Exposure to Toxic Metals (As, Cd, Hg, Pb) in Two Artisanal and Small-scale Gold Mining (ASGM) Areas in Zimbabwe

Stefan Rakete
LMU University Hospital
Given Moonga
LMU University Hospital
Anna-Maria Wahl
LMU University Hospital
Viola Mambrey
LMU University Hospital
Dennis Shoko
Tailjet Consultancy
Dingani Moyo
University of the Witwatersrand
Shamiso Muteti-Fana
University of Zimbabwe
Myriam Tobollik
Federal Environmental Agency: Umweltbundesamt
Nadine Steckling-Muschack
LMU University Hospital
Stephan Böse-O'Reilly (stephan.boeseoreilly@med.uni-muenchen.de)
Institute and Clinic for Occupational-, Social- and Environmental Medicine, LMU University Hospital, Ziemssenstr. 1, D-80336 Munich, Germany
https://orcid.org/0000-0003-0204-3103

Research

Keywords: Arsenic, cadmium, lead, mercury, toxic metals, artisanal and small-scale gold mining

Posted Date: September 18th, 2020

DOI: https://doi.org/10.21203/rs.3.rs-76679/v1

Abstract

Background: People living and working in ASGM areas are exposed to toxic metals such as arsenic (As), cadmium (Cd), mercury (Hg) and lead (Pb). Whereas Hg is purposely used to extract the gold from the ore, the other toxic metals can be liberated from the ore during the mining process. These metals can contaminate drinking water and food and are thus, a source of exposure for people living in these mining areas. However, there is limited to no data about the exposure to toxic metals of people living in ASGM areas. Therefore, the purpose of this study was to conduct biomonitoring of people identifying themselves as artisanal gold miners.

Methods: Blood and urine were collected from 207 people living and working in two ASGM areas in Zimbabwe. Hg in whole blood and urine was analyzed by direct mercury analysis, Pb in blood as well as As and Cd in urine were analyzed by GF-AAS. The results were evaluated by descriptive analysis and correlated with the location, age, fish consumption, years living and working in the area.

Results: Median **As** level in urine was 10.0 µg/l (range from below limit of detection to 460 µg/l). Median **Cd** level in urine was 0.3 µg/l (range from below limit of detection to 11.4 µg/l). Median **Hg** levels in blood / urine were 2.7 / 5.2 µg/l (range from 0.2 to 167 µg/l / 0.1 to 612 µg/l). Median **Pb** level in blood was 19.9 µg/l (range from 6.6 to 276 µg/l). As expected, the Hg levels were frequently above international reference values (e. g. NHANES, blood: 34%, urine: 69%). For Pb and Cd, a considerable number of participants (32% and 22% respectively) were above the reference values (NHANES / National Health and Nutrition Examination Survey) and for As 33% of the participants were above the reference value (UBA/ German Environment Agency).

Conclusions: Hg levels were, as expected in ASGM areas, mainly above reference and threshold values. A high proportion of As, Cd and Pb levels were also above reference levels. Therefore, the exposure to toxic metals in the two ASGM areas in Zimbabwe is relevant for public health.

1. Background

1.1. Toxic Metals in Artisanal and Small-Scale Gold Mining (ASGM)

ASGM is a mining-based livelihood strategy in many developing countries rich in gold resources, counting about 15 million miners globally [1]. While there are also a few low-end formal gold miners, it is predominantly an informal, poverty-driven, poorly resourced, comparatively inefficient and transient sector. ASGM in Zimbabwe has been growing very rapidly over the last forty years. This growth was even faster over the last two decades, from an estimated 300,000 miners in 2000 to over 1.5 million today [2, 3]. This has been due to a variety of factors including a persistently shrinking economy across multiple industries along with recurrent droughts during the last two to three decades. Consequently, there has been an inevitable growth in terms of tonnages mined and processed as well as gold produced. At the same time, this is also reflected in the amounts of process chemicals applied, new rock surfaces exposed to weathering and release of various elemental species. This led to the worsening of adverse environmental and public health impacts due to the release of toxic metals, mainly mercury (Hg), but also others such as arsenic (As), cadmium (Cd) and lead (Pb), which are geochemically associated with the gold hosting sulphide ores [4, 5].

More than 90% of gold deposits in Zimbabwe are associated with the largely mafic/basic and cratonic greenstone belts, while the rest is located within the Limpopo Mobile Belt to the south and the Lomagundi meta-sediments in the north. The genesis of most gold deposits in Zimbabwe (> 75%) involved the generation of superheated (> 2000 °C) aqueous hydrothermal sulphide complexes at depth, in the upper mantle to lower crust. Gold is mainly transported in solution as aqueous complexes of hydrogen sulphides/bi-sulphides and chlorides as well as metal complexes [6]. Various elements, and especially heavy metal elements, have an affinity for dissolved hydrogen sulphides and chlorides in hydrothermal ore-forming solutions. This includes, but is not limited to, As, silver (Ag), Cd, Hg, Pb, selenium (Se), antimony (Sb), tellurium (Te), thallium (Tl) and zinc (Zn). These metals and many others are commonly associated with gold ores and are routinely used as pathfinders in gold exploration geochemistry [7]. Most of these metals have densities above five g/cm³ and are called heavy metals. Those with increased toxicity are currently referred to as toxic metals. Toxic metals naturally occur at relatively low concentrations. However, mining, crushing and milling processes, which are used to extract the gold, can result in the increased liberation of toxic metals. The gold is collected at the end of the concentration process as the high-value target mineral, while the other toxic metals end up in the tailings dumps at mining locations. Several oxidation and reduction reactions during mineral processing and within the tailings dumps result in the formation of various compound species as well as the precipitation of native metals. The World Health Organization has classified As, Cd, Hg, and Pb among the top ten chemicals of public health concern [8]. Therefore, this study focused on the biomonitoring of these toxic metals in people that identified themselves as gold miners in two ASGM areas in Zimbabwe to evaluate their actual exposure.

