

Contents lists available at ScienceDirect

Science of the Total Environment



journal homepage: www.elsevier.com/locate/scitotenv

Mercury exposure in female artisanal small-scale gold miners (ASGM) in Mongolia: An analysis of human biomonitoring (HBM) data from 2008

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ARTICLE INFO

Article history: Received 21 July 2010 Received in revised form 19 November 2010 Accepted 22 November 2010 Available online 22 December 2010

Keywords: Mercury Human biomonitoring Mongolia Artisanal small-scale gold mining Amalgam smelting

ABSTRACT

Background: Many poor in developing countries have turned to artisanal small-scale gold mining (ASGM) in an attempt to improve their situation. However, the mercury used to extract gold from ore is discharged in vaporized form into the environment, where it poses a hazard for human health.

Methods: As part of an environmental epidemiological study in Mongolia—to evaluate the burden of environmental mercury contamination—urine, blood and hair samples were collected from residents of areas with or without mercury contamination. A total of 200 blood, urine and hair samples were analyzed for mercury and divided into three subgroups according to mercury content: (1) occupational exposure (high/medium); (2) environmental exposure (low); and (3) no exposure. Internal mercury distributions of the subgroups were compared using the Kruskal–Wallis and Mann–Whitney U-test. The Chi-square test and likelihood ratio proportion were used to compare the findings with threshold limits.

Results: The highest values and greatest differences were seen in the urine samples (p<0.001, Kruskal–Wallis). The occupational group showing the highest exposure with a median mercury level of 4.36 µg/l (control group: 0.10 µg/l, p<0.001), 7.18 µg/g creatinine and 12 results above the threshold limit HBM I (Human Biomonitoring I). Even participants from the low-exposure subgroup showed elevated mercury levels (median 2.88 µg/l urine and 2.98 µg/g creatinine, p<0.001), with 10 individuals above the HBM I threshold limits.

Discussion: The body burden resulting from the use of mercury in artisanal gold mining is high not only in the miners themselves, an increased mercury hazard was also found for inhabitants of mining areas who were not actively involved in mining. Public health support measures are urgently needed to alleviate the situation.

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1. Introduction

Mercury (Hg) is a heavy metal which occurs naturally and is discharged into the environment both geogenic and anthropogenic through human activity (ATSDR, 1999). Mercury exists in three main species: elemental (metallic) mercury, inorganic (ionic) mercury and organic mercury like methyl mercury (Drasch et al., 2004). The first and the third mentioned forms are of particular interest regarding artisanal

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^{0048-9697/\$ –} see front matter 0 2010 Elsevier B.V. All rights reserved. doi:10.1016/j.scitotenv.2010.11.029

small-scale gold mining areas (ASGM). In many developing countries, elemental mercury is used in ASGM, and practices of mercury misuse in ASGM often lead to the contamination of environment and living organisms, posing serious health hazard. Several studies evaluated mercury using human biomonitoring methods to identify the human body burden in gold mining areas. Exposed miners and residents of ASGM areas showed higher values compared to non-exposed control groups (van Straaten, 2000a,b; Drasch et al., 2001; Bose-O'Reilly et al., 2010a,b).

1.1. Gold mining in Mongolia

Gold mining in Mongolia has risen sharply since the early 1990s (Murray, 2003). ASGM became the main livelihood for people who lost their jobs during the economic transition as well as for nomadic herders who lost their livestock during a particularly harsh winter. Due to the seasonal nature of gold mining and the vast area of the country, exact numbers of regular and artisanal miners are not available. It is estimated that the number of artisanal gold miners rose sharply during the early 1990s and is now in the range of 30,000-100,000, which would be at least twice the number of regular miners (Grayson et al., 2004; Appel, 2005; The World Bank, 2006; Lkhasuren et al., 2007). Working and living conditions in mining areas are poor and unhealthy. Apart from the danger of accidents and explosions in the tunnels, the use of mercury for extracting gold without safety equipment compounds the hazard. Mercury use is ubiquitous amongst artisanal hard-rock gold miners and has been adopted by artisanal placer gold miners (Navch et al., 2006; The World Bank, 2006). Residual mercury is released into the environment, where it contaminates lakes, rivers, the soil and the air (Grayson et al., 2004; Lkhasuren et al., 2007).

