Scientia Africana, Vol. 21 (No. 3), December, 2022. Pp 57-74 https://dx.doi.org/10.4314/sa.v21i3.5
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NON-CARCINOGENIC AND CARCINOGENIC RISK ASSESSMENT OF SOME HEAVY METALS IN CARROTS SELECTED FROM WUDIL, KANO, NIGERIA

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Received: 03-07-2022
Accepted: 26-11-2022

ABSTRACT

Fruits and vegetables contaminated with heavy metals can pose health risks to humans. Non-carcinogenic and carcinogenic risks of some metals (Cu, Cd, Cr, Pb, and Ni) in carrots cultivated in Wudil, Kano, Nigeria was investigated by calculating the chronic daily intake (CDI), target hazard quotient (THQ), hazard index (HI), and carcinogenic risk (CR) of the metals. The mean concentrations of the metals were in the following decreasing order: Cu>Cr>Ni>Pb>Cd. The levels of all the metals were below the maximum tolerable limit set by FAO/WHO. The THQ for the heavy metals were all less than one (THQ < 1), showing that these metals did not constitute a non-carcinogenic risk to the population through the consumption of carrots. The percentage contributions of the metals to the total non-carcinogenic risk were in the following order: Cr>Pb>Cu>Cd>Ni. Cr was the major contributor to the total non-carcinogenic risk and accounted for 57.61% of the total risk, while Pb, Cu, Cd, and Ni contributed 15.64%, 12.76%, 7.41%, and 6.58% respectively. The CR were all below the upper limit of 1×10^{-4}, suggesting that lifetime exposure to these metals through the consumption of carrots did not pose a cancer risk to the population. The levels of the investigated metals in carrots in the area did not pose any risk to the consumers. However, there is a need to monitor the metals on regular basis to ensure the safety of the consumers.

Keywords: Heavy metals, Carcinogenic risk, Carrots, Toxicity, Hazard

INTRODUCTION

Fruits and vegetables are of significance in the daily diet as they are rich in proteins, carbohydrates, minerals, vitamins, and fibres (Baghaie and Fereydoni**,** 2019). A diet rich in vegetables and fruits can lower blood pressure, reduce the risk of heart disease and stroke, prevent some types of cancer, and lower the risk of eye and digestive problems. Based on this, the WHO recommended the consumption of a minimum of 400 g of fruits and vegetables daily (excluding potatoes and other starchy tubers) (WHO, 2004). Although vegetables are vital component of human diets, they are also capable of accumulating high levels of toxic metals from the environment (Xiong, 1998; Cobb *et al.* 2000; Adriano, 2001; Molla *et al.* 2021). They bioaccumulate in the roots, stems, fruits, grains, and leaves of plants (Fatoki, 2000).

Due to their properties such as toxicity, persistence, and non-biodegradation, heavy metals has become a serious global environmental threat, particularly in urban areas (Zwolak *et al.* 2019). High levels of these metals in agricultural soils and plants arise mainly from anthropogenic sources, which include mining, smelting, waste disposal, urban effluents, vehicle exhausts, sewage sludge, pesticide, herbicides, and fertilizers

application (Abdullahi *et al.* 2021). The use of agrochemicals and wastewater for irrigation has been implicated as the sources of metals in most of the studies on vegetables (Mahmood and Malik, 2014; Chauhan and Chauhan, 2014; Xu *et al.* 2022). Absorption and accumulation of heavy metals in plant tissues depend upon the plant species and soil properties such as soil type, temperature, moisture, organic matter, pH, and nutrient availability (Hu *et al.* 2017; Onakpa *et al.* 2018). However, soil to plant transfer is not the only route of accumulation of heavy metals from the environment. Aerial deposition onto leaf surfaces and trapping of metals in hairs or rough cuticle surfaces are also significant routes (Marschner, 1995; Onoyima, 2021).

Some of these metals are essential for the normal function of the body, while some have no known function in the body. Higher levels of both essential and non-essential metals in food pose potential health risks to human and animal consumers (Ndibukke and Egbe, 2018). Heavy metals in food plants accumulate in organs resulting in diverse health risks (Mahmood and Malik, 2014; Likuku and Obuseng, 2015; Emurotu and Onianwa, 2017), including cellular damage, alteration of enzyme specificity (Chen *et al.* 2016), damage to the structure of DNA and alterations of gene expression (Hussein *et al.* 2005), carcinogenesis and neurotoxicity (Jomova *et al.* 2011). Toxic metals in food also significantly decrease vitamin C and other essential nutrients in the body, leading to a decline in immunity, deterioration of human functions, and disabilities associated with malnutrition (Liu *et al.* 2005).

