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Concentration and Risk Assessment of Arsenic, Cadmium and Lead in Husked and De-husked Rice Samples from Niger and Kebbi States, Nigeria

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KEYWORDS

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ABSTRACT: Toxic metals are implicated in many health implications especially in areas of indiscriminate mining activities. A total of 100 (50 husked and 50 de-husked) rice samples were randomly collected from Kebbi and Niger States, Nigeria. Arsenic, cadmium and lead were analyzed using Inductively Coupled Plasma–Mass Spectrometry (ICP-MS). Arsenic was highest (172.58 ± 5.75) µg kg^{-1} in husked rice from Koshaba while the de-husked rice samples from Gwadan gaji had highest concentration (114.29 ± 3.97) µg kg^{-1} of arsenic. Highest concentrations of cadmium was obtained in both the husked (43.29 ± 2.06) µg kg^{-1} and de-husked (66.13 ± 1.20) µg kg^{-1} rice samples from Tondi Yauri. Husked rice samples of Dukune had highest lead concentration (53.19 ± 3.09) µg kg^{-1} while the de-husked rice samples of Yauri had highest lead concentration (60.11 ± 2.18) µg kg^{-1}. Percentage daily intake (%TDI) of arsenic was highest in most samples while contamination below detection limits was observed in few samples.

INTRODUCTION

Toxic metals like arsenic (As), cadmium (Cd), and lead (Pb) accumulate in soils and contaminate the food chain, putting ecosystem safety and human health at risk [1]. They may contaminate food crops as a result of their uptake from the soil, especially when the artisan gold miners' use of the same tools for separating gold from their ores for domestic purposes and empty the effluent into the environment. In the year 2010, more than 500 children died in Zamfara State, Nigeria, after consuming lead-contaminated meals and water. Metal poisoning affects people of all ages, but children are more vulnerable due to their low metabolic rate and insufficient establishment of the blood-brain barrier. Crops absorb and accumulate metals differently in various areas of the plant, and there are differences in uptake with translocation between species and cultivars of the same species [3]. Arsenic, Cd, and Pb are absorbed from the soil at a depth of 25 cm, which is where the roots of most cereals are found [4]. When the harmful metals adsorption in the soil is saturated, more of them are disseminated in the aqueous phase, bioavailability is

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increased and high concentrations boost plant uptake [5]. Metals will subsequently be transported from roots to shoots and finally to grains, where they may be geochemically mobile and easily absorbed by roots and translocated to aerial portions [6].

Because arsenic, cadmium, and lead are non-biodegradable, have lengthy biological half-lives, and accumulate in various regions of the human body, they are toxic.There is no good mechanism for their removal from the body, hence, solubility in water increases their toxicity at low concentrations [7]. Foods polluted with As, Cd, and Pb deplete vital nutrients in the body, resulting in decreased immunological defenses, intrauterine development retardation, altered psycho-social behavior, malnutrition-related disabilities, upper gastrointestinal cancer [8] and other health concerns [9]. Rice is an important staple diet for Nigerians, with yearly consumption of over 5.5 million tons and domestic production of roughly 1.8 million tons [10], according to government statistics. Over 3 million rice farmers in Nigeria cultivate 5 million hectares of fertile land to produce rice [11].

Arsenic is a carcinogen [12] that is easily absorbed by the gastrointestinal tract and lungs and is found in most organisms' tissues. Large amounts are found in the liver, kidneys, and skin, whereas lesser amounts are present in the bones and muscles. Arsenic's toxicity varies depending on its valence state (AsO_3 are more toxic than AsO_5). Organic arsenic compounds are removed more quickly than inorganic arsenic compounds, despite the fact that both have short half-lives, with >10 % of the organic form expelled in feces and 80 % discharged in urine in a few days [13]. Its potential to hinder DNA replication or enzyme repairs has been proposed as the mechanism of its genotoxicity [14]. Arsenic chemicals bind to mitochondrial enzymes, resulting in decreased tissue respiration and cellular damage. It reacts with the –SH group of dihydrolipoic acid, causing tissue respiration enzymes to change.

