Evaluation of Polycyclic Aromatic Hydrocarbon Content of Selected Fin and Shell Fish from Named Rivers in Ogoniland, Rivers State, Nigeria

Jumbo Adata Akie¹, M.O Wegwu², D. C. Belonwu², B. M. Onyegeme-Okerenta² and C. T. Iriakuma¹

¹Federal Polytechnic of Oil and Gas, Bonny, Nigeria.
²University of Port Harcourt, UNIPORT, Nigeria.

Authors’ contributions
This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT
Polycyclic aromatic hydrocarbon (PAH) content in selected fin and shell fishes from Bodo and Kaa in Ogoniland were studied. PAH was determined by Gas chromatography, using Texas Natural Resource Conversion Commission, Texas (TNRCC TX) method. There was a total of 16 PAHs detected in the samples from the two sites, test site (Bodo) and control site (Kaa). Similar PAH accumulations were observed in the four species, but the concentrations of the PAH accumulations were different. For samples from Kaa, Mullet showed the highest total mean concentration of PAHs followed by Sompat grunt, Tilapia and the least was Shrimps. However, for samples collected from Bodo, Tilapia showed the highest total mean concentration of PAHs, followed by Sompat grunt, Shrimps, and the least Mullet. These findings were not definitive as to the source of the PAH, seemingly suggesting various or multiple sources of PAHs contamination in the studied sites. These variations may be attributed to their feeding habits. The mean and standard deviations for PAHs from the test site ranged from 0.08±.000 to 23.7±.473, 0.05±.001 to 7.74±.346, 0.02±.001 to 9.48±.002, 0.07±.000 to 11.0±.029 for Tilapia, Mullet, Shrimps and Sompat grunt.

*Corresponding author: E-mail: adatajumbo@gmail.com;
respectively and $0.18 \pm 0.006^a$ to $6.56 \pm 0.064^a$, $0.08 \pm 0.000^a$ to $11.8 \pm 0.555^a$, $0.05 \pm 0.002^a$ to $3.11 \pm 0.036^a$, $0.05 \pm 0.002^a$ to $5.12 \pm 0.059^a$ for samples of Tilapia, Mullet, Shrimp and Sompat grunt respectively from the control site. In conclusion, the calculated potency equivalence concentration (PEC) for all the tested aquatic species collected from Kaa and Bodo were all above the screening value (SV) suggesting that the consumption of these aquatic species from the test and control site at a rate of 68g/day in an adult of about 60kg will expose the individual to a potential risk of cancer.

Keywords: Fin fish; shell fish and polycyclic aromatic hydrocarbons; contamination; potency equivalence concentration.

1. INTRODUCTION

PAHs are ubiquitous environmental pollutants, resulting from the incomplete combustion or pyrolysis of organic matter during industrial processing and various human activities. These compounds (PAHs) are important environmental pollutants, because of their ubiquitous presence and carcinogenicity and are thus considered the most toxic in the hydrocarbon family [1]. The sources of PAHs in the coastal environment are described as either petrogenic (if the source is derived from petroleum, e.g., natural) or pyrogenic (if the source is derived from the incomplete combustion of organic matter and fossil fuel [2,3]. The ratio of low molecular weight PAHs (HMW-PAHs) to high molecular weight PAHs (LMW-PAHs) has been used to characterize the origin of PAHs in the environment [4]. Petrogenic sources of PAHs show characteristically higher proportion of LMW-PAHs such as naphthalene and acenaphthenes while pyrogenic PAHs have characteristically higher proportion of HMW-PAHs such as pyrene and benzo[a]pyrene. Thus, petrogenic sources of PAHs exhibit LMW/HMW ratios > 1, whereas pyrogenic sources of PAHs exhibit LMW/HMW ratios < 1 [5].

In addition to the LMW/HMW ratios, isomeric ratios of PAHs have been widely used as indices for the identification of PAH sources in the environment [6]. For instance, Benzo[a]anthracene/ (Benzo[a]anthracene+Chrysene) i.e. (BaA/(BaA + Chry) ratio >0.35 indicates pyrogenic or combustion sources while a ratio < 0.20 has been attributed to petrogenic sources although these sources are indistinguishable for ratios in the range 0.20–0.35 [6,4].

