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Chronic Exposure to Heavy Metals in Public Water Supply and Human Health Risk Assessment

Etchie Ayotunde Titilayo • Adewuyi Gregory Olufemi • Etchie Tunde Ogbemi*

Department of Chemistry, University of Ibadan, Ibadan, Nigeria

Corresponding author: * **adventurer247@yahoo.com**

ABSTRACT

Historic disasters resulting from heavy metals contamination of drinking water emphasize the need to assess the quality of public water supplies. Currently, millions of people in and around Bangladesh are facing chronic health risk of arsenic poisoning via drinking water. Therefore, to prevent the re-occurrence of such tragic episodes, heavy metal levels in public water supplies at Agodi-residential area of Ibadan, Nigeria were determined and human health risk assessment was done for metals whose levels were higher than the US EPA maximum contaminants level for drinking purpose. Sampling was spread from January to July, 2010 to account for variations in rainy and dry seasons. 10 public taps were sampled at 5 different times, making a total of 50 water samples collected. Metals concentrations were determined by atomic absorption spectrophotometry. The mean concentrations (mg L^{-1}) of Cd, Co, Cr^{+6}, Mn and Pb in the sampled water were 0.05 ± 0.02, 0.14 ± 0.02, 0.21 ± 0.07, 0.17 ± 0.02 and 0.05 ± 0.04, respectively. These values are significantly higher than the US EPA maximum contaminants level in drinking water. Health risk assessment conducted for adult and child residents, via oral and dermal routes of exposure, gave overall hazard index values of 19 and 45, respectively, while the estimated cancer risks values were 2E-03 and 9E-04, respectively. These values significantly exceed the target non-cancer hazard index of 1 and target cancer risk of 1E-06. Hence, the concentrations of the investigating metals in this public water supply are high enough to cause carcinogenic and non-carcinogenic systemic health effects to adults and children.

Keywords: carcinogenic, evaluation, hazard index, hazard quotient, non-carcinogenic

INTRODUCTION

Chronic exposure to heavy metals in drinking water at concentrations above the US EPA maximum contaminant levels have been reported to cause systemic health effects in human (US EPA 2007). These health effects may include damage to the kidney, liver, nervous and skeletal systems, gastrointestinal distress, mental retardation in children and abortion in pregnant women (WHO 2003; WHO 2004a; WHO 2004b; UNEP 2008; US EPA 2010a; Adewuyi *et al.* 2011). Recent studies have reported Cr^{+6} in drinking water as carcinogenic (NTP 2007; Snow 2010). Currently, millions of people living in and around Bangladesh are at risk of organ dysfunction and cancer from chronic arsenic poisoning from drinking water (Saask 2003; Mahmood and Halder 2011). This is the biggest environmental disaster and a major public issue in the world (Mahmood and Ball 2004). Therefore, the principal concern for assessing and monitoring the quality of drinking water is the desire to prevent the re-occurrence of historical disasters caused by ingestion of water contaminated with heavy metals.

The objectives of this study are to determine the concentrations of heavy metals in the public water supplies at the residential areas of Agodi-Gate in Ibadan, and to assess the carcinogenic and non-carcinogenic systemic health risks that the estimated concentrations would pose on the residents who utilize the water for drinking and bathing purposes.

MATERIALS AND METHODS

Site description and sampling

This study covers Agodi-Gate residential areas of the Ibadan metropolis. The description of the Ibadan metropolis is documented elsewhere (Adekunle *et al*. 2004; Oloruntoba 2005; Ajayi *et al*. 2008; Ogedengbe and Akinbile 2010). Ten public taps receiving water directly from the Asejire Water Supply Scheme were sampled. Sampling was done 5 times, spreading from January to July, 2010, in order to obtain the average concentrations of the investigating metals and to account for changes due to seasonal variations. A total of 50 water samples were obtained.

Collection and preservation of samples

One litre of pre-cleaned polythene bottles were used to collect water samples from the taps. On the field, concentrated HNO_3 was added to fix the metals in the water sample, to a pH < 2. The plastic bottles were corked firmly and stored in the ice chest before transporting to the laboratory for analysis. On getting to the laboratory, the samples were quickly transferred to a deep freezer, in order to preserve the integrity of the samples while awaiting analysis.

