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Health Risks Associated with Lead Exposure in Frequently Consumed Foods

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Abstract

This study determined the exposures and risks associated with the ingestion of lead in the diets of three age groups: 5-19; 20-39 and ≥ 40 living in Kumasi. Frequently consumed foods were sampled from the study area and their lead concentration quantified using the graphite furnace AAS method. The @Risk software was used to fit distributions for all elements of exposure. Subsequently, the chronic daily intake (CDI) was determined, and then used to estimate the risks in terms of the margin of exposure (MoE) and incremental lifetime cancer risk (ILTCR). Across the three age groups, the modal CDI ranged between 0.007 and 0.06 µg/kg bw-day. Significantly low modal MoEs (0.009-0.05) were recorded for developmental neurotoxicity, nephrotoxicity and cardiovascular toxicity. De minimis ($<10^{-6}$) modal lifetime cancer risks were recorded, however, the 95th percentile risks show that some consumers are still at risk ($>10^{-6}$). These findings suggest serious public health concerns.

Keywords: Lead exposure; Margin of exposure; Probabilistic risk; Frequently consumed foods

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List of Abbreviations

AT: Averaging Time; BMDL: Benchmark Dose Lower Bound; BW: Body Weight; C: Concentration of Pb; CDI: Chronic Daily Intake; ED: Exposure Duration; EDI: Estimated Daily Intake; EF: Exposure Frequency; GFAAS: Graphite Furnace Atomic Absorption Spectroscopy; ILTCR: Incremental Lifetime Cancer Risk; MF: Mass of Food Consumed; MoE: Margin of Exposure; PF: Potency Factor

Introduction

Lead (Pb) exposure is ranked as the 26th highest risk factor to the disease burden in Western Sub-Saharan Africa [1]. Globally, exposure to Pb is estimated to have caused 1,050,000 deaths in 2017 alone [2]. The majority of Pb exposure in humans occurs through food consumption [3]. Though Pb is not added to food intentionally, it remains a serious contaminant of food, either through deposition from the air or uptake from soil and leaded-agrochemicals during growth [4]. The use of Pb-contaminated water in food processing and possible leaching into food from food contact materials have also been suggested as another exposure pathway [5]. Though Pb has been determined in several foods in Ghana [6-8], risk assessment is hardly monitored by regulatory authorities. There seems to be weak national intervention programmes for the management and or communication of the risk of Pb. This raises a public health concern since Pb has many adverse health effects.

In characterizing the risk of adverse health effects posed by Pb, health-based guidance thresholds provided by regulatory authorities are matched to prevailing Pb exposure levels. Two approaches that have been used are; the methods of margin of exposure (MoE) and incremental lifetime cancer risk (ILTCR). The MoE approach makes use of a benchmark dose, which is the threshold above which a specified level of adverse response occurs [9,10]. Calculated MoE values are then matched with the recommended thresholds to determine whether risk has occurred or not. The European Food Safety Authority (EFSA) has defined MoE values greater than or equal to 10 as of no appreciable risk [11]. On the other hand, MoE values between 1 and 9 imply a low risk whereas values below 1 imply a significant risk. The incremental lifetime cancer risk (ILTCR) is determined as

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a product of the exposure and the potency factor (slope factor) of Pb [12]. Risk estimates lower than 1×10^{-6} (1 out of 1 million persons) are regarded as acceptable; whereas estimates greater than this de minimis imply significant risk [12].

The toxicity of Pb has been attributed to its ability to bind to several important biomolecules. Perhaps, the most known disease endpoint of Pb is its neurotoxicity. Studies have shown that the mechanism underlying Pb neurotoxicity involves a non-competitive inhibition of the N-methyl-D-aspartate receptor [13]. This receptor is critical for memory and learning processes, thus its inhibition results in reduced cognition functions. The neurotoxic effects of Pb include decreased perception of sound and sight, antisocial tendencies, reduced attention span and a decreased learning ability shown as reduced intelligence quotient (IQ) scores [14,15]. Again, Pb causes damage to glomerulus leading to a reduction in the glomerular filtration rate and ultimately chronic kidney disease [3]. Toxicity to kidney usually occurs at relatively high levels of blood(B)-Pb (>60 µg/dL) [3] though other studies suggest adverse effect at 10 µg/dL [16]. It has also been reported that a 15.5 µg/g increase in bone (tibia) Pb content, is said to be associated with a 19% increase in the risk of hypertension [17]. The mechanism of Pb-induced hypertension has been described as involving impairment of the nitric oxide pathway, leading to a downregulation of soluble guanylate cyclase (nitric oxide receptor) [18]. This leads to increased total peripheral resistance, increased arterial pressure and subsequently, hypertension.

