

www.jchr.org



ORIGINAL ARTICLE

Non-carcinogenic and Carcinogenic Risk Potentials of metals Exposure from Vegetables Grown in *Sharada* Industrial Area Kano, Nigeria

Babandi Abba^{*1,2}, Ya'u Murtala^{1,2}, Muhammad Yakasai Hafeez¹, Shehu Dayyabu¹, Babagana Kamaludeen¹, Ibrahim Aminu¹,

Anosike C.A.², Ezeanyika Lawrence U.S²

¹Biochemistry Department, College of Health sciences, Bayero University, PMB 3011, Kano-Nigeria

²Biochemistry Department, University of Nigeria, Nsukka, 410001, Enugu-Nigeria

(Received: 28 January 2019 Accepted: 14 December 2019)

ABSTRACT: Chemical elements such as heavy metals significantly functions as trace elements but their bio-**KEYWORDS** toxicity to human biochemical process poses a great concern for public health. The human exposure to heavy metals via food chain has been a documented risk factor for cancer development and other health-related Vegetables; implications. The levels of some carcinogenic (Chromium Cr; Cadmium Cd and Lead Pb) and non carcinogenic Heavy metals; (Nickel Ni; Cobalt Co; Copper Cu and Zinc Zn) heavy metals in vegetables (Onion, Drumstick, Lettuce, Okra, Carcinogenic risk; and Carrot) grown in Sharada industrial area, Kano were evaluated. The human health risk implications were Health risk; ILCR; also determined by estimating the daily metal consumption, and calculating non cancer and cancer risks HLI: (Incremental Life Cancer Risk, ILCR and Target Hazard Quotients) using US-EPA probabilistic health risk Kano assessment (US-EPA risk predicting model). The mean concentration of lead (Pb) ranged from 11.21 ± 0.55 µg/g in Okra to 16.84 \pm 0.86 µg/g in onion. Carrot had the highest concentration of chromium (65.10 \pm 3.20 µg/g). The levels of various heavy metals found in the studied vegetables/plants differ generally with plant species. The predictive values of human developing cancer disease from the incessant intake of studied plant/vegetables was found higher than the standard threshold risk boundary set by US-EPA, $(>10^4)$ for Cr, Pb, and Cd. The non cancer health risk values also predicted Pb, Cr and Cd as the dominant metal contaminants of the vegetables with the Health Risk Index greater than one (>1), while Ni, Co, and Zn showed safe quotients. Among the studied vegetables/plants, carrot has predicatively, the highest cancer risk (Σ ILCR 1.25 with chromium contributing 92%), followed by lettuce, onion, Moringa oleifera, and okra (Carrot> Lettuce > Onion > Moringa > Okra). It is, therefore, suggests that the industrial area is unsafe for irrigation due to the heavy metals pollutions of the soil and irrigation water and the risk of high consumption of vegetables grown exposes the population to incremental cancer risks potential among others.

INTRODUCTION

Industrial emission of toxic metals and anthropogenic activities pollute human drinking water, agricultural and

irrigation soils as well as vegetables and food crops, posing potential health risks to the community and general population through distinct pathways such as direct oral ingestion, dermal contact and air inhalation [1, 2 and 3]. These metals are persistent, leading to environmental pollution and toxicity. The pollution by these metals in the aquatic environment such as irrigation soil and waters is a major global alarming situation [4,5]. The toxicity of heavy metal to human system is generally due to the reactivity of its ions with cellular and structural bio-molecules such as nucleic acids, structural proteins, enzymes, hormones, and bio-membrane system. The chronic and acute human toxic metals exposure can cause many sickness conditions in humans [6,7and 8]. Plants grown on metal polluted agricultural soils and waters or near industries demonstrate higher heavy metal content possibly above WHO permissible limits [3]. The intake of toxic metals via food chain is the main route of heavy metals exposure to humans [9]. It also, enters the human body through other routes such as topical and nasal inhalation [10]. These metals have the properties of biological accumulation, toxicity and environmental stability [4]. Once entered the living system, metals are deposited and accumulated slowly in different body tissues, causing different degree and type of illness. A metal such as Chromium is carcinogenic, mutagenic, and teratogenic in humans [11, 12]. Also, copper when in excess can cause brain and kidney damage [13]. Uranium, another harmful metal, is implicated in genotoxicity, carcinogenicity and cellular mutations [14].

Contaminated foods (e.g. heavy metals polluted) or unsafe food intake can be an important factor for various chronic and non-chronic human illnesses such as cardiovascular diseases (CVDs), cancers, renal problems, birth defects and diarrhea [15]. Fruits and Vegetables are important components of human diets because of possible healthimproving properties when consumed purely and regularly. Nowadays, people worldwide have been practicing high consumption of plant-based foods or fresh vegetables and fruits as an alternative to meats or meat-base diets for healthy body. The plants/vegetables were reported to reduce significantly the incidences of some chronic human diseases including diabetes, CVDs, human cancers, infections, brain and age related disorders [16]. The hazardous vegetables intake, however, is a critical health issues due contamination with chemicals more specifically heavy metals; which is caused by human anthropogenic activities and the metals grossly implicated in causing various human cancers when bio-accumulated over time [15, 17]. Foodstuff safety is therefore, main public health issues and the significant associated with consumption of edible part of food crops contaminated by these metals have been increased globally [18, 19]. Soil metals uptake has been studied and reported in various plant vegetables such as lettuce, okra, and onion [3] via assessing the heavy metal in edible portion of most plant or vegetables. The use of industrial effluents or wastewater for farming is becoming norms or common practice in big cities of developing countries and also the application of agrochemicals especially metal-based inorganic fertilizers, chemical herbicides and pesticides play a significant part in contaminating farm harvests such as vegetables [20].When a comparison with most world developed countries is made, Nigerians may have greater human risks from metals in vegetables exposure. This is due considerable environmental pollution related to increasing population, a quick development of new Agro allied, manufacturing and leather industries, various anthropogenic activities as well as lack of public environmental risk awareness campaign associated with various degrees of heavy metals exposures. Vast lands of farms in this study area are mostly irrigated by metal-contaminated water and the farmers produced many types of vegetables of economic magnitude [3]. Usual assessment and monitoring of metal concentrations and other food toxicants in vegetables and other food crops cultivated close to mining areas and manufacturing industries have been conducted and documented in most industrialized nations and some developing countries, however, there is an inadequate available data on vegetables heavy metal levels and dietary consumption of these contaminants in the fruits, crops and vegetables as well as

estimated health risk in Nigeria, and specifically, Kano. The results of this health risk assessment study will strongly guide us of the degree of heavy metal contamination in common dietary vegetables and foods. This can provide basis for data comparison to other parts of Nigeria and world at large.

