JJBS

Volume 11, Number 2,June 2018
ISSN 1995-6673
Pages 171 - 177

Jordan Journal of Biological Sciences

An Evaluation of Contaminant Body Burdens in Selected Fish Species: Associating Toxicity to Upgrade the Hazard Assessment

Victor E. Okpashi*, Victor N. Ogugua, Chibuike S. Ubani and Nwamaka O. Juliet

Department of Biochemistry, University of Nigeria, Nsukka, Nigeria

Received August 16, 2017; Revised November 3, 2017; Accepted November 14, 2017

Abstract

The health problems that are related to chronic exposures to polycyclic aromatic hydrocarbons (PAHs) include decreased immunity, Cataracts, kidney and liver damage, breathing problems, asthma or (lung- function abnormalities) skin redness and inflammation. Induced by a repeated contact with PAHs were found to cause various forms of cancer in aquatic animal models, especially a form that can easily integrate with the cell and corrupt the DNA. This study presents an assessment of the recalcitrant body residues of some legacy contaminants in selected fresh fish species. Three different species of fish, namely the African Red snapper (*Lutjanus agennes*), the Great barracuda (*Sphyraena barracuda*), and the African cat- fish (*Clarias gariepinus*), were sampled in a 1-km stretch of the Qua Ibeo River, Nigeria. The specimens were screened for PAHs using gas chromatography-mass spectroscopy. This study revealed the sixteen priority PAHs, listed by the United State Environmental Protection Agency (USEPA) as carcinogenic. The bio-concentration factor was calculated as the ratio of the concentration of particular PAHs in the tissues to its water-free PAHs concentration. Results showed that the PAHs concentration is beyond the permissible limit. Considering these results, one can infer that the Qua Iboe River and the biota are contaminated by PAH, with the risks of bio-accumulation posing threats to human health through the consumption of fish and aquatic organism foods.

Keywords: PAHs, Fish, Body Burden, Risk Assessment, Toxicity, Aquatic Pollution

1. Introduction

On the whole, most scientific reports and researches focus on the ability to measure the body burden of chemicals in biota. Although the bioaccumulation of a contaminant is an important issue, bioaccumulation in and of itself is not a hazard. The critical question is: at what point does bioaccumulation result in body burdens that lead to adverse effects on individual organisms (prey and predator species) and ultimately the whole ecosystems (Meador *et al.*, 1995). One increasingly popular approach linking body burdens of individual organisms to toxicological effects in that organism is the critical body residue (CBR). This approach shifts the focus from quantifying concentrations in water to predicting toxicity by measuring concentrations in tissues. The CBR approach has several advantages over commonplace approaches that measure concentrations in water and sediments, as discussed in McCarty and MacKay (1993). The body burden of polycyclic aromatic hydrocarbons (PAHs) is determined by the balance between uptake and elimination, where each can be influenced by many factors. The determination of uptake and elimination may suggest measuring tissue concentrations at two different times through routine monitoring. The rate at which these processes occur is more instructive, and may be used to compare species differences, and predict steady-state accumulation. Elimination can be accomplished by passive diffusion when the external concentrations are lower than the internal concentrations favoring an outward flux and enzymatic pathways that convert the hydrophobic parent compounds to more polar metabolites that are readily excreted by some taxa with kidneys or kidney-like organs (such as annelids, molluscs and arthropods) (Hom *et al*., 1995). The conversion of the hydrophobic PAHs to a polar metabolite decreases its ability to diffuse through the gill membrane, favoring the excretory route. The rate of elimination may be affected by environmental factors such as temperature, salinity, physiological factors, reproductive state, age, sex, stress, enzyme-induction, route of uptake, chemical hydrophobicity and exposure history (Mckim, 1994). An uptake rate constant describes the fractional changes in water concentration per unit time (hr.-1) and depends on relative sizes of the fish and water compartments (Barron *et al.*, 1990; Landrum *et al.*, 1992). For those organic contaminants that are metabolized, the uptake and the potential for accumulation are more difficult to determine, although the quantification of metabolites will greatly improve the assessment. Even at

* Corresponding author. e-mail: vic2reshu@gmail.com.