1.2. Toxicity of Arsenic, Cadmium, Mercury and Lead

Arsenic (As) has multiple oxidation states, with the inorganic species of As(III) and AS(V) being the toxicologically most relevant. Exposure pathways include the consumption of contaminated drinking water and food, but are also emitted by copper smelters (inhalation). Acute As poisoning is associated with nausea, vomiting, abdominal pain and diarrhea along with the occasional occurrence of encephalopathy and peripheral neuropathy. Furthermore, paresthesia in the limbs and, in severe cases, polyneuropathy is a frequent symptom of exposure to inorganic As. Endpoints of chronic exposure to inorganic As are various forms of cancer (skin, bladder, lung), peripheral neuropathy, cardiovascular disease, and in exposed children neurobehavioral effects. The mechanisms of As toxicity include generation of reactive oxidative species, DNA damage, neuronal degradation and inhibition of enzymes. [9–11]

Cadmium (Cd) can be taken up via the lungs, e.g. by smoking, and the gastrointestinal tract (drinking water and food). Chronic exposure to Cd can cause renal failure, musculoskeletal disorders, neurological disorders, anosmia, male reproduction system problems, and cancer (lung). Cd can directly interact with macromolecules in mammalian cell and is associated with oxidative stress and DNA damage. Furthermore, it is also known to cause an imbalance of Zn, Mg, and Cu in the body by mimicking or displacing these essential physiological ions. [12–14]

Mercury (Hg) uptake strongly depends on its species. Vaporized elemental Hg is easily absorbed by inhalation. The majority of ingested organic Hg, e.g. methylmercury, is absorbed in the gastrointestinal tract, whereas the uptake of inorganic Hg is relatively low. Mercury has a high affinity for sulfhydryl groups and can therefore react with proteins and non-protein thiols, e.g. glutathione. This causes reduced activity of enzymes and oxidative stress. As Hg dominantly affects the nervous system, chronic exposure to Hg leads to ataxia, weakness, tremors, convulsions, sensory and motor impairment as well as emotional and behavioral changes. Furthermore, adverse effects on the kidney, the cardiovascular system and the immune system have been reported. [15–18]

Lead (Pb) has major pathways, namely atmospheric dust, contaminated food and water, and, in the past, automobile emissions. Pb is readily absorbed in the lungs, but less efficient in the gastrointestinal tract. It accumulates in the liver, kidneys, and bone tissue. Pb causes the inhibition of multiple enzymes such as δ-aminolaevulinic acid dehydratase, a key enzyme in hemoglobin synthesis. Furthermore, it causes oxidative stress and epigenetic alterations. Chronic exposure to Pb affects the hemoglobin synthesis, the cardiovascular and central nervous system, the kidneys, the fertility and is considered to cause cancer. During pregnancy, it can also affect fetal growth. Increased exposure of fetuses, children and adolescents to Pb is associated with reduced cognitive capacities and intelligence. [19–21]

1.3. Toxic Metals in other Mining Areas

As exposure in ASGM has been documented in Tanzania where only 10.6% respondents knew about As toxicity [22]. These were mostly miners who had primary education and above. However, this study focused on the knowledge and perceptions regarding Hg and As toxicity and did not measure the levels of these metals in biospecimens. In a Ghanaian study, where urinary As levels were analyzed in 57 males living in a small-scale gold mining community, 34% of the cases had As levels above 100 µg/l [23], which is above US health guidelines [24]. Further studies from Ghana confirm that As levels are increased in mining areas [25–27].

Cd was analyzed in Ghana in the urine of residents of a small-scale gold mining community [28]. The geometric mean of the 57 male residents for Cd was 0.38 µg/l.

Hg exposure in ASGM was described in a pooled analysis, including 17 studies focused on Hg body burden in ASGM communities worldwide [29]. The pooled analysis revealed a central median Hg concentration in urine of 5.9 µg/l and an upper median concentration of 188 µg/l. For blood, the central median Hg concentration was 10.9 µg/l. In a study from Ghana, Hg was measured in the urine of amalgam smelting miners [30]. The results showed a median Hg concentration of 6.56 µg/l. Exposure to mercury of people living in ASGM areas in Zimbabwe has been studied intermittently [31]. Most studies explore exposure to Hg, showing increased levels of Hg within the mining communities, especially for miners. In Zimbabwe in 2004 the median mercury urine level was 36.6 µg/l, respectively 25.8 µg/g crea) and in blood 10.2 µg/l for miners [32].

Pb levels in ASGM areas are a specific problem in Nigeria. In 2010, ASGM communities in North-western Nigeria experienced a severe lead poisonings due to the processing of heavily contaminated ore [33–37]. One study showed that all children below five years of age were poisoned (blood Pb levels ≥ 10 µg/dl) and 97% of the 204 children had blood lead levels ≥ 45 µg/dl [37].

1.4. Study Aim

The data about the exposure to toxic metals of people living in ASGM areas is clearly still limited. The purpose of this study was to analyze As, Cd, Hg and Pb in two mining areas in Zimbabwe.

2. Materials And Methods

2.1. Study Information

2.1.1. Study Design

This cross-sectional study was conducted within two weeks (18th of March 2019 to the 29th March 2019) at two hospitals in the Kadoma and Shurugwi Districts (Zimbabwe), respectively. Kadoma and Shurugwi are old gold mining towns. In Zimbabwe, the largest gold deposits and the highest density of miners can be found in Kadoma [5]. Shurugwi is primarily known for chrome mining, with a growing trend for ASGM activities. Official data on the number of miners in Kadoma and Shurugwi do not exist.

Inclusion criteria for participation were a minimum age of 18 years and that the participants worked for at least one month as artisanal and small-scale miners. There was no preselection regarding the participants' sex as well as state of physical health. Participants were recruited using snowball sampling where participants recruit further participants among their colleagues. This sampling technique was used due to a widespread and hard-to-reach target population. A per diem of US $ 5 was provided for participants to compensate for the loss of income on the study day. The study centers were located at the hospitals in Kadoma and Shurugwi to minimize contamination of the human biomonitoring samples. Each participant signed an informed consent form and material transfer agreement, prior to the data and sample collection. All documents were available in the three main languages English, Shona and Ndebele spoken in Zimbabwe. Altogether, 206 participants consented to participate (130 from Kadoma and 76 from Shurugwi). Participation in the study was voluntary. Participants were asked to fill out questionnaires concerning general information on demographics and exposure risks factors and undergo medical examination concerning symptoms of chronic Hg intoxication. To ensure a confidential analysis, the samples and data were pseudonomized. More information about the study was previously published [31].

2.1.2. Ethical Clearance

Approval for conducting the study was gained from the Ministry of Health in Zimbabwe and the respective local and regional authorities. The Ethics Committee of the Medical Research Council and the Zimbabwe Research Council (MRCZ / A / 2367, September 26, 2018 and February 25, 2019) and the Ludwig Maximilians University of Munich (18–421, October 15, 2018) approved the study protocols and gave their permissions. In agreement with the Helsinki Declaration of Ethics Code for experiments with human subjects the study was performed.

2.2. Methods

2.2.1. Urine and Blood Collection

All sample containers were labelled with the participant's code for future allocation. Spot urine samples were collected using disposable urine collection cups. For transport and analysis, aliquots of the urine samples were transferred into a urine monovette (Sarstedt®). To prevent bacterial growth and degradation, the samples were acidified with nitric acid to a pH of approximately 2, which was tested with a pH strip. Trained health professionals took venous blood samples into 7 ml Lithium-Heparin-coated tubes for trace metal analyses (Sarstedt®) from all participants. All samples were continuously stored at 4 °C. Once located in the laboratory, samples were stored at − 18 °C.

