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Assessing environmental exposure and health impacts of gold mining in Ghana

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Understanding the extent to which people are being exposed to environmental contaminants helps to identify those populations which may be disproportionately exposed to the contaminants of potential public health concern. This study represents the first report of a comparison of heavy metal arsenic (As), mercury (Hg), cadmium (Cd), cobalt (Co), copper (Cu), lead (Pb), platinum (Pt), zinc (Zn), and manganese (Mn) concentrations in the blood of residents of mining and non-mining communities in Ghana. Blood sampling, health records, and interviews were used in this study to establish the links between the levels of contaminants and health effects in humans within a mining and non-mining community in Ghana. Results of this study show that mean concentration of As in blood in Tarkwa Nsuaem Municipality/Prestea-Huni Valley District (TNMA/PHVD) (mining communities) and Cape Coast Metropolis (CCMA) (non-mining community) was 18- and 2-fold higher, respectively, than the WHO guideline value of 0.002 mg L⁻¹. The mean concentrations of As, Cd, Pb, Hg, and Mn were elevated up to 20-fold higher in the blood of resident adults and children in TNMA/PHVD than in CCMA. The risk of acute respiratory infections in the exposed populations of TNMA and PHVD were approximately 41- and 12-fold greater than the unexposed group. The risk of diabetes mellitus in the exposed populations of TNMA and PHVD were also approximately 20- and 4-fold higher than the unexposed group. In TNMA and PHVD, 40 blood donors were suffering from hyperkeratosis or pigmentation unlike their CCMA counterparts. Data suggest a potential association between mining activities and the levels of these heavy metals. However, the proportion of anthropogenic contribution to the levels of these metals in the blood remains uncertain. This uncertainty seems to be the umbrella under which both government and mining companies continue to hide, leading to inaction.

Keywords: heavy metals; biomarkers; mining; human health; risk; Ghana

Introduction

Exposure of populations to environmental contaminants and its associated adverse health effects remains a continuous problem for science and policy. Understanding the extent to which populations are exposed to environmental contaminants helps to identify potential public health concerns and those who may be disproportionately exposed to contaminants

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or uniquely vulnerable (Landrigan et al. 2004; Watson and Mutti 2004). Children may have disproportionately heavy exposures to environmental contaminants because infants drink more water, breathe more air, and eat more food per kilogram body weight than adults (Sexton et al. 1992; Materia and Baglio 2005). Further, children may be more vulnerable to some environmental contaminants depending on the stage of development during which exposure occurs (Landrigan et al. 2004; WHO 2006). The environmental public health paradigm has been the traditional approach to establish linkages between human exposure to contaminants and adverse human health effects (Sexton, Callahan, and Ryan 1995; Collier 2003). The approach involves measurements of human exposure to environmental contaminants in the ambient environment (air, water, and land), at the point of human contact, or after contact, and contaminant entry into humans has occurred (Landrigan et al. 2004). The focus is on human biomonitoring, which involves the measurement of human tissues or excreta for direct or indirect evidence of exposure to chemical, biological, or radiological substances.

The use of biological markers (or biomarkers) builds on the more traditional exposure assessment approach, providing more information on the extent to which a contaminant enters, remains, and acts in the body (Engel-Cox et al. 2008). Biomarker information attempts to determine the extent to which a contaminant is present in the body after entering through portals of entry such as eyes, skin, stomach, intestines, or lungs (Wilcox 2005). Given the complex set of factors that govern contaminants that are absorbed and distributed in the body, a direct measurement of the levels of a contaminant or related “marker” in the body offers more information about exposure than measured ambient levels alone (Hu 1998; Hewitt et al. 2003). In general, a biomarker reports the level of a substance or a marker (i.e., the product of an interaction between an agent and some target molecule or cell) present in samples collected from the body or produced by the organism. Biomarkers of exposure measure concentrations of a contaminant, its metabolite(s), or reaction product(s) in body fluids or tissue, most commonly blood or urine. Measurements may also be taken from a variety of other body compartments, such as feces, breast milk, hair, nails, exhaled air, and tissues obtained through biopsy or autopsy. The exposure measure used to answer this question focuses on biomarkers of exposure. Biomarkers of exposure do not predict whether biological alterations and potential adverse health effect might result.

Epidemiological studies of mining environments in developed countries established the toxicity of specific metals and the diseases that are likely to occur when such toxic chemicals are consumed by humans above the recommended threshold doses (Balaan and Banks 1998). A summary of established human health effects from exposure to arsenic (As), cadmium (Cd), cobalt (Co), copper (Cu), manganese (Mn), lead (Pb), platinum (Pt), mercury (Hg), and zinc (Zn) is presented in Table 1.

