ORIGINAL RESEARCH ARTICLE

Assessment of the health risks associated with human dietary exposure to polycyclic aromatic hydrocarbons in Nile tilapia from Agboyi creek, Southwest Nigeria

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ABSTRACT

Increasing levels of persistent organic pollutants in aquatic ecosystems have been a major challenge in many regions of the world due to their potential adverse effects on ecological receptors and humans via the food chain. This study assessed the risk associated with dietary exposure to polycyclic aromatic hydrocarbons (PAHs) in muscle tissues of Nile tilapia *Oreochromis niloticus* from Agboyi creek in Southwest-Nigeria. The concentrations of PAHs were determined using Gas Chromatography-Mass Spectrometry (GC-MS) following United State Environmental Protection Agency (USEPA) methods. Of the 16 priority PAHs screened, the mean concentration of Acenaphthene (60.51 ± 69.85 µg/kg), the most dominant of all detected PAHs accounted for 19 % of total PAHs while Benzo (a) pyrene with the lowest mean concentration of 0.08 ± 0.17 µg/kg accounted for 0.03 % of total PAHs recorded. Estimated human daily intake (EDI), Hazard Quotient (HQ) and Hazard Index (HI) of PAHs in fish through human consumption were less than the reference dose (RfD) and threshold value. However, obtained toxic equivalent concentration (TEC) for Benzo (b) fluoranthene (35.79 µg/kg) and Dibenz (a, h) anthracene (56.25 µg/kg) as well as the estimated excess cancer risk (ECR) values for 7 most toxic PAHs in fish tissues exceeded the calculated screening value of 0.0027 µg/g and the 'acceptable' range of risk ($> 10^{-6}$) set by the United State Environmental Protection Agency (USEPA) respectively. Dietary exposure to some PAHs recorded in the tissue of Nile tilapia from Agboyi creek may have consequent health implications on the consumers.

Keywords: *Polycyclic aromatic hydrocarbons, Oreochromis niloticus, human health risk*

INTRODUCTION

Increasing levels of potentially toxic substances in aquatic ecosystems and subsequent bioaccumulation in fishery resources raises a major concern due to the potential risks to human consumers, particularly in populations with high consumption rate. Among the complex mixture of toxic substances in the environment, the occurrence and distribution of persistent organic pollutants (POPs) constitutes a major challenge to the health and sustainability of several ecosystems in many regions of the world due their potential adverse effects on organisms and other natural resources. Of particular concern is the levels of poly aromatic hydrocarbons (PAHs) leached daily into rivers, lakes

and oceans from anthropogenic sources such as waste water, industrial effluents and incomplete combustion of fossil fuel and petroleum products (Ekere *et al.,* 2019). According to earlier studies, they have been found at varying concentrations in different environmental matrices (Chen and Liao, 2006; Ezemonye 2006). Nwaichi and Ntorgbo, (2016), also stated in their report that there is an alarmingly high levels of PAH-based pollutants in the aquatic ecosystem due to significant increases in anthropogenic activities along with unavoidable process of biotransformation and biomagnification. And due to their potential carcinogenic, genotoxic and mutagenic effects, the contamination of aquatic ecosystems with PAHs is receiving considerable attention in recent times (Wu *et al.,* 2012; Behera *et al.,* 2018; Ekere *et al.,* 2019; Olayinka *et al.,* 2019). Fish constitutes an important economic resource and a major cultural food in many regions of the world with proven health benefits. A previous report by Xia *et al.* (2010) however noted that dietary intake constitutes a major pathway of PAHs exposure in humans. In addition, increased risks of cancer in humans have been attributed to dietary exposure to elevated concentrations of PAHs (Yoon *et al.,* 2007; Stacewicz-Sapuntzakis *et al.,* 2008). Hence, the risk derived from exposure to chemical pollutants via frequent consumption of fish has been an issue of concern in contrast to the potential health benefits of dietary fish intake (Nwaichi and Ntorgbo, 2016).