1.2. Extracting the gold

Whereas miners crush ore, usually with ball mills, gold panners add elemental liquid mercury to the ore (amalgamation) during or after milling, causing gold and mercury to form an amalgam. The gold is then recovered by heating the amalgam in smelters until the mercury is vaporized and released into the air as a vapor, leaving behind raw gold. This process does not require high technology, is easy to learn and is widely used; particularly by women, given that men usually engage in heavy physical gold mining. The amalgam is usually smelted in a *ger* (a traditional Mongolian tent), polluting the indoor air with mercury fumes (Grayson et al., 2004; Appel, 2005; Murao et al., 2006; Navch et al., 2006).

Within this gold extraction process, the amount of released mercury depends on the applied technology. Determining technologies of the mercury release in the artisanal gold extraction are on one hand the use of retorts, technical equipment which can trap the mercury vapor during the amalgam smelting process, and on the other hand the amalgamation of the whole ore in contrast to the amalgamation of ore concentrates. While the whole ore amalgamation requires a large extent of mercury, a substantially lesser amount of mercury is necessary to extract gold by the amalgamation of ore concentrates (Veiga and Baker, 2004; UNEP, 2008). While mercury use varies from place to place and different management techniques lead to different exposure risks.

1.3. Health effects of mercury

Mercury is highly toxic. When absorbed, it easily crosses the bloodbrain and the blood-placenta barriers. Methyl mercury absorption is usually the main pathway (Morton et al., 2002). But as well elemental mercury fumes are particularly toxic to the human nervous system (Human Biomonitoring Commission of the Federal Environmental Agency, 1999; Drasch et al., 2004). Typical central nervous system symptoms are tremors and erethism, whereby paresthesias are typical of the peripheral nervous system poisoning. Mercury exposure of children is of particular importance because of its neurodevelopmental toxicity. Target organs for mercury poisoning are the oral cavity and the kidneys, where it causes gingivitis and proteinuria, respectively (Human Biomonitoring Commission of the Federal Environmental Agency, 1999).

1.4. Pathways of mercury contamination in ASGM

In ASGM areas, the major routes of mercury exposure are occupational and environmental. Occupational exposure occurs when workers inhale mercury fumes during amalgam smelting, through direct contact during amalgamation, and through contaminated dust. Environmental exposure results from contaminated soil, air, food (particularly fish) and water (Myers and Davidson, 2000; Drasch et al., 2001; Bose-O'Reilly et al., 2008a,c). Children are at a particularly high risk of mercury poisoning due to additional exposure pathways (Bose-O'Reilly et al., 2008b). Inhaled mercury fumes cross the placenta and the metal is transferred to the fetus *in utero* and to infants when breast feeding (Commission Human Biomonitoring of the Federal Environmental Agency, 1999; Bose-O'Reilly et al., 2008a). Women must therefore be particularly careful when handling mercury (Navch et al., 2006).

Addressing the grave danger posed by mercury to the health of women of reproductive age and their children, the World Health Organization in Ulaanbaatar (Mongolia) and Geneva (Switzerland), the Mongolian Ministry of Health (MoH) and the Austrian University of Health Sciences, Medical Informatics and Technologies (UMIT) carried out an environmental epidemiological study in Mongolia to determine the mercury burden of female gold miners.

2. Materials and methods

In order to evaluate the mercury burden in mining areas and the extent of potential health hazards, health and human biomonitoring data were collected as part of an environmental epidemiological cross-sectional study in September 2008. 157 Mongolian women with mercury exposure and 43 non-exposed Mongolian women were examined. Anamnestic data were collected, including exposure history, and neurological examinations and neuropsychological tests were performed. This article gives the results of the urine, blood and hair analysis.

