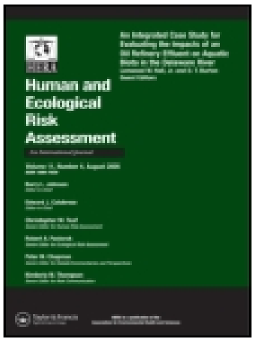

Taylor & Francis
Taylor & Francis Group

Human and Ecological Risk Assessment

ISSN: 1080-7039 (Print) 1549-7860 (Online) Journal homepage: https://www.tandfonline.com/loi/bher20

Cancer and Non-Cancer Risk Assessment from Exposure to Arsenic, Copper, and Cadmium in Borehole, Tap, and Surface Water in the Obuasi Municipality, Ghana

S. Obiri , D. K. Dodoo , D. K. Essumang & F. A. Armah

To cite this article: S. Obiri , D. K. Dodoo , D. K. Essumang & F. A. Armah (2010) Cancer and Non-Cancer Risk Assessment from Exposure to Arsenic, Copper, and Cadmium in Borehole, Tap, and Surface Water in the Obuasi Municipality, Ghana, Human and Ecological Risk Assessment, 16:3, 651-665, DOI: 10.1080/10807031003788907

To link to this article: https://doi.org/10.1080/10807031003788907

Human and Ecological Risk Assessment, 16: 651–665, 2010
Copyright © Taylor & Francis Group, LLC
ISSN: 1080-7039 print / 1549-7860 online
DOI: 10.1080/10807031003788907

Risk Contribution: Around the World

Cancer and Non-Cancer Risk Assessment from Exposure to Arsenic, Copper, and Cadmium in Borehole, Tap, and Surface Water in the Obuasi Municipality, Ghana

S. Obiri,[1,2] D. K. Dodoo,[3] D. K. Essumang,[3] and F. A. Armah[4]
[1]Centre for Environmental Impact Analysis (CEIA), Adisadel, Cape Coast, Ghana; [2]Water Research Institute—CSIR, Achimota, Accra, Ghana; [3]Environmental Research Group, Department Of Chemistry, University Of Cape Coast, Cape Coast, Ghana; [4]Department of Environmental Science, School of Biological Sciences, University of Cape Coast, Ghana

ABSTRACT

Cancer and non-cancer risk assessment from exposure to As, Cd, and Cu by resident adults and children from different water sources in Obuasi Municipality, Ghana, were measured in this study in accordance with the U.S. Environmental Protection Agency's (USEPA's) Human Health Risk Assessment guidelines. The results of cancer health risk for resident adults in Obuasi exposed to As in their tap water for both Central Tendency Exposure (CTE) and Reasonable Maximum Exposure (RME) parameters, respectively, are 6.6×10^{-4} and 5.5×10^{-6}. For resident children in Odumasi, we obtained 4.7×10^{-1} (CTE) and 6.7×10^{-1} (RME). The results of the study obtained in most cases were found to exceed the USEPA's acceptable cancer risk range of 1×10^{-6} to 1×10^{-4} (*i.e.*, 1 case of cancer out of 1,000,000 people to 1 case of cancer out of 10,000 people). Similarly, the results of the non-cancer human health risk for both resident adults and children were also found in most cases to be greater than the USEPA's acceptable non-cancer human health hazard index of 1.

Key Words: cancer and non-cancer health risks, hazard index, River Nyam, River Supu, borehole and tap water, Ghana.

Received 20 April 2009; revised manuscript accepted 22 June 2009.

Address correspondence to Mr. Obiri Samuel, Centre for Environmental Impact Analysis, P.O. Box AD 738, Cape Coast, Ghana or Water Research Institute, P.O. Box AH 40, Accra, Ghana. E-mail: sobiri@ceiagh.com

INTRODUCTION

Water, in addition to being a vital nutrient, is involved in many aspects of human metabolism. It plays important roles in the digestion and absorption of food, transports nutrients in the body, and the elimination of waste products via the excretory organs. The use of water with adequate physical, chemical, and microbial qualities is of fundamental importance to human beings. According to Obiri (2007), surface water in most mining communities in Ghana have become unfit for human consumption due to chemical contamination from gold mining and processing activities (*e.g.*, roasting of arsenopyrites ores and cyanide heap leach methods). Obuasi Municipality, the primary focus of this article, has a long history of gold mining that dates as far back as 1896 (Adimado and Amegbey 2003; Carbo and Serfor-Armah 1997; Akabzaa *et al.* 2005; Hilson 2002). Cyanide spillages and runoff of toxic chemicals from mine sites into rivers in the study area have resulted in toxic exposure of residents in Obuasi and its surrounding villages who consume water and aquatic biota to carcinogenic and non-carcinogenic substances from the impacted rivers.

The Government of Ghana as well as mining companies in the study area as part of their corporate social responsibility have drilled boreholes to provide alternative sources of drinking water for the communities, but in places where the boreholes were not available or not functioning, residents were compelled to drink from the polluted streams, thus posing a health hazard to them. Similarly, groundwater in most mining communities is contaminated due to the chemical nature of the aquifer host rock in the area (Amasa 1975; Amonoo-Neizer and Amekor 1993; Smedley 1996; Obiri 2007).

It is against this background that this study was undertaken to address concerns faced by residents of Obuasi Municipality and Ghanaians in general over exposure to carcinogenic and non-carcinogenic chemicals in tap, borehole, and surface water. The study seeks to answer three fundamental questions that residents in the Obuasi Municipality have posed many times:

- What are the risks associated with exposure to carcinogenic and non-carcinogenic chemicals in tap, borehole, and surface water?
- How serious are they in terms of their levels and prevalence?
- How well can they be estimated?

Answers to these questions have their roots in cancer and non-cancer human health risk assessment and hence provides a strong justification for a study of this nature. The main thrust of this article is to:

- Identify cancer and non-cancer health hazards associated with exposure to As, Cu, and Cd in tap, borehole, and surface water in the study area.
- Estimate chronic, sub-chronic, and acute non-cancer health risk associated with ingesting As, Cu, and Cd in drinking water by resident adults and children in the study area.
- Evaluate cancer health risk associated with ingestion of As, Cu, and Cd in tap water by resident adults and children in the study area.

