



RESEARCH ARTICLE

Health Risk Assessment of Exposures to Polycyclic Aromatic Hydrocarbons from Water and Fish Ingestion in Bayelsa State, Nigeria

Ephraim-Emmanuel, Benson Chukwunweike^{1*}, Enembe, Okokon² and Ordinioha, Best³

¹Africa Centre of Excellence for Public Health and Toxicological Research, University of Port Harcourt, Rivers State, Nigeria

²Department of Community Medicine, University of Calabar, Calabar, Nigeria

³Department of Environmental Health, School of Public Health, University of Port Harcourt, Rivers State, Nigeria

*Corresponding author: Dr. B.C. Ephraim-Emmanuel, Africa Centre of Excellence for Public Health and Toxicological Research, University of Port Harcourt, Rivers State, Nigeria



Abstract

Background: The harmful properties of many polycyclic aromatic hydrocarbons, their capacity to bioaccumulate, and their persistence in the environment, have contributed in making exposures to these compounds to be an issue of great public health concern. This study assessed the health risks associated with PAHs exposure from the ingestion of fish and water in Bayelsa State.

Methods: This study utilized a comparative, cross-sectional design which was conducted in Bayelsa State. It involved 615 adults to whom a food frequency questionnaire was administered to elicit necessary data. PAHs concentrations of the fish and water were also obtained. The Statistical Package for Social Sciences version 25 was used to perform both descriptive and inferential analyses.

Results: Mean incremental lifetime cancer risk associated with the ingestion of fish and water in Sampou, Gbarain and Nembe were $0.010 \pm 0.0019 \times 10^{-3}$ and $0.3452 \pm 0.1357 \times 10^{-3}$; $0.0017 \pm 0.0003 \times 10^{-3}$ and $0.0636 \pm 0.0351 \times 10^{-3}$; $0.0064 \pm 0.0014 \times 10^{-3}$ and $0.0179 \pm 0.0091 \times 10^{-3}$ respectively. The mean hazard index and benzo[a]pyrene equivalents (B[a]P_{eq}) associated with fish and water ingestion in the communities were also computed.

Conclusion: The health of the population in Sampou was slightly at risk for developing cancers due to PAHs exposures from the drinking of water. Constant biomonitoring of environmental media is necessary for ensuring the health of a populace.

Keywords

Risk assessment, Polycyclic aromatic hydrocarbons, Health, Environmental exposures

Introduction

Polycyclic aromatic hydrocarbons (PAHs) are a large subset of ubiquitous organic compounds which possess the capacity to be widely distributed in terrestrial and aquatic ecosystems [1,2]. These organic compounds are environmentally persistent, hardly biodegradable, carcinogenic, mutagenic and environmentally toxic. The level of toxicity of these organic compounds is largely dependent on their molecular weight as the larger molecular weight PAHs (high MW PAHs) having four to seven aromatic rings are not acutely toxic but possess a greater capacity for carcinogenicity [3,4]. PAHs also possess the capability of causing oxidative stress alongside the sequel of cytotoxic effects [5]. Exposure in children has been linked with the manifestation of several pathological outcomes such as symptoms of asthma and poor cognitive development as well as the occurrence of lower pulmonary function and wheezing symptoms in adults [5]. For a number of years now, the problem associated with the release of polycyclic aromatic hydrocarbons (PAHs) into the environment has been worsened by a number of factors. These include increasing levels of artisanal refining activities; intense oil exploration [6,7]; the use of leaded petrol and biomass fuel; inappropriate dumping and burning of toxic waste [3,4] and so on [8-10].

Environmental exposures of organisms to PAHs

can be assessed by monitoring different aspects of their environment (sediment/soil, water, and air) [11]. In a bid to determine how much of an environmental toxicant an organism is exposed to, a risk assessment becomes necessary. A risk assessment is a quantitative and qualitative evaluation of the risk posed to human health and/or the environment by the actual or potential presence and/or use of specific pollutants. It is thus an evaluation of the hazardous properties of environmental pollutants, the dose-response relationship of these pollutants and the extent of human exposure to them [12]. The assessment of risk associated with the exposure to PAHs is necessary in identifying the level of risk experienced by an exposed population. This is particularly important regarding ingested foods and water wherever environmental pollution may lead to elevated levels of impact on the food and water products. For effective assessments, maximum permissible levels of the PAHs are usually put in place by regulatory organizations to protect the health of the public [13,14]. Risk assessment of PAHs uses the results from three steps in giving the probability of the harmful effects of the PAHs in the population. These steps include hazard identification, dose-effect relationship and exposure assessment [15]. Important elements for assessing these exposures include the estimated daily intake of the compound related with ingesting foods in communities to be assessed, the non-cancer risk assessment (hazard index) as well as the cancer risk assessment (Incremental lifetime cancer risk) [16-19].

Considering the harmful properties of many polycyclic aromatic hydrocarbons, their capacity to bioaccumulate, and their persistence in the environment, extensive exposure to these compounds is of great public health concern [17,20,21]. Also, it has been documented that artisanal refining of crude oil coupled with a myriad of other activities which relate to crude oil theft have been responsible for the incessant pollution of the environment with PAH compounds especially within the Niger Delta region of Nigeria [22-25]. They have also been implicated in the occurrence of disastrous damage of the ecosystem and other components of the environment [8,22,26] as well as causing a number of health problems [27-31].

Being one of the states located within the Niger Delta region, residing in oil-producing communities of Bayelsa state could make the populace prone to various carcinogenic and non-carcinogenic risks due to exposure to PAH compounds. It is possible that the problems associated with illegal oil and gas mining activities exist in Bayelsa State [25]. This study thus set out to evaluate the health risks associated with fish or water ingestion within oil-producing Communities in Bayelsa State, Nigeria. It was anticipated that this research would enable a better understanding of the wide-ranging impacts of artisanal refining practices and

can provide a guide for relevant future decisions and actions by the government. This study also provided empirical data on the health risks which the populace residing in these areas communities are exposed to whenever they eat fish or drink water obtained from within these communities.

Materials and Methods

Study design and study area

This study utilized a comparative, cross-sectional design and was conducted in three communities in Bayelsa state. These included Ogbolomabiri in Nembe Local Government Area (LGA), which has been impacted by artisanal refining activities and Gbarain in Yenagoa LGA which has been impacted by both artisanal refining and gas flaring activities. The third community: Sampou located within the Kolokuma/Opokuma LGA was chosen to serve as a reference group because it is a community where neither artisanal refining nor gas flaring activities have been reported.

Study population and sampling

Study population: The study population included healthy persons whom had resided in the Communities for not less than a period of 2 years and were aged between 18 years and 65 years.

Sample size determination and sampling techniques: Formula for sample size for comparing two proportions was used in calculating the sample size used for this study [32]. Proportions of the attributes of interest in PAHs exposed and unexposed groups = 28.0% and 17.0% were obtained from a study that assessed petroleum contaminated water and health symptoms in a rural Nigerian community [33]. A sample size of 615 was finally obtained and was proportionately distributed across the 3 selected communities. Multistage sampling was used in the selection of the study respondents from the different Communities. The first stage was simple random sampling to randomly select wards in the Local Government Areas (LGAs) by balloting. Second stage was simple random sampling by balloting, to select communities within each selected ward. The third stage involved simple random sampling to select houses within selected communities. Each house formed a cluster, and everyone who met the inclusion criteria were recruited. Where more than one household was present in a house, simple random sampling by balloting was used to select only one household. During the course of sampling, when any respondent chose not to participate in the study, sampling was extended to compensate for that. Sampling continued until the minimum sample size was met. A food frequency questionnaire was used to elicit data on the consumption rates of fish and water for each respondent in this study. This was used in the assessment of the level of risk they were exposed to whenever they ate fish or drank water.

