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Non Occupational Health Risk Assessment from Exposure to Chemical Contaminants in the Gold Mining Environment of Tarkwa, Ghana

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ABSTRACT

This study is part of a larger on-going environmental monitoring and assessment of water, sediment and biota in the Tarkwa Nsuaem Gold mining area. Assessment of risks is central to risk mitigation and reduction of human health impacts. The study set out to assess health risks via the Central Tendency Exposure (CTE) and Reasonable Maximum Exposure (RME) scenarios to resident adults and children from exposure to arsenic, manganese and lead in surface and groundwater. The results indicate that hazard quotients for these pollutants are mostly within range of the United States Environment Protection Agency (USEPA) acceptable risk. The RME of Mn through oral contact for Children (hazard quotient of 18) is considerably higher than the USEPA acceptable risk value of 1.0. While at some locations, the RME of Mn through oral contact for adults also exceeded the standard, a comparison of contributions of oral and dermal contact to the health risk, shows that the former accounts for more. Carcinogenic risks to resident adults arising from exposure to arsenic show RME values ranging from 5.0×10^{-4} to 1.1×10^{-3}. These values were up to 11 times greater than the USEPA acceptable range for excess risk of cancer. Arsenic-related cancer risks to resident adults for CTE ranged from 3.7×10^{-4} to 6.7×10^{-4}; also higher than the USEPA acceptable range for excess risk of cancer. Likewise, the cancer risks to resident children for both CTE and RME scenarios were greater than the USEPA acceptable range for excess risk of cancer. These values were up to 210 times greater than the USEPA acceptable range for excess risk of cancer. Consequently, it is imperative to implement a strategy to reduce exposure to carcinogenic compounds in the area in order mitigate cancer and non-cancer risks within this mining area.

Key words: Environmental risk, trace metals, children, adults, gold mining, contamination

INTRODUCTION

The exposure to some heavy metals has been associated with a wide variety of adverse health effects, including cancer (Adeyemi *et al*., 2007; Ghanem and Ghannam, 2010). Although some elements are essential for humans, they can be dangerous at relatively high exposure levels (Domingo, 1994; Goorzadi *et al*., 2009). Health impacts associated with ingestion of trace metals such as arsenic (As), leads (Pb), manganese (Mn), are many and well documented (Picado *et al*., 2010; Babaei *et al*., 2006). In order to assess risks arising from ingestion of these trace metals, their

fate and transport in the physical environment must be understood (Korre *et al.*, 2007; Ansari *et al.*, 2004; Ekosse, 2008). It is important to understand not only the physiological characteristics of individual organisms but also their food habits, behavioural patterns and habitat requirements (Rikken and Lijzen, 2004; Hacon *et al.*, 2010). These factors all have important effects on the exposure of individual organisms to these trace metals and the associated risk of this exposure (Hacon *et al.*, 2010; Ndimele *et al.*, 2011). The chemical analysis of pollutant concentrations in different environmental compartments (i.e., air, soil, vegetation, water, sediments) may be a significant indirect methodology for human health risk assessment. Human exposure may be considered to occur through two routes: direct and indirect. Direct exposure is the sum of exposure to pollutants by direct pathways, such as inhalation, dermal absorption or water ingestion (USEPA, 1989). Sequentially, pollutants can ultimately reach humans after crossing one or several paths and they have been released by at least one intermediate (Rikken and Lijzen, 2004; Zaimoglu *et al.*, 2006).

In Ghana, individuals residing in mining environments have been exposed to trace metals particularly As (metalloid), Pb and Mn in surface and groundwater over the last few decades (Armah *et al.*, 2010; Obiri *et al.*, 2010a). High concentrations of these trace metals have been identified in various environmental compartments in mining communities, particularly surface and groundwater bodies (Obiri *et al.*, 2006; Essumang, 2009). Quantifying trace metals in ecological receptors in Ghana has received much attention but only few studies have attempted to quantify the risk posed to human receptors particularly among residents living in these contaminated mining areas. Obiri *et al.* (2006) focused on occupation-based risk assessment arising from exposure to selected heavy metals onsite while Obiri *et al.* (2010a) considered cancer and non-cancer risk assessment based on exposure to As, Cu and Cd in surface and groundwater. Obiri *et al.* (2010b) also quantified risks to resident children based on exposure to Pb and Hg. An assessment of the risks such trace metal contaminated surface and groundwater bodies pose to individuals living in mining communities is therefore of the essence. Although, there are many approaches to evaluating such risks, the USEPA risk assessment framework has received extensive recognition given its widespread application around the world. Consequently, this study employs this method to evaluate the risk posed to resident adults and children in the mining community of Tarkwa, Ghana where gold mining activity is pervasive and longstanding.