2.1. Study region and study population

In 2008 a field mission was performed in Bornuur sum (Борнуур сум), Jargalant sum (Жаргалант сум) and Khushaat sum (Хушаат сум). The sample consisted of volunteers chosen at random. A total of 200 Mongolian women aged 15–35 were examined. Exclusion criteria were age <15 and incomplete data (one person per group). The study population comprised 198 women of reproductive age and was subdivided into a control and an exposed group, the latter subdivided into two groups: low exposure (environmental exposure in ASGM areas but no direct occupational exposure) and medium to high (active occupational exposure from ASGM). The group with medium/ high exposure comprised women working as smelters or panners, whereas the low-exposure group comprised women living in mining areas but not working in a mine. The control group in Khushaat sum lived in an area without gold mining activity. Their sociodemographic structures are comparable. Khushaat sum was selected because of an ongoing WHO project in the area (safe water for the hospital).

2.2. Laboratory methods

Blood, spontaneous urine and hair samples were collected from every participant (except 7 missing hair samples in the control group). The blood and urine samples were continuously kept cool during transport.

Urine specimens from the control group were shipped to the National Institute for Minamata Disease, Japan (control group). The urine specimens were decomposed in sealed Teflon containers and total mercury concentration was measured by cold vapor atomic absorption spectrophotometry (CVAAS) using SnCl₂ for reduction after enrichment on a gold–platinum net. The detection limit (LOD) was 0.1 µg/l.

The Institute of Forensic Medicine in Munich (Germany) analyzed the urine specimens from the exposure groups. The total amount of mercury in the samples was determined using CVAAS (Analytik Jena ZEEnit 650 spectrometer®) with an amalgamation unit (HS 60, Analytik Jena, Germany®) without further pretreatment. Sodium borohydride (NaBH₄) was used to reduce all mercury (inorganic and organic). Although NaBH₄ reduces inorganic mercury faster than organic mercury (e.g. methyl mercury), this method can be used to determine the correct amount of total mercury because all nascent mercury fumes were collected on a gold-platinum net. In a second step, the net was heated and all trapped metallic mercury released at once to be quantified with CVAAS. The accuracy of the method for inorganic as for organic mercury compounds was proven by inorganic and methyl mercury standard solutions. The LOD with this method was 0.20 µg/l. All analyses were done under strict internal and external guality control. Clin Chek Level II (Recipe, Germany®) was used as a standard matrix-matched control sample.

All blood samples were analyzed by the Health and Safety Laboratory, Buxton (United Kingdom). Aliquots of the blood samples were diluted 1:10 using an alkaline solvent (1% m/v EDTA, 1% v/v NH₃, 1% v/v Triton X-100, 1 mg/l gold and 10 µg/l platinum [internal standards]). Samples were analyzed using ICP-MS (Perkin Elmer 6100 ICP-MS, Beaconsfield, UK®) with standard addition using matrixmatched standards. Internal and external (Bio-Rad Laboratories) quality control samples were run at the start and at the end of each analysis and a check standard of 10 µg/l was analyzed after every 10 samples. The LOD of mercury in blood was 0.20 µg/l.

The hair samples from the control group were sent to the National Institute for Minamata Disease (Japan), those from the exposed group to the National Institute of Public Health in Ulaanbaatar (Mongolia). Both were analyzed using CVAAS (Yasutake et al., 2003). The hair samples were washed well with detergent, and rinsed two times with acetone to dry. Sample digestion was performed with HNO₃ HClO₄ and H₂SO₄ followed by reduction to Hg⁰ by SnCl₂. The total mercury levels were analyzed with an oxygen combustion-gold amalgamation method.

