Original Research Article

POLLUTION STATUS AND HEALTH RISK ASSESSMENT OF POLYCYCLIC AROMATIC HYDROCARBONS IN SURFACE WATER, SEDIMENT AND FISH FROM EZU-RIVER, ANAKU, ANAMBRA STATE, NIGERIA.

UNDER PEER REVIEW

ABSTRACT

The distribution of the sixteen polycyclic aromatic hydrocarbons (PAHs) was studied in surface water, sediment and three fish species (African catfish (*Clarias gariepinus*), Trout fish (*Mormyrus rume*) and *Hetrobranchus longefilis)* from Ezu-river, Anaku, Anambra State, Nigeria. The samples were analysed for PAHs by means of Gas chromatography-mass spectrometry. The results of PAHs showed that, in surface water, the highest concentration was related to benzo(a)pyrene whereas benzo(k)fluoranthene was the most important pollutant in sediment. For the fish samples, *Hetrobranchus longefilis* recorded the highest concentration in Naphthalene while Anthracene was the most dominant pollutants in *Mormyrus rume* and in *Clarias gariepinus*, benzo(b)fluoranthene was the highest pollutant. The data generated were subjected to statistical analysis, the Correlation matrices showed positive values showing that the PAHs in the samples were from a similar source with migration route and vice versa for negative correlation. The Health and exposure risk assessment was conducted for carcinogenic and non-carcinogenic exposure in adults and children, where the cumulative cancer risk and hazard index were within USEPA regulatory standard. This makes all sample sources fit for consumption, as extreme care must be taken into consideration for children exposure as compared to adults.

1.0 INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are a large group of organic compounds with two or more fused aromatic rings in linear, angular or cluster arrangements (Yu *et al.*, 2019). It is made up of carbon and hydrogen atoms that range from naphthalene ($C_{10}H_8$, two rings) to coronene ($C_{24}H_{12}$, seven rings) with molecular masses ranging from 128 to 278 Da. They are formed during the incomplete combustion or high pressure processes of coal, oil gas, wood, garbage, or other organic substances. PAHs are widely distributed in water, soil sediment and air

(Omokpariola *et al.*, 2021; Gong, *et al.*, 2011). Upon entry into the aquatic environment, it either mixes with water or sinks into the sediment, causing severe damage to benthic organisms. Hydrocarbon pollution affects the fishes in the water; it causes an objectionable odour and flavour, thereby reducing their market value and acceptability (Farrington, 2014). These fishes are exposed to PAHs through ingestion of contaminated food and by diffusion of water across their gills and skin.(Arnot and Quinn, 2015) PAHs have a relatively low solubility in water, but are highly lipophilic, they are mostly colourless, white, or pale yellow solids. Due to their low water solubility, PAHs are easily absorbed by particles and colloids when transferred into the water and sediment. (Kumar *et al*., 2017). They generally have low vapour pressure and are globally distributed in atmospheric, terrestrial and aquatic systems (Kumar *et al*., 2014).

Polycyclic aromatic hydrocarbons are classified into two main groups: Low molecular weight (LMW) polycyclic aromatic hydrocarbons and High molecular weight (HMW) polycyclic aromatic hydrocarbons. This is based on their physical and biological properties and also number of fused aromatic rings contained in their structure. LMW PAHs such as naphthalene, acenaphthene, acenaphthylene, fluorene,anthracene, phenanthreneetc tend to have a core structure of two to three benzenoid rings (six-sided aromatic rings of carbon). They are usually related to naturally occurring PAHs, either of petrogenic or pyrogenic origin. HMW PAHs have molecular structures of four or more benzenoid rings (e.g. fluoranthene, pyrene, benzo[a]pyrene, and benzofluoranthenes).They are emitted from combustion processes (Lee, 2010; Abou-Arab, 2014). The HMW PAHs are more persistent and recalcitrant (less readily bio-degraded by indigenous microorganisms) than LMW PAHs. They can persist in an aqueous environment and bioaccumulate in aquatic organisms like fish and shrimps and are more carcinogenic (Olayinka, 2019). Although, the LMW PAHs are less carcinogenic, they can also pose toxic risks to many aquatic organisms. (Brown and Peake, 2006). Polycyclic aromatic hydrocarbons (PAHs) and heavy metals have been known to be environmental contaminants for decades and several monitoring programmes have been conducted to estimate the pollution of sediment, water, biota and air by PAHs and heavy metals.

The Ezu-river is located in Anaku, Anambra State between Latitude: 6° $21^{'}$ 40" N and Logitude: 6° 51' 38" N in Ayamelum Local Government Area. It is bordered by "Omambala", the native name of Anambra River. It is mostly dominated by the Igbos and occupations in the community

UNDER PEER REVIEW

are predominantly fishing, farming and hunting. The aim of this work is to determine the level of polycyclic aromatic hydrocarbons in water, sediment and fish samples and their associated risk value in Ezu-river, Anaku, Anambra state.

2.0 MATERIALS AND METHODS

2.1 Chemicals, Reagents and Equipment

All solvents used for this study were of analytical grade and were purchased from Sigma-Aldrich Co. USA. Sodium sulphate, Hexane, Dichloromethane, silica gel and standard containing the US EPA 16 priority PAHs 2000µg/ml. The GCMS system consist of an agilent 6890 gas chromatograph equipped with auto sampler connected to an agilent 5973N mass selective detector, Rotary evaporator, sonicator.

2.2 Study area

The Ezu-river is located in Anaku, Anambra State between Latitude: 6° 21' 40" N and Logitude: 6° 51' 38" N in Ayamelum Local Government Area. It is bordered by "Omambala", the native name of Anambra River. It is mostly dominated by the Igbos. The occupations in the community are predominantly fishing, farming and hunting.

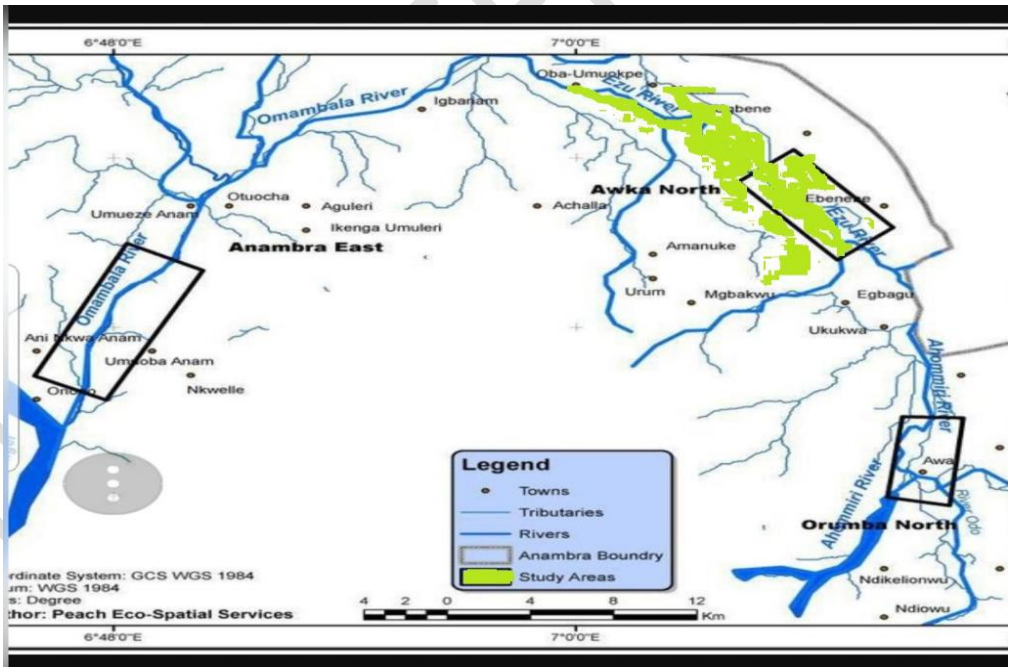

Figure 1: Map showing the location of Ezu-River, Anaku, Anambra State, Nigeria.

2.3 Sample Collection and pre-treatment

2.3.1 Surface water sample: Water samples (2.5L) were collected in a clean glass bottles at the water surface and 50cm below water level from four different locations and homogenized to get a composite sample. The bottle was tightly capped and immediately transported to the laboratory for analysis.

2.3.2 Sediment sample: 2kg of surface sediment samples were collected with a clean bottle in four different locations in the water at 5cm depth and homogenized into a composite sample. It was stored in labeled polythene bags and taken to the laboratory for analysis. The Sediment samples were air dried for 2 days, after which stones and debris were removed from the samples and then pulverized and passed through a 2mm mesh sieve to remove other unwanted materials.