- Recommend to government and other stakeholders ways of reducing cancer and non-cancer health risk associated with ingestion As, Cu, and Cd in tap, borehole, and surface water in the study area.

Risk assessment is defined as a process used to identify in qualitative sense, and then to quantify scientifically, the risk associated with a given exposure. It is also a process used to estimate health effects that might result from exposure to carcinogenic and non-carcinogenic chemicals (Asante-Duah 1996; Obiri *et al.* 2006; USEPA 2001a and 2001b). Risk assessment of toxic chemicals involves, as proposed by the U.S. Environmental Protection Agency (USEPA), involves four basic steps. These are:

- Hazard identification—this is the first step of the risk assessment process that is used to establish a link between of the toxic chemicals identified ad their health effects on residents in the study area.
- Exposure assessment—is used to identify the chemicals of concern and to estimate the magnitude of human exposure to the chemicals of concern. It also identifies the various pathways in which residents in the study could be exposed to the chemicals of concern.
- Toxicity assessment—identifies the toxicity criteria to be used to evaluate human risk associated with each carcinogenic and non-carcinogenic chemical in the study area.
- Risk characterization/estimation—incorporates information gathered from hazard identification, exposure assessment and toxicity assessment to evaluate the potential health risk residents in the study area face. These steps are part of the USEPA Conceptual Model for Environmental Risk Assessment shown in Figure 1 and forms the basis of this study.

METHODOLOGY

Sampling Techniques and Sample Collection

Random sampling techniques were adopted in selecting the sample sites. The samples were collected between March to June 2006. In all, 60 samples were collected within this period. Samples collected for each month were analyzed separately for As, Cu, and Cd and their mean values were computed at the end of study. One and a half liters of the water samples were collected from each sampling point into plastic bottles that had been pre-washed with detergent and rinsed with 1:1 nitric acid and double-distilled water. The samples were acidified with 10% nitric acid, stored in an ice chest at 4°C and were conveyed to the laboratory for analysis.

Analysis of Arsenic, Copper, and Cadmium

One hundred ml of the acidified sample was mixed with 5 ml concentration H_2SO_4 and concentration 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 0.45 μm Whatman filter paper. The final volume was adjusted to 100 ml with double-distilled water and stored for analysis (USEPA 1991; AWWA 1998).

The concentrations of Cu and Cd were determined using flame AAS (Atomic Absorption Spectrophotometer) Shimadzu model 6401F. In the determination of

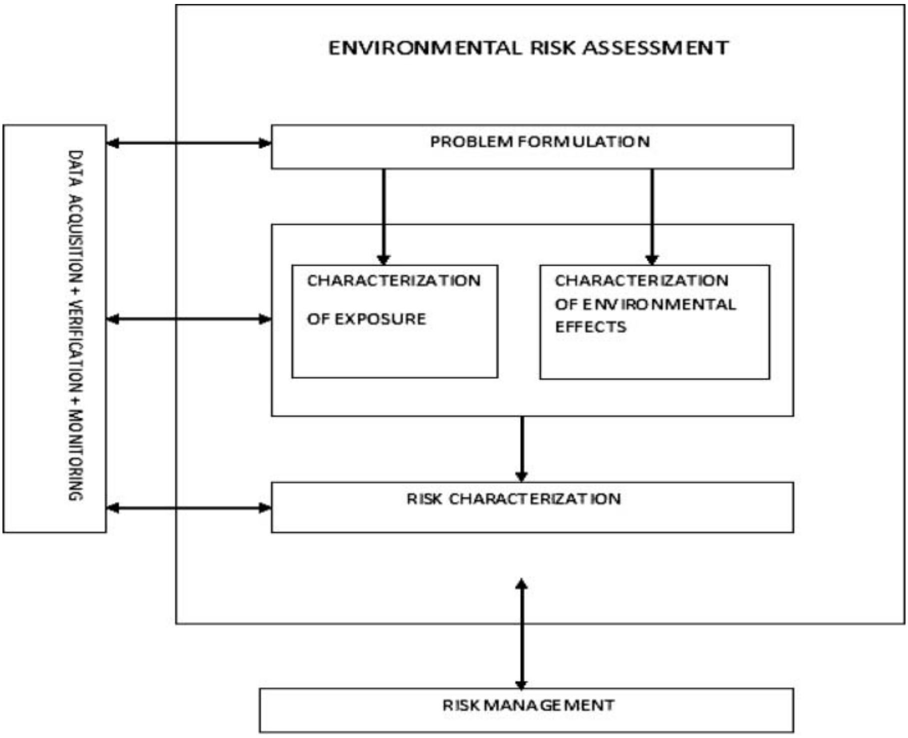

Figure 1. The USEPA conceptual model for environmental risk assessment (modified from Nelson *et al.* 2005).

As, 5 ml of 0.5% $NaBH_4$ and 5 ml of 0.5M HCl were added to each of the digested water samples to reduce all the As in the samples to arsine gas, in the arsine gas generator, which was coupled to the flame AAS Shimadzu model 6401F. The minimum detection limits for As, Cd, and Cu are 0.05 mg/l, 0.010 mg/l, and 0.05 mg/l, respectively.

Quality Control

Reproducibility and recovery studies were conducted. In the reproducibility studies, 1.0 mg/l standard solutions of As, Cd, and Cu were each measured (ten times) using flame Shimadzu model 6401F. The percentage of As, Cu, and Cd recovered in the recovery studies were 92%, 95%, and 100%, respectively. Similar results were obtained for the reproducibility studies. The percentage of As, Cd, and Cu recovered in the reproducibility studies ranged from 96.3 to 99.6% (standard error ± 0.005 to 0.560). The standard error is less than 1, which suggests that the analysis methods employed were reproducible.