2.3. Controlling for confounders

Several potential confounders for mercury concentration in the human body were considered. The quantity of dental amalgam fillings is associated with mercury concentrations in urine and blood (Kingman et al., 1998; Dye et al., 2005). Alcohol can induce neurological symptoms similar to mercury poisoning (Drasch et al., 2001). Smoking also needs to be controlled for, given that smokers show a higher mercury concentration than do non-smokers (Slojewski et al., 2009). Fish also can provide a relevant source of methyl mercury (International Programme on Chemical Safety and World Health Organization, 1990), and this was included in the questionnaire, as were other potential routes of contact with mercury like storing mercury or work clothes at home.

2.4. Thresholds levels

Threshold levels (Table 1) were used to characterize the health risk. UNIDO recommends using the German Human Biomonitoring (HBM) values for urine and blood in ASGM (Veiga and Baker, 2004). Values below the HBM I value ($<7 \mu g$ Hg/l urine, $<5 \mu g$ Hg/g creatinine, $<5 \mu g$ Hg/l blood) are not known to cause any adverse health effects. Values between HBM I and HBM II should be treated as warning signs and followed up by control interventions. Values above HBM II ($>25 \mu g$ Hg/l urine, $>20 \mu g$ Hg/g creatinine, $>15 \mu g$ Hg/l blood) tend to indicate a risk of adverse health effects for susceptible individuals. The burden level should be reduced and medical

Table 1

Toxicologically established threshold limits for mercury in blood, urine and hair. HBM = Human Biomonitoring (Human Biomonitoring Commission of the Federal Environmental Agency, 1999).

	Hg blood (µg/l)	Hg urine (µg/l)	Hg urine (µg/g creatinine)	Hg hair (µg/g)	
<hbm i<="" td=""><td>0–<5</td><td>0–<7</td><td>0-<5</td><td>0-<1 (*)</td><td>Normal</td></hbm>	0–<5	0–<7	0-<5	0-<1 (*)	Normal
HBM I–HMB II	5–<15	7–<25	5-<20	1-<5 (*, **)	Alarm level
>HBM II	≥15	≥25	≥20	≥5 (**)	High level

*US EPA benchmark (U.S. Environmental Protection Agency, 1997); ** derived (Drasch et al., 2001).

examinations done (Ewers et al., 1999; Human Biomonitoring Commission of the Federal Environmental Agency, 1999).

For the hair threshold limits, the US EPA benchmark was used for HBM I ($\geq 1 \mu g$ Hg/g hair) (U.S. Environmental Protection Agency, 1997). A second threshold limit ($\geq 5 \mu g$ Hg/g hair) was derived in analogy to the Human Biomonitoring Commission of the Federal Environmental Agency (1999) from Drasch et al. (2001).

2.5. Statistics

The study population was divided into three groups: low exposure, medium/high exposure, and controls. Statistical significance (alpha level p < 0.05) in sociodemographic differences between subgroups was tested for using the Kruskal–Wallis test because there was no normal distribution. Human biomonitoring data were not distributed normally either, but right-shifted. Therefore in addition to the arithmetic mean, which is given for comparability with other studies, the median (50th percentile) was given. Laboratory data were evaluated using tests for unpaired samples (Kruskal–Wallis and Mann–Whitney U-test). While Kruskal–Wallis tests all three subgroups together, the U-test compares the medians of both exposure groups with the control group separately. The Chi-square was used to test the independence of subgroups regarding the HBM profile. The likelihood ratio was calculated to classify test validity.

Statistics were calculated using SPSS® 16.0.

3. Results

Table 2 shows the results for the study population (divided into exposure subgroups) according to sociodemographic data and potential confounders after Kruskal-Wallis testing. The study population comprised 198 Mongolian women aged 15-35 years. The subgroups differed sociodemographically. Women in the highexposure group were significantly older than the controls (32 vs. 28 years), followed by women in the low-exposure group (25 years). Women of the control group (no exposure) had a significantly higher mean body weight (62.3 kg) vs. a total mean weight of 58.4 kg in the exposed groups. The subgroups did not differ in height. The miners worked with mercury for 1–12 years (mean of 3.88 ± 3.05 years). The three exposure groups showed significant differences regarding storage of mercury and work clothes at home. While 59.4% (n = 38)of the miners stored mercury at home, 67.2% (n = 43) of them took their work clothes home as well. By comparison, only 10.9% (n = 10) of the women in the low-exposure group who were not engaged in mining stored mercury at home. There were no significant differences in potential confounders like smoking, amalgam fillings, alcohol or eating fish.