The International Agency for Research on Cancer (IARC) has classified inorganic Pb as "probably carcinogenic" to humans (Group 2A) [19]. This was based on "sufficient evidence of carcinogenicity" in animals, but "inadequate evidence of carcinogenicity" in humans. However, organic Pb is "not classifiable to carcinogenicity" (Group 3**)** because of "inadequate evidence of carcinogenicity" in humans as well as animals [19]. The mechanism of Pb carcinogenesis involves the ability of Pb to inhibit DNA repair and induce oxidative stress in cells. This in turn adversely impacts the tumor suppressor proteins [20]. Increased lung cancer deaths following Pb exposure has been reported [21]. On the contrary, other studies suggest that there could be other unknown factors involved [22,23]. It appears that the status of Pb as a carcinogen is inconclusive because of the absence of clear biomarkers of Pb exposure [24].

Exposure assessment to Pb is largely based on the concentration of Pb in foods and the frequency of consumption of these foods. Though several approaches for exposure assessment exist, it is necessary that these approaches are able to accurately estimate levels of exposure. The total diet study (TDS) approach, which involves sampling of frequently consumed foods, homogenizing and analyzing them for toxic chemicals is commonly used [25]. This approach provides the most accurate estimate of the concentration of Pb and other chemicals ingested through food [26]. Assessment of exposure to Pb has also been achieved using biomarkers such as bone-Pb, blood-Pb and δ-aminolevulinic acid (δ-ALA). Exposure to Pb results in the inhibition of δ-aminolevulinic acid dehydratase (δ-ALAD) activity and a consequent increase in δ-ALA excretion [27]. Bioaccumulated bone-Pb, as measured by non-invasive x-ray fluorescence, has also been used as a biomarker of long-term exposure since Pb is deposited in the bone [28]. Therefore, the use of biomarkers in exposure assessment allows for the determination of the total body burden of Pb since the route of contamination is of no interest.

The presence of Pb in foods and its exposure is not a contemporary issue, because many studies have reported Pb in foods [6-8]. However, public health concerns still persist since Pb has many adverse health effects. The problem is that though tools exist for the determination of the burden of risk of contamination in foods, there is paucity of information relating to Pb intake risks in many communities. It is therefore necessary to evaluate the extent of risk periodically in order to inform food safety policies. This study therefore sought to determine the exposure of Pb through the consumption of 'frequently consumed foods' and also determine the associated risks.

Materials and Methods

Materials

All reagents used in the study were of analytical grade. HNO_3 was obtained from Surechem Products (England). H_2O_2 was obtained from BDH Chemical Ltd. (UK). Pb standard was obtained from Merck (Darmstadt, Germany).

Study area

The area for this study was the Kumasi metropolis. Eleven locations in the study area; Asafo, Atonsu, Bantama, Kaase, Kotei, Kronom, Santaase, Suame, Tafo, Tanoso and Tech Junction, were selected as sampling sites.

Sampling of foods and sample preparation

The most frequently consumed foods in the Kumasi metropolis have been determined in a total diet study to be rice, *banku, fufu and kenkey* [29]. These foods together with their accompaniments were randomly sampled from selected locations in the study area for a period of 1 week. Sampled foods were homogenized using a Crompton blender (Taura TD71, India), packaged into Ziploc bags and stored at -16°C pending further analyses.

Digestion of foods

The wet acid method was used to digest a mass of 0.5 g of the homogenized food sample weighed into a digestion tube [30]. A volume of 3 mL HNO_3 (65%) and 1 mL H_2O_2 (30%) was then added to the sample in the digestion tube and heated in a Tecator Digester System 20 (1015, US) at 120°C for 3 h. The digestate was then transferred into a 20 mL volumetric flask and topped to the mark using deionized water.