In Ghana, research on human exposure to contaminants and associated health outcomes is nascent. Research on heavy metals has disproportionately focused on their levels in environmental media. Few studies assessed levels in biota and still fewer studies focused on levels in humans. There is limited research on heavy metals as biomarkers in both mining and non-mining communities in Ghana. Further, such studies focused on only one metalloid (As) in the case of Essumang (2009) or Hg (total and methyl) in the case of Donkor et al. (2006) and Voegborlo et al. (2010). The paucity of research on heavy metals as biomarkers exposes the gaps in knowledge regarding the correlation between the levels of heavy metals in environmental media and concentrations in biota. Therefore, this study aimed at measuring the levels of heavy metals in the blood of residents of a mining and non-mining community and determined whether the reported health effects in the

Table 1. Established human health effects of the selected metals.

Toxic chemicals	Established associated health effects
Cadmium	Respiratory tract infection, lung toxicity, bronchitis, kidney damage, gastrointestinal irritation, nausea, vomiting, diarrhea, pain, metallic taste in the mouth, reproductive, and developmental toxicity.
Copper	Irritation of eyes, mouth, nose; nausea, diarrhea and abdominal pains; dizziness and drowsiness; headache, liver and kidney effects.
Lead	Impaired growth, induces weakness in the fingers, wrist and ankles; increased blood pressure and hypertension; anemia; damage to kidney; spontaneous abortion in women and damage to male reproductive system leading to sterility, low IQ development.
Manganese	Neurotoxin, tremors, lethargy, speech disturbance, mask – like face, psychological disturbance, respiratory effects such as increased incidence of cough and bronchitis and increased susceptibility to infectious lung disease, reproductive/developmental effects such as impotence and loss of libido, low IQ.
Mercury	Kidney damage, low IQ, irritation, nausea, vomiting, pain, ulceration, diarrhea, toxicity to the brain and nervous system, abdominal pains.
Zinc	Gastrointestinal effects, impaired lung functioning, respiratory irritations
Cobalt	Respiratory irritation, diminished pulmonary function, wheezing, asthma, pneumonia, and fibrosis.
Platinum	Watering of the eyes, sneezing, tightness of the chest, wheezing, breathlessness, cough, eczematous and urticarial skin lesions, signs of mucous membrane inflammation.
Arsenic	Cancers of the skin, liver, lung, bladder and blood; upper respiratory infections, damages to the nervous system, skin pigmentation such as hyperpigmentation, keratoses, cerebral neuropathy; gastrointestinal diseases, nausea, vomiting, diarrhea.

Source: Adopted from Obiri et al. (2010); Ogola, Mitullah, and Omulo (2002); Tseng (2005) and USEPA (2001).

mining community are associated with human exposure to the heavy metal contaminants. This study, representing the first report of heavy metal concentrations in the blood of residents in both mining and non-mining communities in Ghana, was intended to fill the aforementioned gaps. In this study, the following heavy metals were considered: As, Cd, Co, Cu, Mn, Pb, Pt, Hg, and Zn. Some of these chemicals may be classified as carcinogens, neurotoxins, irritants; others may produce reproductive failure or birth defects, and disrupt the normal functioning of the endocrine system of human beings (Lu et al. 2005; Table 1).

Materials and methods

Study area

The Tarkwa Nsuaem Municipality (TNMA) and Prestea-Huni Valley District (PHVD) carved out of the Wassa West District occupies the mid-southern part of the Western region of Ghana with Tarkwa as its administrative capital. The population of the two districts is approximately 236,000 and is mainly composed of the indigenous Wassa tribe, but all tribal entities in Ghana are well represented. Subsistence farming is the main occupation of the people although rubber, oil palm, and cocoa are also produced.

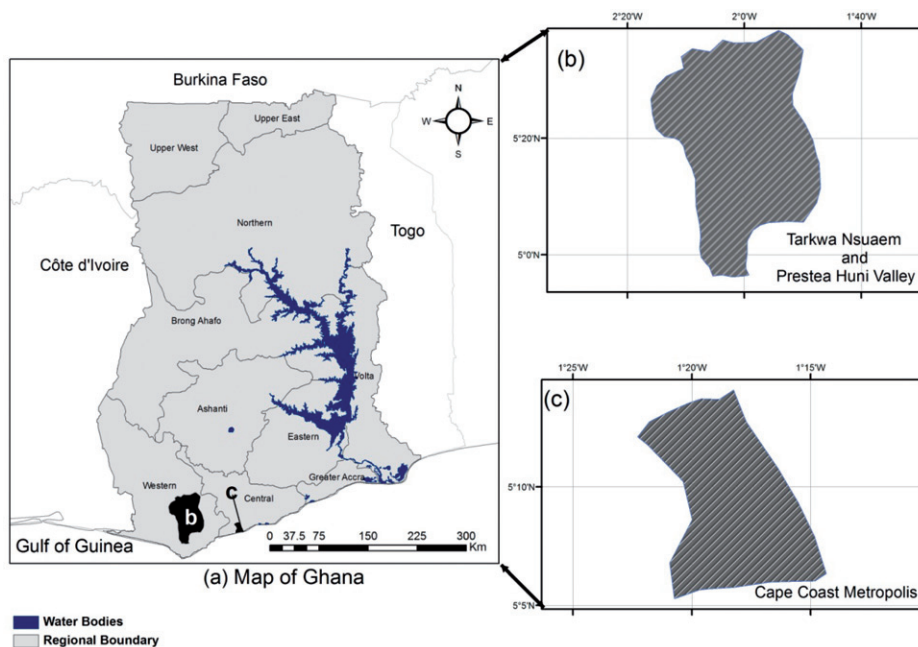


Figure 1. Map of study areas.