2.2.2. Analysis of Trace Metals in Urine and Blood

All samples were at least analyzed in duplicate after thawing on a roll mixer. For quality control ClinChek® -Control (Recipe, Munich, Germany) reference materials for whole blood and urine were used. Results below the limit of detection were divided by 2 and further analysis was performed with those modified values. The samples of one participant had to be excluded from analysis due to contamination.

Analysis of Total **As** in Urine

Total As in urine was analyzed by graphite furnace atomic absorption spectroscopy (GF AAS) at a detection wavelength of 193.7 nm. Urine samples were diluted six fold with 0.01% Triton-X in 0.13% nitric acid. 20 µl of this dilution were automatically pipetted into the graphite tube of the GF-AAS (AAnalyst 600, Perkin Elmer, Rodgau, Germany). 5 µg Pd (as $Pd(NO_3)_2$) and 3 µg $Mg(NO_3)_2$ were added as matrix modifiers. The furnace program was according to the recommendations of the manufacturer. Quantification based on the standard addition method. In detail, 10 and 20 pg As were directly added to the sample in the graphite tube, respectively. The limit of detection (LOD) was at 0.5 µg/l.

Analysis of **Cd** in Urine

Cd in urine was analyzed by graphite furnace atomic absorption spectroscopy (GF AAS) at a detection wavelength of 228.8 nm. Urine samples were diluted fourfold with 0.01% Triton-X in 0.13% nitric acid. 20 µl of this dilution were automatically pipetted into the graphite tube of the GF-AAS (AAnalyst 600, Perkin Elmer, Rodgau, Germany). 50 µg $NH_4H_2PO_4$ and 3 µg $Mg(NO_3)_2$ were added as matrix modifiers. The furnace program was according to the recommendations of the manufacturer. Quantification based on the standard addition method. In detail, 0.5 and 1 pg Cd were directly added to the sample in the graphite tube, respectively. The LOD was at 0.5 µg/l.

Analysis of **Hg** in Urine and Blood

Urine and blood specimen were analyzed without any pre-processing by direct Hg analysis using a DMA80-evo instrument (MLS-Mikrowellen GmbH, Leutkirch, Germany). 100 µl of the sample were directly pipetted into quartz tubes for analysis. All of the following steps were part of the automated Hg analysis. One sample at a time was transferred into the oven where it was incinerated under an oxygen atmosphere at 750 °C. After the catalytic removal of matrix components, the Hg released from the sample was concentrated in an amalgamator. Finally, Hg was released by heating the amalgamator and detected by atomic absorption at 253.5 nm. The quantification was based on an external calibration. The LOD for total Hg in urine and blood was at 0.05 µg/l.

Analysis of **Pb** in Blood

Pb in blood was analyzed by graphite furnace atomic absorption spectroscopy (GF AAS) at a detection wavelength of 193.7 nm. Blood samples were diluted tenfold with 0.05% Triton-X. 20 µl of this dilution were automatically pipetted into the graphite tube of the GF-AAS (AAnalyst 600, Perkin Elmer, Rodgau, Germany). 50 µg $NH_4H_2PO_4$ and 3 µg $Mg(NO_3)_2$ were added as matrix modifiers. The furnace program was according to the recommendations of the manufacturer. Quantification based on the standard addition method. In detail, 5 and 10 pg Pb were directly added to the sample in the graphite tube, respectively. The LOD was at 1.0 µg/l.

2.2.3. Analysis of Creatinine in Urine

Creatinine in urine samples was determined for creatinine-corrected levels of toxic metals in urine. Creatinine-corrected urine values were considered in order to account for the influence of the effect of urine dilution on the exposure indicator. Urine samples were sent to the central laboratory of University hospital of LMU and analyzed with Cobas C702 using the Jaffé-method. Creatinine adjusted urine values (As-urine crea-corr, Cd-urine crea-corr, Hg-urine crea-corr) with creatinine levels < 0.3 g/l and > 3.0 g/l were excluded from analysis.

2.3. Statistical Methods

All the data analysis were done in SPSS (version 26, IBM) and R Studio (version 3.6.2, RStudio Inc.). We conducted descriptive analysis and reported the summary statistics. Boxplots were used to visualize the distribution of the data. We assessed correlation of toxic metals and other covariates using the Pearson correlation coefficient and the Spearman correlation for non-normally distributed covariates. We also reported tests on difference in the medians of continuous variables using Wilcoxon Rank Sum test.

3. Results

3.1. Descriptive Analysis of the Data

The urinary levels of As, Cd and Hg as well the Pb and Hg blood levels were measured for the entire study population of 206 participants (results in µg/l). Additionally, the urinary As, Cd and Hg levels were creatinine-corrected for 193 participants (results in µg/g creatinine). Urine samples with creatinine levels below 0.3 or above 3.0 g/l were excluded from analysis. For As and Cd in urine, 12 and 120 samples were below the limit of detection (LOD), respectively.

The levels for toxic metals were compared between the two mining sites, Kadoma and Shurugwi, (see Table 1 and **Fig. 1**). For As in urine (corrected for creatinine), the levels in Kadoma were significantly higher compared to Shurugwi. For Hg in urine, the levels in Shurugwi were significantly higher compared to Kadoma. Otherwise, there are no significant differences in the median levels between the two mining areas. The results stratified by gender can be found in the supplemental information.

Table 1: Descriptive analysis of biomonitoring results for urinary As, Cd, Hg and blood Hg and Pb.

Parameter	Unit	Overall										Kadoma		Shurugwi		Mann-Whitney-U test
		n	< LOD	Min	Mean	Median	Percentiles				Max	n	Median	n	Median	
							5%	25%	75%	95%						
As urine	µg/l	206	12	0.3	16.3	10.0	0.42	4	18.1	46.9	460.5	130	10.9	76	9.7	p = 0.11
As urine crea. corr.	µg/g creatinine	193		0.1	11.3	6.5	0.4	2.7	13.1	31.9	250.3	122	7.3	71	4.7	p = 0.02*
Cd urine	µg/l	206	120	0.3	0.87	0.3	0.3	0.3	0.9	2.9	11.4	130	0.3	76	0.3	p = 0.83
Cd urine crea. corr.	µg/g creatinine	193		0.1	0.54	0.3	0.1	0.2	0.6	1.7	4.9	122	0.3	71	0.3	p = 0.50
Hg blood	µg/l	206	0	0.2	7.2	2.7	0.5	1.3	6.2	29.7	166.8	130	2.1	76	3.25	p = 0.09
Hg in urine	µg/l	206		0.1	24.3	5.2	0.2	1	21.6	122.3	612.3	130	3.6	76	6.5	p = 0.03*
Hg urine crea. corr.	µg/g creatinine	193		0.1	17.1	4.0	0.2	0.6	16.3	73.1	478.4	122	3.2	71	5.3	p = 0.05
Pb blood	µg/l	206	0	6.6	30.1	19.9	8.2	12.5	34.5	75.9	275.7	130	20.6	76	18.4	p = 0.20

Mann-Whitney-U test to analyze possible significant difference in median between the two study locations. *: significant ($p < 0.05$); Min.: Minimum; Max.: Maximum; crea. corr.: Corrected for urinary creatinine. Creatinine correction was not applied for urine samples with creatinine levels below 0.3 or above 3.0 g/l.; LOD: limit of detection.