Instrumentation

Measurements were performed using an Analytik Jena GF AAS (novAA® 400P, Germany) equipped with a transversely heated graphite atomizer. A hollow cathode lamp operating at a wavelength of 283.3 nm, current of 10 mA and a slit width

of 0.7 nm was employed. A deuterium (D2) lamp was used to achieve background correction. Argon was used as the carrier gas at a flow rate of 20 mL/min and an integration of 3 s used for measurements. Pb standard solutions of concentrations 5, 10, 20, 30 and 50 µg/L were used to calibrate the GF AAS system before the analysis. A linear calibration curve (r^2=0.998) was achieved. Limit of detection and limit of quantification were established as 0.05 µg/L and 0.10 µg/L respectively.

Quality assurance

All glassware used in the study were soaked (3 h) in 20% HNO_3 followed by thorough rinsing with deionized water. Five food samples were spiked (1 mg/L Pb standard) and digested following the same procedure for unspiked samples. A mean recovery of 90% was obtained, indicating accuracy of the method employed.

Data analysis

Food consumption data used in this study was obtained from a previous study [29], where the elements of consumption; mass of food consumed per day (MF), exposure frequency (EF), exposure duration (ED) and body weight (BW) were taken. Distributions were then fitted for these elements as well as the Pb concentration (C) using the Palisade @Risk software. In estimating CDI, averaging times (AT) of 70 years and 30 years were used for carcinogenic and non-carcinogenic risks respectively [12].

$$CDI = \frac{C \times MF \times EF \times ED}{BW \times AT} \quad (1)$$

The MoE approach, which was employed to characterize non–cancer risks was based on Equation 2 [31]. The BMDL for the various disease endpoints used in the determinations of the MoEs were; $BMDL_{01}$ of 0.50 µg/kg bw-day for developmental neurotoxicity; $BMDL_{01}$ of 1.50 µg/kg bw-day for effects of systolic blood pressure (cardiovascular toxicity) and a $BMDL_{10}$ of 0.63 µg/kg bw-day for chronic kidney disease (nephrotoxicity) [11].

$$MoE = \frac{BMDL}{CDI} \quad (2)$$

The cancer risk (ILTCR) was also determined using Equation 3 [12] based on lead's oral potency factor (PF) value of 0.0085 (mg/kg bw-day)$^{-1}$ [32].

$$ILTCR = CDI \times PF \quad (3)$$

All calculations were performed at 100,000 iterations in a Monte Carlo simulation using the Palisade @Risk software.

Results and Discussion

Concentration of lead

Presented in **Table 1** are the concentrations of Pb found in the samples collected from the study area of which detectable levels of Pb was found in 53% of sampled rice. Relative to what was obtained in this study, other studies in Iran had reported higher Pb content of 10.3 µg/g in cooked rice dishes [33]. In fact, a rather lower value of 0.03 µg/g had even been reported in South Korea [34]. The Pb level in rice-based meals sampled across Europe has also been reported to be 12 µg/kg [35] which is more than 400 times lower than the mean Pb level for rice (5.41 µg/g) obtained in the present study. Apart from handling the raw food materials, the observed differences in Pb content in foods samples from the other reported studies, may be attributed to the rice cultivars and the soil content of Pb [36]. Indeed, it has been reported that different rice cultivars have different rates of uptake and bioaccumulation of Pb and other heavy metals [37].

For *fufu*, Pb was detected in 54% of samples analyzed, though with a modal concentration of 0 µg/g, but levels up to 25.35 µg/g were recorded. Again, though Pb was not detected in most *kenkey* and *banku* samples Pb levels, as high as 30.83 µg/g and 28.05 µg/g were recorded for *kenkey* and *banku* respectively. The possible use of Pb alloys in the manufacture of mill plates and cooking pots and their subsequent wearing into foods during processing may also account for the high Pb levels in the present study [38]. The inappropriate use of Pb containing pesticides and fertilizers may also have contributed to their high levels [4,39].

Presented in **Table 2** is the statistical distribution and central tendency metrics of the concentration of Pb in all the 106 foods analyzed. Pb was detected in 59% of the food samples collected from the study area. The concentration of Pb was distributed as "Expon" (6.7972, -0.064124) ranging from safe areas where no Pb was found in foods, to 30.83 µg/g. Though a modal concentration of 0 µg/g was recorded in the study area, indicating safe levels of Pb in frequently consumed foods, the uncertainty is that there could be concentrations ranging from 0.2 µg/g (5^{th} percentile) to as high as 20.3 µg/g (95^{th} percentile) (**Table 2**).

