



# Dietary exposure and health-risk assessment of toxic and essential metals in plantain from selected communities in Rivers State, Nigeria

Kingsley Chukwuemeka Patrick-Iwuanyanwu<sup>1</sup>,  
Nnaemeka Arinze Udowelle<sup>2</sup>

<sup>1</sup>Department of Biochemistry (Toxicology Unit), Faculty of Science, University of Port-Harcourt, Nigeria, <sup>2</sup>Department of Experimental Pharmacology and Toxicology, Faculty of Pharmaceutical Sciences, University of Port-Harcourt, Nigeria

**Address for correspondence:**  
Kingsley Chukwuemeka Patrick-Iwuanyanwu, Department of Biochemistry (Toxicology Unit), Faculty of Science, University of Port-Harcourt, Nigeria.  
E-mail: kctrendy@yahoo.co.uk

**Received:** March 28, 2017

**Accepted:** May 16, 2017

**Published:** July 01, 2017

## ABSTRACT

**Aim:** This study was conducted to assess the levels of lead (Pb), cadmium (Cd), copper (Cu), iron (Fe), zinc (Zn), cobalt (Co), manganese (Mn), and magnesium (Mg) in plantain samples collected from 10 selected communities in Rivers State, aiming to estimate the potential health risk of heavy metal exposure.

**Materials and Methods:** The samples were washed, oven-dried, and digested using a mixture of nitric acid and perchloric acid at a volume ratio of 4:1 at 150°C for 4 h. The samples were further analyzed using flame atomic absorption spectrometer. **Results:** The concentration (mg/kg) range in decreasing order was Mg (285-525) > Fe (60-345) > Cu (56.9-74.8) > Zn (5.48-96.0) > Mn (0.48-5.23) > Pb (0.78-2.0) > Co (0.23-1.28) > Cd (0.16-0.72). The results from the study exceeded the permissible limit set by WHO/FAO. A wide range of variation was observed in the estimated daily intake (EDI) of metals when compared with the tolerable daily intake of different regulatory bodies. The calculated EDI values for Pb, Cd, Cu, Fe, Zn, Mn, Co and Mg in samples from communities under study were highest in Etche, Nkpolu, Emohua, Emohua, Eleme, Eleme, Nkpolu, and Rumuosi, respectively. Target hazard quotient (THQ) values > 1 were recorded in some samples under study indicating a health risk, whereas the hazard index (HI) for plantain samples from all the communities under study was found to be > 1. The average lifetime carcinogenic risk of Pb and Cd through the consumption of plantain from selected communities in Rivers State ranged between 2.9E-05 – 5.6E-05 and 2.2E-04 – 9.1E04 for Pb and Cd, respectively. **Conclusion:** The overall study suggests that frequent consumption of plantain from these selected communities may cause a potential health risk to the exposed population.

**KEY WORDS:** Estimated daily intake, food security, health risk assessment, Niger Delta, plantain, target hazard quotient, Hazard index

## INTRODUCTION

The Niger Delta region of Nigeria is well known for its crude oil exploration activities. It is a region with abundant natural resources including good weather and fertile land for agriculture [1]. However, the negative impact of oil exploration, which includes the destruction of wildlife, loss of fertile soil, pollution of air and water, and damage to ecosystem of the communities [2] could pose a risk to the population in this area. Over the years, the boom in oil and gas industries combined with the population explosion and rapid urbanization in Rivers State could be a rich source of various toxic metals finding their way into the environment thereby contaminating food crops. The negative effects of ubiquitous and nonbiodegradable heavy metals, persist for several decades, and even longer [3]. These developments have led to metal dispersion in the environment and consequently

impaired health of the population by the consumption of food contaminated by harmful elements [4]. Heavy metals with adverse health effect in human metabolism such as lead (Pb), cadmium (Cd), mercury (Hg), arsenic (As), copper (Cu), nickel (Ni), zinc (Zn), and chromium (Cr) present obvious concerns due to their documented potential for serious health concerns [5]. The excessive ingestion of heavy metals can have severe public health implications; therefore, monitoring of these metals in seafood and food crops grown around the Niger Delta of Nigeria is of utmost importance [6]. Reports have shown that at low concentrations, elements such as Ni, Cd, Cr, and Pb are harmful [7-9]. Although Zn, cobalt (Co), iron (Fe), and Cu are essential elements, their excessive concentration in food and feed plants are of great concern because of their toxicity to humans and animals [10].

Plantain (*Musa paradisiaca*) is native to South East Asia and India but is now extensively cultivated in Nigeria and many

other parts of Africa where it serves as a major staple food. It belongs to the family *Musaceae* and the genus *Musa*. It is also locally known as *Ogede agbagba* (Yoruba), *Ayaba* (Hausa), and *Ogadejioko* (Igbo). Although various scientists have reported the ethnopharmacological relevance of plantain in the management of several ailments [11-14]. There is a high frequency of consumption of plantains among the natives of the Niger Delta, especially the ijaws of Bayelsa and Rivers States who prepare various delicacies using both unripe and ripe plantain. Furthermore, in view of the high consumption of plantain coupled with the high demand by diabetics who are majorly placed on unripe plantain diets, this study was carried out to determine the concentration of toxic and essential metals, estimate the dietary daily intake, the target hazard quotient (THQ), and general health risk assessment through the consumption of plantain.

## MATERIALS AND METHODS

### Reagents

All reagents used in this study were of analytical grades with high purity.