3.1. Laboratory data

Table 3 shows the results for mercury in urine, blood and hair for each subgroup. Mercury concentrations differed significantly between exposure groups, with the greatest difference seen in urine. While the

Table 2

Sociodemographic characteristics and relevant habits by subgroups.

	Khushaat	Bornuur and Jargalant		Total	Kruskal–Wallis test
	Control group	Low exposure	Medium/high exposure		(p value)
Total number	42	92	64	198	
<i>Sociodemographic data</i> Age in years (median) Mean weight (kg) Mean height (cm)	28 62.4 (n=41) 155 (n=41)	25 55.9 (n=89) 156 (n=89)	32 59.4 (n=62) 155 (n=62)	28 58.4 (n = 192) 155 (n = 192)	<0.001 0.003 0.947
Potential confounders Smoking Drinking alcohol (\geq 1 per month) Dental amalgam fillings Eating fish \geq 1 per month Keeping mercury at home Keeping work clothes at home	0 0 2 0 0	5 8 0 5 10 2	3 11 0 1 38 43	8 19 0 8 48 45	0.825 0.112 1.000 0.467 <0.001 <0.001

non-exposed women consistently showed low urine mercury levels (median of <LOD), the mercury value rose significantly with the exposure. Higher urine mercury levels were found both in the low-exposure and medium/high-exposure groups, with medians of 2.88 μ g/l and 2.98 μ g/g creatinine (low exposure) and 4.36 μ g/l and 7.18 μ g/g creatinine (medium/high exposure). The highest uncorrect-ed urine mercury value (78.5 μ g/l) was found in the low-exposure group (Fig. 1), the highest creatinine-corrected urine mercury value (311 μ g/g creatinine) in the medium/high-exposure group (Fig. 2).

Blood analysis showed low median mercury levels for all three subgroups (<LOD, $0.2 \mu g/l$). But both exposed groups showed signifi-

Table 3

Laboratory data.

	Khushaat	Bornuur and Jargalant			
Hg urine in µg/l	Control group (n=42)	Low exposure (n=92)	Medium/high exposure (n=64)		
Mean	0.19	4 78	5.67		
Median	<i.od< td=""><td>2.88</td><td>4 36</td></i.od<>	2.88	4 36		
Minimum	<lod< td=""><td><lod< td=""><td><lod< td=""></lod<></td></lod<></td></lod<>	<lod< td=""><td><lod< td=""></lod<></td></lod<>	<lod< td=""></lod<>		
Maximum	1.24	78.5	51.5		
95th percentile	0.87	10.5	17.6		
Mann-Whitney U-test		< 0.001	< 0.001		
Kruskal–Wallis test (each exp	osed group vs. contro	l group): p<0.	.001		
	0 1	0 17 1			
Hg urine in μ g/g creatinine		(n=85)	(n=58)		
Mean	no data available	7.83	18.81		
Median	/	2.98	7.18		
Min	/	0.16	0.15		
Max	/	78.7	311		
95% percentile	/	26.09	68.76		
Kruskal–Wallis test (each exp	osed group vs. contro	l group): $p = 0$	0.008		
Hg blood in ug/l	(n = 42)	(n = 92)	(n = 64)		
11g 51000 in pg/1	((11 02)	(11 0 1)		
Mean	0.30	0.32	0.55		
Median	<lod< td=""><td>0.20</td><td><lod< td=""></lod<></td></lod<>	0.20	<lod< td=""></lod<>		
Min	<lod< td=""><td><lod< td=""><td><lod< td=""></lod<></td></lod<></td></lod<>	<lod< td=""><td><lod< td=""></lod<></td></lod<>	<lod< td=""></lod<>		
Max	3.60	7.60	9.60		
95% percentile	2.28	0.87	2.40		
Mann–Whitney U-test		0.001	0.019		
Kruskal-Wallis test (each exposed group vs. control group): $p = 0.006$					
Hg hair in µg/g	(n=35)	(n=92)	(n=64)		
Mean	0.13	0.24	0.34		
Median	0.10	0.11	0.23		
Min	0.10	0.10	0.10		
Max	0.62	1.61	2.71		
95th percentile	0.37	0.83	0.81		
Mann–Whitney U-test		0.001	< 0.001		
(Kruskal–Wallis test (each exposed group vs. control group): p<0.001					