2.3.3 Fish sample: A sample of three fish species namely: African catfish (*Clarias gariepinus*), Trout fish (*Mormyrus rume*) and *Hetrobranchus longefilis* samples were purchased from local fishermen at sampling locations. All samples were weighed (g), washed with distilled water then wrapped in aluminum foil and transported immediately to the laboratory on ice packs. The fish samples were allowed to thaw; the scales were removed using knife and washed with distilled water. The tissues were dissected into smaller parts and were put in the sample bottles. The samples were weighed using an analytical weighing balance (wet weight) and homogenized with anhydrous sodium sulphate in a mortar with pestle. The mixture was labeled and was left till the next day to cake, prior to extraction.

2.4 Analytical procedures

Analytical procedures for PAHs used in this study have been described previously (USEPA, 2016; USEPA, 1986).

2.4.1 Surface water: 100ml of surface water sample was measured into a clean separating funnel and 50ml of 1:1 Hexane-Acetone mix solvent was added. The separating funnel was sealed and shaken for 2 minutes with periodic venting to release the inbuilt pressure. The mixture was allowed to stand for 10minutes for separation into distinct layers. The organic layer (i.e the upper layer) was collected in a round bottom flask. The extraction procedure was repeated until all the oil is extracted and concentrated to 2ml using Rotary evaporator. The concentrated sample was transferred into the fractionating column and eluted with 10ml dichloromethane into a flat bottom flask. The eluates was transferred into a round bottom flask and pipetted into a Teflon screw-cap vial and analyzed for PAH using the Gas Chromatography-mass spectrometer.

UNDER PEER REVIEW

2.4.2 Sediment sample: 10grams of sediment sample was weighed and homogenized in a mortar with 10grams of anhydrous sodium sulphate until a completely dried homogenate was obtained. 20ml of dichloromethane was added to the dried homogenate sediment samples inside a 100ml beaker and then placed in the sonicator for about 15minutes at about 70^{o}C. (Note this was done in triplicates to extract all analyte present in the sample). After sonication, 10g of anhydrous sodium sulphate was added to the sample to remove any residual water molecules. This was allowed to stand for about 15minutes. The extracts were then transferred into a round bottom flask and then concentrated at about 2ml using a rotary evaporator. 1.5ml of the concentrated sample was pipetted into the already conditioned column and eluted with 15ml of dichloromethane. The eluate was collected in a solvent rinsed round bottom flask and then concentrated to 1.5ml. The concentrated sample was pipette into a clean GC vial bottle and capped tightly. The sample was then injected into the GC for PAH analysis using the Gas Chromatography Agilent 6890 model.

2.4.3 Fish samples: The whole samples of biota were analyzed for PAHs. Analytical procedures for PAHs used in this study are described in detail previously (US EPA, 1986).

5g of fish samples that had been previously homogenized with anhydrous sodium sulphate were poured into 100ml beakers and 40ml of n-hexane and dichloromethane (1:1 vol/vol) was used as an extracting solvent. The Samples were homogenized for 25 minutes at 100 rpm and mixed further with 5g of anhydrous sodium sulphate. The extract was decanted into a clean conical flask, then 20ml of fresh solvent was added, and the process repeated. It was filtered through a small glass funnel containing a layer of anhydrous sodium sulphate over a plug of glass wool into a receiving conical flask. The resulting solvent was eluted with 50 ml n-hexane solvent and evaporated again. The eluates were then concentrated to 1ml using a rotary evaporator under a gentle stream of pure nitrogen. Determination of PAHs in the fish samples was carried out following standard procedures using GCMS (Agilent 6890 gas chromatograph equipped with auto sampler connected to an agilent 5973N mass detector).

2.5 Data Analysis: Microsoft Excel 2019 data analysis was utilized for determination of correlation matrix, which evaluates the strength and direction of a linear relationship between two variables (PAHs). Correlation coefficient of value greater than 0.71 is accepted for correlation matrix at significance level of 0.05. (Thepanodh *et al.*, 2005).

UNDER PEER REVIEW

2.6 Human Health Risk Assessment: Human health risk assessment was carried out to estimate the probability of adverse health effects in humans as a result of exposure to PAHs through contact with the sediment and consumption of contaminated water and fish in the studied river. Cancer risk (CR) and Hazard Quotient (HQ) are indices developed USEPA risk assessment models for evaluation of carcinogenic and non-carcinogenic health risk in adults and children. All calculations were done based on USEPA standards (USEPA 1991; USEPA 2020; USEPA, 1996).

2.6.1 Chronic daily intake (CDI) (mg/kg/day) of PAHs in sediment sample

$$CDI-ingestion = \left(\frac{CS \times IRs \times EF \times ED \times RBA \times TR}{BW \times AT}\right) (1)$$

$$CDI-dermal = \left(\frac{CS \times SA \times K_p \times EF \times AF \times ED \times TR}{BW \times AT \times GIABS}\right) (2)$$

Where CS is PAHs concentration in the sediment (mg/kg), IRs is sediment ingestion rate (mg/day) (100mg/day for adults and 200mg/day for children), EF is exposure frequency (350-day year^{-1}), ED is exposure duration (26 years for adults and 6 years for children), TR is target risk (1×10^{-6} mg/mg), BW is body weight (80kg for adults and 15kg for children), AT is average time (non-carcinogens = ED×365 days), (carcinogen =70×365), SA is skin surface area (6032cm^2/day for adults and 2373 cm^2/day for children), Kp: dermal permeability constant (0.001); AF is water adherence factor: (0.2mgcm^{-2} for adults and 0.07mgcm^{-2} for children), GIABS is fraction of contaminant absorbed in gastrointestinal tracts (unit-less) (1.0 for adults and children)

2.6.2 Chronic daily intake (CDI) (mg/kg/day) of PAHs in surface water

$$CDI-ingestion = \left(\frac{CS \times IR_W \times EF \times ED \times TF}{BW \times AT}\right) \quad (3)$$

$$CDI-dermal = \left(\frac{CS \times SA \times ET_W \times EF \times AF \times ED \times TF}{BW \times AT}\right) \quad (4)$$

UNDER PEER REVIEW

Where CS is PAHS concentration in water (mg/L), IR_w is daily water ingestion rate (L/day) (2.5L/day for adults and 0.78L/day for children), EF is exposure frequency (350-day $year^{-1}$), ED is exposure duration (26 years for adults and 6 years for children), TR is target risk (1×10^{-6} mg/mg) for carcinogen**,** BW is body weight (80kg for adults and 15kg for children), AT is average time (non-carcinogens = ED×365 days), (carcinogen =70×365), SA is skin surface area (19652cm^2 for adults and 6365cm^2 for children), AF is water adherence: (0.2$mgcm^{-2}$ for adults and children), ABS_{sk} is fraction of chemical absorbed through the skin (unit-less) (0.001 for adults and children) and ET_w is exposure time during work event (1h/event for adults and children)(USEPA 2015; USEPA 2017; USEPA 2020).

2.6.3 Chronic daily intake (CDI) (mg/kg/day) of PAHs in fish

The CDI (mg/kg/day) of PAHs were calculated with equation 5.

$$\text{CDI}-\text{Fish ingestion} = \left(\frac{\text{CS} \times \text{IR}_F \times \text{EF} \times \text{ED} \times \text{TF}}{\text{BW} \times \text{AT}}\right) \quad (5)$$

Where: CS is concentration of PAHs in mg/kg, IRF is food ingestion rate 0.0548 kg/capital/day, EF is exposure frequency (350-day $year^{-1}$), ED is exposure duration (26 years for adults and 6 years for children), TR is target risk (1×10^{-6} mg/mg) for carcinogen**,** BW is body weight (80kg for adults and 15kg for children), AT is average time (non-carcinogens = ED×365 days), (carcinogen =70×365).