MATERIALS AND METHODS

Study site

Kano (Latitude 12°02 N, Longitude 08°30 E) is one of the largest and highly populated Nigeria's state, with a projected human population of over 10 million according to the 2006 national population census reported. It is the historic sub-Saharan trade and business nerve center of Northern Nigeria. The large population of the state is basically due to economic, industrial and other economic

activities. The main industrial areas of the state are Bompai, Sharada (Phase I, II and III), and Challawa all located within a two river tributaries. The discharge of crude domestic and industrial effluents in these rivers is posting a great threat to the surrounding environment, the water resources and the health of people living in the area [21]. According to Chen [22] rivers, generally have economic and ecological benefits to the community, but these rivers are prone to contamination due to their easy accessibility, naturally open, and used extensively in agricultural irrigation, industrial, and domestic activities [23]. The industrial discharges are generally used for growing vegetables beside the water bank, thus, metal and other contaminants can accumulate in vegetables via food chain thereby exposing local farmers and consumers to various health problems.



Figure 1. Map of Kano State, northwestern Nigeria, showing the study region. Source: www.nigerianmuse.com/20100527092749zg/maps -various states-and-their-local-governments-in-Nigeria

Sample collection/Design

Plant and soils (about 30cm deep) samples were collected from the irrigation sites near *Sharada* industries. Water samples were collected using containers (rinsed with sample water thrice prior to collection) of 0.5 dm³ capacity. This was done in replicate. Plants samples obtained were: Onion (*Allium cepa*), Drumstick (*Moringa oleifera*), Lettuce (*Lactus sativa*), Okra (*Hibiscus esculentus*) and Carrot (*Daucus carota*), and were then taken immediately to the laboratory.

Sample preparation

Each plant (edible portion) was cut using stainless steel knife and then allowed to oven dry at 105°C to constant weight. The samples were then pounded to a powder form after which were put in labeled containers and kept in desiccators.

Heavy metals determination

The prepared plants and soil samples (1g each) were digested with 5ml diacid mixture of nitric acid: Percholoric acid in 5: 2 ratios [24]. Later, the de-ionized water was added after digestion and then filtered with Whatman-42 paper. Metals in the digested samples were detected by Atomic Absorption Spectrophotometery (AAS) method, using Buck 210VGP machine model as reported by AOAC [24]. Manufacturer's instructions giving all the necessary detailed information regarding the apparatus and operational procedures of the AAS were strictly followed.

Health Risk Assessment of Heavy metals

For heavy metals, the cancer risks are predicted as the lifetime incremental likelihood of a person developing cancer disease as a consequence of potential heavy metal exposure.

The health risk calculation of metals contamination of edible plants

For human health risk estimation of these heavy metals, the U.S EPA recommended health risk assessment model was employed [25]. The cancer risk and the hazard index were widely employed to predict carcinogenic and non-carcinogenic effects of toxicants respectively. Previously, many studies have reported and adopted this model for the estimation, assessing and prediction the implications of the heavy metals to human's health [26, 27].

Human health risk of heavy metals by consumption of metal contaminated vegetables or plants is determined by

hazard quotient (HQ) index [25]. The HQ is defines as the ratio between metal exposure and the standard computed oral reference Dose (RD) of metals. When the computed HQ is less than 1 (One), it signified no apparent human health risk. The HQ of the study samples is determined by the equations below:

HLI/HQ = vegetable daily intake (kg/day)* standard oral reference dose (mg/kg of body weight/day)* human body weight (kg)....(1)

Daily Intake Rate (DIR) = Metal concentration $(mg/kg) \times$ standard conversion factor \times vegetable intake (Kg person/day of fresh weight)...(2) [28, 29].

Where: The standard conversion factor is given as 0.085

The factor (0.085) is place toward converting fresh plant vegetables weight to constant dry weight based on the above Equation [30]. The mean daily metal intake for adult humans is set at 0.345 kg/person/day (fresh weight). The average weight of adults was set at 60 kg in this study [25, 31].

Chronic Daily Intake of metal (CDI) = Estimate of Daily consumption of vegetable * Exposure Frequency* (365days/Year)* Life time exposure Duration/ A*T (365days/year*52years)

Where: Estimated Daily consumption of vegetables= 0.345kg for adults [31].Exposure frequency for individual = 365days/year; Exposure duration = 52years which is the average life expectancy of Nigerians [32].

Incremental Lifetime Cancer Risk (ILCR) = *Chronic Daily Intake (CDI)* Cancer Slope Factor (Oral)* was determined by the method of Wongsasuluk and colleagues [33].

ILCR=CDI*CSF-oral

The cumulative cancer risk due to exposure to many cancercausing metals from human intake of a plants is believed as total of a person metal incremental risk and determined as suggested by Liu and colleagues [19] equation:

$$\sum ILCR_1 + \sum ILCR_2 + \sum ILCR_3 \dots + \sum ILCR_n$$

Where: n = 1, 2, 3 ... n is the individual toxic metals or the vegetables/plants in this study.

Total Hazard Quotient (THQ)

THQ is calculated by the equation of Michael et al. [34].

THQ = CDI/RfD

Hazard Index (HI)

 $HI = THQ_1 + THQ_2 + \dots + THQ_n$

Statistical analyses

In order to calculate the risk indices, Excel Microsoft Office 2003 and the SPSS software (version 20) were used for mean and SD determinations and also for descriptive statistics. *Origin PRO 2015* software was used for the plot. P<00.5 was considered significant.