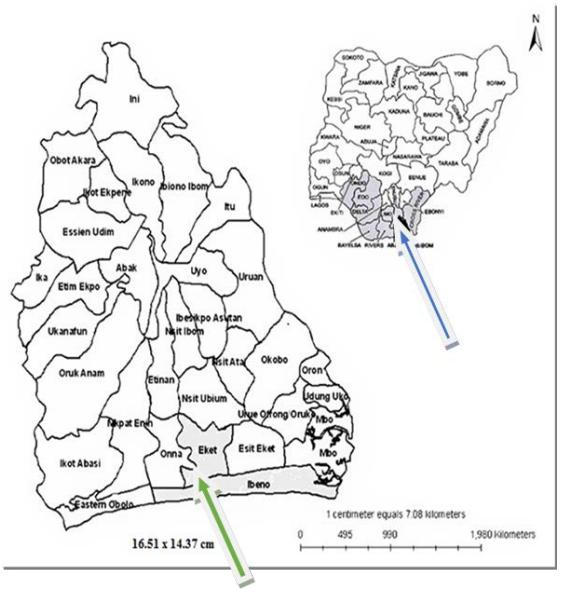
extremely-low environmental concentrations of PAHs (i.e., sometimes below detection limit by analytical instruments), aquatic organisms will accumulate PAHs because partitioning to the organism is thermodynamically favored, and the bio-concentration factors are quite high - (generally in the range of $l0^2$ - $l0^6$) - (Connell, 1991). The pattern of synergism in PAHs uptake, in the presence of other contaminants, has been rarely studied. One study found inhibition in the uptake of naphthalene by Eastern oyster (*Crassostrea virginica*) when presented with PCBs and benzo[a]pyrene (BaP) (Fortner and Sick, 1985). When the oysters were exposed to seawater with only naphthalene, uptake of both dissolved and particulate naphthalene produced tissue burdens of this compound that were always slightly higher.

Conversely, the authors concluded that naphthalene and PCBs had no effect on BaP uptake. In contrast, Stein *et al.* (1984) observed higher body burdens of BaP metabolites in the English sole (*Parophrys vetulus*) exposed to sediment containing BaP and PCBs than in fish exposed to sediment containing only BaP. This may occur because enzyme-induction led to a greater metabolism and higher amounts of metabolites that were not excreted. In the pelagic environment, fish and invertebrates may take up PAHs from water through diffusion across their gills and integuments and via their diet (Knezovich *et al*., 1987). Fish and invertebrates associated with sediment, either living in the substrate (infaunal) or on the surface of sediments (epibenthic) may accumulate PAHs via the diffusion of waterborne PAHs across their gills and integuments, and by dietary uptake. The contribution of PAHs through water and dietary sources to tissue burdens was examined by modeling. A series of curves showing the percentage uptake from water as a function of hydrophobicity was developed for two different modes of feeding (Hom *et al*., 1995). Generally, the metabolism of parent compounds is the principal confounding factor in assessing the exposure to PAHs. Therefore, the - measurement of parent hydrocarbons in any organism that is actively metabolizing PAHs may yield partial information on the total PAHs-derived body burden. Consequently, a direct measurement of parent hydrocarbon would be a useful quantitative parameter in animals that have minimal capacity for PAHs metabolism (Barron *et al*., 1990). Neff *et al.* (1976) reported that the rate of the release of a PAH is dependent on molecular weight and presumably hydrophobicity, with higher molecular weight compounds being released much slowly than low molecular weight compounds. Parent PAHs and their unbound metabolites are not highly persistent in fish, and their residual levels remaining in tissues and fluids two weeks after the exposure are generally minimal (Varanasi *et al*., 1989). However, reactive PAHs metabolites that covalently bind to DNA are quite persistent in fish. In studies with PAHs and nitrogen-containing aromatics, Stein *et al.* (1993), showed that BaP, which is a model mutagenic PAH, and 7H-dibenzo- [c,g]carbazole; a model nitrogen containing mutagenic PAH, form highly persistent DNA adducts in fish. The bio-concentration factor (BCF) is the ratio of the concentration of a particular chemical in the tissue to its concentration in the water. It should be kept in mind that the BCF is relevant for the accumulation from water. Therefore, in order to compare BCFs among different biota, it is important to confirm that water is the only route of uptake (Macarty, 1986).

