

Research Article

Petroleum Hydrocarbon Carcinogens in Organs of Commercially Available Fish Species from Crude Oil Polluted Escravos River in Delta State, Nigeria

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Abstract

This study investigates the presence and distribution of petroleum hydrocarbon carcinogens in the organs of commercially available fish species collected from the Escravos River in Delta State, Nigeria. The Escravos River is known to be heavily impacted by crude oil pollution due to oil exploration and production activities in the region. The bioaccumulation of petroleum hydrocarbons, including polycyclic aromatic hydrocarbons (PAHs), in fish tissues poses potential risks to both aquatic organisms and human consumers. Samples of various commercially important fish species were collected from different locations along the Escravos River and analyzed for the presence of petroleum hydrocarbon carcinogens using gas chromatography-mass spectrometry (GC FID 5890 SERIES II) techniques. All data were subjected to one-way Analysis of Variance (ANOVA) using SPSS version 16 to test for the significant level of the parameters across the groups. Preliminary findings indicate elevated levels of petroleum hydrocarbon carcinogens, particularly PAHs, in the organs of fish sampled from the polluted areas of the Escravos River. Liver and adipose tissues exhibited higher concentrations of PAHs compared to muscle tissue, suggesting organ-specific bioaccumulation patterns. The presence of these carcinogens in commercially available fish species highlights potential health risks to consumers, particularly those reliant on fish as a dietary staple. This study underscores the urgent need for comprehensive environmental monitoring and management strategies to mitigate the impact of crude oil pollution on aquatic ecosystems and human health in the Escravos River region. Further research is warranted to assess the long-term effects of petroleum contamination on fish populations and human communities' dependent on aquatic resources in the study area.

Keywords: Total Petroleum Hydrocarbon, Total Aliphatic Hydrocarbon, Polycyclic Aromatic Hydrocarbon, Tilapia Zilli, Carcinogen, Escravos River.

1. Introduction

Research on petroleum hydrocarbon carcinogens in fish species from crude oil-polluted areas primarily focuses on understanding the bioaccumulation and health effects of these pollutants [1-4]. Fish living in crude oil-polluted environments can accumulate petroleum hydrocarbons in their tissues through various pathways, including ingestion of contaminated food, water, and direct contact with sediment [5]. This bioaccumulation can lead to high concentrations of carcinogenic compounds within the fish. Studies have shown that different organs of fish species may accumulate petroleum hydrocarbons to varying extents [6-8]. For example, liver and adipose tissues often exhibit higher concentrations compared to muscle tissue. This non-uniform distribution of hydrocarbons suggests differential uptake and metabolism within the fish's body [9]. Petroleum hydrocarbons contain numerous compounds known or suspected to be carcino-

genic, such as polycyclic aromatic hydrocarbons (PAHs) [2-4]. These compounds can induce DNA damage, disrupt cellular function, and promote tumor formation in exposed organisms, including fish. Chronic exposure to low levels of carcinogens in fish tissues from polluted areas may increase the risk of cancer development in both fish and humans consuming contaminated fish [6, 7]. Different fish species exhibit varying sensitivities to petroleum hydrocarbon exposure and differences in their ability to metabolize and eliminate these compounds. Some species may bioaccumulate higher levels of carcinogens than others, depending on factors like feeding habits, metabolic rates, and lipid content [1, 2]. Consumption of fish contaminated with petroleum hydrocarbons poses health risks to humans, including an increased risk of cancer and other adverse health effects [2].

Regulatory agencies often set limits on allowable concentra-

tions of these contaminants in fish for human consumption to mitigate health risks. Beyond health implications for individual organisms, petroleum pollution can have significant ecological consequences, including population declines, ecosystem disruption, and long-term degradation of habitats. Overall, research suggests that fish species inhabiting crude oil-polluted environments can bioaccumulate petroleum hydrocarbons, including carcinogenic compounds, in their tissues, with implications for both ecosystem health and human consumption. Continued monitoring and research are essential to understand the long-term effects of petroleum pollution on aquatic ecosystems and human health.

The interaction with the young and aged people living in kurutie/Okerenkoko, along Escravos River revealed that the community have recorded over times cancerous diseases, tumours, deafness, excessive skin diseases and many other mutagenic ailments. These diseases could be traced to the bioaccumulation of millions of carcinogenic polyaromatic hydrocarbons in fishes from the river, since fishing is their major occupation, rate of its consumption is high, and these must have long accumulated causing different ailments.

The purpose of this project is to quantitatively determine the total hydrocarbon carcinogens in organs of commercially available fishes from Escravos River in Delta State, Nigeria. Before considering this research topic, my interest was geared towards health implications of the aquatic animals consumed in the local communities cut across Escravos River. This is because the habitats of these communities have limited lands, as most part of their environments are covered by water. This has caused a lot of eye sight problems and environmental degradations as they do not have waste disposal site as those living on dry lands. Waste in these communities are indiscriminately disposed into the water as it could be expensive to transport them to the land. Industrial activities have also increased the population of the people living in these areas, which causes increase in the amount of generated wastes per period of time. The water body serve as sink for all the generated wastes in the environment. It was also observed that most of habitat builds their toilet at the top of the water suspended by woods, because in most of the communities, the habitats are refugees and cannot afford to build modern houses. Most of these generated wastes into the river are carcinogenic.