Mining is the main industrial activity in the area (Avotri et al. 2002). The area lies within the main gold belt of Ghana that stretches from Axim in the southwest, to Konongo in the northeast. The Cape Coast Metropolis (CCMA) has 71 settlements with a population of 82,291. Out of the 71 settlements in the Metropolitan area, 54% or 76% of them have populations less than 1000 persons and account for only 10% of the metropolis population. The CCMA Assembly area is synonymous with a City District that lies along the Ghanaian coastline. This is because Cape Coast Township is the most populous settlement in the Metropolis with a hierarchy of functions that make it the nerve center of economic activity for both the district and the region (Figure 1). The methods used involved sampling to determine the concentrations of As, Hg, Cd, Co, Cu, Pb, Pt, Zn, Mn in the blood of residents in one mining and non-mining community, health records data collection, and interviews.

Blood sampling

The donors were asked to complete a set of questionnaire which described their lifestyles or habits that might predispose them to exposure to As, Pb, Cd, Mn, Co, Cu, Pt, and Zn. Resident adults and children whose lifestyle or habit predisposed them to these metals in addition to environmental exposures were excluded from this study. In all, 360 blood samples of resident adults ($n = 280$) and children ($n = 80$) in TNMA, PHVD, and CCMA who satisfied the above criteria were taken by lab staff of Tarkwa Nsuaem Municipal Hospital and Cape Coast Metropolitan Hospital. Donors from TNMA and PHVD (contiguous locations) were combined for the reason that the same hospitals and health centers serve both communities. Adult participants were asked to complete consent form,

for children consent were sought from their parents. The blood samples were collected from resident adults and children who voluntarily consented for the blood samples to be taken by the staff of the Laboratory Departments of Tarkwa Municipal Hospital and Cape Coast Metropolitan Hospital, respectively. Donors lived in the study area their entire life.

Lab analysis of heavy metals

The blood samples were analyzed at the National Nuclear Research Institute of Ghana Atomic Energy Commission (GAEC) using the Neutron Activation Analysis (NAA). Three and two milliliters of venous blood samples were collected from the antecubital region of the arm into a Becton Dickinson (BD) vacutainer with and without ethylenediaminetetraacetic acid (EDTA K₂) anticoagulant, respectively. The blood samples without anticoagulant were allowed to clot. The serum was separated by spinning at 2500 g for 5 min with centrifugation. Serum was transferred, whole blood and the serum were labeled and stored in the refrigerator at 4°C at the collection site, and later conveyed to the GAEC for the analysis. In addition, risk assessment of heavy metals was carried out using the USEPA framework which involves four steps, namely hazard identification, exposure assessment; toxicity assessment, and risk estimation/characterization (unreported results).

Blood samples were shaken to ensure uniformity before weighing. Samples were prepared by weighing 500 mg of the sample. Single Standard Reference materials of concentration 10 ppm of the various elements of interest from the National Institute of Standards and Technology and blanks were equally prepared in the same manner as the test samples. Samples and controls were irradiated in the Ghana Research Reactor (GHARR-1) at the GAEC, operating at 15 KW at a thermal flux of $5 \times 10^{11} \text{ n cm}^{-2} \text{ s}^{-1}$. Samples were transferred into irradiation sites via the pneumatic transfer system at a pressure of 0.60 Mpa. The irradiation was categorized according to the half-life of the element of interest. For, Mn and Cu, the samples were irradiated for two min and counted for 10 min. For medium-lived radionuclides like Hg, Cd, As, and Pt, the samples were irradiated for 1 h and delayed for 24 h with 10 min counting. After the irradiation, radioactivity measurement of the induced radionuclide was performed by a PC-based γ -ray spectrometry set-up. It consists of an *n*-type HPGe detector coupled to a computer-based multi-channel analyzer (MCA) via electronic modules. The relative efficiency of detector is 25% and its energy resolution is 1.8 keV at a γ -ray energy of 1332 keV belonging to ⁶⁰Co. Through appropriate choice of cooling-time, detector's dead time was controlled to be less than 10%. Identification of γ -ray of product radionuclide was identified through the energies, and quantitative analysis of the concentration was achieved using the γ -ray spectrum analysis software, ORTEC MEASTRO-32.

Health records data collection

It was hypothesized that there is a relationship between heavy metals used in gold mining operations in the PHVD and TNMA and diseases often reported by residents at the various hospitals, clinics, or health centers. The CCMA was chosen as the control for the reason that it falls within the Tarkwaian and Birimian rock system which is rich in gold ores as PHVD and TNMA, although no active gold mining has been undertaken in the area since 1880 when active mining operations began in PHVD and TNMA areas. In order

Table 2. Population size, number of hospital admissions for TNMA, PHVD, and CCMA.

