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Dietary exposure and risk characterization of selected toxic metals in crude palm oil (*Elaeis guineensis Jacq*) from six states in Niger Delta, Nigeria

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Abstract

The increase in toxic metal (TM) contamination in crude palm oil (CPO) due to anthropogenic activities, method of processing and handling makes it necessary to assess the contents of metals of toxicological interest to ensure safety. The contents of TMs [lead (Pb), cadmium (Cd), chromium (Cr), nickel (Ni) and arsenic (As)] were quantified in CPO samples locally produced from six selected states in Niger Delta, Nigeria using atomic absorption spectrophotometer. The TM contents and dietary intakes were compared with permitted limits of international agencies such as the European Food Safety Authority (EFSA) and Joint FAO/WHO Expert Committee on Food Additives (JECFA). The TM contents exceeded the permissible limits of JECFA with Cr in Akwa Ibom State samples (3.08 mg kg⁻¹) being the highest. The dietary monthly intake of Cd for consumption of 25 g day⁻¹ of CPO for adults and children were 40.36 µg kg-bw⁻¹ month⁻¹ (161%) and 91.6 µg kg-bw⁻¹ month⁻¹ (366%), respectively. The margin of exposure (MOE) values of Pb due to consumption of CPO ranged from 1.39 to 3.83 (nephrotoxic effects) and 3.32 to 9.12 (cardiotoxic effects) for adults, and 0.49 to 1.34 (neurotoxic effect), 0.61 to 1.69 (nephrotoxic effects) and 0.61 to 4.01 (cardiotoxic effects) for children. Furthermore, MOE values of inorganic arsenic (iAs) for carcinogenic effects due to consumption of CPO for adults and 0.14 to 11.69, respectively. The results highlight the necessity to set up monitoring program for TMs in CPO to reduce exposure level.

Keywords: Crude Palm Oil; Toxic Metals; Dietary Intake; Lifetime Cancer Risk; Margin of Exposure; Niger Delta

1. Introduction

Vegetable oils are being widely used all over the world in cooking, cosmetic, pharmaceutical and chemical industries. The presence of essential fatty acids, phytosterols and a tocopherol, as well as high levels of antioxidative nutrients like monosaturated fatty acids, enhances the nutritional value of vegetable oils. Oils and fats have many vital functions in the human body as such as energy source, structural component and vitamins in the human body [1] as well as their cholesterol-lowering effect.

The quality of edible oils with regard to their freshness, storability/shelf life and influence on nutritional value and human health is directly linked to the concentration of some metals [2, 3]. The toxic metals (TMs) can be incorporated naturally into the oil from the soil, use of fertilizers and by unwholesome farming practices where the raw ingredients were grown for the seed-based and/ or during the production process such as in the extraction or refining processes of edible oils [4, 5].

The toxic metals can be very deleterious even at low concentration when ingested over a long period. The presence of some trace elements like Fe, Cu, Ca, Mg, Co, Ni and Mn in oil can increase the rate of oxidation by increasing the generation of free radicals from fatty acids or hydroperoxides [4], while others like Pb, Cd, Cr, Ni, and As are very important on account of their potential toxicity and metabolic roles when they have been consumed by humans [4]. It

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is therefore, of great significance to assess the levels of toxic metals in CPOs and to report possible contamination that would represent a health hazard. Food consumption had been identified as the major pathway of human exposure to toxic metals, compared with other ways of exposure such as inhalation and dermal contact.

Intake of TMs from food consumption is dependent on the TM concentrations in food and the quantity of food consumed by individuals. International agencies like US Environmental Protection Agency (USEPA) and Joint FAO/WHO Expert Committee on Food Additives (JECFA) have provided guidelines on the intake of TMs by humans. The JECFA recommended permissible tolerable weekly intakes (PTWIs) and acceptable daily intakes as guidelines for food additives and certain contaminants in foods. Also, the US EPA provided reference dose (RfDo) values in ug kg-bw⁻¹day⁻¹ for some TMs.

The presence in vegetable oil of TMs such as Pb, Cd, Cr, Ni or As is mostly due to environmental contamination. For some of these metals, limit values in palm oil have been established by Codex Alimentarius (Codex Alimentarius Standard 193-1995) and the European Commission (EC No. 1881/2006 – contaminants in foodstuffs) [6].