RESULTS

The concentration of some heavy metals in the plant and soil samples collected from the study site was presented in Table 1.

Table 1. Heavy Metals Concentration of edible portion of plants and soil (µg/g) in Sharada industrial site farmland

Sample	Lead	Chromium	Cadmium	Copper	Cobalt	Zinc	Nickel
Carrot	12.80±2.01	65.10±3.20	6.50±1.71	6.01±1.00	8.08±2.40	83.30±7.20	2.90±1.11
Lettuce	15.43 ± 1.20	57.20±4.00	4.61±1.31	1.20 ± 0.01	7.52±1.10	70.90±8.03	31.09±6.50
Onion	16.84±0.86	59.12±5.30	8.70±3.04	8.07±0.23	5.06 ± 2.00	47.22±9.20	28.70±3.07
Moringa	14.30±1.01	46.00±2.40	5.10±2.40	1.05 ± 0.04	15.10 ± 3.08	43.30±6.40	35.80±4.76
Okra	11.21±0.55	43.00±2.00	6.00±2.01	2.41±0.22	13.03±3.40	17.04±3.06	22.02±7.30
Soil	25.20±2.00	96.20±12.00	13.00±1.04	10.25±2.02	18.82 ± 0.04	98.20±0.07	35.70±3.05

Data are presented as Mean \pm SD; n=5

Heavy metals accumulated in foods and vegetables have a direct or indirect consequences or effect on human health when these vegetables are frequently consumed. Thus, levels of heavy metals in these vegetables might be a major health issues to inhabitants. The levels of metals in edible portion of plants studied are presented in Table 1. It differed among various vegetables, because of the differences in metal accumulation capacity, plant part analyzed, possibly a variation of metal concentrations in soils, pH of the soil, soil cation/anion exchange ability, soil organic matter levels, variety and types of plant species, and also, the plant stage of development or age are contributing factors [35].

The levels vary with metals type and vegetable species. Lead found to be highest accumulating metal in onion followed by lettuces with okra accumulated low concentration of the lead metal (Table 1). The levels of chromium also vary with vegetable/plant species investigated; Carrot had the highest concentration while okra showed fewer tendencies for chromium accumulation. For cadmium, the onion shows the highest concentration than other metals studied (Table 1). The level of copper was high in onion also, compared to other studied plant vegetables. Meanwhile, zinc and nickel accumulated in lettuce and *Moringa* respectively (Table 1).



Figure 1. Heavy metal Load index (HLI) of Sharada industrial site farm

USEPA risk assessment protocol state that, if the values of calculated HQ is above one (1.0) as in figure 1, there is likelihood or probability of undesirable human health risks because the metal exposure is generally high [36, 37]. From this study, the HQs found were above one (1), for Pb, Cr, Cd, Cu, and Co. This predicts that humans inhabiting *Sharada* industrial site, that depend majorly on the vegetables and fruits might be at higher risks of diseases related to high level of heavy metal pollutants in the investigated vegetables/plants. Total Hazard Quotient (THQ)>1 indicates health risk concern for humans. The Heavy metal Load Index values (HLI) value of >1

represents that the population is at risk. In the present study, the HLI for all the heavy metals studied were >1 except zinc and nickel. This showed that the population is at risk of cadmium, lead, chromium, cobalt, and copper toxicity. Evaluation the HLI/HQ for health risks associated with intake of plants cultivated in *Sharada* industrial site is based on the following assumptions:(a) the ingested concentration of heavy metal pollutants from the vegetables is equal to the absorbed concentration by an individual [25](b) Cooking/processing of the vegetables has no effect on the heavy metal pollutants levels [38].



Figure 2. Daily intake rate (DIR) of some heavy metals in vegetables/plants from Sharada Industrial farm.

Daily consumption of vegetables was estimated via a research survey to estimate the mean intake of plants/vegetables per individual per day for an adult. The fresh vegetable weight of the consumed was afterward changed to dry weight of the samples by multiplying the fresh weight with 0.085. The extent of metal toxicity to human is dependent on the daily intake [39]. This data was adopted to estimate the daily intake Rate of the toxic metals.

Daily Intake Rate of metal (DIR) as function of intake and the body weight

Result of this study shows that lead and cadmium both show DIR values >1. The health department, New York [40] suggests that, when a ratio of Estimated Daily Intake of metal to its standard Reference Dose (i.e., EDI/RfD) is equivalent to or lower than the metal oral RfD, the health risk from the metal is low or minimum. When the EDI is less than 1-5 times the standard oral reference dose of the metal, the risk from the metal exposure is low, But if its greater than 5-10 times the standard oral reference dose, the health risk perceptibly moderate, conversely, if its >10 times standard RfD, the risk is very high. HLI/HQ works with individual toxic metal; however, vegetables/plants have numerous other toxic metals. Thus, it is important to compute the metal hazard index (HI). This is the summation of all the total hazard quotients computed. The H-index, similar to HLI, must not go beyond one (1) [41, 42], if it is higher than 1, then it is a strong indication for public health risk potentials.

Since Zn, Cu and Co were not yet reported to cause any potential cancer threat because their cancer slope factor oral (CFSo) was not been documented [43] the, Total Risk values for dietary consumption of Cd, Ni, Cr and Pb only shows the cancer-causing potential effects. The cancer slope factor established by USEPA [43] is given in Table 2.

Table 2. Carcinogenic potency slope factor (CSF - oral) and standard reference dose (RFD) of metals determined.

Metals	CSF oral (mg/kg) bw/day	RFD-oral mg/kg/day
Со		3.0×10^{-4}
Ni	1.7	$2.0 imes 10^{-2}$
Zn		3.0×10 ⁻¹
Cu		$4.0 imes 10^{-1}$
Cd	$3.8 imes 10^{-1}$	$5.0 imes10^{-4}$
Cr	5×10^{-1}	$3.0 imes 10^{-3}$
Pb	$8.5 imes10^{-5}$	3.6×10^{-3}

The hazard index (HI) = \sum THQ: The HI for lettuce, Carrot, *Moringa*, Onion and Okra are 490.25, 574.11, 417.19, 447.44

and 381.86 (Pb, Cd and Cr) respectively (Table 3). This result indicates non-carcinogenic health risk, for examples more than 500 times for carrot.