	Year	TNMA	PHVD	CCMA
Population size	2007	38,229	29,910	77,873
	2008	38,939	30,465	79,319
	2009	39,661	31,030	80,791
	2010	40,397	31,607	82,291
All admissions	2007	26,641	5427	3116
	2008	35,425	7303	6504
	2009	37,843	10,277	7117
	2010	38,269	12,502	9005
Skin diseases	2007	8507	1409	2279
	2008	10,827	2639	3001
	2009	11,894	3544	3005
	2010	7002	4390	3189
Diabetes mellitus	2007	939	250	105
	2008	1626	376	261
	2009	2857	458	300
	2010	1610	670	376
Acute respiratory infection	2007	16,839	3768	837
	2008	22,511	4664	3503
	2009	22,837	6733	4112
	2010	12,594	6890	4533

to understand the prevalence of diseases in the study areas associated with the exposure to metals such as As, Cd, Cu, Co, Pb, Hg, Zn, Pt, and Mn as well as other diseases that are not directly linked with exposure to these metals, diseases reported annually at all the health posts in the study areas as well as the health directorate was extracted from health records. The health records covered the period 2004 to 2010. The records for the period 2004 to 2008 are valid for both TNMA and PHVD as within this period, both areas were part of the Wassa West District. The obtained hospital records covered the period 2004 to 2010 for TNMA, whereas that of CCMA covered the period of 2007 to 2009. In addition, interviews were conducted to ascertain self-reported reproductive health outcomes of 420 women of child-bearing ages within the study area. Risk ratios based on the health records data were computed. Further explanation of this ratio is given by Fung, Luginaah, and Gorey (2007).

Results

Sample characteristics

The demographic characteristics of the respondents are as follows. The gender distribution in CCMA is 49.53% females and 50.47% males. Out of 1100 resident adults interviewed in TNMA, 61.53% are females and 38.47% are males. The results of the collated data on the disease profile in the study areas have been presented in Table 2. In Table 3, the risk of acute respiratory infections in the exposed populations of TNMA and PHVD were approximately 41- and 12-fold higher than the unexposed group. The risk of diabetes mellitus in the exposed populations of TNMA and PHVD were approximately 20- and

Table 3. Risk ratio* of TNMA and PHVD compared to CCMA.

	2007	2008	2009	2010
<i>All admissions</i>				
TNMA	17.42	11.09	10.83	8.66
PHVD	4.53	2.92	3.76	3.61
<i>Skin diseases</i>				
TNMA	7.60	7.35	8.06	4.47
PHVD	1.61	2.29	3.07	3.58
<i>Diabetes mellitus</i>				
TNMA	18.22	12.69	19.40	8.72
PHVD	6.20	3.75	3.97	4.64
<i>Acute respiratory infections</i>				
TNMA	40.98	13.09	11.31	5.76
PHVD	11.72	3.47	4.26	3.96

Note: *Calculated as risk of event in experimental group/risk of event in control group.

4-fold greater than the unexposed group. Risk ratios for all diseases (Table 3) were generally higher in TNMA than PHVD.

Trace metals and heavy metals in the blood of children and adults

The results of the analysis of As, Cu, Pb, Pt, Mn, Co, Zn, Cd, and Hg in the blood of resident adults and children in TNMA and CCMA are shown in Table 4. The WHO permissible limits are shown in parentheses.

Heavy metal exposure and self-reported reproductive health outcomes

One hundred and sixty-seven women refused to answer the question on fertility-related health outcomes, whereas 18 women refused to answer the question on pregnancy-related health outcomes. This was expected as such issues are rather sensitive. The menstrual cycle disorders, irregular menstrual rates of women in TNMA and PHVD were 31.5% and 19.3% respectively, which were approximately 2-fold higher in women from CCMA (15.8% and 10.1%). The incidence of abdominal pain of women from TNMA/PHVD and CCMA were 61.2% and 42.1%, respectively, which was statistically significant. Perceptions of residents regarding reproductive health outcomes are summarized in Table 5. This table shows that infertility rate in married women in TNMA/PHVD is 13.7%, spontaneous abortion rate of 50.8%, and stillbirth rate of 6.8%, respectively, higher than the 3.3%, 10.9%, and 1.9% reported by married women in CCMA. Post-natal infant mortality rate in the first year of women from TNMA/PHVD was 4.7%, which was higher than that of women from CCMA.

Discussion

Relationship between disease patterns and exposure to toxic chemicals

Acute respiratory tract infectious diseases, as well as skin diseases and diabetes mellitus (type II diabetes) are some of the common diseases reported at health posts in the

Table 4. Mean blood heavy metal levels in adults and children in TNMA and CCMA.