Hazard Identification

Arsenic

Arsenic is an element that is present in soil, water, and food. As in the environment exists in many different forms. In water, for example, As exists primarily as the inorganic form As (+III)—arsenite and As (+V)—arsenate, while in food arsenic exists primarily in organic forms (seafood, for example, contains As as arsenobite), a form that is easily absorbed but rapidly excreted unchanged. The speciation of As (+III) and As (+V) in mine tailings and river sediments within the study area has been given extensive description by Ahmad and Carboo (2000).

According to Tseng *et al.* (1968), chronic dermal exposure to As causes skin cancer. The prevalence of skin cancer is very high in areas where chronic exposure to inorganic As is very high. Tseng (1977) has noted that hyper pigmentation, keratosis, and possibly vascular complications were seen at LOAEL = 0.17 mg/l (0.014 mg/kg/day). The occurrence of acute gastrointestinal symptoms, central and peripheral neuropathy, bone marrow suppression, hepatic toxicity, and mild mucous membrane and cutaneous changes are well documented in the literature. The inorganic form of As 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 As primarily through drinking of As-contaminated water. Again, an increased mortality from multiple internal cancers (liver, kidney, lung, and bladder) and an increased incidence of skin cancer had been observed in populations consuming drinking water with high inorganic As concentration.

Cadmium

Cadmium has been shown to be toxic to human populations from occupational inhalation exposure and accidental ingestion of Cd-contaminated food. Inhalation of Cd dust in certain occupational settings may be associated with an increased incidence of lung cancer. Other symptoms include: irritation of upper respiratory tract, metallic taste in the mouth, cough, and chest pains (Foulkes 1986; Shakah and Smith 1976). Ingestion of elevated levels of Cd has resulted in toxicity to the kidney and skeletal system and may be associated with an elevated incidence of hypertension and cardiovascular disease.

Cadmium is poorly absorbed from the lung, gastrointestinal tract, and skin. Individuals with dietary deficiencies of iron, calcium, or protein exhibit higher absorption of ingested Cd. Cadmium in the body binds readily to certain sulphur-containing proteins, such as metallothionein. Binding to metallothionein is thought to reduce the toxicity of Cd. Following ingestion, faecal excretion is high due to poor gastrointestinal absorption. Most Cd that has been absorbed, however, is excreted very slowly, with faecal and urinary excretion being about equal. Urinary Cd levels are an indicator of body burden.

Copper

Acute toxicity has been observed in humans following deliberate ingestion of Cu salts. Symptoms observed include vomiting, lethargy, acute haemolytic anaemia, renal, and liver damage, and in some cases, coma and death. It has been reported that occupational exposure to high concentrations of Cu fume results in metal fume fever (a flu-like illness). Long-term exposure to Cu can cause irritation of the nose, mouth, and eyes and it causes headaches, stomach aches, dizziness, vomiting, and diarrhea. Intentionally high uptakes of Cu may cause liver and kidney damage and even death.

Industrial exposure to Cu fumes, dusts, or mists may result in metal fume fever with atrophic changes in nasal mucous membranes. Chronic exposure to Cu results in Wilson's disease, characterized by hepatic cirrhosis, brain damage, demyelination, renal disease, and Cu deposition in the cornea (IPCS 1998; NOHSC 1999).

Exposure Assessment

An exposure assessment is used to identify constituents of concern (COCs), and to estimate the magnitude of human exposure to As, Cu, and Cd. It also evaluates the pathways by which individuals could be exposed to the constituents of concern. Ions of these three contaminants in drinking water exist in different oxidation states. In this study, cancer and non-cancer human health risk associated with oral and dermal exposure of total As, Cu, and Cd in tap, borehole, and surface water in the study area were determined. The exposure scenario evaluated in this study is a residential setting. In this scenario, ingestion and dermal contact of tap, borehole, and surface water in the study area by resident adults and children were evaluated based on both Central Tendency Exposure (CTE) and Reasonable Maximum Exposure (RME) parameters, respectively. CTE exposure parameters were used so that health risks associated with typical or average exposures to the COCs can be calculated. RME parameters were also used so that health risks associated with high-end exposures

Table 1. Cancer health risks results faced by both resident children and adults from exposure to arsenic in the study area.

			Cancer health risks			
Sampling point	Exposure media	Exposure route	CTE adults	RME adults	CTE children	RME children
Borehole at Oduamsi	Water	Ingestion	3.7×10^{-2}	5.6×10^{-2}	4.7×10^{-2}	6.7×10^{-2}
		Dermal	2.8×10^{-3}	1.9×10^{-3}	3.2×10^{-3}	5.1×10^{-3}
Tap water at Odumasi	Water	Ingestion	9.8×10^{-3}	2.9×10^{-3}	7.6×10^{-4}	2.7×10^{-3}
		Dermal	1.8×10^{-4}	1.4×10^{-4}	5.2×10^{-4}	4.0×10^{-4}
Tap water at Obuasi	Water	Ingestion	6.6×10^{-4}	5.5×10^{-6}	4.3×10^{-4}	4.6×10^{-4}
		Dermal	2.9×10^{-5}	2.0×10^{-5}	3.1×10^{-5}	6.0×10^{-5}
Kaw	Water	Ingestion	8.6×10^{-4}	2.5×10^{-3}	6.7×10^{-5}	2.4×10^{-4}
		Dermal	1.6×10^{-4}	1.2 × 10–4	4.6×10^{-5}	3.5×10^{-5}
Kwabrafo	Water	Ingestion	5.4×10^{-3}	4.1×10^{-3}	7.1×10^{-2}	3.8×10^{-2}
		Dermal	2.5×10^{-3}	1.9×10^{-3}	3.7×10^{-3}	5.5×10^{-3}
Pompo	Water	Ingestion	9.3×10^{-2}	2.8×10^{-1}	7.2×10^{-3}	2.6×10^{-3}
		Dermal	1.7×10^{-3}	1.3×10^{-2}	4.9×10^{-3}	3.8×10^{-3}
Akapori	Water	Ingestion	2.7×10^{-6}	8.1×10^{-5}	2.1×10^{-5}	7.6×10^{-5}
		Dermal	5.0×10^{-6}	3.9×10^{-5}	1.4×10^{-5}	1.1×10^{-5}
Jimi	Water	Ingestion	1.4×10^{-3}	4.1×10^{-3}	6.1×10^{-3}	3.8×10^{-3}
		Dermal	2.5×10^{-3}	1.9×10^{-3}	7.2×10^{-3}	3.8×10^{-3}
Akyerempe	Water	Ingestion	1.1×10^{-4}	3.4×10^{-4}	8.9×10^{-3}	3.2×10^{-2}
		Dermal	2.1×10^{-4}	1.6×10^{-4}	6.0×10^{-3}	4.7×10^{-2}
Supu	Water	Ingestion	3.5×10^{-3}	5.1×10^{-3}	1.3×10^{-2}	4.8×10^{-2}
		Dermal	1.7×10^{-3}	2.3×10^{-3}	3.0×10^{-2}	6.9×10^{-2}