At the period of this research, it was also observed that at a given period of time, fishes always die and float at the top of the river. This is a clear indication that the water is highly polluted. Crude oil mining is a major industrial activity in the communities cut across Escravos River. Oil spillage is one of the major causes that contributes to pollution of the river. Fishes and other consumed aquatic animals in the environment have permeable skins which can bioaccumulate these wastes when dissolved in the water thereby finding their way into food chains of the habitats. The results of this research will provide insight into the total hydrocarbon carcinogens in organs of commercially available fishes from Escravos River in Delta State, Nigeria. This research is one of its kind in the selected area of study.

1.1. Study Area

The Escravos River is a river in southern Nigeria. "Escravos" is a Portuguese word meaning "slaves" and the area was one of the main conduits for slave trade between Nigeria and the United States in the 18th century. The length of the river is 57km with its source from Niger River, having a link to Atlantic Ocean and Gulf of Guinea. It lies within the coordinate of Latitude: 5° 34' 59.99" N Longitude: 5° 09' 60.00" E [10].



Figure 1: Map of Nigeria showing study area. Source: Google Map 2014.

1.2. Fish Sampling Method

Tilapia fish species from Escravos River location were collected from local fisher men at the point of fishing, wrapped in a sterile aluminium foil, transported and stored at -20°C until further analysis.

1.3. Fish Sample Processing

The fish samples were removed from refrigerator where it was stored, thawed and cleaned in tap water to remove any dirt. The thawed fishes were dissected using aseptic instrument and dishes and obtained the liver, gills, muscles, and kidney and were placed in a sample bottle and labelled for further analysis.

1.4. Total Petroleum Hydrocarbons Extraction

Fish samples were crushed with mortar and pestle. A 10g aliquot of well crushed sample (Gill, Liver, Kidney and Muscle) were weighed into a clean 250ml beaker. A mixture of solvent containing 50:50 ml of acetone and dichloromethane were prepared in a different beaker. 50ml of the solvent mixture was added into the beakers containing 10g of each sample. To ensure high purity for consistent results, samples were spiked with 1ml of surrogate mixture. Samples were placed in a sonicator and agitated for 15 minutes at 70°C. To obtain a clear extract, 10g of anhydrous sodium sulphate were added. Extracts were separated from the mixture into a round bottom flask. This processes were repeated with additional 50ml of solvent mixture, sonicate and allowed the beaker to settle and decanted into the same round bottom flask. Extract were concentrated in rotary evaporator to 3ml [11].

1.5. Column Preparation

Columns were packed with 10g of 100-200 mesh silica gel and glass fibre wool pre-conditioned (baked) at 105°C overnight. The column was made slurry by adding 10ml of n-hexane [11].

1.6. Fractionation and re-concentration of extracts

Concentrated extracts were ready to be fractionated into aliphatic and aromatic fractions. This process was done in a column packed with a glass fibre wool and silica gel. Fractionation of Polyaromatic Hydrocarbon was carried out in the prepared column by running dichloromethane (DCM) through the column containing the extract. This solvent DCM was used because it has affinity for PAHs. Fractionation of Total Petroleum Hydrocarbon (TPH) was carried out with n-hexane because of their affinity for TPH. The fractionated sample of individual component were transferred into a round bottom-flask and concentrated into 2ml. The concentrates were stored in a chromatographic vial ready for TPH/PAH analysis by GC FID 5890 SERIES II. The samples in the vial is were stored at 4°C prior to GC analysis.

1.7. Gas Chromatography Analysis

Each extract transferred to a 1.5ml vial was loaded into a gas chromatography system GC FID 5890 SERIES II, with flame ionization detector (FID) and cold on-column injection. 1µL portion of the sample was injected and analysed for TPH (C8-C40). The analytical separation was carried out with a HP-5 column having the dimensions 30m × 0.25mm with a stationary phase thickness of 0.25µm. The carrier gas was purified nitrogen held at a flow rate of 5mL/min. The operating temperature was started at 60°C for 2mins and then increased at the rate of 10°C/min to 300°C for 10min. The injector and detector temperature were maintained at 250°C and 300°C respectively. The minimum detection limit for all the compounds analysed was 0.1µg/kg wet weight.

1.8. Statistical Analysis

All data were subjected to one-way Analysis of Variance (ANOVA) using SPSS version 16 to test for the significant level of the parameters across the groups. The level of significance was chosen at P<0.05 and the results were presented as mean ± standard error.