Table 3. Chronic daily intake and THQs of carcinogenic metals in vegetables grown in Sharada industrial site.

Plant	Metal	Chronic Daily Intake (CDI)	THQ
Lettuce		4.5 x 10 ⁻³	1.25
Carrot		4.0 x 10 ⁻³	1.11
Moringa	Pb	4.3 x 10 ⁻³	1.19
Onion		5.2 x 10 ⁻³	1.44
Okra		3.1 x 10 ⁻³	0.86
Lettuce	Cd	6.8×10 ⁻²	136

Carrot		9.5 x 10 ⁻²	190
Moringa		6.8×10 ⁻²	136
Onion		6.8×10^{-2}	136
Okra		6.4×10 ⁻²	128
Lettuce		106×10^{-2}	353
Carrot		115×10 ⁻²	383
Moringa	Cr	84×10 ⁻²	280
Onion		93×10 ⁻²	310
Okra		76×10 ⁻²	253

Table 3. Continued.

The carcinogenic risk characterization of metals utilizes, also contaminant intakes and toxicity values (Cancer slope factor-oral; CFS-oral). According to EPA, the CFS-oral is reasonable upper bound approximate of the prospect of a body response per unit ingestion of a hazardous substance over a life span. This factor also, is important in predicting the upper bound possibility of exposed person to develop cancer disease due to a lifetime contact to an individual level of possible cancer causing chemicals such as heavy metals [44]. Cancer risk is expressed as probability of an individual developing cancer at given exposure level in life time, wherein the lifetime exposure level (ADD_{life}), according to USEPA, AT is 70 years[45].The Σ ILCR of the studied plants out weight the standard threshold boundary (> 10⁻⁴) set by USEPA. The carcinogenic metals in lettuce show that chromium is the dominant carcinogen. It has estimated to have highest cancer risk contributing about 99% of the Σ ILCR followed by cadmium (0.64%) and lead (0.42%). In Carrot, Onion, *Moringa*, and Okra cadmium has the highest cancer risk contributor with 7.60, 93, 92.31 and 89.41% respectively, followed by Cr and Pb (Table 4). Among the studied plants, the carrot (Σ ILCR 1.25) has the highest cancer risk, followed by lettuce, onion, *moringa*, and okra (Corrot>Lettuce>Onion>Moringa>Okra).

Table 4. Estimate daily intake of metals, Carcinogenic risk, and Incremental cancer Risks.

Plant	Metal	EDI	Cancer risk (CR)	%	Lifetime increment cancer risk (<u>[L/CR</u>)
	Pb	0.53	4.50E-03	0.42	
	Cr	2.11	1.06E+04	99	
	Cd	0.18	6.80E-03	0.64	
Lettuce	Cu	0.04	-		1.07
	Co	0.25	-		
	Zn	2.61	-		
	Ni	1.11	-		
	Pb	0.47	4.00E-03	0.32	
	Cr	0.25	1.15E+04	92	
	Cd	0.22	9.50E-02	7.6	
Carrot	Cu	0.29	-		1.25
	Co	2.95	-		
	Zn	0.1	-		
	Ni	-	-		
	Pb	0.61	5.20E-03	0.52	
Onion	Cr	1.86	9.30E-01	93	1.00
	Cd	0.18	6.80E-02	6.8	

	Cu	0.28	-		
	Co	1.72	-		
	Zn	1.64	-		
	Ni	-	-		
	Pb	0.51	4.30E-03	0.47	
	Cr	1.68	6.80E-02	7.47	
	Cd	0.18	8.40E-02	92.31	
Moringa	Cu	0.04	-		0.91
	Co	0.51	-		
	Zn	1.58	-		
	Ni	1.25	-		
	Pb	0.36	3.10E-03	3.65	
	Cr	1.51	8.40E-02	9.88	
	Cd	0.22	7.60E-01	89.41	
Okra	Cu	0.08	-		0.85
	Co	0.47	-		
	Zn	0.61	-		
	Ni	1.11	-		

DISCUSSION

Health risk estimation for carcinogens such as heavy metals is generally done by calculating risk indices. Those include the estimated daily intake of carcinogens (EDI), the target hazard quotient (THQ), the hazard (HI) and the human target cancer risk (TR) [25]. The predictive health risk indices not only depend on chemical carcinogens ingestion, but also the human exposure frequency and exposure period, the mean body weight of the individual and the oral reference dose of the chemical(s). Total hazard quotient, a dimensionless quantity, is defined as the ratio of metal level in the vegetable to its oral reference dose, weight by exposure frequency and duration, consumption level and body weight of an individual [46]. THQ of chemicals should generally not be more than one (1); it else therefore, indicates potential non cancer risk to exposed individual and the general population [46, 47 and 48]. THO is not a measurement of potential health risk rather a reflection of human health concern level [46, 49]. The THQ in this study revealed an alarming concern over heavy metals toxicity in the area (Table 3), showing an indices that predicted non

cancer risks concern related to the intake of vegetables studied. According to Covello and Merkhoher [50], the health risk analysis of chemical involve three stages; identification of hazard (identifying the risk chemicals, the conditions and events via which the chemical agents potentially produce adverse effects to public or their surrounding environment), the assessment of health risk (describing and quantifying it) and the evaluation of risk (which involves comparing the risk and ruling out its significance). The main purpose of these stages is to give partly important information required to support Risk management strategy (i.e., identifying, choosing and implementing appropriate control measures).