Lead is found in the environment in its elemental form only infrequently, but as ore with other elements. pH, hardness, salinity, and the presence of humic material all affect the solubility of lead compounds in water. Lead is strongly adsorbed to soil, maintained in the upper layers of soil, and does not leak into the subsoil as it sinks into the soil and sediment. Lead is non-biodegradable and accumulates in high proportions in plants and animals when it is released into the environment. Edible vegetables and fruits can absorb lead from the soil via the root system or by direct foliar uptake and transfer inside the plant. The amount of bioavailable lead to plants is determined by the soil's cation exchange capacity, pH, organic matter content, and fertilizer type [15]. A plant with a high lead concentration creates reactive oxygen species (ROS), which damage lipid membranes and, as a result, chlorophyll and photosynthetic activities, limiting overall plant growth [16]. The most common route of lead exposure in pregnant animals is through the mouth, and lead absorption increases during pregnancy [17]. Due to an inadequate BBB, lead crosses the placenta and accumulates in the embryonic brain (due to its potential to substitute calcium ions) [18]. Lead binds covalently with glutathione –SH groups and antioxidant enzymes, inactivating glutathione reductase, peroxidase, and S-transferase and lowering glutathione levels [19]. Lead inhibits antioxidant enzyme activity by replacing zinc ion (an essential cofactor in the catalytic site of several antioxidant enzymes) [20]. Its toxicity stems from its ability to substitute for other monovalent and divalent cations (Na^+, $Ca^{2+,}$ $Mg^{2+,}$ Fe^{2+}), disrupting the body's core biological activities [21]. Lead disturbs many intracellular biological activities by interfering with the regulating action of calcium on cell functioning at the molecular level. This leads to neurological impairments after replacing calcium ions and is able to permeate the BBB at a significant rate. Lead substitutes zinc in various proteins that operate as transcriptional regulators, including protamines, because it can generate ROS and cause oxidative damage to DNA.

Aspartate and alanine aminotransferase (AST and ALT respectively) activities are stimulated after ingesting lead acetate, although total soluble protein and albumin levels in plasma are dramatically reduced. The activity of cholinesterase is suppressed, whereas alkaline and acid phosphates, as well as lactate dehydrogenase, are enhanced.

Lead exposure causes encephalopathy, which results in the gradual deterioration of particular brain regions [22].

Cadmium is a by-product of zinc ore processing that is widely spread in the environment, lingering for a long time in soils and sediments. Plants gradually absorb the metal and concentrate it as it moves up the food chain, eventually reaching man at the top of the food chain. When cadmium attaches to cystein-rich proteins like metallothionein, its concentration rises by 3000 times. Hepatotoxicity is caused by the cadmium-metallothionein complex, which circulates in the kidney and accumulates in renal tissue, causing nephrotoxicity. Cadmium causes iron deficit by binding to cystein, glutamate, histidine, and aspartate ligands [23]. It has the same oxidation state as zinc and can thus substitute zinc in metallothionein, blocking it and robbing it of its free radical scavenging activity [24]. Cadmium cannot produce free radicals directly, but it can produce superoxide, hydroxyl, and nitric oxide radicals indirectly [25]. It may also replace iron and copper in cytoplasmic and membrane proteins (ferritin), resulting in the release and increase of unbound iron and copper ions. The Fenton reactions involve these free ions that produce oxidative stress [26]. Because Cd binds closely to thiol groups, the liver GSH is the first line of defense against Cd hepatotoxicity, and diethyl maleate depletion increases cadmium-induced hepatotoxicity [27]. Cd-generated alpha-(4-pyridyl-1-oxide)-N-tert-butylnitrone (POBN) radical adduct signals are produced in the bile, which is a critical component of cadmium-induced oxidative stress in the liver [28]. Cadmium's carcinogenicity is due to its propensity to hinder DNA damage repair [29]. Cadmium is carcinogenic in mammalian cells and amplifies the mutagenicity of UV radiation, alkylation, and oxidation. Base excision repair, nucleotide excision repair (NER), mismatch repair, and the removal of the premutagenic DNA precursor 8-oxodGTP are all inhibited by it. [30] Cadmium inhibits uracil (U) repair in DNA, arising from both misincorporation and cytosine (C) deamination. In light of the foregoing, this study was conducted to establish the risk associated with consuming rice polluted with arsenic, cadmium, and lead.

MATERIALS AND METHODS

Materials

Equipments

Weighing balance (Model PA214, OHAUS Corporation), distilled de-ionized water, concentrated HNO_3 (BDH England), H_2O_2 (JDH China), HF (BDH England)), hot plate, digestion flasks, filter papers, electric blender (KENWOOD China).

Rice samples

One hundred (50 de-husked d and 50 husked) rice samples were collected from the sampling zones. From each of the five locations in a zone (The zones include; Gwadangaji, Argungu, Yauri, Kalgo, Suru, Mokwa, Kwakwuti, Bida, Doko and Wushishi, all within Niger and Kebbi States). 500 g each of de-husked and husked rice samples were randomly collected from Kebbi State (latitude 12^{o}27' 57.88"N and longitude 4^{o}11' 58.28" E) and Niger state (latitude 9^{o}32' 54.86"N and longitude 4^{o}11' 51.94"E). The samples were air dried to ensure proper moisture free and each sample was kept in separately labeled polythene bags.