Formula for sample size for comparing two means was also used in calculating the sample size for environmental samples used for this study [34]. The mean and standard deviation of the attribute of interest (PAHs concentration in water) in a PAHs non-exposed group was obtained from the study by Adekunle, et al. at Ife north in Osun state [35]. Also, the mean and standard deviation of the attribute of interest (PAHs concentration in water) in a PAHs exposed group was obtained from the study conducted by Aigberuaat Imiringi in Bayelsa state [36].

Exposure assessment

Environmental monitoring of the PAHs levels of the water, soil and fish was done and PAHs levels were determined using the gas chromatography/flame ionization detector (GC/FID) method as described by [36]. Water samples were collected from five points in each study community using previously cleaned 1 litre capacity glass bottles. The geographic locations for these points (areas on the map with orange-coloured pins) in Nembe ($4^{\circ}N53'32''$ $6^{\circ}E$ $40'31''$, $4^{\circ}N$ $52'95''$ $6^{\circ}E$ $39'82''$ $4^{\circ}N$ $53'04''$ $6^{\circ}E$ $39'26''$), Gbarain ($5^{\circ}N$ $02'79''$ $6^{\circ}E$ $28'22''$, $5^{\circ}N$ $01'05''$ $6^{\circ}E$ $29'28''$) and Sampou ($5^{\circ}N$ $14'78''$

$6^{\circ}E$ $35'42''$, $5^{\circ}N$ $14'46''$ $6^{\circ}E$ $35'04''$) are shown in Figure 1.

Five fish samples of Tilapia fish (*O. niloticus*) harvested at the study sites were collected from each of the three sampling sites. These samples were immediately delivered to the Analytical concepts Ltd., Elemenwo, Port Harcourt, Rivers State, Nigeria; for gas chromatographic analysis of 16 PAHs. This was done using the Gas Chromatographic System (6890 series and 6890 plus) version A.03.08 equipped with a dual detector (FID-ECD), dual column and TriPlus AS auto-sampler with helium carrier gas and a quadrupole Mass Spectrometer (Agilent 5975 MSD) based on USEPA method 8100 (US EPA, 1986).

Extraction and clean-up of PAHs from samples

The liquid-liquid extraction technique was applied to extract PAHs in surface water samples using the method given by the [37] with slight modification on the procedure applied in another study [38]. Firstly, the 250 mL water sample was homogenized before emptying the entire volume into a 500 mL separating funnel. Afterwards, PAHs were extracted by a three-batch extraction process using 20 mL of dichloromethane

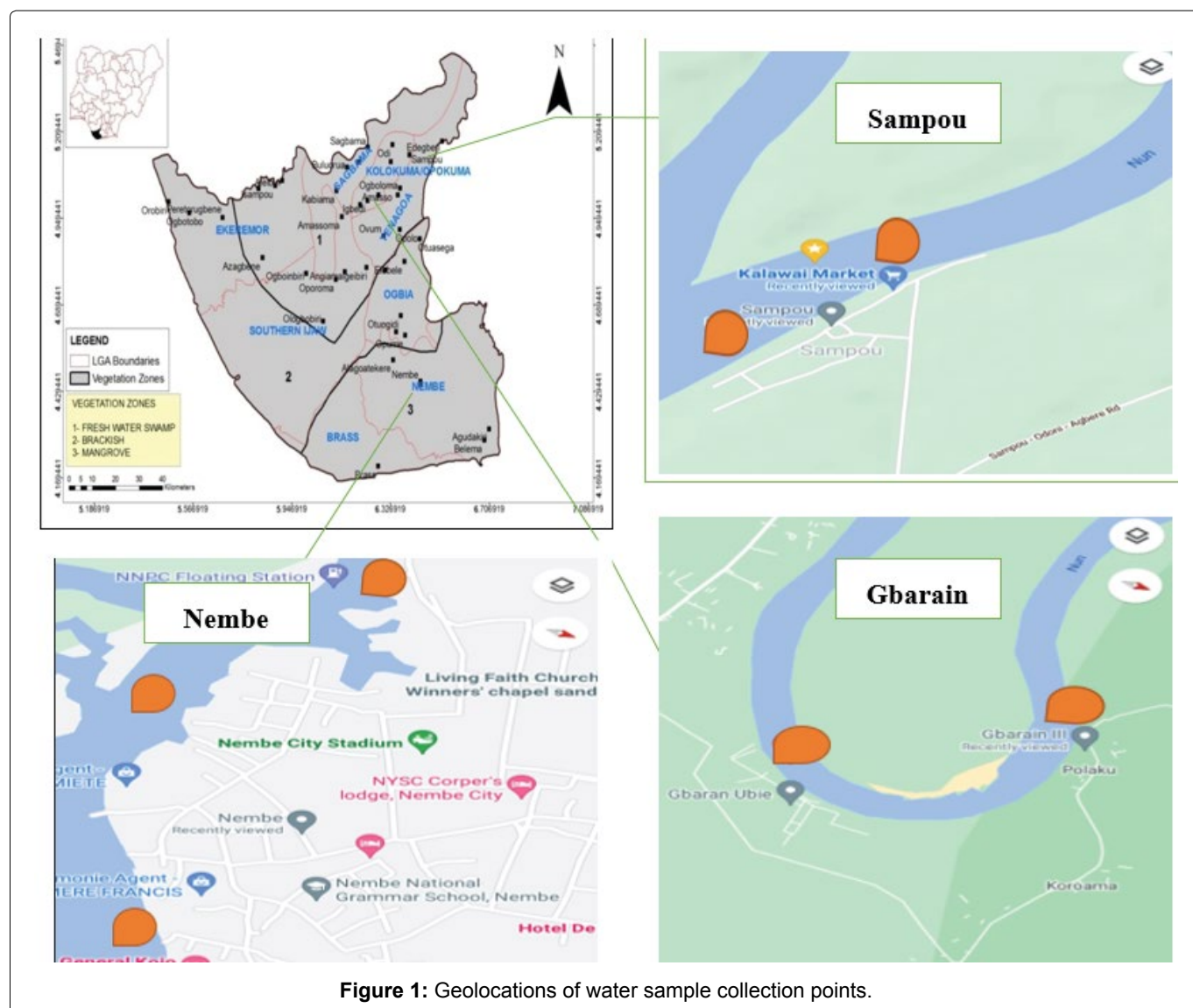


Figure 1: Geolocations of water sample collection points.

(DCM)/n-hexane (1:3 v/v) mixed solvents at each time. The sample-solvent mixture in the separating funnel was vigorously agitated with intermittent ejection of built-up pressure from the tap of glass funnel. This was done to eliminate the risk of blowing up the glass material. Thereafter, the organic extract was dehydrated by filtering through anhydrous sodium sulphate. Organic contaminants in filtered extracts were cleaned by eluting through a 10 mm I.D (internal diameter) × 250 mm long chromatographic column packed with glass wool, slurry of silica gel and anhydrous sodium sulphate. The cleaned-up extract was reconstituted to about 1.0 mL, after being concentrated on a temperature regulated water bath at 35-40 °C. Finally, sample extracts were transferred into glass vials with rubber-crimped caps. Another 250 mL portion of water sample was transferred into a separating funnel and spiked with pre-deuterated PAHs mixture (naphthalene-d8, phenanthrene-d10, chrysene-d12 and perylene-d12) as internal standards, to establish the efficiency of the extraction protocol. The recovery rates ranged between 92% and 107%. Exactly 20 mL of dichloromethane (DCM)/n-hexane (1:3 v/v) mixed solvents were added to the sample mixture, thoroughly mixed and kept standing to allow for adequate phase separation prior to dehydration and filtration, followed by clean-up and elution through a chromatographic column. Afterwards, the eluted extracts were concentrated to 1.0 mL volume and stored in air-tight rubber-crimp cap glass vials [36].