Toxic heavy metals (TMs) are widely present and dispersed in the environment. Their accumulation in plants is particularly important because nutritional substances might translocate from plants in the food chain and finally, they can accumulate in humans [5]. Due to the high nutritional importance of palm oil and their key role in the diet, the increased content of TMs might pose a significant health risk for their consumers. Most of the TMs entering the human body through the consumption pathway originate from the plant products grown in soil, which as the results of the geogenic or anthropogenic factors, may pose a threat of their migration to edible plants due to increased concentration or mobility [5].

Considering the importance of palm oil and their derivatives in the human diet, the potential contamination of this food group with toxic metals and the potential health risks derived from the dietary exposure, the objectives of this study were: (i) quantification of selected five toxic heavy metals (Pb, Cd, Cr, Ni, and As) in CPOs consumed by the population of the selected states in Niger Delta, Nigeria, (ii) assessment of the exposure to these of TMs from the consumption of CPOs and via estimation of the dietary intakes in comparison with health-based guidance values (tolerable intakes) provided by European Food Safety Authority (EFSA), Joint FAO/WHO Expert Committee on Food Additives (JECFA) and United State Environmental Protection Agency (USEPA), iii) the correlation, similarity and source of these metals in samples using Pearson correlation analysis, (iv) characterization of potential health risks using lifetime cancer risk (LCR) and margin of exposure (MOE). On this regard, the work was conducted with the purposes of contributing to a better understanding and to the monitoring of the adequacy and safety of the consumption of crude red palm oil of these consumers in particular and Nigerians in general.

2. Materials and Methods

2.1. Sampling locations and sampling

Samples of CPO were collected in clean sterilized and over-dried amber glass bottles directly from local palm oil mills in the six states (Akwa, Ibom, Bayelsa, Cross, River, Delta, Edo, and Rivers) that make up the states in Niger Delta, Nigeria. Six (6) samples were collected from each state making a total of 36 in all. The sampling sites in the various states are shown in Figure 1. These CPOs were produced from the states using traditional method.

2.2. Reagents (Chemicals)

All chemicals and reagents used in this study were analytical grade. Nitric acid (HNO_3) and hydrogen peroxide (H_2O_2) were purchased from Sigma Aldrich, Darmstadt, Germany. Distilled water was used throughout the work for preparation and dilutions of standard solutions.



Figure 1 Map of study area

2.3. Sample Preparation and Analytical Methods

2.3.1. Instrument Measurements

The certified standard solutions of Pb, Cd, Cr, Ni, and As (1 mg mL⁻¹) were purchased from the National Institute of Metrology, China. The working standard solutions were prepared from the standard solutions with Milli-Q water (18.2 MΩ cm resistivity) from a Millipore Milli-Q system (Thermo Scientific EASYpure II, Waltham, MA, USA). Calibration was performed by analyzing the prepared working standard solutions and two agent blank samples. Concentrations of Pb, Cd, Cr, Ni, and As were determined using an Inductively Coupled Plasma-Atomic Emission Spectrometry instrument (SPECTRO Analytical Instruments GmbH, Kleve, Germany) with axial viewing configuration. Total concentrations of As were determined using Atomic Fluorescence Spectrometry (AFS-930) (Beijing Jitian instrument Ltd., Beijing, China).

2.3.2. Sample Treatment

One gram of each homogenized sample was weighed in Teflon tubes using an analytical precision balance. Two mL of 30% hydrogen peroxide (H₂O₂) and 4 mL of 65% concentrated nitric acid (HNO₃) were added. Samples were subjected to wet digestion in acid medium with microwave digestion system. Three replicates were prepared for each sample. The microwave digestion process is based on a one-hour program divided into stages:

- Initial stage of fifteen minutes until reaching 100 °C maintained for five minutes;
- Second stage of ten minutes until reaching 180 °C, maintained for 15;
- Third stage of fifteen minutes of lowering the temperature to ambient temperature. Finally, the digested samples are placed in 10 ml volumetric flasks and made up to volume with distilled water obtained from the distillation system (Millipore, Burlington, MA, USA) [7].