Methods

Sample preparation

De-husked rice

A 100g each of the de-husked rice samples were weighed and milled with electric blender to obtain a fine powder after which 10g of powdered samples were measured and stored in the refrigerator at -4°C for the metal analysis. The blender was pre-cleaned with 10 % nitric acid before each sample milling.

Husked rice

A 100 g husked rice sample was milled with prewashed mortar and pestle and powder obtained by milling with electric blender. 10 g of each sample was stored refrigerator at -4°C for metal analysis.

Digestion of the rice samples

A 0.3 g of each milled samples were weighed into crucible and predigested 5 ml HNO_3 65% supra-pure and 5 ml H_2O_2 30% in Teflon vessels by thermal heating at 85°C for eight hours. The samples were allowed to cool at room temperature 33°C, filtered and quantitatively transferred into labeled 25mL volumetric flask and made up to volume with distilled deionized water [31].

Analysis of samples by inductively coupled plasma mass spectrometry (ICP-MS)

Samples were analyzed using a Thermo Scientific XSERIES 2 Inductively Coupled-Plasma Mass Spectrometry (ICP-MS) (Thermo Fisher Scientific, Bremen, Germany) at National Center for Energy Science and Nuclear Technology (CNESTEN), Morocco. The advantages of ICP-MS over other methods include sensitivity and precision for its ability to quantify elements in any source like blood, serum, urine and water (about 40 specimens per hour), ability to measure more than one element simultaneously, simple specimen preparation, and favorable detection limits (0.01-0.1) µ L^{-1} or many elements.

Principle of (ICP-MS)

This technique couples the use of an ICP with MS for elemental analysis by generation of ions. The ICP is involved in generation of a high temperature through which the pre-treated sample is passed. The elements in the sample at such high temperature are ionized and directed further into the MS. The MS then sorts the ions according to their mass/charge ratio, directing them to an electron multiplier tube detector. This detector then identifies and quantifies each ion [32].

Procedure

A quadrupole works by setting voltages and radio frequencies to allow ions of a given mass-to-charge ratio to remain stable within the rods and pass through the detector. The spectrometer was optimized to provide minimal values of the ratios CeO^+/Ce^+ and Ba^{2+}/Ba^+ and optimum intensity of the analytes. External calibration was performed using monoelement solutions SPEX CertiPrep. Rhodium 0.01 mg L^{-1} and Iridium 0.01 mg L^{-1} were used as internal standards [33].

Estimation of Exposure Rate and Risk Assessment

Estimated daily intake (EDI) and percentage tolerable daily intake (% TDI) of arsenic, cadmium and lead in rice samples were evaluated by the method of [34] as approved by JECFA.

The formula: EDI (mg kg^{-1} bodyweight/day) = DI µg kg^{-1} bodyweight/1000 was used to estimate the daily intake. Where bodyweight is the average weight of rice consumers given as 61 kg by World Health Organization.

Statistical analysis

The data were analyzed by One-way Analysis of Variance (ANOVA) using Statistical Package for Social Science (SPSS) version 20. The results were expressed as mean ± standard deviation (SD) of triplicate analysis (n=3). The differences in means among various samples were compared by "Duncan multiple Range Test". P-value less than 0.05 were considered significant ($P < 0.05$).

RESULTS

Table 1. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in Husked Rice Samples from Gwadangaji Zone

Location	As	Cd	Pb
Gwandangaji	94.26 ± 4.04^{d}	13.07 ± 1.12^{b}	7.74 ± 0.34^{c}
Ambursa	75.77 ± 5.85^{c}	14.36 ± 0.69^{b}	3.78 ± 0.26^{a}
Zauro	38.36 ± 3.27^{b}	14.96 ± 0.86^{b}	4.56 ± 0.09^{a}
Yauri	71.21 ± 4.47^{c}	9.20 ± 1.03^{a}	4.30 ± 0.60^{a}
Sawa	5.50 ± 2.24^{a}	41.54 ± 0.60^{c}	6.19 ± 0.00^{b}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$)

Table 2. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in Husked Rice Samples from Argungu Zone

Location	As	Cd	Pb
Argungu	70.26 ± 2.15^{b}	7.82 ± 0.60^{a}	11.69 ± 0.60^{c}
Tungan dole	141.90 ± 4.04^{c}	6.36 ± 0.25^{a}	3.78 ± 0.52^{a}
Rinto	138.55 ± 5.93^{c}	8.26 ± 0.69^{b}	7.65 ± 1.03^{b}
Zaradi Sere	161.94 ± 9.12^{d}	5.93 ± 0.52^{a}	4.04 ± 1.81^{a}
Runto	61.06 ± 0.95^{a}	7.99 ± 0.43^{b}	6.45 ± 1.03^{b}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$)

Table 3. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in Husked Rice Samples from Yauri Zone.