Adults						
Metal	TNMA/PHVD (<i>n</i> = 160)			CCMA (<i>n</i> = 120)		
	Mean concentration (mg L ⁻¹)	Min–Max		Mean concentration (mg L ⁻¹)	Min–Max	
Arsenic	0.035 (0.002)	0.001–0.29		0.0032	0.001–0.028	
Mercury	0.043 (0.002)	0.01–0.22		0.011	0.01–0.02	
Cadmium	0.134 (0.0003)	0.01–1.09		0.046	0.01–0.42	
Cobalt	0.035 (–)	0.01–0.35		0.011	0.01–0.03	
Copper	3.30 (0.8)	0.01–24.65		1.80	0.01–7.09	
Lead	0.028 (0.05)	0.01–0.18		0.024	0.01–0.08	
Platinum	0.01 (–)	–		0.01	–	
Zinc	1.405 (6.0)	0.01–7.68		2.911	0.01–6.03	
Manganese	0.195 (0.002)	0.01–0.95		ND	ND	
Children						
Metal	TNMA/PHVD (<i>n</i> = 60)			CCMA (<i>n</i> = 20)		
	Mean concentration (mg L ⁻¹)	Standard deviation	Min–Max	Mean concentration (mg L ⁻¹)	Standard deviation	Min–Max
Arsenic	0.035	0.0045	0.001–0.29	0.0032	0.0003	0.001–0.028
Mercury	0.043	0.0020	0.01–0.22	0.011	0.0050	0.01–0.02
Cadmium	0.134	0.0014	0.01–1.09	0.046	0.0100	0.01–0.42
Cobalt	0.035	0.0005	0.01–0.35	0.011	0.0021	0.01–0.03
Copper	3.30	0.0023	0.01–24.65	1.80	0.0040	0.01–7.09
Lead	0.028	0.0001	0.01–0.18	0.024	0.0005	0.01–0.08
Platinum	0.01	0.0001	–	0.01	0.0001	–
Zinc	1.405	0.0001	0.01–7.68	2.911	0.0001	0.01–6.03
Manganese	0.195	0.0001	0.01–0.95	ND	N/A	ND

Note: ND, not detected.

Table 5. Self-reported prevalence of reproductive health of women of child-bearing ages (*n* = 420).

Reproductive health outcomes	TNMA/PHVD	CCMA
<i>Menstrual-related</i>	<i>n</i> = 102	<i>n</i> = 151
Infertility (%)	13.7*	3.3
Menstrual disorder (%)	31.5*	15.8
Irregular menses (%)	19.3*	10.1
Leucorrhea (%)	43.2	32.6
Stomach aches (%)	61.2*	42.1
Lumbago (%)	52.6	3.2
<i>Pregnancy-related</i>	<i>n</i> = 191	<i>n</i> = 211
Spontaneous abortion (%)	50.8*	10.9
Still birth (%)	6.8*	1.9
Live birth (%)	51.3	59.7
Infant mortality (%)	4.7*	1.4

Note: *Significant at *p* < 0.05.

study areas. The respiratory tract diseases in PHVD and TNMA include: bronchiolitis, bronchitis, croup, pneumonia, laryngitis, and pharyngitis. These diseases are known to be associated with the exposure to Cd, Mn, Hg, and As (USEPA 2001; Ogola, Mitullah, and Omulo 2002), which are directly linked with gold mining operations in those areas because blasting, excavating, milling of the ore bearing rocks by both mining companies and galamsey operators leads to the release of fine particles of sand which contains silicon and other metals such as Mn and As. The acute respiratory diseases recorded in CCMA according to medical records within the metropolis are usually common cold, flu, and in some cases asthma as well as tuberculosis. Skin diseases recorded in CCMA are usually, abrasion or skin lesion, waxing, whereas in PHVD and TNMA it is hyperpigmentation and keratosis. These diseases were found to be linked with the exposure to As via oral and dermal contact (Table 1). Data from Tarkwa Municipal Hospital covers the two administrative districts, as the hospital is the major referral health post serving that area. In TNMA and PHVD, 40 of the blood donors were suffering from hyperkeratosis or pigmentation. The presence of hyperkeratosis in the study population is a signal of the potential effects of As. Arsenic was implicated as a bladder carcinogen in separate studies from Argentina, Chile, and Taiwan (Hopenhayn-Rich, Biggs, and Smith 1998).

Cancer of skin, lung, liver, blood, and breast were reported in TNMA and PHVD, which may be linked to the exposure to elevated levels of As in the blood of resident adults and children in those areas. The outcomes of this study corroborate the results of several other studies, suggesting a relationship between As exposure (Steinmaus et al. 2003; Chen and Ahsan 2004; Smith et al. 2006). Hopenhayn-Rich, Biggs, and Smith (1998) and also found evidence that As ingestion increased the risk of kidney cancer in Cordoba, Argentina. Our results seem to support these findings.

Comparing the results of blood levels of the metals with OPD and in-patients' diseases reported at health facilities in the study areas, the following picture emerges. The high number of cases of acute respiratory as well as upper tract respiratory infection recorded at both OPD and In-Patient Department (IPD) in TNMA and PHVD as compared to CCMA is linked to the presence of elevated levels of As, Cd, Co, and Zn in the blood of resident adults and children, which was attributed to inhalation of toxic fumes in the environment within in the study areas. The non-cancer health risk results (unreported) which regard to systemic diseases such as acute and upper respiratory tract diseases from exposure to the afore-mentioned metals in most cases were greater than 1.