2. Materials and Methods

2.1. Research Area

Eket is the second largest city in Akwa Ibom State in Nigeria. The name also refers to the indigenous ethnic group of the region and their language. The area of Eket's local government occupies the South central portion of Akwa Ibom State. It lies entirely in the tropics with territorial expense, spanning Northwards between Latitude 4° 33″ and 4° 45″ and Eastward between Longitude 7° 52″ and 5° 02″. Eket is bounded on the north by Nsit Ubium LGA, on its east by Esit Eket LGA, on the west by Onna LGA, and on the south by Ibeno LGA/Bright of Bonny Figure 1. At present, there are activities involving oil exploitation from Shell and Exxon Mobil, a thriving hub of a new oil and gas business, with more than 250 companies providing support services such as catering, flights and exports. An oil refinery is currently under construction in the outskirts of the city along the Oron road.



Key: The green arrow point to Map of Eket

Figure 1. Geography of the Project location showing the map of Nigeria and Akwa Ibom State

2.2. Description of the Study Sites

The study was conducted in the Qua Ibeo River (aka Kwa Ibo River), that rises near Umuahia in Abia State in Nigeria, and flows in a southeastern direction through Akwa Ibom State to the Atlantic ocean. The river feeds a zone of mangrove swamps linked by creeks and lagoons separated from the sea by a low and narrow ridge of sand. Ibeno, on the eastern side of the River ≈ 3 km from the river mouth, is one of the largest fishing settlements on the Nigerian coast. Four sampling stations were established, 1km apart, in the upper and middle reaches of the River based on ecological settings and the intensity of human activities. At each station, three sampling sites were selected, making a total of twelve sample points for the present study.

Station 1: This station was located in the upper reaches of the river at Ikot Ikpe and Ikot Akpoenang at latitude 4 - 55.8" and longitude 7 - 40.8". The main socio-economic activities in this area are mainly fishing and boat-building along the river banks. The water quality is relatively good at this location based on visual evaluation.

Station 2: This location receives effluents from a slaughter house located along the Ikot Aroku and Ikot Naidiba village road. There is also discharge of domestic and municipal waste into the river. The main socio-economic activities in this area include sand-mining for both domestic- and commercial purposes. It is about 1 km downstream from Station 1, straddling latitude 4 - 22.9", and longitude 7 - 13.8".

Station 3: This site is located along Eket-Etinan road, within Ebiyan and Ndon directly opposite the Onna local government area. Socio-economic activities at this location include washing of cars and motor bikes, laundry and bathing. The vegetation is dominated by bamboo trees. The site lies about 1km downstream of Station 2, straddling latitude 4 - 23.2"and longitude 7- 40.2".

Station 4: This is located at Ndilla. The opposite villages across the river are Odio and Ale Ebukuku in the lower reaches of the river near the estuary. This descended into Ibeno local government area, see Figure 2. The socio-economic activities include fishing and sand mining. The vegetation is mainly mangrove forests. The area is turbid due to sediments from sand mining as well as the impacts of socio-economic activities upstream. This site is also very deep due to the mining activities which excavate the river bed. It is located 1 km downstream of Station 3 straddling latitude 4- 43.5" and longitude 7 - 53.3".

Figure 2. Map of Eket local government area of Akwa Ibom State, Nigeria

2.3. Sample Collection and Analysis

The river water samples were collected using twelve amber bottles, each 100mL from twelve different points in Eket area of Akwa Ibom state on the second of September, 2015 between 10 a.m and 3 p.m along the coast. The samples collected were extracted with n-hexane before concentrating the analytes or target compounds within thirty minutes.

Fish sampling was randomly conducted to ensure that the representative samples of biota were included in the samples for analysis. Some variables that influenced the site selection based on field work included the proximity to oil-well locations, gas flaring from Bonny Bright, high population density, socio-economic activities, particularly fishing within the area, with heavy presence of sewage, indiscriminate disposal of solid waste in and around the shore of Qua-Iboe river. Locally-consumed fresh Yellow tail (*Seriola lalandi*), Atlantic Croker (*Micropogonias undulates*), and Tilpia (*Oreochromis niloticus*) were collected for this study by a resident fisherman using set nets. These species of fishes, irrespective of their sex and age were weighed. The fishes were wrapped in hexane-rinsed aluminum foil. They were labelled and placed inside closed-glass vessels containing ice pack, and were kept at below -20°C before being taken for laboratory analysis.

