Carcinogenic Risk Assessment of Polycyclic Aromatic Hydrocarbons in Water from Qua Iboe River Estuary, South-South, Nigeria

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ABSTRACT

This study was conducted to determine the levels of carcinogenic and non-carcinogenic polycyclic aromatic hydrocarbon (PAHs) in water from Qua Iboe river estuary (QIRE), Ibeno-Nigeria and their carcinogenic health risk via ingestion of water from the river. Water samples were collected from five designated stations and one reference station along QIRE during the dry and wet seasons and analysed for PAHs using gas chromatography with flame ionization detector (GC/ FID). Carcinogenic health risk assessment was computed using models recommended by USEPA. Concentration for total PAHs ranged from 6.403E-04 (for the control) to 5.986E-02 mg/l. The total PAHs and total carcinogenic PAHs (ΣC-PAHs) in water at all the sites except the control sites were higher than the standards for European Union and WHO. Values of Benzo(b) pyrene (B(b)F) and Indeno(1,2,3-cd)pyrene (Ind(1,2,3-cd)P) at all the sampling sites for both seasons were above the WHO standard for individual PAHs in coastal and surface water. Carcinogenic risk (CR) values indicated potential risk for B(b)F and Ind(1,2,3-cd)P via ingestion of water and the total CR values at all the sites were above the regulatory limit. This study revealed detectable quantities of PAHs in water at all the sampling sites for both seasons with potential carcinogenic risk specifically for B(b)F and Ind(1,2,3-cd)P and health risk resulting from possible synergistic effect of all the C-PAHs. National legislation to minimise gas flaring and the use of advance PAH treatment technology to minimise the level of pollution of the estuary is recommended.

Keyword: Polycyclic Aromatic Hydrocarbon, Carcinogenic Risk Assessment, Water Pollution, Qua Iboe River Estuary, Gas Flaring, Oil Pollution

INTRODUCTION

Water is one of the most valuable and precious resources on the earth as all living things on earth rely on it for survival. The pollution of water bodies due to disposal of industrial and domestic waste in developing countries has been reported[1]. In the Niger Delta region of Nigeria, oil production activities result in oil pollution from seismic survey, gas flaring, oil spills and effluents from oil field that may discharged directly into water bodies. Oil pollution is the release of contaminants or pollutants associated with the extraction, storage or transportation of crude oil into the environment.

According to Olusi[2], there is a correlation between exposures to oil pollution and development of health problems. Diseases such as cancer, respiratory problems, skin ailments, eye problems and gastro-intestinal disorders and water borne diseases may be linked with oil pollution. Animal studies conducted by feeding rats and other experimental animals with food and water contaminated with crude oil indicate that exposure to Nigerian crude oil could lead to infertility, hemotoxicity, hepatoxicity and carcinogenesis[3,4].

Polycyclic Aromatic hydrocarbons are neutral, non-polar organic compound that consist of two or more fused aromatic rings arranged in various configurations and are known to be ubiquitous in both marine and terrestrial environment [5]. Out of the sixteen PAHs included in the European Union priority pollutant list because of their toxicity and ease of chemical detection, EPA has classified seven PAH compounds as probable human carcinogens. They are: benzo(b) fluoranthene, benzo (a) anthracene, benzo (a) pyrene, benzo(k) fluoranthene, chrysene, dibenzo (a,h) anteracene and indeno (1,2,3-cd) pyrene[6].

Usually oil production activities and crude oil spills result in the release of large amounts of hydrocarbons such as polycyclic aromatic hydrocarbon which contaminates terrestrial and aquatic environment[7]. Acute exposures to aromatic hydrocarbons which are common constituents of oil are known to cause respiratory problems, skin tumours as well as chromosal disorder[8]. High molecular weight

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polycyclic aromatic hydrocarbons are of significant concern because of their carcinogenicity, mutagenic properties and bioaccumulation in organic tissues due to their lipophilic character.

Water from QIRE is used by the people living around vicinity for several activities such as fishing, drinking, water sport, wading and swimming and consequently, they are exposed to several dangers and the risk of getting cancer. Carcinogenic risk assessment (CR) is the incremental probability of an individual developing any type of cancer over a lifetime as a result of exposure to a potential carcinogen[9,10].