Chemicals and reagents

Analytical grade reagents were used. Nitric acid (HNO_3) and the metal standards solutions were purchased from Sigma-Aldrich, Fluka, Switzerland, while the deionised water was purchased from the International Institute of Tropical Agriculture (IITA), Ibadan, Nigeria.

Heavy metals digestion methods

Nitric acid digestion of water samples was done for the metals following standard procedures (Hseu 2004; Momodu and Anyakora 2010). The method has also been described recently (Adewuyi *et al*. 2011; Etchie *et al*. 2012). To ensure the removal of organic impurities from the samples and thus prevent interference in analysis, the samples were digested with concentrated nitric acid. Five mL of the concentrated nitric acid was added to 5 mL of the water

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samples and the mixture evaporated on a hot plate to a final volume of 3 mL. Another 5 mL of concentrated HNO_3 was added to the mixture and refluxed for 30 min, after which the mixture was heated on hot plate while the concentrated HNO_3 was added until the mixture was light coloured. The resulting digests were filtered and the filtrate made up to 25 ml with deionised water before analysis.

Heavy metals analysis

The digests from the water samples were analysed for investigating metals using an atomic absorption spectrophotometer (Buck Scientific 210VGP.s). The instrument's setting and operational conditions were done in accordance with the manufacturer's specifications. The instrument was calibrated with analytical grade metal standard stock solutions (1 mg L^{-1}). A blank was run for each digestion procedure to correct the measurements and to check all reagents and procedure for interferences and cross contamination.

Risk assessment

The risk assessment method used in this study is consistent with that described in the US EPA (1989, 1991, 2004, 2007). Five metals, Cd, Co, Cr^{+6}, Mn and Pb, were identified at levels above the US EPA maximum contaminant levels and health risk was evaluated for adult and child residential exposure to the metals in the water supplies. Two exposure pathways were considered to effectively contribute to residential exposure, these are; oral ingestion; by drinking the water, and dermal contact; by bathing with the water. Risk assessment via oral route was evaluated for the metals. However, only Cd and Cr^{+6} were considered for the dermal exposure pathway, since they have been reported to contribute more than 10% of the oral exposure (US EPA 2004).

The general equations for estimating the systemic chronic health risk of non-carcinogenic and carcinogenic contaminants are as follows:

Non-carcinogenic risk equations

$$\text{Oral route equation: } HQ_O = \frac{ORI_{NC}}{RfD_o} = \frac{Cw \times IR \times EF \times ED}{RfD_o \times BW \times AT_{nc}}$$

$$\text{Dermal route equation: } HQ_D = \frac{DRI_{NC}}{RfD_o \times ABS_{GI}};$$

$$= \frac{k_p \times Cw \times t_{event} \times EV \times ED \times EF \times SA}{BW \times AT_{nc} \times RfD_o \times ABS_{GI}}$$

Carcinogenic risk equations:

$$\text{Oral route equation: } Risk_O = ORI_C \times SF_o = \frac{Cw \times IR \times EF \times ED \times SF_o}{BW \times AT_c}$$

$$\text{Dermal route equation: } Risk_D = \frac{DRI_C \times SF_o}{ABS_{GI}};$$

$$= \frac{k_p \times Cw \times t_{event} \times EV \times ED \times EF \times SA \times SF_o}{BW \times AT_c \times ABS_{GI}}$$

All the parameters have been defined in **Tables 1-4**. IR, EF, ED, EV, BW, SA, AT and t_{event} are site-specific exposure characteristics; however in the absence of site specific values, the US EPA default parameters, which march site exposure assumptions, were used. ORI_{NC} and ORI_C are estimated oral route intakes for non-carcinogenic and carcinogenic risks, while DRI_{NC} and DRI_C are the dermal route intakes for non-carcinogenic and carcinogenic risks, respectively. ABS_{GI}, SF_o, RfD_o and kp are chemical-specific and the default values are also presented in **Tables 2** and **3**, while Cw is the mean metal concentration in the municipal water supplies. Hazard quotient, HQ is the non-carcinogenic risk value for oral (HQ_I) and dermal (HQ_D) exposure routes, while $Risk_O$ and $Risk_D$ represent the carcinogenic risk values for oral and dermal routes, respectively.