In Nigeria, a number of studies have shown a steady increase in the levels of pollutants of priority concerns including PAHs and their bioaccumulation in fishery resources from several ecosystems (Nkpaa *et al.,* 2013; Nwaichia and Ntorgbo, 2016; Tongo *et al.,* 2017; Usese *et al.,* 2017; Igbo *et al.,* 2018; Ekere *et al.,* 2019; Olayinka *et al.,* 2019). The Agboyi creek, a socioeconomically important water body in Southwestern Nigeria and a home to a wide array of fishery resources is also shown to be vulnerable to increasing anthropogenic pressures such as the indiscriminate discharge of untreated domestic and industrial effluents from the surrounding city centers. A previous study revealed contamination of the creek surface water and sediment with organochlorine pesticides (Williams, 2013). However, studies estimating the risk associated with human dietary exposure to reported levels of potentially toxic substances including PAHs in socioeconomically important fish species from Agboyi creek are quite limited. In an effort to aid public health safety, this study was designed to assess the levels and risk associated with human intake of PAHs in muscle tissues of adult tilapia fish, *Oreochromis niloticus* from Agboyi creek.

MATERIALS AND METHODS

Sample Collection and Preservation

Samples of adult *O. niloticus* (n = 40) of average length, 11.3 -15.5cm and corresponding weight of 34.4 - 88.8g were obtained from Agboyi creek during the wet months (May and August, 2019) with the help of a professional fisherman. The fish samples were washed with creek surface water, then wrapped in aluminum foil and immediately transported in polythene bags to the Aquatic Toxicology and Ecophysiology Laboratory located at the Department of Marine Sciences, University of Lagos. In the laboratory, whole fish samples were thoroughly cleaned with distilled water to remove any external dirt and dissected to remove the muscle tissues. They were kept frozen at -4 °C until extraction (Tongo *et al.*, 2018).

Preparation, extraction and clean-up of fish samples

Extraction and pretreatment of samples for PAHs screening followed a step by step procedure for persistent organic pollutants described previously (Tongo *et al.,* 2018; Uyimadu *et al.,* 2018) with slight modification. A homogenous tissue sample was created using a Kenwood commercial-grade food blender (BLP900BK) previously washed with phosphate-free soap and water; then rinsed with hexane between samples to avoid cross contamination. Each sample was grounded to ensure a

homogenous sample. Then 3g of homogenized tissue was weighed into a contaminant-free 150 mL Pyrex Berzelius beaker and mixed with 20g of anhydrous sodium sulfate (sodium sulfate granular, Supelco 2-0296) which had been previously dried by heating to 140ºC overnight. The mixture was stirred frequently, until it was dry and free-flowing, containing no large lumps. The beakers were then numbered with appropriate sample numbers and weighed. Sample extraction was done by a column extraction method using 15 mL of petroleum ether to rinse the column. The sample mixture was then poured into the column, after which 50 mL of acetone/petroleum ether was added to the sample beaker and stirred. The stopcock was closed as the solvent began to elute. At this point, the column was lightly stirred with a glass rod to remove trapped air. Elution was then continued at the rate of 1-2 mL per minute until the solvent level reached the beginning of the sample mixture. Another 50 mL of acetone/petroleum ether was added and elution was continued at the same rate. The stopcocks were rinsed with acetone/petroleum ether to wash any residue lipids or analytes into the concentration tube. The eluent was concentrated to 1 mL by placing the concentrator tubes in a Kuderna-Danish TurboVap concentrator and the extract was then transferred to a 2 mL graduated vial with isooctane. A column chromatography method was used for the clean-up by adding 3 g of activated silica gel which was deactivated with 1 mL distilled water before use. The column was topped with 1 cm of preheated sodium sulfate and then rinsed by eluting twice with 20 ml hexane and discarded. The concentrated extract in iso-octane was transferred to the column and eluted with 50 ml of 20 + 80 DCM / hexane (v/v ratio). The eluent was collected in a 100 ml round bottom flask and then reduced by volume with rotary evaporator to 3 ml. The solvent was then exchanged to iso-octane and the volume further reduced to 1 ml in a stream of nitrogen.