This study focuses on the Tarkwa-Nsuaem municipality in the Western region of Ghana, within which mining activities have taken place for over a century (Armah *et al.*, 2010). The risks evaluated in this assessment are of two types. The first is a cancer risk in children and adults in the municipality from exposure to arsenic. The acceptable level of risk based upon the United States Environmental Protection Agency guideline is an excess risk of cancer that is not more than one in ten thousand to one in one million greater than that of the general population. The second type of risk is a non-cancer risk from exposure to arsenic, lead and manganese through oral and dermal pathways. This is measured through the use of a hazard quotient that is derived primarily through the use of USEPA (1989, 1991, 1999, 2001) guidance documents. Each of these four documents contributes to various facets of the risk assessment process.

Regarding the risk assessment approach, evaluating possible effects from exposure to trace metals in mining communities, suggests the need to carefully evaluate doses likely to be received by individuals throughout their lifetime or at critical periods within their life cycle.

Typical USEPA human health risk assessment consists of the following four steps:

- **Hazard identification:** Does a chemical contaminant represent a specific threat to human health? Establishment of cause-effect relationships is central to this component. Usually involves the qualitative determination of whether or not a particular hazardous agent is associated with health effects of sufficient importance to warrant further scientific investigations
- **Defining dose-response:** What is the relationship between the magnitude of the exposure and the probability of an adverse health effect?
- **Exposure assessment:** What is the potential for human exposure to the chemical of concern? Exposure scenarios evaluated in this study includes residential setting for resident adults (male and female) and resident children (male and female). Both exposure scenarios evaluated oral incidental ingestion and dermal contact with the chemicals of concern (COCs)-As, Mn and Pb. Both receptors were assumed to be exposed to arsenic, lead and manganese
- **Risk characterization:** What is the potential magnitude of risk to human health given the predicted exposure and dose response data? What is the uncertainty associated with this risk estimate? Cancer and non-cancer health risks were evaluated using RISC 4.02 human health risk software (Spence 2002). The estimated intakes and Cancer Slope Factors (CSFs) are combined to calculate excess cancer risk according to the following equation (USEPA 1989):

$$\text{Cancer risk} = \text{Intake}\left[\left(\text{mg kg}^{-1} - \text{day}\right)\right] \times \text{CSF} \tag{1}$$

According to USEPA (1989), a hazard index is calculated by using the equation:

$$\text{Hazard index} = \frac{\text{Intake}\left[\left(\text{mg kg}^{-1} - \text{day}\right)\right]}{\text{RFD}\left(\text{mg kg}^{-1} - \text{day}\right)} \tag{2}$$

$$HI = Hq_{oral} + HQ_{dermal} \tag{3}$$

This extensively used software has in built mechanisms for calculating the RfD and CSF.

Arsenic is a naturally occurring element in the earth's crust. It is ranked as twentieth in abundance among the elements in the Earth's crust. Arsenic is found in the deep bedrock materials as well as the shallow glacial materials in the study areas. They are also found alongside the gold ores such as arsenopyrites (FeAsS). Arsenic is usually present in the environment in inorganic form. Arsenic can exist in four valence states: -3, 0, +3 and +5 (Naja and Volesky, 2009). Under reducing conditions, arsenite, As (III), is the dominant form; arsenate, As (V), is generally the stable form in oxygenated environments (Naja and Volesky, 2009). Elemental arsenic is not soluble in water. Arsenic salts exhibit a wide range of solubilities, depending on pH and ionic environment (Amonoo-Neizer and Amekor, 1993). The inorganic arsenic easily dissolves and enters underground and surface waters. Inorganic compounds consist of water-soluble arsenite (As III), the most toxic form and arsenate (As V), the less toxic form and such pollutants have been associated with many health problems such as skin lesions, keratosis (skin hardening), lung cancer and bladder cancer (Rashid and Mridha, 2007; Naja and Volesky, 2009). The presence of arsenic in the environment

may be attributed to one of the following sources: residual arsenic from former pesticidal use, smelter emission from ores of gold such as arsenopyrites from the sulphur treatment plant. According to Abernathy *et al.* (1999), inorganic form of arsenic is classified as a class A Carcinogen (Human Carcinogen). This classification is based on sufficient evidence from human data. That is, increased lung cancer mortality was observed in multiple populations exposed to arsenic primarily through drinking of arsenic contaminated water. Arsenic in the study area is very high in river water (Obiri, 2007).

Lead is a well known neurotoxin (Obiri *et al.*, 2010b). It affects the central nervous system. Children exposed to high levels of lead-contaminated water have low IQS. Other symptoms associated with exposure to lead are behavioural disorders, tremors, etc. The presence of lead in the study area is due to weathering and leaching of the metal from waste rocks dumps (AGC, 2001). Another source of lead is the weathering of the Birimian and Tarkwanian rocks which contains high levels of lead. Similarly, improper disposal of lead-acid batteries also accounts for high levels of lead in the study area. Although lead pollution from mining activities presents a relatively localized problem, its magnitude is significant and particularly on the water pollution side which is compounded by the occurrence of other trace metals (Naja and Volesky, 2009). Lead is a cumulative poison, with fetuses, infants, children up to six years of age and pregnant women (because of their fetuses) being most susceptible to adverse health effects. Lead can severely affect the central nervous system (Naja and Volesky, 2009). Overt signs of acute intoxication include dullness, restlessness, irritability, poor attention span, headaches, muscle tremor, hallucinations and loss of memory (ATSDR, 1990).