RESULTS AND DISCUSSION

Table 1 shows the default parameters used in estimating the carcinogenic and non-carcinogenic risks of exposure. The cancer and non-cancer toxicity data summary, showing the target cancer risk and non-cancer hazard quotient (HQ) are presented in **Tables 2** and **3**, while **Tables 4**, **5** and **6** show the mean concentrations of metals in the water supplies (mg L^{-1}), the exposure concentrations (intake in mg kg^{-1} day^{-1}) and the calculated cancer and non-cancer risk values respectively.

From **Table 4**, the mean concentrations in mg L^{-1} of Cd, Co, Cr^{+6}, Mn and Pb in the public water supplies are 0.05 ± 0.02, 0.14 ± 0.02, 0.21 ± 0.07, 0.17 ± 0.02 and 0.05 ± 0.04 respectively. These values are significantly higher than the US EPA MCLs of 0.005, 0.005, 0.1, 0.05 and 0.015 mg L^{-1}, respectively for the metals. The order of decreasing magnitude for the metals are: Cr^{+6} > Mn > Co > Cd > Pb. This result is consistent with that obtained for drinking water in urban areas of Tigray region of Northern Ethiopia (Mebrahtu and Zerabruk 2011).

Cadmium occurs mostly in association with Zn-coated pipes and fittings (WHO 2004a; Mebrahtu and Zerabruk 2011). According to the UNEP (2008), chronic ingestion of water contaminated with cadmium above 0.005 mg L^{-1} may cause renal failure and skeletal system damage. Recently, Kumber (2011) reported that the incidence of cadmium poisoning in Toyama Perfecture in Japan resulting into the *itai itai* disease was partly caused by drinking cadmium contaminated water. The concentrations of Cd in this study (0.05 ± 0.02 mg L^{-1}) is comparable to that (0.01-0.08 mg/L) reported previously in a drinking water source in South Africa prior to an intervention programme carried out to reduce Cd contamination of the water (Fatoki and Muyima 2003; Fatoki *et al*. 2004).

Also, the mean concentration of cobalt (0.14 ± 0.02 mg L^{-1}) in this study is significantly higher than the average concentration of 0.0022 mg L^{-1} reported in drinking water sources of the United States (ATSDR 2001). However, similar mean concentration was reported in the Northern part of Nigeria (Raji *et al*. 2010). In addition to increased erythrocyte count, chronic intake of cobalt have been reported to exhibit toxic effects on the thyroid, the heart and the kidney of humans (EVM 2002).

Furthermore, the concentration of Cr^{+6} (0.21 ± 0.07 mg L^{-1}) in the water supplies appears to be the highest among all the metals studied. This is consistent with the results of Adekunle *et al*. (2004) and Ajayi *et al*. (2008), who previously recorded higher levels of chromium in drinking water in Ibadan, compared to the concentrations of the other metals studied. The levels of Cr^{+6} may have increase during the process of aeration of the raw water in the treatment facility, as aeration is done to precipitate Fe and Mn from the water and to oxidize organic compounds including microorganisms. However, aeration of water during water treatment causes the oxidation of insoluble Cr^{+3} to soluble Cr^{+6}, thus increasing the levels of Cr^{+6} in the water. Also the Asejire water treatment facility uses slaked lime to precipitate soluble metals from water. However, there is evidence that Cr^{+6} is poorly precipitated by slake lime (E.M. 2001). A recent study of a Chinese population exposed to Cr^{+6} in drinking water provided evidence of an increased risk of stomach cancer (Beaumont *et al*. 2008; Smith and Steinmaus 2009; NJDHSS and NJDEP 2010).