2. Results

Table 1: Mean ± SE of Total aliphatic hydrocarbon component from organs of tilapia fish (mg/kg)

Components	Muscle	Gill	Liver	Kidney	Min.	Max.	P-Value
Octane (C8)	144.33±0.46	BDL	56.25±0.31	78.17±0.03	55.52	149.45	P<0.05
Nonane (C9)	1946.70±0.03	2155.49±0.44	697.29±0.21	869.02±0.41	658.25	2210.08	P<0.05
Decane(C10)	BDL	BDL	BDL	BDL	-	-	P<0.05
Undecane(C11)	310.37±0.05	303.22±0.32	215.32±0.14	198.58±0.18	185.08	310.37	P<0.05
Dodecane(C12)	80.05±0.32	71.95±0.01	86.33±0.02	88.03±0.12	70.18	90.12	P<0.05
Tridecane(C13)	86.92±0.33	157.98±0.21	64.08±0.16	65.02±0.28	64.08	159.36	P<0.05
Tetradecane(C14)	BDL	BDL	BDL	BDL	-	-	P<0.05
Pentadecane(C15)	119.13±0.22	63.05±0.30	60.87±0.04	57.54±0.11	55.97	120.54	P<0.05
Hexadecane(C16)	300.92±0.30	413.82±0.21	287.96±0.18	301.54±0.33	287.02	418.69	P<0.05
Heptadecane(C17)	53.53±0.20	37.68±0.04	58.21±0.03	47.01±0.01	36.85	58.87	P<0.05
Pristane	398.69±0.15	327.69±0.35	348.77±0.14	289.67±0.13	288.03	399.87	P<0.05
Octadecane(C18)	41.82±0.17	22.24±0.01	36.55±0.05	29.36±0.11	21.34	38.40	P<0.05
Phytane	51.69±0.24	77.98±0.02	59.02±0.20	68.66±0.15	51.08	79.58	P<0.05
Nonadecane(C19)	654.82±0.03	477.07±0.23	587.98±0.40	478.09±0.55	472.05	659.36	P<0.05
Eicosane(C20)	104.54±0.08	77.25±0.01	98.14±0.01	66.57±0.28	64.88	109.48	P<0.05
UncosaneC21	74.37±0.05	79.33±0.34	81.02±0.01	46.21±0.42	44.01	81.90	P<0.05
Docosane(C22)	334.72±0.31	408.41±0.33	298.46±0.03	329.07±0.36	299.47	411.77	P<0.05
Tricosane(C23)	403.07±0.20	592.23±0.52	421.02±0.12	408.55±0.70	400.98	599.60	P<0.05
Tetracosane(C24)	96.23±0.15	99.49±0.15	87.02±0.19	75.12±0.07	79.58	101.24	P<0.05
Pentacosane(C25)	357.05±0.13	274.36±0.08	125.22±0.14	149.28±0.09	125.22	358.09	P<0.05
Hexacosane(C26)	596.29±0.12	693.02±0.03	478.98±0.19	357.48±0.17	354.55	693.02	P<0.05
Heptacosane(C27)	209.99±0.25	83.12±0.20	124.09±0.22	147.98±0.05	85.22	212.08	P<0.05
Octacosane(C28)	187.99±0.41	88.58±0.40	96.03±0.08	95.55±0.02	84.06	189.34	P<0.05
Nonacosane(C29)	146.66±0.33	327.17±0.23	108.08±0.02	147.02±0.12	108.66	330.58	P<0.05
Triacosane(C30)	215.80±0.08	102.40±0.16	145.09±0.14	125.22±0.33	101.51	216.01	P<0.05

Untriacontane(C31)	103.99±0.01	107.37±0.20	111.87±0.16	86.69±0.03	86.01	114.09	P<0.05
Dotriacontane(C32)	236.72±0.07	95.06±0.50	158.15±0.07	251.11±0.11	94.07	254.10	P<0.05
Trtriacontane(C33)	83.70±0.22	309.67±0.06	65.74±0.31	125.67±0.17	65.74	310.71	P<0.05
Tetratriacontane(C34)	797.46±0.12	1230.76±0.44	258.39±0.08	143.54±0.33	143.07	1230.76	P<0.05
Pentatriacontane(C35)	52.13±0.02	125.05±0.02	88.88±0.23	77.32±0.140	52.01	92.44	P<0.05
Hexatriacontane(C36)	317.87±0.03	367.54±0.47	489.18±0.31	229.36±0.24	224.58	490.10	P<0.05
Heptatriacontane(C37)	470.19±0.30	222.55±0.36	189.57±0.71	217.64±0.05	189.07	474.05	P<0.05
Octatriacontane(C38)	49.87±0.45	59.16±0.18	44.02±0.41	33.21±0.04	32.51	61.23	P<0.05
Nonatriacontane(C39)	109.06±0.03	147.67±0.04	106.15±0.05	108.24±0.33	105.33	149.04	P<0.05
Tetracontane(C40)	165.63±0.24	789.82±0.05	136.81±0.02	188.93±0.18	134.84	790.17	P<0.05
TOTAL ALIPHATIC (mg/kg)	9302.33±1.08	10388.16±0.98	6270.64±0.85	5980.56±1.20	5978	10390	P<0.05