cantly higher maximum values than the controls, and the highest blood level was seen in the group with medium/high exposure (9.60 µg/l). The low-exposure and the control group consistently showed low levels, apart from two and three outliers, respectively with mercury levels $> 1 \mu g/l$ (Fig. 3).

All subgroups showed very low mercury concentration in hair, with the highest median 0.23 μ g/g in the occupational exposure group. The maximum mercury hair levels were 0.62, 1.61 and 2.71 μ g/g for controls, low exposure and medium/high exposure, respectively. There was a clear tendency of mercury concentration in hair with rising external mercury exposure (Fig. 4).

The mercury concentrations in urine, as in blood and hair, indicate only a low background burden in the control group. The Kruskal– Wallis and the Mann–Whitney U-test confirmed significant betweensubgroup differences for internal mercury concentration in urine, blood and hair (Table 3).

3.2. Threshold levels

Table 4 shows the results of the laboratory analysis by subgroups regarding threshold limits as defined by the German Human Biomonitoring Commission. All non-exposed women (n = 42) showed mercury urine levels under HBM I. Almost 10% of the low-exposure and nearly 20% of the medium/high-exposure group exceed the HBM I threshold mercury urine concentration (creatinine-corrected 36% and 64%, respectively). Three individuals showed mercury urine levels above



Fig. 1. Box plots of mercury in urine: HBM I (7 µg/l) and HBM II (25 µg/l) reference lines.



Fig. 2. Box plots of mercury in urine (creatinine-corrected): HBM I ($5 \mu g/g \text{ crea}$) and HBM II ($20 \mu g/g \text{ crea}$) reference lines. In the control group creatinine in urine was not assessed by mistake.

the HBM II value (18 with creatinine-corrected analysis). The Chi-square test revealed a significant positive association between mercury values and exposure groups for the urine samples.

Regarding the results of mercury blood levels, the HBM I value was exceeded by one woman in the low- and one in the medium/highexposure group, but without reaching the HBM II value. The values of the control group always fell below HBM I. The results for the hair samples were comparable. One woman in the medium/high-exposure and two in the low-exposure group exceeded the HBM I value.

4. Discussion

4.1. Descriptive statistics

Women of child-bearing age were chosen as a group because of their responsibility both for themselves as well as for their (as yet unborn) children. The control group was chosen because of the very low background mercury levels in their area of residence, living at a considerable distance from the mining areas and not being associated with gold mining in any way.



Fig. 3. Box plots of mercury in blood: HBM I (5 µg/l) and HBM II (15 µg/l) reference lines.



Fig. 4. Box plots of mercury in hair: threshold value I $(1 \ \mu g/g)$ and threshold value 2 $(5 \ \mu g/g)$ reference lines.

There was a significant age difference between the subgroups. Women in the medium/high-exposure group were slightly older (mean 32 years) than those in the low- or no-exposure group (mean 25 resp. 28 years). The mean age of the women living or working in ASGM areas (mean 28 years) was the same as that of control group. The weight of non-exposed women was significantly higher than that of exposed women. This difference is probably due to the fact that mainly more experienced and hence older women work in gold extraction.

Further studies on mercury use in gold mining report that miners are in the habit of storing mercury and work clothes at home (Navch et al., 2006; Bose-O'Reilly et al., 2010a,b). This was also found in this study, and suggests a lack of awareness of mercury toxicity by the miners. Even though women with low exposure were not actively involved in gold extraction, some of them reported storing mercury and work clothes at home, which in turn could indicate family members being involved in mining activities.