Most studies in Qua Iboe river estuary and its associated creeks were carried out on the concentration of PAHs[11,12, 13]. However, there is little or no data available on the carcinogenic risk assessment of PAHs in water from the estuary. This study seeks to quantify and evaluate the human health risk induced by ingestion of PAHs in water from the Qua Iboe river estuary.

METHODOLOGY

Study area and sites description

Qua Iboe river is one of the major rivers in the Niger Delta region of Nigeria. It originates from a waterfall in Umuahia hills in Abia state and flows southwards across Umudike into Akwa Ibom state. Five sampling sites are located at the lower reach of Qua Iboe River in Ibeno local government area close to a petrochemical effluent treatment and discharge plant while the control site is located at Ekpene Ukpa in Etinan local government area of Akwa Ibom State, about 27km from the examined sites and is free from oil exploration and production activities. Ibeno lies on the eastern side of Qua Iboe River about 3km from the river and is one of the largest fishing settlements in the Nigerian coast. The global positioning system (GPS) coordinates of the different sites are:

Okoroutip (4°55'5"N - 7°54'47"E), Ukpenekang (4°27'2"N - 8°3'5"E), Iwochang (4°36'50"N - 7°50'03"E), Douglas creek (4°30'55"N - 8°07'E), Stubb creek (4°34'41"N 7°59'47"E), Ekpene Ukpa (4°47'90"N - 7°50'03"E). Figure 1 is a map of the study area indicating the sampling sites.

SP1 = Okoroutip, SP2 = Ukpenekang, SP3 = Iwoachang, SP4 = Douglas creek, Sp5 = Stubb creek, SP 6 = Ekpene Ukpa

Figure 1. Akwa Ibom State, Nigeria showing study locations

Samples and sampling

Sample containers and glassware for PAH analyses were rinsed with hexane and dichloromethane to remove adhering polar and non-polar compounds[5]. Surface water samples for the analysis of PAHs were collected using an amber-coloured borosilicate 1litre capacity glass bottles with teflon-lined caps to prevent photochemical degradation and 3 drops of 1M HCl was added to prevent bacterial degradation of the sample respectively[14]. Sampling was conducted monthly from November, 2013 to October, 2014. Sub-samples from five points per sampling site were homogenized to form a composite sample. A total of 360 subsamples and 72 composite samples were collected.

Extraction and Clean up

The extraction of the samples was carried out by liquid-liquid extraction method protocol described by Anyakora and Coker[15] and the clean-up was carried out in a column chromatography packed with $Na_2SO_4$and silica gel

Determination of PAHs concentration

The concentration of PAHs was determined using standard protocol described by Essumang *et al*.[16] Separation occurs as the vapour constituents' partition between the gas and liquid phases. The sample was automatically detected as it emerges from the column by a flame ionization detector (FID) by measuring the retention time. Usually the identification of the PAH compounds (analyte peaks) was achieved using Chemstation software and was based on matching their retention time with calibrated PAH standards while quantification was obtained from the corresponding areas of the respective chromatograms. Procedural blanks and solvent blanks were analysed and quantified with no PAHs found in these blanks. Prior to use, the GC was calibrated using a five point calibration curve established using dichloromethane-based standards (Accustandard PAH mix, 1000g/ml in CH_2Cl_2). The coefficient of determination values (R^2) were greater than 0.87. Prior to extraction, four surrogate standards were added to the sample to take care of unusual matrix effect. These internal surrogate standards were acenaphthene-d_{10}, phenanthrene-d10, chrysene-d12 and perylene-d12 from Smart solutions, Florida, USA.

Chromatographic Conditions

The gas chromatograph was Agilent Hewlett Packard 5890 series II, coupled with flame ionization detector (FID) powered with HP Chemstation Rev. A. 09:01 (10206) software, Wilmington, DE, USA. To identify and quantify PAH components, the following GC operating conditions was utilized:

Detector: Hydrogen at 35 ml/min; air at 250ml/mins and nitrogen at 30 ml/min.

Oven: Initial temperature 65°C; final temperature; 325°C, Run time: 30 minute

Inlet: Splitless injection was adopted using a rubber septum and the volume injected was 1l. The inlet temperature was 275°C, with a pressure of 14.8 Psi and total flow rate of 65.4ml / min.