The risk assessment is a strong tool or method to evaluate the association between the peoples' health and environment. This can be quantitatively assessed (hazard degree) [51]. Previously, Muhammad et al. [10] reported the risk potentials of heavy metal in drinking water from northern Pakistan region which suggested anthropogenic and geogenic processes were majorly responsible for the drinking water contamination. This conforms to our findings anthropogenic activities as such as industrializations of Sharada, and domestics' run-off contaminated the water and subsequently used for agricultural irrigation by local farmers. Also, Cher et al. [52] found that agricultural irrigation with industrially treated waste water can potentially decrease the projected daily metal intake and target HQ by more than 85% for Cr, Pb, Cu and Zn. Thus, Sharada industrial water must be treated to forestall the health risk hazard from the heavy metals accumulated in the plants' edible parts. Rivers are important for industrial, domestic and agricultural irrigation activities. The pollution of rivers due to huge industrial discharges and run offs from irrigation farms pose an increase environmental concern [53] and other life process as observed in this study.

The non cancer risk for each metal was determined by calculating target HQ as described by Michael et al. [34] equation. It indicates that intake of Cr, Cd and Pb via consumption of plants poses substantial non carcinogenic risk while Zn and Ni show relatively safe quotients. The consumption of heavy metals polluted foods and other toxic elements were suggested to decrease significantly the concentration of iron (Fe) and Ascorbic acids(C vitamins) resulting to a deterioration of human systems' functions, decline of immunity of the body and even some disabilities related with malnutrition as reported by Liu et al. [54]. The incremental cancer risk index is the lifetime probability of a person developing cancer from exposure to carcinogenic heavy metals [55]. The standard ILCR acceptable level of cancer risk is 10^{-6} - 10^{-4} . The regulatory purpose is considered in the series (1-10) [56]. The carcinogenic risk determined from this study is greater than the standard tolerable regulatory risk (Table 4). It ranges from 10^{-3} - 10^{-2} . The incremental risk is more pronounced in carrot followed by lettuce and the other studied plants.

Snow [57] reported a main mechanism of metal induced carcinogenesis in humans as the DNA repair mechanism inhibition and the DNA-protein cross linking through Reactive Oxygen Species (ROS) production. The ROS (HO, O_2^- and H_2O_2) bring about a homeostasis imbalance between pro-oxidants and anti-oxidants bio-molecules. This leads to oxidative stress and its stress related cellular damage to bio-molecular compounds like DNA, lipids and proteins [58, 59]. Bower and colleagues reported that epigenetic and genetic effects of toxic metals which is generally related to increase risk of various types of cancer in humans [60]. The carcinogenic potentials of metals are primarily dependent on the metal oxidation state, the complex form and solubility of the metals. Also, the metal physicochemical characteristics, the soil uptake, cellular transportation, the tissue distribution and the bioavailability of metals contribute to its cancer causing property [61]. Heavy metal ions with similar essential ions properties such as molecular size and charges predicatively compete with body bio-essential ions for protein binding site which result to bio-molecular structural perturbation, lost of protein structure and functions and metal homeostatic disturbances [61]. The generation of free radical(ROS and RNS) cumulatively in the system is called oxidative stress. This induces radox imbalance in the cell and eventually lead to incidences of cancers in humans [62, 63]. The general mechanisms for metal carcinogenicity in human are: (1) Oxidative stress induction and cellular components damage especially DNA; (2)DNA repair system interference leading to instability of genome (3) cellular growth and replication interruption through signaling pathways and dysregulation of tumor suppressor genes or oncogenes [61].

The IARC (International Agency for Research on cancer) placed Cd as carcinogenic metal to human [64]. The published data from previous researches proposed that a relative low Cd exposure may lead to tubular kidney and skeletal damages [65, 66]. Thus, the carcinogenic risk from Cd exposure from this study is enormous as high levels of the metal has been recorded in both soil and vegetables, putting the consumers at risk of skeletal and kidney damages and possibly cancers over a long period of

exposure times. Chromium is a strong clastogen and produces both sister chromatid exchange and chromosomal aberrations in vivo, breaking of DNA strands, oxidized nitrogenous bases damages, DNA-protein and DNA-DNA cross linking are generally formed [57]. Cytochrome P450s, specifically human CYP1A1 is implicated in carcinogens metabolism. These compounds include heterocyclic amines and polycyclic aromatic hydrocarbons (PAHs) which are commonly distributed in our environment via automobile exhausts, industrial wastes, charcoal broiled cooking and cigarette smoke. CYP1A1 inhibits PAH induced carcinogenesis unlike other P450s such as dihydrodiol dehydrogenase and epoxide hydrolase that induce Benzo(a)pyrene and PAHs carcinogenesis. Thus, inhibition of CYP1A1 by heavy metals such as Cr can lead to accumulation of PAHs in the system [67]. This indicates the possibility of this metal to inhibit enzymes involved in xenobiotic metabolisms such as CYP1A1, causing accumulation of the carcinogens and eventually cancers.

Nickel ion (Ni²⁺) induces carcinogenesis through several and different processes or mechanisms. These mechanisms are hypermethylation of DNA (Monomethylation and dimethylation), ROS production, mutation of DNA, inhibiting histone acetylation, conversion of tumour suppressor genes to heterochromatin and considerable increase of H2A and H2B ubiquitination [68]. Thus, Ni ions play a vital role in genes silencing and gene suppression [69]. These mechanisms usually play significant role in silencing tumour suppressor genes and others that are implicated in carcinogenic pathways and thus increases the probability of developing cancers in an exposed individual. This metal is capable to break DNA strands, DNA-protein cross links, and inhibits DNA repair mechanisms. It can also form complex with certain amino acids, peptides, and proteins which can facilitate the production of ROS in vivo [70]. It is obviously believed that toxicity of this metal leads to many genetic aberrations; which is a hallmark of many human cancers.

Lead (Pb) is not an effective genotoxin in human cells, but shows co-mutagenic activity with N-methyl-N nitro Nnitrosoguanidine and Ultraviolet light possibly due to the inhibition of DNA repair system [71]. Pb poisoning can characteristically lead to irritability, headaches abdominal pain and many disorders associated with nervous system. Also acute Pb exposure is reported to leads to proximal kidney tubular damage in humans [72]. The chronic or long term exposure to Pb could give raise to renal injury and brain related disorders. Even though, the research findings implicating Pb as human carcinogen is weak, the most related Pb poison illness are gliomas, stomach and lung cancers [73].