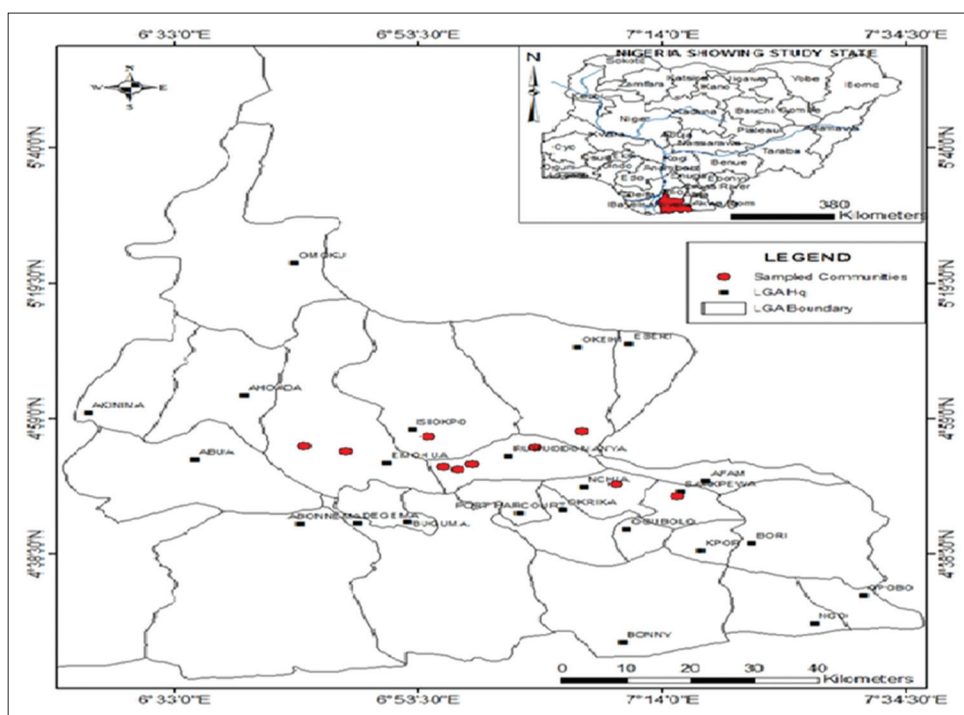
### Sample Collection and Processing

Samples of plantain grown and sold in different parts of Rivers state were either purchased from the open markets or collected from farm lands in 10 different communities namely: Nkpolu, Emohua, Elele, Choba, Rumukwurushi, Rumuosi, Etche, Ogoni, Eleme, and Aluu [Figure 1] in January 2015 and kept in plastic bags. The sources of plantain were authenticated from the seller at the point of purchase to validate they were locally

grown in the area. Only edible parts of the samples were used for the analysis. Selected samples of plantain were washed with clean water, peeled, sliced before being oven dried at 70°C for 72 h to constant weight. The samples were then pulverized with a ceramic mortar and pestle to fine powder. About 0.5 g of dried powdered sample was weighed and transferred into a pyrex beaker and were subjected to wet digestion with 10 mL  $\text{HNO}_3:\text{HClO}_4$  (at a volume ratio of 4:1) at 150°C for 4 h. Following this, samples were left to cool, filtered using a filter paper, transferred into a 50 mL volumetric flask and a final volume of 25 mL was made by adding deionized water. For each of the samples, triplicate digestion was carried out together with blank reagent and kept in refrigerator until analysis using atomic absorption spectroscopy (AAS).

### AAS Analysis

For each of the metals, AAS was calibrated using standard of the metals, which are given below as: Cr ( $\lambda$ ) = 357.90 nm, Cd ( $\lambda$ ) = 228.80 nm, Pb ( $\lambda$ ) = 283.30 nm, Zn ( $\lambda$ ) = 213.9 nm, Mn ( $\lambda$ ) = 279.50 nm, and Fe ( $\lambda$ ) = 248.3 nm. 5 g of the samples was digested in 20ml 10% hydrochloric acid (HCl) on a heating mantle to near dryness. Zn and Mn were analyzed using hollow cathode lamp in a flame atomizer AAS. Cd and Pb were analyzed using electrodeless discharge lamp in the flame atomizer AAS. The extract was aspirated directly into the AAS machine. The limit of detection for the heavy metals Cd, Cr, Cu, Pb, Zn, Fe, Mn, magnesium (Mg), and Co were 0.001, 0.007, 0.005, 0.001, 0.01, 0.1, 0.2, 0.01, and 0.05 mg/L, respectively, with blank values reading as 0.00  $\mu\text{g/g}$  for the metals in deionized water with an electrical conductivity value of  $<5 \mu\text{S cm}^{-1}$ . Standard quality control measures were carried out to reduce the risk of contamination and assure reliability of the results.



**Figure 1:** Map of Rivers state showing study areas

## Heavy Metal Health-Risk Assessment

### Estimated daily intake (EDI)

The health risks associated with the consumption of heavy metal contaminated plantain were assessed based on the EDI of heavy metals, THQ, and target carcinogenic risk (CR).

$$EDI = \frac{C_{\text{metal}} \times D_{\text{food intake}}}{BW_{\text{average}}}$$

Where:

$C_{\text{metal}}$  is the metal concentration in plantain in mg/kg,

$D_{\text{food intake}}$  is the daily intake of food in kg person<sup>-1</sup> and

$BW_{\text{average}}$  is average body weight in kg person<sup>-1</sup>.

An average daily consumption of 0.2 kg of plantain was assumed in this study. This method was adopted because plantain is widely consumed as a major part of the diet. Average adult body weight was considered to be 60 kg.

