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Occupational exposure to airborne mercury during gold mining operations near El Callao, Venezuela

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Abstract ***Objective***: The National Institute for Occupational Safety and Health (NIOSH) recently conducted a cross–sectional study during gold mining operations near El Callao, Venezuela. The purpose of the study was to assess mercury exposures and mercury-related microdamage to the kidneys. The study consisted of concurrent occupational hygiene and biological monitoring, and an examination of the processing techniques employed at the different mining facilities. Mercury was used in these facilities to remove gold by forming a mercury-gold amalgam. The gold was purified either by heating the amalgam in the open with a propane torch or by using a small retort. ***Methods***: Thirty-eight workers participated in this study. Some participants were employed by a large mining company, while others were considered "informal miners" (self-employed). Mercury exposure was monitored by sampling air from the workers' breathing zones. These full-shift air samples were used to calculate time-weighted average (TWA) mercury exposure concentrations. A questionnaire was administered and a spot urine sample was collected. Each urine sample was analyzed for mercury, creatinine, and *N*-acetyl-ß-D-glucosaminidase (NAG). ***Results***: The range for the 8-h TWA airborne mercury exposure concentrations was 0.1 to 6,315 μg/m^3, with a mean of 183 μg/m^3. Twenty percent of the TWA airborne mercury exposure measurements were above the NIOSH recommended exposure limit (REL) of 50 μg/m^3, and 26% exceeded the American Conference of Governmental Industrial Hygienists (ACGIH) threshold limit value (TLV) of 25 μg/m^3. The mean urine mercury concentration was 101 μg/g creatinine (μg/g-Cr), and the data ranged from 2.5 to 912 μg/g-Cr. Forty–two percent of the study participants had urine mercury concentrations that exceeded the ACGIH biological exposure index (BEI) of 35 μg/g-Cr. Urinary NAG excretion is considered a biological marker of preclinical, nonspecific microdamage to the kidney's proximal tubule cells. The mean urine NAG concentration was 3.6 International Units/g-Cr (IU/g-Cr) with a range of 0.5 to 11.5 IU/g-Cr. Three workers had urine NAG levels in excess of the reference values. Correlation analyses found statistically significant correlations between airborne mercury exposure and urine mercury level ($P = 0.01$), and between urine mercury level and urine NAG excretion ($P = 0.01$). In addition, the airborne mercury exposure data and urine mercury data were segregated by job tasks. A Wilcoxon rank sum test revealed significant correlations between tasks and mercury exposure ($P = 0.03$), and between tasks and urine mercury level ($P = 0.02$). ***Conclusions***: The tasks with the highest mean airborne mercury exposures were "burning the mercury-gold amalgam" and "gold refining/smelting". Recommendations were provided for improving the retort design to better contain mercury, for ventilation in the gold shops, and for medical surveillance and educational programs.

Key words Gold mining · Mercury · Biological monitoring · NAG · Occupational exposure

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Introduction

Over two decades ago, the South American countries of Venezuela, Ecuador, Peru, Columbia, and the Amazon region of Brazil saw a large increase in gold mining activities due to the rising price of gold [9, 16, 27]. Recent attention has been focused on the problem of occupational and environmental mercury contamination as it relates to the gold mining and refining industries in these countries [14, 19, 20].

A series of case studies are being conducted by the National Institute for Occupational Safety and Health (NIOSH) at gold and silver mines to characterize workers' exposures to mercury during refining operations. NIOSH recently conducted an occupational health study to determine if overexposure to mercury existed at selected mining operations in the gold mining region of Bolivar State, Venezuela. The goals of this study were to determine the extent of mercury exposures in the mining community of El Callao, and to identify exposure sources in work areas.

Precious metals, such as gold, are usually found in two types of deposits, lode or alluvial. In a lode deposit, the gold is embedded in the host rock. Since the gold is an integral part of the host rock, the ore must be crushed and processed. Alluvial deposits, also referred to as placer deposits, are formed when erosion breaks up a lode deposit over time, and deposits the gold as sediments with sand and gravel. Alluvial gold may be found in the beds of streams or lakes or in sediments that have been buried over time [9]. The gold mined in the El Callao area is primarily from lode deposits and this study focused on those types of operations.