Column: The column was lined with 1,3-dimethyl polysiloxane with capillary HPS type of 30m length, 0.32mm wide bore diameter, 0.25m film diameter

Carcinogenic risk assessment

Carcinogenic risk (CR) values of polycyclic aromatic hydrocarbon in water via ingestion pathway was predicted from their chronic daily intake (CDI) obtained from the equation predicted by [9,17].

CDI x SF……...…….(1)

Where: CR = Cancer Risk; SF = Slope factor' CDI = Chronic daily Intake ingestion pathwa*y

*Slope factor for individual carcinogenic PAHs were used[6]

Chronic daily intake via ingestion were calculated by equation

$$CDI = \frac{C \times IR \times EF \times ED}{Bw \times AT} \quad \text{.....(2)}$$

where CDI is the chronic daily intake via ingestion (mg/kg /day), C is the concentration of PAHs in mg/l, IR is the intake rate (2L per day for adults and 0.61L for children), BW is the body weight of the exposed person (70kg for normal adult and 30kg for children), EF is the exposure frequency (365days/ year), ED is the exposure duration over a life time (70 years for adults and 10 years for children), AT is the averaging time in days (70 years x 365 days/year) for adults and 3650 days for children.

Statistical Analysis

All values are expressed as mean of six determinations ± standard deviation. Student's t-test was used to compare between the mean of total PAH values in both the dry and wet seasons and a $P < 0.05$ was considered statistically significant. Statistical analyses were performed using SPSS statistics 17.0 windows.

RESULTS

Concentration of PAHs

The distribution of PAH at various locations of QIRE for the wet and dry seasons are shown in Tables 1 and 2. The total PAHs for the wet season ranged between 6.4 X 10-4 and 5.45 X 10-2 mg/l. For the dry season the values ranged between 7.87 X 10^{-4} and 5.3 X 10^{-2}. Total PAHs were significantly higher in the studied sites than the control station (Ekpene Ukpa). Comparisons between total PAHs for both seasons revealed that the total PAHS were significantly higher($P< 0.05$) in the dry season than in the wet season at Okoroutip and Ukpenekang. In contrast the total PAHs were significantly higher ($P< 0.05$) in the wet season at Douglass and Stubb Creeks. Carcinogenic PAHs (C-PAHs) were dominant in the water samples accounting for between 87% and 99% of PAHs. The mean total carcinogenic PAHs (ΣC-PAHs) in water from QIRE in both dry and wet seasons are shown in Figures 1 and 2. ΣC-PAHs ranged between 2.82 X10^{-2} and 5.4 X 10^{-2} in the wet season and between 1.32 X 10^{-2} and 4.4 X 10^{-2} for the dry season. For non carcinogenic PAHs the values were between 2.6 X 10^{-5} and 8.1 X 10^{-5}. ΣC-PAHs were significantly higher ($P<0.05$) in the dry season at Okoroutip and Ukpenekang but significantly lower ($P>0.05$) at Douglass and Stubb Creeks compared to the wet season.

Computed Carcinogenic Health Risk Induced by PAHs via Ingestion of Water from QIRE

Table 1. Distribution of PAHs in Water from QIRE during the Wet Season

PAH Mixture	Okoroutip	Ukpenekang	Iwoachang	Douglas creek	Stubb Creek	Ekpene Ukpa
Total PAHs	2.825E-02	3.541E-02	3.477E-02	5.986E-02	5.450E-02	6.403E-04
% non-C-PAHs	0.070	0.107	1.312	0.363	0.407	3.677
% C-PAHs	99.927	99.892	98.687	99.636	99.592	99.322

Table 2. Distribution of PAHs in Water from QIRE during the Dry Season

PAH Mixture	Okoroutip	Ukpenekang	Iwoachang	Douglas creek	Stubb Creek	Ekpene Ukpa
Total PAHs	4.447E-02	5.030E-03	3.297E-02	3.800E-02	1.448E-02	7.872E-04
% non-C-PAHs	1.10	12.5	3.50	7.50	9.10	29.6
% C-PAHs	98.9	87.5	96.5	92.5	90.9	70.4