3.2. Comparison to reference and threshold levels

For reference values, U.S. data from the “National Health and Nutrition Examination Survey” (NHANES, 95th percentiles for the age group “20 + years” from the 2015–2016 survey) published by the CDC were used [38]. Furthermore, data from the “German Environmental Surveys” (GerES, 95th percentiles for the age group 18 to 69 years) published by the “German Environment Agency” (UBA) were used [39, 40]. The UBA also released internationally well accepted threshold levels for adults, called HBM-I and HBM-II (HBM = human biological monitoring). Levels below the HBM-I value are considered as safe, between HBM-I and HBM-II as alerting and levels above HBM-II as health-threatening [39, 41].

The reference and threshold values for As, Cd, Hg and Pb as well as the percentage of participants that exceeded these values are given in Table 2. For urinary As, between 2 and 33% of the participants were above the reference levels, if the reference values from the NHANS study or the UBA were used. The reference values of Cd in urine were exceeded by 22% (NHANES) or 27% (UBA), respectively. Additionally, 4% of the participants were above the HBM-II-value for Cd in urine. For Hg, 34% (NHANES) or 73% (UBA) of the Hg levels in urine and blood were above reference values. The HBM-II value for Hg in blood was exceeded by 11% of the participants, the HBM-II value for Hg in urine by 21%. Finally, blood Pb levels of 32% of the participants exceeded the reference values given by the NHANES study. The UBA provides individual blood Pb reference values for women and men. Here, 24% of the women and 20% of the men exceeded the reference value. Additionally, 13% of the participants had blood Pb levels above the threshold value proposed by NIOSH.

Table 2
Results with As, Cd, Hg and Pb levels above reference or threshold levels.

		As		Cd	Hg			Pb
		Urine [µg/l]	Urine [µg/g crea]	Urine [µg/l]	Blood [µg/l]	Urine [µg/l]	Urine [µg/g crea]	Blood [µg/l]
Reference values	Agency	NHANES (CDC)						
	Value	49.9	56.2	1.08	4.66	1.22	1.15	28.9
	% above	3.6	2.0	22	34	69	67	32
	Agency	UBA						
	Value	15.0	n.a.	0.8	2.0	1.0	n.a.	30.0 (♀)
								40.0 (♂)
	% above	33		27	60	73		24 (♀)
								20 (♂)
Threshold values	Agency	n.a.	n.a.	HBM-II (UBA)				NIOSH (CDC)
	Value			4.0	15.0	25.0	20.0	50.0
	% above			4	11	23	21	13

NHANES = National Health and Nutrition Examination Survey, U.S.; UBA = German Environment Agency; CDC = Center for Disease Control and Prevention, U.S.; NIOSH = National Institute for Occupational Safety and Health, U.S.; n.a. = not applicable, no level determined; HBM II = human biological monitoring alert level. [38–43].

3.3. Correlations

To analyse possible correlations between the different metals as well as age, years living in the area and years working, a Spearman's correlation test was performed (Table 3). A strong correlation was found for Hg in urine with Hg in blood. Otherwise, only Hg (in urine) and Cd (in urine) showed a weak correlation. The age of the participants or the years they lived in the area did not correlate with the toxic metal levels. In contrast, the years worked as a miner showed a weak, but significantly positive correlation with Hg levels in urine. Fish consumption is a potential confounder for As and Hg levels. Therefore, the results for the toxic metal levels in the samples were stratified by fish consumption (less than once a weak vs at least once a weak, for more details see supplemental information). Although As and Hg levels were higher in the high fish group, no significant effect of fish consumption on As or Hg levels was observed.

Table 3
Correlation between the different toxic metals (µg/l) and other parameters (Spearman's correlation).

		Hg in urine	Hg in blood	Pb in blood	As in urine	Cd in urine
Hg in blood	Correlation coefficient	0.737**				
	Sig.	0.000				
Pb in blood	Correlation coefficient	0.231**	0.242**			
	Sig.	0.001	0.000			
As in urine	Correlation coefficient	0.243**	0.121	0.009		
	Sig.	0.000	0.082	0.896		
Cd in urine	Correlation coefficient	0.315**	0.178*	0.021	0.197**	
	Sig.	0.000	0.010	0.759	0.004	
Age	Correlation coefficient	-0.090	-0.047	-0.090	-0.046	-0.118
	Sig.	0.199	0.504	0.198	0.510	0.090
Years living	Correlation coefficient	0.022	-0.021	-0.049	-0.101	-0.065
in area	Sig.	0.755	0.767	0.483	0.149	0.355
Years mining	Correlation coefficient	0.173*	0.157*	0.053	-0.012	-0.082
	Sig.	0.012	0.024	0.450	0.865	0.242

Correlation coefficient are considered strong > 0.7, moderate > 0.5 and weak > 0.3, ** Correlation is significant with $p < 0.01$, * Correlation is significant with $p < 0.05$, n = 206 (entire study population).

4. Discussion

4.1. Discussion of the Results

In general, the levels of As, Cd, Hg and Pb in the samples showed a log-normal distribution, which was expected for this population. As explained in the introduction, toxic metals can occur together with gold-containing ores in ASGM areas. Therefore, it seems plausible that elevated As, Cd and Pb levels in the participants together with the exposure to elemental Hg may be attributed to the mining activities in Kadoma and Shurugwi. However, these levels are influenced by various factors. Below, we discuss the results for the individual metals.

In contrast to the other metals, the reference values for As in urine from UBA and CDC differ considerably from one another (15.0 vs 49.9 µg/l). This is likely due to different exposures to As, e.g. by fish consumption and other sources such as drinking water. However, reference values represent the actual exposure in the general population and cannot be used for toxicological evaluation. In this study, the As levels are more comparable to the reference values of the NHANES study than to the levels in Germany (UBA). However, we did not analyze the As species to differentiate the intake of inorganic species (As(III) and As(V)), and organic As (e.g. arsenobetaine). In fact, inorganic As species are considered carcinogenic and its exposure should be limited to a minimum. Inorganic As can be ingested via drinking water, that could be contaminated due to mining activities or via fish consumption, which had a positive, but not significant, effect on urinary As levels. Furthermore, the ingestion of airborne dust may have been an additional source of exposure. The differences found for urinary As (corrected for creatinine) between the study locations may be explained by different exposure due the mining or dietary patterns.