CONCLUSIONS

The potential carcinogenic effect of consuming vegetables from Sharada was determined as the likelihood tendency or probability that a person developing cancers over lifetime period as a result of exposure to metal from predicted metal intake and cancer slope factors. The concentration of some metals in the studied samples differs generally with plant species. The levels of Pb, Cr, and Cd in plant samples were all found to be above WHO permissible limit. The possibility of a person developing cancers from dietary intake of those vegetables were found higher than the standard threshold risk limits (US-EPA,>10⁻⁴) for Cr, Pb, and Cd. The non carcinogenic risk values also predicted Cd and Pb as the dominant metal contaminants of the vegetables with the Health Risk Index greater than one (>1), while Ni, Co, and Zn showed safe quotients. It, therefore, recommend that the industrial region is unsafe for cultivating dietary vegetables because of potential heavy metals pollutions and the risk of high consumption of those vegetables which exposes the consumer population to cancer risks among others.

Safety recommendations

According to this study, the following measures were suggested as consideration should be given to the

impending risks of heavy metals: (1) Soil Alkalation (Deacidification): to avoid the transport of metals into the immediate irrigation environment and finally vegetables (2) Remediation of polluted soil: This could be carry out by the government or any other agencies concerned; (3) The local fermers should desist from planting edible plants/fruits on polluted soils or vicinity of industrial areas; and (4) Public awareness campaign: Consumers should be enlighten and select cautiously the source of their vegetables to avoid intake of local dietary vegetables contaminated with metals (5) Children and Pregnant women generally have greater risk than adult men and thus, it is important to assess the health risk implications in pregnancy and childhood.

ACKNOWLEDGEMENTS

We sincerely acknowledged and thank the Department of Biochemistry, Bayero University, Kano, for their contributions to the success of this study more especially Prof. M.K Atiku and Prof. A.J. Alhassan. Also our profound gratitude goes to all laboratory staff, technicians and those farmers who volunteered their farmland for this study.

REFERENCES

1. Zhuang P., Zou B., Li N.Y., Li Z.A., 2009. Heavy metal contamination in soils and food crops around Dabaoshan mine in Guangdong, China: Implication for human health. Environ Geochem Health.31, 707–715.

2.Sun H.F., Li Y.H., Ji Y.F., Yang L.S. Wang W.Y., Li H.R., 2010. Environmental contamination and health hazard of lead and cadmium around Chatian mercury mining deposit in western Hunan province, China. Transport of Nonferrous Metals. Soc. China. 20, 308–314.

3. Babandi A., Atiku M.K., Alhassan A.J., Ibrahim A., Shehu, D., 2012.Level Of Heavy Metals In Soil and Some Vegetables Irrigated With Industrial Waste Water Around Sharada Industrial Area, Kano, Nigeria. Chemsearch J. 3(2), 34–38. 4. Pekey H., Karaka D., Bakoglu M., 2004. Source apportionment of trace metals in surface waters of a polluted stream using multivariate statistical analyses. Marine Poll Bull. 49(9), 809-818.

5. Islam M.S., Ahmed M.K., Raknuzzaman M., Habibullah-Al-Mamun M., Islam M.K., 2015. Heavy metal pollution in surface water and sediment: A preliminary assessment of an urban riverina developing country. Ecol Ind. 48, 282–291.

6. Jarup L., 2003. Hazards of heavy metal contamination. British Med Bull. 68, 167–182

7. JavedM., UsmaniN., 2012.Toxic effects of heavy metals (Cu, Ni, Fe Co, Mn, Cr, Zn) to the hematology of *Mastacembelusarmatus* thriving in Harduaganj Reservoir, Aligarh, India. Global J Med Res. 12, 59–64.

8. Javed M., Usmani N., Ahmad I., Ahmad M., 2015. Studies on the oxidative stress and gill histopathology in *Channapunctatus* of the canal receiving heavy metal-loaded effluent of Kasimpur Thermal Power Plant. Environ Monit Assesst. 187, 41-79.

9. Powers K.M., Smith-Weller T., Franklin G.M., Longstreth W.T., Swanson P.D., Checkoway H., 2003. Parkinson's disease risks associated with dietary iron, manganese, and other nutrient intakes. Neurology. 60, 1761–1766.

10. Muhammad S., Shah MT., Khan S., 2011. Health risk assessment of heavy metals and their source apportionment in drinking water of Kohistan region, northern Pakistan. Microchemical J. 98(2), 334-343.

11. Wei X., Gao B., Wang P., Zhou H., Lu J., 2015. Pollution characteristics and health risk assessment of heavy metals in street specks of dust from different functional areas in Beijing, China. Ecotox & Environ. Saf. 112, 186–192.

12. Li N., Kang Y., Pan W., Zeng L., Zhang Q., Luo J., 2015.Concentration and transportation of heavy Metals in vegetables and risk assessment of human exposure to bioaccessible heavy metals in soil near waste-incinerator site, South China. Sci Total Environ. 521–522, 144–151.

 Ali H., Khan E., Sajad MA., 2013.Phytoremediation of heavy metals—Concepts and applications. Chemosphere. 91, 869–881.

14. Hu B., Wang C., Xu X., Zhang S., Bao S., Li Y., (2016). Assessment of radioactive materials and heavy metals in the surface soil around uranium mining area of Tongliao, China. Ecotox & Environ. Safety. 130, 185–192.

15. Akram H.S.M., Richard S., 2013. Cancer Control in Bangladesh. Japan J Clin Oncology. 43(12), 1159–1169.

16. Prakash D., Upadhyay G., Gupta C., PushpangadanP., Singh KK., 2012. Antioxidant and free radical scavenging activities of some promising wild edible fruits. Int Food Res J. 19, 1106–1116.