Both large and small lode mining operations are found around El Callao, but the majority are small-scale informal mines. The small-scale mines employ from one to ten people and only the larger of these have their own milling facility at the mine site. Most small-scale miners depend on cooperative mills to process their ore. At the small mills the ore was crushed and run through a water concentrator to remove the majority of the waste rock. The concentrates were then de-greased and panned with mercury to capture the gold in a mercury-gold amalgam. Because of the inefficiencies of this process, a significant amount of mercury remained with the waste material, called tailings, resulting in mercury finding its way into the groundwater and rivers. To purify the gold, the mercury-gold amalgam was heated, resulting in vaporization of the mercury. The gold was further refined at the gold shops. Few, if any, preventative measures were taken to protect the mill or gold shop workers from hazardous mercury vapors. Mercury toxicity in gold miners, mill operators, refiners, and individuals living near these processes has been documented [5].

The two large underground mines near El Callao employed approximately 1,000 people. These large-scale mines used modern mining methods and cyanide leaching to extract the gold, therefore mercury was not generally used during processing. The large mine included as a part of this study had mercury present in the plant because the company purchased the tailings from small, nearby mills. The tailings still contained about 10 g of gold per tonne because of the inefficiencies associated with mercury separation methods at the small mills. The run-of-mine ore, which averaged 4 g of gold per tonne, was blended with the tailings to achieve an overall mill feed of 5 g of gold per tonne.

The health effects related to exposure to elemental mercury through inhalation are well documented [2, 3, 8, 10, 35]. The target organs for mercury toxicity are the central nervous system (CNS) and the kidneys. The kidneys are sensitive target organs because a large proportion of the absorbed mercury dose accumulates in the renal cortex. Chronic exposure is characterized by proteinuria (albumin, ß-2-microglobulin, retinol-binding protein, etc.) and enzymuria (ß-galactosidase, *N*-acetyl-ß-D-glucosaminidase (NAG), ß-glucuronidase, etc.) [4, 6, 11, 13, 22, 28, 31, 32, 34]. These manifestations can diminish the ability of proximal tubule segments to reabsorb water, proteins, and other substances, thus affecting the kidneys' ability to maintain volume and composition of body fluids within normal limits.

NAG is a large hydrolytic enzyme, and is abundant in the lysosomes of proximal tubule cells [32]. An increase in urinary NAG levels is considered an indicator of nonspecific microdamage to proximal tubule cells, that is, cell breakdown, necrosis, or increased cellular turnover [18, 24]. In fact, urinary NAG excretion is clinically used as a biological marker of disease-related renal damage in cases of diabetes mellitus, hypertension, and rheumatoid arthritis [29, 30]. NAG has also been used in cross-sectional studies as a nonspecific indicator of cadmium- and mercury-related microdamage to the kidneys' proximal tubules [4, 11, 17, 23, 28, 32].

Methods

This study was conducted in El Callao, Venezuela, a small mining area of Bolivar State, where extensive gold extraction is carried out. El Callao is a small town of approximately 5,000 people with the majority of households relying on income from mining activities. The mining-related jobs were easily classified into three employment categories: large mining company, small mining operations (informal miners), and those working in gold shops where jewelry was manufactured. The intent was to survey workers from all three employment categories. Within the employment categories there were workers who performed different tasks. In the case of the informal miner, tasks that an individual performed could change daily. In order to determine if exposures differed according to the task being performed each day, specific tasks were identified. The informal miners carried out the tasks of mining, milling, burning, and smelting. Mining consisted of excavating the ore and transporting it to the mills. Milling included operating the ore crusher and water concentrator, and panning the concentrates with mercury to form the amalgam. Burning referred to burning the amalgam to vaporize the mercury to separate the gold, and the smelting task was performed to refine the gold further. The individuals employed at the large mining company primarily worked in the plant, operating equipment, managing the cyanide leach process, handling materials, and conducting maintenance and repairs. In one case, a worker was involved in milling operations, which entailed crushing the ore.

Personnel from Fundacite Guayana (the Foundation for the Development of Science and Technology in Bolivar State) traveled to El Callao prior to the arrival of the study team to solicit participation by holding a town hall meeting with the residents of El Callao, to explain the purpose of the study. The only criterion for participation in the study was the possibility of occupational exposure to mercury. Prior to their participation in the study, all individuals were asked to read and sign an informed consent form. An attempt was made to pick a cross-section of the volunteers to obtain a representative sample of the three employment categories. The study design was cross-sectional, consisting of concurrent airborne mercury exposure assessments and biological monitoring of the same cohort.