2.7 Assessment of non-carcinogenic health risks

Assessment of non-carcinogenic health risks was achieved by estimating the hazard quotient (HQ). For non-carcinogenic risks from exposure to PAHs, the HQ was calculated as the quotient between the CDI against the reference dose (RfD) (Equation 6)

$$Hazard\ Quotient, HQ = \frac{CDI}{RfD} \quad (6)$$

$$Hazard\ index, HI = \sum_{i=1}^{n} HQi = HQ_{ingestion} + HQ_{dermal} \quad (7)$$

UNDER PEER REVIEW

The hazard index, which estimates the total risk from multiple contaminant pathways, was obtained by summing the HQ of each contaminant pathway

2.8 Carcinogenic Risk Assessment

Carcinogenic risk assessment was determined using CDI of dermal and ingestion as shown below (USEPA 1992; USEPA 2015; USEPA 2020):

$$Cancer\ risk = CDI \times SF \qquad (8)$$

$$\text{Risk}_{total} = \text{Risk}_{der} + \text{Risk}_{ing}$$

$$= ([\text{CDI(der)} \times \text{CSF}] + [\text{CDI(Ing)} \times \text{OSF}]) \qquad (9)$$

Where Risk is a unit-less probability of an individual developing cancer over a lifetime, ADI (E) is average daily intake (exposure), CSF is Cancer slope factor of PAHs and heavy metals (mg/kg/day), Risk $_{total}$ is the total excess lifetime cancer calculated from risk pathway.

Table 1: Reference value for polycyclic aromatic hydrocarbons (PAHs)

TPAHs	**Dermal**		**Ingestion**	
	CSF	RfD	OSF	RfD
Naphthalene (Nap)	NA	0.02**	NA	0.04
Acenaphthene (Acy)	0.073*	0.02**	0.073*	0.006
Acenaphthylene (Ace)	0.0073*	0.06**	0.0073*	0.06
Fluorene (Flu)	NA	0.04**	NA	0.04
Phenanthrene (PA)	NA	NA	NA	0.04
Anthracene (Ant)	NA	0.3**	NA	0.3
Fluoranthene (Flt)	0.073*	0.04**	0.073*	0.04
Pyrene (Py)	0.73*	0.03**	0.73*	0.03
Benzo[a]anthracene (BaA)	0.73*	0.03**	0.73*	0.03
Chrysene (Cry)	0.0073*	0.03**	0.0073*	0.03
Benzo[b]fluoranthene (BbF)	0.73*	0.03**	0.73*	0.03

UNDER PEER REVIEW

Benzo[k]fluoranthene (BkF)	0.0073*	0.03**	0.0073*	0.03
Benzo[a]pyrene (BaP)	7.3*	0.03**	7.3*	0.03
Dibenzo[a,h]anthracene (DBA)	7.3*	0.03**	7.3*	0.03
Indeno [1,2,3-cd] pyrene (IND)	0.73*	0.03**	0.73**	0.03
Benzo[ghi]perylene (BghiP)	0.073*	0.03**	0.073*	0.03
Total PAHs	7.3*	0.03**	7.3*	0.03

Where: *(USEPA, 2005a; USEPA, 2005b), **(USEPA, CEPA, Verbruggen, 2012). CSF: cancer slope factor (mg/kg/day), OSF: oral slope factor (mg/kg/day), RfD: reference dose

3.0: RESULTS AND DISCUSSION

3.1 Concentration of polycyclic aromatic hydrocarbons in surface water, sediment and fish samples.

Table 2 and Figure 2 depicts the mean concentration of polycyclic aromatic hydrocarbons (PAHs) determined from different samples (surface water, sediment, *Hetrobranchus longefillis, Mormyrus rume, Clarias gariepinus*) in Ezu-River, Anaku, Anambra state, Nigeria. Surface water showed that the 16 priority PAHs was below detection limit, BDL (<0.001mg/l) except for BbF (0.02mg/l) and BaP (0.023mg/l), as regards to sediment, Nap, Acy, Flu, Flt, Py, Chy were 0.01mg/kg respectively, while in decreasing mean PAHs concentration was BkF (0.147 mg/kg) > Ant (0.08 mg/kg) > IND (0.033 mg/kg) >BghiP (0.03mg/kg) >BbF (0.027 mg/kg) >Phen; Ace (0.02 mg/kg) > DBA (0.017 mg/kg) >BaA; BaP (0.013mg/kg).*Hetrobranchus longefillis* mean assessment of PAHs indicated that Nap was highest at 2.807 mg/kg as compared to Flt, Chy, BkF, IND, DBA and BghiP, which were BDL respectively. The mean PAHs concentrations in Mormyrus rume were present in Nap, Acy, Ace, Flu, Ant, Flt, BbF and BaP individually ranging between 0.01 – 0.047 mg/kg. For *Clarias gariepinus*, PAHs ranged between BDL (<0.001mg/kg) to 0.017 mg/kg. BbF and BaP were present across all samples. The concentration of BaP across all samples exceeded the EU recommended safe limit of 0.002 mg/kg for human fish consumption (EU, 2006). As one can see, high molecular weight (HMW) PAHs displayed high cumulativex concentration in surface water, sediment and *Clarias gariepinus* and vice versa (lower molecular weight, LMW PAHs) for *Hetrobranchus longefillis* and *Mormyrus rume*, which is due to bioaccumulation and biological distribution pattern of PAHs across different

UNDER PEER REVIEW

sample source (Tella *et al.,* 2017). LMW PAHs is a conglomeration of carbon rings, C–2 to C–3, which implies that all sample source had LMW ranged between 0.03 – 2.90 except for surface water that was below detection level (<0.001 mg/l) by GC instrument. High molecular weight (HMW) is aggregate of aromatic carbon ring, C–4 to C–6, which depicts that sediment was highest as compared to *Clarias gariepinus*. As shown in Table 4.3, the cumulative sum of carcinogenic PAHs (cPAHs) in decreasing order: Sediment (0.30 mg/kg)>*Hetrobranchus longefillis* (0.053 mg/kg) >*Mormyrus rume*(mg/kg) and water (0.043 mg/l) >*Clarias gariepinus* (0.03 mg/kg).

Table 2: PAHs concentration of surface water, sediment and three fish species.

PAHs	**Surface water**	**Sediment**	***Hetrobranchusl ongefillis***	***Mormyrus-rume***	***Clarias gariepinus***
Naphthalene (Nap)	<0.001±0	0.01±0	2.807±0.021	0.03±.010	0.01±0
Acenaphthylene (Acy)	0±0	0.01±0	0.033±0.006	0.027±0.006	0.01±0
Acenaphthene (Ace)	<0.001±0	0.02±0.01	0.02±0	0.01±0	0.01±0
Fluorene (Flu)	<0.001±0	0.01±0	0.02±0	0.013±0.006	<0.001±0
Anthracene (Ant)	<0.001±0	0.08±0.017	0.01±0	0.047±0.006	<0.001±0
Phenanthrene (Phen)	<0.001±0	0.02±0.01	0.01±0	<0.001±0	<0.001±0
Fluoranthene (Flt)	0±0	0.01±0	<0.001±0	0.01±0	<0.001±0
Pyrene (Py)	<0.001±0	0.01±0	0.013±0.006	<0.001±0	<0.001±0
*Benz[a]anthracene (BaA)	<0.001±0	0.013±0.006	0.013±0.006	<0.001±0	<0.001±0
*Chrysene (Chy)	<0.001±0	0.01±0	<0.001±0	<0.001±0	<0.001±0
*Benzo[b]fluoranthene (BbF)	0.02±0.001	0.027±0.015	0.01±0	0.02±0	0.017±0
*Benzo[k]fluoranthene (BkF)	<0.001±0	0.147±0.021	<0.001±0	<0.001±0	0.01±0
*Benzo[a]pyrene (BaP)	0.023±0.006	0.013±0.006	0.03±0	0.023±0.006	0.01±0
*Indeno(1,2,3-cd)perylene (IND)	<0.001±0	0.033±0.006	<0.001±0	0±0	0±0
*Dibenz[a,h]anthracene (DBA)	<0.001±0	0.017±0.006	<0.001±0	<0.001±0	<0.001±0
*Benzo[ghi]perylene (BghiP)	<0.001±0	0.03±0	<0.001±0	0±0	0±0
LMW	0.00	0.143	2.90	0.127	0.03
HMW	0.043	0.290	0.067	0.053	0.03
ΣcPAHs	0.043	0.2833	0.053	0.043	0.03
Total	0.043	0.4333	2.967	0.180	0.06

UNDER PEER REVIEW

Values presented as mean ± standard deviation; <0.001 = below detection limits (BDL); *PAHs: carcinogenic PAHs; LMW: sum total of Nap – Phen; HMW: sum total of Flt – BghiP.