There is a rise in research related to food safety due to human health risks posed by the consumption of metal-contaminated food. Human health risk assessment is the characterisation of the potential adverse effects on humans as a result of exposure to environmental hazards, and is expressed in terms of non-carcinogenic and carcinogenic health risks (USEPA, 1989). As recommended by the United States Environmental Protection Agency (USEPA), non-carcinogenic risk values are calculated using chronic daily intake (CDI), hazard quotient (HQ), and hazard index (HI), while the carcinogenic risk is calculated with chronic daily intake (CDI) and cancer slope factor (CSF). Non-carcinogenic risk assessment makes use of threshold reference dose (RfD) (a dose below which no adverse health effect will be observed), while there is no safe threshold for exposure to carcinogenic chemicals, which implies there is a chance of cancer developing with exposure at low doses (USEPA, 1986).

The uptake of heavy metals from the environment by plants has been widely studied (Abba *et al.* 2020; Aendo *et al.* 2020; Karimi *et al.* 2021; Onoyima, 2021; Xu *et al.* 2022). Vegetables are known to be good absorbers of heavy metals from the soil. Leafy vegetables and root vegetables have the greatest ability to accumulate these metals (Chauhan and Chauhan, 2014; Zwolak *et al.* 2019). Carrot is a root vegetable, largely cultivated in Northern Nigeria, including Kano, Kaduna, Plateau, Sokoto, and Jos. The root is consumed by many on daily basis and contains high quantities of alpha and beta carotenes, vitamins B and K. They are widely used in many cuisines, especially in the preparation of salads. The present study aims to assess the non-carcinogenic and carcinogenic risks posed by the consumption of carrots by both the adult and children population of Wudil, Kano, Nigeria. Human health risk assessment uses risk as an evaluation index to link environmental pollution with human health and quantitatively describes the risk of

Scientia Africana, Vol. 21 (No. 3), December, 2022. Pp 57-74 https://dx.doi.org/10.4314/sa.v21i3.5
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pollution to human health (Tian *et al.* 2019). The study will give information about any threat of heavy metals contamination in the vegetables, as consumption of contaminated vegetables constitutes an important route of human exposure to heavy metals. The main aim is to provide useful information to policymakers and regulators.

MATERIALS AND METHODS

Sample collection and analysis

Freshly harvested carrots were selected directly from the farmers at three different locations in the area. The samples were washed repeatedly with running tap water and rinsed three times with distilled water. They were sliced into small pieces, oven-dried at 70 ^{o}C for 48 h, and ground into a powder with mortar and pestle. The powder was then sieved with a 3 mm sieve to obtain a fine powder. Approximately 1 g of the powdered sample was transferred into a beaker and a mixture of 5 ml of 60 % HCl and 10 ml of 70 % HNO_3 was added and digested in a hot plate at 100 ^{o}C for 2 h. Then, 20 ml of distilled water was added and heated until a colourless solution was obtained. The solution was removed and allowed to cool. Distilled water was then added to make it up to 100 ml in a volumetric flask. The solution was stirred and filtered and the filtrate was used to determine the concentration of the heavy metals using Atomic Absorption Spectrophotometry.

Exposure assessments

Exposure assessment using the chronic daily intake of metals was evaluated to estimate the average daily loading of metal into the body system of a specified body weight of a consumer. The daily intake of metal in this study was calculated based on the formula reported by Sajjad *et al.* (2009) as shown:

$$CDI = \frac{C_m \; x \; CF \; x \; DFI}{BW} \qquad (1)$$

Where, C_m, CF, DFI, and BW represent the concentration of the heavy metals, conversion factor, daily food intake, and body weight respectively. A conversion factor (CF) of 0.085 was used to convert the fresh sample weight to dry weight, while the average adult and children daily vegetable intake rate of 0.345 and 0.175 kg/person/day respectively, and body weight of 60 kg and 32.7 Kg for adults and children respectively were used as reported in the literature (Wang *et al.* 2005; Likuku and Obuseng, 2015).