For Cd, most of the samples were below the LOD. This speaks for a generally low exposure. Still, a considerable number of participants were above the references and threshold values. Cd levels are generally elevated in smokers. Unfortunately, we do not have the data for smoking in our study. In Zimbabwe's mining areas, far more men compared to women smoke [5]. However, this was not reflected in our results, where women were more frequently above international reference and threshold values for Cd (data not shown).

The high number of samples above reference and threshold values found for Hg in urine and blood can be explained by the occupational use of Hg to extract gold. Interestingly, the results were not significantly confounded by the consumption of fish, although levels of Hg in blood and urine in the high fish group were elevated compared to low fish group. An increase in Hg levels was especially expected for blood, since the uptake of MeHg, which is the predominant Hg species in fish, would result in elevated Hg levels in blood rather than in urine. Eventually, fish consumption did not contribute significantly to the total Hg body burden. This may be explained by that the fact that the Hg levels in the consumed fish were relatively low (see also limitations) compared to the exposure to elemental Hg. Unfortunately, data on the Hg species was not available to further investigate the source of exposure. The differences found for urinary Hg between the study locations may be attributed to the more frequent application of protective equipment and safety precautions, since the Kadoma region has more experience with ASGM. Additionally, the years of mining in ASGM areas showed a weak positive correlation with Hg levels in blood and urine. This finding may be explained by the fact that chronic exposure to Hg leads to the accumulation of Hg in the kidney, resulting in a generally elevated urinary elevation [42]. However, we expect that the acute exposure to elemental Hg outweighs the effect of years mining in this population. As exposure to Hg can be assessed in either blood or urine, both matrices are highly correlated to each other. We also found a weak correlation between Hg and Cd, which has no plausible explanation. We considered this to be the result of a "statistical" chance by applying multiple statistical tests as we did.

For Pb, about a third of the participants were above international reference values. The exposure to lead could be via contaminated drinking water, although the uptake of Pb in the gastrointestinal tract of adults is considered relatively low, and airborne dust due to mining activities. Besides this, Pb exposure could be due to emissions of leaded gasoline, which was used longer in Africa than in the US or Europe [43]. Therefore, the recent exposure to these emissions might still be reflected by elevated blood Pb levels. Furthermore, we cannot exclude that leaded gasoline might be still used in Zimbabwe, especially in remote areas.

4.2. Comparison of the results with other studies in mining areas.

The results of this study were compared to the results from other studies in mining areas around the globe (see Table 4). Not all of these studies were analyzing the same range of toxic metals, but single elements up to all four parameters. We used, if given, the median as well as the 25th and 75th percentile for comparison. In general, our results were in line to what has been previously found in studies conducted in current and former mining areas.

For As, the studies from Tanzania [44] and Ghana [23] show similar results for ASGM areas. But also in non-active mining areas in Mexico, comparable urinary As levels were found [45]. In contrast, studies from Spain and Guatemala found relatively low levels of As the urine [46, 47]. For Cd in urine, the studies from Ghana, Spain and Guatemala showed very similar results [23, 46, 47]. The levels of Hg in urine found in our study are comparable to what has been found in other ASGM areas with known Hg use [48–52]. In areas without known or recent use of Hg for gold extraction, urinary Hg levels were considerably lower [45–47]. For Hg in blood, the results from other ASGM areas are relatively similar [48, 49, 53]. But also in non-ASGM mining areas, the median of Hg in blood was relatively high [47]. In contrast, pregnant women living in ASGM areas showed lower Hg levels in blood [44]. For Pb, our results are mainly similar to what has been found in other studies [47, 53–55]. However, in a study from Mexico, blood Pb levels were significantly higher [45].

Although some of the studies found similar results, the exposure to toxic metals likely depends on multiple factors such as the concentrations of metals in soil and water, the diet, the use of Hg for gold extraction, living area and many others. Generally, living close to mining areas seems to have a significant effect on the body burden [46, 47, 53]. One possible explanation is that contaminated mining tailings contribute to the exposure to toxic metals by causing elevated concentrations in water, food and airborne dust [45].

Table 4
Comparison of the study results (all values are given in µg/l) with the literature

Parameter [µg/l]	Values measured in this Study	Values measured in studies in current and former mining areas (country)	Reference
	25th –median – 75th	25th –median – 75th	
As in urine	4.0–10.0–18.5	4.9–9.4–15.1 (Tanzania)	[44]
		73.2–100.2–135.3 (Ghana)	[23]
		11.1–16.5–19.4 (Mexico)[1]	[45]
		0.5–1.17–1.93 (Spain)[1]	[46]
		0.06 (Guatemala)[#]	[47]
Cd in urine	0.3–0.3–0.9	0.25–0.36–0.60 (Ghana)	[23]
		0.13–0.29–0.46 (Spain)[1]	[46]
		0.11 (Guatemala)[#]	[47]
Hg in urine	1.0–5.2–21.3	3.11*, 2.32** (Ghana)	[30]
		1.04–2.94–11.0 (Ghana)	[51]
		< LOQ – 0.7–0.9 (Mexico)[1]	[45]
		0.33–1.13–2.0 (Spain)[1]	[46]
		0.11 (Guatemala)[#]	[47]
		0.7–1.8–5.4 (Ecuador)	[52]
		12.6*, 1.76** (Tanzania)	[49]
		10.2/22.4*, 5.3/7.8** (Indonesia)	[48]
Hg in blood	0.2–2.7–6.2	0.72–1.24–1.69 (Tanzania)	[56]
		2.4 (Guatemala)[#]	[47]
		4.62*, 1.98** (Tanzania)	[49]
		10.6/13.3*, 9.5/9.2** (Indonesia)	[48]
		63.0 (Ghana)[##]	[53]
Pb in blood	12.5–19.9–34.5	16.9–25.4–33.7 (Tanzania)	[56]
		64–94 – 113 (Mexico)[1]	[45]
		26.7 (Guatemala)[#]	[47]
		13.0 (Zambia)[#]	[55]
		21.5 (Nigeria)[#]	[54]
		28.0 (Ghana)[##]	[53]

25th : 25th percentile; 75th : 75th percentile; * amalgam smelters, ** non-smelting gold miners; [1] children, [#] median, [##] mean.

4.3. Limitations

For some samples and parameters, the sensitivity of the analytical method was too low. In detail, As levels in urine, 13 out of 206 samples were below the limit of detection (LOD). For Cd, 120 samples were below the LOD. In contrast, Hg levels in urine and blood as well as Pb levels in blood were above the LOD in all samples. Although Cd levels were not detectable in the majority, the LOD of Cd in urine is still below the reference values (UBA and NHANES) and alert levels (HBM-II) for adults and therefore considered a relatively safe level. For statistical analyses, all samples below the LOD were set to a value of LOD/2 as an approximation. For Cd, a relatively high number of participants were above international reference values (22% > NHANES, 27% > UBA). This may be explained by the fact that the Cd levels of 51 samples are still below 3 times the LOD, which equals the limit of quantitation. In this range, there might be some uncertainty regarding absolute quantitation. Furthermore, information on the smoking status as a potential confounder for Cd was not collected in the study. In this study, As and Hg were not speciated, thus limiting the comparability with other studies. Unfortunately, we had no information on the levels of the analyzed metals in either drinking water or food, e.g. fish. Furthermore, we did not inquire a full dietary questionnaire. Therefore, other food sources than fish could not be considered in statistical analysis and discussion of this study. The fact that the number of participants in the low fish group was relatively low (38 vs 155 in the high fish group) may also impaired a powerful statistical analysis.