Using Kruskal–Wallis test confounders like smoking and drinking were not of significance. The descriptive statistics (Table 2) also showed no burden from eating fish. This is not surprising, given that Mongolia is landlocked and has limited access to water. Also, rivers are frozen most of the year (Batima et al., 2005), and there is little fish available during the rest of the year to provide a major food source (Kachondham et al., 1992).

These differences between the subgroups are therefore both explainable and negligible. Potential confounders were controlled for and comparability of the three groups was assured.

4.2. Laboratory data and threshold values

As expected, the mercury levels in urine, blood and hair were significantly higher in the group of women directly exposed to the element (Table 3). The largest differences between the three exposure subgroups were seen for urine. Table 4 shows the laboratory data in HBM categories. While blood mercury levels reflect predominantly the acute organic and inorganic exposure, urine mercury mirrors more the chronic inorganic exposure, and hair relates mainly to chronic organic exposure (Drasch et al., 2004). Together with the comparably low total mercury concentrations in hair this indicates that the main exposure is to inorganic mercury.

The results from the representative German epidemiological study, the GerESIII are useful as low background levels (Becker et al., 2002,

Table 4

Laboratory data in Human Biomonitoring (HBM) categories.

		Khushaat	Bornur and Jargalant			
		Control group	Low exposure	Medium/high exposure		
Urine						
<hbm (<7="" i="" l)<="" td="" µg=""><td>n</td><td>42</td><td>82</td><td>52</td></hbm>	n	42	82	52		
	%	100	89.1	81.2		
HBM I-HBM II (7-<25 μg/l)	n	0	8	11		
	%	0	8.7	17.2		
>HBM II (≥25 μg/l)	n	0	2	1		
m . 1	%	0	2.2	1.6		
lotal	n	42	92	64		
Chi square: $p = 0.042$ likelihood i	%	100	100	100		
c_{11} -square. $p = 0.045$, likelihood l	dli0.	p=0.008				
Urine creatinine						
<HBM I (<5 µg/g crea)	n	/	54	21		
	%	,	63.5	36.2		
HBM I-HBM II (5-<20 µg/g crea)	n	,	23	27		
	%	,	27.1	46.6		
>HBM II ($\geq 20 \ \mu g/g \ crea$)	n	/	8	10		
	%	/	9.4	17.2		
Total	n	/	85	58		
	%	/	100	100		
Chi-square: $p = 0.006$, likelihood i	atio:	p = 0.005				
Blood		10	01	62		
<hbm (<5="" i="" i)<="" td="" μg=""><td>n</td><td>42</td><td>91</td><td>63</td></hbm>	n	42	91	63		
	%	100	98.9	98.4		
HBIVI I–HBIVI II $(5-<15 \mu\text{g/I})$	0/	0	1 1 1	16		
> HPM II (> 15 µg/l)	/0 D	0	1.1	1.0		
\geq 11BW II (\geq 15 µg/1)	%	0	0	0		
Total	70 n	42	92	64		
Total	%	100	100	100		
Chi-square: $p = 0.730$, likelihood i	atio:	p = 0.599	100	100		
$c_{11} = 0.555$						
Hair						
<1 µg/g	n	35	90	63		
	%	100	97.8	98.4		
1–<5 µg/g	n	0	2	1		
	%	0	2.2	1.6		
\geq 5 μ g/g	n	0	0	0		
	%	0	0	0		
Total	n	35	92	64		
	%	100	100	100		
Chi-square: $p = 0.679$, likelihood ratio: $p = 0.522$						

2003). The control group showed compared to GerESIII even lower background mercury levels. This might depend mainly on different fish consumption habits, with very low fish consumption in Mongolia.