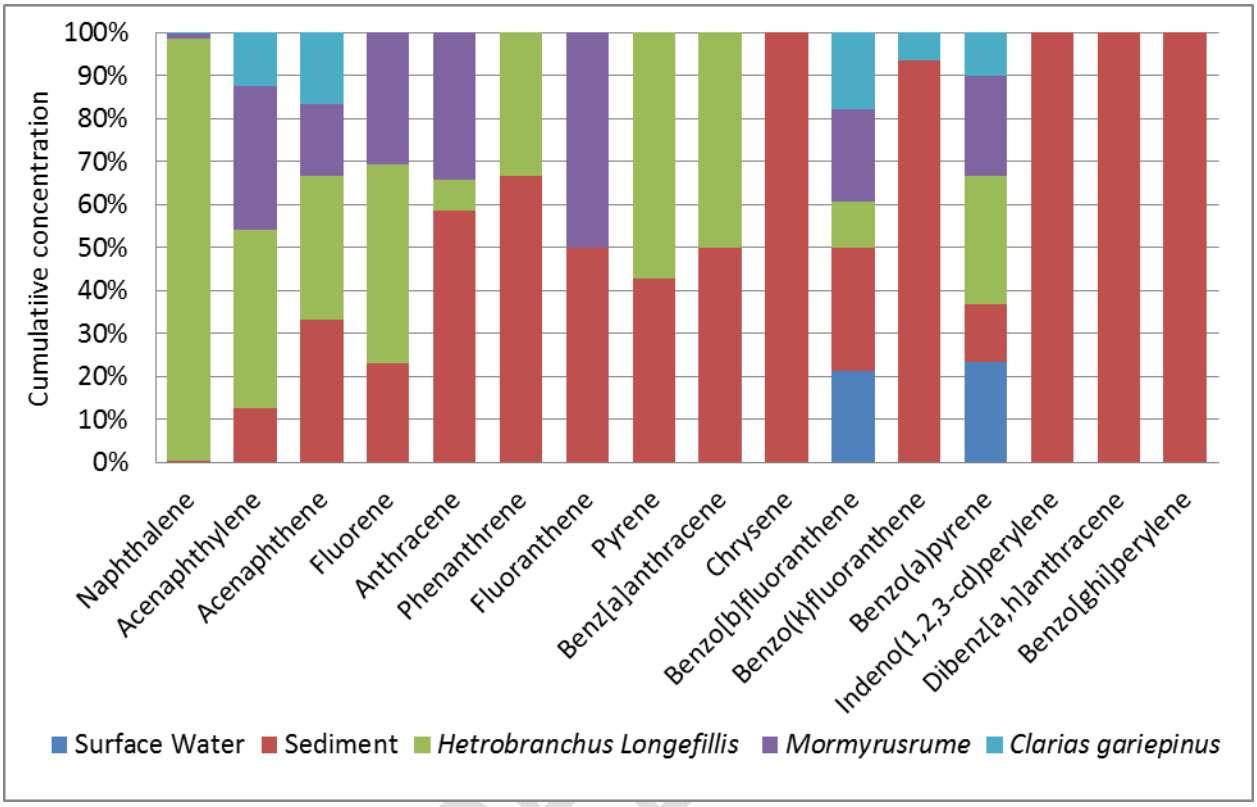

Figure 2: Percentage stark column of PAHs in analyzed samples

3.2 Correlation matrices of surface water, sediment and fish samples

Pearson correlation was conducted to get important information about relationship of PAHs constituents of fish samples in tandem with surface water as shown in Table 3.1,3.2 and 3.3 respectively. As we can see in Table 3.1, *Hetrobranchus longefilis* and surface water correlation produced strong correlation grade (>0.75) between 0.877 – 1.00, as such Nap, Acy, Flu, Ant and Phen correlated strongly with all 14 PAHs except Flt, Chy, BbF, BkF, BaP, IND, DBA and BghiP, which were absent (0.00) or moderate correlation (0.75 to 0.50). For sediment and *Hetrobranchus longefilis*, there was predominantly negative correlation across the 14 PAHs components, which implies that sediment contributed least in relation to biological assimilation; as such one can therefore state that PAHs transfer was predominant in surface water than sediment. A cursory review of Table 3.2 in correlative potential of surface water and sediment with *Mormyrus rume*, showed that PAHs correlation in surface water was predominantly absent

due to instrumental detection limit, as one cannot entirely rule out that PAHs could be present below <0.001 mg/kg in *Mormyrus rume* (Trout fish). As regards sediment, there was dominant strong correlation (having positive and negative regression) across all PAHs components, as positive values show that PAHs are from similar or mutual source and response mode, while negative is associated to different contaminating source and biological interaction taken place no associated to surface water and sediment samples (Ojaniyi *et al.*, (2021).

Table 3.3 correlation of *Clarias gariepinus* in relation with surface water and sediment showed predominantly weak (0.35 – 0.00) and strong correlation (1.00 – 0.75), which was due to similar polluting sources and biotransformation across all PAHs component in tandem to physiochemical interaction in fish samples.

UNDER PEER REVIEW

Table 3.1: Correlation matrices between *Hetrobranchus longefilis* in relation to surface water and sediment

		ap	cy	ce	u	nt	ien	t	r	aA	ay	oF	kF	aP	ID	BA	ghiP	
Surface water	Nap		959	5E-17	936	.962	.655	.447	718	.004	.447	.687	.987	928	.979	.92	.999	Sediment and
and	Acy	978		4E-17	90	.925	.629	.43	834	194	.43	.66	.949	393	.942	.88	.962	*Hetrobranchus*
Hetrobranchus	Ace	999	979		.304	238	378	.775	401	.612	.775	714	1564	322	.7E-17	.34	.4E-17	*longefillis*
longefillis	Flu	999	979	00		.949	.843	.105	594	248	.105	.884	.963	304	.952	.79	.936	
	Ant	999	979	00	00		529	307	.566	.097	307	311	992	.79	914	777	962	
	Phen	999	979	00	00	00		.293	.472	.463	.293	309	575	.608	773	502	554	
	Flt	447	456	447	447	447	447		.529	533	00	.307	340	.581	348	587	447	
	Py	877	957	879	879	879	879	393		20	.529	.241	.638	3416	.753	.84	.721	
	BaA	877	957	879	879	879	879	393	00		533	.486	.08	.131	.071	138	00	
	Chy	00	00	00	00	00	00	00	00	00		.307	34	.581	348	587	447	
	BbF	.654	.628	.655	.654	.654	.655	293	.576	.576	00		783	.434	7	417	587	
	BkF	00	00	00	00	00	00	00	00	00	00	00		.857	957	346	987	
	BaP	707	599	707	707	707	707	532	622	522	00	14E-17	00		.947	.00	.928	
	IND	00	00	00	00	00	00	00	00	00	00	00	00	00		941	979	
	DBA	00	00	00	00	00	00	00	00	00	00	00	00	00	00		92	
	BghiP	00	00	00	00	00	00	00	00	00	00	00	00	00	00	00		

UNDER PEER REVIEW

Table 3.2: Correlation matrices between *Mormyrus rume* in relation to surface water and sediment

		Nap	Acy	Ace	Flu	Ant	Phen	Flt	Py	BaA	Chy	BbF	BkF	BaP	IND	DBA	BghiP	
Surface water and *Mormyrus-rume*	Nap		.643	0.567	.847	0.732	0.74	.612	0.39	0.762	0.387	0.306	0.854	.84	0.84	0.8	0.86	Sediment and *Mormyrus-rume*
	Acy	.82		0.608	.478	0.816	0.79	.657	0.42	0.817	0.415	0.328	0.916	.495	0.917	0.85	0.94	
	Ace	.929	.968		0.775	.885	.756	0.926	.878	.265	0.293	.926	.7642	0.159	.642	.346	.654	
	Flu	.97	.734	.88		0.683	0.82	.837	0.53	0.414	.092	0.665	0.764	.662	0.742	0.57	0.71	
	Ant	.919	.94	.99	.905		.722	0.777	.832	.623	.151	.657	.914	0.328	.78	.579	.847	
	Phen	.00	.00	.00	.00	.00		0.922	.382	.538	0.02	.62	.8635	0.622	.927	.786	.854	
	Flt	.927	.968	.997	.877	.987	.00		0.63	0.286	.316	0.875	0.777	.343	0.763	0.51	0.71	
	Py	.00	.00	.00	.00	.00	.00	.00		.181	0.2	.791	.5826	.108	.35	.061	.447	
	BaA	.00	.00	.00	.00	.00	.00	.00	.00		.819	0.109	.8143	0.755	.815	.903	.879	
	Chy	.00	.00	.00	.00	.00	.00	.00	.00	.00		0.632	.3403	0.542	.35	.587	.447	
	BbF	.0E-17	.022	.4E-17	.00	.4E-17	.00	.062	.00	.00	.00		.4845	.086	.381	.048	.353	
	BkF	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00		0.641	.958	.846	.986	
	BaP	.227	0.17	.00	.336	.0511	.00	.025	.00	.00	.00	.612	.00		0.751	0.86	0.72	
	IND	0.43	0.86	0.707	0.286	0.648	.00	0.705	.00	.00	.00	2.6E-17	.00	.50		.941	.98	
	DBA	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00	.00		.92	
	BghiP	0.43	0.86	0.707	0.286	0.649	.00	0.705	.00	.00	.00	2.6E-17	.00	.50	.00	.00		