17. Cui Y.J., Zhu Y.G., Zhai R.H., Chen D.Y., Huang Y.Z., Qiu Y., Liang J.Z., 2004. Transfer of metals from soil to vegetables in an area near a smelter in Nanning. China. Environ Int. 30, 785–791.

18. DMello J.P., 2003. Food safety: Contaminants and Toxins. CABI Publishing, Cambridge. pp. 191-215.

19. Liu X.M., Song Q., Tang Y., Li W.L., Xu J.M., Wu J.J., 2013. Human health risk assessment of heavy metals in the soil-vegetable system: A multi-medium analysis. Sci Total Environ. 463–464,530–540.

20. Naser H.M. Shil N.C., Mahmud N.U., Rashid M.H., Hossain K.M., 2009.Lead, cadmium and nickel contents of vegetables grown in industrially polluted and non-polluted areas of Bangladesh. Bangladesh J Agric. 34(4), 545-554.

21. Bichi M.H., Anyata B.U., 1999. Industrial Waste Pollution in the Kano River Basin. Environ Mgmt Health. 10(2),112-11.

22. Chen W.Y., 2017. Environmental externalities of urban river pollution and restoration: A hedonic analysis in Guangzhou (China). Lands and Urban Planning. 157, 170–179.

23. Li Z., Mao X.Z., Li T.S., Zhang S.Y., 2016. Estimation of river pollution source using the space-time radial basis collocation method. Adv. in Water Resources.88, 68–79.

24. AOAC., 1984. *Official Methods of Analysis* 14th edn.AOAC, Inc. Washington. pp.152-162.

25.USEPA (the United States Environmental Protection Agency)., 1989. Choice of Water Regulations and Standard: a Guidance manual for assessing human health risks from chemically contaminated, fish and shellfish U.S. Environmental Protection Agency, Washington, DC; EPA-503/8-89-002.

26. Cai L.M., Xu Z.C., Qi J.Y., Feng Z.Z., Xiang T.S., 2015. Assessment of exposure to heavy metals and health risks among residents near Tonglushan mine in Hubei, China. Chemosphere.127, 127–135.

27. ChristouA., Theologides C.P., Costa C., Kalavrouziotis I.K., Varnavas S.P., 2017. Assessment of toxic heavy metals concentrations in soils and wild and cultivated plant species in Limni abandoned copper mining site, Cyprus. J Geochem Expl.178, 16–22.

28. Arora M., Kiran B., Rani S., Rani A., Kaur B. and Mittal N., 2008. Heavy metal accumulation in vegetable irrigated with water from different sources. Food Chem. 111, 811-815.

29. Sajjad K., Robina F., Shagufta S., Mohammad A., Maria S., 2009. Health Risk Assessment of Heavy Metals for Population via Consumption of Vegetables. World Appld Sci J. 6,1602-1606.

30. Rattan RK., Datta SP., Chhonkar PK., Suribabu K., Singh AK., 2005. The long-term impact of irrigation with sewage effluents on heavy metal content in soils, crops and groundwater-a case study. Agric .Ecosyst Environ. 109, 310e - 322.

31. EPA., 2010. The limits of pollutants in food. China: State Environmental Protection Administration. GB2762.

32. UNICEF., 2012. At a glance: Nigeria. https://www. unicef.org/infobycountry/nigeria_statistics.html.

33. Wongsasuluk P., ChotpantaratS., Siriwong W., Robson M., 2014. Heavy metal contamination and human health risk assessment in drinking water from shallow groundwater wells in an agricultural area in Ubon Ratchathani province, Thailand. Environ Geochem Health. 36, 169-182.

34. Micheal B., Patrick O., Vivian T., 2015. Cancer and non-cancer risks associated with heavy metal exposures from street foods: Evaluation of roasted meats in an urban setting. J Environ Poll Human Health. 3, 24–30.

35 Alloway B.J., DaviesB.E., 1971.Trace element content of soils affected by base metal mining in Wales. Geoderma. 5, 197–208.

36. U.S. Environmental Protection Agency. 2004. Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E, Supplemental Guidance for Dermal Risk Assessment); USEPA: Washington, DC, USA,2004.

37. Nkoom M., Cobbina J.S., Kumi M., 2013. Assessment of endocrine disrupting trace metals in River Samre at Samreboi in the Wassa Amenfi west district of the western region of Ghana. J Water Resources & Protection. 5(10), 983–992.

38. Forti E., SalovaraS., CetinY., Bulgheroni A., Pfaller RW., PrietoP., 2011. *In vitro* evaluation of the toxicity induced by nickel soluble and particulate forms in human airway epithelial cells. Toxicol In vitro. 25, 454–461.

39. Singh A., Sharma R.K., Agrawal M., Marshall, F.M., 2010. Risk assessment of heavy metal toxicity through contaminated vegetables from waste water irrigated area of Varanasi, India. Tropical Ecol. 51(2S), 375-387.

40. NYSDOH (New York State Department of Health)., 2007. Hopewell precision area contamination: appendix C-NYS DOH. Procedure for evaluating potential health risks for contaminants of concern. http://www.health. ny. gov/environmental /investigations /hopewell/appendc. htm

41. Islam M.S., Ahmed M.K., Al-Mamun M.H., Islam K.N., Ibrahim M., Masunaga S., 2014. Arsenic and lead in foods: a potential threat to human health in Bangladesh. Food Additives and Contaminants Part A. doi:10.1080/19440049.2014.974686.

42. Zodape G.V., 2014. Metal contamination in commercially important prawns and shrimps species collected from Kolaba market of Mumbai (west coast) India. Int J Agric Sci. 4, 160–169.

43. USEPA (the United States Environmental Protection Agency), 2012. EPA Region III Risk-Based Concentration (RBC) Table 2008 Region III, 1650 Arch Street, Philadelphia, Pennsylvania 19103.

44. EPA, 1999. Guidance for performing aggregate exposure and risk assessments.Office of Pesticide Programs, Environmental Protection Agency (EPA), Washington, DC.