Quantification of PAHs

Exactly 1 µL portion of the reconstituted extract was injected into the gas chromatograph-flame ionization detector (GC-FID) using hypodermic syringe. Nitrogen served as the carrier gas while a combination of hydrogen and air were used to create an ionization environment at the detector head. The various fractions of the aromatic compounds were automatically detected at the FID (whose response is dependent on the composition of the eluted vapour) as they emerged from the column. Results were expressed in µg/L units. Standard pre-set operating conditions of the GC-FID were ensured [36]. The instrument conditions above are based on manufacturer recommendations and PAHs method suitability for repeatability of analytical data on the HP 6890 Plus GC-FID, version A.03.08. Quality assurance/quality control (QA/QC) parameters applied during GC-FID analysis included the spike concentration, concentration obtained, percentage recovery, limit of detection (LOD) and limit of quantification (LOQ) [36]. The surface water (SW) matrix was used to calculate extraction recovery efficiency for the different PAHs: Naphthalene (Nap), Acenaphthylene (Acy), Acenaphthene (Ace), Fluorene (Flr), Phenanthrene (Phe), Anthracene (Ant), Fluoranthene (Flt), Pyrene (Pyr), Benz (a) anthracene (BaA), Chrysene (Chr), Benzo (b) fluoranthene (BbF), Benzo (k)fluoranthene

(BkF), Indeno-1,2,3-cd pyrene (IndP), and Dibenzo (a,h) anthracene (DahA). The instrument limit of detection (LOD) and limit of quantification (LOQ) were also estimated and ranged between 0.001-0.04 µg/mL and 0.004-0.10 µg/mL respectively. The acceptable recovery range of the equipment was stipulated between 90 and 110% [36].

Health risk assessment in relation to fish and water consumption

Weight of study participants was measured using the standard protocols. Using the retrieved data, the estimated daily intake of PAHs from the consumption of fish/water was calculated using the formula;

$$EDI_f = \frac{C_f \times IR_f}{BW}$$

where,

EDI = Dietary dose of exposure of an individual to PAHs from fish/water per day (µg/kg/ bw/day),

C_f = PAHs concentration in fish/water sample (µg/kg),

IR_f = Ingestion rate of fish OR the weight of fish consumed by an average individual category (0.0548 kg) [39]. Also, an average daily consumption of 0.0548 kg/day/person (for an adult population) of fish has been proposed by the Food Agriculture Organization (FAO) data on Fishery and aquaculture statistics [40]. Water drinking frequency and quantity per day was obtained from the data of the food frequency questionnaire.

BW = Specific body weights of assessed population.

Non-cancer risk assessment: The non-carcinogenic risk was estimated by using the target hazard quotient (THQ) model. The hazard quotient (HQ) is the ratio of determined dose of a pollutant taken in per day (EDI: mg/kg/day) to a toxicological endpoint which is the oral reference dose (RfD: Reference dose considered unarmful in mg/kg/day) of that pollutant. The reference dose can be described as an estimate of a continuous exposure occurring among human populations (including sensitive sub-groups) that is likely to have the absence of appreciable risk of deleterious effects during a lifetime [16].

The HQ is estimated using the equation [17,18]:

$$HQ = \frac{EDI}{RfD}$$

$$HQ = \frac{Efr \times ED \times IR \times C}{RfD \times BW \times ATn} \times 10^{-3}$$

Where:

Efr - Exposure frequency (365 days/year)

ED - Exposure duration (58 years, equivalent to average life time of a Nigerian adult)

IR - Intake rate

C - Contaminant concentration ($\mu\text{g}/\text{kg}$)

RfDo - Oral reference dose ($\text{mg}/\text{kg}/\text{day}$)

BW - Body weight of participant (kg)

ATn - Average exposure time for non-carcinogen in days ($\text{Efr} \times \text{ED}$)

10^{-3} - Conversion factor.

The calculated hazard quotients for different pollutants having similar target organ effects can be summed-up to constitute what is known as the hazard index (HI).

Thus,

$$HI = \sum i HQ_i$$

Where,

HI: Hazard index

HQ_i: Individual hazard quotients having similar target organ effects

When the HI values are lesser than one, it can be stated that the health of the exposed population is not at risk due to the consumption of fish or water from the area where they were sourced [17]. The same interpretation can be given to hazard index (HI) values that are lesser than one [16,19].

Cancer risk assessment: The Incremental lifetime cancer risk (ILCR) was estimated by the multiplication of the pollutant oral slope factor (SF) with the estimated daily intake (EDI) or daily exposure doses averaged over a life time. Therefore, the PAHs Cancer Risk (CR) for ingestion of water or fish can be estimated using the following equation [41]:

$$CR = EDI \times SF \times CF$$

Where,

ILCR = Incremental lifetime cancer risk (ILCR) (in $\text{mg}/\text{kg}/\text{bw}/\text{day}$),

EDI = Dietary exposure of an individual to PAHs per day ($\mu\text{g}/\text{kg}/\text{bw}/\text{day}$),

SF = oral slope factor (in mg of pollutant per kg of body weight) every day for a lifetime.

CF is the conversion factor: 1×10^{-3}

The United States Environmental Protection Agency (USEPA) has stated that when the level of carcinogenic health risk is at 10^{-6} for a pollutant, it will result in a relatively negligible cancer risk.

Benzo[a]pyrene equivalent (B[a]Peq) estimation: Considering that benzo[a]pyrene (B[a]P) is considered the most potent carcinogenic PAH, the total PAHs concentration is usually expressed as B[a]P equivalents (B[a]Peq) to illustrate the toxic potency [17]. The B[a]Peq can be calculated as the sum of the B[a]Peq_i values for individual PAHs. Each B[a]Peq_i value can

be calculated for each PAHs using its concentration in the sample (cPAH_i) multiplied by its toxic equivalency factor (TEFPAH_i). When the B[a]Peq value lies below the estimated screening value (SV) of 3.556 mg/kg , it is generally considered that ingesting food or water polluted by PAHs in the affected area does not pose a cancer risk to the health of the inhabitants of the communities [18,39]. The following formula can be used in calculating the benzo[a]pyrene equivalent [17].

$$B[a]Peq = \sum (BaPeq_i) = \sum (cPAH_i \times TEFPAH_i)$$

Data analysis

The Statistical Package for Social Sciences (SPSS) version 25 (IBM, Armonk, New York, USA) was used to perform both descriptive and inferential analyses. The One-way Analysis of variance (ANOVA) was used to compare the concentrations of PAHs, the Estimated Daily Intake (EDI) of fish and water, the Hazard Index (HI) related with fish and water ingestion among the three communities and the incremental lifetime cancer risk (ILCR) related with fish and water ingestion among the three communities. All analysis were conducted at the 95% confidence level and p-value ≤ 0.05 was considered as being statistically significant.