can be calculated. The potential receptors evaluated in this study are resident adults and children aged between 20–80 years and 2–19 years, respectively.

The intake of As, Cu, and Cd from ingestion of tap, borehole, and surface water in the study area was calculated as follows:

$$\text{Intake}[(\text{mg/kg} - -\text{day})] = [(\text{EPC} \times \text{IR} \times \text{EF} \times \text{ED} \times 10^{-6})/(\text{BW} \times \text{AT})] \qquad (1)$$

where, EPC is Exposure Point Concentration for As, Cu, and Cd in the water samples (mg/l), IR is Ingestion Rate of water samples (mg/day), EF is Exposure Frequency (day-equivalents/year), ED is Exposure Duration (years), BW is Body Weight (kg) and AT is Averaging Time (days). In the same vein, intake of As, Cu, and Cd from dermal contact with tap, borehole, and surface water in study area by resident adults

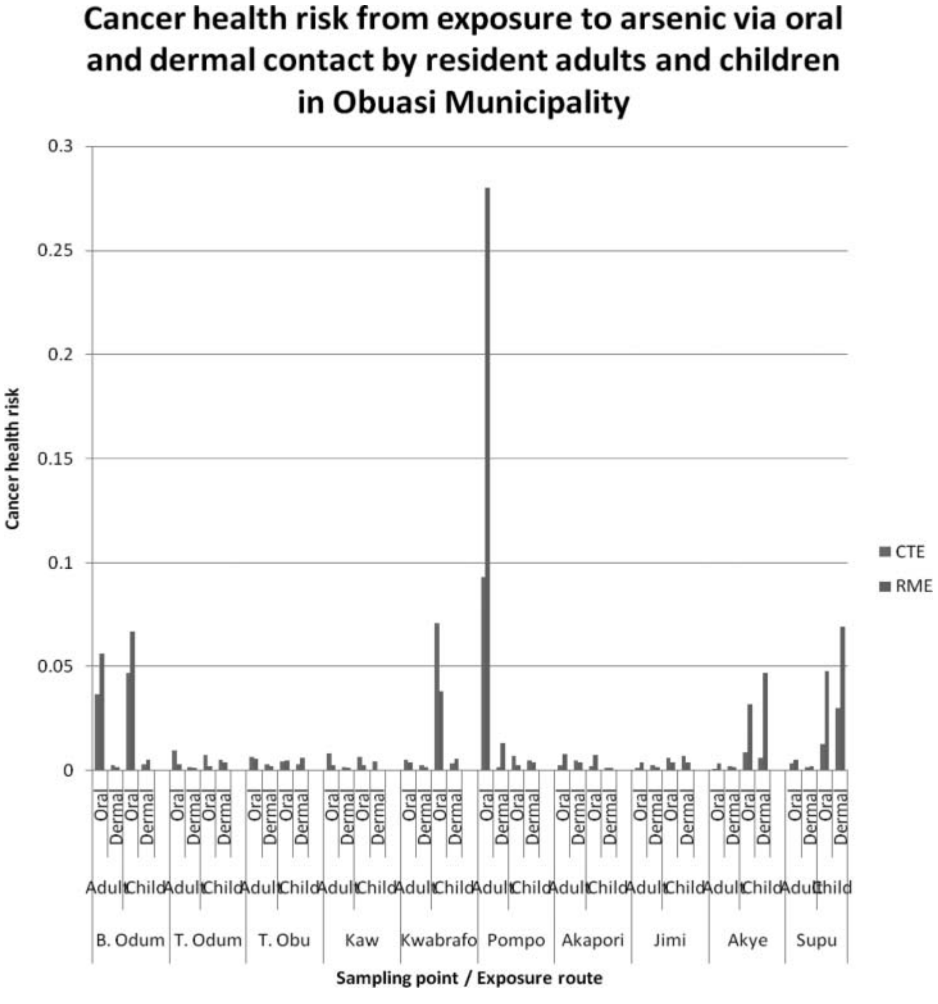

Figure 2. Cancer health risk faced by resident adults and children in Obuasi Municipality.

and children was calculated using the equation below:

$$\text{Intake}[(\text{mg/kg} - \text{day})] = [(\text{EPC} \times \text{Kp} \times \text{SA} \times \text{EF} \times \text{ED} \times 10^{-6})/(\text{BW} \times \text{AT})] \qquad (2)$$

where, Kp is the skin permeability constant, SA is skin surface area exposed to As, Cu, and Cd in the water sample and the other terms in Eq. (2) have the same meanings as described earlier.

The exposure durations (ED) for the acute, sub-chronic, and chronic conditions for both resident adults and children in this study are 3 months, 4 years, and 20 years, respectively, for acute, sub-chronic, and chronic exposures. These values were used based on the fact that resident adults and children are exposed to the same concentrations of As, Cd, and Cu in drinking water.

