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.

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* benz(b)fluoranthene was the highest pollutants. The Health and exposure risk assessment was conducted for carcinogenic and non-carcinogenic exposure in adults and children which shows that the cumulative cancer risk and hazard index were within USEPA regulatory standard. Calculated Hazard Index for fish and water samples were less than one and thus be recommended for consumption.

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 *et al.*, 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.*, 2017).

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, phenanthrene etc 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. HMW PAHs have molecular structures of four or more benzenoid rings (e.g. fluoranthene, pyrene, benzo[a]pyrene, and benzofluoranthenes) and 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 *et al.*, 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.

This study is aimed at assessing the health risk of polycyclic aromatic hydrocarbons (PAHs) and heavy metals in surface water, sediments and fishes in Ezu-River, Anaku,Anambra state, Nigeria. The main objectives of this study were (i) To determine the concentration of polycyclic aromatic hydrocarbons (PAHs) in the surface water, sediment and fish samples using Gas

Chromatography-Mass Spectrophotometer(GC-MS) (ii) To check the health risk factor of PAHs and heavy metals in surface water, sediment and fish using USEPA methods.

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 whose end source is River Niger.. It is mostly dominated by the Igbos. The occupations in the community are predominantly fishing, farming and hunting.

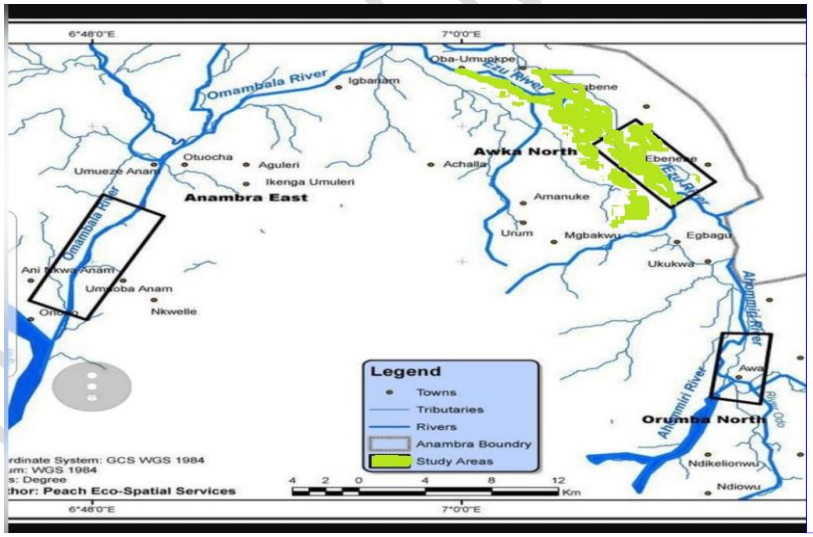

Figure 1: Map showing the study area showing sampling location.

Comment [D(O1]: A little more information on the Geology/hydrogeology, vegetation and stratigraphy of the study area.
Insert the sampling locations on the map.

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 placed on ice packs. It was immediately transported to the laboratory and refrigerated at 4°C prior to analysis.

Comment [D(O2]: How was the water sample preserved prior to analysis?

2.3.2 Sediment sample: 2kg of surface sediment samples were collected using a Van-Veen grab sampler at four different locations in the river and homogenized into a composite sample. The sample was wrapped in aluminium foil and was kept at 4°C. In the laboratory, Stones and debris were removed from the sample and then frozen-dried before the extraction procedure.

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. They were refrigerated at 4◦C until extraction (Ezemonye *et al*., 2008).

2.4 Analytical procedures

Preparation of packed column

Ten grams (10g) of 100mm mesh silica gel was baked at 105°C in an oven overnight. The baked silica gel was mixed with 15ml dichloromethane to form slurry. The fractionating column was packed with glass wool followed by the slurry silica gel and 3grams of anhydrous sodium sulphate was then added to absorb water.

2.4.1 Surface water: The PAHs in the sample was determined according to USEPA, 2016 method.

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 organic phase 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. 2ml of the concentrated sample was pipetted into a Teflon screw-cap vial and analyzed for PAH using the Gas Chromatography-mass spectrometer.

2.4.2 Sediment sample: The PAHs in the sample was determined according to USEPA, 2016 method.

10grams of sediment sample was weighed and homogenized 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 an ultrasonicator bath for 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 to about 2ml using a rotary evaporator. 1.5ml of the concentrated sample was pipetted into the vertical 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-MS 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 as shown below:

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 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).