Manganese is an essential element that becomes toxic at high concentrations. In the current study area, the Birimian and the Tarkwanian rock systems contain high levels of iron and other toxic chemicals such as manganese (AGC, 2001). A high level of manganese in humans is associated with the following symptoms; tremors, weakness in the legs, staggering gait, behavioural disorders, etc. Children exposed to high levels of manganese exhibit low IQS (Obiri *et al.*, 2010a). There is clear evidence from studies of humans exposed to manganese dusts in mines and factories that inhalation of high levels of manganese can lead to a series of serious and ultimately disabling neurological effects in humans. However, in Ghana, manganese in mining communities is primarily due to leaching of manganese from waste rocks (AGC, 2001).

MATERIALS AND METHODS

Study area: Tarkwa is a town located in the southwest of Ghana, approximately a distance of 193 km west of Accra. Since 2005, it was estimated to have a population of 40,397. It is a noted centre of gold and manganese mining. Tarkwa Mine which is a large open-caste gold mine is situated to the North West of the town and Nsuta manganese mine is situated to the East of the town (Kuma and Younger, 2004). The Iduapriem Gold Mine is an open-pit gold mine situated 10 km south of the town.

Sample collection and preparation: Figure 1 shows the locations where samples were collected from April to June 2010. Random sampling techniques were used to collect surface and groundwater samples within the study area. The sampling technique was influenced by the fact that some of the groundwater outlets were capped restricting the sampling locations to only uncapped outlets; hence, the clustered nature of the random sampling pattern. Water samples were obtained from rivers, streams, boreholes and hand-dug wells in the study area and 1.5 L of water