5. Conclusions

This is, to the best of our knowledge, the first study that analyzed the levels of As, Cd and Hg as well as Hg and Pb in blood in people identifying themselves as artisanal and small-scale gold miners in Zimbabwe. Hg levels were, as expected in ASGM areas, mainly above reference and threshold values. A high proportion of As, Cd and Pb levels were above reference levels. Therefore, the exposure to toxic metals in the two ASGM areas in Zimbabwe is relevant to public health and should be the subject of further investigation to clarify the influence of possible confounders, e.g. diet and living.

Declarations

Ethics approval and consent to participate: Approval for conducting the study was gained from the Ministry of Health in Zimbabwe and the respective local and regional authorities. The Ethics Committee of the Medical Research Council and the Zimbabwe Research Council (MRCZ / A / 2367, September 26, 2018 and February 25, 2019) and the Ludwig Maximilians University of Munich (18-421, October 15, 2018) approved the study protocols and gave their permissions. In agreement with the Helsinki Declaration of Ethics Code for experiments with human subjects the study was performed.

Availability of data and materials: The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Competing interests: The authors declare no conflict of interest. The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Funding: This research was funded by the German Federal Ministry for the Environment, Nature Conservation and Nuclear Safety (BMU IG II 2 45083 - 4/0).

Author Contributions: Conceptualization: SR, VM, DS, DN, SM, NS, SB; Data curation: GM, VM, SB; Formal analysis: SR, GM, VM; Funding acquisition: VM, DS, DM, NS, SB; Investigation: SR, VM, DS, DM, SM, MT, SB; Methodology: SR, GM, VM, DM, SM, NS, SB; Project administration: VM, DS, DM, NS, SB; Supervision: SR, NS, SB; Validation: SR; Visualization: GM, AW, SB; Writing – original draft, SR, GM, AW, VM, DS, DM, SM, MT, SB; Writing – review & editing: SR, GM, AW, VM, DS, DM, SM, MT, NS, SB.

Acknowledgments: The authors would like to thank the funding institution and our team in Zimbabwe and Germany. Especially we would like to thank Evans Rudzvidzo, Dr. Godknows Madziva, Brian Chizaza, Josephine Mashamba, Jana Becker, James Rooney, and Friederike Butscher.

References

1. Seccatore J, Veiga M, Origliasso C, Marin T, De Tomi G. An estimation of the artisanal small-scale production of gold in the world. Science of The Total Environment. 2014;496:662–7.
2. Mkodzongi G, Spiegel SJ. **Mobility, temporary migration and changing livelihoods in Zimbabwe's artisanal mining sector**. *The Extractive Industries and Society* 2020.
3. Mudzwiti PM, Mungoni N, M.: **A golden opportunity**: **Scoping Study of Artisanal and Small Scale Mining in Zimbabwe** In. Edited by Zimbabwe P. Harare, Zimbabwe; 2015: 174.
4. Mudzwiti P, Mukwakwami N, Mungoni M, I. M: **A golden opportunity**: **Scoping Study of Artisanal and Small Scale Mining in Zimbabwe** In. Edited by PACT. Harare, Zimbabwe; 2015.
5. Billaud P, Laperche V, Maury-Brachet R, Boudou A, Shoko D, Kahwai S, Freyssinet P. **Removal of barriers to the introduction of cleaner artisanal gold mining and extraction technologies in Kadoma, Zimbabwe - Final report, part A Environmental assessment. UNIDO Project EG/GLO/01/G34 No.03/089. BRGM Project Nr 53320-FR** In. Orleans, France: BRGM (Bureau de Recherches Géologiques et Minières), ; 2004.
6. Foster RP. Major Controls of Archaean Gold Mineralization in Zimbabwe. Trans Geol Soc S Afr. 1985;88:109–33.
7. Saunders J, Hofstra A, Goldfarb R, Reed MH. **Geochemistry of Hydrothermal Gold Deposits** *Treatise on Geochemistry: Second Edition* 2013, 13:383–424.
8. **Ten chemicals of major public health concern** [https://www.who.int/ipcs/assessment/public_health/chemicals_phc/en/].
9. Nurchi VM, Djordjevic AB, Crisponi G, Alexander J, Bjorklund G, Aaseth J. **Arsenic Toxicity: Molecular Targets and Therapeutic Agents**. *Biomolecules* 2020, 10(2).
10. Bjørklund G, Tippairote T, Rahaman MS, Aaseth J. Developmental toxicity of arsenic: a drift from the classical dose–response relationship. Arch Toxicol. 2020;94(1):67–75.
11. Medda N, Patra R, Ghosh TK, Maiti S. **Neurotoxic Mechanism of Arsenic: Synergistic Effect of Mitochondrial Instability, Oxidative Stress, and Hormonal-Neurotransmitter Impairment**. *Biological Trace Element Research* 2020.
12. Lee W-K, Thévenod F. **Cell organelles as targets of mammalian cadmium toxicity**. *Archives of Toxicology* 2020.
13. Reyes-Hinojosa D, Lozada-Pérez CA, Zamudio Cuevas Y, López-Reyes A, Martínez-Nava G, Fernández-Torres J, Olivos-Meza A, Landa-Solis C, Gutiérrez-Ruiz MC, Rojas del Castillo E et al: **Toxicity of cadmium in musculoskeletal diseases**. *Environmental Toxicology and Pharmacology* 2019, **72**:103219.
14. Zhang H, Reynolds M. Cadmium exposure in living organisms: A short review. Science of The Total Environment. 2019;678:761–7.
15. Afrifa J, Opoku YK, Gyamerah EO, Ashiagbor G, Sorkpor RD. **The Clinical Importance of the Mercury Problem in Artisanal Small-Scale Gold Mining**. *Frontiers in Public Health* 2019, 7(131).
16. Raj D, Maiti SK. Sources, toxicity, and remediation of mercury: an essence review. Environ Monit Assess. 2019;191(9):566.
17. Takahashi T, Shimohata T. **Vascular Dysfunction Induced by Mercury Exposure**. *International journal of molecular sciences* 2019, 20(10).
18. Yang L, Zhang Y, Wang F, Luo Z, Guo S, Strähle U. Toxicity of mercury: Molecular evidence. Chemosphere. 2020;245:125586.