2.3.3. Determination of Toxic Metals

The determination of Pb, Cd, Cr, Ni and As was performed with an atomic absorption spectrophotometer (AS-800, Perkin Elmer, USA) with a graphite chamber (HGA-800, Perkin Elmer, USA) (GF-AAS). Atomic absorption spectrophotometry (AAS) is the analytical method approved by Regulation 333/2007 (CE) modified by Regulation 836/2011 [8] for the official control of levels of lead, cadmium and mercury in food products. The determination of Hg was carried out with a cold vapour atomic absorption spectrophotometer (AS-800, Perkin Elmer, USA) (CV-AAS) with a flow injection system (FIMS-400, Perkin Elmer, USA). The instrumental wavelengths (nm) were: Cd (228.8), Pb (283.3), and Hg (253.7); the instrumental limits of quantification (LOQ) of the method were: Cd (0.013 mg/kg), Pb (0.040 mg/kg), and Hg (0.10 mg/kg). Instrumental conditions are shown in Table 2. Reference material (NIST SRM 1577

BL, Sigma Aldrich, Germany) was used for the determination of Hg. For Cd and Pb, the reference material NIST1567B Wheat four (Sigma Aldrich, Germany) was used. The recovery study, subjecting the reference material to the same conditions as the samples, yielded recovery values above 97% in all cases.

2.3.4. Quality Assurance and Control

All apparatus and glassware used through the entire laboratory analysis were sterilized. They were first washed with tap water and liquid detergent in order to keep the analyte from contamination and ensure a grease-free surface. Then, they were soaked with deionized water and followed by rinsing with 0.5 N HNO₃, and rinsed again with distilled water and a corresponding analyte solution to be analysed based on its necessity.

2.4. Methodology for Health Risk Assessment

The method for health risk assessment is based on the following:

2.4.1. Dietary Exposure Assessment

Estimated daily intake

The estimated daily intake (EDI) is defined as the amount of a chemical element/substance/contaminant that is ingested with a portion of a food or foods per body weight. The EDIs were calculated considering a 25 g day⁻¹ consumption scenario of the CPO and the detected concentration of the toxic metal (Eqn. 1). The calculation of this value makes it possible to evaluate the risk when comparing it with the reference value established by food safety institutions.

The EDI was calculated as follows:

EDI (mg kg - bw - 1 day - 1) =
$$\frac{\text{Ci} * \text{IRi}}{\text{BW}}$$
......(1)

where C_i is the concentration of THM i, (mg kg⁻¹), IR_i is the daily consumption rate (kg day⁻¹) and BW is the body weight (kg) of the consumer. Likewise, average body weights for an adult and a child of 63 kg and of 21.1 kg, respectively were used [9].

Estimated monthly intake (EMI) was calculated as per Eqn. (2):

The contribution percentage allows obtaining a percentage value over the reference value (or health-based guidance value). It was calculated as per Eqn. (3):

Contribution (%) =
$$\frac{\text{DI}}{\text{HBGV}} * 100 \dots \dots \dots (3)$$

where DI is dietary intake, which could be either daily (EDI) or monthly (EMI), and HBGV is health-based guidance value, which could be the corresponding tolerable daily intake (TDI) or monthly intake (TMI). Tolerable daily intake (TDI) is the highest quantity of a toxic metal that a person can consume each day throughout a lifetime without an appreciable health risk while TMI is the highest quantity of a toxic metal that a person can consume monthly throughout a lifetime without an appreciable health risk.

For the Cd, Cr, and Ni risk characterization, the TDI and TMI were used: Cd: $25 \ \mu g \ kg \ bw^{-1} \ month^{-1} \ [10]$; Cr: $300 \ \mu g \ kg \ bw^{-1} \ day^{-1} \ [11]$ and Ni: $13 \ \mu g \ kg \ bw^{-1} \ day^{-1} \ [12]$. Equation 3 was used to estimate the contribution percentage of the estimated intakes to the TDI and TMI.

Chronic daily intake

The chronic daily intake (CDI) was calculated as per Eqn. (4):

$$CDI (mg kg - bw - 1 day - 1) = \frac{Ci * EDI * EFi * ED}{ATc} \dots \dots \dots (4)$$

Where CDI is chronic daily dietary intake (mg kg-bw⁻¹ day⁻¹), EF is exposure frequency (day), ED is exposure duration (year) and AT_c is the average time for carcinogen (ED * LT) (day-year), LT is lifetime expectancy (year).

2.4.2. Risk characterization

The risk was characterized by two quantitative parameters namely lifetime cancer risk (LCR) [13] and margin of exposure (MOE) [14].

2.4.3. Lifetime Cancer Risk Estimates

The lifetime cancer risk (LCR) of population groups in six selected states in Niger Delta, Nigeria caused by THM dietary intake was calculated based on Eq. (5).