The data from the exposure assessment study are presented in Table 1. Forty workers completed questionnaires on the first day of the study. After completing the questionnaire, two people chose not to continue in the study. Of these 38 workers, 34 participated in the airborne mercury exposure monitoring on day 2, with five invalid samples, and 35 on day 3, with three invalid samples. A sample was considered invalid if the personal sampling pump was not functioning at the end of the work shift. On the morning of the third day of the study, 33 workers supplied a urine sample. Four workers from the large mining company and one informal miner chose not to provide a urine sample.

Of the workers studied, 37 (97%) were male and 1 (3%) was female. The mean age was 35.7 years, with a range of 20 to 61 years. The duration of employment in their current jobs ranged from 0.4 to 16 years, with a mean of 5.6 years.

Exposure assessment study

The exposure assessment study consisted of two consecutive days of airborne mercury exposure air monitoring. Workers' inhaled mercury concentrations were determined by sampling the air from the workers' breathing zones. These full-shift samples were used to determine the workers' time-weighted average (TWA) mercury exposure concentrations.

The air sampling and analysis method used to determine mercury exposures was NIOSH Method 6009 [26]. This method entails drawing air through a solid sorbent tube containing 200 mg of hopcalite at a nominal flow rate of 200 cm^3/min. The samples were prepared by adding 2.5 ml each of concentrated nitric acid and hydrochloric acid to a vial containing the hopcalite granules and glass wool plugs. After this preparation, the acid extracts were diluted to volume and analyzed using a Perkin-Elmer Model 3100 Flow Injection cold vapor atomic absorption spectrometer.

The limit of detection (LOD) and limit of quantification (LOQ) for NIOSH Method 6009 were 0.01 and 0.034 µg mercury per sample (µg/sample), respectively. The minimum detectable concentration (MDC) and minimum quantifiable concentration

Table 1 Summary of all mercury exposure data segregated by employment category (*Cr* creatinine, *IU* International Units)

Employment category	Airborne mercury (µg/m^3)		Urine mercury (µg/g-Cr)	Urine NAG (IU/g-Cr)	Task	
	Day 2	Day 3			Day 2	Day 3
Informal miner	10.6	62.0	191	4.2	Milling	Burning
	4.4	153	33.9	1.8	Milling	Milling
	1.5	0.8	2.5	3.0	Mining	Mining
	641	1040	682	7.3	Smelting	Smelting
	6.6	Invalid	110	2.6	Smelting	Smelting
	14.1	7.9	89.9	2.1	Smelting	Smelting
	0.9	28.0	63.2	2.8	Milling	Burning
	518	1518	14.8	5.8	Smelting	Smelting
	1.9	7.8	24.6	3.1	Smelting	Smelting
	0.5	4.7	15.0	2.6	Smelting	Smelting
	0.5	0.5	26.9	2.0	Milling	Milling
	6315	2.0	39.3	3.4	Burning	Burning
	16.1	18.1	131	7.9	Milling	Milling
	0.3	0.6	3.7	1.0	Milling	Milling
	0.3	0.3	4.9	3.6	Milling	Milling
	29.5	20.3	75.9	2.0	Smelting	Smelting
	3.0	4.3	45.3	4.5	Milling	Milling
	200	30.2	912	2.5	Burning	Burning
	0.8		27.7	9.8	Milling	
		4.4	447	11.5		Burning
			159	2.4		
	68.9				Smelting	
Jeweler	84.3	2.0	9.8	3.7	Smelting	Smelting
	10.3	11.4	127	7.1	Smelting	Smelting
	0.2	0.1	2.5	2.2	Smelting	Smelting
	0.4	0.6	16.9	5.4	Smelting	Smelting
Large mining company	17.0	52.7	9.3	1.1	Plant work	Plant work
	Invalid	1.9	49.9	1.0	Plant work	Plant work
	Invalid	0.5	2.5	5.8	Plant work	Plant work
	6.8	Invalid	12.6	0.7	Plant work	Plant work
	Invalid	29.6	2.5	0.5	Plant work	Plant work
	Invalid	202	2.5	1.2	Plant work	Plant work
	Invalid	Invalid	13.4	2.6	Plant work	Plant work
		0.5	2.5	2.1	Plant work	Plant work
	0.5	6.5			Plant work	Plant work
	0.4	0.5			Milling	
	0.5	2.2			Plant work	Plant work
		3.0			Plant work	Plant work

(MQC) are measures of the sensitivity of the air sampling and analysis protocol, that is, the lowest mercury exposure concentration that could be detected and quantified in this study. In determining the MDC and MQC for these data, a sampling period of 8 h (480 min) and a flow rate of 200 cm^3/min were used to calculate an air sample volume of 96 l (0.096 m^3). This results in an MDC of 0.1 μg/m^3 and an MQC of 0.35 μg/m^3.