### Non-carcinogenic Health Effect

#### Calculation of THQ

Non-CR estimation of Pb and Cd consumption was determined using THQ values. THQ is a ratio of the determined dose of a pollutant to a reference level considered harmful. THQ values were determined based on the following formula [15].

$$THQ = \frac{Efr \times ED \times FIR \times C}{RfDo \times B_{\text{average weight}} \times ATn \times 10^{-3}}$$

Where Efr is exposure frequency in 156 days year<sup>-1</sup>, ED is exposure duration in 70 years (equivalent to an average lifetime) [16], FIR is average daily consumption in Kg person<sup>-1</sup> day<sup>-1</sup>, C is concentration of metal in food sample in mg/kg, RfDo is reference dose in mg/kg day<sup>-1</sup>, and ATn is average exposure time for non – carcinogens in days. The following reference doses were used (Pb = 4.0 × 10<sup>-3</sup>, Cd = 0.001, Cu = 0.04, Fe = 0.7, Zn = 0.3, Mn = 0.014, and Co = 0.043). THQs were calculated according to the methodology described by the Environmental Protection Agency (EPA) in the USA [17]. Doses were calculated using the standard assumption for an integrated risk analysis and an average adult body weight of 65 kg [17,18]. In addition, based on EPA guidelines, it was assumed that ingested doses were equal to absorbed contaminant doses [17,19].

### Hazard Index (HI)

The HI has been developed to estimate the risk to human health through more than one heavy metals [20]. It is the sum of the hazard quotients for all heavy metals and calculated by the equation below [21].

$$THI = \sum HQ = HQ_{Pb} + HQ_{Cd} + HQ_{Cu} + HQ_{Fe} + HQ_{Zn} + HQ_{Co} + HQ_{Mn} + HQ_{Mg}$$

### Carcinogenic Health Effect

CR

Slope factor is used to assess cancer risk. A slope factor and the accompanying weight-of-evidence determination are the toxicity data most commonly used to evaluate potential human CRs. In general, the slope factor is a plausible upper-bound estimate of the probability of a response per unit of a chemical over a lifetime. The slope factor is used in risk assessments to estimate an upper-bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen.

For carcinogen, which United States EPA (USEPA) identifies by a weight-of-evidence classification of the chemical, the estimated daily dose and the cancer slope factor are multiplied together to find the lifetime cancer risk posed by the chemical. Cancer slope factors are estimates of carcinogenic potency and are used to relate estimate daily dose of a substance over a lifetime exposure to the lifetime probability of excess tumors.

### Ingestion Cancer Slope Factors

The ingestion cancer slope factors evaluate the probability of an individual developing cancer from oral exposure to contaminants levels over a lifetime. Ingestion cancer slope factors are expressed in units of (mg/kg/day)<sup>-1</sup>.

Lifetime probability of contracting cancer due to exposure to site-related chemicals is calculated as follows:

$$\text{Lifetime probability of cancer, CR} = EDI \times CSF_{\text{ing}}$$

Where:

EDI is the estimated daily intake of each heavy metal (mg/kg/day).

$CSF_{\text{ing}}$  is ingestion cancer slope factor (mg/kg/day)<sup>-1</sup>.

The USEPA [22] states that 10<sup>-6</sup> (1 in 1,000,000) to 10<sup>-4</sup> (1 in 10,000) represent a range of permissible predicted lifetime risks for carcinogens. Chemical for which the risk factor falls below 10<sup>-6</sup> may be eliminated from further consideration as a chemical of concern. The risk associated with the carcinogenic effect of target metal is expressed as the excess probability of contracting cancer over a lifetime of 70 years [Table 1].

## RESULTS

The mean concentrations of Pb, Cd, Cu, Zn, Fe, Co, Mn, and Mg levels (mg/kg) in plantain consumed in selected communities under study are shown in Table 2. The concentration range of various metals in plantain was 0.78-2.0, 0.158-0.723, 7.48-5.69,

60-345, 5.48-33.9, 0.48-5.23, 0.13-1.28, and 283-525 mg/kg for Pb, Cd, Cu, Zn, Fe, Co, Mn, and Mg, respectively. The EDI of metals from the consumption of plantain is shown in Table 3. The highest EDI for Pb was observed in samples from Etche (0.0066) followed by Nkpolu (0.0058) and Rumukwurushi (0.0055) community while the highest EDI value of Cd was observed in samples from Nkpolu (0.0024) followed by samples from Elele (0.0023). Highest EDI for Cu, Fe, Zn, Mn, Co, and Mg was seen in Emohua (0.247), Emohua (1.14), Eleme

(0.32), Eleme (0.017), Nkpolu (0.0042), and Rumuosi (1.73) community, respectively. Figure 2 shows the THQ value from exposure to heavy metals through consumption of plantain. Pb (Nkpolu, Rumukwurushi, Rumuosi, Etche), Cd (Nkpolu, Emohua, Elele, Choba), Fe (Emohua), and Zn (Eleme) all had THQ values >1 while all other samples were <1. The result of the HI values (the sum of all THQs) through the consumption of plantain is shown in Figure 3. The HI for plantain samples from all the communities under study was found to be >1. In general, the HI of heavy metals from multiple consumption pathways is in the decreasing order Nkpolu > Emohua > Elele > Etche > Eleme > Rumukrushi > Rumuosi > Ogoni > Choba > Aluu. The average lifetime CR of Pb and Cd through the consumption of plantain from selected communities in Rivers State, in the Niger Delta region of Nigeria, is shown in Table 4. The result showed that average CR ranged between 2.9E-05 – 5.6E-05 and 2.2E-04 – 9.1E-04 for Pb and Cd, respectively.