45. H2M Group, 1997. Baseline human health risk assessment report: Operable unit 1. Lockheed Martin Tactical Defense Division of Lockheed Martin Tactical Systems, Inc., (Former Unisys Corp. Site) Great Neck, New York, January 1997. pp. 4-16, 38, 39.

46. Harmanescu M., Alda L.M., Bordean D.M., Gogoasa I., Gergen I., 2011. Heavy metals health risk assessment for population via consumption of vegetables grown in old mining area; a case study: Banat County, Romania. Chem Central J. 5,64.

47. Abdou H.M., Hassan M.A., 2014. Protective role of Omega-3 polyunsaturated fatty acid against lead acetate-induced toxicity in liver and kidney of female rats. Biomed Res Int. doi:10.1155/2014/435857.

48. JovicM., StankovicS., 2014. Human exposure to trace metals and possible public health risks via consumption of mussels *Mytilusgalloprovincialis* from the Adriatic coastal area. Food and Chem Toxicol. 70, 241–251.

49. Khan S., Farooq R., Shahbaz S., Khan MA., Sadique M., 2009. Health risk assessment of heavy metals for population via consumption of vegetables. World Appld Sci. J. 6, 1602–1606.

50. Covello V.T.,Merkhoher M.W., 1993. Risk AssessmentMethods: Approaches for Assessing Health and Environmental Risks.Plenum Press, USA.pp. 3-5.

51. Ma Y., Egodawatta P., McGree J., Liu A., Goonetilleke A., 2016. Human health risk assessment of heavy metals in urban stormwater. Sci Total Environ.557–558, 764–772.

52. Cher A., Achour M., Cher M., Otmani S., Morsli A., 2015. Health risk assessment of heavy metals through the consumption of vegetables irrigated with reclaimed urban wastewater in Algeria. The process of Safety & Environ Protection. 98, 245–252.

53. Obiri S., 2005. Risk assessment of toxic chemicals in mining operations in Ghana master's thesis. Kumasi, Ghana: Department of Chemistry, Kwame Nkrumah University of Science and Technology. pp.30.

54. Liu H., Probst A., Liao B., 2005. Metal contamination of soils and crops affected by the Chenzhou lead/zinc mine spill (Hunan, China). Sci & Total Environ. 339, 153–166.

55. Li S., Zhang Q., 2010. Risk assessment and seasonal variations of dissolved trace elements and heavy metals in the Upper Han River, China. J Hazardous Mat. 181, 1051-1058.

56. Li P.H., Kong S.F., GengC.M., Han B., Lu B., Sun R.F., Zhao R.J., BaiZ.P., 2013. Assessing the Hazardous Risks of Vehicle Inspection Workers' Exposure to Particulate Heavy Metals in Their Work Places. Aerosol and Air Qual Res.13, 255-265.

57. Snow E.T., 1992. Metal carcinogenesis: mechanistic implications. Pharmacol &Therap. 53, 31-65.

58. Sies H., 1997. Oxidative stress: oxidants and antioxidants. Exp Physiol. 82, 291-5.

59. Vertuani S., Angusti A., Manfredini S., 2004. The antioxidants and pro-antioxidants network: an overview. Curr Pharmacol Dis. 10, 1677-1694.

60. Bower J.J., Leonard S.S., Shi X., 2005.Conference overview: Molecular mechanisms of metal toxicity and carcinogenesis. Mol Cell Biochem. 279, 3-15.

61. Beyersmann D., HartwigA., 2008. Carcinogenic metal compounds: Recent insight into molecular and cellular mechanisms. Arch of Toxicol. 82, 493–512.

62. Poli G., Leonarduzzi G., Biasi F., Chiarpotto E., 2004. Oxidative stress and cell signalling. Curr Med Chem. 11, 1163–1182.

63. Valko M., Rhodes C.J., Moncol J., Izakovic M., Mazur M., 2006. Free radicals, metals, and antioxidants in oxidative stress-induced cancer. Chem & Bio Interact.160, 1–40. 64. IARC 1993. Cadmium and cadmium compounds. IARC Monograph: Evaluation of carcinogenic risk of humans. 58,119-237.

65. Staessen J.A., Roels H.A., Emelianov D., Kuznetsova T., Thijs L., VangronsveldJ., Fagard R., 1999.Environmental exposure to cadmium, forearm bone density, and risk of fractures: a prospective population study. Lancet. 353(9159), 1140–4.

Alfven T., Elinder C.G., Carlsson M.D., Grubb A.,
Hellstrom L., PerssonB., Pettersson C., Spang G., Schutz
A., Jarup L., 2000. Low-level cadmium exposure and osteoporosis. J Bone and Mineral Resources.15(8), 1579–86.

67. Wu J.P., Chang L.P., Yao H.T., 2008. Involvement of oxidative stress and activation of aryl hydrocarbon receptor in elevation of CYP1A1 expression and activity in lung cells and tissues by arsenic: An *in vitro* and *in vivo* study. Toxicol & Sci. 107, 385-93.

68. Ke Q., Ellen T.P., Costa M., 2008. Nickel compounds induce histone ubiquitination by inhibiting histone deubiquitinating enzyme activity. Toxicol & Appld Pharmacol. 228, 190-9.

69. Lee Y.W., Broday L., Costa M., 1998. Effects of nickel on DNA methyl-transferase activity and genomic DNA methylation levels. Mut Res.415, 213-8.

70. Kasprzak K.S., 1995. The possible role of oxidative damage in metal-induced carcinogenesis. Cancer Invest.13, 411-30.

71. HartwigA., 1994. Role of DNA repair in inhibition in lead and cadmium-induced genotoxicity: a review. Environ Health Perspectives. 102, 45-50.

72. WHO., 1995. Environmental health criteria 165: inorganic lead Internet. Geneva, Switzerland: World Health Organization; 1995 cited 2015 Nov 11. pp. 300. Available from: http:// www. Inchem . org / documents / ehc / ehc / ehc165 . htm

73. Steenland K., Boffetta P., 2000. Lead and cancer in humans: where are we now? Am J of Ind and Med. 38(3), 295-9.