Fish can easily bio-accumulate PAHs from water through their gills and skin [7]. They also ingest PAH-contaminated particle matter along with their food when they feed [8,9], especially soil sediments [10]. PAHs are lipophilic and so they accumulate in the fatty tissues of fish following their uptake [11]. Different species of fish bioaccumulate PAHs to different degrees. These different degrees of bioaccumulation of PAHs expressed by different species of fish is a reflection of their different feeding habits [12].

2. METHODOLOGY

2.1 Reagents

All reagents used were of analytical standard

2.2 Study Sites

The study sites Bodo (test) and Kaa (control) are located in Ogoniland of Rivers State, with a population of about 832000, and land area covering 1000km².

2.3 Collection of Samples

Fresh samples of selected fin and shell fish were collected from Bodo and Kaa Rivers of Gokana and Khana local government areas of Rivers State, Nigeria. 10 samples of each fish species were collected at each sight. The collected samples were cleaned and wrapped in aluminium foil plates, and cooled in an ice chest at -15°C, before transportation to the laboratory. The identification of fish samples was done in the Department of Fisheries, Faculty of Agricultural Science, University of Port Harcourt.

2.4 Sample Preparation

Fresh fish samples were oven dried, and ground to powder using a wedgwood mortar and pestle, and then kept in an air tight container ready for extraction.

2.5 Extraction Procedure

Two grams of sample was weighed into a clean cylindrical conical flask. Extraction solvent (dichloromethane) 10mls, was added into the samples and mixed thoroughly, and then allowed to settle. The mixtures were carefully filtered into clean solvent rinsed extraction bottles using filter paper fitted into Buchner funnels. After extraction, the filtrates were concentrated to 2ml and transferred for clean-up / separation.
Fig. 1. Map of Ogoniland showing the study sites; Kaa (Khana L.G.A) and Bodo (Gokana L.G.A) [13]
2.6 Clean Up / Separation

One cm of moderately packed glass wool was placed at the bottom of 10mm ID x 250mm long chromatographic column. Slurry of 2g activated silica in 10ml methylene chloride was prepared and placed into the chromatographic column. To the top of the column was added 0.5cm of sodium sulphate. The column was rinsed with additional 10ml of methylene chloride. The column was pre-eluted with 20ml of dichloromethane. This was allowed to flow through the column for about 2 minutes, until the liquid in the column was just above the sulphate layer. Immediately, 1ml of the extracted sample was transferred into the column. The extraction bottle was rinsed with 1ml of dichloromethane and added to the column as well. The stop cock of the column was opened and the eluant was collected with a 10ml graduated cylinder. Just prior to the exposure of the sodium sulphate layer to air, dichloromethane was added to the column in 1 – 2ml increments. Accurately measured volumes of 8 – 10ml of the eluant were collected and labelled aliphatic.

2.7 Gas Chromatographic Analysis

The concentrated aliphatic fractions were transferred into labelled glass vials with rubber crimp caps for GC analysis. 1µl of the concentrated sample was injected by means of a hypodermic syringe through a rubber septum into the column. Separation occurred as the vapour constituent partitioned between the gas and liquid phases. The sample was automatically detected as it emerged from the column at a constant flow rate by the flame ionization detector (FID) whose response is dependent upon the composition of the vapour.

2.8 Chromatographic Conditions

The gas chromatography was Hewlett Packed 5890 series II, gas chromatography apparatus, coupled with FID (Hewlett Packard, Wilmington, DE, USA), powered with HP ChemStation Rev. A 09:01 (10206) software to identify and quantify compounds. The GC operating conditions were as follows: fused silica column (30m*0.25µmfilmof HP-5(thickness)); the inlet and injection temperature were set at 275°C to 310°C. Split injection was adopted with a split ratio of 8:1, using rubber septum and volume injected was 1µl. The column temperature was programmed as follows: held at 65°C for 2min; 65-260°C at 12°C /min; 260-320°C at 15°C /min and maintained at 310°C for 8 minutes and oven temperature was set at 65°C. Nitrogen was used as carrier gas. The hydrogen and compressed air pressure were 30psi. The oven initial temperature was at 65°C. Verification of peaks was carried out based on retention times compared to those of external PAHs. Procedural blank and solvent blanks were analysed and quantified, but no PAHs were found in these blanks.