BDL = Below Detectable Limit

Table 2: Mean ± SE of Polynuclear Aromatic Hydrocarbon component from organs of tilapia fish (mg/kg)

Components	Muscle	Gill	Liver	Kidney	Min	Max	P-Value
Naphthalene	4.32±0.12	3.06±0.11	3.54±0.01	7.36±0.05	2.85	8.33	P<0.05
Acenaphthylene	11.08±0.33	3.86±0.25	4.54±0.02	4.26±0.14	3.20	12.09	P<0.05
Acenaphthene	7.70±0.14	26.32±0.03	15.34±0.12	8.26±0.11	7.52	26.77	P<0.05
Fluorene	7.37±0.18	2.96±0.15	3.69±0.30	4.65±0.25	2.47	8.30	P<0.05
Phenanthrene	3.98±0.02	9.99±0.13	8.14±0.12	7.77±0.24	3.42	10.23	P<0.05
Anthracene	135.57±0.33	85.12±0.01	122.36±0.33	129.39±0.18	85.02	136.88	P<0.05
Fluoroanthene	35.68±0.21	7.58±0.11	26.36±0.31	13.26±0.22	6.55	36.09	P<0.05
Pyrene	16.51±0.33	8.25±0.02	8.87±0.05	12.22±0.04	8.25	17.87	P<0.05
Chrysene	3.32±0.25	6.15±0.14	3.95±0.12	8.69±0.13	3.08	8.94	P<0.05
Benz(a)anthracene	6.78±0.17	2.69±0.54	5.37±0.19	4.23±0.08	2.33	7.45	P<0.05
Benzo(b)fluoranthene	3.76±0.24	4.90±0.20.16	4.09±0.27	5.64±0.17	3.24	6.80	P<0.05
Benzo(k)fluoranthrene	3.95±0.18	3.76±0.18	14.02±0.24	12.08±0.31	3.47	12.66	P<0.05
Benzo(a)pyrene	20.95±0.32	3.12±0.11	7.14±0.08	16.65±0.11	2.85	21.12	P<0.05
Indeno(1,2,3-cd)pyrene	36.33±0.44	14.17±0.30	24.56±0.15	18.21±0.36	13.18	38.02	P<0.05
Dibenz(a,h)anthracene	8.94±0.14	6.08±0.22	6.23±0.11	7.09±0.09	5.54	9.30	P<0.05
Benzo(g,h,i)perylene	7.10±0.17	4.90±0.05	7.89±0.01	6.88±0.05	4.80	7.95	P<0.05
Total PAH (mg/kg)	313.43±0.67	192.96±0.45	266.17±0.81	266.72±0.36	192.96	318.02	P<0.05

Table 3: Mean ± SE of Total Petroleum Hydrocarbon (TPH)

Components	Muscle	Gill	Liver	Kidney
ΣAliphatics	9302.33±1.85	10388.16±1.52	6270.64±1.87	5980.56±1.08
ΣPAHs	313.43±1.64	192.96±1.09	266.17±1.35	266.72±1.07
ΣTPH (mg/kg)	9615.76±1.06	10581.13±1.09	6536.81±1.54	6247.29±1.87

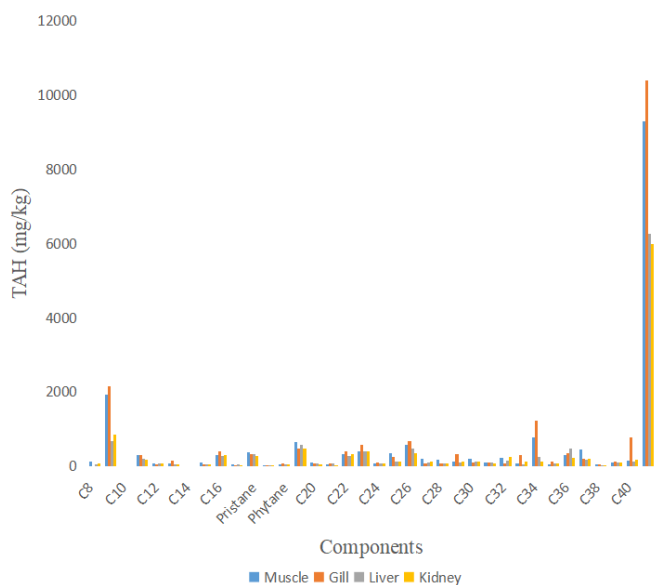


Figure 2: Total aliphatic hydrocarbon content (mg/kg).