UNDER PEER REVIEW

Table 3.3: Correlation matrices between *Clarias gariepinus* in relation to surface water and sediment

		ap	cy	ce	u	nt	ıen	t	r	aA	ıy	oF	kF	aP	lD	BA	ghiP	
Surface water and *Clarias gariepinus*	Nap		00	00	00	00	00	00	00	00	00	00	00	00	00	00	00	Sediment and *Clarias gariepinus*
	Acy	997		00	00	00	00	00	00	00	00	00	00	00	00	00	00	
	Ace	00	997		.293	795	756	.29	378	265	.29	809	771	309	542	346	554	
	Flu	00	00	00		324	.02	00	.2	819	00	.43	333	092	35	586	447	
	Ant	00	00	00	00		328	324	553	794	324	534	996	325	926	796	969	
	Phen	00	00	00	00	00		.02	382	538	.02	599	363	516	927	786	354	
	Flt	447	51	4472	00	00	00	.2	819	00	.43	333	092	35	587	447		
	Py	00	00	00	00	00	00	00		181	.20	676	591	783	35	061	447	
	BaA	00	00	00	00	00	00	00	00		819	036	309	512	315	903	379	
	Chy	00	00	00	00	00	00	00	00	00		.43	333	092	35	587	447	
	BbF	.19	.146	.186	00	00	00	415	00	00	00		518	335	442	164	424	
	BkF	00	997	00	00	00	00	447	00	00	00	.186		797	956	840	985	
	BaP	.91	.894	.907	00	00	00	.266	00	00	00	552	.907		565	462	731	
	IND	.71	.705	.707	00	00	00	.316	00	00	00	657	.707	341		941	98	
	DBA	00	00	00	00	00	00	00	00	00	00	00	00	00	00		92	
	BghiP	.71	.705	.707	00	00	00	.316	00	00	00	657	.707	341	00	00		

UNDER PEER REVIEW

3.3 Health and exposure risk assessment of polycyclic aromatic hydrocarbons

Table 4.1 shows the carcinogenic risk assessment conducted on PAHs samples in Ezu-River, Anaku, Anambra state using USEPA risk formulas as regards different exposure patterns measured in mg/kg/day. The cumulative PAHs for both adults and children are: surface water – oral (5.25E-10; 9.47E-10), surface water – dermal (4.24E-07; 4.19E-07), sediment – accidental ingestion (2.52E-07; 5.37E-07), sediment – dermal (1.06E-09; 1.27E-09), *H. longefillis* (8.39E-10; 1.79E-09), *Mormyrus rume* (5.63E-11; 1.20E-10) and *C. gariepinus* (2.25E-11; 4.80E-11). A review shows that some PAHs analytes were absent that influenced the cumulative PAHs values across all samples and exposure medium.

Table 4.2 depicts the non-carcinogenic CDI evaluation of PAHs across different samples(surface water, sediment and three fishes), as the cumulative non-carcinogenic CDI for both adults and children exposure are surface water – oral (1.23E-09; 1.12E-08), surface water – dermal (9.89E-07; 4.95E-06), sediment – accidental ingestion (5.87E-07; 1.25E-06), sediment – dermal (2.48E-09; 1.51E-08), *H. longefillis* (1.96E-09; 2.12E-08), *Mormyrus rume* (1.31E-10;1.42E-09) and *C. gariepinus*(5.25E-11;5.68E-10).

The cumulative PAHs CDI influence of adults to children was evaluated using similar model in eq. 10 to assess carcinogenic and non-carcinogenic PAHs, as shown in Figure 3. The results are: surface water – oral (55.4%; 11.0%), surface water – dermal (101%; 20%), sediment accidental ingestion (46.9%; 47.0%), sediment dermal (83.5%; 16.4%), *H.Longefillis*(46.9%; 9.25%); *Mormyrus rume* (46.9%; 9.23%) and *C.gariepinus* (46.9%; 9.24%), as such this shows that surface water – dermal exposure was dominant, while *H. Longefillis*, *Mormyrus rume* and *C. gariepinus* were least across all samples due to PAHs concentration for adults to children CDI evaluations respectively.

UNDER PEER REVIEW

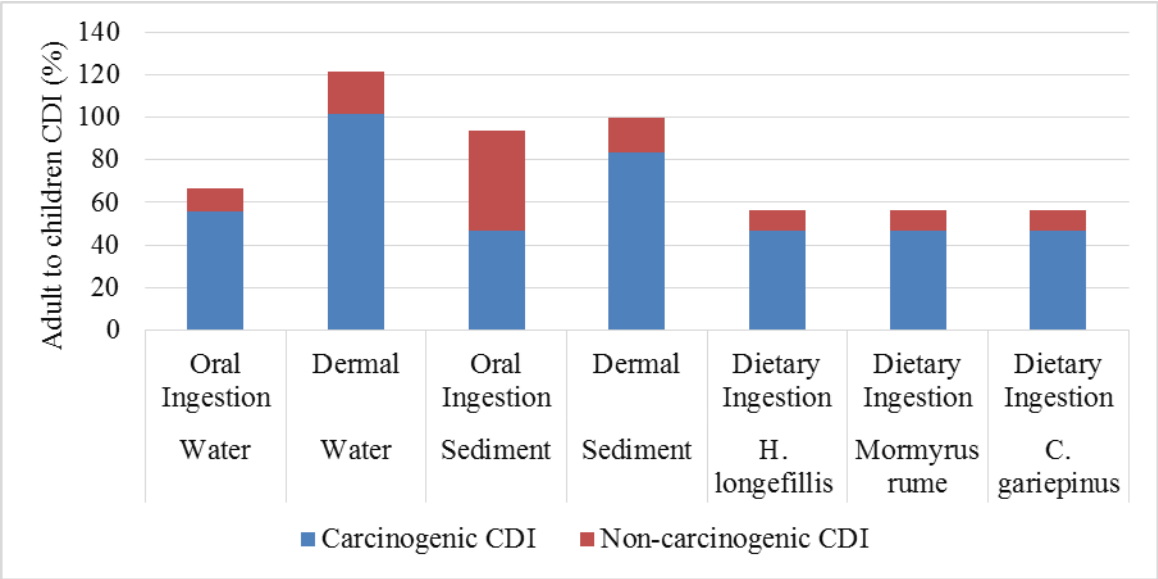

Figure 3: Cumulative CDI influence of adults to children

Table 4.1: Carcinogenic CDI of polycyclic aromatic hydrocarbons

	Surface water		Sediment		*Hetrobranchus Longefillis*	*Mormyrus rume*	*Clarias gariepinus*
Adult Exposure	Oral Ingestion	Dermal	Oral Ingestion	Dermal	Dietary Ingestion	Dietary Ingestion	Dietary Ingestion
Nap	BDL	BDL	5.14E-09	2.17E-11	7.97E-10	1.13E-11	2.82E-12
Acy	BDL	BDL	5.14E-09	2.17E-11	8.45E-12	5.63E-12	2.82E-12
Ace	BDL	BDL	1.54E-08	6.51E-11	5.63E-12	2.82E-12	2.82E-12
Flu	BDL	BDL	BDL	BDL	5.63E-12	5.63E-12	BDL
Phen	BDL	BDL	1.03E-08	4.34E-11	2.82E-12	BDL	BDL
Ant	BDL	BDL	5.14E-08	2.17E-10	2.82E-12	1.41E-11	BDL
Flt	BDL	BDL	BDL	BDL	BDL	2.82E-12	BDL
Py	BDL	BDL	5.14E-09	2.17E-11	2.82E-12	BDL	BDL
BaA	BDL	BDL	5.14E-09	2.17E-11	2.82E-12	BDL	BDL
BkF	BDL	BDL	8.73E-08	3.69E-10	BDL	BDL	2.82E-12
BbF	2.63E-10	2.12E-07	2.05E-08	8.68E-11	2.82E-12	5.63E-12	8.45E-12
BaP	2.63E-10	2.12E-07	1.03E-08	4.34E-11	8.45E-12	8.45E-12	2.82E-12
DBA	BDL	BDL	5.14E-09	2.17E-11	BDL	BDL	BDL
IND	BDL	BDL	1.54E-08	6.51E-11	BDL	BDL	BDL
BghiP	BDL	BDL	1.54E-08	6.51E-11	BDL	BDL	BDL
∑ PAHs	5.25E-10	4.24E-07	2.52E-07	1.06E-09	8.39E-10	5.63E-11	2.25E-11