NIOSH, the World Health Organization (WHO), and the American Conference of Governmental Industrial Hygienists (ACGIH) all have occupational exposure criteria for workers with potential inhalation exposure to mercury. The NIOSH recommended exposure limit (REL) for mercury is 50 μg/m^3 as a TWA exposure for up to 10 h/day, 40 h/week [25]. In 1980, the WHO recommended an 8-h TWA mercury exposure standard of 25 μg/m^3 [35]. Finally, in 1994, the ACGIH lowered the threshold limit value (TLV) for mercury to 25 μg/m^3 as an 8-h TWA exposure, 40 h/week [1].

Biological monitoring

The biological monitoring consisted of the administration of a questionnaire and the collection of a spot urine sample. Spot urine samples were collected from 33 individuals. Urine sampling was conducted at the medical clinic in El Callao. Approximately 50 ml of urine were obtained from each worker in sterile, mercury-free containers. The samples were kept at 4 °C until required for analysis. Each sample was analyzed for mercury, creatinine, and NAG.

Because urinary water output varies between urinations, a dilution correction is used to normalize the volume portion of the urine mercury concentration. The preferred method for dilution correction is to express the urine mercury concentration in micrograms of mercury per gram of creatinine (μg/g-Cr).

The urine was analyzed for mercury by placing a 25-ml aliquot of the urine in an acid-washed glass bottle. The pH of the urine sample was adjusted to below 2. During the analysis, the mercury in the urine sample was reduced using tin chloride and was determined by cold vapor atomic absorption spectroscopy [7, 33]. The LOD for this method was 5 μg/l. Both the WHO and the ACGIH have established evaluation criteria for urine mercury concentrations. The WHO recommends a urine mercury limit of 50 μg/g-Cr [35], and the ACGIH biological exposure index (BEI) for urine mercury is 35 μg/g-Cr [1]. The average mercury concentration for people without occupational exposure to mercury is less than 5 μg/g-Cr [3].

Creatinine concentration was determined using a Jaffe rate method [15]. Creatinine in the urine sample combines with picric acid to produce a creatinine-picrate complex. This complex has a red color, and the development and intensity of this color are directly proportional to the mass of creatinine in the sample. The samples were analyzed with a bichromatic detection system consisting of a light source, beam splitter, 520-nm sample detector, 560-nm reference detector, and two photo detectors. Normal creatinine values are between 0.5 and 3.0 g/l [1].

NAG was analyzed in urine using the Kamiya Biomedical K-Assay Kit KAE-001. The basis for the analysis was the measurement of NAG activity by the ultraviolet (UV) rate method [12]. In this method, NAG catalyzes the hydrolysis of the substrate (6-methyl-2-pyridyl-*N*-acetyl-1-thio-ß-D-glucosaminide), liberating 6-methyl-2-pyridinethiol. The concentration of the liberated substance is measured as a function of time to obtain the rate of reaction (rate of increase in UV absorbence at 340 nm), which is converted to NAG activity. The reported urine NAG concentration range for this method was 0.04 to 300 IU/l. Urinary NAG levels were considered abnormal when they exceeded 9.3 IU/g-Cr in women and 7.9 IU/g-Cr in men [21].

Statistical analyses

Statistical analyses were performed using Statistical Analysis System Version 6.12. Measures of airborne mercury exposure, urine mercury, and urine NAG were log-transformed for analyses. Pearson's correlation coefficients were used to measure the linear association between the log-transformed airborne mercury exposure measurements, urine mercury levels, and urine NAG levels. One-way analysis of variance was used to test whether the log-transformed airborne mercury exposure measurements and urine mercury levels differed by job task. The results of a statistical test were considered statistically significant if $P \leq 0.05$.

Results

Data from the airborne mercury exposure assessments are presented in Table 2. A total of 61 personal breathing zone samples were collected over two consecutive days. The mean for the 61 samples was 183 μg/m^3 with a range of 0.1 to 6,315 μg/m^3. Twenty percent (12 of 61) of the airborne mercury exposure concentrations were over the NIOSH REL of 50 μg/m^3 and 26% (16 of 61) were above the ACGIH TLV of 25 μg/m^3. Elevated airborne mercury exposure concentrations were measured in all three employment categories.