LCR = CDI * SF(5)

where ILCR= the incremental lifetime cancer risk of the dietary exposure (dimensionless); SF = oral cancer slope factor (Pb: 0.0085, Cd: 0.38, Cr: 0.5, Ni: 1.7, As: 1.5 kg day mg⁻¹) [13].

According to the magnitude of LCR values, the LCR level is divided into three grades, including acceptable or inconsequential (LCR < 1×10^{-6}), Cautionary ($1 \times 10^{-6} \le LCR \le 1 \times 10^{-4}$) and Unacceptable risk (LCR > 1×10^{-4}) [15].

2.4.4. Margin of Exposure

The risks to human health resulting from Pb and iAs exposure in consumed palm oil were characterized by applying the Margin of Exposure (MOE) method in line with the recommendations of the European Food Safety Authority [14]. Margin of exposure is the margin between the dose that caused a ten per cent increase in tumour frequency in animals and the dose that humans are normally exposed to. The MOE is used for assessing the risk of substances with genotoxic and carcinogenic properties. It is the ratio between the lower confidence level of benchmark dose (BMDL) (mg kg-bw⁻¹ day⁻¹) determined in experimental animals and the CDI (mg kg-bw⁻¹ day⁻¹). The benchmark dose was the lower limit of 99% confidence interval of the BMDL at the dose resulting in biological effect, or a predetermined measure for toxicity resulting in an adverse effect of 10% or 1% compared with the control group. The MOE value was calculated based on the Eq. (6):

$$MOE = \frac{BMDL}{CDI}\dots\dots\dots(6)$$

As report by the EFSA Panel, MOE values $<10^4$ are interpreted as possible concern, 10^4 - 10^5 as low concern, $>10^5$ as negligible concern as long as actions are taken to minimize further exposure, while $>10^6$ values are translated as negligible concern.

According to EFSA Panel on Contaminants in the Food Chain (CONTAM Panel) the Pb current tolerable weekly intake (TWI) of 25 μ g kg-bw⁻¹ is no longer appropriate as there is no evidence for a threshold for critical lead-induced effects and confirmed by Joint FAO/WHO Expert Committee on Food Additives (JECFA) as well. Therefore, the following benchmark dose lower confidence limits (BMDLs) for lead (Pb) [16]:

- BMDL₀₁ for children (neurotoxicity) 0.50 μg kg-bw⁻¹ day⁻¹;
- BMDL₁₀ for adults (nephrotoxicity) 0.63 μg kg-bw⁻¹ day⁻¹;
- BMDL₀₁ for adults (cardiovascular disorders) 1.50 µg kg-bw⁻¹ day⁻¹.

The EFSA Panel on Contaminants in the Food Chain (CONTAM Panel) established the lower limit of the benchmark dose (BDML₀₁) for adults (cancer incidence) with inorganic As (iAs) induced lung cancer as the end point of toxicity effect between 0.3 to 8 μ g kg-bw⁻¹ day⁻¹ for a 1% increased risk of cancer of the lung, skin and bladder, as well as skin lesions [17]. In this study, according to the safety protection principle of risk assessment the benchmark dose lower confidence limits (BMDLs) for inorganic arsenic (iAs): BMDL₀₁ for children and adults (carcinogenicity) - 0.3 μ g kg-bw⁻¹ day⁻¹

2.5. Statistical Analysis

For every sample, each parameter was measured three times, and the average values for every state were given as mean ± SD. Multiple comparisons were performed using Kruskall-Wallis ANOVAs. The degree of linear dependence between the various parameters assayed was evaluated using the Beauvais-Pearson test.

3. Results and Discussion

3.1. Content of Toxic Metals in Crude Palm oil

The quantification of TM content in edible palm oil is vital, since several TMs play major roles in various metabolic processes in the human body. Moreover, these TMs are toxic if consumed in excessive quantities. Table 1 shows the mean average concentration of toxic metals (Pb, Cd, Cr, Ni, As) in the CPOs samples collected from local producers in six selected states of states in Niger Delta, Nigeria. The mean concentrations of Pb, Cd, Cr, Ni and As were 0.823, 0.346, 1.746, 0.344 and 0.0640 mg kg⁻¹, respectively. All the values, except As, exceeded permissible limits of international bodies such European Food Safety Authority (EFSA) and Joint FAO/WHO Expert Committee on Food Additives (JECFA). In general, the toxic metals analyzed follow the descending order of Cr > Pb > Cd = Ni > As.