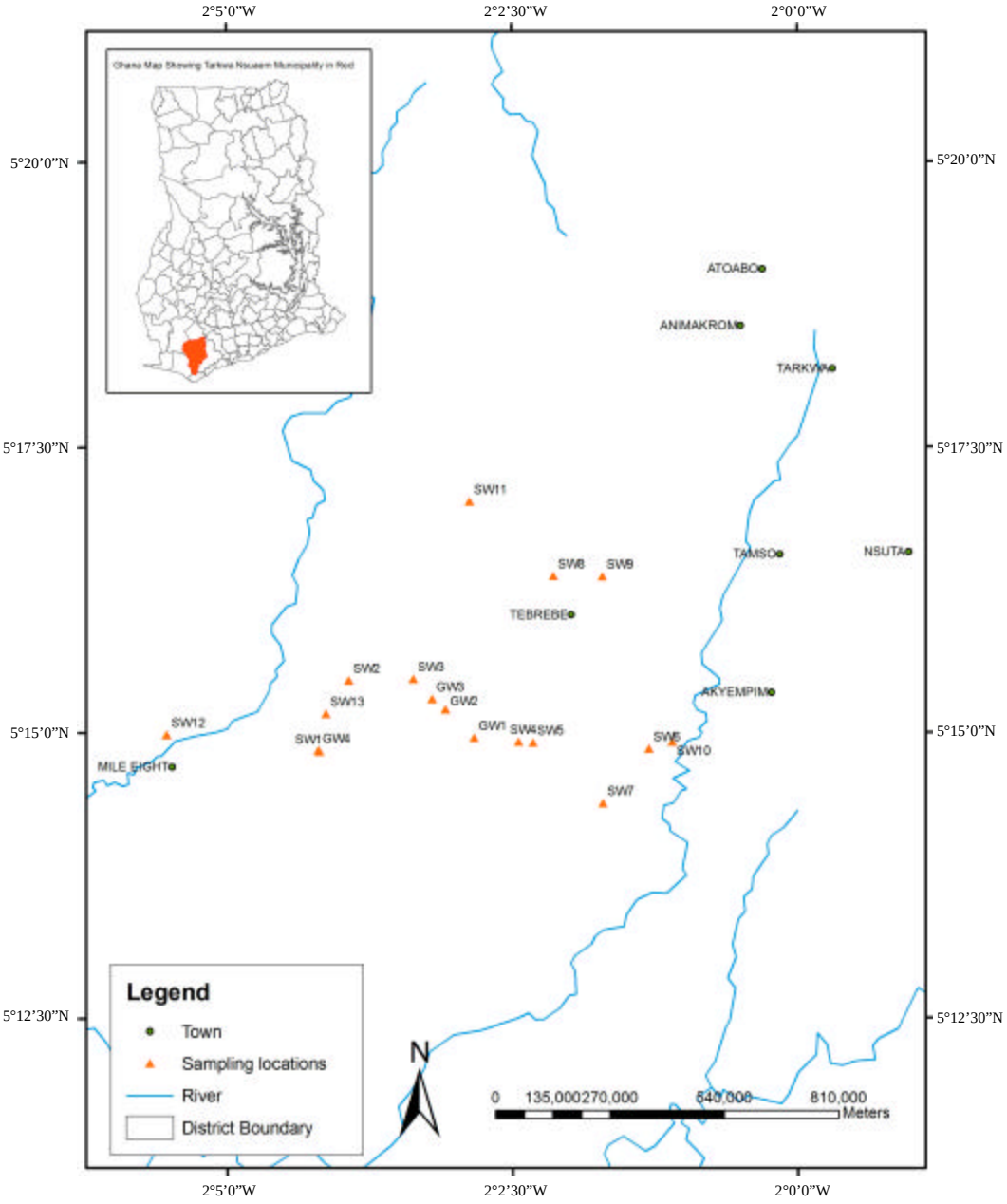

Fig. 1: Study area showing sampling locations

samples were collected into plastic containers that had been rinsed with 1:1 conc. HNO_3 acid and double-distilled water and fixed with identification labels. A total of 250 samples were collected between April 2010 and June 2010 from 16 locations. The samples were stored in an ice-chest at a temperature of 4°C and later conveyed to the laboratory for analysis.

In the laboratory, the acidified samples were filtered using Whatman's No. 0.45 μm filter paper. The 0.45 μm membrane filter paper was used because the analyte of interest in this work is the

total dissolved metals. The pH, turbidity, total dissolved solids and conductivity of the samples were determined *in situ* (APHA, 1998) using a Horiba U52G multi-parameter water quality tester. Other parameters determined included nitrates, sulphates, chemical oxygen demand, cyanides (free, weak acid dissociable and total). Five milliliter each of conc. $HClO_4$ and conc. HNO_3 were added to 100 mL of the sample. Concentrated HNO_3 dissolves all common metals with the exception of aluminum and chromium which are passive to the reagent as a result of surface oxide formation. It also readily oxidizes many organic substances. $HClO_4$ is a potent oxidizing agent that leads to the formation of highly soluble perchlorate salts. $HClO_4$ dehydrates and oxidizes organic samples very efficiently. There is little danger of $HClO_4$ explosions as long as sufficient nitric acid is present to decompose the bulk of the organic matter, hence the addition of HNO_3 to the former. The mixture was heated until the volume was reduced to 20 mL. It was allowed to cool to room temperature and filtered through 0.45 μm filter paper into 100 mL volumetric flask, the final volume was adjusted to 100 mL using double-distilled water and stored for the analysis of As, Pb and Mn (APHA, 1998).

Reproducibility and recovery studies were conducted. In the reproducibility studies, 1.0 mg L^{-1} standard solutions of As, Pb and Mn were each measured (ten times) using flame Atomic Absorption Spectrophotometer Shimadzu model 6401 F. The percentage of As, Pb and Mn recovered in the recovery studies were 95, 97 and 100%, respectively. Similar results were obtained for the reproducibility studies. The percentages of As, Pb and Mn recovered ranged from 95.5 to 99.6% (standard error±0.005 to 0.550). With standard error less than 1, this indicates that the analytical methods employed were reproducible.

For quantitative risk assessment, the dataset from the 16 locations were used. We focused on constituents that had the potential to cause the greatest risk. Screening procedures were used to limit the number of constituents of potential concern (COPC) in each medium (USEPA, 1989). Two screening procedures were used for the risk assessment data sets to ensure that the proper trace metals were being assessed.

The methodologies for the screening procedures used in the risk assessment are addressed in the following paragraphs. The first step in the screening procedure for the risk assessment involved an evaluation of the frequency of detection in each of the data sets. Constituents that were infrequently detected were considered as potential errors in the data due to sampling, analytical, or other procedures and, therefore, may not be related to site operations or disposal practices. Hence for risk assessment, a constituent detected in 5% or fewer samples in any given environmental medium (e.g., groundwater, surface water) was omitted from the quantitative risk assessment for that medium. In addition, constituents that were considered to be essential nutrients, such as aluminum, calcium, sodium, magnesium, potassium and iron, were excluded. The comparison method was used as second step in the screening procedure and this was applied to each of the data sets. With the comparison method, the maximum concentration detected in a medium or data set was compared to screening values. However, constituents that are known human carcinogens were retained as COPCs if they exceeded screening values, regardless of the frequency of their detection. Table 1 presents receptor specific data used in the study.

Mean concentrations of As, Pb and Mn, were calculated in SPSS and then keyed in Risk Integrated Software for Clean ups (RISC 4.02), a human health risk assessment computerized software.

