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Evaluation of lead and mercury neurotoxic health risk by resident children in the Obuasi municipality, Ghana

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ABSTRACT

This study assesses neurotoxic effects associated with exposure to lead and mercury in borehole, tap and surface water by resident children in the Obuasi municipality in accordance with USEPA risk assessment guidelines. From the results of the study, the hazard quotient for oral ingestion of mercury in tap water in Obuasi is 7.4 and 15 respectively via both central tendency exposure (CTE) and reasonable maximum exposure (RME) parameters, respectively. This means that approximately 7 and 15 (by both CTE and RME parameters, respectively) resident children in Obuasi are likely to show neurologic effects associated with exposure to mercury and lead such as increased nervousness, loss of memory and/or decrease in concentration, impaired writing ability and tremor.

1. Introduction

Gold mining in Ghana has played a significant role in the socio-economic and socio-political life of the nation for the past 200 years. However, gold mining in recent times has become unpopular as it is regarded as a significant source of Hg, Pb and other heavy metal contamination of the environment. This is due to activities such as mineral exploitation, ore transportation, smelting and refining, disposal of the tailings and mine waste into rivers/streams (Adriano, 2001).

In the study area, large deposits of mine wastes, ore stockpiles and waste rocks have become heap of mountains around the plants. Weathering of the heaped waste materials result in the release of toxic chemicals into the environment especially, into aquatic bodies. Harmful metals released from mine tailings include mercury, arsenic, lead, cadmium, etc. Workers at the processing and smelting sections who inhale the vapours of mercury oxides and lead oxides will have their central nervous system affected. Moreover, people (especially resident children) living in and around the mining communities inhale fumes of oxides of mercury and lead just to mention a few (Davies and Ballinger, 1990; Merrington and Alloway, 1994; Amonoo-Neizer and Amekor, 1994; Obiri, 2007).

According to a study conducted by Taayeli (2008) to measure trends of some indicators of internal efficiency in public junior secondary schools in the Obuasi municipality, he found out that repetition and dropouts' rates among students of junior secondary schools in the municipality were high. This, he attributed to exposure to high levels of lead and mercury in soil, water and food crops in the study area which have serious implication on intellectual abilities of resident children in the study area.

Mercury and lead toxicities are particularly severe to a developing brain and exposures during pregnancy are therefore most hazardous (USEPA, 1997; IPCS, 1990). Cognitive deficits have recently been reported in children in Brazil with known exposures to high levels of neurotoxicants such as mercury and lead (Grandjean et al., 1997). Determining the sources (potential pathways of exposure) of neurotoxic metals in water bodies in mining communities is important for mitigating impacts aimed at reducing concentrations and limiting human exposure. However, no work has been carried out in this regard in Ghana. There is therefore the need to accurately quantify neurotoxicological risk face by children in the contaminated environments.

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Table 1
Exposure factors for a resident Ghanaian child in Obuasi municipality, Ghana.

Factor/parameter	Symbol	Units	Exposure scenario	Data source
Exposure duration	ED	20 years	Residential setting	USEPA (2008) USEPA (1997)
Exposure frequency	EF	365 days/years	Residential setting	USEPA (2008) USEPA (1997)
Water ingestion rate	IR_w	L/day	Residential setting	USEPA (2008) USEPA (1997)
Body weight	BW	13.5 kg	Residential setting	GLSS (2003)
Contaminant concentration	C	mg/kg	Residential setting	Refer to Table 2 for the analytical concentration of Pb and Hg in the samples
Averaging time	AT	225 days	Residential setting	USEPA (2008)

The main thrust of the study is to evaluate neurotoxicological health risks faced by resident children from exposure to mercury and lead, and their effects on their cognitive development.