The contents of Pb in the samples were found to be in the range of 0.340 to1.50 mg kg⁻¹, with an average value of 0.823 mg kg⁻¹. The Pb contents obtained in CPO from the study area were significantly higher than the maximum tolerable limit set by European community and Joint FAO/WHO Expert Committee on Food Additives (JECFA) in vegetable oils (0.1 mg kg⁻¹) [18, 19].

Table 1 Mean concentration (mg kg⁻¹) of toxic metals in the crude palm oil samples from six states, six selected states in Niger Delta, Nigeria

ТМ	mean	SD	min	max	Maximum Levels		
					EC [19]	FAO/WHO [20]	
Pb	0.823	0.422	0.340	1.500	0.1	0.1	
Cd	0.346	0.969	0.001	3.390	0.05	0.5	
Cr	1.746	0.788	0.820	3.080			
Ni	0.344	0.250	0.020	0.690	-	0.2	
As	0.064	0.077	0.010	0.283	0.1	0.1	

The contents of Pb in the samples were found to be in the range of 0.340 to1.50 mg kg⁻¹, with an average value of 0.823 mg kg⁻¹. The Pb contents obtained in CPO from the study area were significantly higher than the maximum tolerable limit set by European community and Joint FAO/WHO Expert Committee on Food Additives (JECFA) in vegetable oils (0.1 mg kg⁻¹) [18, 19]. Lead is a cumulative element that has no biological role and have carcinogenic effects. Acute Pb exposure can induce appetite loss, fatigue, headaches, stomach discomfort, hypertension, and insomnia [21, 22] while chronic exposure affects multiple body systems, including the gastrointestinal, neurological, cardiovascular, haematological and renal systems [1, 23]. It causes sterility, neonatal mortality, morbidity, sub-cellular changes, increased systolic blood pressure and cardiovascular diseases in adults as well as neuro-developmental disorders (reduces intellectual performance and cognitive development) in children [24, 25]. Furthermore, Pb displaces Ca in bone tissues leading to bone formation mechanisms, alteration of compositional properties, and disruption of mineralization [1].

The concentrations of Cd in the samples were found to be in the range of ND (not detected) -3.390 mg kg⁻¹, with an average value of 0.728 mg kg⁻¹. All the investigated samples showed lower Cd levels, except at Edo State, than the recommended maximum permissible limits of 0.5 mg kg⁻¹ [18]. Cadmium is a nonessential toxic element that has no biological role and classified as a human carcinogen. Cadmium absorbed through intake is known to bio-accumulate in the liver and kidney and the human body takes from 10 to 30 years to excrete this element [26]. The resultant health effects are reproductive deficiencies, kidney dysfunction, bone demineralization, skeletal damage, prostate proliferative changes as well as cancer [27- 30].

Chromium, an essential element of biological interest, can exist in two oxidation states (Cr⁺³ and Cr⁺⁶). Cr⁺³ acts as a critical cofactor in insulin action and plays an important role in the metabolism of lipid, glucose, and protein. Cr⁺⁶ is carcinogenic according to the International Agency for Research on Cancer [31] and acute Cr poisoning can be mutagenic and carcinogenic [24, 32] and the toxic effects of Cr⁺³ intake include skin rash, nose irritation, bleeding, upset stomach, kidney and liver damage. The highest and lowest contents of Cr were observed in samples from Rivers and Delta, respectively. Cr concentrations varied from 0.820 to 2.780 mg kg⁻¹ with an average value of 1.515 mg kg⁻¹.

The levels of Ni in the oil samples from the studied areas ranged from 0.020-0.690 mg kg⁻¹ with a mean value of 0.344 mg kg⁻¹. Except in Cross River State, the levels were higher than the maximum permissible limit (0.2 mg kg⁻¹) set by World Health Organisation [20]. Trace amounts of Ni may be beneficial as an activator of some enzyme systems, influences iron absorption and metabolism and may be an essential component of the hemopoietin process [33]. However, Ni exposure is associated with adverse health effects, such as dermatitis, vomiting and nausea, cyanosis, gastrointestinal discomfort, pulmonary fibrosis, cardiovascular and kidney diseases, increased lung and nose cancer risk, and even death [34, 35].