Toxicity Assessment

The USEPA has provided oral cancer slope factor (CSF_{oral}) and oral reference dose (RfD_{oral}) values in its on-line toxicity database file for use in assessing cancer, chronic, sub-chronic, and acute non-cancer health risk from exposure to As, Cu, and Cd in tap, borehole, and surface water. In line with USEPA risk assessment guidelines for assessing dermal health risks from dermal exposures to the three contaminants, if oral absorption of the chemical of concern is 100%, there is no need to adjust the oral reference dose to calculate the dermal reference dose as well as the dermal cancer slope factor. Since oral absorption of As is about 95% in drinking water

Table 2. Non-cancer health risks results faced by resident children from exposure to arsenic in the study area.

Sampling point	Exposure media	Exposure route	Chronic		Sub chronic		Acute	
			CTE	RME	CTE	RME	CTE	RME
Borehole at Oduamsi	Water	Ingestion	1.2	34	7.1	2.3	5.1	10
		Dermal	0.98	11	0.34	1.1	0.0023	0.015
Tap water at Odumasi	Water	Ingestion	0.040	0.14	5.1	10	5.1	10
		Dermal	0.027	0.21	0.0083	0.015	0.0023	0.015
Tap water at Obuasi	Water	Ingestion	0.02	0.71	2.7	5.3	0.002	0.071
		Dermal	0.013	0.01	0.0043	0.0078	0.0013	0.010
Kaw	Water	Ingestion	0.17	6.3	230	480	0.71	6.3
		Dermal	0.12	0.91	0.38	0.79	0.12	0.91
Kwabrafo	Water	Ingestion	2.7	99	3,700	7,400	2.7	99
		Dermal	1.9	140	6.0	11	1.9	14
Pompo	Water	Ingestion	1.9	67	3,700	730	2,500	5,000
		Dermal	1.3	9.8	6.0	1.2	4.1	7.3
Akapori	Water	Ingestion	1.3	2.0	74	150	74	150
		Dermal	0.055	0.29	0.12	0.21	0.12	0.21
Jimi	Water	Ingestion	0.27	9.8	370	740	370	1.1
		Dermal	0.19	1.4	0.60	1.1	0.60	0.062
Akyerempe	Water	Ingestion	0.23	8.3	170	320	310	620
		Dermal	0.16	1.2	0.35	0.72	0.51	0.90
Supu	Water	Ingestion	0.34	12	460	920	460	920
		Dermal	0.23	1.8	0.75	1.3	0.75	1.3

(USEPA 1999), no adjustment would be made to CSF_{oral}, $RfD_{oral,}$ or sub-chronic RfD_{oral} to assess health risks from dermal exposures to As in tap, borehole, and surface water in the study area. Default values were used from RISC 4.02 software for CSF_{dermal} and RfD_{dermal}, respectively, for assessing health risks associated with exposure to As, Cu, and Cd in drinking water. The values are; As ($CSF_{oral} = 3.0 \times 10^{-4}$; $CSF_{dermal} = 3.0 \times 10^{-4}$ mg/kg-day; $RfD_{oral} = 1.5$ mg/kg-day; $RfD_{dermal} = 1.5$ mg/kg-day), Cd ($RfD_{oral\ and\ dermal} = 5.0 \times 10^{-4}$ mg/kg-day), and Cu ($RfD_{oral\ and\ dermal} = 3.72 \times 10^{-2}$ mg/kg-day). The basis for the use of these values has been explained in the USEPA *Exposure Factors Handbook* (USEPA 1997; 1999).

Risk Characterization

Cancer and non-cancer health risks were evaluated using RISC 4.02 human health risk software. Eqs. (3) and (4) have been incorporated in the software. The estimated intakes and CSFs are combined to calculate excess cancer risk according to the following equation (USEPA 1989):

$$\text{Cancerrisk} = \text{Intake}[(\text{mg/kg} - \text{day})] \times \text{CSF}[(\text{mg/kg})] \tag{3}$$

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

$$\text{HazardIndex} = \text{Intake}[(\text{mg/kg} - \text{day})/\text{RfD}(\text{mg/kg} - \text{day})] \tag{4}$$

Table 3. Non-cancer health risk results from exposure to cadmium by resident children in the study area.

Sampling point	Exposure media	Exposure route	Chronic		Sub chronic		Acute	
			CTE	RME	CTE	RME	CTE	RME
Borehole at Oduamsi	Water	Ingestion	2.3	3.0	200	400	200	400
		Dermal	0.45	1.0	0.33	0.58	0.10	0.58
Tap water at Odumasi	Water	Ingestion	0.0004	0.014	240	490	0.18	6.5
		Dermal	0.00027	0.0021	0.40	0.71	0.12	0.95
Tap water at Obuasi	Water	Ingestion	0.18	6.5	240	490	0.0021	0.011
		Dermal	0.12	0.90	0.40	0.71	0.0015	13
Kaw	Water	Ingestion	0.36	13	480	970	0.36	1.9
		Dermal	0.24	1.9	7.9	1.4	0.24	13
Kwabrafo	Water	Ingestion	0.54	1.9	730	1,500	0.54	19
		Dermal	0.37	2.8	1.2	2.1	0.37	2.8
Pompo	Water	Ingestion	1.2	43	730	1,500	1,600	3,200
		Dermal	0.82	6.3	1.2	2.1	2.6	4.7
Akapori	Water	Ingestion	0.51	18	690	1,400	690	1,400
		Dermal	0.35	2.7	1.1	2.0	1.1	2.0
Jimi	Water	Ingestion	0.23	8.1	300	610	300	610
		Dermal	0.15	1.2	0.50	0.89	0.50	0.89
Akyerempe	Water	Ingestion	0.23	8.2	140	0.026	310	620
		Dermal	0.16	1.2	0.29	0.60	0.50	0.90
Supu	Water	Ingestion	0.42	15	560	1,100	560	1,100
		Dermal	0.28	2.2	0.92	1.6	0.92	1.6

RESULTS AND DISCUSSIONS

The results of cancer health risks for both resident adults and children from exposure to As in drinking water in the Obuasi Municipality have been presented in Table 1.