Location	As	Cd	Pb
Yauri	122.46 ± 15.22^{c}	10.58 ± 0.86^{b}	4.04 ± 0.77^{b}
Yelwa	105.52 ± 5.33^{c}	11.26 ± 1.12^{b}	4.73 ± 0.17^{b}
Tondi	1.72 ± 0.47^{a}	43.77 ± 2.06^{d}	10.66 ± 0.69^{c}
Koko	46.44 ± 1.37^{b}	4.21 ± 0.86^{a}	15.48 ± 0.02^{d}
Zaha	133.99 ± 1.20^{c}	14.79 ± 0.95^{c}	3.01 ± 0.26^{a}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscriptsare significantly different ($p < 0.05$)

Table 4. Arsenic, Cadmium and Lead concentrations (µg kg^{-1}) in Husked Rice Samples from Kalgo Zone

Location	As	Cd	Pb
Kalgo	46.87 ± 3.87^{a}	27.60 ± 0.17^{c}	8.51 ± 0.43^{a}
Jega	113.09 ± 2.15^{c}	24.59 ± 0.77^{b}	6.62 ± 1.12^{a}
Aliero	79.72 ± 1.98^{b}	4.47 ± 0.43^{a}	12.99 ± 0.60^{c}
Kardi	85.83 ± 2.32^{b}	28.04 ± 0.69^{b}	5.25 ± 0.61^{a}
Gindi	56.76 ± 4.21^{a}	31.05 ± 2.06^{c}	8.17 ± 0.26^{b}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$)

Table 5. Arsenic, Cadmium and Lead concentrations (µg kg^{-1}) in Husked Rice Samples from Suru Zone

Location	As	Cd	Pb
Suru	69.57 ± 4.90^{b}	15.99 ± 0.95^{d}	7.57 ± 0.34^{b}
Bunza	89.27 ± 3.12^{c}	11.27 ± 0.60^{b}	<LD
Tunga dannupe	118.68 ± 2.32^{d}	19.69 ± 0.43^{d}	3.44 ± 0.34^{a}
Garadi	4.47 ± 0.61^{a}	12.04 ± 0.60^{b}	9.03 ± 0.17^{c}
Maidehini	93.31 ± 4.90^{c}	8.51 ± 0.34^{a}	8.34 ± 0.77^{c}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$); LD: Below the limit of detection.

Table 6. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in Husked Rice Samples from Mokwa Zone

Location	As	Cd	Pb
Dukun	<LD	<LD	<LD
Dukune	12.48 ± 4.60^{a}	31.15 ± 2.39^{b}	53.19 ± 3.09^{a}
Koshaba	172.58 ± 5.75^{b}	3.45 ± 0.27^{a}	50.00 ± 1.24^{a}
Raba	<LD	<LD	<LD
Gunjigi	<LD	<LD	<LD

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$); LD: Below the limit of detection.

Table 7. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in De-husked Rice from Gwadangaji Zone

Location	As	Cd	Pb
Gwadangaji	114.29 ± 3.97^{c}	2.58 ± 0.60^{b}	17.45 ± 0.77^{a}
Ambursa	63.73 ± 1.46^{b}	7.14 ± 1.29^{c}	28.72 ± 1.63^{c}
Zauro	56.07 ± 4.39^{a}	7.48 ± 0.43^{c}	29.32 ± 8.94^{c}
Yaurawa	52.63 ± 8.08^{a}	13.24 ± 0.34^{d}	23.65 ± 2.84^{b}
Sawa	103.45 ± 8.34^{c}	0.94 ± 0.09^{a}	21.41 ± 3.27^{b}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$)

Table 8. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in De-husked Rice from Argungu Zone

Location	As	Cd	Pb
Argungu	58.13 ± 5.76^{c}	7.13 ± 0.68^{a}	31.30 ± 3.01^{d}
Tungadole	43.52 ± 2.63^{b}	24.77 ± 2.80^{c}	10.66 ± 1.97^{b}
Rinto	100.53 ± 5.67^{d}	11.18 ± 1.81^{b}	11.52 ± 1.63^{b}
Zaradisere	107.67 ± 5.59^{d}	11.69 ± 1.29^{b}	7.31 ± 0.10^{a}
Runto	29.84 ± 3.78^{a}	64.58 ± 2.84^{d}	22.45 ± 0.69^{c}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$)