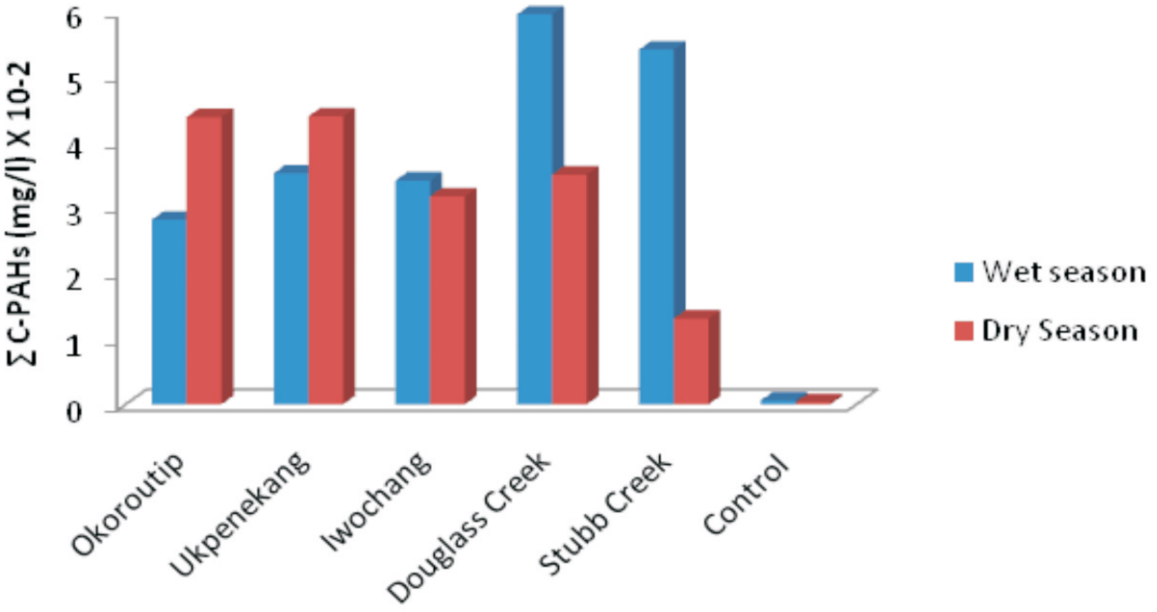

Figure 1. Total carcinogenic PAHs in water from Qua Iboe River Estuary, Nigeria

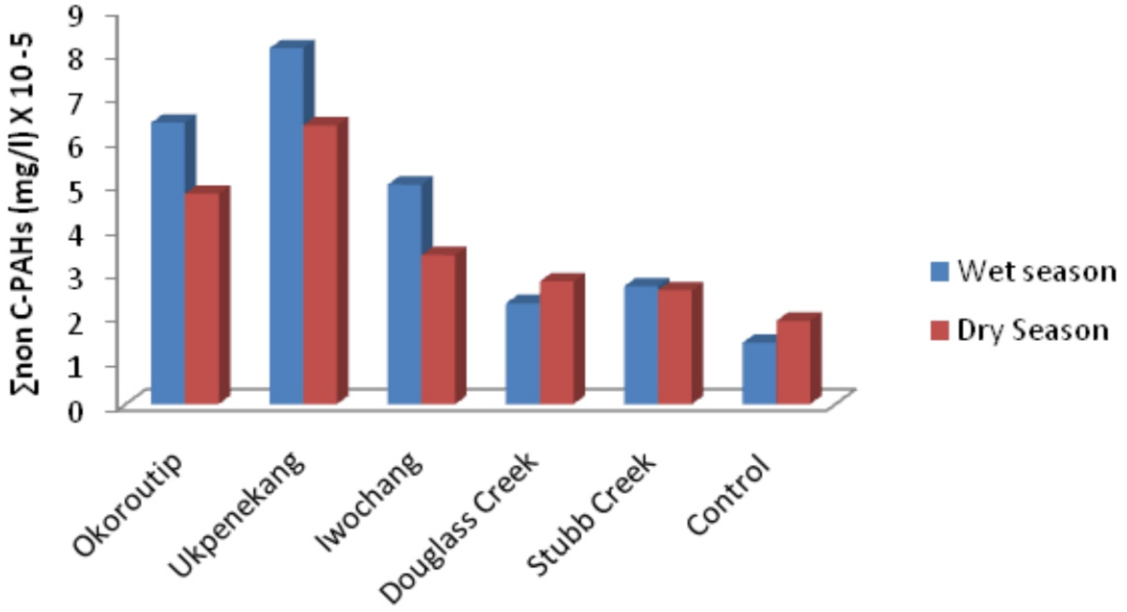

Figure 2. Total non-carcinogenic PAHs in water from Qua Iboe River Estuary, Nigeria