3. RESULTS

Estimation of the carcinogenic risk from exposure to PAHs in fish, was done by following the USEPA guideline, as described by Cheung et al. [14] In this method, Benzo[a]Pyrene is used as a marker for the occurrence and effect of carcinogenic PAHs in foods and, therefore, the overall carcinogenic health risk from the measured PAHs was estimated based on toxic equivalency factors (TEFs) derived from the cancer potencies of individual PAH compounds relative to the cancer potency of Benzo[a]Pyrene. The product of the PAH concentration (µg/g) and
its TEF gives a Benzo[a]Pyrene equivalent concentration (BaPeq) for each PAH. All the individual Benzo[a]Pyrene equivalent concentrations were then summed up to give a carcinogenic potency equivalent concentration (PEC) of all the PAHs according to equation (1) [15].

\[
\text{PEC} = \sum (\text{TEF} \times \text{Concentration})
\]

Potency equivalent concentration values were then compared with a screening value for carcinogenic PAHs. The screening value was calculated from Equation (2) Russell et al. [16].

\[
\text{SV} = \left( \frac{\text{RL}}{\text{SF}} \times \text{BW} \right) / \text{CR}
\]

Where SV = screening value (µg/g)
RL = maximum acceptable risk level (dimensionless)
SF = USEPA oral slope \(^{-1}\) factor (µg/g day)
BW = body weight (g)
CR = consumption rate (g/day).

Screening value (SV) is the threshold concentration of total PAHs in fish tissue that is of potential public health concern; BW is the average body weight (g) and was set to 60000 g (i.e., 60 kg) for the adult population, CR is the consumption rate (g/day). Fish consumption rate was set to 68.5 g/day from the annual per capita fish consumption of 25 kg for Nigeria. RL is the maximum acceptable risk level (dimensionless), which was set to \(10^{-5}\) [17] so that the maximum risk would be one additional cancer death per 100000 persons, if an adult weighing 60 kg consumed 68.5 g of fish daily with the same measured concentrations of PAHs for 70 years; SF is the USEPA oral slope factor for PAHs, used to estimate an upper-bound probability of an individual developing cancer as a result of a lifetime (70 years) exposure to carcinogenic PAHs and has a value of \(7.30\) (µg/g day)\(^{-1}\) [18]. For safety reasons, a consumption rate of 1 g/day was used to estimate the minimum level that a consumer may be protected from the carcinogenic effects of PAHs detected in these fishes.

3.1 PAHs Levels in Fish species

3.1.1 Tilapia guineensis (Tilapia)

A total of 16 PAHs were detected in the samples from the two sites. The mean PAH concentrations for Tilapia ranged from below detection limit of 0.0001 to 23.7±0.473. The highest mean PAH concentrations were recorded for Benzo(K)Fluoranthene, at 6.56 ±0.064 for tilapia collected from Kaa, and 23.7±0.473 for tilapia collected from Bodo. All mean PAH concentrations in tilapia from Kaa were significantly lower at (p<0.05) than PAH concentrations in Tilapia from Bodo, except for Acenaphthalene, acenaphthene and Benzo(a)anthracene, which were below detection level for tilapia collected from both sites (Kaa and Bodo), and Anthracene, Fluorene and pyrene, which were significantly higher at p<0.05 for samples collected from Kaa than samples collected from Bodo. The total PAH concentrations were 21.0±0.221 and 39.8±0.519 for samples collected from Kaa and Bodo respectively.

The calculated potency equivalence concentration (PEC) for tilapia collected from Kaa and Bodo were 2.08 and 5.68 respectively, which is very much higher than the screening value (SV), thus indicating that the consumption of tilapia from both sites at a rate of 68g/day in an adult of about 60kg, exposes the individual to a potential risk of cancer [4].

The LMW-PAH/HMW-PAH ratio for tilapia collected from Kaa and Bodo were <1, indicating that the sources of these PAHs in the fish are mainly pyrogenic [5,19] a clear indication of anthropogenic pollution of PAHs [20].