Table 9. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in De-husked Rice from Yauri Zone

Location	As	Cd	Pb
Yauri	36.55 ± 1.73^{b}	63.38 ± 3.01^{d}	60.11 ± 2.18^{d}
Yelwa Yauri	93.39 ± 2.83^{d}	16.08 ± 0.60^{b}	17.20 ± 1.03^{b}
Tondi Yauri	27.95 ± 3.87^{a}	66.13 ± 1.20^{d}	35.86 ± 1.63^{c}
Koko	22.87 ± 1.27^{a}	29.49 ± 1.03^{c}	12.04 ± 1.19^{a}
Zaha	49.19 ± 4.21^{c}	9.89 ± 0.69^{a}	20.29 ± 2.75^{b}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$)

Table 10. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in De-husked Rice from Kalgo Zone

Location	As	Cd	Pb
Kalgo	23.13 ± 3.01^{a}	27.09 ± 1.20^{b}	9.03 ± 0.43^{b}
Jega	83.72 ± 5.42^{d}	7.74 ± 0.86^{a}	39.22 ± 3.44^{c}
Aliero	34.14 ± 2.94^{b}	5.76 ± 1.04^{a}	<LD
Kardi	70.26 ± 4.64^{c}	8.34 ± 1.03^{b}	38.18 ± 4.30^{c}
Gindi	88.32 ± 11.78^{d}	8.26 ± 0.43^{b}	2.32 ± 0.60^{a}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$); LD: Below the limit of detection.

Table 11. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in De-husked Rice from Suru Zone

Location	As	Cd	Pb
Suru	45.49 ± 1.63^{c}	60.11 ± 1.38^{d}	32.68 ± 2.67^{c}
Bunza	37.15 ± 0.94^{b}	15.48 ± 0.52^{b}	28.12 ± 0.69^{c}
Tungadannupe	56.76 ± 3.78^{d}	10.92 ± 0.86^{a}	3.54 ± 0.85^{a}
Garadi	66.99 ± 2.83^{e}	8.68 ± 0.02^{a}	15.56 ± 0.77^{c}
Maidehini	26.23 ± 4.73^{a}	52.80 ± 1.89^{c}	9.98 ± 0.60^{b}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$)

Table 12. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in De-husked Rice from Kwakuti Zone.

Location	As	Cd	Pb
Kwakuti	22.74 ± 1.77^{a}	3.81 ± 0.44^{b}	<LD
Kafinkoro	17.26 ± 1.41^{a}	2.48 ± 0.53^{a}	8.85 ± 0.62^{a}
Jibidiga	19.20 ± 4.87^{a}	1.41 ± 0.44^{a}	12.92 ± 1.95^{b}
Paiko	63.37 ± 2.74^{b}	<LD	<LD
Kamache	28.85 ± 9.73^{a}	3.81 ± 1.06^{b}	<LD

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$); LD: Below the limit of detection.

Table 13. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in De-husked Rice from Bida Zone

Location	As	Cd	Pb
Badegi	48.94 ± 3.27^{c}	15.93 ± 2.12^{b}	<LD
Ndaloke	20.44 ± 1.89^{a}	<LD	13.72 ± 2.04^{b}
NCRI	<LD	<LD	20.53 ± 0.20^{c}
Majingari	48.59 ± 2.57^{c}	15.22 ± 0.44^{b}	2.12 ± 1.23^{a}
Mazeri	29.91 ± 3.99^{b}	2.66 ± 0.18^{a}	5.75 ± 3.19^{a}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$); NCRI: National Cereals Research Institute LD: Below the limit of detection.

Table 14. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in De-husked Rice from Doko Zone

Location	As	Cd	Pb
Doko	13.01 ± 1.63^{a}	1.95 ± 0.09^{a}	8.05 ± 0.62^{b}
Boku	11.86 ± 2.74^{a}	<LD	28.76 ± 5.58^{d}
Kupafu	9.91 ± 1.95^{a}	5.75 ± 0.89^{b}	4.98 ± 0.62^{a}
Batagi	<LD	<LD	<LD
Kpanje	16.11 ± 4.25^{a}	5.75 ± 0.97^{b}	9.82 ± 0.71^{c}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$); LD: Below the limit of detection.

Table 15. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in De-husked Rice from Mokwa Zone

Location	As	Cd	Pb
Dukun	12.74 ± 3.36^{a}	0.18 ± 0.00^{a}	2.04 ± 0.013^{a}
Dukune	14.43 ± 3.01^{a}	1.59 ± 0.89^{a}	1.24 ± 0.27^{a}
Koshaba	50.45 ± 2.83^{c}	<LD	29.03 ± 1.95^{b}
Raba	60.09 ± 5.39^{d}	2.83 ± 0.15^{a}	<LD
Gunjigi	37.79 ± 4.51^{b}	<LD	123.52 ± 4.60^{c}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$); LD: Below the limit of detection.