Chromatographic analysis of extract

The final extracts from fish muscle tissues were screened for Naphthalene (NaP), Acenaphthylene (AcPY), Acenaphthene (AcP), Fluorene (Flu), Phenanthrene (Phe), Anthracene (Ant), Pyrene (Pyr), Fluoranthene (FL), Benz[a]anthracene (BaA), Chrysene (Chr), Benzo[b]fluoranthene (BbFL), Benzo[k]fluoranthene (BkFL), Benzo[a]pyrene (BaP), Benzo[g,h,i]perylene (BP), Indeno[1,2,3c,d]pyrene (Ind) and Dibenz[a,h]anthracene (DBA). The analysis of PAHs was carried out on a GC-MS (QP-2010 series, Shimadzu, Japan) following the procedure previously reported by Unyimadu *et al.* (2018). The injector port was set at 300 °C while the oven temperature was held initially at 40 °C and then increased to 120 °C at 25 °C min^{-1}, then to 160 °C at 10°C min^{-1} and finally to 300°C at 5°C min^{-1}. Purified extracts (10 µl) were injected without splitting. The PAHs were separated on a Rxi®-5Sil-MS capillary column (30 m × 0.25 mm i.d. × 0.25 µm film thickness of 1,4-bis (dimethyl siloxy) phenylene dimethyl polysiloxane). Ultrapure helium (99.99%) was used as the carrier gas.

Quality Control

Appropriate quality assurance and controls were performed including analysis of procedural blanks to check for purity of reagents, potential laboratory contamination and inferences. Random duplicate samples were analyzed (standard deviation <5) to check the precision of the instrument. PAHs were quantified using the internal calibration method based on five-point calibration curves for individual compounds. Calculated concentrations were reported as less than the limit of detection if the peak area did not exceed the specified threshold (three times the noise). Concentrations below the detection limit (BDL) were assigned zero values for the statistical analysis. The PAHs were denoted by their International Union of Pure and Applied Chemistry numbers. Genuine standards of PAHs (certified reference standard from Accustandard, New Haven, CT, USA) of various concentrations (20 ppm, 40 ppm, 50 ppm and 80 ppm) were used to calibrate the GC-FID before analysis. Recoveries of authentic standards for the individual target PAHs ranged from 75% to 96%.

Human Health Risk Assessment of PAHs levels in Fish

Potential risk to humans associated with PAH intake via fish consumption was assessed through estimation of dietary intake (EDI), Hazard Index, Toxic Equivalent Quotient (TEQ) and Excess Cancer Risk Further toxicological risk from PAHs levels in fish muscles was assessed by comparison with legal limits (Tongo *et al.*, 2017).

Estimated Dietary intake

The daily intake of PAHs from fish was evaluated by multiplying the respective PAH concentration in each fish sample by the fish ingestion rate (IFR) of an average weight adult (70 kg) from Nigeria using (Eqn. 1) as reported by Nasher *et al.* (2016) thus:

Estimated Dietary Intake (µg/kg/day) = (Ci x IFR) / BW (1)

Where: Ci is concentration of individual PAH in fish

IFR is the consumption rate per person per day (0.036 kg/capita/day) (Worldfish, 2018)

BW is the body weight (average adult weight of 70kg in Nigeria)

Estimation of Carcinogenic and Non-Carcinogenic Risk

According to USEPA (1986), values of HQ and HI of contaminants below one (< 1) are considered as safe whereas there may be cause for concern from potential non-carcinogenic health risks when HQ is > 1. The HQ for carcinogenic and non-carcinogenic risks from exposure to PAHs in fish muscle tissues were calculated using equations 2, 3 and 4:

Hazard quotient (HQ non-carcinogenic) = EDI / RfD (2)
Hazard quotient (HQ carcinogenic) = EDI X SF (3)
Hazard index (HI) = (4)

Where; EDI is the estimated daily intake,

RfD is the reference dose and SF is the slope factor in mg/kg/day which was adopted from United State Environmental Protection Agency (USEPA, 2012) following the report of Li *et al.* (2016). Thus, RFD values used in risk estimation were 0.02, 0.02, 0.06, 0.02, 0.04, 0.3 and 0.04 for NaP, AcPY, AcP, Flu, BaP, Phe, Ant, Pyr and FL respectively. On the hand, SF values used in the estimation of risk posed by consumption of BaA, Chr, BbFl, BkFl, BaP, BP, Ind and DBahA in fish tissues were 0.73, 0.007, 0.73, 0.073, 7.3, 0.04, 0.73 and 7.3 respectively.

To estimate the carcinogenic potency from exposure to PAHs in fish, the toxic equivalent quotient was calculated using equation 5 and 6.

$$TEC = TEF \times C_iPAH \quad (5)$$

Where: TEC is the toxic equivalent concentration of individual PAH
TEF is the toxic equivalence factor of individual PAH.

CiPAH is the concentration of individual PAH in fish muscle tissues.