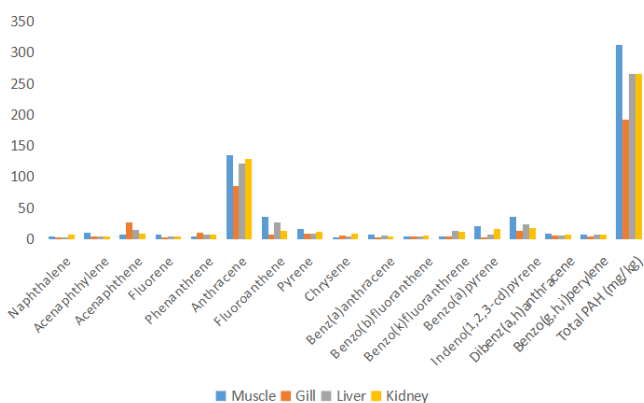


Figure 3: Polynuclear Aromatic Hydrocarbon Content (mg/kg).

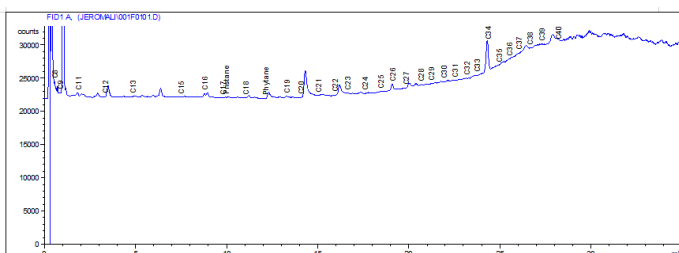


Figure 4: Chromatogram for the Aliphatic Hydrocarbons from the muscles of the tilapia specie.

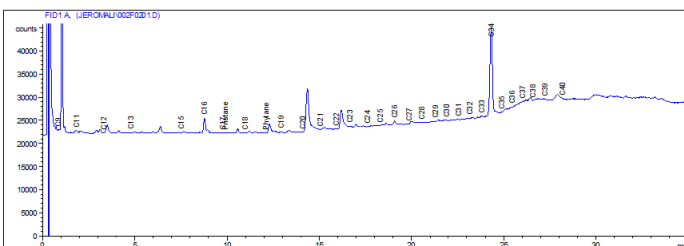


Figure 5: Chromatogram for the Aliphatic Hydrocarbons from the Gills of the tilapia specie

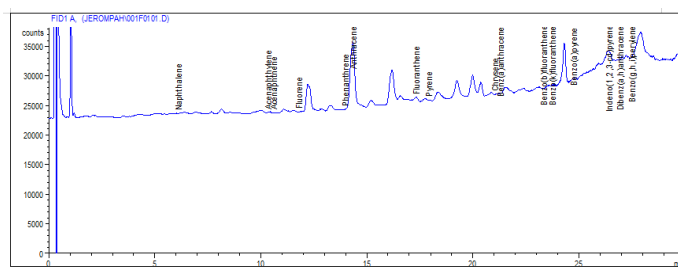


Figure 6: Chromatogram for the Polyaromatic Hydrocarbons from the muscles of the tilapia specie

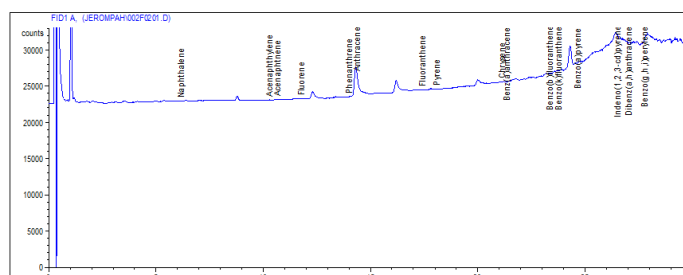


Figure 7: Chromatogram for the Polyaromatic Hydrocarbons from the Gills of the tilapia specie

3. Discussion of Results

The interaction with the young and aged people living in kurutie/Okerenkoko, along Escravos River revealed that the community have recorded over times cancerous diseases, tumours, deafness, excessive skin diseases and many other mutagenic ailments. These diseases are to be traced to the bioaccumulation of millions of carcinogenic polyaromatic hydrocarbons in fishes from the river, since fishing is their major occupation, rate of its consumption is high, and these must have long accumulated causing different ailments.

In a given environment (air, water and sediments) polyaromatic hydrocarbons does not exist as individual compound. They exist as a mixture of many other polynuclear aromatic hydrocarbons. Research have revealed that weakly or non-carcinogenic polyaromatic hydrocarbons which exist as a mixture can modify the carcinogenicity effects of a given polyaromatic hydrocarbon such as benzo (a) pyrene [12]. All fish ingest petroleum hydrocarbons directly or indirectly from contaminated water as food and sediments leading to massive destruction of aquatic biota [9]. Both aliphatic and polycyclic aromatic hydrocarbon fractions of dissolved petroleum are readily absorbed by most finfish and shellfish because of their high lipid solubility and are bioconcentrated in them [7]. Humans are exposed to total petroleum hydrocarbons through air, water, food, or soil. However, dietary intake has been shown to be a major for route for human exposure to petroleum hydrocarbons (PAHs) [13].