**Table 1: Toxicological characteristics of the investigated heavy metals**

USDOE, 2011 USEPA, 2011 FAO/WHO, 1993	Ingestion reference dose	Ingestion carcinogenic slope factor
Symbol Factor unit	RfD <sub>ing</sub> (mg kg <sup>-1</sup> d <sup>-1</sup> )	CSF <sub>ing</sub> (mg kg <sup>-1</sup> d <sup>-1</sup> ) <sup>-1</sup>
Metals		
Cr	1.5	0.5
Cd	0.001	0.38
Pb	0.0035	0.0085
Mn	0.14	0
Zn	0.3	0
Fe	0.8	0

Cr: Chromium, Cd: Cadmium, Pb: Lead, Mn: Manganesh, Zn: Zink, Fe: Iron

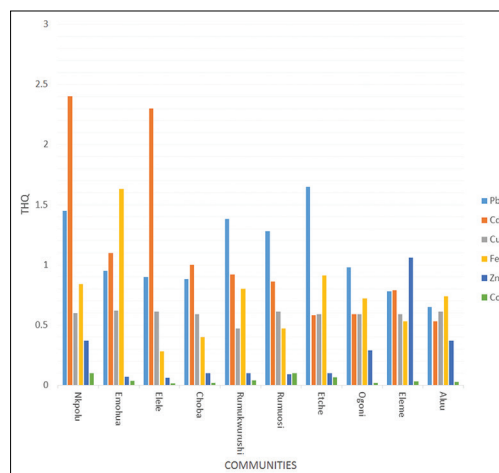
**Table 2: Mean concentration (mg/kg) of selected heavy metals in plantain samples from selected communities in Rivers state Nigeria**

Samples	Pb	Cd	Cu	Fe	Zn	Mn	Co	Mg
Nkpolu	1.75	0.723	72.9	180	33.8	3.5	1.28	283
Emohua	1.15	0.348	74.8	345	6.43	1.95	0.45	503
Elele	1.08	0.683	73.8	60	5.48	0.48	0.18	500
Choba	1.05	0.305	72.4	85	8.88	4.38	0.23	482
Rumukwurushi	1.68	0.278	56.9	170	8.80	3.63	0.53	473
Rumuosi	1.53	0.260	73.9	100	8.10	4.50	0.13	525
Etche	2.00	0.175	71.5	193	9.30	4.93	0.85	480
Ogoni	1.18	0.195	71.6	152	26.8	3.25	0.23	340
Eleme	0.95	0.240	71.6	112	96.0	5.23	0.38	487
Aluu	0.78	0.158	74.2	158	33.9	2.25	0.35	313
Permissible limit	0.3 <sup>1</sup>	0.2 <sup>1</sup>	73.3 <sup>1</sup>	30-150 <sup>2</sup>	60 <sup>3</sup>	500 <sup>4</sup>	-	-

Pd: Lead, Cd: Cadmium, Cu: Copper, Zn: Zinc, Fe: Iron, Mn: Manganese, Co: Cobalt, Mg: Magnesium. <sup>1</sup>FAO/WHO 2007, <sup>2</sup>Demirezen and Uruc 2006, <sup>3</sup>WHO 1982, <sup>4</sup>CODEX 2001

**DISCUSSION**

The study investigated the potential hazard of exposure to heavy metals through intake of *M. paradisiaca* (Plantain) collected from selected communities in Rivers State; a Niger



**Figure 2: Target hazard quotient calculated for heavy metals in plantain samples collected from selected c communities in Rivers state, Nigeria**

**Table 3: Estimated daily intake of metals (mg/kg day<sup>-1</sup> Person<sup>-1</sup>) for plantain in investigated communities for a 60 kg individual day<sup>-1</sup>**

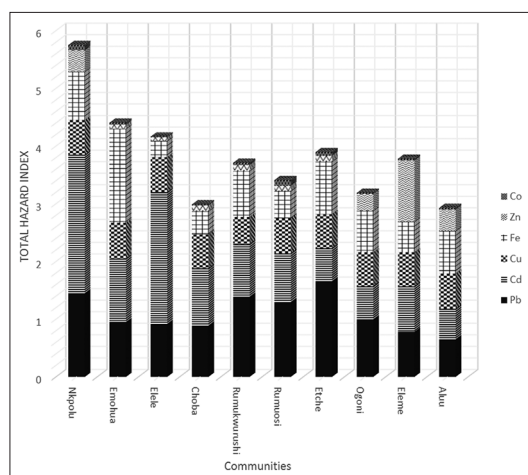
Samples	Pb	Cd	Cu	Fe	Zn	Mn	Co	Mg
Nkpolu	0.0058	0.0024	0.241	0.59	0.11	0.012	0.0042	0.93
Emohua	0.0038	0.0011	0.247	1.14	0.021	0.0064	0.0015	1.66
Elele	0.0036	0.0023	0.244	0.19	0.018	0.0016	0.0006	1.65
Choba	0.0035	0.00102	0.239	0.28	0.029	0.015	0.0008	1.59
Rumukwurushi	0.0055	0.00092	0.188	0.56	0.029	0.012	0.0017	1.56
Rumuosi	0.0051	0.00086	0.244	0.33	0.027	0.015	0.0004	1.73
Etche	0.0066	0.00058	0.236	0.64	0.031	0.016	0.0028	1.58
Ogoni	0.0039	0.00059	0.236	0.50	0.088	0.011	0.0008	1.12
Eleme	0.0031	0.00079	0.236	0.37	0.32	0.017	0.0013	1.61
Aluu	0.0026	0.00053	0.245	0.52	0.11	0.007	0.0012	1.03
TDI	NIL	0.001 <sup>1</sup>	0.04 <sup>1</sup>	0.83 <sup>1</sup>	0.3-1 <sup>1</sup>	-	0.0014 <sup>2</sup>	4.2 <sup>3</sup>