Urine mercury and urine NAG concentrations are presented in Table 3. The mean urine mercury concentration for the 33 workers was 101 μg/g-Cr, with a range of 2.5 to 912 μg/g-Cr. Forty-two percent (14 of 33) of the workers had urine mercury concentrations that exceeded the ACGIH BEI of 35 μg/g-Cr. Only three workers had urine NAG levels in excess of the reference values.

The airborne mercury exposure measurements (using the mean of the log-transformed values for the 2 days) were significantly correlated with the log-transformed urine mercury levels ($r = 0.44$, $P = 0.01$) (Fig. 1), but were not significantly correlated with the log-transformed urine NAG concentrations ($r = 0.02$, $P = 0.92$).

Table 2 Airborne mercury exposure data (μg/m^3) (*SD* standard deviation, *NIOSH* National Institute for Occupational Safety and Health, *REL* recommended exposure limit, *ACGIH* American Conference of Governmental Industrial Hygienists, *TLV* threshold limit value)

	No.	Mean	SD	Range	> NIOSH REL[a]	> ACGIH TLV[b]
All exposure data:	61	183	837	0.1–6,315	12/61 (20%)	16/61 (26%)
Day 2	29	274	1,170	0.2–6,315	6/29 (21%)	7/29 (24%)
Day 3	32	101	318	0.1–1,518	6/32 (19%)	9/32 (28%)
Large mining company	15	21.6	52.0	0.4–202	2/15 (13%)	3/15 (20%)
Informal miners	38	283	1,050	0.3–6,315	9/38 (24%)	12/38 (32%)
Jewelers	8	13.6	28.9	0.1–84.3	1/8 (13%)	1/8 (13%)

[a] NIOSH REL = 50 μg/m^3
[b] ACGIH TLV = 25 μg/m^3

Table 3 Urine mercury and urine *N*-acetyl-ß-D-glucosaminidase (*NAG*) data (*SD* standard deviation, *ACGIH* American Conference of Governmental Industrial Hygienists, *BEI* biological exposure index, *WHO* World Health Organization, *Cr* creatinine, *IU* International Units)

	No.	Mean	SD	Range	> ACGIH BEI[a]	> WHO[b]	> NAG ref[c]
Urine mercury (µg/g-Cr)	33	101	201	2.5–912	14/33 (42%)	11/33 (33%)	
Large mining company	8	11.9	16.1	2.5–49.9	1/8 (13%)	0	
Informal miners	21	148	240	2.5–912	12/21 (57%)	10/21 (48%)	
Jewelers	4	39.1	58.9	2.5–127	1/4 (25%)	1/4 (25%)	
Urine NAG (IU/g-Cr)	33	3.6	2.7	0.5–11.5			3/33 (9%)
Large mining company	8	1.9	1.7	0.48–5.8			0
Informal miners	21	4.1	2.8	1.0–11.5			3/33 (9%)
Jewelers	4	4.6	2.2	2.2–7.13			0

[a] ACGIH BEI = 35 µg/g-Cr
[b] WHO limit = 50 µg/g-Cr
[c] NAG reference value = 7.9 IU/g-Cr for men and 9.3 IU/g-Cr for women

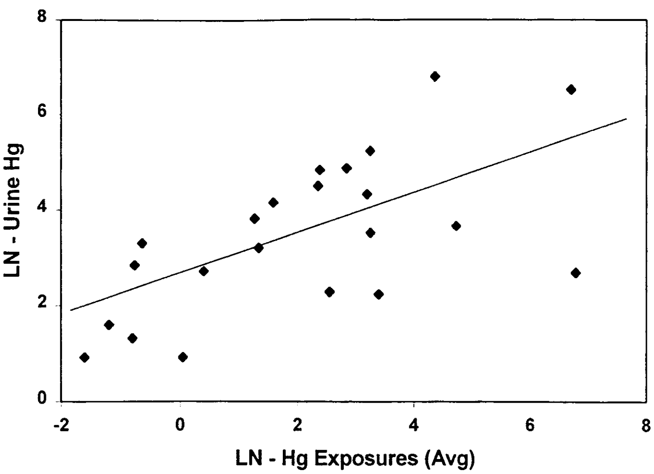

Fig. 1 Correlation between log-transformed airborne mercury exposure values and the log-transformed urine mercury values ($P = 0.01$, $r = 0.44$)