Non-carcinogenic risk assessment

The non-carcinogenic risk estimation of heavy metals consumption was determined using the Target Hazard Quotient (THQ) and Hazard Index (HI).

$$THQ = \frac{CDI}{RfD} \qquad (2)$$

Where RfD is the oral reference dose (mg/kg/day), defined as the daily oral exposure to a substance that will not result in any deleterious effect in a lifetime for a given human population. The RfD values for the assessed metals are as follows: Cd = 0.001, Cu = 0.04, Ni = 0.02, Pb = 0.0035, and Cr = 0.003 (FAO/WHO, 2013).

The target hazard quotient (THQ) based on average daily intake (CDI) of THQ $\leq$ 1 indicates no risk (USEPA, 1989).

The hazard index (HI) was calculated as the summation of the Target Hazard Quotient (THQ) arising from all the metals examined.

$$HI = \Sigma THQ \qquad (3)$$

The value of the hazard index is proportional to the magnitude of the toxicity of the vegetables consumed.

Carcinogenic risk assessment

Carcinogenic risk (CR) assessment estimates the probability of an individual developing cancer over a lifetime due to exposure to the potential carcinogen. CR was calculated using equation 4 below:

$$CR = CSF \times CDI \quad (4)$$

Where CDI and CSF are the chronic daily intake (mg/kg/day) and cancer slope factors $(mg/kg/day)^{-1}$ respectively. The ingestion CSF is a reasonable upper-bound approximation of the prospect of a body response per ingestion of hazardous substances over a lifespan. The CSF for the studied heavy metals with oral carcinogenic data are: Cd = 0.38; Pb = 0.0085; Cr = 0.5 (USEPA, 1999).

According to USEPA, (2011) CR between 1×10^{-6} to 1×10^{-4} represents a range of permissible predicted lifetime risks for carcinogens. Chemicals for which the risk factor falls below 1×10^{-6} may be eliminated from further consideration as a chemical of concern.

RESULTS AND DISCUSSION

Heavy metal concentrations in carrots

The mean concentrations (mg/kg) of heavy metals in carrots selected from different locations in Wudil, Kano are presented in Table 1. The values showed that the decreasing order of the mean concentrations of the heavy metals was: Cu > Cr > Ni > Pb > Cd. Copper (Cu) has the highest mean concentrations (0.4300±0.0153 - 0.4567±0.0460 mg/kg), which are all below the maximum tolerable limit of 40.00 mg/kg set by FAO/WHO, (2001). The values are far lower than those obtained for carrots cultivated in an industrial area of Kano by Abba *et al.* (2020) (6.01±1.00); in Kano metropolis by Lawal and Audu, (2011) (0.69±0.12 – 7.5±1.08), and those in Berlin Germany (5.4 – 23.2 mg/kg) and in Wisley UK (4.43 mg/kg) by Säumel *et al.* (2012) and Alexander *et al.* (2006) respectively. The values were comparable to those of Doherty *et al.* (2012) in Lagos Nigeria; and higher than results obtained in Jos, Nigeria by Mafuyai *et al.* (2020) (0.155±0.02 mg/kg), and in Chenzhou City, China by Zhou *et al.* (2016) (0.227±0.011 mg/kg) and Xu *et al.* (2022) (0.1861 mg/kg). Sources of Cu in agricultural soil include electrical equipment, chemicals, paints, agricultural pesticides (e.g. copper sulphate), preservatives, vehicular emission, and brake pad wear, etc. (Cui *et al.* 2019; Al-Edresy *et al.* 2019).

The levels of Cr in the carrots ranged from 0.1263±0.0029 to 0.1690±0.0104 mg/kg. These values are below the FAO/WHO safe limit of 2.3 mg/kg. The values are similar to the results obtained for carrots in Jos, Nigeria (0.158±0.04) (Mafuyai *et al.* 2020) but lower than those reported by Abba *et al.* (2020) (65.10±3.20) and Udibia *et al.* (2018) (1.19 – 3.76 mg/kg) in an industrial area in Kano, Nigeria; and Säumel *et al.* (2012) (0.10 – 2.39 mg/kg) in Berlin Germany. The values of Cr in carrots obtained in this study were higher than those reported by Guerra *et al.* (2012) (0.09 mg/kg), Pan *et al.* (2016) (0.057 mg/kg), and Antoine *et al.* (2017) (0.031 mg/kg). Sources of Cr in the agricultural soil include application of chemical fertilizers and pesticides, wastewater from industries (Islam *et al.* 2009; Bhuiyan *et al.* 2011), atmospheric deposition from coal-burning dust, electroplating, paints, and laboratory effluents (Cui *et al.* 2019; Astatkie *et al.* 2021).