Total Aliphatic Hydrocarbon (TAH) were recorded in the tilapia fish sample with distinct varying concentrations. This study analysed both TAH and PAH in the muscles, gills, livers and kidneys of tilapia fish species from Escravos river cut across kurutie/Okerenkoko in Delta state, Nigeria. Thirty-five components of n-alkanes which makes up the

TAH has been analysed. From the results, the gills had the highest mean concentrations (10388.16 ± 0.98 mg/kg) of TAH while the Kidney had the lowest mean concentration (5980.56 ± 1.20 mg/kg) of the TAH. Eneuku et al., showed that the gills of fish analysed for TAH had the highest mean accumulation while the muscle recorded the lowest mean concentration of TAH [14]. The lower mean concentration of TAH in kidneys suggests that bioaccumulation is lower as against uptake demonstrated by the gills which showed highest bioaccumulation of TAH. The high mean concentration of TAH in the gills could be traced to the constant interaction of the gills which are highly vascularized with the pollution sources. Respiration keeps this organ constantly exposed to pollutants in water.

Table 1 and figure 2 show the mean \pm standard error of the total aliphatic hydrocarbon component from organs of tilapia fish (mg/kg). The results of bioaccumulation of octane (C8) in the four organs of tilapia fish showed that the minimum and maximum concentrations of octane are 55.52 and 149.45 mg/kg respectively. The concentration of C8 hydrocarbon in the gills of the tilapia fish was below detectable limit. The muscle and the liver of the tilapia specie recorded the highest and least concentrations of 144.33 ± 0.46 and 56.25 ± 0.31 mg/kg of octane respectively. The research work of Eneuku et al., (2019), the four organs of commercially available fishes from Oliha market show no record of C8 aliphatic hydrocarbon.

The bioaccumulation result for nonane (C9) aliphatic hydrocarbon showed that the organs of the analysed tilapia fish recorded highest concentration of C9 aliphatic hydrocarbon amongst other aliphatic hydrocarbons. The minimum and maximum concentrations of C9 aliphatic hydrocarbon are 658.25 and 2210.08 mg/kg respectively. Similar research work on the organs of fish samples showed lower concentration of C9 aliphatic hydrocarbon ranging from 3.72-22.32 mg/kg [14].

The C10 (decane) aliphatic hydrocarbon concentration were all beyond detectable limit from the four fish organs studied. The C11 (Undecane) aliphatic hydrocarbon concentration from the organs ranges from 185.08-310.37 mg/kg. The muscle and kidney showed highest (310.37 ± 0.05 mg/kg) and lowest (198.58 ± 0.18 mg/kg) bioaccumulation of C11 aliphatic hydrocarbon. The C12 (Dodecane) aliphatic hydrocarbon concentrations ranges from 70.18-90.12 mg/kg. The highest and lowest concentrations were recorded in kidney (88.03 ± 0.12) and gills (71.95 ± 0.01) mg/kg. Several studies have reported the negative effects of petroleum hydrocarbon to human health [7-9]. Recently, studies have shown that most human cancers such as prostate and lung cancer can be attributed to dietary sources [4, 14].

The C13 (tridecane) aliphatic hydrocarbon concentration ranges from 64.08-159.36 mg/kg, the liver showed the lowest mean concentration (64.08 ± 0.16 mg/kg) while the gill recorded the highest mean concentration (157.98 ± 0.21 mg/kg). Tetradecane (C14) was not detected from the organs. This shows that their concentration and bioaccumulation in

the organs were below the detectable limit of the GC-FID.

The C15, C16, C17, Pristane, C18, Phytane, C19 and C20 minimum concentration from the organs are 55.97, 287.02, 36.85, 288.03, 21.34, 51.08, 472.05 and 64.88 mg/kg respectively while the maximum concentration across the four organs are 120.54, 418.69, 58.87, 399.87, 38.40, 79.58, 659.36 and 109.48 mg/kg respectively. The minimum concentrations of TAH in C21, C22, C23, C24, C25, C26, C27, C28, C29 and C30 are 44.01, 299.47, 400.98, 79.58, 125.22, 354.55, 85.22, 84.06, 108.66 and 101.51 mg/kg respectively while their maximum concentrations are 81.90, 411.77, 599.60, 101.24, 358.09, 693.02, 212.08, 189.34, 330.58 and 216.01 mg/kg respectively.

The minimum concentrations of C31, C32, C33, C34, C35, C36, C37, C38, C39 and C40 are 86.01, 94.07, 65.74, 143.07, 52.01, 224.58, 189.07, 32.51, 105.33 and 134.84 mg/kg respectively, while their maximum concentrations are 114.09, 254.10, 310.71, 1230.76, 92.44, 490.10, 474.05, 61.23, 149.04 and 790.17 mg/kg respectively. The high concentrations of the TAH in the organs of the tilapia fishes analysed, clearly indicates that the Escravos river has been highly exposed to contaminants and pollutants, these pollutions could be the major causes of the ailments in the riverine areas across the Escravos river. This finding is in agreement with the findings of [1, 4, 6, 14].