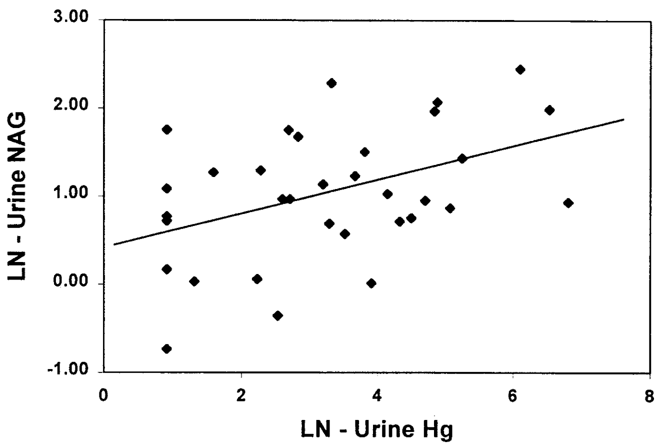

Fig. 2 Correlation between log-transformed urine mercury levels and the log-transformed urine *N*-acetyl-ß-D-glucosaminidase (*NAG*) values ($P = 0.01$, $r = 0.43$)

There was also a statistically significant correlation between the log-transformed urine mercury levels and the log-transformed urine NAG concentrations ($r = 0.43$, $P = 0.01$) (Fig. 2).

The workers were asked to report the tasks they performed each day to ascertain whether airborne mercury exposure was related to a specific job task (Table 4). The tasks reported by the workers varied, depending upon their place of employment. The large mining company utilized cyanide leaching to remove the gold, so the task these participants reported most frequently was plant working. The informal miners were either self-employed or worked for a small mining operation. The tasks reported by these informal miners were milling, mining, smelting, and burning. The task reported by the participants who worked for a gold shop or jewelry manufacturer was smelting. The workers who performed the task of burning the mercury-gold amalgam had the highest mean mercury exposure concentrations (mean of 949 µg/m^3 followed by those workers who smelted or further refined the gold (mean of 181 µg/m^3). The mean airborne mercury exposure concentration for workers who performed the tasks of plant working, milling, and mining were below the RELs.

A similar comparison was done to determine whether urine mercury concentrations were related to specific job tasks. Table 4 also shows a comparison of urine mercury concentrations by job tasks. In order to do this comparison, we used only workers who did the same task on both days. Again, the workers who performed the task of burning the mercury-gold amalgam had the highest mean urine mercury concentrations (mean of 475 µg/g-Cr), followed by the workers who smelted the gold (mean of 106 µg/g-Cr).

The Wilcoxon rank sum test was used to determine the association between airborne mercury exposure data and job tasks, and urine mercury concentrations and job tasks. The results from this test yielded evidence that the job tasks burning, smelting, plant working, milling, and mining did not have the same average airborne mercury exposure level ($P = 0.03$). In addition, the average urine mercury concentrations for the job tasks were found to differ significantly ($P = 0.02$).

Table 4 Comparison of mercury exposures by job tasks

Task	Airborne mercury exposures (μg/m^3) (P = 0.03)			Urine mercury levels[a] (μg/g-Cr) (P = 0.02)		
	No.	Mean	Range	No.	Mean	Range
Burn	7	949	2.0–6,315	2	475	39.2–912
Smelt	22	181	0.1–1,518	11	106	2.5–682
Plant	13	24.9	0.5–202	6	41	3.7–131
Mill	16	13.4	0.3–153	8	12	2.5–50
Mine	2	1.1	0.8–1.47	1	2.5	2.5

[a] Workers doing the same job task both days

Discussion

This study was not intended to characterize mercury exposure risk in the Venezuelan gold mining population, but was performed to discover if potential mercury exposure hazards exist among the miners in El Callao. More in-depth exposure assessment and epidemiological studies would be needed to characterize the mercury exposure risk in this population. Nonetheless, our data indicate that there is a potential for overexposure to mercury for Venezuelan gold miners, in some cases huge overexposures, and that the exposures in our study cohort (albeit small) were at levels that could produce mercury-related symptoms and health effects. The methodology employed in this study was successfully used to characterize a wide range of exposures, and could be utilized in another population or town with varying degrees of exposure to mercury. If a larger population-based study were to be conducted, the results of this study would be useful in determining the appropriate methodology to be used.