The BaA/(BaA+Chry) ratio for tilapia collected from Kaa and Bodo were both zero (0), suggesting that the source of PAH input is petrogenic in contrast to the LMW-PAH/HMW-PAH ratio. The mean concentration of Benzo (a) pyrene in tilapia from Kaa and Bodo was 0.44±0.007 and 1.21±0.012 respectively, and were both below the European Union (EU) permissible limit of 2µg/kg [21] suggesting that consumption of tilapia from Kaa and Bodo will not predispose the consumer to cancer, in contrast to the PEC values which suggest otherwise.

3.1.2 Liza falcipinis (Mullet)

The mean PAH concentrations for mullet ranged from below detection limit of 0.0001 to 11.8 ±0.555. The highest mean PAH concentrations were recorded for Benzo(K)Fluoranthene at 11.8±0.555, for mullet from Kaa, and pyrene at 7.74±0.346 for mullet collected from Bodo. The mean PAH concentrations for Benzo(b)Fluoranthene, Benzo(a)anthracene,
Chrysene, Dibenzo(a,b)-anthracene, Fluoranthene, Indeno(1,2,3-c,d)-pyrene, Phenanthrene and Pyrene were higher for mullet samples collected from Bodo, than those collected from Kaa, while the mean PAHs concentrations for Anthracene, Benzo(a)pyrene, Benzo(g,h,i)-perylene, Benzo(k)fluoranthene, Fluorene and Naphthalene were all higher for mullet samples collected from Kaa.acenaphthylene and Acenaphthene were below detection level (BDL) for both sites, while Benzo(a)anthracene and phenanthrene were below detection level for samples collected from Kaa only. The total PAH concentrations were 28.1±1.802 and 15.7±0.418 for samples collected from Kaa and Bodo respectively. There was a significant statistical difference at p<0.05 for all PAH detected for samples from Kaa and Bodo.

The potency equivalence concentration (PEC) for mullet samples collected from Kaa and Bodo were 3.29 and 2.20 respectively, which is higher than the screening value indicating that the consumption of mullet from both Bodo and Kaa at a rate of 68g/day in an adult of about 60kg will expose the individual to a potential health risk of cancer [4].

The LMW-PAH/HMW-PAH ratio for mullet collected from Kaa and Bodo were 0.07 and 0.19 respectively, which are both <1. Thus, indicating that the sources of these PAHs in the fish are mainly pyrogenic [5,19] a clear indication of anthropogenic pollution of PAHs [20].

The BaA/(BaA + Chry) ratio for mullet were 0 and 0.434 for Kaa and Bodo respectively. This suggests that the PAHs detected in mullet from Kaa are of petrogenic origin, disagreeing with the LMW-PAH/HMW-PAH ratio, while the PAHs detected in mullet from Bodo are of pyrogenic origin, (Yunker et al., 2002) agreeing with the LMW-PAH/HMW-PAH ratio. The mean concentration of Benzo (a) pyrene in mullet was 0.66±0.004 and 0.19±0.002 for Kaa and Bodo respectfully which is below the European Union (EU) permissible limit of 2g/kg, suggesting that consumption of mullet from Kaa and Bodo will not predispose the consumer to cancer [21].

Table 1. Polycyclic Aromatic Hydrocarbon (PAH) concentrations (mean ± S.E.M, µg/kg) in fin and shell fishes from study sites [Bodo (test site) and Kaa (control site)]