Cancer health risk is defined as the incremental probability that an individual would develop cancer during his or her lifetime due to chemical exposure under specific exposure scenarios evaluated. The term "incremental" means the risk is more than the background cancer risk by individuals in the course of life. For example, in the United States, approximately one out of four individuals die of cancer, in this case, the cancer background risk is 0.25 or 250,000 in 1,000,000 Americans would die of cancer-related diseases (ACS 2000).

From Table 1, the results of estimated lifetime cancer risk for adult receptors (20–80) residents in Odumasi who are exposed to water from a borehole is 3.7×10^{-2} and 5.6×10^{-2} for oral ingestion route based on CTE and RME parameters, respectively. This means that on the average, 4 and 6 cases of cancer are likely to be recorded via oral exposure pathway in every 100 resident adults in Odumasi by CTE and RME parameters, respectively. For dermal exposure route, we have 2.8×10^{-3} and 1.9×10^{-3} based on CTE and RME parameters, respectively.

Similarly, the results of estimated lifetime cancer health risk for resident children aged 2–19 yrs in Odumasi exposed to water from a borehole is 4.7×10^{-2} (CTE)

Table 4. Non-cancer health risks results faced by resident children from exposure to copper in the study area.

Sample point	Exposure media	Exposure route	Chronic		Sub chronic		Acute	
			CTE	RME	CTE	RME	CTE	RME
Borehole at Oduamsi	Water	Ingestion	3.2	4.3	2.7	5.3	2.7	3.3
		Dermal	0.56	0.098	0.0043	0.0077	0.0043	0.0077
Tap water at Odumasi	Water	Ingestion	0.028	0.0099	0.21	0.42	0.0016	0.056
		Dermal	0.019	0.0014	0.0034	0.0061	0.0011	0.082
Tap water at Obuasi	Water	Ingestion	0.0016	0.0056	0.037	0.074	0.028	0.099
		Dermal	0.0011	0.0082	0.061	0.011	0.019	0.014
Kaw	Water	Ingestion	0.0038	0.14	5.1	10	0.0038	0.020
		Dermal	0.0026	0.020	0.0084	0.015	0.0026	0.14
Kwabrafo	Water	Ingestion	0.044	1.6	59	120	0.038	0.34
		Dermal	0.030	0.23	0.097	0.017	0.070	0.16
Pompo	Water	Ingestion	0.053	1.9	59	120	0.038	0.34
		Dermal	0.036	0.28	0.097	0.013	0.070	0.16
Akapori	Water	Ingestion	0.015	0.56	21	42	21	42
		Dermal	0.011	0.081	0.034	0.061	0.034	0.061
Jimi	Water	Ingestion	0.014	0.50	19	37	19	37
		Dermal	0.0094	0.073	0.030	0.054	0.030	0.054
Akyerempe	Water	Ingestion	0.003	0.11	8.8	16	4.0	8.0
		Dermal	0.002	0.016	0.018	0.037	0.0065	0.012
Supu	Water	Ingestion	0.003	0.036	1.3	2.7	1.3	2.7
		Dermal	0.0068	0.0053	0.0023	0.0039	0.022	0.0039

and 6.7 × 10^{-2} (RME) via oral ingestion route, which means that approximately 5 and 7 children out of 100 children in Odumasi who drink water from a borehole are likely to show symptoms of cancer-related diseases such as skin cancer, cancer of the liver, and so on.

The cancer health risk results obtained from exposure to As by resident children via oral and dermal contact of Rivers Supu, Akyerempe, and Kwabrafo are greater than the result obtained for resident adults, as shown in Figure 2 and Table 1. These values are significant judging from the fact that the children have low body weight coupled with the fact that most of their organs responsible for detoxifying toxic chemicals are not well developed. Hence, they stand a high risk of showing symptoms of cancer-related diseases. The cancer health risk obtained from exposure to As via oral ingestion was found to be greater than that from the dermal exposure route. However, in certain cases cancer health risk results obtained from dermal exposures were found to be higher than the oral route. This may be due to other factors such as heterogenous makeup of the exposed persons. That is, resident adults with very low fatty tissues underneath their skin are likely to experience greater risk to dermal contact with As as compared to the oral ingestion as seen Tables 1 and 3.

The results of non-cancer health risk from exposure to As, Cd and Cu in tap, borehole, and surface water have been presented in Tables 2, 3, 4, 5, 6, and 7. Non-cancer health risks are expressed as hazard indices rather than as probabilities. From Table 2, acute non-cancer health risk from exposure to As via oral contact

Table 5. Non-cancer health risks results faced by resident adults from exposure to arsenic in the study area.

Sample point	Exposure media	Exposure route	Chronic		Sub chronic		Acute	
			CTE	RME	CTE	RME	CTE	RME
Borehole at Oduamsi	Water	Ingestion	2.4	4.4	1.8	5.6	5.1	10
		Dermal	0.048	0.01	0.98	1.1	0.083	0.015
Tap water at Odumasi	Water	Ingestion	1.7	1.5	1.3	2.3	0.0017	0.015
		Dermal	3.1	7.0	0.025	0.053	0.0031	0.007
Tap water at Obuasi	Water	Ingestion	16	3.1	0.25	0.46	0.0004	0.0031
		Dermal	2.7	1.4	0.005	0.001	0.0006	0.0014
Kaw	Water	Ingestion	0.16	1.3	110	200	0.15	1.3
		Dermal	0.27	0.62	0.22	0.46	0.27	0.62
Kwabrafo	Water	Ingestion	2.3	2.1	1,700	3,200	2.7	99
		Dermal	4.3	9.7	3.5	7.3	1.9	14
Pompo	Water	Ingestion	1.6	14	1,700	3,200	1,200	2,200
		Dermal	3.0	6.6	3.5	7.3	2.4	5.0
Akapori	Water	Ingestion	0.047	0.42	35	63	35	0.15
		Dermal	0.086	0.19	0.070	0.15	0.070	0.032
Jimi	Water	Ingestion	0.23	2.7	370	740	170	320
		Dermal	0.43	0.97	0.60	1.1	320	0.72
Akyerempe	Water	Ingestion	0.20	1.8	170	320	150	270
		Dermal	0.36	0.82	0.35	0.72	0.29	0.61
Supu	Water	Ingestion	0.29	2.6	220	390	220	390
		Dermal	0.54	1.2	0.44	0.91	0.44	0.91

to water from the borehole and tap water at Odumasi by resident children is 5.1 (CTE) and 10 (RME). The results show that the probability of resident children in Odumasi experiencing diseases associated with As intoxication is very high.