2.4. Sample Preparation, Extraction and Clean-Up Procedure of Fish Samples for Analysis

Prior to extraction, the fish samples were descaled using knife, and subsequently dissected to obtain the tissues. From each specimen, an excision of 15 g of the fish tissue was placed in a clean mortar, and ground with pestle with 40g of anhydrous sodium sulphate - until the sample was completely dried and homogenized. The sample extraction was carried out using dichloromethane (DCM). A sub-sample of 10g of the homogenized sample was placed in 50ml extraction bottle and 1mL of 60ng/mL of 1- Chloro-octadecane surrogate standard was added in the extraction bottle. The content was agitated or vortexed for five hours, and was allowed to settle for one hour. The sample was then carefully filtered through a funnel fitted with cotton wool, silica gel, and Sodium Sulphate (Na_2SO_4) in a clean volumetric flask. The residue was further washed and made-up to volume using the extracting solvent. The sample was concentrated to 2 ml for PAHs analysis using a gas Chromatography Tandem-Mass Spectroscopy (GC-MS).

2.5. Extraction of Fish and Water Samples for PAH Determination

The liquid-liquid extraction procedure was used in this analysis as follows. One litre of the sample was extracted in a 2L glass separator funnel fitted with a glass stopper using 30 mL of hexane as extract. The separator funnel was vigorously shaken for three minutes, and the organic layer was allowed separating clearly from the aqueous phase for a minimum of five minutes, and - the organic layer was then collected into a separate glass bottle. The extraction was repeated thrice for each sample. Water residues were expelled from the organic layer by passing extracts through funnels containing anhydrous Sodium Sulphate. Extracts were concentrated using rotary evaporators with the water bath preset at 85^0 C. Concentrated extracts were transferred to a pre-weighed sample bottle and were evaporated to dryness.

2.6. Calculation of Bio -Concentration Factor (BCF)

The bio-concentration factor was investigated using the McCarty (1986) method. The bio-concentration factor (BCF) is the ratio of the concentration of a particular chemical in the tissue to its concentration in water

(equation 1) below. Considering that - BCF is relevant only for the accumulation from water to compare with other BCFs, it is important to confirm that water is the only route of uptake. Conversely, the bioaccumulation factor (BAF) which is generally computed as the ratio between the contaminant concentrations in the tissues and multiple external sources (e.g., sediment, water, and diet) is useful in determining the tendency of hydrophobic compounds to accumulate in the tissue.

$$BCF\ with\ free\ PAHs\ in\ water = \frac{Tissue}{Water\ free} \quad Equation\ 1$$

BCF, (bio-concentration factor with free PAH in water) = [Tissue]/ [Water free]

BCF is BCF predicted. For example, the equation from McCarty (1986); = 0.046 KOw.

2.7. Statistical Analysis

All investigations were carried out in triplicate, and the data obtained were presented as mean ± standard deviation using descriptive statistics. The one-way analysis was used to compare mean variance among the samples. Significance was accepted at $p < 0.05$ level using SPSS software, (version 18).

3. Results

3.1. Bioaccumulated PAHs in African Red Snapper (Lutjanus agennes) Fish

Table 1 shows the mean concentration of individual PAHs in river-water samples, mean bio-concentration of individual PAHs in the fish tissue, and the bioaccumulation factor of individual PAHs in *L. agennes* specimen. Extrapolated data revealed that *L. agennes* had the highest PAHs body load compared with the other fish species analyzed. For instance, the BCF for BaP was 2.15±6.19, while indeno (1, 2, 3, cd) pyrene was the highest body load or burden at 2.19±1.13. Meanwhile, other PAHs compounds recorded higher BCF values beyond the permissible limit. For example, naphthalene had 1.27±8.78, 2-methylnaphthalene at 1.07±0.23, acenaphthylene at 1.81±0.36, acenaphthene at 1.09±0.04, fluorene at 1.03±0.18, phenanthrene at 1.07±0.17 and anthracene 1.07±2.11, respectively.