Relative to the mean Pb level found in this study (6.80 µg/g), other studies have recorded much lower levels of 36 µg/kg across Europe [35]. Relatively, levels such as 0.128 and 1.095 µg/g in China [40] and Iran [41] respectively, have been reported. Though the mean Pb level (6.80 µg/g) of foods in the present study is higher than what has been reported in other studies, it is consistent with findings in Ghana where relatively high levels have been reported [6,8].

Chronic exposures to lead

The exposure to Pb, as measured by the chronic daily intake, showed a wide variation (**Table 3**). The non-detection of Pb in about 41% of the food samples analyzed resulted in no chronic exposures to some consumers across the various age groups.

A maximum non-cancer exposure of 1,712.12 µg/kg bw-day was observed among adults (40 and above), followed by exposures of

Table 1: Concentration of lead in frequently consumed foods.

Food	Number of samples (Pb%)	Pb concentration (µg/g)	
		Mean ± Standard deviation	Min-Max
Banku	26 (58)	7.50 ± 9.54	0.00-28.05
Fufu	24 (54)	4.78 ± 6.89	0.00-25.35
Kenkey	26 (73)	9.55 ± 10.74	0.00-30.83
Rice	30 (53)	5.41 ± 7.99	0.00-20.80

Pb%: Percentage of samples that Pb was detected in. Pb concentrations below LOD were taken as zero.

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Table 2: Statistical distribution and metrics of the elements of exposure.

Age group	Variable	Statistical distribution	Min	Max	Central tendency metrics			Percentiles	
					Mean	Mode	Median	5th	95th
	C (µg/g)	Expon (6.7972, -0.064124)	0.0	30.83	6.8	0.0	2.4	0.3	20.3
5 – 19	MF (g/day)	Kumaraswamy (0.65047,1.8164,77.038,1046.96)	77	997	309	104	231	81	776
	EF (day/year)	Uniform (48.494, 367.51)	52	364	213	52	156	64	352
	ED (year)	Triang (1,1,20.083)	1	18	8	1	7	1	16
	BW (kg)	Kumaraswamy (1.3579,1.7725,18.637,65.884)	19	65	40	24	39	22	59
20–39	MF (g/day)	Triang (95.251,199.47,979.01)	104	939	405	166	347	163	793
	EF (day/year)	Uniform (50.340,365.66)	52	364	230	52	208	53	350
	ED (year)	Triang (1,1,38.880)	1	38	12	1	9	2	30
	BW (kg)	LogLogistic (-8.5909,75.010,11.663)	37	100	67	53	67	50	88
40 and above	MF (g/day)	Triang (54.804,138.89,1067.5)	66	1032	418	139	384	120	851
	EF (day/year)	Uniform (48.241,367.76)	52	364	202	52	156	64	352
	ED (year)	Expon (20.917,0.75099)	1	73	22	1	20	2	63
	BW (kg)	ExtValue (67.484,11.254)	49	120	74	66	72	55	101

Table 3: Chronic exposures to lead (µg/kg bw-day).

Age group	Min	Max	Central tendency metrics			Percentiles	
			Mean	Mode	Median	5th	95th
	Non-carcinogenic exposures						
5 - 19	0	776.21	8.10	0.03	2.67	0.09	33.30
20 - 39	0	725.33	11.32	0.06	4.32	0.15	45.88
40 and above	0	1,712.12	16.20	0.02	4.74	0.13	68.87
	Carcinogenic exposures						
5 - 19	0	332.66	3.47	0.01	1.14	0.04	14.27
20 - 39	0	310.85	4.85	0.02	1.85	0.06	19.66
40 and above	0	733.77	6.94	0.01	2.03	0.06	29.52

776.21 and 725.33 µg/kg bw-day in children and adolescents (5-19) and young adults (20-39) respectively. Though these maxima exposures were observed, the 95th percentiles ranged between 33.30 and 68.87 µg/kg bw-day across the three consumer groups. The trend of exposure for carcinogenic endpoints was similar to that for the non-cancer endpoints. The modal exposures observed in this study were very low across all age groups (0.01-0.06 µg/kg bw-day) however, there is still cause for concern, since there is no safe level of Pb exposure, especially in children [15]. The median exposures of 1.14-4.74 µg/kg bw-day across the consumer groups present a grave outlook.