Ethical considerations

Ethics approval for the research was obtained from the Research Ethics Committee of the University of Port Harcourt (Approval number: UPH/CEREMAD/REC/MM72/097). Permission to conduct the research was also obtained from the Bayelsa State Ministry of Health (Approval number: BSHREC/Vol. 1/21/02). Permission to conduct this study was also sought from necessary authorities of the Communities involved. Every part of the research protocol was explained to the respondents and their consent sought before commencement of instrument administration and health assessment. In the course of collection of samples from the environment, it was ensured that the appropriate techniques were applied and that no harm came to the environment in the course of doing so.

Results

Socio-demographic characteristics of respondents

Out of the 615 respondents who took part in this study, 205 of them were selected from each community. It was found that the majority of the respondents were male 318 (51.7%) and majority of the respondents were aged between 18 and 44 years 490 (82.9%) with a mean age of 32.77 ± 11.13 years. The largest proportion of the respondents had an income of less than 500 naira per day 234 (38.6%), were single 281 (45.7%) and were self-employed 290 (47.2%). Most of the respondents had received secondary school education 319 (52.3%). These details are shown in Table 1.

Table 1: Demographic data of respondents.

Variables	Frequency	Percentage (%)
Community		
Sampou	205	33.3
Gbarain	205	33.3
Nembe	205	33.3
Total	615	100
Sex		
Male	318	51.7
Female	297	48.3
Total	615	100
Age (years)		
18-44	490	82.9
45-60	90	15.2
> 60	11	1.9
Total	591	100
Employment status		
Unemployed	208	33.9
Self-employed	290	47.2
Employed by others	116	18.9
Total	614	100
Income (naira)		
< 500	234	38.6
500-1000	165	27.2
1000-2000	59	9.7
2000-3000	33	5.4
> 3000	116	19.1
Total	607	100
Variables	Frequency	Percentage (%)
Marital status		
Single	281	45.7
Married	270	43.9
Divorced	17	2.8
Widow(er)	8	1.3
Cohabiting	39	6.3
Total	615	100
Educational qualification		
None	30	4.9
Primary	27	4.4
Secondary	319	52.3
Tertiary	206	33.8
Vocational/Technical	28	4.6
Total	610	100

Table 2: Mean and total concentrations of PAHs from fish.

PAHs (µg/kg)	Sampou	Gbarain	Nembe
Total	95.43	64.75	78.35
Mean	5.62	3.81	4.61
Standard deviation	5.92	5.57	5.33
PAH4 index (µg/kg b.w./day)	22.17	11.06	24.39

Polycyclic Aromatic Hydrocarbons (PAHs) concentrations in fish

Assessment of the concentrations of the 16-priority PAHs in the fish obtained from the three study locations showed that the mean concentration in the samples obtained from Sampou (control group) was 5.62 ± 5.92 µg/kg with ΣPAHs of 95.43 µg/kg (highest among the 3 communities) and the PAH4 index of the samples was also found to be 22.17 µg/kg. The mean concentration in fish samples obtained from Gbarain was 3.81 ± 5.57 µg/kg with ΣPAHs of 64.75 µg/kg and the PAH4 index of the samples was found to be 11.06 µg/kg. Finally, the mean concentration in fish samples obtained from Nembe was 4.61 ± 5.33 µg/kg with ΣPAHs of 78.35 µg/kg and the PAH4 index of the samples was found to be 24.39 µg/kg (highest among the 3 communities. These are shown in [Table 2](#).

In fish samples obtained from Sampou, mean concentration of non-carcinogenic PAHs was found to be 4.17 ± 6.91 µg/kg with non-carcinogenic ΣPAHs of 29.16 µg/kg accounting for 30.56% of ΣPAHs. In samples obtained from Gbarain, mean concentration of non-carcinogenic PAHs was found to be 4.09 ± 8.61 µg/kg with non-carcinogenic ΣPAHs of 28.65 µg/kg accounting for 44.25% of ΣPAHs. In samples obtained from Nembe, mean concentration of non-carcinogenic PAHs was found to be 1.84 ± 2.29 µg/kg with non-carcinogenic ΣPAHs of 12.85 µg/kg accounting for 16.40% of ΣPAHs. These are shown in [Table 3](#).

Polycyclic Aromatic Hydrocarbons (PAHs) concentrations in water

Assessment of the concentrations of the 16-priority PAHs in water obtained from the three study locations showed that the mean concentration in the samples obtained from Sampou (control group) was 3.50 ± 4.51 µg/l with ΣPAHs of 59.59 µg/l and the PAH4 index of the samples was found to be 9.32 µg/l (highest among the 3 communities). The mean concentration in water samples obtained from Gbarain was 1.76 ± 4.35 µg/l with ΣPAHs of 29.87 µg/l and the PAH4 index of the samples was found to be 2.03 µg/l. Finally, the mean concentration in water samples obtained from Nembe was 1.90 ± 4.20 µg/l with ΣPAHs of 32.25 µg/l and the PAH4 index of the samples was found to be 1.66 µg/l. These are shown in [Table 4](#).

In water samples obtained from Sampou, mean concentration of non-carcinogenic PAHs was found

Table 3: Mean and total concentrations of carcinogenic and non-carcinogenic PAHs in fish.

PAHs (µg/kg)	Sampou				Gbarain				Nembe			
	Mean	S.D.	Total	Percent (%) of ΣPAHs	Mean	S.D.	Total	Percent (%) of ΣPAHs	Mean	S.D.	Total	Percent (%)
Non-carcinogenic PAHs	4.17	6.91	29.16	30.56	4.09	8.61	28.65	44.25	1.84	2.29	12.85	16.40
Carcinogenic PAHs	6.63	7.22	66.27	69.44	3.61	2.74	36.10	55.75	6.55	6.47	65.50	83.60
	10.8	14.13	95.43	100	7.7	11.35	64.75	100	8.39	8.76	78.35	100

Carcinogenic PAHs: Fluoranthene, Pyrene, Benzo(a)anthracene, Chrysene, Benzo(b)fluoranthene, Benzo(k)fluoranthene, Benzo(a)pyrene, Dibenz(a,h) anthracene, Indeno(1,2,3NDcd)pyrene, Benzo(g,h,i)perylene.

Non-carcinogenic PAHs: Naphthalene, 2-methylnaphthalene, Acenaphthylene, Fluorene, Acenaphthene, Phenanthrene, Anthracene.

Table 4: Mean and total concentrations of PAHs from water.

PAHs (µg/l)	Sampou	Gbarain	Nembe
Total	59.59	29.87	32.25
Mean	3.50	1.76	1.90
Standard deviation	4.51	4.35	4.20
PAH4*(µg/kg b.w. per day)	9.32	2.03	1.66

Table 5: Mean and total concentrations of carcinogenic and non-carcinogenic PAHs in water.

PAHs (µg/kg)	Sampou				Gbarain				Nembe			
	Mean	S.D.	Total	Percent (%) of ΣPAHs	Mean	S.D.	Total	Percent (%) of ΣPAHs	Mean	S.D.	Total	Percent (%)
Non-carcinogenic PAHs	1.57	2.10	10.99	18.44	0.27	0.36	1.90	6.36	0.80	1.80	5.53	17.15
Carcinogenic PAHs	4.86	5.90	48.60	81.56	2.80	5.67	27.97	93.64	2.68	5.41	26.72	82.85
Total	6.43	8	59.59	100	3.07	6.03	29.87	100	3.48	7.21	32.25	100

Carcinogenic PAHs: Fluoranthene, Pyrene, Benzo(a)anthracene, Chrysene, Benzo(b)fluoranthene, Benzo(k)fluoranthene, Benzo(a)pyrene, Dibenz(a,h) anthracene, Indeno(1,2,3NDcd)pyrene, Benzo(g,h,i)perylene.