Table 1. Bio-accumulated PAHs in African Red snapper (*Lutjanus agennes*) fish.

Polycyclic aromatic hydrocarbons (PAHs)	Mean ± SD of Contaminant in African Red snapper fish (ppm)	Mean ± SD Bio-concentration factor (BCF)
Naphthalene (ppm)	29.763±1.001	1.277±8.780*
2-Methylnaphthalene (ppm)	30.260±0.060	1.072±0.231
Acenaphthylene (ppm)	30.100±0.081	1.817±0.368
Acenaphthene (ppm)	30.133±0.049	1.092±0.040
Fluorene (ppm)	28.106±0.023	1.031±0.187
Phenanthrene (ppm)	30.533±0.367	1.074±0.174
Anthracene (ppm)	30.360±0.467	1.074±2.113*
Fluoranthene (ppm)	27.416±5.035	0.997±12.556*
Pyrene (ppm)	28.773±1.157	1.020±1.060*
Benzo (a) anthracene (ppm)	21.480±2.295	0.817±10.432*
Triphenylene (ppm)	30.183±0.119	1.038±0.361
Benzo (e) pyrene (ppm)	26.623±6.160	0.965±29.194*
Benzo (a) pyrene (ppm)	29.773±0.687	2.146±6.189*
Indeno (1,2,3,cd) pyrene (ppm)	30.116±0.241	2.186±1.131*
Benzo (g,h,i) perylene (ppm)	30.536±0.730	1.075±3.476*
Dibenzo (a,h) anthracene (ppm)	30.526±0.180	1.078±1.782*
000053-70-3-benzo(e) pyrene ppm)	29.490±1.625	1.059±9.503*

Results are mean ± SD for 3 determinations

Column marked with asterisk (*) indicates standard deviation greater than the mean. This is because the water free PAHs were considerably higher than the partitioned PAHs in fish tissue after computation.

3.2. Bio-Accumulated PAHs in Great Barracuda (Sphyraena barracuda) Fish

Table 2 shows the mean concentration of individual PAHs in river water samples, mean bio-concentration of individual PAHs in the fish tissue, and the bioaccumulation factor of individual PAHs in the Great Barracuda (*Sphyraena barracuda*) fish. The bio-concentration values of PAHs in the fish tissue indicated a low PAH contamination in all the fish samples analyzed. The BCF values are considerably lower compared to other fish species. For example, BeP had 0.361±1.602, BaP 0.683±6.099 and Indeno (1, 2, 3, cd) pyrene at 0.689±1.709. Other PAHs compounds such as 2-methylnaphthalene, acenaphthylene, acenaphthene, fluorine, phenanthrene, anthracene, fluoranthene and pyrene were recorded in lower BCFs, respectively.

Table 2. Bio-accumulated PAHs in the Great Barracuda (*Sphyraena barracuda*) fish.

Polycyclic aromatic hydrocarbons (PAHs)	Mean ± SD of Contaminant in Barracuda fish (ppm)	Mean ± SD Bio-concentration factor (BCF)
Naphthalene (ppm)	9.360±0.795	0.402±6.974*
2-Methylnaphthalene (ppm)	9.550±0.446	0.339±1.930*
Acenaphthylene (ppm)	9.4300±0.578	0.569±2.627*
Acenaphthene (ppm)	9.806±0.596	0.355±0.492
Fluorene (ppm)	8.777±1.154	0.322±9.382*
Phenanthrene (ppm)	8.726±0.903	0.307±0.43
Anthracene (ppm)	10.086±0.030	0.357±0.135
Fluoranthene (ppm)	9.766±0.516	0.355±1.286*
Pyrene (ppm)	9.753±0.575	0.346±0.527
Benzo (a) anthracene (ppm)	9.913±0.375	0.377±1.705*
Triphenylene (ppm)	9.826±0.422	0.337±1.283*
Benzo (e) pyrene (ppm)	9.960±0.338	0.361±1.602*
Benzo (a) pyrene (ppm)	9.473±0.677	0.683±6.099*
Indeno (1,2,3,cd) pyrene (ppm)	9.490±0.364	0.689±1.709*
Benzo (g,h,i) perylene (ppm)	9.800±0.616	0.345±2.933*
Dibenzo (a,h) anthracene (ppm)	10.093±0.565	0.357±5.594*
000053-70-3-benzo(e) pyrene ppm)	9.676±0.480	0.348±2.807*

Results are mean ± SD for 3 determinations

Column marked with asterisk (*) indicates standard deviation greater than the mean. This is because the water free PAHs were considerably higher than the partitioned PAHs in fish tissue after computation.