<sup>1</sup>Joint Expert Committee on Food Additives=JECFA (1982, 1983, 2010), <sup>2</sup>Dutch National Institute for Public health and the environment=RIVM (2001), <sup>3</sup>Science Committee on Food=SCF (2001). TDI: Tolerable Daily Intake. Pd: Lead, Cd: Cadmium, Cu: Copper, Zn: Zinc, Fe: Iron, Mn: Manganese, Co: Cobalt, Mg: Magnesium

**Table 4: Lifetime cancer risk calculation**

Communities	Pb	Cd
Nkpolu	4.9E-05	9.1E-04
Emohua	3.2E-05	4.2E-04
Elele	3.1E-05	8.7E-04
Choba	2.9E-05	3.9E-04
Rumukwurushi	4.7E-05	3.5E-04
Rumuosi	4.3E-05	3.3E-04
Etche	5.6E-05	2.2E-04
Ogoni	3.3E-05	2.2E-04
Elemo	2.6E-05	3.0E-04
Aluu	2.2E-05	2.0E-04

\*(ILCR=10<sup>-6</sup>) is the level of risk considered acceptable or inconsequential. \*(ILCR=10<sup>-4</sup>) is considered serious and of great public health concern. Pb: Lead, Cd: Cadmium



**Figure 3: Hazard index of heavy metals in plantain samples collected from Rivers state, Nigeria**

Delta region of Nigeria. The levels of toxic metals (Pb, Cd) and essential metals (Mg, Mn, Zn, Fe, Cu, and Co) were investigated in line with the recommendation of European Union regulation for hazardous metals [23]. The toxic metals (Pb and Cd) detected in various plantain samples are known to cause adverse effect to human health even at low levels [24]. Pb has been reported to be toxic at any concentration and also appears not to be beneficial or necessary as no safe level has been found [25]; hence, any concentration detected in food samples is of potential public health concern considering the frequency of exposure among the population. Pb and Cd are among the most common heavy metals of public health concern because they bio-accumulate in foods [26] and they are also readily absorbed in the body [27]. The increase of toxic metals along the food chain has been attributed to their nonbiodegradable and persistent nature [28,29]. However, the concentration range of Pb (0.78-2.0 mg/kg) and Cd (0.16-0.72 mg/kg) detected, in our present study, was higher than values reported by Orisakwe *et al.* [30] in 2012 for plantain grown in South-Eastern Nigeria, and lower than those reported by Zango *et al.* and Bortey-Sam *et al.* [31,32] all in Ghana. Although the European commission and WHO/FAO set a permissible limit of 0.3 and 0.2 mg/kg for Cd and Pb, respectively, there was a 100% and 40% violation, respectively, when compared with results of the present study.

In cases of clinical toxicity due to exposure to Cd, there is a reduction in glutathione, distortion of protein structure due to Cd binding to SH groups [33], these outcomes are exacerbated by interaction with other toxic metals such as Pb and As [34]. This indicates that an exposed population could probably be at risk of Pb and Cd related health diseases [35-37]. Among possible effects of lead poisoning includes neurological disorders in children and interstitial nephritis in adults, it can also lead to high blood pressure and cardiovascular disease after long-term consumption [38]. Other possible toxicological risks from long-term exposure to Cd includes Parkinson's and Wilson's diseases, impairment of Vitamin D metabolism in the kidney, renal tubular dysfunction, anemia, and calcium malabsorption [39,40]. Asides from cigarette smoke which is the most significant source of human exposure to Cd, ingestion of contaminated food is also a major route of exposure and could lead to long-term health effects [41]. In this study, Etche community had the highest concentration of Pb (2 mg/kg), followed by Nkpolu (1.75 mg/kg) and Rumukwurushi (1.68 mg/kg). The high levels of these toxic elements detected in plantain samples may be due to the presence of oil installations, vehicular activities and sparse bioremediation of crude oil polluted soils in these communities. Orisakwe *et al.* [42] reported high Blood Pb level among unexposed workers in Nigeria; this suggests that the major pathway of Pb exposure among Nigerians could be via consumption of contaminated foods.

Cu is an essential trace element with multiple biological roles; they act as a prosthetic group in several key enzymes [43]. Exposure to increased doses or frequent exposure to Cu can lead to headaches, dizziness, nausea, and diarrhea [44]. The levels of Cu in this study ranged between 56.9 and 74.8 mg/kg with the highest level observed in Emohua which is higher than the WHO permissible limit for Cu in food of 40 mg/kg [45]. The primary source of Fe exposure to humans is mainly through dietary consumption and its deficiency is a generally acknowledged problem due to its role as an essential metal for the synthesis of blood pigments [46]. The concentration of Fe in plantain ranged between 60-345 mg/kg and it is higher than the allowable limit of Fe in food crops which is in the range of 30-150 mg/kg [26]. Concentration of heavy metals above the permissible limits in food crops has been reported to be of serious public health concern as exposure to very high concentrations may induce oxidative stress for the exposed population [47]. The EDI levels of Fe in plantain varied from 0.19-1.14 mg/kg bodyweight (bw)/day. Plantain sample from Emohua which had the highest intake of Fe (1.14 mg/kg bw/day) was higher than the TDI of 0.83 mg/kg bw/day as shown in Table 3.

Zn requirements change throughout lifetime with numerous disorders associated with its deficiency resulting from insufficient dietary intake, reduced absorption and excessive elimination [47]. Ingestion of small amounts of Zn is essential for normal body metabolism [48]. The concentration of Zn in this study was below the WHO permissible limit of Zn in foods. More so, calculated EDI values were below the TDI of 0.3-1 mg/kg bw/day [46]. However, Zn can also induce toxicity when consumed at a concentration higher than the recommended dose [49].