The concentrations of manganese (0.17 ± 0.02 mg L^{-1}) n the water supplies is high enough to pose chronic health risk, as Mn has been known to cause neurological disorders in humans (NIS 2007). Epidemiological study of chronic exposure to naturally occurring manganese in drinking water found correlations between Mn intake and neurological effects in elderly persons and children (WHO 2004b; Ljung *et al*. 2007). Pb concentrations (0.05 ± 0.04 mg L^{-1}) in the water supplies exceed the USEPA maximum concentration levels (0.015 mg L^{-1}) in drinking water. This indicates that the consumers are exposed to a high concentra-

Table 1 USEPA default parameters used for oral and dermal exposure assessment.

Parameters	Receptor	Value	Exposure route
IR, Ingestion Rate	Adult	2 L day^{-1}	Oral
	Child	1 L day^{-1}	Oral
ED, Exposure Duration	Adult	30 years	Oral, Dermal
	Child	6 years	Oral, Dermal
EF, Exposure Frequency	Resident	350 days $year^{-1}$	Oral, Dermal
BW, Body Weight	Adult	70 kg	Oral, Dermal
	Child	15 kg	Oral, Dermal
Cancer Averaging Time	Resident	25550 days	Oral, Dermal
Non cancer Averaging Time	Resident	ED × 365 days $year^{-1}$	Oral, Dermal
EV, Event Freqency	Resident	1 event day^{-1}	Dermal
$t_{event,}$ Event Duration	Adult	0.58 h	Dermal
	Child	1 h	Dermal
SA, Skin Surface Area	Adult	18,000 cm^2	Dermal
	Child	6,600 cm^2	Dermal
Kp, Skin Permeability Coefficient; Cd	-	1E-03 cm h^{-1}	Dermal
Cr^{+6}	-	2E-03 cm h^{-1}	Dermal

Reference: USEPA (2004, 2006).

Table 2 Cancer toxicity data summary. Pathway: Oral Ingestion, Dermal.

Chemical of Concern	$SF_{o,}$ (mg $kg^{-1}day^{-1})^{-1}$	$SF_d = SF_o/ABS_{GI}$ (mg $kg^{-1}day^{-1})^{-1}$	ABS_{GI} (%)	Target Cancer Risk	Reference
Cr^{+6}	5E-01	2E+01	2.5	1E-06	USEPA (2004, 2010b)

SF_o= Oral cancer slope factor; SF_d = Dermal cancer slope factor; ABS_{GI} = Gastrointestinal tract absorption fraction.

Table 3 Non-cancer toxicity data summary.

Chemical of concern	RfD_o (mg $kg^{-1}day^{-1}$)	$RfD_d = RfD_o \times ABS_{GI}$ (mg $kg^{-1}day^{-1}$)	ABS_{GI} (%)	Target Hazard Quotient, HQ	Exposure Pathway
Cd	5E-04	2.5E-05	5	1	Oral, Dermal
Cr^{+6}	3E-03	7.5E-05	2.5	1	Oral, Dermal
Co	3E-04	-	-	1	Dermal
Mn	2.4E-02	-	-	1	Dermal

RfD_o = Oral reference dose; RfD_d = Dermal reference dose; ABS_{GI} = Gastrointestinal tract absorption fraction; References: USEPA (2004, 2010b).

Table 4 Mean concentrations of heavy metals in water supply and USEPA maximum contaminant levels.

Concentrations	Heavy metals				
(mg L^{-1})	Cd	Co	Cr^{+6}	Mn	Pb
Cw	0.05±0.02	0.14±0.02	0.21±0.07	0.17±0.02	0.05 ± 0.04
MCL	0.005	0.005	0.05	0.05	0.015

Cw = Mean heavy metal concentration in water supply; MCL = USEPA Maximum contaminant level; Reference: USEPA (2010).