3.3. Bioaccumulated PAHs in the African Cat- Fish (Clarias gariepinus)

Table 3 shows the mean concentration of individual PAHs in river water samples, mean bio-concentration of individual PAHs in the fish tissue, and the bioaccumulation factor of individual PAHs in *Clarias gariepinus* samples. Results followed a similar trend to table 1. Though there is dissimilarity in values in terms of concentration and the bio-concentration factor. Considering PAH body load or burden in cat fish, results showed that the PAHs compounds accumulated were tolerable. For instance, Benzo (a) anthracene recorded 0.365±2.722 ppm, and Benzo (e) pyrene was 0.367±0.286 ppm. These values are below the 0.5 ppm threshold given by McCarthy *et al*., (1986) as the toxic level in aquatic organisms.

Table 3. Bio-accumulated PAHs in cat-fish *(Clarias gariepinus).*

Polycyclic aromatic hydrocarbons (PAHs)	Mean ± SD of Contaminant in cat - fish (ppm)	Mean ± SD Bio-concentration factor (BCF) ppm
Naphthalene (ppm)	10.517±0.453	0.451±3.974*
2-Methylnaphthalene (ppm)	10.476±0.551	0.371±2.385*
Acenaphthylene (ppm)	10.170±0.500	0.613±2.272*
Acenaphthene (ppm)	10.097±0.023	0.366±0.019
Fluorene (ppm)	10.146±0.115	0.372±0.934
Phenanthrene (ppm)	10.400±0.294	0.366±0.140
Anthracene (ppm)	10.373±0.458	0.367±2.072*
Fluoranthene (ppm)	11.646±0.803	0.423±2.002*
Pyrene (ppm)	10.360±0.312	0.367±0.286
Benzo (a) anthracene (ppm)	10.613±0.512	0.404±2.327*
Triphenylene (ppm)	10.206±0.215	0.351±0.653
Benzo (e) pyrene (ppm)	10.323±0.195	0.374±0.924
Benzo (a) pyrene (ppm)	9.436±1.411	0.680±12.711*
Indeno (1,2,3,cd) pyrene (ppm)	9.656±1.064	0.701±4.995*
Benzo (g,h,i) perylene (ppm)	10.133±1.021	0.357±4.862*
Dibenzo (a,h) anthracene (ppm)	10.336±0.275	0.365±2.722*
000053-70-3-benzo(e) pyrene ppm)	10.193±0.020	0.366±0.117

Results are mean ± SD for 3 determinations

Column marked with asterisk (*) indicates standard deviation greater than the mean. This is because the water free PAHs were considerably higher than the partitioned PAHs in fish tissue after computation.

4. Discussion

Studies on wildlife have revealed some modification of the gonadal, reproductive development and hormones (Cynthia *et al.,* 2004). A number of abnormalities in the reproductive system of various wildlife species correlate with abnormalities found in human population. These substances in our environment, food, and consumer products interfere with hormone biosynthesis and metabolism resulting in a deviation from normal homeostatic control or reproduction. Reports from aquatic animal models, human clinical observations, and epidemiological studies show that endocrine disrupting compounds are a significant concern for public health. The available field evidence gives proof to the ubiquitous nature of PAHs in environmental media and fish.