The results obtained from the computation of the carcinogenic risk assessment induced by PAH through the ingestion of water from QIRE in both the dry and wet seasons are shown in Tables 3 and 4. The carcinogenic risk computed for the dry season for adult were between 9.003E - 0.09 to 9.711E-04. The range for the CR values for children during the dry season was between 8.088E-10 and 4.355 E-04. For B(b)F at Iwochang. In the wet season, the range for children was between 2.701 E- 08 and 4.840E -04 while the range for adults was between 9.083E-and 9.771E-04. For both children and adult the CR values for all PAHs were significantly higher ($P < 0.05$) in the sampled station than the control.

The total CR during the dry season ranged from 5.874E-06 to 5.247E -04 for children and 8.2557E-06 to 7.374E-04 for adult with Ekpene Ukpa (control site) and Douglas creek recording the least and highest values respectively. Total CR range for wet season was from 9.278E-06 to 8.906E-04 for children and from 1.303E-05 to 7.207E-03 for adults.

DISCUSSIONS

The concentrations of the carcinogenic

Table 3: Cancer Risk (CR) Values of Carcinogenic PAH in Water from QIRE during the Dry Season for Children and Adults

	Okoroutip		Ukpenekang		Iwoachang		Douglas Creek		Stubb Creek		Ekpene Ukpa	
PAHs	Children	Adult	Children	Adult	Children	Adult	Children	Adult	Children	Adult	Children	Adult
B(a)A	9.578E-08	1.346E-07	1.243E-06	1.747E-06	1.478E-07	2.780E-07	1.273E-07	1.787E-07	8.315E-08	1.168E-07	2.701E-08	3.795E-08
Chrysene	5.112E-08	7.185E-08	2.714E-08	3.814E-08	8.592E-08	1.207E-07	4.838E-08	6.799E-08	2.706E-08	3.804E-08	1.171E-08	1.647E-08
B(b)F	6.810E-05	9.571E-05	2.030E-05	2.853E-05	2.930E-04	4.119E-04	4.848E-04	6.813E-04	1.648E-04	2.317E-04	4.468E-06	6.279E-06
B(k)F	4.188E-07	5.885E-07	6.386E-07	8.974E-06	4.468E-06	6.278E-06	3.714E-07	5.220E-07	6.225E-07	9.552E-07	5.240E-08	7.364E-08
B(a)P	2.148E-06	3.020E-06	4.370E-06	6.142E-06	2.042E-06	2.869E-06	3.493E-06	4.909E-06	3.474E-06	4.884E-06	6.170E-07	8.672E-07
Ind(cd)P	5.833E-05	8.198E-05	4.369E-07	6.140E-07	1.726E-04	2.452E-04	2.850E-05	4.006E-05	3.532E-05	4.963E-05	6.170E-07	8.673E-07
D(ah)A	6.215E-08	8.734E-08	4.177E-05	5.902E-05	6.442E-07	9.053E-07	5.820E-07	8.180E-07	8.325E-07	1.170E-06	8.094E-08	1.137E-07
Total	1.485E-04	1.815E-04	6.900E-05	9.698E-05	4.731E-04	6.648E-04	5.247E-04	7.374E-04	1.983E-04	2.799E-04	5.874E-06	8.255E-06

Table 4: Cancer Risk (CR) Values of Carcinogenic PAH in Water from QIRE during the Wet Season for Children and Adults