The concentrations of Ni ranged from 0.0900±0.0100mg/kg to 0.1467±0.0623mg/kg, which are all below the maximum permissible limit set by FAO/WHO (1.0

Scientia Africana, Vol. 21 (No. 3), December, 2022. Pp 57-74 *https://dx.doi.org/10.4314/sa.v21i3.5*
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mg/kg). The values were also below the ones reported by Abba *et al.* (2020) (2.90±1.11 mg/kg) and Lawal and Audu, (2011) (2.02±0.35 mg/kg) in Kano, Nigeria, but comparable to those reported by Guerra *et al.* (2012) in Sao Paulo, Brazil (0.11 mg/kg) and Säumel *et al.* (2012) in Berlin Germany (0.07 – 1.93 mg/kg). Ni is widely distributed in nature, forming about 0.008% of the earth's crust (IARC, 1990). It is released into the soil and then plants from Cu-Ni smelting, burning of diesel oil containing Ni, city effluents, bio-solid, impurities in fertilizers, mining, etc. (Alloway, 1990).

The mean concentrations of Pb in carrots in this study were between 0.0213±0.0048 mg/kg to 0.0717±0.0152 mg/kg. The levels of Pb are all below the maximum permissible limit of 0.3 mg/kg set by FAO/WHO, (2001). These values obtained in this study are also lower than those reported by Mafuyai *et al.* (2020) (0.545±0.03 mg/kg) in Jos Nigeria; Udibia *et al.* (2018) (0.34 – 1.03 mg/kg) and Lawal and Audu, (2011) (21.22 – 35.28 mg/kg) in Kano Nigeria; Guerra *et al.* (2012) (0.38 mg/kg) in Sao Paulo, Brazil; and Zhou *et al.* (2016) (0.233±0.001 mg/kg) in Chenzhou, China. The results were, however, comparable to those reported by Pan *et al.*, (2016) (0.034 mg/kg) and Xu *et al.* (2022) (0.0153 mg/kg). Sources of Pb include automobile exhaust, wastewater sludge, pesticides, batteries, solders, bearings, cable covers, ammunition, and pigments (Wuana and Okieimen, 2011).

The levels of Cd (ND - 0.0143±0.0043 mg/kg) in the samples of carrots in this study are all below the maximum allowable limit (0.20 mg/kg). The values are also lower than that reported by Sobukola *et al.* (2010) (0.09 mg/kg) in Lagos, Nigeria, Udibia *et al.* (2018) (0.20 – 0.36 mg/kg) in Kano, Nigeria, Zhou *et al.* (2016) (0.023±0.005 mg/kg) in Chenzhou, China, and Guerra *et al.* (2012) (0.03mg/kg) in Sao Paulo, Brazil. The results are comparable to those reported by Doherty *et al.* (2012) (0.001 – 0.003 mg/kg) in Lagos Nigeria, and Mafuyai *et al.* (2020) (0.013±0.10 mg/kg) in Jos, Nigeria. Significant sources of Cd in agricultural soil include discharge from phosphate fertilizers, sewage sludge (Abdullahi *et al.* 2021), polluted irrigation water (Karimi *et al.* 2021; Xu *et al.* 2022), and industrial sources (electroplating, batteries, paints) (WHO, 2007).

Table 1: Mean concentrations (mg/kg) of heavy metals

	Cd	**Cu**	**Ni**	**Pb**	**Cr**
A	0.0143±0.0043	0.4567±0.046	0.1033±0.0233	0.0717±0.0152	0.1690±0.0104
B	0.0033±0.0033	0.4300±0.0153	0.1467±0.0623	0.0477±0.0412	0.1263±0.0029
C	ND	0.4500±0.0058	0.0900±0.0100	0.0213±0.0048	0.1473±0.0037