Table 1: Receptor specific data used in the study

Parameter	Adult resident (CTE)	Adult resident (RME)	Child resident (CTE)	Children resident (RME)
Lifetime (year)	70	70	70	70
Body weight (kg)	70	70	15	15
Exposure frequency for groundwater (events $year^{-1}$)	350	350	350	350
Exposure duration for groundwater (year)	9	30	6	6
Ingestion rate for groundwater (day^{-1})	1.1	2	0.5	1
Total skin surface area (cm^2)	18400	23000	6800	7280
Time of exposure while washing (h day^{-1})	0.12	0.2	0.12	0.2
Exposure frequency for swimming (events $year^{-1}$)	20	36	5	36
Exposure duration for swimming (year)	9	30	6	6
Ingestion rate while swimming (mL h^{-1})	10	50	10	50
Time spent swimming (h day^{-1})	2.6	2.6	2.6	2.6

These values are default values that were generated using RISC 4.02 software

Data processing and analysis: All the mathematical and statistical computations were made using Statistical Package for Social Sciences (SPSS) version 16 (Gerber and Finn, 2005). Equation 1 and 2 are in built in the RISC 4.02 software. Cancer and non-cancer risks were calculated using the RISC 4.02 software.

RESULTS

Cancer risks within the USEPA target range of 1×10^{-4} to 1×10^{-6} for resident adults and children constitute acceptable risks. That is, for a person meeting the conditions of the reasonably maximal exposed individual, the risk is less than one in ten thousand. The evaluation of cancer risks arising from exposure to arsenic for SW1 revealed that the cancer risks to resident adults for RME ranged from 5.0×0^{-4} to 1.1×10^{-3}. These values were up to 11 times greater than the USEPA acceptable range for excess risk of cancer. This is due to arsenic in a tributary of the Agonabeng stream near a mine waste dump. However, this location is neither used as a drinking water source nor household supply. The evaluation of cancer risks from exposure to arsenic showed that the cancer risks to resident adults for CTE ranges from 3.7×10^{-4} to 6.7×10^{-4}. These values were also higher than the USEPA acceptable range for excess risk of cancer but considerably lower than results obtained by Obiri *et al.* (2010a) in surface water within the Obuasi mining area. Total excess cancer risks for all other surface water locations regarding arsenic were all within the USEPA acceptable range for adult residents for both CTE and RME (Table 2). This contrasts with the results obtained by Obiri *et al.* (2010a) for the Obuasi mining area. The evaluation of cancer risks arising from exposure to arsenic revealed that, except for three surface water locations (SW5, SW6 and SW8); the cancer risks to resident children for both CTE and RME were greater than the USEPA acceptable range for excess risk of cancer. CTE values ranged from 4.0×10^{-5} to 2.1×10^{-2} (Table 2) while RME values ranged from 2.3×10^{-5} to 3.8×10^{-1} (Table 2). These values were up to 210 times greater than the USEPA acceptable range for excess risk of cancer. The risks posed to children are much higher than adults in this case. Children are generally more vulnerable than adults consequently there is the need to mitigate the high risk posed to them.

Arsenic related non-carcinogenic health risks via a single exposure route (either dermal or oral) for both children and adults in the CTE and RME scenarios were within the USEPA acceptable risks (Table 2). From Table 2, summation of As-related hazard quotients for oral and dermal contact