	Okoroutip		Ukpenekang		Iwoachang		Douglas Creek		Stubb Creek		Ekpene Ukpa	
PAHs	Children	Adult	Children	Adult	Children	Adult	Children	Adult	Children	Adult	Children	Adult
B(a)A	2.660E-07	3.739E-07	2.545E-07	3.576E-07	1.319E-07	1.853E-07	1.596E-07	2.224E-07	9.127E-08	1.282E-07	6.105E-09	8.580E-09
Chrysene	2.036E-08	2.861E-08	6.463E-09	9.083E-09	1.947E-08	2.736E-08	1.810E-09	2.544E-09	9.198E-09	1.292E-08	8.088E-10	1.136E-09
B(b)F	2.646E-04	3.718E-04	1.384E-04	1.957E-04	4.345E-04	6.106E-04	1.887E-04	2.652E-04	3.076E-04	4.323E-04	2.545E-08	3.576E-08
B(k)F	4.838E-08	6.823E-08	1.153E-07	1.627E-07	4.127E-08	5.820E-08	3.493E-08	4.926E-08	1.475E-06	2.081E-06	2.545E-08	3.089E-08
B(a)P	2.218E-06	3.118E-06	1.246E-06	1.751E-06	3.548E-06	4.986E-06	1.918E-06	2.696E-06	4.194E-06	5.894E-06	1.475E-07	2.073E-07
Ind(cd)P	1.527E-04	2.146E-04	3.849E-04	5.410E-04	7.334E-05	1.030E-04	6.952E-04	9.771E-04	4.953E-04	6.962E-04	8.714E-06	1.224E-05
D(ah)A	3.065E-06	4.300E-06	3.659E-06	5.143E-06	1.081E-06	1.517E-06	4.670E-06	6.563E-06	3.855E-07	5.418E-07	3.592E-07	5.049E-07
Total	4.226E-04	5.942E-04	5.285E-04	7.429E-04	5.126E-04	7.203E-04	8.906E-04	7.251E-03	8.090E-04	1.138E-03	9.278E-06	1.303E-05

PAHs (C-PAHs) in both seasons were higher than the non-carcinogenic PAHs (non-C-PAHs). The European Union permissible limit for total C-PAHs is 100ng/l (10^{-4}mg/l). Apart from the control sites, the concentration of C-PAHs in water samples from all the examined sites in both season were higher than the standard. This may be due to increase anthropogenic activities at the examined site Anyakora *et al*[15]reported that most carcinogenic PAHs show greater resistance to microbial degradation. The result for C-PAHs in this study is similar to the findings of Karyab *et al.*[18].

The concentrations of B(a)P in water from all the stations were below the standard of 0.7µg/l stipulated by Institute of standards and industrial research of Iran [19, 20] in both the dry and wet seasons. The concentration of individual C-PAHs stipulated by WHO in coastal and surface water was reported as 0.05µg/l which is equivalent to 5 x 10^{-5}mg/l[6]. Values of D(ah)A at Ukpenekang in the dry season, B(b)F and lnd (1,23-cd)P for all the station except the control station in both season were above the WHO limit. Similarly, Inam *et al.*[1] recorded high values of B(b)F and lnd (1,2,3-cd) P for PAHs in water from Ogba/ Egbema/ Ndoni communities in the Niger Delta of Nigeria.

Bikey *et al.*[21] stated that individual concentration of C-PAHs above the WHO limit indicates some level of contamination and toxicity. The results obtained from this study imply that the consumption of fish and other seafood from the above sites may be harmful to the consumers since the above-mentioned C-PAHs may gain entry into man. Man may accumulate these PAHs in its adipose tissue and since carcinogenic tendency is transgeneric, cancer prone genes may be inherited by the next filial generation[22].

The high level of PAHs in water especially at Stubb creek, Douglas creek and other examined sites may be due to atmospheric fall-out from gas flaring, burning of domestic waste, waste water from a petrochemical plant discharge plant, leaching and abrasion of coal tar treated boats, creosote treated woods and substantial amount of petroleum products released by water craft engine operating in the area. Essumang *et al.*[16] reported that pathways for PAHs to enter surface water include atmospheric fallout, urban-run off, municipal effluents, sewage discharge, industrial effluents and oil spillage while Ana *et al.*[23] linked the high levels of B(k)F and lnd(1,2,3-cd)P in water from the Niger Delta to run-off from petroleum by-products.

PAHs are potent carcinogens that produce tumors in some organisms even at single dose. They also create advance glycogen end-products which lead to an increase risk of coronary heart disease[16] Laboratory animals exposed to PAHs experience different forms of cancer depending on their pathway of entry into the body or route of administration[23]. Cancer of the larynx in humans and colon cancer are linked with exposure to PAHs through inhalation and ingestion pathways respectively[24].

Carcinogenic risk is the product of the chronic daily intake via the exposure pathway and the carcinogenic slope factor in mg/kg/day[25]. Under most regulatory programs a CR value over 1.00E-05 indicates potential carcinogenic risk while the safe level of risk which requires risk management decision is 10^{-6}[6,26].