2. Methodology

2.1. Sampling techniques and sample collection

Random sampling techniques were adopted in selecting the sample sites as outlined by USEPA. The samples were collected between March and June 2008. In all, 60 samples were collected within this period. The sample containers were washed with detergent and rinsed with 1:1 nitric acid and double distilled water. 1.5 L of the water samples were collected from each sampling point into sampling bottles. During sampling, the containers were rinsed several times with the sample before the final sample was taken. The samples were acidified with 10% nitric acid, stored in an ice-chest at 4 °C and were conveyed to the laboratory for analysis. Samples collected for each month were analyzed separately for mercury and lead and the mean results for lead and mercury were computed at the end of study (AWWA, 1998).

2.2. Analysis of lead and mercury

For the determination of lead, 100 mL of the acidified sample were mixed with 5 mL each of conc. H_2SO_4 and conc. HNO_3. The mixture was heated until the volume was reduced to about 15–20 mL on a hot plate. The digested samples were allowed to cool to room temperature and then filtered through a 0.45 μm Whatman filter paper. The final volume was adjusted to 100 mL with double distilled water and stored for analysis (AWWA, 1998). For the determination of Hg, to 100 mL of the sample, 5 mL of conc. H_2SO_4 and 2.5 mL of conc. HNO_3 were added after which the mixture was shaken thoroughly. Additional 15 mL of 5% (w/w) $KMnO_4$ and 8 mL of 5% (w/w) potassium perusulphate were also added to the mixture. The sample was then heated at 90 °C for 2 h. It was allowed to cool to room temperature and 6 mL of 12% (w/w) hydroxylamine hydrochloride were added to the resulting solution to reduce the excess permanganate. The digested solution was stored for analysis (AWWA, 1998).

The concentration of Pb was determined using flame Atomic Absorption Spectrophotometer (AAS) Shimadzu model 6401F. In the determination of Hg, a carrier solution containing 3% (v/v) HCl and a reducing agent 1.1% (m/v) $SnCl_2$ in 3% (v/v) HCl was added to digested sample to generate mercury vapour which was determined by cold vapour AAS Shimadzu model 6401F. The detection limit for lead and mercury is 0.01 mg/L and 0.01 μg/L, respectively.

2.3. Quality control

Reproducibility and recovery studies were conducted. The percentage of lead and mercury recovered in the recovery studies are 95% and 100% for lead and mercury, respectively. Similar results were obtained for the reproducibility studies. The percentage of mercury and lead recovered in the reproducibility studies ranges from 96.3% to 99.6% (standard error ±0.005–0.560). The standard error is less than 1, suggesting that the method employed to analyze lead and mercury is reproducible.

2.4. Theory/calculation

2.4.1. Risk assessment process

Risk assessment is defined as the process of estimating the probability of the occurrence of an event and the probable magnitude of adverse health effects on human exposures to environmental hazards (Kollunu et al., 1996; Paustenbach, 2002; NRC, 1983). It consists of four interactive steps, namely: the hazard identification, exposure assessment, toxicity assessment and risk characterization. This study assessed neurotoxicological health risk to a resident child in the affected area and who is exposed to Hg and Pb present in contaminated waters. The hazard identification process was accomplished through field sampling of contaminated waters, and the subsequent determination of the contaminant levels for Hg and Pb in these samples.

2.4.2. Exposure assessment

The exposure assessment identifies the pathways by which humans are potentially exposed to the toxicants and estimates the magnitude, frequency and duration of these actual and/or potential exposures. In this study, the exposure assessment involved the analysis contaminant releases, identifying exposed populations, identification all potential pathways of exposure, estimating exposure point concentrations for specific pathways and estimating contaminant intakes for specific pathways. The average daily dose (ADD) of the contaminant via the identified pathways (i.e. drinking water pathway) is the quantity of Pb and Hg ingested per kilogram of body weight per day (Kollunu et al., 1996; Paustenbach, 2002) that resident children in the study area are exposed to. In calculating the average daily dose, Eq. (1) below was used:

$$\text{ADD} = \frac{C \times \text{IR} \times \text{ED} \times \text{EF}}{\text{BW} \times \text{AT} \times 365} \quad (1)$$

