-07	3.00E-07	1.29E-06	9.44E-07	8.58E-07
Acp	2.15E-07	7.29E-07	5.29E-07	4.43E-07	5.58E-07	4.95E-07
Flr	7.51E-07	8.58E-07	9.22E-07	7.51E-07	9.44E-07	8.45E-07
Ant	1.24E-06	5.15E-07	1.29E-07	2.15E-07	3.65E-07	4.93E-07
Fit	3.00E-07	5.15E-07	1.24E-06	1.50E-07	6.44E-08	4.55E-07
Pyr	4.29E-07	5.72E-07	1.14E-07	5.72E-08	2.17E-06	6.69E-07
HI_d	4.87E-06	4.69E-06	3.97E-06	3.12E-06	6.72E-06	4.67E-06
Dermal HQs for Children						
Nap	3.29E-05	1.65E-05	2.15E-05	6.33E-06	4.94E-05	2.53E-05
Acy	2.40E-05	2.78E-05	8.86E-06	3.80E-05	2.78E-05	2.53E-05
Acp	6.33E-06	2.15E-05	1.56E-05	1.31E-05	1.65E-05	1.46E-05
Flr	2.22E-05	2.53E-05	2.72E-05	2.22E-05	2.78E-05	2.49E-05
Ant	3.67E-05	1.52E-05	3.80E-06	6.33E-06	1.08E-05	1.46E-05
Fit	8.86E-06	1.52E-05	3.67E-05	4.43E-06	1.90E-06	1.34E-05
Pyr	1.27E-05	1.69E-05	3.38E-06	1.69E-06	6.41E-05	1.97E-05
HI_d	1.44E-04	1.38E-04	1.17E-04	9.20E-05	1.98E-04	1.38E-04
Ingestions HQs for Adults						
Nap	0.00223	0.00111	0.00146	0.00043	0.00334	0.00171
Acy	0.00163	0.00188	0.00060	0.00257	0.00188	0.00171
Acp	0.00043	0.00146	0.00106	0.00088	0.00111	0.00099
Flr	0.00150	0.00171	0.00184	0.00150	0.00188	0.00169
Ant	0.00248	0.00103	0.00026	0.00043	0.00073	0.00098
Fit	0.00060	0.00103	0.00248	0.00030	0.00013	0.00091
Pyr	0.00086	0.00114	0.00023	0.00011	0.00434	0.00134
HI_i	0.009717	0.009361	0.007920	0.006221	0.013413	0.009326
Ingestions HQs for Children						
Nap	0.00324	0.00162	0.00212	0.00062	0.00486	0.00249
Acy	0.00237	0.00274	0.00087	0.00374	0.00274	0.00249
Acp	0.00062	0.00212	0.00154	0.00129	0.00162	0.00144
Flr	0.00218	0.00249	0.00268	0.00218	0.00274	0.00246
Ant	0.00362	0.00150	0.00037	0.00062	0.00106	0.00143
Fit	0.00087	0.00150	0.00362	0.00044	0.00019	0.00132
Pyr	0.00125	0.00166	0.00033	0.00017	0.00632	0.00194
HI_i	0.01415	0.01363	0.01153	0.00906	0.01953	0.01358

Table S2: incremental life cancer risks (ILCR) for the different communities

PAH	Incremental life cancer risk due to dermal contact (ILCRd) for Aldult					
	A	B	C	D	E	mean
BaA	3.59E-05	8.64E-05	4.60E-05	7.35E-05	4.87E-05	5.81E-05
Chr	4.14E-06	1.96E-05	3.59E-06	5.74E-06	6.62E-06	7.93E-06
BbF	1.56E-04	2.00E-04	1.65E-04	2.65E-04	2.55E-04	2.08E-04
BkF	4.78E-05	4.85E-04	2.81E-04	4.50E-04	3.60E-04	3.25E-04
BaP	5.13E-03	8.54E-03	2.16E-03	3.46E-03	3.88E-03	4.63E-03
DhA	6.42E-03	6.63E-03	1.95E-03	3.12E-03	4.82E-03	4.59E-03
IcP	1.47E-04	2.57E-04	4.17E-04	6.68E-04	2.80E-04	3.54E-04
∑ILCRd	0.0119	0.0162	0.0050	0.0080	0.0097	0.0102
Incremental life cancer risk due to dermal contact (ILCRd) for Child						
BaA	0.00016	0.00021	0.00051	0.00027	0.00028	0.00029
Chr	0.00002	0.00002	0.00012	0.00002	0.00002	0.00004
BbF	0.00156	0.00092	0.00118	0.00098	0.00289	0.00150
BkF	0.00344	0.00028	0.00286	0.00166	0.00238	0.00213
BaP	0.01215	0.03027	0.05040	0.01275	0.00879	0.02287
DhA	0.00363	0.03786	0.03911	0.01150	0.05023	0.02847
IcP	0.00164	0.00087	0.00151	0.00246	0.00179	0.00165
∑ILCRd	0.0226	0.0704	0.0957	0.0296	0.0664	0.0569
Incremental life cancer risk due to ingestions (ILCRi) for Adults						
BaA	1.65E-05	2.15E-05	5.17E-05	2.75E-05	2.86E-05	2.92E-05
Chr	1.82E-06	2.48E-06	1.17E-05	2.15E-06	1.65E-06	3.96E-06
BbF	1.59E-04	9.36E-05	1.19E-04	9.91E-05	2.93E-04	1.53E-04
BkF	3.49E-04	2.86E-05	2.91E-04	1.68E-04	2.41E-04	2.16E-04
BaP	1.23E-03	3.07E-03	5.11E-03	1.29E-03	8.92E-04	2.32E-03
DhA	3.69E-04	3.84E-03	3.97E-03	1.17E-03	5.10E-03	2.89E-03
IcP	1.66E-04	8.81E-05	1.54E-04	2.50E-04	1.81E-04	1.68E-04
∑ILCRi	0.0023	0.0071	0.0097	0.0030	0.0067	0.0058
Incremental life cancer risk due to ingestions (ILCRi) for Children						
BaA	4.81E-06	6.25E-06	1.51E-05	8.01E-06	8.33E-06	8.49E-06
Chr	5.29E-07	7.21E-07	3.41E-06	6.25E-07	4.81E-07	1.15E-06
BbF	4.62E-05	2.72E-05	3.48E-05	2.88E-05	8.53E-05	4.45E-05
BkF	1.02E-04	8.33E-06	8.46E-05	4.90E-05	7.02E-05	6.28E-05
BaP	3.59E-04	8.94E-04	1.49E-03	3.77E-04	2.60E-04	6.76E-04
DhA	1.07E-04	1.12E-03	1.16E-03	3.40E-04	1.48E-03	8.41E-04
IcP	4.84E-05	2.56E-05	4.47E-05	7.28E-05	5.28E-05	4.89E-05
∑ILCRi	0.00067	0.00208	0.00283	0.00088	0.00196	0.00168