<table>
<thead>
<tr>
<th>PAHs</th>
<th>TILAPIA</th>
<th>MULLET</th>
<th>SHRIMP</th>
<th>SOMOPAT GRUNT</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>KAA</td>
<td>BODO</td>
<td>KAA</td>
<td>BODO</td>
</tr>
<tr>
<td>Acenaphthylene</td>
<td>BDL</td>
<td>BDL</td>
<td>BDL</td>
<td>BDL</td>
</tr>
<tr>
<td>Acenaphthene</td>
<td>BDL</td>
<td>0.05±0.003</td>
<td>BDL</td>
<td>0.05±0.003</td>
</tr>
<tr>
<td>Anthracene</td>
<td>0.48±0.003</td>
<td>0.17±0.010</td>
<td>0.07±0.001</td>
<td>0.02±0.001</td>
</tr>
<tr>
<td>Benzo[a]anthracene</td>
<td>BDL</td>
<td>BDL</td>
<td>0.21±0.005</td>
<td>BDL</td>
</tr>
<tr>
<td>Benzo[a]pyrene</td>
<td>0.44±0.007</td>
<td>0.65±0.004</td>
<td>0.19±0.002</td>
<td>0.43±0.005</td>
</tr>
<tr>
<td>Benzo[g,h,i]perylene</td>
<td>0.54±0.004</td>
<td>5.78±0.013</td>
<td>0.58±0.004</td>
<td>0.27±0.001</td>
</tr>
<tr>
<td>Benzo[k]fluoranthene</td>
<td>0.45±0.011</td>
<td>0.75±0.010</td>
<td>0.97±0.001</td>
<td>1.86±0.018</td>
</tr>
<tr>
<td>Benzo[k]fluoranthene</td>
<td>6.56±0.064</td>
<td>11.8±0.555</td>
<td>1.25±0.006</td>
<td>4.2±0.014</td>
</tr>
<tr>
<td>Chrysene</td>
<td>0.19±0.005</td>
<td>0.08±0.000</td>
<td>0.28±0.001</td>
<td>0.17±0.003</td>
</tr>
<tr>
<td>Dibenzo[a,h]anthracene</td>
<td>0.18±0.006</td>
<td>0.39±0.001</td>
<td>0.25±0.019</td>
<td>0.07±0.003</td>
</tr>
<tr>
<td>Fluoranthene</td>
<td>1.11±0.013</td>
<td>1.60±0.004</td>
<td>1.32±0.178</td>
<td>1.95±0.036</td>
</tr>
<tr>
<td>Indeno(1,2,3-c,d)pyrene</td>
<td>0.33±0.008</td>
<td>0.71±0.006</td>
<td>0.97±0.016</td>
<td>1.06±0.005</td>
</tr>
<tr>
<td>Fluorene</td>
<td>5.13±0.066</td>
<td>0.49±0.003</td>
<td>0.66±0.019</td>
<td>0.27±0.003</td>
</tr>
<tr>
<td>Naphthalene</td>
<td>0.48±0.004</td>
<td>0.50±0.001</td>
<td>1.08±0.004</td>
<td>0.58±0.000</td>
</tr>
<tr>
<td>Phenanthrene</td>
<td>BDL</td>
<td>0.08±0.000</td>
<td>BDL</td>
<td>0.26±0.003</td>
</tr>
<tr>
<td>Pyrene</td>
<td>5.16±0.027</td>
<td>4.55±0.993</td>
<td>7.74±3.469</td>
<td>1.78±0.001</td>
</tr>
<tr>
<td>Total PAHs</td>
<td>21.1±2.211</td>
<td>39.8±5.190</td>
<td>28.1±1.802</td>
<td>15.7±0.180</td>
</tr>
</tbody>
</table>

Values are expressed as mean ± standard error of mean (S.E.M) of three replicates, (n=3). Values with different superscript (a, b) in the same row are significantly different at the 0.05 levels (p< 0.05)
3.1.3 Penaeus notialis (Shrimps)

The mean PAH concentrations for shrimps ranged from below detection limit of 0.0001 to 9.48±0.002. The highest mean PAH concentrations were recorded for pyrene in shrimp collected from Bodo at 9.48±0.002 and Benzo(k)fluoranthene in shrimp collected from Bodo at 4.27±0.014. The mean concentrations of Acenaphthylene, Anthracene, Benzo(g,h,i)perylene, Benzo(b)fluoranthene, Indeno(1,2,3-cd)pyrene, Fluorene, and phenanthrene were higher for shrimp samples collected from Kaa than those collected from Bodo, while mean concentrations of Acenaphthene, Benzo(a)pyrene, Benzo(k)fluoranthene, Chrysene, Dibenz(a,h)anthracene, Fluoranthene, Naphthalene and pyrene were higher for shrimp samples collected from Bodo. Benzo(a)anthracene and Fluoranthene were below detection level for samples collected from Kaa. The total PAH concentrations in samples were 9.38±0.086 and 18.3±0.063 for samples collected from Kaa and Bodo respectively. All the PAH concentrations showed a statistically significant difference at p<0.05, for all shrimp samples collected from Kaa and Bodo, except for Benzo(a)anthracene which was below detection level for shrimps collected from both Kaa and Bodo.