Mn acts as a cofactor in various enzymes and is also required for normal mammalian physiological processes [50,51]. Over exposure to this metal however can be toxic, though there has never been any report of Mn toxicity from dietary exposure. Intoxication in occupationally exposed welders, miners, smelters and battery factory workers abound in literature [52-54]. The observable concentrations range of Mn in this study (0.48-5.23 mg/kg) was below the permissible limit of 500 mg/kg for Mn in food crops [55,56]. Furthermore, the calculated EDI values cannot be compared with any known standard (TDI) due to limitation in human data, and the EFSA in 2006 concluded that an upper intake for Mn cannot be set.

Co controls significant biological functions of few specific cellular proteins [57,58]. The major pathway by which Co get into the body is through the diet. The calculated EDI presented higher intake in Nkpolu, Emohua, Rumukwurushi, and Etche communities when compared with the TDI of 0.0014 mg/kg bw/day for Co as set by the RIVM in 2001 [59]. Toxic outcomes from excessive intake of this metal include overproduction of erythrocytes and fibrosis [60]. However, deficiency of Co in the diet is linked to Vitamin B<sub>12</sub> synthesis which might cause anemia and hypofunction of thyroid [61].

Mg metal serves as a cofactor in hundreds of enzymatic reaction, also its deficiency is linked to a number of chronic diseases [62,63]. The Science Committee on Food (SCF) in 2001 set an intake limit of 250 mg/day (approximately, 4.16 mg/kg for a 60 kg individual) for readily dissociable Mg salts. This reference intake limit does not include Mg normally present in food and beverages [64], as Mg in food has not been demonstrated to induce any adverse effect as stated above. Hence, the calculated values in this study cannot be compared with reference standards as Mg is present in crops.

Long-term consumption of foods contaminated with heavy metals and which are above their permissible limits have harmful effects and can cause noncarcinogenic threats such as neurological impairment, headache, and hepatotoxicity [65].

The THQ and corresponding HI values which are >1 for Pb and Cd suggests that consumption of plantain grown in these communities might be unsafe for consumption. The communities at high risk of toxic metal poisoning from contaminated plantain include Nkpolu, Rumukrushi, Rumuosi, and Etche for Pb toxicity on the one hand and Nkpolu, Emohua, and Elele for Cd toxicity on the other hand. THQ and HI values of <1 are suggestive that the exposed population is assumed to be safe. The calculated cancer risk of Pb and Cd with range 10E-05 – 10E-04 is of public health concern according to USEPA standard.

## CONCLUSION

The result from the study demonstrates that plantain grown and sold in selected communities of Rivers State under study are contaminated with heavy metals, and could pose a major health problem to the exposed population as the concentrations of Pb, Cd, Cu, Fe, Zn, Mn, Mg, and Co present in the most plantain

samples violated the permissible limits set by WHO/FAO. The levels of essential metals (Cu, Fe, Zn, Mn, Co, and Mg) in this study were present in high concentrations as shown in data from the study and the mean concentration was in the decreasing order Mg > Fe > Cu > Zn > Mn > Co. The EDI of the essential metals was observed to be mostly below the Recommended Daily Intake as proposed by WHO and SCF. The current study indicates that frequent consumption of plantain (*M. paradisiaca*) collected from the vicinity of study communities may be major sources of heavy metal exposure among the population and could lead to adverse effect with THQ value of >1 observed in Pb, Cd, Fe and Zn.

## REFERENCES

1. Abii TA, Nwosu TP. The effect of oil spillage on the soil of Eleme in Rivers state of the Niger Delta area of Nigeria. *Res J Environ Sci* 2009;3:316-20.
2. Aghalino SO. Petroleum exploration and the agitation for compensation by oil mineral producing communities in Nigeria. *J Environ Policy Issues* 2000;1:11-20.
3. Fantke P, Friedrich R, Jolliet O. Health impact and damage cost assessment of pesticides in Europe. *Environ Int* 2012;49:9-17.
4. Zukowska J, Biziuk M. Methodological evaluation of method for dietary heavy metal intake. *J Food Sci* 2008;73:R21-9.
5. Zhang X, Zhong T, Liu L, Quyang X. Impact of soil heavy metal pollution on food safety in China. *PLoS One* 2015;10:e0135182.
6. Osakwe SA, Okolie PL. Physicochemical characteristics and heavy metals contents in soils and cassava plants from farmlands along a major highway in Delta State, Nigeria. *J Appl Sci Environ Manage* 2015;19:695-704.
7. Golia EE, Dimirkou A, Mitsios IK. Influence of some soil parameters on heavy metals accumulation by vegetables grown in agricultural soils of different soil orders. *Bull Environ Contam Toxicol* 2008;81:80-4.
8. Kirkillis CG, Pasiadis IN, Miniadis-Meimaroglou S, Nikolaos ST, Zabetakis I. Concentration levels of trace elements in carrots, onions, and potatoes cultivated in Asopos region, Central Greece. *Anal Lett* 2012;45:551-62.
9. Parsafar N, Marofi S. Heavy metal concentration in potato and in the soil via drainage water irrigated with wastewater. *Irrig Drain* 2014; 63:682-91.
10. Kabata-Pendias A, Mukherjee AB. *Trace Elements from Soil to Human*. New York: Springer-Verlag; 2007.
11. Agarwal PK, Singh A, Gaurav K, Goel S, Khanna HD, Goel RK. Evaluation of wound healing activity of extracts of plantain banana (*Musa sapientum* var. *Paradisiaca*) in rats. *Indian J Exp Biol* 2009;47:32-40.
12. Surbhi G, Vipin KG, Pramod KS, Anita S. Analgesic activity of aqueous extract of *M. paradisiaca*. *Pharm Sin* 2011;4:74-7.
13. Jawla S, Kumar Y, Khan MS. Antimicrobial and antihyperglycemic activities of *M. paradisiaca* flowers. *Asian Pac J Trop Biomed* 2012;2:S914-8.
14. Nirmala M, Girija K, Lakshman K, Divya T. Hepatoprotective activity of *M. paradisiaca* on experimental animal models. *Asian Pac J Trop Biomed* 2012;2:11-5.
15. Singh A, Sharma RK, Agrawal M, Marshall FM. Risk assessment of heavy metal toxicity through contaminated vegetables from waste water irrigated area of Varanasi, India. *Trop Ecol* 2010;51:375-87.
16. Bennett DH, Kastenber WE, McKone TE. A multimedia, multiple pathway risk assessment of atrazine: The impact of age differentiated exposure including joint uncertainty and variability. *Reliab Eng Syst Saf* 1999;63:185-98.
17. United States Environmental Protection Agency (USEPA). Mid-Atlantic Risk Assessment: Human Health Risk Assessment. Available from: <http://www.epa.gov/reg3hwmd/risk/human/index.htm>. [Last accessed on 2014 Oct].
18. Wang X, Sato T, Xing B, Tao S. Health risks of heavy metals to the general public in Tianjin, China via consumption of vegetables and fish. *Sci Total Environ* 2005;350:28-37.
19. Cooper CB, Doyle ME, Kipp K. Risks of consumption of contaminated