The values of TEF for 7 carcinogenic PAHs (BaA, Chr, BbF, BkF, BaP, InP and DBahA) used in the estimation are 0.1, 0.01, 0.1, 0.01, 1.0, 0.1 and 5.0 respectively (Nisbet and Lagoy 1992).

TEQ = (6)

Where: TEQ is the sum of the toxic equivalent concentration (TEC) of PAH in fish.

The evaluated TEQ value was also compared with a calculated screening value (SV) to assess human health risks posed by PAHs to humans from consumption of fish. According to a previous study, SV is the threshold concentration of total PAHs in fish tissue that is of potential public health concern (Nyarko and Klubi, *et al.,* 2011). The SV was calculated using Eqn. 7 (Tongo *et al.,* 2017).

SV (µg/kg) = [(RL/SF) × BW]/CR (7)

Where:

RL = maximum acceptable risk level (10^{-5} USEPA, 2000)

SF = USEPA oral slope factor (7.30 µg/g day USEPA, 1993)

BW = body weight (70000 g)

CR = fish consumption rate (36 g/day Worldfish, 2018).

The Excess cancer risk was also calculated using the equation below (Tongo *et al.,* 2017; Xia *et al.,* 2010)

ECR =

Where: TEC is the toxic equivalent concentration of individual PAH
IFR is the consumption rate per person per day (0.036 kg/capita/day) (Worldfish, 2018)

BW is the body weight (average adult weight of 70 kg in Nigeria)

Q is the cancer potency of BaP which was assessed as 7.3 µg/g/day by the integrated risk information system of the USEPA (USEPA, 2001).

ED is the exposure duration (70years)

ATn is the average life span (25550 days)

Statistical Analysis

Data obtained from the analysis of duplicate samples were subjected to descriptive statistics and analysis of variance (ANOVA) at 0.05 ($P < 0.05$) level of significance using Microsoft Excel and the Statistical Package for the Social Sciences software (SPSS) version 20.0 for Windows respectively. The results are reported as mean ± standard deviation.

RESULTS

The mean concentrations of total PAHs obtained as 17.74 ± 16.30 µg/kg was observed in the order AcP > BbFL > Bp > BkFL > NaP > Flu > AcPy > Chr > Ant > Ind > Pyr > DBA > Phe > BaA > Bap > Fl in fish muscle tissues. Of all the detected individual PAHs congeners in examined fish muscles, Acenaphthene (AcP) with a mean concentration of 60.51 ± 69.85 µg/kg was the most dominant PAHs (Fig.1). Fluoranthene was below the detection limit (0.002 µg/kg) used in the study. The result also showed relatively low but measurable concentrations of Benzo[a]pyrene (BaP) in examined fish

muscles with a total mean value of 0.084 ± 0.17 µg/kg. Mean concentrations for total carcinogenic PAHs (sum of BaA (1. %), Chr (15%), BkFL (22.2%) BaP (0.1%), BbFL (23.7%), Ind (7.8%), DBA (7.4), BP (22.8) accounted for 47% of the total PAHs in fish.

Generally, the total concentration of higher molecular weight (HMW) PAHs obtained as 162.87 µg/kg was relatively higher than the total lower molecular weight (LMW) PAHs in fish (156.47 µg/kg).