Table 16. Arsenic, Cadmium and Lead Concentrations (µg kg^{-1}) in De-husked Rice from Wushishi Zone

Location	As	Cd	Pb
Zungeru	<LD	6.02 ± 1.15^{c}	30.00 ± 0.35^{d}
Tungakawo	62.83 ± 3.94^{c}	0.35 ± 0.04^{a}	6.28 ± 0.44^{a}
Bankogi	9.47 ± 5.49^{a}	3.09 ± 0.89^{b}	11.95 ± 1.24^{b}
Maito	16.82 ± 3.01^{b}	2.57 ± 0.71^{b}	25.75 ± 1.24^{c}
Kanko	8.41 ± 2.21^{a}	2.74 ± 0.27^{b}	13.98 ± 1.77^{b}

Values are expressed as mean ± standard deviation (n=3). Values on the same column with different superscripts are significantly different ($p < 0.05$); LD: Below the limit of detection.

Table 17. Exposure and Risk Assessment of As, Cd and Pb in Husked Rice Samples

Zone	Metal	Average daily intake of rice (D) ($x10^{-3}$) (Kg day^{-1})	Calculated daily intake (DI) from the zone (µg kg^{-1})	Adult weight (Kg)	Estimated daily dietary intake (EDI) (µg kg^{-1} bw day^{-1})	TDI (µg kg^{-1} bw)	%TDI
Gwadangaji	As	330.50	18.85	61	0.00	15	0.00
	Cd		6157.22		0.10	7	1.43
	Pb		1754.96		0.02	25	0.08
Argungu	As	330.50	37921.50	61	0.62	15	4.13
	Cd		2402.74		0.04	7	0.57
	Pb		2220.96		0.04	25	0.16
Yauri	As	330.50	27110.92	61	0.44	15	2.93
	Cd		5592.06		0.09	7	1.29
	Pb		2505.19		0.04	25	0.16
Kalgo	As	330.50	25266.73	61	0.41	15	2.73
	Cd		7651.08		0.13	7	1.86
	Pb		2746.46		0.05	25	0.20
Suru	As	330.50	24807.33	61	0.41	15	2.73
	Cd		4461.75		0.07	7	1.00
	Pb		1877.24		0.03	25	0.12
Mokwa	As	330.50	12231.81	61	0.20	15	1.33
	Cd		2287.06		0.04	7	0.57
	Pb		6821.52		0.11	25	0.44

Table 18. Exposure and Risk Assessment of As, Cd and Pb in De-husked Rice Samples.

Zone	Metal	Average daily intake of rice (D) ($x10^{-3}$) (Kg day^{-1})	Calculated daily intake (DI) from the zone (µg kg^{-1})	Adult weight (Kg)	Estimated daily dietary intake (EDI) (µg kg^{-1} bw day^{-1})	TDI (µg kg^{-1} bw)	%TDI
Gwadangaji	As	330.50	25788.92	61	0.42	15	2.80
	Cd		2075.54		0.03	7	0.43
	Pb		7968.36		0.13	25	0.52
Argungu	As	330.50	22454.17	61	0.37	15	2.47
	Cd		7889.04		0.13	7	1.86
	Pb		5436.73		0.09	25	0.36
Yauri	As	330.50	15199.70	61	0.25	15	1.67
	Cd		12225.20		0.20	7	2.86
	Pb		9617.55		0.16	25	0.64
Kalgo	As	330.50	19800.26	61	0.32	15	2.13
	Cd		3780.92		0.06	7	0.86
	Pb		5866.38		0.10	25	0.40
Suru	As	330.50	15374.86	61	0.25	15	1.67
	Cd		9782.80		0.16	7	2.29
	Pb		5942.39		0.10	25	0.40
Kwakwuti	As	330.50	10007.54	61	0.16	15	1.07
	Cd		760.15		0.01	7	0.14
	Pb		1437.68		0.02	25	0.08
Bida	As	330.50	9776.19	61	0.16	15	1.07
	Cd		2234.18		0.04	7	0.57
	Pb		2782.81		0.05	25	0.20
Doko	As	330.50	3364.49	61	0.06	15	0.40
	Cd		889.05		0.01	7	0.14
	Pb		3410.76		0.06	25	0.24
Mokwa	As	330.50	11600.55	61	0.19	15	1.27
	Cd		304.06		0.00	7	0.00
	Pb		10301.69		0.17	25	0.68
Wushishi	As	330.50	6448.06	61	0.10	15	0.67
	Cd		974.98		0.02	7	0.29
	Pb		5813.50		0.10	25	0.40