Non-carcinogenic PAHs: Naphthalene, 2-methylnaphthalene, Acenaphthylene, Fluorene, Acenaphthene, Phenanthrene, Anthracene.

to be 1.57 ± 2.10 µg/l with non-carcinogenic ΣPAHs of 10.99 µg/l accounting for 18.44% of ΣPAHs. In samples obtained from Gbarain, mean concentration of non-carcinogenic PAHs was found to be 0.27 ± 0.36 µg/l with non-carcinogenic ΣPAHs of 1.90 µg/l accounting for 6.36% of ΣPAHs. In samples obtained from Nembe, mean concentration of non-carcinogenic PAHs was found to be 0.80 ± 1.80 µg/l with non-carcinogenic ΣPAHs of 5.53 µg/l accounting for 17.15% of ΣPAHs. This is shown in [Table 5](#).

Calculation of estimated daily intake (EDI)

Assessment of the Estimated Daily Intake (EDI) of PAHs in fish in this study showed that at Sampou the EDI for consumption of fish was highest for acenaphthylene 0.016 ± 0.003 µg/kg/bw/day. Similarly, at Gbarain, the EDI for consumption of fish was highest for acenaphthylene 0.019 ± 0.003 µg/kg/bw/day. From Nembe, the EDI for consumption of fish was highest

for pyrene 0.016 ± 0.003 µg/kg/bw/day. The difference in the mean EDI values for fish ingestion among the communities was not statistically significant. This data is shown in [Table 6](#).

Assessment of the Estimated Daily Intake (EDI) of PAHs in water in this study showed that at Sampou the EDI for ingestion of water was highest for indeno-1,2,3-(cd)pyrene 1.165 ± 0.458 µg/litre/bw/day. Similarly, at Gbarain the EDI for ingestion of water was highest for indeno-1,2,3-(cd)pyrene 0.936 ± 0.517 µg/litre/bw/day and from Nembe, the EDI for ingestion of water was highest for benzo (g,h,i) perylene 0.977 ± 0.499 µg/litre/bw/day. The difference in the mean EDI values for water ingestion among the communities was however statistically significant. This data is shown in [Table 7](#).

Evaluation of the Hazard Index (HI) related with fish ingestion

Evaluation of the non-carcinogenic risk of ingesting

Table 6: Estimated Daily intake (EDI) of PAHs via fish ingestion.

EDI	Sampou		Gbarain		Nembe	
	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation
Naphthalene	0.0003	0.0001	0.0003	0.000	0.0002	0.000
Acenaphthylene	0.0164	0.0031	0.0194	0.0034	0.0052	0.0011
Fluorene	0.0008	0.0001	0.0013	0.0002	0.0009	0.0002
Acenaphthene	0.0010	0.0002	0.0005	0.0001	0.0004	0.0001
Phenanthrene	0.0014	0.0003	0.0006	0.0001	0.0007	0.0002
Anthracene	0.0016	0.0003	0.0004	0.0001	0.0016	0.0003
Fluoranthene	0.0016	0.0003	0.0020	0.0003	0.0016	0.0003
Pyrene	0.0127	0.0024	0.0067	0.0012	0.0155	0.0034
Benzo(a)anthracene	0.0046	0.0009	0.0055	0.0010	0.0061	0.0013
Chrysene	0.0120	0.0023	0.0031	0.0005	0.0125	0.0027
Benzo(b)fluoranthene	0.0009	0.0002	0.0004	0.0001	0.0017	0.0004
Benzo(k)fluoranthene	0.0052	0.0010	0.0027	0.0005	0.0032	0.0007
Benzo(a)pyrene	0.0012	0.0002	0.0002	0.0000	0.0007	0.0001
Dibenz(a,h) anthracene	0.0042	0.0008	0.0026	0.0005	0.0025	0.0005
Indeno(1,2,3-cd)pyrene	0.0106	0.0020	0.0044	0.0008	0.0105	0.0023
Benzo(g,h,i)perylene	0.0030	0.0006	0.0023	0.0004	0.0022	0.0005
Total	0.0774	0.0147	0.0525	0.0093	0.0656	0.0141
Sampou > Nembe > Gbarain (The difference between each was not statistically significant; p-value = 0.586, df:2; Kruskal Wallis: 1.070)						

Table 7: Estimated Daily intake (EDI) of PAHs via water ingestion.

EDI water	Sampou		Gbarain		Nembe	
	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation
Naphthalene	0.005	0.002	0.003	0.002	0.002	0.001
Acenaphthylene	0.351	0.138	0.048	0.027	0.288	0.147
Fluorene	0.022	0.008	0.003	0.002	0.004	0.002
Acenaphthene	0.024	0.010	0.006	0.003	0.004	0.002
Phenanthrene	0.056	0.022	0.004	0.002	0.003	0.002
Anthracene	0.235	0.093	0.026	0.015	0.021	0.011
Fluoranthene	0.035	0.014	0.008	0.005	0.015	0.008
Pyrene	0.368	0.145	0.094	0.052	0.063	0.032
Benzo(a)anthracene	0.197	0.077	0.066	0.036	0.059	0.030
Chrysene	0.232	0.091	0.024	0.014	0.031	0.016
Benzo(b)fluoranthene	0.149	0.059	0.006	0.003	0.007	0.003
Benzo(k)fluoranthene	0.098	0.039	0.051	0.028	0.002	0.001
Benzo(a)pyrene	0.031	0.012	0.008	0.004	0.002	0.001
Dibenz(a,h) anthracene	0.260	0.102	0.099	0.055	0.035	0.018
Indeno(1,2,3-cd)pyrene	1.165	0.458	0.936	0.517	0.396	0.202
Benzo(g,h,i)perylene	0.645	0.253	0.134	0.074	0.977	0.499
Total	3.875	1.523	1.516	0.838	1.909	0.975
Sampou > Nembe > Gbarain (The difference between control and test communities was statistically significant; p-value: 0.015, df:2); Kruskal Wallis: 8.427						

fish in the studied communities was done using the hazard index. The hazard quotient of each PAH that had a corresponding oral reference dose was first obtained by dividing the estimated daily intake of the PAHs by their respective oral reference doses. The hazard index (summation of the hazard quotients of the

individual PAHs) showed that the mean HI associated with consumption of fish polluted by PAHs in Sampou, Gbarain and Nembe were 0.0046 ± 0.0009 , 0.0009 ± 0.0002 and 0.0028 ± 0.0006 , respectively. The difference in the mean HI values for fish consumption among the communities were not statistically significant (Kruskal

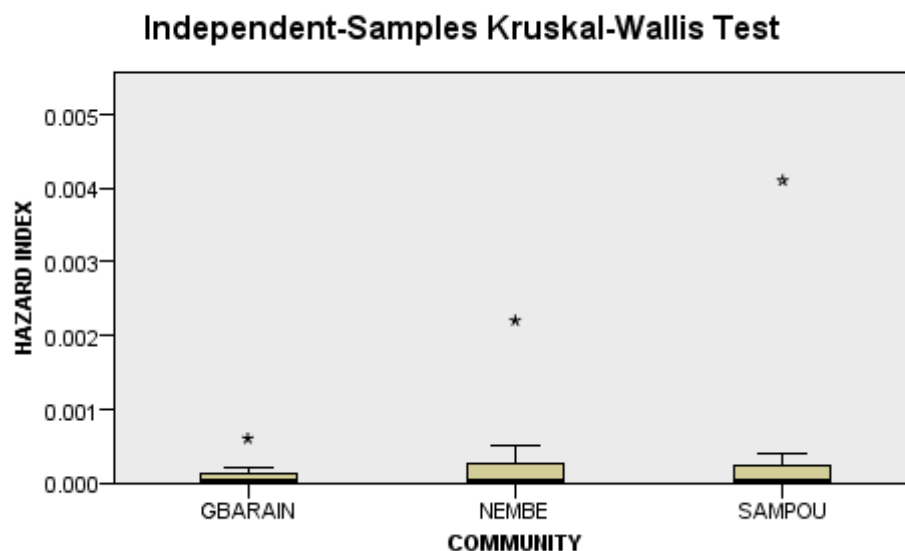


Figure 2: Comparison of HI for eating fish in the three communities.

