Heavy Metal Pollution Of Fish Of Qua-Iboe River Estuary: Possible Implications For Neurotoxicity

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Citation

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Abstract

The Qua-Iboe River rises from Umuahia in Abia State. It traverses about 150 km and empties into the Atlantic Ocean via Eket (Ibeno LGA) Akwa Ibom State, Nigeria. The Social, economic and cultural lives of the people are significantly linked to the river. Its estuary situates close to Exxon-Mobile crude oil effluent processing and treatment facility. The heavy metal pollution status of the estuary was investigated in the Qua-Iboe Terminal (QIT) community on the bank of the estuary, using the method of Franson (1975). Based on WHO safety reference standards, the results showed that the water was polluted with respect to all the metals tested except Mn and Zn. The mean (X) (n=10) was Ni = 0.21± 0.01, Cr = 0.53 ± 0.01, Cd = 0.03 ± 0.01, Mn = 0.14 ± 0.09 and Pb = 0.3 ± 0.1 mg/L respectively. The fish bioaccummulated some of the metals: Mean (X) (n=7) for Ni = 1.9 ± 0.1,Cr = ND, Cd = 0.38 ± 0.06, Mn = 12.85 ± 0.66 and Pb = 25.58 ± 1.2 mg/g respectively. Based on WHO safety reference standard, the fish were polluted with respect to all the metals except Zn and Cr, which was not detected. These metals could pass to humans through the food chain. Cd, Mn, Pb, Zn and Ni are known to be neurotoxic. This may predispose the water and fish consumers to possible neurotoxicity, especially the QIT community. Regular medical checks may be necessary to prevent residual neurotoxicity in the community. Regulatory environmental measures should be put in place to ensure the safety of the estuary and its aquatic life.

INTRODUCTION

Pollution describes the introduction of foreign substances into the biosphere. As xenobiotics, some of these pollutants sometimes find their way into the human system through the food chain. In the body, they may undergo biotransformation, metabolism and excreted without the risk of toxicity depending on the chemical characteristics of the compound and the dose. However, some of the pollutants resist chemical and biological transformation and accumulate in the tissues, including the nerves, to cause toxicity. The adverse effects of these pollutants on the nerves give rise to neurotoxicity.

Some heavy metals are neurotoxic. For instance lead, mercury, nickel, zinc, cadmium, chromium and manganese ([1,2,3,4]). The commonest source of heavy metal pollution are industrial and mining activities, petroleum exploration, exploitation, processing and effluent management, atmospheric condensation and sewage disposal. Natural phenomena such as earthquake, landslides, tornadoes and cyclones have been implicated ([5,6]). Nuclear reactor accidents, and solid weapons sometimes discharge heavy metal pollutants, which constitute potential dangers to the environment. For instance, the Chernobyl (Ukraine, April 1986) radioactive metal pollutants left about 4000 dead and 60000 at the risk of dieing from thyroid cancer. The Jintsu river, Japan, victims died after eating methyl mercury contaminated fish polluted by an industrial effluent discharged into the river ([7]).

Qua-Iboe River is about 150km long and empties into the Atlantic Ocean. The estuary situates in close proximity to the Exxon-Mobile oil effluent treatment and discharge plant. The wastes are discharged into the Atlantic Ocean but may recycle into the estuary due to tidal motion. This understanding gave rise to the current study. The objective was to find out if the estuary was polluted with neurotoxic heavy metals, the type and possible effect of the metals – the sources of the pollution, the influence on some marine life and the possible effect on the fishermen and the oil workers who make up the population of the estuary community, whose economic and cultural lives depend on the estuary. QI – terminal is the first community closest to the effluent discharge facility. This study would attempt to provide answers to some of the questions raised above as it affects the QIT community on the bank of the estuary.

The information obtained may provided the basis for rational policies that would regulate the state of pollution of the estuary, and save the inhabitants from possible metal neurotoxicity arising from use of the water and consumption of its aquatic life. At the moment, there is no reported case of neurotoxicity, neither a study on the possible neurotoxic effect of the heavy metal pollutants of the estuary. This provides a strong basis for the present study.

MATERIALS AND METHODS

A field survey was done in Que-Iboe estuary communities. Qua-Iboe Terminal (QI-T) community was selected for the study of heavy metal neurotoxic potentials because of its proximity to the effluent discharge and processing plant of Exxon-Mobil crude oil facility. Water was randomly collected during the rainy and dry seasons, February – March, July-August.

The concentrations of Pb, Mn, Cr, Cd, Zn and Ni were determined using Atomic Absorption spectrophotometric (AAS) technique. Stock solutions were prepared from which serial dilutions were done for the working standards. Calibration curve for each element was plotted following the procedure in the Technical Bulletin 27 ($_{8}$). The standard method for the examination of water and waste water ($_{9}$) was used.

Mixed species of fish were directly procured from the artisan fishermen in the QIT community at the beach of the estuary. They were stored in coolers under-ice-flakes to preserve their freshness. They were subsequently stored in a refrigerator before the study.

Each fish was descaled and dried in an electric oven at 80°C for 3 hours. They were pulverized in a clean mortar, dried again and stored dried in a polythene bottles. The methods of Capon as reported by Obodo ($_{10}$) was used to digest the fish: 1.0g of pulverized fish was weighed into a 200ml kjeldah's flask. 10ml of $HclO_4$ and 100ml of HNO_3 were added to 20ml of the digestate. The mixture was gently swirled and digested in a fume cupboard at increasing temperature for 10 minutes until the brown fumes of NO_2 escaped. A golden yellow liquid was obtained after 3 hours of digestion. The liquid was cooled and transferred into 50ml volumetric flask and made up to the mark with 0.7% HNO_3 solution. The metal concentrations were determined using AAS (Hitachi model 180-70 polarized zeeman) by adopting specific cathode-ray tube for each heavy metal.