According to the USEPA risk assessment guidelines, a hazard index greater than 1 indicates a high probability of such diseases being associated with those chemicals. Severe respiratory effects, including respiratory distress, hemorrhagic bronchitis, and pulmonary edema, were reported in some cases of acute oral As poisoning at doses of $0.08 \text{ mg As kg}^{-1}$ and above (Fincher and Koerker 1987; Levin-Scherz et al. 1987; Moore, Ficklin, and Johns 1988; Civantos et al. 1995). These effects may be secondary to injury to the pulmonary vasculature. In addition, bronchitis and sequelae (bronchiectasis, broncho-pneumonia) were observed in patients and at autopsy in some chronic poisoning cases (Zaldivar 1974).

High number of cases of type II diabetes (diabetes mellitus) reported at both OPD and IPD in TNMA and PHVD as compared to CCMA was also associated with the presence of elevated levels of As in whole blood and serum of residents. It is noteworthy that there was no data on the number of type II diabetes cases recorded in the CCMA. This may imply that either the prevalence of the disease is not elevated in the area or health officials in the metropolis are not recording it. The high numbers of type II diabetes (diabetes mellitus) in TNMA as well as PHVD can be linked to the presence of high levels of As in

whole/serum blood. Tseng (2005) found that in areas with the most elevated levels of environmental As in drinking water, there is a proposed relationship to type II diabetes, as As may produce resistance to insulin and impaired pancreatic cell functions, including insulin synthesis and secretion. Type II diabetes compromises fertility (Tseng 2005), making As a potential endocrine-disrupting chemical. Another mechanism of As toxicity is through the formation of reactive oxygen and nitrogen species that produce non-specific damage such as oxidative damage to DNA and lipid peroxidation that contribute to reproductive problems (Valko, Morris, and Cronin 2005). There are low birth weight infants, more spontaneous abortions, and congenital malformations in female employees and women living close to Cu smelters, as reported in Sweden and Bulgaria (Nordstrom, Beckman, and Nordenson 1979; Tabacova et al. 1994).

The relatively high number of cancer diseases reported by in-patients at health posts in TNMA and PHVD from the period January 2004 to June 2010 is in agreement with the results obtained from the cancer risk assessment. The cancer health risk estimates in most cases exceeded the acceptable USEPA cancer health risk range of 1×10^{-4} to 1×10^{-6} where 1 case of cancer-related disease in every 10,000 people to 1 case of cancer in every 1,000,000 people. Cancer health risk resulted from the oral ingestion of water from the borehole in Dumasi is 7.1×10^{-2} (by CTE parameters). This indicates that approximately seven resident adults in Dumasi are likely to suffer from various cancer-related diseases by ingesting As-contaminated water. This result confirms the cancer cases that have been reported in health posts in the area. Despite these findings, there are still pertinent but unanswered questions. Is there a causal or by chance relationship between the metals and specific diseases prevalent in the mining communities?

It should be noted that as a result of the complexities in establishing causality, these results need to be interpreted with caution. Nonetheless, the association signals a potential problem with adverse health effects in these areas. For instance, the instrument used in this study measured the total concentrations of As, Cd, Pb, Hg, and Mn in the blood of donors. It fails to distinguish between geogenic and anthropogenic sources. Levels of heavy metals in blood are made of up both anthropogenic and natural contribution. This implies that the proportion of the concentration that is attributable to anthropogenic factors remains uncertain. To date, no research in Ghana has documented the levels of heavy metals in the blood of residents before mining operations commenced in these communities centuries ago. This fact therefore leaves a caveat in terms of making definitive causal statements on mining as a precursor of the current levels of these metals in the blood of residents. The levels of heavy metals in the blood of residents also reflect multiple exposure routes and pathways. Consequently, it is difficult to ascertain how much of the heavy metals in blood contributed by the different exposure routes (dermal contact, ingestion, inhalation, oral) and/or pathways (air, water, soil, sediment). However, it needs to be borne in mind the fact that all the sampled metals exceeded the WHO guidelines for adverse effects. Further, the hyperkeratosis or pigmentation observed in blood donors cannot conclusively be attributed to As alone, although this metal is known to induce these diseases. It is possible that these dermal characteristics have multiple causes. In addition, some individuals may never show these symptoms because of latency factors. Confounding factors such as those that are occupation-related may enhance uncertainty of the results. This uncertainty is further exacerbated when genetic and other variables are considered. For most prospective studies, as in this case, it is difficult to recruit and screen for volunteers with the same background (age, diet, education, geography, etc.), and in historical studies there can be similar variability.