UNDER PEER REVIEW

Children Exposure	Oral Ingestion	Dermal	Oral Ingestion	Dermal	Dietary Ingestion	Dietary Ingestion	Dietary Ingestion
Nap	BDL	BDL	1.1E-08	2.6E-11	1.7E-09	2.4E-11	6.01E-12
Acy	BDL	BDL	1.1E-08	2.6E-11	1.8E-11	1.2E-11	6.01E-12
Ace	BDL	BDL	3.29E-08	7.8E-11	1.2E-11	6.01E-12	6.01E-12
Flu	BDL	BDL	BDL	BDL	1.2E-11	1.2E-11	BDL
Phen	BDL	BDL	2.19E-08	5.2E-11	6.01E-12	BDL	BDL
Ant	BDL	BDL	1.1E-07	2.6E-10	6.01E-12	3E-11	BDL
Flt	BDL	BDL	BDL	BDL	BDL	6.01E-12	BDL
Py	BDL	BDL	1.1E-08	2.6E-11	6.01E-12	BDL	BDL
BaA	BDL	BDL	1.1E-08	2.6E-11	6.01E-12	BDL	BDL
BkF	BDL	BDL	1.86E-07	4.42E-10	BDL	BDL	6.01E-12
BbF	4.73E-10	2.09E-07	4.38E-08	1.04E-10	6.01E-12	1.2E-11	1.8E-11
BaP	4.73E-10	2.09E-07	2.19E-08	5.2E-11	1.8E-11	1.8E-11	6.01E-12
DBA	BDL	BDL	1.1E-08	2.6E-11	BDL	BDL	BDL
IND	BDL	BDL	3.29E-08	7.8E-11	BDL	BDL	BDL
BghiP	BDL	BDL	3.29E-08	7.8E-11	BDL	BDL	BDL
∑ PAHs	9.47E-10	4.19E-07	5.37E-07	1.27E-09	1.79E-09	1.2E-10	4.8E-11

BDL: Below detection limit; ∑ PAHs: sum total of polycyclic aromatic hydrocarbons

Table 4.2: Non-carcinogenic CDI of polycyclic aromatic hydrocarbons

	Surface water		Sediment		*Hetrobranchus Longefillis*	*Mormyrus rume*	*Clarias gariepinus*
Adult Exposure	Oral Ingestion	Dermal	Oral Ingestion	Dermal	Dietary Ingestion	Dietary Ingestion	Dietary Ingestion
Nap	BDL	BDL	1.2E-08	5.06E-11	1.86E-09	2.63E-11	6.57E-12
Acy	BDL	BDL	1.2E-08	5.06E-11	1.97E-11	1.31E-11	6.57E-12
Ace	BDL	BDL	3.6E-08	1.52E-10	1.31E-11	6.57E-12	6.57E-12
Flu	BDL	BDL	BDL	BDL	1.31E-11	1.31E-11	BDL
Phen	BDL	BDL	2.4E-08	1.01E-10	6.57E-12	BDL	BDL
Ant	BDL	BDL	1.2E-07	5.06E-10	6.57E-12	3.28E-11	BDL
Flt	BDL	BDL	BDL	BDL	BDL	6.57E-12	BDL
Py	BDL	BDL	1.2E-08	5.06E-11	6.57E-12	BDL	BDL

UNDER PEER REVIEW

BaA	BDL	BDL	1.2E-08	5.06E-11	6.57E-12	BDL	BDL
BkF	BDL	BDL	2.04E-07	8.6E-10	BDL	BDL	6.57E-12
BbF	6.13E-10	4.95E-07	4.79E-08	2.02E-10	6.57E-12	1.31E-11	1.97E-11
BaP	6.13E-10	4.95E-07	2.4E-08	1.01E-10	1.97E-11	1.97E-11	6.57E-12
DBA	BDL	BDL	1.2E-08	5.06E-11	BDL	BDL	BDL
IND	BDL	BDL	3.6E-08	1.52E-10	BDL	BDL	BDL
BghiP	BDL	BDL	3.6E-08	1.52E-10	BDL	BDL	BDL
∑ PAHs	1.23E-09	9.89E-07	5.87E-07	2.48E-09	1.96E-09	1.31E-10	5.25E-11

Children Exposure	Oral Ingestion	Dermal	Oral Ingestion	Dermal	Dietary Ingestion	Dietary Ingestion	Dietary Ingestion
Nap	BDL	BDL	2.56E-08	3.08E-10	2.01E-08	2.84E-10	7.1E-11
Acy	BDL	BDL	2.56E-08	3.08E-10	2.13E-10	1.42E-10	7.1E-11
Ace	BDL	BDL	7.67E-08	9.23E-10	1.42E-10	7.1E-11	7.1E-11
Flu	BDL	BDL	BDL	BDL	1.42E-10	1.42E-10	BDL
Phen	BDL	BDL	5.11E-08	6.15E-10	7.1E-11	BDL	BDL
Ant	BDL	BDL	2.56E-07	3.08E-09	7.1E-11	3.55E-10	BDL
Flt	BDL	BDL	BDL	BDL	BDL	7.1E-11	BDL
Py	BDL	BDL	2.56E-08	3.08E-10	7.1E-11	BDL	BDL
BaA	BDL	BDL	2.56E-08	3.08E-10	7.1E-11	BDL	BDL
BkF	BDL	BDL	4.35E-07	5.23E-09	BDL	BDL	7.1E-11
BbF	5.6E-09	2.48E-06	1.02E-07	1.23E-09	7.1E-11	1.42E-10	2.13E-10
BaP	5.6E-09	2.48E-06	5.11E-08	6.15E-10	2.13E-10	2.13E-10	7.1E-11
DBA	BDL	BDL	2.56E-08	3.08E-10	BDL	BDL	BDL
IND	BDL	BDL	7.67E-08	9.23E-10	BDL	BDL	BDL
BghiP	BDL	BDL	7.67E-08	9.23E-10	BDL	BDL	BDL
∑ PAHs	1.12E-08	4.95E-06	1.25E-06	1.51E-08	2.12E-08	1.42E-09	5.68E-10

BDL: Analytical data below detection limit; ∑ PAHs: sum total of polycyclic aromatic hydrocarbons.

UNDER PEER REVIEW

The cancer risk was evaluated as shown in Table 4.3 to derive the cumulative cancer risk (cancer-total). As we can see, the cumulative PAHs are Nap, Flu, Phen and Ant were absent due to lack of laboratory data and no cancer slope factor for determined values with the cumulative cancer total for adults and children having 1.85E-06 and 2.00E-06, which were within USEPA reference values respectively (Verbruggen, 2012).

Hazard quotient was evaluated using non-carcinogenic CDI for different exposure medium in assessed samples to derive the hazard, with the cumulative hazard quotient (hazard index) for adults and children were 4.98E-05 and 2.02E-04, which means that both population conglomerate will not have significant health related issues over a period of time (USEPA, 2020).