The exposure to Benzo (a) pyrene may damage the reproductive system and cause cancer. Ingestion of Benzo (e) pyrene may cause gastrointestinal irritations. Dermal contact with Benzo (a) pyrene may lead to skin irritation. In the natural environment, Benzo (a) pyrene occurs as part of a mixture of polycyclic aromatic hydrocarbons (PAHs). The full effects of Benzo (a) pyrene on human health are unknown, however studies have shown that inhalation of PAHs or dermal contact with PAHs for extended periods of time can cause cancer. The maximum permissible concentrations (MPCs), the serious risk concentrations (SRC) for ecosystems, and the sixteen known polycyclic aromatic hydrocarbons (PAHs) were derived by Verbruggen (2012). This was computed for all individual PAHs in water and fish. Bioavailability and organism's physiology are the two important variables with major effects on chemical contaminant body burdens. Considering the wide spread of environmental PAHs, only the bioavailable fraction can enter the organism. Results revealed different mean PAHs bio-accumulation in the African Red snapper (*Lutjanus agennes*). These bio-accumulated toxicants in the fish are beyond the USEPA permissible limits - and the recommendation limit postulated by McCarty (1986) of = 0.046- KOw. Physiological factors, including lipid levels, the rates of uptake and elimination i.e. (metabolism, diffusion, and excretion), determine the contaminant body burden (Landrum *et al.*, 1994). Uptake is an important factor when determining body burden, since this is controlled by factors associated with bioavailability and organismal physiology. It is believed that the process of uptake of hydrophobic compounds is passive (vis-a-vis active transport) and is controlled by diffusion pressure (fugacity) because of the difference between the environmental matrix and the tissue load (Mckim, 1994). The uptakes of PAHs from water, and factors that control the concentration of the free PAHs (non-sorbed) are important, and can be attributable to toxicity and risk.

Comparatively, the Great Barracuda fish (*Sphyraena barracuda)* bio-accumulated lesser contaminants than the African Red Snapper fish. For this model, Benzo (a) pyrene 0.683±6.099 ppm and Indeno (1, 2, 3, cd) pyrene 0.689±1.709 ppm had the highest concentration, and posed a bigger risk burden on the organism. This study presents evidence that differential ability of fish species and tissues to bio-transform PAHs to more water-soluble metabolites and metabolites half-life may result in differences in body burden. Some studies indicate that a fraction of persistent PAH remains in the tissues of organisms that are exposed to PAHs for long periods, even in species capable of metabolizing these xenobiotics. Results of PAHs bio-accumulation indicate a higher degree and variability of PAHs body burdens in *C.gariepinus*. Comparing the toxicant body burdens in these aquatic fish species showed that they differ from other species, although they were all fished from the same river. Several factors may be attributed to the variation in concentration. Understanding the mechanism of chronic exposure to PAHs by fish is desirable when evaluating the population of aquatic animal models and the possibility of transferring contaminant body burdens in fish to humans via the food chain. Additionally, the carcinogenicity of PAHs presents a human health risk because some PAHs are bio-transformed to reactive metabolites which interact with DNA adducts. The ability to predict tissue burdens in aquatic organisms is important in the assessment of hazard and toxic effects. Considering the lethal body burdens of non-ionic hydrophobic compounds such as parent PAHs compounds, acting by narcosis, they are believed to be in the range of 2-6 pmol/g (wet wt) (McCarty 1990). For species with no capacity to metabolize PAHs, assessing contaminant body burdens in light of this synergetic reactions will be beneficial in determining deleterious effects. Perhaps, these body burdens can be applicable in the relationships between chemical exposure and ecological effects on aquatic species.

5. Conclusion

This study evaluated contaminant body load from existing bioaccumulation, using model organisms as screening tools. The use of indigenous fish species was desirable since the results of these species would have greater ecological significance to predict perceived risks or hazards associated with aquatic pollution. The direct measurement of bio-concentration gives an insight into toxicant tissue residues at the time and location of the sampling. Direct measurement was accomplished by measuring tissue concentrations in the field-collected organisms. It is concluded that the toxicant body burdens in the assessed fish species were beyond the permissible limits as described by USEPA.

References

Barron MG, Stehly GRand Hayton WL. 1990. Pharmacokinetic modeling in aquatic animals. I. Models and concepts. *Aquatic Toxic* **17**: 187-212.

Connell DW. 1991. Extrapolating test results on bioaccumulation between organism groups. *In*: **Bioaccumulation in Aquatic Systems-Contributions to the Assessment**, R. Nagel R and R. Loskill R. (Eds) , Proceedings of an international workshop, Berlin, 1990. VCH Publishers, Weinheim, pp 133-149.