- seafood: The Quincy Bay case study. *Environ Health Perspect* 1991;90:133-40.
20. United States Environmental Protection Agency (USEPA). Risk assessment guidance for superfund. In: Human Health Evaluation Manual Part A, Interim Final. Vol. I. Washington, DC: United States Environmental Protection Agency; 1989.
  21. Guerra K, Konz J, Lisi K, Neebren C. Exposure Factors Handbook. Washington, DC: USEPA; 2010.
  22. United States Environmental Protection Agency (USEPA). Screening Level (RSL) for Chemical Contaminant at Superfund Sites. Washington, DC: U.S. Environmental Protection Agency; 2011.
  23. Council of the European Union. Setting maximum levels for certain contaminants in food stuffs. *Off J Eur Commun* 2006;L364:5.
  24. Hossen MF, Hamdan S, Rahman MR. Review on the risk assessment of heavy metals in Malaysian clams. *ScientificWorldJournal* 2015;2015:905497.
  25. Flora G, Gupta D, Tiwari A. Toxicity of lead: A review with recent updates. *Interdiscip Toxicol* 2012;5:47-58.
  26. Demirezen D, Uruç K. Comparative study of trace elements in certain fish, meat and meat products. *Meat Sci* 2006;74:255-60.
  27. Krejpcio Z, Trojanowska E. The effect of lead (II) and cadmium (II) ions on pepsin and trypsin activity *in vitro*. *Bromatol Chem Toksykol* 2000;33:43-8.
  28. Khan MU, Malik RN, Muhammad S. Human health risk from heavy metal via food crops consumption with wastewater irrigation practices in Pakistan. *Chemosphere* 2013;93:2230-8.
  29. Bhuiyan MA, Suruvi NI, Dampare SB, Islam MA, Quraishi SB, Ganyaglo S, *et al*. Investigation of the possible sources of heavy metal contamination in lagoon and canal water in the tannery industrial area in Dhaka, Bangladesh. *Environ Monit Assess* 2011;175:633-49.
  30. Orisakwe OE, Nduka JK, Amadi CN, Dike DO, Bede O. Heavy metals health risk assessment for population via consumption of food crops and fruits in Owerri, South Eastern, Nigeria. *Chem Cent J* 2012;6:77.
  31. Zango MS, Anim-Gyampo M, Ampadu B. Health risks of heavy metals in selected food crops cultivated in small-scale gold-mining areas in Wassa-Amenfi-West district of Ghana. *J Nat Sci Res* 2013;3:96-105.
  32. Bortey-Sam N, Nakayama SM, Ikenaka Y, Akoto O, Baidoo E, Mizukawa H, *et al*. Health risk assessment of heavy metals and metalloids in drinking water from communities near gold mines in Tarkwa, Ghana. *Environ Monit Assess* 2015;187:397.
  33. Valko M, Morris H, Cronin MT. Metals, toxicity and oxidative stress. *Curr Med Chem* 2005;12:1161-208.
  34. Whittaker MH, Wang G, Chen XQ, Lipsky M, Smith D, Gwiazda R, *et al*. Exposure to Pb, Cd, and as mixtures potentiates the production of oxidative stress precursors: 30-day, 90-day, and 180-day drinking water studies in rats. *Toxicol Appl Pharmacol* 2011;254:154-66.
  35. Edwards JR, Prozialeck WC. Cadmium, diabetes and chronic kidney disease. *Toxicol Appl Pharmacol* 2009;238:289-93.
  36. Sughis M, Penders J, Haufroid V, Nemery B, Nawrot TS. Bone resorption and environmental exposure to cadmium in children: A cross - Sectional study. *Environ Health* 2011;10:104.
  37. Youness ER, Mohammed NA, Morsy FA. Cadmium impact and osteoporosis: Mechanism of action. *Toxicol Mech Methods* 2012;22:560-7.
  38. World Health Organization (WHO). Lead: International Programme on Chemical Safety (IPCS), WHO Food Additives Series 44. Geneva, Switzerland: World Health Organization; 2000.
  39. Montgomery EB Jr. Heavy metals and the etiology of Parkinson's disease and other movement disorders. *Toxicology* 1995;97:3-9.
  40. Ogawa T, Kobayashi E, Okubo Y, Suwazono Y, Kido T, Nogawa K. Relationship among prevalence of patients with Itai-itai disease, prevalence of abnormal urinary findings, and cadmium concentrations in rice of individual hamlets in the Jinzu river basin, Toyama prefecture of Japan. *Int J Environ Health Res* 2004;14:243-52.
  41. Bernhoft RA. Cadmium toxicity and treatment. *Sci World J* 2013;2013:394652.
  42. Orisakwe OE. Environmental pollution and blood lead levels in Nigeria: Who is unexposed? *Int J Occup Environ Health* 2009;15:315-7.
  43. Jaiser SR, Winston GP. Copper deficiency myelopathy. *J Neurol* 2010;257:869-81.