Table 4.3a: Cancer Risk of polycyclic aromatic hydrocarbons in adults

	Water		Sediment		*Hetro-branchus longefillis*	*Mormyru-srume*	*Clarias gariepinus*	
Adult Exposure	Oral Ingestion	Dermal	Oral Ingestion	Dermal	Dietary Ingestion	Dietary Ingestion	Dietary Ingestion	Total CR
Nap	No Data	No Data	No CSF	No CSF	No CSF	No CSF	No CSF	0.00+00
Acy	No Data	No Data	3.75E-10	1.58E-12	6.17E-13	4.11E-13	2.06E-13	3.78E-10
Ace	No Data	No Data	1.13E-10	4.75E-13	4.11E-14	2.06E-14	2.06E-14	1.13E-10
Flu	No Data	No Data	No Data	No Data	No CSF	No CSF	No Data	0.00+00
Phen	No Data	No Data	No CSF	No CSF	No CSF	No Data	No Data	0.00+00
Ant	No Data	No Data	No CSF	No CSF	No CSF	No CSF	No Data	0.00+00
Flt	No Data	No Data	No Data	No Data	No Data	2.06E-13	No Data	2.06E-13
Py	No Data	No Data	3.75E-09	1.58E-11	2.06E-12	No Data	No Data	3.77E-09
BaA	No Data	No Data	3.75E-09	1.58E-11	2.06E-12	No Data	No Data	3.77E-09
BkF	No Data	No Data	6.38E-10	2.69E-12	No Data	No Data	2.06E-14	6.40E-10
BbF	1.92E-10	1.55E-07	1.5E-08	6.33E-11	2.06E-12	4.11E-12	6.17E-12	1.70E-07
BaP	1.92E-09	1.55E-06	7.5E-08	3.17E-10	6.17E-11	6.17E-11	2.06E-11	1.62E-06
DBA	No Data	No Data	3.75E-08	1.58E-10	No Data	No Data	No Data	3.77E-08
IND	No Data	No Data	1.13E-08	4.75E-11	No Data	No Data	No Data	1.13E-08
BghiP	No Data	No Data	1.13E-10	4.75E-13	No Data	No Data	No Data	1.13E-10
∑ PAHs	2.11E-09	1.7E-06	1.47E-07	6.23E-10	6.85E-11	6.64E-11	2.7E-11	**1.85E-06**

Table 4.3b: Cancer Risk of polycyclic aromatic hydrocarbons in children

Surface water	Sediment	*Hetrobranchus longefillis*	*Mormyrus rume*	*Clarias gariepinus*

UNDER PEER REVIEW

Children Exposure	Oral Ingestion	Dermal	Oral Ingestion	Dermal	Dietary Ingestion	Dietary Ingestion	Dietary Ingestion	Total CR
Nap	BDL	BDL	No CSF	No CSF	No CSF	No CSF	No CSF	0.00+00
Acy	BDL	BDL	8E-10	1.9E-12	1.32E-12	8.77E-13	4.38E-13	8.05E-10
Ace	BDL	BDL	2.4E-10	5.7E-13	8.77E-14	4.38E-14	4.38E-14	2.41E-10
Flu	BDL	BDL	BDL	BDL	No CSF	No CSF	BDL	0.00+00
Phen	BDL	BDL	No CSF	No CSF	No CSF	BDL	BDL	0.00+00
Ant	BDL	BDL	No CSF	No CSF	No CSF	No CSF	BDL	0.00+00
Flt	BDL	BDL	BDL	BDL	BDL	4.38E-13	BDL	4.38E-13
Py	BDL	BDL	8E-09	1.9E-11	4.38E-12	BDL	BDL	8.02E-09
BaA	BDL	BDL	8E-09	1.9E-11	4.38E-12	BDL	BDL	8.02E-09
BkF	BDL	BDL	1.36E-09	3.23E-12	BDL	BDL	4.38E-14	1.36E-09
BbF	3.46E-10	1.53E-07	3.20E-08	7.59E-11	4.38E-12	8.77E-12	1.32E-11	1.85E-07
BaP	3.46E-09	1.53E-06	1.60E-07	3.8E-10	1.32E-10	1.32E-10	4.38E-11	1.69E-06
DBA	BDL	BDL	8.00E-08	1.9E-10	BDL	BDL	BDL	8.02E-08
IND	BDL	BDL	2.4E-08	5.7E-11	BDL	BDL	BDL	2.41E-08
BghiP	BDL	BDL	2.4E-10	5.7E-13	BDL	BDL	BDL	2.41E-10
∑ PAHs	3.80E-09	1.68E-06	3.15E-07	7.47E-10	1.46E-10	1.42E-10	5.75E-11	**2.00E-06**

BDL: Below detection limit; No CSF: reference value unavailable; ∑ PAHs: sum total of polycyclic aromatic hydrocarbons.

3.4 DISCUSSION

In the PAHs study conducted in in Ezu-River, Anaku, Anambra state, Nigeria, different concentration were detected as shown in **Table 4.3**, where benzo [a] pyrene and benzo[b] fluoranthene were detected across all sample sources. According to USEPA (2020) and WHO (2017), benzo[a]pyrene (BaP) is used as a reference value for determining the carcinogenicity of any sample in relation to other 15 priority PAHs due to its ecological and health effect to flora and faunas and subsequently human contact over a period of time in addition to different environmental matrices. Several studies have shown that PAHs is highly hazardous to human health with genotoxic, neurotoxic and behavioral changes to both adult and children (Sharma, 2014; Loganathan *et al*, 2014; Niu *et al,* 2010; Mukhopadhyay *et al.,* 2010). Continuous exposure of PAHs for a period of 350 days as stipulated by USEPA (2020) shows that PAHs can cause damages to human immune, nervous and reproductive system, often reacts with other ions

UNDER PEER REVIEW

that produces highly toxic and carcinogenic PAHs compounds, as 1% of the toxicity of benzo[a]pyrene can damage or destroy red blood cells that is detrimental to persons with leukemia (Ian, 1992; Russo *et al.,* 2006). Naphthalene was highest in *Hetrobranchus longefillis*, as studies by Santucci and Shah (2000) shows that it can cause glucose deficiency in human body; they said that over 400 million people suffer from glucose-6-phosphate dehydrogenase (G6PD) deficiency, with symptoms such as: hemolytic anemia which is commonly found in children, fatigue, lack of appetite, restlessness, and pale skin in addition to nausea, vomiting, diarrhea, blood in urine and jaundice (Otto *et al.,* 1997). Benzo[b]fluoranthene has been associated with gasoline engine exhaust; emissions from burning coal and from oil-fried heating; broiled and smoke food; oils and margarine (Lawal, 2017); and soils, ground water and surface waters at hazardous waste site (WHO, 2017). A close overview of carcinogenic PAHs has shown that continuous human exposure of these PAHs can lead to negative cancer related illnesses, which is evident in studies conducted with rat and mice, where reported negative results for dermal and oral exposure with present of tumor incidence, bladder cancer, degeneration of cognitive reasoning and death (Sepela *et al.,* 2016; Dutta *et al.,* 2010; Ramesh *et al.,* 2010; Mc Callister *et al.,* 2008; Vaanale *et al.,* 2005). Human health risk assessment conducted showed varying chronic daily intake for ingestion and dermal contact via non-carcinogenic and carcinogenic models in all samples (Omokpariola and Omokpariola, 2021). The cancer risk and hazard index were within acceptable USEPA standards, which makes it all samples sources fit for human consumption, as extreme care must be taken into consideration for children exposure as compared to adults. As such, the exposure period of any population is dependent on age, sex, body weight, location and proximity to pollution (Verbruggen, 2012). It is worth mentioning that chances of children exposure to the carcinogenic risk is higher than for adults (Karyab *et al.* 2015), as it is necessary that extra caution should be taken by everyone using the aquatic environment, while regulatory bodies should as well ensure that the pollution of the aquatic environment is controlled to ensure the safety of human and aquatic lives.

Conclusion

The research has revealed the influence of polycyclic aromatic hydrocarbons to aquatic environment in diverse concentrations in Ezu-River, Anaku, Anambra state, Nigeria. We see that pollution has the capacity to alter the natural balance of diverse locations, as water bodies are

UNDER PEER REVIEW

encompassed by numerous pollution sources and migratory influence; there is a need to constantly monitor diverse water bodies suited to the study locations to ascertain possible cause and mitigate any impending pollution to the ecological system. Human health risk assessment showed that both hazard index and total cancer risk were within acceptable limit, as such, proper advocacy and sensitization is needed to assist inhabitants on the health impact of heavy PAHs for their survival. Therefore, the following recommendations are advocated:

i. Reducing emissions to a minimal level. If this not possible, then at least a workable pollution mitigation plan

ii. Public awareness and education about the sources and health effects of exposure to PAH should be improved.

iii. Aquatic environment should be monitored all year round and not only seasonally.

COMPETING INTERESTS DISCLAIMER:

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

UNDER PEER REVIEW

References

Abou-Arab, A.A, Abou-Donia, M.A, El-Dars, F.M, Ali, O.I and Hossam A,G.(2014). Detection of polycyclic aromatic hydrocarbons levels in Egyptian meat and milk after heat treatment by gas chromatography-mass spectrometry. International Journal of Current Microbiology and Applied Science. 3(7): 294-305.

Arnot, J.A and Quinn, C.L.(2015). Development and Evaluation of a Database of Dietary Bioaccumulation Test Data for Organic chemicals in Fish. Environmental science & Technology.49(8):4783-4796.

Brown, J.N and Peake, B.M.(2006). Sources of heavy metals and polycyclic aromatic hydrocarbons in urban stormwater runoff. Science Total Environment. 359(1-3):145-55.