Figure 4 and 5 represent the Chromatogram for the Aliphatic Hydrocarbons from the muscles and gills of the tilapia specie respectively while Figure 11 and 12 represent the Chromatogram for the Polyaromatic Hydrocarbons from the muscles and gills of the tilapia specie. Table 2 and figure 8 represent the Mean \pm Standard error of Polynuclear Aromatic Hydrocarbon component from organs of tilapia fish (mg/kg). In this research, sixteen components of aromatics which makes up the PAH's has been analysed. Polycyclic aromatic hydrocarbons (PAHs) are ubiquitous anthropogenic pollutants that can be biologically amplified to high concentrations in food webs. Due to their lipophilicity, persistence, and high toxicity, these residues are readily accumulated in the tissues of non-target living organisms where they may cause detrimental effects. PAHs are toxic, carcinogenic, and mutagenic to all organisms, including humans [15, 16]. The metabolites of PAHs may bind to proteins and DNA, which causes biochemical disruption and cell damage in animals and cancer in human [12, 16].

Naphthalene, Acenaphthylene, Acenaphthene, Fluorene, Anthracene, Fluoranthene and Pyrene are less carcinogenic, while benzo(a)anthracene, chrysene, benzo(k)fluoranthene, benzo(a)pyrene, benzo(b)fluoranthene, indeno(1,2,3)perylene, dibenzo(a,h)anthracene and benzo(g,h,i)perylene are highly carcinogenic [17].

For the PAH, the maximum and minimum concentrations of naphthalene are 8.33 and 2.85 mg/kg respectively. Highest average bioaccumulation was recorded in the kidney of the tilapia fish as 7.36 ± 0.05 mg/kg while the gills recorded the least value of 3.06 ± 0.11 mg/kg. Acenaphthylene and ace-

naphthene minimum bioaccumulation across the organs are 3.20 and 7.52 mg/kg respectively while their maximum concentrations are 12.09 and 26.77 mg/kg respectively. The highest average concentration of acenaphthylene and acenaphthene were observed in the muscles (11.08 ± 0.33 mg/kg) and gills (26.32 ± 0.03 mg/kg) of the tilapia fish respectively, while their lowest mean concentrations were recorded in the gills (3.86 ± 0.25 mg/kg) and the muscles (7.70 ± 0.14 mg/kg) of the tilapia fish respectively. The PAH stated here are far higher than the values reported by Olaji et al., in the investigation from four species of fish at Degele Community, Nigeria. The high mean concentrations recorded in the gills and muscles are similar to the fact that they have direct interaction with the contaminated medium, thereby ingesting higher concentrations [1].

The minimum concentrations of Fluorene, Phenanthrene, Anthracene, Fluoroanthene, Pyrene and Chrysene are 2.47, 3.42, 85.02, 6.55, 8.25 and 3.08 mg/kg while their maximum concentrations are 8.30, 10.23, 136.88, 36.09, 17.87 and 8.94 mg/kg respectively. Anthracene showed a very high mean concentration across the organs. The muscle concentration is as high as 135.57 ± 0.33 mg/kg while the gill recorded 85.12 ± 0.01 mg/kg. Anthracene when consumed targets the human skin, blood, intestines, blood and the lymph system. Exposure to high doses of anthracene for a short time can cause skin damage. It can also cause itching, burning and edema, a build-up of fluid in tissues. Humans exposed to anthracene experiences headache, loss of appetite, nausea, swelling or inflammation of the stomach and intestines. Anthracene has also been recorded to have caused tumours in laboratory animals that were exposed to anthracene, through their foods, breathing from contaminated air and direct skin application [18].

The minimum concentrations of benzo (a) anthracene, benzo (b) fluoranthene, benzo (k) fluoranthrene and benzo(a) pyrene are 2.33, 3.24, 3.47 and 2.85 mg/kg respectively while their maximum concentrations are 7.45, 6.80, 12.66 and 21.12 mg/kg respectively. In a report of Faust, when a pregnant mice were fed with high doses of PAH (benzo (a) pyrene) they experienced reproductive problems. The offspring of the pregnant mice showed birth defects and a decrease in their body weight. The animal study reported that exposure of mice to 308 ppm of PAH (benzo(a)pyrene) in food for 10 days (short term exposure) caused birth defects of the offspring, while mice exposed to 923 ppm of benzo(a) pyrene in food for months caused liver and blood problems [18].

The minimum concentrations of indeno (1,2,3-cd) pyrene, Dibenz (a,h) anthracene and benzo (g,h,i) perylene are 13.18, 5.54 and 4.80 mg/kg respectively while their maximum concentrations are 38.02, 9.30 and 7.95 mg/kg respectively. Biological monitoring of exposure to PAHs is of primary interest, due to the widespread diffusion of these compounds and to their toxicological relevance. However, the health effects of individual PAHs are not exactly alike. In fact, the International Agency for Research on Cancer classifies some PAHs as known, possibly, or probably carcinogenic to humans (Group

1, 2A or 2B). Among these are benzo[a]pyrene (Group 1), naphthalene, chrysene, benz[a]anthracene, benzo[k]fluoranthene and benzo[b]fluoranthene (Group 2B) [19, 20]. Some PAHs are well known as carcinogens, mutagens, and teratogens and therefore pose a serious threat to the health and the well-being of humans. The most significant health effect to be expected from inhalation exposure to PAHs is an excess risk of lung cancer [20].