Table 2: Carcinogenic risk and hazard quotients from exposure to arsenic

			Cancer health risk				Non cancer health risk			
			Children		Adults		Children		Adults	
Sample ID	Mean concentration (mg L^{-1})	Exposure route	CTE	RME	CTE	RME	CTE	RME	CTE	RME
SW1	0.093	Oral contact	0.0017	0.0047	0.00037	0.0011	0.0043	0.11	0.0063	0.057
		Dermal contact	0.0012	0.0064	0.00067	0.00050	0.003	0.017	0.012	0.026
SW2	0.032	Oral contact	0.00059	0.015	1.3×10^{-7}	3.8×10^{-6}	0.0015	0.039	0.0022	0.020
		Dermal contact	0.00004	0.0022	2.3×10^{-7}	1.7×10^{-6}	0.001	0.0057	0.0040	0.0040
SW4	0.055	Oral contact	0.001	0.20	2.2×10^{-7}	6.5×10^{-6}	0.0036	0.067	0.0037	0.034
		Dermal contact	0.000069	0.38	4.0×10^{-7}	3.0×10^{-6}	0.0018	0.00568	0.0068	0.0090
SW5	0.022	Oral contact	4.0×10^{-8}	1.0×10^{-6}	1.8×10^{-7}	2.6×10^{-6}	0.0010	0.0027	0.0015	0.013
		Dermal contact	2.7×10^{-8}	1.5×10^{-6}	1.6×10^{-7}	1.2×10^{-6}	0.00074	0.0039	0.0027	0.0062
SW6	0.028	Oral contact	5.8×10^{-8}	1.3×10^{-6}	1.2×10^{-7}	1.1×10^{-6}	0.0013	0.034	0.0019	0.019
		Dermal contact	2.7×10^{-8}	1.9×10^{-7}	2.0×10^{-7}	2.0×10^{-7}	0.00071	0.0050	0.0035	0.035
SW7	0.037	Oral contact	0.0068	0.0017	1.5×10^{-7}	4.6×10^{-6}	0.0018	0.045	0.0025	0.023
		Dermal contact	0.0046	0.0025	2.7×10^{-6}	2.0×10^{-6}	0.0012	0.066	0.0046	0.010
SW8	0.032	Oral contact	0.000059	0.015	1.3×10^{-7}	3.8×10^{-6}	0.0015	0.039	0.0022	0.020
		Dermal contact	0.000004	0.0022	2.3×10^{-7}	1.7×10^{-6}	0.0010	0.0057	0.0040	0.0040
SW9	0.047	Oral contact	0.0087	0.0022	1.8×10^{-7}	5.5×10^{-6}	0.0022	0.057	0.0032	0.029
		Dermal contact	0.0059	0.033	3.4×10^{-7}	2.5×10^{-6}	0.0015	0.0084	0.0059	0.013
SW10	0.037	Oral contact	0.0068	0.0017	1.2×10^{-7}	1.1×10^{-7}	0.0018	0.045	0.0019	0.019
		Dermal contact	0.0046	0.0025	2.0×10^{-7}	2.0×10^{-7}	0.0012	0.066	0.0035	0.035
SW11	0.076	Oral contact	0.014	0.036	3.0×10^{-7}	8.9×10^{-6}	0.0036	0.093	0.0052	0.046
		Dermal contact	0.0095	0.052	5.5×10^{-7}	4.1×10^{-6}	0.0025	0.014	0.0095	0.021
SW12	0.045	Oral contact	0.0082	0.0021	1.8×10^{-7}	5.3×10^{-6}	0.0021	0.055	0.0031	0.027
		Dermal contact	0.0056	0.0031	3.2×10^{-7}	2.4×10^{-6}	0.0015	0.0080	0.0056	0.013
SW13	0.033	Oral contact	0.00060	0.00016	1.3×10^{-7}	3.9×10^{-6}	0.0016	0.040	0.0022	0.020
		Dermal contact	0.00041	0.000023	2.4×10^{-7}	1.8×10^{-6}	0.0011	0.0059	0.0041	0.0093
SW14	0.039	Oral contact	0.0071	0.0018	1.5×10^{-7}	4.6×10^{-6}	0.0019	0.048	0.0026	0.024
		Dermal contact	0.0049	0.0027	2.8×10^{-7}	2.1×10^{-6}	0.0013	0.0069	0.0049	0.011
GW1	0.035	Oral contact	0.00064	0.00016	1.4×10^{-7}	4.1×10^{-6}	0.0017	0.043	0.0024	0.021
		Dermal contact	0.00044	0.00024	2.5×10^{-7}	1.9×10^{-6}	0.0011	0.0062	0.0044	0.0098
GW2	0.074	Oral contact	0.014	0.0035	2.9×10^{-7}	8.7×10^{-6}	0.0035	0.090	0.0050	0.045
		Dermal contact	0.0092	0.0051	5.3×10^{-7}	4.0×10^{-6}	0.0024	0.013	0.0092	0.021
GW3	0.069	Oral contact	0.00013	0.0032	2.7×10^{-7}	8.1×10^{-6}	0.0033	0.084	0.0047	0.042
		Dermal contact	0.00086	0.0047	5.0×10^{-7}	3.7×10^{-6}	0.0022	0.012	0.0086	0.019
GW4	0.114	Oral contact	0.021	0.054	4.5×10^{-7}	1.3×10^{-5}	0.0054	0.14	0.0077	0.070
		Dermal contact	0.014	0.0078	$8.2\times1^{-0^{7}}$	6.2×10^{-6}	0.0037	0.020	0.014	0.032

USEPA acceptable risk values for cancer risk = 1.0×10^{-4} to 1.0×10^{-6}, USEPA acceptable risk values for non-cancer risk ≤1.0

did not result in HI values greater than 1, consequently, arsenic did not pose any significant non-cancer risks to children in the local population.

Non-cancer health hazard quotients below 1.0 for resident adults and children constitute acceptable risks. Exposure to surface and groundwater poses significant human health risks. Hazard quotient ranging from 0.1 to 18 (Table 3) were obtained from the evaluation of risk. The primary contributor to these health hazards is manganese as measured in surface and groundwater. The highest risk particularly refers to the RME of Mn through oral contact for