ND = not detected

Exposure assessment

Risk depends on the concentration of chemicals present in an environmental medium, the amount of contact (exposure) a person has with the pollutant in the medium, and the toxicity of the chemical. Exposure is the amount of pollutants absorbed by the human body through different pathways (inhalation, ingestion, dermal contact) (IPCS, 2010). Exposure assessments, using the chronic daily intake (CDI) (mg/person/day), show that the exposure to the studied heavy

metals through ingestions of carrots in the area by the adult population are in the following range: Cd (1.613×10^{-6} - 7.00×10^{-6}), Cu (2.047×10^{-4} - 2.232×10^{-4}), Ni (4.399×10^{-5} - 7.170×10^{-5}), Pb (1.041×10^{-5} - 3.504×10^{-5}), Cr (6.173×10^{-5} - 8.260×10^{-5}). These show that the average human exposure to the heavy metals are in the following order: Cu > Cr > Ni > Pb > Cd. There was no significant difference between exposure to adults and children. It was observed that all the CDI values were below the tolerable limit set by FAO/WHO, **(**2013**).** One limitation of the daily intake estimate is that it does not take into account the possible metabolic ejection of metals. However, it is a good estimate of the possible ingestion rate of a particular metal and is a significant step in health risk assessment (Latif *et al.* 2018).

Table 2: Chronic daily intake of metals (CDI) (mg/person/day)

LOCATION		**Cd**	**Cu**	**Ni**	**Pb**	**Cr**
A	Adult	7.00×10^{-6}	2.23×10^{-4}	5.05×10^{-5}	3.50×10^{-5}	8.26×10^{-5}
	Children	6.51×10^{-6}	2.08×10^{-4}	4.70×10^{-5}	3.26×10^{-5}	7.69×10^{-5}
B	Adult	1.61×10^{-6}	2.10×10^{-4}	7.17×10^{-5}	2.33×10^{-5}	6.17×10^{-5}
	Children	1.50×10^{-6}	1.96×10^{-4}	6.67×10^{-5}	2.17×10^{-5}	5.75×10^{-5}
C	Adult	-	2.20×10^{-4}	4.40×10^{-5}	1.04×10^{-5}	7.20×10^{-5}
	Children	-	2.05×10^{-4}	4.09×10^{-5}	9.69×10^{-6}	6.70×10^{-5}

Non-carcinogenic risk assessment

Target hazard quotient (THQ) and hazard index (HI) were used to assess the potential non-carcinogenic risk posed by the consumption of carrots contaminated with heavy metals to the human population in the study area. THQ indicates the potential non-carcinogenic risk probability in a population exposed to the particular toxicant, while HI is the cumulative effect of all the studied pollutants. The results of the THQ were presented in Figure 1. The results indicate that the THQ for the studied heavy metals are all less than one (THQ < 1), showing that these metals did not constitute non-carcinogenic risk to the population through the consumption of carrots.

The THQ for Cd ranged between 0.002 – 0.007. Cd has no known essential biological function. Its toxicity is thought to be partly related to its chemical similarity to an essential trace element Zn; it can substitute Zn and cause the malfunctioning of many metabolic processes (Campbell, 2006). The main target organ following chronic oral exposure is the kidney, resulting in glomerular toxicity, kidney dysfunction, or failure (ATSDR, 1999; Bawaskar *et al.* 2010; EFSA, 2012). Cd also affects other organs and causes skeletal disorders (osteomalacia, and/or osteoporosis), diabetic complications (Buha *et al.* 2019), liver damage, cardiovascular diseases, and hypertension (Wu *et al.*, 2010), dysfunction of the sexual glands, and disrupting of mineral balance in the body (Zwolak *et al.* 2016). It is reported to induce premature birth and reduces birth weight in infants (Jaishnkeer *et al.* 2014). Cd is a severe pulmonary and gastrointestinal irritant, leading to symptoms like abdominal pain, burning sensations, nausea, vomiting, salivation, muscle cramps, shock, and loss of consciousness (Baselt and Cravey, 1995).

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The THQ for Cu ranged from 0.005 – 0.006. Cu is an essential part of several enzymes, and it is necessary for the synthesis of haemoglobin (Khan *et al.* 2014). However, Cu has been reported to result in acute toxicity from excessive ingestion resulting in gastrointestinal distress, nausea, vomiting, and diarrhoea (Lam *et al.* 1985; Pizzaro *et al.* 1999; Madilonga *et al.* 2021). It also affects hepatic, renal, and central nervous systems (Koupaie and Eskicioglu, 2015), and can accumulate in the liver, brain, kidney, and cornea and result in Wilson's disease (hepatolenticular degeneration) (Frydman *et al.* 1985; ATSDR, 2002). Free radical-induced oxidative damage has been mostly implicated in Cu-induced cellular damage (Lippard, 1999; Gaetke *et al.* 2014).