Knowledge gaps and implications for decision-making and policy

In the face of these scientific uncertainties, policy decisions still need to be taken. The results of this study remain equivocal. Yet, there is a plausible link between the diseases reported and the levels of metals detected in the blood. Consequently, the results are still relevant for decision-making. In this particular context, dealing with this situation involves hard political pressure, disputed values, and high decision stakes. The dominant belief in science for policy inquiries is that inappropriate control of environmental risks is due only to insufficient scientific knowledge. However, this ignores both the socioeconomic influence on the construction of the scientific evidence and the influence of political contexts on the use of such evidence for communication and action. Stakeholders can strategically use science in public debates on causality (Hellström 1996; Van der Sluijs 2006). In some cases, the existence of contradictory expertise may be the result of a “manufactured uncertainty,” which is intended to favor prolongation of the debate (Michael 2005; Maxim and van der Sluijs 2007). The consequence is mistrust, conflict, and low chances for mutually respectful dialogue among interested parties.

Conclusion

This article attempted to show that establishing causal links between environmental exposure and adverse health effects is a complex endeavour. Concentration of arsenic in the whole blood in Tarkwa Nsuaem Municipality/Prestea-Huni Valley District (TNMA/PHVD) and Cape Coast Metropolis (CCMA) was significantly higher than the WHO guideline value. This work represents the first report of heavy metal concentrations in the blood of residents of mining and non-mining communities in Ghana. The mean concentrations of arsenic, cadmium, lead, mercury and manganese were also up to twenty times higher in blood serum of resident adults and children in TNMA/PHVD than in CCMA. Yet, we are uncertain of the respective contribution of anthropogenic and natural sources to these elevated concentrations. Consequently, there is the need to implement long term epidemiological studies particularly cohort studies in the mining communities to enhance our understanding of the linkages between adverse human health effects and gold mining. In seeking to establish causality there is the need to adopt robust methods such as multi-methods and systems approaches and also involve interested but varied stakeholders in the design and implementation of research projects. This will ensure that they contribute to a more nuanced understanding of the issues at stake. This will likely inform policy decisions and provide a framework for dealing with conflicting situations in mining communities.

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References

- Avotri, T.S.M., N.A. Amegbey, M.A. Sandow, and S.A.K. Forson. 2002. *The health impact of cyanide spillage at Gold fields Ghana Ltd.* May, Tarkwa: Funded by Goldfields Ghana Limited (GFGL).
- Balaan, M.R., and D.E. Banks. 1998. Silicosis. In *Environmental and occupational medicine*. 3rd ed, ed. W.N. Rom, 435–48. Philadelphia: Lippincott-Raven.

- Chen, Y., and H. Ahsan. 2004. Cancer burden from arsenic in drinking water in Bangladesh. *American Journal of Public Health* 94: 741–4.
- Civantos, D.P., A. Lopez Rodriguez, J.M. Aguado-Borruey, and J.A. Narvaez. 1995. Fulminant malignant arrhythmia and multiorgan failure in acute arsenic poisoning. *Chest* 108: 1774–5.
- Collier, T.K. 2003. Forensic ecotoxicology: Establishing causality between contaminants and biological effects in field studies. *Human and Ecological Risk Assessment* 9: 259–66.
- Donkor, A.K., J.C. Bonzongo, V.K. Nartey, and D.K. Adotey. 2006. Mercury in different environmental compartments of the pra river basin, Ghana. *Science of the Total Environment* 368: 164–76.
- Engel-Cox, J.A., B.V. Houten, J. Phelps, and S.W. Rose. 2008. Conceptual model of comprehensive research metrics for improved human health and environment. *Environmental Health Perspectives* 116: 583–92.
- Essumang, D.K. 2009. Levels of arsenic in human hair as biomarkers of arsenic exposure in a mining community in Ghana. *Bulletin of the Chemical Society of Ethiopia* 23: 275–80.
- Fincher, R.M.E., and R.M. Koerker. 1987. Long-term survival in acute arsenic encephalopathy follow-up using newer measures of electrophysiologic parameters. *American Journal of Medicine* 82: 549–52.
- Fung, K.Y., I. Luginaah, and K.M. Gorey. 2007. Impact of air pollution on hospital admissions in Southwestern Ontario, Canada: Generating hypotheses in sentinel high-exposure places. *Environmental Health* 6: 18.
- Hellström, T. 1996. The science-policy dialogue in transformation: Model-uncertainty and environmental policy. *Science and Public Policy* 23: 91–7.
- Hewitt, L.M., M.G. Dub, J.M. Culp, D.L. MacLatchy, and K.R. Munkittrick. 2003. A proposed framework for investigation of cause for environmental effects monitoring. *Human Ecological Risk Assessment* 9: 195–211.
- Hopenhayn-Rich, C., M.L. Biggs, and A.H. Smith. 1998. Lung and kidney cancer mortality associated with arsenic in drinking water in Cordoba, Argentina. *International Journal of Epidemiology* 27: 561–9.
- Hu, H. 1998. Bone lead as a new biologic marker of lead dose: Recent findings and implications for public health. *Environmental Health Perspectives* 106: 961–7.
- Landrigan, P.J., C.A. Kimmel, A. Correa, and B. Eskenazi. 2004. Children's health and the environment: Public health issues and challenges for risk assessment. *Environmental Health Perspectives* 112: 257–65.
- Levin-Scherz, J.K., J.D. Patrick, F.H. Weber, and C.J. Garabedian. 1987. Acute arsenic ingestion. *Annals of Emergency Medicine* 16: 702–4.
- Lu, J., G. Getz, E.A. Miska, E. Alvarez-Saavedra, J. Lamb, D. Peck, A. Sweet-Cordero, et al. 2005. MicroRNA expression profiles classify human cancers. *Nature* 435: 834–8.
- Materia, E., and G. Baglio. 2005. Health, science and complexity. *Journal of Epidemiology and Community Health* 59: 534–5.
- Maxim, L., and J.P. van der Sluijs. 2007. Uncertainty: Cause or effect of stakeholders' debates? Analysis of a case study: The risk for honeybees of the insecticide Gaucho®. *Science of the Total Environment* 376: 1–17.
- Michael, D. 2005. Industry groups are fighting government regulation by fomenting scientific uncertainty: Doubts is their product. *Scientific American* 292: 96–101.
- Moore, J.N., W.H. Ficklin, and C. Johns. 1988. Partitioning of arsenic and metals in reducing sulfidic sediments. *Environmental Science and Technology* 22: 432–7.
- Nordstrom, S., L. Beckman, and I. Nordenson. 1979. Occupational and environmental risks in and around a smelter in northern Sweden. Part V: Spontaneous abortion among female employees and decreased birth weight in their offspring. *Hereditas* 90: 291–6.
- Obiri, S., D.K. Doodoo, D.K. Essumang, and 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: 651–65.