  44. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Copper. Atlanta, GA: U.S. Department of Health and Human Services, Public Health Service; 2004.
  45. Bahemuka TE, Mubofu EB. Heavy metals in edible green vegetables grown along the sites of the Sinza and Msimbazi rivers in Dares Salaam, Tanzania. *Food Chem* 1999;66:63-6.
  46. Lynch SR, Baynes RD. Deliberations and evaluations of the approaches, endpoints and paradigms for iron dietary recommendations. *J Nutr* 1996;1269 Suppl:2404S-9.
  47. Jaishankar M, Tseten T, Anbalagan N, Mathew BB, Beeregowda KN. Toxicity, mechanism and health effects of some heavy metals. *Interdiscip Toxicol* 2014;7:60-72.
  48. Plum LM, Rink L, Haase H. The essential toxin: Impact of zinc on human health. *Int J Environ Res Public Health* 2010;7:1342-65.
  49. Gopalani M, Shahare M, Ramteke DS, Wate SR. Heavy metal content of potato chips and biscuits from Nagpur City, India. *Bull Environ Contam Toxicol* 2007;79:384-7.
  50. Aschner M, Guilarte TR, Schneider JS, Zheng W. Manganese: Recent advances in understanding its transport and neurotoxicity. *Toxicol Appl Pharmacol* 2007;221:131-47.
  51. Guilarte TR. Manganese and Parkinson's disease: A critical review and new findings. *Environ Health Perspect* 2010;118:1071-80.
  52. Huang CC, Chu NS, Lu CS, Wang JD, Tsai JL, Tzeng JL, *et al*. Chronic manganese intoxication. *Arch Neurol* 1989;46:1104-6.
  53. Keen CL, Lonnerdal B. Toxicity of essential and beneficial metal ions. *Handbook of Metal-Ligand Interaction in Biological Fluids*. New York: Marcel Dekker; 1995. p. 683-8.
  54. Ono K, Komai K, Yamada M. Myoclonic involuntary movement associated with chronic manganese poisoning. *J Neurol Sci* 2002;199:93-6.
  55. Codex Alimentarius Commission (FAO/WHO). Food Additives and Contaminants. Joint FAO/WHO Food Standards Program, ALINORM 01/12A. Geneva, Switzerland: Codex Alimentarius Commission; 2001. p. 1-289.
  56. WHO/FAO. Joint FAO/WHO Food Standard Programme Codex Alimentarius Commission 13<sup>th</sup> Session. Report of the Thirty Eight Session of the Codex Committee on Food Hygiene, ALINORM 07/30/13. Houston, United States of America: WHO/FAO; 2007.
  57. Klaassen CD, Casarett LJ, Doull J. Casarett and Doull's Toxicology-The Basic Science of Poisons. 8<sup>th</sup> ed. New York: McGraw-Hill.
  58. Scharf B, Clement CC, Zolla V, Perino G, Yan B, Elci SG, *et al*. Molecular analysis of chromium and cobalt-related toxicity. *Sci Rep* 2014;4:5729.
  59. Baars AJ, Theelen RM, Janssen PJ, Hesse JM, van Apeldoorn ME, Meijerink MC, *et al*. Re-Evaluation of Human-Toxicological Maximum Permissible Risk Levels, RIVM Report 711701025. Bilthoven, Netherlands: National Institute of Public Health and the Environment; 2001.
  60. Lombaert N, Lison D, Van Hummelen P, Kirsch-Volders M. *In vitro* expression of hard metal dust (WC-Co) - Responsive genes in human peripheral blood mononucleated cells. *Toxicol Appl Pharmacol* 2008;227:299-312.
  61. Battaglia-Hsu SF, Akkiche N, Noel N, Alberto JM, Jeannesson E, Orozco-Barrios CE, *et al*. Vitamin B12 deficiency reduces proliferation and promotes differentiation of neuroblastoma cells and up-regulates PP2A, proNGF, and TACE. *Proc Natl Acad Sci U S A* 2009;106:21930-5.
  62. Kupetsky-Rincon EA, Uitto J. Magnesium: Novel applications in cardiovascular disease - a review of the literature. *Ann Nutr Metab* 2012;61:102-10.
  63. Reed BN, Zhang S, Marron JS, Montague D. Comparison of intravenous and oral magnesium replacement in hospitalized patients with cardiovascular disease. *Am J Health Syst Pharm* 2012;69:1212-7.
  64. European Commission Health & Consumer Protection Directorate-General. Opinion of the Scientific Committee on Food on the Tolerable Upper Intake Level of Magnesium, 26 September; 2001.
  65. United States Environmental Protection Agency (USEPA). Risk-based Concentration Table. Washington, DC: United State Environmental Protection Agency; 2000.

© **EJManager**. This is an open access article licensed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0/>) which permits unrestricted, noncommercial use, distribution and reproduction in any medium, provided the work is properly cited.

**Source of Support: Nil, Conflict of Interest: None declared.**