There was no non-carcinogenic risk due to Ni as the THQ ranged from 0.002 to 0.004. The toxicity of Ni is due to the interference with the metabolism of essential metals such as Fe, Mn, Ca, Zn, and Mg (Kasprzak *et al.* 2003). It is believed that Ni may interfere with general body functions by replacing other metals in metalloenzymes. It is also possible that high Ni doses may interfere with the transport of the trace essential elements (ABC, 1988). The most widely reported health effect of Ni is Ni-induced contact dermatitis (which is more common in women than men), mainly through dermal contact, but with some reported cases of systemic contact dermatitis through the oral route (Veien and Menne, 1990; Buxton *et al.* 2019). Exposure to Ni through inhalation leads to health risks such as fibrosis, chronic bronchitis, impaired pulmonary functions, and emphysema (IARC, 1990). However, the most sensitive target for non-cancer effects of oral exposure is the kidney, specifically decreased glomerular function (Vyskocil *et al.* 1994). Oral exposure to Ni has also been observed to result in decreased immune function (Schiffer *et al.* 1991), decreased body weight (ABC, 1988), and inhibition of enzymatic activities (Ndibukke and Egbe, 2018). Ni can cross the placenta, and several oral studies have reported increased neonatal mortality of doses below those resulting in maternal toxicity. Congenital heart defect in infants has been associated with Ni exposure in mothers (Smith *et al.* 1993; Zhang *et al*. 2019).

This study indicated that there was no risk due to Pb in carrots in the study area (THQ < 1). Pb mimic or inhibit the action of Ca. It binds to biological molecules (e.g. enzymes) and interferes with their function (ATSDR, 2007). The effect of Pb toxicity is greater in children (because Pb is excreted more in adults than in children), and results in neurodegenerative disease, interfering with brain function, lowering intelligence quotient (IQ), and prenatal exposure is thought to cause foetal abnormality (NRC, 1993; Karri *et al.* 2018). Pb also interferes with heme biosynthesis and the proper function of the mitochondria, impairing respiration, causing anaemia, swelling of the brain, paralysis, and death (Oluyemi *et al.* 2008). It has also been reported to disrupt, vitamin D metabolism (leading to osteoporosis), and disorders of the immune and reproductive systems (Bonde *et al.* 2002; Zwolak *et al.* 2019). Acute exposure leads to liver and kidney tubular damage (WHO, 1995).

The THQ for Cr in the samples are all less than unity (THQ <1), and ranged from 0.019 to 0.028, indicating that it is not of serious concern for non-carcinogenic risk. While Cr (III) is considered an essential trace nutrient with some physiological roles, Cr (VI) is highly toxic due to its ability to generate reactive oxygen species (ROS) which damage cell biomolecules, and cause functional

degradation (Shi and Dalal, 1992; Patlolla *et al.* 2009; Sobol and Schiest, 2012). Cr (VI) is known to cause damage to the nasal epithelia and other skin surfaces, allergic contact dermatitis, and asthma (Tan *et al.* 2016); ulceration, kidney, liver, and nerve tissue damage (Akomaye *et al.* 2018); higher pregnancy loss, miscarriage, low birth rate, perinatal jaundice, and congenital disabilities (Ferreira *et al.* 2019).

The cumulative effects of all the studied heavy metals using HI showed the following results: Site A (adults = 0.054, children = 0.049), Site B (adults = 0.039, children = 0.035, Site C (adults = 0.034, children = 0.032). The results showed low HI (HI < 1), indicating low non-carcinogenic risk due to all the studied metals. There was no significant difference between the risk posed to the adult population and the children. There was also no variation between the sampling locations. The percentage contribution of each of the metals to the total non-carcinogenic risk (Figure 2) can be arranged in the following order: Cr > Pb > Cu > Cd > Ni. The result shows that Cr is the major contributor to the total non-carcinogenic risk and accounted for 57.61% of the total risk, while Pb, Cu, Cd and Ni contributed 15.64%, 12.76%, 7.41%, and 6.58% respectively.