The As content in the samples were in the range of 0.000 to 0.10 mg kg⁻¹ with an average value of 0.058 mg kg⁻¹. The obtained values fell far below the action level recommended by FAO/WHO of 0.5 mg kg⁻¹ [20]. Arsenic (As) is a non-essential element. It occurs in several forms, of which inorganic arsenic is the most toxic form. Inorganic As (iAs, i.e., sum of As⁺³ and As⁺⁴) form is carcinogenic. Chronic As exposure due to its metabolism process might damage each human body organ; including respiratory, cardiovascular, neurological, gastrointestinal, hematological, renal, hepatic, developmental, and reproductive disorders and carcinogenic, mutagenic, and immunological effects such as hepatocarcinoma as well as cancers of the skin, skin lesions (in the form of hyperkeratosis, hyperpigmentation or hypopigmentation) [36, 37].

Some researchers in Nigeria have conducted several studies that showed metals (Pb, Cd, Cr, Ni, and As) in palm oil produced and sold in different parts of Nigeria. Table 2 shows the mean concentrations of heavy metals of this study compared with other studies in Nigeria. Some of the detected metal contents in these studies were reported below the permissible limits [38-43].

Pb	Cd	Cr	Ni	As	References
0.823	0.346	1.746	0.344	0.064	This study
0.032	0.050	0.025	0.052	0.002	[38]
0.178	0.022	-	-	-	[39]
0.039	0.645	0.178	0.26	-	[40]
-	-	-	0.38	-	[41]
<0.001	0.154	0.04	-	0.163	[42]
0.001	0.006	-	0.001	-	[43]

Table 2 Mean concentration of toxic metals in crude red palm oil and other studies in Nigeria

3.2. Pearson Correlation coefficient

Table 3 The Pearson correlation coefficients of toxic metals in palm oil

	Pb	Cd	Cr	Ni	As			
Pb	1							
Cd	0.501 ^a	1						
Cr	0.734 ^a	0.205	1					
Ni	0.756 ^a	0.465 ^b	0.672 ^a	1				
As	0.352	-0.109	0.393	0.394	1			
^a Correlatio	^a Correlation is significant at the 0.01 level (2-tailed); ^b Correlation is significant at the 0.05 level (2-tailed)							

Correlations support the interpretation of meaningful measurements. This approach also helps to identify common factors, inducing the observation of metal relationships.

A high value of the coefficient reveals a high linear correlation between the contents of two metals. Table 3 shows the Pearson correlation coefficient (r) between the metals in oil samples. Pearson correlation coefficients showed moderate positive correlations between Pb and Cr (0.734), Pb and Ni (0.756), and Cr and Ni (0.672). Overall, the correlation

behaviour of metals in the studied samples remained noticeably diverse, which may be attributed to the disproportions of the origin of samples.

3.3. Exposure assessment of toxic metals via consumption of CPOs

In order to evaluate dietary-related daily or monthly contaminant exposure in humans, the computed EDIs and EMIs were compared with their corresponding PTDIs and PTMIs, respectively.

Table 4 summarizes the dietary intake (EDI: µg kg-bw⁻¹ day⁻¹; and EMI: µg kg-bw⁻¹ month⁻¹) values via consumption of 25 g day⁻¹ of CPO in children and adults. It also shows the percentage of contribution to the provisional tolerable daily and monthly intakes (PTDIs/PTMIs) for both children and adults.

The EMI values of Cd for adults and children for EDS were 36.32 and 27.57 μ g kg-bw⁻¹ month⁻¹ respectively, and 145.3 and 110.3%, respectively above the PTMI values. These will cause major health hazard to both adult and children's consumers from that area. The EDI values for other metals (Cr and Ni) for adults and children were considerably lower than their corresponding PTDIs, and represent contributions to PTDI_{Cr} and PTDI_{Ni} of 0.10 to 0.37% and 0.05 to 1.87% for adults and 0.07 to 0.28% and 0.04 to 1.2% for children, respectively and pose no health risk for both age-groups of consumers.