DISCUSSION

The binding of mitochondrial enzymes by arsenic compounds results in impaired tissue respiration which may be related to cellular toxicity. Rice is one of the most efficient silica accumulators among all crops and As (III) enters through silicic acid transporters in rice. Arsenic is highest (94.26 ± 4.04) µg kg^{-1} in husked rice sample collected from Gwadangaji town in Gwadangaji zone six (Table 1). Samples collected from Koshaba in Mokwa zone had the highest (172.58 ± 5.75) µg kg^{-1} concentration of arsenic (Table 6) while samples from Tondi in Yauri zone had lowest (1.72 ± 0.47) µg kg^{-1} concentration (Table 3). Because arsenic is grown under flooded conditions (where irrigation water is often contaminated with arsenic), rice absorbs more arsenic than other food crops. More so, the

irrigation points are close to River Niger and Sunti Sugar Company that discharge the effluents directly into the water body. Arsenic based pesticides are also heavily used on crops and inorganic arsenic can persist in soil indefinitely [35]. Acute exposure to inorganic arsenic may cause nausea, vomiting, profuse diarrhea, arrhythmia, a decrease in red and white blood cell production, loss of blood volume, and encephalopathy [36]. Organic forms of arsenic however, have little acute toxicity compared to later and arsine gas [37]. Prolonged arsenic ingestion leads to its accumulation in vital organs with small amounts in the muscles, nervous system, gastrointestinal and tract spleen [38]. Arsenic toxicity inactivates enzymes in cellular energy pathways and DNA replication and repairs where arsenic is substituted for phosphate in ATP. [39] examines lung and bladder cancers as evidence of low-dose effect during the risk assessment. The husked rice samples from Dukun, Raba and Gunjigi (Table 6), NCRI (Table 13), Batagi (Table 14) and Zungeru (Table 16) had values below the ICPMS detection limit. Highest arsenic concentration (114.29 ± 3.97) µg kg^{-1} was obtained in de-husked rice samples collected from Gwadangaji in Gwadangaji zone (Table 7) and this agrees with the report of [38]. Rice has higher levels of inorganic arsenic than other foods because as the plant grows, the grains tend to absorb arsenic more readily than other food crops. Rice is a major staple food in most Nigerian homes and long-term exposure to arsenic contaminated rice can cause adverse health effects. This exposure is associated with cardiovascular diseases, diabetes and in-utero and early childhood exposure is linked to negative impacts on cognitive development and increased deaths in young adults [40]. Studies by [41] have demonstrated negative impacts of arsenic exposure on cognitive development, intelligence, and memory. Arsenic persists in rice because of non-adherence to good agricultural practices such the use of arsenic pesticides and the use of natural arsenic chelators. Paddy rice is grown in paddy fields that require high quantities of irrigation water that may contain arsenic from effluents.

Cadmium concentration increases 3,000 fold when it binds to cystein-rich metallothionein as well as glutamate, histidine and aspartate ligands leading to the deficiency of iron [23]. Highest concentration (43.77 ± 2.06) µg kg^{-1} of cadmium was detected in husked rice samples from Tondi Yauri located in Yauri Zone (Table 3) while it was below detection limit in de-husked samples of Dukune, Raba and Gunjigi in Mokwa zone (Table 6). Cadmium and zinc have the same oxidation states and hence cadmium replaces zinc in metallothionein, thereby inhibiting it, and depriving its activity as a free radical scavenger [24]. Polished rice from Thailand was reported to have significant value of $0.0166\ mg\ Cd\ kg^{-1}$ [42] while [43] reported a geometric mean content of Cd in rice across twenty-two countries to be $0.02\ mg\ Cd\ kg^{-1}$. The de-husked rice samples from Tondi Yauri in Yauri Zone (Table 9) had highest concentration (66.13 ± 1.20) µg kg^{-1} of cadmium while the samples from Dukun, Raba, Gunjigi (Table 6), Paiko (Table 12), Ndaloke and NCRI (Table 13), had cadmium levels below the detection limits. The husked rice sample from Tondi in Yauri zone with cadmium level of 43.77 ± 2.06 (Table 3) had cadmium level of 66.13 ± 1.20 (Table 9) after being de-husked. In some instances, de-husking process aided in the reduction of the metal. Low but steady concentration of cadmium may lead to inhibition of the activity of 1-hydroxycholecalciferol hydroxylase responsible converting 25-$(OH)D_3$ to 1,25-$(OH)_2D_3$ in the kidney. According to [44], prolonged intake of cadmium contaminated rice in Japan caused *itai-itai* disease in 1950s. After a chronic low-level exposure, the highest concentration found in the renal cortex causes cell death manifesting with tubular proteinuria and urinary excretion of low molecular weight serum protein (β-2-microglobin) and a concomitant increase in urinary cadmium [45]. Cd plays no role in metabolism but is a sulphur seeking agent binding with S-CH_3 and S-H groups in enzymes. The Cd^{2+} and Zn^{2+} are similar in their properties including size, where Cd^{2+} replaces Zn^{2+} in enzymes that contain the later [46]. Cd-containing enzymes do not perform the same function as Zn-containing enzymes and the presences of Cd in tissues when bound to matallothioneins, allows it to be transported to the blood by the erythrocytes or bound to large molecular weight proteins. The cystein residues of