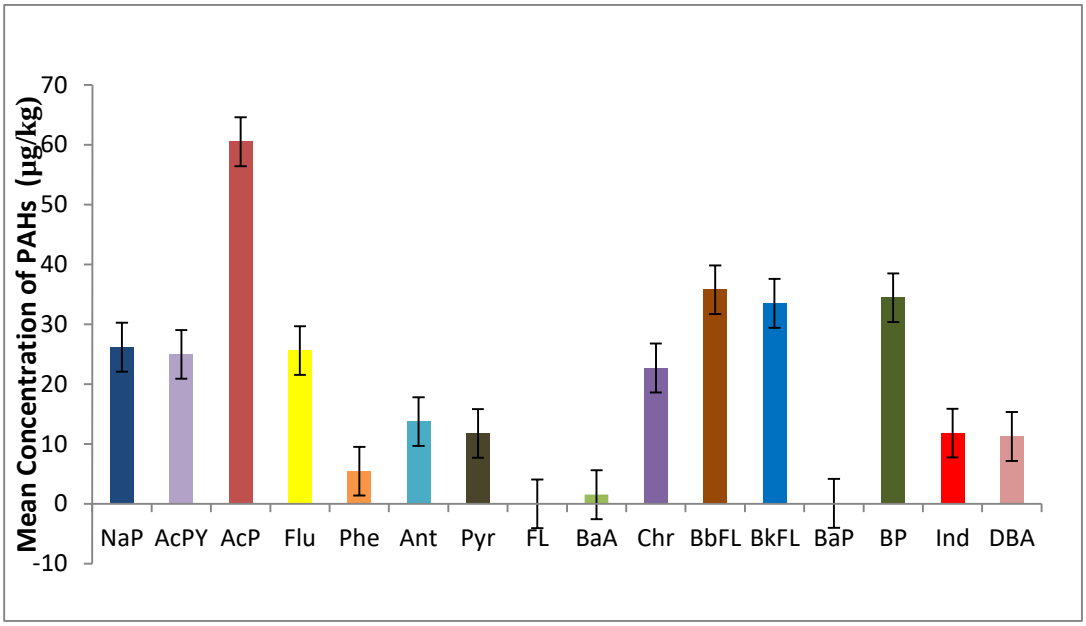

Figure 1: Mean concentration of PAHs in muscle tissues of *Oreochromis niloticus*from Agboyi creek

Human Health Risk

The mean EDI for the seven most toxic PAHs in fish muscle tissues varied from 4.0×10^{-5} to 1.8×10^{-2} µg/kg bw/day. Relatively higher EDI values were recorded for Benzo(b)fluoranthene and Benzo (g, h, i,) perylene, while Benzo (a) pyrene recorded the lowest EDI value of 4.0×10^{-5} µg/kg bw/day (Table I)

Potential Non-Dietary Risk Exposures

The results of non-dietary exposures estimated using HQ for individual PAHs in fish muscle tissue and HI through consumption of fish by adult consumers are presented in Table I. HQ and HI were observed to be less than the threshold value of 1. For carcinogenic risk, the estimated excess cancer risk values for individual toxic PAHs congeners varied from $8.7 \times 10^{-7}$to $5.8. \times 10^{-4}$, all exceeding the USEPA acceptable guideline value of 1×10^{-6} except for Benzo (a) pyrene (Table 1).

Table I: Estimated daily intake, Hazard quotient, Hazard index and Excess cancer risk for 7 most toxic

PAHs in *Oreochromis niloticus* from Agboyi creek.

PAH	Chr	BbFL	BkFL	BaP	BP	Ind	DBA
Estimated dietary intake (µg/kg bw/day)	0.012	0.018	0.017	4.0E-05	0.018	0.006	0.006
Cancer slope factor (mg/kg/day)	0.007	0.73	0.073	7.3	NA	0.73	7.3
Hazard quotient (HQ)	8.2E-08	0.013	0.0013	0.0003	0.129	0.0044	0.042
Excess cancer risk	2.30E-06	3.70E-05	3.4E-05	8.7E-07	3.50E-06	1.2E-05	0.00058
Hazard Index ($\sum_{i=1}^{n} HQ$)	0.19						

An estimated SV value of 0.0027 µg/g was obtained in the present study. Further risk estimation revealed relatively higher toxic equivalent concentration (TEC) values for Benzo (b) fluoranthene (3.58), Benzo (k) fluoranthene (3.35) and Dibenz (a, h) anthracene (56.25) respectively when compared to the SV (Table II).

Table II: Toxic equivalent concentration TEC and Toxic equivalent quotient TEQ for 7 most toxic PAHs in *Oreochromis niloticus*

PAH	Mean (ug/kg)	TEF (Nisbet and Lagoy 1992)	TEC
Chr	22.7 ± 25.19	0.01	0.227
BbFL	35.79 ± 41.24	0.1	3.579
BkFL	33.51 ± 38.57	0.1	3.351
BaP	0.084 ± 0.17	1	0.084
BP	34.45 ±39.33	0.01	0.344
Ind	11.81 ±13.53	0.1	1.181
DBA	11.25 ±13.02	5	56.25
Mean ± SD	21.37 ± 14.0		
TEQ	65.46		

DISCUSSION

Dietary pathways have been identified as a predominant exposure route of contaminants including PAHs in humans (Cheung, *et al.*, 2007; Wu *et al.*, 2012). In the present study, examined fish muscle tissues had relatively low but measurable concentrations of detected USEPA priority PAHs; thus, signifying the potential contamination of Agboyi creek with PAHs. The absence or rather low detection of certain PAHs in fish tissues may also be attributed to their rapid depuration or biotransformation (Dhananjayan, and Muralidharan, 2012). Furthermore, various factors including route and duration of exposure, lipid content of tissues, environmental factors, differences in species,

age, and sex, as well as exposure to other xenobiotics may influence the accumulation and depuration of PAHs in fish (Dhananjayan, and Muralidharan, 2012).