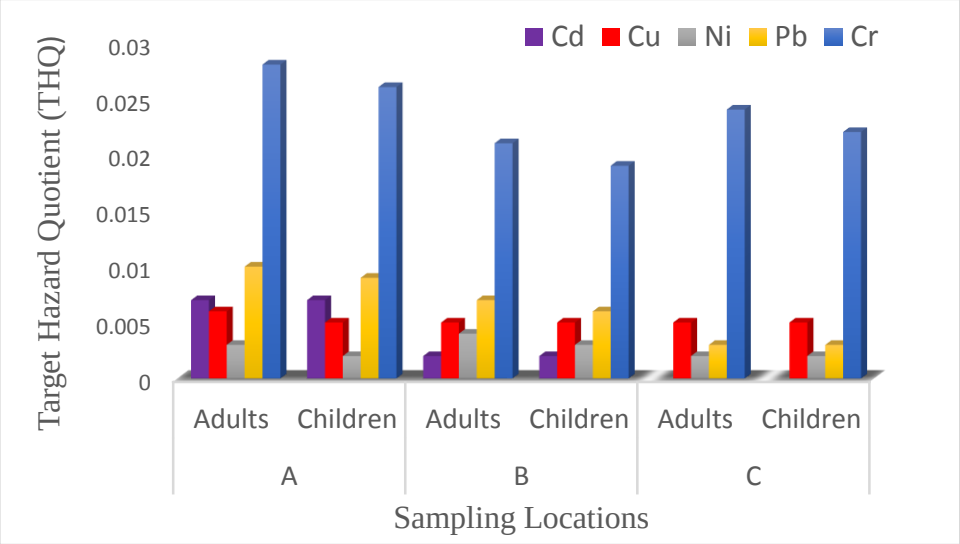

Figure 1: Target Hazard Quotient (THQ) of heavy metals in the carrots

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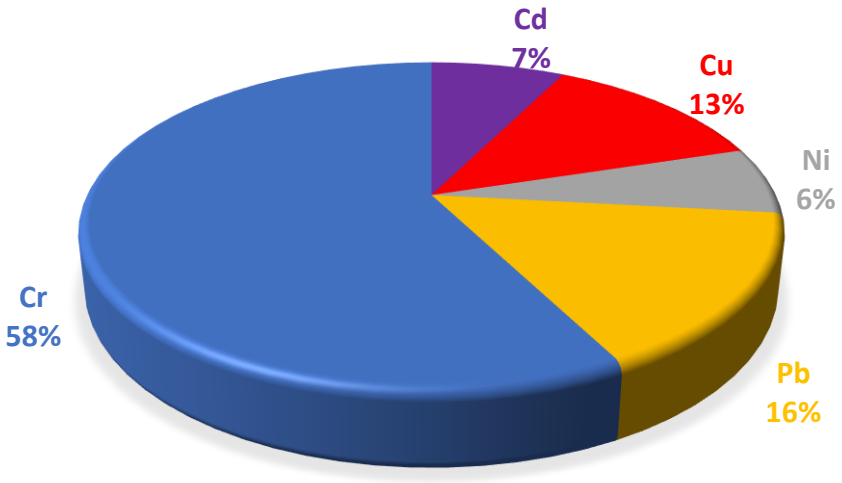

Figure 2: Percentage contributions of the metals to non-carcinogenic risk

Carcinogenic risk assessment

Carcinogenic Risk (CR) is the incremental risk or the probability of an individual developing cancer over a lifetime (Gebeyehu and Bayissa, 2020). Cancer risk lower than 1×10^{-6} is considered to be negligible, cancer risk above 1×10^{-4} is considered unacceptable, and CR lying in the range of 1×10^{-6} to 1×10^{-4} is generally considered an acceptable range (USEPA, 1989). The results of the CR for Cd, Pb, and Cr, calculated for both adults and children, as presented in Table 3 show that CR for Cd ranged from 5.70×10^{-7} - 2.66×10^{-6}, Pb ranged from 8.25×10^{-8} - 2.98×10^{-7}, and Cr from 2.87×10^{-5} - 4.13×10^{-5}. These values are all below the upper limit of 1×10^{-4}, suggesting that lifetime exposure to these metals through the consumption of carrots did not pose a cancer risk to the adult and children population in the area.