The majority of workers who are overexposed work as informal miners. The elevated exposures within this group most likely resulted from the way they process the mercury-gold amalgam. Forty-two percent (14 of 33) of the workers had urine mercury concentrations above the ACGIH BEI. Twelve of those workers were employed as informal miners, one as a jeweler, and one at the large mining company. The individuals who performed the task of burning the mercury-gold amalgam, and those who further refined the gold receive the highest exposures. Three of the 33 workers had urine NAG concentrations above the reference values; all three worked as informal miners.

Some of the smaller milling and processing facilities used a retort to separate the gold from the mercury of the amalgam. Heat was applied to the amalgam, and the mercury was distilled off as a vapor. Gold remained in the retort while the mercury vapor was released through a ventilation pipe. At one site, no attempt was made to collect the mercury vapors. This process could easily be modified by directing the outlet of the ventilation pipe into a container of water. The mercury vapors would condense in the water and could then be re-used. Occupational exposure could be reduced significantly if all the informal miners would utilize simple retorts. These retorts can be built with standard plumbing water pipes found at local hardware stores. Another option would be for the informal miner to take their gravity concentrates to the "Processing Centers" or "Amalgamation Centers" that have been established within areas of Venezuela. The amalgamation centers use trained operators to run the retorts and melting furnaces. Fume hoods with activated charcoal filters are used to capture any mercury vapors so that emissions are minimized.

Our study found that individuals employed as goldsmiths or jewelers also had a high likelihood of overexposure to mercury. The gold shop workers buy the gold nuggets and then refine them by melting the gold, which vaporizes any residual mercury. Generally, the workers smelted the gold in enclosed areas without adequate ventilation. Despite the small amount of mercury reburned in the shops when compared with total mercury used in the whole process, reburning presents a potential risk to a worker as well as to those living in the vicinity of the gold shops. Fumes released in gold shops could be captured using a simple fume hood with an activated charcoal filter. The use of simple technology could result in a significant reduction of mercury emissions and poisoning.

A medical surveillance program, including biological monitoring, would be very beneficial to those workers potentially exposed to mercury. Medical monitoring involves periodically evaluating exposed workers to ensure they are not experiencing adverse effects. An evaluation should include a complete medical history, a signs and symptoms questionnaire (with emphasis on the target organs for chronic exposure), a baseline handwriting sample, and a urinalysis. At least twice a year, chronically exposed workers should have their urine analyzed for mercury to determine how much mercury has been absorbed into their body. Results from these monitoring programs would help convince the miners that they are being affected by mercury vapors, and thus they may seek to adopt safer processing methods.

Perhaps the real benefit of this study was notifying the participants of their urine mercury results. The majority of the participants had never had their urine tested for mercury and therefore, had no idea if the processing techniques currently being used were causing overexposures. Education of the miners is a prerequisite for long-term solutions. The miners need to understand the health hazards associated with mercury exposure, and the environmental problems associated with mercury pollution, so that they will adopt safer methods. Some low-cost alternatives that could be done at the community level are workshops within the mining communities, distribution of educational pamphlets or brochures, establishment of mining associations or cooperatives, and incorporation of environmental and health issues associated with mercury into the formal school system. National and international organizations should join local institutions and authorities in developing proper programs to combat this serious health problem.