Table 4 Estimated dietary in	ntakes of toxic metals (Cd,	Cr, and Ni) in crude	palm oil compared with	۱ the tolerable intakes

ТМ	Parameter	AKS	BAS	CRS	DES	EDS	RVS	
For adu	lts of body weight of 63 kg	5						
Cd	EMI (µg kg-bw ⁻¹ month ⁻¹)	0.60	0.24	0.18	0.48	40.36	1.01	
	%TMI	2.38	0.95	0.74	1.90	161.43	4.05	
Cr (III)	EDI (mg kg-bw ⁻¹ day ⁻¹)	0.00	0.00	0.00	0.00	0.00	0.00	
	%TDI	0.34	0.26	0.15	0.11	0.20	0.33	
Ni	EDI (µg kg-bw-1 day-1)	0.21	0.15	0.04	0.12	0.16	0.14	
	%TDI	1.63	1.18	0.27	0.93	1.22	1.07	
For chil	dren of body weight of 21.	ght of 21.1 kg						
Cd	EMI (µg kg-bw-1 month-1)	1.35	0.54	0.42	1.08	91.58	2.30	
	%TMI	5.40	2.16	1.67	4.32	366.31	9.18	
Cr (III)	EDI (mg kg-bw ⁻¹ day ⁻¹)	2.29	1.76	1.01	0.74	1.36	2.26	
	%TDI	0.76	0.59	0.34	0.25	0.45	0.75	
Ni	EDI (µg kg-bw-1 day-1)	0.48	0.35	0.08	0.27	0.36	0.32	
	%TDI	3.71	2.67	0.62	2.11	2.77	2.42	

EDI: estimated daily intake; EMI: estimated monthly intake; TDI: tolerable daily intake; TMI: tolerable monthly intake.

3.4. Risk Characterization

3.4.1. Cancer risk

Table 5 is a summary of the cancer risk (CR) and the (Σ CR) values for exposure to TMs due to consumption of 25 g day⁻¹ of CPO. The CR for Pb, Cd, Cr, Ni and As for children and adults ranged from were 1.94 x 10⁻⁷; 1.56 x 10⁻⁶; 7.86 x 10⁻⁶; 2.77 x 10⁻⁵ and 2.26 x 10⁻⁵, and 2.26 x 10⁻⁷; 1.82 x 10⁻⁶; 9.16 x 10⁻⁶; 3.23 x 10⁻⁵ and 2.63 x 10⁻⁵, respectively. The lifetime cancer risk (LCR) values of Pb and Cd, except at EDS, for the population across the states were insignificant, 1.79 x 10⁻⁵ - 7.26 x 10⁻⁵ below 1 x 10⁻⁶. In comparison, the cancer risk (CR and LCR) values of Cr, and Ni for all the states were within the "cautionary risk" range of 1.0 x 10⁻⁶ - 1.0 x 10⁻⁴, except As (1.88 x 10⁻⁴) for consumers from Akwa Ibom State. The LCR_{iAs} for CPOs from Akwa Ibom and Delta states are above 1 x 10⁻⁴ while the total lifetime cancer risk (except Cross River State) are above the threshold of 1 x 10⁻⁴. The LCR values were in the following descending orders: AKS > DES > EDS = RVS > BAS > CRS. Palm oil samples from Akwa Ibom State pose the highest potential LCR while those from Cross River State pose the less LCR to consumers (figure 2).

ТМ	AKS	BAS	CRS	DES	EDS	RVS				
Adult	Adults									
Pb	3.13E-07	2.49E-07	2.11E-07	1.14E-07	2.49E-07	2.21E-07				
Cd	2.62E-07	1.05E-07	8.13E-08	2.10E-07	1.78E-05	4.46E-07				
Cr	1.33E-05	1.03E-05	5.90E-06	4.33E-06	7.95E-06	1.32E-05				
Ni	5.02E-05	3.62E-05	8.45E-06	2.86E-05	3.76E-05	3.29E-05				
As	6.69E-05	1.18E-05	6.21E-06	3.94E-05	1.24E-05	1.45E-05				
∑CR	1.31E-04	5.86E-05	2.09E-05	7.26E-05	6.09E-05	6.12E-05				
Child	ren									
Pb	2.68E-07	2.13E-07	1.81E-07	9.77E-08	2.13E-07	1.90E-07				
Cd	2.25E-07	9.00E-08	6.97E-08	1.80E-07	1.53E-05	3.82E-07				
Cr	1.14E-05	8.80E-06	5.06E-06	3.71E-06	6.82E-06	1.13E-05				
Ni	4.31E-05	3.10E-05	7.25E-06	2.46E-05	3.22E-05	2.82E-05				
As	5.74E-05	1.01E-05	5.33E-06	3.37E-05	1.07E-05	1.24E-05				
∑CR	1.12E-04	5.02E-05	1.79E-05	6.23E-05	5.22E-05	5.25E-05				