The calculated potency equivalence concentration (PEC) for shrimp collected from Kaa and Bodo were 0.86 and 2.70 respectively, which is <1, indicating that the consumption of shrimp from both sites at a rate of 68g/day in an adult of about 60kg will expose the individual to a potential risk of cancer [4].

The LMW-PAH/HMW-PAH ratio for shrimps collected from Kaa and Bodo were 0.68 and 0.05 respectively, which is <1, indicating that the sources of the PAHs in the shrimps from both sites are mainly pyrogenic[5,19], a clear indication of anthropogenic pollution of PAHs [20].

The BaA/(BaA + Chry) ratio for shrimp collected from Kaa and Bodo were both zero (0). This suggests that the source of PAH input in the shrimp samples is petrogenic [6], in contrast to the LMW-PAH/HMW-PAH ratio. The mean concentration of Benzo(a)pyrene in shrimp from Kaa and Bodo was 0.14±0.006 and 0.43±0.005 respectively, which were both below the European Union (EU) permissible limit of 2µg/kg. This means that consumption of shrimps from Kaa and Bodo does not predispose the consumer to cancer [21] in contrast to the calculated PEC values.

3.1.4 Pomadasys jubelini (Sompat grunt)

The mean PAH concentrations for Sompat grunt ranged from below detection limit of 0.0001 to 11.0±0.029. The highest mean concentrations were recorded for benzo(k)fluoranthene 11.0±0.029, and 10.1±0.179 for sompat grunt collected from Bodo, and Kaa respectively. All the mean PAH concentrations for sompat grunt collected from Kaa were significantly lower at p<0.05, than mean PAH concentrations for sompat grunt collected from Bodo, except for Naphthalene and Anthracene, which were significantly higher at p<0.05 for sompat grunt from Kaa than sompat grunt from Bodo, and Acenaphthene, Acenaphthylene and Benzo(a)anthracene level for sompat grunt collected from both Kaa and Bodo which were all below detection levels. The total mean PAH concentration for Sompat grunt were 24.6±0.285 and 28.4±0.158 for Sompat grunt collected from Kaa and Bodo respectively.

The calculated potency equivalence concentration (PEC) for sompat grunt collected from Kaa and Bodo were 2.95 and 6.91 respectively, which are both higher that the screening value (SV), suggesting that consumption of tilapia from both sites at a rate of 68g/day in an adult of about 60kg would expose the individual to a potential risk of cancer [4].

The LMW-PAH/HMW-PAH ratio for sompat grunt collected from Kaa and Bodo were both <1 for Kaa and Bodo, indicating that the sources of the PAHs in the sompat grunt samples from Kaa and Bodo are mainly of pyrogenic origin [5,19] a clear indication of anthropogenic pollution of [20].

The BaA/(BaA + Chry) ratio for sompat grunt from Kaa and Bodo were both zero (0) , (ie<0.20) suggesting that the source of PAH input in the fish samples is petrogenic (Yunker et al., 2002) in contrast to the LMW-PAH/HMW-PAH ratios. The mean concentration of Benzo(a)pyrene in sompat grunt from Kaa and Bodo was 0.54±0.005 and 0.70±0.009 respectively, which are both below the European Union (EU) permissible limit of 2µg/kg, suggesting that consumption of tilapia from Kaa and Bodo will not predispose the consumer to cancer [21] in contrast to the calculated PEC values.
4. DISCUSSION

The extent of the damage inflicted on aquatic and terrestrial wildlife by oil spillage is dependent on several factors which include; the type of hydrocarbons, the quantity of the spill, the temperature at the time of occurrence and the season (my paraphrase) [22].