Comment [D(O3]: Oven temperature program of GC-MS is not mention.

How did the authors authenticate the accuracy of the analytical data?(No LOD, LOQ , Recovery % etc. is mentioned as the sample matrices were not spiked with deuterated PAHs)

Instrumental and analytical conditions

An Agilent 6890 gas chromatograph equipped with auto sampler connected to an Agilent 5973N mass selective detector was used. 1µl of sample solution was injected in the spiltless mode onto a 30m x 0.25mm META X_5 coated fused capillary column with a film thickness of 0.25µm. Helium was used as the carrier gas and the column head pressure was maintained at 13 psi to give constant flow 1.0 ml/min. Other operating conditions were pre-set, purge time 2.00 mins, purge flow 20.0 ml/min, total flow of 23.7 ml/min, and injection temperatures 250^{o}C. The column temperature was initially held at 70^{o}C for 2mins, increased to a final temperature of 300^{o}C at a rate of 12^{o}C/min and held for 8mins. The mass spectrometer (MS) condition was electron impact positive ion mode. The Aromatic compound identification time was based on retention time since each of the Aromatic compounds has their separate retention time in the column. Those with lower retention times were identified first followed by those with longer retention time.

Quality control

The blanks were analysed the same way as the samples. The surface water, sediment and fish samples were spiked. These fortified matrices were used as calibration standards and the range of concentrations added to their matrices were used to produce the calibration curves of 20 - 100 mgkg-1. The surrogate internal standards were added to the spiked surface water, sediment and fish samples at100 mgkg-1. The response factors were then calculated using the response obtained from desorption of a standard solution containing 40 mgkg-1 of the 16 PAHs of interest and 100 mgkg-1 of each internal standard. Spiked samples were extracted and analyzed. Recovery yields were 75 - 110% and limit of detection for individual PAHs ranged from 0.02 to 30.00 mgkg- 1 in the samples with a signal to noise ratio of three (3) and limit of quantization of signal to noise ratio of ten (10).

2.5 Data Analysis: Microsoft Excel 2019 data analysis was utilized for determination of mean and standard deviation

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 by 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).

Comment [D(O4]: by

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

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

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

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),RBA is relative bioavailability for sediment calculation, 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)

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

$$\text{CDI-ingestion} = \left(\frac{\text{CS} \times \text{IR}_W \times \text{EF} \times \text{ED} \times \text{TR}}{\text{BW} \times \text{AT}}\right) \quad (3)$$

$$\text{CDI-dermal} = \left(\frac{\text{CS} \times \text{SA} \times \text{ET}_W \times \text{EF} \times \text{AF} \times \text{ED} \times \text{TR}}{\text{BW} \times \text{AT}}\right) \quad (4)$$

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 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.

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

Where: CS is concentration of PAHs in mg/kg, IR_F 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).

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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
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 were below detection limit (<0.001mg/l) except for BbF

(0.02mg/l) and BaP (0.023mg/l) with mean concentration of 0.003mg/kg. In sediment, the mean concentration of PAHs was 0.027 mg/kg respectively. *Hetrobranchus longefillis* concentration of PAHs indicated that Nap has the highest concentration (2.807mg/kg) while the mean concentration of PAH detected is 0.185mg/kg. The mean concentrations of PAH in *Mormyrus rume* 0.011 mg/kg while in *Clarias gariepinus*, mean concentrations of PAH is 0.004mg/kg. The concentration of BaP across all samples exceeded the EU recommended safe limit of 0.002 mg/kg for human fish consumption (EU, 2006). High molecular weight (HMW) PAHs displayed high concentration in surface water, sediment and *Clarias gariepinus* than lower molecular weight (LMW) PAHs) for *Hetrobranchus longefillis* and *Mormyrus rume*, which is due to bioaccumulation and biological distribution pattern of PAHs across different 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-MS analysis. High molecular weight (HMW) is aggregate of aromatic carbon ring, C–4 to C–6, which depicts that sediment recorded the highest concentration as compared to surface water, *Hetrobranchus longefillis*, *Mormyrus rume* and *Clarias gariepinus*. As shown in Table 4.3, the cumulative sum of carcinogenic PAHs (cPAHs) in decreasing order was; 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).