Table 3: Hazard quotients of Mn

			Non cancer health risk			
			Children		Adults	
Sample ID	Mean concentration (mg L^{-1})	Exposure route	CTE	RME	CTE	RME
SW1	1.387	Oral contact	0.066	1.70	0.094	0.85
		Dermal contact	0.076	0.42	0.29	0.66
SW2	5.644	Oral contact	0.27	6.90	0.0035	3.40
		Dermal contact	0.31	1.70	0.38	2.70
SW4	1.023	Oral contact	0.049	1.20	0.069	0.62
		Dermal contact	0.056	0.31	0.22	0.49
SW5	4.743	Oral contact	0.23	5.80	0.32	2.90
		Dermal contact	0.26	1.40	1.00	2.30
SW6	1.264	Oral contact	0.060	1.50	0.086	0.77
		Dermal contact	0.069	0.38	0.27	0.60
SW7	2.136	Oral contact	0.10	2.60	0.14	1.30
		Dermal contact	0.12	0.65	0.45	1.00
SW8	1.028	Oral contact	0.049	1.30	0.07	0.63
		Dermal contact	0.056	0.31	0.22	0.49
SW9	1.056	Oral contact	0.050	1.30	0.072	0.64
		Dermal contact	0.058	0.31	0.22	0.50
SW10	5.644	Oral contact	0.25	6.90	0.38	3.40
		Dermal contact	0.31	1.70	1.20	2.70
SW11	5.410	Oral contact	0.26	6.60	0.37	3.30
		Dermal contact	0.30	1.60	1.10	2.60
SW12	5.587	Oral contact	0.27	6.80	0.38	3.40
		Dermal contact	0.31	1.70	1.20	2.70
SW13	14.987	Oral contact	0.71	18.00	1.00	9.20
		Dermal contact	0.82	4.50	3.20	7.20
SW14	13.481	Oral contact	0.64	16.00	0.092	8.20
		Dermal contact	0.74	4.10	2.90	6.40
GW1	3.333	Oral contact	0.16	4.10	0.26	2.00
		Dermal contact	0.18	1.00	0.71	1.60
GW2	11.963	Oral contact	0.57	15.00	0.76	6.80
		Dermal contact	0.66	3.60	2.40	5.30
GW3	1.963	Oral contact	0.093	2.40	0.13	1.20
		Dermal contact	0.11	0.59	0.42	0.94
GW4	1.039	Oral contact	0.04	1.30	0.07	0.63
		Dermal contact	0.057	0.31	0.22	0.50

USEPA acceptable risk values for cancer risk = 1.0×10^{-4} to 1.0×10^{-6}, USEPA acceptable risk values for non-cancer risk ≤1.0

Children. While at some locations, the RME of Mn through oral contact for adults also exceeded the standard, a comparison of contributions of the two pathways, oral and dermal, to the health risk shows that the former accounts for more.

HI for three locations namely SW13 (1.53), SW14 (1.38) and GW2 (1.23) were greater than 1 indicating that drinking water from these locations constitutes potential health risk for children.

Generally, regarding manganese relatively high hazard quotients were obtained for resident children in the RME scenario while low hazard quotients were obtained for resident adults in the

Table 4: Hazard quotients of Pb

			Non cancer health risk			
			Children		Adults	
Sample ID	Mean concentration (mg L^{-1})	Exposure route	CTE	RME	CTE	RME
SW1	0.044	Oral contact	0.00017	0.0045	0.00025	0.0022
		Dermal contact				
SW2	0.069	Oral contact	0.00027	0.0070	0.00039	0.38
		Dermal contact				
SW4	0.023	Oral contact	0.000091	0.0023	0.00013	0.0012
		Dermal contact				
SW5	0.039	Oral contact	0.00015	0.0040	0.00022	0.0020
		Dermal contact				
SW6	0.027	Oral contact	0.00011	0.0027	0.00015	0.0014
		Dermal contact				
SW7	0.035	Oral contact	0.00014	0.0036	0.00020	0.0018
		Dermal contact				
SW8	0.034	Oral contact	0.00013	0.0035	0.00019	0.0017
		Dermal contact				
SW9	0.042	Oral contact	0.00017	0.0043	0.00024	0.0021
		Dermal contact				
SW10	0.055	Oral contact	0.00022	0.0056	0.00031	0.0028
		Dermal contact				
SW11	0.054	Oral contact	0.00021	0.0055	0.00031	0.0027
		Dermal contact				
SW12	0.073	Oral contact	0.00029	0.0074	0.00041	0.0027
		Dermal contact				
SW13	0.092	Oral contact	0.00036	0.0094	0.00052	0.0047
		Dermal contact				
SW14	0.083	Oral contact	0.00033	0.0084	0.00047	0.0042
		Dermal contact				
GW1	0.051	Oral contact	0.00020	0.0052	0.00029	0.0026
		Dermal contact				
GW2	0.042	Oral contact	0.00017	0.0043	0.00024	0.0021
		Dermal contact				
GW3	0.075	Oral contact	0.00030	0.0076	0.00042	0.0038
		Dermal contact				
GW4	0.051	Oral contact	0.00020	0.0052	0.00029	0.0026
		Dermal contact				

Lead is not absorbed through the skin as such there are no values for dermal contact. USEPA acceptable risk values for cancer risk = 1.0×10^{-4} to 1.0×10^{-6}, USEPA acceptable risk values for non-cancer risk≤1.0

CTE scenario. Non-carcinogenic health risks arising from exposure to lead for both children and adults in the CTE and RME scenarios were within the USEPA acceptable risks (Table 4). Apart from manganese, arsenic and lead in groundwater do not account for the hazard quotient of the resident children in the CTE and RME exposure scenarios. The hazard quotient for resident children based on arsenic in groundwater is far less than 1.0. Arsenic in groundwater may likely be due to naturally occurring arsenic in soil and not related to site activities. Additionally, the concentrations of arsenic in groundwater are within the WHO drinking water limits.