The Carcinogenic risk was calculated only for the metals with carcinogenic or mutagenic data which include Cr, Cd, and Pb. Although Ni compounds have earlier been classified as Group 1 (carcinogenic to humans), and metallic Ni as group 2B (possibly carcinogenic to humans) (IARC, 2012), there is inadequate evidence in humans for the carcinogenesis of Ni, and inadequate database for the oral carcinogenicity of soluble and insoluble Ni salts (Kasprzak *et al.* 2003). Ni compounds appear to be associated with tumour response only through the inhalation route (USEPA, 1986). The ability of metals to induce cancer depends on the oxidation state, complex form, and solubility of the metal (Beyersmann and Hartwig, 2008). While soluble Ni salts are believed to produce mutations via reactive oxygen species which inhibit DNA repair (Lynn *et al.* 1997; Han *et al.* 2018), carcinogenesis of insoluble Ni salts appears to be due to interaction with heterochromatin (a densely packed DNA that has low transcriptional activity) (Costa *et al.* 1994; Kumar *et al*. 2017).

The cancer risk in this study was ranked in the order of Cr > Cd > Pb. The CR for Cr is within the USEPA normal range of $1 \times 10^{-6} – 1 \times 10^{-6}$. Cr is a category 1 carcinogen (IARC, 1989). The mechanisms of Cr (VI) carcinogenesis are mainly through reactive oxygen species-induced oxidative stress (from the transition of Cr (VI) to Cr (III)), epigenetic change, chromosomal and DNA damage, and

mutagenesis (Ding and Shi, 2002). Although Cr (III) was not previously classified as a carcinogen, some studies have shown that high concentrations of Cr (III) can exert genetic toxicity by the production of damaging reactive oxygen species (Ateeq *et al.* 2016; Headlam and Lay, 2016), DNA damage (Wang *et al.* 2017), and interference with DNA base pairing mode (Fang *et al.* 2014). Cr (VI) exposure is linked to stomach and lung cancer (Wise and Wise, 2012).

Cd ranked second in carcinogenic risk in this study, with a CR range of 5.70×10^{-7} to 2.66×10^{-6}, which indicates that there is no cancer risk due to Cd. Cd inhibits the biosynthesis of certain proteins leading to the transformation of normal epithelial cells into carcinogenic cells (Sharma *et al.* 2015). Cd is a group 1 carcinogen known to cause lung, prostate, jugular, and pancreatic cancer (Djordjevic *et al.* 2019). It also causes lesions on the kidney and muscular layer of the heart (myocardium) and appears to be associated with renal cancer (Il'yasova and Schwartz, 2005). Long-term exposure has been linked to breast cancer (Kippler *et al.* 2012).

The CR for Pb in this study is negligible. Pb is classified as a 2B carcinogen (IARC, 1989). The most common tumours found for Pb were of the respiratory and digestive systems, although renal adenocarcinoma has been reported in workmen with prolonged occupational exposure to Pb (Baker *et al.* 1980). Pb and its compounds cause genetic damage by inhibition of DNA synthesis and repair, oxidative damage, and interaction of DNA-binding protein and tumour suppressor proteins (Roy and Rossman, 1992).

Table 3: Carcinogenic Risk (CR) of heavy metals

		Cd	**Pb**	**Cr**
A	Adults	2.66×10^{-6}	2.98×10^{-7}	4.13×10^{-5}
	Children	2.48×10^{-6}	2.78×10^{-7}	3.84×10^{-5}
B	Adults	6.13×10^{-7}	1.98×10^{-7}	3.09×10^{-5}
	Children	5.70×10^{-7}	1.84×10^{-7}	2.87×10^{-5}
C	Adults	-	8.85×10^{-8}	3.60×10^{-5}
	Children	-	8.24×10^{-8}	3.35×10^{-5}

CONCLUSION

The mean concentrations of the heavy metals were in the following decreasing order: Cu > Cr > Ni > Pb > Cd. The levels of all metals were below the maximum tolerable limit set by FAO/WHO. The exposure to the studied heavy metals also follows the same order. The study showed that there was no significant difference between the risk posed to the adult population and the children. There was also no variation between the sampling locations. The percentage contribution of the metals to the total non-carcinogenic risk was in the following order: Cr > Pb > Cu > Cd > Ni. Cr was the major contributor to the total non-carcinogenic risk and accounted for 57.61% of the risk. The investigated heavy metals did not pose non-carcinogenic risk, and there was also no carcinogenic risk concern due to the consumption of carrots in the area.

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