metallothioneins play important roles in the toxicokinetics and the mechanism of Cd toxicity to the kidney [47].

Lead serves no useful purpose in human body and its presence in the body will lead to multisystem damage regardless of the route of exposure [46]. At the molecular level, the mechanisms of toxicity involve biochemical processes that include its ability to inhibit or mimic the actions of calcium affecting calcium dependent processes. Lead also interacts with proteins that have sulphydryl, amine, phosphate and carboxyl groups [48]. The husked rice collected from Dukune in Mokwa zone had highest concentration (53.19 ± 3.09) µg kg^{-1} of lead (Table 6) while no lead was detected in husked rice samples from Dukun, Raba, Gunjigi and Bunza. On the other hand, de-husked rice samples collected from Yauri in Yauri zone had highest (60.11 ± 2.18) µg kg^{-1} concentration of Pb (Table 9) with undetectable limit recorded in Badegi (Table 13), Batagi, kwakuti, Paiko, Kamache and Raba samples. Niger and Kebbi States have several illegal mining sites where the artisan gold miners are actively involved in the dredging of the earth for gold. Lead and gold appear as ore during mining and lead is discharged into the environment after the separation which may be a probable mechanism that lead gets into the soil and to the rice. The mitochondrial enzyme- δ-aminolevulinic acid synthase (δ-ALAS) catalyzes the synthesis of δ-aminolevulinic acid (δ-ALA) and porphobilinogen is produced from two δ-ALA molecules, in the presence of the cytosolic enzyme δ-aminolevulinic acid dehydratase. The ferrochelatase then catalyzes the insertion of Fe^{2+} into protoporphyrin IX to form heme [49]. δ-ALAD is a crucial enzyme in lead toxicity where its inhibition decreases heme production and increases the quantity of δ-ALA found in the blood and urine of subjects with lead exposure [50]. When δ-ALA accumulates during lead exposure, there is autoxidation resulting in the conversion of oxyhemoglobin to methemoglobin [51]. Ferrochelatase inhibition by lead allows the substitution of iron by zinc producing zinc protoporphyrin (ZPP) resulting in increased excretion in urine of coproporphyrinogen and accumulation of protoporphyrin in erythrocytes. Lead also reduces the life span of erythrocytes in the bloodstream as it binds to 98–99 % of the erythrocytes with destabilizing effect on cellular membranes. In RBC, lead causes a decrease of cell membrane fluidity and an increase of erythrocyte hemolysis rate associated with anemia [52]. Lead exposure is one factor of many that may contribute to the onset and development of hypertension [53]. It is estimated that there are about 20.8 million cases of hypertension in Nigeria among people aged 20 years in 2010, with a prevalence of 28 % in both sexes, 30.7 % among men, and 25.2 % among women [54]. Exposure to lead is generally associated with mild, moderate, and severe toxicities with occasional abdominal discomfort, lethargy, tremor, arthalgia and fatigue [55].

Diet is the main route of exposure to environmental pollutants for non-occupational exposed individuals. The highest estimated daily intake (EDI) of arsenic (0.42 µg kg^{-1} bodyweight/day) with corresponding percentage total daily intake (%TDI) (2.80 µg kg^{-1} bodyweight) in de-husked rice was recorded from samples of Gwadangaji zone (Table 18). In the same way, highest EDI of Cd (0.20 µg kg^{-1} bodyweight/day) and Pb (0.17 µg kg^{-1} bodyweight/day) in the de-husked rice were obtained from Yauri and Mokwa zones respectively.

CONCLUSIONS

The husked rice samples from all zones though, contain some levels of Pb, Cd and As but de-husking was able to reduce the levels of some metals and invariably the %TDI.

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Conflict of interest

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