RESULTS

Figure 1

Table 1: Mean concentration of heavy metals in water and fish at Qua-Iboe Terminal (QIT).

Heavy metal	Water (mg/L)		Fish mg/g		Exon-Mobil Effluent (mean± SD) mg/L
	Mean* (n=10)	WHO standard	Mean (n=10)	WHO standard	
Lead	0.3 ± 0.1	0.01	25.58±1.2	1.5	1.18 ± 0.09
Manganese	0.14±0.09	0.5	12.85±0.66	2.5	0.29 ± 0.03
Chromium	0.53±0.01	0.05	ND	0.15	0.04 ± 0.04
Cadmium	0.03±0.01	0.005	0.38±0.06	0.2	0.03 ± 0.03
Nickel	0.21±0.01	0.1	1.9±0.1	0.4	0.23 ± 0.27
Zinc	0.14±0.02	3.0	6.65±0.45	150	0.34 ±0.03

The water was polluted with the metals except Mn and Zn. The fish was similarly polluted with the metals except for Zn and Cr, which was not detected.

*Mean ± Standard Deviation (X ±SD)

DISCUSSION AND CONCLUSION

Table 1 shows the mean concentrations of the heavy metals in fish and water. The mean Pb concentration was 0.3 ± 0.1 mg/L in water and 25.55 ± 1.2 mg/g in the fish. These were above the WHO($_{11}$) safety standard of 1.5 mg/L and 0.01 mg/g respectively. The results indicated that the water and the fish were polluted based on WHO standards. Obodo ($_{10}$) reported the case of polluted species of fish from the lower course of the River Niger at Onitsha. His work showed that tilapia was polluted with Pb having a bioconcentration of 68.36 mg/g.

Victims of Pb intoxication have been shown to manifest various forms of neurological syndrome such as lead palsy and lead encephalopathy, especially in children ($_{2}$). Some of the clinical manifestations include muscular weakness, fatigue, which are more pronounced in the fingers, wrist, toes and forearm; clumsiness, ataxia, headache, insomnia, irritability, grandmal seizures and gait. The fatality rate of Pb neurotoxicity is about 25% while about 40% of the survivors have to live with neurological sequelae such as mental retardation, optic atrophy and cerebral palsy ($_{1}$). The fish was polluted (25.58 ± 1.2 mg/g) against the WHO safety limit of 2.5 mg/g.

The role of manganese in neuropsychiatric disorders is also documented ($_{12,13}$). The water was not polluted with respect to Mn. The mean concentration in the water was 0.14 ± 0.09 mg/L, which was below the WHO reference standard of 0.5 mg/L. However, manganese is neurotoxic and manifests in the form of impulsive and aggressive behaviour; in some cases euphoria and sexual stimulation. Manganese selectively accumulates in the CNS, the bones, liver and

kidney ([14]).

Chromium was not detected in the fish at the QIT but the water was laden with the heavy metal (0.5±0.01 mg/L). It is possible that the species of fish in the QIT have efficient biotransformation mechanism for chromium ([2]); or that the fish species selectively exclude chromium from their food chain. These agree with the findings of Obodo ([10]) and the report of Nemerow ([5]). Cr is associated with the cancer of the lungs and kidney ([13]).

Cadmium occurs naturally in association with Pb, Zn, or Ni. The acute neurological effects of Cd toxicity manifests in the form of nausea and abdominal cramps, bloody diarrhea and vomiting, dizziness and chest pain. The so-called "itai-itai" disease found in Fuchu, Japan, after the WW II was traced to Cd contaminated rice field. The source was an industrial effluent discharged into the farm. The disease was characterized by rheumatic arthritis, muscular pain and osteomalacia in the elderly; and nephrotoxicity, carcinogenicity and testicular necrosis in rats ([2]). In this study the water and fish in QI estuary were polluted with Cd: 0.38 mg/L and 0.03 mg/g relative to WHO safety values of 0.2 mg/L and 0.005 mg/g. The source of the Cd may be the petroleum processing and effluent discharge facility at the QIT. Klaassen ([2]) reported that coal and fossil fuel are known sources of Cd pollution; besides, shellfish and animal liver are known to concentrate Cd. The residual deposits of the metal from oil spillage may be another source.

The Cd levels in the estuary and fish pose potential dangers for the QIT community who depend on the estuary and its aquatic life for subsistence.

Zn is a neurotoxin. By its ability to chelate and deplete the neuronal concentration of glutathione (GSH), it causes neuronal cell death in a dose dependent manner ([15]). The water and fish were not polluted with respect to Zn, 0.14 ± 0.02 mg/L and 6.65 ± 0.45 mg/g; when compared with the WHO standards of 3.0 mg/L and 150mg/g respectively.

There is so far no known study of the possible adverse neurological effects of the heavy metal pollution on the QI estuary. Besides, there are no reported cases of neurotoxicity arising from the direct use of the water or fish consumption from the estuary. However, this does not rule out the fact that it might exist. There are no sufficient data to enable a public health study or to initiate an ecological protection framework. The Qua-Iboe estuary and fish were polluted with respect to all the metals studied except Zn at the QIT community.

The work has provided some data and information that may be useful for such studies and policy formulation. There is the need for regular public health checks on the level of heavy metals among the community that border the estuary.

Measures should be put in place to control the treatment and discharge of effluent into the neighbourhood of the estuary.

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