In this study, the total concentration of n-alkanes is reported as Σ Aliphatics, total concentration of Polycyclic Aromatic Hydrocarbon as Σ PAH and Σ TPH for the total petroleum hydrocarbon. The values reported followed different trends. For the Σ Aliphatics, the gills had the highest average concentration while the kidney had the lowest average concentration. For Σ PAH, the muscle had the highest average concentration while the gills had the lowest average concentration. In summation Σ TPH, the gill had the highest average concentration while the kidney had the lowest average concentration. In other studies, the Σ Aliphatics and Σ PAH followed similar trend. The gills recorded the highest average concentration while the muscles recorded the lowest concentrations. The reported mean concentrations of TAH and PAH in the organs of tilapia fish were far higher than the European Union recommended limit of 2 μ g/kg; wet weight for fish. This claim, arrayed the extent of human impacts on the Escravos river.

4. Conclusion

In conclusion, our study highlights the significant presence of petroleum hydrocarbon carcinogens, particularly polycyclic aromatic hydrocarbons (PAHs), in the organs of commercially available fish species from the crude oil-polluted Escravos River in Delta State, Nigeria. The findings reveal organ-specific bioaccumulation patterns, with liver and adipose tissues showing higher concentrations of PAHs compared to muscle tissue. These results underscore the potential health risks posed to both aquatic organisms and human consumers relying on fish from the polluted river for sustenance. The elevated levels of petroleum hydrocarbon carcinogens in fish organs emphasize the urgent need for effective environmental management strategies to mitigate the impact of crude oil pollution on aquatic ecosystems and human health in the Escravos River region.

Regulatory measures aimed at controlling industrial activities and reducing pollutant discharges into the river are imperative to safeguard environmental quality and public health. Furthermore, our findings underscore the importance of ongoing monitoring and research efforts to assess the long-term effects of petroleum contamination on fish populations and human communities dependent on aquatic resources in the study area. Collaborative initiatives involving government agencies, industry stakeholders, and local communities are essential to address the complex challenges associated with crude oil pollution and ensure the sustainable management of freshwater resources in the Escravos River basin. Ultimately, proactive measures aimed at reducing pollution, restoring ecosystem health, and promoting sustainable fishing practices are crucial for safeguarding the ecological integrity of the Escravos River and protecting

the well-being of both aquatic life and human populations in Delta State, Nigeria.

Recommendations

Based on the findings of our study on petroleum hydrocarbon carcinogens in organs of commercially available fish species from the crude oil-polluted Escravos River in Delta State, Nigeria, the following recommendations are proposed: Environmental Remediation: Implement immediate and comprehensive remediation efforts to reduce the levels of crude oil pollution in the Escravos River. This may include cleanup operations, restoration of contaminated habitats, and implementation of technologies for treating oil-contaminated water and sediments.

Pollution Control Measures: Strengthen regulations and enforcement mechanisms to control industrial activities and prevent further discharge of pollutants into the Escravos River and its tributaries. Implement best practices in waste management, spill prevention, and environmental monitoring to minimize the risk of future contamination incidents. Fisheries Management: Develop and enforce sustainable fisheries management practices to prevent overfishing and ensure the long-term viability of fish populations in the Escravos River. This may include the establishment of fishing quotas, seasonal closures, and habitat restoration initiatives to support fish reproduction and migration.

Public Health Awareness: Increase public awareness and education initiatives to inform local communities about the potential health risks associated with consuming fish from the polluted areas of the Escravos River. Provide guidance on safe fishing practices, proper cooking methods, and alternatives for obtaining safe sources of protein.

Monitoring and Surveillance: Establish a robust monitoring and surveillance program to regularly assess the levels of petroleum hydrocarbon carcinogens in fish organs and water samples from the Escravos River. This will facilitate early detection of contamination trends, inform risk assessments, and guide decision-making processes for environmental management and public health protection.

Community Engagement: Foster collaboration and engagement with local communities, indigenous groups, and stakeholders to develop participatory approaches for addressing environmental challenges and promoting sustainable development in the Escravos River basin. Empower communities to actively participate in decision-making processes and contribute to the implementation of solutions that prioritize environmental conservation and social well-being.

Research and Innovation: Encourage further research and innovation in the fields of environmental science, toxicology, and ecological risk assessment to advance our understanding of the impacts of crude oil pollution on aquatic ecosystems and human health. Support interdisciplinary studies and knowledge exchange initiatives to develop evidence-based solutions for mitigating pollution and promoting resilience in the Escravos River region.

By implementing these recommendations in a coordinated and collaborative manner, stakeholders can work together to address the complex challenges posed by crude oil pollution in the Escravos River and safeguard the health and well-being of both ecosystems and communities in Delta State, Nigeria.

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