Table 5 Estimated exposure concentrations (intake, mg $kg^{-1}day^{-1}$) of heavy metals

Receptor	Metal	Oral Route Intake		Dermal Route Intake		Overall Exposure Concentration
		ORI_{NC}	ORI_C	DRI_{NC}	DRI_C	
Adult	Cd	1.4E-03	-	3.1E-06	-	1.4E-03
	Co	3.8E-03	-	-	-	3.8E-03
	Cr	5.8E-03	2.5E-03	2.6E-05	1.1E-05	5.8E-03
	Mn	4.7E-03	-	-	-	4.7E-03
	Pb	1.4E-03	-	-	-	1.4E-03
Child	Cd	3.2E-03	-	5.3E-06	-	3.2E-03
	Co	8.9E-03	-	-	-	8.9E-03
	Cr	1.3E-02	1.2E-03	4.4E-05	3.8E-06	1.3E-02
	Mn	1.1E-02	-	-	-	1.1E-02
	Pb	3.2E-03	-	-	-	3.2E-03

ORI_{NC} = Calculated oral route intake for non-carcinogenic risk; ORI_C = Calculated oral route intake for carcinogenic risk; DRI_{NC} and DRI_C = Calculated dermal route intake for non-carcinogenic and carcinogenic risks respectively

Table 6 Calculated cancer and non-cancer risk values.

Receptor	Metal	HQ_O	HQ_D	HI	HI_T	$Risk_O$	$Risk_D$	$Risk_T$
Adult	Cd	2.9	0.3	3.2		-	-	-
	Co	13.0	-	13.0		-	-	-
	Cr^{+6}	1.9	0.8	2.7		1E-03	5E-04	1.5E-03
	Mn	0.2	-	0.2	19.1	-	-	-
Child	Cd	6.8	0.9	7.7		-	-	-
	Co	30.1	-	30.1	-	-	-	-
	Cr^{+6}	4.5	2.0	6.5		6E-04	3E-04	9E-04
	Mn	0.5	-	0.5	44.8	-	-	-

HQ_O & HQ_D = Calculated hazard quotient for oral ingestion and dermal exposure respectively; HI, Hazard index = (HQ_O + HQ_D); HI_T = Total hazard index; $Risk_O$, $Risk_D$ & $Risk_T$ = Estimated cancer risk for oral and dermal exposure routes, and total cancer risk for both routes respectively.

tion of Pb. Several studies have also reported high Pb concentrations in drinking water sources of the Ibadan metropolis (Adekunle *et al*. 2004; Ajayi *et al*. 2008; Olusegun 2010). Pb is an element of major concern due to its many industrial and domestic uses; the presence of tetraethyl Pb in gasoline and the largely uncontrolled dumping of Pb-

containing residues, have severely contaminated soil and water supplies in many parts of Nigeria (Alatise and Schrauzer 2010; Adewuyi *et al*. 2011). Consequently, a large segment of the population of Nigeria is chronically exposed to Pb (Alatise and Schrauzer 2010). In a study conducted in the South-Western Nigeria, 70% of children aged 6 to 35 months, had blood Pb levels in excess of 10 µg dL^{-1}, while occupationally Pb exposed auto-mechanics exhibited mean blood and hair Pb levels of 49 ± 9 µg dL^{-1} and 18 ± 5 µg g^{-1} respectively (Alatise and Schrauzer 2010).

The result of **Table 5** shows that the adults and children are exposed to considerable amount of Cd, Co, Cr^{+6}, Mn and Pb in the water supplies. Adults' exposure (mg $kg^{-1}d^{-1}$) to Cd, Co, Cr^{+6}, Mn and Pb are 1E-03, 4E-03, 6E-03, 5E-03 and 1E-03, respectively, whereas children exposure are 3E-03, 9E-03, 1E-02, 1E-02 and 3E-03 mg $kg^{-1}d^{-1}$, respectively. Adults and children exposures to the metals are in the decreasing order of magnitude, Cr^{+6} > Mn > Co > Cd ≥ Pb. There is evidence that ingested Cd, Cr, and Pb, when absorbed interact *in vivo* with selenium (Se), a nutritionally essential trace element whose anti-carcinogenic properties have been demonstrated in numerous animal tumor model systems (Alatise and Schrauzer 2010). The interactions are part of natural metal detoxification processes, but result in the metabolic inactivation of Se, and at sufficiently high exposure levels may over time produce a state akin to Se deficiency (Alatise and Schrauzer 2010). Also, absorbed Cr^{+6} is converted to the Cr^{+3} in cells and forms tightly bound adducts with DNA and proteins, thus initiating the formation of cancer (Williams *et al*. 2000). Mn occurs in manganese superoxide dismutase (MnSOD), which exists in several genotypes of which some are believed to cause oxidative damage to DNA and increase breast cancer risk (Alatise and Schrauzer 2010).