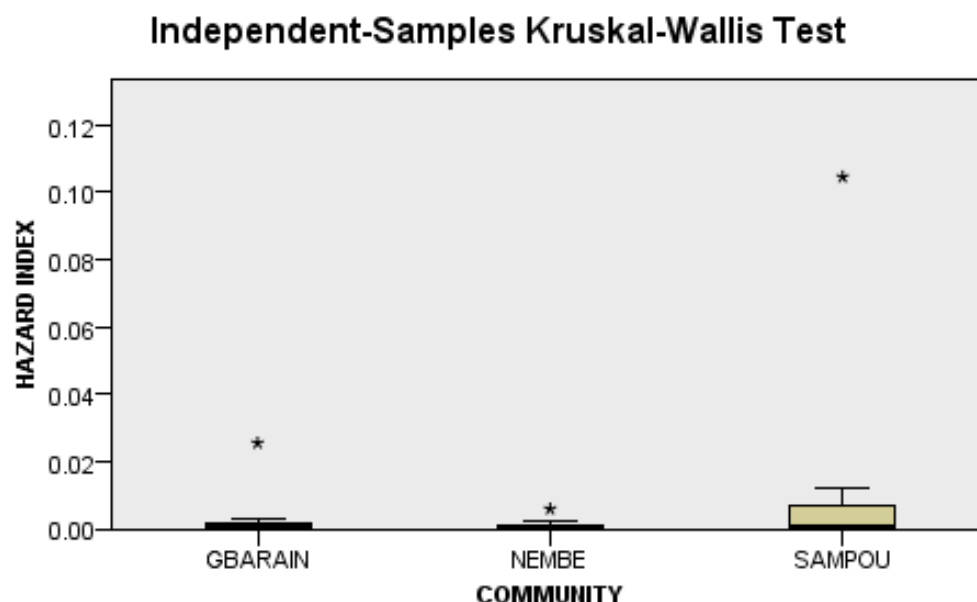


Figure 3: Comparison of HI for drinking water in the three communities.

Wallis: 0.578; p-value: 0.749). This data is shown in [Figure 2](#).

Evaluation of the Hazard Index (HI) related with water ingestion

Evaluation of the non-carcinogenic risk of ingesting water in the studied communities was also done using the hazard index. The hazard index (summation of the hazard quotients of the individual PAHs) showed that the mean HI associated with drinking water polluted by PAHs in Sampou, Gbarain and Nembe were 0.1200 ± 0.0472 , 0.0292 ± 0.0162 and 0.0087 ± 0.0045 , respectively. The differences in the mean HI values for fish consumption among the three communities were not statistically significant (Kruskal Wallis: 4.883; p-value: 0.087). This data is shown in [Figure 3](#).

Cancer risk assessment

Estimation of the incremental lifetime cancer risk (ILCR) associated with ingesting fish or water polluted with PAHs in this study (using their respective slope factors) showed that the mean ILCR values associated with the consumption of fish polluted by PAHs in Sampou, Gbarain and Nembe were $0.010 \pm 0.0019 \times 10^{-3}$, $0.0017 \pm 0.0003 \times 10^{-3}$ and $0.0064 \pm 0.0014 \times 10^{-3}$ respectively. Also, mean ILCR values associated with the consumption of water polluted by PAHs in Sampou, Gbarain and Nembe were $0.3452 \pm 0.1357 \times 10^{-3}$, $0.0636 \pm 0.0351 \times 10^{-3}$ and $0.0179 \pm 0.0091 \times 10^{-3}$ respectively. None of the differences in the mean ILCR among the communities was statistically significant. These data are shown in [Table 8](#).

Table 8: Incremental lifetime cancer risk (ILCR) associated with fish and water consumption.

ILCR (10^{-3})	Sampou		Gbarain		Nembe	
	Mean	Standard deviation	Mean	Standard deviation	Mean	Standard deviation
Fish						
Benzo(b)fluoranthene	0.0007	0.0001	0.0003	0.0001	0.0013	0.0003
Benzo(k)fluoranthene	0.0004	0.0001	0.0002	0.0000	0.0002	0.0001
Benzo(a)pyrene	0.0090	0.0017	0.0012	0.0002	0.0049	0.0010
Total	0.0101	0.0019	0.0017	0.0003	0.0064	0.0014
(Kruskal Wallis: 1.412; p-value: 0.494, df:2)						
Water						
Benzo(b)fluoranthene	0.1089	0.0428	0.0041	0.0023	0.0048	0.0024
Benzo(k)fluoranthene	0.0072	0.0028	0.0037	0.0021	0.0002	0.0001
Benzo(a)pyrene	0.2292	0.0901	0.0558	0.0308	0.0130	0.0066
Total	0.3452	0.1357	0.0636	0.0351	0.0179	0.0091
(Kruskal Wallis: 3.298; p-value: 0.193, df:2)						

Table 9: Benzo[a]pyrene equivalent (B[a]Peq) estimation associated with fish and water.

	Sampou	Gbarain	Nembe
$\Sigma B[a]Peq$	$\Sigma B[a]Peq$	$\Sigma B[a]Peq$	$\Sigma B[a]Peq$
Fish	28.581	18.091	19.005
Water	24.644	11.981	4.067

Benzo[a]pyrene equivalent (B[a]Peq) estimation

Estimation of the benzo[a]pyrene equivalent of the PAHs done by multiplying the individual PAHs concentrations with their Toxic Equivalency Factor (TEF) for the 3 study communities showed that the $\Sigma B[a]Peq$ for the consumption of fish in Sampou, Gbarain and Nembe communities were 28.581, 18.091 and 19.005 respectively. Also, the $\Sigma B[a]Peq$ for the ingestion of water in Sampou, Gbarain and Nembe communities were 24.644, 11.981 and 4.067 respectively. This is shown in [Table 9](#).