From **Table 3**, relatively higher chronic mean exposures (3.47-16.20 µg/kg bw-day) and 95th percentile exposures (14.27-29.52 µg/kg bw-day) across the consumer groups were obtained in the current studies relative to some studies reported in China. Indeed, lower mean exposures (1.601- 2.104 µg/kg bw-day) and 95th percentile exposures (2.473-3.250 µg/kg bw-day) have been reported [40]. In fact, exposures in the present study are high relative to lower chronic exposures in children (1.03 µg/kg bw-day), adolescents (0.55 µg/kg bw-day) and adults (0.50 µg/kg bw-day) that have been reported [35]. Relative to the findings of the present study, where the highest exposure occurred in adults, the EFSA study recorded the highest exposures among infants and the lowest among adults [35]. Differences in the elements of exposure; mass of food consumed, exposure frequency and duration and the concentration of Pb in foods, may be responsible for the differences in findings [42]. However, there could be differences in age limits used in classification of the age groups relative to the EFSA study [35]. In spite of the EFSA study, another study reported a high daily exposure to Pb (1.46 µg/kg-bw day) among male adults (>65 years) relative to the lowest exposures (1.11 µg/kg-bw day) among female adolescents (10-19 years) [43]. Though these findings are relatively lower compared to the exposures in the present study, the trend of higher intake among adults was also observed in the present study.

Margin of exposure

Based on the prevalence of these disease endpoints among specific age groups [11,44], the risks of developmental neurotoxicity was characterized for children and adolescents only (age 5-19). On the other hand, the risks of chronic kidney disease and systolic blood pressure were characterized among young adults (age 20-39) and adults (age 40 and above). According to EFSA [11], the MoE for developmental neurotoxicity, which ranged from a minimum of 0.0 (<1), imply a worst case risk, whereas a maximum MoE of 53,951(>10) obtained in this study indicate no appreciable risk (**Table 4**). Thus, MoEs greater than 10 are deemed safe for children. Though the maximum MoE (53,951)

presents a safe situation, the value might be an outlier relative to the simulated 95th percentile value of 4.40 (<10). Thus, there is still public health concern.

According to EFSA, MoEs of between 1 and 10 present a low risk of adverse health effects to consumers, but they do not have to be totally dismissed as of no concern [11]. Compared to the evaluation of the present median MoE (0.18), a relatively higher median MoE (0.57-0.66) has been reported in the Netherlands [45]. The disparities probably arising from differences in Pb exposures in the populations [42]. A mean MoE of 1.10 was recorded in this study, indicating low risks, relatively, a higher mean MoE of between 3 and 13 was reported in a study in Ireland [44]. Similar to what was recorded in this study, the second French total diet study recorded a mean MoE of 0.9 for children aged between 3 and 17 [46], meaning Pb exposure is of global concern.

The reference point (BMDL of 0.50 µg/kg bw-day) used in computing the MoE for neurotoxicity correspond to a benchmark response of 1% (1-point) reduction in intelligence quotient (IQ) [11]. The high exposures recorded in this study (**Table 3**) indicate possibly lower IQ scores in children in the study area. The low modal MoE (0.02) recorded in the study area is worrying because it is well below the recommended MoE of 10 [11]. It has been estimated that a 1% decrease in IQ results in a 2% decrease in worker productivity later in life [47], thus, such high exposures are not acceptable. Again, the high level of exposures in children may result in developmental neurotoxicity which is linked to criminal behavior in adult life [48]. In fact, the Institute for Health Metrics and Evaluation (IHME) has reported that 63.8% of the global burden of idiopathic developmental intellectual disability is attributable to Pb exposure [49]. It must be a matter of grave concern that there is high exposure to Pb among children in the study area. Since this level of exposures could lead to increased risks of developmental neurotoxicity in children, every effort must not be spared to effectively manage this problem.