In the wet season, B(a)A, B(k)F, B(a)F and chrysene recorded low CR values while most values recorded for B(a)P and D(ah) A were within USEPA's superfund risk level (10^{-6}) and this calls for high risk management decision. Except for the control sites CR values for B(b)F and lnd (1,2,3-cd) P in water from QIRE were quite high indicating the possibility of adverse biological effect. Inam *et al.*[1] recorded high CR values for B(b)F and lnd (1,2,3, cd)P and the low CR values for chrysene and D(ah)A. Similar trend was observed for individual carcinogenic PAHs during the dry season.

Higher total CR values recorded in the wet season compared to the dry season may be due to flooding, possible pipeline vandalisation and oil spillage that occurred in the month of July during the sampling period. Higher total CR values were recorded at Douglas creek and Stubb creek while the control site recorded the least value. This may be due to atmospheric fallout (deposition) from constant gas flaring and sewage. Eduok *et al.*[13] reported that in Douglas creek, much of the PAHs are released into the atmosphere via soots from gas flaring which eventually reaches the water body by direct deposition or deposition on vegetables in addition to inputs from sewage, used motor oil from diffused sources and periodic discharge from oil installations.

The CR values of B(a)F and lnd(1,2,3-cd)P were above the USEPA's regulatory limit. Apart from the control sites and Ukpenekang

during the dry season, the total CR values for the investigated carcinogenic PAHs in most of the sampling sites were above the regulatory limit indicating potential cumulative carcinogenic risk. The result for individual CR values in this study is comparable to the research of other authors[1,9,10].

Falco *et al.*[27] reported that in all carcinogenic PAHs, after metabolic activation in mammalian cells to diol-epoxides and phenol compounds, bind covalently to cellular macromolecules such as RNA, protein and DNA. DNA adducts cause error in DNA replication and mutation which initiates carcinogenic process. The phenyl groups in PAHs attach to nitrogenous base of DNA. This alteration of DNA can trigger initiation of events that results in the growth and replication of neoplastic (cancerous) cells. In this study, the CR values for children were slightly lower than that of adults. This may be due the age-group specific body weight as indicated by other authors [1,9]. However, the susceptibility of children to ingested contaminants is higher because of their high food intake in proportion to their body weight [24].

PAHs are lipid soluble, as a result, they can cross the placenta and may cause genetic damage to the developing fetus. High levels of B(a)P during pregnancy leads to decreased body weight in offspring, lower intelligence quotient and childhood asthma. Children between the ages of 6 and 7 years exposed to high levels of PAHs were found to suffer from anxiety and depression[28]. Prenatal exposure of children to PAHs has resulted in DNA aberration in specific chromosomes, low birth weight, premature delivery, heart malfunction and intra uterine growth restriction[29].

Although the computed CRs in this study revealed carcinogenic risks associated with exposure to individual PAHs at the QIRE, it is unclear at this point if the risks with respect to whole PAH mixture is additive, synergistic or antagonistic. CR estimation assumes that doses of similar chemical components have similar toxicological action and that interactions among PAH mixtures do not occur at levels of exposure typically encountered in the environment[30]. The limitation in this study is that cancer risks from non PAH compounds, heterocyclic, substituted and unmeasured PAHs in the water samples were not estimated.

CONCLUSION

The level of PAHs in water from QIRE and the carcinogenic health risk induced by the ingestion of PAHs in water was evaluated in this study. The total PAHs and total C-PAHs at all the sites except the control site were higher than the standard of European Union and WHO respectively. The values of B(b)F and Ind(1, 2, 3-cd)P at all the sampling sites during both seasons, were above the WHO standard of 5×10^{-5}mg/l for individual PAHs in coastal and surface water. The result for the carcinogenic risk (CR) induced by C-PAHs through the ingestion of water reveals that B(b)F and Ind (1,2,3-cd)P and the total CR values for water in both seasons for all the sampling sites except the control site were above the USEPA regulatory limit. The result of the above investigation reveals that the risk of B(b)F, lnd(1,2,3-cd)P and cumulative risk of other carcinogenic PAHs is higher in the lower reach of QIRE (examined sites) than the control site (Ekpene Ukpa). National legislation to minimise gas flaring and the use of advance PAH treatment technology to minimise the level of pollution of the estuary is recommended.

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