Discussion

Evaluation of the hazard index (HI) which is also known as the non-carcinogenic risk posed by exposure to the PAHs in this study showed that the mean HI associated with the consumption of fish and water polluted by PAHs in Sampou, Gbarain and Nembe communities were less than one. This shows that the health of the exposed populations was not at risk due to the consumption of fish and drinking of water from these communities. This finding is in agreement with the findings of the study conducted in China that assessed human and ecological risk of 16 PAHs from a reservoir source of drinking water with a possible PAHs contamination from activities relating to crude oil [41]. In the present study, it was also found that carcinogenic PAHs were major constituents in water in communities where artisanal refining and gas flaring activities were practiced; as well as major constituents in fish for the community where only artisanal refining was known to

be practiced. Benzo(a)pyrene concentration values in water in this study were found to be high in all three communities when compared with the concentration limit of $0.1 \mu\text{g/l}$ [41]. Considering that benzo(a)pyrene is one of the most carcinogenic PAHs and generally used as an exposure marker for risk assessments, the risk of exposure to its carcinogenic effects can be said to be elevated [42]. However, estimation of the benzo[a]pyrene equivalent of the PAHs which is a measure of the carcinogenic potency after exposure to the PAHs for the 3 study communities showed that the $\Sigma B[a]Peq$ for the consumption of fish in Sampou, Gbarain and Nembe communities were $28.581 \mu\text{g/kg}$, $18.091 \mu\text{g/kg}$ and $19.005 \mu\text{g/kg}$ respectively. Also, the $\Sigma B[a]Peq$ for the ingestion of water in the 3 communities were $24.644 \mu\text{g/kg}$, $11.981 \mu\text{g/kg}$ and $4.067 \mu\text{g/kg}$ respectively. These values suggest that the ingestion of the water or fish obtained from these communities does not pose a risk to the health of the inhabitants of the communities considering that they lie below the estimated screening value (SV) of 3.556 mg/kg [39,18]. This is further buttressed by the PAH4 index (which has been described as the level that can serve as a marker for carcinogenic PAHs in food) for both water and fish consumed in these communities which were below the benchmark dose concentration of $0.34 \text{ mg/kg/b.w. per day}$ [43].

Estimation of the incremental lifetime cancer risk (ILCR) in this study however showed that most of the mean ILCR values associated with the consumption of fish and water polluted by PAHs in the 3 studied communities were within the normal limits which would pose no carcinogenic health risk (between 1×10^{-4} and 1×10^{-6}) [17,18] to dwellers in these communities. This is also in agreement with the study conducted to assess human and ecological risk associated with drinking water from a reservoir in which the values of ILCR through both ingestion and dermal adsorption were all less than 1×10^{-4} except for Chrysene [44]. The only

ILCR that was slightly elevated in the present study was related with drinking water from Sampou (the control community): $3.452 \pm 0.1357 \times 10^{-4}$ which is in agreement with reports of increased cancer risk resulting from the ingestion of contaminated sea food with the resultant occurrence of varying susceptibilities [36]. This slightly elevated ILCR occurring in the control community and not in the test communities is however contrary to expectations considering that oil and gas activities are non-existent in the community. A possible explanation for this finding could be the location of the control community. Although, being one of the northern-most communities in Bayelsa state, it shares boundaries with surrounding oil-producing communities in Rivers and Delta states. This means that Sampou community is located downstream in relation to these other communities where oil and gas activities are existent, thus polluting the river water flowing through Sampou and exposing the residents to PAHs [45-47].

The occurrence of potential adverse effects caused by PAHs that do not have reference values have also been reported to be a possibility for having the increased cancer risk in Sampou community [44]. Co-toxicity as a result of different pollutants is also another possibility which can arise from a variety of interactions either directly among the co-occurring pollutants or indirectly through the effect posed by one pollutant on the different processes involved in the transport, metabolism and detoxification of the co-occurring pollutant(s) within life forms [17,18]. The implication of the finding of absent non-cancer and cancer-risks related with ingesting fish and water in this study is that despite the various oil and gas activities that are going on within various parts of Bayelsa state in Nigeria, the health of the residents of involved communities are faced with negligible to no risk. This notwithstanding, it should be noted that the HI parameter does not estimate the risks; it is a proxy indicator of the risk level associated with pollutants exposure [17,18]. It should not be forgotten that PAHs have the capacity to travel long distances and that other harmful pollutants are released into the environment during oil and gas related activities. It is thus necessary that regulations for ensuring environmental safety be adhered to during the conduct of these activities within the oil-rich region of the Niger Delta. Furthermore, illicit activities such as artisanal refining which result in crude oil spillage should be prohibited because apart from environmental pollution problems, occupational exposures during such activities could result in the occurrence of more acute health problems for the artisanal refiners [11,48-50].

Conclusion

The health of the exposed populations was not at risk due to PAHs exposure from the consumption of fish and drinking water from these communities although the ILCR associated with drinking water in Sampou

was slightly elevated. Recommendations included ensuring constant environmental monitoring of water, fish and other environmental media so as to ensure that exposures and associated health risks are kept as minimal as possible. Also, PAHs pollution from other known sources should be minimized as much as possible through the use of more environmentally-safe methods for cooking and waste management.

Study Limitation

Alongside utilizing a food frequency questionnaire to assess the risk which the respondents were exposed to by drinking water and eating fish from the study communities, it would have been ideal to evaluate concentrations of PAHs markers in study respondents. This could however not be achieved in the present study due to limited resources as at the time of conducting the research.

Funding

This work was partly supported by the World Bank Group, Zeelicious foods and FeyiKola foundation.

Author Contribution

All authors listed above contributed equally in the conduct of this research and the development of this manuscript. All authors have read through and agree with the findings in this manuscript.

References

- Boisa N, Ideriah TJK, Okechie CS (2019) Evaluation of polycyclic aromatic hydrocarbons and total petroleum hydrocarbons profiles in some Nigerian crude oils. *J Sci Res Rep* 23: 1-14.
- Lawal AT (2017) Polycyclic aromatic hydrocarbons - A review. *Cogent Environ Sci* 71: 1-89.
- Nwaichi EO, Agbam P, Iwu PI (2017) Polycyclic Aromatic Hydrocarbons (PAHS) and some trace metals in yam, cassava, orange and papaya from two oil and gas flaring impacted communities in southern Nigeria. *J Appl Scie Environ Manag* 21: 1057-1062.
- Singh L, Varshney JG, Agarwal T (2016) Polycyclic aromatic hydrocarbons' formation and occurrence in processed food. *Food Chem* 199: 768-781.
- Padula AM, Balmes JR, Eisen EA, Mann J, Noth EM, et al. (2015) Ambient polycyclic aromatic hydrocarbons and pulmonary function in children. *J Exp Sci Environ Epidemiol* 25: 295-302.
- Adeniji AO, Okoh OO, Okoh AI (2018) Analytical methods for polycyclic aromatic hydrocarbons and their global trend of distribution in water and sediment: A review. *Recent Insight Petrol Sci Engin* 10: 393-428.
- Obenade M, Amangabara GT (2014) Perspective: The environmental implications of oil theft and artisanal refining in the Niger Delta Region. *Asian Rev Environ Earth Sci* 1: 25-29.
- Anyanwu BO, Ezejiogor AN, Igweze ZN, Orisakwe OE (2018) Heavy metal mixture exposure and effects in developing nations: an update. *Toxics* 6: 65.