Similarly, in accordance with USEPA risk assessment guidelines, a hazard index greater than 1.0 means that the probability for the adverse health effects associated with exposure to such a chemical is very high (USEPA 1989; 1997; 2001aa). Hence, in Tables 2, 3, 4, 5, 6, and 7 where the hazard index is greater than 1.0, resident adults and children are prone to the following diseases associated with exposure to As, Cd, and Cu: headache, dizziness, gastrointestinal, skin hyper-pigmentation, kidney failures, and so on.

The toxicity of a given chemical in eliciting its adverse response is affected by several factors such as the chemical composition (salt, free base, anion, *etc.*), physical characteristics (particle size, liquid, or solid), chemical properties (volatility, solubility, *etc.*), the presence of impurities (may alter absorption or toxicity), and the stability of products in the mixture are examples. Similarly, the heterogeneous make up of the exposed persons, that is, the genetic status of the organism, its immunologic status, nutritional status, hormonal status, and so on do affect the way and manner in which the toxic effects of a given chemical may be manifested. This explains the discrepancies in risk results via oral and dermal contact with the contaminated water sources in the study area for acute exposures to Cd by resident children and adults.

Table 6. Non-cancer health risks results faced by resident adults from exposure to cadmium in the study area.

Sample point	Exposure media	Exposure route	Chronic		Sub chronic		Acute	
			CTE	RME	CTE	RME	CTE	RME
Borehole at Oduamsi	Water	Ingestion	94	170	9.4	17	200	400
		Dermal	0.19	0.39	1.9	3.9	0.33	0.58
Tap water at Odumasi	Water	Ingestion	0.16	1.4	110	210	0.16	1.4
		Dermal	0.29	0.64	0.23	0.48	0.29	0.64
Tap water at Obuasi	Water	Ingestion	8.7	16	1.4	2.5	0.0018	0.016
		Dermal	1.8	7.6	0.0027	0.0057	0.0034	0.0076
Kaw	Water	Ingestion	0.31	2.8	230	410	0.31	2.8
		Dermal	0.57	1.3	0.46	0.95	0.57	1.3
Kwabrafo	Water	Ingestion	0.46	4.2	340	620	0.54	19
		Dermal	0.85	1.9	0.69	1.4	0.37	2.8
Pompo	Water	Ingestion	10	93	330	615	760	1,400
		Dermal	1.9	4.4	0.75	2.4	1.5	3.2
Akapori	Water	Ingestion	0.44	3.9	320	590	320	590
		Dermal	0.086	1.8	0.65	1.4	0.65	1.4
Jimi	Water	Ingestion	0.19	1.7	300	610	140	260
		Dermal	0.36	0.80	0.50	0.89	0.29	0.60
Akyerempe	Water	Ingestion	0.20	1.8	140	260	150	260
		Dermal	0.36	0.81	0.29	0.60	0.29	0.61
Supu	Water	Ingestion	0.36	3.2	260	480	260	480
		Dermal	0.66	1.5	0.53	1.1	0.53	1.1

Table 7. Non-cancer health risks results faced by resident adults from exposure to copper in the study area.

Sample point	Exposure media	Exposure route	Chronic		Sub chronic		Acute	
			CTE	RME	CTE	RME	CTE	RME
Borehole at Oduamsi	Water	Ingestion	1.3	2.3	1.3	2.3	2.7	5.3
		Dermal	0.0025	0.0052	0.0025	0.0052	0.0043	0.0077
Tap water at Odumasi	Water	Ingestion	0.0012	0.012	0.099	0.18	0.0013	0.012
		Dermal	0.0025	0.055	0.002	0.0041	0.0025	0.0055
Tap water at Obuasi	Water	Ingestion	0.015	0.021	0.018	0.032	0.0024	0.0021
		Dermal	0.024	0.098	0.0035	0.073	0.0044	0.0098
Kaw	Water	Ingestion	0.003	0.030	2.4	4.4	0.033	0.030
		Dermal	0.006	0.014	0.0049	0.010	0.060	0.014
Kwabrafo	Water	Ingestion	0.038	0.34	28	51	0.044	1.6
		Dermal	0.070	0.16	0.056	0.12	0.030	0.23
Pompo	Water	Ingestion	0.046	0.41	28	51	34	62
		Dermal	0.084	0.019	0.056	0.12	0.068	0.14
Akapori	Water	Ingestion	0.013	0.12	9.8	18	9.8	18
		Dermal	0.024	0.055	0.02	0.041	0.020	0.041
Jimi	Water	Ingestion	0.012	0.11	19	37	8.8	16
		Dermal	0.022	0.049	0.030	0.054	0.018	0.037
Akyerempe	Water	Ingestion	0.025	0.023	8.8	16	1.9	3.4
		Dermal	0.043	0.010	0.018	0.037	0.0039	0.0079
Supu	Water	Ingestion	0.086	0.077	0.64	1.2	0.64	1.2
		Dermal	0.016	0.036	0.0013	0.0027	0.0013	0.0023

CONCLUSION

Given the limitation of routine analytical techniques for measuring total As, Cd, and Cu and the presence of the various forms of these three contaminants in surface, tap, and borehole water, it follows that considerable uncertainty exists regarding the actual toxicities of the various forms of As, Cd, and Cu in water to human beings.