Also presented in **Table 4** are the MoEs for cardiovascular (systolic blood pressure) effects. A median MoE of 0.33 was obtained for consumers aged between 20 and 39 and a median of 0.30 for consumers aged 40 and above. A higher median MoE of 3.7 (meaning lower risk) has been reported in other studies [45], indicating that the findings in the present study was not acceptable. A 95th percentile MoE of 7.90 (age 20-39) and 9.17 (age 40 and above) was obtained in this study. This is similar to findings from a study in France where the 95th percentile MoE of 8.0 was obtained for consumers aged between 18 and 79 [46]. On the basis of the 95th percentile, exposures in the present study are safer relative to what has been reported in Canada (0.81) [50]. However, a worrying trend of MoEs was consistently observed in the adult population relative to the younger population. For instance, the most frequent (modal) MoE (0.05) was recorded among consumers aged between 20 and 39 and an even lower modal MoE (0.02) among consumers aged 40 and above. These MoEs are lower than 1 and present significantly unacceptable risks of cardiovascular toxicity which is manifested as an increase in systolic blood pressure [11]. Such increases have been linked to the occurrence of cardiovascular disorders such as stroke, heart attack and coronary artery disease [18]. In fact, the IHME estimates that 3.1% and 3% of the global burden of stroke and coronary artery disease respectively, is attributable to Pb exposure [49].

Table 4: Distribution metrics of MoE across consumer groups.

Age group	Min	Max	Central tendency metrics			Percentiles	
			Mean	Mode	Median	5th	95th
5–19[1]	0.00	53,951	1.10	0.02	0.18	0.01	4.40
20–39[2]	0.00	207,568	4.37	0.05	0.33	0.03	7.90
40 and above[2]	0.00	41,509	2.33	0.02	0.30	0.02	9.17
20–39[3]	0.00	87,178	1.84	0.02	0.14	0.01	3.32
40 and above[3]	0.00	17,434	0.98	0.009	0.13	0.008	3.85

[1]MoEs for developmental neurotoxicity; [2]MoEs for systolic blood pressure; [3]MoEs for chronic kidney disease.

Table 5: Lifetime cancer risk of consumer groups.

Age group	Min	Max $\times10^{-3}$	Central tendency metrics			Percentiles	
			Mean $\times10^{-5}$	Mode $\times10^{-7}$	Median $\times10^{-5}$	5th $\times10^{-7}$	95th $\times10^{-4}$
5 - 19	0	2.83	2.95	1.22	0.97	3.32	1.21
20 - 39	0	2.64	4.13	2.06	1.57	5.52	1.67
40 and above	0	6.24	5.90	0.60	1.73	4.72	2.51

Table 6: Standardized regression coefficients (β) of risk factors.

Age group	C	MF	EF	ED	BW
5 - 19	0.47	0.34	0.21	0.28	-0.14
20 - 39	0.55	0.26	0.24	0.36	-0.09
40 and above	0.44	0.24	0.20	0.42	-0.07

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The effects of increasing systolic blood pressure have also been linked to increased incidences of chronic kidney disease. In this study, the risk of chronic kidney disease has been characterized and the MoEs presented in **Table 4**. MoEs of 3.32 and 3.85 (95th percentiles) were obtained for consumers between 20 to 39 years and consumers above 40 years respectively. This indicate very low risks (MoE between 1 and 10) among the low exposed groups of consumers in these age groups. Relative to the 95th percentile MoE of 0.90 obtained in Netherlands [45], the present study presented relatively safer outcome(3.32-3.85). Relatively lower risks also prevailed in Ireland, where a median MoE ranging between 5 and 16 was reported compared to what was obtained in this study (**Table 4**) [44]. Variations that are common among the findings of this study and other studies could be attributable to different population characteristics and concentration of Pb found in their diets [42]. Though a modal MoE of 0.02 was recorded for the age group 20-39, in the current study, an even lower modal MoE of 0.009 was obtained for consumers aged 40 and above. The implication of these findings is that most of the consumers are likely to be at risk (MoE<1) of chronic kidney disease.

Lifetime cancer risk

From **Table 5**, the risk of carcinogenesis ranged from cases of acceptable risk (0), observed across all groups of consumers, to a highest of 6.24×10^{-3} (6 out of 1000 persons at risk) which was observed among consumers aged 40 and above.