Cynthia A, Heinlein R and Chawnshang C. 2004. Androgen receptor in prostate cancer. *Endocrine Revi.*,**25**(2):276–308.

Fortner AR and Sick LV. 1985. Simultaneous accumulations of naphthalene, a PCB mixture, and benzo(a)pyrene, by the oyster, *Crassostrea virginica. Bull Environ Contam Toxicol*. **34**:256-264.

Hom T. Varanasi U. Stein JE. Sloan CA. Tilbury, KL and Chan SL. 1995. Assessment of the exposure of subsistence fish to aromatic compounds following the Exxon Valdez oil spill. In: **Exxon Valdez Symposium Proceedings**, Rice SD, Spies RB, Wolfe DA and Wright BA (Eds). American Fisheries Society, Washington, DC.

Knezovich JP. Harrison FLand Wilhelm RG 1987. The bioavailability of sediment-sorbed organic chemicals: a review. *Water Air Soil Pollut* **32**:233-245.

Landrum PF. Dupuis WS Kukkonen J., 1994. Toxicokinetics and toxicity of sediment-associated pyrene and phenanthrene in *Diporeia* spp.: examination of equilibrium-partitioning theory and residue-based effects for assessing hazard. *Environ Toxicol Chem.*, **13**: 1769-1780.

Landrum PF. Lee H Lydy MJ. 1992. Toxicokinetics in aquatic systems: model comparisons and use in hazard assessment. *Environ Toxicol Chem***., 1** (1): 1709-1725.

McCarty LS. 1986. The relationship between aquatic toxicity QSARS and bioconcentration for some organic chemicals. *Environ Toxic Chem.*, **5**: 1071- 1080.

McCarty LS. 1990. A kinetics-based analysis of quantitative structure-activity relationships in aquatic toxicity and bioconcentration bioassays with organic chemicals. Ph.D. thesis, University of Waterloo, Waterloo, Ontario, Canada.

McCarty LS and MacKay D. 1993. Enhancing ecotoxicological modeling and assessment: Body residues and modes of toxic action. *Environ. Sci. Technol*. **27:**1719-1728.

McKim JM. 1994. Physiological and biochemical mechanisms that regulate the accumulation and toxicity of environmental chemicals in fish. *In*: **Bioavailability: Physical, Chemical, and Biological Interactions**, Hamelink JL. Landrum PF. Bergman HL and Benson WH (Eds). Lewis Publishers, Boca Raton, FL, pp 179-201.

Meador JP. Casillas E. Sloan CA Varanasi U. 1995. Comparative bioaccumulation of polycyclic aromatic hydrocarbons from sediment by two infaunal organisms. *Mar Ecol Prog Ser*., **123**: 107-124.

Neff JM. 1976. Polycyclic aromatic hydrocarbons. *In*: **Fundamentals of Aquatic Toxicology,** Rand GM and Petrocelli SR (Eds). Hemisphere Publishing Co., New York, pp 416-454.

Stein JE, Hom T, and Varanasi U. 1984. Simultaneous exposure of English sole (*Parophrys vetulus*) to sediment-associated xenobiotics: Part 1 -uptake and disposition of '4C-polychlorinated biphenyls and 3~-benzo[a]pyrene. *Mar Environ Res.*, **13**:97-119.

Stein JE, Reichert WL, French B and Varanasi U. 1993. 32P-postlabeling analysis of DNA adduct formation and persistence in English sole (Pleuronectes vetulus) exposed to benzo[a]pyrene and 7H-dibenzo[c,g]carbazole. *Chem Biol Interact*., **88(1):**55-69.

Varanasi U, Stein JE and Nishimoto M. 1989. Biotransformation and disposition of polycyclic aromatic hydrocarbons (PAH) in fish. *In*: **Metabolism of Polycyclic Aromatic Hydrocarbons in the Aquatic Environment,** Varanasi U (Ed). CRC Press, Boca Raton, FL, pp 94-149.

Verbruggen EMJ. 2012. Environmental risk limits for polycyclic aromatic hydrocarbons (PAHs) for direct aquatic, benthic, and terrestrial Toxicity. National Institute of Public Health and the Environment. RIVM report *607711007/2012*; pp: 165-182.