The high levels of heavy metals detected in drinking water of residential areas have led risk assessors from different parts of the world to estimate risk of carcinogenic and non-carcinogenic health effects from oral and/or dermal exposure routes using the US EPA risk assessment methodology (Fatoki *et al*. 2004; Sadiq *et al*. 2004; Momot and Synzynys 2005; Ni *et al*. 2009; Nguyen *et al*. 2009; Zeng *et al*. 2009; Li and Qian 2011; Etchie *et al*. 2012). Risk assessment is the estimation of the frequency and consequences of undesirable events which can produce harm (Sadiq *et al*. 2004). It is used to predicting adverse health effects due to exposure to pollutants in environmental media.

A pollutant is regarded as safe or acceptable; when the non-cancer HQ and cancer risk values are less than 1 and 1E-06 respectively (US EPA 2010b). Conversely, an exposed population of concern will experience health risks if the dose is equal to or greater than these values respectively (Zeng *et al*. 2009). The estimated Cd HQ_O and HQ_D for adult residents are 2.9 and 0.3, respectively (**Table 6**). While the HQ_O is greater than the acceptable or target HQ of 1, the HQ_D is lower than the target HQ. However, the overall Hazard Index, HI for adult exposure to Cd, which is the summation of HQ_O and HQ_D is 3.2, this value is 3 times higher than the target HI of 1. Also, the HQ_O and HQ_D for child exposure are 6.8 and 0.9, respectively. Again, the HQ_O is higher than the target HQ, while the HQ_D is lower. These values are greater than the HQs of adult exposure to Cd. Furthermore, the estimated HI for the child exposure is 7.7, and is 7 times the target HI and 2 times the adult HI. In comparison, the HI values for the adult and child residential exposures estimated in this study are by far greater than the risk values reported for Cd via drinking water in a rural community in South Africa (Fatoki *et al*. 2004) and in Hunan Province, China (Sun *et al*. 2010).

Also, the estimated HQ_O and HQ_D values for adult exposure to Cr^{+6} are 1.9 and 0.8 respectively. The HQ_O is greater than the target HQ, while the HQ_D is lower. The overall HI for adults' exposure to Cr^{+6} via both routes is 2.7, which is about 3 times the target HI. Likewise, the HQ_O and HQ_D for the child exposure are 4.5 and 2.0, respectively. Again these values are significantly greater than those obtained for the adult residents and the acceptable HQ value. Therefore, the overall HI for child exposure is 5.5, via both routes, and is about 2 times that of adult' and 6 times the target HI.

The estimated HQ_O values for adult and child exposure to Co are 13.0 and 30.1, respectively, while Mn HQ values are 0.2 and 0.5, respectively. The HQ_O values for Co for the two receptors are greater than the target HQ, whereas the Mn HQ_O values for both receptors are lower than the target HQ. Thus, Co poses significant non-carcinogenic health risks to the residents but Mn does not. Note that the HQ_D was not estimated for Co and Mn because these metals do not contribute significantly through the dermal exposure route (US EPA 2004).