19. Gidlow DA. Lead toxicity. Occup Med. 2015;65(5):348–56.
20. Kumar A, Kumar A, M MSC, Chaturvedi AK, Shabnam AA, Subrahmanyam G, Mondal R, Gupta DK, Malyan SK. S SK et al: **Lead Toxicity: Health Hazards, Influence on Food Chain, and Sustainable Remediation Approaches**. *International journal of environmental research and public health* 2020, 17(7).
21. Mitra P, Sharma S, Purohit P, Sharma P. Clinical and molecular aspects of lead toxicity: An update. Crit Rev Clin Lab Sci. 2017;54(7–8):506–28.
22. Charles E, Thomas DS, Dewey D, Davey M, Ngallaba SE, Konje E. A cross-sectional survey on knowledge and perceptions of health risks associated with arsenic and mercury contamination from artisanal gold mining in Tanzania. BMC Public Health. 2013;13:74.
23. Basu N, Nam D-H, Kwansaa-Ansah E, Renne EP, Nriagu JO. Multiple metals exposure in a small-scale artisanal gold mining community. Environ Res. 2011;111(3):463–7.
24. Agency for Toxic Substances and Disease Registry (ATSDR). **Toxicological profile for Arsenic**. **Atlanta**, **GA**: **U.S**. **Department of Health and Human Services**, **Public Health Service**.. In. Edited by U.S. Department of Health and Human Services PHS. Atlanta: Agency for Toxic Substances and Disease Registry (ATSDR),; 2007.
25. Adu-Poku B, Asiedu N, Akoto O, Ataki J, Stafilov T. **Modelling the distribution of arsenic and mercury in urine using chemometric tools**. *Cogent Chemistry* 2019, 5(1).
26. Abrefah R, Adotey D, Mensimah E, Ampomah-Amoako E, Sogbadji R, Opata N. **Biomonitoring of Occupational Exposure to Total Arsenic and Total Mercury in Urine of Goldmine Workers in Southwestern Ghana**. *Environmental Research, Engineering and Management* 2011, 56.
27. Asante KA, Agusa T, Subramanian A, Ansa-Asare OD, Biney CA, Tanabe S. Contamination status of arsenic and other trace elements in drinking water and residents from Tarkwa, a historic mining township in Ghana. Chemosphere. 2007;66(8):1513–22.
28. Basu N, Nam DH, Kwansaa-Ansah E, Renne EP, Nriagu JO. Multiple metals exposure in a small-scale artisanal gold mining community. Environ Res. 2011;111(3):463–7.
29. Basu N, Horvat M, Evers DC, Zastenskaya I, Weihe P, Tempowski J. A State-of-the-Science Review of Mercury Biomarkers in Human Populations Worldwide between 2000 and 2018. Environ Health Perspect. 2018;126(10):106001.
30. Kwaansa-Ansah EE, Armah EK, Opoku F. Assessment of Total Mercury in Hair, Urine and Fingernails of Small-Scale Gold Miners in the Amansie West District, Ghana. J Health Pollut. 2019;9(21):190306.
31. Mambrey V, Rakete S, Tobollik M, Shoko D, Moyo D, Schutzmeier P, Steckling-Muschack N, Muteti-Fana S, Bose-O'Reilly S. Artisanal and small-scale gold mining: A cross-sectional assessment of occupational mercury exposure and exposure risk factors in Kadoma and Shurugwi, Zimbabwe. Environ Res. 2020;184:109379.
32. Baeuml J, Bose-O'Reilly S, Gothe RM, Lettmeier B, Roider G, Drasch G, Siebert U. Human Biomonitoring Data from Mercury Exposed Miners in Six Artisanal Small-Scale Gold Mining Areas in Asia and Africa. Minerals. 2011;1(1):122–43.
33. Thurtle N, Greig J, Cooney L, Amitai Y, Ariti C, Brown MJ, Kosnett MJ, Moussally K, Sani-Gwarzo N, Akpan H, et al. Description of 3,180 Courses of Chelation with Dimercaptosuccinic Acid in Children #5 y with Severe Lead Poisoning in Zamfara, Northern Nigeria: A Retrospective Analysis of Programme Data. PLoS Med. 2014;11(10):pmed.1001739.
34. Greig J, Thurtle N, Cooney L, Ariti C, Dargan P. Association of Blood Lead Level with Neurological Features in 972 Children Affected by an Acute Severe Lead Poisoning Outbreak in Zamfara State, Northern Nigeria. PLoS ONE. 2014;9(4):e93716.
35. Plumlee GS, Durant JT, Morman SA, Neri A, Wolf RE, Dooyema CA, Hageman PL, Lowers HA, Fernette GL, Meeker GP, et al. Linking geological and health sciences to assess childhood lead poisoning from artisanal gold mining in Nigeria. Environ Health Perspect. 2013;121(6):744–50.
36. Lo YC, Dooyema CA, Neri A, Durant J, Jefferies T, Medina-Marino A, de Ravello L, Thoroughman D, Davis L, Dankoli RS, et al. Childhood lead poisoning associated with gold ore processing: a village-level investigation-Zamfara State, Nigeria, October-November 2010. Environ Health Perspect. 2012;120(10):1450–5.
37. Dooyema CAea. Outbreak of Fatal Childhood Lead Poisoning Related to Artisanal Gold Mining in Northwestern Nigeria. Environ Health Perspect. 2010;120(4):601–7.
38. Centers for Disease Control and Prevention. **Fourth National Report on Human Exposure to Environmental Chemicals - Updated Tables**. In. Edited by U.S. Department of Health and Human Services CfDCaP. Atlanta: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention,; 2019.
39. **[Reference values (RV95) for antimony, arsenic and metals (Pb, Cd, Ni, Hg, Pt, Tl, U) in urine or in the blood]** https://www.umweltbundesamt.de/sites/default/files/medien/4031/dokumente/tab._referenzwerte_-_metalle_30._september_2019_aktualisiert.pdf.
40. Schulz C, Wilhelm M, Heudorf U, Kolossa-Gehring M, Human Biomonitoring Commission of the German Federal Environment A. Update of the reference and HBM values derived by the German Human Biomonitoring Commission. Int J Hyg Environ Health. 2011;215(1):26–35.
41. Ewers U, Krause C, Schulz C, Wilhelm M. Reference values and human biological monitoring values for environmental toxins. Report on the work and recommendations of the Commission on Human Biological Monitoring of the German Federal Environmental Agency. Int Arch Occup Environ Health. 1999;72(4):255–60.
42. Clarkson TW, Magos L. The Toxicology of Mercury and Its Chemical Compounds. Crit Rev Toxicol. 2006;36(8):609–62.
43. Todd DT, Hazel. **Outcome and Influence Evaluation of the UNEP Partnership for Clean Fuels and Vehicles (PCFV)** *United Nations Environment Programme (UNEP)* 2010.
44. Nyanza EC, Bernier FP, Manyama M, Hatfield J, Martin JW, Dewey D. Maternal exposure to arsenic and mercury in small-scale gold mining areas of Northern Tanzania. Environ Res. 2019;173:432–42.