Table 5 Cancer (CR) and total cancer (Σ CR) risk of toxic metals due to consumption of 25 g day⁻¹ of CPO samples from six selected states in Niger Delta, Nigeria

AKS - Akwa Ibom State, BAS- Bayelsa State, CRS - Cross River State, DES - Delta State, EDS - Edo State, RVS - River State



Figure 2 Lifetime cancer risk (LCR) and total lifetime cancer risk (∑LCR) due to consumption of 25 g day⁻¹ of palm oil samples from six selected states in Niger Delta, Nigeria

3.5. Margin of exposure of Pb and iAs

The MOE values of Pb for adults and for children from consumption of CPO (25 g day⁻¹) were 1.46 to 6.43 (nephrotoxic effects) and 3.47 to 15.32 (cardiotoxic effects), and 1.41 to 6.21 (neurotoxic effect), 1.77 to 7.83 (nephrotoxic effects) and 4.22 to 18.63 (cardiotoxic effects), respectively. Likewise, the MOE values of iAs from consumption of palm oil (25

g day⁻¹) for adults and children ranged between 0.00 to 104.16 (carcinogenic effects) and 0.00 to 126.70 (carcinogenic effects), respectively. An MOE value of 10,000 or higher is considered of low concern from a public health view point with respect to carcinogenic effect. Hence, a small MOE represents a higher risk than a larger MOE. Considering the BMDL of 0.63 μ g kg-bw⁻¹ day⁻¹ (nephrotoxic effects) and 1.5 μ g kg-bw⁻¹ day⁻¹ (cardiotoxic effects), the MOE values obtained indicate potential nephrotoxicity and cardiotoxicity due to Pb and carcinogenicity due to exposure to iAs for adults, and potential neurotoxicity, nephrotoxicity and cardiotoxicity (Pb) and carcinogenicity (iAs) for children.

Table 6 Margin of Exposure (MOE) of Pb and iAs due to daily consumption of 25 g of palm oil from six selected statesin Niger Delta, Nigeria

Sample states	Pb				iAs		
	CDI	MOE	MOE	MOE	CDI	MOE	
		Neurotoxicity	Nephrotoxicity	Cardiotoxicity		Carcinogenicity	
For adult of body weight of 63 kg							
AKS	0.452		1.393	3.316	0.064	4.681	
BAS	0.359		1.754	4.177	0.011	26.526	
CRS	0.306		2.062	4.909	0.275	1.092	
DES	0.165		3.826	9.108	0.931	0.322	
EDS	0.359		1.754	4.177	0.012	25.200	
RVS	0.319		1.972	4.696	0.014	21.600	
For children of	body w	eight of 21.1 kg					
AKS	1.027	0.487	0.614	1.461	0.145	2.063	
BAS	0.815	0.614	0.773	1.841	0.026	11.690	
CRS	0.693	0.721	0.909	2.163	0.014	22.211	
DES	0.374	1.338	1.686	4.014	0.086	3.507	
EDS	0.815	0.614	0.773	1.841	0.027	11.105	
RVS	0.725	0.690	0.869	2.069	0.03	9.519	

AKS - Akwa Ibom State, BAS- Bayelsa State, CRS - Cross River State, DES - Delta State, EDS - Edo State, RVS - River State

4. Conclusion

The results obtained from the samples of edible crude oil palm oil from six selected states of the Niger Delta, Nigeria exceed the maximum permissible limits for toxic metal contents established by the World Health Organization and European Union.

The Pb exposure from consumption of crude palm oil could have toxic effects such as nephrotoxicity and cardiotoxicity in both adults and children as well as neurotoxicity in children. Also, consumption of crude palm oil could have carcinogenicity due to exposure to iAs for both adults and children. The calculated total cancer risk (Σ CR) (for both children and adult consumers for both age groups from Akwa Ibom State) and total lifetime cancer (Σ LCR) values from exposure to Pb, Cd, Cr, Ni, and iAs exceeded the benchmark (10⁻⁵), which illustrated potential carcinogenesis among consumers. It can be concluded that the crude palm oil samples were not free of any chemical element contamination. The results highlight the need to set up educational for the artisanal or local producers as well as monitoring and surveillance programs for the safety of consumers of palm oil in these selected states in Niger Delta, in particular and Nigeria in general.

Compliance with ethical standards

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Disclosure of conflict of interest

The Authors declare that there is no conflict of interest

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