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References

1. ACGIH (1997) 1997 Threshold limit values for chemical substances and physical agents and biological exposure indices. American Conference of Governmental Industrial Hygienists, Cincinnati, Ohio
2. Agocs M, Thomas C, et al (1992) Mercury toxicity. Am Fam Physician 46: 1731–1741
3. ATSDR (1994) Toxicological profile for mercury. U.S. Dept. of Health and Human Services, Agency for Toxic Substances and Disease Registry, DHHS (ATSDR) Publication No. TP-93/10, Atlanta, Ga
4. Barregard L, Hultberg B, et al (1988) Enzymuria in workers exposed to inorganic mercury. Int Arch Occup Environ Health 61: 65–69
5. Branches FJP, Erickson TB, Aks SE, et al (1993) The price of gold: mercury exposure in the Amazonian rain forest. J Toxicol Clin Toxicol 31: 295–306
6. Buchet J, Roels H, et al (1980) Assessments of renal function of workers exposed to inorganic lead, cadmium, and mercury. J Occup Med 22: 741–750
7. Carson BL, Ellis HV, McCann JL (1987) Toxicology and biological monitoring of metals in humans. Lewis, Mich, USA
8. Clayton G, Clayton F (eds) (1981) Patty's industrial hygiene and toxicology. Wiley, New York
9. Cleary D (1990) Anatomy of the Amazon gold rush. 1st edn, University of Iowa Press, Iowa City, Iowa, USA
10. Doull J, Klaassen CD, Amdur MO (eds) (1991) Casarett and Doull's toxicology: the basic science of poisons, 4th edn. Macmillan, New York
11. Ehrenberg R, Vogt R, et al (1991) Effects of elemental mercury exposure at a thermometer plant. Am J Ind Med 19: 495–507
12. Furuhata N, Shiba K, Shiina S (1992) Basic study on *N*-acetyl-ß-D-glucosaminidase (NAG) measuring reagent using new substrate 6-methyl-2-pyridyl-*N*-acetyl-1-thio-ß-D-glucosaminide (MPT-NAG). J Clin Lab Instruments Reagents 15: 839–844
13. Himeno S, Watanabe C, et al (1986) Urinary biochemical changes in workers exposed to mercury vapor. Ind Health 24: 151–155
14. Homewood B (1991) Mercury poisoning confirmed among Amazon villagers. New Scientist 9: 18
15. Jaffe MZ (1886) Über den Niederschlag welchen Pikrinsäure in normalem Harn erzeugt und über eine neue Reaction des Kreatinins. Physiol Chem 10: 391–400
16. Lacerda LD, Pfeiffer WC, Marins RV, Rodrigues S, Souza CMM, Bastos WR (1991) Mercury dispersal in water, sediments and aquatic biota of a gold mining tailing deposit drainage in Pocone, Brazil. Water Air Soil Pollut 55: 283–294
17. Langworth S, Elinder C, et al (1992) Renal and immunological effects of occupational exposure to inorganic mercury. Br J Ind Med 49: 394–401
18. Lauwerys R, Bernard A (1989) Preclinical detection of nephrotoxicity: description of the tests and appraisal of their health significance. Toxicol Lett 46: 13–29
19. Malm O, Pfeiffer WC, Sousa CMM, Reuther R (1990) Mercury pollution due to gold mining in the Madeira river basin. Braz Ambio 19: 11–15
20. Martinelli LA, Ferreira JR, Forsberg BR, Victoria RL (1988) Mercury contamination in the Amazon: a gold rush consequence. Ambio 17: 252–254
21. Maruhn D, Fuchs I, et al (1976) Normal limits of urinary excretion of eleven enzymes. Clin Chem 22: 1567–1574
22. Meyer R, Fischbein A, et al (1984) Increased urinary enzyme excretion in workers exposed to nephrotoxic chemicals. Am J Med 76: 989–998
23. Mueller PW, Jay-Smith S, et al (1989) Chronic renal tubular effects in relation to urine cadmium levels. Nephron 52: 45–54
24. Mutti A (1989) Detection of renal diseases in humans: developing markers and methods. Toxicol Lett 46: 177–191
25. NIOSH (1992) Recommendations for occupational safety and health, compendium of policy documents and statements. US Dept. of Health and Human Services, Public Health Service, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 92–100, Cincinnati, Ohio
26. NIOSH (1994) NIOSH manual of analytical methods, 4th edn, US Dept. of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 94–113, Cincinnati, Ohio
27. Pfeiffer WC (1989) Mercury concentrations in inland waters of gold mining areas in Rondonia, Brazil. Sci Total Environ 87/88: 233–240
28. Piikivi L, Ruokonen A (1989) Renal function and long-term low mercury vapor exposure. Arch Environ Health 44: 146–149
29. Price R (1982) Urinary enzymes, nephrotoxicity and renal disease. Toxicology 23: 99–134
30. Rocskay A, Robins T (1994) Assessment of a screening protocol for occupational renal disease. J Occup Med 36: 1100–1109
31. Roels H, Gennart J, et al (1985) Surveillance of workers exposed to mercury vapor: validation of a previously proposed biological threshold limit value for mercury concentration in urine. Am J Ind Med 7: 45–71
32. Rosenman K, Valciukas J, et al (1986) Sensitive indicators of inorganic mercury toxicity. Arch Environ Health 41: 208–215
33. Schaller KH, Angerer J (eds) (1985) Mercury. In: Analyses of hazardous substances in biological materials, vol 2. The German Science Foundation, pp 195–211
34. Stonard M, Chater B, et al (1983) An evaluation of renal function in workers exposed to mercury vapour. Int Arch Occup Environ Health 52: 177–189
35. WHO (1980) Recommended health-based limits in occupational exposure to heavy metals. Technical Report, Series 647, World Health Organization, Geneva, Switzerland