9. Opio-Odongo J (2013) Africa environment outlook (AEO 3): Our environment, our health; summary for policy makers. UNEP.
10. Ugochukwu CNC (2008) Sustainable environmental management in the Niger Delta region of Nigeria: Effects of hydrocarbon pollution on local economy. BTU Cottbus-Germany.
11. Srogi K (2007) Monitoring of environmental exposure to polycyclic aromatic hydrocarbons: A review. *Environ Chem Letters* 5: 169-195.
12. United-States-Environmental-Protection-Agency (2021) Risk assessment.
13. Ephraim-Emmanuel BC, Ordinioha B (2021) Exposure and public health effects of polycyclic aromatic hydrocarbon compounds in Sub-Saharan Africa: A systematic review. *Int J Toxicol* 40: 250-269.
14. Onojake M, Nwokonko V, Osakwe JO (2020) Human health risks assessment of polycyclic aromatic hydrocarbons in selected seafood from Niger Delta Nigeria. *Niger J Chem Res* 25: 2020-2052.
15. Shi M, Zhang R, Wang Y, Zhang Y, Zhang Y, et al. (2020) Health risk assessments of polycyclic aromatic hydrocarbons and chlorinated/brominated polycyclic aromatic hydrocarbons in urban air particles in a haze frequent area in China. *Emerg Contamin* 6: 172-178.
16. Chem Safety PRO, C (2021) How to Calculate Hazard Quotient (HQ) and Risk Quotient (RQ). Toxicology and Health Risk Assessment.
17. Ozoani H, Ezejiofor AN, Amadi CN, Chijioko-Nwauche I, Orisakwe OE (2020) Safety of honey consumed in Enugu State, Nigeria: A public health risk assessment of lead and polycyclic aromatic hydrocarbons. *Rocz Panstw Zakl Hig* 71: 57-66.
18. Udowelle NA, Igweze ZN, Asomugha RN, Orisakwe OE (2017) Health risk assessment and dietary exposure of polycyclic aromatic hydrocarbons (PAHs), lead and cadmium from bread consumed in Nigeria. *Rocz Państw Zakł Hig* 68: 269-280.
19. United-States-Environmental-Protection-Agency U (1998) Risk and Hazard Characterization. In: Human Health Risk Assessment Protocol, 1-7.
20. Almeda R, Wambaugh Z, Chai C, Wang Z, Liu Z, et al. (2013) Effects of crude oil exposure on bioaccumulation of polycyclic aromatic hydrocarbons and survival of adult and larval stages of gelatinous zooplankton. *PLoS One* 8: E74476.
21. Brandt M, Einhenkel-Arle D (2016) Polycyclic aromatic hydrocarbons harmful to the environment! toxic! inevitable? *German Environ Agency Section IV 2*: 1-26.
22. Amangabara GT, Njoku JD (2012) Assessing groundwater vulnerability to the activities of artisanal refining in Bolo and environs, Ogu/Bolo Local Government Area of Rivers State; Nigeria. *Bri J Environ Climate Change*, 2: 28-36.
23. Kadafa AA (2012) Environmental impacts of oil exploration and exploitation in the Niger Delta of Nigeria. *Glob J Sci Frontier Res Environ Earth Sci* 12: 19-28.
24. Odalonu BH (2015) Upsurge of oil theft and illegal bunkering in the Niger Delta region of Nigeria: Is there a way out? *Mediterran J Soc Sci* 6: 563.
25. Odalonu BH, Eronmhomsele J (2015) The irony of amnesty programme : Incessant oil theft and illegal bunkering in the Niger Delta region of Nigeria. *Int J Humanit Soc Sci Invent* 4: 9-18.
26. Yabrade M, Tanee FBG (2016) Assessing the impact of artisanal petroleum refining on vegetation and soil quality: A case study of warri south west salt wetland of delta state, Nigeria. *Environ Toxicol* 10: 205-212.
27. Niranjana R, Thakur AK (2017) The toxicological mechanisms of environmental soot (black carbon) and carbon black: Focus on oxidative stress and inflammatory pathways. *Front Immunol* 8: 763.
28. Nriagu J, Udofia EA, Ekong I, Ebuk G (2016) Health risks associated with oil pollution in the Niger Delta, Nigeria. *Int J Environ Res Public Health* 13: 346.
29. Okonkwo FO, Njan AA, Ejike CECC, Nwodo UU, Onwurah INE (2018) Health implications of occupational exposure of butchers to emissions from burning tyres. *Ann Glob Health* 84: 387.
30. Ordinioha B, Brisibe S (2013) The human health implications of crude oil spills in the Niger delta, Nigeria: An interpretation of published studies. *Niger Med J* 54: 10.
31. Parent M, Siemiatycki J, Renaud G (1996) Case-control study of exposure to carbon black in the occupational setting and risk of lung cancer. *Am J Industr Med* 30: 285-292.
32. Pandis N (2012) Sample calculations for comparing proportions. *Am J Orthod Dentofacial Orthop* 141: 666-667.
33. Kponee K Z, Chiger A, Kakulu II, Vorhees D, Heiger-Bernays W (2015) Petroleum contaminated water and health symptoms: A cross-sectional pilot study in a rural Nigerian community. *Environ Health* 14: 86.
34. Pandis N (2012) Sample calculations for comparison of two means. *Statist Res Des* 141: 519-521.
35. Adekunle A, Oyekunle J, Oladele A, Ojo O, Maxakato N (2020) Evaluation of Polycyclic Aromatic Hydrocarbons (PAHs) and health risk assessment of surface water and sediments of river sasa, ife north local government area, Nigeria. *Chem Africa* 3: 1109-1122.
36. Aigberua AO (2020) A Survey of concentrations and source characterization of polycyclic aromatic hydrocarbons in surface waters of the imiringi river system. *Int Res J Pure Appl Chem* 21: 71-84.
37. United States Environmental Protection Agency (USEPA) (2001) Separatory funnel liquid-liquid extraction method 3510C.
38. Nekhavhambe T, Van Ree T, Fatoki O (2014) Determination and distribution of polycyclic aromatic hydrocarbons in rivers, surface runoff and sediments in and around Thohoyandou, Limpopo Province, South Africa. *Water SA* 40: 415-424.
39. Tongo I, Ogbeide O, Ezemonye L (2017) Human health risk assessment of polycyclic aromatic hydrocarbons (PAHs) in smoked fish species from markets in Southern Nigeria. *Toxicol Rep* 4: 55-61.
40. Food-Agriculture-Organization (2014) Fishery and aquaculture statistics 2014. Statistics and Information Service of the Fisheries and Aquaculture Department/Service.
41. Ekere NR, Yakubu NM, Oparanozie T, Ihedioha JN (2019) Levels and risk assessment of polycyclic aromatic hydrocarbons in water and fish of Rivers Niger and Benue confluence Lokoja, Nigeria. *J Environ Health Sci Engineer* 17: 383-392.

42. Patel AB, Shaikh S, Jain KR, Desai C (2020) Polycyclic aromatic hydrocarbons: Sources, toxicity, and remediation approaches. *Front Microbiol* 11: 562813.
43. European Food Safety Authority E (2008) Scientific opinion of the panel on contaminants in the food chain on a request from the European Commission on Polycyclic Aromatic Hydrocarbons in food. *EFSA J* 724: 1-114.
44. Sun C, Zhang J, Qiyun M, Yanan C (2015) Human health and ecological risk assessment of 16 polycyclic aromatic hydrocarbons in drinking source water from a large mixed-use reservoir. *Int J Environ Res Public Health* 12: 13956-13969.
45. Honda M, Suzuki N (2020) Toxicities of polycyclic aromatic hydrocarbons for aquatic animals. *Int J Environ Res Public Health* 17: 1-23.
46. Nkwocha EE, Egejuru RO (2008) Effects of industrial air pollution on the respiratory health of children. *Int J Environ Sci Technol* 5: 509-516.
47. Yakubu OH (2018) Particle (soot) pollution in port Harcourt rivers state, Nigeria-double air pollution burden? Understanding and tackling potential environmental public health impacts. *Environ* 5: 2.
48. Ifegwu C, Osunjaye K, Fashogbon F, Oke K (2012) Urinary 1-hydroxypyrene as a biomarker to carcinogenic polycyclic aromatic hydrocarbon exposure. *Biomarkers Cancer* 4: 7-17.
49. Rao PSS, Kumar S (2015) Polycyclic aromatic hydrocarbons and cytochrome P450 in HIV pathogenesis. *Front Microbiol* 6: 550.
50. World-Bank (WB), Institute for Health Metrics and Evaluation (IHME) (2016) The cost of air pollution: Strengthening the economic case for action. Washington, DC, USA.