where C is the concentration of the contaminant in the environmental media (mg/kg or mg/L), IR is the ingestion rate per unit time (mg/day or L/day), ED is the exposure duration (years), EF is the exposure frequency (days/year), BW is the body weight of the receptor (kg), and AT is the averaging time (years), equal to the life expectancy for non carcinogen, and 365 is the conversion factor from year to days. The values of C, IR, ED, EF, BW and AT which were used in Eq. (1) above have been presented in Table 1 below. With the exception of BW, the rest were default values in human health risk assessment computerized software, RISC version 4.02 developed by USEPA for the Superfund sites.

The exposure scenario evaluated in this study is a residential setting. In this scenario, ingestion of tap, borehole and surface water in the study area by resident children was evaluated based on both central tendency exposure (CTE) and reasonable maximum exposure (RME) parameters, respectively. The CTE exposure parameters were used so that health risks associated with typical or average exposures to the constituents of concern (COC's) can be calculated based on the 50th percentile of the mean concentration of the COCs. RME parameters were also used to calculate the health risks associated with high-end exposures also based on 95th percentile of the mean concentration of the COCs. The potential receptors evaluated in this study are resident children aged between 2 and 19 years.

2.4.3. Toxicity assessment

In order to evaluate neurotoxic non-carcinogenic health risk associated with exposure to lead and mercury by resident children in the study area, toxicity assessment was carried out using oral reference dose from Integrated Risk Information Systems (IRIS) database. Default values from RISC 4.02 human health evaluation software were used. The basis for these values has been described in detail in the USEPA Child-Specific Exposure Factors Handbook (USEPA, 2008). The oral reference dose (RfD) is based on the assumption that thresholds exist for certain toxic effects such as cellular necrosis or tremor. It is expressed in units of mg/kg/day. In general, the RfD is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime.

2.4.4. Risk characterization

Neurotoxic risks refer to the non-carcinogenic harm done to the central nervous systems due to exposures to neurotoxic chemicals. The extent of the harm incurred is expressed in terms of hazard quotient as shown in Eq. (2) below:

$$\text{Hazard quotient (HQ)} = \frac{\text{ADD}}{\text{RfD}} \quad (2)$$

where, RfD_{oral} is the oral reference dose. The reference dose is the daily dosage that enables the exposed individual to sustain this level of exposure over a prolonged time period without experiencing any harmful effect. In accordance with USEPA risk assessment guidelines, hazard quotients are calculated for each receptor and exposure route, and then summed across the different exposure routes to calculate

Table 2
Mean concentrations of mercury and lead in the samples.

Sample location	Concentration of mercury (μg/L)	Concentration of lead (mg/L)
Borehole at Odumasi	0.020	0.120
Tap water at Odumasi	0.054	0.044
Tap water at Obuasi	0.692	0.021
River Kaw	0.020	2.47
River Kwabrafo	0.020	15.2
River Pompo	0.020	13.3
River Akapori	0.020	2.27
River Jimi	0.054	1.05
River Akyerempe	0.982	0.059
River Supu	0.894	2.27

Table 3
Descriptive statistical analysis on the concentrations of lead and mercury.

Metal concentration	*N*	Mean	Standard deviation
Mercury (μg/L)	10	0.2776	0.40547
Lead (mg/L)	10	3.6804	5.67652

the hazard index. Hazard index (HI) indicates whether the estimated exposures for individual present a potentially significant non-cancer neurotoxic health risk based on comparison to a USEPA recommended RfD_{oral}. If the HI is less than 1.0, then the neurotoxic non-carcinogenic adverse effect due to this exposure pathway or chemical is assumed to be negligible. In this study, the value of the oral reference dose for mercury is 3×10^{-4} mg/kg/day whilst that of lead is 6.0×10^{-2} mg/kg/day.

3. Results and discussion

3.1. Contamination levels for lead and mercury

The mean concentrations of lead and mercury in tap, borehole and surface water in the study area have been presented in Table 2 below (refer to Appendix A for the monthly results).