As such, the toxic effect from chronic exposure to As, Cd, and Cu in tap, borehole, and surface water by resident adults and children is very small compared to the acute and sub-chronic exposures. Most of these contaminants in drinking water might have broken down into less toxic compounds.

The results of cancer and non-cancer health risk assessment indicate high prevalence of diseases associated with exposure to As, Cd, and Cu via oral and dermal contact with tap water, borehole, and surface water in the study area. The cancer and non-cancer health risk values from Obuasi Municipality in most cases exceeded the USEPA acceptable cancer health risk range of 1×10^{-4} to 1×10^{-6}, meaning, one case of cancer in every 10,000 to 1,000,000 people and acceptable hazard index of 1.0 (USEPA 1989; 1997).

Comparing the cancer and non-cancer health risk results obtained in this study to the average life expectancy figures of 55.4 years for men and 58.6 years for women in Ghana, it could be inferred that resident adults and children in the study area are likely to have reduced life expectancy figures (GLSS 2003). Government agencies and other stakeholders responsible for providing potable water should act accordingly to provide clean and safe drinking water for residents in the Obuasi Municipality.

ACKNOWLEDGMENT

We are grateful to WACAM and its partners for funding this project.

REFERENCES

Adimado AA and Amegbey NA. 2003. Incidents of cyanide spillage in Ghana, mineral processing and extractive metallurgy. Trans. Inst. Min. Metall. C 112: C1–5

Ahmed K and Carboo D 2000. Speciation of As (III) and As (V) in some Ghanaian gold tailings by a simple distillation method. Water, Air and Soil Pollution 122: 317–326

Akabzaa TM Baneong, Yakubu BK, and Sekyire JS. 2005. Impact of mining activities on water resources in the vicinity of the Obuasi mine. West.Journ. App. Eco. 11: 34–44

Amasa SK. 1975. Arsenic pollution at Obuasi gold mine, Town and surrounding countryside. Environmental Health Perspectives. 12:131–135

American Cancer Society, ACS. 2000. Statistics: Cancer facts and figures. www.cancer.org/statistics/index

Amonoo-Neizer EH and Amekor EMK. 1993. Determination of total arsenic in environmental samples from Kumasi and Obuasi, Ghana. Environ Health Perspect 101(1):46–9

Asante-Duah DK. 1996. Managing Contaminated Sites: Problem Diagnosis and Development of Site Restoration. John Wiley, West Sussex, UK

AWWA, 1998. Standards methods for the examination of waste water. APHA–AWWA–WEF, 20th Ed. Washington DC. pp 320–330

Carboo D and Serfor–Armah Y. 1997. Arsenic in stream and sediments in Obuasi area. Proceeding of the Symposium on the Mining Industry and the Environment. KNUST/IDRC 1997:114–9

Foulkes EC. 1986. Absorption of cadmium. In: Foulkes EC (ed), Handbook of Experimental Pharmacology, vol 80. Springer Verlag, Berlin, Germany pp. 75–100

Hilson G. 2002. The environmental impact of small–scale gold mining in Ghana: identifying problems and possible solutions. Geo. Journ. 168(1): 57–72

GLSS (Ghana Living Standard Survey). 2003. Ghana Demographic and Health Survey. Ghana Statistical Service, Accra, Ghana

IPCS (International Programme on Chemical Safety). 1998. Copper: Environmental Health Criteria 200. World Health Organisation, Geneva, Switzerland

Nelson PO, Thayumanavan P, Azizan MF, *et al.* 2005. Evaluation methodology for Environmental Impact Assessment for industrial wastes used as highway materials: An overview with respect to U.S. EPA's Environmental Risk Assessment Framework. Handbook of Environ Chem 5(1):271–91

NOHSC (National Occupational Health and Safety Commission). 1999. List of Designated Hazardous Substances. Sydney, NSW, Australia

Obiri S, 2007. Determination of heavy metal in water from boreholes in Dumasi in the Wassa West District of Western Region of Republic of Ghana. Environ Monit Assess 130:455–63

Obiri S, Dodoo DK, Essumang DK, *et al.* 2006. Non-cancer health risk assessment from exposure to cyanide by resident adults from the mining operations of Bogoso Gold Limited in Ghana. Environ Monit Assess 118:51–63

Shaikh ZA. and Smith JC. 1976. The biosynthesis of metallothionein in rat, liver and kidney after administration of cadmium. Chem. Biol. Interact 15: 327–336

Smedley PL. 1996. Arsenic in rural ground water in Ghana. J African Earth Sci 22(4):459–70

Tseng P. 1977. Effects and dose-response relationship of skin cancer and black foot disease with arsenic. Environ Health Perspect 19:109–119

Tseng P, Chu HM, and How SW, 1968. Prevalence of skin cancer in an endemic area of chronic arsenicism in Taiwan. Natl Cancer Inst L10(3):453–462

USEPA (US Environmental Protection Agency). 1989. Risk Assessment Guidance for Superfund (RAGS), vol I, Human Health Evaluation Manual (Part A). OWSER Directive 9285. 7-01A. EPA-540/1-89 -002. Office of Emergency and Remedial Response, Washington, DC, USA

USEPA, 1991. Methods of determination of metals in environmental samples. Office of research and development. Cincinnati, Ohio

USEPA. 1997. Exposure Factor Handbook NCEA-W-005. 1995.107. USEPA: Exposure Factors Handbook, vol I: General Factors. Office of Research and Development, Washington, DC, USA

USEPA. 1999b. Risk Assessment Guidance for Superfund, vol I: Human Health Evaluation Manual-Supplemental Guidance (Final Draft). Dermal Risk Assessment. Interim Guidance (Final Draft). Office Of Emergency and Remedial Response, Washington, DC, USA

USEPA. 2001a. Derivation of Acute and Subchronic Oral RfD Doses for Inorganic Arsenic. USEPA Region 8, Kansas City, KS, USA

USEPA. 2001b. Integrated Risk Information System (IRIS). National Centre for Environmental Assessment, Office of Research and Development, Washington, DC, USA