- Ogola, J.S., W.V. Mitullah, and M.A. Omulo. 2002. Impact of gold mining on the environment and human health: A case study in the Migori Gold Belt, Kenya. *Environmental Geochemistry and Health* 24: 141–58.
- Sexton, K., M.A. Callahan, and E.F. Ryan. 1995. Estimating exposure and dose to characterize health risks: The role of human tissue monitoring in exposure assessment. *Environmental Health Perspectives* 103, Suppl. 3: 13–29.
- Sexton, K., S.G. Selevan, D.K. Wagener, and J.A. Lybarger. 1992. Estimating human exposures to environmental pollutants: Availability and utility of existing databases. *Archives of Environmental Health* 47: 398–407.
- Smith, A.H., G. Marshall, Y. Yuan, C. Ferreccio, J. Liaw, O. von Ehrenstein, C. Steinmaus, N.M. Bates, and S. Selvin. 2006. Increased mortality from lung cancer and bronchiectasis in young adults after exposure to arsenic in utero and in early childhood. *Environmental Health Perspectives* 114: 1293–6.
- Steinmaus, C., Y. Yuan, N.M. Bates, and A.H. Smith. 2003. Case-control study of bladder cancer and drinking water arsenic in the western United States. *American Journal of Epidemiology* 158: 1193–201.
- Tabacova, S., D.D. Baird, L.D. Balabaeva-Lolova, and I. Petrov. 1994. Placental arsenic and cadmium in relation to lipid peroxides and glutathione levels in maternal–infant pairs from a copper smelter area. *Placenta* 15: 873–81.
- Tseng, G.H. 2005. The potential biological mechanisms of arsenic-induced diabetes mellitus. *Toxicology and Applied Pharmacology* 197: 67–83.
- USEPA. 2001. *Integrated risk information system (IRIS)*. Washington, DC: National Centre for Environmental Assessment, Office of Research and Development.
- Valko, M., H. Morris, and M.T.D. Cronin. 2005. Metals, toxicity and oxidative stress. *Current Medicinal Chemistry* 12: 1161–208.
- Van der Sluijs, J. 2006. Uncertainty, assumptions, and value commitments in the knowledge base of complex environmental problems. In *Interfaces between science and society*, eds. Á. Guimarães Pereira, S. Guedes Vaz, and S. Tognetti, 67–84. London: Greenleaf.
- Voegborlo, R.B., A. Matsuyama, A.A. Adimado, and H. Akagi. 2010. Head hair total mercury and methylmercury levels in some Ghanaian individuals for the estimation of their exposure to mercury: Preliminary studies. *Bulletin of Environmental Contamination and Toxicology* 84: 34–8.
- Watson, W.P., and A. Mutti. 2004. Role of biomarkers in monitoring exposures to chemicals: Present position, future prospects. *Biomarkers* 9: 211–42.
- Wilcox, B.A. 2005. Emerging infectious diseases: Bridging the divide between biomedical and bioecological science. *EcoHealth* 2: 167–8.
- World Health Organization. 2006. Principles for evaluating health risks in children associated with exposure to chemicals. *Environmental Health Criteria* 237: 10–330.
- Zaldivar, R. 1974. Arsenic contamination of drinking water and foodstuffs causing endemic chronic poisoning. *Beitrag zur pathologischen Anatomie und zur allgemeinen Pathologie* 151: 384–400.