The noncarcinogenic risk of Pb was not estimated, since there is no reported oral reference dose for Pb. However, the risk of Pb was assess base on comparison of the intake values with the provisional tolerable daily intake (PTDI) of 3.5E-03 mg $kg^{-1}day^{-1}$ reported by the Joint FAO/WHO Expert Committee on Food Additives (Sun *et al*. 2010). Hence Pb intake by an adult receptor is over 2.5 times lower the PTDI, while the child receptor is close to the PTDI value. However, bearing in mind that this risk is an incremental estimate and exposures to Pb from other sources have not been added, the result is still fearful, especially when there is previous report that over 300 children under the ages of 5 years have been confirmed dead as a result of Pb poisoning in the northern part of Nigeria (Punch Newspaper 2010). Furthermore, a study has shown that breast cancer among Nigerian females is directly linked to Pb exposure (Alatise and Schrauzer 2010).

The result of the non-carcinogenic risk shows that children are by far more prone to the toxic effects of the metals than adults. This trend has been observed before (Zeng *et al*. 2009) and was attributed to the sensitive nervous system, low bile excretion and high gastrointestinal absorption of children (Ljung *et al*. 2007; Zeng *et al*. 2009).

Furthermore, of all the metals studied, only Cr^{+6} has toxicity values for estimating cancer risk of exposure (US EPA 2010b). Recent studies have documented carcinogenic risk via drinking water contaminated with Cr^{+6} (NTP 2007; Snow 2010). In addition, the US EPA (2004) has reported that the dermal route of exposure could be considered for cancer risk estimation if there is evidence of carcinogenic risk through the oral route. However, the carcinogenicity of the other metals via oral ingestion pathway is still inconclusive (US EPA 2007).

From the result (**Table 6**), the $Risk_O$ and $Risk_D$ reported for Cr^{+6} are 1E-03 and 5E-04, respectively for adult residents and 6E-04 and 3E-04 for child residents. The overall cancer risks $Risk_T$ for the adult and child residents via oral and dermal exposure routes are 2E-03 and 9E-04 respectively. Again, these values greatly exceed the target risk value of 1E-06 set by the US EPA (2010b). This implies that about 2 adults in 1,000 and 9 children in 10,000 may suffer from cancer as a result of intake of Cr^{+6} from the water supplies. This is similar to the report of Nguyen *et al*. (2009) who documented cancer risk of 4 people in 10,000 and 50 people in 10,000 on consumption of treated and untreated groundwater, respectively in Vietnam. Also, a recent study (EWG 2011) reported that at least 74 million Americans in 42 states are drinking tap water polluted with Cr^{+6}, even though the US EPA analysis of the metal's toxicity cited significant cancer concern linked to exposure via drinking. Another report proposed that 6 to 331 persons in each million may die per year in Northwest China as a result of drinking water contaminated with carcinogens (Li and Qian 2011). Likewise, a recent study in Nigeria proposed that 4 adults and 2 children in every 1000 are at risk of cancer as a result of Cr^{+6} intake from groundwater (Etchie *et al*. 2012)

In addition, unlike the non-cancer risk, the cancer risk result revealed that adults are at more risk of getting cancer than children. This inference disagrees with that of Obiri *et al.* (2010), who proposed that children are at more cancer risk than adults as a result of exposure to carcinogenic

metals in borehole water. However, considering that the cancer risk is averaged over 70 years with exposure duration of 30 years for adults and 6 years for children, the probability of a child getting cancer because of exposure in 6 years should be less than that of an adult getting cancer over 30 years (Etchie *et al*. 2012). More so, cancer cases in children are rare (WHO/ICO 2010; Etchie *et al*. 2012).

CONCLUSION

The result of the analysis of water samples collected from the public water supplies of the Agodi-Gate residential areas of the Ibadan metropolis has revealed high concentrations of Cd, Co, Cr^{+6}, Mn and Pb in the water samples and health risk assessment has shown that there are potential cancer and non-cancer systemic health risks for the water consumers, as long as they continue to utilize the groundwater for their domestic purposes. In order to prevent this menace that may surface in the nearest future, it is recommended that appropriate water treatment processes that can effectively remove these metals be adopted. Also, general maintenance such as frequent washing and cleaning of the water treatment compartments at reasonable intervals and assessment of levels of trace metals in each batch of treated water before distribution will help improve the quality of the water. Furthermore, routine assessment of metals levels in supplies at supply terminals will go a long way in checking re-contamination in pipes and storage tanks

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