Similar PAH assemblages were observed in the four species, but the concentrations of the PAH assemblages were different. These findings are not definitive as with the source of the PAH, seemingly suggesting various or multiple sources of PAH contamination in the studied sites, as suggested by Knutzen and Sortland [23] who reported that different pollution sources give rise to different PAH. For samples from Kaa, Mullet showed the highest total mean concentration of PAHs followed by Sompat grunt, Tilapia and the least was Shrimps. However, for samples collected from Bodo, Tilapia showed the highest total mean concentration of PAH, followed by Sompat grunt, Shrimps, and the least Mullet. These variations may be attributed to their feeding habits, as discussed earlier. Some of these aquatic species feed on large planktonic organisms, sponges, dead organisms, worms, plant materials and soil particles at the bottom of the sea, which could contain PAHs deposits in agreement with [24,25]. This may explain why some of these species have a significantly higher concentration of PAHs.

The LMW-PAH/HMW-PAH ratios observed in the four species from the test and control sites were < 1, indicating that the sources of these PAHs in the fish analyzed are mainly pyrogenic as suggested by Yunker et al., 2002 and Rocher et al., 2004. The LMW- PAH/HMW-PAH ratios indicate that the HMW-PAHs were generally predominant compared to the LMW-PAHs. The predominance of HMW-PAHs may be due to the fact that LMW-PAHs are preferentially degraded during PAH transport and buried into sediments as proposed by [26]. In contrast to this, BaA/(BaA+Chry) ratios in all the samples were <0.02, this suggests that the sources of PAH are petrogenic in agreement with Yunker et al., 2002, except in Mullet from Bodo which >0.35, agreeing with the LMW-PAH/HMW-PAH ratio, that suggests the source of PAH are pyrogenic. This further reinforces the possibility that there are multiple sources of PAH contamination (pyrogenic and petrogenic) in the study sites.

The calculated potency equivalence concentration (PEC) for all the tested aquatic species collected from Kaa and Bodo were all above the screening value (SV) suggesting that the consumption of these aquatic species from the test and control site at a rate of 68g/day in an adult of about 60kg will expose the individual to a potential risk of cancer [4]. However, in contrast to this evaluation, the concentration of Benzo(a)pyrene in all the species analyzed from Kaa and Bodo were all below the European Union permissible limit of 2µg/Kg, suggesting that the consumption of tested species from Kaa and Bodo would not predispose the consumer to cancer [21]. As pointed out by Chen and Liao, 2006, it is very important however to note that the Benzo[a]Pyrene equivalent-based approach used for carcinogenic risk assessment is limited to a few PAHs that have been monitored in ambient air, and does not account for the toxicity of all PAHs to which the general population is exposed, and thus, may not be a very reliable determinant of carcinogenicity / toxicity assessment of PAH.

The presence of detectable levels of hydrocarbon pollution in the control site (Kaa) could be attributed to seepage from previously existing oil spillage site, as reported by the UNEP, 2011 which stated that “Observations and scientific investigations found that oil contamination in Ogoniland is widespread and severely impacting many components of the environment, and that even though the oil industry is no longer active in Ogoniland, oil spills continue to occur with alarming regularity.” This could explain the presence of pyrogenic sources of PAHs found in both sites. The presence of detectable levels of hydrocarbon pollution in the sea foods collected from the control site (Kaa) could also be as a result of migration of sea animals from the polluted site (Bodo) to the test site.

5. CONCLUSION

It can be concluded from the findings of this research that there is significant pollution of the study sites, with the presence of high levels of hydrocarbon pollutants in the test site (Bodo) and the control site (Kaa) studied, though, in much higher levels in the test site than the control site, and though fish tend to leave polluted areas in search of cleaner water according to UNEP, 2011, the possible spread to new areas of the already existing petroleum pollution in Ogoniland is a major threat to the aquatic life, and hence the lives of the Ogoni people. Furthermore, the population living around the study areas may be...
exposed to substantial levels of PAH, and thus are predisposed to a lot of health risks, including cancer. However, a consumption rate of 1 g/day may be protective from the carcinogenic effects of the current PAH levels. This is because the PEC values associated with a consumption rate of 1 g/day are found to be less than the screening value as reported by Russell et. al. [16] Although, it most unlikely that people living in these regions will consume only 1 g/day of fish.

ETHICAL APPROVAL

As per international standard or university standard ethical approval has been collected and preserved by the authors.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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