The modal risks obtained in the present study is relatively lower compared to the risk range of 1.3×10^{-4} and 2.4×10^{-4} reported in Malaysia [51]. The differences between this current study and the study in Malaysia could be arising from the approaches of the two studies since their study used a deterministic approach. It has been reported that deterministic approaches are conservative and do not account for variability in elements of exposure due to the use of single estimates [52]. From **Table 5**, lower cancer risks were obtained for consumers between the ages of 5 and 19, relative to consumers in age groups: (20-39) and those aged 40 and above. The trend reported in this study is in contrast with findings from another study where higher risks were observed in children [53]. The differences might be resulting from the approach of the study. While the lifetime exposure method (Equation 1) was used in this study, the other study [53] used the average daily intake or estimate daily intake (EDI) method.

Cancer risks have a de minimis (acceptable risk) of 1×10^{-6}, therefore risk estimates lower than the de minimis present acceptable risks to consumer populations [12]. Since a risk value (median) of 9.7×10^{-6} (10 out of 1 million) was observed in consumers aged between 5 and 19, it implies the risk is unacceptable (**Table 5**). While the modal ILTCR was acceptable among all the age groups ($<10^{-6}$), the 50th percentiles presented cautiously acceptable risks (2×10^{-5}) of '2 out of 100 thousand' consumers for the two adult consumers groups. Again, this must be interpreted carefully since the 95th percentiles across all three consumer groups show unacceptable risk ($<10^{-4}$), below the risk management threshold (1×10^{-4}) set by US EPA [54].

The regression analysis of the risk descriptors as presented in **Table 6** shows the highest standardized regression (β) coefficients across all the three consumer groups to be concentration of Pb (C). This means the highest impact on the risk were all from the concentration of Pb in the foods analyzed.

There is paucity of information regarding the impact (standardized regression coefficient) of elements of exposure on risk estimates. However, a study on some hazards [29], in the same study area, showed that the concentration of hazard had the highest impact on risk, similar to what was observed in the present study. For the young adults age group (20-39), the exposure duration (β=0.36) and mass of food consumed (β=0.26), had the next higher impacts. The reverse was observed in children and teenagers (5-19) where the mass of food consumed had a bigger impact (β=0.34) on cancer risk relative to the exposure duration (β=0.28). The body weights of all three consumer groups had negative regression coefficients. This implies that higher body weight of consumers had a negative impact on the cancer risk. This observation does not suggest that in managing the risk of cancer in this study area, increased body weight should be recommended. It simply means that there is an inverse relationship between risk and body weight. Risk management efforts of Pb must rather focus on the reduction of the concentration of Pb in the diets of consumers.

Conclusion

This study showed the presence of Pb in 59% of the 'frequently consumed foods' analyzed, with a median concentration of 2.4 µg/g. A trend of lower chronic exposures was observed in children and adolescents (5-19) as compared to the exposures in young adults and adults (≥ 40). Though low modal non-carcinogenic exposures of 0.01-0.06 µg/kg bw-day were found in the study area, the high 95th percentile exposures (33.30-68.87 µg/kg bw-day) suggest significant consumers might be at risk. This fact was further buttressed by the findings from the carcinogenic exposures. Very low modal MoEs (0.009-0.05) were recorded for all consumers in the study area, indicating that most children in the study area were at risk of developmental neurotoxicity. Adults, on the other hand, were at risk of chronic kidney disease and increased systolic blood pressure. Though the modal cancer risks (1.22×10^{-7} - 6.0×10^{-8}) were below de minimis (1×10^{-6}), the 95th percentile risks (1.21×10^{-4} - 2.51×10^{-4}) across the three consumer groups were above the threshold (1×10^{-4}) required for management action. Thus, findings from this study indicate that there is the need for sustained or regular risk assessment to inform risk management actions.

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Declaration of Competing Financial Interests

The authors declare they have no actual or potential competing financial interests.

Contribution of Authors

Edmund O. Benefo, collected and processed the data and also drafted the initial manuscript; Gloria M. Ankar-Brewoo and Herman E. Lutterodt, made critical suggestions to the initial manuscript while correcting certain portions; Michelle Oppong Siaw collected and processed parts of the data for this manuscript. Isaac W. Ofosu, designed the study and made significant corrections of the draft manuscript prior to submission.

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