The results in Table 2 above were used as input parameters in calculating the average daily dose (ADD) that resident children in the study area ingested. Comparing the results of the mean concentrations of Pb and Hg in the water samples in the study area, it is clear that the mean concentrations of Hg and Pb in the water samples exceeds WHO standards. The WHO permissible guideline values for Pb and Hg are 0.01 μg/L and 0.05 mg/L, respectively. Thus it poses significant health hazards to residents who use it. Hence regular consumption of water from these sources by resident children could pose serious neurological health problem from a long-term Hg and Pb exposure in the vicinity of the mine which would eventually affect their academic performance. The results of descriptive statistical analysis on the concentrations of lead and mercury as well as *t*-test for equality of means for the concentrations of lead and mercury have been presented in Tables 3 and 4 below, respectively.

Table 4
t-Test for equality of means for metal concentrations.

t-Test for equality of means			
T	d.f.	*p*-value	Mean difference
−1.891	18	0.075	−3.40280

The mean concentration of mercury in μg/L is 0.278 whilst the mean concentration of lead in mg/L is 3.680. These values are greater than the WHO permissible value of 0.05 and 0.01, respectively. From Table 3, there is variability in the concentration of mercury but this variability is very small (standard deviation = 0.40547). In contrast with variability in the concentration of lead which is rather high (standard deviation = 5.67652). From Table 4 above, the *t*-test for equality of means the *p*-value was found to be 0.075 which is greater than an α-value of 0.05. This implies that there is no significant difference in the average concentrations of mercury and lead. Even though, differences may exist as can be seen from the standard deviations, statistically these differences are not significant at 5% level of significance.

3.2. Evaluation of neurotoxic health risk

Neurotoxic (non-carcinogenic) health risks are expressed in terms of hazard quotients. The results of hazard quotients from exposure to lead and mercury from ingestion of contaminated tap, borehole and surface water by resident children in Obuasi municipality has been presented in Table 5 below.

From Table 5 above, the hazard quotient values recorded in this study for mercury were found to be greater than 1.0. Therefore, there is high toxic risk associated with ingestion of mercury in drinking water by resident children. The HQ values via CTE exposure parameters ranges from 2.1 to 100 for the borehole at Odumasi to River Akyerempe whilst that of RME parameters ranges from 4.3 to 210 for the borehole at Odumasi to River Akyerempe. Similarly, the results of HQ values for exposure to lead by resident children via CTE parameters ranges from 0.19 to 130 i.e. for tap water at Obuasi to River Kwabrafo whilst that of RME parameters ranges from 0.37 to 270, respectively.

Comparing the neurotoxic risk results expressed in terms of both hazard quotients and hazard indexes for exposure to mercury to that of lead reveals that resident children in the study area face significant neurotoxic risks from exposure to lead than mercury. This is due to the fact that there are multiple exposures to lead by the resident children in the study area. For example, the lead in the study area includes those accumulated from vehicular fallout of vehicles that run on leaded fuel and rampant discharge of mine waste which contains elevated levels of lead. According to US Center for Disease Control (CDC) very low concentrations of lead poses serious public health hazard to sensitive populations such as

Table 5
Hazard quotient (HQ) and Hazard index (HI) for exposure to Hg and Pb by resident children in Obuasi municipality.

Sampling location	Hazard quotients (HQ)				Hazard indexes (HI)	
	Mercury		Lead		Mercury	Lead
	CTE	RME	CTE	RME		
Borehole at Odumasi	2.1	4.3	1.1	2.1	2.22	1.11
Tap water at Odumasi	5.8	12	0.39	0.78	5.75	0.76
Tap water at Obuasi	7.4	15	0.19	0.37	7.37	0.186
River Kaw	2.1	4.3	22	44	2.13	21.9
River Kwabrafo	2.1	4.3	130	270	2.13	135
River Pompo	2.1	4.3	120	240	2.13	118
River Akapori	2.1	4.3	20	40	2.13	202
River Jimi	5.8	12	9.3	19	5.75	9.32
River Akyerempe	100	210	0.52	1.0	105	0.524
River Supu	2.2	4.4	1.1	2.2	95.3	20.2

infants, children, and pregnant women (as surrogates for foetuses). This work confirms the observations of CDC.