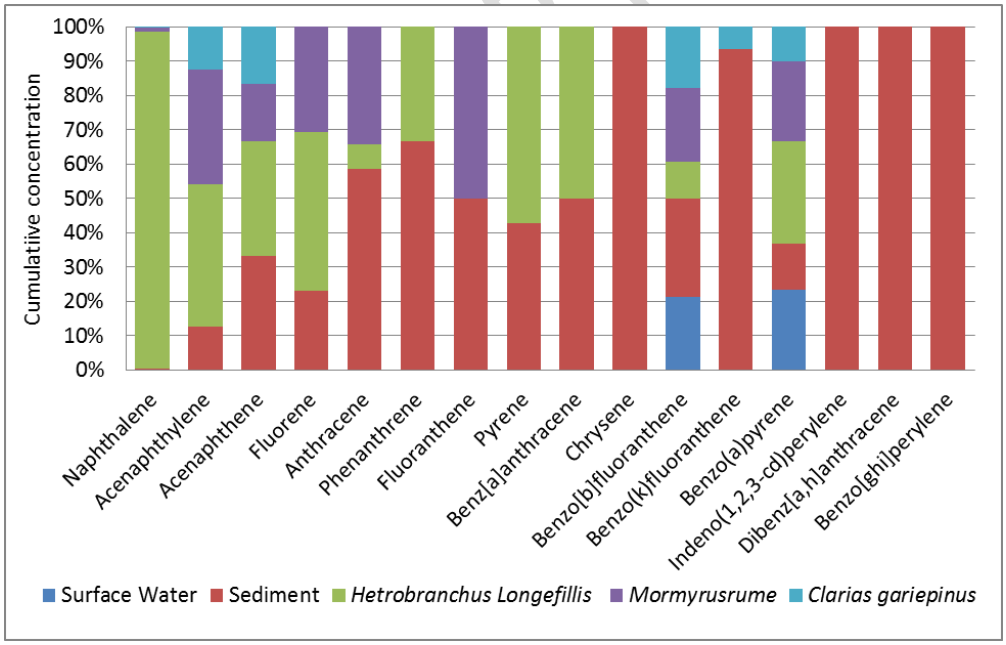
Comment [D(O8]: GC-MS analysis

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
Mean	0.003	0.027	0.185	0.011	0.004

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



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237

238 **Figure 2: Percentage stark column of PAHs in analyzed samples**

3.3 Health 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 as follows: 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.

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 from adults to children was evaluated using similar model to assess the carcinogenic and non-carcinogenic assessment, as shown in Figure 3. The results are are as follows:

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.

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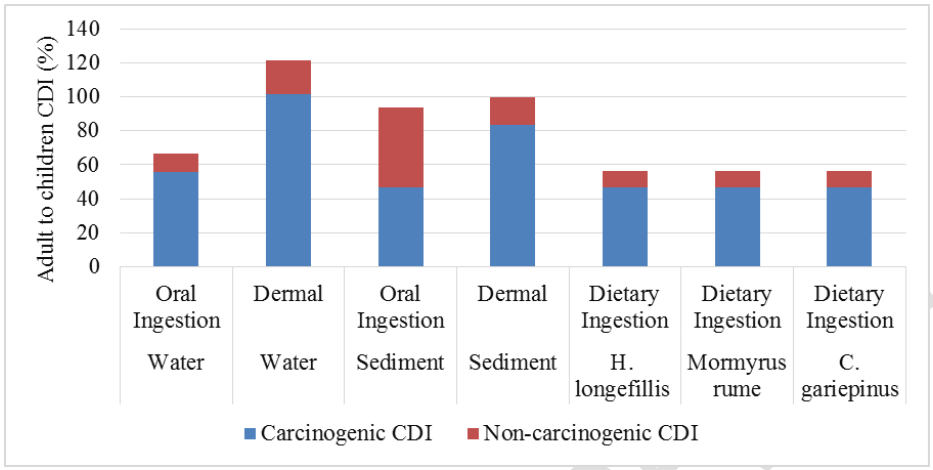

Figure 3: Cumulative CDI influence of adults to children

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

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
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.

According to table 4.3, the cumulative cancer total for adults and children are 1.85E-06 and 2.00E-06, which were within USEPA reference values respectively.

The hazard index for adults and children were less than one which shows that exposed population 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

Comment [D(O11)]: Keep the table at landscape orientation

	Surface water		Sediment		*Hetrobranchus longefillis*	*Mormyrus rume*	*Clarias gariepinus*	
Children	Oral	Dermal	Oral	Dermal	Dietary	Dietary	Dietary	Total CR

Exposure	Ingestion		Ingestion		Ingestion	Ingestion	Ingestion	
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.

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

ii. 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

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