The internal mercury concentrations in volunteers from other countries with ASGM activities are significantly higher. In Indonesia e.g., maximum levels of 5240 µg/l (urine), 1697 µg/g crea (urine), 429 µg/l (blood) and 792 µg/g (hair) were found (Bose-O'Reilly et al., 2010a). Levels in this study reached 78.5 µg/l in urine, 311 µg/g crea, $9.6 \,\mu\text{g/l}$ in blood and $2.71 \,\mu\text{g/g}$ in hair. These differences can be attributed to higher mining activity, the use of different mining technologies, and higher fish consumption in other countries. The results of this study cannot be directly compared to those of gold mining in coastal areas (e.g., Indonesia). In addition to exposure from mining, there is a great exposure to methyl mercury from mercury polluted fish in all these regions, which in turn increases the overall internal mercury content (Grandjean et al., 1999; Drasch et al., 2001; Cortes-Maramba et al., 2006; Li et al., 2008). However the results from this study still indicate significant mercury exposure, especially environmental exposure (low-exposure group) where levels in some individuals were on a par with occupational exposures.

The results of this paper show that women in small-scale gold mining areas are affected more by inorganic mercury and less by methyl mercury. Women with medium/high exposure show higher urine mercury values than women with low exposure. Clearly, women living in an ASGM area but not working as miners are exposed indirectly (as through soil and water) whereas women miners are exposed not only to smelting and amalgamation, but also due to their habit of keeping mercury and work clothes at home (Table 2). The highest mercury urine value was found in a woman in the low-exposure group. The creatinine-corrected mercury level was 57.7 μ g/g indicating some dehydration of the women. It is possible as well that a family member was actively involved in smelting and took mercury home. Still, this case is an outlier, as the general tendency is evident.

The results of this analysis underline the urgent requirement of mercury reducing strategies to decline the mercury exposure of involved and non-involved people in ASGM regions. Several functional strategies are already identified and tested, like the introduction of mercury-free technologies or corresponding technical trainings. Since serious health effects were observed in Mongolia and elsewhere health education campaigns are urgently needed (Bose-O'Reilly et al., 2008c). Beside the need of actions on the level of the gold miners, regional capacity-building measures and regulations on the governmental level are also supporting strategies to reduce the mercury exposure in ASGM (Spiegel and Veiga, 2005; Spiegel, 2009a,b).

Based on the United Nations Global Mercury Project, which analyzed human exposure and health, as well as environmental effects of smallscale gold mining areas in different countries, *international guidelines* on mercury management in small-scale gold mining were recently published (Spiegel and Veiga, 2010). These guidelines are developed to assist locals and stakeholders to develop mercury reducing strategies to reach the minimization of the corresponding risk.

4.2.1. Limitations of the study

Urine and hair samples from the control and the exposure groups were analyzed by different laboratories. In spite of strict adherence to quality standards, some deviation cannot be ruled out.

No multivariate analysis adjusting for confounding was performed, therefore, it cannot be excluded that the shown effects are not causal.

A further limitation could be that there might be other target groups (e.g. miners with a higher mercury burden) who were not included because they did not participate, thus yielding exposure values which were too low. Particularly high values were seen in some few individuals from the exposed groups, which would seem to suggest this.

Also, the relatively small number of cases does not allow much extrapolation. Still, despite the small group sizes, the results are significant and indicate a clear tendency.

5. Conclusions

This study proved the mercury body burden caused by occupational mercury use in artisanal gold mines in Mongolia. Aside from the mining itself, environmental exposure also poses a serious public health hazard in mining areas. Residents who do not engage in mining also show high internal mercury levels, in some cases exceeding occupational guidance values. These findings indicate the extent to which people are affected by artisanal gold mining in Mongolia. Political measures are needed to support the population groups thus affected; particularly women of child-bearing age, to prevent secondary mercury exposure of fetuses and infants.

Acknowledgements

The authors would like to thank all volunteers for participating in the study and providing samples. Thanks are also due to all the scientists and aides for data collection during the field mission, as well as the various laboratory teams. We would also like to thank Jenny Bäuml for her help with data maintenance.

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