Mercury(II) ions ingested by resident children in drinking water, inhibits enzymes and proteins, especially those of the thiol groups, typical of zinc and copper binding sites, to produce stable methylmercury derivatives. The methylmercury derivatives due to its high lipid solubility are able to cross biological membranes more easily and enter the brain, spinal cord and the peripheral nerves as well as the placenta. For this reason, even though the mother may not show symptoms of mercury poisoning, cerebral palsy may result in the foetus in the uterus through mercury intoxication and this may affect the cognitive development of their unborn children (Weed et al., 1962).

4. Conclusion

This study assessed neurotoxic health risk associated with exposure to lead and mercury in drinking water bodies via oral ingestion by resident children in the Obuasi municipality, Ghana.

The outcomes of the risk assessment show that the neurotoxic health risk (HI) in most cases exceeded the USEPA acceptable value of 1.0. This means that exposure to lead and mercury in drinking water is likely to affect their cognitive development. As exposures to even very low levels of lead and mercury produce the following adverse effects:

- Impairments of central nervous system (CNS) and other organ development in foetuses.
- Impairments in cognitive function and initiation of various behavioural disorders in young children.

According to Taayeli (2008) there are high repetition and dropout rates in all the schools in the study area. Their repetition rate is 25% whilst the dropout rate is 12% compared to the national average of 12% repetition rate and 5% dropout rate (GLSS, 2003). It is likely that exposure to the high levels of lead and mercury in their drinking water is contributing to their low academic performance.

From the results of the study, it is likely that the ingestion of elevated levels of neurotoxic metals i.e. lead and mercury by resident children in Obuasi municipality is likely to have serious effect on their cognitive development which is also affecting their academic performance. Hence, a similar study should be conducted to determine the concentrations of lead and mercury in blood plasma of resident children in the study area. The concentrations of mercury and lead in the blood plasma of resident children in the study should be used as input parameter to evaluate the neurotoxic health risk associated with the ingestion of mercury and lead in their drinking water.

Conflict of interest statement

I write on behalf of my co-authors to state that in the design, execution of the study as well as the preparation of this manuscript that there was no conflict of interest.

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Appendix A. Monthly concentrations of lead and mercury in water samples in the study area

Sample location	March		April		May		June		Mean	
	Hg	Pb	Hg	Pb	Hg	Pb	Hg	Pb	Hg	Pb
Borehole at Odumasi	0.020	0.120	0.019	0.0118	0.019	0.124	0.021	0.121	0.020	0.120
Tap water at Odumasi	0.052	0.044	0.052	0.040	0.051	0.046	0.058	0.048	0.054	0.044
Tap water at Obuasi	0.692	0.021	0.690	0.0118	0.696	0.021	0.688	0.028	0.692	0.021
River Kaw	0.020	2.47	0.019	2.35	0.019	2.57	0.021	2.49	0.020	2.47
River Kwabrafo	0.020	15.2	0.021	14.6	0.019	15.5	0.019	15.6	0.020	15.2
River Pompo	0.021	13.3	0.018	12.6	0.020	13.8	0.022	13.4	0.020	13.3
River Akapori	0.020	1.17	0.019	2.27	0.019	3.35	0.021	2.29	0.020	2.27
River Jimi	0.056	1.15	0.055	1.07	0.052	1.05	0.052	0.95	0.054	1.05
River Akyerempe	1.002	0.059	0.982	0.070	0.965	0.052	0.980	0.055	0.982	0.059
River Supu	0.902	2.27	0.894	1.17	0.898	3.35	0.876	2.29	0.894	2.27

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