Dutta, K., Gosh, D., and Nazmi, A.(2010). A common carcinogen benzo[a]pyrene causes neuronal death in mouse via microglial activation Plos one 5: e 9984.

European Union Commission Regulation EU. (2006). European Union Commission Regulation EU No 1881/2006 of 19 December 2006. Setting maximum levels for certain contaminants in foodstuff, 2006.

Farrington, J.W. (2014). Oil pollution in the marine environment II: Fates and Effects of oil spills. Environmental Science Policy, Sustainability and Development. 56(4):16-31.

Gong, P.,Wang, X and Yao, T (2011). Ambient distribution of particulate and gasphase n-alkanes and polycyclic aromatic hydrocarbons in the Tibetan Plateau.Environmental Earth Sciences,64(7): 1703–1711.

Ian,C.T.N., and Lagos.K.P.,(1992) Toxic equivalent Factor (TEFs)for PAHs,16:290-300.

Kumar, A., Schimmelmann, A., Sauer, P.E and Brassell, S.C. (2017). Distribution and sources of polycyclic aromatic hydrocarbons (PAHs) in laminated Santa Barbara Basin sediments. Organic Geochemistry, Vol.113, Pp: 304-314. https://doi.org/10.1016

Lee B.K.(2010). Sources, distribution and toxicity of polyaromatic hydrocarbons (PAHs) in particulate matter.DOI:10.5772/10045

Loganathan, P., Nugyen, T. C., Nugyen T.V., Vigneswaran, S. Kandasame, J., Sly, D., Stevenson, G., and Naidu, R.(2014). Polycyclic aromatic hydrocarbons in road deposited sediments, water sediments and soil in Sydney Australia : comprises of concentration distribution, sources and potential toxicity. Eco toxicity and environmental safety.104: 339-348

Mc Callister, N.M., Maguire, M., and Ramesh, A.(2008). Prenatal exposure to benzo[a]pyrene impairs later life. Cortical neuronal function neurotoxicology, vol.29 pg 846-854.

Mukhopadhyay, D., Nnadi, P., and Varghese, A . C. (2010) The vitro effect of benzo[a]pyrene on human sperm hyper activation and acrosome reaction. Fertil.Sterli (in press).

Niu, Q., Zhang, H., and Li, X.(2010). Benzo[a]pyrene- induced neurobehavioral function and neuro transmitter alternation s in coke oven worker occup. Environ. Med. Vol.67 pg 444-448.

Ojaniyi, O.F., Okoye, P.A.C., Omokpariola, D.O. (2021). Heavy Metals Analysis and Health Risk Assessmentof Three Fish Species, Surface Water and SedimentSamples in Ogbaru Axis of River Niger, AnambraState, Nigeria. *Asian Journal of Applied Chemistry Research*. 9(1): 64-81.

Omokpariola D.O and Omokpariola P.L. (2021). Health and Exposure risk assessment of heavy metals in rainwater samples from selected locations in Rivers State, Nigeria. *Physical Science Reviews.* 1-14. https://doi.org/10.1515/psr-2020-0090.

Omokpariola, J.O., Omokpariola, D.O. and Omokpariola, E.C.O. (2021). Risk assessment of polycyclic aromatic hydrocarbons and total petroleum hydrocarbons in oilfield produced water and seawater at Gulf of Guinea Oilfield, Nigeria. *Advanced Journal of chemistry – section B.* 3(1): 68 – 85. https://doi.org/10.22034/ajcb.2021.121909.

Olayinka, O.O., Adewusi, A.A.,Olujimi, O.O and Aladesida, A.A. (2019). Polycyclic aromatic hydrocarbons in sediment and health risk of fish, crab and shrimp around Atlas cove, Nigeria. J. health pollut. 9(24):191204.

UNDER PEER REVIEW

Ramesh, A., Archibong, A.E., and Niaz, M.S.(2010). Ovarian susceptibility to benzo[a]pyrene: Tissue burden of metabolites and DNA adducts in F-344 rats. J. toxicol. Environ. Health. (in press) .

Russo, A., Troncoso, N., and Sanchez, F.(2006). Propoles protects humans spermatozoa from DNA damage caused benzo[a]pyrene and exogenous reactive oxygen species. Life. Sci. vol. 78 pg 1401-1406.

Santucci K, Shah B (2000). Association of naphthalene with acute hemolytic anemia. AcadEmerg Med. 7 (1): 42–7. doi:10.1111/j.1553-2712.2000.tb01889.x. PMID 10894241.

Seopela, M.P., McCrindle, R.I., Combrinck, S. and Regnier, T. J-C. (2016) "Hazard assessment of polycyclic aromatic hydrocarbons in water and sediments in the vicinity of coalmines."Journals of soil and Sediments, vol. 16 pg 2740-2725.

Sharma, T.(2014). In- silico investigation of polycyclic aromatic hydrocarbon against bacteria 1,2 dioxygenase journal of chemical and pharmaceutical research. 6: 873-877

Tella, A.C., Ajayi, S.O., Abiola, B.E., Adekunle, A.K., Akintelu, A.S. and Olabemiwo, O.M. (2017).Assessment of the levels of polycyclic aromatic hydrocarbons in Wazo market topsoil, Ogbomoso, Nigeria.Ew J Anal & Environ Chem 3(1), 111 - 118.

Thepanodh, S., Ayer, G.P and Hooper, M.A. (2005). Analysis of precipitation chemistry in Northern Thailand. Clean Air Environ Quantity. 39:43-47.

U.S. EPA. (2015). Report on the 2015 U.S. Environmental protection Agency (Epa) International Decontamination Research and Development conference. U.S. Environmental Protection Agency, Washigton, Dc, EPA/600/R-15/283.

U.S. EPA. (2017). Regional screening levels (RISLS)- user's guide- Risk assessmeent. Retrived January 19, 2012, from United States Environmental protection agency Web Archive: http://hawaii.gov/health/environmental/ hazard/index.html (2017).

US.EPA (1992). United States Environmental Protection Agency (US EPA). Guidance for data usability in risk assessment (Part A), Final, PB92-963356.

US.EPA (2005a). United States Environmental Protection Agency. Guidelines for carcinogen risk assessment [EPA Report]. (EPA/630/P-03/001F). Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. http://www2.epa.gov/osa/guidelines-carcinogen-risk-assessment.

US.EPA (2005b). United States Environmental Protection Agency. Supplemental guidance for assessing susceptibility from early-life exposure to carcinogens.(EPA/630/R-03/003F).Washington, DC: U.S. Environmental Protection Agency, Risk Assessment Forum. https://www3.epa.gov/airtoxics/childrens_supplement_final.pdf

US.EPA. (1991). U.S. Environmental Protection Agency. Human Health Evaluation Manual, Supplemental Guidance: Standard Default Exposure Factors; USEPA:Washington, DC, USA, 1991.

US.EPA. (2016). Test methods for evaluating Solid Waste, SW –846, 3rd Edition (USEPA, Office of solid Waste and Emergency Response, Washington, DC, November). pp 200-206.

USEPA (2020). Regional Screening levels (RSLs) Table. Updated May 1, 2020.United States Environmental Protection Agency Washington D.C. https://www.epa.gov/risk/regional-screening-levels-rsls-generic-tables.

UNDER PEER REVIEW

Vaanale, V., Mella, M., and Kalliokoski, P.(2005). Dermal exposure to polycyclic aromatic hydrocarbons among road pavers. Ann. Occup. Hyg.Vol. 49 pg 167-178.

Verbruggen, E.M.J. (2012) Environmental risk limits for polycyclic aromatic hydrocarbons (PAHs) for direct aquatic, benthic and terrestrial toxicity. RIVM Report 607711007/2012. National Institute for Public Health and the Environment Ministry of Health, Welfare and Sport. https://www.rivm.nl/bibliotheek/rapporten/60771107.pdf

Wang, Z., Chen, J.,Yang, P., Qiao, X and Tian, F.(2007). Polycyclic Aromatic Hydrocarbons in Dalian soils: distribution and toxicity assessment, Journal of Environment Monitoring., 9(2): 199–204.

WHO(2017):Guideline for drinking water quality recommendation,5th edition, Geneva, Switzerland.

Yu, H., Li, T., Liu, Y and Ma, L. (2019).Spatial distribution of polycyclic aromatic hydrocarbon contamination in urban soil of China. J. Chemosphere, Vol.230, pp: 498-509. https://doi.org/10.1016.

UNDER PEER REVIEW