DISCUSSION

The risk estimate provided by this analysis clearly shows that the communities in this mining area are at excess risk for As related cancer. Human health concerns associated with lead intoxication in children include brain damage and/or mental retardation, behavioral problems, anaemia, liver and kidney damage, hearing loss, hyperactivity and developmental delays (Stosh and Bagchi, 1995; Goyer, 1993; Gohar and Mohammadi, 2010; Rajaganapathy *et al*., 2011) whereas in adults poor muscle coordination, nerve damage to the sense organs and nerves controlling the body, increased blood pressure, hearing and vision impairment, reproductive problems (e.g., decreased sperm count) and retarded fetal development even at relatively low exposure levels may arise (Stosh and Bagchi, 1995; Surendran and El-Fawal, 2008). Health effects of arsenic exposure include neurological damage (Tchounwou *et al*., 2003), cardiovascular disease and cancer (Tchounwou *et al*., 2004). Effects from manganese are primarily to the central nervous system (Wasserman *et al*., 2006; Bhattacharya *et al*., 2008). In the cases of Mn and As, local redox conditions will play a large role in the transport of these metals (Tchounwou *et al*., 2003).

Arsenic related cancer risk values obtained in this study were less than the values obtained in drinking water from West Bengal, India (Roychowdhury *et al*., 2003); however, it is consistent with the findings of Li *et al*. (2010) in the Luanhe River Basin, China. Also, arsenic related cancer risk values obtained in this study were less than values obtained by Lim *et al*. (2008) in the vicinity of an abandoned goldmine in South Korea. Pb related non-cancer health risk values obtained in this study were far less than the values obtained in the Taihu Lake in China (Liang *et al*., 2011). Mn related non cancer risk values were however greater than values obtained by Liang *et al*. (2011). Mn and Pb related non-cancer risks values are consistent with the findings of Wcislo *et al*. (2002) regarding an abandoned metal smelter in Poland.

The average person was assumed to have a body weight of 70 kg. This default assumption may underestimate risks to children and more highly sensitive individuals. Risk estimates assume that people spend their lifetime (70 years) exposed to the annual average hazardous water pollutant concentrations estimated for their area. This assumption could lead to overestimation of potential health risks if pollution levels decline over time. It could lead to underestimation if people live in "hotspots" where water pollutant concentrations are higher than area averages. Exposures to hazardous air pollutants through other media, such as food were not taken into account. Omission of these exposures may underestimate potential human health risks. This type of risk estimate provides a way to screen for those pollutants that are of public health significance in order to prioritize research and regulatory intervention efforts (Albering *et al*., 1999; Tchounwou *et al*., 2003). For example, it was assumed that exposure levels, exposure duration and exposure frequency do not change in a lifetime and that human activities remain the same. It is apparent that the likelihood of this is very small. Notwithstanding these limitations, the findings of the study are relevant and hold several policy implications.

The findings of this study hold several implications for policy. Previously, most mining communities depended on surface water as drinking water sources. However, the contamination of surface water particularly via small-scale mining activities (Armah *et al*., 2010) made it imperative for government and other non-state stakeholders to resort to groundwater. However, this study and other studies (Obiri *et al*., 2010a, b) have shown that the quality of groundwater is also questionable. Policy makers need to be appraised of the situation so that they can formulate regulations that make it mandatory to test sources of drinking water in mining communities on a regular basis. Where boreholes have been tested, communities need to be notified about

contaminant levels so that it can inform their daily decision-making regarding access to safe drinking water (Berg *et al*., 2007). Apart from hand-dug wells and boreholes, other mitigation actions on the household level include rainwater collection and sand filters (Berg *et al*., 2007). Overall, the results indicate there is a critical need for a clearly laid out strategy to mitigate risk in this area.

CONCLUSION

The study calculated the health risks to resident adults and children from exposure to three trace metals: arsenic, manganese and lead in surface and groundwater within a mining community in Ghana. Both carcinogenic and non-carcinogenic risks were evaluated. Mining facilities located in Tarkwa constitute a relevant source of pollution by heavy metals. Moreover, the presence of these facilities does pose a notable risk for the health of the population living in the vicinity. The risk estimate provided by this study clearly shows that the communities in this mining area are at excess risk for As related cancer. Cancer risk values were up to 210 times greater than the USEPA acceptable range for excess risk of cancer. Groundwater was considered to be a useful alternative drinking water in the mining communities. However, the findings of the study indicate that indiscriminate reliance on groundwater without preliminary testing of water quality could present potential health risks to the surrounding population. Consequently, a monitoring programme is clearly advisable, while some efforts should be focused on reducing the environmental levels of As, Pb and Mn in surface and groundwater sources in the mining communities.

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