45. Moreno ME, Acosta-Saavedra LC, Meza-Figueroa D, Vera E, Cebrian ME, Ostrosky-Wegman P, Calderon-Aranda ES. Biomonitoring of metal in children living in a mine tailings zone in Southern Mexico: A pilot study. Int J Hyg Environ Health. 2010;213(4):252–8.
46. Molina-Villalba I, Lacasaña M, Rodríguez-Barranco M, Hernández AF, Gonzalez-Alzaga B, Aguilar-Garduño C, Gil F. Biomonitoring of arsenic, cadmium, lead, manganese and mercury in urine and hair of children living near mining and industrial areas. Chemosphere. 2015;124:83–91.
47. Basu N, Abare M, Buchanan S, Cryderman D, Nam D-H, Sirkin S, Schmitt S, Hu H. A combined ecological and epidemiologic investigation of metal exposures amongst Indigenous peoples near the Marlin Mine in Western Guatemala. Science of The Total Environment. 2010;409(1):70–7.
48. Bose-O'Reilly S, Drasch G, Beinhoff C, Rodrigues-Filho S, Roider G, Lettmeier B, Maydl A, Maydl S, Siebert U. Health assessment of artisanal gold miners in Indonesia. Science of The Total Environment. 2010;408(4):713–25.
49. Bose-O'Reilly S, Drasch G, Beinhoff C, Tesha A, Drasch K, Roider G, Taylor H, Appleton D, Siebert U. Health assessment of artisanal gold miners in Tanzania. Science of The Total Environment. 2010;408(4):796–805.
50. Kwaansa-Ansah EE, Armah EK, Opoku F. Assessment of Total Mercury in Hair, Urine and Fingernails of Small-Scale Gold Miners in the Amansie West District, Ghana. J Health Pollut. 2019;9(21):190306–6.
51. Rajaee M, Long RN, Renne EP, Basu N. Mercury Exposure Assessment and Spatial Distribution in A Ghanaian Small-Scale Gold Mining Community. Int J Environ Res Public Health. 2015;12(9):10755–82.
52. Schutzmeier P, Berger U, Bose-O'Reilly S. **Gold Mining in Ecuador: A Cross-Sectional Assessment of Mercury in Urine and Medical Symptoms in Miners from Portovelo/Zaruma**. *International journal of environmental research and public health* 2016, 14(1).
53. Obiri S, Yeboah PO, Osae S, Adu-Kumi S. Levels of arsenic, mercury, cadmium, copper, lead, zinc and manganese in serum and whole blood of resident adults from mining and non-mining communities in Ghana. Environ Sci Pollut Res. 2016;23(16):16589–97.
54. Gottesfeld P, Meltzer G, Costello S, Greig J, Thurtle N, Bil K, Mwangombe BJ, Nota MM. Declining blood lead levels among small-scale miners participating in a safer mining pilot programme in Nigeria. Occup Environ Med. 2019;76(11):849–53.
55. Yabe J, Nakayama SMM, Nakata H, Toyomaki H, Yohannes YB, Muzandu K, Kataba A, Zyambo G, Hiwatari M, Narita D, et al. Current trends of blood lead levels, distribution patterns and exposure variations among household members in Kabwe, Zambia. Chemosphere. 2020;243:125412.
56. Nyanza EC, Dewey D, Bernier F, Manyama M, Hatfield J, Martin JW. Validation of Dried Blood Spots for Maternal Biomonitoring of Nonessential Elements in an Artisanal and Small-Scale Gold Mining Area of Tanzania. Environ Toxicol Chem. 2019;38(6):1285–93.

Figures

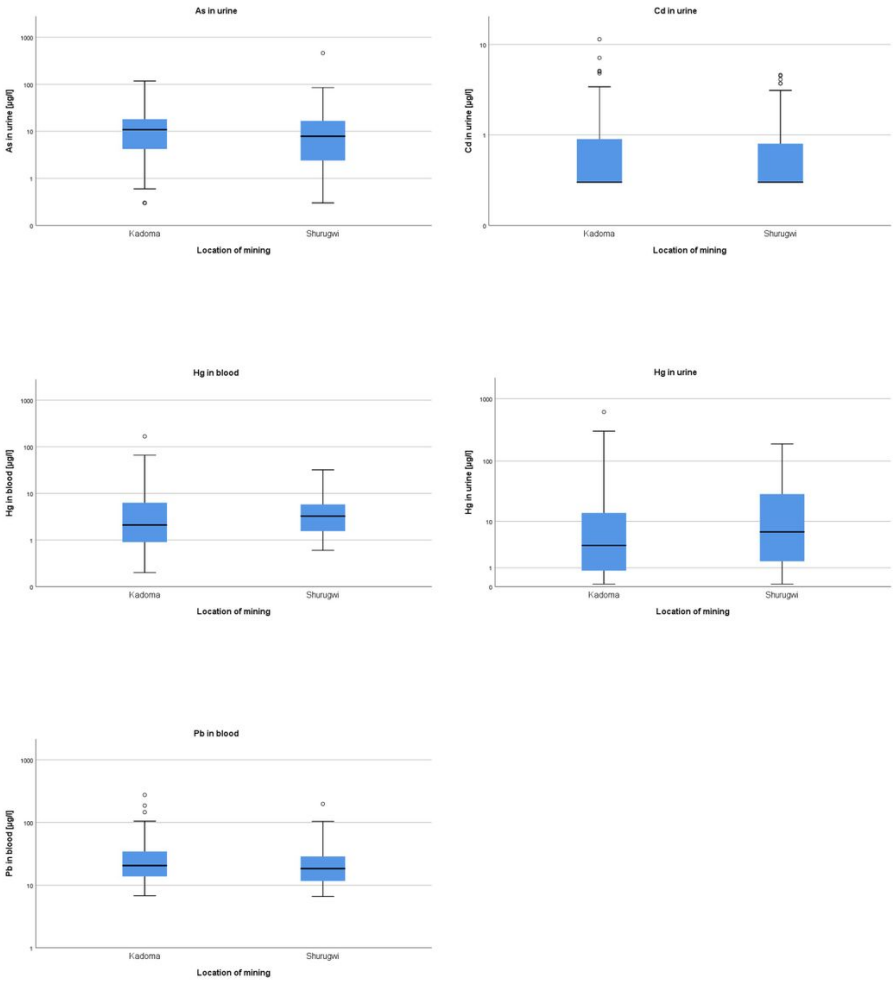

Figure 1

Figure 1 demonstrates the location of mining

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- supplementalinformation.docx
- supplementalinformation.docx