Generally, measured PAHs composition showed a considerable predominance of higher molecular weight PAHs suggesting anthropogenic origin. According to Nwaichia and Ntorgbob (2016), high concentration of heavy molecular weight PAHs indicates a predominant pyrolytic origin for the PAHs pollution. Elsewhere, significantly higher mean percentage concentration of higher molecular weight PAHs, accounting for 88% of the total PAHs in fish has been reported (Tongo *et al.* 2018). Although compositional profile of PAHS in environmental and biological samples are frequently used to identify potential sources of PAHs, Pulster *et al.* (2020) however noted that such comparisons are difficult to assess within organisms due to the complex interactions and species-specific differences between bioaccumulation (e.g., uptake, metabolism and elimination) and the physiochemical parameters affecting chemical exposure and bioavailability in the surrounding environment.

The levels of most of the USEPA priority PAHs detected in this study is lower when compared to the levels observed in muscle tissues of *C. gariepinus* from Ovia River in Southern Nigeria by Tongo *et al.* (2017). In their report, it was observed that the concentration of the lower molecular weight PAHs (LWPAHs) was higher than the higher molecular weight PAHs (HWPAHs) in fish muscle tissues. This was attributed to the omnivorous and detritus feeding habit of *C. gariepinus* as well as the lipophilic nature of PAHs. Relatively higher concentrations of individual PAHs congeners when compared to the results of the present study were also reported in fish muscle tissues from crude oil polluted waters of Ogoniland (Nkpaa, *et al.,* 2013).

In addition, Benzo (a) pyrene (B(a)P) usually used as a marker for the occurrence and effect of carcinogenic PAHs in food (Tongo *et al.,* 2017) were recorded in low but measurable concentrations. The observed BaP levels when compared to levels reported elsewhere (Tongo *et al.,* 2017) was relatively low and did not exceed the existing EU recommended safe limit of 0.002mg/kg for human fish consumption. However, the relatively low levels recorded might still pose unacceptable adverse effects in human consumers and populations with high fish consumption rate. Similarly, higher levels of Flu have been reported in fish tissues by Ohiozebau *et al.* (2017) contrary to the result of the present study. Generally, the obtained levels for individual PAHs congeners in fish muscle tissue varied considerably when compared to previously reported PAHs levels in fish from most contaminated ecosystems in Nigeria (Tongo *et al.,* 2017; 2018; Nkpaa *et al.,* 2013).

The results of estimated human daily intake of PAHs, HQs and HI values for PAHs in examined fish which were lower than the reference dose (RfD) and set threshold value of 1 indicates no potential adverse health effect in consumers. This finding is similar to an earlier report for fish and shellfish from Amariaria Community, downstream of Bonny River, Southern Nigeria (Tongo *et al*., 2018). On the contrary, the toxic equivalent concentration values of Benzo (b) fluoranthene, Benzo (k) fluoranthene and Dibenz (a, h) anthracene which exceeded the calculated screening value, indicates the potential for carcinogenic risk in adult consumers over a life time of exposure. Except for Benzo (a) pyrene, the estimated excess cancer risk (ECR) from lifetime exposure to the 7 most toxic PAHs through fish consumption was also slightly higher than the acceptable risk value ($> 10^{-6}$). This has implications for consumer safety and calls for concern.

CONCLUSION

The present study detected 15 PAHs in muscle tissues of *Oreochromis niloticus* from Agboyi Creek with higher molecular weight PAHs being the most predominance in the fish samples. Estimation of noncarcinogenic health risk indicates no potential negative effects in humans. However, the obtained concentrations of Benzo (a) pyrene (BaP) and the toxic equivalent concentration (TEC) values for

Benzo (b) fluoranthene, Dibenz (a, h) anthracene which exceeded EU recommended safe limit and USEPA screening value in fish respectively, as well as the estimated excess cancer risk (ECR) for 7 most toxic PAHsin adult consumers over a life time exposure may have implications for consumer safety. Hence, there is need for continuous monitoring and stringent environmental regulations in the area.

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